2025 TROPICAL HEALTH ORIENTATION MANUAL

for health practitioners in Northern Australia (3rd edition)



Tropical Health Orientation Manual

FOR HEALTH PRACTITIONERS IN NORTHERN AUSTRALIA

An updated and expanded edition of *Tropical Health in the Top End (2003)*

2025

MENZIES SCHOOL OF HEALTH RESEARCH 2025

Copyright© Flinders University, Tropical Health Orientation Manual (2020)

The intellectual property rights contained within THOM2020 are owned by Flinders University and have been licensed to Menzies School of Health Research. Modification or alteration is strictly prohibited without approval.

This Manual was developed by the Menzies School of Health Research (Menzies) and Northern Territory Primary Health Network (NT PHN) based on the THOM2020. It is an introduction to tropical health in Northern Australia and not a source of management guidelines for the treatment of individual patients. Neither Menzies, NT PHN or Flinders University nor any individual contributor to this guide shall be liable to users of the guide or other persons for any loss or harm arising from errors or omissions that this revised guide may contain.

Images provided by Professor Bart Currie (Menzies School of Health Research) are available for teaching purposes, with acknowledgment.

Copying and reproduction

The text in this work is licensed under the Creative Commons Attribution-NonCommercial-ShareAlike 3.0 Unported Licence. To view a copy of this licence, see https://www.creativecommons.org/licenses/by-nc-sa/3.0 or write to Creative Commons, PO box 1866, Mountain view, CA 94042, USA. You may only copy, distribute, display or perform the work (and make derivative works) subject to the terms of the Creative Commons licence that applies to it (which, among other things, preserves the moral rights of the Remote Primary Health Care Manuals joint venture and imposes obligations of attribution upon users).

You may not make use of this work for commercial purposes.
For permissions beyond the scope of this licence, contact the publisher.

This licence does not apply to the photographs in the work.

ISBN: 978-0-646-98127-7

An updated and expanded edition of *Tropical Health in the Top End 2003*
(ISBN 1877021024)

Published by Menzies School of Health Research
PO Box 41096, Casuarina NT 0811 Australia

Website: www.menzies.edu.au

Suggested citation
2025 Tropical Health Orientation Manual. Menzies School of Health Research, 2025.

Legal Disclaimer
While the Australian Government helped fund this document, it has not reviewed the content and is not responsible for any injury, loss or damage however arising from the use of or reliance on the information provided herein.

Menzies School of Health Research does not accept any contractual, tortious or other liability whatsoever in respect of the contents or consequences arising from their use. While all advice and recommendations are made in good faith, Menzies School of Health Research does not accept legal liability or responsibility for such advice or Recommendations.



Cover illustration by Chidanpee (Norma Benger)
Reproduced under licence.

STOP THE CURLEW CRYING

The curlew story is one of prevention. When our children play around the billabong at dusk, the mothers must go down and rub out all the children's footprints. This will stop the curlew crying. If the curlew cries this can bring bad luck or make you sick. The curlew cries because she thinks that the footprints are of her lost children who went missing while playing around the billabong, a long time ago in the dreamtime...

Introduction

The Tropical Health Orientation Manual (THOM) provides an introductory overview of clinically important conditions encountered by health staff in tropical Australia. THOM aims to complement existing management guidelines and links to resources for further guidance.

Tropical Health in the Top End was developed and first published by the Northern Territory Top End Division of General Practice in 2003 in response to a need identified by general practitioners working in rural and remote health. The second edition was published in 2020 by the Centre for Remote Health. It updated existing topics and in response to requests from clinicians it was broadened to be helpful for other northern Australian jurisdictions, noting the similarities across the tropical regions. The second edition of THOM was aligned with and complemented the Remote Primary Health Care Manuals (RPHCM), which continue to be developed by health practitioners, for health practitioners working in rural and remote locations.

This third edition of the THOM is supported by the Northern Territory Primary Health Network (NT PHN) and published by the Menzies School of Health Research. As with the prior editions, there are many clinical colleagues who have generously given their own time to update topics and provide new content. Emphasis has been placed on adding direct links to relevant guidelines and resources, especially CARPA, Therapeutic Guidelines and NT HealthPathways. While the added links to resources for the individual topics are often Northern Territory-focussed, the topics and content remain relevant for colleagues working elsewhere in rural and remote Australia, with the tropical focus continuing. Adding links to resources specific for Western Australia and Queensland will hopefully follow, at least for the electronic version.

I hope that THOM will be of value to health staff and students across health disciplines who are working or contemplating working in central and northern Australia. Thanks again to all those who have contributed to the THOM over the last 25 years.

Bart Currie

Menzies School of Health Research and Royal Darwin Hospital

Acknowledgements

Editor: Bart Currie

Production editor: Sara Noonan

Specialist editorial advice: Sonja Janson, Vicki Krause

A team spirit of voluntary collaboration and cooperation between many people and different health organisations has made this edition possible. Those colleagues are greatly appreciated and listed below.

2025 contributors: Brooke Ah Shay, Rodney Campain, Anne Chang, Jerry Chen, Chris Clohesy, Imogen Couchman, Roxane Craig, Lucy Crawford, Bart Currie, Jane Davies, Robert Duguid, Hayley Dyke, Dana Fitzsimmons, Howard Flavell, Robert Forbes, Catherine Gargan, Manoji Gunathilake, Subash Heraganahally, Sonja Janson, Alexandra Kay, Vicki Krause, Greta Lindenmayer, Sarah Lynar, Genevieve Martin, Catherine Marshall, Marsilla Marzukie, Melanie Matthews, Malcolm McDonald, Ella Meumann, Bianca Middleton, Nerida Moore, Stephanie Naidu, Sara Noonan, Alyssa Pidgeon, Anna Ralph, Sophia Scrimgeour, Maddison Sims, Harsimran Singh, Emma Smith, Simon Smith, David Szmulewicz, Ju Ann Tan, Sze Tay, Dev Tilakaratne, Angela Titmuss, Vicki Wade, Christine Watson, Rosalind Webby, Bernie Westley, Kristof Wing, Anna Wood, Ashish Yadav.

Acknowledgements to previous editions

2020 contributors: Wendy Page (Working Group Chair), Enzo Binotto, Rowena Boyd, Monique Chilver, Heather Cook, John Cooper, Bart Currie, Jane Davies, Ruth Derkenne, Keith Edwards, Lloyd Einsiedel, Dana Fitzsimmons, Howard Flavell, Keith Forrest, Catherine Gargan, Belinda Greenwood-Smith, Manoji Gunathilake, Rory Hannah, Kate Hardie, Hugh Heggie, Alex Hope, Brendan Johnson, John Kelly, Vicki Krause, Clinton Leahy, Rosemary Lee, Stephanie Mackie-Schneider, Catherine Marshall, Peter Markey, Libby Massey, John McBride, Ian McCrossin, Diana Mosca, Jacqueline Murdoch, Victoria Orpin, Naomi Porter, Sandeep Reddy, Lesley Scott, Jenny Shield, Rick Speare, Geoffrey Stewart, Kylie Strate, Vipin Tayal, Steven Tong, Fran Vaughan, William Walker, Susan Wearne, Rosalind Webby, Andrew Webster, Bernard Westley, Sheree Zadow.

The 2020 edition was dedicated to the memory of Meredith Arnold.

2003 contributors: Fay Johnston (Editor), Paul Armstrong, Meredith Arnold, Paul Burgess, Alan Clough, Christine Connors, Bart Currie, Keith Edwards, Nilva Egana, Dan Ewald, Dana Fitzsimmons, Chris Harrison, Paul Kelly, Vicki Krause, Sue Lenthall, Justine Mayer, Jeanette McGregor, Jacki Mein, Simon Morgan, Peter Morris, Hung The Nguyen, Patrick O'Brien, Wendy Page, Barbara Paterson, Brett Ritchie, Jan Savage, Jan Schmitzer, Steven Skov, Tania Wallace, Kate Walker, Jane Walters, Peter Whelan, Jo Wright, Annie Whybourne, Nathan Zweck.

We wish to acknowledge and honour the traditional owners of Australia and the Torres Strait Islands.

This manual was produced with funding from The Commonwealth Department of Health through the Northern Territory Primary Health Network.



How to use this manual

This manual introduces conditions frequently encountered in Northern Australia. It is intended to be used as a starting point for information about these conditions and is not a source of detailed guidelines for individual patient management.

Each entry stands alone, and most are presented with the following format:

- **Disease in Northern Australia** — a summary of the epidemiology and local relevance of each condition.
- **Aetiology and pathogenesis** — presented briefly
- **Clinical picture** — highlights important symptoms, signs and investigations.
- **Differential diagnosis** — particularly highlights related conditions that are locally relevant.
- **Principles of management** — summarises approaches to management. Detailed treatment protocols are not reproduced unless there is no alternative and easily accessible source of information.
- **Further information** — lists sources of telephone advice, management guidelines, clinical resources and further reading.

In addition to the disease summaries; Cultural safety in healthcare, Medication supply issues, and Antimicrobial resistance provide an overview of these approaches in the Northern Territory.

Several clinical case studies are included to highlight approaches to some common presentations in more detail. Some treatment information is included in the case studies as examples of current practice. However, approaches and treatment recommendations are constantly changing, and the current disease management guidelines should always be used to guide decisions about individual patients.

The appendices contain:

- A list of acronyms and abbreviations that occur frequently throughout the book.
- A list of local contacts, and health and community resource agencies.

As advised, contributors to the Tropical Health Orientation Manual have respectfully employed the term First Nations peoples in preference to Indigenous.

Feedback is welcome on content and suggestions for improvement. Please direct feedback to globalhealthadmin@menzies.edu.au

An electronic version of this manual is available via the Northern Territory HealthPathways: https://nt.healthpathwayscommunity.org

Table of Contents

Cultural safety in healthcare x
Antimicrobial resistance xii
Medication supply issues in the Northern Territory xvi

SECTION 1 — BACTERIAL INFECTIONS AND POST-STREPTOCOCCAL SYNDROMES

Group A Streptococcal (Strep A) infections 3
Staphylococcal infections 7
Case study — Skin sores 11
Acute post-streptococcal glomerulonephritis (APSGN) 13
Acute rheumatic fever (ARF) and rheumatic heart disease (RHD) 15
Pneumococcal disease 21
Case study — Pneumonia 25
Melioidosis 29
Tuberculosis (TB) 37
Nocardia 43
Leprosy 45
Non-tuberculous mycobacteria 49
Acinetobacter baumannii pneumonia 53
Bronchiectasis and chronic suppurative lung disease 55
Crocodile bites and water-associated infections 57

SECTION 2 — VIRUSES, SPIROCHETES, CHLAMYDIAL AND VECTOR BORNE DISEASES

Mosquito borne diseases 63
Scrub typhus 71
Leptospirosis 73
Trachoma 77
Bat borne viruses 81
Hepatitis A 85
Hepatitis B 87
Human T-cell lymphotropic virus-I infection (HTLV-1) 91
Influenza 95

SECTION 3 — INFESTATIONS, PARASITIC AND FUNGAL INFECTIONS

Cryptococcal meningitis and pneumonia 99
Tinea corporis 101
Onychomycosis 105
Scabies 107
Head lice 115
Paederus australis (the 'acid beetle') 117
Other bites and stings 119
Hookworm 123
Strongyloidiasis 127
Trichuriasis (whipworm) 131

SECTION 4 — SEXUALLY TRANSMITTED INFECTIONS

An overview of sexually transmitted infections (STIs) 135
Gonorrhoea 139
Syphilis 143
Chlamydia 149
Donovanosis 153
Trichomoniasis 155
Human immunodeficiency virus (HIV) 157
Case study — Urethral discharge 161
Case study — Genital ulcer 163
Case study — Lower abdominal pain: more than just a pain 165

SECTION 5 — TOXINS

Snakebites 169
Ciguatera poisoning 175
Box jellyfish 177
Irukandji syndrome 181
Stonefish 183
Cone snails 185
Kava use 187
Volatile substance misuse 189

SECTION 6 — OTHER CONDITIONS AFFECTING CHILDREN

Otitis media 193
Diarrhoea in First Nations children 197
Case study — Recurrent diarrhoea 201
Case study — Anaemia 205

SECTION 7 — OTHER CONDITIONS

Systemic lupus erythematosus (SLE) 211
Machado-Joseph disease (MJD) 213
Approach to planned immunosuppression 217
Case study — Immunosuppression 221
Asymptomatic eosinophilia 223
Approach to type 2 diabetes mellitus (T2DM) 225
Vaccine preventable diseases and immunisation 231

APPENDIX

Acronyms and abbreviations 237
General information 240
Index 241

Cultural safety in healthcare

FIRST NATIONS CULTURES

First Nations Australians belong to the oldest continuing living culture in the world, dating back over 65,000 years. Across the nation there is also much cultural diversity with over 500 languages spoken before colonisation through a complex yet related kinship system.

This kinship system is grounded in a deep connection to land, sea and water through dreaming and spiritual belief systems. Furthermore, First Nations people are inextricably connected to country. As such, health belief systems reflect a connectedness that is holistic and extends beyond the physical wellbeing of an individual to the social, emotional and culturally wellbeing of families and communities.

First Nations sociomedical model reflects social and spiritual (including supernatural) elements of health in contrast to the dominant view of western health and White structures. Conflicting health perspectives are key barriers to safe and effective care. These barriers manifest both at systems levels and at individual levels, particularly within cross cultural settings. It is essential that all health professionals develop intercultural communication skills that foster authentic relationships and trust across cultural divides. This is the only way to provide safe and effective care.

In Australia, First Nations peoples, particularly those living in remote areas, endure the highest communicable and non-communicable health burdens in the country. This burden reflects the complex interaction between the economic and socio-political environmental conditions attributable to colonisation and ongoing institutional racism. The result is high rates of cardiac, kidney, respiratory, skin and mental illness. There are profound sequalae. Current interventions based on colonial health service models have not, and will not, relieve this burden. We need new health service models based on cultural foundations and owned by First Nations Australians.

LANGUAGE AND COMMUNICATION

More than 100 Indigenous languages are spoken in the Northern Territory, and about 60% of First Nations people speak an Indigenous language as their first language. Language is more than just communication; it is a central aspect of culture. This supports and strengthens health and wellbeing. Poor communication in Northern Territory health services has led to unnecessary deaths, lack of informed consent, needless elongated hospital stays, amputations without patient permission, discharge against medical advice and distrust of healthcare providers. Ineffective communication also places a large amount of stress on medical staff who need to make important decisions, potentially in the absence of critical information. Good communication requires effort. We need to make the effort.

Primary care staff often lament apparent insurmountable barriers to improving clinical outcomes, including the need for systemic change outside their scope of practice. One example is better housing to reduce household crowding, a chief driver of otitis media, childhood pneumonia & diarrhoea, scabies, impetigo, post-streptococcal glomerulonephritis and acute rheumatic fever. First Nations peoples make up 70% of people admitted to the Royal Darwin Hospital. They face huge obstacles trying to navigate complex health systems that have been built on western health philosophies and colonial practices. Institutional racism exists across the great majority of Australian hospitals and health systems. It is typically embedded and concealed in institutional policies and practices. Whereas, at the service coal face, racism is often unthinking rather than malicious. As such it is hard to identify and address by the authorities.

These conflicting systems and paradigms perpetuate ongoing trauma and suffering for First Nations peoples. The concept of cultural safety was developed to counter racism in healthcare and is a proposed solution to address health inequities that result from negative attitudes to cultural differences. Effective clinical care for First Nations peoples must be culturally safe.

CULTURALLY SAFE CARE

Cultural safety involves healthcare providers and institutions recognising and reflecting on their own cultures, while making efforts to address any behaviours or practices that undermine, devalue, or disempower patients' identities. *"Culturally safe practise is the ongoing critical reflection of health practitioner knowledge, skills, attitudes, practising behaviours and power differentials in delivering safe, accessible and responsive healthcare free of racism."* (Australian Health Practitioner Regulation Agency 2020). Culturally safe health care means a patient feels emotionally, spiritually and culturally strong in their identity.

Culturally safe communication is required for the delivery of safe and effective health care. Cultural safety embraces a communication style that is effective, respectful and free from bias. For many non-Indigenous health providers this requires mindfulness in all interactions. In this way healthcare providers can create an environment where power is shared between patient and provider, resulting in a culturally safe clinical consultation. When health providers engage in critical self-reflection, they are better able to consider the social determinants of health and can recognise interpersonal and institutional racism. The task is to change it. Early engagement of language interpreters invariably enriches intercultural communication, helps build relationships, and improves health outcomes.

TRAUMA-INFORMED CARE

Trauma has immediate, lasting and profound transgenerational consequences, and damage from trauma can often echo down many generations. As such, it is important to acknowledge the ongoing impact that traumatic colonisation still has on the health and wellbeing of First Nations peoples. When First Nations peoples today face discrimination and racism in the health system it compounds past trauma and impacts on health and wellbeing.

Trauma-Informed Care is an organisational change process that requires all individuals, practices and protocols, and environments to engage in universal precautions for trauma. Similar to how healthcare professionals put on gloves when needing to touch patients as universal precautions for pathogens, being trauma-informed requires putting on metaphorical gloves by changing interactions, policies, and environments to prevent the possibility of re-traumatization.

RESOURCES

The Communicate Study Partnership aims to improve First Nations peoples' experience and outcomes of healthcare through culturally safe communication practices. The team based at Menzies School of Health Research developed a cultural education podcast called, *Ask the Specialist: Larrakia, Tiwi and Yolŋu stories to inspire better healthcare*. These podcasts aim to answers doctors' questions about working with First Nations patients at Top End hospitals, and address questions such as *Is it okay to make eye contact?* and *I want to know what Aboriginal people feel like when we talk to them, what makes them think that we're racist?*

Menzies School of Health Research	■ Ask the Specialist: Larrakia, Tiwi and Yolŋu stories to inspire better healthcare (podcasts) ■ The benefits of working with Aboriginal interpreters at Royal Darwin Hospital	Available online
NT Government	■ Aboriginal Interpreter Service (bookings) ■ Plain English Health Dictionary	Available online
Menzies School of Health Research	Innovate Reconciliation Action Plan	Available online
Australian Human Rights Commission	Race Discrimination	Available online

FURTHER READING

Kerrigan V, McGrath SY, Baker RD, et al. "If They Help Us, We Can Help Them": First Nations Peoples Identify Intercultural Health Communication Problems and Solutions in Hospital in Northern Australia. J Racial Ethn Health Disparities. 2024

Kerrigan V, McGrath SY, Majoni SW, et al. "The talking bit of medicine, that's the most important bit": doctors and Aboriginal interpreters collaborate to transform culturally competent hospital care. Int J Equity Health. 2021;20(1):170.

Kerrigan V, McGrath SY, Majoni SW, et al. From "stuck" to satisfied: Aboriginal people's experience of culturally safe care with interpreters in a Northern Territory hospital. BMC Health Serv Res. 2021;21(1):548.

Kerrigan V, Lewis N, Cass A, et al. "How can I do more?" Cultural awareness training for hospital-based healthcare providers working with high Aboriginal caseload. BMC Med Educ. 2020;20(1):173.

Mithen V, Kerrigan V, Dhurrkay G, et al. Aboriginal patient and interpreter perspectives on the delivery of culturally safe hospital-based care. Health Promot J Austr. 2021;32 Suppl 1:155-165.

Antimicrobial resistance

The Northern Territory has some of the highest rates of antimicrobial resistance in Australia.

ANTIMICROBIAL RESISTANCE IN NORTHERN AUSTRALIA

Antimicrobial resistance rates in common bacteria that cause infections such as *Staphylococcus aureus, Escherichia coli and Klebsiella pneumoniae* are high in Northern Australia and worsening over time. This is thought to be primarily due to high rates of antibiotic prescribing, variable drug regimen uptake and completion, sepsis and severe infections, and the poor living conditions and overcrowding often found in remote communities. Antimicrobial resistance poses a significant threat to public health because antibiotics underpin so much of routine clinical practice.

AETIOLOGY AND PATHOGENESIS

Antimicrobial Resistance occurs when antibiotics are used for too long or not used for long enough, or the antibiotics are not targeted enough to an infection (e.g. broad-spectrum antibiotics) so that the antibiotics used partially affect other bacteria in the body e.g. in the gut or on the skin. Bacteria develop resistance by evolving to avoid being killed by antibiotics, particularly if they are exposed to antibiotics repeatedly or for a long duration.

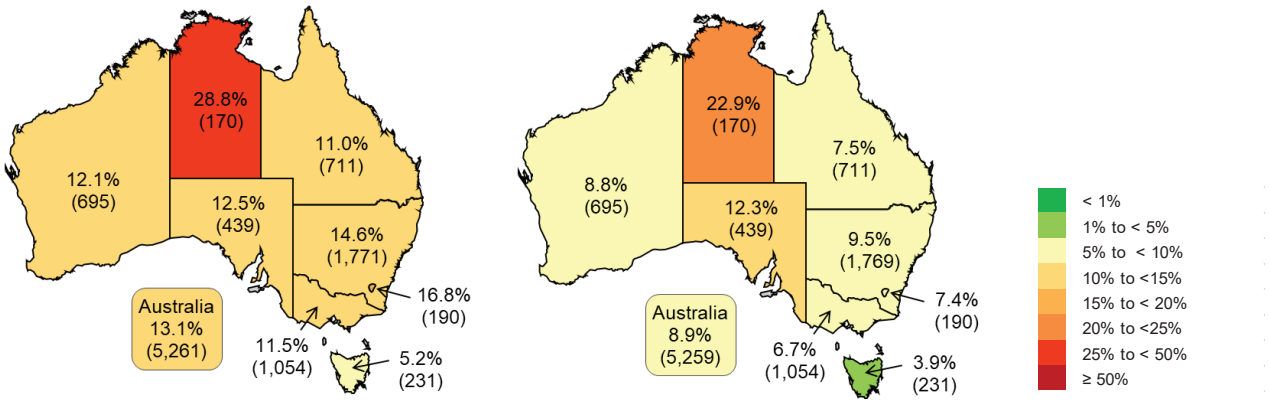


Figure 1: Percentage of *Escherichia coli* from patients with bacteraemia with resistance, as defined by EUCAST, to third-generation cephalosporins (left) and aminoglycosides (right), Australia, AGAR, 2022

Note: Third-generations cephalosporins refers to ceftriaxone and/or ceftazidine; Aminoglycosides refers to gentamicin or tobramycin.

Source: Surveillance Outcome Programs: Bloodstream Infections, 2022 report. Australian Group on Antimicrobial Resistance.

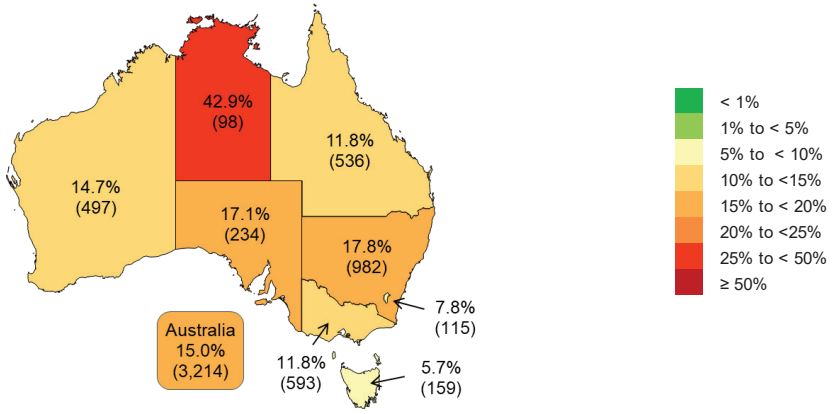


Figure 2: Percentage of *Staphylococcus aureus* from patients with bacteraemia with resistance to methicillin, Australia, AGAR, 2022

Source: Surveillance Outcome Programs: Bloodstream Infections, 2022 report. Australian Group on Antimicrobial Resistance.

CLINICAL PICTURE

Antibiograms are tables showing how susceptible groups of common bacteria are to different antibiotics. They provide information about trends in resistance over time and can inform the development of antibiotic guidelines and prescribing policies. HOTspots is a digital surveillance platform from the CSIRO that uses local pathology data to give antibiograms for different regions of Northern Australia and is a primary care prescribing resource.

For example, by tracking antibiograms in the Top End of the Northern Territory it can be seen that the proportion of all *E. coli* isolates cultured at Territory Pathology that were resistant to ceftriaxone or ceftazidime (i.e. extended beta lactamase producing (ESBL) or AmpC beta lactamase producing) was 12% in 2022 and 18% in 2024. In Central Australia, the proportion reached 25% in 2024. Central Australia has Australia's highest rates of ESBL *E.coli*, with the Top End not far behind.
The rates of bacterial resistance are increasing over time, and this will have significant healthcare implications if unchecked.

PRINCIPLES OF MANAGEMENT

Controlling antimicrobial resistance will help reduce superinfections, premature death and disability, hospital admissions and hospital costs in Northern Australia.

Antimicrobial Stewardship is a systematic approach to "using antimicrobials wisely" to reduce rates of antimicrobial resistance. The Antimicrobial Stewardship Clinical Care Standard contains eight quality statements that describe the care that should be provided to patients with an infection, or patients at risk of an infection. Northern Territory hospitals have Antimicrobial Clinical Decision Support and Antimicrobial formulary and restrictions to support clinicians in the daily practice of Antimicrobial Stewardship.

Below is a scenario-based framework for how clinicians can incorporate four moments of antibiotic decision making into daily clinical practice using Antimicrobial Stewardship principles.

Antimicrobial resistance

Table 1: Scenario-based framework for how clinicians can incorporate 4 moments of antibiotic decision-making into daily practice using Antimicrobial Stewardship principles

Hypothetical scenario incorporating the 4 moments of antibiotic decision-making into daily practice			
Moment	**Scenario**	**Patient and symptom description**	**Decision**
1	Does the patient have an infection that requires antibiotics?	Patient is a 34-year-old, previously healthy woman with dysuria, fever and flank pain.	Patient has signs and symptoms concerning for pyelonephritis.
2	Have I ordered appropriate cultures before starting antibiotics? What empirical antibiotic therapy should I initiate?	Urine dipstick indicates pyuria and bacteriuria.	Urine and blood culture are obtained prior to administering antibiotic therapy. Cefazolin and gentamicin are prescribed as empirical therapy for pyelonephritis as per Antibiotic Clinical Decision Support. Broader therapy is not indicated because the patient does not have risk factors for an antibiotic-resistant infection.
3	A day or more has passed. Can I stop antibiotics? Can I narrow therapy? Can I change from intravenous to oral therapy?	Patient has an appropriate response to therapy and no longer has a fever. A renal ultrasound shows no collection. Urine cultures grow *E. coli* resistant to Augmentin but sensitive to Bactrim.	Patient quickly defervesced so a second dose of gentamicin was not given at 24 hours. Because *E. coli* recovered in the urine; has oral treatment options available that penetrate kidney tissue, cefazolin is stopped, and Bactrim is initiated. The patient is able to tolerate oral therapy and shows clinical improvement: this the patient is switched to oral therapy.
4	What duration of antibiotic therapy is needed for this patient's diagnosis?	Patient is on day 3 of therapy and is ready to be discharged home.	Treatment with Bactrim for 7 days has shown to be effective for pyelonephritis. The patient is discharged home to complete an additional 4 days of therapy.

Adapted from TAMMA, PD et al. Rethinking how antibiotics are prescribed. JAMA. 2019;321(2):130-140.

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call infectious disease physician or Antimicrobial Stewardship team.

MANAGEMENT GUIDELINES

National		
Australian Commission on Safety and Quality in Health Care	Antimicrobial Stewardship Clinical Care Standard	Available online

RESOURCES

CSIRO	HOTspots	Available online
NT Health	NT Antimicrobial Stewardship	Available online

FURTHER READING

Bell JM, Fajardo Lubian A, Partridge SR, et al. Australian Group on Antimicrobial Resistance (AGAR) Australian Gram-negative Surveillance Outcome Program (GnSOP) Bloodstream Infection Annual Report 2022. Commun Dis Intell. (2018). 2022;17:46.

Tamma PD, Miller MA, Cosgrove SE. Rethinking How Antibiotics Are Prescribed: Incorporating the 4 Moments of Antibiotic Decision Making Into Clinical Practice. JAMA. 2019;321(2):139-140.

Medication supply issues in the Northern Territory

MEDICATION SUPPLY IN NORTHERN AUSTRALIA

Geographic isolation, a transient healthcare workforce, unique disease epidemiology, and infrastructure gaps in the Northern Territory are compounded by the tropical climate where extreme seasonal changes, flooding, and road and airstrip closures frequently disrupt access to essential services and supplies, including medication supplies.

Domestic medicine management remains an ongoing challenge, which is increasingly frequently compounded by international supply disruptions. The successful navigation of such shortages depends on coordinated efforts across national, state, and local levels.

MANAGING MEDICATION SUPPLY ISSUES

Governments proactively manage medicine supplies affecting their primary health care services and hospitals, including remote health and mental health services. Strategies include:

- Pharmacists monitoring medicine availability at wholesale level, including sourcing alternate medicines when required.
- Liaising with the Therapeutic Goods Administration (TGA), pharmaceutical wholesalers, pharmaceutical companies, other states and territory agencies and jurisdictional colleagues around medication supply.
- Conserving available medication supplies in the hospitals through consolidation and altering prescribing habits (including the removal of imprest stock).
- Liaising with internal and external stakeholders including clinicians to guide medication conservation activities, including for example, identifying patients and specific conditions for prioritisation of remaining stock, and assessing alternate treatments including overseas registered alternatives where indicated.
- Developing resources to communicate these changes.

The Aboriginal Medical Services Alliance of the Northern Territory (AMSANT) has pharmacists who oversee support and coordination of medication supplies to Aboriginal Controlled Community Health Centres.

STRATEGIES FOR HEALTHCARE PROVIDERS IN MANAGING A MEDICATION SHORTAGE

Collaboration between prescriber, pharmacists and patients are the key to navigating local medication shortages. The following principals and strategies can be considered:

- **Share contact details between healthcare professionals and prepare a local strategy.** Provide direct contact information to facilitate timely communications between healthcare professionals involved in the impact of delivering patient care. Consider strategies that can be implemented prior to the shortage occurring, such as prescribers leaving a standing order or instruction for the pharmacist. In local outreach settings, contacting the clinic managers or healthcare providers and the contracted community pharmacy service providers, can be a practical start to collaborating a solution.
  - The TGA is an important resource used to anticipate and monitor medicine supply issues.
  - Often multiple healthcare professionals participate in the collaborative response to managing a medicine supply issue. Pharmacists are well placed to discuss and problem-solve supply impacts.
  - Medicine supply issues that are anticipated to have large impacts on delivering patient care are often discussed at a government executive level in collaboration with multiple healthcare discipline representatives to navigate solutions.
- **Consider alternate therapies.** Could an alternative strength or formulation of the medicine be prescribed? Is an alternative medicine appropriate when assessing safety and efficacy? Is access to overseas registered alternatives appropriate? Is a pharmacy compounded alternative appropriate? Are there any additional costs to the patient for alternate medicine? Is there a risk of flow-on effect from a protracted shortage? Collaboration is key to determine the appropriate alternate medicine.
- **Seek expert advice as required.** Seek advice early and often from other clinicians, specialists and hospital pharmacists about alternative medications, especially for patients with limited options due to tolerability or lack of efficacy, or if needing to engage with suppliers for overseas registered medicines.
- **Counsel and empower the patient.** Explain the situation, discuss the alternate option(s), address concerns about safety and efficacy and provide clear instruction on the safe use of the alternative medication, set expectations for the potential duration of the shortage, and empower patients to ask questions and express their concerns.

MORE INFORMATION

Guidance for health service organisations - Conserving medicines | Australian Commission on Safety and Quality in Health Care	Useful resource on principles and resources for conserving medicines during a medicine shortage.	Available online
Guidance for clinicians - Antimicrobial Shortages | Australian Commission for Safety and Quality in Healthcare	Useful resource provides general information on managing shortages of antimicrobials.	Available online
Shortages | Therapeutic Goods Administration (TGA)	TGA homepage for Medicine Shortage Alerts, current medicine shortages and discontinuations, and accessing alternative medicines during a shortage.	Available online
Serious Scarcity Substitution Instruments (SSSIs) | Therapeutic Goods Administration (TGA)	Useful resources for community pharmacies to substitute specific medicines without prior approval from the prescriber.	Available online
National Centre for Antimicrobial Stewardship	Medicine Shortage Fact Sheets, including information about prescribing alternatives for common infections.	Available online
Don't Rush to Crush - Advanced Pharmacy Australia	Useful resource for administering tablets and capsule for consumers with swallowing difficulties when oral liquid medicines are in shortage.	Available online
Medicine Shortages | Medicines Management Team	A repository of information intended for use by NT Health clinicians for critical medicine shortages affecting the Northern Territory.	Login required (NT intranet access only)
Your local Primary Health Network | Australian Government Department of Health and Aged Care	Useful resource to facilitate communication in your local primary health network.	Available online

SECTION 1 — BACTERIAL INFECTIONS AND POST-STREPTOCOCCAL SYNDROMES

Group A streptococcal (Strep A) infections

Community surveys demonstrate that up to half of First Nations children may have streptococcal skin sores at any one time.

GROUP A STREPTOCOCCAL INFECTIONS IN NORTHERN AUSTRALIA

Invasive Strep A infections cause a great deal of morbidity and mortality in Northern Australia. *Streptococcus pyogenes (Group A streptococcus* (Strep A)) causes skin and throat infections, and invasive disease such as bacteraemia, necrotising fasciitis, toxic shock syndrome and septic arthritis.

Post-infectious sequelae include acute post-streptococcal glomerulonephritis and acute rheumatic fever, both of which have extremely high incidence rates in Northern Australia. Community surveys demonstrate that up to half of First Nations children in tropical regions may have streptococcal skin sores at any one time.

AETIOLOGY AND PATHOGENESIS

Strep A can be part of normal skin and throat flora. Newly introduced and invasive strains spread quickly particularly overcrowded and unhygienic conditions. Strep A causes infection by adherence and invasion. It subverts the host immune system by resisting phagocytosis, lysing host immune cells, and degrading immunoglobulins.

CLINICAL PICTURE

Risk factors for Strep A infection include overcrowding, poor hygiene practices and conditions of immune compromise such as the extremes of age, HIV, diabetes mellitus, corticosteroid treatment and liver failure.

Symptoms and signs depend on the site of infection. Locally acquired skin infections in tropical Australia (skin sores, pyoderma, impetigo) are usually due to Strep A They are often secondary to scabies infection but may follow minor trauma and insect bites. These lesions are usually seen on the limbs, whereas in some other populations lesions on the face are more common.

Typical symptoms for Strep A pharyngitis include a sudden-onset fever and sore throat with signs of an inflamed pharynx and tonsils, purulent tonsillar exudate, and cervical lymphadenopathy. In contrast to the high prevalence of impetigo, Strep A pharyngitis is uncommonly seen in many remote First Nations communities in Northern Australia.



Figure 3: Streptococcal pyoderma (skin sore)
Source: Bart Currie — Menzies School of Health Research



Figure 4: Streptococcal cellulitis
Source: Bart Currie — Menzies School of Health Research



Figure 5: Streptococcal necrotising fasciitis
Source: Bart Currie — Menzies School of Health Research

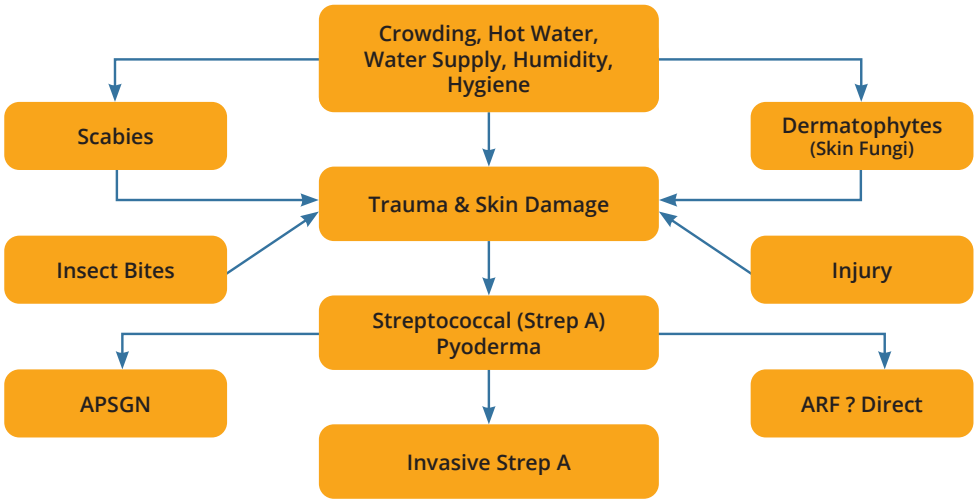


Figure 6: Factors affecting skin disease in First Nations communities

Cellulitis appears as a swollen, red, warm area of skin, often with a clearly demarcated border between the cellulitis and normal skin. The most common causes of cellulitis are Strep A and *Staphylococcus aureus*.

Although rare, it is important to recognise warning symptoms and signs for the more severe forms of Strep A infections. Necrotising fasciitis is characterised by fever, swelling and severe pain over the infected area, and often out of keeping with the skin examination findings. Streptococcal toxic shock syndrome is defined as invasive Strep A with hypotension and organ dysfunction and may accompany necrotising fasciitis.

Investigations. Take swabs of impetigo lesions if they are not improving with standard therapy. Swabs for sore throat should be taken to look for Strep A. If acute rheumatic fever is suspected: take swabs of the throat, bloods for anti-streptococcal antibodies (anti-streptolysin O titre [ASOT] and anti-DNAse B), CRP, FBC, ESR, do an ECG and consult a medical specialist. Echocardiogram is ideally conducted in all such cases. If post-streptococcal glomerulonephritis is suspected check a dipstick urine analysis (and if dipstick positive for blood and/or protein, then urine for albumin creatinine ratio and microscopy) and check complement (C3, C4) levels. Febrile and septic patients should have blood cultures taken.

DIFFERENTIAL DIAGNOSIS

Bullous or pustular impetigo is more frequently due to *S. aureus*. Always look for concurrent scabies infection. If boils — see *Principles of Management* in Staphylococcal Infections (page 9). Several other bacteria and some fungi can cause skin infections. Invasive Strep A infection can be similar to sepsis syndromes from other bacteria such as *S. aureus, Escherichia coli, Pseudomonas aeruginosa, Vibrio* species, and melioidosis, leptospirosis and scrub typhus.

Group A Streptococcal (Strep A) infections

PRINCIPLES OF MANAGEMENT

Skin sores are not normal and should be treated. The initial treatments of choice are a single dose of benzathine benzylpenicillin IM or a course of oral trimethoprim-sulfamethoxazole. Sore throat should be treated with a single dose of benzathine benzylpenicillin IM or a course of oral antibiotics. The aim of treatment in both skin sores and sore throat is to eradicate the Strep A and minimise the risk of transmission to other children and post-streptococcal diseases. Invasive Strep A infections, necrotising fasciitis and toxic shock syndrome are often rapidly progressive and require early intravenous antibiotics after blood cultures, discussion with a medical specialist and transfer to hospital. Guidelines are available for contact tracing and antibiotic prophylaxis for selected contacts of patients with invasive Strep A infection. Post-streptococcal syndromes such as glomerulonephritis and rheumatic fever require hospitalisation. Underlying conditions such as diabetes mellitus and scabies should be managed.

It is important to discuss regular skin washing to reduce the ongoing risk of Strep A skin infection. Identify problems that may prevent daily washing including problems with health hardware (e.g. plumbing, fridges, food preparation and cooking facilities, equipment for cleaning). These should be reported to the local council and Environmental Health Officer. The family should also be advised about washing linen and airing out mattresses to minimise the risk of reinfection.

The emergence and spread of community methicillin-resistant *S. aureus* (MRSA) have implications for antibiotic guidelines. In severe sepsis resulting from skin and soft tissue infections, addition of vancomycin is now recommended during evacuation to hospital or in the Emergency Department. In skin sores without sepsis healing still usually occurs with benzathine benzylpenicillin, irrespective of whether MRSA grows from swab culture. Therefore, for skin sores initial empirical therapy as per guidelines without the need for skin swab cultures is recommended, but if sores persist despite therapy, then skin swab cultures are required to direct therapy.



Figure 7: Recurrent Strep A and MSSA skin sores with mild cellulitis in 3 year old – rapid response to cephalexin (right foot), see Figure 8

Source: Hannah Woerle — Menzies School of Health Research



Figure 8: Recurrent Strep A and MSSA skin sores with mild cellulitis in 3 year old – rapid response to cephalexin (left foot), see Figure 7

Source: Hannah Woerle — Menzies School of Health Research

A NOTE ABOUT PENICILLIN

Penicillin remains the mainstay of treatment for Strep A diseases. It is commonly prescribed in remote areas of Northern Australia, and several preparations are available. Prescribers should refer to the most recent edition of the Therapeutic Guidelines or regional guidelines (e.g. CARPA Standard Treatment Manual) for the treatment of specific conditions.

Phenoxymethyl penicillin (penicillin V)

This is an acid stable penicillin that may be given orally, six or twelve hourly. Food impairs its absorption. In children over the age of two years, its half-life, and that of other oral penicillins, may be extended by concurrent administration of probenecid.

Benzylpenicillin (crystalline penicillin, penicillin G)

This is an intravenous or intramuscular preparation of penicillin, which needs to be administered 4–6 hourly. It is the treatment of choice for many infections.

Procaine penicillin

This is an intramuscular preparation of penicillin commonly used in the treatment of mild com-munity-acquired pneumonia. **As of 2023 it is no longer available in Australia.**

Benzathine benzylpenicillin

This is an intramuscular preparation that provides low levels of penicillin from one day after administration for up to four weeks. It is used in the treatment of syphilis (1-3 doses depending on stage of disease), infected skin sores (single dose), and as a 4-weekly injection to prevent recurrent rheumatic fever.

> Invasive Strep A (confirmed by culture or PCR from a sterile site) is a notifiable condition to be reported by LABORATORIES in the Northern Territory. Cases are to be reported to the local Centre for Disease Control/Public Health Unit.

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call infectious disease physician or general physician.

MANAGEMENT GUIDELINES

National		
The Australian Healthy Skin Consortium	National Healthy Skin Guideline: for the Diagnosis, Treatment and Prevention of Skin Infections for Aboriginal & Torres Strait Islander Children and Communities in Australia, 2023	Available online
National Healthy Skin Guideline	Recognising and Treating Skin Infections: A visual clinical handbook	Available online
Northern Territory		
NT Centre for Disease Control (CDC)	■ Public health management of invasive Group A streptococcal infection ■ Healthy Skin Program — Guidelines for Community Control of Scabies, Skin Sores and Crusted Scabies in the Northern Territory, 2015 ■ Public Health and Notifiable Diseases	Available online

RESOURCES

Menzies School of Health Research	Flip Chart and Video — Healthy Skin Story	Available online
NT Health	Fact sheet - Group A streptococcus infection	Available online
NT Centre for Disease Control (CDC)	Patient information — Streptococcal infection (iGAS) Low literacy version	Available online

FURTHER READING

Abo YN, Oliver J, McMinn A, et al. Increase in invasive group A streptococcal disease among Australian children coinciding with northern hemisphere surges. Lancet Reg Health West Pac. 2023;41:100873.

Cannon JW, Bowen AC. An update on the burden of group A streptococcal diseases in Australia and vaccine development. Med J Aust. 2021;215(1):27-28.

Lacey JA, Marcato AJ, Chisholm RH, et al. Evaluating the role of asymptomatic throat carriage of Streptococcus pyogenes in impetigo transmission in remote Aboriginal communities in Northern Territory, Australia: a retrospective genomic analysis. Lancet Microbe. 2023;4(7):e524-e533.

May PJ, Tong SYC, Steer AC, et al. Treatment, prevention and public health management of impetigo, scabies, crusted scabies, and fungal skin infections in endemic populations: a systematic review. Tropical Medicine & International Health. 2019;24(3):280-293.

Wiegele S, McKinnon E, van Schaijik B, et al. The epidemiology of superficial Streptococcal A (impetigo and pharyngitis) infections in Australia: A systematic review. PLoS One. 2023;18(11):e0288016.

Staphylococcal infections

Staphylococcal infections tend to be purulent, acute in onset, and localise rapidly.

STAPHYLOCOCCAL INFECTIONS IN NORTHERN AUSTRALIA

Infections caused by *Staphylococcus aureus (S. aureus)* are common and include bullous impetigo, cellulitis and skin and soft tissue abscesses. Staphylococcal toxins may also cause rapid-onset, self-limited food poisoning outbreaks. Serious infections such as septicaemia, endocarditis and pneumonia cause considerable morbidity. Staphylococcal septicaemia has a high case fatality rate. *S. aureus* is the most common cause of septic arthritis, osteomyelitis and pyomyositis (muscle abscess). Resistance to standard antibiotics has been present for more 3 decades and is increasing across Australia — community methicillin-resistant *S. aureus* (cMRSA).

AETIOLOGY AND PATHOGENESIS

S. aureus forms part of the normal flora on skin and mucous membranes. It is pathogenic when it produces toxins, breaches epithelial surfaces, and overcomes immune defences. There are at least two successful strains of *S. aureus* described in Northern Australia that produce large amounts of toxins and are a prominent cause of both skin and soft tissue infections and severe invasive infections.

Methicillin-resistant *S. aureus* (MRSA) causes major infection control problems in hospitals, and in some regions, cMRSA now also constitutes up to 50% of all community acquired staphylococcal infections. Widespread use of antibiotics in remote communities helps to select and favour the emergence and dissemination of cMRSA.

CLINICAL PICTURE

Risk factors. These include close contact with people carrying virulent strains, kidney disease, and diabetes mellitus.

Symptoms and signs. Superficial skin infections present as weeping or crusted sores, while deeper infections present as cellulitis, boils, or hot tender abscesses. Infections tend to be purulent, relatively acute in onset and localise rapidly. They can be recurrent.

S. aureus is the most common cause of septic arthritis and osteomyelitis. *S. aureus* bacteraemia should be suspected in someone who is febrile and septic, with skin, soft tissue, osteoarticular, prosthetic or vascular access infections. Staphylococcal pneumonia is a well-known cause of secondary pneumonia following influenza.

Investigations. Cultures from infected sites should be taken if the patient is unwell. This helps guide subsequent antibiotic therapy. Uncomplicated skin and soft tissue infections do not require routine cultures unless the patient is not improving. Febrile and septic patients should have blood cultures taken.



Figure 9: Community MRSA boil
Source: Bart Currie — Menzies School of Health Research



Figure 10: Severe Community MRSA skin pustules and empyema
Source: Bart Currie — Menzies School of Health Research

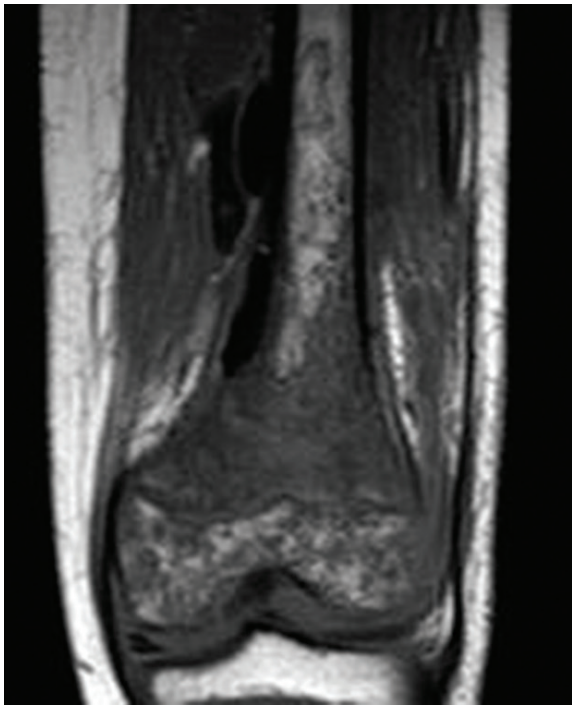


Figure 11: MRI Community MRSA femoral osteomyelitis eight-year-old
Source: Bart Currie — Menzies School of Health Research



Figure 12: MRI Community MRSA tibial osteomyelitis eight-year-old
Source: Bart Currie — Menzies School of Health Research



Figure 13: Community MRSA pneumonia
Source: Bart Currie — Menzies School of Health Research



Figure 14: Severe Community MRSA necrotising pneumonia
Source: Bart Currie — Menzies School of Health Research

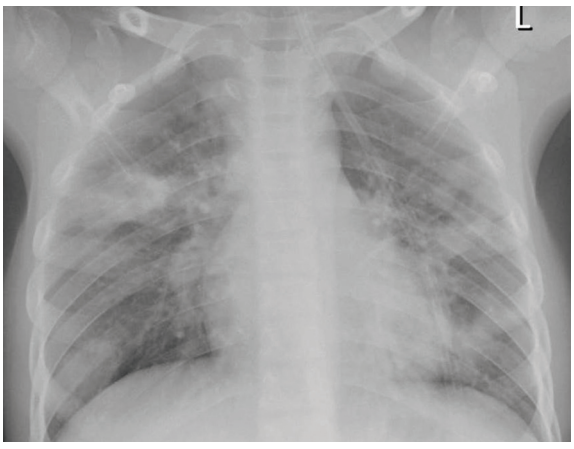


Figure 15: Nine-year-old with severe ST93 Community-MRSA pneumonia post-influenza A
Source: Bart Currie — Menzies School of Health Research

Staphylococcal infections

DIFFERENTIAL DIAGNOSIS

Staphylococcal infection should be differentiated from other causes of bacterial skin or respiratory infection. Look for signs of scabies (page 107). Infections of the occipital scalp are usually associated with infestations of head lice (page 115). The differential diagnosis for pneumonia is described in *Case study — Pneumonia* (page 25). Differentials for *S. aureus* septic arthritis include acute rheumatic fever, gout, shingles, and gonococcal arthritis.

PRINCIPLES OF MANAGEMENT

Treatment depends on antibiotic sensitivity of the organism and the seriousness of infection. Drainage of focal collections is a key tenet. Boils and skin abscesses should be drained; many collections require surgical drainage in an operating theatre. Septic joints should be washed out and deep-seated infected foci removed if possible. Deep infections in body cavities (e.g. pneumonia, osteomyelitis and endocarditis) require hospitalisation and long-term high dose antibiotics.

Septic patients should initially receive antibiotics to cover both methicillin-susceptible *S. aureus* (MSSA), and methicillin-resistant *S. aureus* (MRSA) according to the Therapeutic Guidelines: Antibiotic. Less severe infections can be treated initially with agents directed at methicillin susceptible *S. aureus*, such as flucloxacillin/dicloxacillin, with attention to surgical management and close monitoring of response.

The Staphylococcus aureus Network Adaptive Platform (SNAP) trial is currently being led by Australian researchers internationally. Results from this trial are not yet available, however will likely lead to changes in guidelines in antibiotics used for *S. aureus* bacteraemia as well as duration of treatment.

Recurrent household staphylococcal abscesses/boils may warrant a staphylococcal eradication program including skin washes and nasal mupirocin (see Therapeutic Guidelines or regional guidelines, e.g. CARPA Standard Treatment Manual for details). Underlying conditions (e.g. scabies, diabetes mellitus) should be treated appropriately. Superficial skin sores are predominantly streptococcal and therefore usually respond to therapies directed at Group A streptococcus (Strep A) alone.

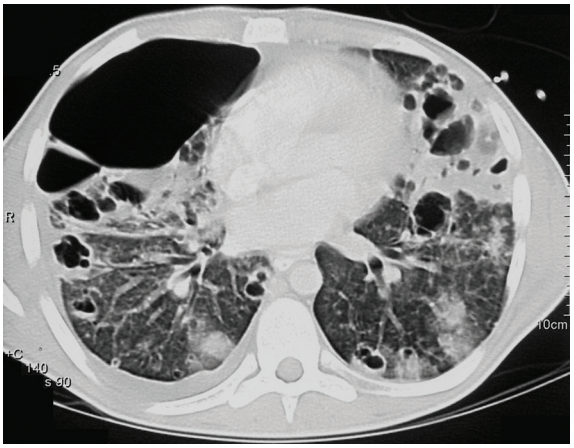


Figure 16: Severe Community MRSA necrotising pneumonia
Source: Bart Currie — Menzies School of Health Research

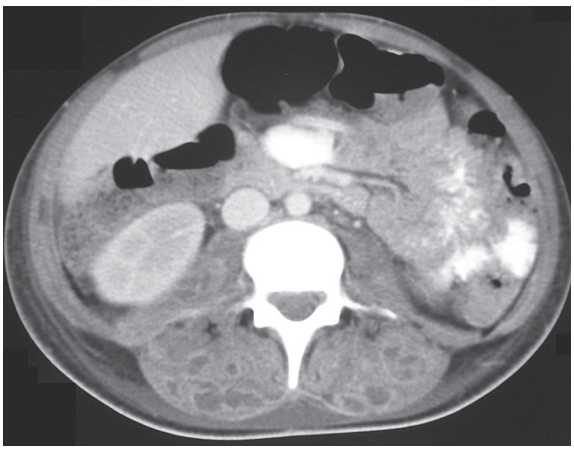


Figure 17: Severe Community MRSA psoas and paraspinal muscle abscesses
Source: Bart Currie — Menzies School of Health Research



Figure 18: Shingles (herpes zoster) post immunosuppression (differential diagnosis)
Source: Bart Currie — Menzies School of Health Research

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call infectious disease physician or general physician or local CDC/PHU.

MANAGEMENT GUIDELINES

National		
The Australian Healthy Skin Consortium	National Healthy Skin Guideline: for the Diagnosis, Treatment and Prevention of Skin Infections for Aboriginal & Torres Strait Islander Children and Communities in Australia, 2023	Available online
National Healthy Skin Guideline	Recognising and Treating Skin Infections: A visual clinical handbook	Available online
Northern Territory		
NT Centre for Disease Control (CDC)	Healthy Skin Program — Guidelines for Community Control of Scabies, Skin Sores and Crusted Scabies in the Northern Territory, 2015	Available online

RESOURCES

Menzies School of Health Research	Flip Chart and Video — Healthy Skin Story	Available online

FURTHER READING

Cameron JK, Hall L, Tong SYC, et al. Incidence of community onset MRSA in Australia: least reported where it is most prevalent. Antimicrob Resist Infect Control. 2019;8:33.

Coombs GW, Daley DA, Shoby P, Mowlaboccus S. Australian Group on Antimicrobial Resistance (AGAR) Australian Staphylococcus aureus Surveillance Outcome Program (ASSOP) Bloodstream Infection Annual Report 2022. Commun Dis Intell (2018). 2023;47.

Tong SYC, Mora J, Bowen AC, et al. The Staphylococcus aureus Network Adaptive Platform Trial Protocol: New Tools for an Old Foe. Clinical Infectious Diseases. 2022;75(11):2027-2034.

Tong SY, Varrone L, Chatfield MD, et al. Progressive increase in community-associated methicillin-resistant Staphylococcus aureus in Indigenous populations in northern Australia from 1993 to 2012. Epidemiology and Infection. 2015;143(7):1519-1523.

Tong SY, Davis JS, Eichenberger E, et al. Staphylococcus aureus infections: epidemiology, pathophysiology, clinical manifestations, and management. Clinical Microbiology Reviews. 2015;28(3):603-661.

Case study — Skin sores

The clinic Health Worker asks you to see Miranda, a two-year-old girl, whose growth and weight gain are poor. Miranda is often seen at the clinic for skin sores. Today she has several pustules around her wrists, crusty lesions on her buttocks and oozing sores on her feet.

What underlying issues should you consider, and how will you treat Miranda?

Miranda's poor growth and skin condition both require attention. You investigate and work out an action plan with the Health Workers according to local guidelines. The plan includes weekly weighs at the clinic, home reviews and ongoing education and support for her family. You schedule her for review by the visiting paediatrician.

You treat her skin infection with a single dose of benzathine benzylpenicillin IM and advise daily bathing to minimise Strep A carriage. It is clear that the underlying cause is scabies, so you also treat with permethrin cream and make arrangements to treat her household contacts for scabies. For household treatment to be effective, it should be done under careful instruction and supervision by a Health Worker. Oral ivermectin may also be an option.

What are some of the reasons for recurrent skin sores?

- Household crowding is number one.
- Poor health hardware such as places to bathe babies and toddlers, non-functioning toilets, lack of working showers &r taps.
- Recurrent scabies in the household. Also look for a possible super-spreader (e.g. with crusted scabies) in the household or nearby.
- Malnutrition or other health issue leading to lower immunity.

What are some of the conditions associated with recurrent skin sores?

- Acute post-streptococcal glomerulonephritis and chronic kidney disease.
- Acute rheumatic fever.
- Cellulitis.
- Secondary bacteraemic sepsis.
- Osteomyelitis.

What public health issues might you consider after seeing Miranda and other children with similar presentations?

- Address health hardware and household crowding - advocate on the family's behalf for urgent housing repair and/or prioritisation for any new housing developments.
- Run a community-wide 'Healthy Skin' program to reduce scabies and Strep A infections. Make sure it is sustainable. Lack of sustainability is the reason these programs have failed in the past.
- Explore community-based growth promotion initiatives (e.g. Strong Women Strong Babies Strong Culture Program).
- In cooperation with local Public Health authorities, establish coordinated and integrated public health and clinical programs focused on scabies eradication.
- Provide training and local capacity building of local Indigenous Health Workers in your community.

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call community paediatrician or local child health team.



Acute post-streptococcal glomerulonephritis (APSGN)

Associated with scabies, skin sores and crowded living conditions, and is a risk factor for chronic renal disease in First Nations adults.

ACUTE POST-STREPTOCOCCAL GLOMERULONEPHRITIS

Acute post-streptococcal glomerulonephritis occurs as individual sporadic cases, and outbreaks that can involve numerous children within one household or several related communities. In the past large outbreaks were common but are now rare. Incidence rates remain significantly higher in First Nations children (>150 cases/10^5/yr) compared with non-Indigenous children (~11 cases /10^5/yr).

AETIOLOGY AND PATHOGENESIS

Acute post-streptococcal glomerulonephritis (APSGN) is an acute inflammatory condition of the kidneys occurring one to three weeks after pharyngeal infection or three to six weeks after skin infection with nephritogenic strains of Group A streptococci (Strep A) (page 3). It is immune mediated with circulating immune complexes and IgA depositing on, and damaging, the glomerular basement membrane. In Northern Australia it is usually associated with skin rather than throat infections. As infection with a newly introduced nephritogenic Strep A strain spreads rapidly, geographic and temporal clustering of cases can occur, often with multiple cases in one household. Periodically there are larger outbreaks, some even extending across the continent.

CLINICAL PICTURE

Risk factors. Children under 15 years are most at risk, although it can occur at any age. Household crowding is one of the key factors, together with geographic or temporal proximity to, or contact with, known cases. Ask about recent Strep A pharyngitis or skin infection.

Symptoms. More than half the cases (up to 70%) are subclinical and only detected on screening. The commonest clinical presentations in children are new onset painless haematuria and/or facial oedema. Other symptoms may include lethargy, anorexia and headache.

Signs. The key findings are sudden weight gain, facial and/or limb oedema plus hypertension. Blood pressure must be assessed using age & sex tables for children (https://publications.aap.org/view-large/7674623). Check the urine for visible haematuria.

Investigations. Urine dipstick is usually positive for blood and protein. But it should always be checked with microscopy, looking for dysmorphic red blood cells 10/mm^3 and red blood cell casts. Swab the throat and any skin sores for Strep A. The blood test for serum complement (C3/C4) is essential; in APSGN C3 will be low, especially in the first 2 weeks of the illness. Other blood results may show elevated ESR, mild normochromic normocytic anaemia, and elevated creatinine. Evidence of recent Strep A infection is essential for the diagnosis. Anti-streptolysin O titre [ASOT] or anti-DNase B titres are usually high, consistent with recent streptococcal infection. However, streptococcal infections are common in First Nations children and antibodies are often elevated even when otherwise healthy.

DIFFERENTIAL DIAGNOSIS

APSGN typically has nephritic triad: haematuria, hypertension and oedema. As such it should be differentiated from other kidney conditions such as IgA nephropathy, membranoproliferative glomerulonephritis, systemic lupus erythematosus, and Henoch-Schönlein disease.

Patients with IgA nephropathy may have an exacerbation following an upper respiratory illness, but the latent period is usually shorter (less than 5 days versus more than 10 in acute post-streptococcal glomerulonephritis). Other infections occasionally associated with nephritis are hepatitis B virus and infective endocarditis. Haematuria may also be caused by renal calculi or urinary tract infections.

PRINCIPLES OF MANAGEMENT

Treatment. All cases should be managed in consultation with a paediatrician or consultant nephrologist. Subclinical or mild cases are often managed in the community with fluid restriction and close observation of weight and blood pressure. Red flags for immediate hospitalisation include oliguria and/or abnormal kidney function, uncontrollable blood pressure, dyspnoea &/or tachypnoea, persistent vomiting and seizure.

Most clinical features settle after 2 weeks although proteinuria and microhaematuria typically persist for months. Mortality is rare (less than 1%), and recurrences are uncommon. Nevertheless, childhood APSGN is subsequently accompanied by a 4-fold increased risk of kidney disease later in life.

Prevention. Reducing the incidence of further cases is main aim. All confirmed, probable and possible cases should be immediately reported by telephone to the regional Public Health authority.

Contact tracing is usually coordinated by the Public Health team and should occur as soon as possible following a case and within 14 days of confirmation of the index case. All close contacts should be examined clinically for signs and symptoms of APSGN. Close contacts aged 12 months to 14 years, and all other contacts with skin sores, should receive a single dose of benzathine benzylpenicillin IM. Regular bathing, especially of children, should be promoted and scabies treated.

> Acute post-streptococcal glomerulonephritis is a notifiable condition to be reported by CLINICIANS in the Northern Territory. Only confirmed and probable cases should be notified to the CDC/PHU. Possible cases should be reported to CDC/PHU but not notified to the Northern Territory Notifiable Disease System.

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call renal physician, general physician, or paediatrician, or local CDC/PHU.

MANAGEMENT GUIDELINES

Northern Territory		
NT Centre for Disease Control (CDC)	■ Public Health Management of Acute Post-Streptococcal Glomerulonephritis (APSGN) ■ Healthy Skin Program — Guidelines for Community Control of Scabies, Skin Sores, Tinea, and Crusted Scabies in the Northern Territory ■ Public Health and Notifiable Diseases	Available online
Remote Primary Health Care Manuals (RPHCM)	CARPA Standard Treatment Manual — Post-streptococcal glomerulonephritis	Available online

RESOURCES

Menzies School of Health Research	Healthy Skin Story	Available online

FURTHER READING

Chaturvedi S, Boyd R, Krause V. Acute post-streptococcal glomerulonephritis in the Northern Territory of Australia: a review of data from 2009 to 2016 and comparison with the literature. American Journal of Tropical Medicine and Hygiene. 2018; 99(6):1643-1648.

Chong HC, Hung TY, Hohls A, et al. Clinical characteristics of hospitalised children with acute post-streptococcal glomerulonephritis in the Top End of Australia. J Paediatr Child Health. 2023;59(5):735-742.

Acute rheumatic fever (ARF) and rheumatic heart disease (RHD)

First Nations peoples in Northern Australia have among the highest published incidence of acute rheumatic fever in the world.

ACUTE RHEUMATIC FEVER AND RHEUMATIC HEART DISEASE IN NORTHERN AUSTRALIA

Since the mid-1900s there has been a shift in the epidemiology of acute rheumatic fever (ARF) resulting in a decline in developed countries. This is primarily due to improvements in housing environments and public health. However, in much of the developing world and in discreet groups within some developed countries, ARF and rheumatic heart disease (RHD) continue to cause significant morbidity and mortality.

Many cases of ARF are not detected, either because people do not seek treatment, or because the diagnosis is missed. This results in missed opportunities for secondary prevention and progress to RHD. A high proportion of patients have progression to RHD at the time of first diagnosis. Clinicians must maintain a high index of suspicion when assessing any patient presenting with joint pain in a high-risk setting, even if there is a history of trauma. Secondary prophylaxis in the form of regular benzathine benzylpenicillin G (BPG) injections is required to prevent ARF recurrences.

AETIOLOGY AND PATHOGENESIS

ARF is a self-limiting, inflammatory illness of the heart, joints, central nervous system and subcutaneous tissues mediated by the immune response to infection with Group A Streptococci (*Streptococcus pyogenes*/ Strep A). The paradox in First Nations communities is that Strep A throat carriage rates are usually low and symptomatic pharyngitis uncommon, whereas streptococcal skin infections are common.

RHD is a complication of ARF characterised by damage to the heart valves resulting in regurgitation and/or stenosis. Complications of RHD include heart failure, atrial fibrillation, stroke and bacterial endocarditis, resulting in increased morbidity and early mortality. ARF tends to recur, and RHD is more likely to develop and progress with each ARF recurrence.

CLINICAL PICTURE

Risk factors include socio-economic disadvantage, household crowding, and repeated Strep A infections. ARF typically occurs in school aged children. It is rare in children aged less than four years but still seen up to middle age.

Symptoms and signs of ARF can be subtle. There are two distinct clinical presentations and disease progressions; they do NOT occur together (see Table 2). Consider the diagnosis in any First Nations patient presenting with arthralgia, arthritis, fever of unknown origin, a new heart murmur or new heart failure. Alternatively, there may be new onset involuntary movements (Sydenham chorea). The presence of Sydenham chorea alone confirms the diagnosis of ARF.

Since 2012, the diagnostic criteria for ARF in Australia include different criteria for people at highest risk. This includes First Nations peoples living in rural or remote settings, First Nations, Māori and Pacific Islanders living in crowded urban settings, and migrants from other high-risk countries.



Figure 19: Polyarthritis in the fingers during ARF
Source: Bart Currie — Menzies School of Health Research



Figure 20: Sixteen year old with ARF post-infected scabies
Source: Bart Currie — Menzies School of Health Research

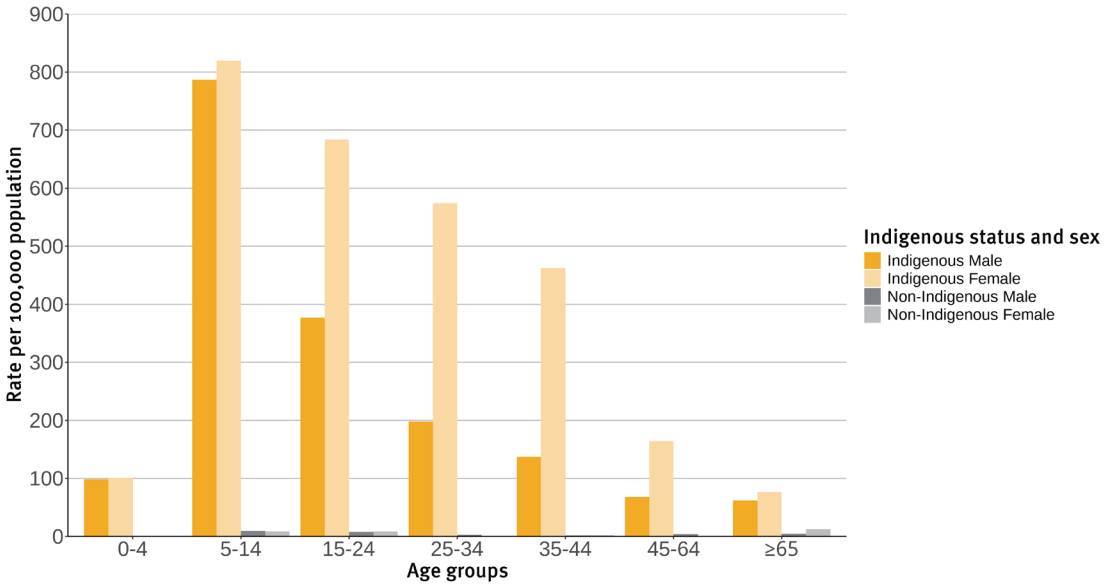


Figure 21: Rates of ARF and RHD incidence (combined) in the Northern Territory, by age group, Indigenous status and sex, 2018-2024

Source: Northern Territory Notifiable Diseases System (NTNDS)

Table 2: Different presentations of ARF

ARF – acute febrile illness with joint symptoms	ARF – neurological illness with chorea (25-30%)
■ Onset: 2-4 weeks after Step A infection ■ Fever ■ Acute joint symptoms: asymmetrical large joint mono/oligoarthritis ■ Carditis: clinical & subclinical [ECG: P-R interval] ■ Skin manifestations & subcutaneous nodules: rare in Australia ■ Raised CRP & ESR ■ Evidence of preceding Strep A infection [ASOT & anti-DNAase B] ■ Rapid symptomatic response to NSAID/aspirin ■ Duration 4-6 weeks ■ Long term RHD ~ 40% and this escalates with each ARF recurrence	■ Later onset: 2-6 months after Strep A infection ■ Usually no fever ■ Behavioural disorder and distinctive Sydenham's chorea. Females>>males ■ Active carditis > 30%: commonly subclinical ■ Often normal inflammatory markers ■ ASOT less helpful, anti-DNAase B commonly raised ■ Duration 3-6 months with relapse ~ 30% ■ Treatment: may require valproate, carbamazepine, etc ■ Repeat episodes ~ 30% ■ Long-term RHD > 50%. This escalates with each ARF recurrence

Acute rheumatic fever (ARF) and rheumatic heart disease (RHD)

Table 3: 2020 Updated Australian criteria for ARF diagnosis

	High risk groups†	Low-risk groups
Definite initial episode of ARF	2 major manifestations + evidence of preceding Strep A infection, **OR** 1 major + 2 minor manifestations + evidence of preceding Strep A infection‡	
Definite recurrent§ episode of ARF in a patient with a documented history of ARF or RHD	2 major manifestations + evidence of preceding Strep A infection, **OR** 1 major + 2 minor manifestations + evidence of preceding Strep A infection‡, **OR** 3 minor manifestations + evidence of a preceding Strep A infection‡	
Probable or possible ARF (first episode or recurrence§)	A clinical presentation in which ARF is considered a likely diagnosis but falls short in meeting the criteria by either: ■ one major or one minor manifestation, **OR** ■ no evidence of preceding Strep A infection (streptococcal titres within normal limits or titres not measured) Such cases should be further categorised according to the level of confidence with which the diagnosis is made: ■ probable ARF (previously termed 'probable: highly suspected') ■ possible ARF (previously termed 'probable: uncertain')	
Major manifestations	Carditis (including subclinical evidence of rheumatic valvulitis on echocardiogram) Polyarthritis¶ or aseptic monoarthritis or polyarthralgia Sydenham chorea†† Erythema marginatum‡‡ Subcutaneous nodules	Carditis (including subclinical evidence of rheumatic valvulitis on echocardiogram) Polyarthritis¶ Sydenham chorea†† Erythema marginatum‡‡ Subcutaneous nodules
Minor manifestations	Fever§§ ≥38°C Monoarthralgia¶¶ ESR ≥30 mm/h or CRP ≥30 mg/L Prolonged P-R interval on ECG†††	Fever ≥38.5°C Polyarthralgia or aseptic monoarthritis¶¶ ESR ≥60 mm/h or CRP ≥30 mg/L Prolonged P-R interval on ECG†††

† High-risk groups are those living in communities with high rates of ARF (incidence >30/100,000 per year in 5–14 year olds) or RHD (all-age prevalence >2/1000). Aboriginal and Torres Strait Islander peoples living in rural or remote settings are known to be at high risk. Data are not available for other populations but Aboriginal and Torres Strait Islander peoples living in urban settings, Māori and Pacific Islanders, and potentially immigrants from developing countries, may also be at high risk.

‡ Elevated or rising antistreptolysin O or other streptococcal antibody, or a positive throat culture or rapid antigen or nucleic acid test for Strep A infection.

§ Recurrent definite, probable, or possible ARF requires a time period of more than 90 days after the onset of symptoms from the previous episode of definite, probable or possible ARF.

¶ A definite history of arthritis is sufficient to satisfy this manifestation. Note that if polyarthritis is present as a major manifestation, polyarthralgia or aseptic monoarthritis cannot be considered an additional minor manifestation in the same person.

†† Chorea does not require other manifestations or evidence of preceding Strep A infection, provided other causes of chorea are excluded.

‡‡ Care should be taken not to label other rashes, particularly non-specific viral exanthems, as erythema marginatum.

