TROPICAL HEALTH ORIENTATION MANUAL

for health practitioners in Northern Australia

Tropical Health Orientation Manual

FOR HEALTH PRACTITIONERS IN NORTHERN AUSTRALIA

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

2020

CENTRE FOR REMOTE HEALTH
ALICE SPRINGS, 2020

Images provided by Professor Bart Currie (Menzies School of Health Research) are available for teaching purposes as part of the National Health and Medical Research Council (NHMRC) funded 'Improving Health Outcomes in the Tropical North: A multidisciplinary collaboration (Hot North)', grant identification number 1131932: www.hotnorth.org.au

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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...

Foreword

The Tropical Health Orientation Manual (THOM) provides an introductory overview with background knowledge on clinically important conditions prevalent in tropical Australia. THOM complements existing quality management guidelines and recommends resources for further exploration.

Health practitioners unfamiliar with tropical diseases and new to rural and remote communities need be prepared for practice in Northern Australia. This preparation includes awareness of locally important tropical health conditions which impact on the high burden of disease in the diverse tropical north, and being alert to the diagnoses that are not to be missed. Managing an unwell patient in an isolated community where specialist resources may be hundreds of kilometres away is challenging. THOM provides accessible, foundational information about many of the clinically significant conditions found in tropical Australia, identified by local clinicians as core curriculum for Northern Australia.

Tropical Health in the Top End was developed and published by the Top End Division of General Practice in 2003 in response to the need identified by remote general practitioners. Health professionals with expertise and passion for their topics collaborated to share their knowledge and are gratefully acknowledged for laying the foundations for this new edition. This second edition updates existing topics and has been broadened to include Northern Australia due to the similarities across the tropical regions, and in response to requests from clinicians.

THOM is now associated with and complements the Remote Primary Health Care Manuals (RPHCM) which also continue to be developed by health practitioners, for health practitioners working in rural and remote locations.

Working and living in communities in Northern Australia provides a unique opportunity to discover the tropical north with its rich cultural heritage and spectacular scenery ranging from the coast to the desert. The warm weather and wide, open spaces entice one to be closer to nature and embrace the tropical biodiversity, including unseen microorganisms.

We hope you will use this resource as a key stepping-stone to discover more about the fascinating and challenging tropical health conditions in Northern Australia.

Wendy Page MBBS, FRACGP, FACRRM, MPH&TM, GCHPE
Chairperson
Tropical Health Orientation Manual Working Group

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

In memory of Meredith Arnold who inspired us with dedication, commitment, and a vision of improved health for all.

Australian Government
Department of Health

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How to use this manual

This manual contains an introduction to 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, educational resources and further reading.

In addition to the disease summaries several clinical case scenarios are presented. These 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 up-to-date references should always be used for management decisions about individual patients.

The appendices contain:

- A list of acronyms that occur frequently throughout the book
- A list of local resource agencies for health and community services in tropical Australia.

Please note: As advised, contributors to the Tropical Health Orientation Manual have respectfully employed the term Aboriginal and Torres Strait Islander in preference to Indigenous. Non-Indigenous refers to non-Aboriginal and Torres Strait Islanders.

An electronic version of this manual is available via the Remote Primary Health Care Manuals website: **https://remotephcmanuals.com.au** and Northern Territory HealthPathways: **https://nt.healthpathwayscommunity.org**

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SECTION 1 — BACTERIAL INFECTIONS AND POST-STREPTOCOCCAL SYNDROMES

Group A Streptococcal (GAS) infections

Community surveys demonstrate that up to half of Aboriginal and Torres Strait Islander children may have streptococcal skin sores at any one time.

GROUP A STREPTOCOCCAL INFECTIONS IN NORTHERN AUSTRALIA

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

The 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 Aboriginal and Torres Strait Islander children may have streptococcal skin sores at any one time.

AETIOLOGY AND PATHOGENESIS

GAS can be part of normal skin and throat flora. Newly introduced and invasive strains spread quickly particularly in unhygienic and overcrowded conditions. Streptococci cause infection by adherence and invasion, and subvert the host immune system by resisting phagocytosis, lysing host immune cells, and degrading immunoglobulins.

CLINICAL PICTURE

Risk factors for streptococcal infection include overcrowding, poor hygiene practices and conditions of immune compromise such as the extremes of age, HIV, diabetes, steroid 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 streptococci and are often secondary to scabies.

Lesions can follow minor trauma and insect bites but are also commonly secondary to scabies. These lesions are usually seen on the limbs whereas in other populations lesions on the face are more common.

Typical symptoms for Group A Streptococcal (GAS) pharyngitis include a sudden-onset fever with sore throat with signs of an inflamed pharynx and tonsils, purulent tonsillar exudate, and cervical lymphadenopathy. In contrast to the high prevalence of impetigo, GAS pharyngitis appears to be uncommonly seen in many remote Aboriginal and Torres Strait Islander communities.

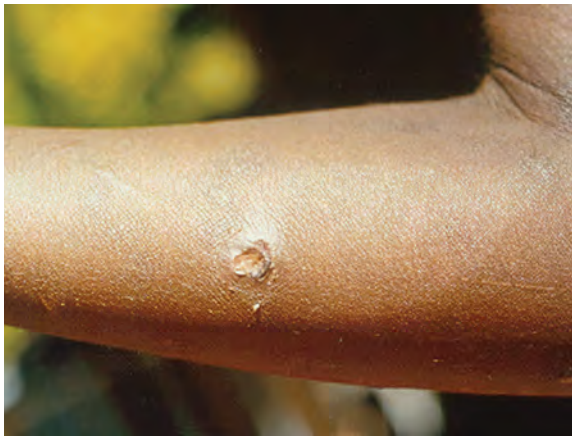

Figure 1: Streptococcal pyoderma (skin sore)
Source: Bart Currie — Menzies School of Health Research

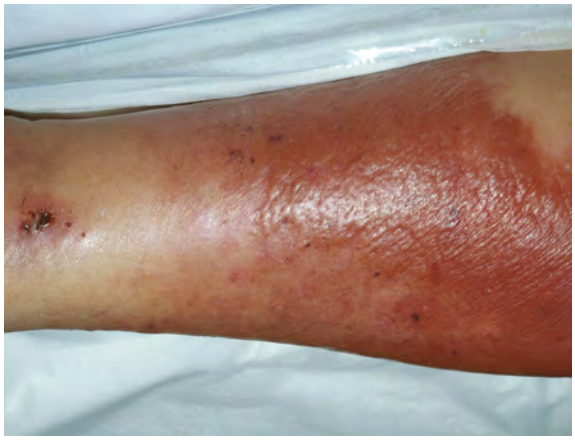

Figure 2: Streptococcal cellulitis
Source: Bart Currie — Menzies School of Health Research

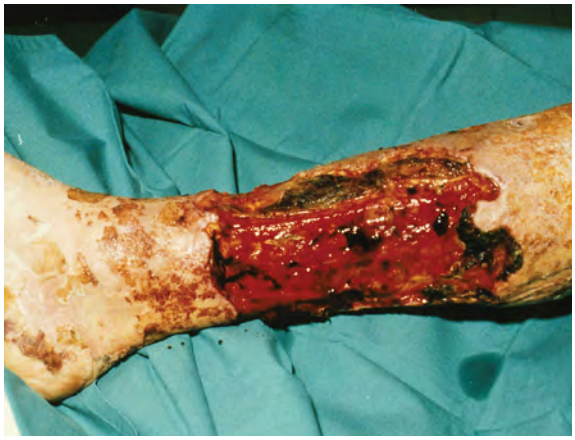

Figure 3: Streptococcal necrotising fasciitis
Source: Bart Currie — Menzies School of Health Research

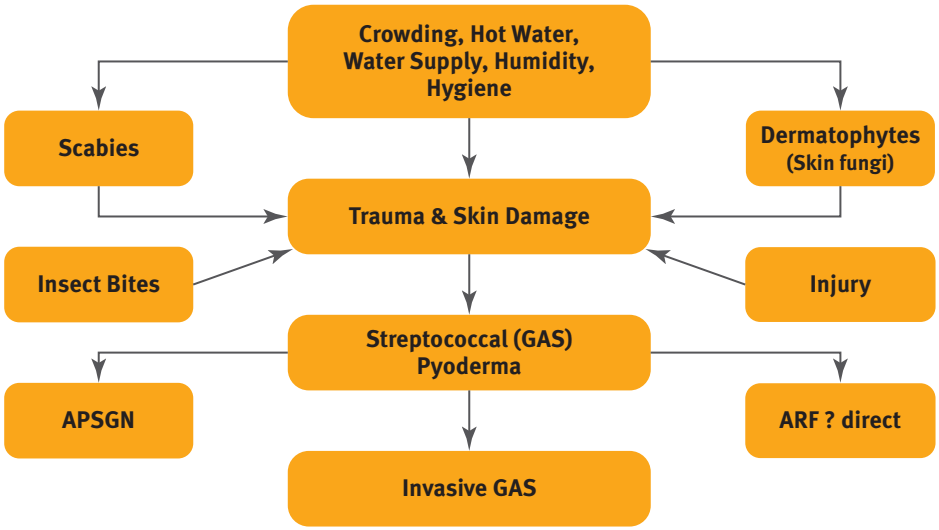

Figure 4: Factors affecting skin disease in Aboriginal and Torres Strait Islander communities.
GAS = Group A streptococcus; APSGN = acute post-streptococcal glomerulonephritis; ARF = acute rheumatic fever.

Source: Currie BJ, Carapetis JR. Skin infections and infestations in Aboriginal communities in northern Australia. The Australasian Journal of Dermatology. 2000 Aug;41(3):139-143.

Cellulitis appears as a swollen, red, warm area of skin. The most common causes of cellulitis are GAS and *Staphylococcus aureus*.

Although rare, it is important to recognise warning symptoms and signs for the more severe forms of GAS 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 GAS with hypotension and organ dysfunction, and may accompany necrotising fasciitis.

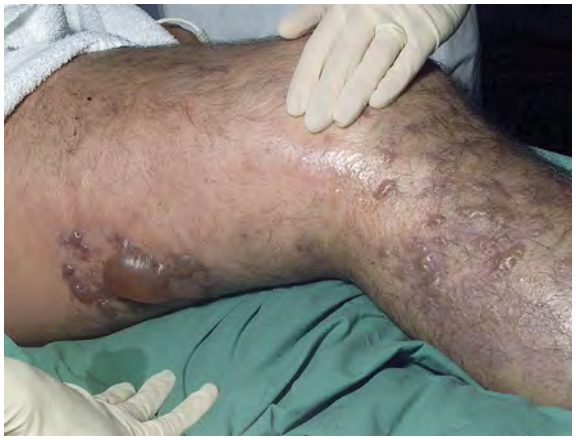

Figure 5: Vibrio vulnificus bullous cellulitis (differential diagnosis)
Source: Bart Currie — Menzies School of Health Research

Investigations. Take swabs of impetigo lesions if they are not improving with standard therapy. Swabs for sore throat should be performed to look for GAS. If acute rheumatic fever is suspected: take swabs of the throat, bloods for anti-streptococcal antibodies (anti-streptolysin O titre [ASOT] or anti-DNAse B), CRP, FBC, ESR, perform an ECG and consult a medical specialist. If post-streptococcal glomerulonephritis is suspected perform a dipstick urine analysis (and albumin creatinine ratio if positive) 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 (*p9*). A number of other bacteria and some fungi can cause skin infections.

Group A Streptococcal (GAS) 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. The aim of treatment in both skin sores and sore throat is to eradicate the streptococcus and minimise the risk of transmission to other children and post-streptococcal diseases. Serious infections and post-streptococcal syndromes such as glomerulonephritis and rheumatic fever require hospitalisation. Underlying conditions such as diabetes and scabies should be managed.

It is important to discuss regular skin washing to reduce the risk of streptococcal skin infection. Identify problems that may prevent daily washing including problems with health hardware (eg 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.

Cellulitis requires a course of anti-streptococcal antibiotics. For more severe infections, antibiotic cover for staphylococcal infection is also required, as per your local guidelines. Severe infections such as sepsis, necrotising fasciitis and toxic shock syndrome require immediate discussion with a medical specialist, urgent antibiotic treatment and urgent surgical assessment.

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.

A NOTE ABOUT PENICILLIN

Penicillin remains the mainstay of treatment for streptococcal 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 (eg *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 that extends the half-life of benzyl penicillin. It provides therapeutic blood levels for up to 24 hours, and is commonly used in the treatment of mild community-acquired pneumonia as a daily injection for five days.

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 commonly used in the treatment of syphilis, infected skin sores, and as a 4-weekly injection to prevent recurrent rheumatic fever.

> Invasive Group A Streptococcal (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.

FURTHER INFORMATION

TELEPHONE ADVICE

Contact infectious disease physician, general physician in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines see *Appendix p200*.

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
National Healthy Skin Guideline	Recognising and Treating Skin Infections: A visual clinical handbook	Available online
Northern Territory		
Centre for Disease Control (CDC)	Publications/Protocols: ■ Public health management of invasive Group A streptococcal infection ■ Healthy Skin Program — Guidelines for Community Control of Scabies, Skin Sores and Crusted Scabies ■ NT Guidelines for the Control of Acute Post-Streptococcal Glomerulonephritis ■ Notifiable diseases	Available online
Kimberley		
Kimberley Aboriginal Medical Services (KAMS)	Clinical Protocols/Guidelines — Acute Post-Streptococcal Glomerulonephritis	Available online
North Queensland		
Queensland Health	Communicable Disease Control Guidance — Invasive Group A Streptococcal Disease	Available online

EDUCATIONAL RESOURCES

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

KEY REFERENCES AND FURTHER READING

May PJ, Tong SYC, Steer AC, Currie BJ, Andrews RM, Carapetis JR, 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 Mar;24(3):280-93. DOI: 10.1111/tmi.13198

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Bowen AC, Tong SY, Chatfield MD, Carapetis JR. The microbiology of impetigo in Indigenous children: associations between Streptococcus pyogenes, Staphylococcus aureus, scabies, and nasal carriage. BMC Infectious Diseases. 2014 Dec 31;14:727. DOI: 10.1186/s12879-014-0727-5

Bowen AC, Tong SY, Andrews RM, O'Meara IM, McDonald MI, Chatfield MD, Currie BJ, Carapetis JR. Short-course oral co-trimoxazole versus intramuscular benzathine benzylpenicillin for impetigo in a highly endemic region: an open-label, randomised, controlled, non-inferiority trial. Lancet. 2014 Dec 13;384(9960):2132-40. DOI: 10.1016/S0140-6736(14)60841-2

Boyd R, Patel M, Currie BJ, Holt DC, Harris T, Krause V. High burden of invasive group A streptococcal disease in the Northern Territory of Australia. Epidemiology and Infection. 2016 Apr;144(5):1018-27. DOI: 10.1017/S0950268815002010

Andrews RM, Kearns T, Connors C, Parker C, Carville K, Currie BJ, Carapetis JR. A regional initiative to reduce skin infections amongst Aboriginal children living in remote communities of the Northern Territory, Australia. PLoS Neglected Tropical Diseases. 2009 Nov 24;3(11):e554. DOI: 10.1371/journal.pntd.0000554

Walker MJ, Barnett TC, McArthur JD, Cole JN, Gillen CM, Henningham A, Sriprakash KS, Sanderson-Smith ML, Nizet V. Disease manifestations and pathogenic mechanisms of Group A Streptococcus. Clinical Microbiology Reviews. 2014 Apr;27(2):264-301.DOI: 10.1128/CMR.00101-13

Staphylococcal infections

Staphylococcal infections tend to be purulent, acute in onset, and to 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 associated with aspiration 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 is increasing across Australia — community methicillin-resistant *S. aureus* (MRSA).

AETIOLOGY AND PATHOGENESIS

S. aureus forms part of the normal flora on skin and mucous membranes. It is pathogenic because it produces toxins, can breach epithelial surfaces and resist 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) has caused major infection control problems in hospitals, and now also constitutes more than 25% of all community-acquired staphylococcal infections in some regions. Widespread use of antibiotics in remote communities may have selected and favoured the emergence and dissemination of MRSA.

CLINICAL PICTURE

Risk factors. These include close contact with people carrying virulent strains of the bacteria, renal disease and diabetes.

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 to localise rapidly. They can be recurrent.

Staphylococcus aureus (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 can be a cause of secondary bacterial pneumonia following influenza.

Investigations. Cultures from infected sites should be taken if the patient is unwell, to help 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.

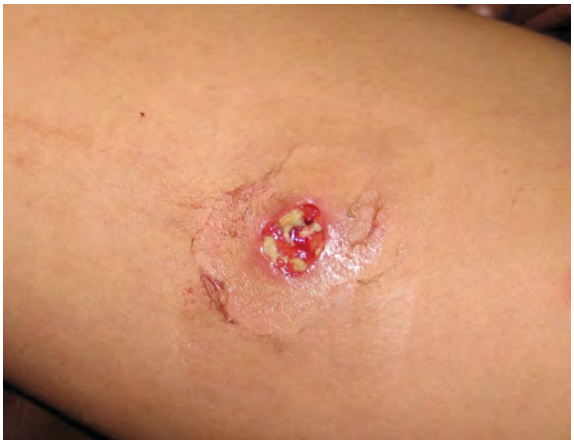

Figure 6: Community MRSA boil
Source: Bart Currie — Menzies School of Health Research

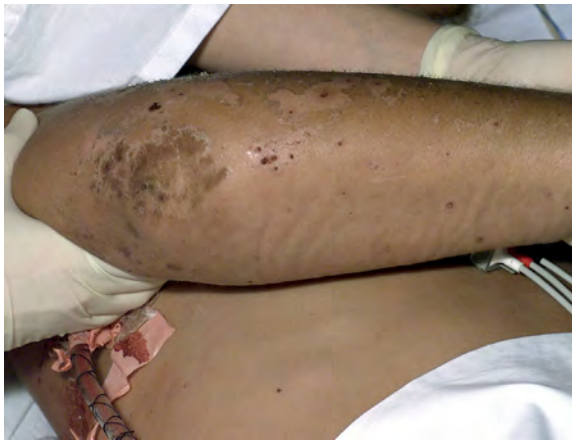

Figure 7: Severe Community MRSA skin pustules and empyema
Source: Bart Currie — Menzies School of Health Research

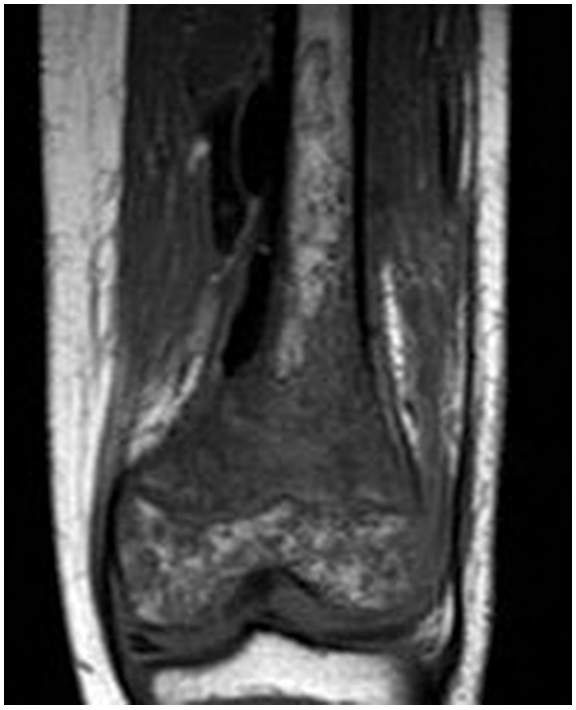

Figure 8: MRI Community MRSA femoral osteomyelitis eight year old
Source: Bart Currie — Menzies School of Health Research

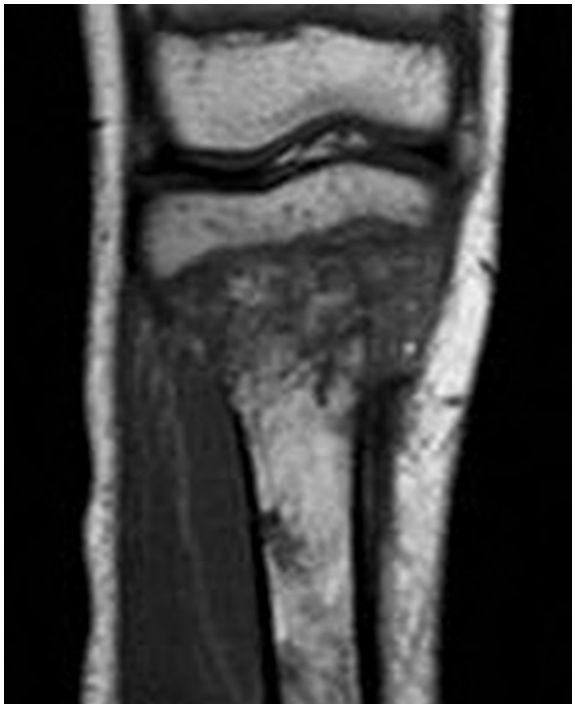

Figure 9: MRI Community MRSA tibial osteomyelitis eight year old
Source: Bart Currie — Menzies School of Health Research

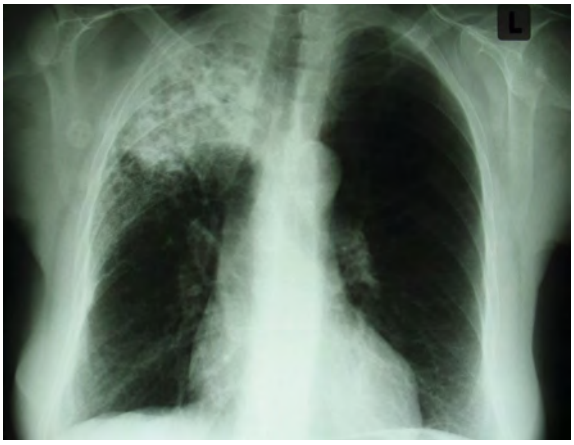

Figure 10: Community MRSA pneumonia
Source: Bart Currie — Menzies School of Health Research

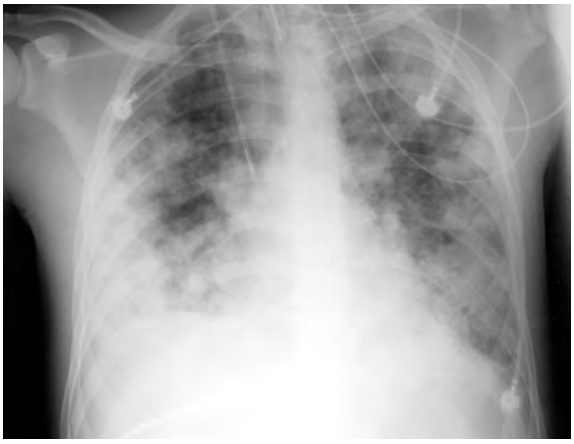

Figure 11: Severe Community MRSA necrotising pneumonia
Source: Bart Currie — Menzies School of Health Research

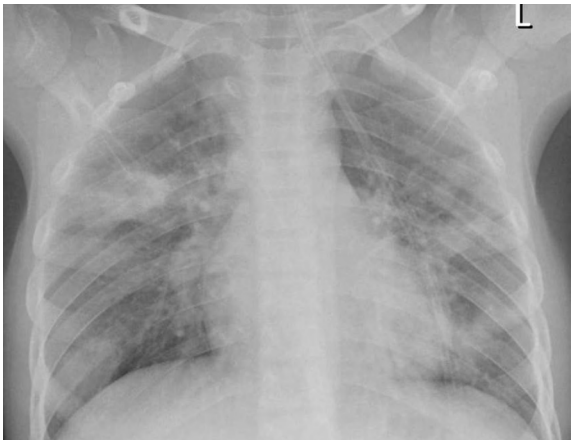

Figure 12: Nine year old with severe ST93 Community-MRSA pneumonia post-influenza A
Source: Bart Currie — Menzies School of Health Research

Staphylococcal infections

DIFFERENTIAL DIAGNOSIS

Staphylococcal infection should be differentiated from other causes of bacterial skin or respiratory infection. Look for signs of scabies (*p93*). Infections of the occipital scalp are usually associated with infestations of head lice (*p99*). The differential diagnosis for pneumonia is described in *Case study — Pneumonia (p45)*. 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 infections is a key tenet of management. Boils and skin abscesses should be drained and many cases 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 (eg 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), eg flucloxacillin, and methicillin-resistant *S. aureus* (MRSA), eg vancomycin. Less severe infections can be treated initially with agents directed at methicillin-susceptible *S. aureus*, such as flucloxacillin/dicloxacillin or cephalexin, with attention to surgical management and close monitoring of response.

Recurrent staphylococcal abscesses may warrant a staphylococcal eradication program including skin washes and nasal mupirocin (see *Therapeutic Guidelines* or regional guidelines for details). Underlying conditions (eg scabies, diabetes) should be treated. Superficial skin sores are predominantly streptococcal and therefore usually respond to therapies directed at Group A streptococcus (GAS) alone.

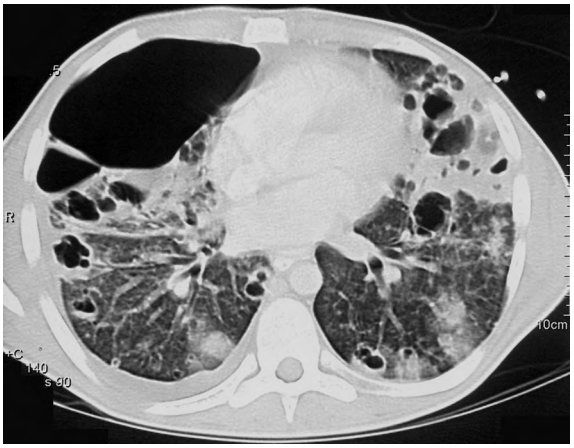

Figure 13: Severe Community MRSA necrotising pneumonia
Source: Bart Currie — Menzies School of Health Research

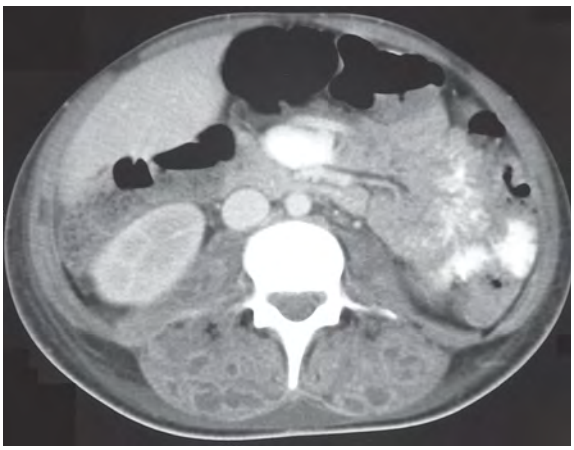

Figure 14: Severe Community MRSA psoas and paraspinal muscle abscesses
Source: Bart Currie — Menzies School of Health Research

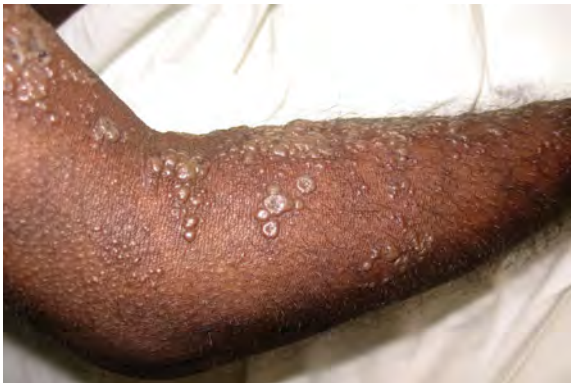

Figure 15: Shingles (herpes zoster) post immunosuppression (differential diagnosis)
Source: Bart Currie — Menzies School of Health Research

FURTHER INFORMATION

TELEPHONE ADVICE

Contact infectious disease physician, general physician, CDC/PHU in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines see *Appendix p200*.