§§ In high-risk groups, fever can be considered a minor manifestation based on a reliable history (in the absence of documented temperature) if anti-inflammatory medication has already been administered.

¶¶ If polyarthritis is present as a major criterion, monoarthritis or arthralgia cannot be considered an additional minor manifestation.

††† If carditis is present as a major manifestation, a prolonged P-R interval cannot be considered an additional minor manifestation.

CRP, C-reactive protein; ECG, electrocardiogram; ESR, erythrocyte sedimentation rate.

Source: Australian guideline for prevention, diagnosis and management of acute rheumatic fever and rheumatic heart disease (3rd edition)

Investigations for ARF include: FBC, ESR, CRP, antistreptococcal antibodies (anti-streptolysin O [ASOT] and Anti-DNase B titres), ECG, echocardiogram and blood cultures if the patient is febrile. Collect a throat swab for Strep A MC&S.

The ECG needs interpretation using age adjusted values for normal PR intervals.

Table 4: Upper limits of normal of P–R interval

Age group (years)	Sec
3–11	0.16
12–16	0.18
17+	0.20

Source: Australian guideline for prevention, diagnosis and management of acute rheumatic fever and rheumatic heart disease (3rd edition)

Table 5: Suggested upper limits of normal (ULN) for serum streptococcal antibody titres in children and adults

Age group (years)	ULN (U/ml)	
(years)	ASO titre	Anti-DNase B titre
104	170	366
5-14	276	499
15-24	238	473
25-34	177	390
35	127	265

Anti-DNase B, antideoxyribonuclease B; ASO, antistreptolysin O; ULN, upper limit of normal.

Source: Australian guideline for prevention, diagnosis and management of acute rheumatic fever and rheumatic heart disease (3rd edition)

DIFFERENTIAL DIAGNOSIS

An ARF diagnosis calculator developed for the Australian context is available as a smart phone application. The calculator supports the clinician through the steps for diagnosis and management of ARF, with aim of helping to reduce error and inconsistency.

The differential diagnosis can be challenging as symptoms may be subtle. There is no single laboratory test for ARF, therefore, diagnosis remains a clinical decision based on medical history, symptoms and clinical markers. If ARF is being considered, it is recommended to assess the patient with input from a paediatrician, physician or cardiologist to confirm or rule out the diagnosis.

Polyarthritis must be distinguished from the arthritis of arboviral infections (Ross River and Barmah Forrest viruses, gonococcal arthritis, reactive arthritis or other causes of immune mediated arthritis such as systemic lupus erythematosus (page 211). Subacute rheumatic carditis with little or no joint involvement is uncommon but may be confused with cardiac conditions such as viral myocarditis or tuberculosis pericarditis. Subclinical ARF can present as a vague illness associated with aches and pains or unexplained tachycardia.

Note that arthritis during ARF is frequently mis-attributed to recent trauma such as from sport. Sydenham chorea may initially present with an acute behavioural episode.

RHD is diagnosed on clearly defined criteria using echocardiography.

PRINCIPLES OF MANAGEMENT

Generally, people suspected to have ARF should be hospitalised for investigation and management planning. It is important to fully investigate all suspected and potential cases because of the high chance of ARF recurrence and seriousness of the cardiac sequelae.

Initial treatment includes a Benzathine benzylpenicillin G (BPG) injection to eradicate any residual Strep A infection. Additional treatment is based on symptom support for joint pain, fever, and other complications. Treatment of Sydenham chorea may require specialist input.

If there is doubt about the diagnosis, BPG injections should be commenced while waiting for investigation results or specialist review.

The next step is development of a sustainable, long-term management plan in consultation with a specialist cardiologist, general physician or paediatrician. The aim of treatment is to prevent ARF recurrence. This will usually be developed around regular injections of BPG and careful follow-up.

All cases of ARF and RHD must be reported to the local RHD register, which is used to help coordinate care.

People should also be encouraged to seek immediate treatment if they have a sore throat, scabies or skin sores. In addition, they should present if there are recurrent ARF symptoms.

Acute rheumatic fever (ARF) and rheumatic heart disease (RHD)

People with RHD also need prophylactic antibiotics prior to certain procedures to prevent endocarditis; refer to the Therapeutic Guidelines or national or regional guidelines, (e.g. CARPA Standard Treatment Manual).

Women and girls with rheumatic heart disease. It is important that multidisciplinary care with a whole of life approach, encompassing age appropriate and reproductive health needs of girls and women is provided as part of the regular care during review of RHD. Where contraception is required, long-acting reversible contraceptives such intra-uterine device or etonogestrel implant is recommended. Oestrogen containing contraceptives are associated with a higher risk of thrombosis and should be avoided.

Rheumatic Heart Disease in pregnancy. Health practitioners in clinics and communities with known high-risk populations for ARF should be on alert for RHD and carefully assess all women at the first ante-natal presentation. Ideally, all women with known RHD should be assessed prior to pregnancy by their GP and cardiologist. Women with moderate or severe disease should have access to a reliable form of contraception until assessed by a cardiologist to plan a safe pregnancy.

Pregnant women with moderate or severe RHD should be seen by a cardiologist and obstetrician in the first trimester and then according to their specific management plan. Women with mild disease should be seen by their GP in each trimester and a cardiologist in the second trimester. Any pregnant woman with a new murmur should be assessed for undiagnosed RHD as early as possible.

All ARF **(including first and all recurrent definite, probable and possible cases)** and RHD are notifiable conditions that must be reported by CLINICIANS in the Northern Territory. All suspected and confirmed cases should be reported to the RHD Control Program and the local Centre for Disease Control/Public Health Division.

RHD Control Programs

- Darwin 08 8922 8454
- Alice Springs 08 8951 6909

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call specialist physician, paediatrician or local CDC/PHU.

MANAGEMENT GUIDELINES

International		
World Heart Federation	World Heart Federation guidelines for the echocardiographic diagnosis of rheumatic heart disease. 2023	Available online
National		
Menzies School of Health Research	Australian guideline for prevention, diagnosis and management of acute rheumatic fever and rheumatic heart disease (3rd edition)	Available online
Northern Territory		
Remote Primary Health Care Manuals (RPHCM)	CARPA Standard Treatment Manual — Acute rheumatic fever (ARF) and rheumatic heart disease (RHD)	Available online
NT Primary Health Network (NT PHN)	HealthPathways - Acute rheumatic fever (ARF)	Available online (CLICK HERE to request access)
NT Centre for Disease Control (CDC)	Public Health and Notifiable Diseases	Available online

RESOURCES

Menzies School of Health Research	Guidelines and ARF Diagnosis Calculator App	Available online
NT Health	■ Fact sheet - Acute rheumatic fever and rheumatic heart disease ■ NT Rheumatic Heart Disease Program (and Register)	Available online

FURTHER READING

Wyber R, Ralph AP, Bowen AC, et al. Improving primary care for Aboriginal and Torres Strait Islander people with rheumatic heart disease: What can I do? Aust J Gen Pract. 2022;51(12):959-964.

Pneumococcal disease

Streptococcus pneumoniae is a common cause of pneumonia, otitis media and meningitis in Northern Australia. More than 100 distinct serotypes have been identified.

PNEUMOCOCCAL DISEASE IN NORTHERN AUSTRALIA

Streptococcus pneumoniae (S. pneumoniae) **causes substantial morbidity and mortality worldwide. Rates of infection in First Nations Australians are much higher compared to non-Indigenous Australians, with the overall rate of Invasive Pneumococcal Disease (IPD) in First Nations peoples reported as eight times the rate in the non-Indigenous population in 2023. The Northern Territory has the highest rate of IPD in Australia, with 30 cases per 100,000 vs the Australian average of 10.9 per 100,000. Pneumonia is the most common presentation. *S. pneumoniae* is also a leading cause of, ear infection, sinusitis, bacterial meningitis and sepsis.**

IPD has been notifiable by laboratories in the Northern Territory since 1995 and nationally since January 2001. Conjugate pneumococcal vaccines, initially covering 7, then 10 and now 13 pneumococcal serotypes have been available for young children since 2001 and resulted in significant IPD reduction in target groups, with some evidence of herd immunity for other age groups. Reductions in hospitalised pneumococcal pneumonia have been reported in Australia along with vaccine serotype specific pneumococcal otitis media. However, otitis media remains a major problem for First Nations children in Northern and Central Australia. Serotypes 19A and 8 were the most common in the Northern Territory in 2023, but overall, there is significant variability.

AETIOLOGY AND PATHOGENESIS

S. pneumoniae is an encapsulated gram-positive diplococcus. The polysaccharide capsule enhances virulence by protecting it from phagocytosis and providing antigenic variation. The predominant serotypes vary in their distribution between populations, disease types, age groups, geographic areas and in response to vaccination programs. Pneumococci bind to human nasopharyngeal cells and spread to anatomically contiguous sites such as the eustachian tubes or nasal sinuses causing local disease including otitis media, sinusitis and bronchitis.

Various *S. pneumoniae* virulence factors determine the capacity to invade, disseminate and cause severe disease including pneumonia, meningitis and bacteraemia.

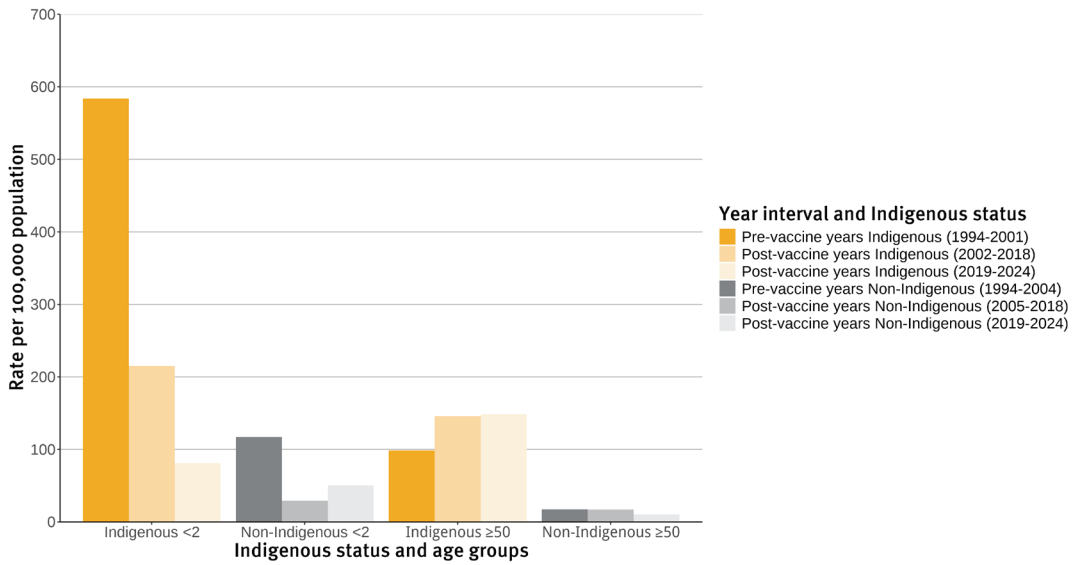


Figure 22: Rates of invasive pneumococcal disease in the Northern Territory before and after conjugate vaccine introduction, by Indigenous status and age group, 1994-2024

Source: Northern Territory Notifiable Diseases System (NTNDS)

CLINICAL PICTURE

Risk factors

- IPD commonly affects people at the extremes of age. For First Nations Australians this is younger than 2 and older than 50 years of age.
- Other factors associated with the highest risk —
  - > First Nations Australian (3 times more likely)
  - > Household crowding and childhood disadvantage
  - > Functional or anatomical asplenia
  - > Immunodeficiencies
    - – Corticosteroids
    - – Malignancies
    - – Chronic kidney failure
- Conditions with an increased risk —
  - > Cardiac disease
  - > Chronic lung disease
  - > Diabetes mellitus
  - > Hazardous alcohol use
  - > Tobacco smoking

Symptoms and signs. *S. pneumoniae* is the most common cause of community acquired bacterial pneumonia. In severe cases, this can be accompanied by a bacteraemia. This can present with sepsis and even shock. It can also be complicated by an parapneumonic effusion or empyema. Outside the lungs, pneumococcus can cause meningitis manifesting as the triad of fever, severe headache and neck stiffness. In infants, classical signs of meningeal inflammation may be atypical.

Less common forms of IPD include septic arthritis, sometimes on a background of pre-existing osteoarthritis, osteomyelitis and rarely endocarditis.

S. pneumoniae is a key pathogen in acute otitis media, especially in First Nations infants and children living in remote communities. This often leads to chronic ear problems including chronic suppurative otitis media (CSOM). CSOM is a major cause of hearing impairment in these children and has life-long consequences.

Investigations aim to recover and identify the infectious agent. Sputum can be sent for microscopy and culture. Blood cultures are essential in all febrile patients and CSF is collected when meningitis is suspected.

A Gram stain of sputum will reveal gram-negative lancet-shaped diplococci and polymorphonuclear leukocytes. A urine pneumococcal antigen test has a moderate sensitivity (0.66) but good specificity (0.90); false positives can occur in children with chronic carriage. PCR testing of blood, CSF or pleural fluid may be used, especially in children, however culture remains the preferred diagnostic procedure for serotyping and antibiotic susceptibility testing.

Chest X-rays in adults usually show a segmental or lobar distribution, whereas in children and the elderly a patchier or bronchopneumonic picture is common.



Figure 23: Pneumococcal septicaemia — post splenectomy
Source: Bart Currie — Menzies School of Health Research

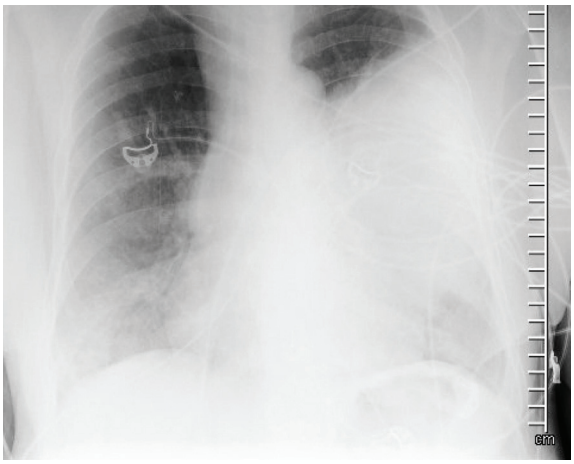


Figure 24: Fatal pneumococcal pneumonia - serotype 3
Source: Bart Currie — Menzies School of Health Research

Pneumococcal disease

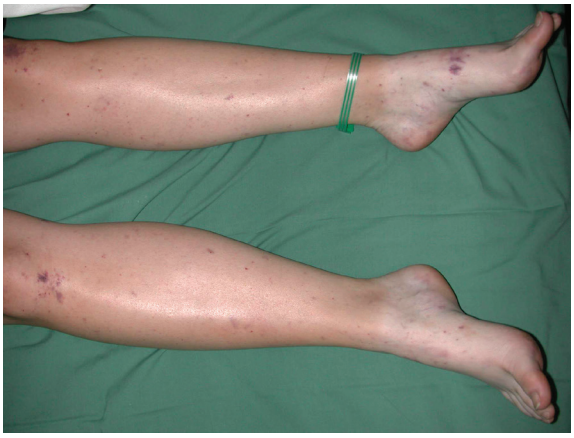


Figure 25: Meningococcal septicaemia rash (differential diagnosis)
Source: Bart Currie — Menzies School of Health Research



Figure 26: Imported measles — will usually have bad cough and fever (differential diagnosis)
Source: Bart Currie — Menzies School of Health Research

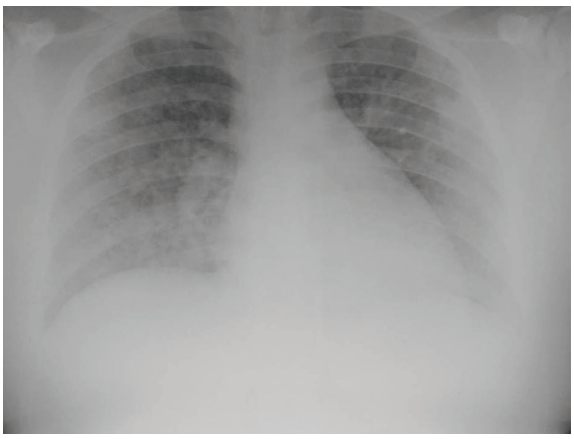


Figure 27: Sixteen year old with varicella pneumonia
Source: Bart Currie — Menzies School of Health Research

DIFFERENTIAL DIAGNOSIS

Other causes of community acquired pneumonia should be considered, especially in the Top End, including melioidosis and *Acinetobacter* pneumonia. While melioidosis tends to be the 'wet season' pneumonia, pneumococcal pneumonia is more often seen in the 'dry season' in adults.

PRINCIPLES OF MANAGEMENT

Treatment. Specific treatment will depend on the clinical condition. While historically the mainstay of treatment of pneumococcal diseases is penicillin, empirical protocols have also been developed to address decreased penicillin susceptibility in some strains of *S. pneumoniae* and to cover other causes of potentially fatal causes of pneumonia such as Melioidosis (page 29) and *Acinetobacter* (page 53).

For confirmed pneumococcal pneumonia penicillin remains excellent therapy but for meningitis the appropriate antibiotic depends on the *S. pneumoniae* MIC (sensitivity) results.

Presentations with overwhelming sepsis need to be treated aggressively. Clinicians should refer to their regional guidelines (e.g. CARPA Standard Treatment Manual) or the Therapeutic Guidelines: Antibiotic for the treatment of community-acquired pneumonia (also see *Case Study — Pneumonia* page 25).

Prevention. The mainstay of control of pneumococcal disease is prevention through the immunisation of high-risk individuals. There are currently two vaccine types:

- The conjugate vaccine (currently mainly 13vPCV, also 15vPCV) has 13-15 polysaccharide serotypes conjugated to a diphtheria carrier protein and routinely included in a 3-dose childhood immunisation schedule at 2, 4, and 12 months, with First Nations children and at-risk children in the Northern Territory receiving a 4th dose at age 6 months. Refer to the Australian Immunisation Handbook. All children over 12 months of age and adults with medical conditions associated with an increased risk of IPD are also recommended to receive a single dose of the 13vPCV conjugate vaccine.

- The polysaccharide vaccine (23vPPV) contains 23 polysaccharide serotypes. In the Northern Territory it is recommended for all First Nations children and adults aged over 50, and anyone aged over one year with increased risk to have two doses of 23vPPV, five years apart.

First Nations children remain less likely to be fully vaccinated against pneumococcus by age 12 months. Although the Northern Territory comparatively has the highest rates of pneumococcal disease nationwide, it was trailing other states in adult coverage with 13vPCV at only 22% in 2022, and vaccination should be readily encouraged.

The Northern Territory pneumococcal vaccination and revaccination guideline is available online — see *Further Information* below.

> Invasive pneumococcal disease is a notifiable condition to be reported by LABORATORIES in Australia and isolates are sent for serotype testing.
>
> **Adverse vaccine reactions** are notifiable by CLINICIANS and should be reported to the local Centre for Disease Control/Public Health Unit.

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call infectious disease physician or general physician or local CDC/PHU.

MANAGEMENT GUIDELINES

National		
Australian Immunisation Handbook	Pneumococcal disease	Available online
Northern Territory		
NT Centre for Disease Control (CDC)	NT Pneumococcal vaccination and revaccination guideline	Available online
NT Department of Health	■ Immunisation Schedules ■ Public Health and Notifiable Diseases	Available online

RESOURCES

Australian Government Department of Health	Invasive Pneumococcal Disease Surveillance	Available online
National Centre for Immunisation Research and Surveillance	History of immunisation in Australia — Significant events in pneumococcal vaccination practice in Australia	Available online
NT Centre for Disease Control (CDC)	Fact sheet — Pneumococcal Disease	Available online

FURTHER READING

Collaro AJ, Chang AB, Marchant JM, et al. Early Childhood Pneumonia Is Associated with Reduced Lung Function and Asthma in First Nations Australian Children and Young Adults. J Clin Med. 2021;10(24):5727.

Lima FJ, Lehmann D, McLoughlin A, et al. Risk factors and comorbidities for invasive pneumococcal disease in Western Australian Aboriginal and non-Aboriginal people. Pneumonia. 2014;4(1):24.

Ranzani OT, Prina E, Menendez R, et al. New sepsis definition (sepsis-3) and community-acquired pneumonia mortality. A validation and clinical decision-making study. American Journal of Respiratory and Critical Care Medicine. 2017;196(10):1287-1297.

Robins-Browne KL, Cheng AC, Thomas KAS, et al. The SMART-COP score performs well for pneumonia risk stratification in Australia's Tropical Northern Territory: a prospective cohort study. Tropical Medicine and International Health. 2012;17(7):914-919.

Case study — Pneumonia

Sandra is a 51-year-old First Nations woman who is brought in by a relative to the remote Top End community health centre where you work. She has been ill for five days with a fever and productive cough and is now having difficulty breathing. She has Type 2 diabetes and frequently has high blood glucose readings. She has smoked tobacco for 30 years and has had several previous admissions to Royal Darwin Hospital for 'bronchitis'. The wet season has arrived, and you had been wondering, before Sandra came in, whether the community airstrip was safe for a medical evacuation.

On examination Sandra is obese and has respiratory distress. She is febrile 38.5C, with a HR of 100, RR of 32, and BP of 115/80. Her finger prick glucose reading is 23mmol/L. ECG and urine dipstick are normal except for glycosuria. She is breathless at rest and oxygen saturation on room air is 89%.

Auscultation of her chest reveals left sided crackles and bronchial breathing. Her yellow/green sputum is copious but not visibly bloodstained. You diagnose Sandra with community-acquired pneumonia.

What organisms are likely to be responsible?

The organisms most likely to be responsible for the pneumonia are bacterial and include *Streptococcus pneumoniae, Burkholderia pseudomallei* (melioidosis), *Staphylococcus aureus*, and *Acinetobacter baumannii*.

Given the short history of illness, infection due to *Mycobacterium tuberculosis* and *Cryptococcus gattii* are less likely. Atypical pneumonia appears to be less common in Northern Australia than in southern states, especially in remote communities. *Mycoplasma, Chlamydophila*, and *Legionella* are less common in remote areas.

Influenza occurs in clusters in remote locations, where it usually has higher morbidity and mortality and may precede bacterial infections, as can COVID-19. While August–September influenza peaks occur as in the rest of Australia, the Top End often also has wet season clusters early in the calendar year. CDC regularly update GPs on the local influenza risk.

The important causes of death in high-risk groups which require specific antibiotics are *Burkholderia pseudomallei* (melioidosis) and *Acinetobacter baumannii.*

On what parameters can you grade this pneumonia as mild, moderate or severe?

The parameters that help to grade the pneumonia are twofold:

- Clinical picture (including observations and bloods where available).
- Individual patient risk factors.

General clinical examination parameters to grade pneumonia include:

- Respiratory rate — more than 30 breaths/min in adults indicates severe pneumonia.
- BP — systolic less than 90 and diastolic less than 60 indicates severe pneumonia and a high risk for developing septic shock.
- Oxygen saturation less than 90% on room air.
- Confusion.

Therapeutic Guidelines discusses pneumonia severity scoring tools for community-acquired pneumonia and notes that CRB-65 and CURB-65 can identify low-risk patients who can usually be managed in the community. However, these tools do not account for comorbidities and hypoxaemia and importantly they can underestimate the severity of pneumonia in patients under the age of 50 years. It is also noted that the PSI (Pneumonia Severity Index) which used to be recommended is considered too complex for routine use in practice.

In the setting of Northern Australia, **local risk factors are important** to determine the risk of severe, life-threatening pneumonia caused by *Acinetobacter baumannii* or melioidosis. Sandra is a First Nations woman; she is obese, has diabetes, is a smoker, has a history of respiratory infections and it is the early wet season. Even if the pneumonia was not severe, with this presentation and her underlying risk factors, broad antibiotic cover and transfer to hospital should be immediately considered and discussed with the rural medical practitioner and hospital medical registrar on call.

What risk factors are particularly important to remember?

In the Top End, risk factors for severe pneumonia caused by *Acinetobacter baumannii* or *Burkholderia pseudomallei* include chronic medical conditions such as diabetes mellitus, hazardous alcohol use, chronic lung disease, chronic renal disease, long-term steroid treatment or other immunosuppression, and kava use.

Would you grade Sandra's pneumonia as mild, moderate or severe?

According to the clinical criteria Sandra's pneumonia is severe: she is febrile, has a high respiratory rate, is mildly tachycardic and is hypoxic.

What are the organisms most likely to cause Sandra's death?

Streptococcus pneumoniae, Burkholderia pseudomallei (melioidosis), *Staphylococcus aureus* (including community MRSA), and *Acinetobacter baumannii*. In some First Nations groups in Central Australia invasive pneumococcal infection rates are as high as 200 per 100,000 people.

How would you initially manage Sandra in your health centre?

While urgent transfer to hospital is being arranged, immediate management consists of:

- Oxygen via mask (6L/min or to maintain oxygen saturation of more than 90%).
- Intravenous (IV) access.
- Blood cultures (to be sent with the patient) immediately prior to the administration of antibiotics.
- Consider IV fluids if BP falls to less than 90 systolic.
- Consider collecting a sputum for culture specifically for melioidosis if the purple Ashdown's medium is available. If not available, collect in a standard specimen container and write on the request form "Melioidosis culture also please".
- Prompt IV antibiotics as per Therapeutic Guidelines or regional protocols (e.g. CARPA Standard Treatment Manual).

Penicillin is **not** recommended as a first line agent for moderate or severe pneumonia for people with risk factors in tropical Australia, as it does not adequately cover potentially lethal organisms. In hospital once the causative organism is identified the antibiotic regimen can be rationalised. While atypical pneumonia cover with doxycycline had previously been considered optional for treatment of CAP in remote communities, more recent advice is to now include it.

> Sandra survived, largely because of your prompt treatment and transfer. She stayed in hospital for 10 days and *Burkholderia pseudomallei* was grown from both sputum and blood cultures. She completed the last 4 days of her intravenous therapy in the self-care unit with 'hospital in the home' providing infusion of ceftazidime via a peripherally inserted central catheter. She returned to your community to complete her eradication therapy under supervision of the clinic and with follow up with Royal Darwin Hospital Infectious Diseases Department.

What longer term issues need consideration?

Several issues need discussion with Sandra when she returns home:

- Listen to her experience of this illness and hospitalisation. Explore and understand her perspective and priorities for her future
- Regardless of causative organism, you should discuss pneumococcal and yearly influenza and COVID-19 vaccines — refer to the use of the newer conjugate as well as polysaccharide vaccine for pneumococcus
- Smoking cessation, diabetes control and healthy nutrition are all priorities for Sandra's well-being. Develop or review her chronic disease care plan. Check she is on your health centre's recall system
- Be alert for the possibility of TB particularly in steroid dependent patients, or lung cancer in long-time tobacco smokers. If the initial chest X-ray was abnormal, a follow-up chest X-ray should be performed at six weeks to ensure that there is no residual consolidation requiring investigation
- Diabetes educator input and medication optimisation for enhanced diabetes control.
- Melioidosis eradication therapy and follow-up in consultation with Royal Darwin Hospital Infectious Diseases Department.

Case study — Pneumonia

FURTHER INFORMATION

See *General Information* (page 240)

MANAGEMENT GUIDELINES

Northern Territory		
NT Primary Health Network (NT PHN)	HealthPathways - Community-acquired Pneumonia (CAP) in adults	Available online (CLICK HERE to request access)

FURTHER READING

Phung DT, Wang Z. Risk of pneumonia in relation to body mass index in Australian Aboriginal people. Epidemiology and Infection. 2013;141(12):2497-502.

Ranzani OT, Prina E, Menendez R, et al. New sepsis definition (sepsis-3) and community-acquired pneumonia mortality. A validation and clinical decision-making study. American Journal of Respiratory and Critical Care Medicine. 2017;196(10):1287-97.

Rémond MGW, Ralph AP, Brady SJ, et al. Community-acquired pneumonia in the central desert and north-western tropics of Australia. Internal Medicine Journal. 2010;40:37-44.

Robins-Browne KL, Cheng AC, Thomas KAS, et al. The SMART-COP score performs well for pneumonia risk stratification in Australia's Tropical Northern Territory: a prospective cohort study. Tropical Medicine and International Health. 2012;17(7):914-919.



Melioidosis

The environmental Gram-negative bacillus *Burkholderia pseudomallei* causes melioidosis, which can affect any organ of the body. Improved diagnosis and treatment have decreased the previously high mortality rate in the Top End of the Northern Territory to under 10%.

MELIOIDOSIS IN NORTHERN AUSTRALIA

***Burkholderia pseudomallei* is endemic in tropical northern Australia, including the Top End of the Northern Territory, Far North Queensland, and in the Kimberley region of Western Australia. Yearly case numbers fluctuate depending on rainfall, severe weather events such as cyclones or flooding, the El Niño Southern Oscillation, and construction activities associated with environmental disturbance. Yearly case numbers vary between 30 and 90 in both the Top End of the Northern Territory and far north Queensland, with under 10 cases yearly in Western Australia.**

AETIOLOGY AND PATHOGENESIS

B. pseudomallei is a gram-negative bacillus found in soil and water across northern Australia. It occurs further south including in Central Australia, Southeast Queensland, and Southwest Western Australia after periods of heavy rains and flooding. Humans are exposed to the organism by percutaneous inoculation, inhalation/aspiration or ingestion. Haematogenous dissemination of infection can occur. The most common site of acute infection is the lungs, although lesions can develop in other organs.

CLINICAL PICTURE

Risk Factors. Diabetes mellitus is the most important risk factor and is present in over 40% of cases. Other risk factors include chronic renal disease, chronic lung disease, hazardous alcohol use and kava intake, malignancy, immunosuppression - especially steroid therapy, and being aged above 45 years. Occupational and recreational exposure can occur through activities that result in contact with soil or water.

Over 80% of cases occur during the wet season, with an incubation period of 1–21 days (median 4 days). During periods of heavy rain and wind, inhalational infection can result in critical illness within 1–2 days.

Although melioidosis usually affects adults with risk factors, it can affect healthy adults and children. First Nations peoples are disproportionately affected. Clinicians should have a low threshold for considering melioidosis as a potential diagnosis across a range of presentations.

Symptoms and signs will depend on the mode and site of infection. Pneumonia is the most common presentation and has a case fatality rate of 10–40%, with the lowest mortality seen when there is timely access to appropriate antibiotics and intensive care unit management.

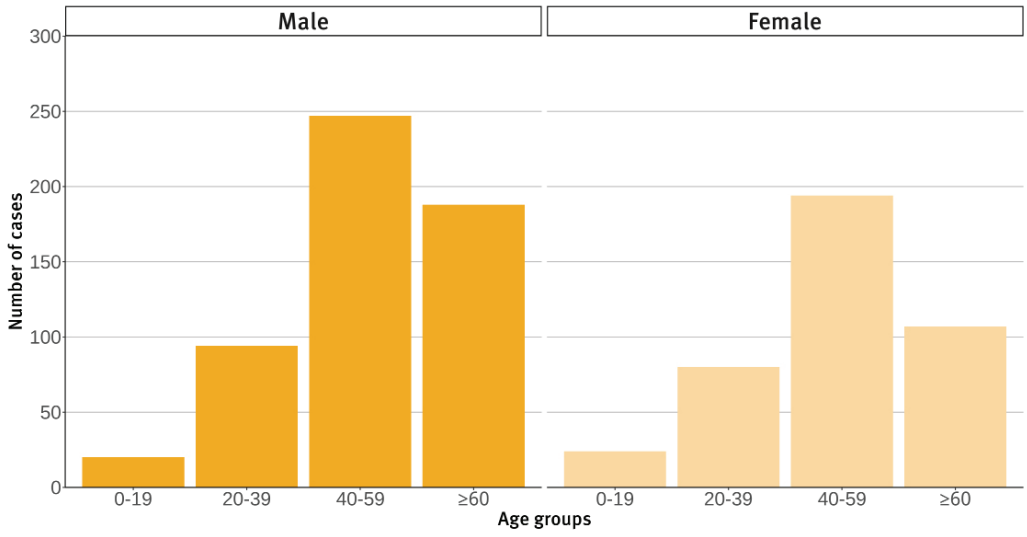


Figure 28: Number of Melioidosis cases in the Northern Territory by sex and age group, 2010-2024
Source: Northern Territory Notifiable Diseases System (NTNDS)

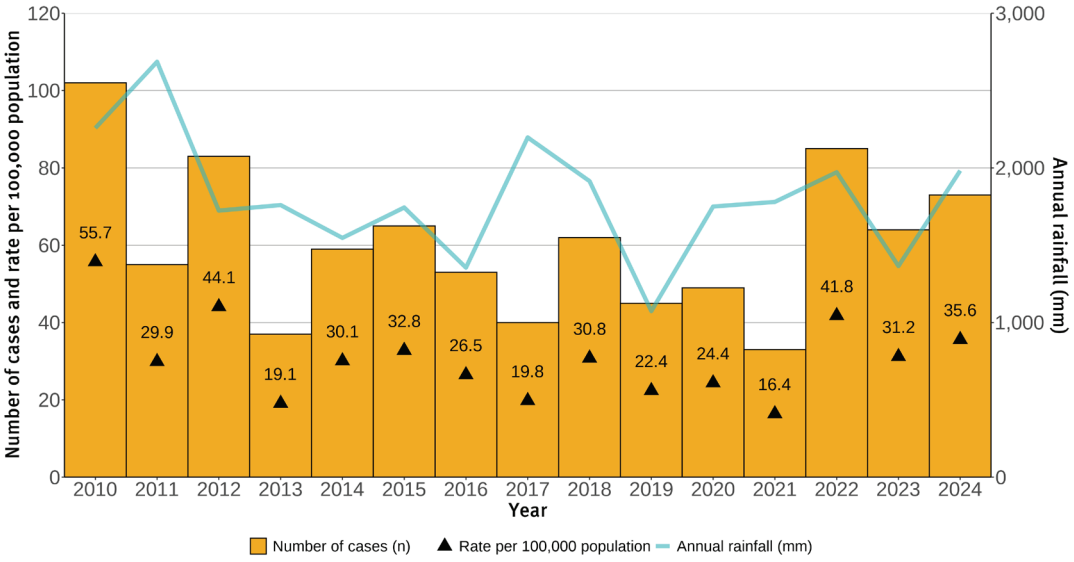


Figure 29: Number and rates of Melioidosis in the Top End (Darwin Urban, Darwin Rural, Katherine & East Arnhem; not including cases with unknown, interstate or overseas address) and annual rainfall by year, 2010-2024

Source: Northern Territory Notifiable Diseases System (NTNDS)

(Rainfall data from Bureau of Meteorology www.bom.gov.au/)

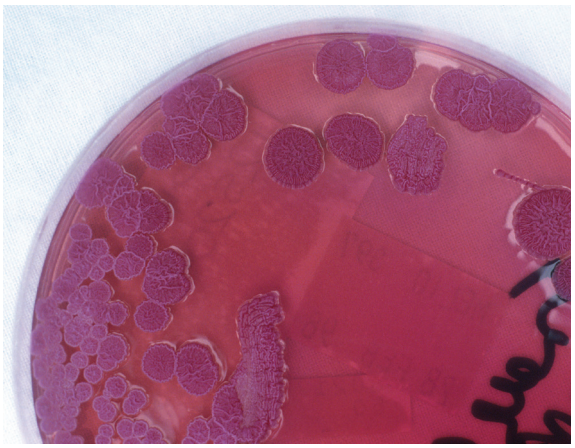


Figure 30: Burkholderia pseudomallei growing on agar plate

Source: Bart Currie — Menzies School of Health Research

Melioidosis can cause severe sepsis leading to septic shock, abscesses of the internal organs, genitourinary infection (often with prostate abscess), osteomyelitis, septic arthritis, pustular skin lesions or chronic non-healing ulcers, and rarely, neurological infection with encephalomyelitis, meningitis, and/or brain abscess.

Examples of important presentations of melioidosis are:

(i) Pneumonia: usually acute, with imaging showing localised consolidation or multifocal infiltrates consistent with bacteraemic spread, but sometimes chronic, with fevers, weight loss and cavitation with or without lymphadenopathy on imaging (mimicking tuberculosis).

(ii) Prostate melioidosis: non-specific abdominal pain, dysuria, diarrhoea, fever and sometimes urinary retention.

(iii) Melioidosis encephalomyelitis: fever, headache, cranial nerve palsies with or without limb weakness or flaccid paraparesis.

Some people are found to be seropositive in the absence of symptoms or known history of the disease. Some seropositive people may have latent *B. pseudomallei* infection, which can activate years later (as is seen in tuberculosis) – however this is rare. It is thought that activation accounts for less than 5% of cases in the Top End.

Melioidosis



Figure 31: *Burkholderia pseudomallei* osteomyelitis and discharging ulcer
Source: Bart Currie — Menzies School of Health Research



Figure 33: Melioidosis — skin abscess in a healthy child who was otherwise well
Source: Bart Currie — Menzies School of Health Research



Figure 32: Melioidosis cutaneous post animal bite
Source: Bart Currie — Menzies School of Health Research



Figure 34: Melioidosis cutaneous
Source: Bart Currie — Menzies School of Health Research

Investigations. Diagnosis is made by culture of *B. pseudomallei* from clinical specimens. Patients with suspected melioidosis should have blood cultures, urine, sputum, and swabs or aspirates from other sites (depending on the presentation) sent with a request to culture for *B. pseudomallei*, with associated clinical details included on the request slip. Swabs can be placed in Ashdown's selective enrichment broth if it is available, however standard bacterial culture swabs should also be collected and *B. pseudomallei* can be isolated from these but add "please culture for melioidosis" on the request slip. Although rapid diagnostic tests such as PCR or antigen detection are highly specific, neither has the sensitivity needed to replace culture and these are not routinely done on clinical specimens. Serology has poor sensitivity early in acute illness, and poor specificity due to high background seropositivity in endemic areas. It can be helpful in situations where obtaining clinical samples for culture is difficult, for example brain abscess or mediastinal mass.

Do not do throat and rectal swabs in remote clinics where Ashdown's media is not available. For skin sores collect standard swabs and place in bacterial transport medium tubes. For these AND for sputum and urine samples, add to the laboratory request form: "Melioidosis culture also please".

Chest X-ray may show cavitation and consolidation. CT scans may identify other sites of infection, and all cases should have CT scan or ultrasound of the abdomen and pelvis.



Figure 35: Fatal melioidosis pneumonia — lung autopsy
Source: Bart Currie — Menzies School of Health Research



Figure 38: Nocardia cutaneous (differential diagnosis)
Source: Bart Currie — Menzies School of Health Research



Figure 36: Sporotrichosis (differential diagnosis)
Source: Bart Currie — Menzies School of Health Research



Figure 39: Sporotrichosis (differential diagnosis)
Source: Bart Currie — Menzies School of Health Research

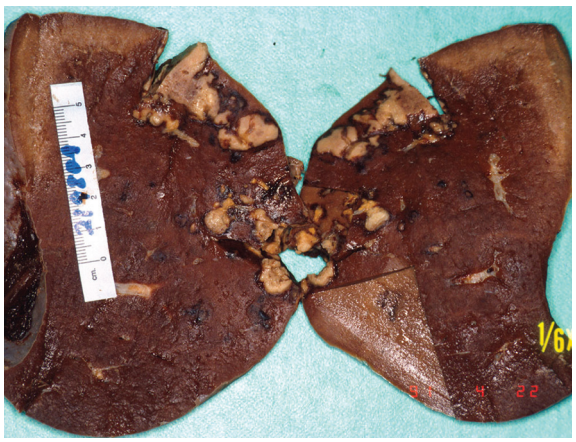


Figure 37: Melioidosis splenic abscesses requiring splenectomy
Source: Bart Currie — Menzies School of Health Research

Melioidosis

DIFFERENTIAL DIAGNOSIS

While pneumonia is the most common presentation, melioidosis can affect any organ of the body often causing multiple abscesses, and the differential diagnosis is wide (including Nocardia infection, page 43). Progressive upper lobe disease mimics tuberculosis and bronchoscopy may be required to make a definitive diagnosis if cultures of sputum are negative.

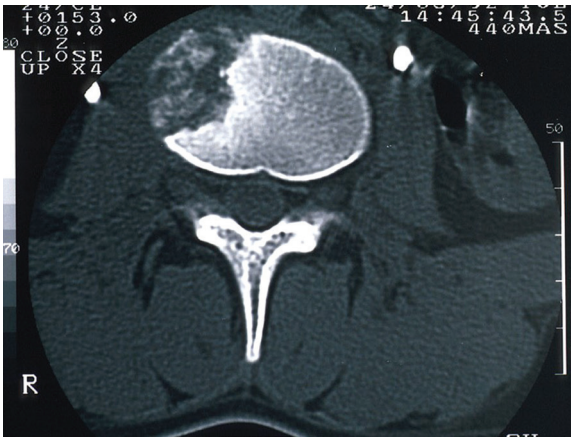


Figure 40: Melioidosis vertebral osteomyelitis
Source: Bart Currie — Menzies School of Health Research

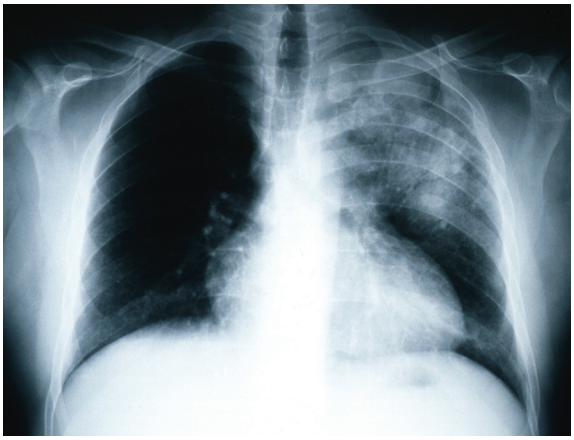


Figure 41: Melioidosis — acute pneumonia
Source: Bart Currie — Menzies School of Health Research

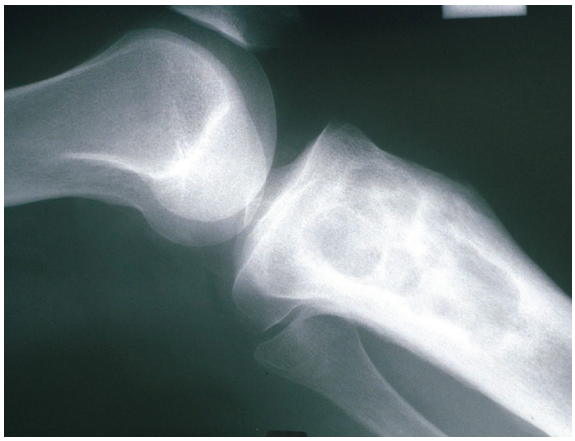


Figure 42: Melioidosis tibial osteomyelitis
Source: Bart Currie — Menzies School of Health Research

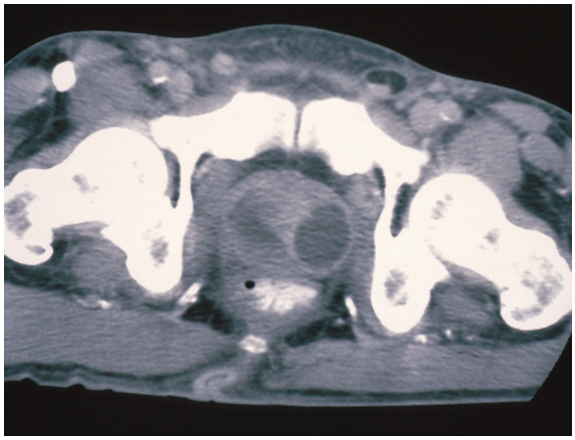


Figure 43: Melioidosis — prostatic abscesses
Source: Bart Currie — Menzies School of Health Research

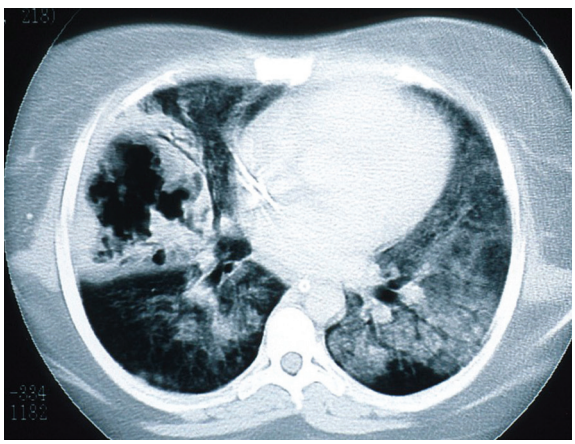


Figure 44: Melioidosis fatal necrotising pneumonia
Source: Bart Currie — Menzies School of Health Research

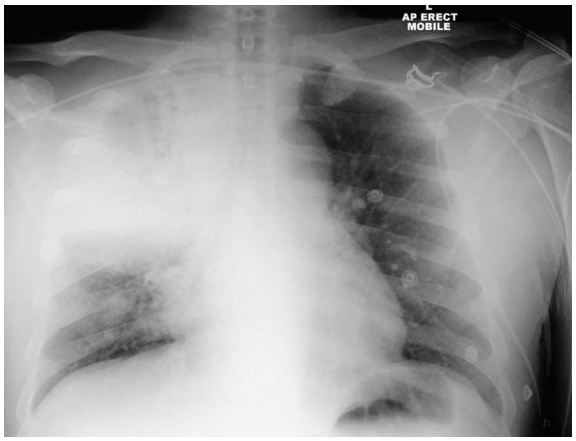


Figure 45: Thirty-six-year-old with fatal nocardia pneumonia (differential diagnosis)
Source: Bart Currie — Menzies School of Health Research

PRINCIPLES OF MANAGEMENT

Prevention. Avoiding contact with soil or muddy water by wearing appropriate shoes and using gloves when gardening or working outdoors. Also avoiding high pressure hosing during the wet season, or as a minimum, using a protective face mask. These measures are particularly important for people with diabetes or other risk factors. People with risk factors should also be advised to stay indoors during strong winds and heavy rain, when the bacteria may become aerosolised.

Treatment. Early diagnosis and appropriate antibiotic therapy decrease mortality. Until diagnosis is confirmed follow the Therapeutic Guideline: Antibiotic or regional guidelines (e.g. CARPA Standard Treatment Manual) for community-acquired pneumonia, which cover the most important pathogens in Northern Australia (see *Case study — Pneumonia* page 25).

Treatment of confirmed melioidosis requires intravenous antibiotics for at least 2 weeks and surgical drainage of abscesses, followed by oral eradication therapy for at least three months. Eradication therapy and close clinical follow-up are extremely important to prevent relapse. All cases in the Northern Territory are managed and followed up in consultation with the Royal Darwin Hospital Infectious Diseases Department.

Subclinical infection or seropositivity alone does not warrant treatment in most instances, however follow-up for these is important.

> Melioidosis is a notifiable condition to be reported by LABORATORIES in the Northern Territory. Cases are to be reported to the local Centre for Disease Control/Public Health Unit.

Melioidosis

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call infectious disease physician or local CDC/PHU.

MANAGEMENT GUIDELINES

Northern Territory		
Remote Primary Health Care Manuals (RPHCM)	CARPA Standard Treatment Manual — Melioidosis	Available online
NT Department of Health	■ Melioidosis guideline ■ Public Health and Notifiable Diseases	Available online
NT Primary Health Network (NT PHN)	HealthPathways - Melioidosis	Available online (CLICK HERE to request access)

RESOURCES

NT Centre for Disease Control (CDC)	■ Fact sheet - Melioidosis ■ Melioidosis- Wet Season Factsheet for Health Professionals ■ Poster – Don't get melioidosis	Available online

FURTHER READING

Currie BJ. Melioidosis: evolving concepts in epidemiology, pathogenesis, and treatment. Seminars In Respiratory And Critical Care Medicine. 2015;36(1):111-125.

Currie BJ, Janson S, Meumann EM, et al. The 2024 revised Darwin Melioidosis Treatment Guideline. NT Dis Control Bull. 2023;30(4):3-15.

Meumann EM, Currie BJ. Approach to melioidosis. CMI Communications. 2024;1(1):100008.

Smith S, Hanson J, Currie BJ. Melioidosis: An Australian Perspective. Trop Med Infect Dis. 2018 Mar 1;3(1):27.



Tuberculosis (TB)

Tuberculosis is caused by *Mycobacterium tuberculosis* and most commonly presents with lung or systemic symptoms, but can affect any body system.

TUBERCULOSIS IN NORTHERN AUSTRALIA

Australia has one of the lowest rates of tuberculosis (TB) in the world (incidence rate of 5–6 cases per 100,000) with people born overseas accounting for 85–90% of new cases. In the Northern Territory, annualised incidence between 2019 and 2023 were 11.9 cases per 100,000 population (95% CI 10.0 to 13.9) with overseas-born people accounting for 54% of cases, First Nations Australians for 43%, and non-Indigenous Australian-born people representing less than 3%. While there have been outbreaks in remote and urban First Nations communities in the past 10 to 20 years such outbreaks are now decreasing.

Beyond country of birth, the three most common risk factors for active TB disease are contact with a case of active pulmonary TB, hazardous alcohol use, and immunosuppressing conditions or medications. Our near neighbours, Indonesia, Timor-Leste and Papua New Guinea have high rates of TB, as do other countries in the region, including Vietnam, Myanmar, Nepal, India, Thailand and the Philippines. People from countries with high TB burdens have an increased risk of infection and disease.

Worldwide, untreated HIV infection is the strongest risk factor for TB infection progressing to active TB disease. Currently there is very little TB-HIV co-infection in Northern Australia.

While multi-drug-resistant TB (MDRTB) and extensively-drug resistant TB (XDRTB) are a concern in much of the rest of the world only a few cases of MDRTB have been notified in the Northern Territory. However, the high rate of MDRTB in Papua New Guinea is a continuing threat to North Queensland. Preventing the emergence of MDRTB relies on having a well-coordinated TB control program. Preventing the spread of MDRTB from those already affected requires timely diagnosis and appropriate curative treatment and infection control.

AETIOLOGY AND PATHOGENESIS

Tuberculosis is an infectious bacterial disease spread by coughing. The causative organism, *M. tuberculosis* complex, is inhaled by contacts of active pulmonary TB patients and deposited in the lungs. Fairly close and prolonged contact is usually required for transmission with an estimated average of 10–15 patients infected by an untreated pulmonary case of TB over a 12-month period. The number infected however is dependent on other factors such as overcrowding and susceptibility of contacts such as through diabetes mellitus, HIV infection, head and neck cancers, or very young age.

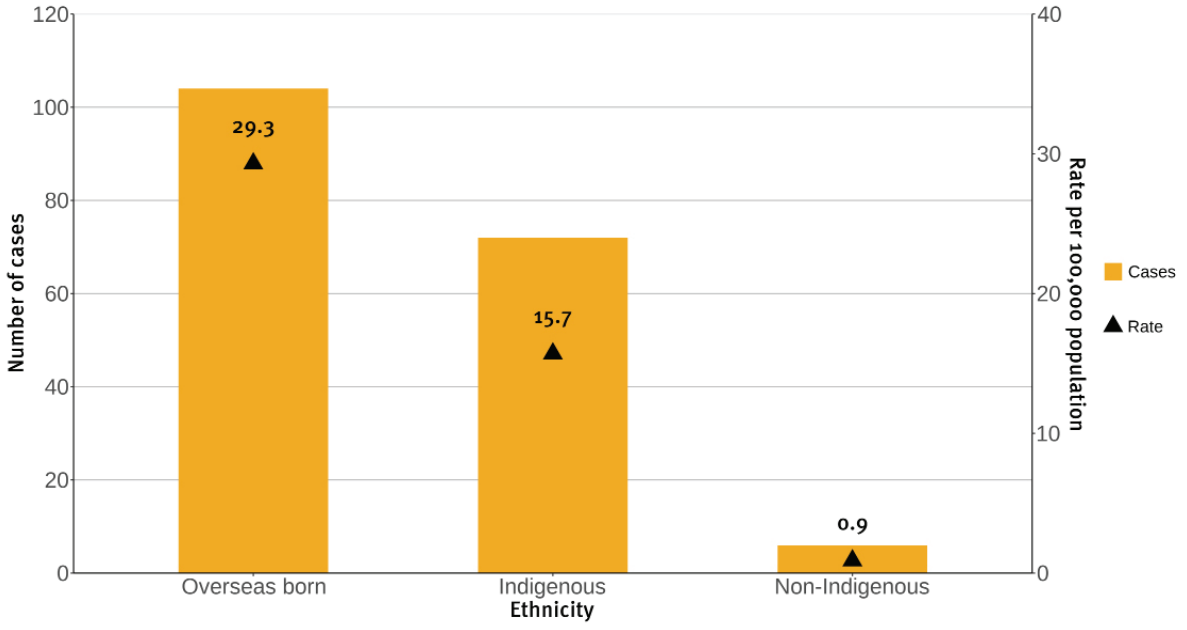


Figure 46: Number and rates of TB in the Northern Territory by ethnicity, 2019-2024
Source: Northern Territory Notifiable Diseases System (NTNDS)

Once infected, a person will usually have a positive tuberculin skin test (TST), also called a Mantoux test, or will test positive via a blood test called an interferon-gamma release assay (IGRA). The most commonly used IGRA in the NT is a QuantiFERON-TB Gold assay. A person who is infected with *M. tuberculosis* but who does not have disease is said to have latent TB Infection (LTBI). Persons with LTBI have no signs or symptoms of active TB and are not infectious. A person with LTBI has a 5–10% lifetime risk of developing active TB disease, which is symptomatic and may, if present in lungs, transmit TB to others. This risk is higher in an individual who develops immunosuppression or other risk factors (see below). The first two years after infection are the highest risk for progression of LTBI to disease and therefore a very important time to find LTBI and consider treatment. This will prevent progression to active disease in the individual, and where pulmonary disease is prevented (the most common TB presentation) will stop transmission to others.

CLINICAL PICTURE

Risk factors. High risk groups in the Northern Territory include contacts of TB cases (always ask patients about this and contact your local CDC or PHU for information), migrant groups from countries where TB is endemic, First Nations people, people who overuse alcohol, and those with diabetes mellitus, chronic kidney disease, malignancies (particularly of the head and neck), leukaemia or lymphoma.

Patients with LTBI who become immunosuppressed due to HIV infection, cancer chemotherapy, long term steroids, or other immunosuppressive therapy are at increased risk of developing active TB.

Sites of presentation. In more than 80% of cases globally the primary site of TB is pulmonary only or extrapulmonary with pulmonary involvement. The remaining 15–20% of cases are extrapulmonary (in low HIV prevalence countries) and include:

- Lymph nodes.
- Urogenital — kidney, bladder, genital tract.
- Skeletal: commonly spine or hip.
- Meningeal.
- Miliary — disseminated TB in lungs and elsewhere.
- Serous membranes — pleurisy, pericarditis, peritoneal.
- Ileocaecal (bowel).
- Almost any other site, eye, breast, liver.



Figure 47: TB lymphadenitis
Source: Bart Currie — Menzies School of Health Research

Symptoms and Signs. Active TB disease is characterised by:

- Cough with sputum for 2 weeks or more +/- haemoptysis.
- Fevers.
- Night sweats.
- Weight loss.
- Lethargy and tiredness.
- Chest pain.
- Localised chest signs in upper/mid zones.
- Pleural effusion.
- Enlarged matted lymph nodes, usually non-tender and most commonly around head and neck.
- Localising symptoms and signs: headache, swollen joint, back pain, etc.

If TB is suspected and the patient is being referred into a hospital ED, the clinician should always call ahead to ensure appropriate transport measures and immediate isolation on arrival.

Investigations. Microbiological confirmation of the diagnosis of TB is essential:

- **Pulmonary TB** — sputum smear examination for acid fast bacilli (AFB) and mycobacterial culture. Other considerations in the differential diagnosis may include collecting sputum for cytology, or to diagnose fungal disease or melioidosis.
  - > Collect sputum specimens **preferably** early in the morning on three different days — however if it needs to be done within 24 hours collect at least 8 hours apart with one collection being early in the morning.

  - > Care should be taken not to infect others — sputum should be collected in a well-ventilated area or outside.
  - > If the patient cannot produce sputum alternative methods are gastric aspiration, induced sputum, or bronchoscopy .
- **For TB in other sites** — a fine needle or formal biopsy for culture is recommended (specimens must not be placed in formalin).
- Rapid diagnostic tests such as Nucleic Acid Amplification Tests (NAAT) using GeneXpert or an MPT64 antigen test can be used by laboratories on different specimen types. They may provide a more rapid diagnosis or help differentiate between *M. tuberculosis* and other mycobacterial species. These usually require prior laboratory discussion.
- Radiology — chest X-ray, CT, MRI, PET.
- Haematology — haemoglobin, white cell count and differential, CRP.
- Histology — may support the diagnosis if granulomatous inflammation found or AFBs seen on staining but is not specific and tissue should always be sent for mycobacterial culture.
- The TST/Mantoux test and interferon-gamma release assays (IGRAs) are helpful in determining previous TB exposure, although they cannot differentiate between latent and active TB. A negative test does not exclude TB. For detailed information about indications for, and interpretation of, Mantoux tests and IGRAs see the CDNA National Guidelines for the Public Health Management of TB.



Figure 48: Tuberculosis — newly arrived in Northern Territory
Source: Bart Currie — Menzies School of Health Research

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of a patient with symptoms and/or a chest X-ray suggestive of pulmonary TB include:

- Common infections in the same risk groups — melioidosis, non-tuberculous mycobacteria, staphylococcal pneumonia.
- Carcinoma — primary bronchogenic or secondary.
- Less common infections — cryptococcosis, nocardiosis, actinomycosis, aspergillosis, pulmonary strongyloidiasis.
- Other — bronchiectasis, sarcoidosis, pneumoconiosis, lymphoma.

PRINCIPLES OF MANAGEMENT

Well-supported, curative treatment with anti-TB medications and adequate nutrition provides the foundations of the clinical management of active TB. Patients require close monitoring for side-effects of treatment, for barriers to treatment, and for signs of clinical improvement. All treatment of either active or latent TB infection should be coordinated through specialist TB or Infectious Diseases units.

The most effective public health interventions are prompt diagnosis and adequate treatment of TB cases with curative treatment, and identification and treatment of LTBI to prevent active TB. In the Northern Territory, people with smear positive pulmonary TB are isolated, usually in hospital, for a minimum of 2 weeks while treatment is initiated. Clearance from isolation depends on several factors including duration of effective therapy and clinical response. Re-testing of sputum may also be requested by clinicians to assess clinical response to treatment. Some people may require hospitalisation for longer than two weeks if they have complicated disease or are likely to be discharged to a high-risk environment.

For active TB cases, a combination of 4 drugs (usually rifampicin, isoniazid, pyrazinamide, and ethambutol) is given for 2 months, followed by 4 months treatment with 2 drugs (usually rifampicin and isoniazid). A shorter course may be possible in some children with pulmonary TB; longer treatment may be required depending on the location of the TB and the medications used. Pyridoxine (vitamin B6) is usually added to prevent neuropathic side effects. MDRTB treatment options have improved significantly in recent years, and the majority can now also be treated with all-oral combination drug regimens.

Treatment of active TB requires a collaborative and patient-centred approach that aims at supporting the patient to obtain adequate number of doses to achieve cure. This includes provision of education and resources that are understandable and in language (if needed), as well as active and ongoing engagement with the patient and their family. Treatment may be best provided through direct observation therapy (DOT), video or text messaging for each dose. Supporting the patient through the full course of treatment greatly increases the chance of cure, while minimising the risk of onward transmission, relapse, and development of drug resistance.

Treatment of LTBI aims to reduce the lifetime risk of developing active TB by more than 90%. Regimens include weekly rifapentine/isoniazid combination therapy for 12 weeks, daily rifampicin for 4 months, daily isoniazid for 9 months, or daily rifampicin/ isoniazid combination therapy for 3 months.

General practitioners and other healthcare workers contribute to TB control by prompt investigation of clinical presentations suggestive of TB, and by having a high index of suspicion in high-risk settings. They may also assist in contact tracing, supporting TB treatment and clinical monitoring in remote communities.

TB Units provide expert advice and treatment and clinical management and follow-up. They have educational materials to share with GPs and healthcare workers, and information sheets and flip charts for patients.

> TB is a nationally notifiable condition to be reported by CLINICIANS and LABORATORIES. **Report cases urgently by telephone** to the local Centre for Disease Control/Public Health Unit.

Tuberculosis (TB)

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call infectious disease physician or local CDC/PHU.

MANAGEMENT GUIDELINES

National		
Communicable Diseases Network Australia (CDNA)	■ National Guidelines for Public Health Units — Management of TB ■ Tuberculosis surveillance case definition	Available online
Northern Territory		
NT Centre for Disease Control (CDC)	■ Guidelines for the Treatment of Tuberculosis in the Northern Territory ■ Public Health and Notifiable Diseases	Available online
Remote Primary Health Care Manuals (RPHCM)	CARPA Standard Treatment Manual —Tuberculosis	Available online
NT Public Health Network (NT PHN)	Northern Territory HealthPathways — Tuberculosis	Available online (CLICK HERE to request access)

RESOURCES

NT Centre for Disease Control (CDC)	■ Fact sheet — Tuberculosis ■ Fact sheet — Tuberculosis (for patients)	Available online
Stop Tuberculosis Partnership	Stop TB Partnership	Available online
International Union Against Tuberculosis and Lung Disease	Crofton's Clinical Tuberculosis — Third Edition	Available online

FURTHER READING

Australian Government Department of Health and Aged Care. National Tuberculosis Advisory Committee. (Updated 5 September 2024).

Coorey NJ, Kensitt L, Davies J, et al. Risk factors for TB in Australia and their association with delayed treatment completion. Int J Tuberc Lung Dis. 2022;26(5):399-405.

National Tuberculosis Advisory Committee for the Communicable Diseases Network Australia. The Strategic Plan for Control of Tuberculosis in Australia, 2021-2025. Communicable Diseases Intelligence. 2022;46.



Nocardia

Nocardiosis is a rare but serious infection which is more common in people with significant immunocompromise.

NOCARDIOSIS IN NORTHERN AUSTRALIA

***Nocardia* species are rare but serious bacterial pathogens which can cause pneumonia, brain abscesses and non-healing wounds. Similar to infections from other environmental pathogens, nocardiosis occurs more commonly in Tropical Australia than elsewhere.**

AETIOLOGY AND PATHOGENESIS

Nocardia species are saprophytic environmental organisms that are found worldwide. There are many species of *Nocardia* which have different geographic ranges. Infection with *Nocardia* species usually occurs via inhalation or direct inoculation into the skin, with pneumonia and skin disease the most commonly observed infections. In immunocompromised hosts, the infection can spread, and cause disseminated disease.

CLINICAL PICTURE

Immunocompromising conditions, such as solid organ transplantation, are major **risk factors** for the development of nocardiosis. In the Top End, hazardous alcohol use and diabetes have also been reported to be common in people diagnosed with this infection. Chronic lung disease is a risk factor for pulmonary infection.

Signs and symptoms. Nocardiosis may present as pneumonia, which can be severe (Figure 50), brain abscess(es) (particularly in immunocompromised hosts) or skin lesions. Skin lesions can have varied appearances, be single or multiple and can be associated with lymphangitic spread with overlying nodules (Figure 49, also see Figure 38). Nocardia bacteria can also colonise lungs in people with chronic lung disease.

Investigations to diagnose nocardiosis should focus on obtaining specimens for culture from the site of disease (such as sputum for pneumonia or wound swabs of skin lesions). As *Nocardia* species can be difficult to grow in the laboratory, special culture techniques may be needed and cases of suspected nocardiosis should be discussed with the microbiologist to ensure these are performed, with laboratory forms labelled “please look for nocardia”. Individuals with suspected nocardiosis should be tested for important differentials, based on the focus of infection, as these may be clinically indistinguishable.

DIFFERENTIAL DIAGNOSIS

Nocardiosis can mimic other serious infections found in Tropical Australia including melioidosis, tuberculosis, atypical mycobacterial lung infections, pulmonary cryptococcosis, cutaneous sporotrichosis and *Acinetobacter baumannii* pneumonia. Sarcoidosis is an important non-infective differential diagnosis.



Figure 49: Nocardia cutaneous post-partum with ankle inoculation site from a thorn puncture
Source: Bart Currie — Menzies School of Health Research

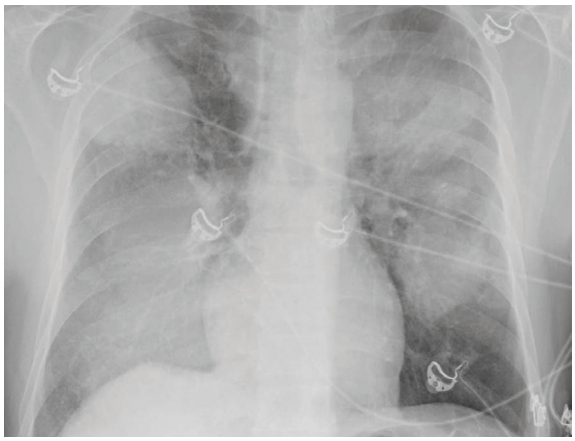


Figure 50: Severe nocardia pneumonia
Source: Bart Currie — Menzies School of Health Research

PRINCIPLES OF MANAGEMENT

Management of nocardiosis should be discussed with an Infectious Diseases specialist. In individuals who are immunocompromised or have neurological symptoms, MRI or CT imaging of the brain is indicated to exclude disseminated disease.

Antibiotic treatment of *Nocardia* is outlined in Therapeutic Guidelines. Antibiotic susceptibility testing is important, but results may be delayed as Nocardia species are slow growing. As susceptibility to key antibiotics varies between *Nocardia* species, an understanding of local epidemiology is required to guide empiric antibiotic choice. For localised cutaneous disease disease in the immuncompetent patient, monotherapy is used, with dual antimicrobials chosen in other situations; duration is 3-12 months depending on disease severity. Key empirical antibiotics used are trimethoprim/sulfamethoxazole, linezolid and amikacin. Occasionally nocardia is cultured from a single sputum sample in a patient without pneumonia and in such cases repeat culture is recommended with close follow up, as therapy may not be necessary if repeat cultures remain negative. This is analogous to atypical mycobacterial infections but different from melioidosis, where any culture of *B. pseudomallei* mandates therapy.

Nocardiosis is not notifiable in the Northern Territory or Queensland.

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

For diagnostic or management advice, contact the on-call infectious diseases physician.

FURTHER READING

Davidson N, Grigg MJ, Mcguinness SL, et al. Safety and Outcomes of Linezolid Use for Nocardiosis. Open Forum Infect Dis. 2020;7(4):ofaa090.

Margalit I, Lebeaux D, Tishler O, et al. How do I manage nocardiosis? Clin Microbiol Infect. 2021;(4):550-558.

McGuinness SL, Whiting SE, Baird R, et al. Nocardiosis in the Tropical Northern Territory of Australia, 1997-2014. Open Forum Infect Dis. 2016;3(4):ofw208.

Leprosy

Leprosy is caused by *Mycobacterium leprae*, which affects the skin, mucous membranes of the nose and peripheral nerves.

LEPROSY IN NORTHERN AUSTRALIA

Although leprosy has become a rare disease in Northern Australia, continued vigilance is required as the incubation period can be as long as 30 years. Recent cases in the Kimberley and Northern Territory may represent past infection rather than ongoing transmission of *M. leprae*, but Queensland has had a recent increase in leprosy cases in the Torres Strait Islands linked to high rates of leprosy still present in regions of Papua New Guinea.

In addition, there are many people in Northern Australia, born in Australia or overseas, who are bacteriologically cured but continue to require lifelong support because of leprosy related disabilities such as reduced visual acuity, amputations, and anaesthetic hands and feet leading to injury. Their close contacts require ongoing surveillance.

AETIOLOGY AND PATHOGENESIS

The leprosy bacillus, *Mycobacterium leprae*, is thought to be predominantly transmitted by inhalation of aerosols generated from infection in the nasal mucosa of people with leprosy. *M. leprae* primarily infects the skin and mucous membranes of the nose and peripheral nerves. There is a continuous spectrum of disease between two forms, **tuberculoid** and **lepromatous** leprosy, depending on the body's immune response to the invading bacilli. The position of individuals on this continuum determines their infectivity, prognosis, likely complications and treatment.

CLINICAL PICTURE

Risk factors. These include being from a high prevalence country, First Nations Australian, or having family or household contact with multibacillary leprosy and crowded living conditions.

Symptoms and signs. Early disease may be asymptomatic. The cardinal signs of leprosy are hypopigmented skin lesions with reduced sensation and/or sweating and thickening of peripheral nerves in leprosy-prone sites. The ulnar, median, radial, common peroneal (lateral popliteal), posterior tibial and sural nerves are most commonly affected.

Investigations.

1. Demonstration of acid-fast bacilli (AFB) in slit-skin smears from standardised sites and lesions.
2. Typical histology, read by an experienced pathologist for skin or nerve biopsies.
3. Nerve conduction studies.

Tips for clinical examination for leprosy

- The entire skin surface needs to be examined — lesions may be only on the buttocks which may require same-gender examiners.
- Skin lesions can present as hypopigmented or erythematous coppery patches with loss of sensation (e.g. to monofilament or ballpoint pen) and an absence of sweating in a lesion (tuberculoid) OR as non-anaesthetic nodules (lepromatous).
- Thickened nerves (e.g. greater auricular, facial, ulnar nerves, among others) on examination.



Figure 51: Borderline lepromatous leprosy with Type I reaction
Source: Bart Currie — Menzies School of Health Research



Figure 52: Leprosy anaesthetic hypopigmented skin patch
Source: Bart Currie — Menzies School of Health Research

- Impaired function of peripheral nerves (e.g. ulnar, common peroneal, median, and tibial nerve, among others), with sensory loss and/or weakness of hands and feet.
- Loss of eyebrow hair, thickened ear lobes and "leonine" facial appearance.
- Inner nasal septum mucosal inflammation +/- ulceration and even perforation.

DIFFERENTIAL DIAGNOSIS

Differential diagnosis includes tinea (especially pityriasis versicolor), systemic lupus erythematosus with skin involvement (discoid lupus), lupus vulgaris (skin TB), sarcoidosis, vitiligo and yaws. Peripheral neuropathy from causes such as diabetes, alcohol excess or syringomyelia do not usually cause primary skin lesions. Leprosy should always be considered when chronic skin disease and peripheral nerve disease co-exist.

PRINCIPLES OF MANAGEMENT

The main objective of treatment is the prevention of disability. Management is complex and all cases must be notified to the Centre for Disease Control/ Public Health Unit and managed by appropriately trained staff.

Treatment is with multi-drug therapy for six to 24 months. At any stage of the disease, and especially when multi-drug therapy is commenced the sudden liberation of antigen from killed *M. leprae*, can precipitate reactions with inflammation of skin lesions and nerves and other manifestations, including new painful tender erythematous skin nodules (erythema nodosum leprosum - ENL), arthritis, orchitis, eye involvement and nephritis. Leprosy reactions are classified as Type I or Type II (i.e. ENL). Neuritis (especially with Type 1 reactions) can be symptomatic but is often silent, and if undetected and untreated can result in irreversible disability.

The key monitoring activity is a monthly standardised motor and sensory test to detect loss of neurological function. Prednisolone initially at high doses may be required to reverse neuritis and prevent disability.

Established impairments such as anaesthetic and anhidrotic skin, or paralysis with contracture, can be managed in primary care through a multidisciplinary team by encouraging the patient towards a daily self-care routine of:

- Inspection for early tissue damage and resting the part if required.
- Soaking and abrading thickened ulcer-prone skin followed by application of a moisturiser.
- Appropriate footwear for protection.
- Active and passive exercising of muscles and joints.
- Identifying and avoiding hazardous activities and negotiating new ways of performing daily tasks.

A person who has been diagnosed with leprosy should be screened for tuberculosis with a Mantoux test, chest X-ray and clinical review as the 'at risk' groups overlap, and treatment of leprosy may jeopardise treatment of TB. Contact tracing of new leprosy cases is undertaken by CDC/PHUs and prophylaxis with single dose rifampicin for selected contacts is beneficial.