National		
The Australian Healthy Skin Consortium	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		
Centre for Disease Control (CDC)	Healthy Skin Program — Guidelines for Community Control of Scabies, Skin Sores and Crusted Scabies	Available online
Kimberley		
Kimberley Aboriginal Medical Services (KAMS)	Clinical Protocols/Guidelines — Skin infection	Available online
North Queensland		
Queensland Health	Health Conditions Directory. Bacterial infections: Staphylococcus aureus infection	Available online
Children's Health Queensland Hospital and Health Service	Recurrent boils: Guidelines for management and Staphylococcal decolonisation	Available online

EDUCATIONAL RESOURCES

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

KEY REFERENCES AND FURTHER READING

May PJ, Bowen A. Tong SYC, Steer AC, Currie BJ, Andrews RM, Carapetis JR, Bowen AC. Treatment, prevention and public health management of impetigo, scabies, crusted scabies and fungal skin infections in endemic populations: a systematic review. Tropical Medicine and International Health. 2019 Mar;24(3):280-293. DOI: 10.1111/tmi.13198

Engelman D, Hofer A, Davis JS, Carapetis JR, Baird RW, Giffard PM, Holt DC, Tong SY. Invasive Staphylococcus aureus Infections in Children in Tropical Northern Australia. Journal of the Pediatric Infectious Diseases Society.2014 Dec;3:304-11. DOI: 10.1093/jpids/piu013

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Case study — Skin sores

The baby-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.

How will you treat Miranda and what underlying issues should you consider?

Miranda's poor growth and skin condition both require attention. You investigate and work out an action plan according to local guidelines. The plan includes weekly weighs at the clinic, medical reviews and ongoing education and support for her family. You list 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 streptococcal carriage. As the distribution of her sores is typical of scabies infestation you also treat with permethrin cream and make arrangements to examine or treat her household contacts for scabies.

What are some of the reasons for recurrent skin sores?

- Challenges keeping clean because of poor health hardware such as toilets, showers or taps that don't work
- Recurrent scabies
- Overcrowding
- Undiagnosed case of crusted scabies in the house — assess all household contacts for this.

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

- Acute post-streptococcal glomerulonephritis
- Anaemia of chronic disease
- Secondary bacteraemic sepsis
- Acute rheumatic fever (but maybe not directly from skin bacteria)
- Poor growth in children.

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

- Address health hardware and overcrowding
- Run a ‘Healthy Skin’ program to reduce scabies and streptococcal infections
- Community based growth promotion initiatives (eg Strong Women Strong Babies Strong Culture program)
- Coordinated and integrated public health and clinical programs focused on scabies eradication.

FURTHER INFORMATION

TELEPHONE ADVICE

Contact community paediatrician or child health team in your local jurisdiction.

Acute Post-streptococcal Glomerulonephritis (APSGN)

Associated with scabies, skin sores and crowded living conditions, and is a risk factor for chronic renal disease in Aboriginal and Torres Strait Islander adults.

ACUTE POST-STREPTOCOCCAL GLOMERULONEPHRITIS

Acute post-streptococcal glomerulonephritis occurs as individual sporadic cases, and in epidemics that can involve numerous children within one household or several related communities. In the past very large outbreaks were common but are now rare. Rates are significantly higher in Aboriginal and Torres Strait Islander children compared with non-Indigenous children.

AETIOLOGY AND PATHOGENESIS

Acute post-streptococcal glomerulonephritis (APSGN) is an acute renal inflammatory syndrome occurring two to three weeks after a cutaneous or pharyngeal infection with certain 'nephritogenic' strains of Group A streptococci (GAS) (*p3*). It is thought to be immunologically mediated but the exact mechanism is unclear. In Northern Australia it is usually associated with skin rather than throat infections. As infection with a newly introduced streptococcal strain spreads rapidly, there may be geographic and temporal clustering of cases, often with multiple cases in one household. Periodically there are larger outbreaks.

CLINICAL PICTURE

Risk factors. Children under school age are those most at risk, although it can occur at any age. Geographic or temporal proximity to or contact with known cases, and recent streptococcal pharyngitis or skin infection are risk factors.

Symptoms. It is likely that some cases are so mild they are subclinical; if symptoms do occur they may include lethargy, anorexia, headache and dull back pain. In an outbreak there are usually about three asymptomatic cases for each clinical case.

Signs. Facial or limb oedema, hypertension (diastolic BP more than 80 if 13 years or younger, or more than 90 if older) and visible haematuria.

Investigations. Urine dipstick may show protein and more than 2+ haemoglobin or blood. Microscopy may show dysmorphic red blood cells 10/mm³, white blood cells, and hyaline, granular and red blood cell casts. Blood tests usually demonstrate elevated ESR, mild normochromic normocytic anaemia, and elevated creatinine. Anti-streptococcal antibodies (anti-streptolysin O titre [ASOT] or Anti-DNase B) titres are usually high, consistent with recent streptococcal infection. However, as streptococcal infections are common in Aboriginal and Torres Strait Islander children and antibodies are often elevated, a reduced C3 complement level usefully confirms the presence of an acute post infective process, and occurs in almost all cases of APSGN.

DIFFERENTIAL DIAGNOSIS

Acute post-streptococcal glomerulonephritis (APSGN) should be differentiated from other causes of glomerulonephritis such as membranoproliferative glomerulonephritis, IgA nephropathy, Systemic lupus erythematosus, and Henoch-Schönlein disease. Evidence of recent streptococcal infection is important in diagnosis and APSGN usually begins to resolve within 1–2 weeks following presentation, unlike these other often more chronic conditions.

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 close observation of weight and blood pressure. More severe cases, particularly children, should be hospitalised for management of fluid balance and hypertension. Mortality is rare (less than 1%) and unlike rheumatic fever, recurrences are uncommon.

Prevention. Reducing risk of incidence of further cases is important. All suspected cases in the NT should be notified by telephone to the Centre for Disease Control (CDC), and the *Northern Territory Guidelines for the Control of Acute Post-Streptococcal Glomerulonephritis* should be followed. In WA notification should be made to the Kimberley Population Health Unit (PHU) and the *Acute Post-Streptococcal Glomerulonephritis — Kimberley control Measures* followed. In Queensland the condition is not notifiable, but it is recommended that the NT guidelines be followed.

All close contacts aged 12 months to less than 17 years, and all other contacts with skin sores, should receive a single dose of benzathine benzylpenicillin IM to eradicate the virulent streptococcal strain. Washing, especially of children, should be promoted and scabies

treated. If more than one case is suspected, liaise with the CDC or PHU. Two unrelated clinical cases in a week or three in a month suggests an outbreak, and community wide screening may be required. This is coordinated in consultation with CDC or PHU who will provide advice (and where possible resources) regarding contact screening, testing and follow-up.

Acute post-streptococcal glomerulonephritis is a notifiable condition to be reported by CLINICIANS in the Northern Territory and Western Australia. Report both suspected and confirmed cases to the local Centre for Disease Control/ Public Health Unit.

FURTHER INFORMATION

TELEPHONE ADVICE

Contact infectious disease physician, general physician, paediatrician, CDC/PHU in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines see *Appendix p200*.

Northern Territory		
Centre for Disease Control (CDC)	■ NT Guidelines for the Control of Acute Post-Streptococcal Glomerulonephritis ■ Healthy Skin Program — Guidelines for Community Control of Scabies, Skin Sores and Crusted Scabies ■ Notifiable diseases	Available online
Remote Primary Health Care Manuals (RPHCM)	CARPA Standard Treatment Manual — Post-streptococcal Glomerulonephritis	Available online
Kimberley		
Kimberley Aboriginal Medical Services (KAMS)	■ Clinical Protocols/Guidelines — Acute Post-streptococcal Glomerulonephritis ■ Notification of infectious diseases and related conditions	Available online
North Queensland		
Queensland Health	Primary Clinical Care Manual — Acute Post-streptococcal Glomerulonephritis	Available online

EDUCATIONAL RESOURCES

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

KEY REFERENCES AND 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. The American Journal of Tropical Medicine and Hygiene. 2018 Dec;99(6):1643-8. DOI: 10.4269/ajtmh.18-0093

Hoy WE, White AV, Tipiloura B, Singh G, Sharma SK, Bloomfield H, et al. The multideterminant model of renal disease in a remote Australian Aboriginal population in the context of early life risk factors: lower birth weight, childhood post-streptococcal glomerulonephritis, and current body mass index influence levels of albuminuria in young Aboriginal adults. Clinical Nephrology. 2015;83(7 Suppl 1):75-81.

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Eison TM, Ault BH, Jones DP, Chesney RW, Wyatt RJ. Post-streptococcal acute glomerulonephritis in children: clinical features and pathogenesis. Pediatric Nephrology. 2011 Feb;26(2):165-80. DOI: 10.1007/s00467-010-1554-6

Boyd R, Markey P. Continued high incidence of acute post-streptococcal glomerulonephritis in the Northern Territory. The Northern Territory Disease Control Bulletin. 2013;20(2):1-4.

Mansfield K, Gunn J, Wilson N, Scott L. Acute post-streptococcal glomerulonephritis and opportunistic trachoma screening in an Indigenous community in the Northern Territory, 2011. The Northern Territory Disease Control Bulletin. 2011. 18(4):8-12.

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

Aboriginal and Torres Strait Islander people in Northern Australia have amongst the highest published incidences of acute rheumatic fever in the world.

ACUTE RHEUMATIC FEVER AND RHEUMATIC HEART DISEASE IN NORTHERN AUSTRALIA

Over the past five decades, major changes have occurred in the epidemiology of acute rheumatic fever (ARF) resulting in a decline of the disease in developed countries. However, in the developing world, and in Indigenous groups within developed countries, it remains common and causes significant morbidity and mortality from valvular heart disease.

Many cases of ARF are not detected resulting in missed opportunities for secondary prevention and a relatively high proportion of patients who have established valvular heart disease at the time of first diagnosis. A high index of suspicion must be maintained when assessing any patient presenting with joint pain, even if there is a history of trauma.

AETIOLOGY AND PATHOGENESIS

ARF is an 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)*. It was believed that rheumatic fever followed *S. pyogenes* infection of the throat. The paradox in Aboriginal and Torres Strait Islander communities is that throat carriage rates of *S. pyogenes* are usually low and that symptomatic pharyngitis is uncommon, whereas streptococcal pyoderma is very common — see *Group A Streptococcal Infections p3*.

Rheumatic Heart Disease (RHD) is a complication of acute rheumatic fever (ARF) which involves dysfunction of the heart valves leading to regurgitation and stenosis, and may result in early heart failure and death at a young age. It is more likely to develop and/or progress with each recurrence of ARF.

CLINICAL PICTURE

Risk factors include socio-economic disadvantage, overcrowding and repeated streptococcal infections. ARF typically occurs in school aged children internationally however new cases of ARF also occasionally occur in middle aged people. The diagnosis should be considered in patients of any age.

Symptoms and signs can be very subtle. Consider the diagnosis in any Aboriginal or Torres Strait Islander patient presenting with arthralgia, arthritis, fever of unknown origin, a new murmur, new heart failure, or involuntary movements (chorea). Chorea may occur without any other features of ARF.