Figure 53: Leprosy nerve damage
Source: Bart Currie — Menzies School of Health Research



Figure 54: Leprosy foot ulcer from nerve damage
Source: Bart Currie — Menzies School of Health Research

Leprosy

Leprosy is a nationally notifiable condition to be reported by CLINICIANS and LABORATORIES. Report cases to the local Centre for Disease Control/Public Health Unit.

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call infectious disease physician or local CDC/PHU.

MANAGEMENT GUIDELINES

National		
World Health Organization	Towards zero leprosy. Global leprosy (Hansen's Disease) strategy 2021–2030	Available online
American Leprosy Missions	International Textbook of Leprosy 2016	Available online
National		
Communicable Diseases Network Australia (CDNA)	Australian notifiable diseases and case definitions - Leprosy	Available online
Northern Territory		Available online
NT Centre for Disease Control (CDC)	■ Guidelines for the control of leprosy in the Northern Territory 2018 ■ Public Health and Notifiable diseases	Available online

RESOURCES

Infolep	Leprosy information services	Available online
NT Centre for Disease Control (CDC)	Fact sheet - Leprosy	Available online

FURTHER READING

Barkla S, Modi S. Lepromatous leprosy: A rare presentation in Australia. The Australasian Medical Journal. 2013;6(4):175-177.

Glennie M, Gardner K, Dowden M, Currie BJ. Active case detection methods for crusted scabies and leprosy: A systematic review. PLoS Negl Trop Dis. 2021;15(7):e0009577.



Non-tuberculous mycobacteria

Non-tuberculous mycobacteria (NTM) are environmental microorganisms found in soil and tap water. When isolated in clinical samples, they could represent a colonizer, pathogen or contaminant. While NTM can cause severe infections, not all positive NTM cultures require treatment.

NON-TUBERCULOUS MYCOBACTERIA IN NORTHERN AUSTRALIA

NTM include species of mycobacteria other than *Mycobacterium tuberculosis* complex (MTBC) and *Mycobacterium leprae.* NTM disease is being increasingly recognised globally with higher reported incidences in recent years, and the epidemiology of NTM species can differ geographically and regionally. Between 1989 and 2021, 226 cases of NTM disease were notified in the NT giving an annualised incidence of 3.6 cases per 100 000 population. Of these, there were 131 (58%) pulmonary infections, 73 (32%) soft tissue infections, 7 (3%) lymphadenitis, 4 (2%) gastrointestinal infections, and 3 (1%) disseminated NTM infections.

AETIOLOGY AND PATHOGENESIS

There are more than 190 NTM species which can be divided into rapid growers (growth within a week on culture media) and slow growers (growth requiring weeks). The commonest pathogenic rapid growers are *M. abscessus, M. fortuitum* and *M. chelonae*. Slow growers include *M. avium* complex (MAC, such as *M. avium* and *M. intracellulare*), *M. simiae, M. ulcerans, M. marinum*, and *M. kansasii*. Each pathogenic species is associated with a different clinical picture and susceptibility profile. Host factors and the type of infection are important factors in determining clinical presentation and prognosis. Multi-drug resistance and treatment toxicities add to the challenges of treating NTM and consequently lead to a high rate of treatment failure, morbidity and mortality.



Figure 55: Characteristic appearance of an early *Mycobacterium ulcerans* ulcer
Source: Prof John McBride



Figure 57: *Mycobacterium ulcerans* ulcer
Source: Bart Currie — Menzies School of Health Research



Figure 56: *Mycobacterium abscessus* ulcer
Source: Bart Currie — Menzies School of Health Research



Figure 58: *Mycobacterium fortuitum* ulcer
Source: Bart Currie — Menzies School of Health Research

CLINICAL PICTURE

Risk factors for pulmonary infection or colonisation. People with structural lung diseases such as COPD, bronchiectasis, prior TB, cystic fibrosis; oesophageal reflux/motility disorders; immunosuppression; rheumatoid arthritis; inhalational exposures (such as hot tub use with contaminated water and gardening); malnutrition; and certain body morphologies that may result in poor tracheobronchial secretion drainage or ineffective mucociliary clearance (such as women over 50 with pectus excavatum and/or mitral valve prolapse).

Risk factors for skin infection and disseminated disease. Direct environmental inoculation via broken skin through trauma, natural disasters, insect/animal bites, seawater, fish pedicure, fish tank and marine life handling; immunosuppression (such as HIV, immunosuppressant use, malignancy and diabetes mellitus); and healthcare-associated infections (such as surgery and peritoneal catheter-related infections).

Symptoms and signs. Four distinct clinical syndromes are described below. In addition, NTM can also cause catheter-related, CNS, ocular, and ear infections.

1. Pulmonary disease (chronic nodular bronchiectatic lung disease, cavitating lung disease and hypersensitivity pneumonitis).
   - Chronic cough with or without sputum, fatigue, fever, dyspnoea, haemoptysis and weight loss (Common causative species: MAC, *M. abscessus, M. simiae, M. fortuitum*).
2. Localized cutaneous, joint and soft tissue infection
   - Small violet papules on limbs that may progress to shallow, crusty ulcerations and scar formation, usually singular lesions. May progress to multiple ascending lesions resembling sporotrichosis (Common causative species: *M. fortuitum, M. abscessus, M. marinum, M. ulcerans, M. chelonae*).
   - Slow healing ulcers (often with undermined edge) not responding to usual antibiotics with exposure to endemic areas (Common causative species: *M. ulcerans*).
3. Lymph node disease
   - Enlarging lymph node with or without fistula formation to the skin (Common causative species: MAC, *M. scrofulaceum, M. abscessus, M. fortuitum*).
4. Disseminated disease
   - Common presentation in advanced HIV (Common causative species: MAC).



Figure 59: *Mycobacterium marinum* ulcer from fish tank exposure
Source: Bart Currie — Menzies School of Health Research

   - Non-HIV immunosuppressed patients (Common causative species: *M. abscessus, M. chelonae, M. fortuitum*).

Of note, *M. gordonae* is the most commonly isolated specimen contaminant of low virulence. Expert opinion should be sought if there are persistent positive cultures over months in patients with a clinically compatible disease.

Investigations and diagnosis of pulmonary infections. As transient pulmonary colonisation and environmental contamination can be frequent, diagnosis for NTM pulmonary requires both clinical and microbiological criteria to be met as below:

Clinical and radiologic (both required)

1. Pulmonary or systemic symptoms.
2. Nodular or cavitary opacities on chest radiograph, or a high-resolution CT scan that shows multifocal bronchiectasis with multiple small nodules/tree-in-bud appearance.

With appropriate exclusion of other diagnoses. Consider pulmonary function testing to confirm respiratory impairment.

Microbiological

1. Positive culture results from **at least 2 separate** expectorated sputum samples (should be the same NTM species or subspecies). If the results are non-diagnostic, consider repeat sputum AFB smears and cultures, Or
2. Positive culture results from at least one bronchial washing sample or bronchoalveolar lavage, Or

3. Transbronchial or other lung biopsy with mycobacterial histologic features (granulomatous inflammation or AFB) and positive culture for NTM OR biopsy showing mycobacterial histologic features (granulomatous inflammation or AFB) and one or more sputum or bronchial washings that are culture positive for NTM.

Investigations for non-pulmonary disease. Diagnosis is based on blood cultures, tissue cultures, cultures of aspirate/pus, polymerase chain reaction (PCR), and histology. In disseminated diseases, rapid growers can be isolated from conventional blood cultures. If there is clinical suspicion of disseminated slow-grower infection such as MAC, please contact microbiology / infectious diseases doctors to discuss appropriate blood culture incubation requests or other investigations. For ulcers suspicious of *M. ulcerans* infection, two dry swabs (bacterial culture) of tissue beneath the undermined edges of the lesion should be sent for AFB stain, *M. ulcerans* PCR and culture with clear clinical information provided on pathology form.

DIFFERENTIAL DIAGNOSIS

Differential diagnosis includes other infective causes such as tuberculosis, melioidosis, fungal lung infections (e.g. aspergillosis and cryptococcosis). Non-infective causes include malignancy, while differentials for non-healing skin lesions or ulcers are venous stasis, pressure areas, ischaemic vasculopathy, neuropathic ulcer, skin cancer, pyoderma gangrenosum, leprosy, and arthropod bites.

PRINCIPLES OF MANAGEMENT

Treatment. All cases should be managed in consultation with experienced practitioners in this field (Infectious diseases and CDC). In the NT, the TB Unit can provide expert advice regarding the management of NTM disease. A careful assessment of the pathogenicity of the organism, risks and benefits of therapy, the patient's wish, and ability to tolerate treatment as well as treatment goals should be discussed with patients before initiating treatment. All management should include optimisation of any underlying risk factor(s) management and/or reduction in immunosuppression where possible. In general, antibiotic monotherapy should be avoided as this may lead to drug resistance.

Pulmonary disease. Not all patients who meet the diagnostic criteria for NTM pulmonary disease require antibiotic treatment. In some instances, "watchful waiting" may be the preferred course of action. Optimisation of underlying lung conditions (e.g. chest physiotherapy/airway clearance techniques), nutrition, and treatment of reflux symptoms can help improve symptomatology and chance of cure as well as stop disease progression. Poor prognostic factors such as cavitary disease, low body mass index, low albumin, and/or elevated inflammatory markers should warrant consideration of antibiotic treatment. Adjuvant surgery should be considered in selected patients with localised disease and medical management failure, cavitary disease, drug-resistant isolates (e.g. *M. abscessus* disease), or complications such as haemoptysis or severe bronchiectasis. As surgery can be associated with significant morbidity, it must be subjected to a rigorous risk–benefit analysis within a multidisciplinary setting.

Skin and soft tissue disease. For uncomplicated lesions, surgery with wide margins alone can result in a cure. In more extensive or complicated diseases, antibiotic treatment should complement surgical management. Repeated surgeries may be needed to achieve a cure.

Ulcers from *Mycobacterium ulcerans* are uncommon in the Northern Territory, but have occurred. Primary therapy is with antibiotics. (See Muhi et al, 2025 under Further Reading, page 52)

Overall, established NTM infections are associated with significant morbidity and mortality due to inherent drug multi-drug resistance limiting treatment options, significant treatment toxicities, underlying host factors, and prolonged course of antibiotic treatment (weeks/months) required. Multidisciplinary approach to the management of NTM disease is important for best patient outcome.

Non-tuberculous mycobacterial (NTM) disease including *M. ulcerans* is a notifiable condition to be reported by CLINICIANS and LABORATORIES in the Northern Territory. Report cases to the local Centre for Disease Control/Public Health Unit.

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call infectious disease physician or local CDC/PHU.

MANAGEMENT GUIDELINES

Northern Territory		
NT Centre for Disease Control (CDC)	■ Non-tuberculous mycobacteria (NTM) Guidelines for Health Professionals in the Northern Territory ■ Public Health and Notifiable Diseases	Available online

RESOURCES

NT Centre for Disease Control (CDC)	Fact sheet— Non-tuberculous mycobacterial lung disease	Available online

FURTHER READING

Daley CL, Iaccarino JM, Lange C, et al. Treatment of nontuberculous mycobacterial pulmonary disease: an official ATS/ERS/ESCMID/IDSA clinical practice guideline. Eur Respir J. 2020;56:2000535.

Haworth CS, Banks J, Capstick T, et al. British Thoracic Society guidelines for the management of non-tuberculous mycobacterial pulmonary disease (NTM-PD). Thorax. 2017;72(Suppl 2):ii1–ii64.

Muhi S, Cox VR, O'Brien M, et al. Management of Mycobacterium ulcerans infection (Buruli ulcer) in Australia: consensus statement. Med J Aust. 2025.

Nohrenberg M, Wright A, Krause V. Non-tuberculous mycobacterial skin and soft tissue infections in the Northern Territory, Australia, 1989-2021. International Journal of Infectious Diseases. 2023;135:125-131.

To K, Cao R, Yegiazaryan A, et al. General Overview of Nontuberculous Mycobacteria Opportunistic Pathogens: *Mycobacterium avium* and *Mycobacterium abscessus*. J Clin Med. 2020;9(8):2541.

Acinetobacter baumannii pneumonia

Acinetobacter community-acquired pneumonia is severe with most deaths occurring within 48 hours.

ACINETOBACTER BAUMANNII PNEUMONIA IN NORTHERN AUSTRALIA

***Acinetobacter baumannii* is a Gram-negative coccobacillus that is ubiquitous in the environment and is a human skin and throat commensal. It is well-known because of the development of multiple drug resistance and as causing hospital acquired infections.**

Important in Northern Australia for its ability to cause community-acquired infections in those with hazardous alcohol use and diabetes, with rapidly progressive fulminant pneumonia with sepsis. Local experience shows that appropriate initial treatment can reduce mortality from 60% to 11%.

AETIOLOGY AND PATHOGENESIS

Acinetobacter species are ubiquitous in the environment and have also been found to colonise humans, animals, lice, and present in a wide variety of food products. Most reports of *Acinetobacter* community-acquired pneumonia have been from tropical and subtropical Asia-Pacific countries. *Acinetobacter* community-acquired pneumonia is severe with most deaths occurring within 48 hours, and mortality rates over 60% without appropriate initial treatment.

Most cases in tropical Australia occur during the warmer, wetter months of October to April. The distribution is tropical and subtropical regions.

CLINICAL PICTURE

Risk factors include hazardous alcohol use, diabetes, chronic lung disease, smoking, and chronic renal failure.

Symptoms and signs. Patients present with cough, dyspnoea and fever in most cases. The illness progresses rapidly, and most patients meet the criteria for severe sepsis. Lobar consolidation is usually seen on chest X-ray.



Figure 60: Severe *Acinetobacter baumannii* community-acquired pneumonia
Source: Bart Currie — Menzies School of Health Research

PRINCIPLES OF MANAGEMENT

Acinetobacter community-acquired pneumonia and melioidosis are both wet season diseases and management includes antibiotic coverage for these organisms in community-acquired pneumonia. The *Acinetobacter* strains that cause community acquired pneumonia in the Top End are sensitive to antibiotics including gentamicin, meropenem, and ciprofloxacin. Since the standard Northern Territory treatment for patients with severe tropical wet season community acquired pneumonia and sepsis has included an initial dose of gentamicin (and meropenem is given in those patients sick enough to be managed in ICU), mortality has fallen from more than 60% to 11%.

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call infectious disease physician or local CDC/PHU.

FURTHER READING

Meumann EM, Anstey NM, Currie BJ, et al. Genomic epidemiology of severe community-onset Acinetobacter baumannii infection. Microb Genom. 2019;5(3):e000258.

Bronchiectasis and chronic suppurative lung disease

Bronchiectasis is abnormal widening of the bronchi or their branches, which is both caused by and increases the risk of lung infections.

BRONCHIECTASIS AND CHRONIC SUPPURATIVE LUNG DISEASE IN NORTHERN AUSTRALIA

The incidence of early childhood respiratory infections in First Nations populations is about 5–10 times that of non-Indigenous populations. Bronchiectasis has a reported incidence of 14.7 per 1000 in First Nations children under 15 years of age in Central Australia. The prevalence in First Nations adults is estimated 19.4 per 1000 in the Top End of the Northern Territory. Of Central Australian patients with bronchiectasis, 43 out of 69 (70%) had a history of recurrent childhood respiratory infections as the only potential cause. Bronchiectasis causes a decline in lung function, quality of life and life expectancy. Effective management improves well-being and reduces morbidity.

Bronchiectasis is currently diagnosed by high resolution computed tomography. Children with symptoms of bronchiectasis but no confirmatory high-resolution computed tomography are labelled as having chronic suppurative lung disease. For most purposes, the management of chronic suppurative lung disease and bronchiectasis are the same.

AETIOLOGY AND PATHOGENESIS

CLINICAL PICTURE

Early signs are a chronic cough and recurrent respiratory infections. Chronic wet cough (more than 4 weeks duration in children; >8 weeks in adults) or recurrent wet cough (more than 2 episodes/year) are important. Over time the chronic damage and disease can lead to growth failure, finger clubbing, chest wall deformity, hyperinflation, and added respiratory sounds. In adults, concurrent presence of bronchiectasis and chronic obstructive pulmonary disease (COPD) is common, partly due to high prevalence of tobacco smoking. Although COPD and bronchiectasis share several similar clinical features, the management of these conditions differs; hence, in clinical practice, identifying which one is predominant condition is extremely critical for appropriate management.

Experience has shown that First Nations people commonly under-report cough. This, along with late and non-presentations for respiratory illnesses, make early diagnosis of chronic suppurative lung disease challenging. Keep a high index of suspicion in children and seek additional information from the family and community. In adults, chronic productive cough and shortness of breath are most commonly reported. Lung function parameters more often display a restrictive pattern among patients with bronchiectasis in isolation and demonstrate much more severe and mixed impairment when COPD co-exists.

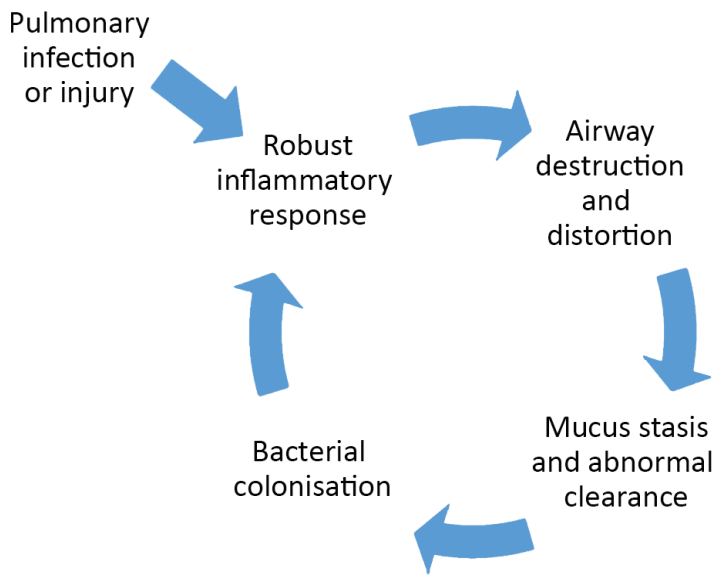


Figure 61: Cole's vicious cycle diagram
Source: Cole PJ. Inflammation: a two-edged sword - the model of bronchiectasis. European Journal of Respiratory Diseases. Supplement 1986; 147:6-15.

Risk factors include cystic fibrosis, underlying immune deficiency and features of primary ciliary dyskinesia, recurrent aspiration or inhaled foreign body. Environmental risk include exposure to tobacco and vape smoke.

Symptoms and signs. Children present with a recurrent wet cough lasting for more than 4 weeks with or without other features (faltering growth, exertional dyspnoea, recurrent chest infections, digital clubbing, hyperinflation and chest wall deformity). Adults have a chronic productive cough with recurrent exacerbations.

Investigations for children include FBC, sweat test for chloride testing, sputum microbiology, spirometry (if aged over 6 years) and chest radiology. For adults, also consider Alpha-1-antitrypsin levels if there is evidence of chronic obstructive pulmonary disease/ emphysema, autoimmune serology, bronchoscopy.

PRINCIPLES OF MANAGEMENT

Management aims to improve symptoms and quality of life, while preserving lung function and reducing exacerbation frequency. In children, an additional aim is to achieve reversibility. The most important elements for primary practitioners are initial recognition of the condition, diagnosis, referral to specialist care, nutrition and minimising further lung injury from environmental pollutants and recurrent infections. Airway clearance techniques are vital, and discussion with, and referral to chest physiotherapists where available, is necessary (See Bronchiectasis Toolbox in the Resources section below). In adults, when there is concurrent airway eosinophilia or concurrent COPD, pharmacotherapy such as inhaled beta-2 agonists and muscarinic antagonists can be considered with airflow obstruction. Inhaled corticosteroids (ICS) should be cautiously used with predominant bronchiectasis. Antimicrobial agents should be considered during exacerbations as per local guidelines.

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call respiratory, infectious diseases or general physician or paediatrician.

MANAGEMENT GUIDELINES

Northern Territory		
Remote Primary Health Care Manuals (RPHCM)	CARPA Standard Treatment Manual ■ Chronic suppurative lung disease and bronchiectasis in children ■ Chronic obstructive pulmonary disease (COPD) and bronchiectasis in adults	Available online
NT Primary Health Network (NT PHN)	HealthPathways - Bronchiectasis in Adults	Available online (CLICK HERE to request access)

RESOURCES

Menzies School of Health Research	The CRE in Lung Health especially for First Nations children	Available online
Bronchiectasis Toolbox	Bronchiectasis Toolbox	Available online
Children's Bronchiectasis Education, Advocacy and Research Network	News & Resources	Available online

FURTHER READING

Chang AB, Bell SC, Byrnes CA, et al. Thoracic Society of Australia and New Zealand (TSANZ) position statement on chronic suppurative lung disease and bronchiectasis in children, adolescents and adults in Australia and New Zealand. Respirology. 2023;28(4):339-349.

Gibbs C, Howarth T, Ticoalu A, et al. Bronchiectasis among Indigenous adults in the Top End of the Northern Territory, 2011–2020: a retrospective cohort study. Med J Aust. 2024;220(4):188-195.

Heraganahally SS, Howarth T, Chen W. A clinical approach to chronic respiratory disorders in 'Aboriginal and Torres Strait Islander in primary care. Aust J Gen Pract. 2024;Supplement 53(12 Suppl):S3-S9.

Howarth T, Gibbs C, Heraganahally SS, Abeyaratne A. Hospital admission rates and related outcomes among adult Aboriginal Australians with bronchiectasis - a ten-year retrospective cohort study. BMC Pulm Med. 2024;24(1):118.

Schultz A, Chang A. Review of bronchiectasis among Aboriginal and Torres Strait Review of bronchiectasis among Aboriginal and Torres Strait Islander children and young people (0-18 years old). Journal of the Australian Indigenous HealthInfoNet. 2025;6(2).

Welford A, McCallum GB, Hodson M, Johnston H. Physiotherapy management of first nations children with bronchiectasis from remote top end communities of the Northern Territory: a retrospective chart audit. Front Pediatr. 2023;11:1230474.

Crocodile bites and water-associated infections

Fatalities from crocodile attacks in northern Australia now outnumber deaths from snakebite and jellyfish envenoming combined.

CROCODILES IN NORTHERN AUSTRALIA

Between 1971 and 2004 there were 62 definite, unprovoked attacks by wild saltwater crocodiles (*Crocodylus porosus*) across northern Australia, with 17 being fatal (10 in the Northern Territory, 5 in Queensland and 2 in Western Australia). Crocodile numbers and individual's size are increasing and there have been further fatalities in the Northern Territory and Queensland since 2004. While saltwater crocodile attacks are predominantly in coastal and estuarine environments, saltwater crocodiles can be found hundreds of kilometres inland in freshwater rivers, such as the Katherine River. The much smaller freshwater crocodile (*Crocodylus johnstoni*) can bite if provoked but has not been associated with fatalities.

AETIOLOGY AND PATHOGENESIS

Death can occur on site from drowning and massive blood loss. In survivors, extensive soft tissue and bone injuries are common from the blunt and shear force trauma as well as from deeply penetrating teeth. Legs and arms are most commonly bitten, but truncal, head and neck bites also occur. As well as compound fractures and devitalised tissue with resulting necrosis, secondary infections and sepsis are common, especially if prehospital wound care is delayed and/or inadequate.

As with other animal bites and penetrating injuries from fish spines or inanimate object such as fishhooks, spears and knives, and with coral cuts, the infecting pathogens (usually bacteria) can be

1. From the surrounding water and soil environment, or
2. From the oral flora of the animal or on the surface of the penetrating spine or object, or
3. From the cutaneous flora of the person, or
4. A combination of these.

The oral flora of crocodiles includes anaerobes and many Gram-negative species including various Pseudomonads.

There is a diversity of marine pathogens in saltwater, with *Vibrio* species such as *V. vulnificus* especially important. Freshwater and brackish water and mud are notable for *Aeromonas hydrophila*.

The human cutaneous flora that are most implicated in many secondary infections are staphylococci (MSSA and MRSA) and streptococci (especially Strep A, *Streptococcus pyogenes*).

Table 6: Risk factors for moderate to severe skin and soft tissue injuries

Risk factors for moderate to severe infections
Presentation to medical care delayed by 8 hours or more
Wound is a puncture wound that cannot be debrided adequately
Wound involved hands, feet, groin or face
Wound involves deeper tissue (such as bones, tendons, joints)
Wound involves an open fracture (refer to Surgical Antibiotic Prophylaxis TEHS Guideline)
Patient is immunocompromised (e.g. asplenia, immunosuppressive therapy, liver disease, iron overload, diabetes, malignancy)

Source: Guideline for Water-Associated Skin & Soft Tissue Infection and Crocodile/Shark Bite (Adults), NT Health. 2023

PRINCIPLES OF MANAGEMENT

Crocodile bites

Prehospital management of crocodile bites includes

1. Lifesaving resuscitation and blood loss control.
2. Meticulous wound care with copious irrigation, with soap and/or disinfectant.
3. Splint fractures and dress wounds with sterile dressings.
4. Commence antibiotics, based on severity (Table 7).
5. Arrange evacuation. In hospital, meticulous and often extensive wound debridement is most important. Because of the extent of wound contamination, delayed primary closure after aggressive initial debridement is often necessary.

Other water-associated infections

As noted above, these infections can be from the environmental organisms and/or the patient's own skin bacteria. They can be mild, moderate or severe and can be restricted to skin with or without soft tissue infection or can result in sepsis which can progress to severe septic shock. Patients with co-morbidities (such as diabetes, chronic kidney disease, chronic liver disease, hazardous alcohol use, immunocompromised) are particularly at risk of more severe water-associated infections such as with *Vibrio* species and *Burkholderia pseudomallei* (melioidosis).

1. Wound care and assessment of whether associated sepsis is present, and evacuation is required.
2. If delayed presentation, take wound swabs and blood cultures (if sepsis present).
3. Commence antibiotics (Table 7).



Figure 62: Saltwater crocodile bite



Figure 63: Freshwater crocodile bite –secondary *Aeromonas hydrophila* infection



Figure 64: *Vibrio vulnificus* bullous cellulitis and severe sepsis in a fisherman with skin saltwater exposure
Source: Bart Currie — Menzies School of Health Research

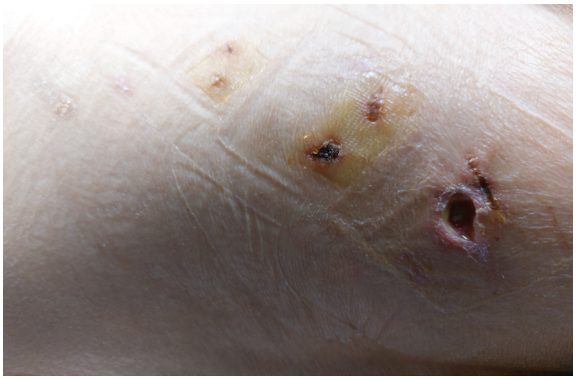


Figure 65: Saltwater crocodile bite



Figure 66: Saltwater crocodile bite - delayed closure after debridement and antibiotics



Figure 67: *Vibrio vulnificus* infection from catfish barb penetrating injury

Crocodile bites and water-associated infections

Table 7: Empiric treatment option for mild, moderate and severe infection

Severity	Criteria	Empiric treatment options (adult doses)
Mild (Seawater)	**NONE OF THE FOLLOWING** - risk factors as listed in Table 6 - infection with systemic features - infection involving deeper tissues - shark or crocodile bite	Doxycycline 100mg orally, 12-hourly AND Dicloxacillin 500mg orally, 6-hourly OR Doxycycline 100mg orally, 12-hourly AND Cefalexin 500mg orally, 6-hourly (if delayed, non-severe penicillin hypersensitivity) **If sewage or soil or mud contaminated wound** Amoxicillin/clavulanate 875/125mg orally, 12-hourly AND Doxycycline 100mg orally, 12-hourly
Mild (Fresh/ brackish or aquarium)	**NONE OF THE FOLLOWING** - risk factors as listed in Table 6 - infection with systemic features - infection involving deeper tissues - shark or crocodile bite	Trimethoprim+sulfamethoxazole 320+1600mg orally, 12-hourly (monotherapy) OR Doxycycline 100mg orally, 12-hourly AND Dicloxacillin 500mg orally, 6-hourly OR Doxycycline 100mg orally, 12-hourly AND Cefalexin 500mg orally, 6-hourly (if delayed, non-severe penicillin hypersensitivity) **If sewage or soil or mud contaminated wound** Amoxicillin/clavulanate 875/125mg orally, 12-hourly AND Doxycycline 100mg orally, 12-hourly
Moderate	**ANY OF THE FOLLOWING** - Risk factors as listed in Table 6 - Infection involving deeper tissues - Shark or crocodile bite	**No systemic features, reliable for clinical follow up** Ciprofloxacin 750mg orally, 12-hourly AND Dicloxacillin 500mg orally, 6-hourly OR Ciprofloxacin 750mg orally, 12-hourly AND Cefalexin 500mg orally, 6-hourly (if delayed, non-severe penicillin hypersensitivity) If sewage OR soil OR mud contaminated wound OR shark or crocodile bite, ADD Clindamycin 450mg orally, 8-hourly - IF extensive wounds or delay beyond 24 hours from time of bite **Any systemic features (but not sepsis or septic shock)** Ciprofloxacin 400mg IV, 8-hourly AND Flucloxacillin 2g IV, 6-hourly OR Ciprofloxacin 400mg IV, 8-hourly AND Cefazolin 2g IV, 8-hourly (if delayed, non-severe penicillin hypersensitivity) If sewage OR soil OR mud contaminated wound OR shark or crocodile bite, ADD Clindamycin 450mg orally, 8-hourly ** Consider de-escalation from IV to oral regimen after 24-48 hours if clear improvement.*
Severe infection (including sepsis and septic shock)		Ciprofloxacin 400mg IV, 8-hourly AND Meropenem 1g IV, 8-hourly (wet season) OR Piperacillin-tazobactam 4.5g IV, 6-hourly (dry season) AND Vancomycin (loading dose 25 to 30mg/kg IV; also chart regular vancomycin IV) If shark OR crocodile bite OR necrotising infection, ADD Clindamycin 600mg IV, 8-hourly

Source: Guideline for Water-Associated Skin & Soft Tissue Infection and Crocodile/Shark Bite (Adults), NT Health. 2023

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call Rural Medical Practitioners (RMP) or Duty Medical Officer (DMO) or medical retrieval service:

- CareFlight (Top End) 1300 655 855
- Medical Retrieval and Consultation Centre (Central Australia) 08 8951 8200

RESOURCES

Northern Territory		
NT Health	Water-Associated Skin & Soft Tissue Infection and Crocodile/Shark Bite (Adults)	Available online
NT Government	■ Crocodiles ■ Management Program for the Saltwater Crocodile (Crocodylus porosus) in the Northern Territory of Australia 2024-2034 ■ Be Crocwise	Available online

FURTHER READING

Caldicott DG, Croser D, Manolis C, et al. Crocodile attack in Australia: an analysis of its incidence and review of the pathology and management of crocodilian attacks in general. Wilderness Environ Med. 2005;16(3):143-159.

Fukuda Y, Manolis C, Saalfeld K, Zuur A. Dead or Alive? Factors Affecting the Survival of Victims during Attacks by Saltwater Crocodiles (Crocodylus porosus) in Australia. PLoS One. 2015;10(5):e0126778.

SECTION 2 — VIRUSES, SPIROCHETES, CHLAMYDIAL AND VECTOR BORNE DISEASES

Mosquito-borne diseases

Murray Valley encephalitis, West Nile (Kunjin strain) virus, Ross River fever, and Barmah Forest fever are endemic to Northern Australia. Japanese encephalitis had occasionally occurred in the Torres Strait Islands and adjacent Cape York (probably from wind-blown mosquitoes), but then in 2022 unexpectedly caused devastating outbreaks in commercial piggeries across southeast Australia, with also human cases and fatalities. Dengue outbreaks occasionally still occur in Far North Queensland. Australia remains free of endemic malaria.

MOSQUITO BORNE DISEASES IN NORTHERN AUSTRALIA

Mosquito-borne diseases have a prominent role in clinical and public health practice in Northern Australia. Detailed travel history is a crucial component of history for any patient presenting with fever. Mosquito vectors are present throughout the region for Murray Valley encephalitis (MVE) and West Nile (Kunjin strain) viruses, malaria, Ross River virus (RRV), Barmah Forest virus (BFV) and Japanese encephalitis (JE) virus.

***Aedes aegypti* and *Aedes albopictus*, the mosquito vectors for dengue fever, Chikungunya and Zika are present in Far North Queensland and the Torres Strait Islands respectively, but not currently in the Northern Territory (except for a Tennant Creek focus of *Ae. aegypti* being actively managed for elimination) or Western Australia. The re-introduction or spread of Aedes mosquito vectors for dengue into the Northern Territory or Western Australia are a constant concern. *Aedes aegypti* that carry the symbiotic bacteria, Wolbachia, do not transmit dengue as well as wild type mosquitoes. Their successful introduction in North Queensland has been associated with decreased dengue transmission in the area.**

Mosquitoes capable of carrying dengue, Chikungunya and Zika viruses are present in Timor-Leste, Indonesia, Papua New Guinea and North Queensland and imported cases of these arboviruses and of malaria can occur from these northern neighbour countries into anywhere in Australia.

MVE occurs sporadically throughout Northern Australia with the highest incidence in the northern parts of Western Australia and the Top End of the Northern Territory, occasional cases in central Australia, and exceptional outbreaks in southern Australia such as occurred in 2023 following La Niña-associated heavy rainfall. JE virus transmission had occurred in some years on the northern Torres Strait Islands, presumably secondary to incursion of wind-blown mosquitoes from countries to the north, with rare incursions into the Cape York region. In February 2021 there was a fatal case of JE from the Tiwi Islands, which was followed in February 2022 by outbreaks on commercial piggeries in NSW, Victoria and Queensland, with human cases in those States and also South Australia. It was declared a Communicable Disease Incident of National Significance (the first since COVID-19). By winter 2022 there had been over 80 piggeries affected and 42 human cases nationally (7 fatal). All recovered JEV isolates, including from the sentinel 2021 Northern Territory case, were Genotype 4. How the virus entered northern Australia remains unknown, but the spread southward was linked to the heavy rainfall and postulated to involve a combination of infected mosquitoes and birds migrating southward, with also virus amplification in feral pigs. Despite predictions of JEV becoming endemic in Australia, there were no further cases in Australia in 2023 or 2024, while in 2023 there were 8 MVE cases (2 fatal) from the Northern Territory. However, JE cases have re-emerged in early 2025 in the four eastern states, supporting that JEV has become endemic in Australia.

RRV and BFV infections are relatively common and occur each wet season.

Health authorities are alert to the possibility of the introduction of Chikungunya and Zika viruses into areas where their mosquito vectors occur. In 2024, Oropouche virus, another arbovirus, was added to that list following massive outbreaks in the Americas.

Occasional limited transmission of malaria occurs in Far North Queensland and the Torres Strait Islands, but without re-establishment of endemic malaria. The last case of malaria acquired in the Northern Territory was in Roper River in 1962. The patchy but widespread presence of the *Anopheles* mosquito vectors in Northern Australia means that sporadic transmission is likely to continue to occasionally occur. Each person notified with malaria is investigated to determine where the disease was acquired, and whether there is a risk of transmission to local mosquitoes. Ongoing vector surveillance is conducted at airports and wharf areas.

AETIOLOGY AND PATHOGENESIS

Malaria in humans is caused by one of at least six *Plasmodium* parasites. *Plasmodium falciparum* is the more serious form of the disease that causes cerebral malaria whilst *Plasmodium vivax* can relapse due to a dormant liver stage. *Plasmodium malariae* and *Plasmodium ovale* are much less common. *Plasmodium knowlesi* is now the most common malaria species in Sabah, Malaysia. The parasites cause fever and chills when they rupture out of red cells, and organ-specific pathology by microvascular occlusion. *P. falciparum* can infect all red cells whilst *Plasmodium vivax* infects mainly young red cells, which means the former can achieve much higher parasite counts.

Arboviruses fall into two main groups:

- *Alphaviruses:* RRV and BFV can also infect other species such as macropods. The arthritis and rash are caused by immunological responses to infection, with rash usually more prominent with BFV and arthritis usually more prominent with RRV. Chikungunya is also an alphavirus.
- *Flaviviruses:* dengue, MVE, West Nile (Kunjin strain in Australia), JE and Zika viruses. Dengue only occurs in humans and there are four viral serotypes. Infection with a particular serotype results in lifelong immunity to that serotype. Macrophage and monocyte infection are the most important aspects of the pathogenesis of dengue fever and the more severe complicated dengue with haemorrhagic fever and/or dengue shock syndrome. In the more severe forms, cross reactivity from previous infection with different serotypes leads to 'priming' of the immune system and subsequent release of cytokines. This causes the characteristic increased vascular permeability and bleeding seen in severe dengue.

MVE and West Nile (Kunjin strain) viruses also infect wildlife including certain waterbirds. Western Australia and the Northern Territory have a viral monitoring program, historically through serology of sentinel chicken flocks, but with the Northern Territory having retired the chickens and now using PCR detection of viral RNA in trapped mosquitoes. The main JE virus host is pigs. The encephalitis caused by these flaviviruses in humans follows a viraemia and direct neuronal infection. Symptoms and sequelae are partly caused by the infection and partly from the secondary oedema and inflammatory response.

CLINICAL PICTURE

Malaria can be a severe illness and requires urgent medical intervention. Fever is the most common presentation. Other features include headache, myalgia, and sometimes vomiting and diarrhoea. Rash is not usually a feature. Blood smear remains the gold standard for diagnosis. Rapid antigen tests are also available, to complement but not replace, microscopy with sensitivities of over 95% for *P. falciparum*, but only 70% for *P. vivax*. PCR is now also used to detect malaria DNA.

Table 8: Aetiology, vector and immunity in mosquito-borne diseases in Northern Australia

Organism	Main vector or potential vector	Vector distribution	Immunity after infection	Illness
Plasmodium spp.	*Anopheles farauti*	Patchy throughout Northern Australia	No	Malaria
Dengue virus (serotypes I-IV)	*Aedes aegypti* *Aedes albopictus*	North Queensland	Yes (serotype specific)*	Dengue fever, dengue haemorrhagic fever and shock
West Nile (Kunjin strain) virus	*Culex annulirostris*	Common throughout Northern Australia	Probable	Rare, encephalitis uncommon
Murray Valley encephalitis (MVE) virus	*C. annulirostris*	Common throughout Northern Australia	Probable	Moderate to severe encephalitis, death, residual disability
Japanese encephalitis (JE) virsu	*C. annulirostris, Culex gelidus*	Common throughout Northern Australia	Probable	Moderate to severe encephalitis, death, residual disability
Ross River virus (RRV)	*Aedes vigilax, C. annulirostris*	Common throughout Northern Australia	Probable	Ross River fever, polyarthritis and rash, fatigue
Barmah Forest virus (BFV)	*Aedes vigilax, C. annulirostris*	Common throughout Northern Australia	Probable	Barmah Forest fever, rash often more prominent than with Ross River virus, and arthritis less prominent

* risk of dengue haemorrhagic fever if infected with another serotype

Mosquito-borne diseases

BFV and **RRV infection** have similar clinical pictures, although rash is more common in the former and arthritis in the latter. Both diseases are self-limiting with arthralgia lasting from days to months mostly affecting the wrist, knee, ankle, hands and feet. In many patients, the onset of arthritis is followed by a maculopapular non-pruritic rash mainly affecting the trunk and limbs. The rash resolves within seven to 10 days, followed by a fine desquamation.

Dengue also known as 'break bone fever' commonly presents with back pain, myalgia, retro-orbital pain and headache accompanying the sudden onset of fever. Typically, rash occurs a few days into the illness, affecting the whole body.

It can be maculopapular, petechial or confluent erythema that blanches with pressure. Complicated dengue with haemorrhagic fever/dengue shock syndrome may occur in people who have previously been infected with another serotype of dengue. Dengue haemorrhagic fever has an abrupt onset characterised by fever, lymphadenopathy, hepatomegaly, scattered petechiae and rapid deterioration due to intravascular fluid loss.

MVE, West Nile (Kunjin strain) and **JE** virus infections that when symptomatic are manifested by fever, headache, seizures especially in children and sometimes nausea and vomiting, rapidly followed by the features of encephalitis. It has been estimated from serological studies that only 1:1000 people infected with MVE virus become symptomatic for the disease. The rate of symptomatic infection with JE virus is in the range of 1:30–300. These estimates remain speculative. Children are particularly susceptible to the severe form of the disease. Symptomatic infections are associated with high rates of mortality (up to 33%) and neurological sequelae (up to 33%).

There are at least seven genetic lineages of West Nile virus. West Nile (Kunjin strain) is a small but genetically distinct sublineage, designated as 1b. The epidemics of severe disease in humans and animals during the past decade have been due to West Nile Virus sublineage 1a, which has never been found in Australia. Prior to 2010, West Nile (Kunjin strain) was known as Kunjin virus.

Diagnostic investigation of mosquito-borne infections

Malaria: If clinical suspicion for malaria, send EDTA for

1. Thick and Thin films (via Haematology).
2. Malaria Antigen rapid diagnostic test (RDT) (via Haematology).
   - Using Abbott Bioline™ Malaria Ag P.f/Pan test [detection of histidine-rich protein 2 (HRP2) antigen of *Plasmodium falciparum* and common *Plasmodium lactate* dehydrogenase (pLDH) of *Plasmodium* species].
3. Mosquito-borne panel for nucleic acid detection (done if number 1 or 2 positive, or if high suspicion; most sensitive test).

Dengue: For dengue, the detection of the NS1 protein in blood, is sensitive, specific and readily available — but cannot be used to determine serotype. After 7–10 days the diagnosis relies on detection of antibody in blood.

If clinical suspicion for Dengue, send Serum/ EDTA for

1. Lateral Flow Assay for Dengue NS1 Antigen
   - If negative and strong clinical suspicion, discuss with the laboratory.
2. Mosquito-borne panel for nucleic acid detection.

Arboviruses (apart from dengue): In the early symptomatic stage of most arboviral infections direct detection of virus nucleic acid from blood (EDTA/ serum), CSF and/or urine is the most appropriate method of diagnosis. Isolation of the virus by culture is available in reference laboratories. RT-PCR, however, is more sensitive and can be used for detection in the first seven days. Serology responses to flaviviruses cross react with each other — making a specific diagnosis specialised, especially in separating MVE from JE. Serological diagnosis of alphaviruses (BFV and RRV) is also problematic and false positive IgM results are relatively common. For both flaviviruses and alphaviruses, in the absence of viral detection, confirmation of the diagnosis requires both the presence of a clinically suggestive illness and compatible serology, preferably paired sera to show a rising titre. Repeat serological testing is required for up to 6- 12 months to establish a diagnosis in some cases.

If clinical suspicion for arbovirus (apart from dengue):

1. Mosquito-borne panel for nucleic acid detection.
   a. Send EDTA/ Serum/ Urine/ CSF.
   - Send all four specimen types on all patients (apart from Dengue and Malaria).

- Will be run on CSF if WCC / protein elevated and/ OR clinical indication.

b. This panel includes:

- Dengue i-iv RNA (see below)
- Ross River Virus RNA
- Chikungunya RNA
- Barmah Forest RNA
- West Nile Virus RNA
- Zika Virus RNA
- Murray Valley Encephalitis RNA
- Japanese Encephalitis RNA
- Kunjin Virus RNA
- Malaria (see below)

2. If Mosquito-borne panel is POSITIVE then samples (excluding dengue/ malaria) will be sent to reference laboratory (Westmead) for repeat PCR + serology.

If clinical suspicion for **Oropouche,** discuss with lab.

If clinical suspicion for **Mpox***

1. In house PCR assay (screens for all orthopox viruses and distinguishes Mpox).
   - Swab from base of lesion fluid.
   - Lesion specimens are preferred; however, throat swabs are also suitable specimens if a lesion swab is not possible.
   - Rectal swab if proctitis.

*note syphilis and Mpox swabs need to be collected separately because they are sent to different reference laboratories

PRINCIPLES OF MANAGEMENT

All the diseases described in this section, apart from those caused by RRV and BFV, require expert advice from an infectious diseases physician. Malaria is the only disease which has specific treatment. Anti-malarial drug resistance is common worldwide, and it is important that current protocols are consulted prior to commencing treatment. Hospital assessment is required for all cases of *P. falciparum, P. malariae, P. knowlesi* and malaria cases where the species cannot be confirmed within 24 hours. As *P. falciparum* can be life-threatening this allows specialist assessment and observation of first doses of antimalarials. Where gametocytes are present this initial management is in an environment protected from mosquitoes to minimise the risk of transmission of the parasite to local mosquitoes.

Follow-up is then arranged through 'Hospital in the Home' when parasitaemia is low and other criteria are met. *P. vivax* malaria can be managed as an outpatient



Figure 68: Barmah Forest virus arthritis and rash
Source: Bart Currie — Menzies School of Health Research



Figure 69: Barmah Forest virus rash
Source: Bart Currie — Menzies School of Health Research



Figure 70: Dengue blanching rash — acquired in Timor Leste
Source: Bart Currie — Menzies School of Health Research

Mosquito-borne diseases

Treatment of severe dengue, or dengue with warning signs (refer to the American CDC Dengue Case Management Guidelines) requires close monitoring and intravenous fluid administration in hospital. Aspirin and other NSAIDs should not be taken for suspected or confirmed dengue fever. Uncomplicated dengue and other viral infections have no specific treatment although paracetamol, fluids and rest offer some relief of symptoms.

MVE and JE require admission to hospital for close monitoring and supportive therapy. Effective vaccines for the prevention of JE are used in the northern Torres Strait Islands and were distributed in 2022 to selected populations in JE-implicated locations. Patient advice is important for RRV and BFV infections. Queensland Health and CDC Darwin have excellent fact sheets available on their website — see *Educational Resources* page 69.

Public health surveillance and management are coordinated through regional public health services:

- Urgent notification is required for suspected cases of dengue fever, malaria, MVE, Kunjin, JE and Zika infection. This leads to prompt investigation including identification of the place where the infection was acquired, mosquito control measures, contact tracing, and if necessary, trapping and testing of mosquitoes close to the location of the case.
- New technologies with virus nucleic acid detection by PCR from mosquito traps in selected locations has replaced the sentinel chickens in the Northern Territory.
- Public health warnings are given when mosquito numbers and/or clinical cases increase above the expected levels and/or mosquito surveillance tests positive, especially for MVE and/or JE virus.

Remember: In cases of fever in Northern Australia, always take a travel history — 'Where have you been and when?'



Figure 71: Mpox – visitor from Africa, indicating the importance of travel history
Source: Bart Currie — Menzies School of Health Research

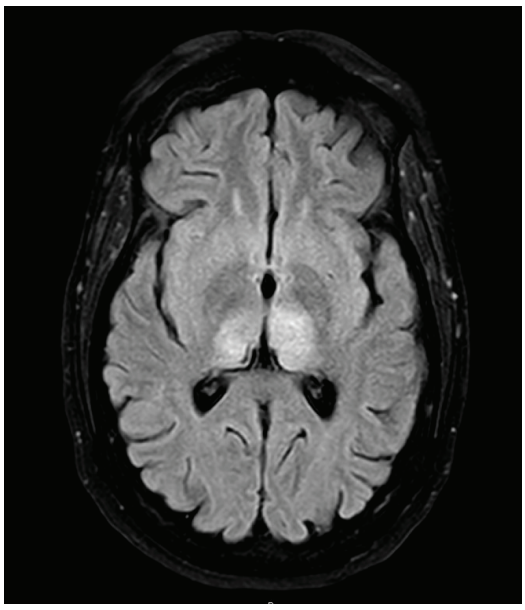


Figure 72: Fatal Japanese encephalitis with intense bilateral thalamic inflammation on MRI
Source: Bart Currie — Menzies School of Health Research

Table 9: Summary of clinical and diagnosis of vector borne diseases

Illness	Fever	Rash	Other symptoms	Diagnostic test
Malaria	+	-	Headache, chills, rigors	Blood film, antigen test, PCR
Dengue	+	+	Retro orbital headache, myalgias	RT–PCR, NS1, Serology, FBC and LFT (incr AST/ALT) (↓ WCC, platelets)
Ross River virus (RRV), Barmah Forest virus (BFV)	+/-	+/-	Arthralgia/arthritis	Serology (negative if done too early)
Murray Valley encephalitis (MVE), West Nile (Kunjin strain) Japanese encephalitis (JE)	+	-	Altered mental state, seizures, focal CNS signs, coma	CSF, blood and urine RT–PCR, CSF and blood serology

These diseases are nationally notifiable. All suspected cases of MVE virus, West Nile (Kunjin strain) virus, JE virus, malaria, dengue fever, Chikungunya and Zika should be notified by CLINICIANS and LABORATORIES. Cases of BFV and RRV, are notifiable by LABORATORIES only. **Report cases urgently by telephone** to the local Centre for Disease Control/Public Health Unit.

Mosquito-borne diseases

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call infectious disease physician or local CDC/PHU.

Contact the local medical entomologist.

MANAGEMENT GUIDELINES

International		
US Department of Health and Human Services	Response to Dengue Cases in Non-Endemic Areas of the United States	Available online
National		
Communicable Diseases Network Australia (CDNA)	■ National Guidelines for Public Health Units: > Dengue > Murray Valley encephalitis > Zika > Mpox ■ Australian notifiable diseases case definitions	Available online
Therapeutic Guidelines	Antibiotics: Malaria	Available online (Login required)
Northern Territory		
NT Centre for Disease Control (CDC)	■ Guidelines for Malaria ■ Public Health and Notifiable Diseases	Available online
NT Primary Health Network	HealthPathways - Mosquito-borne Diseases	Available online (CLICK HERE to request access)

EDUCATIONAL RESOURCES

NT Centre for Disease Control (CDC)	Fact sheets — Ross River virus, Mosquito-borne diseases, Murray Valley encephalitis, Japanese encephalitis, Dengue, Chikungunya, Barmah Forest	Available online

FURTHER READING

Floridis J, McGuinness SL, Kurucz N, et al. Murray Valley encephalitis virus: an ongoing cause of encephalitis in Australia's north. Tropical Medicine and Infectious Disease. 2018;3(2):49.

Furuya-Kanamori L, Gyawali N, Mills DJ, et al. The Emergence of Japanese Encephalitis in Australia and the Implications for a Vaccination Strategy. Trop Med Infect Dis. 2022;7(6):85.

Gray TJ, Trauer JM, Fairley M, et al. Imported malaria in the Northern Territory, Australia--428 consecutive cases. Commun Dis Intell Q Rep. 2012;36(1):107-113.

Knope KE, Doggett SL, Jansen C, et al. Arboviral diseases and malaria in Australia, 2014-15: Annual report of the National Arbovirus and Malaria Advisory Committee. Communicable Diseases Intelligence Quarterly Report. 2019;43(1):1-69.

Proudmore K, Krause VL, Currie BJ, Baird R. Fallibility and flaviviruses: a diagnostic lesson in Japanese encephalitis. Med J Aust. 2023;219(6):253-254.

Waller C, Tiemensma M, Currie BJ, et al. Japanese Encephalitis in Australia - A Sentinel Case. N Engl J Med. 2022;387(7):661-662.



Scrub typhus

Scrub typhus is a type of rickettsial infection caused by bacteria transmitted via mite bite in wet forested areas of tropical Northern Australia.

SCRUB TYPHUS IN NORTHERN AUSTRALIA

Scrub typhus is endemic to the Asia-Pacific and Northern Australia and is characteristically patchy in distribution (mirroring the mite vector habitats). Compared to other countries, scrub typhus is relatively uncommon in the Northern Territory. There were ten serologically proven notifications between 2019 and 2024 in the Northern Territory, with half acquired overseas. The Top End has seen one fatality and several severe cases.

Scrub typhus was previously more common; the reduction is speculated to be from improved national park maintenance and less visitor exposure to mites. Most Northern Territory cases have occurred in workers or visitors to rainforest areas within Litchfield Park south-west of Darwin, but other foci of scrub typhus have also been found.

Far North Queensland, including the Torres Strait Islands, and Western Australia have also reported cases of scrub typhus. Clusters of cases have also occurred in military personnel undertaking training in coastal sites close to Innisfail, North Queensland.

AETIOLOGY AND PATHOGENESIS

Scrub typhus is a zoonotic infection in which humans are accidental hosts. The main causative organism is *Orientia tsutsugamushi* (previously known as *Rickettsia tsutsugamushi*), a gram-negative obligate intracellular bacterium. The bacterium is transmitted to humans by the trombiculid mite (chigger), *Leptotrombidium deliense*, which acts as both a vector and reservoir.

The infection is maintained in the mite population via transovarial vertical transmission. The mites rely on small rodents for blood meals. In humans, bacteria proliferate at the site of the mite bite before disseminating throughout the body. The incubation period is 5–18 days.

CLINICAL PICTURE

Risk factors include time and exposure in endemic areas, particularly walking and camping.

Symptoms and signs include an inoculation eschar (black scab) of approximately 1cm in diameter at the bite site, which is often found in warm moist areas, such as the buttocks, genitalia, lower trunk or armpit. The eschar may go unnoticed by the patient or may look like an ulcer. Occasionally no eschar can be found, even on full body inspection. Symptoms are largely non-specific including fever, regional lymphadenopathy, myalgias, rash and severe headache. The rash is typically maculopapular and truncal in distribution. In a small number of patients, the disease may be severe with delirium, tremors, slurred speech and multi-organ failure. Severe disease mimics a widespread (i.e. systemic) small vessel vasculitis.

Investigations. Serology (indirect microimmunofluorescence test for *O. tsutsugamushi*) should be performed on serum taken at presentation and two weeks later. A fourfold rise in titre is diagnostic. Eschar material should be obtained by punch biopsy, and PCR is rapid and relatively sensitive.



Figure 73: Scrub typhus eschar on buttock
Source: Bart Currie — Menzies School of Health Research



Figure 74: Scrub typhus eschar and multi-organ failure requiring dialysis
Source: Bart Currie — Menzies School of Health Research

DIFFERENTIAL DIAGNOSIS

The differential diagnosis is broad. The non-specific symptoms of fever, myalgia and headache can be due to other rickettsial diseases, viral infections, toxoplasmosis, leptospirosis, melioidosis, Ross River virus infection, Murray Valley encephalitis or bacterial sepsis. The patient's travel history determines the likelihood of other infections, such as dengue and typhoid.

PRINCIPLES OF MANAGEMENT

Management depends on the severity of infection. In mild disease, oral doxycycline is recommended and is usually rapidly effective with a lower risk of relapse. Duration of therapy is guided by clinical response but is usually seven days. In more severe disease with multi-organ failure, hospital-based supportive therapy is required, and recovery is sometimes delayed. Combination therapy with intravenous azithromycin and doxycycline has been shown in one study to be superior to monotherapy for severe disease. Antibiotic resistance is rare.

Mortality in untreated cases varies but can be up to 70% in severe cases. Prompt therapy may be lifesaving, so consider and test for scrub typhus in patients with flu-like illnesses and a history of environmental exposure from an endemic area. Do not wait for confirmation before commencing empiric therapy.

Prevention includes maximising skin coverage by wearing tall footwear and long trousers in high-risk areas. Use N-diethylmeta-toluamide (DEET) containing repellents on skin and socks/trousers with a groundsheet for sitting. Doxycycline can be considered for prophylaxis for those at particularly high risk, such as soldiers on field patrol.

> Scrub typhus is a notifiable condition to be reported by LABORATORIES in the Northern Territory. Report cases to the local Centre for Disease Control/Public Health Unit.

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call infectious disease physician or local CDC/PHU.

Contact the local medical entomologist.

MANAGEMENT GUIDELINES

NT Centre for Disease Control (CDC)	Fact sheet — Scrub Typhus	Available online

FURTHER READING

Frances SP. Rickettsial diseases of military importance: an Australian perspective. Journal of Military and Veterans' Health. 2011;19(4).

Kala D, Gupta S, Nagraik R, et al. Diagnosis of scrub typhus: recent advancements and challenges. 3 Biotech. 2020;10(9):396.

Ralph A, Raines M, Whelan P, Currie BJ. Scrub typhus in the Northern Territory: exceeding the boundaries of Litchfield National Park. Communicable Diseases Intelligence Quarterly Report. 2004;28(2):267-269.

Leptospirosis

Leptospirosis is an acute generalised infectious disease caused by the spirochaete *Leptospira*.

LEPTOSPIROSIS IN NORTHERN AUSTRALIA

The average notification rate of leptospirosis in Australia is 0.66/100,000 population/year in Australia (1991–2023), with Queensland having the highest average notification rate of 2.1/100,000 population. The Northern Territory has the second highest average notification rate of 1.62/100,000 population.

Activities associated with cases include living or working on farms and rural blocks, working in abattoirs or in the crocodile industry, hunting of animals such as duck, goose and turtle, and water-based recreational activities.

AETIOLOGY AND PATHOGENESIS

Leptospirosis occurs worldwide but is more common in tropical and subtropical environments with most cases occurring during the wet season. Many mammals carry *Leptospira* — vectors include rodents, bats, marsupials, cattle, pigs, and dogs.

There are 17 species pathogenic to humans. Humans are infected through contact of abraded skin or mucous membranes with urine and body fluids of infected animals or soil contaminated by infected animals. Human cases are rarely infectious to others.

After penetration of the skin or mucous membrane, leptospires migrate throughout the body, including to the cerebrospinal fluid, muscle, lung, liver and kidneys, causing inflammation, vasculitis and immune mediated damage. The incubation period in humans is usually 5–14 days.



Figure 75: Leptospirosis conjunctival injection
Source: Bart Currie — Menzies School of Health Research

CLINICAL PICTURE

Risk factors. Occupational/recreational exposure to urine or fluids/tissues from infected animals (e.g. fruit farm workers, cattle station workers, veterinarians, crocodile egg collectors). Skin exposure to contaminated soil or vegetation (e.g. bush walking) or waters (e.g. swimming or wading in creeks or floodwaters).

Symptoms. The severity of illness varies. Approximately 90% of cases are self-limiting , with 10% exhibiting a more severe and potentially fatal illness. The latter is characterised by any combination of kidney or liver injury, and pneumonitis associated with pulmonary haemorrhage. Mortality rates with severe disease range from 5%–40%.

The illness most often presents acutely. In severe cases there is rapidly progressive pulmonary haemorrhage often with acute renal failure. The disease can be biphasic, although a clear demarcation between the two phases is uncommon. Defervescence for a day or two may even occur between phases. The first phase lasts 5–7 days and the second phase may last up to 30 days after onset.

Early symptoms of mild illness are flu-like with marked fever and myalgia. Headache, abdominal pain, respiratory symptoms, vomiting and diarrhoea may also be present. Symptoms in the second phase may be headache, photophobia, eye pain, jaundice, muscle pain, respiratory symptoms and haemoptysis.

Signs. In the first phase of illness physical signs may be minimal. Conjunctival suffusion (redness without exudate) is often present and should raise the possibility of leptospirosis. Muscle tenderness is common and in severe disease, reduced urine output, signs of pneumonitis and pulmonary haemorrhage and signs of myocarditis may be present. Lymphadenopathy, splenomegaly, hepatomegaly and a maculopapular pretibial rash may also occur. If a second, immune phase occurs, then possible features include fever, headache, jaundice, signs of meningitis, uveitis, hepatomegaly, splenomegaly, myocarditis and pulmonary symptoms.

Investigations. Routine laboratory tests are non-specific. They include an elevated CRP/ESR, a low to normal WCC with left shift (but both neutrophilia and pancytopaenia are possible). Thrombocytopaenia can occur. LFTs often show a moderate elevation in transaminases and bilirubin. Serum creatine kinase is usually raised; elevated muscle creatine kinase is an important indicator. Renal function tests may show renal impairment. Hypokalaemia and hyponatraemia are common. Cerebrospinal fluid may show an aseptic meningitis picture with a lymphocytic pleocytosis. In severe disease, chest X-ray changes include modular densities and progressive confluent consolidation or ground glass appearance consistent with pulmonary haemorrhage.

Appropriate leptospirosis directed testing depends on the timing of the infection (Figure 78). During the first week leptospires are present in the blood and can be detected by inoculating special culture media (contact local microbiology laboratory) with several drops of aseptically collected whole blood. This is incubated for up to six weeks and it usually takes 1–2 weeks for organisms to be seen under dark field microscopy.

Leptospires can also be detected in blood by a nucleic amplification method (contact local microbiology laboratory). This is usually by PCR and is more rapid but less sensitive than culture. In the second week of illness serology tests have better sensitivity. IgM antibodies are detectable within 5–10 days of illness onset but the IgM test can be false positive for a number of reasons. A positive IgM result should not be considered definitive evidence of current leptospirosis.

There is no leptospire specific IgG test. The equivalent test to demonstrate seroconversion is the microscopic agglutination test. This reports a titre and an infecting serovar. Commonly there are cross reacting antibodies against a number of serovars. The microscopic agglutination test is often negative on first testing and should be repeated 10–14 days after the initial test to demonstrate a rise in antibody titre.

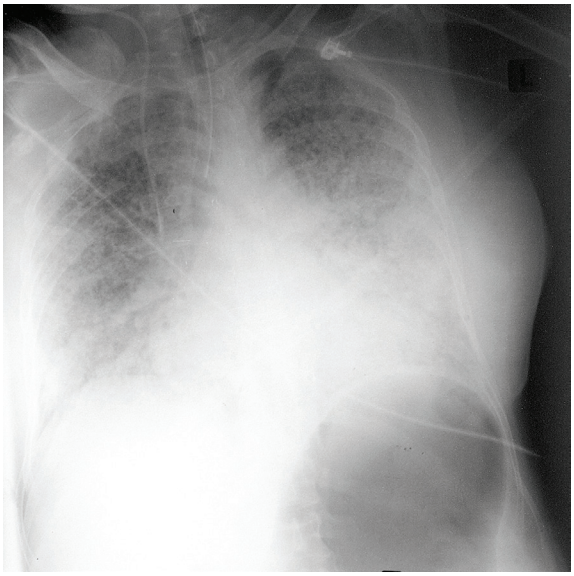


Figure 76: Severe leptospirosis with pulmonary haemorrhage
Source: Bart Currie — Menzies School of Health Research

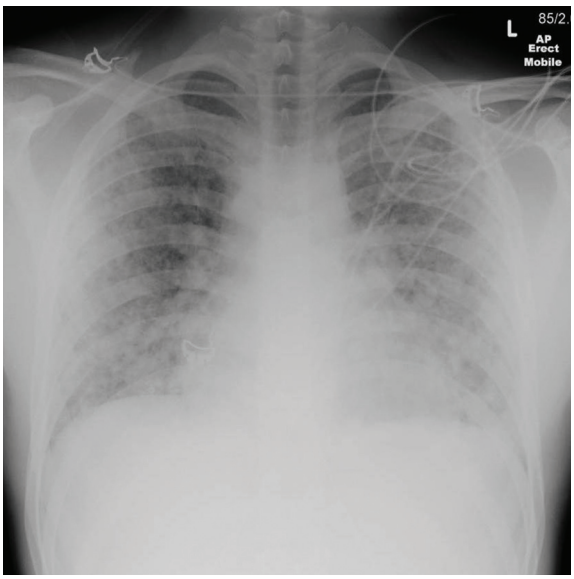


Figure 77: Nineteen-year-old with leptospirosis severe pneumonia
Source: Bart Currie — Menzies School of Health Research

Leptospirosis

DIFFERENTIAL DIAGNOSIS

The differential diagnosis is broad as leptospirosis symptoms tend to be non-specific and variable. For early or mild disease leptospirosis should be differentiated from other febrile illnesses associated with headache and muscle pain such as influenza, scrub or tick typhus, viral hepatitis, Q fever, brucella, melioidosis, arbovirus infections, HIV, syphilis, cytomegalovirus or Epstein Barr virus infections. In travellers also consider malaria, typhoid, dengue, Hantavirus infections, and other rickettsial diseases. Immune mediated conditions such as systemic lupus erythematosus should be included in the differential diagnosis.

The important pointers to leptospirosis are an occupational/recreational exposure risk history and a flu-like illness with disproportionately severe myalgia and headache.

PRINCIPLES OF MANAGEMENT

Prevention. The risk of acquiring leptospirosis can be reduced by not swimming or wading in water that might be contaminated with animal urine and eliminating contact with potentially infected animals. Protective clothing or footwear should be worn by those exposed to contaminated water or soil because of work or recreational activities. Vaccination of domestic and companion animals is available. Annual revaccination is needed, and animals may still excrete leptospires in urine despite not developing overt clinical disease.

There is no clear evidence for chemoprophylaxis (before or after possible exposure to leptospires). Oral doxycycline 200mg per week prevented infection in young male army recruits training in a highly endemic area in one study, however this was not replicated in other studies. Doxycycline is associated with photosensitivity and is contraindicated in pregnancy. Expert opinion from an infectious diseases physician should be sought if chemoprophylaxis is considered.

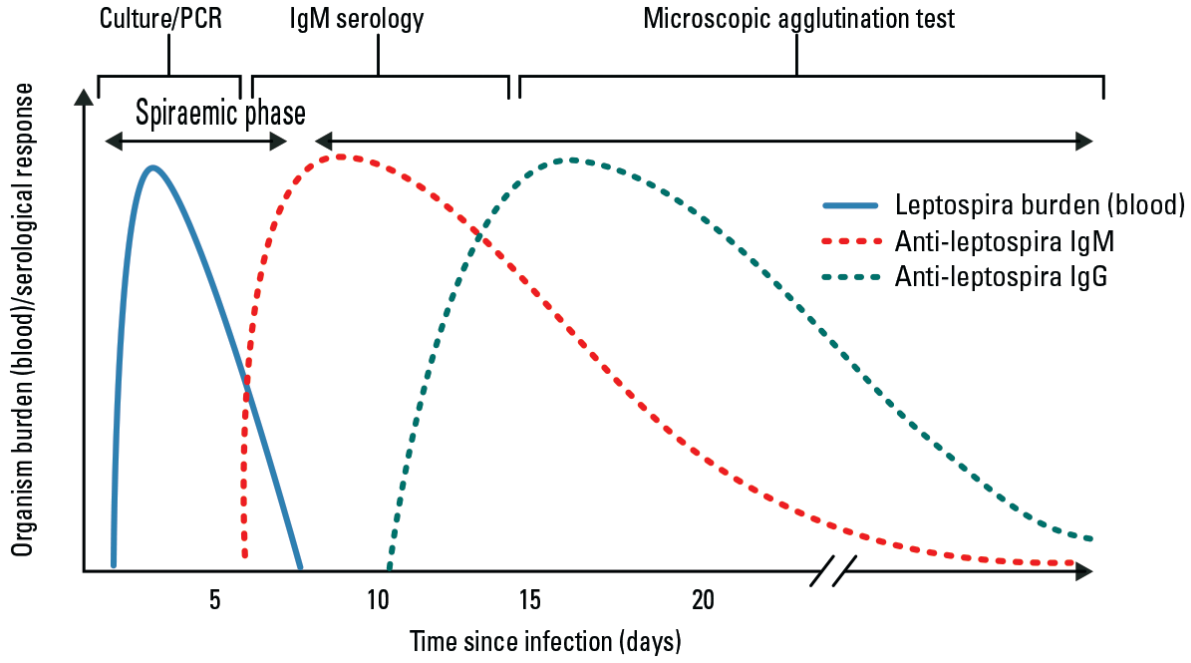


Figure 78: Prototypic antibody response in Leptospirosis against time. Appropriate diagnostic testing for leptospirosis is dependent on accurate timing of infection.

Source: The Royal Australian College of General Practitioners from Slack A. Leptospirosis. Australian Family Physician 2010;39(7):495–498. Reproduced with permission.

Treatment includes supportive treatment and antibiotic therapy. Refer to Therapeutic Guidelines: Antibiotic for details. If leptospirosis is suspected, start treatment before the diagnosis is confirmed. Patients who recover before the diagnosis is made do not require antibiotic treatment. Generally, doxycycline is the preferred empirical treatment as it also covers rickettsial infections with similar presentations. Amoxicillin has been used in an adult oral dose of 500mg 4 times a day where doxycycline cannot be used, as has ceftriaxone.

For more severe disease, referral to hospital is required for intravenous antibiotics and supportive therapy for electrolyte disturbance, hepatic/renal failure, respiratory distress/haemorrhage or hypotension. Intravenous benzylpenicillin/ampicillin and ceftriaxone are equally efficacious. Jarisch-Herxheimer reactions which manifest as fevers, rigors, and hypotension following antibiotic therapy have been reported in patients treated with penicillin. Seek expert advice.

> Leptospirosis is a nationally notifiable condition to be reported by CLINICIANS and LABORATORIES. Report cases to the local Centre for Disease Control/Public Health Unit.

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call infectious disease physician or local CDC/PHU.

MANAGEMENT GUIDELINES

Northern Territory		
NT Centre for Disease Control (CDC)	Public Health and Notifiable Diseases	Available online

RESOURCES

NT Centre for Disease Control (CDC)	Fact sheet —Leptospirosis	Available online

FURTHER READING

Brown DR, Peiris R, Waller C, et al. Communicable Diseases Intelligence 2022 - An outbreak of leptospirosis associated with cattle workers during the wet season, in the Northern Territory of Australia, 2021. Commun Dis Intell (2018). 2022;46.

Smith S, Kennedy BJ, Dermedgoglou A, et al. A simple score to predict severe leptospirosis. PLoS Negl Trop Dis. 2019;13(2):e0007205.

Stark AM, Nohrenberg M, Draper AD, et al. A cluster of leptospirosis cases associated with crocodile workers in the Northern Territory, Australia, 2022. Commun Dis Intell 2018. 2023;47.

Win TZ, Perinpanathan T, Mukadi P, et al. Antibiotic prophylaxis for leptospirosis. Cochrane Database Syst Rev. 2024;3(3):CD014959.

Trachoma

Chlamydia trachomatis is an intracellular bacterium that causes trachoma, which is the leading infectious cause of blindness world-wide.

TRACHOMA IN NORTHERN AUSTRALIA

Trachoma is endemic in 51 countries and has caused visual impairment in about 1.8 million people, of whom 0.5 million are irreversibly blind. In Australia trachoma remains an important cause of blindness in First Nations people. Elimination targets for trachoma are detailed in the global strategy for neglected tropical diseases: Ending the neglect to attain the Sustainable Development Goals; A road map for neglected tropical disease 2021-2030.

Treatment programs started in the Northern Territory in the 1980s following surveys that showed a disease prevalence of over 20%. These programs were time consuming, unpleasant for children, and had poor uptake by the community. In the 1990s trachoma control programs ceased in many regions. By 2008 the Northern Territory-wide mean prevalence in remote communities was 29%.

The National Trachoma Elimination Project's program commenced in the Northern Territory in 2007. Since then, the observed prevalence of trachoma in the Northern Territory has decreased to 2.3% and has effectively been eliminated in East Arnhem and Darwin regions (0%). Significant reductions in trachoma have occurred in the remaining regions since 2007:

- **Alice Springs (remote) – from 22% to 7% (in 2023)**
- **Barkly – from 30% to 2%**
- **Katherine – from 20% to 13%**

The Northern Territory Trachoma Program continues to screen communities in these regions and treat trachoma, including with community-wide treatment programs.

AETIOLOGY AND PATHOGENESIS

Trachoma is a chronic kerato-conjunctivitis caused by infection with *Chlamydia trachomatis*, an obligate intracellular bacterium. The incubation period is 5 to 12 days. The active infective stages of trachoma are usually found in children. After years of infection the eyelid becomes so scarred that it turns inwards, and the eyelashes scratch the cornea. Repeated corneal damage leads to blindness. Eyelid surgery can prevent this eye damage.

Children host and transmit trachoma within families. The severity of scarring and risk of subsequent blindness depends on the intensity and duration of the inflammation. The presence of scarring increases with age.

CLINICAL PICTURE

Risk factors. Poor individual and community hygiene and limited access to water are important risk factors. Flies can transmit chlamydia.

Symptoms are often less intense than would be expected from the clinical signs, and people are often asymptomatic. There may be a mild mucopurulent discharge, irritation of the eye, or photophobia (from the associated keratitis). Once the eyelid turns inwards (entropion) and eyelashes grow inwards (trichiasis), pain and irritation from corneal abrasions may occur. Blindness can result and is intractable.

Signs. The World Health Organization has developed a grading system for diagnosis and assessment of trachoma (Table 10).

Investigations. Trachoma is predominantly a clinical diagnosis usually made without microbiological testing. Community and individual screening require assessment by people trained in the clinical diagnosis and grading of trachoma.

Laboratory methods for diagnosis of chlamydia are the classical direct microscopy, immunofluorescence, ELISA tests and, more recently, more sensitive polymerase chain reaction (PCR) and ligase chain reaction testing. Although PCR testing is more sensitive than other laboratory tests, it should not be used for routine screening communities, but to confirm the clinical diagnosis in a proportion of cases. Similarly, investigation of trachoma outbreaks may benefit from some confirmatory microbiology.

Examination of the eye for trachoma

Each eye must be assessed separately using binocular loupes (x 2.5) in good light. Signs must be clearly seen to be considered present. Examine the inside of the everted upper eyelid (tarsal conjunctiva) for follicles, inflammation and scarring. To examine for trichiasis, either in-turned eyelashes or previously removed lashes, the upper lid needs to be pushed upwards slightly to expose the lid margins. Examine the cornea for opacities.

DIFFERENTIAL DIAGNOSIS

Trachomatous inflammation (follicular): viral infections, hypersensitivity to topical medications.

Trachomatous inflammation (intense): chronic blepharitis, allergic conjunctivitis, bacterial conjunctivitis, contact lens related problems.

Trachomatous conjunctival scarring: atopic conjunctivitis, prolonged use of steroids.

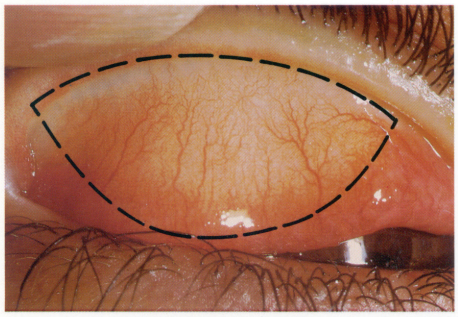
Table 10: WHO Trachoma simplified grading card

TRACHOMA GRADING CARD

– Each eye must be examined and assessed separately.
– Use binocular loupes (x 2.5) and adequate lighting (either daylight or a torch).
– Signs must be clearly seen in order to be considered present.

The eyelids and cornea are observed first for inturned eyelashes and any corneal opacity. The upper eyelid is then turned over (everted) to examine the conjunctiva over the stiffer part of the upper lid (tarsal conjunctiva).

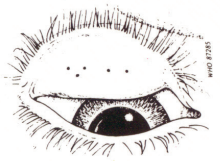
The normal conjunctiva is pink, smooth, thin and transparent. Over the whole area of the tarsal conjunctiva there are normally large deep-lying blood vessels that run vertically.



Normal tarsal conjunctiva (x 2 magnification). The dotted line shows the area to be examined.

***TRACHOMATOUS INFLAMMATION – FOLLICULAR (TF)**: the presence of five or more follicles in the upper tarsal conjunctiva.*

Follicles are round swellings that are paler than the surrounding conjunctiva, appearing white, grey or yellow. Follicles must be at least 0.5mm in diameter, i.e., at least as large as the dots shown below, to be considered.

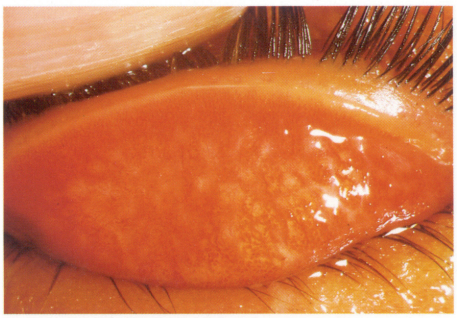




Trachomatous inflammation – follicular (TF).

***TRACHOMATOUS INFLAMMATION – INTENSE (TI)**: pronounced inflammatory thickening of the tarsal conjunctiva that obscures more than half of the normal deep tarsal vessels.*

The tarsal conjunctiva appears red, rough and thickened. There are usually numerous follicles, which may be partially or totally covered by the thickened conjunctiva.

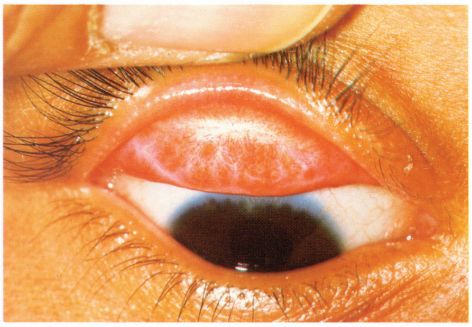


Trachomatous inflammation – follicular and intense (TF + TI).

Table 10: WHO Trachoma simplified grading card (continued)

***TRACHOMATOUS SCARRING (TS)**: the presence of scarring in the tarsal conjunctiva.*

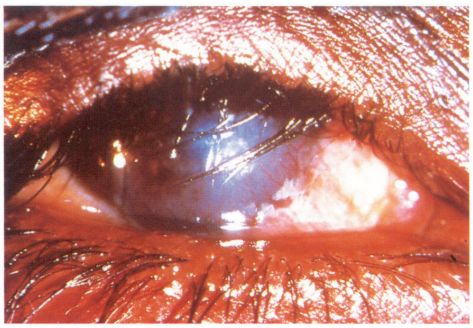
Scars are easily visible as white lines, bands, or sheets in the tarsal conjunctiva. They are glistening and fibrous in appearance. Scarring, especially diffuse fibrosis, may obscure the tarsal blood vessels.



Trachomatous scarring (TS)

***TRACHOMATOUS TRICHIASIS (TT)**: at least one eyelash rubs on the eyeball.*

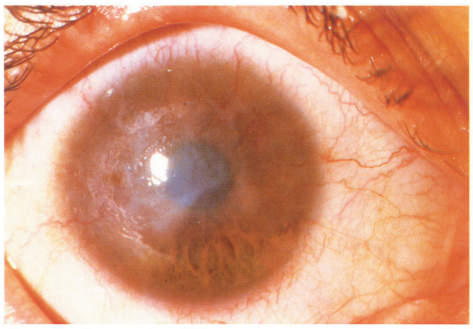
Evidence of recent removal of inturned eyelashes should also be graded as trichiasis.



Trachomatous trichiasis (TT)

***CORNEAL OPACITY (CO)**: easily visible corneal opacity over the pupil.*

The pupil margin is blurred viewed through the opacity. Such corneal opacities cause significant visual impairment (less than 6/18 or 0.3 vision), and therefore visual acuity should be measured if possible.



Corneal opacity (CO)

TF:– give topical treatment (e.g. tetracycline 1%).
TI:– give topical and consider systemic treatment.
TT:– refer for eyelid surgery.

WORLD HEALTH ORGANIZATION
PREVENTION OF BLINDNESS AND DEAFNESS

Support from the partners of the WHO Alliance for the Global Elimination of Trachoma is acknowledged.

Reproduced with permission

Note: Australian guidelines recommend single-dose systemic treatment, rather than topical treatment.

PRINCIPLES OF MANAGEMENT

Prevention. Improved hygiene (especially face and hand washing) and improved environmental and socio-economic conditions are the most important factors in preventing trachoma.

Treatment. Cases are treated with a single dose of oral azithromycin (see the Therapeutic Guidelines or CARPA Standard Treatment Manual). All household contacts should also be treated with a single dose of oral azithromycin.

Screening and community wide treatment. The Northern Territory Trachoma Program undertakes community screening and targets all 5-9-year-olds in communities. The calculated prevalence of trachoma in the community is then used to determine the type of treatment for the community (if required). Generally, if prevalence of trachoma is estimated to be above 5%, then community-wide treatment may be indicated. An in-depth explanation of community-wide treatments can be found in the CDNA Series of National Guidelines (SoNG) for Trachoma.

The chronic sequelae of trachoma (trachomatous conjunctival scarring, trichiasis, corneal opacity and blindness) occur in adults. All adults who lived as a child in a First Nations community with endemic or higher levels of trachoma require an annual eye examination looking for trachoma. Refer people with trachomatous trichiasis for prompt ophthalmological review and sight-saving lid surgery.

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call infectious disease physician, paediatrician, ophthalmologist, or local CDC/PHU

MANAGEMENT GUIDELINES

National		
Communicable Disease Network Australia (CDNA)	National Guidelines for Public Health Units — Trachoma	Available online
Northern Territory		
Remote Primary Health Care Manuals (RPHCM)	CARPA Standard Treatment Manual — Trachoma	Available online

RESOURCES

University of Melbourne — Indigenous Eye Health Unit	Trachoma (resources)	Available online
Fred Hollows Foundation	The Indigenous Australia Program	Available online
Nt Health	Fact sheet - Trachoma	Available online

FURTHER READING

Cowling CS, Liu BC, Snelling TL, et al. Australian trachoma surveillance annual report, 2013. Communicable Diseases Intelligence Quarterly Report. 2016;40(2):E255-E266.

Lange FD, Baunach E, McKenzie R, Taylor HR. Trachoma elimination in remote Indigenous Northern Territory communities: baseline health promotion study. Australian Journal of Primary Health. 2014;20(1):34-40.

Melese M, Alemayehu W, Lakew T, et al. Comparison of annual and biannual mass antibiotic administration for elimination of infectious trachoma. JAMA. 2008;299(7):778-784.

Renneker KK, Abdala M, Addy J, et al. Global progress toward the elimination of active trachoma: an analysis of 38 countries. The Lancet Global Health. 2022;10(4):e491- e500.

Shattock AJ, Gambhir M, Taylor HR, et al. Control of trachoma in Australia: a model based evaluation of current interventions. PLoS Neglected Tropical Diseases. 2015;9(4):e0003474.

Bat borne viruses

Australian bat lyssavirus and Hendra virus are rare and carry a high mortality. Lyssavirus is universally fatal whilst Hendra virus has a case fatality rate of over 50%.

Lyssavirus in Northern Australia

Australian bat lyssavirus (ABLV) is a rabies-like virus. ABLV has been documented in most bat families in Australia, and it is assumed that all bats have the potential to carry the virus.

There have been 3 human cases, all from Queensland and each resulting in death. Serological testing of bats demonstrates that lyssavirus is widely distributed throughout Australia. The virus has previously been detected in bats in the Northern Territory; however no infected bats were detected in the period 2019-2023.

If someone is bitten or scratched by a bat they should be considered to be exposed to the virus and offered post-exposure prophylaxis with rabies vaccine and human rabies immunoglobulin (HRIG). Those at occupational risk of handling bats should be immunised against ABLV with the rabies vaccine.

Data from the Northern Territory Centre for Disease Control show that between 2019 and 2023 an average of 19 bat exposures were documented annually, and that 60% required treatment with HRIG.

AETIOLOGY AND PATHOGENESIS

Australian bat lyssavirus infection is caused by a lyssavirus, genotype 7, which is very similar to the genotype 1 lyssavirus that causes classical human rabies. It was identified in Queensland in 1996 following the first human case. It appears to be transmitted by bat saliva, or possibly by inhalation of aerosolised bat secretions. It is thought not to be transmitted by contact with bat droppings, urine, or blood. Provided bats are cooked well, the meat and organs other than the brain can be eaten without risk of transmission.

Once the virus enters the body it migrates up nerve pathways to the central nervous system. The incubation period is variable, ranging from 7 days to over 1 year (mean of 1–2 months). This probably depends on the amount of virus introduced, the amount of tissue involved, host defence mechanisms, and the actual distance that the virus has to travel from the site of inoculation to the central nervous system. Incubations of several years have been described with other lyssavirus infections.

CLINICAL PICTURE

Risk factors. The main risk factor is a history of bat bite or scratch. While lyssavirus is more likely to be found in aggressive, sick, or injured bats — bats that appear healthy may also be infectious.

Symptoms. The clinical manifestations are assumed to be very similar to rabies. Rabies can be divided into four stages: a non-specific prodrome, acute encephalitis, profound brainstem dysfunction, and death.

The prodromal period (1–4 days) is generally marked by fever, headache, malaise, myalgias, anorexia, nausea and vomiting, sore throat, and dry cough. The one specific symptom is paraesthesia at the virus inoculation site in 50–80% of patients. The encephalitic phase is usually marked by agitation and confusion with lessening lucid periods until the patient lapses into coma. The prominence of early brainstem dysfunction distinguishes rabies from other viral encephalitides and accounts for the rapid downhill course. The median period of survival after the onset of symptoms is 4 days, with a maximum of 20 days, unless artificial supportive measures are instituted. Death occurs because of apnoea after involvement of the respiratory centre.

Signs. Early signs such as fever may be non-specific. Paraesthesias and cranial nerve palsies may be present later in the disease.

Investigations. If possible, the bat should be sent to specialised veterinary laboratories for testing. Infected people are usually diagnosed on clinical and historical grounds, but diagnosis can be confirmed through PCR testing of suitable specimens (cerebrospinal fluid, saliva, tissue sampling of brain or salivary gland).

DIFFERENTIAL DIAGNOSIS

ABLV is clinically indistinguishable from rabies infection and, while Australia is rabies free, this should be considered as a differential in people with a suspicious clinical illness and a history of animal bite in endemic areas (notably Southeast Asia, including Bali).

Unusual neurologic illnesses that may require differentiation from lyssavirus infection include Guillain-Barré syndrome, Murray Valley encephalitis and Japanese encephalitis. Other, more common causes of encephalitis, such as herpes simplex virus should also be excluded.

Polio may also present as a paralytic illness and should be kept in mind in people presenting from areas where polio still exists.

PRINCIPLES OF MANAGEMENT

Prevention is the cornerstone of management as lyssavirus infections including rabies are universally fatal. Pre-exposure vaccination and boosters are given to those at risk through their work, such as Parks and Wildlife staff and veterinary staff who may handle bats.

Post-exposure prophylaxis is recommended as soon as possible after bat bites or scratches and consists of a 4–dose course of rabies vaccine and HRIG if not more than 7 days has elapsed since the start of vaccination. The decision to vaccinate, its timing, and bat testing is complex and should be discussed with the Centre for Disease Control.

Bats should not be handled. Do not attempt to recover bats responsible for scratches or bites. If bat testing is warranted, the Centre for Disease Control or the on-call rural medical practitioner in your area can liaise with the appropriate authorities to do this. However, staff from agencies such as Parks and Wildlife are not routinely available outside office hours or away from major urban centres.

First Aid if scratched or bitten:

- Wash the wound **thoroughly** with soap and running water for approximately 15 minutes as soon as possible. Proper cleaning of the wound is the most effective way to reduce transmission of the virus.
- Apply a virucidal antiseptic solution such as povidone-iodine after washing.
- Cover the wound and seek medical attention immediately for consideration of post-exposure prophylaxis even if already vaccinated against rabies.

First Aid if bat saliva in the mouth, eyes or nose:

- Flush the area **thoroughly** with water.
- Seek medical attention immediately for consideration of post-exposure prophylaxis even if already vaccinated against rabies.

Management of persons with suspected lyssavirus illness is largely supportive and requires hospital based intensive care. The three cases documented so far have been fatal.

> Australian bat lyssavirus infection is a nationally notifiable condition to be reported by both CLINICIANS and LABORATORIES. **Report cases urgently by telephone** to the local Centre for Disease Control/Public Health Unit.

Hendra virus in Northern Australia

AETIOLOGY AND PATHOGENESIS

Hendra virus belongs to the paramyxovirus genus *Henipavirus* whose hosts are fruit bats/flying foxes (*Pteropus* species). It was first identified in 1994.

Horses become infected from feed contaminated with saliva, urine and reproductive fluids from infected bats. Human infection can result from exposure to bodily fluids of infected horses — high risk exposures include respiratory secretions and post-mortem examinations.

No direct bat to human or human to human transmissions have been documented and there have been no infections, to date, of either horses or humans in the NT. However, cases are documented in NSW and QLD, and there is evidence that Hendra virus exists in flying foxes in the Northern Territory.

CLINICAL PICTURE

Risk factors. Exposure to sick horses, particularly their respiratory secretions, is the primary risk factor for veterinarians, horse trainers and stable workers.

Symptoms and signs. Limited data, based on 7 cases, indicate the incubation period is between 5–21 days. Most cases have involved an influenza-like illness with either pneumonia or aseptic meningitis/encephalitis. There have been 4 deaths among the 7 people infected.

Investigations. Hendra virus can be tested for using PCR testing of suitable samples (including blood, respiratory samples, urine and tissue) at state-based reference laboratories. If there is clinical suspicion of Hendra virus infection, discuss with infectious disease specialists and public health authorities to facilitate early testing.

PRINCIPLES OF MANAGEMENT

There is no specific prophylaxis or treatment of Hendra virus and care is supportive and should be directed by infectious disease specialists. Monoclonal antibodies may be protective against lethal infection and are only stocked in Queensland.

> Hendra virus is a notifiable condition to be reported by CLINICIANS and LABORATORIES in the Northern Territory. **Report cases urgently by telephone** to the local Centre for Disease Control/ Public Health Unit.

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call infectious disease physician, general physician, or local PHU/CDC.

MANAGEMENT GUIDELINES

International		
World Health Organization	Frequently asked questions on rabies	Available online
National		
Communicable Disease Network Australia (CDNA)	National Guidelines for Public Health Units: ■ Rabies and other lyssavirus ■ Hendra virus	Available online
Australian Immunisation Handbook	Rabies post-exposure prophylaxis (PEP): bat exposures	Available online
Northern Territory		
NT Centre for Disease Control (CDC)	Public Health and Notifiable Diseases	Available online

RESOURCES

NT Centre for Disease Control (CDC)	NT Fact Sheet – Australian bat lyssavirus and rabies	Available online

FURTHER READING

McGuinness S, Denholm JT, Leder K. Notifiable Australian zoonotic infections. Medicine Today. 2013;14(10):55-64.

Paterson BJ, Butler MT, Eastwood K, et al. Cross sectional survey of human-bat interaction in Australia: public health implications. BMC Public Health. 2014;14:58.

Young JR, Selvey CE, Symons R. Hendra virus. The Medical Journal of Australia. 2011;195(5):250-251.

Hepatitis A

The Hepatitis A incubation period is 50–150 days with a mean of 30 days.

HEPATITIS A IN NORTHERN AUSTRALIA

Hepatitis A is a self-limiting viral infection of the liver caused by the hepatitis A virus (HAV). Where there is poor sanitation and hygiene, cumulative rates approach 80% by the age of two years.

Free hepatitis A vaccine is offered to all First Nations infants born on or after 1 May 2004 as part of a national immunisation program. In the Northern Territory, this vaccine is offered routinely at 18 months and 4 years of age. Since the introduction of this vaccine for First Nations infants there have been very few cases of hepatitis A acquired in the Northern Territory (Figure 79).

AETIOLOGY AND PATHOGENESIS

Hepatitis A virus (HAV) is transmitted by the faecal-oral route and possibly also via blood during the viraemic stage of the illness. The virus directly infects hepatocytes causing abnormal liver function. The symptoms and signs listed below are due to acute hepatitis.

CLINICAL PICTURE

Risk factors include ingestion of sewage via contaminated water or shellfish; being in preschools or childcare centres; being intellectually disabled; anal sex with an infected person; sharing IV needles with an infected person; and travel to areas with poor sanitation within Australia, and low- and middle-income countries with poorly developed sanitation systems.

Symptoms and signs. The infectious period extends from 2 weeks before until one week after symptoms appear. No carrier state exists and lifelong immunity results. Fulminant HAV occurs in less than 0.5% of cases. Infected children under the age of five do not usually become jaundiced and are therefore rarely diagnosed. Adults are usually symptomatic with fever, tiredness, nausea and vomiting, jaundice, abdominal pain, and dark urine. Symptoms may last from a few days to about a month. Signs include jaundice, tender enlarged liver, dark urine from bilirubinuria, and pale stools.

Investigations include urine dipstick for bilirubinuria, as well as blood tests for LFTs, and hepatitis serology. HAV serology includes Hep A IgM (positive in acute infection) and Hep A IgG (remains positive after infection or vaccination).

DIFFERENTIAL DIAGNOSIS

Acute hepatitis may result from infection (e.g. hepatitis A, B, C, D, E, arboviruses, EBV, CMV or leptospirosis), toxins (such as alcohol), medications (such as isoniazid, rifampicin, flucloxacillin) or autoimmune disease. A history of infection risk factors including travel and occupation, as well as drug or medication ingestion is useful.

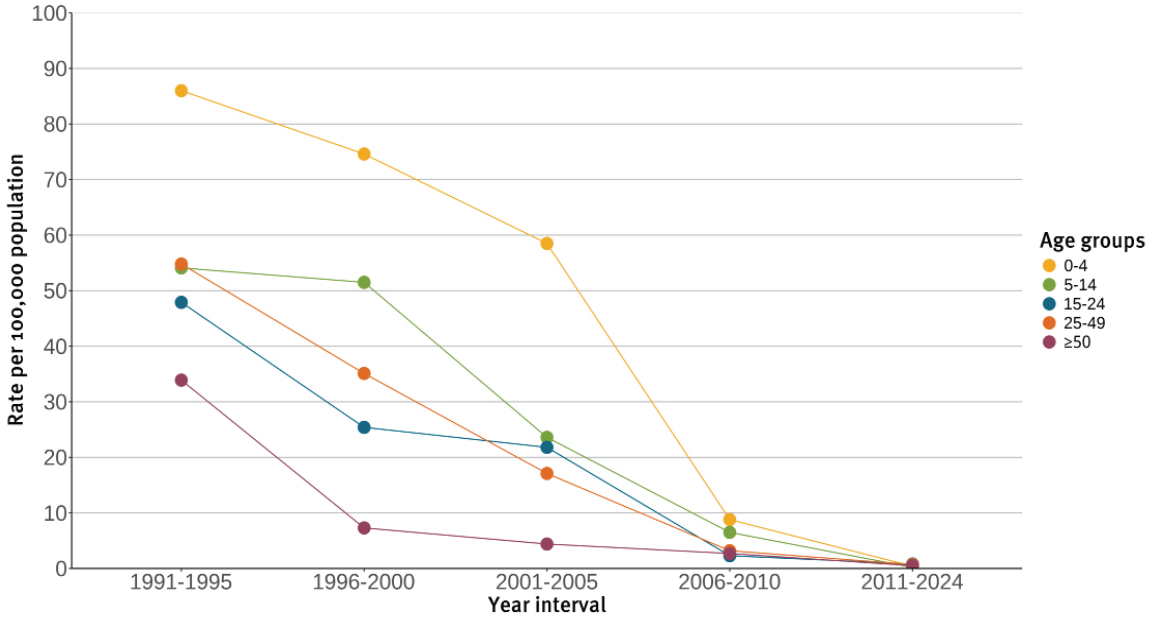


Figure 79: Age-specific rates of Hepatitis A in the Northern Territory by year intervals, 1991-2024
Source: Northern Territory Notifiable Diseases System (NTNDS)
Note: There were no cases of locally acquired hepatitis A 2011–2015.

PRINCIPLES OF MANAGEMENT

Treatment of acute infection is supportive and includes rest, maintaining hydration and avoiding alcohol. Once positive, there is no need for repeat serology. Household and family contacts of acute HAV should be made aware of infection routes and arrangements made for immediate testing for serologic evidence of infection. All non-immune household and family contacts should be offered the hepatitis A vaccine.

Normal human immunoglobulin may be given to children less than 1 year of age or to people who are immune suppressed within 72 hours of exposure. Infections in areas of high transmission risk (e.g. disability or childcare centres, preschools) may require treatment of more than household and family contacts. Telephone CDC/PHU for advice.

Prevention of HAV infection includes good sanitation and vaccination. Many people born before 1950 and most First Nations adults have immunity. While serological testing prior to vaccination is not generally recommended it may be worth checking in these groups. Two doses of HAV vaccine 6–12 months apart are recommended for high-risk groups such as rural health professionals, people raised in low prevalence communities who move to live in remote Northern Territory communities, travellers to low- and middle-income countries, child care workers, disability carers, men who have sex with men, and plumbers. Boosters or serology post vaccination are not necessary.

> Hepatitis A is a nationally notifiable condition to be reported by all CLINICIANS and LABORATORIES. **Report cases by telephone** to the local Centre for Disease Control/Public Health Unit.

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call infectious disease physician, specialist physician, or local CDC/PHU.

MANAGEMENT GUIDELINES

National		
Communicable Disease Network Australia (CDNA)	■ National Guidelines for Public Health Units — Hepatitis A ■ Australian notifiable diseases case definitions	Available online
Australasian Sexual Health Alliance (ASHA)	Australian STI Management Guidelines for use in Primary Care — Hepatitis A	Available online
Northern Territory		
Remote Primary Health Care Manuals (RPHCM)	CARPA Standard Treatment Manual — Hepatitis	Available online
NT Cenre for Disease Control (CDC)	Public Health and Notifiable Diseases	Available online

RESOURCES

NT Centre for Disease Control (CDC)	Fact Sheet — Hepatitis A	Available online

FURTHER READING

Gandhi AP, Al-Mohaithef M, Aparnavi P, et al. Global outbreaks of foodborne hepatitis A: Systematic review and meta-analysis. Heliyon. 2024;10(7):e28810.

Hepatitis B

Hepatitis B (HBV) is a blood-borne viral infection that can cause acute or chronic liver inflammation and damage.

HEPATITIS B IN NORTHERN AUSTRALIA

Hepatitis B surface antigen was first identified in a First Nations person from Central Australia and was initially known as the Australian antigen. Although chronic hepatitis B (CHB) sero-prevalence in Australia overall is low at 1%, in high-risk groups such as First Nations peoples and culturally and linguistically diverse communities (CALD), the seroprevalence of CHB can be up to 10%.

The Northern Territory has the highest overall percentage (1.9%) of people living with chronic HBV of any primary healthcare network in Australia. The prevalence in First Nations people who make up 67% of those living with chronic HBV in the Northern Territory is 6%. The majority of the remaining 33% are from CALD communities.

First Nations people living with chronic HBV in the Northern Territory have a unique sub-genotype called C4. HBV C4 variant has a high potential to cause advanced liver disease and has genetic mutations that may increase the risk of vaccine failure and liver cancer.

Without treatment, 15–25% of people living with CHB will die from liver cirrhosis or hepatocellular carcinoma (HCC) as a direct consequence of their infection.

AETIOLOGY AND PATHOGENESIS

HBV is transmitted via blood and secretions. Infection causes an immune mediated hepatitis which can be acute or chronic. In the Northern Territory where universal birth-dose and childhood vaccination has been in place since 1988 for First Nations children, and 1990 for all children, most people living with CHB infection acquired it early in life from mother to child or early horizontal transmission. Acute HBV is now relatively uncommon in the Northern Territory.

CLINICAL PICTURE

Risk factors. Being born to a CHB infected mother; blood to blood contact particularly in early life; unprotected sex with an infected person; sharing injecting equipment, toothbrushes or razors and needlestick injuries.

Symptoms and signs. Most people with chronic hepatitis B are asymptomatic. In acute infection the incubation period is 45–180 days. The infectious period extends for several weeks before symptoms appear, until the resolution of the illness unless chronic infection develops (defined as the presence of HBsAg positivity for greater than 6 months). The infection is cleared in 90% of adults and lifelong immunity results, however if HBV is acquired during the process of birth or before the age of 5 years (without any intervention) it is more than 90% likely that the individual will develop CHB.

People living with CHB require lifelong regular follow-up as they may require treatment at some point in their life as well as screening for hepatocellular carcinoma. CHB is a dynamic disease process which fluctuates over time. Without appropriate follow-up and management approximately 25% of those living with CHB will die prematurely of cirrhosis or HCC. These outcomes can be prevented with publicly funded treatments.

In acute infection and during flares in chronic infection symptoms can include fever, malaise, nausea and vomiting, abdominal pain, myalgias, rash or arthritis. The acute illness may last for weeks, and tiredness can persist for months. The signs of HBV infection include jaundice, tender enlarged liver, dark urine, pale stools. Individuals with chronic HBV may present with signs and symptoms of cirrhosis, either compensated or decompensated, or HCC, as chronic infection can often be asymptomatic until this point.

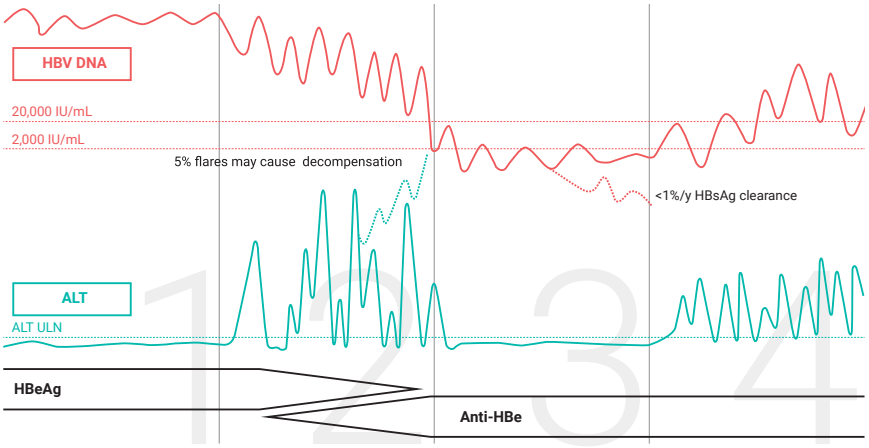
Investigations. Include FBC, UEC, LFTs, INR and Hepatitis A IgM and IgG, HBsAg, anti-HBs, anti-HBc, HBeAg, anti-HBe, Hepatitis B viral load, Hepatitis C IgG, Hepatitis D antibody* and HIV serology. A baseline ultrasound and non-invasive assessment of fibrosis (elastometry by Fibroscan® if available, otherwise APRI or Hepascore) is recommended.

* HDV testing is not necessary in First Nations people unless there is clinical concern as it has never been identified in a Northern Territory First Nations person. However, testing should be routine at baseline in non-Indigenous people with chronic hepatitis B.

DIFFERENTIAL DIAGNOSIS

Acute hepatitis may result from infection (hepatitis A, B, C, D, E, arboviruses, EBV, CMV, leptospirosis), toxins (such as alcohol), medications (e.g. rifampicin, flucloxacillin), or autoimmune disease (e.g. systemic lupus erythematosus). A history of risk factors for infection including travel, and drug or medication ingestion, is useful. Chronic HBV infection (defined as HBsAg positive for more than 6 months) may be detected incidentally by screening, or by follow-up after an acute illness.

Patients with CHB must be **regularly re-evaluated** to determine which phase they are in and managed accordingly.



HBeAg-positive chronic infection (Immune tolerance)	HBeAg-positive chronic hepatitis (Immune clearance)	HBeAg-negative chronic infection (Immune control)	HBeAg-negative chronic hepatitis (Immune escape)
• **HBV DNA: high†** >10^7 IU/mL • **ALT: normal** • **HBeAg positive**	• **HBV DNA: high†** >20 000 IU/mL • **ALT: elevated** Elevated is >30 IU/L men; >19 IU/L women • **HBeAg positive**	• **HBV DNA: low†** <2000 IU/mL • **ALT: normal** • **HBeAg negative** • **anti-HBe positive**	• **HBV DNA high†** >2000 IU/mL • **ALT: elevated** Elevated is >30 IU/L men; >19 IU/L women • **HBeAg negative** • **anti-HBe positive**
Treatment not required	**Refer to s100 community prescriber or specialist for consideration of treatment** Risk of progression to cirrhosis and HCC	**Treatment not required**	**Refer to s100 community prescriber or specialist for consideration of treatment** Risk of progression to cirrhosis and HCC

† Medicare covers HBV DNA testing once per year for patients not on treatment and 4 times per year for patient on treatment.

Figure 80: Phases of disease in chronic hepatitis B infection and when to consider treatment.
Source: Decision making in hepatitis B. Sydney: ASHM; 2024

PRINCIPLES OF MANAGEMENT

Treatment of acute infection is usually supportive. Serology is repeated at 6 months. If HBsAg is then still positive the patient has chronic hepatitis B.

Treatment of chronic hepatitis B. People living with chronic hepatitis B should be followed up regularly for life with assessment of their phase of hepatitis B and their liver disease including 6 monthly LFTs, FBC, kidney function tests and INR and annual HBV viral load as well as a baseline ultrasound and elastometry.

Establishing the phase of chronic HBV infection and the patient's cirrhotic status is essential in determining the need for antiviral treatment.

Note: CHB is a dynamic disease and can change over time hence the need for ongoing regular review.

Effective anti-viral treatment is available with either entecavir or tenofovir and should be considered if an individual is in the immune clearance (HBeAg positive, VL>20,000IU/mL and elevated ALT) or immune escape phase (HBeAg negative, VL>2000IU/mL and elevated ALT) and for all those with cirrhosis. These antiviral agents effectively suppress disease while they're being taken. They are NOT curative, and disease is likely to re-emerge if treatment is stopped for any reason.

***Note*:** Upper limit of normal for ALT is 19 for females and 30 for males.

Six-monthly HCC screening is recommended for those individuals outlined in Figure 81 below.

Hepatitis B

Hepatocellular carcinoma (HCC) surveillance (6 monthly ultrasound and AFP) is recommended in these HBsAg + groups:

- First Nations people >40 years born in the Northern Territory
- Africans >20 years
- Asian men >40 years
- Asian women >50 years
- Patients with cirrhosis
- HCC family history

Figure 81: HCC recommendations for the Northern Territory
Source: Clinical practice guidelines for hepatocellular carcinoma surveillance for people at high risk in Australia. Cancer Council Australia. 2023.

The Australasian Society for HIV, Viral Hepatitis and Sexual Health Medicine (ASHM) provides the S100 GP training required to prescribe hepatitis B antivirals. Treatment can also be initiated by specialist viral hepatitis services at both Royal Darwin Hospital and Alice Springs Hospital. Within the Northern Territory the Hep B PAST partnership program is establishing region and clinic specific core clinical care groups (including streamlined access to a prescriber, trained AHP, ultrasound and Fibroscan® elastometry) for First Nations people living with CHB in the Northern Territory — for further information please email: hepbpast@menzies.edu.au

Referral to a specialist viral hepatitis or liver clinic should be considered for:

- Consistently elevated ALT (more than the upper normal limit).
- All individuals with cirrhosis.
- Co-infection with another blood borne virus.
- Those about to commence immunosuppression (HBsAg positive individuals and isolated anti-HBc positive individuals).
- Pregnant women (as treatment for the prevention of mother to child transmission is available for those with a HBV viral load >200,000 IU/mL at 28 weeks).
- Children with chronic HBV (age less than 16 years).
- Anyone else you are concerned about.

Prevention. Vaccination is the foundation stone of prevention. However, First Nations peoples are identified as high-risk in the National Hepatitis B Strategy and should all be screened for HBV. Once the results are available the patient's serostatus should be recorded in their electronic health record problem list. If documented to be non-immune vaccination should be arranged. HBV status should also be checked at the first ante-natal visit of every pregnant female in Australia.

See CDC NT Hepatitis B vaccination and public health guidelines for detailed information about contact tracing and immunisation. Household and sexual contacts of people with acute or chronic HBV infection should be educated about infection risks and tested for evidence of infection. Non-immune contacts should be offered hepatitis B vaccine. Contacts of people with acute infection should also be offered immunoglobulin if within 72 hours of exposure.

Prevention of HBV infection includes routine immunisation (and is part of the NT Immunisation Schedule for all babies and children), safe sex practices and needle exchange programs. HBV vaccination is recommended for high-risk groups such as: health professionals, disability carers, men who have sex with men, and sex industry workers. Boosters are not necessary except for immunosuppressed individuals such as renal dialysis patients and HIV positive individuals.

> Hepatitis B is a nationally notifiable condition to be reported by all CLINICIANS and LABORATORIES. Report cases to the local Centre for Disease Control/Public Health Unit.

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call infectious diseases specialist physician or local CDC/PHU.

MANAGEMENT GUIDELINES

National and international		
The Australasian Society for HIV, Viral Hepatitis and Sexual Health Medicine (ASHM)	B Positive: Hepatitis B for Primary Care	Available online
Communicable Diseases Network Australia (CDNA)	Australian notifiable diseases case definitions – Hepatitis B (newly acquired)	Available online
The Australasian Society for HIV, Viral Hepatitis and Sexual Health Medicine (ASHM)	Decision making in Hepatitis B in the Northern Territory	Available online
Gastroenterological Society of Australia (GESA)	Australian consensus recommendations for the management of hepatitis B infection	Available online
Northern Territory		
NT Centre for Disease Control (CDC)	■ Northern Territory Hepatitis B Vaccination and Public Health Guidelines ■ Public Health and Notifiable Diseases	Available online
Remote Primary Health Care Manuals (RPHCM)	■ CARPA Standard Treatment Manual — Hepatitis ■ Women's Business Manual — Hepatitis in pregnancy	Available online
NT Primary Health Network (NT PHN)	Northern Territory HealthPathways — Hepatitis B	Available online (CLICK HERE to request access)

RESOURCES

The Australasian Society for HIV, Viral Hepatitis and Sexual Health Medicine (ASHM)	HBV Prescriber Program	Available online
Menzies School of Health Research	Hep B Story — Yolngu Matha and English (Mobile app — free download from Apple App Store and Google Play Store)	Available online
Centre for Disease Control (CDC)	Fact Sheet — Hepatitis B	Available online

FURTHER READING

Australian Government Department of Health. Fourth National Hepatitis B Strategy 2023-2030. (Draft for consultation (June 2023)

Davies J, Li SQ, Tong SY, et al. Establishing contemporary trends in hepatitis B sero-epidemiology in an Indigenous population. PloS One. 2017;12(9):e0184082.

Hosking, K, Binks P, De Santas T, et al. Evaluating a novel model of hepatitis B care, Hep B PAST, in the Northern Territory of Australia: results from a prospective, population-based study. The Lancet Regional Health – Western Pacific. 2024;48:101116.

MacLachlan JH, Romero N, Purcell I, Cowie BC. Viral Hepatitis Mapping Project: Hepatitis B National Report 2022. Darlinghurst, NSW, Australia: ASHM; 2024.

Human T-cell lymphotropic virus-1 infection (HTLV-1)

HTLV-1 causes haematological malignancies and inflammatory diseases and is communicable.

HUMAN T-CELL LYMPHOTROPIC VIRUS-1 IN NORTHERN AUSTRALIA

Human T-cell leukaemia virus type 1 (HTLV-1) is present largely in confined populations, particularly in Central Australia, Southwestern Japan, parts of the Caribbean, Pacific Islanders of Melanesian descent, South America and central Africa. HTLV-1 is highly endemic in parts of Central Australia, where seropositivity rate among hospitalised adults exceed 30%, and up to 48% in some remote communities surveyed (the highest community prevalence in the world). It is estimated that approximately 5000 adults with HTLV-1 reside in the Central desert region of the Northern Territory.

High rates of HTLV-1 infection have also been reported in a community in the Kimberley region of Western Australia and among adults admitted to Alice Springs Hospital from communities in Western Australia and South Australia. Infection rates are lower to the north of central Australia and the virus is uncommon in Arnhem Land and Darwin rural communities.

AETIOLOGY AND PATHOGENESIS

HTLV-1 is a human retrovirus (distantly related to HIV) that is predominantly transmitted via infected lymphocytes by breastfeeding, unprotected sexual intercourse and blood contact. The epidemiology of HTLV-1 in the Northern Territory and the proportions of infections by breastfeeding, unprotected sexual intercourse and other modes remains very unclear. However, sexual transmission is considered the major mode of transmission worldwide. HTLV-1 infection is often asymptomatic but predisposes to other infections including crusted scabies, symptomatic strongyloidiasis, and mycobacterial infections. Studies in the Northern Territory suggest a link to bronchiectasis and progressive lung disease. After many years of infection, it can cause:

1. A rapidly progressive haematological malignancy (adult T-cell leukaemia/lymphoma, ATL).
2. Inflammatory disorders, such as HTLV-1 associated myelopathy, HTLV-1 associated infective dermatitis, HTLV-1 associated polymyositis and HTLV-1 associated uveitis.

CLINICAL PICTURE

Risk factors include exposure to the transmission routes described above, and belonging to particular endemic groups described above.

Symptoms and signs. HTLV-1 infection is often asymptomatic. It is sometimes found when investigating for underlying immunological problems in patients with crusted scabies. Data derived from hospitals in Japan and the Caribbean suggests that the life-time risk of developing ATL and HTLV-1 associated myelopathy is 1–5% and 0.3–4%, respectively. The life-time risk of developing ATL is estimated to be 20% among those who acquire HTLV-1 infection in early childhood. In a recent community survey in Central Australia, HTLV-1 associated diseases and infections were found in 16.4% of adult participants with HTLV-1 compared to 3.3% without HTLV-1. Risk was associated with higher HTLV-1 pro-viral DNA load (a higher number of HTLV-1 infected cells in peripheral blood).

ATL is typically highly aggressive, resulting in a median survival of less than 10 months in 75% of cases despite advances in chemotherapy and supportive therapy. Lymphadenopathy is common and many patients also have hepatosplenomegaly and skin lesions. Individuals with ATL are highly immunosuppressed and often succumb to opportunistic infections. Lobulated lymphocytes, hypercalcaemia (from lytic bone lesions) and elevated LDH are common laboratory findings.

HTLV-1 associated myelopathy typically presents as a slowly progressive, symmetrical, spastic paresis involving the lower limbs. Difficulty walking and bladder dysfunction are often the initial symptoms. Cognitive and upper limb functions are usually preserved. Positive HTLV1 serology or antigen in the CSF forms part of the WHO diagnostic criteria. There is an average of eight years delay in diagnosis and treatment due to lack of awareness and testing. The condition results in considerable disability for those living in remote communities.

A variety of skin diseases have been reported, which can lead to invasive bacterial infections in residents of impoverished communities. Infective dermatitis is a chronic condition that typically affects children and results in exudates and crusting involving the scalp, ears, paranasal skin, eyelid margins, neck, axilla and groin. The condition responds rapidly to treatment with topical steroids and long-term antibiotics, such as sulfamethoxazole/trimethoprim.

HTLV-1 infection leads to higher parasite burdens resulting in crusted scabies and complicated strongyloidiasis and a predisposition to mycobacterial infections including TB. These individuals may serve as core transmitters in communities.

Recent studies in the Northern Territory suggest a link between HTLV-1 and bronchiectasis and progressive lung disease. In these circumstances, management plans for the bronchiectasis need emphasis, including ensuring that all vaccinations are up to date, access to physiotherapy support, strategies to optimise sputum clearance and when to use antibiotics. Co-infections with mycobacteria should be sought and treated.

In Central Australia, higher HTLV-1 proviral DNA load was associated with a fourfold increased risk of premature death, especially in people who suffer from bronchiectasis.

Investigations. Diagnosis of HTLV-1 infection is made by positive serology. All patients with crusted scabies, symptomatic strongyloidiasis, bloodborne virus infections, TB, T-cell lymphoma, unexplained severe dermatitis or spastic paraparesis should be tested for HTLV-1 especially in endemic regions.

DIFFERENTIAL DIAGNOSIS

Differential diagnosis depends on the presenting symptoms. ATL can be confused with other malignancies (e.g. cutaneous T-cell lymphomas, mycosis fungoides) or infectious diseases, and HTLV-associated myelopathy with various genetic syndromes, or infectious diseases such as late syphilis. An important differentiating question is whether the patient is from Central Australia or another endemic region with the above symptoms. Note that HTLV-1 is also associated with other malignant and inflammatory conditions.

PRINCIPLES OF MANAGEMENT

Although there is no current treatment for HTLV-1 infection, treating infective complications such as scabies, strongyloidiasis, skin, lung and urinary tract infections make it safer to live with HTLV-1. ATL may respond to chemotherapy and antiviral therapy. Early referral for an allogenic haematopoietic stem cell transplantation should be considered. Steroids may be beneficial for progressive myelopathy and pain control. Symptomatic management and physiotherapy can improve quality of life for patients living with myelopathy. Patients should be screened and treated for strongyloidiasis, TB, hepatitis B, scabies, HIV, hepatitis C and melioidosis (in the Top End) before commencement of steroids or immunosuppressants. Measures to reduce transmission should be stressed, i.e. safe sex and no blood donations. Although HTLV-1 is transmitted by breastmilk, this is less common in the first six months and current NT policy does not discourage breastfeeding.

HTLV-1 must be requested in screening tests following needlestick injuries involving a First Nations person from the Katherine region or the southern region of the NT. The Blood Borne Virus (BBV) Exposure – Clinical Management NT Health Guideline offers guidance on post exposure prophylaxis.

> HTLV-1 is a notifiable condition in the Northern Territory to be reported by CLINICIANS and LABORATORIES (it is not notifiable outside the Northern Territory). Report cases to the local Centre for Disease Control/Public Health Unit.

Human T-cell lymphotropic virus-1 infection (HTLV-1)

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call infectious disease physician or local CDC/PHU.

MANAGEMENT GUIDELINES

Northern Territory		
NT Centre for Disease Control (CDC)	Public Health and Notifiable diseases	Available online

FURTHER READING

Araujo A, Bangham CRM, Casseb J, et al. Management of HAM/TSP: Systematic Review and Consensus-based Recommendations 2019. Neurol Clin Pract. 2021;11(1):49-56.

Einsiedel L, Pham H, Talukder MR, et al. Very high prevalence of infection with the human T cell leukaemia virus type 1c in remote Australian Aboriginal communities: Results of a large cross-sectional community survey. PLoS Neglected Tropical Diseases. 2021;15(12):e0009915.

Einsiedel L, Pham H, Wilson K, et al. Human T-Lymphotropic Virus type 1c subtype proviral loads, chronic lung disease and survival in a prospective cohort of Indigenous Australians. PLoS neglected tropical diseases. 2018;12(3):e0006281.

Einsiedel L, Spelman T, Goeman E, et al. Clinical associations of Human T-Lymphotropic Virus type 1 infection in an Indigenous Australian population. PLoS Neglected Tropical Diseases. 2014;8(1):e2643.

Einsiedel L, Cassar O, Bardy P, et al. Variant human T-cell lymphotropic virus type 1c and adult T-cell leukemia, Australia. Emerging Infectious Diseases. 2013;19(10):1639-1641.

Einsiedel LJ, Pepperill C, Wilson K. Crusted scabies: a clinical marker of human T-lymphotropic virus type 1 infection in central Australia. Medical Journal of Australia. 2014;200(11):633-634.



Influenza

Remote communities may be the first geographic locations within Australia to experience influenza outbreaks.

INFLUENZA IN NORTHERN AUSTRALIA

Influenza occurs throughout the year in the Top End (Figure 69). This pattern is different from that seen in temperate parts of Australia and the timing of epidemics is less predictable. Patterns of respiratory virus circulation, including influenza, have changed since the COVID-19 pandemic and previously predictable peaks have not occurred. Influenza remains an important disease because epidemics evolve rapidly, cause widespread morbidity and serious complications, particularly from viral and bacterial pneumonias.

Immunisation remains the cornerstone to controlling the impact of influenza. It is recommended that all health staff and patients in risk categories be immunised as soon as that year's influenza vaccine is available, usually in February or March. Continue to promote the vaccine to unimmunised people in these groups throughout the year until the following year's vaccine becomes available. There are national programs for providing free vaccines to high-risk individuals. Information about these programs is available under Management Guidelines following.

Remote communities may be among the first geographic locations within Australia to experience outbreaks sometimes due to new antigenic strains of influenza virus. The identification of new antigenic strains is of national and international importance for influenza vaccine development and pandemic preparedness.

Clinicians should notify outbreaks of influenza-like illness to the CDC (lab will notify individual cases of confirmed influenza) and collect appropriate specimens (nasopharyngeal swabs) for viral diagnosis (PCR or rapid antigen testing (RAT)). The World Health Organization Collaborating Centre for Influenza Reference and Research at the Doherty Institute in Melbourne performs surveillance testing of specimens from the Top End to inform vaccine design and public health responses; samples are forwarded by the testing laboratory and do not need to be sent directly by clinicians.

There are several online national surveillance systems (see *Management Guidelines* page 96). The Australian Sentinel Practices Research Network (ASPREN) is an influenza and infectious disease surveillance system for GPs associated with the Royal Australian College of General Practitioners and the Australian College of Rural and Remote Medicine. FluTracking monitors influenza in the communities and across the nation, and now also includes data from New Zealand. Both provide reports and updates and depend on ongoing and regular input of volunteers.

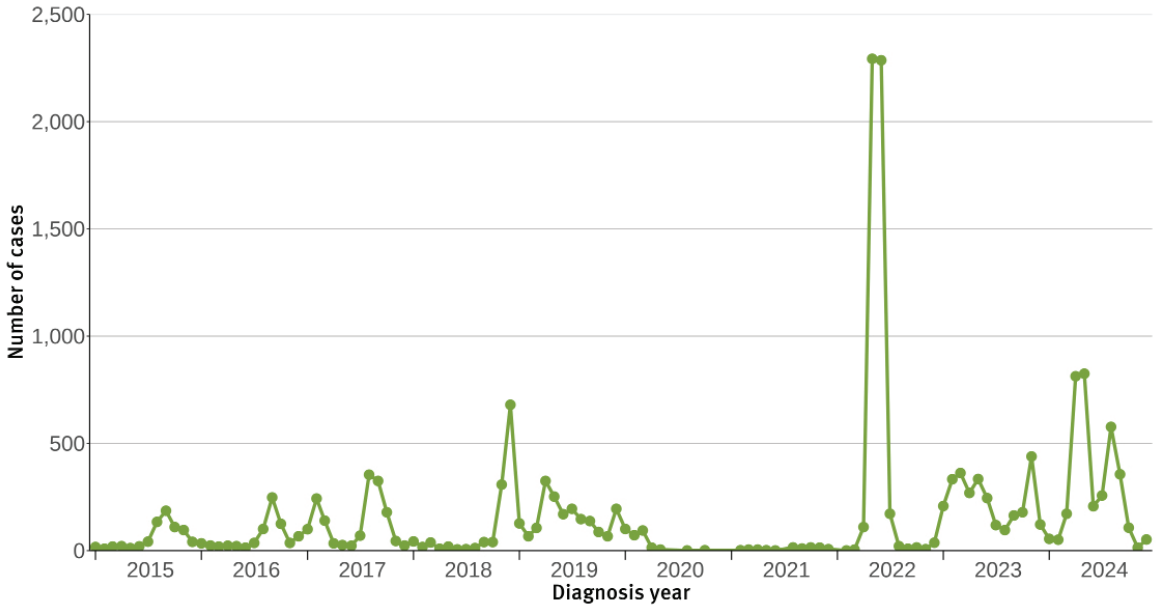


Figure 82: Number of Influenza cases in the Northern Territory by year and month of diagnosis, 2015-2024
Source: Northern Territory Notifiable Diseases System (NTNDS)

PRINCIPLES OF MANAGEMENT

Treatment is usually supportive with early use of oseltamivir recommended for patients at risk of severe disease (detailed recommendations in Therapeutic Guidelines).

Influenza is a nationally notifiable condition to be reported by LABORATORIES (individual cases do not need to be notified by clinicians). CLINICIANS should report possible outbreaks to the local Centre for Disease Control/Public Health Unit.

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call specialist physician or local CDC/PHU.

MANAGEMENT GUIDELINES

National		
Australian Government, Department of Health	■ Flu (influenza) immunisation ■ ATAGI advice on seasonal influenza vaccines ■ Australian Influenza Surveillance Report and Activity Updates ■ Australian Health Management Plan for Pandemic Influenza (AHMPPI) ■ The Australian Sentinel Practices Research Network (ASPREN) ■ FluTracking (Australia and New Zealand)	Available online
Communicable Diseases Network Australia (CDNA)	■ National Guidelines for Public Health Units — Influenza ■ Australian notifiable diseases case definitions	Available online
Northern Territory		
NT Centre for Disease Control (CDC)	Public Health and Notifiable diseases	Available online

RESOURCES

NT Centre for Disease Control (CDC)	Fact sheet — Influenza	Available online
NT Primary Health Network (NT PHN)	HealthPathways - Influenza	Available online (CLICK HERE to request access)

FURTHER READING

Betts JM, Weinman AL, Oliver J, et al. Influenza-associated hospitalisation and mortality rates among global Indigenous populations; a systematic review and meta-analysis. PLOS Glob Public Health. 2023;3(4):e0001294.

Nixon JC, Freeman K, Baird RW. Altered epidemiological patterns of Respiratory Syncytial Virus and influenza detections in a tropical Australian setting 2020 to 2023. Aust N Z J Public Health. 2024;48(4):100172.

Uyeki TM, Hui DS, Zambon M, et al. Influenza. Lancet. 2022;400(10353):693-706.

Weinman AL, Sullivan SG, Vijaykrishna D, et al. Epidemiological trends in notified influenza cases in Australia's Northern Territory, 2007-2016. Influenza Other Respir Viruses. 2020;14(5):541-550.

SECTION 3 — INFESTATIONS, PARASITIC AND FUNGAL INFECTIONS

Cryptococcal meningitis and pneumonia

Cryptococcus is a genus of yeast-like fungi that cause respiratory and neurological illness.

CRYPTOCOCCAL MENINGITIS AND PNEUMONIA IN NORTHERN AUSTRALIA

Cryptococcus gattii is endemic in Northern Australia, particularly western Arnhem Land. It causes meningitis, focal brain lesions, pneumonia, and focal lung lesions in patients without specific immune deficiency. Cryptococcus neoformans is an important cause of meningitis in immunosuppressed people.

Several species of Eucalypts, classically the River Red Gum (Eucalyptus camaldulensis) have been shown to be the environmental niche for C. gattii in Australia and it is thought to be transmitted to humans via contact with these trees. Although River Red Gums are not common in the Top End, C. gattii has been grown from various eucalypts (including the Darwin Woollybutt, Eucalyptus miniata) and other trees in Arnhem Land.

AETIOLOGY AND PATHOGENESIS

Cryptococcus species cause infection via inhalation, then dissemination, commonly to the central nervous system. *Cryptococcus gattii* is mostly restricted to tropical and subtropical locations. Disease incidence Australia wide is 0.61 per million but in the Northern Territory it is 12.9 per million.

CLINICAL PICTURE

Symptoms and signs.

- Early symptoms — non-specific, may include chronic headache or subtle changes in exercise tolerance. Fever is an unreliable sign.
- Respiratory — can be asymptomatic or present with subacute symptoms of lower respiratory tract infection.
- Neurological — chronic headache often without neck stiffness is the most common presentation. Other signs may include papilloedema (this is common and may indicate raised intracranial pressure and the need for urgent neurosurgical intervention), impaired consciousness, seizures, and/or focal neurological signs.

Symptoms are often indolent. The most common presentation of recent Northern Territory cases has been persistent headache without other clinical features of acute meningitis, so the diagnosis is easy to miss and yet a simple cryptococcal antigen test on blood will make the diagnosis. *Cryptococcus gattii* should be considered if someone is behaving differently, sleeping more or has new onset chronic headaches.

Data from 45 cases in the Northern Territory between 1996 and 2018 showed that the median time to diagnosis from the first symptoms was 26 days, with a median time to diagnosis of 47 days in those with isolated central nervous system (CNS) disease. Most cases (62%) presented with CNS involvement, 44% had both neurological and lung infection, and 36% presented with lung involvement only. Of the CNS infections meningoencephalitis (96%) and brain involvement with cryptococcomas(68%) were common. The Northern Territory cases have clustered around the western Arnhem Land communities of Maningrida and Gunbalanya.

Investigations. Cryptococcal antigen (CrAg) in serum and cerebrospinal fluid has excellent sensitivity and specificity. Recent Australian data demonstrates the CrAg is positive in serum in >97% of cases of C gattii infection. Lumbar puncture (LP) is almost always diagnostic in central nervous system disease. On cerebrospinal fluid microscopy, encapsulated yeast may be seen with an India ink stain. Perform a CT brain prior to lumbar puncture if there is a decreased level of consciousness, focal neurological signs or seizures. In respiratory disease sputum fungal cultures should be sent and a chest X-ray will usually show the solid mass lesion of pulmonary cryptococcoma.

DIFFERENTIAL DIAGNOSIS

Respiratory disease may mimic pulmonary tuberculosis or lung cancer. Consider other causes of subacute meningitis especially tuberculous meningitis. CT brain and lumbar puncture are mandatory to exclude cryptococcal meningitis in suspected cases.

PRINCIPLES OF MANAGEMENT

Management of *C. gattii* infection includes intensive phase therapy with intravenous liposomal amphotericin and oral 5-flucytosine, usually for 4–6 weeks followed by prolonged oral eradication therapy with fluconazole for 12–18 months. Recently voriconazole has been successfully used as a replacement for fluconazole in a small number of cases with extensive disease. For CNS disease, aggressive surgical management of raised intracranial pressure or serial therapeutic lumbar puncture is often necessary and for pulmonary disease there is a role for lobectomy for large cryptococcomas. Treatment in the setting of HIV infection is slightly different, and cure is less certain. Regular clinical follow-up with an infectious diseases physician is critical.



Figure 83: Cryptococcoma in lung
Source: Bart Currie — Menzies School of Health Research



Figure 84: Cryptococcoma surgically excised from lung
Source: Bart Currie — Menzies School of Health Research

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call infectious disease physician or local CDC/PHU.

FURTHER READING

Coussement J, Heath CH, Roberts MB, et al. Current epidemiology and clinical features of Cryptococcus infection in patients without human immunodeficiency virus: A multicenter study in 46 hospitals in Australia and New Zealand. Clin Infect Dis. 2023;77(7):976-986.

O'Hern JA, Koenen A, Janson S, et al. Epidemiology, management and outcomes of Cryptococcus gattii infections: A 22-year cohort. PLOS Neglected Tropical Diseases. 2023;17(3):e0011162.

Tinea corporis

Tinea corporis is a fungal infection that commonly affects children and teenagers.

TINEA CORPORIS IN NORTHERN AUSTRALIA

Tinea corporis is endemic in tropical areas and is ubiquitous in many First Nations communities. The predominant species in the Top End is Trichophyton rubrum. High levels of such anthropophilic dermatophyte infections reflect the poor living conditions and overcrowding often found in remote communities.

AETIOLOGY AND PATHOGENESIS

Tinea corporis is a dermatophyte infection of the trunk, legs, or arms as distinct from fungal infections of the groin (tinea cruris), hands (tinea manuum) or feet (tinea pedis). A group of filamentous fungi, also known as ringworm fungi, cause tinea corporis. While *Trichophyton rubrum* is by far the commonest dermatophyte in the Top End, *Trichophyton tonsurans, Epidermophyton* and *Microsporum* species also occur. *Trichophyton rubrum* is only spread person to person, has no animal reservoir, and can cause extensive and severe disease. Other dermatophyte species such as Microsporum have primary animal hosts and may be acquired from cats and dogs.

CLINICAL PICTURE

Risk factors. Direct contact with skin or nail lesions of infected people. Children and teenagers are commonly affected.

Symptoms and signs. The clinical presentation depends upon the site of infection, the immunological response of host and the fungal species. There are usually no symptoms although mild pruritus may be present. The lesions are dry and scaly with an active border of advancing infection with central clearing. In pale skin the advancing edge is often erythematous, while in dark skin there is usually just a silvery scale, but hyperpigmentation can occur. Infections are often extensive, covering up to 50% of the body with exposed areas affected more commonly. Secondary bacterial infection may occur.

Investigations. Diagnosis is usually clinical. Skin scrapings may be useful in difficult cases for definitive diagnosis. Up to 50% of suspicious material may not contain any fungus. To take a skin scraping, clean the skin with alcohol to decrease bacterial contamination and collect a dry sample from the raised borders. Scrape outwards with glass microscope slide or blunt scalpel held perpendicular to the skin using light pressure. Superficial bleeding is common when scraping as the *Trichophyton rubrum* scales are very adherent. Skin flakes should be collected in a sterile container and stored in the refrigerator. Microscopy results should be available quickly, but fungal culture may take up to 6 weeks.

DIFFERENTIAL DIAGNOSIS

The main differential diagnosis is pityriasis versicolor caused by *Malassezia furfur*, otherwise known as 'lace handkerchief' skin. Other possibilities include eczema, psoriasis, kava affected skin, extensive scabies or crusted scabies and leprosy.



Figure 85: Tinea corporis
Source: Ian McCrossin



Figure 86: Tinea corporis
Source: Ian McCrossin



Figure 87: Tinea corporis discrete lesions
Source: Bart Currie — Menzies School of Health Research

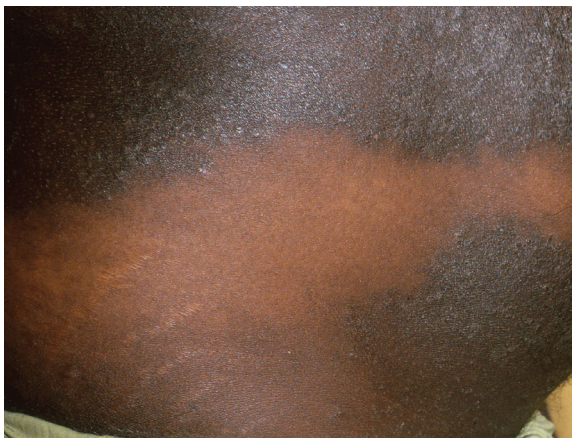


Figure 88: Tinea corporis extensive pigmentation
Source: Bart Currie — Menzies School of Health Research



Figure 89: Tinea – hand and nails
Source: Bart Currie — Menzies School of Health Research

Tinea corporis

PRINCIPLES OF MANAGEMENT

Smaller lesions respond to topical anti-fungals such as terbinafine 1% cream or miconazole 2% cream applied over lesions and to 3cm beyond the active margin twice per day for 2-4 weeks.

Extensive lesions require oral anti-fungals. While griseofulvin was the standard therapy for many years, this drug is less effective and requires treatment for many weeks. Two weeks terbinafine (fungicidal) orally daily is usually curative for tinea corporis. However, reinfection is common and recrudescence from residual nail disease occurs if nail disease is present and not treated with a longer course of terbinafine.

If greater than 2 weeks of terbinafine is planned, liver function should be checked before starting treatment. Similarly, if the patient has acute or chronic liver disease, renal disease, is over 40 years or consumes excessive alcohol, check UECs, LFTs and FBC before terbinafine treatment. Re-check LFTs and FBC at 2 and 4 weeks of treatment. Therapy and monitoring can continue if there is only mild liver function abnormality, but therapy must be stopped immediately if neutropenia develops. Wait until after pregnancy and breast feeding before treating tinea corporis.

Clinical assessment of household contacts is recommended as they may also have tinea which leads to reinfection.

During the past decade highly infectious and aggressive *Tinea indotineae* infections have been reported globally, having emerged in India, and are now being seen in Australia. Isolates are often resistant to terbinafine. Clinicians should be aware of this infection and perform scrapings for fungal microscopy and culture in patients where the tinea corporis infection has not responded to antifungal treatment or where the tinea infection is particularly widespread.



Figure 90: Tinea (pityriasis) versicolor — not T. rubrum (differential diagnosis)
Source: Bart Currie — Menzies School of Health Research

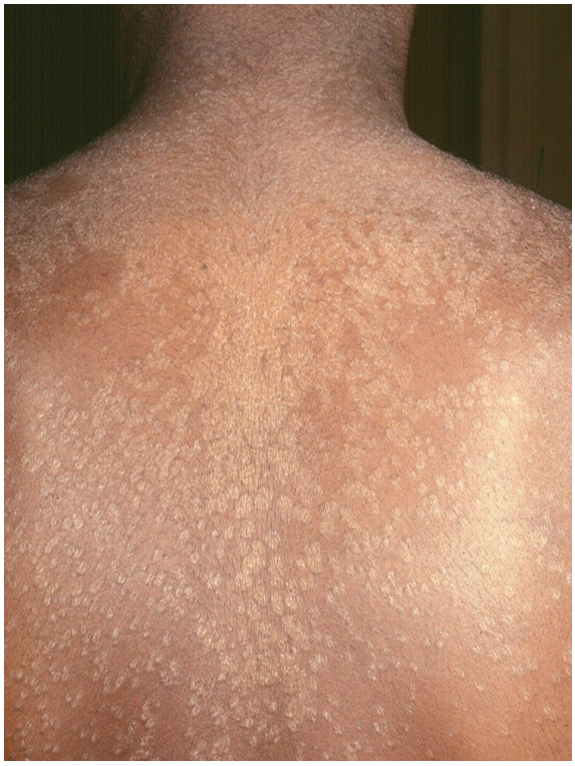


Figure 91: Tinea (pityriasis) versicolor — not T. rubrum (differential diagnosis)
Source: Bart Currie — Menzies School of Health Research

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call infectious disease physician and local dermatologist.

MANAGEMENT GUIDELINES

Northern Territory		
NT Centre for Disease Control (CDC)	Healthy Skin Program — Guidelines for Community Control of Scabies, Skin sores, Tinea and Crusted Scabies in the Northern Territory	Available online

RESOURCES

Green, Allen	A Handbook skin conditions in Aboriginal populations of Australia. Carlton South: Blackwell Science Asia; 2007	Available online
Menzies School of Health Research	Healthy Skin Story — Flip Charts	Available online
Remote Primary Health Care Manuals (RPHCM)	CARPA Standard Treatment Manual - Tinea	Available online

FURTHER READING

Chua KYL, Halliday CL, Chen SCA, et al. Treatment-resistant tinea caused by Trichophyton indotineae in Australia. Medical Journal of Australia. 2024;221:192-194.

May PJ, Tong SYC, Steer AC, et al. Treatment, prevention and public health management of impetigo, scabies, crusted scabies and fungal skin infections in endemic populations: a systematic review. Tropical Medicine & International Health: TM & IH. 2019;24(3):280-293.

Onychomycosis

Fungal infections of the finger and toe nails are common in tropical climates.

ONYCHOMYCOSIS IN NORTHERN AUSTRALIA

Onychomycosis is usually a dermatophyte infection of the finger or toenails and is usually secondary to other fungal infections, such as tinea corporis (page 101). Fungal infections are also passed on to other people via skin or nail contact, or contact with contaminated surfaces, such as in bathrooms.

AETIOLOGY AND PATHOGENESIS

Trichophyton rubrum is the dermatophyte responsible for nearly all cases of onychomycosis, with other non-dermatophyte yeasts and moulds causing a minority of cases.

CLINICAL PICTURE

Symptoms and signs. Toenails are more commonly affected than fingernails. The nails usually turn white or yellow, crack and have irregular edges. Changes first appear at the free distal edge of the nail. Proximal subungual onychomycosis is often associated with immunosuppression. Subungual hyperkeratosis may cause the nail to become detached from the nail bed. Frequently not all nails are involved, even in chronic infections of many years' duration. Tinea pedis, manuum or corporis is usually present. Paronychial inflammation is absent.

Investigations. Clip the distal nail with nail clippers and scrape material from under the distal nail and infected nail surface with a curette, or spatula, for microscopic examination and culture.

Laboratory diagnosis is important for the following reasons:

1. Onychomycosis may be caused by a mould or yeast that will not respond to oral terbinafine.
2. Treatment is needed for a long time.
3. Treatment is expensive.
4. Treatment has potential side effects (need to monitor liver function and FBC).
5. Only proven cases of fungal infection are eligible for the Pharmaceutical Benefit Scheme subsidy.

DIFFERENTIAL DIAGNOSIS

- Psoriasis — can be clinically difficult to differentiate. Look for evidence of psoriasis elsewhere in scalp, ears and on elbows and knees.
- Other skin diseases such as lichen planus and eczema.
- *Candida albicans* — usually in proximal nail plate with paronychial inflammation and lacks gross distortion and subungual debris.
- Bacterial paronychia.
- Subungual hyperkeratosis.



Figure 92: Onychomycosis — nail tinea with Trichophyton rubrum
Source: Bart Currie — Menzies School of Health Research

PRINCIPLES OF MANAGEMENT

Explain to patients that oral treatment is often required for three months (fingernails) to six months (toenails). Regular cutting and thinning of the nails by a podiatrist may improve cure rate. Oral terbinafine is the treatment of choice. As treatment is needed for more than 2 weeks, renal function, liver function and FBC should be checked before and during treatment. Therapy and monitoring can continue if there is only mild liver function abnormality, but therapy must be stopped immediately if neutropenia develops. Wait until after pregnancy and breast feeding before treating onychomycosis. Footwear worn during the time of infection should be thrown away and new footwear purchased, and bathroom floors bleached. Most nails with extensive onychomycosis will still look abnormal after a course of three months of oral terbinafine as new nails can take nine to 12 months to grow. If the treatment is working, there should be healthy nail growing from the proximal aspect of the fingernail and treatment can be continued. If this is not the case, then referral to a skin specialist is recommended.

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call infectious disease physician and local dermatologist.

FURTHER READING

Adams C, Athanasoula E, Lee W, et al. Environmental and Genetic Factors on the Development of Onychomycosis. J Fungi (Basel). 2015;1(2):211-216.

Scabies

The scabies mite typically causes persistent, generalised, nocturnally- aggravated itch. Secondary bacterial skin infections can lead to life-long kidney and heart disease.

SCABIES IN NORTHERN AUSTRALIA

Scabies infestation and associated streptococcal skin infections are among the most common and important skin conditions in First Nations people in Northern Australia. The high prevalence of streptococcal skin infection contributes to the extremely high rates of post-streptococcal glomerulonephritis and rheumatic fever.

Surveys in First Nations communities have shown high but variable prevalence rates of scabies infestation ranging from 30–50% in children under the age of 15 years, with approximately 70% of infants being affected in the first year of life, and up to 45% of children with streptococcal skin sores. At least one-in-three adults were infected with scabies at any one time, with generally milder clinical manifestations.

Decreased skin barrier function resulting from mites burrowing into the skin, combined with itch-induced excoriations provides a niche for bacterial skin infections, particularly *Streptococcus pyogenes* and *Staphylococcus aureus*.

AETIOLOGY AND PATHOGENESIS

Scabies is caused by a parasitic mite, *Sarcoptes scabiei* var. *hominis*, which is typically transmitted from person to person through close household contact. The gravid female mite burrows and deposits two to three eggs a day in the skin's stratum corneum. The nymphs emerge as adults on the surface of the skin after a series of moults over about 2 weeks. The mature mites then mate and reinvade the skin of the same or another host.

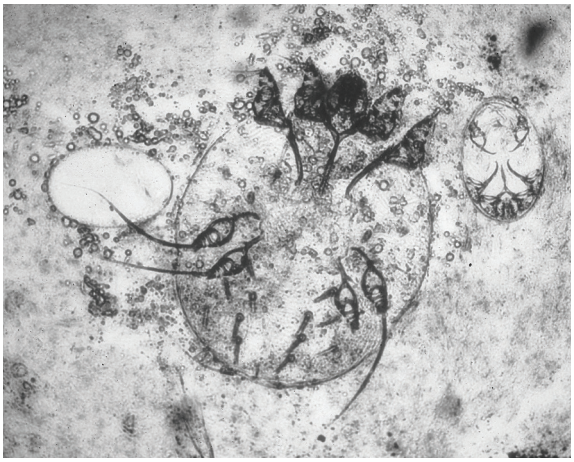


Figure 93: Scabies female with eggs
Source: Bart Currie — Menzies School of Health Research

Initially, the infestation is asymptomatic. After 4–6 weeks the host becomes sensitised to the excreta of the mites and itchy lesions develop. With subsequent episodes of infestation, the host develops the symptomatic hypersensitivity reaction much sooner.

Crusted scabies (also known as Norwegian scabies) is a severe form of the disease, typically observed in people with immune deficiency, the elderly, as well as those with impaired ability to scratch, such as physical disability or severe mental illness. In the Northern Territory, the underlying immune predisposition may be more complex and subtle, such as in Central Australia, where HTLV-1 (page 91) is a common link. In crusted scabies, mite replication escapes the body's containment mechanisms, leading to hosts becoming hyper-infested with thousands-to-millions of mites, developing initially scaly skin, then thickened skin with hyperkeratosis (increase in adherent dead skin cells) or crusting (plasma extruding through an eroded epidermis). They are highly infectious to others due to shedding of hyper-infested hyperkeratotic skin and are themselves highly susceptible to reinfestation and recurrent episodes of crusted scabies.

CLINICAL PICTURE

Risk factors. Scabies is typically transmitted through direct contact with an infected person and through contact with fomites such as bedding, clothing and soft furnishings. Poverty and overcrowded living conditions are risk factors for scabies. People with crusted scabies are 'key transmitters' as they carry large numbers of mites. It is always worth considering whether scabies is the underlying cause of skin sores in high-risk individuals.

Symptoms and signs.