Since 2012, the diagnostic criteria for ARF in Australia include different criteria for people at high risk. This includes Aboriginal and Torres Strait Islanders living in rural or remote settings, and Aboriginal and Torres Strait Islanders, Māori and Pacific Islanders living in crowded urban settings. These ARF criteria based on risk category were adopted internationally in 2015. The 2020 Australian guideline for prevention, diagnosis and management of acute rheumatic fever and rheumatic heart disease (3rd edition) is now available: **https://www.rhdaustralia.org.au/arf-rhd-guideline**

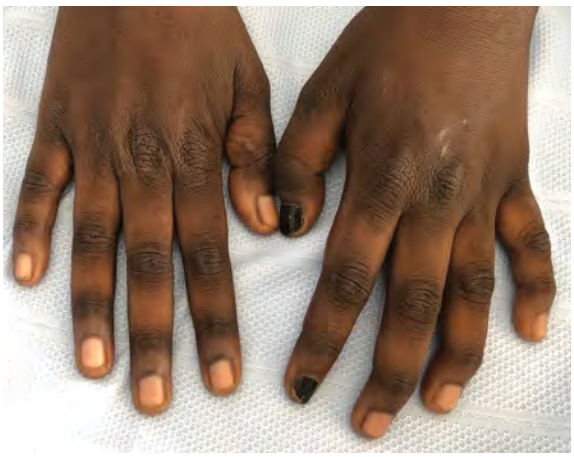

Figure 16: Acute Rheumatic Fever presenting as polyarthritis
Source: Bart Currie — Menzies School of Health Research

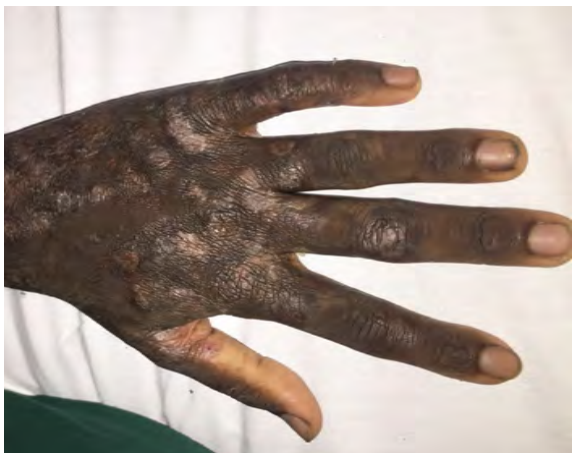

Figure 17: Sixteen year old with ARF post-infected scabies
Source: Bart Currie — Menzies School of Health Research

Table 1: 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: 2020 Australian guideline for prevention, diagnosis and management of acute rheumatic fever and rheumatic heart disease (3rd edition)

Table 2: Recommended duration of secondary prophylaxis

Diagnosis	Definition	Duration of prophylaxis
Possible ARF (no cardiac involvement)	Incomplete features of ARF with normal echocardiogram and normal ECG§ throughout ARF episode	12 months (then reassess)
Probable ARF	Highly suspected ARF with normal echocardiogram	Minimum of 5 years after most recent episode of probable ARF, or until age 21 years (whichever is longer)
Definite ARF (no cardiac involvement)	ARF with normal echocardiogram and normal ECG§ throughout ARF episode	Minimum of 5 years after most recent episode of ARF, or until age 21 years (whichever is longer)
Definite ARF (with cardiac involvement)	ARF with carditis or RHD on echocardiogram, or with atrioventricular conduction abnormality on ECG§ during ARF episode	According to relevant RHD severity
Borderline RHD (≤20 years of age only)	Borderline RHD on echocardiogram without a documented history of ARF	Not usually recommended¶
Mild RHD††	Mild RHD on echocardiogram, or atrioventricular conduction abnormality on ECG§ during ARF episode	■ If documented history of ARF: Minimum of 10 years after the most recent episode of ARF, or until age 21 years (whichever is longer)
Moderate RHD†† §§	Moderate RHD on echocardiogram	■ If documented history of ARF: Minimum of 10 years after the most recent episode of ARF or until age 35 years (whichever is longer)
Severe RHD§§ ¶¶	Severe RHD on echocardiogram, OR Previous valve repair or prosthetic valve replacement	■ If documented history of ARF: Minimum of 10 years after the most recent episode of ARF or until age 40 years (whichever is longer)

† All people receiving secondary prophylaxis require a comprehensive clinical assessment and echocardiogram prior to cessation. Risk factors including future exposure to high Strep A burden environments need to be considered.

‡ Echocardiography may be more frequent based on clinical status and specialist review.

§ Normal ECG means no atrioventricular (AV) conduction abnormality during the ARF episode – including first-degree heart block, second degree heart block, third-degree (complete) heart block and accelerated junctional rhythm.

¶ Secondary prophylaxis may be considered in some circumstances, including family preference, family history of rheumatic heart valve surgery, or suspected retrospective history of ARF. If prophylaxis is commenced, consider ceasing after 1-3 years if no history of ARF and if echocardiographic features have resolved or not progressed to definite RHD.

†† Prophylaxis may be considered for longer in women considering pregnancy who live in high-risk circumstances for ARF.

	Conditions for ceasing prophylaxis†	Timing of echocardiography after cessation‡
	■ No signs and symptoms of ARF within the previous 12 months ■ Normal echocardiogram	At 1 year
	■ No probable or definite ARF within the previous 5 years ■ Normal echocardiogram	At 1, 3 and 5 years
	■ No probable or definite ARF within the previous 5 years ■ Normal echocardiogram	At 1, 3 and 5 years
		Medical review and repeat echocardiogram at 1, 3 and 5 years after diagnosis
■ If NO documented history of ARF and aged <35 years‡‡: Minimum of 5 years following diagnosis of RHD or until age 21 years (whichever is longer)	■ No probable or definite ARF within the previous 10 years, no progression of RHD ■ Stable echocardiographic features for 2 years	At 1, 3 and 5 years
■ If NO documented history of ARF and aged <35 years‡‡: Minimum of 5 years following diagnosis of RHD or until age 35 years (whichever is longer)	■ No probable or definite ARF within the previous 10 years ■ Stable echocardiographic features for 2 years	Initially every 12 months
■ If no documented history of ARF†††: Minimum of 5 years following diagnosis of RHD or until age 40 years (whichever is longer)	Stable valvular disease/cardiac function on serial echocardiogram for 3 years **OR** Patient or family preference to cease due to advancing age and/or end of life care	Initially every 6 months

‡‡ If diagnosed with mild or moderate RHD aged ≥35 years (without ARF), secondary prophylaxis is not required.

§§ Rarely, moderate or severe RHD may improve on echocardiogram without valve surgery. In these cases, the conditions for ceasing prophylaxis can change to follow the most relevant severity category. For instance, if moderate RHD improves to mild on echocardiogram, recommendations for mild RHD can then be instigated.

¶¶ Risk of ARF recurrence is low in people aged ≥40 years, however, lifelong secondary prophylaxis is usually recommended for patients who have had, or are likely to need, heart valve surgery.

††† If diagnosed with severe RHD aged ≥40 years (without ARF), specialist input is required to determine the need for secondary prophylaxis.

Source: 2020 Australian guideline for prevention, diagnosis and management of acute rheumatic fever and rheumatic heart disease (3rd edition)

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

Investigations should include: FBC, ESR, CRP, anti-streptococcal antibodies (anti-streptolysin O [ASOT] and Anti-DNAse B titres), throat swab for MC&S, ECG, echocardiogram and blood cultures if the patient is febrile.

ECGs need interpretation using age adjusted values for normal PR intervals.

Table 3: Upper limits of normal of P–R interval

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

Source: 2020 Australian guideline for prevention, diagnosis and management of acute rheumatic fever and rheumatic heart disease (3rd edition)

DIFFERENTIAL DIAGNOSIS

Rheumatic Heart Disease Australia (RHDA) has created a mobile phone app that includes the rheumatic fever diagnosis calculator. The calculator provides a text and reference for each technical state of a diagnosis, to minimise diagnosis error and inconsistency — see *Educational Resources p20*.

The differential diagnosis of acute rheumatic fever (ARF) depends upon its mode of presentation. If you are considering ARF as a diagnosis the patient should be assessed in conjunction with a paediatrician, physician or cardiologist to confirm the diagnosis or diagnose an alternative condition.

Polyarthritis must be distinguished from the arthritis of arboviral infections (Ross River and Barmah Forrest viruses *p51*), gonococcal arthritis, reactive arthritis or other causes of immune mediated arthritis such as systemic lupus erythematosus (*p191*). Subacute rheumatic carditis with little or no joint involvement may be confused with cardiac conditions such as viral myocarditis or tuberculosis pericarditis. Subclinical ARF can present as a vague illness in Aboriginal and Torres Strait Islander people, associated with aches and pains or unexplained tachycardia.

Note that arthritis from ARF has often been initially mis-attributed to recent trauma such as from sport.

PRINCIPLES OF MANAGEMENT

All cases of ARF, or probable ARF require hospitalisation for investigation and commencement of a management plan. It is important to fully investigate all potential cases of ARF because of the seriousness of the cardiac sequelae and the high chance of recurrence.

If there is doubt about the diagnosis, commence intramuscular benzathine benzylpenicillin before waiting for investigation results or specialist review. All cases should be notified to the local Centre for Disease Control and commenced on secondary prophylaxis. Secondary prophylaxis consists of 4 weekly benzathine benzylpenicillin injections and aims to decrease recurrences of ARF. For the duration of secondary prophylaxis required refer to Table 2 p17.

Environmental factors that promote transmission of *Streptococcus pyogenes* such as overcrowding and limited access to working showers and hand washing facilities should be assessed and patients or their families should be assisted where means exist, to reduce these environmental risks.

Long term management of people with acute rheumatic fever (ARF) and/or rheumatic heart disease (RHD) needs to be developed in consultation with their specialist cardiologist, general physician or paediatrician. People should be encouraged to seek treatment if they have a sore throat, scabies or skin sores, or symptoms of ARF recurrence.

People with rheumatic heart disease need antibiotics prior to certain procedures to prevent endocarditis; refer to the *Therapeutic Guidelines* or regional guidelines, (eg *CARPA Standard Treatment Manual*). All patients with a history of ARF and/or RHD should have an annual dental review.

Rheumatic Heart Disease (RHD) in pregnancy.
All women with RHD should be assessed prior to pregnancy by their GP and cardiologist. Women with moderate or severe rheumatic heart 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 subsequently as per their specific management plan. Women with mild rheumatic heart 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 rheumatic heart disease as early as possible.

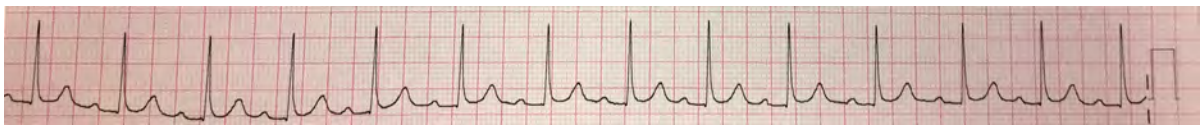

Figure 18: Prolonged PR interval, first degree heart block
Source: Bart Currie — Menzies School of Health Research

Acute rheumatic fever (including recurrent episodes) is a notifiable condition that must be reported by CLINICIANS in the Northern Territory, Queensland, South Australia, New South Wales and Western Australia. All suspected and confirmed cases should be reported to the RHD control program and the local Centre for Disease Control/Public Health Unit.

RHD Control Programs

- NT — Darwin 08 8922 8454
  Alice Springs 08 8951 6909
- QLD — Cairns 1300 135 854 / 07 4226 5544
- WA — Perth 1300 622 745
- SA — Adelaide 08 7425 7146
- NSW — Sydney 1300 066 055

FURTHER INFORMATION

TELEPHONE ADVICE

Contact specialist physician, paediatrician, CDC/PHU in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines see *Appendix p200*.

National		
RHDAustralia	The 2020 Australian guideline for prevention, diagnosis and management of acute rheumatic fever and rheumatic heart disease (3rd edition).	Available online and includes the ARF Diagnostic Calculator.
Northern Territory		
Remote Primary Health Care Manuals (RPHCM)	CARPA Standard Treatment Manvual — Acute rheumatic fever (ARF) and rheumatic heart disease (RHD)	Available online
NT Public Health Network (NT PHN)	Northern Territory HealthPathways ■ Acute rheumatic fever ■ Rheumatic heart disease	Available online
Kimberley		
Kimberley Aboriginal Medical Services (KAMS)	■ Clinical Protocols/Guidelines — Acute Rheumatic Fever (ARF) ■ Notification of infectious diseases and related conditions	Available online
North Queensland		
Queensland Health	Primary Clinical Care Manual — Acute Rheumatic Fever	Available online

EDUCATIONAL RESOURCES

RHDAustralia	Resources and Guidelines ■ Patient Education (including Take Heart App, Treatment Tracker App) ■ Guideline and diagnosis calculator app ■ Control Program (NT, WA, QLD, NSW, SA)	Available online

KEY REFERENCES AND FURTHER READING

RHDAustralia. Guidelines and diagnosis calculator app. http://www.rhdaustralia.org.au/apps

Ralph AP, Fittock M, Schultz R, Thompson D, Dowden M, Clemens T, Parnaby MG, Clark M, McDonald MI, Keith N Edwards KN, Carapetis JR, Bailie RS. Improvement in rheumatic fever and rheumatic heart disease management and prevention using a health centre-based continuous quality improvement approach. BMC Health Services Research. 2013;13(1):525. DOI: 10.1186/1472-6963-13-525

Pneumococcal disease

Streptococcus pneumoniae is a common cause of pneumonia, otitis media and meningitis in Northern Australia. To date over 90 distinct serotypes have been identified.

PNEUMOCOCCAL DISEASE IN NORTHERN AUSTRALIA

Streptococcus pneumoniae (S. pneumoniae) **causes significant morbidity and mortality worldwide. Rates of infection in Aboriginal and Torres Strait Islander Australians are much higher compared to non-Indigenous Australians with the overall Aboriginal and Torres Strait Islander rate of Invasive Pneumococcal Disease (IPD) reported as nine times the rate in the non-Indigenous population in 2011. The NT had the highest rate of IPD in Australia, having 55.8 cases per 100,000 vs the Australian average of 8.3, with pneumonia being the most common presentation. *S pneumoniae* is also a leading cause of non-bacteraemic pneumonia, sinus and ear infections.**

IPD has been notifiable by laboratories in the NT since 1995 and nationally since January 2001. Conjugate pneumococcal vaccines covering — progressively over time — 7, 10 and now 13 pneumococcal serotypes has been available for young children since 2001 and has resulted in significant IPD reduction in those groups targeted for the vaccine, with some evidence of herd immunity for other age groups. Reductions in hospitalised pneumococcal/lobar pneumonia have been reported in Australia along with vaccine-serotype specific pneumococcal otitis media, however middle ear disease remains a major problem for Aboriginal and Torres Strait Islander children in Northern Australia.

AETIOLOGY AND PATHOGENESIS

Streptococcus pneumoniae (S. pneumoniae) is an encapsulated gram-positive coccus. The polysaccharide capsule enhances the virulence of the pneumococcus by protecting it from phagocytosis and providing antigenic variation. To date over 90 distinct serotypes have been identified. The predominant serotypes vary in their distribution between different populations, disease types, age groups, geographic areas and more recently in response to vaccination programs. The 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.