Simple scabies: The distribution and severity of scabies infestation is directly related to the body's immune response. The younger the child, the more likely they are to have a greater number and wider distribution of itchy papular, pustular and nodular lesions, with associated dermatitis and/or secondary infection (impetiginisation). Older children and immunocompetent adults often have fewer lesions, limited to wrists, ankles, and in the interdigital web-spaces on hands and feet. In infants and young children, the lesions may be generalised, and it can manifest as a purely pustular eruption on palms and soles. Such pustular eruptions are not necessarily infected with bacteria, but rather, caused by the immune response to the mite. The elderly can also

develop a similar pustular eruption limited on the trunk as the only manifestation of the condition, hampering diagnosis. Contrary to popular belief, scabies lesions may also be found on the face and scalp in infants and the elderly. Hypersensitivity nodules are areas of extreme immunological response to scabies mite infestation, manifesting typically as 5-10mm extremely itchy nodules on the buttocks, upper thighs, genitalia and the nipple-areola complex. There does not appear to be a correlation between the location of a hypersensitivity nodule and any individual mite, but rather a manifestation of the immune response. Instances of secondary infection typically manifest as crusted, pustular or weeping lesions.

Crusted (Norwegian) scabies is often not itchy and presents as areas of hyperkeratosis and / or crusting, anywhere on the body, including of the limbs, trunk, and lower back. Lesions without secondary infection are dry, silvery, and powdery when scraped. They are often poorly demarcated. Examination should encompass the full surface of the skin, including in skin folds, of the natal cleft, and buttocks by a practitioner of the same gender. In more subtle cases examination of the web spaces of the hands (interdigital folds) may reveal fine scale and silvered skin, typically of the dominant hand first.

Clinicians should become suspicious of the presence of a household contact with crusted scabies when seeing recurrent presentations of scabies within an



Figure 94: Scabies
Source: Bart Currie — Menzies School of Health Research



Figure 96: Sixteen year old with infected scabies
Source: Bart Currie — Menzies School of Health Research



Figure 95: Scabies with streptococcal pyoderma
Source: Bart Currie — Menzies School of Health Research



Figure 97: Sixteen year old with infected scabies
Source: Bart Currie — Menzies School of Health Research

Scabies

individual or household. Circular reinfestation (where transmission to a family member has already occurred by the time a symptomatic individual has been treated) within the household may also account for recurrent infestations with scabies.

Scabies, and in particular crusted scabies, is a stigmatised condition. This stigma may substantially delay presentation to the health service.

Investigations. Investigations are not routinely required for simple scabies because the clinical picture is usually pathognomonic, but skin scrapings are mandatory for diagnosing crusted scabies. Careful examination of the skin using wearable loupes or a dermatoscope will often allow identification of burrows, often between fingers or at the wrist crease and examination under a dermatoscope can also reveal the front legs and mouthparts of the mites as a dark granular isosceles triangle at the end of the burrow, also known as the "delta" or "contrail" signs. Burrows can be hard to identify in patients with darker skin types but are frequently identifiable in these patients on their palms and soles. Scrapings can be taken using a scalpel and examined under a light microscope for further confirmation if needed, however, false negative results are common in patients with low numbers of mites. By contrast, in crusted scabies, scrapings are much more sensitive due to the sheer number of mites in affected skin – it is worth wearing gloves and performing strict hand hygiene due to the highly infectious nature of this condition. Where diagnosis remains uncertain, Reflectance Confocal Microscopy has been used at Royal Darwin Hospital for real-time in-vivo analysis, as a non-invasive diagnostic method. A swab of any associated skin sores will usually culture streptococcus and often staphylococcus.

Patients with crusted scabies should be investigated to exclude underlying immune deficiencies such as renal failure, diabetes, systemic lupus erythematosus (SLE) and Human T-cell lymphotropic virus type 1 (HTLV-I). Recommended tests are in the Centre for Disease Control Guidelines for Community Control of Scabies, Skin Sores, Tinea and Crusted Scabies in the Northern Territory — see *Management guidelines* page 113.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis for scabies includes dermatitis, tinea, and psoriasis. Differentiating between crusted scabies, dermatitis, impetiginised dermatitis, impetiginised scabies, tinea and discoid lupus can be difficult as these conditions can all produce hyperkeratosis and crusting.

PRINCIPLES OF MANAGEMENT

For treatment details refer to scabies and skin sore guidelines in your region (e.g. NT CDC Guidelines, CARPA Standard Treatment Manual). Overdiagnosis of scabies has been reported in the Darwin region. Simply having itch and being from a scabies endemic location does not meet the threshold for making a diagnosis of scabies. The International Alliance for the Control of Scabies criteria for the diagnosis of scabies stratifies the basis for the diagnosis (Figure 109), and it is encouraged that clinicians document the basis for the diagnosis in case the patient fails to improve with treatment and clinicians must then distinguish between treatment failure and an incorrect diagnosis of scabies.

The mainstay of **treatment for simple scabies** consists of two whole-body applications of 5% permethrin cream, administered one week apart for the individual and all symptomatic household contacts. Attention needs to be paid to ensure application to all creases, under nails, soles of feet, face, scalp and neck, ensuring all skin is treated (larger individuals may need 2 tubes per application to achieve this). Permethrin can be given at the same time as benzathine benzylpenicillin or cotrimoxazole for streptococcal infection. Two 200mcg/kg stat doses of oral ivermectin can be given (with fatty food such as a glass of milk) one week apart accompanying the permethrin cream treatments either as first line treatment or if monotherapy with topical permethrin fails. Asymptomatic household contacts will require a single application of permethrin.

To prevent circular reinfestation, it is vital that linen, clothing, and furniture are treated with hot washes, freezing, or fumigation as appropriate and as outlined in local guidelines.

Improvement in itch often lags behind the treatment of scabies, sometimes by several weeks, or it can persist for months afterwards, termed "post-scabetic itch". Some patients develop other dermatologic complications such as dermatitis, prurigo nodularis or delusions of parasitosis so it is important to be vigilant for these sequelae and refer to Dermatology if the itch is recalcitrant.

Community wide treatment of dogs effected by sarcoptic mange is unnecessary as the dogs have different types of mites that are unlikely to infect people, although they may cause irritant dermatitis which can result in secondary bacterial skin infections.

Crusted scabies treatment involves both topical treatment with permethrin and a keratolytic cream to soften the crust and allow permethrin to penetrate as well as several doses of oral ivermectin. The exact dosing and intervals are determined by the severity of infestation and clinicians should refer to the CDC and Royal Darwin Hospital crusted scabies protocols, or patients can be discussed with an infectious diseases physician. Patients with crusted scabies almost always need treatment in hospital, which also allows time for community clinic staff and environmental health officers to educate the family and advice regarding optimal household cleaning, treatment of household contacts and have supports implemented for long term keratolytic moisturiser application and the creation of a scabies free zone, in conjunction with the patient's GP and local support services.

The main complication of crusted scabies is septicaemia. A study conducted at Royal Darwin Hospital in the mid 1990's showed that people with severe crusted scabies had a 5-year mortality rate of 50%, which is higher than that of many malignancies. All the deaths were due to septicaemia following bacterial penetration through cutaneous fissures. Aggressive antibiotic therapy and supportive care has since reduced this mortality.

Healthy skin programs. Community programs to reduce the prevalence of scabies and streptococcal skin disease have been run in Top End communities. Guidelines are available from CDC. The programs involve education, development of local resources and a one-off treatment of the entire community (Mass Drug Administration) with oral ivermectin and permethrin cream for young children and in pregnancy. MDA provides initial reductions in disease prevalence, but there is often gradual return to prior infestation rates as scabies is reintroduced to the community.

A. Confirmed scabies

At least one of:

- A1: Mites, eggs or faeces on light microscopy of skin samples
- A2: Mites, eggs or faeces visualised on an individual using a high-powered imaging device
- A3: Mites visualised on an individual using dermoscopy

B. Clinical scabies

At least one of:

- B1: Scabies burrows
- B2: Typical lesions in a typical distribution and two history features

C. Suspected scabies

One of:

- C1: Typical lesions in a typical distribution and one history feature
- C2: Atypical lesions or atypical distribution and two history features

History features

- H1: Itch
- H2: Positive contact history

Figure 98: Summary of the 2020 International Alliance for the Control of Scabies Concensus Criteria for the Diagnosis of Scabies
Source: Engelman D, Yoshizumi J, Hay RJ, et al. The 2020 International Alliance for the Control of Scabies Consensus Criteria for the Diagnosis of Scabies, British Journal of Dermatology. 2020;183(5)1:808-820.

Scabies



Figure 99: Crusted scabies
Source: Bart Currie — Menzies School of Health Research



Figure 100: Crusted scabies
Source: Bart Currie — Menzies School of Health Research



Figure 101: Crusted Scabies with fissures
Source: Bart Currie — Menzies School of Health Research



Figure 102: Crusted scabies with residual disease under nails
Source: Bart Currie — Menzies School of Health Research



Figure 103: Severe crusted scabies with exfoliation and fissures
Source: Bart Currie — Menzies School of Health Research



Figure 104: Less severe crusted scabies with scale rather than crusts
Source: Bart Currie — Menzies School of Health Research



Figure 105: Nodular scabies, with secondary herpes simplex infection



Figure 106: Scabies with secondary pyoderma, not crusted scabies



Figure 107: Crusted scabies with extensive scaling hyperkeratosis rather than crusting
Source: Bart Currie — Menzies School of Health Research



Figure 108: Nodular scabies treated as crusted scabies



Figure 109: Scabies with secondary pyoderma, not crusted scabies

Scabies

Crusted scabies is a notifiable condition to be reported by LABORATORIES in the Northern Territory. Cases are reported to the local Centre for Disease Control/Public Health Unit.

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call infectious disease physician, dermatologist or local CDC/PHU.

MANAGEMENT GUIDELINES

National		
The Australian Healthy Skin Consortium	National Healthy Skin Guideline: for the Prevention, Treatment and Public Health Control of Impetigo, Scabies, Crusted Scabies and Tinea for Indigenous Populations and Communities in Australia	Available online
Northern Territory		
NT Health	Public Health Management of Crusted Scabies	Available online
NT Centre for Disease Control (CDC)	■ Healthy Skin Program — Guidelines for Community Control of Scabies, Skin Sores, Tinea and Crusted Scabies in the Northern Territory ■ NT Guidelines for the Control of Acute Post-streptococcal Glomerulonephritis ■ Public Health and Notifiable Diseases	Available online
Remote Primary Health Care Manuals (RPHCM)	CARPA Standard Treatment Manual — Scabies	Available online
NT Primary Health Network (NT PHN)	HealthPathways - Scabies	Available online (CLICK HERE to request access)

RESOURCES

CRANAplus	Crusted Scabies in the NT (online seminar, 50min)	Available online
NT Health	■ Scabies and crusted scabies - Resources ■ Crusted scabies and hospital story - English (video)	Available online

FURTHER READING

Einsiedel LJ, Pepperill C, Wilson K. Crusted scabies: a clinical marker of human T-lymphotropic virus type 1 infection in central Australia. Medical Journal of Australia. 2014;200(11):633-634.

Engelman D, Yoshizumi J, Hay RJ, et al. The 2020 International Alliance for the Control of Scabies Consensus Criteria for the Diagnosis of Scabies. Br J Dermatol. 2020;183(5):808-820.

Hasan T, Krause VL, James C, Currie BJ. Crusted scabies; a 2-year prospective study from the Northern Territory of Australia. PLoS Negl Trop Dis. 2020 Dec 18;14(12):e0008994.

Kearns TM, Andrews R, Speare R, et al. Prevalence of scabies and strongyloidiasis before and after MDA in a remote Aboriginal community in Northern Territory, Australia. International Journal of Infectious Diseases. 2014;21:252.

Kearns TM, Speare R, Cheng AC, et al. Impact of Ivermectin mass drug administration on scabies prevalence in a remote Australian Aboriginal community. PLoS Neglected Tropical Diseases. 2015;9(10):e0004151.

Lokuge B, Kopczynski A, Woltmann A, et al. Crusted scabies in remote Australia, a new way forward: lessons and outcomes from the East Arnhem Scabies Control Program. The Medical Journal of Australia. 2014;200(11):644-648.

May PJ, Tong SYC, Steer AC, et al. Treatment, prevention and public health management of impetigo, scabies, crusted scabies and fungal skin infections in endemic populations: a systematic review. Tropical Medicine & International Health. 2019;24(3):280-93.

Head lice

Head lice are small wingless insects, which are usually transmitted via head to head contact.

HEAD LICE IN NORTHERN AUSTRALIA

The prevalence of head lice in Northern Australia is high, particularly in remote First Nations communities. It is estimated that up to 50-60% of all primary school children in the Northern Territory have experienced head lice. High levels of humidity increase the prevalence during the build-up and wet seasons.

AETIOLOGY AND PATHOGENESIS

Head lice are 2–3mm long, opaque to dark-brown wingless insects. Nits are the small eggs laid by the lice which are bonded onto the hair shaft close to the scalp. They are yellow to white in colour and are difficult to remove. The eggs hatch in 7–10 days. They die quickly on brushes, hats, pillows and furniture. Notably in hot and humid environments they can live longer outside of the hair. The lice are highly transferable, for example when an adult hugs a child.

CLINICAL PICTURE

Head lice affect people of all ages and are most common in children aged 3-13 years.

Risk factors include being female (longer hair), greater number of children in the household and sharing beds. Infestation may be asymptomatic, although it often causes an itchy scalp. Itch and irritation can affect quality of sleep and increase stress levels. Head lice can also lead to skin infections through extreme itching enabling entry of bacteria. Cleanliness does not stop head lice, so all close contacts need to be checked. To find lice and nits, look at the scalp / hair behind the ears and nape of the neck with a strong light. The are more difficult to see on fairer hair.

Complications include dermatitis, crusted sores with bacterial infection and tender lymph nodes.

DIFFERENTIAL DIAGNOSIS

Other causes of scalp itching include over-treating with insecticide, eczema, psoriasis and contact dermatitis. Occipital scalp abscesses are often associated with head lice infestation.

PRINCIPLES OF MANAGEMENT

Insecticides are no longer routinely used due to concerns relating to toxicity, irritation, discomfort and resistance.

Current recommended treatment is with an occlusive product containing dimethicone that coats and smothers the lice, and nerolidol that kills the nits. The product is applied to clean dry hair covering each hair from root to tip. The stunned (and dead) lice and nits are then able to be removed using a fine-toothed comb. Comb in sections, wiping on a tissue to check for lice and nits. Check in one week using a fine-tooth comb. The treatment can be repeated every 7 days as required.

In all cases:

- Check and treat all family members at the same time.
- Recommend regular hair checks to rapidly identify re-infestation.
- Support school-based eradication campaigns to decrease the rate of re-infestation.
- Keep long hair tied back or braided or consider keeping the hair short.

Detailed information is available in the Head Lice Advice from CDC and in the product information.

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call paediatrician or local CDC/PHU.

MANAGEMENT GUIDELINES

Northern Territory		
NT Centre for Disease Control (CDC)	Information for Parents, Schools and Child Care Centres. Head Lice Advice	Available online
Remote Primary Health Care Manuals (RPHCM)	CARPA Standard Treatment Manual — Head lice (nits)	Available online

RESOURCES

NT Centre for Disease Control (CDC)	■ Fact Sheet — Head Lice and Nits ■ Kid's nit booklet ■ Head lice advice posters	Available online

FURTHER READING

Amanzougaghene N, Fenollar F, Raoult D, Mediannikov O. Where Are We With Human Lice? A Review of the Current State of Knowledge. Front Cell Infect Microbiol. 2020;9:474.

Cook S, Ellis I, Knight S, Lenthall S. Headlice: a precursor to Group A Streptococcal infection in remote Indigenous children. Primary Intention: The Australian Journal of Wound Management. 2007;15(4):181.

Paederus australis (the 'acid beetle')

Paederus australis is a beetle that causes an irritant contact dermatitis characterised by linear streaks and 'kissing lesions'.

PAEDERUS AUSTRALIS IN NORTHERN AUSTRALIA

Paederus australis, (the 'acid beetle' or 'whiplash rove beetle'), lives in swamps and riverbanks, feeds on other insects and decaying animal or vegetable matter and is attracted to lights at night. Communities close to flood plains can experience plagues of beetles, especially following heavy rainfall. Outbreaks can be so severe that evacuation of an entire outstation may be required.

AETIOLOGY AND PATHOGENESIS

Paederus australis is thin and 5–10mm long with brightly coloured sections of blue/black and orange. It produces a defensive toxic substance called Pederin, which is released by squashing, swatting or swiping the beetle on the skin.

Initial contact with the beetle often occurs at night and is painless. Skin damage takes several hours and even a day to occur, and patients may not associate the symptoms with the beetle and may often not be aware of the beetle contact that has occurred when asleep.

CLINICAL PICTURE

Symptoms and signs. *Paederus australis* causes an irritant contact dermatitis characterised by linear streaks and 'kissing lesions' when two skin surfaces are in contact with each other, such as above and below the elbow on flexing and face/neck/shoulder from head tilting such as when asleep.

The erythematous rash appears about 24 hours after contact with the beetle and subsequently often blisters, usually becoming very painful then itchy. After about a week the crusted lesions become dry and then desquamates. Healing occurs during the second week, leaving an initially dark pigmented area that fades but may persist for weeks. It has also been reported to cause acute keratoconjunctivitis with marked oedema of the eyelids. Contact with multiple beetles can cause extensive blistering over limbs, trunk and face that requires hospitalisation for wound care and can become secondarily infected.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include herpes simplex or zoster, impetigo, other forms of irritant or contact dermatitis such as from plants or caterpillars, conjunctivitis, and the periorbital swelling of acute glomerulonephritis.

PRINCIPLES OF MANAGEMENT

Treatment is symptomatic as for superficial minor burns taking care to avoid secondary infection. Hospitalisation is sometimes required for extensive acid beetle burns. Simple analgesics may be needed. Topical steroids have been recommended by some clinicians, but they are not advised for circumstances where secondary bacterial infection is common.



Figure 110: Acid or whiplash rove beetle Paederus australis
Source: Bart Currie — Menzies School of Health Research



Figure 111: Acid beetle burn — kissing lesion
Source: Bart Currie — Menzies School of Health Research



Figure 112: Acid beetle also called blister beetle – kissing lesions above and below elbow

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the local medical entomologist or local CDC/PHU.

KEY REFERENCES AND FURTHER READING

Whelan P. Bites and stings in the Top End and how to avoid them. The Northern Territory Disease Control Bulletin. 2005;12(3):20-27.

Other bites and stings

Other stings and contact skin reactions can be seen in the following pictures.



Figure 113: Sea urchin spines
Source: Bart Currie — Menzies School of Health Research



Figure 115: Sea urchin spines extracted from skin after 17 days
Source: Bart Currie — Menzies School of Health Research



Figure 114: Orb-weaving spider
Source: Jayde Hopkins — Menzies School of Health Research



Figure 116: Bite from an Orb-weaving spider. Note inflammation from contact with the spider's leg hairs
Source: Jayde Hopkins — Menzies School of Health Research



Figure 117: Mango sap rash
Source: Bart Currie — Menzies School of Health Research



Figure 118: Stings from Freshwater mangrove itchy caterpillar Euproctis lutea
Source: Bart Currie — Menzies School of Health Research



Figure 119: Caterpillar Euproctis lutea
Source: Bart Currie — Menzies School of Health Research



Figure 120: Biting midges (Culicoides) in a fishing doctor new to the Top End
Source: Bart Currie — Menzies School of Health Research



Figure 121: Biting midges (Culicoides) in a fishing scientist new to the Top End
Source: Bart Currie — Menzies School of Health Research

Other bites and stings



Figure 122: Phytodermatitis
Inflammation and severe itch with delayed onset of 24 hours or more – in this case from contact pulling down dieffenbachia vines with gloves protecting hands but not forearm. Rapid response to topical steroids and antihistamines. (See Figure 123)
Source: Emma Smith – Royal Darwin Hospital



Figure 123: Dieffenbachia vines
Source: Emma Smith – Royal Darwin Hospital



Figure 124: Likely centipede bite – note the wide distance between punctures from the venomous claws (maxillipedes)



Figure 125: Palm thorn lacerations (See Figure 126)
Source: Bojana Simsic – Royal Darwin Hospital



Figure 126: Palm thorns
Timely washing and wound care prevents infection from environmental bacteria or fungi (thorn-associated or soil) or own staphylococci or streptococci flora.
Source: Bojana Simsic – Royal Darwin Hospital



Figure 127: Palm thorn lacerations
Source: Bojana Simsic – Royal Darwin Hospital

Hookworm

Hookworm infections cause iron deficiency anaemia, but regular deworming programs can dramatically reduce this problem.

HOOKWORM IN NORTHERN AUSTRALIA

Hookworm is a common faecal/soil transmitted helminth that affects 480 million people globally. It is particularly common in tropical areas and used to be a major health issue in remote First Nations communities in Northern Australia. While it is an important cause of anaemia globally, regular deworming programs in Northern Australia have dramatically reduced its contribution to iron deficiency anaemia.

AETIOLOGY AND PATHOGENESIS

There are two species of human hookworm, *Ancylostoma duodenale* (Old World hookworm) and *Necator americanus* (New World hookworm). The zoonotic *Ancylostoma ceylanicum* is a species seen across many regions of SE Asia and is present in the Northern climes of Australia. *A. duodenale* is the dominant hookworm present in Northern Australia. The usual mode of transmission is by penetration of the skin by filariform larvae. However, *A. duodenale* can also be transmitted by ingestion or breastfeeding.

After entering the host, the larvae are carried in the blood to the right side of the heart. They enter the alveoli, ascend the bronchial tree and are swallowed. In the small intestine they mature into adult worms, attach to the small bowel mucosa where they feed on the intestinal villi and suck approximately 0.2mL of blood per day. Adult *A. duodenale* live for less than a year and produce up to 30,000 eggs per day. The period from skin invasion to appearance of eggs in the faeces is about 6–8 weeks, but during the dry season, the larvae can remain dormant in the tissues for months, until just before the onset of the wet season. Egg laying begins approximately 1 month before the beginning of the wet season. Pregnancy also induces this dormant state with resumption of development on parturition. The eggs are deposited with faeces in

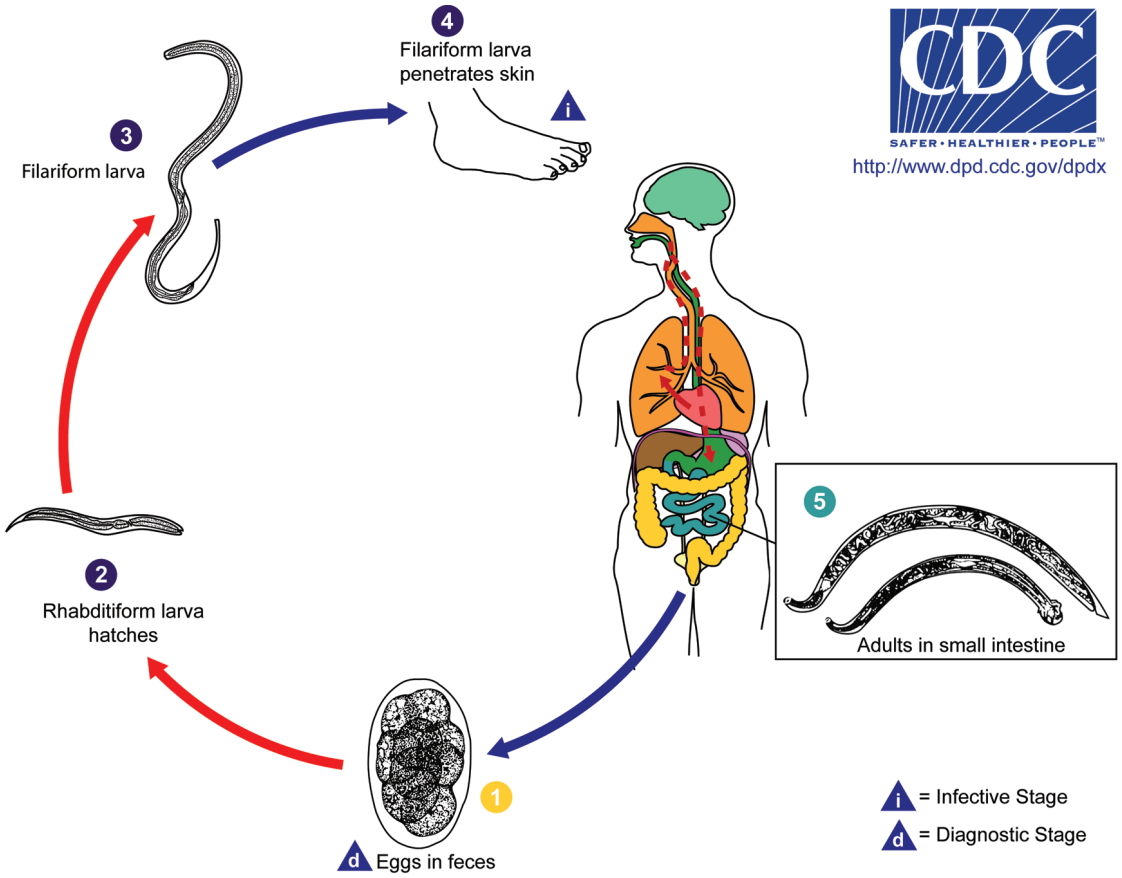


Figure 128: The life cycle of hookworm
Source: https://www.cdc.gov/dpdx/hookworm/index.html

soil where they hatch into rhabditiform larvae which develop over one week into the filariform larvae which can invade the host.

Helminth, including hookworm, infections have been shown to immunomodulate the host. There is evidence that hookworms not only blunt responses to vaccines but also reduce risk of progression to type 2 diabetes and other inflammatory conditions.

CLINICAL PICTURE

Risk factors. Exposure to contaminated soil, particularly in settings of poor sanitation where hookworm is prevalent.

Symptoms and signs. Most hookworm infections are asymptomatic. People with low worm loads are considered carriers, whereas heavy loads (expressed as ≥4,000 eggs per gram) cause disease. Infective larvae may cause pruritic maculopapular dermatitis ('ground itch') at the site of penetration. Serpiginous tracts of subcutaneous migration (similar to cutaneous larva migrans) may occur in previously sensitised hosts. Larvae migrating through lungs occasionally cause mild transient pneumonitis. Early intestinal infection may cause epigastric pain, inflammatory diarrhoea, or other abdominal symptoms accompanied by eosinophilia.

An important consequence of chronic hookworm infection is iron deficiency which may be severe with weakness, shortness of breath and pallor. In pregnant women infection contributes to low birth weight and prematurity. Chronic anaemia in children causes growth retardation and delayed cognitive development.

Investigations. Faecal microscopy may demonstrate characteristic oval eggs. Stool concentration techniques may be required. Blood tests may demonstrate hypochromic, microcytic anaemia, eosinophilia or hypoalbuminaemia.

DIFFERENTIAL DIAGNOSIS

Larvae may be confused with those of *Strongyloides stercoralis*. *Ancylostoma caninum*, the dog hookworm, has been identified as a cause of human eosinophilic enteritis, especially in northeastern Australia and diagnosed in a case at Numbulwar in the Northern Territory. Cutaneous larva migrans is caused by larvae of the cat and dog hookworm *(Ancylostoma braziliense)* migrating slowly through the skin of humans who are not the preferred host. *A ceylanicum* has been detected in north-east Arnhem Land.



Figure 129: Ancylostoma duodenale egg.
Source: https://www.cdc.gov/dpdx/hookworm/index.html



Figure 130: Hookworm (ancylostoma duodenale)

Hookworm

PRINCIPLES OF MANAGEMENT

Treatment: Anthelmintic drugs can safely and effectively treat hookworm infection, with albendazole being the current preferred anthelmintic. Regular deworming treatments are part of child health programs in remote communities in Northern Australia. Severe hookworm disease with protein loss and malabsorption is rare and requires additional nutritional support. In the absence of reinfection, most worms are eliminated spontaneously within 2 years. Cutaneous larva migrans responds rapidly to anthelmintic drugs (see Therapeutic Guidelines: Antibiotic).

Prevention includes community education, avoiding skin contact with contaminated soil, and improved sanitation infrastructure and practices. For more information about the prevention and treatment of anaemia, see *Case study — Anaemia* page 205.



Figure 132: Cutaneous larva migrans from Ancylostoma braziliense (cat–dog Hookworm)
Source: Bart Currie — Menzies School of Health Research



Figure 131: Cutaneous larva migrans from Ancylostoma braziliense (cat–dog Hookworm)
Source: Bart Currie — Menzies School of Health Research

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call paediatrician or local CDC/PHU.

MANAGEMENT GUIDELINES

Northern Territory		
Remote Primary Health Care Manuals (RPHCM)	CARPA Standard Treatment Manual — Worms	Available online

FURTHER READING

Beknazarova M, Whiley H, Traub R, Ross K. Opportunistic Mapping of Strongyloides stercoralis and Hookworm in Dogs in Remote Australian Communities. Pathogens. 2020;9(5):398.

Pierce DR, McDonald M, Merone L, et al. Effect of experimental hookworm infection on insulin resistance in people at risk of type 2 diabetes. Nat Commun. 2023;14(1):4503.

Strongyloidiasis

Strongyloidiasis is a nematode infection that can affect any organ of the body. Disseminated Strongyloides is a potentially fatal sequelae of the disease but has only been documented in immunosuppressed people.

STRONGYLOIDIASIS IN NORTHERN AUSTRALIA

Strongyloidiasis is caused by the parasitic nematode, *Strongyloides stercoralis*. It is common throughout tropical regions of the world where warmth, moisture and poverty favour its spread. Worldwide, an estimated 370 million people are thought to be infected and prevalence rates of above 5% have been defined as hyperendemic.

Strongyloidiasis occurs throughout Northern Australia. Historically, some Arnhem Land communities demonstrated positive isolation rates from stools ranging from 15% to 41% and seropositivity in up to 60% of people tested. In Central Australia, it also occurs in conjunction with HTLV-1.

AETIOLOGY AND PATHOGENESIS

Strongyloides stercoralis is a faecal soil transmitted helminth. The most common mode of transmission is penetration of the skin by the infective filariform larvae.

Most larvae are then carried in the bloodstream to the right side of the heart. They enter the alveolar spaces in the lungs, ascend the bronchial tree and are swallowed. In the small intestine the larvae mature into adult female worms (2mm long) and tunnel through the absorptive epithelium of the jejunum where they can lay up to 40 eggs a day. The parasitic females can reproduce without males by parthenogenesis. This begins 17–28 days after the initial infection. The eggs hatch in the intestinal mucosa, releasing rhabditiform larvae that migrate to the lumen of the bowel.

Three cycles are possible:

- **Direct host-soil-host cycle.** (as above) The rhabditiform larvae pass out of the body with the faeces. They enter the soil and become filariform larvae that can then infect the host by penetrating the skin.
- **Indirect cycle.** The rhabditiform larvae pass out of the body with the faeces, enter the soil and develop into free-living male and female adults. The total life span in the soil is considered to be up to 3 weeks.
- **Autoinfection, hyper infection and disseminated infection.** A proportion of the rhabditiform larvae will mature within the colon to filariform larvae, they will re-enter the circulation from there and migrate to the lungs and then be swallowed. This is described as autoinfection and allows the parasite to complete its lifecycle without leaving the host. Under certain physiological conditions such as immunosuppression and malnutrition the proportion of larvae following this route intensifies greatly. Disseminated infection occurs when large numbers of larvae migrate to organ systems outside of the autoinfection route such as the CNS and kidneys.

CLINICAL PICTURE

Risk factors. A history of living in areas with a high prevalence of strongyloides such as First Nations communities in Northern Australia and many tropical countries. This includes workers in First Nations communities, immigrants, returned travellers and service personnel. Infection can be lifelong without effective treatment.

Symptoms and signs. Infection in immunocompetent people is mostly asymptomatic but gastrointestinal, pulmonary and most importantly disseminated infection can occur.

- Gastrointestinal: subacute obstruction or segmental ileus (pseudo-intestinal obstruction) is seen especially with primary infection in young children, but diarrhoea and non-specific gastrointestinal symptoms can also occur with higher parasite burdens.
- Respiratory: dyspnoea and bronchospasm with migration of parasites through the lung, rarely gross haemoptysis.
- Skin: “larva currens”, lesions over lower back and buttocks, recurrent urticaria, resulting from autoinfection with larval migration.
- Neurological: gram-negative meningitis can occur especially with disseminated infection, secondary to migrating larvae causing gram-negative bacteraemia
- Systemic: gram-negative bacteraemia and sepsis secondary to disseminated strongyloidiasis.

There are three important clinical patterns of severe infection with *S. stercoralis* in Northern Australian First Nations communities:

1. Acute gastrointestinal infection in children, often with diarrhoea, hypokalaemia and wasting. Pseudo-intestinal obstruction can occur.

2. Disseminated strongyloidiasis can present initially as gram-negative meningitis/septicaemia, as the parasite facilitates penetration of gut bacteria into the circulation. There have been several deaths in Central Australia from sepsis associated with *S. stercoralis* infection, some with HTLV-1 comorbidity.

3. Disseminated strongyloidiasis is nearly always associated with immunosuppression. Historically there were several deaths in the Top End in patients on high dose immunosuppressive therapy but none in the last decade since the introduction of the Prevention of opportunistic infections in immunosuppressed patients in the tropical Top End of the Northern Territory.

Investigations. Faecal testing in chronic strongyloidiasis ideally requires three samples due to intermittent shedding of low numbers of larvae. Agar plate is the most sensitive culture technique but requires fresh faeces with live larvae and not all labs can perform this. *S. stercoralis* larvae die at low and high temperatures, keep sample in air-conditioned room. Fresh faeces may demonstrate larvae of *S. stercoralis* on microscopy. However, if faeces are examined a few days later, any hookworm eggs that also may be present may have hatched into larvae. Larvae should therefore be accurately identified, especially if specimens have been sent from a remote site. PCR testing of faeces is now available.

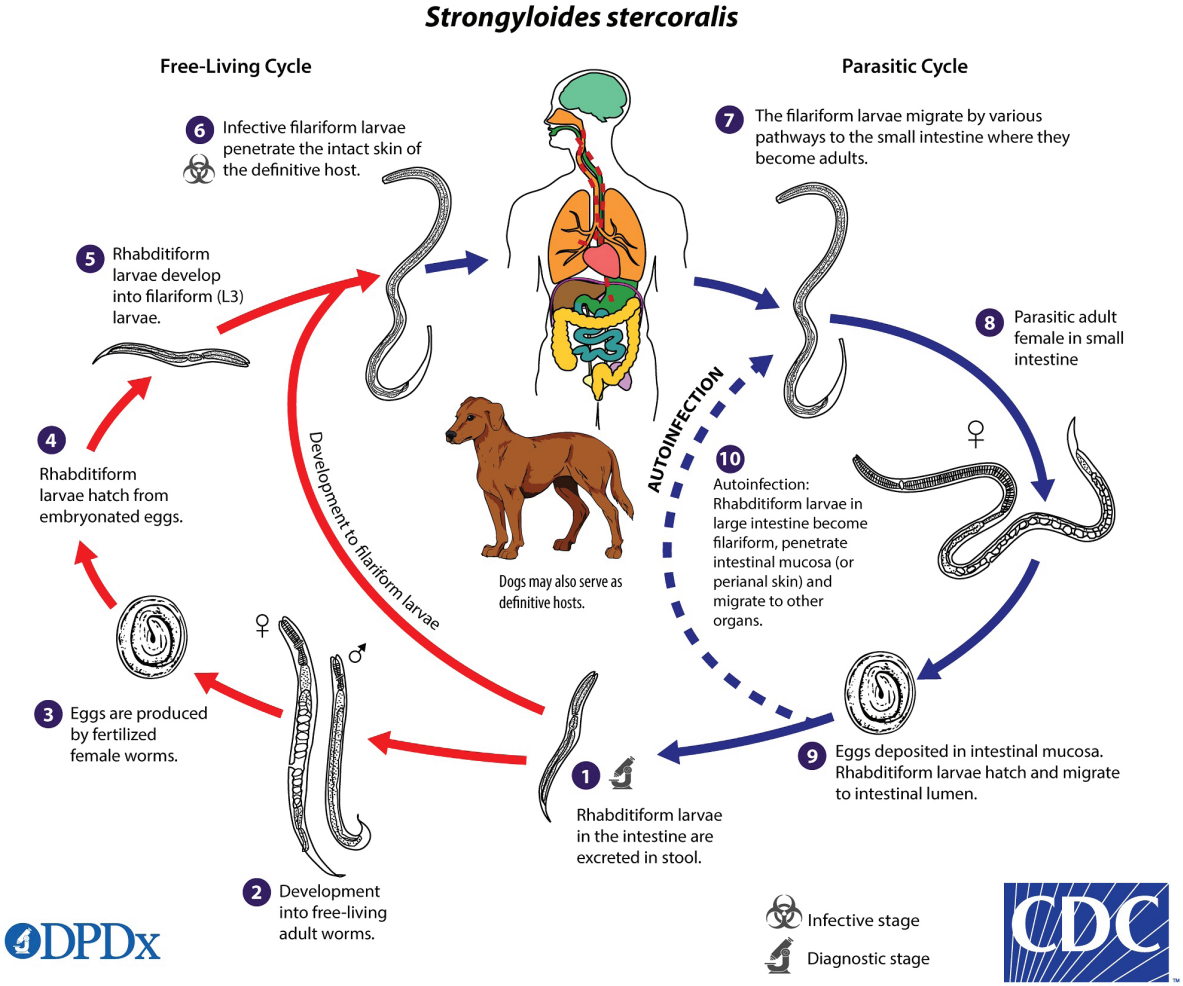


Figure 133: The life cycle of S. stercoralis.
Source: https://www.cdc.gov/dpdx/strongyloidiasis/index.html

Strongyloidiasis

Eosinophilia is present in 10–50% of cases. Strongyloides serology is used in diagnosing chronic strongyloidiasis, particularly in adults, and for individual follow-up as titres have been shown to usually decrease after 6 months with successful treatment. Serology results in an endemic area need to be interpreted in the light of the clinical picture as false negatives can occur in acute strongyloidiasis (with a window period for seroconversion) and disseminated strongyloidiasis (when individuals are no longer able to mount an immune response) as well as false positives from cross-reaction.

In disseminated disease, sputum wet mount examination is useful in addition to stool examination for parasites. Strongyloidiasis may also cause an abnormal chest X-ray.

DIFFERENTIAL DIAGNOSIS

The differentials of eosinophilia include atopy, drug reaction other soil-based helminths and scabies, as documented in the Asymptomatic Eosinophilia section (Page 223).

The differentials related to the often insidious symptoms of chronic strongyloidiasis such as gastrointestinal upset are broad but include other endemic soil-based helminths such as whipworm and hookworm.

PRINCIPLES OF MANAGEMENT

Treatment. Ivermectin is the drug of choice for non-pregnant people. Second line and significantly less effective is albendazole. Ivermectin is given with full fat milk as it increases absorption two-fold. The dosing schedule is dependent upon the clinical scenario.

The most important principle is to prevent hyperinfection / disseminated disease in immunosuppressed people. In the case of planned corticosteroid or immunosuppression, patients should have urgent stool sample x 3 and strongyloides serology and if positive, treated, before immunosuppression begins. Treatment includes 2 doses of ivermectin (200 microgram/kg PO rounded up to closest 3mg tablet) 7-10 days apart. Refer to the current Therapeutic Guidelines: Antibiotic for Strongyloidiasis and *Strongyloides stercoralis*. It may be prudent in this scenario to follow up with household contacts and treat as required as well as assess for immunosuppressed contacts.

Regardless of initial testing, anyone from an endemic area who is deemed immunosuppressed should be offered empirical 3 monthly ivermectin for the duration of immunosuppression to prevent disseminated disease. If immunosuppressive therapy has already commenced the number of doses is increased as per referenced protocols.

If a patient presents as asymptomatic but serology positive, then they should be assessed for underlying conditions and the serology titres interpreted prior to treatment.

In the setting of an asymptomatic eosinophilia, with no established other cause, updated protocols recommend empiric treatment with albendazole-ivermectin combination without strongyloides serology. The rational being the treatment of hookworm, whipworm and strongyloides at one time.

Prevention. Infection may be transmitted by direct contact with faeces or soil contaminated by faeces from an infected person. There is emerging evidence of the role of dogs and any control program may require a One Health approach. Prevention therefore includes community education about disease transmission, avoiding skin contact with contaminated soil and improved sanitation.

> Strongyloidiasis (extraintestinal) is a notifiable condition to be reported by LABORATORIES in the Northern Territory. Cases are reported to the local Centre for Disease Control/Public Health Unit.

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call infectious disease physician or paediatrician or local CDC/PHU.

MANAGEMENT GUIDELINES

Northern Territory		
Remote Primary Health Care Manuals (RPHCM)	CARPA Standard Treatment Manual — Worms	Available online

RESOURCES

Strongyloides Australia	Strongyloides Information Resource	Available online
ARDS Aboriginal Corporation	Strongyloides Information for Health Professionals	Available online
NT Health	Fact sheet - Strongyloidasis	Available online

FURTHER READING

Beknazarova M, Whiley H, Traub R, Ross K. Opportunistic Mapping of Strongyloides stercoralis and Hookworm in Dogs in Remote Australian Communities. Pathogens. 2020;9(5):398.

Hansen M, Bowden E, Currie BJ, et al. Strongyloides stercoralis seropositivity is not associated with increased symptoms in a remote Aboriginal community. Intern Med J. 2021;51(8):1286-1291.

Hays R, Esterman A, McDermott R. Control of chronic strongyloides stercoralis infection in an endemic community may be possible by pharmacological means alone: Results of a three-year cohort study. PLoS Neglected Tropical Diseases. 2017;11(7):e0005825.

Kearns TM, Currie BJ, Cheng AC, et al. Strongyloides seroprevalence before and after an ivermectin mass drug administration in a remote Australian Aboriginal community. PLoS Negl. Trop. Dis. 2017;11:e0005607.

Miller A, Smith ML, Judd JA, Speare R. Strongyloides stercoralis: systematic review of barriers to controlling strongyloidiasis for Australian Indigenous communities. PLoS Neglected Tropical Diseases. 2014;8(9):e3141.

Page W, Judd JA, Bradbury RS. The unique life cycle of strongyloides stercoralis and implications for public health action. Tropical Medicine and Infectious Disease. 2018;3(2).

Page WA, Judd JA, MacLaren DJ, Buettner P. Integrating testing for chronic strongyloidiasis within the Indigenous adult preventive health assessment system in endemic communities in the Northern Territory, Australia: An intervention study. PLoS Negl Trop Dis. 2020;14(5):e0008232.

Page W, Speare R. Chronic strongyloidiasis - Don't look and you won't find. Australian Family Physician. 2016;45(1):40-44.

Trichuriasis (whipworm)

Whipworm infection occurs globally and may cause anaemia, Trichuris dysentery syndrome, and growth retardation.

TRICHURIASIS IN NORTHERN AUSTRALIA

Trichuriasis or whipworm is a human intestinal infection caused by the helminth *Trichuris trichiura*. Along with other intestinal parasites it is common in many remote First Nations communities, particularly in the Top End of Northern Territory and Queensland. It has a global prevalence of 465 million people with most cases in the tropics and areas with poor sanitation. It was identified in 80% of stool samples examined from one community in East Arnhem Land. Infection may contribute to anaemia, dysentery and growth retardation. Compared to other soil transmitted helminths regional prevalence has remained stubbornly high in the face of deworming programs.

AETIOLOGY AND PATHOGENESIS

Trichuris trichiura has a faecal-soil-oral transmission cycle. The adult worm is 30–50mm in length and has a characteristic whip-like shape. Upon ingestion, eggs pass to the colon where, upon signalling from gut bacteria, they hatch. The larvae bury into the epithelium forming an intracellular syncytium. As they mature the posterior portion emerges from the mucosa of the colon and caecum and lays 3000–7000 eggs per day. The eggs are passed with the faeces and incubate for at least three weeks in the soil before they become infective. The cycle from ingestion to egg-laying takes about three months. Unlike hookworm the adults do not appear to ingest blood from the host, and anaemia is likely due to inflammation and damaged colonic capillary loss.

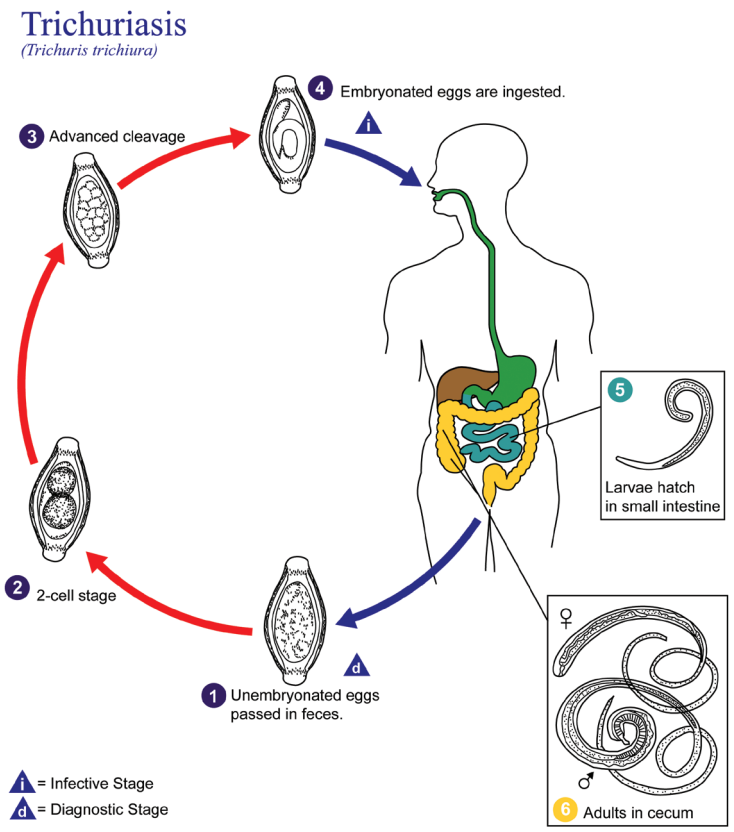


Figure 134: The life cycle of T. trichiura
Note: Eggs hatch in the colon, not the small intestine.
Source: https://www.cdc.gov/dpdx/trichuriasis/index.html

CLINICAL PICTURE

Risk factors. Living in poor sanitary conditions in prevalent areas.

Symptoms and signs. Most whipworm infections are asymptomatic. Abdominal pain, anorexia, bloody or mucoid diarrhoea, and in extreme cases tenesmus may lead to rectal prolapse. Moderately heavy worm loads may contribute to growth retardation and anaemia. Infected patients lose approximately 0.005mL blood per worm per day.

Investigations. A stool sample should be sent to the lab requesting microscopy for OC&P (ova, cysts and parasites). Faecal microscopy may demonstrate characteristic 50 by 20µm lemon-shaped eggs. A full blood count will often show eosinophilia. Proctoscopy may reveal adult worms, 3–5cm in length.



Figure 135: Egg of T. trichiura as seen in an unstained wet mount
Source: https://www.cdc.gov/dpdx/trichuriasis/index.html

PRINCIPLES OF MANAGEMENT

Albendazole should be taken on an empty stomach.

This has been shown to reduce worm burden but is often not curative, and reinfection in endemic areas is common. Therefore, a repeat stool test for ova and parasites should be done 6 months after treatment. There is increasing evidence that combination ivermectin–albendazole is superior to albendazole monotherapy, and this is now recommended when asymptomatic eosinophilia is incidentally identified on blood film. Regular anthelminthic empirical treatment is also part of the child health programs in the Top End.

Prevention. Community health education, improved hygiene, particularly washing hands and food handling practices, and improved infrastructure for sanitation.

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call physician or paediatrician or local CDC/PHU.

MANAGEMENT GUIDELINES

Northern Territory		
Remote Primary Health Care Manuals (RPHCM)	CARPA Standard Treatment Manual — Worms	Available online

FURTHER READING

Holt DC, Shield J, Harris TM, et al. Soil-Transmitted Helminths in Children in a Remote Aboriginal Community in the Northern Territory: Hookworm is Rare but Strongyloides stercoralis and Trichuris trichiura Persist. Trop Med Infect Dis. 2017;2(4):51.

Hürlimann E, Keller L, Patel C, et al. Efficacy and safety of co-administered ivermectin and albendazole in school-aged children and adults infected with Trichuris trichiura in Côte d'Ivoire, Laos, and Pemba Island, Tanzania: a double-blind, parallel-group, phase 3, randomised controlled trial. Lancet Infect Dis. 2022;22(1):123-135.

Le B, Monteiro MAA, Amaral S, et al. The impact of ivermectin, diethylcarbamazine citrate, and albendazole mass drug administration on the prevalence of scabies and soil-transmitted helminths in school-aged children in three municipalities in Timor-Leste: a before-after assessment. Lancet Glob Health. 2023;11(6):e924-e932.

SECTION 4 — SEXUALLY TRANSMITTED INFECTIONS

An overview of sexually transmitted infections (STIs)

Sexually transmitted infections are frequently treated according to their presenting syndrome while waiting for laboratory results.

STIs IN NORTHERN AUSTRALIA

Remote First Nations communities in Northern Australia have disproportionately high notification rates of STIs. Northern Territory data demonstrate a higher prevalence in both First Nations and non-Indigenous populations compared with other parts of Australia.

Among First Nations groups the highest notification rates occur in people aged 15– 34 years. Notification rates are crude measures that are affected by community testing patterns, screening programs, laboratory testing and reporting practices of practitioners. They are likely to underestimate the true prevalence of STIs.

CLINICAL PICTURE

Risk factors. The reasons for high STI rates include a young and highly mobile population, high rates of substance misuse (alcohol, marijuana and other recreational drugs, petrol), barriers to access and delivery of health services including high level of social disadvantage, variable health literacy and lack of access to resources in people's first language.

Clinical manifestations. STIs are a significant clinical and public health problem globally. Acute infections may cause unpleasant and distressing symptoms such as discharge, pain or ulceration. More importantly, STIs have serious potential long-term sequelae, including chronic pelvic pain, infertility, tubal pregnancy, adverse pregnancy outcomes and psychological distress. Mother-to-child transmission of STIs can result in stillbirth, neonatal death, low-birth weight and prematurity, sepsis, neonatal conjunctivitis and congenital deformities. The presence of STIs like herpes, gonorrhoea and syphilis can increase the risk of acquisition of HIV.

Sexually transmitted infections are frequently asymptomatic. As a result, only a minority of patients with STIs may present for treatment. This proportion may be further reduced by unfamiliarity of the significance of symptoms, lack of access to services, fear of unpleasant or embarrassing questions and tests, and the stigma attached to a diagnosis of an STI. This is often compounded in the remote First Nations community setting by cultural and language barriers.

In addition, pelvic inflammatory disease (PID) is frequently subclinical, or presents with mild symptoms which are often attributed to other causes. Pyuria (leucocytes) on urinalysis, in the absence of nitrites, could be due to an STI or PID and requires appropriate further history, examination and testing for STIs. A high degree of clinical suspicion for PID should be maintained for women presenting with lower abdominal pain and/ or dysuria, and a diagnosis of urinary tract infection should be made with caution.

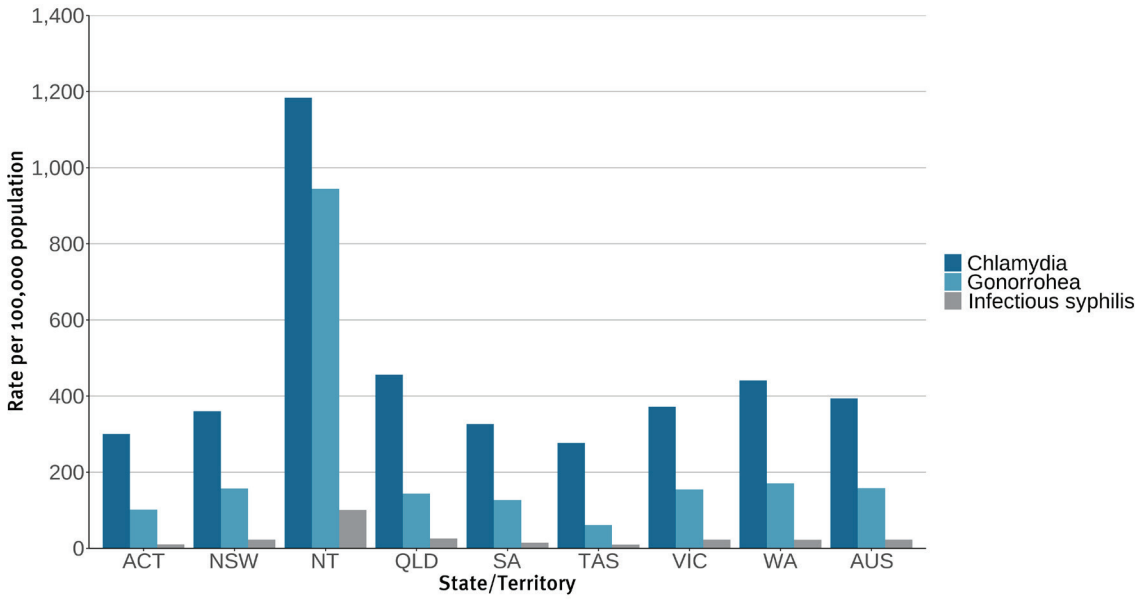


Figure 136: Rates of STI (chlamydia, gonorrhoea and syphilis) by state and territory, 2023-2024 combined
Source: National data from National Notifiable Diseases Surveillance System (NNDSS)

Sexually transmitted infections in children may be transmitted by sexual abuse, perinatally, or rarely by accidental contact. It is necessary for clinicians to be familiar with jurisdictional mandatory reporting requirements and report to Territory families/ appropriate child protection authority if there are reasonable grounds that a child is harmed or exploited.

PRINCIPLES OF MANAGEMENT

Syndromic approach

The diagnosis of the aetiology of STIs on clinical features alone is difficult, even for experienced clinicians. This may be due to similarity of clinical presentation, co-infection with more than one agent and atypical presentations due to self-treatment or secondary infection.

Inaccuracy of clinical STI diagnosis has led to an approach called syndromic management. This involves the identification and treatment of a syndrome, or a set of symptoms and signs associated with a limited number of aetiologies. Treatment at the first visit in the absence of a microbiological diagnosis, results in presumptive cure and a reduction in further transmission and complications of untreated infection. The syndromic approach is considerably less costly with less constraints on time, resources and access to treatment.

Guidelines in Northern Australia use the syndromic approach in STI management, for example, in urethritis, cervicitis and genital ulcers. This strategy is supported by laboratory investigations in almost all cases.

POPULATION APPROACH

Traditionally, health services waited for symptomatic people to present to the clinic for STI treatment. With high rates of asymptomatic infections and significant barriers to service utilisation, this approach misses a large proportion of people in the community with an STI. Effective STI control needs a population-based approach that includes education, the provision of condoms, opportunistic and community screening, specific staff training, and increased accessibility of health services.

CONTACT TRACING

Contact tracing is an essential public health strategy in STI management. But its effectiveness may be limited, particularly in the following circumstances:

- High prevalence of STIs.
- A highly mobile population.
- Community concerns about confidentiality.
- Patient reluctance to disclose sexual activity and partners.
- Large numbers of contacts.
- Anonymous contacts.

Contact tracing is often viewed as resource and time intensive. Nonetheless, it continues to be an integral part of STI control activities in Northern Australia and needs to be done as an important component of comprehensive care. Germ theory (of disease) is a complex idea that is not well understood in some communities. Therefore, it is important that education is provided to the person with the STI and their contacts to help explain why contact tracing is necessary. The local CDC sexual health unit can provide advice/ give direction in contact tracing.

> Chlamydia, gonorrhoea, syphilis, trichomonas, donovanosis and lymphogranuloma venereum (LGV) are LABORATORY notifiable in the Northern Territory.
>
> Mpox, Gonococcal conjunctivitis, chancroid, HIV/AIDS and donovanosis, are notifiable by CLINICIANS in the Northern Territory.
>
> All above cases are reported to the local Centre for Disease Control/Public Health Unit.

An overview of sexually transmitted infections (STIs)

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the local sexual health unit and CDC/PHU.

MANAGEMENT GUIDELINES

International		
World Health Organization	Guidelines for the management of symptomatic sexually transmitted infections. 2021	Available online
National		
The Australasia Society for HIV, Viral Hepatitis, and Sexual Health Medicine (ASHM)	■ Australasian Contact Tracing Guidelines 2022 ■ Australian STI Guidelines for use in primary care	Available online
Northern Territory		
NT Centre for Disease Control (CDC)	■ NT Guidelines for the Management of Sexually Transmitted Infections in the Primary Health Care setting ■ Public health and notifiable diseases	Available online

RESOURCES

NT Primary Health Network (NT PHN)	HealthPathways – Sexual Health Check	Available online (CLICK HERE to request access)
Remote Primary Health Care Manuals (RPHCM)	■ CARPA Standard Treatment Manual — Sexual Health/STI checks for men ■ Women's Business Manual — Sexual Health/STI checks for women	Available online

FURTHER READING

Bell S, Aggleton P, Ward J, et al. Young Aboriginal people's engagement with STI testing in the Northern Territory, Australia. BMC Public Health. 2020;20(1):459.

Lafferty L, Smith K, Causer L, et al; TTANGO2 Collaboration. Scaling up sexually transmissible infections point-of-care testing in remote Aboriginal and Torres Strait Islander communities: healthcare workers' perceptions of the barriers and facilitators. Implement Sci Commun. 2021;2(1):127.

Middleton M, McDonald A. Sexually transmissible infections among young people in Australia: an overview. HIV Australia. 2013;11(1):9-10.

Rahman T, Kong FYS, Williams R, et al. Increasing awareness of sexually transmitted infections (STI) testing and addressing stigma may improve STI testing in Aboriginal and Torres Strait Islander youth: Evidence from the Next Generation Youth Wellbeing Study. Aust N Z J Public Health. 2024;48(6):100203.

Ward J, Guy R, Huang RL, et al. Rapid point-of-care tests for HIV and sexually transmissible infection control in remote Australia: can they improve Aboriginal people's and Torres Strait Islanders' health. Sexual Health. 2012;9(2):109-112.

Gonorrhoea

Neisseria gonorrhoea causes gonorrhoea which can be asymptomatic or have acute symptoms and cause long-term complications.

GONORRHOEA IN NORTHERN AUSTRALIA

Gonorrhoeal genital tract infection is common in Northern Australian remote First Nations communities. Other major risk groups in Australia are men who have sex with men, and people who acquire STIs overseas. Gonorrhoea is more common in the Northern Territory than other states. People aged 15–34 have the highest rates of infection.

AETIOLOGY AND PATHOGENESIS

Neisseria gonorrhoeae is the gram-negative diplococcus that causes gonorrhoea. Mucous membranes lined by columnar or cuboidal cells are susceptible to gonococcal infection, including the urethra, endocervix, fallopian tube, and rectum. Genital gonorrhoea is exclusively a sexually transmitted infection.

CLINICAL PICTURE

Risk factors. See *An overview of sexually transmitted infections* (page 135).

Symptoms and signs. Clinical features of *N. gonorrhoeae* infection range from none, local genital symptoms, and systemic illness.

- **Penile Urethral infection:** Symptoms occur in over 90% individuals. A mucopurulent urethral discharge and/ or dysuria, after an incubation period of about 2–5 days. Local complications include epididymo-orchitis and prostatitis. Coinfection with chlamydia is common.
- **Female urethral infection:** Females may present with dysuria without urinary frequency**.**
- **Endocervical infection** is symptomatic in only about half of cases, manifesting as altered or increased vaginal discharge, dysuria and/ or post coital bleeding. A 'friable' cervix with contact bleeding, frank mucopurulent discharge or an oedematous cervix supports the diagnosis of cervicitis.
- **Infections in other sites.** Pharyngeal infection is common in both sexes and is usually asymptomatic. Most rectal infections are asymptomatic or subclinical. If present, symptoms include perianal/ anal irritation, pain or discomfort. Some may have tenesmus, discharge, painful defecation, disturbed bowel function and bleeding.

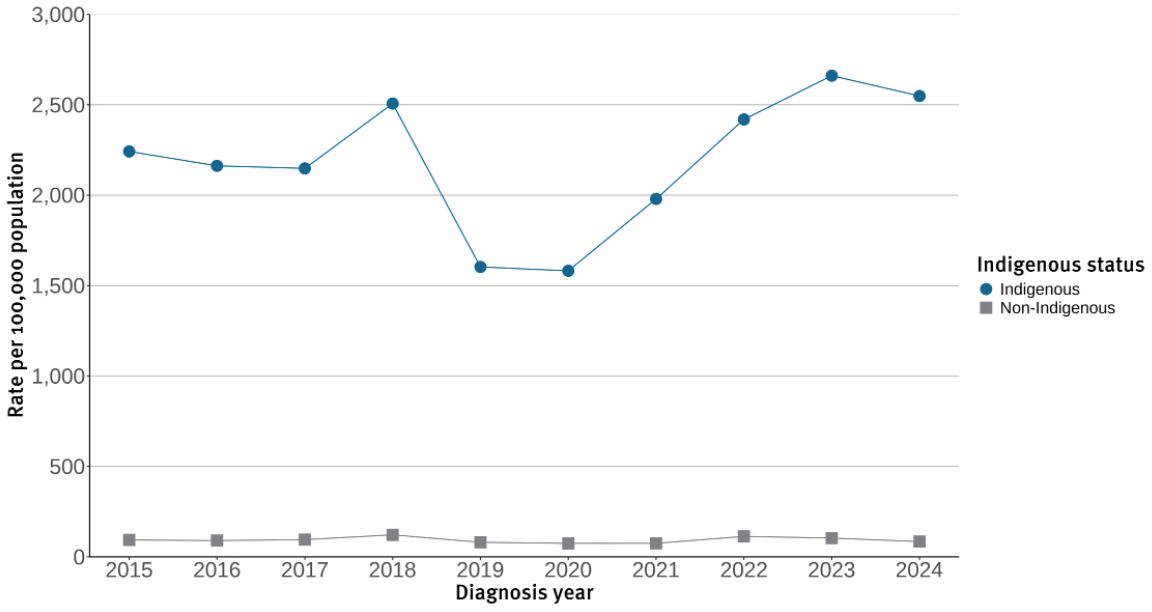


Figure 137: Rates of gonorrhoea in the Northern Territory by Indigenous status and year of diagnosis, 2015-2024
Source: Northern Territory Notifiable Diseases System (NTNDS)

Complicated infections.

- **Pelvic Inflammatory Disease (PID)**: Transluminal/ascending infection from urethra or endocervix can lead to PID. Gonococcal PID may present with any combination of pelvic pain, fever, deep dyspareunia or menstrual irregularities, but can also be subclinical. Cervical excitation and adnexal tenderness are typical features on clinical examination that support a diagnosis of PID. The possible sequelae of gonococcal PID are serious and include tubo-ovarian abscesses with or without rupture, tubal infertility and ectopic pregnancy.

  Complicated gonorrhoea may also be considered in patients presenting with right upper quadrant abdominal pain as part of Fitz-Hugh-Curtis syndrome (perihepatitis secondary to PID). Curiously, symptoms and signs of PID are often not present in such patients and upper abdominal ultrasound is usually unremarkable, but diagnostic sampling is usually the same as for PID.

- **Gonococcal conjunctivitis.** Although uncommon, gonococcal conjunctivitis should always be considered in any neonate with a discharging or red eye. It should also be considered in children or sexually active adults presenting with intense conjunctival inflammation, copious discharge or a persistent conjunctivitis. It requires systemic treatment and may occasionally occur as part of an epidemic. It is important to diagnose this correctly because untreated infection may lead to corneal ulceration, perforation and blindness. A sporadic presentation in a child should raise the suspicion of sexual abuse, although nonsexual transmission is possible.

- **Disseminated gonococcal infection (DGI)**. Haematogenous dissemination can lead to DGI in 0.5–3% of gonorrhoea cases, and manifests most commonly as the arthritis-dermatitis syndrome, with joint swelling, tenosynovitis and skin lesions. It is an important differential in sexually active adolescents or adults presenting with oligo or polyarthritis, a situation where acute rheumatic fever, systemic lupus erythematosus, and endocarditis are the other important differential diagnoses.



Figure 138: Disseminated gonococcal infection — septic arthritis

Source: Bart Currie — Menzies School of Health Research



Figure 139: Disseminated gonococcal infection — tenosynovitis, arthritis, skin lesion

Source: Bart Currie — Menzies School of Health Research



Figure 140: Disseminated gonococcal infection — ankle joint arthritis and skin lesions

Source: Bart Currie — Menzies School of Health Research

Gonorrhoea

DIFFERENTIAL DIAGNOSIS

The *Case study — Urethral discharge* (page 161) discusses the differential diagnosis of penile urethritis. CARPA guidelines recommend empiric treatment for chlamydia and gonorrhoea in people from remote First Nations communities, aged 15–35, who present with penile urethral discharge or dysuria.

The presence of vaginal discharge could represent cervicitis, upper genital tract infection (pelvic inflammatory disease), or vaginitis from candidiasis, trichomoniasis or bacterial vaginosis, as well as non-infective causes. Similarly, dysuria or positive leukocytes on urinalysis could be a manifestation of an STI (with or without pelvic inflammatory disease), vaginitis or a urinary tract infection.

Gonorrhoea should be considered in monoarthritis and tested for by culture and nucleic acid amplification test (NAAT) of joint aspirates. Endocarditis and meningitis can occur, although rare.

PRINCIPLES OF MANAGEMENT

Gonorrhoea in the setting of the STI syndromes is discussed further in the urethral discharge (page 161) and lower abdominal pain (page 165) case studies.

Investigations. It is important to obtain culture samples from affected sites before treating someone for gonorrhoea. Rectal and throat swabs for gonococcal NAAT and culture are routine in the sexual health work up of men who have sex with men. Otherwise, testing is recommended in the settings of STI syndromes/clinical illnesses suggestive of gonorrhoea, contact tracing, sterile pyuria with leukocytes in the urine, those who are in 15–34 year age group and those who are at increased risk of STIs (e.g. new sexual partner, living or travelling to areas of higher prevalence in Australia or overseas, known exposure to any STI, or history of an STI within the past 12 months. However, NAAT is unable to provide sensitivities and returns occasional false positives, and in certain sites (throat and rectal), validity of NAAT is sufficiently questioned as to always warrant an additional culture test. Culture is less sensitive but is important due to its increased specificity and ability to test for antibiotic resistance.

Knowing antibiotic sensitivities is important as antibiotic-resistant *Neisseria gonorrhoeae* are increasing in Australia. Remote Northern Territory has not been considered as a penicillin sensitive area since 2023 as penicillinase producing *N. gonorrhoeae* (PPNG) have been detected.

Testing guidelines vary across Australia, so it is important that clinicians are familiar with local protocols.

Importantly for clinicians, culture requires a swab in transport media and transport at room temperature (as long as less than 40°C), whereas NAAT requires a dry swab (due to inhibitors in the transport media reducing the test's sensitivity), and/or urine sample and usually favours transport at fridge temperatures. Delays in transport reduce the yield of culture.

Screening. The incidence is sufficiently high in the Northern Territory that screening is recommended in men who have sex with men, people from remote First Nations communities, and the mainstream population of the Northern Territory (including antenates). The age range of screening in the latter two groups varies with the local guideline and partner/STI history.

The CARPA Women's Business Manual recommends opportunistic STI checks at least twice a year in First Nations communities of the Northern Territory. All guidelines in Australia recommend NAAT as the main screening test for gonorrhoea due to the increased sensitivity and reasonable specificity compared to culture, and the ability to test multiple sites including urine, genital secretions, throat, rectum, and joints.

Management. As a general rule all patients with presentations with presumptive or confirmed gonococcal infections require parenteral ceftriaxone and oral azithromycin as part of the gonorrhoea treatment.

In some remote WA locations with penicillin sensitive gonorrhoea, if the partner is also from a penicillin sensitive zone, oral amoxicillin/probenecid and azithromycin is the recommended treatment strategy. If the partner is from a penicillin resistant zone or of unknown origin, ceftriaxone and azithromycin is the recommended treatment.

In either scenario two antibiotics of different mechanisms of action — a beta lactam antibiotic and azithromycin — are used simultaneously. This aims to reduce the development of penicillin resistance and is encouraged by the World Health Organization.

The finding of a penicillin resistant isolate in a penicillin sensitive zone is a cause of concern and requires heightened contact tracing.

In the penicillin resistant areas of Northern Australia, culture and sensitivity is still important due to the emerging development internationally of increased

minimum inhibitory concentrations to ceftriaxone. The finding of a ceftriaxone resistant strain (rare in Australia) would be regarded as a public health emergency.

Contact tracing is recommended for up to two months prior to onset of symptoms.

Follow-up depends on the syndrome but will always include a three-month review which would include testing for reinfection.

Test of cure by NAAT is recommended in Australian sexual health guidelines for each site of infection (pharyngeal, anal or cervical), 2 weeks after treatment is completed, especially if no gonococcal culture swab was collected before treatment, or if the culture swab indicated antimicrobial resistance, or if the patient was treated with a non-standard regimen.

> Gonorrhoea is a nationally notifiable condition by LABORATORIES.
>
> CLINICIANS are also required to notify gonococcal conjunctivitis in the Northern Territory. Cases are reported to the local Centre for Disease Control/ Public Health Unit.

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the sexual health clinic or the local CDC/PHU.

MANAGEMENT GUIDELINES

Northern Territory		
NT Centre for Disease Control (CDC)	■ NT Guidelines for the Management of Sexually Transmitted Infections in the Primary Health Care setting ■ Public health and notifiable diseases	Available online
Remote Primary Health Care Manuals (RPHCM)	■ CARPA Standard Treatment Manual — Sexual Health/Gonorrhoea ■ Women's Business Manual — Sexual Health/Gonorrhoea	Available online

FURTHER READING

See *An overview of sexually transmitted infections* (page 137)

Graham S, Chen W, Connor S, Gunathilake M. Men at risk of gonococcal urethritis: a case-control study in a Darwin sexual health clinic. BMC Infect Dis. 2019 Nov 21;19(1):991.

Guy RJ, Natoli L, Ward J, et al. A randomised trial of point-of-care tests for chlamydia and gonorrhoea infections in remote Aboriginal communities: Test, Treat ANd GO- the 'TTANGO' trial protocol. BMC Infectious Diseases. 2013;13:485.

Syphilis

Syphilis is a chronic systemic infection caused by the spirochaete *Treponema pallidum* sub species *pallidum*.

SYPHILIS IN NORTHERN AUSTRALIA

Syphilis has periods of active disease and long periods of latency. Mother to child transmission leading to congenital syphilis infection during pregnancy is an important cause of fetal and child morbidity and mortality.

Syphilis is mostly seen in remote First Nations communities, in men who have sex with men, or is acquired overseas. In 2011, an outbreak of early infectious syphilis swept across from the North Queensland area reaching the Northern Territory in 2013 and has eventually entered the Kimberley and South Australia. This outbreak has led to several cases of congenital syphilis and several infant deaths from congenital infection.

Across Australia, 95 cases of congenital syphilis were reported between 2016 and Q2 2024, including 52 reported in First Nations infants.

The devastating consequences of congenital infection and other sequelae make syphilis testing an important part of the sexual health screening of all Australians, even those at low risk. Maintain a high index of suspicion for possible infection with syphilis, in both asymptomatic and symptomatic patients, especially in high-risk groups.

AETIOLOGY AND PATHOGENESIS

Syphilis is predominantly a sexually transmitted disease, with transmission occurring through direct contact with the highly infectious lesions of primary and secondary syphilis in oral and ano-genital areas. Transmission may also be transplacental and blood borne. *T. pallidum* is very difficult to culture, and serology and direct detection from lesions through PCR are the mainstay of diagnosis.

The classical lesion of primary syphilis, the chancre, develops at the site of inoculation with the organism and occurs 10–90 days after infection. The organism disseminates to the draining lymph nodes, and then to distant organs and tissues. Involvement of the central nervous system, cardiovascular system, eyes, skin and bone manifest at the secondary and tertiary stages of syphilis. There is a long latency period between secondary and tertiary syphilis.

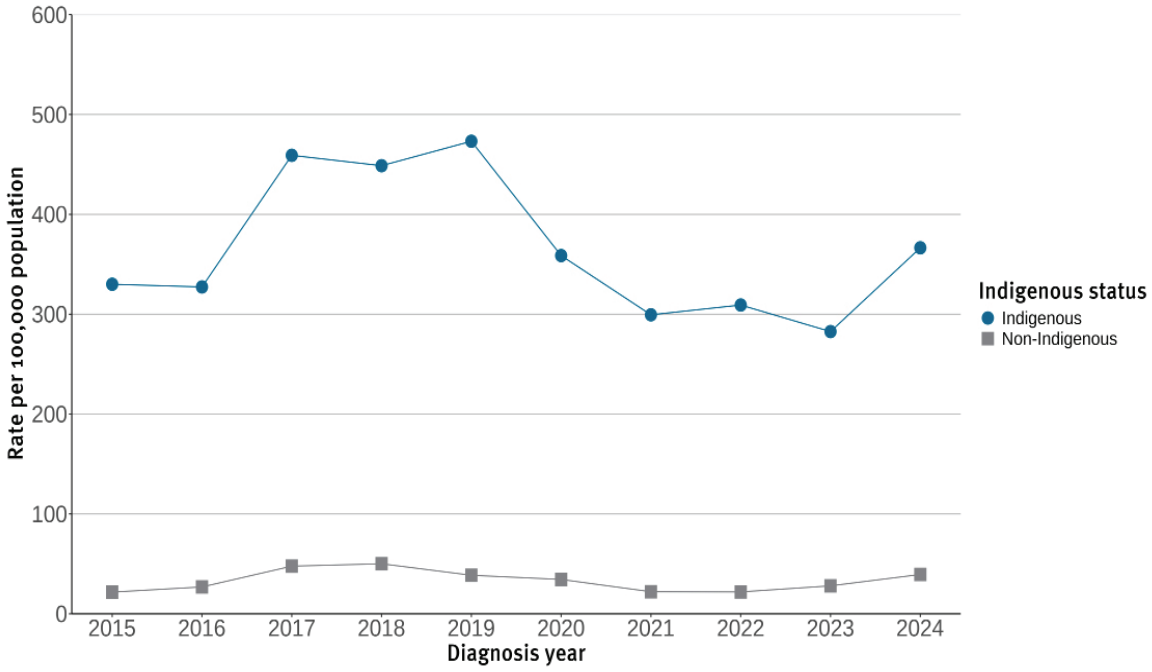


Figure 141: Rates of infectious syphilis in the Northern Territory by Indigenous status and year of diagnosis, 2015-2024
Source: Northern Territory Notifiable Diseases System (NTNDS)

Infection in pregnancy crosses into the foetus transplacentally and the spirochaetes disseminate to the fetal organs including the central nervous system, reticuloendothelial system, skin and mucous membranes and bones.

Intrauterine disease is affected by the stage of disease in the mother, the gestation of the pregnancy and the timeliness of treatment. Transmission of infection to the foetus is 70–100% in primary and secondary syphilis, 40% in early latent syphilis and 8–10% in late latent syphilis. The frequency of vertical transmission increases with gestational age whilst the severity of fetal infection decreases with gestational age. Completion of treatment 30 days prior to birth reduces the infection rate in the baby to 1–2%.

CLINICAL PICTURE

Risk factors. See *An overview of sexually transmitted infections* (page 135).

Syphilis can be classified into early infectious syphilis, late latent syphilis, syphilis of unknown duration, and the consequences of intrauterine infection.

Early infectious syphilis refers to **primary**, **secondary** and **early latent syphilis** which are cases of two or less years duration. Early syphilis is easily sexually transmitted and has a high propensity to cause mother to child transmission. In the Northern Territory the highest rates of syphilis infections occur in the 15–30-year age group with the ensuing risk of transmitting the infection to the foetus during pregnancy.

This makes diagnosis and treatment urgent and an important way of controlling disease transmission and reducing the incidence of the infection. Early syphilis is usually effectively treated with a single 2.4 million unit dose (2 x 1.2 million) of benzathine benzylpenicillin.

Late syphilis refers to late **latent syphilis** and **tertiary syphilis** where the infection has lasted more than two years. Patients are not usually infectious to sexual partners at this stage although 10% of affected antenates will transmit infection to the foetus. The main purpose of diagnosis and treatment at this stage is to prevent the morbidity of tertiary syphilis and prevent vertical transmission in pregnant women. In contrast to early syphilis, where one dose of benzathine benzylpenicillin is usually adequate, treatment usually involves three doses seven days apart, or in the case of neurosyphilis, two weeks of IV benzylpenicillin therapy.

Symptoms and signs.

- **Primary syphilis.** The incubation period for primary syphilis is 10–90 days. The classical presentation is a single, painless, indurated papule that ulcerates into the typical, 1–2cm sized, firm-based chancre, with associated non-tender inguinal lymphadenopathy. However, atypical presentations are very common, including multiple and/or painful ulcers. The untreated chancre usually heals after a few weeks. Due to a lack of pain and spontaneous resolution, chancres are commonly subclinical and may not lead to clinical presentation, particularly in women.



Figure 142: Secondary syphilis
Source: Bart Currie — Menzies School of Health Research

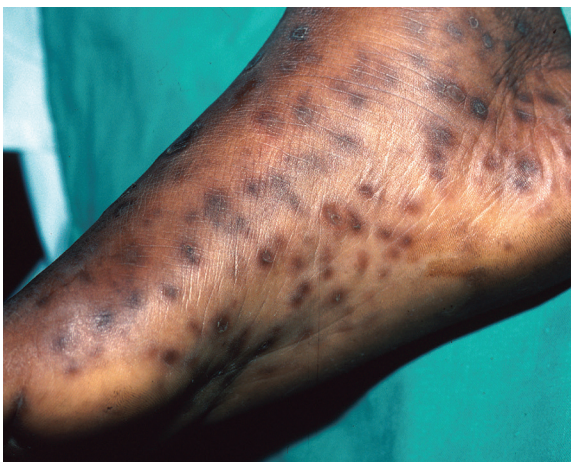


Figure 143: Secondary syphilis
Source: Bart Currie — Menzies School of Health Research

Syphilis

- **Secondary syphilis.** This is the systemic stage of syphilis infection, occurring at the same time as or up to six months after resolution of the primary chancre. It should be considered by clinicians in patients presenting with a non-specific constitutional illness, including symptoms of malaise, headache, sore throat, and fever. Generalised or sometimes localised rash, often involving palms and soles, highly infectious 'wart like' lesions found in warm moist anogenital area called condylomata lata, patchy alopecia, generalised lymphadenopathy and mucosal patches or ulcers (called snail track ulcers in the mouth) are particularly suggestive of the diagnosis and should be actively sought. Other features of secondary syphilis include altered LFTs (transaminitis) and meningitis.

  Secondary syphilis may last for weeks or months before resolution and will relapse in about one quarter of untreated patients over the subsequent few years.

  Patients with secondary syphilis are very infectious.

- **Latency.** The latent stage of syphilis represents ongoing but dormant infection with *Treponema pallidum*. There is serological evidence of infection but no clinical features. This can be divided into early latent infection (syphilis infection present two or less years) and late latent syphilis (syphilis infection present more than 2 years), with major implications to treatment, urgency of treatment, public health actions and contact tracing (early latent syphilis is considered infectious). A third entity is latent syphilis of unknown duration, which is where it is unknown whether the infection has been present more than two years.

  A key point in latent syphilis is that diagnosis requires a clinical assessment looking for absence of symptoms and signs of syphilis in the presence of positive serology results.

- **Tertiary syphilis.** Latent syphilis may last for decades, with about two thirds of untreated patients never manifesting any further signs of disease. The remaining third demonstrate a variety of clinical features, collectively termed tertiary syphilis. The three classical manifestations of tertiary syphilis are:
  - > Cardiovascular: aneurysm of the ascending aorta, aortic regurgitation and coronary artery ostia narrowing.
  - > Neurosyphilis: general paresis, sensorineural deafness without clear alternate cause, tabes dorsalis, meningovascular syphilis and a variety of ocular conditions.
  - > Gummas: granulomatous destructive tumours of skin, bone and viscera.

  Tertiary disease occurs years to decades after primary infection. A detailed history and examination and review of the patient's medical file is usually adequate to rule out these conditions when deciding whether someone has latent or tertiary syphilis.

- **Syphilis in pregnancy.** As the deaths of infants in the recent ongoing syphilis outbreak in First Nations communities in Northern Australia suggests, it is very important to prevent mother to child transmission of syphilis. Adverse clinical outcomes include miscarriage, stillbirth and neonatal death, non-immune hydrops fetalis, preterm delivery, intrauterine growth restriction and congenital syphilis.

  Early congenital syphilis is defined as onset prior to the age of two years. The features of early congenital syphilis are similar to secondary syphilis. Usually it presents 2–8 weeks after birth with failure to thrive, nasal snuffles (40% cases), skin rash, muco-cutaneous lesions, generalised lymphadenopathy, and sometimes osteochondritis. Central nervous system involvement may be symptomatic or asymptomatic. Asymptomatic central nervous system syphilis is diagnosed by abnormalities of the cerebrospinal fluid. Some present with sepsis.

  The onset of late congenital syphilis is defined as manifestations after the age of two but usually seen at or near puberty. Stigmata include sensorineural deafness, interstitial keratitis, Hutchison's teeth (Hutchison's triad), rhagades (cracks/fissures) around mouth, Clutton's joint, osteitis and chondritis (saddle nose, frontal bossing, and sabre tibia), intellectual impairment, hydrocephalus and perforated palate.

  Treatment of early maternal syphilis at least 30 days before delivery is the most important factor influencing the risk of congenital infection. 70–100% of infants born to untreated mothers will be infected compared to 1–2% of those born to women adequately treated during pregnancy. Treatment prior to pregnancy and monitoring for reinfection should reduce this further and help prevent the serious effects of infection of the foetus in the first trimester.

Screening and diagnosis. In low-risk populations, testing for syphilis is part of a routine antenatal screen, an annual sexual health screen, any STI check, for sexual or mother to child contact tracing of confirmed cases of syphilis and investigation of clinical illness suggestive of syphilis.

In high-risk populations, such as the First Nations population of Northern Australia or men having sex with men, the threshold for screening is lower, especially during outbreaks.

Screening for syphilis in antenates in remote First Nations communities of the Northern Territory is recommended at the first visit, 28 weeks, 36 weeks, birth and 6 weeks postpartum (post-partum allowing for the incubation period).

Given the higher occurrence of congenital syphilis, some other Australian states/territories recommend additional syphilis testing in third trimester and at birth in addition to the first screen earlier in pregnancy.

Familiarise yourself with your local screening recommendations as these vary over time.

A nucleic acid test (PCR) for *Treponema pallidum* is recommended for direct identification of syphilis in a suspected chancre, mucosal lesions or condylomata lata.

Serology is the major diagnostic test in the investigation of syphilis. Interpretation of syphilis serology can be difficult and often beyond the experience of the primary health care provider.

For example, a positive Syphilis antibody (CMIA/EIA) and *Treponema pallidum* particle agglutination assay (TPPA) with a negative Rapid Plasma Reagin (RPR) can be seen in early primary syphilis, early latent syphilis, late latent syphilis, latent syphilis of unknown duration, treated syphilis, tertiary syphilis and congenital syphilis. All tests can be negative during the first few days of primary syphilis. Proper interpretation relies on a clinical assessment of the likely stage of disease, sexual contact history, patient's prior treatment history, previous serology results and HIV status. An awareness of the incubation period and biological false positives also helps.

The staff of the syphilis registers in public health units are key people to seek help for previous testing/treatment details. They maintain a syphilis serology and treatment database of most patients in their jurisdictions and will provide advice on the correct interpretation of serology. Their advice and access to the database is encouraged, even for those who understand the serology. Register staff must be rung about every case, so they can update the register with new details.

PRINCIPLES OF MANAGEMENT

Penicillin remains the mainstay of treatment for syphilis. The length and type of adequate penicillin therapy depends on the stage of disease.

Usual recommendations are:

- For early syphilis benzathine benzylpenicillin IM single dose of 2.4 million units (2 x 1.2 million).
- For late latent syphilis or latent syphilis of unknown duration and for most non-neurological tertiary syphilis, benzathine benzylpenicillin IM dose of 2.4 million units (2 x 1.2 million) weekly for 3 weeks.
- For suspected neurosyphilis a neurologist/infectious disease physician/sexual health physician should be contacted. Hospital admission may be necessary as intravenous penicillin is required.

A few important points are worth remembering when giving treatment:

1. Baseline syphilis serology on the actual day of first treatment is recommended.
2. 2.4 million units of benzathine benzylpenicillin is two syringes not one (one syringe contains 1.2 million units).
3. All treatments must be notified to the local syphilis register.
4. Warn patients about possible Jarisch-Herxheimer reactions (up to 40% of early syphilis cases, usually presenting as a fever for three to four hours 6–12 hours after treatment given). The reaction is due to dying spirochaetes releasing endotoxin-like products in response to antibiotic treatment. Other symptoms include chills, rigor, hypotension, headache, tachycardia, hyperventilation, vasodilatation, muscle aches, exacerbation of skin lesions and anxiety.
5. Obtain pregnancy test in all women of childbearing years. A positive pregnancy test would require management (and sometimes preventative treatment) of the Jarisch-Herxheimer reaction and its potential propensity to cause uterine contractions, fetal distress, pre-term birth and still birth, monthly follow-up blood tests for treatment response, ultrasound for overt evidence of syphilis (if gestation more than 20 weeks) and particular follow-up, treatment and investigations of the neonate from birth onwards.

Syphilis

Contact tracing depends on the stage of disease, being 3 months plus duration of symptoms for primary syphilis, 6 months plus duration of symptoms for secondary syphilis, 12 months for early latent syphilis and long-term partners for late latent syphilis.

Test of cure is with repeat RPR testing at **3 months**, then at **6 months** and (if necessary) at **12 months** after completing treatment, all performed at the same laboratory to allow accurate assessment of a fourfold drop in the titre of the non-treponemal specific serology (i.e. RPR). (An exception to this particular follow-up would be patients with low titre such as RPRs 1:4 or less whose RPR is already low.)

As with all STIs, screen for other STIs and consider the potential significance of the HIV window period.

Advise no sexual contact for **7 days** after treatment, and no sex with partners from the last **3 months** (primary syphilis) and **6 months** (secondary syphilis) until the partners have been tested and treated if necessary.

Empirical therapy for syphilis is required in those with non-painful ulcers, clinical suspicion for secondary syphilis, sexual contacts of confirmed cases of early syphilis, and neonates at risk of having congenital syphilis.

The *Case study — Genital ulcer* (page 163) provides further discussion of the differential diagnosis, investigations and management of genital ulcers.

> Syphilis is a nationally notifiable condition to be reported by Australian LABORATORIES. Cases are reported to the local Centre for Disease Control/ Public Health Unit.

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the local syphilis register, sexual health clinic, and CDC/PHU.

MANAGEMENT GUIDELINES

Northern Territory		
NT Centre for Disease Control (CDC)	■ Congenital syphilis guidelines for the Northern Territory ■ NT Guidelines for the Management of Sexually Transmitted Infections in the Primary Health Care setting ■ Health Alert – Increasing Syphilis Outbreak Cases in the Northern Territory (2023) ■ Public health and notifiable diseases	Available online
Remote Primary Health Care Manuals (RPHCM)	■ CARPA Standard Treatment Manual — Sexual Health/Syphilis ■ Women's Business Manual — Sexual Health/Syphilis	Available online
NT Primary Health Network (NT PHN)	HealthPathways — Syphilis	Available online (CLICK HERE to request access)

FURTHER READING

See *An overview of sexually transmitted infections* (page 137)

Australian Centre for Disease Control. National syphilis surveillance quarterly report: Quarter 2: 1 April-30 June 2024.

Ramachandran PS, Baird RW, Markey P, et al. Neurosyphilis: Still prevalent and overlooked in an at risk population. PLoS One. 2020;15(10):e0238617.

Rode NBA, Ryder N, Su JY. An audit on the management and outcomes of infants at risk of congenital syphilis in the Top End of the Northern Territory, Australia, 2005-2013. Commun Dis Intell (2018). 2018;42:S2209-6051(18)00018-0.

Chlamydia

Chlamydia is caused by *Chlamydia trachomatis* bacteria that can cause long term complications, even when those infected have no symptoms.

CHLAMYDIA IN NORTHERN AUSTRALIA

Genital tract infection with chlamydia is common in Northern Australia and causes high rates of pelvic inflammatory disease (PID) and infertility in women. Notification rates for chlamydia are particularly high in the Northern Territory. The majority of this excess burden of disease is in First Nations people, with rates five times that of the national rate. The highest rates of infection are in 15–24-year-olds.

AETIOLOGY AND PATHOGENESIS

Chlamydia trachomatis is one of four species within the genus *Chlamydia*. Two other species are also pathogens of humans: *Chlamydia psittaci,* the cause of *psittacosis*, and *Chlamydia pneumoniae,* a common respiratory pathogen.

Specific *Chlamydia trachomatis* serovars are responsible for a variety of human diseases. Serovars A–C cause trachoma; L1–L3 cause lymphogranuloma venereum, an STI not endemic to Australia, except for some cases in men who have sex with men; and serovars D–K are responsible for genital tract chlamydial infections and inclusion conjunctivitis. Chlamydia are obligate intracellular organisms with a unique growth cycle and cannot be cultured on artificial media. Attachment and penetration of columnar epithelial cells of the urethra, rectum, cervix and fallopian tubes leads to a vigorous immune response, with local inflammation and tissue damage. Genital tract chlamydia is a sexually transmitted infection.

CLINICAL PICTURE

Risk factors. See *An overview of sexually transmitted infections* (page 135).

Symptoms and signs. The clinical manifestations of infection with *C. trachomatis* have close parallels with those from *Neisseria gonorrhoeae,* predominantly genital tract symptoms, and symptoms of pharyngitis or proctitis in both men and women, occasional systemic disease as well as asymptomatic infection. Asymptomatic chlamydial infection is common, with reported rates up to 75% in women and 30% in men, and as a result, clinicians should have a very high index of suspicion for diagnosis.

- **Men.** After a 7–21-day incubation period, *C. trachomatis* infection presents typically as dysuria and a scant to moderate white or clear urethral discharge. Examination is usually otherwise unremarkable. Rectal infection may occur in men having sex with men and is commonly asymptomatic. Epididymo-orchitis is a well-recognised local complication of *C. trachomatis* in men; the role of this infection in non-bacterial prostatitis is inconclusive.

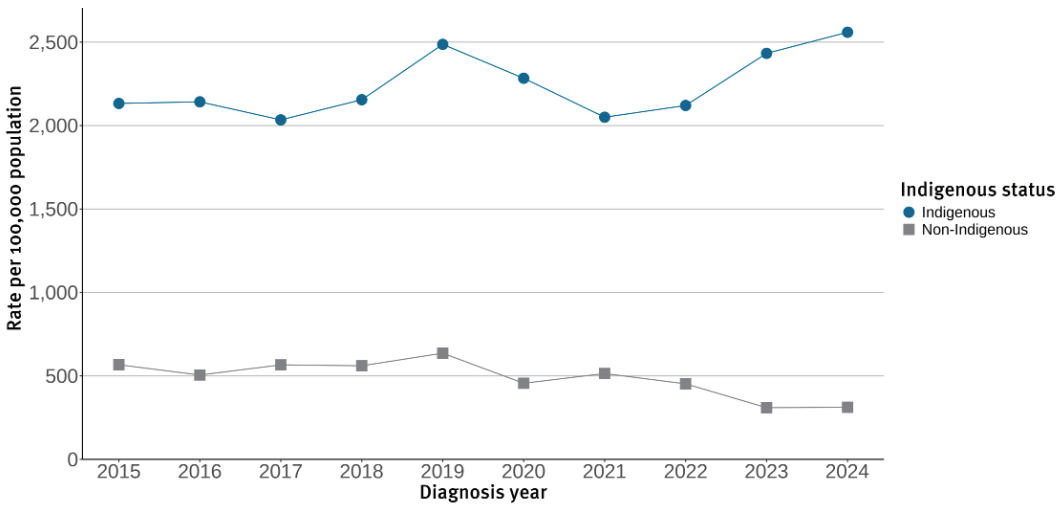


Figure 144: Rates of chlamydia in the Northern Territory by Indigenous status and year of diagnosis, 2015-2024

Source: Northern Territory Notifiable Diseases System (NTNDS)

- **Women.** *C. trachomatis* may infect the cervix and urethra, as well as the upper genital tract. Importantly, most cases are asymptomatic. In symptomatic women with cervicitis, *C. trachomatis* infection most commonly presents as mucopurulent vaginal discharge. Common findings on examination include mucopurulent cervicitis, a 'friable' cervix and/or hypertrophic ectopy. Urethritis is commonly associated with cervicitis and may present as dysuria and frequency (the 'urethral syndrome'). Bartholinitis is a recognised local complication of *C. trachomatis* infection.

  As with gonorrhoea, the most common and serious complications of *C. trachomatis* in women include pelvic inflammatory disease (PID), salpingitis, and endometritis. Chlamydial PID is frequently subclinical, though it may present with any combination of pelvic pain, fever, dyspareunia or menstrual irregularities. Cervical excitation and adnexal tenderness on clinical examination support a diagnosis of PID. The possible sequelae of PID include tubal infertility and ectopic pregnancy.

  Perihepatitis (Fitz-Hugh-Curtis syndrome) is an uncommon systemic complication of infection with *C. trachomatis* in women that presents with right upper quadrant pain but often little in the way of pelvic symptoms and signs and an unremarkable upper abdominal ultrasound. Cultures are nevertheless obtained in the same way as for PID.

- **General.** *C. trachomatis* is an important cause of conjunctivitis and chlamydial pneumonia in neonates acquired at birth from infected mothers. It may cause conjunctivitis in sexually active adults. It is not prone to the epidemics or blinding complications of gonorrhoea. It is also one of the bacteria which in certain individuals can cause reactive arthritis. Culture negative endocarditis and meningoencephalitis due to *C. trachomatis* have been reported but are rare.

DIFFERENTIAL DIAGNOSIS

See *Case study — Urethral discharge* (page 161) for discussion of the differential diagnosis of urethritis in men. In women, the presence of vaginal discharge could represent cervicitis, pelvic inflammatory disease (PID) or vaginitis from candidiasis or trichomoniasis, as well as non-infective causes. Similarly, dysuria or abnormal urinalysis could be a manifestation of an STI with or without PID, vaginitis or a urinary tract infection.

PRINCIPLES OF MANAGEMENT

Investigations. Screening for chlamydia is recommended in Australia. The age range for screening varies per state and territory, partner and STI history and First Nations status.

Testing is also recommended for STI syndromes, as sexual partner testing as part of safe sex, screening, investigation of sterile pyuria and as part of contact tracing and follow up of STI. See the case studies for *Urethral discharge* (page 161) and *Lower abdominal pain* (page 165) for more detail on the syndromes.

In the Northern Territory, the finding of leukocytes in urine of 15–35-year-olds in remote First Nations communities should prompt consideration of empirical testing and treatment for both gonorrhoea and chlamydia.

Nucleic acid amplification testing (NAAT) has replaced chlamydia culture.

In women — self-collected vaginal swabs have a better yield than first void urine.

In men who have sex with men, rectal and throat swabs for chlamydia are part of the sexual health screen.

The finding of chlamydia on rectal swab NAAT in a man presenting with proctitis should prompt special laboratory testing to see if it is *Lymphogranuloma venereum*.

Pelvic inflammatory disease (PID) is notoriously difficult to diagnose, and a high degree of clinical suspicion must be maintained. Treatment should be syndromic and empirical. All women with possible PID and particularly those with confirmed chlamydia or endocervicitis, should be assessed for PID by appropriate history and examination.

Treatment and follow-up. Treatment of chlamydia depends on the syndrome. Doxycycline 100 mg PO, BD for 7 days is the recommended antibiotic regimen in Australian Guidelines. Azithromycin 1g oral stat dose is considered an alternative treatment. As a minimum, abstinence from sex is recommended for six days post treatment of partners and patient.

Resistance is not an issue at this stage. Co-infection with gonorrhoea is common in those populations at increased risk of gonorrhoea.

Contact tracing may need to go back six months prior to the onset of symptoms.

Chlamydia

Patient delivered partner therapy (PDPT) is legal in the Northern Territory but not in Western Australia or Queensland. See the information sheets on the NT CDC website.

Test of cure is not normally recommended but repeat testing three months post STI is recommended to rule out reinfection.

Chlamydia infection is a nationally notifiable condition by Australian LABORATORIES. Cases are reported to the local Centre for Disease Control/ Public Health Unit.

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the local sexual health unit and CDC/PHU.

MANAGEMENT GUIDELINES

National		
The Australasia Society for HIV, Viral Hepatitis, and Sexual Health Medicine (ASHM)	Australian STI Guidelines for use in primary care - Chlamydia	Available online
Northern Territory		
NT Centre for Disease Control (CDC)	NT Guidelines for the Management of Sexually Transmitted Infections in the Primary Health Care setting	Available online
Remote Primary Health Care Manuals (RPHCM)	■ CARPA Standard Treatment Manual — STI management/Chlamydia ■ Women's Business Manual — STI management for women/Chlamydia	Available online
NT Primary Health Network (NT PHN)	HealthPathways - Chlamydia	Available online (CLICK HERE to request access)

RESOURCES

NT Centre for Disease Control (CDC)	Fact Sheet - Chlamydia	Available online

FURTHER READING

See *An overview of sexually transmitted infections* (page 137)

Engstrom T, Baliunas D, Smith A, et al. Immediate and Ongoing Impact of COVID-19 on Chlamydia Treatment in Australia. Sex Transm Dis. 2023;50(6):363-369.

Munari SC, Goller JL, Hellard ME, Hocking JS. Chlamydia prevention and management in Australia: reducing the burden of disease. Med J Aust. 2022;217(10):499-501.

Lewis D, Newton DC, Guy RJ, et al. The prevalence of chlamydia trachomatis infection in Australia: a systematic review and meta-analysis. BMC Infectious Diseases. 2012;12:113.

Donovanosis

Treatment should be syndromic and empirical in most cases of genital ulcer disease (GUD).

DONOVANOSIS (GRANULOMA INGUINALE) IN NORTHERN AUSTRALIA

Donovanosis is thought to have finally been eradicated from Central and Northern Australia, where it had previously been diagnosed predominantly in First Nations people. This is the result of the introduction of treatment with azithromycin and dedicated donovanosis management programs, with the last confirmed case in Australia from 2014 and the last notified from the Northern Territory in 2008.

However, the disease has serious complications and ongoing awareness of this infection is very important. Consequently, all guidelines in Northern Australia that address remote First Nations communities still recommend routinely testing for donovanosis in patients presenting with genital nodules or ulcers. Treatment should be syndromic and empirical in most cases of genital ulcer disease.

AETIOLOGY AND PATHOGENESIS

The causative organism of donovanosis is a bacterium called *Klebsiella granulomatis* (formerly known as *Calymmatobacterium granulomatis*). Cells infected with this organism demonstrate characteristic "Donovan bodies" when appropriately stained. The bacterium is extremely difficult to culture.

Donovanosis is a sexually transmitted infection with many unusual epidemiological features, including a low incidence and transmission rate.

CLINICAL PICTURE

Risk factors. See *An overview of sexually transmitted infections* (page 135).

Symptoms and signs. Donovanosis has an uncertain incubation period, from weeks to many months. The first sign of the disease is usually a small nodule that ulcerates. The classical presentation of established donovanosis is a beefy-red, granulomatous, painless genital ulcer, which bleeds readily when touched. When complicated by secondary bacterial infection, it is often accompanied by a foul smell and may also become painful. Other variants include hypertrophic or verrucous (warty), cicatricial (scarring) and necrotic types. Donovanosis involves the genitals and/or inguinal lymph nodes in most cases, though extragenital lesions can occur, especially perianal ulcers. The sequelae of untreated donovanosis are progressive local destruction of genital tissue (which can be very severe and debilitating) and genital deformity (e.g. saxophone penis) with some case reports of associated genital tract carcinoma. Very rarely, the infection may disseminate.

Investigations. Nucleic acid amplification testing (NAAT) for donovanosis is recommended in remote First Nations communities for patients presenting with suspected genital nodules or ulcers. Vulval carcinoma is another important differential diagnosis, especially in East Arnhem Land. Suspected cases, based on a positive NAAT, or a lack of an alternative cause for a genital lesion, or when a biopsy is being considered, are best discussed with a public health or infectious disease or sexual health specialist. Specialist laboratory staining of impression smears or biopsies may be required, looking for "Donovan bodies".

PRINCIPLES OF MANAGEMENT

For discussion of the differential diagnosis, investigations and treatment, see *Case study — Genital ulcer* (page 163), or local guidelines (e.g. CARPA Standard Treatment Manual).

Empirical therapy for suspected donovanosis is azithromycin for donovanosis plus benzathine benzylpenicillin for syphilis as syphilis is much more common. Treatment should involve specialist advice as recent suspected cases have been eventually found to have other diagnoses.Donovanosis is a chronic condition that requires close follow up until completely healed. As with all STIs, test for other STIs including HIV, and contact partners of infected patients.

Donovanosis is a notifiable condition by LABORATORIES in Northern Australia. Cases are reported to the local Centre for Disease Control/ Public Health Unit.

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the local sexual health unit or CDC/PHU.

MANAGEMENT GUIDELINES

Northern Territory		
NT Centre for Disease Control (CDC)	■ NT Guidelines for the Management of Sexually Transmitted Infections in the Primary Health Care setting ■ Public health and notifiable diseases	Available online
Remote Primary Health Care Manuals (RPHCM)	■ CARPA Standard Treatment Manual — Sexual Health/Donovanosis ■ Women's Business Manual — Sexual Health/Donovanosis	Available online

RESOURCES

NT Centre for Disease Control (CDC)	Fact Sheet — Donovanosis	Available online

FURTHER READING

See *An overview of sexually transmitted infections* (page 137)

Bowden FJ; National Donovanosis Eradication Advisory Committee. Donovanosis in Australia: going, going.. Sex Transm Infect. 2005 Oct;81(5):365-366.

Trichomoniasis

Trichomoniasis is caused by a protozoan *Trichomonas vaginalis*. It is the most common nonviral STI worldwide. It is the most common STI reported in the Northern Territory.

TRICHOMONIASIS IN NORTHERN AUSTRALIA

***Trichomonas vaginalis* is transmitted almost exclusively by sexual intercourse and is common in First Nations communities of Northern Australia.**

The vaginal epithelium is the principal site of infection with *Trichomonas vaginalis* in women. Less commonly, the parasite is found in the endocervix, the urethra and the Bartholin's and Skene's glands. The clinical features may range from asymptomatic to florid vaginitis.

Symptoms occur in 20–50% of infected women; most commonly a frothy, malodorous, purulent, yellow-green vaginal discharge and dysuria. The vulva and vagina may become erythematous and oedematous. It can cause pruritus and dyspareunia. Though very uncommon, 'strawberry cervix', the appearance of multiple punctate cervical ulcerations, is a highly specific sign for trichomoniasis.

The differential diagnosis of vaginal discharge includes other vaginal infections, such as the white, curdy discharge of candidiasis and the thin, grey discharge of bacterial vaginosis; endocervical infection and/or pelvic inflammatory disease (PID), including gonorrhoea and chlamydia; as well as many non-infective causes. Similarly, dysuria can be a manifestation of an STI with or without PID, vaginitis, vulvitis or a urinary tract infection.

While many men with *T. vaginalis* are asymptomatic, it can cause urethritis presenting as discharge and dysuria. Rare clinical presentations include balanoposthitis, urethral stricture and epididymitis. Associations have also been reported with prostatitis and infertility. Spontaneous resolution of *T. vaginalis* infection appears to occur commonly over a few months, probably a result of anti-trichomonal host immune factors. This is in direct comparison with women, who can be infected for years. See *Case study — Urethral discharge* (page 161) for discussion of the differential diagnosis of urethritis in men.

Trichomonal infection can have significant complications during pregnancy. There are associations with low birth weight infants and pre-term delivery from premature rupture of membranes.

Trichomoniasis associated mucosal inflammation increases the risk of transmission of HIV.

Investigations. STI screening of symptomatic and asymptomatic people in remote First Nations communities and the mainstream population of the Northern Territory includes testing for trichomonas. Nucleic acid amplification testing (NAAT) of urine or self-collected vaginal swabs are the preferred test.

Treatment. First line treatment is metronidazole 400 mg orally with food, BD for 7 days or a single dose of metronidazole 2g orally with food. While Tinidazole 2g as an oral single dose is effective, it is no longer available in Australia. Metronidazole 400mg twice daily is recommended for breastfeeding women, with doses given immediately after feeds as high doses may affect the taste of breast milk.

Metronidazole is a category B2 drug during pregnancy. As a general rule, treatment of pregnant patients with or without symptoms, is indicated as *T. vaginalis* infection has been associated with adverse pregnancy outcomes. However, a stat dose is preferred rather than a longer course of metronidazole. Non-GP specialist advice is recommended if symptoms persist despite treatment as metronidazole resistance can occur.

Comprehensive contact tracing should be undertaken for partners of patients infected with trichomonas. No test of cure is recommended, but nucleic acid testing will usually be negative within two weeks and testing after three months can rule out re-infection.

> Trichomoniasis is notifiable condition by LABORATORIES in the Northern Territory. Cases are reported to the local Centre for Disease Control/ Public Health Unit.

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact sexual health unit, CDC/PHU in your local jurisdiction.

MANAGEMENT GUIDELINES

Northern Territory		
NT Centre for Disease Control (CDC)	NT Guidelines for the Management of Sexually Transmitted Infections in the Primary Health Care setting	Available online
Remote Primary Health Care Manuals (RPHCM)	■ CARPA Standard Treatment Manual — STI management/Trichomonas ■ Women's Business Manual — STI management for women/Trichomonas	Available online

RESOURCES

NT Centre for Disease Control (CDC)	Fact Sheet — Trichomoniasis	Available online

FURTHER READING

See *An overview of sexually transmitted infections* (page 137)

Human immunodeficiency virus (HIV)

The human immunodeficiency virus (HIV) causes acute and then chronic life-long infection. Practitioners need to be alert to the possibilities of local HIV transmission.

HIV IN NORTHERN AUSTRALIA

The incidence of HIV in the Northern Australia is similar to that of comparable non-metropolitan regions within Australia. However, it is considerably less than the national cumulative incidence, which includes notifications for New South Wales and Victoria, where rates are much higher.

In contrast to the rest of the country, the Northern Territory has a higher proportion of heterosexually acquired infection (2014-2023; 45% in the Northern Territory vs 23% nationally). This may reflect Darwin's close proximity to many countries with a high prevalence of HIV infection in the heterosexual population (including Southeast Asia). The index case in half of the heterosexually acquired Northern Territory cases originates from a high HIV prevalence country.

CLINICAL FEATURES

Risk factors. HIV can be transmitted when exposed to blood, semen, vaginal fluid or breast milk of an infected person.

The use of barrier prophylaxis (condoms and dental dams) lowers risk dramatically.

The single most important biological factor in determining risk of transmission is the viral load of the index case. Cases with an undetectable viral load will not transmit HIV (undetectable = untransmissible; U=U). Another key modifiable factor which increases the risk of infection for both parties includes co-infections with bacterial (e.g. chlamydia, gonorrhoea, syphilis), other viral (including herpes viruses), or parasitic sexually transmitted infections (such as trichomonas). Of note, these infections all share common routes of transmission with HIV.

The first confirmed case of HIV in First Nations people in the Northern Territory was in 1991. Since then, First Nations notifications have made up 10% of the total. There remains inadequate testing for HIV amongst First Nations people.

Screening. HIV testing should be offered to all people with an STI. Those undergoing testing must be made aware of the three month window period and be offered follow-up testing.

HIV is now becoming a rare condition in Australia, but transmission still occurs, and morbidity associated with late diagnosis is significant. Testing for HIV is cost effective even if the likelihood of a positive test is 1-in-1000. The list of indicator conditions is broad. Of particular note are patients presenting with any sexually transmitted infection, recurrent bacterial pneumonia, recurrent or multidermatomal shingles, chronic oral candidiasis, chronic diarrhoea of unknown cause, weight loss of unknown cause, lymphadenopathy of unknown cause.

PRINCIPLES OF MANAGEMENT

The Australasian Society for HIV, Viral Hepatitis and Sexual Health Medicine (ASHM) provide nationally accepted protocols on testing and treatment of STI/blood borne viruses with primary care practitioner-oriented resources.

There has been a great deal of progress in the care and treatment of people living with HIV. Life expectancy for HIV-infected people in developed countries approaches that of the general population. HIV is now considered a chronic manageable disease, analogous to disease like diabetes or hypertension. It requires life-long care and treatment and much of this can be done by GPs and primary care clinicians with good engagement of their clients.

Management of the HIV positive patient should involve a multidisciplinary health care team, including a shared care partnership between GP and a sexual health/infectious diseases physician (usually through the local sexual health clinic/hospital). Treatment of HIV with antiretroviral drugs should now be considered for all HIV positive people regardless of CD4 count or phase of illness. The Strategic Timing of Anti-Retroviral treatment (START) study has demonstrated significantly better outcomes for both HIV/AIDs-related morbidity/mortality as well as for non-HIV related outcomes with early commencement of antiretrovirals.

A standard regimen includes 3 or 2 active agents often given as a daily dose with just 1 tablet per day for most patients. Long-acting injectable treatment is also available for eligible patients.

Diagnosis of acute HIV infection has the advantage of providing early treatment with reduction of transmission to others, as transmission risk is highest early in the infection (due to high viral load) and can be minimised by the rapid reduction in viral load associated with treatment.

People with HIV who take antiretroviral therapy as prescribed and maintain undetectable viral load; a very low level of HIV in the blood (< 200 copies/ml)

will not transmit HIV to their HIV-negative partners through sexual intercourse. This is called undetectable = untransmittable (U=U).

GPs can prescribe antiretrovirals after completing the HIV S100 prescriber's course run by ASHM.

> HIV is a nationally notifiable condition by LABORATORIES. Cases are reported to the local Centre for Disease Control/Public Health Unit.

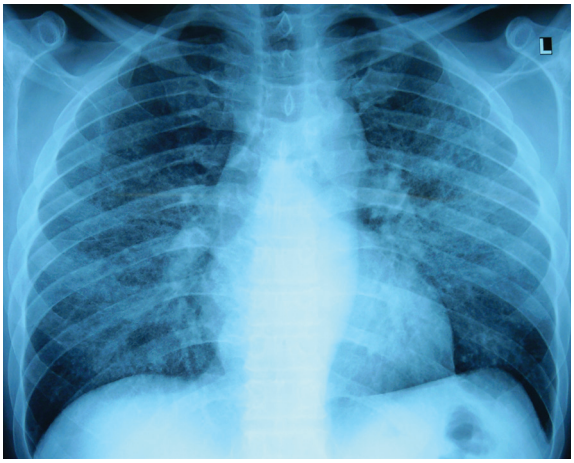


Figure 145: Pneumocystis pneumonia — undiagnosed HIV heterosexually acquired in SE Asia
Source: Bart Currie — Menzies School of Health Research

Human immunodeficiency virus (HIV)

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the local sexual health unit or CDC/PHU.

MANAGEMENT GUIDELINES

RESOURCES

Health Equity Matters	The facts about HIV	Available online
NT Department of Health	HIV	Available online
Australasian Society for HIV, Viral Hepatitis and Sexual Health Medicine (ASHM)	Prevention, testing and diagnosis, resources	Available online
Northern Territory AIDS and Hepatitis Council (NTAHC)	Resources	Available online

KEY REFERENCES AND FURTHER READING

See *An overview of sexually transmitted infections* (page 137)

Aanundsen D. Responding to HIV among Aboriginal and Torres Strait Islander people in the Northern Territory. HIV Australia. 2015;13(3):28.

Insight Start Study Group. Initiation of antiretroviral therapy in early asymptomatic HIV infection. New England Journal of Medicine. 2015;2015(373):795-807.

Kirby Institute. Annual Surveillance Report of HIV, viral hepatitis. STIs 2016.

Kong M, Ward J. HIV and sexually transmissible infections among Aboriginal and Torres Strait Islander people: summary of the latest surveillance data. HIV Australia. 2015;13(3):19-22.

National Institute of Allergy and Infectious Diseases. Starting Antiretroviral Treatment Early Improves Outcomes for HIV-Infected Individuals. 2015

Northern Territory Department of Health, Sexual Health & Blood Borne Viruses Unit, Centre for Disease Control. Northern Territory Sexual Health and Blood Borne Viruses Unit Surveillance Update. 2023.

Templeton DJ, Wright ST, McManus H, et al. Antiretroviral treatment use, co-morbidities and clinical outcomes among Aboriginal participants in the Australian HIV Observational Database (AHOD). BMC Infectious Diseases. 2015;15(1):326.

Ward J, Bryant J, Wand H, et al. Results of the first Australian study of knowledge, risk practices and health service access for Sexually Transmissible Infections (STIs) and Blood Borne Viruses (BBVs) among young Aboriginal and Torres Strait Islander people (The Goanna Survey). 2014.

Case study — Urethral discharge

Victor, a 25-year-old man, presents to the clinic with a two-day history of urethral discharge and dysuria. He admits to having unprotected sex about a week ago. The discharge is profuse and purulent.

What is the most likely diagnosis?

The most likely diagnosis is gonorrhoea. Infection with *Neisseria gonorrhoeae* in men usually produces copious and purulent discharge and dysuria, with an incubation period of about 2–5 days. In contrast, the incubation period of *Chlamydia trachomatis* is longer (7–21 days), with typically a milder urethritis and a scant, mucoid discharge. However, clinical features may range from asymptomatic to florid discharge and dysuria with both infections and therefore differentiating the two clinically is impossible.

What are the possible causes of urethral discharge?

Table 11: Aetiology of urethritis in males

Sexually transmitted	Other causes
■ **Gonococcal urethritis** > *Neisseria gonorrhoeae* ■ **Non-gonococcal urethritis (NGU)** > *Chlamydia trachomatis* > *Mycoplasma genitalium* > *Trichomonas vaginalis* > *Adenovirus* > *Herpes simplex virus* > *Candidiasis* > *Ureaplasma* spp.*	■ Prostatitis ■ Urethral strictures ■ Urinary tract infections ■ Chemical irritation ■ Trauma

* Considered normal urethral flora. Presence does not necessarily indicate an infection or need for treatment

Sexually acquired urethritis is divided into gonococcal and non-gonococcal aetiologies. Approximately half of non-gonococcal urethritis is caused by chlamydia, and the other half from other organisms. *M. genitalium* probably accounts for a significant part of *C. trachomatis* negative non-gonococcal urethritis. *T. vaginalis,* candidiasis and covert urethral herpes simplex infection should also be considered in men with urethral discharge not responding to routine therapy.

What specimens would you collect, and what tests would you order?

1. Obtain pus from the urethra for two urethral swabs.
   > One swab with transport media for MC&S (to be kept at room temperature but under 40°C)
   > One dry swab for chlamydia, gonorrhoea, trichomonas and *Mycoplasma genitalium* NAAT if NGU is suspected.
2. Blood serology for HIV and syphilis +/- hepatitis B if status needs clarification (and repeat HIV and syphilis serology in 6 weeks if window period considered important).
3. Men who have sex with men need throat and anal swabs (one dry and one with transport medium) sent for chlamydia, gonorrhoea and trichomonas.

In the presence of a urethral discharge, NAAT should be requested from a urethral swab and urine NAAT is not necessary. In the absence of a discharge, NAAT should be requested on urine. The best urine specimen for NAAT is first void.

When testing for gonorrhoea, a specimen should be sent for culture. Cultures enable antibiotic sensitivity testing and help maintain surveillance for antibiotic resistant strains.

Syphilis and HIV tests should be offered. Serology may be performed as a baseline, but the window periods need to be explained to the patient.

Would you offer Victor any presumptive treatment?

Yes. He should be offered immediate presumptive treatment for his urethral discharge. Delaying treatment until microbiological diagnosis is unnecessary and potentially harmful from both an individual and public health perspective, in terms of risk of transmission. The most common complication of gonococcal urethritis is epididymoorchitis, which has been reported to occur in about 20% of untreated patients.

What treatment would you give?

Victor should receive immediate 'syndromic management'. Despite differences in the typical presentations of gonococcal urethritis and non-gonococcal urethritis, it is impossible to make an absolute distinction on clinical grounds. Moreover, co-infection is common, with studies showing *C. trachomatis* isolation rates of 15–25% from urethral samples from men with gonococcal urethritis.

Victor needs treatment as a 'urethritis syndrome'. Empirical therapy should cover gonococcal urethritis and the common causes of non-gonococcal urethritis and *C. trachomatis*.

Historically, gonococci in remote communities in the Northern Territory remained sensitive to penicillin and an all-oral regimen of amoxicillin/probenecid and azithromycin was used. Sporadically, penicillinase producing resistant *N. gonorrhoeae* (PPNG) would enter and circulate for some time in some communities. In recent years PPNG has become more pervasive and as of 2024, the syndromic treatment is now ceftriaxone and azithromycin for all the NT. As with all STIs, comprehensive contact tracing should be undertaken usually going back to six months prior to the onset of symptoms. Victor should abstain from intercourse until one week after his and his partner's treatment. See him for follow-up one week later.

Education on safe sex is important, i.e. using condoms and testing of both partners prior to sex when with a new partner.

How should you follow-up Victor?

Follow-up is usually at one week and at three months post treatment.

At one week Victor should be asked about his well-being, re-assessed for symptoms and signs of urethritis (i.e. dysuria, expressible pus), given his results, and contact tracing progress reviewed.

Persistent symptoms at one week is called persistent urethritis, which should prompt testing for *M. genitalium* with NAAT and if not previously done, *T. vaginalis*. Testing for other organisms is sometimes considered. Some cases of urethritis, even though effectively treated, take two or three weeks to settle.

Test of cure (i.e. a repeat test to see if the organism is no longer there) is not routinely recommended except for *M. genitalium* infection.

A follow-up visit in three months is arranged to test for re-infection of the identified pathogens.

FURTHER READING

See *An overview of sexually transmitted infections* (page 137)

Chen W, Connor S, Gunathilake M. Men at risk of gonococcal urethritis: a case-control study in a Darwin sexual health clinic. BMC Infect Dis. 2019;19(1):991.

Case study — Genital ulcer

Cathy is a 28-year-old First Nations woman from a remote community who reluctantly presents to you for a 'check-up'. You offer a general 'well woman's check' which includes a speculum cervical screening test as it is unclear what is worrying her. On vaginal examination, you notice a large deep painless labial ulcer about 1cm in size which is red and non-tender. She also has non-tender bilateral inguinal lymphadenopathy. She says the sore has been present for a few days.

What is Cathy's most likely diagnosis?

The most likely diagnosis is primary syphilis. The classical chancre of primary syphilis presents as a single, painless, and indurated genital ulcer. This compares with the multiple, small, painful, superficial ulcers of herpes simplex virus infection (HSV) and the beefy red, fleshy, granulomatous and painless ulcers of donovanosis, the two other ulcerative sexually transmitted infections (STIs) found in remote Australia. Non-tender bilateral inguinal lymphadenopathy often accompanies the primary chancre of syphilis infection. This differs from the tender lymphadenopathy of herpes simplex virus infection and the usual absence of lymph node involvement in donovanosis.

The above 'classic' textbook descriptions of specific genital ulcers are often not found in clinical practice, and even experienced clinicians may be misled by appearance alone. Atypical presentations may be further altered by secondary bacterial infection. For example, primary syphilis chancre can be tender, especially when superinfected. Though herpes simplex virus ulcers are usually small (1–2mm) and multiple, they may coalesce into larger, solitary lesions. This emphasises the need to attempt microbiological diagnosis and offer syndromic treatment.

What are the other possible causes of genital ulceration?

Table 12: Aetiology of genital ulcer disease

Sexually transmitted	Non-sexually transmitted
■ **Syphilis** — both primary and secondary *Treponema pallidum* ■ **Genital herpes** Herpes Simplex Virus (HSV) — types 1 and 2 ■ **Donovanosis** (remote First Nations communities in Northern Australia, very rare) *Klebsiella granulomatis* ■ **Chancroid** (imported cases only in Australia) *Haemophilus ducreyi* ■ **Lymphogranuloma venereum** (mostly in men who have sex with men from urban areas or overseas) *Chlamydia trachomatis* ■ **Scabies** (excoriated) *Sarcoptes scabiei* ■ **Mpox** (Predominantly in men who have sex with men from urban areas or overseas) Orthopox monkeypox	■ Malignancy or neoplasia (a particular concern in Arnhem Land) ■ Severe candidiasis with fissuring ■ Fixed drug eruption ■ Behcet's syndrome ■ Non-sexually acquired ulceration (NSGU)

The prevalence of these specific aetiologies of genital ulcer varies according to the population. HSV is by far the commonest ulcerative STI in Australia. Syphilis notification rates are significantly higher among First Nations people in Northern Australia compared to non-Indigenous people. Similarly, donovanosis, although very rare in recent years, was historically much more common in First Nations people. Lymphogranuloma venereum may also be considered in men who have sex with men from urban areas with or without overseas contact, especially if they have associated proctitis or inguinofemoral lymph node swelling and/or discharge (bubo), with or without overlying erythema.

What specimens would you collect, and which tests would you order?

Cathy should have the following specimens collected:

- Dry swab from the ulcer for syphilis and HSV NAAT.
- Screen for other STIs i.e. chlamydia, gonorrhoea and trichomonas on first void urine, self-collected swabs and/or endocervical sample.#
- Serology for syphilis and HIV and consider repeating in three months to allow for window period. Hepatitis B serology is performed if immune status needs clarification.
- Pregnancy test (as pregnancy has major implications if syphilis is diagnosed, in terms of subsequent serological follow-up and antenatal/post-natal care).

Trichomonas testing is routine part of STI screening in remote First Nations communities of Northern Australia.

When performing a swab on an ulcer, the base of the lesion must be firmly swabbed to collect cells containing the intracellular virus of HSV. A dry swab is needed as the swabs with transport medium contain inhibitors that reduce the sensitivity of NAAT.

Biopsy and/or referral to a Gynaecologist should be considered in suggestive cases which have negative workups for syphilis, HSV +/- donovanosis, or if there is a particular concern on the history and examination about malignancy.

The presence of one probable STI, as with this case, demands that the practitioner consider the presence of other concurrent STIs. There is now a legal precedent for this in Australia. Therefore, not only should the ulcer be swabbed but other sites sampled to exclude other infections. If possible (if not painful to perform), the cervix should be visualised, and swabs collected for culture and NAAT as above.

In about 80% of patients with a syphilitic chancre the RPR is reactive, usually at titre of 1:16 or less. The *Treponema pallidum* haemagglutination (TPHA) is positive in about 90%. Therefore, while most patients with primary syphilis will have reactive serology, a negative initial result does not exclude the diagnosis. In patients with genital ulcers from secondary syphilis, the TPHA and RPR are almost always positive. NAATs such as syphilis PCR have sensitivity of 91% and specificity approaching 100% in the setting of chancre. They have the ability to detect as few as 10 treponemes per lesion. This is not 100%. Therefore, when clinical suspicion for syphilis remains present after initial testing, serology is worth repeating two weeks later even if the initial NAAT and serology is negative, and then at 3 months if this is still negative, along with HIV testing. Swabs from the ulcer for NAAT should be collected in all such cases.

Herpes simplex NAAT detects viral shedding and may be negative in older herpetic ulcers.

Type specific herpes serology should not be done in this setting. Such tests (if performed) must be interpreted with caution and the possible implications made clear to the patient prior to testing.

Serology for HIV and hepatitis B virus may be performed as a baseline but the window period for HIV must be explained to the patient.

Mpox (Monkeypox) is a viral infection transmitted through sexual and close contact with an infected individual. In Australia, cases are largely internationally imported however community transmission is increasing among men who have sex with men. Symptoms can include a viral prodrome and a painful genital or oral rash which may be ulcerating, as well as proctitis. Consider sending a dry swab of lesion fluid for Mpox NAAT in at risk populations with genital ulcers.

Would you offer Cathy presumptive treatment?

Yes. She should be offered immediate presumptive treatment for her genital ulcer. Delaying treatment until microbiological diagnosis is unnecessary and potentially harmful from both an individual and public health perspective, in terms of risk of complications and transmission, respectively.

What treatment and follow-up would you give?

Cathy should receive immediate 'syndromic management'. Although the testing is the same for all, empirical treatment for both syphilis and donovanosis in remote First Nations communities is reserved for painless lesions, whereas painful lesions are empirically treated for herpes simplex. Cathy should be given benzathine benzylpenicillin 2.4million units (2 x 21.2 million) and reviewed in one week.

Cathy should avoid sex until the lesion has healed and as per the guidelines for the specific STI identified. Contact tracing depends on the cause and starts after laboratory confirmation unless highly suspicious clinically. Confirmed herpes simplex does not normally need contact tracing. For laboratory confirmed primary syphilis partners need to be traced back three months prior to the onset of the chancre. For secondary syphilis trace back six months from onset of symptoms.

Subsequent follow-up and the need to notify the Public Health Unit or Centre for Disease Control depends on the cause of the ulcer.

FURTHER READING

See *An overview of sexually transmitted infections* (page 137)

Case study - Lower abdominal pain: more than just a pain

Serena is a 34-year-old First Nations woman who comes to see you at a community clinic. She has had lower abdominal pain and dysuria for four days and is now 'doubled up' with pain.

Serena has not noticed any vaginal discharge and does not think she is pregnant. Her periods are usually heavy, irregular, painful and last for 7 days. When asked, she thinks her period is about three to four weeks late. She has not used contraception in the 18 months she has been with her current partner and has no other partners. Her bowel habits are regular apart from a small amount of diarrhoea yesterday, with no blood in the stool.

Serena has two children aged six and four and is very keen to have more. She has had several urinary tract infections in the past, but no prior abdominal surgery or gynaecological procedures.

Serena has T 38.1°C, HR 90, RR 20, oxygen saturation 98%, BP 90/60. She is tender over the lower abdomen but has no obvious guarding or rebound. Bowel sounds are normal. On speculum examination she has some whitish cervical discharge, the os is closed and there is no blood. On gentle pelvic digital examination; she is tender in the right fornix, but no mass is palpable, and she is acutely tender on moving the cervix.

What important conditions should be diagnosed or excluded early?

An ectopic pregnancy must be excluded as Serena is not using contraception and her menstrual period is late. Also important are pelvic inflammatory disease (PID) and appendicitis.

What other causes should be considered?

Other conditions to consider include:

- Severe urinary tract infection.
- Ovarian cyst haemorrhage, rupture or torsion.
- Other less likely gynaecological pathology such as septic abortion, haemorrhagic corpus luteum cyst, endometriosis and fibroid infarction.
- Other gastrointestinal pathology less likely —gastroenteritis, acute infective colitis, diverticulitis.

What investigations would you do immediately?

- Urine tests for pregnancy and dipstick urinalysis and mid-stream urine for MC&S.
- STI screen, in order of preference —
  - (1) two endocervical swabs (one dry and one with transport medium), OR
  - (2) two self-collected swabs (one dry and one with transport medium), OR
  - (3) first void urine.

Specimens should be sent for *Neisseria gonorrhoeae* (NAAT and MC&S), *Chlamydia trachomatis* (NAAT) and *Mycoplasma genitalium* (NAAT). As Serena lives in a remote community the pathology form should also include *Trichomonas vaginalis* (NAAT).

- Blood cultures taken in clinic.
- Point-of-care blood Hb and lactate if available.
- Blood for CRP, FBC and film, U+E.
- Blood for STI screen: syphilis, HIV serology +/- hepatitis B serology.

What will you do if Serena's urine pregnancy test is positive?

If the pregnancy test is positive Serena must be managed for suspected ectopic pregnancy:

- Evacuate for urgent ultrasound at nearest centre with surgical facilities.
- Insert two IV lines.
- Frequent observations — especially be alert for PV bleeding and haemodynamic instability.
- Gentle or no further abdominal examinations.
- Pathology samples taken, including blood cultures, can be transported with her.

What will you do if Serena's urine pregnancy test is negative?

If the urine pregnancy test is negative, then pelvic inflammatory disease becomes more likely. Pelvic pain in a woman aged less than 35 years and living in an First Nations community is suspicious of PID, even in the setting of dysuria. Cervical excitation or adnexal tenderness make the diagnosis of PID probable, although other lower abdominal pathology such as an acute appendicitis or ovarian cyst pathology will also give adnexal tenderness. Pus from the cervix or an oedematous, friable or inflamed cervix increases the likelihood of PID further. PID is unlikely in a patient presenting with pain but no adnexal or uterine tenderness or cervical excitation.

Serena's pregnancy test is negative, and signs indicate treatment for PID is needed. However, other conditions, including appendicitis, are possible.

Serena has severe pain, a fever, low blood pressure, pelvic tenderness, and cervical excitation. She needs to be in hospital, and she needs IV fluids and IV antibiotics.

The recommended therapy to initiate while awaiting hospital transfer is as per current antibiotic guidelines for patients awaiting evacuation to regional hospitals.

After management of the acute condition, what follow-up investigation and/or management would be useful if the infection is mild, or if Serena does not wish to leave the community?

If Serena chooses to stay in the community and oral antibiotics are started, and there is no improvement in one to three days, hospitalisation for more detailed assessment, pelvic ultrasound and admission for IV antibiotics should be recommended.

Negative STI laboratory tests do not rule out PID as organisms are only found 70% of the time.

Early initiation of contact tracing, advice to abstain from sex until one week post treatment of both patient and partner, and safe sex advice are important. The extent of contact tracing depends on the organisms identified and the timing of symptoms. Sensitively advise that the risk of infertility increases with each episode of PID. This may empower patients to assist with contact tracing and insist on safe sex in the future.

Check the results of syphilis/HIV +/- hepatitis B tests.

In Serena's case pre-conception counselling is important.

FURTHER READING

See *An overview of sexually transmitted infections* (page 137)

SECTION 5 — TOXINS

Snakebites

All potentially lethal terrestrial snakes in Australia are front-fanged and belong to the family *Elapidae*.

SNAKEBITES IN NORTHERN AUSTRALIA

Snakebites occur more commonly in tropical Northern Australia than in temperate regions. With prompt first aid, access to care and antivenom when needed, deaths are rare.

However, recently there have been recorded envenoming deaths in North Queensland and the Northern Territory. In the Top End the annual incidence of bites in children during the 1990s was 18.3 per 100,000, the highest incidence of bites within Australia. The last confirmed death in the Northern Territory was in March 2023, with intracranial haemorrhage from a likely northern (tropical) brown snake bite in the rural Darwin region. Prior to that, in October 2018 there was a fatality from a sea snake bite with progressive neurotoxicity; the first death from sea snake bite in Australia for over 80 years. Brown snakes accounted for the last four Northern Territory deaths before that, with one in Alice Springs, one in a tourist in a remote Top End coastal camp and two fatalities from intracranial haemorrhage following northern (tropical) brown snake bites occurring in the suburbs of Darwin.

Snakes in the tropical north of Australia are active all year, so envenoming can occur in any month, although it is less common in the cooler months of June to August. Many bites are 'dry' and do not result in envenomation, however all suspected bites should be managed with a period of observation in hospital due to the sometimes-delayed effects of venom. Men are more likely to be envenomed than children or women.

AETIOLOGY AND PATHOGENESIS

Venomous snakes of the region.

All potentially lethal terrestrial snakes in Australia belong to the family *Elapidae*. There are no vipers (family *Viperidae*) in Australia, and tiger snakes *(Notechis* spp.*)* do not exist in tropical regions. A number of potentially lethal sea snakes (family *Hydrophiidae)* are also present in the surrounding seas and occasionally up tidal rivers. Table 13 shows the regional distribution of the potentially lethal terrestrial snakes, by decreasing frequency of envenoming for regions of Northern Australia.

There are many other species of less venomous elapids in the region and several species in the black *(Pseudechis* spp.*)* and brown *(Pseudonaja* spp.*)* snake genera which may not have been associated with fatal human envenoming. In the Top End whip snakes *(Demansia* spp.*)* account for more confirmed elapid bites than all other elapid species, but life-threatening envenoming has never been documented. Whip snakes, active during the day, are fast and aggressive and easily mistaken for taipans or brown snakes. Other less venomous species include the red-bellied black snake *(Pseudechis porphyriacus)* and Collett's black snake *(Pseudechis colletti)* in North Queensland and *Pseudonaja guttata, Pseudonaja ingrami* and *Pseudonaja modesta* in various locations across Northern and Central Australia.

The brown snake (*Pseudonaja)* genus has been recently further split, with the western brown snakes re-classified into 3 distinct species, with the northern (tropical) brown snake retaining the species name *Pseudonaja nuchalis.*

Table 13: The distribution of potentially lethal terrestrial snakes in tropical Australia in decreasing order of bites seen in each region

Tropical Northern Territory
■ **Pseudonaja nuchalis** Northern (Tropical) brown snake
■ **Pseudonaja mengdeni** Western brown snake (Gwardar)
■ **Pseudechis australis** Mulga or King brown snake (a misnomer as it belongs to the black snake *[Pseudechis]* genus)
■ **Acanthophis spp.** Death adder
■ **Oxyuranus scutellatus*** Taipan

* Taipans have been found in the Top End and across to the Kimberley, but are uncommonly encountered in these regions, with no recorded human bites except in snake handlers until early 2019.

Impact of cane toad on snake populations.

The introduction of the toxin-containing cane toad *(Bufo marinus)* from Queensland into the Northern Territory in 1980 and its progression across the Northern Territory (reaching Darwin in 2005) and into the Kimberley in 2009 has had a devastating impact on the native fauna. Frog-eating snakes such as the apex predators the mulga snakes and death adders, have substantially diminished in numbers. Taipans are not frog eaters and they therefore have a selective advantage in areas where cane toads are populous.

This appears to have happened and in early 2019 there was the first recorded taipan bite in the Northern Territory in a person who was not a reptile keeper or catcher.

The non-venomous but aggressive slaty grey snake (*Stegonotus cucullatus)* is immune to cane toad toxins and this snake now accounts for more snakebites in Darwin than any other species.

PATHOGENESIS

Snake venoms are a diverse and complex mixture of proteins. *Elapidae* venoms usually cause only minor local damage at the bite site and systemic effects predominate. The mulga snake *(P. australis)* can be the exception, with occasionally severe local damage, especially if a tight first-aid bandage has been applied around or above the bite site. Each snake species has a combination of venom components which usually cause consistent clinical envenoming syndromes.

The major venom components for Australasian elapids cause:

- **Early transient collapse (hypotension).** Early collapse is a reliable indication of systemic envenomation that may occur up to 30 minutes after the bite, often with a brief loss of consciousness, then full recovery until other features of envenoming occurs. This can be a most dramatic event, especially with brown snakes *(Pseudonaja* spp.*)*. The relative contributions of the various potential pathogenetic mechanisms for early hypotension and collapse are unknown and are likely to vary for different snakes across the world.
- **Neuromuscular paralysis (neurotoxins).** Progressive descending paralysis of the eyes (ptosis, diplopia, blurred vision), bulbar muscles, chest and



Figure 146: Mulga snake
Source: Bart Currie — Menzies School of Health Research



Figure 148: Slaty Grey snake Darwin (non-venomous)
Source: Bart Currie — Menzies School of Health Research



Figure 147: Northern or Tropical (formerly Western) brown snake
Source: Bart Currie — Menzies School of Health Research



Figure 149: Northern Death adder
Source: Bart Currie — Menzies School of Health Research

diaphragm, then limb muscles. Ptosis should be added to the neurological observations of all snake bite victims.

- **Haematological disorders —**
  - > **Venom-induced consumptive coagulopathy (VICC)** caused by activation of the clotting pathway by prothrombin activator toxins and consumption of clotting factors (fibrinogen, factor V and factor VIII) leading to a high INR, prolonged aPTT, low fibrinogen and high D-dimer.
  - > **Anticoagulant coagulopathy** is seen with black snake *(Pseudechis)* bites.
  - > **Thrombotic microangiopathy / microangiopathic haemolytic anaemia (MAHA)** typified by thrombocytopenia, schistocytosis, low haptoglobin, haemoglobinuria, hyperbilirubinaemia, and renal injury.
- **Rhabdomyolysis (myotoxins).** These result in muscle breakdown leading to high CK and myoglobinuria. This is detected by the presence of 'blood' on urinalysis. There may also be muscle pain associated with rhabdomyolysis.
- **Nephrotoxicity.** This can be seen with brown snake (*Pseudonaja* spp.) envenoming but can also be secondary to myoglobinuria from severe rhabdomyolysis which may result in the need for haemodialysis.

Early collapse after a snakebite in Australia correlates strongly with those snakes with potent pro-coagulant venoms. Direct myocardial depression by venom components is also possible.

CLINICAL PICTURE

Figure 150 summarises the clinical manifestations of envenoming from the major Australasian elapids. The four important 'non-specific features of systemic envenoming', headache, nausea, vomiting and abdominal pain, are common to envenoming from all species, but may be absent in bites from brown snakes even in the presence of total fibrinogen consumption. Similarly in death adder (*Acanthophis* spp.) envenoming, progressive neurotoxicity may develop in the absence of these non-specific features. This can be life threatening and is why a sleeping child, who has been bitten, needs waking for neurological observations including checking for ptosis.

THE TIME COURSE OF ENVENOMING

Table 14 shows the progression of envenoming, with features depending on the snake species. The early collapse and recovery, if present, are the first features (5–30 minutes).

Lymph node pain (tenderness on palpation may precede the symptom of pain), early non-specific systemic features and haemostatic abnormalities (manifest by oozing bite site or venepuncture sites,

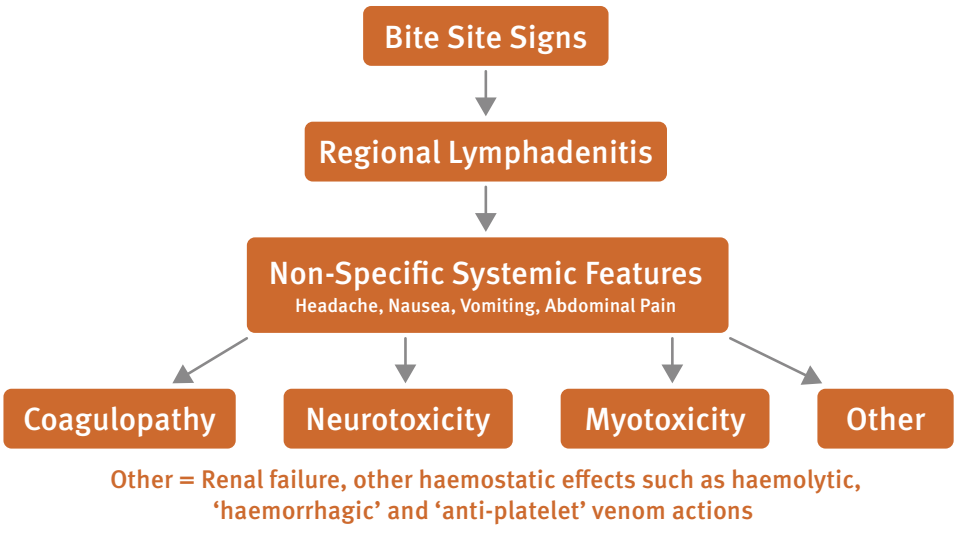


Figure 150: Australasian elapid envenoming

spitting blood, macroscopic or microscopic haematuria or prolonged glass tube whole blood clotting time) usually begin from 30–120 minutes after the bite.

Neuromuscular paralysis onset is often delayed for several hours and occasionally even 24 hours, possibly due to tissue sequestration of venom in the extreme case. First-aid with bandaging and immobilisation may also delay onset. The classical pattern of taipan (*Oxyuranus scutellatus*) envenoming without medical intervention has its onset of paralysis up to four hours after the bite, followed by steady progression over approximately 24 hours to a maximum deficit.

Ptosis is followed by ophthalmoplegia, then bulbar palsy and finally intercostal then diaphragmatic paralysis. Limb weakness is usually less severe and may not be evident. The clinical course of death adder (*Acanthophis* spp.) envenoming may be faster (related to post-synaptic neurotoxins) but may also be delayed and less severe without progression in mild cases.

The potential delay in onset of neurotoxicity, although unusual, justifies all cases of possibly venomous snakebite in tropical Australia, where bitten outside the urban limits (Darwin), being observed in hospital for 24 hours after the bite. In Darwin there is a 12-hour observation policy for urban bites, where death adder envenoming has not occurred.

INVESTIGATIONS

A urine dipstick positive for 'blood' can mean haematuria from consumptive coagulopathy, haemoglobinuria from intravascular haemolysis or myoglobinuria from rhabdomyolysis, or a combination of these.

A glass tube whole blood clotting test (WBCT). This simple test can be very useful to demonstrate procoagulant activity. A clot should normally form in a standard glass tube by 10 minutes. An assay validated in the field is the 20 WBCT, which simply determines whether a clot is formed in the glass tube by 20 minutes. With brown snake envenoming it is not unusual for the blood to remain completely unclotted.

Point of care INR. These tests are inaccurate in snakebite for technical reasons and should not be used. Formal laboratory coagulation testing is required to assess for coagulopathy and confirm any WBCT abnormality.



Figure 151: Northern Death adder fangs
Source: Bart Currie — Menzies School of Health Research



Figure 152: Death adder bite with subtle fang marks
Source: Bart Currie — Menzies School of Health Research



Figure 153: Brown snake coagulopathy
Source: Bart Currie — Menzies School of Health Research

PRINCIPLES OF MANAGEMENT

First aid

Pressure bandage of the limb and strict immobilisation by splinting (PBI) of the patient to slow venom absorption via the lymphatic system are the main first aid measures. Do not wash the bite site, as this may hinder venom identification. The bandage and immobilisation should remain in place until the patient is transported to hospital and can be released under supervision. Bandage the entire limb from distal to proximal (i.e. starting at the toes or the fingers) in one layer of half-overlapping elastic bandage, tensioned as for a sprained joint. Leave the tip of a digit exposed to permit neurovascular observations. Split the limb to prevent movement.

Pre-hospital care

All patients should be admitted/evacuated to hospital for observation, investigation, removal of immobilisation and pressure bandage and administration of antivenom if required. Prior to transfer (if in a remote clinic), establish intravenous access with 2 intravenous lines, monitor vital signs, neurological status including ptosis, oxygen saturation and ECG and keep nil by mouth. Check urine dipstick and whole blood clotting time as described above. Collect the first urine sample and a swab from the bite site if possible and send with the patient to hospital. Record the time of onset of any new clinical features (e.g. ptosis, dysarthria, bleeding). Seek expert advice and obtain detailed management guidelines. Antivenom should only ever be given in discussion with specialists.

The use of antivenom has recently been standardised as a result of a better evidence base and has resulted in significant changes in clinical practice.

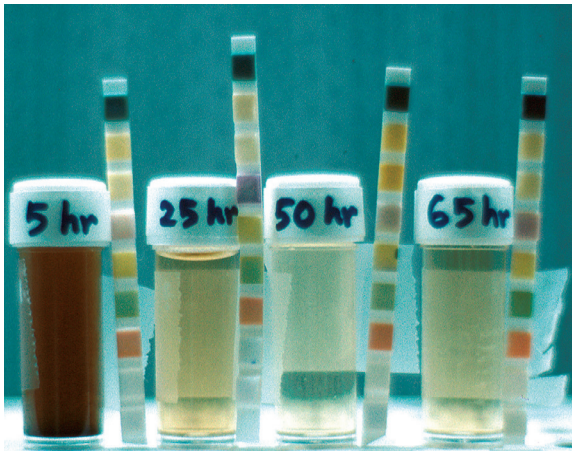


Figure 154: Mulga snake myoglobinuria
Source: Bart Currie — Menzies School of Health Research

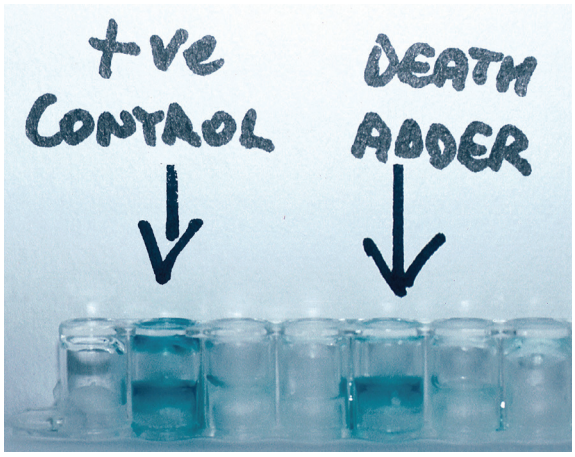


Figure 155: Death adder snakebite venom detection bite swab positive
Source: Bart Currie — Menzies School of Health Research

Table 14: Clinical syndromes of envenoming by the major Australasian snakes

	Early Collapse	Local swelling	Tender regional lymph nodes	Non-specific 'systemic features'[1]	Myotoxicity	Coagulopathy	Neurotoxicity
Brown snakes	++	+/-	+/-	+/-	-	+++[2]	Yes, but uncommon
Mulga snake		++	+	++	++	+[3]	Yes, but uncommon
Death adder	-	+/-	+/-	+/-	-	-	++[4]
Taipans	+	+/-	+	+	+	+[2]	++[5]
Rough-scaled snake[6]	+	+/-	+	+	+	+[2]	+
Eastern small-eyed snake[6]	-	+/-	+	+	+	+/-	?
Whip snakes[7]	-	+	+/-	+/-	-	-	-
Tiger snakes[8]	+	+	+	+	++	+[2]	++[5]

1 Abdominal pain, nausea, vomiting, headache
2 Predominantly procoagulant with fibrinogen depletion
3 Anticoagulant, no fibrinogen depletion, usually mild
4 Predominantly post-synaptic, may be delayed
5 Predominantly pre-synaptic
6-Not in the Northern Territory or Western Australia
7 Not potentially lethal but common
8 Not in the tropics but included for comparison

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call emergency physician.