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

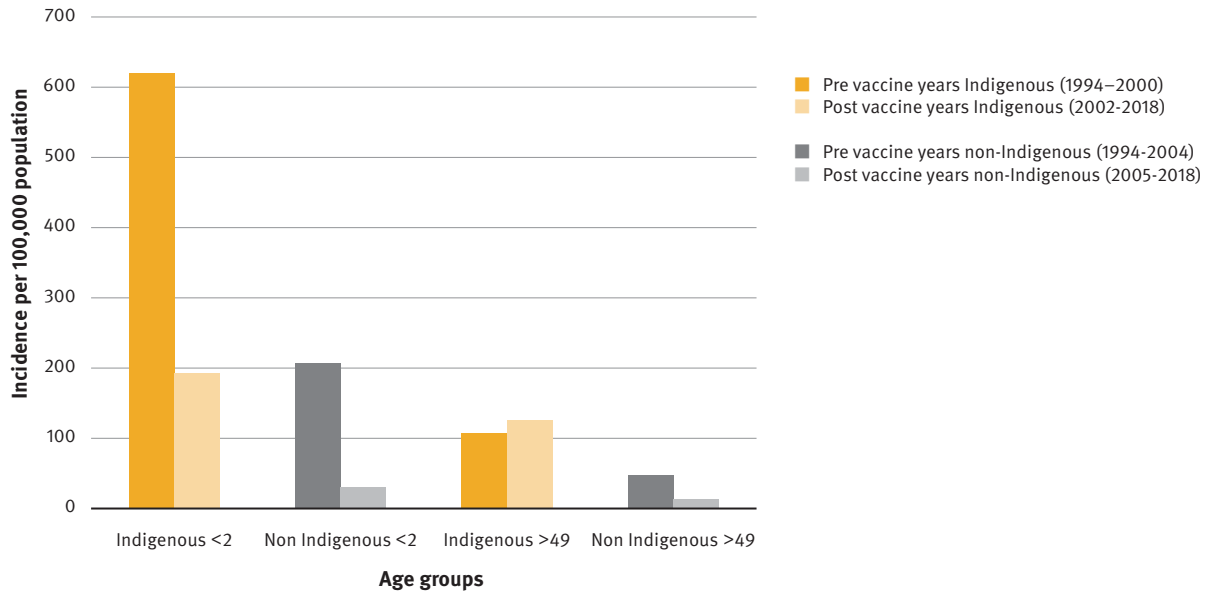

Figure 19: Rates of Invasive Pneumococcal Disease (IPD) in the Northern Territory before and after conjugate vaccine introduction by age group and Indigenous status.
Source: Northern Territory Notifiable Diseases Surveillance System (NTNDSS), Centre for Disease Control, Darwin, accessed June 2019.

CLINICAL PICTURE

Risk factors

- Invasive Pneumococcal Disease (IPD) commonly affects people at the extremes of age. For Aboriginal and Torres Strait Islander Australians this is less than 2 and more than 50 years old
- Other factors associated with the highest risk —
  - > Aboriginal and Torres Strait Islander Australian
  - > Functional or anatomical asplenia
  - > Immunodeficiencies
    - – Steroids
    - – Malignancies
    - – Chronic renal failure
- Conditions with an increased risk —
  - > Cardiac disease
  - > Chronic lung disease
  - > Diabetes
  - > Hazardous alcohol use
  - > Smoking.

Symptoms and signs. The clinical picture will depend on the site of infection. Pneumonia classically presents with the triad of cough, fever and tachypnoea, often preceded by upper respiratory tract signs. Onset of chest symptoms and fever are often abrupt.

Meningitis presents with fever and symptoms and signs of meningeal inflammation (eg, nuchal rigidity, irritability, confusion or altered mental status, headache, photophobia, nausea, vomiting), often preceded by symptoms of upper respiratory tract infection. In infants, signs of meningeal inflammation may be absent or atypical.

Investigations are to identify the infectious agent and site of infection and usually include a FBC, blood for culture and sensitivity, a chest X-ray, and CSF in children and adults with meningeal symptoms and signs.

A Gram stain of the sputum in suspected pneumococcal pneumonia is useful to look for sheets of gram-negative lancet-shaped diplococci and polymorphonuclear leukocytes. A urine pneumococcal antigen test is useful in supporting the diagnosis but 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 more patchy or bronchopneumonic picture is common.

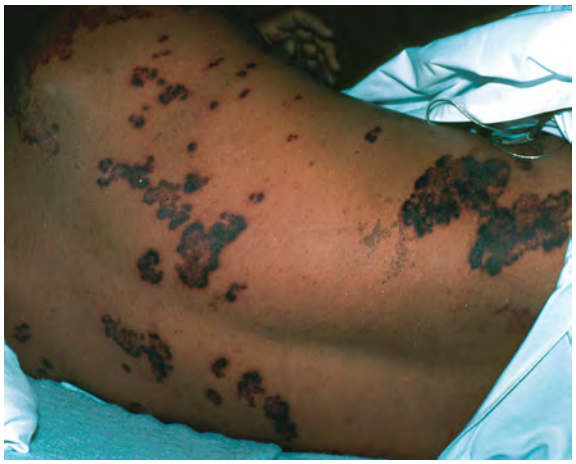

Figure 20: Pneumococcal septicaemia — post splenectomy
Source: Bart Currie — Menzies School of Health Research

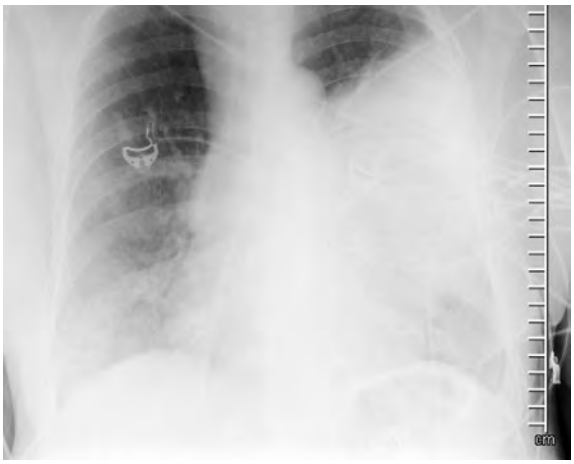

Figure 21: Fatal pneumococcal pneumonia - serotype 3
Source: Bart Currie — Menzies School of Health Research

Pneumococcal disease

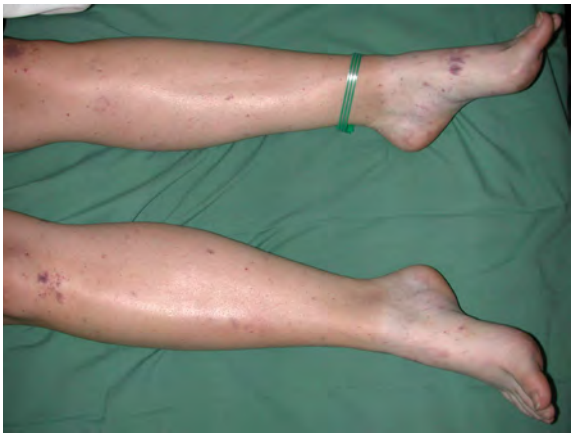

Figure 22: Meningococcal septicaemia rash (differential diagnosis)
Source: Bart Currie — Menzies School of Health Research

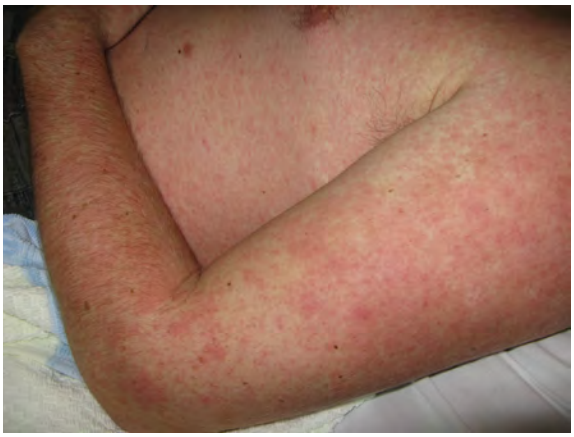

Figure 23: Imported measles — will usually have bad cough and fever (differential diagnosis)
Source: Bart Currie — Menzies School of Health Research

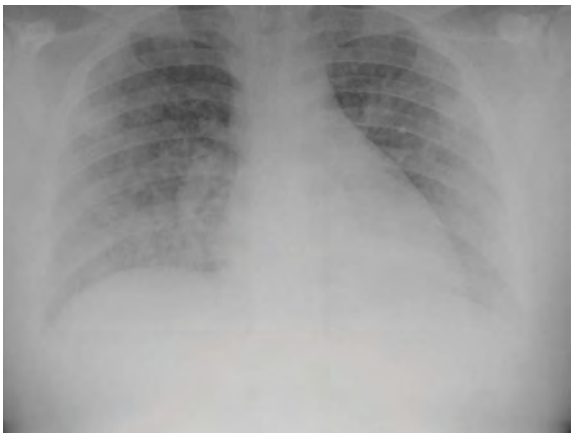

Figure 24: Sixteen year old with varicella pneumonia
Source: Bart Currie — Menzies School of Health Research

DIFFERENTIAL DIAGNOSIS

Pneumococcal pneumonia is a common cause of adult community-acquired pneumonia. The differential diagnosis of a patient with clinical pneumonia includes melioidosis, *Staphylococcus*, *Acinetobacter* and other causes of bacterial 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 was penicillin, detailed empirical protocols have been developed as a strategy for appropriate management in the face of decreased susceptibility in *S. pneumoniae*, and also to cover other causes of potentially fatal causes of pneumonia such as melioidosis (*p25*) and *Acinetobacter* (*p41*).

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 are common and need to be treated aggressively. Clinicians should refer to their regional guidelines (eg *CARPA Standard Treatment Manual*) or the *Therapeutic Guidelines: Antibiotic* for the treatment of community-acquired pneumonia (also see *Case Study — Pneumonia p45*).

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 13vPCV) has 13 polysaccharide serotypes conjugated to a diphtheria carrier protein and routinely included in a 3-dose childhood immunisation schedule at 2, 4, and 6 months, with Aboriginal and Torres Strait Islander children and at-risk children in the NT receiving a 4th (booster) dose at age 18 months. Check the Immunisation Schedule in your jurisdiction. Older children and adults with medical conditions associated with the highest increased risk of Invasive Pneumococcal Disease (IPD) are also recommended to receive a single dose of Prevenar 13® vaccine.

- The polysaccharide vaccine (23vPPV) contains polysaccharides derived from the 23 most frequent or virulent capsular types. In the NT it is funded for non-Indigenous persons aged 65 years and over, and for Aboriginal and Torres Strait Islander persons aged 15 years and over.

The NT 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

TELEPHONE ADVICE

Contact infectious disease physician, general physician, CDC/PHU in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines see *Appendix p200*.

National		
Australian Immunisation Handbook	Pneumococcal disease	Available online
Northern Territory		
Centre for Disease Control	NT Pneumococcal vaccination and revaccination guideline	Available online
NT Department of Health	■ Immunisation Schedules ■ Notifiable diseases	Available online
Kimberley		
WA Department of Health	■ Public health — Immunisation schedule ■ Notification of infectious diseases and related conditions	Available online
North Queensland		
Queensland Health	■ Communicable disease control guidance — Pneumococcal disease (invasive) ■ Immunisation schedule ■ Notifiable conditions ■ Guidelines for PHU	Available online

EDUCATIONAL 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

KEY REFERENCES AND FURTHER READING

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Lima FJ, Lehmann D, McLoughlin A, Harrison C, Willis J, Giele C, Keil AD, Moore HC. Risk factors and comorbidities for invasive pneumococcal disease in Western Australian Aboriginal and non-Aboriginal people. Pneumonia. 2014 Sep 11;4(1):24.

Jayasinghe S, Chiu C, Menzies R, Lehmann D, Cook H, Giele C, Krause V, McIntyre P. Evaluation of impact of 23 valent pneumococcal polysaccharide vaccine following 7 valent pneumococcal conjugate vaccine in Australian Indigenous children. Vaccine. 2015 Nov 27;33(48):6666-74. DOI: 10.1016/j.vaccine.2015.10.089

Menzies R, Andrews R. Immunisation issues for Indigenous Australian children. Journal of Paediatrics and Child Health. 2014 Oct;50(10):E21-5. DOI: 10.1111/j.1440-1754.2011.02079.x

Melioidosis

The bacillus Burkholderia pseudomallei causes melioidosis, which can affect any organ of the body. Until improved diagnosis and therapy decreased mortality to under 10% it was the most common cause of fatal community-acquired bacteraemic pneumonia in the Top End of the Northern Territory.

MELIOIDOSIS IN NORTHERN AUSTRALIA

Bacillus *Burkholderia pseudomallei* that causes melioidosis was formerly known as *Pseudomonas pseudomallei*. Yearly case numbers in the NT over the last decade have fluctuated between 36 and 97, with almost all acquired in the Top End following heavy wet season rains. Over the past 5 years Queensland has averaged 25 cases a year and Western Australia has averaged 6 cases a year. More rapid diagnosis and newer therapies have recently improved outcomes.

In a long-term study of adults admitted to Royal Darwin Hospital with pneumonia, *B. pseudomallei* accounted for 24% of culture positive cases of community-acquired pneumonia and 36% of deaths. *Streptococcus pneumoniae* was the most commonly isolated pathogen overall, accounting for 39% of cases and 20% of fatalities.

AETIOLOGY AND PATHOGENESIS

B. pseudomallei is a gram negative bacillus found in soils and water across the Top End and in parts of Northern Western Australia and Northern Queensland, as well as in some Central Australian locations 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 especially in the subacute stage of infection.