National
Poisons information **13 11 26**

MANAGEMENT GUIDELINES

National		
White J. A	Clinician's Guide to Australian Venomous Bites and Stings, 2013.	Available online
Therapeutic Guidelines	Toxicology and Wilderness	Available online
Northern Territory		
Remote Primary Health Care Manuals (RPHCM)	CARPA Standard Treatment Manual — Bites and stings — snakebites - land and sea	Available online
NT Primary Health Network (NT PHN)	HealthPathways - Snake Bite	Available online (CLICK HERE to request access)

FURTHER READING

Isbister GK. Antivenom availability, delays and use in Australia. Toxicon X. 2022;17:100145.

Isbister GK, Chiew AL, Buckley NA, et al. Real world delays in antivenom administration: patient, snake or hospital factors (ASP-33). Clin Toxicol (Phila). 2024:1-6.

Noutsos T, Currie BJ, Wijewickrama ES, Isbister GK. Snakebite Associated Thrombotic Microangiopathy and Recommendations for Clinical Practice. Toxins (Basel). 2022;14(1):57.

Tasoulis T, Pukala TL, Isbister GK. Investigating Toxin Diversity and Abundance in Snake Venom Proteomes. Front Pharmacol. 2022;12:768015.

Ciguatera poisoning

Ciguatera syndrome is a potentially fatal multisystem condition that results from eating tropical reef fish that have been contaminated with an algal toxin.

CIGUATERA IN NORTHERN AUSTRALIA

Ciguatera occurs throughout the tropical and subtropical waters of the Pacific, Indian and Atlantic Oceans and surrounding areas. Outbreaks and sporadic cases occur in residents and visitors to tropical waters, and people who eat contaminated fish transported from affected areas. The Gove Peninsula is the only known high-risk region for ciguatera poisoning in the Northern Territory. The most recent published outbreak occurred in 1995 when twenty people were affected, of whom six required hospitalisation. All had eaten the same fish. Outbreaks also occur in North Queensland. In 2014, 17 people presented at Townsville Hospital with ciguatera poisoning.

AETIOLOGY AND PATHOGENESIS

Gambierdiscus toxicus and related species are dinoflagellate algae that adhere to dead coral. Under particular environmental conditions they produce toxins which are then biochemically converted to ciguatoxins. Herbivorous fish consume the algae, and the toxins increase in concentration along the food chain. Ciguatoxins particularly concentrates in the head, viscera (guts) and roe (eggs). Large fish tend to be the most toxic. Some of the fish implicated include surgeon fish, file fish, moray eel, coral trout, coral cod, red emperor, parrot fish, sweet lip, barracuda, red snapper, groper, mackerel, trevally, queenfish and estuary cod. Ciguatoxins do not harm the fish and cannot be removed by freezing, cooking or cleaning the fish. They are colourless, odourless and tasteless and the fish does not look spoilt. Symptoms are due to the direct effects of the toxin.

CLINICAL PICTURE

Symptoms and signs. Diagnosis is based on risk exposure and subsequent characteristic symptoms and signs including:

- Respiratory — dyspnoea, sore/dry throat or respiratory depression.
- Cardiovascular — bradycardia or hypotension.
- Gastrointestinal — vomiting, abdominal cramps, explosive diarrhoea.
- Neurological — temperature perception reversal (hot feels cold and vice versa), tingling and numbness around the lips, hands and feet, dental pain, muscle weakness, short term memory loss and headaches.
- Skin — severe pruritus, skin rash.
- Musculoskeletal — joint pain, muscle pain, neck stiffness, difficulties walking.
- Psychological — tiredness, depression.

In severe cases death may occur from respiratory paralysis. Previous exposure does not confer immunity and classically may increase sensitivity to the toxin. The acute illness lasts from 1–8 days, whereas neurological symptoms can last for months. Symptoms may also recur from time to time, for months or years. Recurrence is potentially triggered by certain foods (nuts, dairy products, meat and alcohol), and other situations such as heavy exertion or fatigue. Domestic animals such as cats can also be affected.

Investigations. The toxin can be found in samples of the fish, but there is no approved assay for testing for ciguatera toxins in humans.

PRINCIPLES OF MANAGEMENT

Treatment is supportive according to symptoms. Mannitol may relieve symptoms of ciguatera within 24 hours of the onset — seek specialist advice. Advise that alcohol consumption, exercise and dietary changes such as a high-protein or restricted diet, can make neurological symptoms worse.

Prevention

- Avoid eating fish species that are locally implicated. The fish species of most concern in the Northern Territory are Chinaman cod and red bass.
- Never eat the head, viscera or roe of reef fish.
- Consider all large warm water carnivorous reef fish with suspicion and eat no more than 250 grams of flesh at a first sitting.

> Ciguatera poisoning is a notifiable condition by CLINICIANS in the Northern Territory. Report cases to the local Centre for Disease Control/Public Health Unit.

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call specialist physician or local CDC/PHU.

National
Poisons information **13 11 26**

MANAGEMENT GUIDELINES

National		
Therapeutic Guidelines	Toxicology and Wilderness	Available online
Northern Territory		
Remote Primary Health Care Manuals (RPHCM)	CARPA Standard Treatment Manual — Fish poisoning-Ciguatera	Available online

RESOURCES

NT Centre for Disease Control (CDC)	Factsheet — Ciguatera fish poisoning	Available online
World Health Organization	Food Safety Digest – Ciguatera poisoning	Available online

FURTHER READING

Gould D, Kraa E, Dalton CB, et al. Foodborne disease outbreaks in Australia, 1995 to 2000. Communicable Diseases Intelligence Quarterly Report. 2004;28(2):211-224.

Neville G, Warren R. Ciguatera fish poisoning in Queensland: epidemiological features and a case definition for routine disease surveillance. Environmental Health Australia. 2003;3(4):11.

Box jellyfish

The major box jellyfish *Chironex fleckeri* causes potentially fatal stings.

BOX JELLYFISH IN NORTHERN AUSTRALIA

The world's most venomous box jellyfish species, Chironex fleckeri, seasonally inhabits coastal waters of Northern Australia. C. fleckeri has caused more than 70 deaths over the last 100 years in Australia. Children are most at risk because of their small body mass and the last 18 fatalities in the Northern Territory have occurred in children. Fatalities in north Queensland occurred in teenagers in March 2021 and February 2022.

While the first of October until the first of June is regarded as the peak 'stinger' season, box jellyfish may be present throughout the year. Even from June to September, the time considered to be the safest period for swimming, envenomations are still known to occur.

AETIOLOGY AND PATHOGENESIS

C. fleckeri has a rounded box shape transparent bell that can measure up to 25cm in diameter. The four corners of the bell have fleshy pedalia (feet) from which tentacles trail. Each pedalia can have up to 15 tentacles that can extend to 3 metres in search of prey. There are millions of nematocysts (stinging organelles) on each tentacle which, on contact with skin, fire threads through the epidermis which discharge venom into the dermis.

The toxins and their exact mechanisms are poorly understood. However, death which may result within minutes of being stung, is thought to be due primarily to cardiotoxicity. In addition, the venom is dermo-necrotic and may result in scarring.

CLINICAL PICTURE

Risk factors. Swimming in tropical coastal waters, particularly during the wet season. Young children are at greater risk of severe envenoming.

Symptoms and signs. Mild to moderate stings are much more common than severe envenoming:

- Severe localised pain.
- Respiratory — collapse with respiratory failure and/or cardiac arrest.
- Cardiovascular — arrhythmias and cardiogenic shock.
- Neurological — confusion, agitation, unconsciousness.
- Skin — immediate severe pain, then white welts where tentacles touch the skin. Welts change to erythematous wheals and necrosis and/or blistering may occur after 24 hours.



Figure 156: C. fleckeri jellyfish at Nightcliff beach
Source: Bart Currie — Menzies School of Health Research



Figure 157: C. fleckeri stings
Source: Bart Currie — Menzies School of Health Research

Investigations. A baseline ECG is useful in all but minor stings. Collect nematocysts by putting 4–8cm of ordinary transparent sticky tape over the sting site and then removing and taping it onto a glass slide for microscopy. Investigations are not otherwise necessary for mild stings.

Prevention remains the most important management strategy. Do not enter the sea, and most importantly, do not let children enter the sea during the stinger season — October to the first of June. Stinger suits are advised for water activities. Vinegar should always be available when swimming off the coast of Northern Australia.

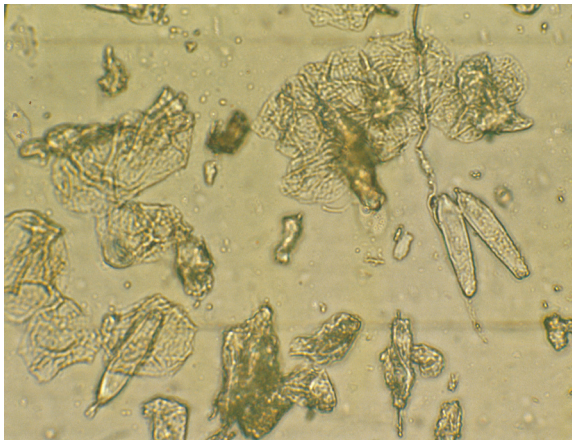


Figure 158: C. fleckeri nematocysts under microscope (x200 magnification) from sticky tape test
Source: Bart Currie — Menzies School of Health Research



Figure 161: C. fleckeri stings
Source: Bart Currie — Menzies School of Health Research

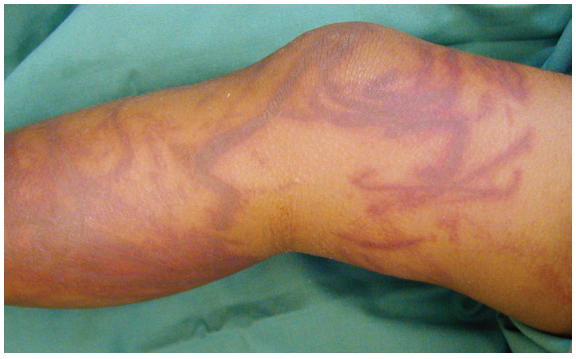


Figure 159: C. fleckeri sting at 5 hours
Source: Bart Currie — Menzies School of Health Research



Figure 162: C. fleckeri sting at 48 hours
Source: Bart Currie — Menzies School of Health Research



Figure 160: C. fleckeri sting at 5 days
Source: Bart Currie — Menzies School of Health Research



Figure 163: C. fleckeri sting at 13 days
Source: Bart Currie — Menzies School of Health Research

Box jellyfish

PRINCIPLES OF MANAGEMENT

- Immediately flood the affected skin with vinegar to prevent further envenoming from nematocysts.
- Carefully remove tentacles from the skin using tweezers, forceps, or gloved fingers.
- Control pain with narcotics — large doses may be needed.
- Severe cases may require cardiopulmonary resuscitation, immediate transfer to hospital and intravenous antivenom.
- Prevention of secondary infection is important to prevent scarring.
- Both ice packs and hot water can be beneficial for pain relief.



Figure 164: C. fleckeri sting at 10 weeks
Source: Bart Currie — Menzies School of Health Research



Figure 165: C. fleckeri delayed hypersensitivity papular uricaria at 19 days
Source: Bart Currie — Menzies School of Health Research



Figure 166: C. fleckeri delayed hypersensitivity 9 days after sting at Mindil Beach, Darwin in February – responded to topical steroids and antibiotics
Source: Bart Currie — Menzies School of Health Research



Figure 167: C. fleckeri delayed hypersensitivity 9 days after sting at Mindil Beach, Darwin in February – responded to topical steroids and antibiotics
Source: Bart Currie — Menzies School of Health Research

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call emergency physician or local CDC/PHU.

National
Poisons information **13 11 26**

MANAGEMENT GUIDELINES

National		
White J. A	Clinician's Guide to Australian Venomous Bites and Stings, 2013	Available online
Therapeutic Guidelines	Toxicology and Wilderness	Available online
Northern Territory		
Remote Primary Health Care Manuals (RPHCM)	CARPA Standard Treatment Manual — Box jellyfish sting	Available online

RESOURCES

NT Centre for Disease Control (CDC)	Fact sheet — Box jelly fish	Available online

FURTHER READING

Berling I, Isbister G. Marine envenomations. Australian Family Physician. 2015;44(1-2):28-32.

Irukandji syndrome

In 2002, one confirmed and one possible fatality from Irukandji syndrome occurred in North Queensland.

IRUKANDJI SYNDROME IN NORTHERN AUSTRALIA

The Irukandji syndrome was named in 1952 after a First Nations group that lived in the Cairns region. It is a characteristic constellation of symptoms that appear 10 to 40 minutes after a jellyfish sting. The symptoms have been attributed to a toxin induced catecholamine release following stings from a number of different carybdeid (four tentacled) box jellyfish. It has been clearly linked with Carukia barnesi in Queensland, but the syndrome also occurs in the Top End of the Northern Territory, Broome region in Western Australia, and other locations where other carybdeids are implicated. In 2002, one confirmed and one possible fatality from Irukandji syndrome occurred in North Queensland.

AETIOLOGY AND PATHOGENESIS

The venom from *Carukia barnesi* caught near Cairns, has been shown to act as a pre-synaptic neuronal sodium channel antagonist, stimulating the release of noradrenaline and causing many of the clinical features of Irukandji syndrome.

CLINICAL PICTURE

Risk factors. Swimming in the tropical waters of north Queensland, Northern Territory and northern Western Australia. Irukandji syndrome-causing box jellyfish are hard to see in the water as they are colourless and very small — the bell is 2.5cm or smaller and the four tentacles range from a few centimetres to 35cm. In divers, Irukandji syndrome and decompression sickness (the "bends") can be clinically similar.

Symptoms and signs:

- Cardiovascular — tachycardia, marked hypertension. The toxin is a direct myocardial depressant. Subsequent hypotension and cardiac failure with pulmonary oedema have been described, although rare.
- Gastrointestinal — abdominal pain, often with cramps and occurring in waves lasting a few minutes, plus nausea and vomiting.
- Skin — sweating and piloerection are common and can be severe.
- Musculoskeletal — back and chest tightness/pain, limb cramps.
- Psychological — marked distress and anxiety, restlessness, 'impending doom'.



Figure 168: Irukandji syndrome post jellyfish bell contact
Source: Bart Currie — Menzies School of Health Research



Figure 169: Irukandji syndrome severe despite minimal jellyfish bell contact
Source: Bart Currie — Menzies School of Health Research



Figure 170: Irukandji syndrome with severe sweating and piloerection
Source: Bart Currie — Menzies School of Health Research

PRINCIPLES OF MANAGEMENT

- Immediately flood the affected skin with vinegar to prevent further envenoming from nematocysts.
- Monitor the blood pressure closely.
- Control pain with narcotics — large doses may be needed.
- Consult with a tertiary emergency physician in severe cases to guide the use of intravenous magnesium and antihypertensives.

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call emergency physician.

National

Poisons information **13 11 26**

MANAGEMENT GUIDELINES

National		
White J. A	Clinician's Guide to Australian Venomous Bites and Stings, 2013	Available online
Therapeutic Guidelines	Toxicology and Wilderness	Available online
Northern Territory		
Remote Primary Health Care Manuals (RPHCM)	CARPA Standard Treatment Manual — Irukandji syndrome	Available online

RESOURCES

NT Centre for Disease Control (CDC)	Fact sheet — Jelly fish	Available online
	Fact sheet - Irukandji syndrome	

FURTHER READING

Berling I, Isbister G. Marine envenomations. Australian Family Physician. 2015;44(1-2):28-32.

Kong EL, Nappe TM. Irukandji Syndrome. 2023. In: StatPearls [Internet]

Neilson M, Skov S. Beware of jellyfish causing Irukandji syndrome. Northern Territory Disease Control Bulletin. 2014;21(3):6-9.

Stonefish

Stonefish envenomation is very painful but rarely life threatening.

STONEFISH IN NORTHERN AUSTRALIA

Stonefish are extremely well camouflaged fish found in the coastal waters of the Indo-Pacific region and grow to 35–50cm. Australia has 2 species —Synanceia horrida (the estuarine stonefish) and Synanceia verrucosa (the reef stonefish). The fish inflict their defensive effect through penetrating injuries caused by 13 dorsal spines each with paired venomous glandular tissue in holding grooves.

Deaths have been recorded in the Seychelles, Mozambique, and Japan and while there have been no reported deaths in Australia, the stonefish antivenom is one of the most frequently administered antivenoms. 265 cases of envenomation were reported between 1965–1981 in Australia.

AETIOLOGY AND PATHOGENESIS

Stonefish venom contains pre- and post-synaptic neurotoxins, vascular permeability factors, tissue necrosis factors and a vasodilator.

CLINICAL PICTURE

Symptoms and signs. Rapid onset of severe pain at the puncture site ("savage and incapacitating") is usually the main clinical effect. Pain may persist for more than 24 hours and radiate proximally with variable degrees of accompanying oedema. Other symptoms include:

- Respiratory — dyspnoea.
- Cardiovascular — dizziness, rarely hypotension, bradycardia and collapse.
- Gastrointestinal — nausea, vomiting.
- Skin — extensive tissue necrosis is usually a result of secondary infection from contamination or retained foreign matter including spines.

Investigations. Plain X-ray or ultrasound may be required to detect retained foreign body.

DIFFERENTIAL DIAGNOSIS

Many other fish have venomous spines, including lionfish and scorpionfish such as the bullrout. While stonefish are the most venomous of all fish, excruciating pain can also occur after stings from other fish with spines. The diagnosis of stonefish envenoming is often a clinical one, without a fish to confirm the species. Whether stonefish antivenom counteracts the venom of other spinous fish remains uncertain, but empirical use of stonefish antivenom in those with severe pain not relieved by narcotics and hot water is a therapeutic option.

PRINCIPLES OF MANAGEMENT

- Immersing the affected limb in hot water (40-45*C) for up to 90 minutes. This relieves pain but may not denature the toxins. Pain recurs when limb is removed from hot water. (Make sure the unaffected limb is immersed first to ensure water temperature is tolerable, preventing burns).
- Remove any visible spines or other foreign bodies present in the wound. Surgical exploration and debridement are usually required for wounds that contain foreign material, involve joints or sterile body cavities, or present late.
- Consider antibiotic treatment as per water related skin infections, especially if delayed presentation.
- IV narcotics are usually required, often with incremental dosing.
- Consider regional anaesthesia.
- Ensure adequate tetanus cover.
- **DO NOT** apply pressure immobilisation.

Antivenom. CSL stonefish antivenom (Equine IgG Fab) is used for severe pain refractory to above measures, or severe oedema or systemic envenoming. The antivenom is given as 1 ampoule (2000u) intramuscularly for every 2 punctures to a maximum of 3 ampoules, regardless of weight or age. It may be diluted and administered intravenously, in the emergency department or ICU setting.

There are no absolute contraindications, however there may be an increased risk of anaphylaxis in those previously treated or with equine sera allergy. Stonefish antivenom is safe in pregnancy.

Adverse reactions may include acute allergy, presenting as erythema or urticaria. Serum sickness may occur 5–14 days after antivenom. Symptoms include fever, rash, arthralgia, and myalgia.

Follow-up. Patients treated with opioid analgesia or antivenom may be discharged if asymptomatic for 4 hours. Warn patient of signs of secondary infection and risk of serum sickness if antivenom administered.

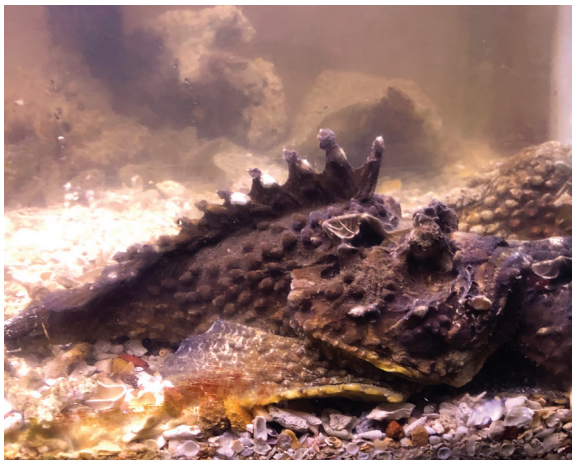


Figure 171: Stonefish
Source: Bart Currie — Menzies School of Health Research

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call emergency physician.

National

Poisons information **13 11 26**

MANAGEMENT GUIDELINES

National		
White J. A	Clinician's Guide to Australian Venomous Bites and Stings, 2013	Available online
Northern Territory		
Remote Primary Health Care Manuals (RPHCM)	CARPA Standard Treatment Manual — Stonefish and catfish sting	Available online

FURTHER READING

Lyon RM. Stonefish poisoning. Wilderness & Environmental Medicine. 2004;15(4):284-288.

Rensch G, Murphy-Lavoie HM. Lionfish, Scorpionfish, and Stonefish Toxicity. 2023. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025.

Cone snails

Cone snail envenoming can cause respiratory arrest.

CONE SNAILS IN NORTHERN AUSTRALIA

Cone snails are 1–12cm marine molluscs commonly found around coastal reefs and rocky outcrops.

All cone snails are predatory. They harpoon prey for food and predators for defence with venomous barbed darts ejected from a highly mobile proboscis. Because species are difficult to identify, all cone shells should be treated with caution.

There is no safe way of picking up a live cone shell. Envenoming has usually occurred when shells are handled out of water, including being placed in pockets.

Deaths from cone snail toxin have been recorded across the Indo-Pacific region. The only recorded death in Australia was at Hayman Island in Northern Queensland in 1935; a 27-year-old male picked up a live *Conus geographus*, developed progressive paralysis and died 5 hours after being stung on the palm. In 2015, CareFlight rescued a man in Northern Queensland, who needed ventilation as a result of cone snail envenoming.



Figure 172: *Conus geographus* (upper) *Conus textile* (lower)
Source: Bart Currie — Menzies School of Health Research

AETIOLOGY AND PATHOGENESIS

Cone snail venom is a complex mix of proteins with a wide range of effects on ion channels.

CLINICAL PICTURE

Symptoms and signs. Pain at the puncture site is usually sharp and may be excruciating but can also be relatively mild. This is rapidly followed by localised numbness which spreads and may be accompanied by partial or complete paralysis of limbs and most importantly of respiratory function with resulting respiratory arrest. Difficulties with speech, swallowing and coordination may precede paralysis. Nausea and hypersalivation can also occur.

DIFFERENTIAL DIAGNOSIS

While a history of handling a shell should usually be evident, in young children or in a paralysed patient the story may not be forthcoming. Under such circumstances, alternative diagnoses initially made could be envenoming from snakebite or blue-ringed octopus and ingestion of poison.

PRINCIPLES OF MANAGEMENT

There is no antivenom for cone snail envenoming. All cases should be triaged to urgent assessment and management in hospital and complete recovery may take up to 3 days. Supportive management includes:

- Pressure bandage with immobilisation as for snakebite, to delay venom absorption.
- Ventilation such as airway protection and bag-valve-mask ventilation until intubation is available (this can be lifesaving).
- Observe for and treat any secondary infection or retained foreign body.
- Ensure adequate tetanus immunisation coverage.

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call emergency physician.

National
Poisons information **13 11 26**

MANAGEMENT GUIDELINES

National		
White J. A	Clinician's Guide to Australian Venomous Bites and Stings, 2013	Available online

FURTHER READING

Saintmont F, Cazals G, Bich C, Dutertre S. Proteomic Analysis of the Predatory Venom of Conus striatus Reveals Novel and Population-Specific κA-Conotoxin SIVC. Toxins (Basel). 2022;14(11):799.

Kava use

Kava is consumed as a beverage, made from the crushed, powdered root of the kava plant (*Piper methysticum*).

KAVA USE IN NORTHERN AUSTRALIA

Kava in traditional beverage form was brought to Arnhem Land First Nations communities in the Northern Territory in early 1982. High consumption levels of this 'Pacific Elixir' soon became common and concerns were expressed about adverse health and social effects.

Based on the information available at the time, regulations were introduced to control kava consumption under the Kava Management Act, and in 2001, the Northern Territory Government introduced the Kava Licensing regimen. In June 2007, the Australian Government restricted the import of commercial quantities of kava to only pharmaceutical or research purposes. This resulted in the cessation of regulated trade of kava in the Northern Territory, but a thriving black market persisted.

From December 2021, Regulation changes have again permitted kava products to be imported into Australia as food items. The Kava Management Act still restricts the supply and sale of any kava product in the Northern Territory.

CLINICAL PICTURE

Kava has been consumed as a traditional beverage in the South Pacific for centuries. Kavalactones are responsible for the psychoactive properties of the plant which include mild intoxication, relief of anxiety and muscle relaxation.

Kava extracts, made into pills, liquids or powdered forms are also commercially available as medicinal anxiolytic products for sale in Australia.

Risk factors. Ingestion of kava.

Symptoms and signs. There is little documented evidence of adverse health effects with moderate consumption of traditional kava beverage. However, high levels of consumption, above 400grams per week of kava powder, have been associated with:

- Kava dermopathy (Figure 175).
- Nausea and indigestion.
- Malnutrition.
- Increase in the liver enzymes gamma glutamyl transferase (GGT) and alkaline phosphatase (ALP).
- Low lymphocyte blood count.
- Seizures and abnormal movements.
- A potential link to melioidosis.

These effects are considered reversible with cessation of use.

Kava dermopathy can be confused with tinea corporis, which is common in the Top End. In areas where kava is used, the diagnosis of fungal skin infection should be microscopically confirmed. A number of other issues have been associated with heavy kava consumption.



Figure 173: P. methysticum

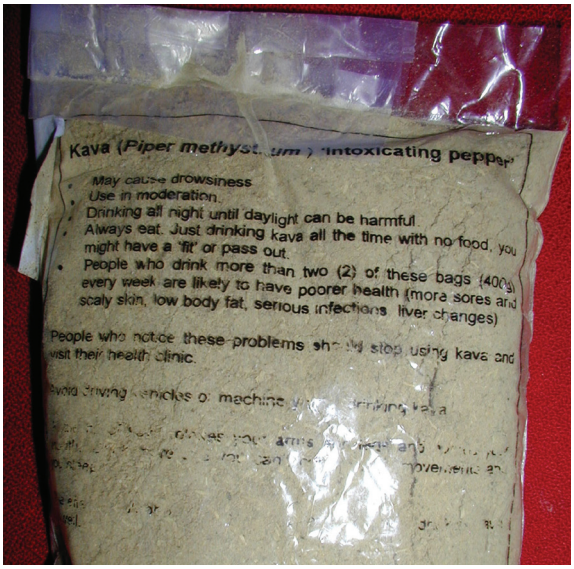


Figure 174: Commercial kava powder prior to being made illegal
Source: Bart Currie — Menzies School of Health Research

- Weight loss may be related to poor food intake.
- Driving may be acutely impaired after high levels of consumption.
- Heavy kava users may meet a criterion for substance use disorder, however no clear withdrawal syndrome has been described.
- Kava is metabolised by the liver, and as such, there is potential for drug interactions with other substances metabolised by the CYP450 pathway.

There are several international case reports of hepatotoxicity after consumption of kava in its traditional beverage form and a number of case reports after consumption of medicinal kava products, however the exact mechanism remains unclear. Kava is not a central nervous system depressant; however, it can increase the effects of alcohol.



Figure 175: Kava dermopathy
Source: Bart Currie — Menzies School of Health Research

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call medical specialist.

Contact the local Alcohol and Other Drugs service.

RESOURCES

National		
Alcohol and Drug Foundation	Kava	Available online
Northern Territory		
Remote Primary Health Care Manuals (RPHCM)	CARPA Standard Treatment Manual — Kava	Available online

FURTHER READING

Abid A, Allen N, Hagelamin A, Henderson C, Tatian A. Diagnosing Acute Kava Dermopathy: A Case Report of a Characteristic Cutaneous Eruption. Med J Aust. 2026;224(1):e70120.

Butt, J. Review of kava use among Aboriginal and Torres Strait Islander people. Australian Indigenous Health Bulletin. 2019;19(2).

Clough AR, Wang Z, Bailie RS, et al. Case-control study of the association between kava use and ischaemic heart disease in Aboriginal communities in eastern Arnhem Land (Northern Territory) Australia. Journal of Epidemiology and Community Health. 2004;58(2):140-141.

Teschke R, Sarris J, Schweitzer I. Kava hepatotoxicity in traditional and modern use: the presumed Pacific kava paradox hypothesis revisited. Br J Clin Pharmacol. 2012;73(2):170-174.

WHO Technical report 2016. Kava: a review of the safety of traditional and recreational beverage consumption.

Volatile substance misuse

Volatile substances are used to create euphoria and intoxication but have significant acute and chronic complications that are potentially fatal.

VOLATILE SUBSTANCE MISUSE IN NORTHERN AUSTRALIA

Volatile substance abuse in the Northern Territory in the 80s and 90s was largely related to petrol sniffing. The associated neurotoxicity from the incidentally ingested lead in petrol caused an outbreak of lead encephalopathy with deaths and considerable resultant long term neurological and cognitive impairment in individuals.

The removal of lead from regular petrol occurred in Australia in 2002. Low aromatic fuel was also introduced to many remote Indigenous communities in 2005. It should be noted that aviation fuel, although considered relatively low aromatic, is still occasionally sniffed and does still contain lead.

The toxicity and social effects of volatile substance misuse can be substantial. In 2005, volatile substance misuse was estimated to cost $78.9 million in Central Australia alone. It has been associated with juvenile crime particularly property damage, poor school performance, and unsafe sexual practices.

Overall rates of volatile substance misuse have declined in Indigenous communities since the early 2000s. However, low level endemic use continues in some communities. Outbreaks continue to be seen in an epidemic pattern in remote communities throughout the Northern Territory.

AETIOLOGY AND PATHOGENESIS

The primary psychoactive ingredients in most commonly abuse inhalants include toluene, benzene, xylene, propane and butane. These intoxicating and highly lipid soluble agents are found in many common commercial and household products including in glues, paints, deodorants, petrol and in fuel for portable stoves.

Most spray cans use propane or butane as a propellant. Some of these products may also contain other potentially dangerous ingredients which will be incidentally ingested at the time of abuse, such as lead in aviation fuel.

CLINICAL PICTURE

Symptoms and signs.

The immediate effects of inhalation include euphoria and intoxication. Acute effects of exposure to volatile agents include:

- Intoxication lasting minutes to hours.
- Sudden sniffers death (a syndrome characterised by sudden collapse shortly after intoxication with cardiac or respiratory arrest).
- Acute renal injury.
- Drug-induce psychosis.
- Seizures.
- CNS depression.
- Chemical pneumonitis.

Long term consequences of volatile substance abuse include cognitive impairment via lead encephalopathy or heavy toluene exposure. Petrol inhalation has been linked to short stature and low body weight, and there is an association with inhalant abuse and increase risk of suicide via various mechanisms. Other associated medical consequences of inhalant misuse include bone marrow suppression, liver dysfunction, burns and increase risk of misadventure.

An approach to managing a patient in this situation is described in the CARPA Standard Treatment Manual.

Prevention. Various interventions have been tried; the most successful of which have been those with community support and participation. Minimising harm strategies include:

- Supply reduction — substitution of petrol for fuels with low level aromatic hydrocarbons which are less intoxicating when sniffed, such as Avgas and OPAL; restricting sales of aerosols.
- Demand reduction — provision of educational activities, recreational programs, counselling, rehabilitation, treatment of underlying mental disorders, and outstation programs such as Mt Theo in Central Australia.
- Harm reduction — use of non-leaded petrol, avoiding use in enclosed spaces or with covering of the head, advising not to sniff near busy roads, avoiding use when alone.

The voluntary regional roll-out of low aromatic OPAL fuel in Central Australia from 2005 has been a particularly successful intervention, with a 97% reduction in the prevalence of petrol sniffing being observed from 2005–2012 in the communities involved.

> Elevated blood lead level more than 5mcg/dL is a notifiable condition by LABORATORIES in the Northern Territory. Cases are reported to the local Centre for Disease Control/Public Health Unit.

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call medical or paediatric specialist.

Contact the local Alcohol and Other Drugs service.

MANAGEMENT GUIDELINES

National		
NHMRC	Consensus-Based Clinical Practice Guideline for the management of Volatile Substance Use in Australia	Available online
Northern Territory		
NT Health	Elevated blood lead level Clinical and Public Health	Available online
Remote Primary Health Care Manuals (RPHCM)	CARPA Standard Treatment Manual — Volatile substance misuse	Available online

FURTHER READING

Australian National Audit Office. Delivery of the Petrol Sniffing Strategy in remote Indigenous communities. Canberra: Department of the Prime Minister and Cabinet; 2015.

Crossin R, Cairney S, Lawrence A, Duncan J. Adolescent inhalant abuse leads to other drug use and impaired growth; implications for diagnosis. Alcohol, tobacco and other drugs. 2017;41(1):99-104.

d'Abbs P, Gillick V Hodson S, et al. Longitudinal research into petrol sniffing and other substance abuse trends in Indigenous communities: final report. School of Public Health University of Queensland July 2019

Marel C, MacLean S, Midford R. Review of volatile substance use among Aboriginal and Torres Strait Islander people. Australian Indigenous HealthInfoNet. 2016.

Shaw G, d'Abbs P. Monitoring trends in prevalence of petrol sniffing in selected Aboriginal communities: an interim report. Darwin: Menzies School of Health Research; 2016.

SECTION 6 — OTHER CONDITIONS AFFECTING CHILDREN

Otitis media

Otitis media causes significant morbidity in First Nations children in Northern Australia. It contributes to conductive hearing loss as well as speech and learning delay.

OTITIS MEDIA IN NORTHERN AUSTRALIA

Rural and remote First Nations children have extremely high rates of otitis media (OM), including the more severe forms of acute otitis media with perforation (AOMwiP) and chronic suppurative otitis media (CSOM). Rates of perforation vary considerably from 5–67%. The World Health Organization considers perforation rates greater than 4% a public health emergency. In remote communities across the Northern Territory, only one in ten First Nations children younger than 3 years has healthy ears; five have otitis media with effusion (OME), or "glue ear"; and four have suppurative OM [acute OM (AOM) with or without perforation or CSOM]. All forms of OM can cause conductive hearing loss, which is associated with language delay, poor school attendance, and reduced education and employment attainment.

AETIOLOGY AND PATHOGENESIS

Otitis media is the presence of fluid in the middle ear space. This usually follows an infection that blocks the eustachian tube and prevents subsequent fluid drainage. Infections can be viral (most commonly *respiratory syncytial virus*, *influenza* and *rhinovirus*) or bacterial (most commonly *Streptococcus pneumoniae*, non-capsular *Haemophilus influenzae* and *Moraxella catarrhalis*).

Mixed infections are common. Bacterial infections are most important in AOMwiP and CSOM. Persistent discharge for longer than 6 weeks is usually associated with secondary infection with multiple additional organisms (most commonly *Pseudomonas aeruginosa*). High rates of antibiotic resistance and increasing tissue damage make this condition extremely difficult to treat.

CLINICAL PICTURE

Risk factors. The most important risk factor appears to be early exposure to other children with persistent nasal discharge, often driven by socio-economic disadvantage. Similar rates of severe forms of otitis media and persistent nasal discharge were seen in the poor neighbourhoods of all cities in the first half of the 20th century. Poorer outcomes from OM are associated with earlier onset of disease and longer duration of disease. Early diagnosis and treatment can prevent hearing loss and associated undesirable outcomes.

Other recognised risk factors from studies in Europe and the USA include recent upper respiratory tract infection, family history of otitis media, childcare attendance, large numbers of siblings, passive smoke exposure, lack of breastfeeding, and use of a dummy.

Symptoms and signs. Classification of otitis media has been published in national guidelines (Figure 176). Ear examination and accurate diagnosis requires careful practice and an auroscope with a bright light and the largest speculum that will fit the ear canal.

- **Non-severe otitis media**
  - **Otitis media with effusion (OME).** Presence of fluid behind an intact tympanic membrane without any of the symptoms or signs of an acute infection. OME is very common in young First Nations and non-Indigenous children. Accurate diagnosis requires pneumatic otoscopy or tympanometry. Bilateral disease is associated with a mean hearing loss of 25dB, which is the equivalent of sticking your fingers in your ears and the sound level of a whisper and can delay language development. It is important to document the presence of OME as well as duration, as bilateral persistent OME (pOME) for longer than 3 months requires antibiotics and close follow up.
  - **Acute otitis media without perforation (AOMwop).** Presence of fluid behind an intact tympanic membrane with either ear pain or bulging. AOM is very common in young First Nations and non-Indigenous children.
- **Severe otitis media**
  - **Acute otitis media with perforation (AOMwip).** Acute otitis media plus perforation of the tympanic membrane within the previous 2 weeks. First Nations children frequently do not report this as painful. Perforations usually heal and re-perforate several times before becoming chronic. Consequently, if there are signs of discharge in the canal, this diagnosis can be made even when the tympanic membrane appears intact.
  - **Chronic suppurative otitis media (CSOM).** Persistent discharge for at least 6 weeks despite appropriate treatment for acute otitis media with perforation. This condition can persist for many years and may result in the complete erosion of the tympanic membrane and adjacent ossicles. In these extreme cases, the associated hearing loss may be as great as 60dB (the sound level of conversation). Untreated, this impacts severely on learning in school. Prevention of chronic suppurative otitis media is a priority in First Nations health.

Investigations. Accurate diagnosis requires the use of either pneumatic otoscopy or tympanometry. Audiometry is essential to measure the degree of hearing loss.

DIFFERENTIAL DIAGNOSIS

Non-severe otitis media

Due to inherent challenges with physical examination, OM can be difficult to diagnose as it frequently occurs in association with other upper respiratory tract infections (e.g. rhinitis). However, the presence of fluid behind an intact tympanic membrane results in a diagnosis of AOM or OME. Unless ear pain is present, these conditions cannot be reliably distinguished without careful otoscopy or ideally pneumatic otoscopy or tympanometry. AOM is confirmed by the presence of a bulging tympanic membrane, where children with OME will have a normal or retracted membrane. Reliable diagnosis of OME requires pneumatic otoscopy to confirm reduced mobility of the tympanic membrane or a type B tympanogram.

Severe otitis media

It is important but sometimes challenging to distinguish between new perforations (AOMwip) and chronic discharge (CSOM). Discharge present for more than 2 weeks should result in a diagnosis of CSOM. Most new perforations occur in the first 2 years of life and are associated with small holes (less than 2% of the tympanic membrane). Children and adults who have longstanding persistent discharge despite appropriate treatment should be re-examined to exclude the presence of a cholesteatoma.

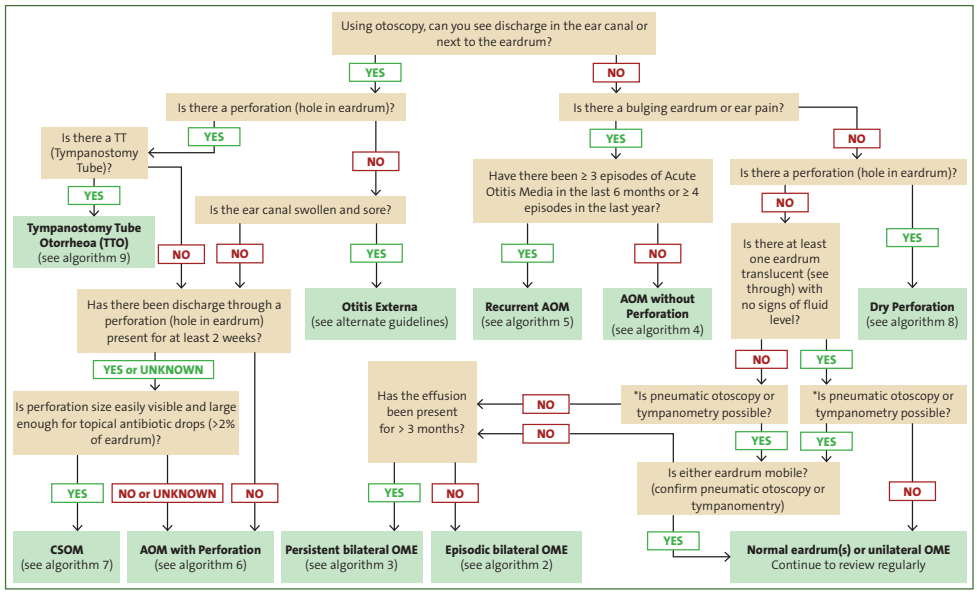
Cholesteatoma

Cholesteatoma can be a difficult diagnosis and should be suspected if there is granulation tissue visible in the canal near the drum, or if there is a retraction pocket with debris in the upper or postero-superior quadrant of the tympanic membrane. It is caused by an abnormal growth of skin cells in the middle ear, which continue to grow and eventually cause erosion of surrounding structures (middle ear ossicles, facial nerve and base of skull). Serious consequences include severe deafness, balance problems, facial nerve palsy and meningitis. An urgent ENT specialist review is indicated to confirm and treat such cases.

Otitis externa

Otitis externa is another important cause of ear discharge. It is usually associated with pain, swelling and discharge. It can be identified by pain on moving the pinna prior to otoscopy. The presence of otitis externa does not exclude severe otitis media since the chronic presence of discharge in the canal may be the cause of the local skin infection.

ALGORITHM 1: DIAGNOSIS Could this child have a middle ear infection (otitis media)?
(Use this Algorithm during examination and management of both ears accordingly)



*Pneumatic otoscopy or tympanometry is required to accurately diagnose OME

Figure 176: Otitis media diagnosis algorithm
Source: OM app https://otitismediaguidelines.com/

(Use this algorithm during diagnosis and management of both ears accordingly.)

Otitis media

PRINCIPLES OF MANAGEMENT

Many First Nations children from remote communities will have persistent otitis media and some associated hearing loss. Education about the importance of good hearing and advice on strategies that limit the effects of mild hearing loss should be provided to all families.

Early identification and clear education to caregivers about the recommended antibiotic therapy and ear toileting is the key to effective medical management. Parents should be advised about the need to bring their child to the clinic should they develop any ear pain or discharge. The overall aim is to prevent persistent ear discharge and to minimise any effects that hearing loss may have on the child's development.

In Australia, a permanent hearing loss of greater than 35dB is regarded as sufficient to warrant referral for hearing aids. Children with persistent hearing loss of 20–35dB should have access to classroom amplification. It is important that all remote schools have an ear health program.

Specific management strategies are dependent on the clinical diagnosis (Figure 176) and further details of each management pathway/algorithm can be found on the OM app.

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call paediatrician.

MANAGEMENT GUIDELINES

National		
Department of Health	Recommendations for Clinical Care Guidelines on the Management of Otitis Media in Aboriginal and Torres Strait Islander Populations	Available online
Northern Territory		
Remote Primary Health Care Manuals (RPHCM)	CARPA Standard Treatment Manual — Ear and hearing problems	Available online
NT Primary Health Network (NT PHN)	■ HealthPathways - Acute Otitis Media in Children ■ HealthPathways - Otitis Media with Effusion (Glue Ear)	Available online (CLICK HERE to request access)

RESOURCES

Aussie Deaf Kids	Glue Ear: A guide for parents	Available online
Deaf Connect	Support and Resources	Available online
Centre of Research excellence in Ear and Hearing Health	Otitis Media Guidelines App(OMapp)	Available online and as an iOS application

FURTHER READING

DeLacy J, Dune T, Macdonald JJ. The social determinants of otitis media in aboriginal children in Australia: are we addressing the primary causes? A systematic content review. BMC Public Health. 2020;20(1):492.

Leach AJ, Morris PS, Coates HL, et al. Otitis media guidelines for Australian Aboriginal and Torres Strait Islander children: summary of recommendations. Med J Aust. 2021;214(5):228-233.

Oguoma VM, Mathew S, Begum T, et al. Trajectories of otitis media and association with health determinants among Indigenous children in Australia: the Longitudinal Study of Indigenous Children. Public Health. 2023;225:53-62.

Diarrhoea in First Nations children

Acute infectious diarrhoea has a significant impact on the health of First Nations infants and children.

DIARRHOEA IN FIRST NATIONS CHILDREN

Historically over one third of First Nations children in the Northern Territory less than one year of age were admitted to hospital with acute gastroenteritis each year. These numbers have decreased significantly since the introduction of the rotavirus vaccine in 2006. Even so, diarrhoeal illness remains an important cause of admission to hospital for children under 5 years of age and their admissions are often prolonged due to multiple co-morbidities.

The high prevalence of diarrhoeal diseases in First Nations communities is related to overcrowding, poor hygiene and sanitation all of which reflect systemic socio-economic disadvantage. Recurrent episodes of diarrhoea can lead to Tropical Environmental Enteropathy Syndrome resulting in asymptomatic malabsorption. Poor growth and nutrient deficiencies associated with malabsorption have a myriad of associations with poor developmental and educational attainment outcomes.

AETIOLOGY AND PATHOGENESIS

The main routes for transmission of infectious agents causing diarrhoea are by contamination of fingers, food, fluids and fomites. Pathogens commonly implicated include bacteria, *(Escherichia coli, Campylobacter, Salmonella* and *Shigella*), viruses (particularly rotavirus) and parasites including nematodes (hookworm, whipworm and *Strongyloides stercoralis*), *Cryptosporidium* and *Giardia*. More than one pathogen is common.

Rotavirus infection is commonly associated with acidosis and osmotic diarrhoea, wasting and hypokalaemia. Infections with *Strongyloides stercoralis* and *Cryptosporidium* are associated with prolonged diarrhoea in children admitted to hospital.

High rates of lactose intolerance (25%) in First Nations children have been reported. The brush border enzyme lactase is reduced from a combination of malnutrition, tropical environmental enteropathy syndrome and acute infection. Malabsorption of lactose results in osmotic diarrhoea with water and electrolyte losses.

TROPICAL ENVIRONMENTAL ENTEROPATHY SYNDROME

Intestinal morphology and function have been shown to vary geographically. In almost all tropical areas, even asymptomatic inhabitants have a different small bowel structure with leaf shaped villi and ridges that are broader and more stunted in architecture. These changes are often associated with an increased inflammatory cell infiltrate and impaired permeability with reduced absorptive capacity.

The abnormalities described are not seen in the foetus and severity appears related to time spent in the tropics. They also affect different ethnic groups similarly. The causes are most likely environmental, with recurrent insults to the gut from infective organisms and changes in the gut microflora. Lack of access to safe drinking water, sanitation, and hygiene services is thought to exacerbate this condition. The underlying mucosal damage predisposes to the development of profuse diarrhoea with severe dehydration, acidosis and hypokalaemia from acute infective enteritis. Malabsorption contributes to early growth faltering in affected children.

CLINICAL PICTURE

Risk factors. Children at greatest risk of complications from diarrhoeal illness include:

- Infants less than 12 months.
- Children with faltering growth or malnutrition.
- Children with immune dysfunction.
- Children with chronic disease, with renal disease, congenital heart disease, metabolic disorders, short-gut syndrome or ileostomy/colostomy.

Signs and symptoms of gastroenteritis. Symptoms diarrhoea, vomiting, reduced oral intake and irritability. Signs of dehydration include tachycardia, reduced urine output, dry mucous membranes and absent tears, altered skin turgor, and decreased level of consciousness. **Recent weight loss provides the best approximation of the amount of dehydration.**

First Nations children presenting with diarrhoea should be thoroughly examined for other co-morbidities including ear, chest, central nervous system and urine infections.

Malnourished and septic children may be less dehydrated than clinical examination would suggest, and physical signs are unreliable in obese infants.

Investigations. Minor self-limiting episodes do not usually require laboratory investigation. Severe or prolonged cases usually require admission to hospital. Investigations for the cause of diarrhoea might include stool samples for bacterial microscopy, culture and sensitivity (MC&S), microscopy for ova, cysts and parasites (OCP), multiplex viral PCR, reducing substances for lactose intolerance. Investigations to screen for complications of severe diarrhoea include blood for electrolytes, acid/base status, serum urea and creatinine. If laboratory or point of care testing are not available, an ECG lead II rhythm strip may be helpful in assessing severe electrolyte disturbances (e.g. hypokalaemia) associated with acute gastroenteritis.

DIFFERENTIAL DIAGNOSIS

It is important to consider other diagnoses, especially in younger children.

Infective. If a fever of ≥39°C is present search for another focus of infection including septicaemia, meningitis, acute otitis media, pneumonia, urinary tract, or soft tissue infections.

Surgical causes may include peritonitis, volvulus, malrotation, pyloric stenosis, acute appendicitis or intussusception.

Metabolic causes include diabetes mellitus with ketoacidosis and inborn errors of metabolism.

Other. anaphylaxis, inflammatory bowel disease, acute food intolerance.

Haemolytic uraemic syndrome has a high mortality in children. Patients may present following a diarrhoeal illness prodrome with a triad of microangiopathic haemolytic anaemia, renal impairment, and thrombocytopenia.

PRINCIPLES OF MANAGEMENT

Treatment is aimed at restoring and maintaining water and electrolyte balance and ensuring adequate nutrition. The mode of replacement depends on the degree of dehydration. Children with **≥5% dehydration (moderate dehydration)** generally require hospital admission for nasogastric or intravenous rehydration. Early discussions with a doctor experienced in managing acutely unwell children is recommended if ongoing fluid resuscitation is required or where a child has **signs of shock (10% dehydration or more)**.

Signs of shock will include one or more of– reduced conscious state, tachycardia, tachypnoea, hypotension, weak peripheral pulses, mottled/cold peripheries, acidosis. Refer to regional rehydration guidelines (e.g. CARPA Standard Treatment Manual).

General principles of managing mild dehydration (<5%) include:

- Continue breastfeeding.
- Use oral rehydration solutions.
- Reintroduce solids early by feeding children if they are hungry.
- Review anti-helminth treatment especially when diarrhoea is prolonged, and avoid antibiotics, antiemetics, and antidiarrhoeals.
- Provide simple instructions for oral rehydration at home.
- Consider admission for—babies less than 6 months old; chronic diseases; if repeat outpatient review is not possible; or difficult social circumstances.
- Encourage hand washing at every opportunity.

Public health actions include following up diarrhoeal diseases occurring in food handlers or in clusters. In urban areas all cases of *Salmonella* and *Shigella* in children under five years of age are investigated.

> Salmonellosis, shigellosis and typhoid are nationally notifiable conditions to be reported by LABORATORIES in the Northern Territory. Rotavirus is notifiable by LABORATORIES and food/water borne disease in 2 or more related cases is notifiable by CLINICIANS. Cases are reported to the local Centre for Disease Control/Public Health Unit.

Diarrhoea in First Nations children

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call paediatrician, or local CDC/PHU.

MANAGEMENT GUIDELINES

Northern Territory		
NT Department of Health	■ Strong Women Strong Babies Strong Culture program — Information for Strong Women Workers, Government and Non-Government Professionals ■ Growing Healthy Kids	Available online
Remote Primary Health Care Manuals (RPHCM)	CARPA Standard Treatment Manual — Child health/Diarrhoea	Available online
NT Primary Health Network (NT PHN)	HealthPathways -Chronic Diarrhoea in Children	Available online (CLICK HERE to request access)

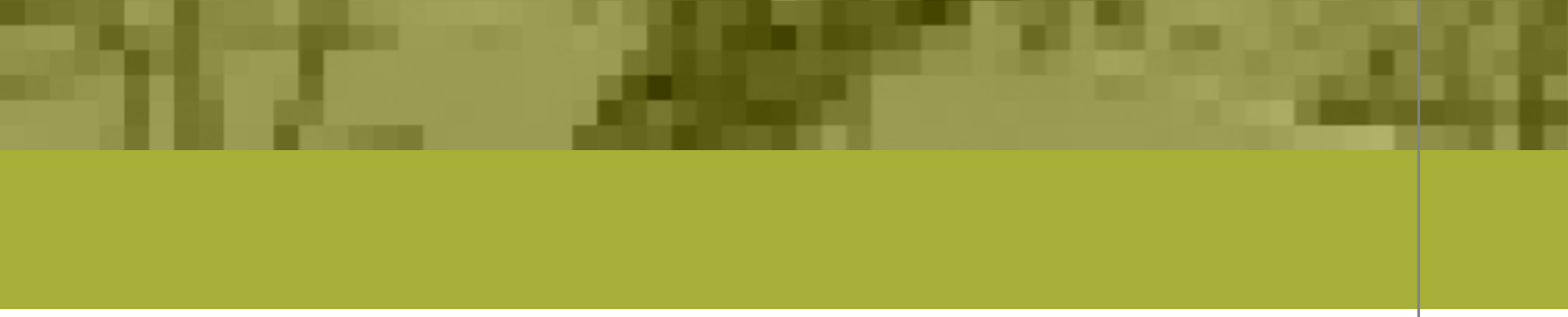
FURTHER READING

Donato CM, Roczo-Farkas S, Thomas S, et al. Australian Rotavirus Surveillance Group. Australian Rotavirus Surveillance Program Annual Report, 2022. Commun Dis Intell (2018). 2024;48.

Hanieh S, Mahanty S, Gurruwiwi G, et al. Enteric pathogen infection and consequences for child growth in young Aboriginal Australian children: a cross-sectional study. BMC Infect Dis. 2021;21(1):9.

Middleton BF, Danchin M, Quinn H, et al. Retrospective Case-Control Study of 2017 G2P[4] Rotavirus Epidemic in Rural and Remote Australia. Pathogens. 2020 Sep 26;9(10):790.

Pavlinac PB, Brander RL, Atlas HE, et al. Interventions to reduce post-acute consequences of diarrheal disease in children: a systematic review. BMC Public Health. 2018;18(208).



Case study — Recurrent diarrhoea

Martin is 8 months old and lives with his extended family in a remote First Nations community. He was born in hospital at term, after an uneventful pregnancy and weighed 2.6kg. The community nurses are concerned because Martin has not gained any weight for the previous two months. He was admitted to hospital at 3 months of age with pneumonia and gastroenteritis, and again at 6 months with gastroenteritis and iron deficiency.

Martin is referred to you after his mother and grandmother brought him to the health centre with a 1-week history of increasingly loose stools and fever. His mother says that Martin is still fully breastfed although he was less interested during the last two feeds. He had a small vomit whilst waiting at the health centre and is still wetting his nappies.

On examination, Martin is crying and irritable but appears vigorous. His temperature is 38.5°C, pulse rate 140/min, respiratory rate 38/min, BP 95/65 and he is well perfused. His lips and mucous membranes are dry, but his skin turgor is normal. His eyes look sunken. His heart sounds are normal, and lung fields are clear. His abdomen is soft and non-tender, and bowel sounds are present. He passes a moderate sized loose offensive stool during the examination. Two weeks ago, he weighed 7.0kg and today he is 6.72kg.

What do you think is the most probable cause of Martin's acute diarrhoea?

Acute infective diarrhoea is the most likely diagnosis. Martin's diarrhoea could be due to bacterial or viral organisms, including rotavirus which typically occurs in epidemics.

Pathogens commonly causing persistent diarrhoea from initial infection include *Shigella*, *Escherichia coli, Cryptosporidium*, *Giardia lamblia*, *Clostridium difficile* and *Salmonella*.

Additional explanations for his loose stools could include concurrent infection with gut parasites and/or lactose intolerance secondary to recent gastroenteritis episodes.

What do you think are probable causes of Martin's poor weight gain?

Martin's poor weight gain may reflect lack of introduction of solids to supplement breastmilk from 6 months of age, as well as acute infective gut insults, relative malabsorption, and current dehydration.

How dehydrated is Martin on the information provided?

Dehydration can be estimated based on clinical signs, weight measurements or a combination of these.

Martin's clinical signs of dehydration are dry lips and mucous membranes. Martin also has sunken eyes, but this could be because he is chronically underweight. He is tachycardic, but this could reflect his temperature rather than hypovolaemia. His acute weight loss is at least 280gm, which is 4% of his body weight measured two weeks prior to this illness.

The percentage of weight loss is equivalent to the percentage of dehydration:

- Mild dehydration is less than 5% weight loss.
- Moderate dehydration is between 5-10% weight loss.
- Severe dehydration is more than 10% weight loss.

Martin's dehydration is mild-moderate based on clinical assessment and degree of weight loss.

Table 15: Clinical assessment of dehydration

SIGN	Mild	Moderate	Severe
Main signs			
General appearance	Well, alert	Thirsty, restless or lethargic but irritable when touched	Drowsy, limp, cold or sweaty +/- unconscious
Eyes	Normal	A bit sunken	Very sunken
Tears	Tears	Less tears	No tears
Mouth and tongue	Moist	Sticky	Dry
Other signs			
Pulse rate	Normal	Fast	Weak, fast
Central capillary refill	Normal - less than 2 seconds	2 seconds	More than 2 seconds
Skin turgor	Normal - goes back quickly	Goes back slowly	Goes back very slowly

Source: CARPA Standard Treatment Manual

Suggest a management plan for Martin

Gather further history about diarrhoea, oral intake, other sicknesses. Examine Martin to exclude sepsis – continue to monitor vital signs and test urine & BGL.

Continue to breastfeed and supplement early with oral rehydration solution via cup, syringe, spoon or bottle. If he is not drinking, consider a nasogastric tube. For children with mild or moderate dehydration, enteral (oral or nasogastric) rehydration is preferable. For children with moderate dehydration, the amount of oral fluid replacement is based on weight:

- 50 mL/hour if under 5 kg
- 50-90 mL/hour if between 5 and 9 kg
- 100-140 mL/hour if 10-14 kg
- 150-200 mL/hour if 15-20 kg

Keep Martin in the clinic and monitor him regularly to check progress, with a full review of hydration status after 2 hours of commencing fluid replacement. Ensure management is discussed with family and the plan with ongoing review is clear.

Martin's grandmother starts to give him oral rehydration solution using a 10mL syringe. He vomits after the second syringe, but she perseveres with his rehydration and remains in the clinic for 4 hours.

Fortuitously a clean catch urine is collected. Urinalysis is weakly positive for protein only. The urine is sent to the lab in town, along with a stool specimen. Martin is given 3 days of albendazole. His mother continues breastfeeding and is recommended to start solid food. She is asked to bring Martin back to the clinic the following morning, or earlier if the vomiting and/or diarrhoea become worse.

Martin weighs 7.1kg the next morning. He looks well and is still breastfeeding. His stools remain loose but less frequent. There are no clinical signs of dehydration. Martin's 380g weight gain = 380mL water gain, which means that he was approximately 5.3% dehydrated.

On review 2 days later, the stool microscopy is negative for ova, cysts and parasites, there are no rotavirus antigens, and culture is negative for bacterial pathogens. Urine culture shows no significant growth and PCR is negative.

Case study — Recurrent diarrhoea

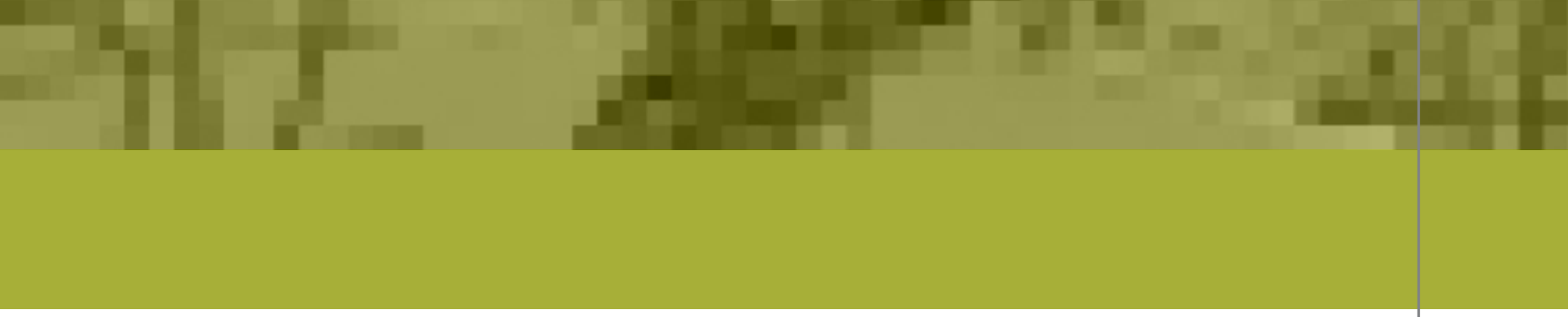
What would you now advise Martin's mother?

Martin should continue to breastfeed, and if the diarrhoea persists, he needs ongoing supplements with oral rehydration solution from the clinic. To improve his weight gain, he must be given more weaning foods, up to 6 times per day.

Provide education around diet, including iron rich foods, fussy eating. Talk about any social worries, family access to supports, maternal mood. Discuss available community-based services and activities that support positive parenting and healthy eating and provide food or advocate for financial and housing issues.

A Growth Action Plan is worked out for Martin according to the CARPA Standard Treatment Manual. The plan involves weekly weighs and reviews at the clinic and ongoing parental education and promotion of frequent nutritious foods for Martin. A multi-disciplinary approach is recommended including child health nurse, GP, paediatrician, dietician & First Nation health workers.

A First Nations Health Practitioner works closely with Martin's mother. She helps make sure that money is budgeted to buy food for Martin. Martin's maternal aunt is on the Strong Women's Strong Babies Strong Culture committee, and she is also asked to assist Martin's mother in learning how to give appropriate foods to Martin.



Case study — Anaemia

It is the first term of the school year. You and the health centre staff have just completed annual screening with the Healthy School-Age Kids Program. This combined health and education program aims to improve the health and learning outcomes of children by health promotion in schools, child health screening and integration of other programs and services for school-age children.

You saw 46 of the 51 children who live in the community. When you review the results, you notice that 14 children aged 4–15 years had haemoglobin (Hb) of less than 110g/L on a finger prick blood sample tested on the HemoCue haemoglobinometer. Two of these children had an Hb below 90g/L (79g/L and 83g/L).

What are the likely causes of the anaemia?

The most common cause of anaemia in remote First Nations communities is dietary iron deficiency. Contributors to iron deficiency anaemia in First Nations children are multifactorial and may include low birthweight, prematurity and maternal anaemia, twin birth, poor quality and late introduction of weaning foods, high rates of infection and tropical enteropathy syndrome associated with failure to thrive, and drinking cow's milk in the first year of life.

It is also recognised that recurrent & chronic infections, even mild viral infections, may transiently lower the Hb by several grams per litre. Children at high risk for anaemia are also at high risk of recurrent and chronic infections [skin, respiratory, ears & teeth]; they lead an 'inflammatory life' with persistently raised levels of inflammatory cytokines. Raised inflammatory cytokines provoke release of hepcidin from the liver. Hepcidin locks iron up in the bone marrow and reduces iron absorption through inactivation of ferroportin in the gut.

Hookworm (*Ancylostoma duodenale*) was historically the most important intestinal helminth in Australia, causing anaemia due to blood loss. It is found north of the Tropic of Capricorn, i.e. north of Tennant Creek. Whipworm (*Trichuris trichiura*) may cause growth faltering but only causes anaemia when infestations are very heavy (heavier than usually seen in the Top End). *Strongyloides stercoralis* rarely causes malabsorption, diarrhoea and growth faltering. However, severe infestation (hyperinfection) and malabsorption, usually in the context of immune suppression, may cause nutritional anaemia in children with diarrhoea.

Anaemia may be due to folate deficiency alone or in combination with iron deficiency. Prevalence of 0.6–15% has been reported in the Northern Territory for folate deficiency. These rates are lower than expected given the high rates of anaemia and malnutrition. This may be due to production of folic acid from bacterial overgrowth in the small bowel.

Inherited haemoglobinopathies are considered rare in First Nations Australians, although alpha thalassaemia carrier status has been identified in communities in the Northern Territory and Northern Western Australia. Melanesian ovalocytosis may occur in people from the Torres Strait.

Is mild iron deficiency anaemia (Hb 90–109g/L) a concern if the child is otherwise well?

Yes, it is a concern. Anaemia suggests that the iron stores are depleted. Iron deficiency can adversely affect psychomotor development during infancy and decrease concentration, reasoning ability and academic attainment in school-age children. Anaemia that develops slowly tends not to cause overt symptoms. Lethargy/weakness may be the only symptom. Untreated mild anaemia may progress to severe anaemia, with pallor and a flow murmur. Signs of heart failure are rare and tend to occur late.

Would you do any further investigations on these children, and if so, what?

Investigation depends on the prevalence of anaemia in the community and the severity of the anaemia. A venous blood sample for FBC and film and red cell folate are recommended if the Hb is below 90g/L and if there are other clinical indications. Iron deficiency anaemia is confirmed if the Hb is less than 110g/L and hypochromic, microcytic red blood cells are reported. Iron studies are mostly unnecessary and often difficult to interpret because chronic or recurrent infection increases inflammatory markers, such as ferritin.

The HemoCue haemoglobinometer, using finger prick blood samples, is a simple and acceptable screening tool which has high sensitivity and specificity. A Top End study confirmed high correlation between finger prick and laboratory Hb results. However, it is essential that staff are trained and follow the manufacturer's instructions and that the cuvettes are stored appropriately.

The haemoglobinometer reading may be used to initiate treatment for mild anaemia (Hb 90–109g/L) when the prevalence of anaemia in the school-age community is high because the positive predictive value of the test will be correspondingly high. When the community prevalence drops to 20% or below, a full blood count and film are recommended as the positive predictive value of the finger prick test is likely to be 62% or less, i.e. about one third with a screening Hb less than 110g/L will not be truly anaemic.

How would you treat these children?

Talk with the family about results and ask about diet, supports, & sicknesses. Possible symptoms from worm infections to ask about include diarrhoea, tiredness, weakness, tummy pain and weight loss.

In your community the coverage of screening was high i.e. (46/51) 90%. The prevalence of anaemia according to the haemoglobinometer reading was 30% so it would be reasonable to treat those children with an Hb 90–109g/L without doing a confirmatory test.

Treatment consists of iron replacement therapy and deworming. Anaemia management charts with dose tables are provided to all health centres.

Iron can be given as a daily dose of oral iron for 3 months, or as a supervised twice weekly oral dose for 3 months, or as a short course of intramuscular iron. Completing oral iron treatment is often a problem in remote communities. Decisions about the type of iron regimen should be made in consultation with children their carers and consider staff resources.

The broad spectrum anthelminthic albendazole is currently recommended as a daily dose for 3 days. A 3-day course of albendazole has a high cure rate for hookworm and may treat trichuriasis and strongyloidiasis which often co-exist in children with anaemia and faltering growth. This guideline may change pending further research into the community prevalence of intestinal parasites.

Iron treatment as per dose table will correct the anaemia and help replace the iron stores. However, if dietary iron intake is not sufficient, anaemia will likely recur.

Would you repeat the Hb, and if so, when?

A repeat Hb is recommended in 4-6 weeks at which time about 80% of treated children show improved Hb. If there has not been an increase in Hb, or it has fallen, then perform an FBC, film and red cell folate. If the child had been prescribed unsupervised daily iron and it is uncertain that all treatment was taken, offer IM or twice weekly supervised oral iron. Also take the opportunity to monitor weight and growth and general health check.

What advice would you give to the parent/carer to prevent their child becoming anaemic again?

Before giving advice, listen to understand the family's strengths, dynamics and challenges. Then propose a plan for the child, which might include:

- Eat regular nutritious meals and snacks.
- Encourage a range of different foods every day.
- Encourage the intake of meat, fish, bush foods, iron fortified cereals such as Weet-Bix, Milo milk drinks and green vegetables.
- Encourage fruit after meals for Vitamin C content.
- Avoid drinking tea with meals, as it decreases the absorption of non-haem iron by 75%. The iron added to cereals such as Wheet-Bix is non-haem, so tea with breakfast may prevent iron absorption, while orange juice will enhance it.

Case study — Anaemia

Good Food for Strong Blood is a resource developed for use with First Nations families of children who have anaemia.

Are there any other preventive measures to be considered?

Anaemia is a significant public health problem in the Northern Territory.

Anaemia surveillance and treatment alone will not solve the problem of iron deficiency anaemia although it may reduce the prevalence and severity. Household crowding, poor environmental health, problems with the availability and cost of fresh food and social and dysfunctional family pressures, including alcohol and substance abuse, and gambling all indirectly contribute towards the problem.

Some options to consider:

- Regular de-worming — where hookworm is or has been common.
- Prevent and treat anaemia in both pregnant and non-pregnant women.
- Advise mothers that their smoking can contribute to iron deficiency anaemia in children. Provide information and encouragement to quit.

Having a systematic approach to identify and manage anaemia in children has been shown to be feasible & acceptable. Some communities have developed successful breakfast and lunch programs with the support of the school and store. Such programs may also have a positive effect on growth, concentration and school achievement. Iron supplementation to prevent iron deficiency anaemia is rarely used in the Northern Territory.

SECTION 7 — OTHER CONDITIONS

Systemic lupus erythematosus (SLE)

The clinical manifestations of SLE are due to disturbed immune regulation. The diagnosis of SLE should be considered in any patient presenting with inflammatory joint pain or one or more features of a connective tissue disease.

SYSTEMIC LUPUS ERYTHEMATOSUS IN NORTHERN AUSTRALIA

Systemic lupus erythematosus (SLE) is a multi-system disorder characterised by the presence of numerous autoantibodies, circulating immune complexes, and widespread immunologically mediated tissue damage. SLE affects women more frequently than men and the prevalence of SLE in First Nations Australians is estimated to be two to four times that of non-Indigenous Australians. First Nations Australians with SLE also have a much higher morbidity, and the rate of mortality is 6 times higher than for non- First Nations Australians. Socioeconomic, environmental and biological factors likely contributing to this difference, however, detailed research in this area is lacking.

AETIOLOGY AND PATHOGENESIS

The cause of SLE is currently unknown but is thought to be a loss of self-tolerance in a genetically susceptible individual with environmental factors playing a role. Clinical manifestations are heterogenous and occur due to the immune dysregulation affecting multiple target organs. Tissue damage occurs from direct cytotoxicity of autoantibodies and complement, and from deposition of immune complexes.

CLINICAL PICTURE

Risk factors. Infections, drugs and UV light are common trigger agents. Kidney involvement, high disease activity, and sepsis associated with immunosuppressive therapy have been identified as risk factors for poorer outcomes.

Symptoms and signs. Arthritis, arthralgia and systemic symptoms such as fatigue and fever are the most common presenting symptoms. While the classic malar rash is said to occur in over two thirds of patients, it is less commonly documented in First Nations people although discoid (skin) lupus may be present. However, concurrent skin conditions such as scabies, fungal and bacterial infections are common. In Northern Australia, SLE sometimes first manifests as crusted scabies in a previously healthy young person. Kidney involvement, particularly proteinuria, occurs in more than 50% of patients and carries a worse prognosis. Haematological involvement with autoimmune haemolytic anaemia and cytopenias is commonly seen in First Nations Australians, as is cardiopulmonary disease such as pericarditis, pleurisy and pulmonary hypertension (seen rarely in non-Indigenous Australians).

Central nervous system involvement includes mild psychiatric disturbances, frank psychosis and seizures. SLE may cause secondary Sjogren's syndrome (dry eyes and mouth) and secondary antiphospholipid antibody syndrome (miscarriages, thrombosis and occasionally chorea).