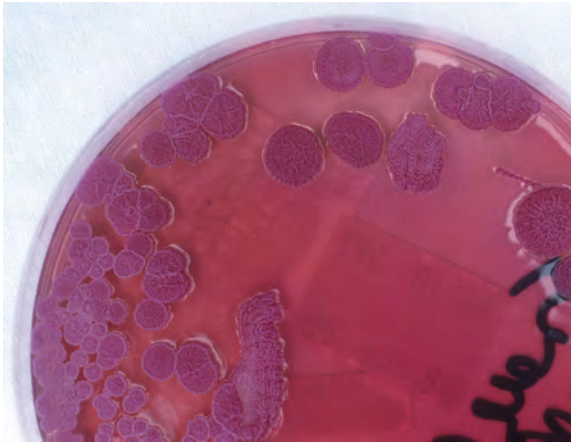

Figure 25: *Burkholderia pseudomallei* growing on agar plate
Source: Bart Currie — Menzies School of Health Research

CLINICAL PICTURE

Risk Factors. Although melioidosis usually affects adults with risk factors, it can affect healthy adults and children. Aboriginal and Torres Strait islander peoples are over-represented in the incidence of contracting melioidosis. Clinicians should have a low threshold for considering melioidosis as a potential diagnosis across a range of presentations.

Diabetes is the most important risk factor being present in over 40% of cases. Others include chronic renal disease, chronic lung disease, hazardous alcohol use and kava intake, malignancy, immunosuppression, skin contact with wet soil and age more than 45 years.

Over 80% of cases occur during the wet season, with an incubation period of 1–21 days (median 9 days). During periods of severe rain and wind, inhalational infection can result in critical illness within 1–2 days.

Symptoms and signs will depend on the mode and site of infection. Pneumonia is the most common manifestation of the disease and has a case fatality rate of 10–40%, with the lowest rates seen when there is rapid diagnosis and access to appropriate antibiotics and state-of-the-art ICU management.

Melioidosis can also cause severe sepsis leading to septic shock, multiple organ abscesses, genito-urinary infection (especially prostate abscesses), osteomyelitis, septic arthritis, pustular skin lesions or ulcers, and rarely, neurological infections with encephalomyelitis.

Two important presentations of melioidosis are:

(i) prostate melioidosis: men with non-specific abdominal pain, dysuria, diarrhoea, fever and possible urinary retention and

(ii) melioidosis encephalomyelitis: fever, headache, cranial nerve palsies +/- limb weakness or flaccid paraparesis.

Some people are found to be seropositive in the absence of symptoms or known past history of the disease. Latent infection may activate many years later in a small proportion of these people, analogous to infection with tuberculosis. It is thought that reactivation accounts for less than 5% of cases in the Top End.

Investigations. Serology for melioidosis is useful but may not be diagnostic for a current active infection and a positive serology may also reflect just past exposure and not acute disease (ie not melioidosis). The likelihood of diagnosis is increased by using modified Ashdown's broth (purple broth bottles), a selective culture media, frequent sampling including culture of sputum, urine, throat, rectal and ulcer swabs and collection of blood cultures.

Culture specimens should specifically request examination for melioidosis. If sputum has been sent in Ashdown's broth then another sputum should also be sent separately for MC&S for other organisms, including request for acid fast bacilli and TB culture where TB is considered possible. Blood tests should include melioidosis serology. 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.

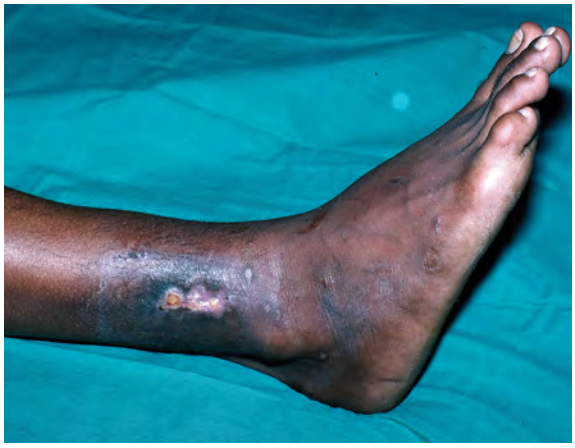

Figure 26: *Burkholderia pseudomallei* osteomyelitis and discharging ulcer
Source: Bart Currie — Menzies School of Health Research

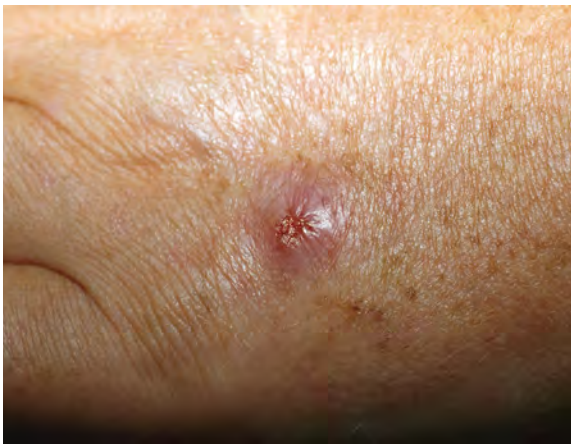

Figure 27: Melioidosis — skin abscess in a healthy child who was otherwise well
Source: Bart Currie — Menzies School of Health Research

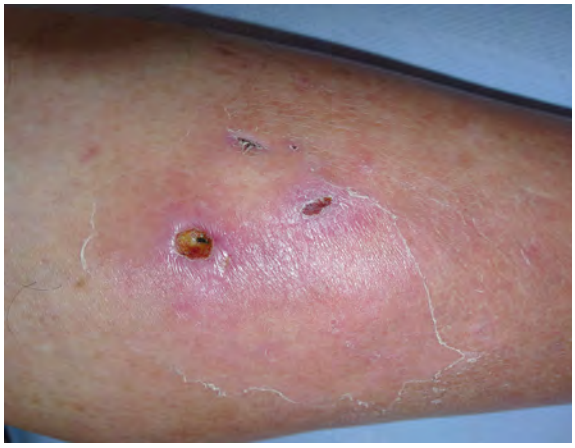

Figure 28: Melioidosis cutaneous post animal bite
Source: Bart Currie — Menzies School of Health Research

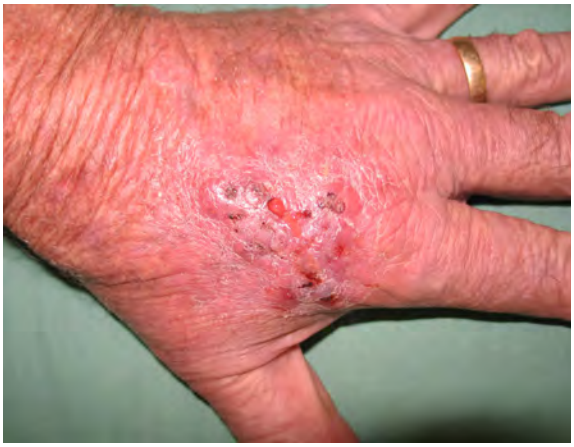

Figure 29: Melioidosis cutaneous
Source: Bart Currie — Menzies School of Health Research

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 and sporotrichosis as shown overpage). Progressive upper lobe disease mimics tuberculosis and bronchoscopy may be required to make a definitive diagnosis if cultures of sputum are negative.

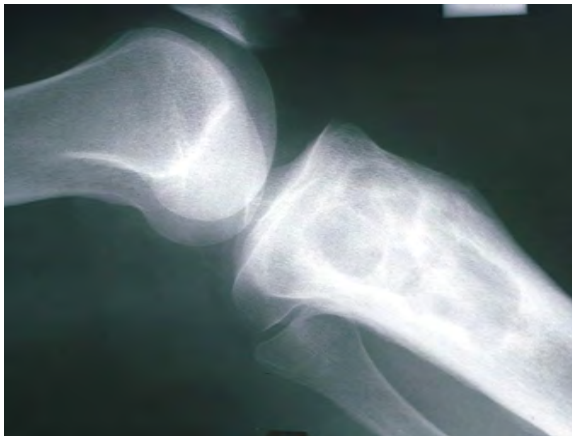

Figure 30: Melioidosis tibial osteomyelitis
Source: Bart Currie — Menzies School of Health Research

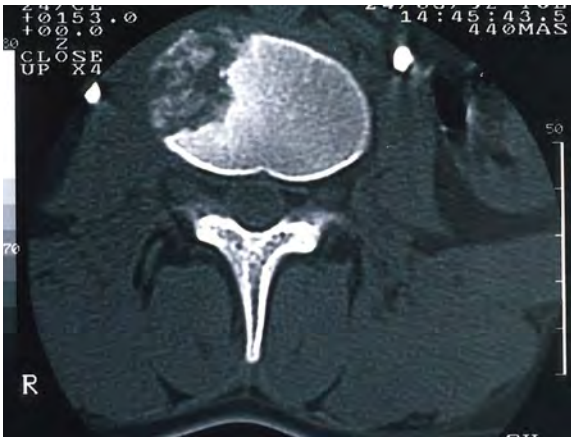

Figure 31: Melioidosis vertebral osteomyelitis
Source: Bart Currie — Menzies School of Health Research

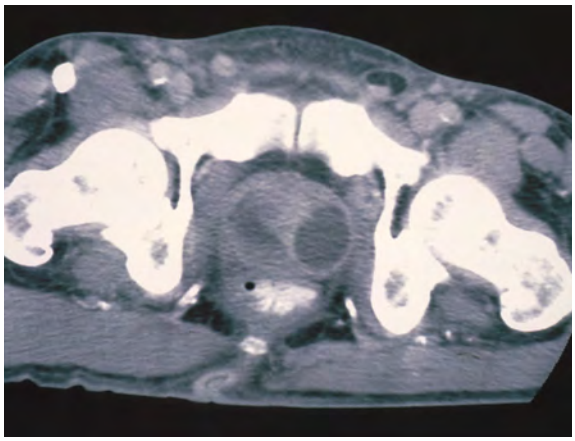

Figure 32: Melioidosis — prostatic abscesses
Source: Bart Currie — Menzies School of Health Research

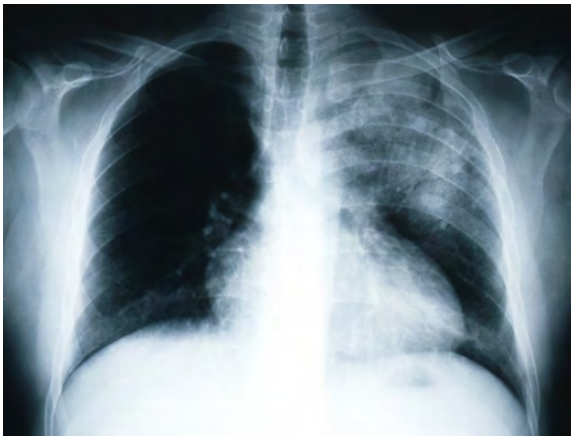

Figure 33: Melioidosis — acute pneumonia
Source: Bart Currie — Menzies School of Health Research

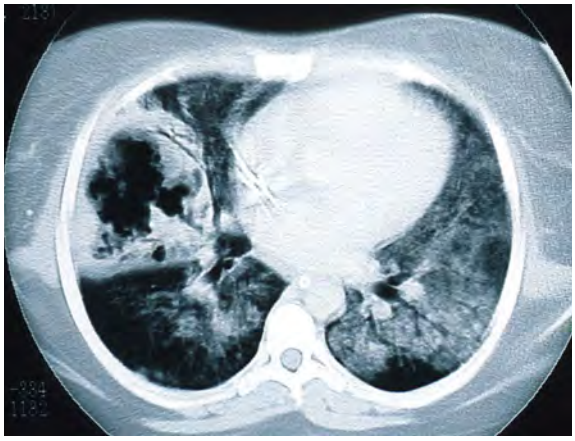

Figure 34: Melioidosis fatal necrotising pneumonia
Source: Bart Currie — Menzies School of Health Research

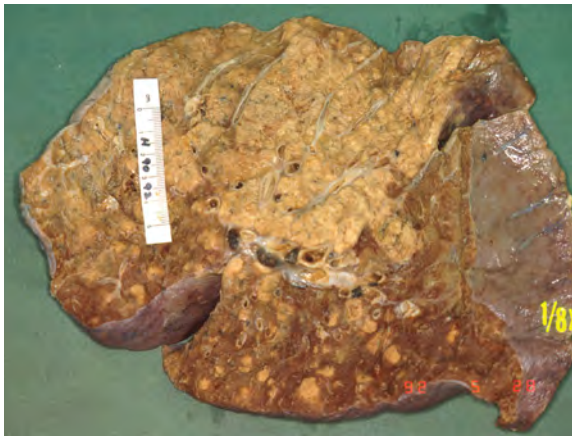

Figure 35: Fatal melioidosis pneumonia — lung autopsy
Source: Bart Currie — Menzies School of Health Research

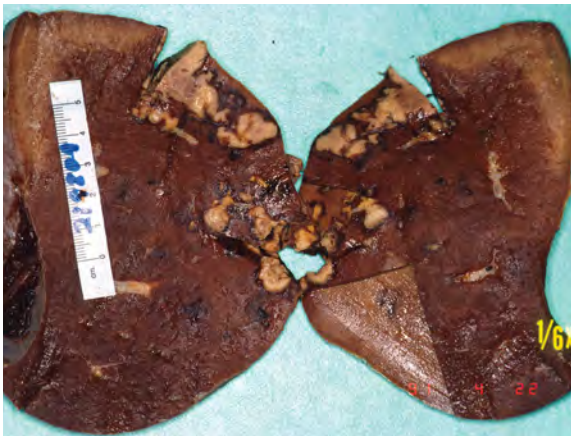

Figure 36: Melioidosis splenic abscesses requiring splenectomy
Source: Bart Currie — Menzies School of Health Research

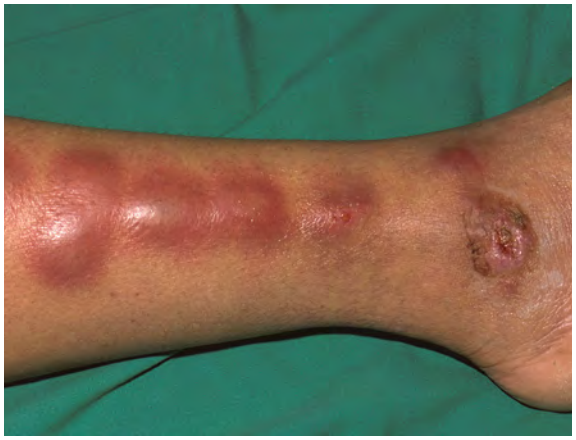

Figure 37: Nocardia cutaneous post-partum with ankle inoculation site (differential diagnosis)
Source: Bart Currie — Menzies School of Health Research

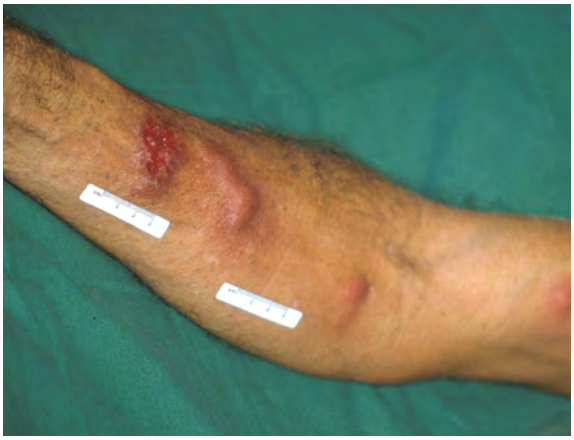

Figure 38: Nocardia cutaneous (differential diagnosis)
Source: Bart Currie — Menzies School of Health Research

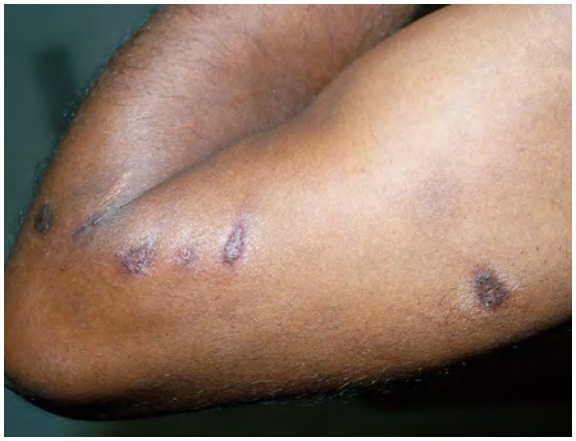

Figure 39: Sporotrichosis (differential diagnosis)
Source: Bart Currie — Menzies School of Health Research

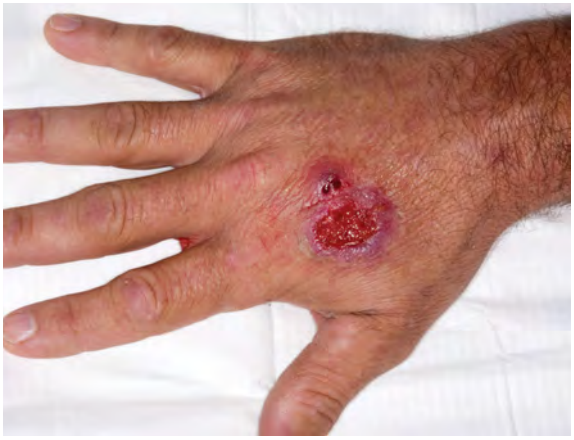

Figure 40: Sporotrichosis (differential diagnosis)
Source: Bart Currie — Menzies School of Health Research

Melioidosis

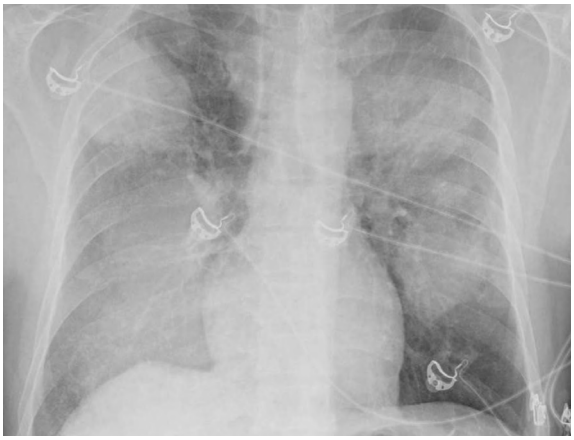

Figure 41: Severe nocardia pneumonia (differential diagnosis)
Source: Bart Currie — Menzies School of Health Research

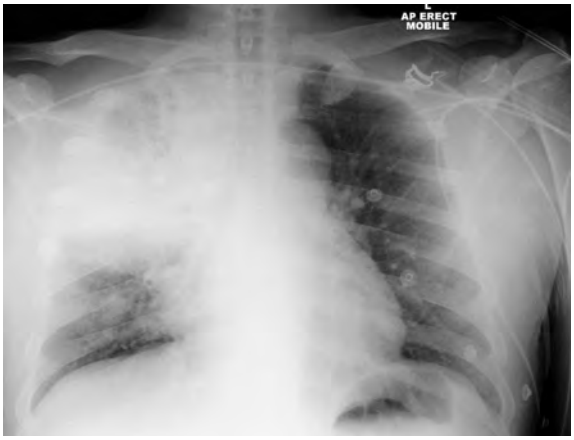

Figure 42: Thirty six year old with fatal nocardia pneumonia (differential diagnosis)
Source: Bart Currie — Menzies School of Health Research

PRINCIPLES OF MANAGEMENT

Prevention. Avoid contact with wet season soils or muddy water by wearing appropriate shoes and using gloves when gardening or working outdoors. Also avoid high pressure hosing during the wet season, or as a minimum use an appropriate protective face mask. These measures are particularly important for people with diabetes or other risk factors.

Treatment. Early diagnosis and appropriate antibiotic therapy decrease mortality. Until diagnosis is confirmed follow the *Therapeutic Guidelines* or regional guidelines (eg *CARPA Standard Treatment Manual*) for community-acquired pneumonia, which cover the most important pathogens in Northern Australia (see *Case study — Pneumonia p45*).

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, Queensland and Western Australia. Cases are to be reported to the local Centre for Disease Control/Public Health Unit.

FURTHER INFORMATION

TELEPHONE ADVICE

Contact infectious disease physician, CDC/PHU in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines see *Appendix p200*.

Northern Territory		
Remote Primary Health Care Manuals (RPHCM)	CARPA Standard Treatment Manual — Melioidosis	Available online
NT Public Health Network (NT PHN)	Northern Territory HealthPathways — Melioidosis	Available online
NT Department of Health	Notifiable diseases	Available online
Kimberley		
WA Department of Health	Notification of infectious diseases and related conditions	Available online
North Queensland		
Queensland Health	■ Communicable disease and control guidance — melioidosis ■ Notifiable conditions	Available online

EDUCATIONAL RESOURCES

NT Centre for Disease Control (CDC)	■ Fact sheet — Melioidosis ■ Poster – Melioidosis	Available online

KEY REFERENCES AND FURTHER READING

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Tuberculosis (TB)

Tuberculosis is caused by *Mycobacterium* tuberculosis and most commonly presents with lungs symptoms but can affect every body system.

TUBERCULOSIS IN NORTHERN AUSTRALIA

Australia has one of the lowest rates of tuberculosis (TB) in the world (incidence rate is between 5–6 per 100,000) with people born overseas accounting for 85–90% of new cases. In the NT annualised incidence between 2014 and 2018 were 9.2 cases per 100,000 population (95% CI 5.8 to 13.8) with overseas-born people accounting for 58% of cases, Aboriginal and Torres Strait Islander Australians for 33%, and non-Indigenous Australian-born people representing less than 9%. While there have been outbreaks in remote and urban Aboriginal communities in 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 TB, hazardous alcohol use, and immunosuppressing conditions or medications. Our near neighbours, Indonesia, East Timor and Papua New Guinea have very high rates of TB, as do other countries in the region, including Vietnam, Myanmar 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 the NT.

While multi-drug resistant TB (MDRTB) is a concern in much of the rest of the world only a few cases of MDRTB have been notified in the NT. However the high rate of MDRTB in Papua New Guinea is a continuing threat to North Queensland. Preventing the emergence of multi-drug resistant TB 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 appropriate 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 is deposited in the lungs. Fairly close and prolonged contact is required for transmission with an estimated 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, eg diabetes, HIV infection, head and neck cancers, a very young age.

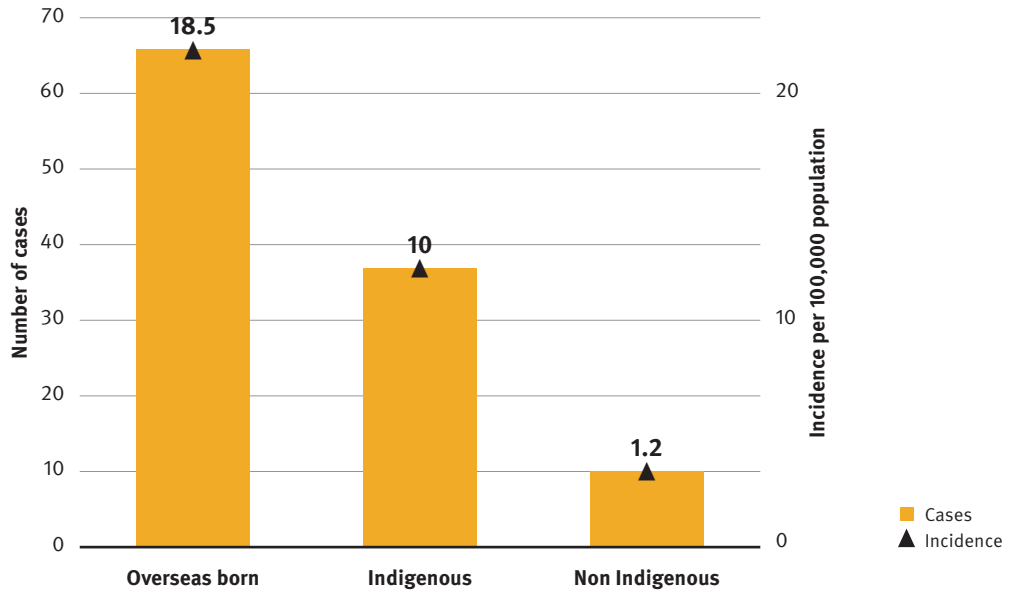

Figure 43: TB cases notified in the NT 2014 to 2018 and incidence by ethnicity
Source: Northern Territory Department of Health: Research, Reporting and Analysis. 2018. Health district Population 1971 to 2017.

Once infected, a person will have a positive tuberculin skin test (TST) or a 'Mantoux test' or will test positive via a similar blood test called an interferon-gamma release assay (IGRA). 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 which is symptomatic and may, if present in lungs, transmit TB to others. 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 treat it. 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 NT 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, Aboriginal and Torres Strait Islander people, people who overuse alcohol, and those with diabetes, chronic renal disease, malignancies (particularly of the head and neck), leukaemias or lymphomas.

Patients with latent *M. tuberculosis* (LTBI) infection 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 — eg renal, bladder, genital tract
- Skeletal: commonly spine and hip
- Meningeal
- Miliary — eg disseminated TB in lungs and elsewhere
- Serous membranes — pleurisy, pericarditis, peritoneal
- Ileocaecal (bowel)
- Almost any other site, eg eye, breast, liver.

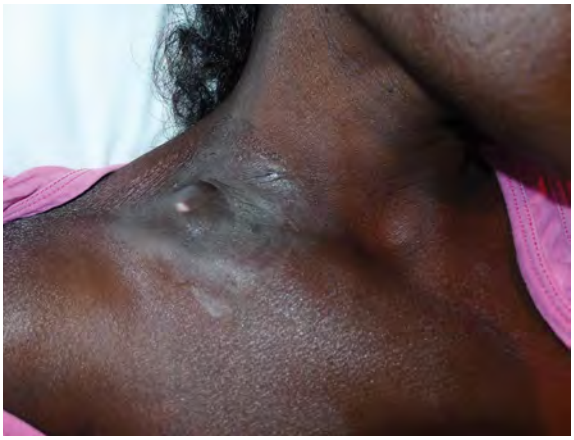

Figure 44: TB lymphadenitis
Source: Bart Currie — Menzies School of Health Research

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, eg headache, swollen joint etc.

Investigations. Microbiological confirmation of the diagnosis of TB should be obtained:

- Pulmonary TB — sputum smear examination for acid fast bacilli (AFB) and culture. Other considerations in the differential diagnosis may include collecting sputum for cytology, or to diagnosis 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 patient cannot produce sputum alternative methods are gastric aspiration, induced sputum, or bronchoscopy

Tuberculosis (TB)

- For TB in other sites — a fine needle or formal biopsy for culture is recommended (do not put it into formalin)
- Radiology — chest X-ray, CT, MRI
- Haematology — haemoglobin, white cell count and differential, ESR and CRP
- Histology — may support the diagnosis but is not specific and tissue needs to be cultured
- The TST/Mantoux test and interferon-gamma release assays (IGRAs) are helpful in determining previous TB exposure and latent TB infection. 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 p34*.