Investigations. The clinical diagnosis can be supported by the presence of antinuclear antibodies which are detected in more than 90% of patients but are also commonly found in other autoimmune diseases (i.e. non-specific). Positive ANA should prompt testing of ENA and anti-dsDNA antibodies. Anti-dsDNA antibodies are more specific but only found in 50% of patients. Inflammatory markers are helpful to determine disease activity: the ESR is usually raised in active disease while the CRP is rarely raised in the absence of infection. FBC may demonstrate leucopenia, anaemia or thrombocytopenia. Low complements (C3 or C4, or both C3/C4) is also indicative of active disease. Evidence of deposition of immune complexes may be found on skin or organ biopsies. If urinalysis is positive a mid-stream urine specimen for identification of active sediment should be sent as the presence of renal involvement will affect management.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses depend on which organ is involved; however commonly considered differentials include acute rheumatic fever, gonococcal arthritis, other autoimmune diseases (e.g. rheumatoid arthritis, mixed connective tissue disease and vasculitis), dermatitis and idiopathic thrombocytopenic purpura.

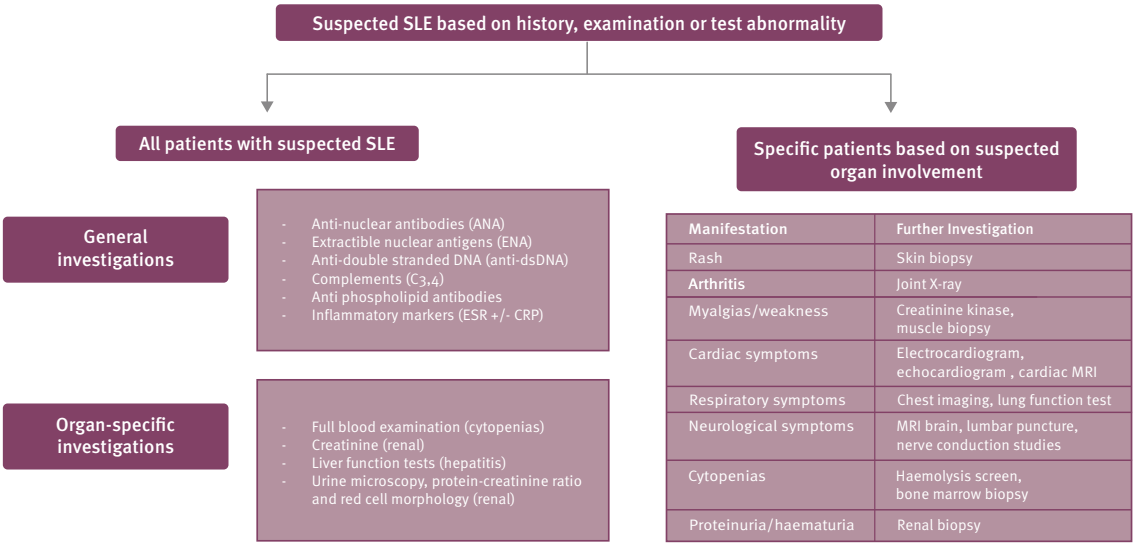


Figure 177: An approach to investigations in the diagnosis of systemic lupus erythematosus.
Source: Connelly K, Morand EF. Systemic lupus erythematosus: a clinical update. Intern Med J. 2021;51(8):1219-1228.

PRINCIPLES OF MANAGEMENT

SLE is not curable and complete remission is rare. The health team, the patient, the family and specialist physician should develop a plan to control day-to-day symptoms and acute episodes. Acute episodes may be life threatening and are usually managed with high dose corticosteroids. The antimalarial hydroxychloroquine is also an immune modulator and is used long term for most patients with additional therapy based on disease manifestations. Patients on daily hydroxychloroquine need regular ophthalmology reviews. People prescribed immunosuppressant medications (such as methotrexate, azathioprine, mycophenolate or rituximab), need regular blood test monitoring and assessment and management of latent infections, particularly **tuberculosis, melioidosis, hepatitis B** and **strongyloidiasis**. Non-pharmacological aspects of care include sun avoidance, not smoking, addressing of cardiovascular risk factors, bone health, keeping vaccinations up-to-date and careful pregnancy planning.

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call specialist physician.

MANAGEMENT GUIDELINES

British Medical Journal	EULAR recommendations for the management of systemic lupus erythematosus: 2023 update	Available online

RESOURCES

Arthritis Australia	Staying moving staying strong LUPUS systemic lupus erythematosus (SLE)	Available online

FURTHER READING

Nikpour M, Bridge JA, Richter S. A systematic review of prevalence, disease characteristics and management of systemic lupus erythematosus in Australia: identifying areas of unmet need. Intern Med J. 2014;44(12a):1170-1179.

Machado-Joseph disease (MJD)

Machado-Joseph disease is an autosomal dominant spinocerebellar ataxia.

MACHADO-JOSEPH DISEASE IN NORTHERN AUSTRALIA

Machado-Joseph disease (MJD) is an inherited neurological disorder. The disease was thought to have been spread around the world by Portuguese and other traders, eventually reaching Arnhem Land.

In 2012 haplotyping studies confirmed that the disease is of the 'Joseph' lineage, which originated in Asia more than 6000 years ago, and not the 'Machado' strain, which is more common in Portugal. The disease was first documented in the Top End in the late 1980s and is now found in 12 remote communities across the Northern Territory. In 2024 there were 51 known affected individuals, another 75 being monitored for the disease, and 855 at risk (i.e. having a parent or grandparent with MJD).

The MJD Foundation was established in 2008 to improve the quality of life for First Nations Australians and their families living with MJD in Arnhem Land and other affected communities in the Northern Territory and Queensland.

AETIOLOGY AND PATHOGENESIS

Machado-Joseph disease, also known as spinocerebellar ataxia type 3 (SCA3), is an autosomal dominant neurodegenerative disorder due to an abnormality of chromosome 14. The affected chromosome 14 produces ataxin3, which prematurely destroys nerve cells, affecting the cerebellum and its connections, including the brainstem, basal ganglia, vestibular nuclei, dorsal root ganglia, anterior horn cell atrophy, and variable long tract degeneration. There is a similar condition in First Nations people of Far North Queensland (SCA7) with a different mutation but almost identical clinical picture plus blindness due to retinal degeneration.

MJD manifests genetic anticipation whereby the abnormal DNA repeat length tends to increase with subsequent generations conferring a much higher risk of earlier disease onset and greater severity, such that children may be affected. As a dominantly inherited disease, only one parent is required to be affected for offspring to have a fifty percent chance of being affected. Critically, in a substantial proportion of people, the clinical features of MJD only emerge after they already have children.

CLINICAL PICTURE

Risk factors. Having a parent or grandparent with MJD.

Symptoms and signs. A complete neurological examination is required. In First Nations peoples, condition is characterised by:

- **Ataxia**: Ataxia is usually the first feature of MJD; early symptoms and signs can be subtle. They involve multiple muscles with incoordination of gait, truncal instability, ataxic upper limb function, slurring of speech (dysarthria), difficulty swallowing (dysphagia and aspiration) and abnormal eye movements (oscillopsia). In the early stages, suspicion, context & a careful history are crucial.
- **Progression**: the clinical features of MJD relentlessly progress but the pace of progression varies widely. The following clinical features become increasingly important over time.
- **Eyes**: there are numerous eye manifestations of MJD, including strabismus (diplopia), nystagmus (spontaneous oscillopsia), vestibular hypofunction (motion-induced oscillopsia; also due to cerebellar impairment), and ophthalmoplegia (does not affect vision unless severe).
- **Somatosensory**: reduced sensory perception, sometimes in a patchy fashion (dorsal root ganglionopathy). Tendon reflexes may be normal, hyperreflexic or hyporeflexic. Back pain is a common symptom.
- **Spasticity and increased muscle tone**: may contribute to gait unsteadiness and reduced upper limb functionality. In severe cases may be associated with contractures.
- **Bladder:** neurogenic bladder (urgency, incontinence sensation as if incomplete bladder emptying) and urinary retention.
- **Bowel:** chronic constipation.
- **Sleep disturbances**: insomnia, obstructive sleep apnoea, restless leg syndrome, REM sleep behaviour disorder (dream enactment risking injury to self or bed partner).
- **Mood**: depression and anxiety.

Investigations. Genetic testing is available for the chromosome 14 abnormality, but the process is fraught with practical, emotional and cultural issues. Before ordering any genetic testing, it is essential to get expert advice, ideally through the MJD Foundation.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses may include other spinocerebellar ataxias, Parkinson's disease and demyelination disorders. There are stories of people in remote communities being arrested for 'drunken behaviour' due to ataxia. Alcohol-related cerebellar ataxia is a diagnosis of exclusion.

PRINCIPLES OF MANAGEMENT

Multidisciplinary assessment and ongoing clinical management and social support are required including the following:

- **Initial discussion of the diagnosis and follow-up** is a critical time; it requires considerable clinical skill and experience. Health practitioners are strongly advised to seek guidance through the MJD Foundation before embarking on the process. In addition to documentation of the history and examination, there are the challenges of explaining the origin of the condition ("it's not your fault"), its likely progression and how the person will be supported over time. The diagnosis invariably brings up a host of family issues, including fear and shame. Some people do not want to know about the diagnosis; this is understandable. Anxiety and depression are common interlopers as the process gets underway, and thereafter.
- **Genetic Counselling for the patient & family** requires expertise unavailable in most remote communities and the MJD Foundation has the best resources.
- **Bladder management** includes assessment of post void volume with ultrasound, management of infections and incontinence with agents such as oxybutynin. Discuss with urologist, rehabilitation consultant & neurologist as required, and refer to the MJD Care Guidelines.
- **Bowel management** includes establishing a bowel routine in consultation with client/carers to suit needs and lifestyle including aperients and promotion of appropriate diet and fluids. Significant faecal unloading is often required (e.g. Movicol) with serial abdominal X-rays to monitor effect.
- **Skin care** includes identifying risks to skin integrity and preventing complications by using appropriate equipment in consultation with occupational therapists and physiotherapists.
- **Spasticity management** includes physiotherapy, antispasmodic agents such as baclofen, Botulinum injections and the treatment of underlying precipitants such as urinary tract infections or faecal impaction.
- **Dysphagia and dysarthria** are assessed and managed in consultation with a speech pathologist. This may include behavioural guidance to avoid aspiration, modified food and drink consistency.
- **Sleep disturbance management** will depend on the manifestations. For insomnia consideration can be given to sleep hygiene and melatonin. Referral to a sleep physician and sleep studies can be considered.
- **Neuropathic symptoms** such as pain, hyperalgaesia and allodynia may be managed with pregabalin or duloxetine. Back pain can be intractable & opiates are unhelpful.
- **Vision management** includes prisms prescribed by optometrists for diplopia and gaze holding exercises prescribed by physiotherapists.
- **Mobility and functional ability** need to be assessed and managed in consultation with occupational therapists and physiotherapists. Management may include antispasmodic agents, prescribed equipment, and preventative exercises.
- **Medication awareness.** People with MJD often do not tolerate certain medications well; they include those with anti-muscarinic properties (many antihistamines, anti-psychotics, anti-depressants and anti-Parkinson drugs), plus pregabalin and similar medications.
- **Vocational, recreational, housing and financial support.** Identify needs and refer to appropriate services such as:
  - > Regional aged and disability service
  - > MJD Foundation
  - > Allied health services
  - > Commonwealth Rehabilitation Service
  - > Centrelink
  - > Community disability legal service.

Machado-Joseph disease (MJD)

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the local neurology, rehabilitation or renal physician, or allied health services team.

MANAGEMENT GUIDELINES

RESOURCES

The MJD Foundation	■ Resources for health professionals ■ Resources for families	Available online

FURTHER READING

LaGrappe D, Massey L, Kruavit A, et al. Sleep disorders among Aboriginal Australians with Machado-Joseph Disease: Quantitative results from a multiple methods study to assess the experience of people living with the disease and their caregivers. Neurobiol Sleep Circadian Rhythms. 2022;12:100075.

Machado-Joseph Foundation. A guide to living with Machado Joseph Disease in Australia. Alyangula, NT: MJD Foundation; 2012.

Massey L, Gilroy J, Kowal E, et al. Aboriginal families living with MJD in remote Australia: questions of access and equity. Int J Equity Health. 2024;23(1):187.

Approach to planned immunosuppression

The population of Northern Australia has a high incidence of several infections of particular significance which occur opportunistically in people who are immunosuppressed.

IMMUNOSUPPRESSION IN NORTHERN AUSTRALIA

Evaluation and management of patients prior to induced immunosuppression is recommended to reduce the risk of serious manifestations of opportunistic infections such as disseminated *Strongyloides stercoralis* or tuberculosis, severe scabies infestation, hepatitis B flare and melioidosis.

It is also important to consider the risk of infections not specific to Northern Australia (e.g. *Pneumocystis jirovecii* pneumonia, herpes viruses, cytomegalovirus and fungal infections). Further advice regarding this is provided in the Therapeutic Guidelines.

IMMUNOSUPPRESSIVE AGENTS

Planned significant immunosuppression with any of the following should prompt baseline screening:

- Cancer chemotherapy.
- Stem cell transplantation.
- Corticosteroids in high or prolonged doses.
- Antibodies directed against B cell antigens (e.g. rituximab) or T cell antigens (e.g. alemtuzumab).
- New targeted therapies used in the treatment of leukaemia, lymphoma or myeloma.
- TNF inhibitors, antimetabolites (e.g. high dose methotrexate), anti-rejection therapy for solid organ transplantation, immune checkpoint inhibitors, cytokine and integrin inhibitors, tyrosine kinase inhibitors, cyclophosphamide.

There may also be specific risks associated with particular drugs, especially the newer biologic agents. The product information should always be reviewed for additional recommendations.

SCREENING

Baseline screening for the following opportunistic infections should occur prior to medically induced immunosuppression.

Tuberculosis. Tuberculin skin test (TST or Mantoux) is the test of choice where available through the CDC TB unit. If this is negative and clinical suspicion of latent TB infection (LTBI) is high or if TST is not available, an interferon gamma release assay should be carried out. Patients with a known post positive TST or interferon gamma release assay (IGRA) do not need repeat testing, however a review by the TB unit is required to establish that LTBI has been appropriately treated.

Hepatitis B. Hepatitis B serology with HBsAg, HBcAb and HBsAb. Hepatitis B DNA viral load should be ordered for patients with either a positive HBsAg or HBcAb.

Hepatitis C. If positive for hepatitis C antibodies, a HCV RNA viral load should be ordered.

HIV serology.

Melioidosis. If melioidosis serology is positive (≥ 1:40), swabs for melioidosis culture should be taken from the throat, rectum and any wounds. Blood, urine and sputum (if available) should be collected for melioidosis culture and a chest x-ray performed. If cultures are positive, full treatment is required in a hospital setting.

Strongyloidiasis. If strongyloides serology is positive or if eosinophilia is present, collect 3 stools for microscopy stating “immunosuppressed ?strongyloidiasis” on the pathology form.

Scabies and skin sores. Examine skin for evidence of scabies infection and pyoderma and treat if seen. In patients with recurrent boils, consider performing *Staphylococcus aureus* skin decolonization as outlined in the Therapeutic Guidelines.

Review of immunisation history. Consult the Australian Immunisation Handbook for full details of vaccination recommendations for immunosuppressed patients. It is important to remember that immunosuppressed patients generally cannot be given live vaccinations and there may be specific recommendations for particular drugs.

PRINCIPLES OF MANAGEMENT

Tuberculosis. All patients with a positive TST or IGRA test must be referred to the local public health unit for further assessment and consideration of treatment. A CXR and clinical assessment is done to rule out active TB. If active TB is excluded and a diagnosis of LTBI is established, treatment should be initiated through the TB unit, if possible, at least one-month duration before initiating immunosuppression.

Hepatitis B. All patients who are HBsAg positive and/or have detectable HBV DNA via PCR should be referred urgently to the Viral Hepatitis Clinic for consideration of antiviral therapy. Patients who are HBsAg and HBV DNA PCR negative but for whom HBcAb is positive and undergoing treatment with B-cell depleting therapy (e.g. rituximab), bone marrow transplantation, haemopoietic stem cell transplantation, or treatment for acute leukaemia or high-grade lymphoma should be referred for antiviral prophylaxis. Patients who are HBsAg and HBV DNA negative but HBcAb positive and not commenced on hepatitis B therapy should be monitored with HBV DNA PCR after one month and then at 3-monthly intervals until at least 6 months following immunosuppression. If HBV DNA becomes detectable, they should be commenced on antiviral therapy. Duration of prophylaxis and follow up should be determined by the Viral Hepatitis Clinic.

Melioidosis. If the patient has high positive serology of ≥ 1:320 but no clinical evidence of current disease, monitor closely and, for high risk patients give secondary prophylaxis with trimethoprim/sulfamethoxazole 160 mg+800 mg (in children, 4 mg/kg/day; dose calculated using the trimethoprim component) orally, daily, while immunosuppressed, year round and regardless of the season. High risk patients with negative melioidosis serology should receive trimethoprim/sulfamethoxazole for primary prophylaxis during the wet (monsoon) season. All patients with immunosuppression of any severity should be discouraged from soil and surface water contact and told to avoid being outside during any rainy and/or windy periods during the wet season. As a minimum, they should wear gardening gloves and footwear when coming into contact with mud or soil.

Strongyloidiasis. If stool microscopy or serology positive, treat with two doses of oral ivermectin 200 micrograms/kg separated by one week between doses, rounded up to the nearest 3mg, taken with a glass of milk/fatty meal). Do not give in pregnancy. Patients who are already immunosuppressed require four doses on days 1, 2, 15 and 16. All patients who have positive stool microscopy should be referred to Infectious Diseases for further assessment to exclude disseminated disease. In ongoing immunosuppression for patients living in or regularly visiting remote First Nations communities, repeat ivermectin dose every three months without further investigation. In an immunosuppressed patient with unexplained pulmonary infiltrates, fever, abdominal pain, Gram negative bacteraemia or septic shock with an unclear focus – always discuss with the Infectious Diseases team and perform microscopy on stool and sputum to look for strongyloides larvae.

Approach to planned immunosuppression

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call infectious diseases physician.

MANAGEMENT GUIDELINES

British Medical Journal	EULAR recommendations for the management of systemic lupus erythematosus: 2023 update	Available online

RESOURCES

Therapeutic Guidelines: Antibiotic	Primary Prophylaxis in immunocompromised adults without HIV infection	Available online

FURTHER READING

Nikpour Davis JS, Ferreira D, Paige E, et al. Infectious Complications of Biological and Small Molecule Targeted Immunomodulatory Therapies. Clin Microbiol Rev. 2020;33(3):e00035-e00019.

Case study — Immunosuppression

Kelvin is a 45-year-old man who is homeless and has been a heavy drinker for many years. He has type 2 diabetes and chronic obstructive pulmonary disease. Five years ago, he was run over by a car when he was lying on the road and had a splenectomy for a ruptured spleen. Kelvin rarely attends his local health service, and when given medications to treat his chronic conditions, he does not take them. Recently he has had several exacerbations of airways disease requiring prolonged courses of oral steroids. He takes these most of the time as he is distressed by dyspnoea.

You are surprised to see Kelvin in your waiting room as the morning clinic is about to commence. The clinic nurse approaches you with a worried frown and asks you to see Kelvin immediately as he looks really sick.

Kelvin says he could not sleep last night; he is short of breath and has pain in the right side of the chest on breathing. He can hardly stand up and says he has been "feeling crook" for a few days.

On examination he looks acutely unwell.

His pulse is 136, respiratory rate 38, BP 95/50, oxygen saturation 86% on room air and temperature 38°C. He has marked respiratory distress and decreased air entry across the right lung fields, and a possible pleural rub.

You call for an ambulance, but they cannot get to you for an hour.

What are the likely causes of Kelvin's presentation?

Kelvin has signs of sepsis probably due to right sided pneumonia. Other sites of infection should be considered and investigated.

What is your immediate management, given that the ambulance is at least an hour away?

Kelvin is hypotensive and hypoxic and needs resuscitation and antibiotics.

1. Provide oxygen via mask, adjust flow rate according to improvement in SpO_2.
2. Insert two IV lines.
3. Administer an initial bolus of IV fluid over the first 30 minutes. 10–15mL/kg of normal saline could be used as a guide.
4. Administer IV antibiotics. Use a regimen that will cover melioidosis as well as other likely organisms as per local guidelines.
5. Draw blood for cultures and other baseline tests (FBC, UEC, LFT, BGL, CRP, ESR).
6. Obtain urine sample for MC&S.

What risk factors does Kelvin have for developing severe infections?

Kelvin's diabetes, malnutrition, alcoholism, splenectomy, and prolonged courses of oral steroids have caused immunosuppression leading to increased risk of infection.

What infections are of particular concern for immunosuppressed patients in the tropical north of Australia?

Immunosuppressed patients are susceptible to all infections occurring in immunocompetent people as well as more opportunistic infections.

These include melioidosis, strongyloides, cryptococcus, TB, crusted scabies and activation of chronic hepatitis B.

What measures should be taken to prevent opportunistic infection when Kelvin is commenced on prolonged oral steroids?

When Kelvin receives more than 0.5mg/kg of prednisolone or equivalent, for more than 14 days, he should be assessed for prevention of opportunistic infections. Steps include:

1. Consider empirical strongyloides treatment when he begins prolonged prednisolone therapy as he lives in a high prevalence area.
2. Assess for and treat latent TB infection if detected.
3. Assess for scabies and associated bacterial infection. Treat pyoderma or scabies if detected.
4. Hepatitis B reactivation occurs with *potent* immunosuppression. This applies to those undergoing chemotherapy, transplant and potent therapy for autoimmune disease. An HBsAg positive patient taking oral steroids for chronic obstructive pulmonary disease should have regular monitoring of their infection.
5. Ensure Kevin is up to date with vaccinations.

What measures should be taken to protect splenectomised patients from infection?

People with anatomical or functional asplenia should be vaccinated against pneumococcus, meningococcus and *Haemophilus influenza*e B and receive an annual influenza vaccination.

See the Spleen Australia website for further details including prophylactic antibiotics, emergency plan and patient education.

Telehealth services can efficiently and effectively improve access to specialist advice for patients and health practitioners in rural and remote areas.

FURTHER INFORMATION

See *General Information* (page 240)

MANAGEMENT GUIDELINES

National		
Monash University	Spleen Australia	Available online
The Australasian Society for HIV, Viral Hepatitis and Sexual Health Medicine (ASHM)	B Positive: A guide for primary care providers	Available online
Australian Immunisation Handbook	Vaccination for people who are immunocompromised	Available online
Therapeutic Guidelines: Antibiotics	Primary prophylaxis in immunocompromised adults without HIV	Available online with subscription

FURTHER READING

Auguste P, Tsertsvadze A, Pink J, et al. Accurate diagnosis of latent tuberculosis in children, people who are immunocompromised or at risk from immunosuppression and recent arrivals from countries with a high incidence of tuberculosis: systematic review and economic evaluation. Health Technology Assessment. 2016;20(38):1-678.

Davis JS, Currie BJ, Fisher DA, et al. Prevention of opportunistic infections in immunosuppressed patients in the tropical top end of the Northern Territory. Communicable Diseases Intelligence Quarterly Report. 2003;27(4):526-532.

Petoumenos K, van Leuwen MT, Vajdic CM, et al. Cancer, immunodeficiency and antiretroviral treatment: results from the Australian HIV Observational Database (AHOD). HIV Medicine. 2013;14(2):77-84.

Asymptomatic eosinophilia

Asymptomatic eosinophilia is commonly identified on routine screening blood tests in otherwise well people in the Northern Territory.

EOSINOPHILIA IN NORTHERN AUSTRALIA

Causes include (but are not limited to) soil-transmitted helminths and scabies, which are endemic in many rural and remote areas of the Northern Territory due to factors such as household crowding, poverty and climate. Significant resources have previously been devoted to resource intensive investigations with limited evidence of clinical efficacy, and as such a pragmatic but safe standardised approach was deemed necessary. The full blood count is tested at least 2 yearly for all First Nations people aged over 15 years in Northern Territory clinics as part of the Adult Health Check, and routinely when patients are admitted to Northern Territory Hospitals.

AETIOLOGY AND PATHOGENESIS

There are many causes of eosinophilia which are well documented and explained in standard clinical practice guidelines. Eosinophilia in the Northern Territory is commonly caused by endemic soil-transmitted helminths, including *Trichuris trichiura* (whipworm) and *Strongyloides stercoralis*. Hookworm used to be very common but is now rarely found in the Top End. Scabies also can cause eosinophilia.

CLINICAL PICTURE

This guideline is for immunocompetent people who have eosinophilia but are otherwise found to be clinically healthy, with no unexplained weight loss, skin rash, lymphadenopathy and after consideration of the differential diagnoses.

DIFFERENTIAL DIAGNOSIS

All patients with eosinophilia in the Northern Territory should have a thorough skin check for clinical features of scabies, as scabies is endemic.

Differentials such as atopy, or drug reactions (such as clozapine-induced eosinophilia) should be considered. **If the eosinophil count is above 1.5 x109/L** the patient should be bought into clinic for semi-urgent review to screen for hyper-eosinophilic syndromes.

PRINCIPLES OF MANAGEMENT

Exclude scabies, atopy and urticaria, drug reaction and ensure patient is otherwise well.

If asymptomatic and eosinophilia of 0.7 - 1.5 x109/L and not pregnant

- Albendazole 400mg PO once a day for 3 days (200mg if child 10kg or less), to cover empirically for *Trichuris trichiura* and hookworm.

AND

- Ivermectin STAT 200 microgram/kg PO dose, to cover for *Strongyloides stercoralis* and *Trichuris trichiura*.

Note: The single dose ivermectin is a pragmatic approach supported by a RCT by Buonfrate and colleagues published in Lancet in 2019, suggesting non-inferiority of single dose ivermectin compared to multiple dose for *Strongyloides stercoralis* infection.

Ivermectin is dosed 200mcg/kg, rounded UP to the nearest 3mg. Give ivermectin with a glass of milk or a fatty meal. If ivermectin cannot be given, use BD albendazole with a repeat course after 7-14 days, dosed as per the Therapeutic Guidelines.

Delay ivermectin and albendazole treatment until after pregnancy.

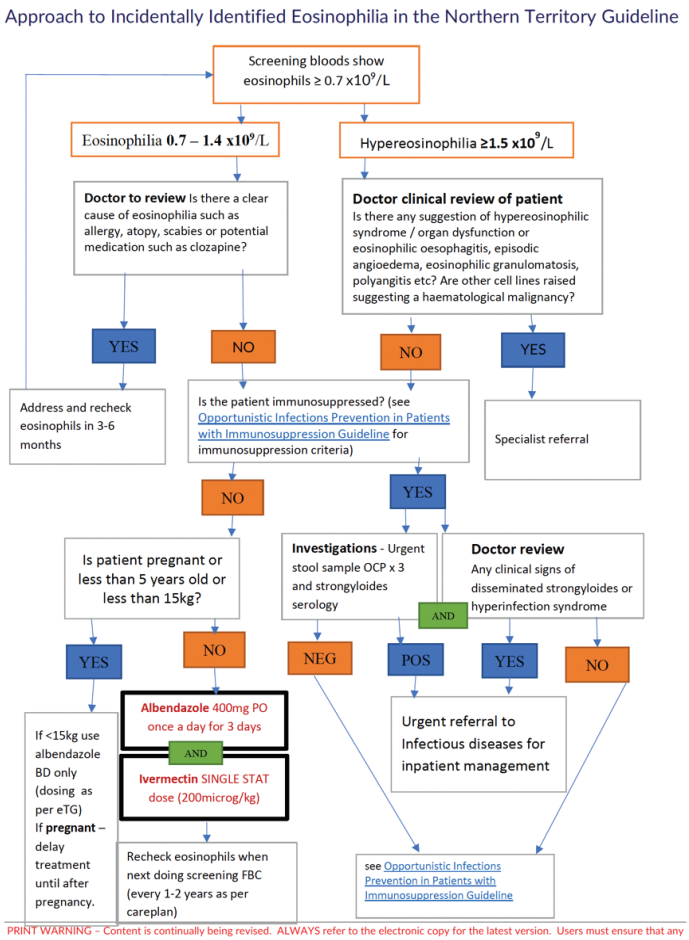


Figure 178: Management of incidentally identified eosinophilia in the Northern Territory
Source: Approach to Incidentally Identified Eosinophilia in the Northern Territory (Guideline), NT Health, 2023

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call duty medical officer (DMO) or general physician.

National Poisons information 13 11 26

MANAGEMENT GUIDELINES

Northern Territory		
Remote Primary Health Care Manuals (RPHCM)	CARPA Standard Treatment Manual – Asymptomatic eosinophilia	Available online

FURTHER READING

Ho YC, Lin HL. Continuation with clozapine after eosinophilia: a case report. Ann Gen Psychiatry. 2017;16:46.
Klion AD. Eosinophilia: a pragmatic approach to diagnosis and treatment. Hematology Am Soc Hematol Educ Program. 2015;(1):92-97.
Roufosse F, Weller PF. Practical approach to the patient with hypereosinophilia. J Allergy Clin Immunol. 2010;126(1):39-44.
Tefferi A. Blood eosinophilia: a new paradigm in disease classification, diagnosis, and treatment. Mayo Clin Proc. 2005;80(1):75-83.

Approach to type 2 diabetes mellitus (T2DM)

Type 2 diabetes mellitus (T2DM) occurs at much higher rates and at a younger age among First Nations peoples than non-Indigenous Australians.

DIABETES IN NORTHERN AUSTRALIA

T2DM is a leading cause of death among First Nations peoples in the Northern Territory and severely shortens life expectancy. With a prevalence as high as 40% in Central Australia, and concerningly high rates among younger people, rates among First Nations peoples in the Northern Territory are among the highest in the world. Early onset T2DM, coupled with kidney and cardiovascular complications, has devastating impacts on communities. If managed well, these complications can be prevented or ameliorated. However, diabetes mellitus is a heterogenous condition and must be managed in the context of individualised holistic care.

AETIOLOGY AND PATHOGENESIS

T2DM accounts for more than 85% of diabetes mellitus in Northern and Central Australia. It is characterised by peripheral insulin resistance and impaired islet cell insulin secretion in response to blood glucose. The liver does not help; it makes glucose (gluconeogenesis) and drives dyslipidaemia.

Other types of diabetes mellitus include type 1 which is due to autoimmune β-cell destruction, monogenic diabetes syndromes (such as neonatal diabetes and maturity-onset diabetes of the young), diseases of the exocrine pancreas (recurrent/chronic pancreatitis is the most common cause in the Northern Territory often called Type 3c), and drug- or chemical-induced diabetes (such as with glucocorticoid use, in the treatment of HIV/AIDS, or after organ transplantation). Gestational diabetes mellitus is diagnosed in pregnancy that was not clearly overt prior to gestation.

Classification is important for determining therapy, but some individuals cannot be clearly classified as having type 1 or type 2 at the time of diagnosis. The traditional paradigms of T2DM occurring only in adults and type 1 only in children are no longer accurate, as both occur in both age groups.

CLINICAL PICTURE

Risk factors. Family history of T2DM (especially mother with T2DM during pregnancy), ethnicity (First Nations Australian, Pacific Islander), overweight or obesity (particularly abdominal obesity), impaired glucose tolerance or prediabetes, medicines (e.g. corticosteroids, antipsychotics). For women: history of gestational diabetes.

Symptoms and signs. The diagnosis is often made incidentally or by routine screening such as a 715 Adult Health Check for First Nations Australians. Many people can be asymptomatic or have symptoms and signs of high blood glucose: polyuria, polydipsia, weight loss, frequent infections, visual changes, dental/oral issues, lethargy.

Screening (if asymptomatic).

Adults:

- First Nations adults should have an HbA1C checked yearly as part of the 715 Adult Health Check.
- Non-Indigenous adults should be screened every 3 years from 40 years of age using the Australian T2DM risk assessment tool (AUSDRISK).

Children and adolescents:

- See Figure 180 (It is important to note that none of the diagnostic criteria for diabetes mellitus have been specifically validated in youth and are all extrapolated from adult definitions).



Figure 179: Example of Acanthosis Nigricans: marker of insulin resistance, thick dark patches, usually in skin creases and folds, such as the sides and back of the neck, armpits, elbow pits, and groin.
Source: Angela Titmuss, Menzies School of Health Research

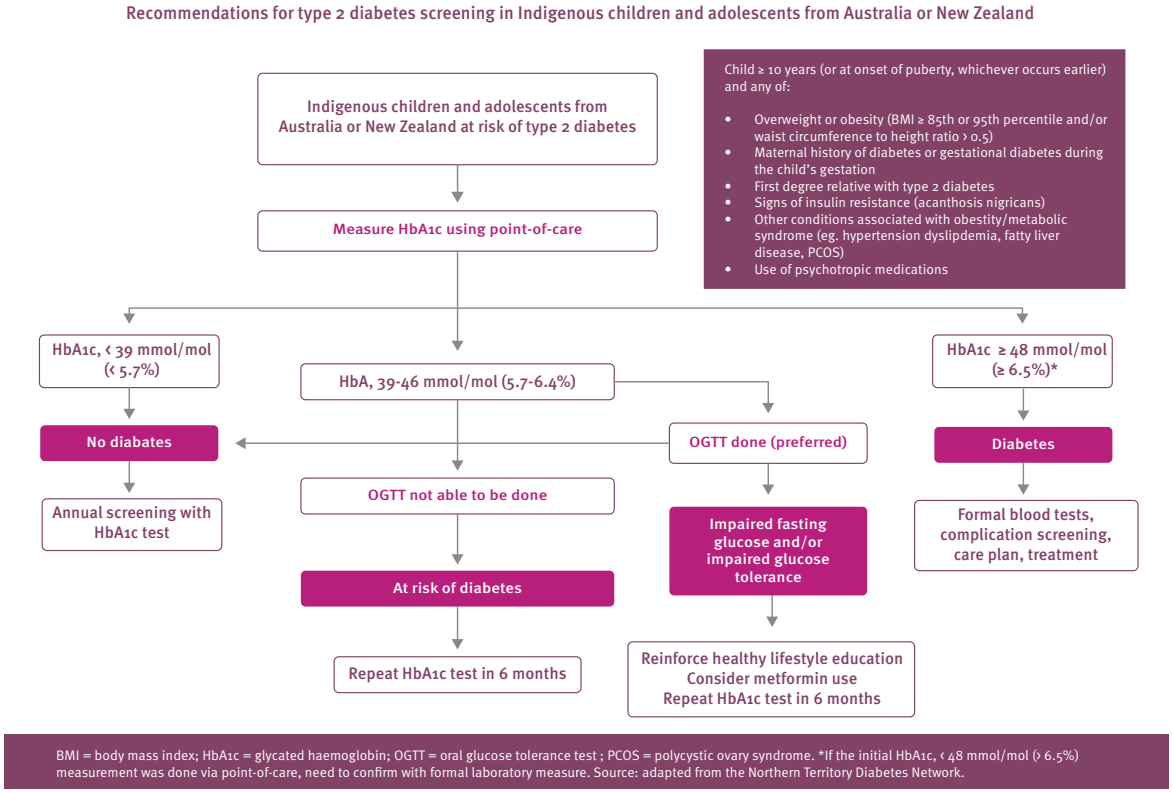


Figure 180: Recommendation for type 2 diabetes screening in Indigenous children and adolescents from Australia or New Zealand. SOURCE: Peña AS, et al. Screening, assessment and management of type 2 diabetes mellitus in children and adolescents: Australasian Paediatric Endocrine Group guidelines. Med. J. Aust. 2020;213: 30-43.

DIFFERENTIAL DIAGNOSIS

Type 1 diabetes mellitus. Always consider type 1 diabetes mellitus and if blood glucose levels >15 mmol/L check ketones. If type 1 is suspected, check diabetes associated antibodies and paired c-peptide and glucose.

If capillary blood ketone levels are elevated (≥0.6 mmol/L) this requires an urgent medical consult and consideration of hospital admission. People with type 1 always need insulin even when fasting.

Type 3c of the exocrine pancreas (DEP). If type 3c diabetes is suspected, usually in skinny people, check paired c-peptide and glucose and faecal elastase. Similar presentation and concerns as for type 1, however this is not an autoimmune condition, but instead involves endocrine and exocrine pancreatic dysfunction. As pancreatitis is a common presentation to Alice Springs and Royal Darwin Hospitals, it is important to consider this differential. Blood glucose management can be particularly difficult in DEP due to the dysfunction of both beta and alpha cells of the pancreas.

Approach to type 2 diabetes mellitus (T2DM)

Table 16: Diagnosis of T2DM

SYMPTOMS	Diabetes criteria	Pre-diabetes criteria*	Issues to consider
Symptomatic	Classic symptoms of hyperglycaemia and random plasma glucose ≥11.1mmol/L		Blood glucose meter readings cannot provide a diagnosis — readings need to be checked with accurate testing method, e.g. venous blood glucose
Asymptomatic			Diabetes diagnosis in any asymptomatic person needs to be confirmed using a different test from the same sample, or with a repeat test on the following day. If two different tests are performed and only one is high the test with the high result should be repeated — diagnosis can then be made based on the repeated test.
	HbA1c ≥48mmol/L (6.5%), or	HbA1c 5.7-6.4% (39-46mmol/mol), or	Do not use HbA1c if less than 4 months postpartum (after childbirth). Caution in interpreting HbA1c if person has a condition that affects red blood cell turnover. Reproducibility is higher for HbA1c than fasting glucose or OGTT in youth.
	Fasting plasma glucose ≥7mmol/L, or	Fasting plasma glucose 6.1-6.9mmol/L, or	Fasting plasma glucose correlates with liver insulin resistance. Fasting plasma glucose is affected by stress and illness. Correct and timely pre-analytical sample processing are important, especially in remote areas where laboratory analysis will be delayed, otherwise glucose levels reduce by 5-7% per hour in a sample due to glycolysis.
	2-hour plasma glucose ≥11.1mmol/L during OGTT	2-hour plasma glucose 7.8-11mmol/L during OGTT	Correct and timely pre-analytical sample processing required to minimize glycolysis.

*Prediabetes is not relevant for people with type 1 diabetes. The prediabetes threshold of 5.7–6.4% for adults is as per the American Diabetes Society guidelines. The term is also not yet universally accepted in children or youth. There are concerns that extrapolating adult data to youth is inaccurate in predicting risk of future T2D or diabetes complications.

PRINCIPLES OF MANAGEMENT

Good care looks after the whole person not just blood glucose. It is important to provide comprehensive self-management education that is patient centred, individualised, and culturally appropriate. This can be via referral to a Diabetes Educator, dietitian or First Nations Health Practitioner if available.

Prevention. The risk of developing T2DM can be reduced with a healthy diet, physical activity, and by maintaining a healthy weight. It should be acknowledged that these may not be possible for some people due to the structural and social determinants of health.

Treatment. Treatment targets: HbA1c is marker of glycaemia in the preceding 3 months. High HbA1c levels increase risk of complications and any decrease in HbA1c is useful.

- Assess for CVD risk, diabetes related eye disease, neuropathy, foot care and nephropathy and mental health.
- Females of reproductive age: assess for pregnancy and counsel regarding diabetes mellitus and pregnancy.

Adults:

- *Treatment target:* HbA1c 53mmol/mol (7%) or less OR if a history of severe hypoglycaemia, limited life expectancy or elderly — HbA1c 64mmol/mol (8%) OR individual target as per care plan.
- *Weight management:* 15% weight loss (for people with overweight or obesity) can result in improvement in glycaemia and other cardiovascular risk factors, including remission for a minority. This can be achieved through intensive dietary intervention (e.g. very low energy diet), medications (e.g. GLP-1 receptor agonists) or bariatric surgery (both public and private bariatric surgery are available in Darwin).
- *Medical treatment:* Refer to the Australian T2DM Glycaemic Management Algorithm.

Some specific notes regarding T2DM management:

- Metformin often causes gastrointestinal side effects, starting at the lowest dose and up- titrating can minimise these side effects, as can using extended-release formulation. If metformin stops for at least 4 weeks, consider restarting at the lowest dose. It is also a large tablet, and people may have difficulty swallowing it. It must be stopped if evidence of acute kidney injury or CKD with eGFR ‹15. Dose reduction is required in Stage 3 and 4 CKD.
- Sulphonlyureas cause weight gain and hypoglycaemia and are no longer routinely second line treatment for diabetes. They are useful if a person is not able to take insulin or has a contraindication to GLP-1 receptor agonists or SGLT-2 inhibitors.
- SGLT-2 inhibitors have evidence for benefit in CVD, especially for heart failure and CKD. These benefits exist for people with and without diabetes hence they are used very frequently. They should be used cautiously in people who are prone to dehydration and with food insecurity. If someone is not eating, they should be withheld and if unwell, finger-prick ketones must be checked.
- GLP-1 receptor agonists have evidence for benefit in CVD, CKD and weight loss. These benefits exist for people with and without T2DM hence they are used frequently. They are associated with significant weight loss and should not be used for people with low BMI and used in caution in the elderly and people with normal BMI. They should not be used if there is a recent history of pancreatitis.

Young people/children:

- Treatment target: HbA1c 48mmol/mol (6.5%) or less.
- There are limited licensed medications available for youth aged ‹18 years (currently only metformin and insulin). Use of GLP-1 receptor agonists and SGLT-2 inhibitors in this age group is currently ‘off-label’. Young people with T2DM may also respond differently to medications than adults. Careful consideration of their use should therefore include review by a paediatrician or paediatric endocrinologist. Young people with T2DM in northern Australia may have a range of phenotypes and intergenerational transmission and in-utero exposures are important. This may also affect medication choice and efficacy.

PBS criteria must be adhered to when prescribing medications for diabetes mellitus.

Complications assessment. Once hyperglycaemia occurs, people with all forms of diabetes mellitus are at risk for developing the same chronic complications, although rates of progression may differ. Assess for microvascular and macrovascular complications and manage accordingly.

Approach to type 2 diabetes mellitus (T2DM)

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the on-call physician or paediatrician.

MANAGEMENT GUIDELINES

National		
Australian and New Zealand Society for Paediatric Endocrinology and Diabetes	Screening, assessment and management of type2 diabetes mellitus in children and adolescents: Australasian Paediatric Endocrine Group guidelines	Available online
Royal Australian College of General Practitioners	Management of type 2 diabetes: a handbook for general practice	Available online
NT Primary Health Network (NT PHN)	HealthPathways - Diabetes	Available online (CLICK HERE to request access)

RESOURCES

National		
Australian Diabetes Society	■ Australian Type 2 Diabetes Glycaemic Management Algorithm ■ The Australian Type 2 Diabetes Risk Assessment Tool (AUSDRISK) | Australian Government Department of Health and Aged Care	Available online
Northern Territory		
Remote Primary Health Care Manuals (RPHCM)	■ CARPA Standard Treatment Manual - Chronic Conditions/Diabetes ■ Women's Business Manual - Diabetes in pregnancy	Available online

FURTHER READING

American Diabetes Association. 2. Classification and Diagnosis of Diabetes: Standards of Medical Care in Diabetes-2021. Diabetes Care. 2021;44(Suppl 1):S15-S33.

Hare MJL, Maple-Brown LJ, Shaw JE, et al. Risk of kidney disease following a pregnancy complicated by diabetes: a longitudinal, population-based data-linkage study among Aboriginal women in the Northern Territory, Australia. Diabetologia. 2023;66(5):837-846.

Hare MJL, Barzi F, Boyle JA, et al. Diabetes during pregnancy and birthweight trends among Aboriginal and non-Aboriginal people in the Northern Territory of Australia over 30 years. Lancet Reg Health West Pac 2020;1:100005.

Titmuss A, Davis EA, O'Donnell V, et al. Youth-onset type 2 diabetes among First Nations young people in northern Australia: a retrospective, cross-sectional study. Lancet Diabetes Endocrinol 2022;10(1):11-13.

Titmuss A, Korula S, Wicklow B, et al. Youth-onset Type 2 Diabetes: An Overview of Pathophysiology, Prognosis, Prevention and Management. Curr Diab Rep. 2024;24:183-195.

Vaccine preventable diseases and immunisation

Vaccination is one of the most effective and cost-effective public health interventions.

Global immunization efforts have saved an estimated 154 million lives over the past 50 years. Australia's National Immunisation Program (NIP) is a comprehensive, publicly funded program that provides free vaccines to eligible individuals across the lifespan. The NIP has been highly successful in controlling and eliminating several infectious diseases in Australia.

It is important for clinicians working in Northern Australia to be aware of local immunisation schedules, particularly for high-risk groups including First Nations people, children under 5, adolescents, pregnant women, the elderly, renal dialysis patients, those at medical risk and people who are immunocompromised. The immunisation schedules provide information on current NIP vaccination advice for children, adolescents and adults and are available on Immunisation program, NT Health. The list of vaccine preventable diseases included in this section is not comprehensive; a full list along with detailed information about the vaccines and recommendations for vaccination can be accessed from the Australian Immunisation Handbook.

VACCINE PREVENTABLE DISEASES IN NORTHERN AUSTRALIA

Pneumococcal disease. Pneumococcal vaccine is recommended for all children under 5 years, First Nations people aged ≥ 50 years and all children, adolescents and adults with risk conditions for pneumococcal disease.

Influenza. Influenza vaccination is recommended for all children 6 months to < 5 years old, and adults aged ≥65 years. Influenza vaccination is also recommended for all First Nations people from 6 months of age, pregnant women, healthcare workers, homeless people and for people with specified medical risk conditions. In Northern Australia, immunisation is recommended as soon as the year's influenza vaccine is available (March or April), and influenza vaccination should be promoted to unimmunised people throughout the year.

Hepatitis A. Hepatitis A vaccine is recommended and funded for First Nations children at 18 months and 4 years of age living in the Northern Territory, Queensland, South Australia and Western Australia. It is also recommended for people who live and work in rural and remote communities in northern Australia, including carers and early childhood educators; people with medical and behavioural risk factors, plumbers and sewage workers and people aged ≥1 year who travel to hepatitis A–endemic areas.

Hepatitis B. Hepatitis B vaccination is recommended and funded for all infants from birth, and for children aged up to 6 months. Hepatitis B vaccination is also recommended for First Nations people, those who are immunocompromised, people with specified medical risk conditions, and people who are at increased risk of exposure to hepatitis B because of their occupation, including healthcare workers.

Tuberculosis (TB). BCG (bacille Calmette–Guérin) vaccine is recommended for First Nations children <5 years in some parts of northern Australia, children born to parents from or travelling to countries with high TB incidence, children who are a household contact of a person with leprosy and healthcare workers with high risk of occupational exposure to TB.

Respiratory syncytial virus (RSV) infection. RSV vaccination is recommended and free via National Immunisation Program (NIP) funding for all pregnant women between 28-34 weeks to protect their babies from birth to about 6 months of age, which is when babies are most at risk of serious RSV disease. Adults aged ≥75 years and all First Nations adults aged ≥60 years and adults aged ≥60 years with risk factors for severe RSV disease are also recommended for RSV vaccine but currently not funded by the NIP. There is no distinct seasonal pattern in northern Australia, although more likely after the wet season (March - May).

Herpes Zoster (shingles vaccine). Shingles vaccine is available and free (NIP funded) for First Nations people aged ≥ 50 years and other Australians aged ≥65 years as a 2-dose schedule of Shingrix®, 2-6 months apart. People aged ≥18 years who are immunocompromised from disease or treatment including people on renal dialysis are also recommended to receive the vaccine.

Rotavirus infection. Rotavirus vaccine (oral) is recommended and funded for all infants <6 months of age; either 2 doses of Rotarix, at 2 and 4 months of age or 3 doses of RotaTeq, at 2, 4 and 6 months of age.

Human papillomavirus (HPV) disease. HPV vaccine is recommended for adolescents and young adults aged 9–25 years, people with severely immunocompromising conditions and men who have sex with men. In the NT, Gardasil 9 ® is available as a single dose for Year 7 students (12 years of age).

IMMUNISATION FOR HIGH-RISK GROUPS

Pregnant Women. Vaccination during pregnancy protects pregnant women against vaccine preventable diseases that may lead to pregnancy complications and generates antibodies that can cross the placenta and provide protection for babies in the first few months of life. Pregnant women are routinely recommended to receive influenza vaccine (anytime during pregnancy), pertussis-containing vaccine (dTpa - reduced antigen diphtheria-tetanus-acellular pertussis, between 20 – 32 weeks) and RSV vaccine (between 28 – 36 weeks). Women planning pregnancy should check their vaccination status for the following diseases and plan catch up vaccinations accordingly – hepatitis B, measles, mumps, rubella, varicella and COVID-19. Live vaccines should be given at least 4 weeks prior to pregnancy and are contraindicated during pregnancy.

Elderly people. Older adults are at higher risk for serious health problems caused by diseases like COVID-19, pneumococcal infection, RSV, influenza and herpes zoster and have reduced immune responsiveness to vaccination. Vaccination against these diseases is routinely recommended for older Australians, and a special formulation of influenza vaccine is routinely recommended for those 65 years of age and older.

Immunocompromised people. People who are immunocompromised have increased risk of disease and may need additional doses of some vaccines to optimise protection. People who are severely immunocompromised should receive annual influenza vaccine, pneumococcal vaccine, meningococcal vaccine and COVID-19 vaccine; those aged ≥18 years should receive 2-doses of herpes zoster vaccine, and those aged ≥ 60 years should receive RSV vaccine. Other inactivated vaccines as required should also be given such as those for human papillomavirus vaccine (HPV) and hepatitis B. Severely immunocompromised people should NOT receive live vaccines including BCG, oral typhoid, measles/ mumps/ rubella/ varicella, rotavirus, or yellow fever. People who are less severely immunocompromised may be able to receive live vaccines (not BCG or oral typhoid) and their case should be discussed with an immunisation expert. Household contacts should be fully vaccinated to protect the person who is immunocompromised. Household contacts can safely receive live vaccines.

Asplenia. People with an absent or dysfunctional spleen are at a lifelong increased risk of fulminant bacterial infection, especially with invasive pneumococcal disease. In addition to all routinely recommended vaccines, people with functional or anatomical asplenia are recommended to receive haemophilus influenza b (Hib) vaccine, influenza vaccine, meningococcal vaccine, pneumococcal vaccine, and RSV vaccine at 60 years and over. (See Spleen Australia)

Renal dialysis patients. Vaccines recommended for people on renal dialysis are pneumococcal, influenza, COVID, herpes zoster and Hepatitis B. People on renal dialysis have a lower response to hepatitis B vaccine and need a larger-than-usual doses of hepatitis B vaccine (dialysis strength or additional dose). Levels of antibody to hepatitis B surface antigen should be checked after the vaccination course.

The National Immunisation Program (NIP) funds many vaccines for all Australians, including high risk groups. Additional vaccines may be funded by individual states and territories. It is important to review local immunisation guidelines.

Vaccine preventable diseases and immunisation

FURTHER INFORMATION

See *General Information* (page 240)

TELEPHONE ADVICE

Contact the local CDC/ PHU immunisation team.

IMMUNISATION GUIDELINES

National		
Australian Immunisation Handbook	■ Pneumococcal ■ Influenza (flu) ■ Hepatitis A ■ Hepatitis B ■ Tuberculosis ■ Respiratory syncytial virus (RSV) ■ Zoster (herpes zoster) ■ Rotavirus ■ Human papillomavirus (HPV) ■ Vaccination for women who are planning pregnancy, pregnant or breastfeeding ■ Vaccination for people who are immunocompromised ■ Catch-up vaccination	Available online
Department of Health and Aged Care	National Immunisation Program	Available online

RESOURCES

National		
Department of Health and Aged Care	■ Australian Technical Advisory Group on Immunisation (ATAGI) ■ Vaccine Preventable Diseases and Vaccination Coverage in Aboriginal and Torres Strait Islander People, Australia, 2016-2019	Available online
National Centre for Immunisation Research and Surveillance	■ SKAI – Sharing Knowledge About Immunisation ■ Vaccination for our Mob ■ Surveillance of adverse events following immunisation in Australia 2022 ■ Annual Immunisation Coverage Report 2024 ■ Summary of National Surveillance Data on Vaccine Preventable Diseases in Australia, 2016-2018	Available online
AusVaxSafety	Vaccine safety in Australia AusVaxSafety summary reports	
Spleen Australia	Vaccinations	Available online
Northern Territory		
NT Health	■ Immunisation program ■ NT Immunisation Schedule Pneumococcal Vaccination ■ RSV Factsheet | NT Health	Available online

APPENDIX

Acronyms and Abbreviations
General Information
Index

Acronyms and abbreviations

ABLV	Australian bat lyssavirus
ACR	albumin creatinine ratio
AFB	acid fast bacilli
AFP	alpha foetoprotein
AIDS	acquired immunodeficiency syndrome
ALP	alkaline phosphatase
ALT	alanine aminotransferase
AMSANT	Aboriginal Medical Services Alliance of the Northern Territory
ANA	antinuclear antibody
anti-DNase B	antibodies against antideoxyribonuclease B
anti-dsDNA	anti-double stranded DNA
anti-HBc	hepatitis B core antibody
anti-HBe	hepatitis B envelope antibody
anti-HBs	hepatitis B surface antibody
anti-HCV	hepatitis C virus antibody
anti-Sm	anti-Smith antibody
AOM	acute otitis media
aPPT	activated partial thromboplastin time
APSGN	acute post-streptococcal glomerulonephritis
ARF	acute rheumatic fever
ASHA	Australasian Sexual Health Alliance
ASHM	The Australasian Society for HIV, Viral Hepatitis and Sexual Health Medicine
ASOT	anti-streptolysin O titres
ASPERN	Australian Sentinel Practices Research Network
ATAGI	Australian Technical Advisory Group on Immunisation
ATSIHP	Aboriginal and Torres Strait Islander Health Practitioner
BBV	blood-borne virus bd
bis die	twice a day
BFV	Barmah Forest virus
BGL	blood glucose level
BMI	body mass index
BP	blood pressure
BPG	benzathine benzylpenicillin G
C3	third component of complement
C4	fourth component of complement
CALD	culturally and linguistically diverse communities
CARPA	Central Australian Rural Practitioners Association
CARPA STM	CARPA Standard Treatment Manual
CDC	Centre for Disease Control
CDI	Communicable Diseases Intelligence
CDNA	Communicable Diseases Network Australia
CHB	chronic hepatitis B
CI	confidence interval
CK	creatine kinase
CKD	chronic kidney disease
cm	centimetre
cMRSA	community-acquired methicillin-resistant staphylococcus aureus
CMV	cytomegalovirus
CNS	central nervous system
COPD	chronic obstructive pulmonary disease
COVID-19	Coronavirus disease 2019
CrAg	cryptococcal antigen
CRP	c-reactive protein
CSF	cerebrospinal fluid
CSIRO	Commonwealth Scientific and Industrial Research Organisation
CSOM	chronic suppurative otitis media
CT scan	computed tomography
CVD	cardiovascular disease
CXR	chest X-ray
dB	decibel
DEET	N,N-diethyl-m-toluamide
DEP	diabetes of the exocrine pancreas
DNA	deoxyribonucleic acid
DOT	directly observed therapy
dsDNA	double stranded deoxyribonucleic acid
EBV	Epstein-Barr virus
ECG	electrocardiogram
EDTA	ethylenediaminetetraacetic acid
eGFR	estimated glomerular filtration rate
ELISA	enzyme-linked immunosorbent assay
ENA	extractable nuclear antigen
ENL	erythema nodosum leprosum

ENT	ear nose and throat
ESBL	extended-spectrum beta-lactamases
ESR	erythrocyte sedimentation rate
FBC	full blood count
FNQ	Far North Queensland
g	gram
GAA	growth assessment and action
GAS	group A streptococcus
GGT	gamma glutamyl transpeptidase
GLP-1	glucagon-like peptide-1
GP	general practitioner
GUD	genital ulcer disease
HAV	hepatitis A virus
Hb	haemoglobin
HbA1c	glycated haemoglobin (test)
HBeAg	hepatitis B envelope antigen
HBsAg	hepatitis B surface antigen
HBV	hepatitis B virus
HCC	hepatocellular carcinoma
HIV	human immunodeficiency virus
hr	hour
HR	heart rate
HRIG	human rabies immunoglobulin
HSV	herpes simplex virus
HTLV-I	human T cell lymphotropic virus
ICU	intensive care unit
ID	infectious disease
IgA	immunoglobulin A
iGAS	invasive Group A Streptococcus
IgG	immunoglobulin G
IgM	immunoglobulin M
IGRA	interferon-gamma release assay
IM	intramuscular
INR	international normalized ratio
IPD	invasive pneumococcal disease
IU	international units
IV	intravenous
JE	Japanese encephalitis
kg	kilogram
L	litre
LFT	liver function test
LTBI	latent tuberculosis Infection
MAC	mycobacterium avium complex
MC&S	microscopy, culture and sensitivity
mcg/dL	micrograms per decilitre
MDA	mass drug administration
MDRTB	multi-drug resistant tuberculosis
mg	milligram
min	minute
MJD	Machado-Joseph disease
mL	millilitre
mm	millimetre
mmol	millimole
Mpox	monkey pox virus
MRI	magnetic resonance imaging
MRSA	methicillin-resistant *staphylococcus aureus*
MSSA	methicillin-sensitive *staphylococcus aureus*
MTBC	*Mycobacterium tuberculosis complex*
MVE	Murray Valley encephalitis
NAAT	nucleic acid amplification test
NGO	non-government organisation
NGU	non-gonococcal urethritis
NIP	National Immunisation Program
NS1	non-structural protein 1
NSAID	non-steroidal ant-inflammatory drug
NT	Northern Territory
NTM	non-tuberculosis mycobacteria
OC&P	ova, cysts and parasites
OM	otitis media
PCCM	Primary Clinical Care Manual
PCR	polymerase chain reaction
PCV	packed cell volume
PHU	Public Health Unit
PID	pelvic inflammatory disease
po	taken orally
PPNG	penicillinase producing *Neisseria gonorrhoeae*
PPNG	penicillinase producing gonorrhoeae
PPV	pneumococcal polysaccharide vaccine

Acronyms and abbreviations

PVC	pneumococcal conjugate vaccine
qid	quater in die — 4 times a day
QLD	Queensland
RAT	rapid antigen test
RDT	rapid diagnostic test
RHD	rheumatic heart disease
RNA	ribonucleic acid
RPR	rapid plasma reagin test
RR	respiration rate
RRF	Ross River fever
RRV	Ross River virus
RSV	respiratory syncytial virus
RT–PCR	reverse transcription — polymerase chain reaction
S100	highly specialised drugs program on Pharmaceutical Benefits Scheme
SCA7	spinocerebellar ataxia Type 7
SE Asia	Southeast Asia
SGLT-2	sodium/glucose cotransporter 2
SLE	systemic lupus erythematosus
STAT	immediately
STI	sexually transmitted infection
Strep A	group A streptococcus
T	temperature
T2DM	type 2 diabetes mellitus
TB	tuberculosis
TGA	Therapeutic Goods Administration
TPHA	treponema pallidum haemagglutination
TST	tuberculin skin test
UEC	urea, electrolytes, creatinine
µL	microliter
UTI	urinary tract infection
WA	Western Australia
WBCT	whole blood clotting test
WCC	white cell count
WHO	World Health Organization
wt	weight

General information

TELEPHONE ADVICE

Hospitals	
Royal Darwin Hospital	**08 8922 8888**
Palmerston Regional Hospital	**08 7979 9200**
Alice Springs Hospital	**08 8951 7777**
Katherine Hospital	**08 8973 9211**
Tennant Creek Hospital	**08 8962 4399**
Gove District Hospital	**08 8987 0211**
Centre for Disease Control (CDC)	
Darwin	**08 8922 8044**
Alice Springs	**08 8951 7540**
Katherine	**08 8973 9049**
Tennant Creek	**08 8962 4259**
Nhulunbuy	**08 8987 0357**

Sexual Health Units	
Clinic 34	
— Darwin	**08 8999 2678**
— Alice Springs	**08 8951 7549**
— Katherine	**08 8973 9049**
— Tennant Creek	**08 8962 4259**
— Nhulunbuy	**08 8987 0357**
NT Syphilis register	
— Darwin	**08 8922 7818**
— Alice Springs	**08 8951 7552**
Alcohol and Other Drugs	
Darwin	**08 8922 8399**
Alice Springs	**08 8951 7580**

NOTIFIABLE CONDITIONS

National		
Communicable Diseases Network Australia (CDNA)	Australian notifiable diseases and case definitions	Available online
Northern Territory		
NT Centre for Disease Control (CDC)	Public Health and Notifiable Diseases	Available online

MANAGEMENT GUIDELINES

National		
Therapeutic Guidelines		Available online (Login required)
The Australian Immunisation Handbook		Available online
Northern Territory		
Remote Primary Health Care Manuals (RPHCM)	■ CARPA Standard Treatment Manual ■ Minymaku Kutju Tjukurpa — Women's Business Manual	Available online
Northern Territory Public Health Network (NT PHN)	HealthPathways	Available online (CLICK HERE to register)
NT Centre for Disease Control (CDC)	Resources and Publications	Available online

Index

Photographs are in *italic type*.

A
acid beetle 117
Acinetobacter baumannii pneumonia 53-54, 25-26
Acute post-streptococcal glomerulonephritis (APSGN) 13-14
Acute rheumatic fever (ARF) and rheumatic heart disease (RHD) 15-20
polyarthritis 15, 17-18
prolonged PR interval 20
Sydenham chorea 15-18
algae toxin — *see* Ciguatera poisoning 175
anaemia 123-125, 131-132, 198, 211
case study 205-207
Antimicrobial resistance xii-xiv, 142
Approach to planned immunosuppression 216-219
arboviral infections 51-56
Asymptomatic eosinophilia 223-224

B
Barmah Forest virus 18, 63-68
arthritis and rash 65
Bat borne viruses 81-84
bites
animal 50
bat 81-82 (*also see* Bat borne virus 81-84)
biting midges (culicoides) 120
crocodile 57-60
insect 3-4 (*also see* Other bites and stings 119-121)
Melioidosis cutaneous post animal bite 31
mites 71 (*also see* Scrub typhus 71-72)
snake 169-174
Box jellyfish 177-180
at beach 177
nematocysts – magnified 178
stings 177-180
Bronchiectasis and chronic suppurative lung disease 55-56, 39, 50-51, 91-92

C
care
culturally safe x-xi
trauma-informed xi
Case studies
anaemia 205-207
genital ulcer 163-164
immunosuppression 221-222
lower abdominal pain 165-166
pneumonia 25-27
recurrent diarrhoea 201-203
skin sores 11
urethral discharge 161-162
Chikungunya virus 63, 66
Chlamydia 77, **149-151**, 155,157 (*also see* Case study – Urethral discharge 161-162)
chronic suppurative lung disease 55-56
Ciguatera poisoning 175-176
community-acquired pneumonia 23, 25-27, 234, **53-54**
community MRSA 5, 7, 9, 26, 57
boil 7
osteomyelitis (scan) 8
pneumonia (scan) 9
pneumonia (x-ray) 8
psoas and paraspinal muscle abscesses (scan) 9
skin pustules and empyema 7
Cone snails 185-186
Crocodile bites and water-associated infections 57-60 (*also see* bites)
crocodile bite 58
treatment (table) 59
crusted scabies 11, 91-92, 101, **107-113**, 221
crusted scabies 111-112
Cryptococcal meningitis and pneumonia 99-100
Cultural safety in healthcare x-xi

D
Dengue fever 63-69, 72, 75
blanching rash 66
Diarrhoea in First Nations children 197-199
diarrhoea, recurrent — case study 201-203
diabetes mellitus 225-229
as risk factor, underlying condition 3, 5, 7, 9, 22, 25-26, 29, 37, 38, 43, 46, 50, 53, 109, 124, 221,
Donovanosis 136, **153-154**, 163-164

E
eosinophilia 124, 129, 132, 217, **223-224**
euproctis lutea (caterpillar) 120

F
fish poisoning 175-176
fractures 57
freshwater crocodile 57
freshwater crocodile bite –secondary Aeromonas hydrophila infection 58
freshwater mangrove itchy caterpillar stings 120

G
genital ulcer 136, 153
case study 163-164
Gonorrhoea 135, **139-142**, 149-150, 155, 157, 161-162, 165
disseminated infection 140
Group A Streptococcal (Strep A) infections 3-6, 9, 11, 13, 15, 16-18, 57
cellulitis 3
necrotising fasciitis 3
pyoderma (skin sore) 3

H
Head Lice 9, **115-116**
Hendra virus 83-84
Hepatitis A 85-86, 87, 231, 233
Hepatitis B 13, **87-90**, 92, 161, 163-166, 212, 217, 221-222, 231-233,
Hookworm 123-126, 129, 197, 205-207, 223
cutaneous larva migrans 125

Photographs are in *italic type*.

egg 124
hookworm 124
life cycle of hookworm (diagram) 123
Human immunodeficiency virus (HIV) 135, 155, **157-159**, 161, 163-164, 217
as risk factor 3, 37, 38, 50, 75, 87, 92, 99, 146
pneumocystis pneumonia (x-ray) 158
Human T-cell lymphotropic virus-I infection (HTLV-1) 91-93
as risk factor 107, 127

I
Influenza 7, 25, 26, 75, **95-96**, 193, 222, 231-233
Immunisation and vaccine preventable diseases 217, **231-233**
immunisation
asplenia 222, 232
catch-up 233
elderly people 232
hepatitis A 85, 231, 233
hepatitis B 89, 231, 233
herpes zoster (shingles vaccine) 231, 233
high-risk groups 232
human papillomavirus (HPV) disease 231, 233
immunocompromised people 232, 233
influenza 95, 231, 233
Japanese encephalitis 54
lyssavirus 70
meningococcal 232
pneumococcal disease 23-24, 231, 233
pregnant women 232, 233
renal dialysis patients 232
respiratory syncytial virus (RSV) 231, 233
rotavirus 197, 231, 233
tetanus 185
tuberculosis 231, 233
immunosuppression 89, 127, 129, **216-219**
as risk factor 26, 29, 50, 105,
case study 221-222
Irukandji syndrome 181-182
post bell contact 181
with severe sweating and piloerection 181

J
Japanese encephalitis 63-69
fatal Japanese encephalitis with intense bilateral thalamic inflammation on MRI 67

K
Kava use 187-188
as risk factor 26, 29,
dermopathy 188
Kunjin virus 163-169

L
Leprosy 45-47
as differential diagnosis 51, 101
nerve damage 46
skin 45
Leptospirosis 73-76
as differential diagnosis 4, 72, 85, 87
conjunctival injection 73
pulmonary haemorrhage (x-ray) 74
severe pneumonia (x-ray) 74
lower abdominal pain — case study 165-166
lymphogranuloma venereum 136, 149, 163
lyssavirus 181-182

M
Machado Joseph disease 213-215
malaria 63-69
mango sap rash 120
mantoux test 38, 39, 46, 217
measles 232
measles rash 23
medication
supply xvi
shortage xvi-xvii
Melioidosis 23, 25-26, **29-35**, 38, 57, 92, 187, 212, 217, 218, 221
as differential diagnosis 39, 43, 51, 72, 75,
Burkholderia pseudomallei growing on agar plate 30
cutaneous 31
osteomyelitis (scan, x-ray) 33
osteomyelitis and discharging ulcer 31
pneumonia (scan, x-ray) 33
pneumonia lung autopsy 32
prostatic abscesses (scan) 33
splenic abscesses 32
meningococcal septicaemia rash 23
methicillin resistant staphylococcus aureus (MRSA) 5, **7-10**, 26, 57,
also see community MRSA
Mosquito borne diseases 63-69
Murray Valley encephalitis 63-69
Mycobacterium ulcerans 51, 52
ulcers 49

N
Nocardia 43-44
cutaneous 32, 43
pneumonia (x-ray) 34
severe pneumonia (x-ray) 43
Non-tuberculous mycobacteria 39, **49-52**

O
Onychomycosis 105-106
nail tinea with trichophyton rubrum 105
orb-weaving spider 119
Otitis media 21, 22, **193-196**

P
Paederus australis (acid beetle) 117-118
beetle 117
kissing lesion 117
palm thorn lacerations 121
pelvic inflammatory disease (PID) 135, 140, 141, 149, 150, 155,

Index

Photographs are in *italic type*.

see More than just a pain — case study of lower abdominal pain 165-166
penicillin 5-6
rheumatic fever prophylaxis 15, 18
sensitive zone 141
petrol sniffing 189-190
Pneumococcal disease 21-24, 231, 232
pneumococcal pneumonia — serotype 3 (x-ray) 22
pneumococcal septicaemia — post splenectomy 22
with varicella pneumonia (x-ray) 23
pneumonia
acinetobacter baumannii pneumonia 53-54
case study 25-27
cryptococcus meningitis and pneumonia 99-100
melioidosis 25-29
pneumococcal 29-34
staphylococcal 7
Post-streptococcal syndromes
acute post-streptococcal glomerulonephritis 13-14, 113
acute rheumatic fever 4, 15-20
rheumatic heart disease 15-20

Q

R
rabies 81-84
rheumatic fever, acute 4, **15-20**
rheumatic heart disease (RHD) **15-20**
Ross River virus 18, 63-69

S
Scabies 3, 4, 9, 11, 13, 18, 91, 92, **107-113**, 211, 217, 221, 222, 223
as differential diagnosis 101, 129, 163,
ARF post-infected scabies 15
crusted 111
female with eggs 107
infected 108
Scrub typhus 71-72
eschar 71
sea urchin spines 119
Sexually transmitted infections — overview 135-137
chlamydia 149-151
donovanosis 153-154
gonorrhoea 139-142
human immunodeficiency virus (HIV) 157-159
syphilis 143-148
trichomoniasis 155-156
see also Case study — Urethral discharge 161-162
see also Case study — Genital ulcer 163-164
see also Case study — More than just a pain lower abdominal pain 165-166
shingles 9, 157, 231
shingles (herpes zoster) post-immunosuppression 9
skin sores — case study 11-12
see also Group A Streptococcal (GAS) infections 3-6
Snakebites 169-174
coagulopathy 172
death adder fang marks 172
fang marks 172
myoglobinuria 173
snakes 170
sporotrichosis 32
Staphylococcal infections 7-10
stings, and bites 119-121
itchy caterpillar 120
fleckeri 177, 178
Stonefish 183-184
streptococcal infections
Group A Streptococcal infections 3-6
Pneumococcal disease 21-24
see also post-streptococcal syndromes
Strongyloidiasis 91, 92, **127-130**
life cycle *S. stercoralis* (diagram) 128
Syphilis 6, 135-136, **143-147,** 153, 161,
as differential diagnosis 75, 92, 163-165
secondary syphilis 144
Systemic lupus erythematosus 109, **211-212**

T
Tinea corporis 101-104
on hands and nails 102
pigmentation 102
tinea corporis, lesions 102
Trachoma 77-80
corneal opacity (CO) 79
trachomatous inflammation - follicular (TF) 78
trachomatous inflammation - follicular and intense (TF + TI) 78
trachomatous scarring (TS) 79
trachomatous trichiasis (TT) 79
Trichomoniasis 141, 150, **155-156**
Trichuriasis (whipworm) 131-132, 206
egg 132
life cycle of *T. trichiura* (diagram) 131
Tuberculosis (TB) 30, **37-41**, 46, 212, 217, 218,
as differential diagnosis 18, 25, 33, 43, 51, 99
lung x-ray 39
lymphadenitis 38
vaccine 231, 233
Type 2 diabetes mellitus, approach to 225-229

U
urethral discharge 139, 141, 149,
case study 161-162

V
vaccines (*also see* immunisations)
Japanese encephalitis 67
Leptospirosis, in animals 75
pneumococcal 21, 23, 24, 26
rabies 81-82,
Vector borne diseases
see Barmah forest virus

Photographs are in *italic type*.

see Chikungunya
see Dengue fever
see Japanese encephalitis
see Malaria
see Murray Valley encephalitis
see Ross River virus
see Scrub typhus
see West Nile (Kunjin strain) virus
see Zika virus
Vibrio vulnificus bullous cellulitis 58
Volatile substance misuse 189-190

W
West Nile (Kunjin strain) virus 63-60
whipworm (*see trichuriasis)*

X

Y

Z
Zika virus 63-69

ISBN 978-1-922770-09-7