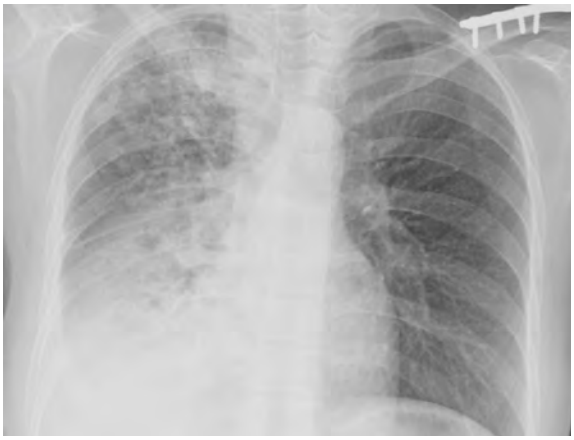

Figure 45: Tuberculosis — newly arrived in NT
Source: Bart Currie — Menzies School of Health Research

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 — cryptococcus, nocardiosis, actinomycosis, aspergillosis, pulmonary strongyloidiasis
- Other — bronchiectasis, sarcoidosis, pneumoconiosis, lymphoma.

PRINCIPLES OF MANAGEMENT

Directly observed therapy (DOT) or well supported curative treatment with anti-TB medications and adequate nutrition are the hallmarks of the clinical management of active TB disease. Patients require close monitoring for side-effects of treatment, for barriers to treatment, and for signs of clinical improvement.

The most effective public health interventions are the prompt diagnosis and adequate treatment of TB cases with curative treatment, and the treatment of latent TB infection (LTBI) to prevent TB usually with isoniazid. In the NT, cases of smear positive pulmonary TB are isolated in hospital until sputum smears are negative.

For active TB cases, a combination of four drugs (usually rifampicin, isoniazid, pyrazinamide, and ethambutol) is given for 2 months, followed by 4 months treatment with two drugs (usually rifampicin and isoniazid). Treatment of latent TB infection (LTBI) is usually 9 months of isoniazid.

General practitioners contribute to TB control by prompt investigation of clinical presentations suggestive of TB, and by having a high index of suspicion of the disease in high risk individuals. GP's may also assist in contact tracing, directly observed therapy and clinical monitoring in remote communities.

TB Control Units provide expert advice and treatment and clinical management and follow-up for all TB patients. They have educational materials to share with doctors 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.

FURTHER INFORMATION

TELEPHONE ADVICE

Contact infectious disease physician, CDC/PHU in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines see *Appendix p200*.

National		
Communicable Diseases Network Australia (CDNA)	■ National Guidelines for Public Health Units — Management of TB ■ Australian notifiable diseases and case definitions	Available online
Northern Territory		
Centre for Disease Control (CDC)	■ Guidelines for the Treatment of Tuberculosis in the Northern Territory ■ 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
Kimberley		
WA Department of Health	■ Notification of infectious diseases and related conditions ■ Public health management	Available online
North Queensland		
Queensland Health	■ Communicable disease and control guidance — Tuberculosis ■ Primary Clinical Care Manual —Tuberculosis ■ Notifiable conditions	Available online

EDUCATIONAL RESOURCES

NT Centre for Disease Control (CDC)	■ Fact sheet — Tuberculosis ■ Fact sheet — Tuberculosis (multiple languages)	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
Queensland Health	Tuberculosis Control in Queensland —Information for the Public and Patients	Available online

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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 NT 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.

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 (M. 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, Aboriginal and Torres Strait Islander 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. Specific forms are available to guide clinical examination for leprosy.

Investigations. Demonstration of acid-fast bacilli (AFB) in slit-skin smears from standardised sites and lesions. Typical histology, read by an experienced pathologist for skin or nerve biopsies and 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 present as hypopigmented or erythematous coppery patches with loss of sensation and an absence of sweating in a lesion
- Impairment or involvement of peripheral nerves, weakness of hands and feet or face.

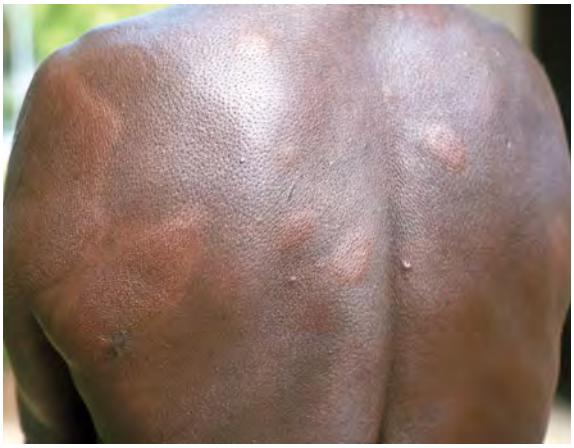

Figure 46: Borderline lepromatous leprosy with Type I reaction
Source: Bart Currie — Menzies School of Health Research

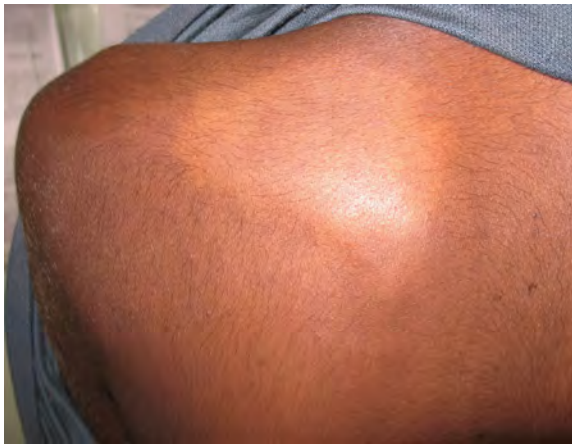

Figure 47: Leprosy anaesthetic hypopigmented skin patch
Source: Bart Currie — Menzies School of Health Research

DIFFERENTIAL DIAGNOSIS

Differential diagnosis includes: tinea (eg pityriasis versicolor), systemic lupus erythematosus, lupus vulgaris (skin TB), sarcoidosis and yaws. Peripheral neuropathy from causes such as diabetes, alcoholism 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.

When multi-drug therapy is commenced, there is a sudden liberation of antigen from killed *M. leprae* which can precipitate a reaction with inflammation of skin lesions and nerves which are classified as Type I or Type II reactional states. Neuritis can be symptomatic but is often silent, and if undetected and untreated, the resulting disability may become irreversible.

The key monitoring activity is a monthly standardised motor and sensory test to detect loss of neurologic function. High dose prednisolone may be required for 4–6 months to reverse neuritis and prevent disability.

Established impairments such as anaesthetic and anhidrotic skin, or paralysis with contracture, can be managed in general practice 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
- 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 TB 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.

> 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.

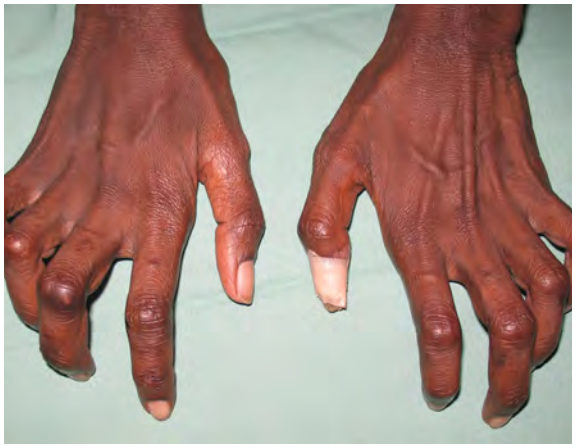

Figure 48: Leprosy nerve damage
Source: Bart Currie — Menzies School of Health Research

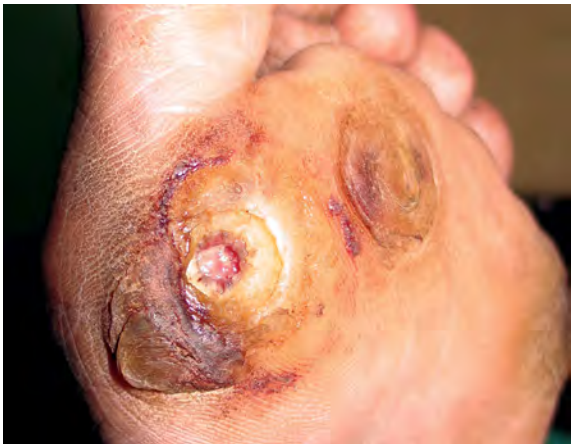

Figure 49: Leprosy foot ulcer from nerve damage
Source: Bart Currie — Menzies School of Health Research

Leprosy

FURTHER INFORMATION

TELEPHONE ADVICE

Contact infectious disease physician, CDC/PHU in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines see *Appendix p200*.

International		
World Health Organization	Global Leprosy Program ■ Global Leprosy Strategy 2016–2020 ■ Leprosy elimination	Available online
National		
Communicable Diseases Network Australia (CDNA)	Australian notifiable diseases and case definitions	Available online
Northern Territory		
Centre for Disease Control (CDC)	■ Guidelines for the control of leprosy in the NT ■ Notifiable diseases	Available online
Kimberley		
WA Department of Health	■ Notification of infectious diseases and related conditions ■ Public health management — Leprosy	Available online
Kimberley Aboriginal Medical Services (KAMS)	Clinical Protocols/Guidelines — Leprosy	Available online
North Queensland		
Queensland Health	■ Primary Clinical Care Manual — Leprosy ■ Communicable disease control guidance — Hansen's diseases ■ Notifiable conditions	Available online

EDUCATIONAL RESOURCES

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

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Mycobacterium ulcerans

Mycobacterium ulcerans causes slowly growing skin ulcers.

MYCOBACTERIUM ULCERANS IN NORTHERN AUSTRALIA

***Mycobacterium ulcerans (M. ulcerans)* causes indolent, necrotising skin lesions which occasionally involve the underlying bone. Most cases in Northern Australia occur in the coastal areas between Mossman and the Daintree River in Queensland. Cases have been described rarely from the Top End of the Northern Territory and around Yeppoon in Central Queensland. Areas around Melbourne have had substantially increasing case numbers in recent years.**

AETIOLOGY AND PATHOGENESIS

The organism produces a toxin called mycolactone that is responsible for both the necrosis and localised immunosuppression. The ulcer is known as the Buruli ulcer, named after the area in Uganda where this disease has been common. In Australia, the condition is also called Daintree ulcer (North Queensland) or Bairnsdale ulcer (Victoria) after the 2 areas from which the disease was initially described. The organism is associated with water and probably an event breaching the skin such as an insect bite, but the modes of transmission to humans are still an area of active enquiry.

CLINICAL PICTURE

Risk factors. The main risk factor is living or visiting an endemic area. Transmission is highly focal geographically.

Symptoms and signs. An initial papule slowly progresses to an ulcer. Other presentations include nodules or non-ulcerative plaques. The ulcers are typically undermined and indurated. The lesions are typically painless. A small proportion of ulcers may spontaneously heal. On the other hand, some lesions progressively enlarge.

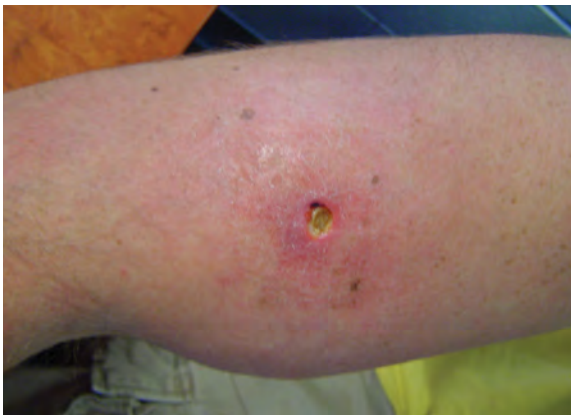

Figure 50: Characteristic appearance of an early ulcer
Source: Prof John McBride

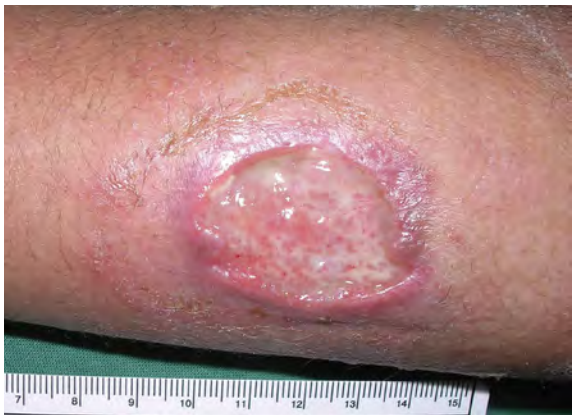

Figure 51: *Mycobacterium ulcerans* ulcer
Source: Bart Currie — Menzies School of Health Research

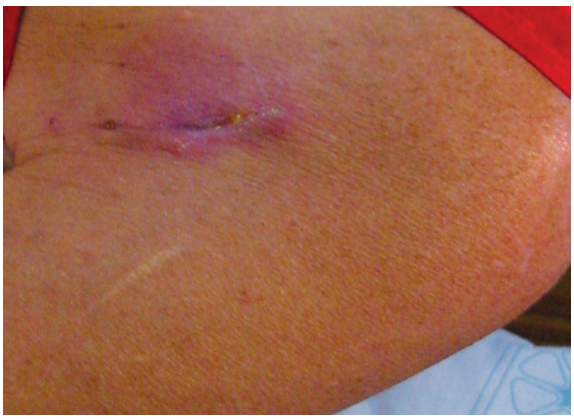

Figure 52: *Mycobacterium abscessus* ulcer
Source: Bart Currie — Menzies School of Health Research

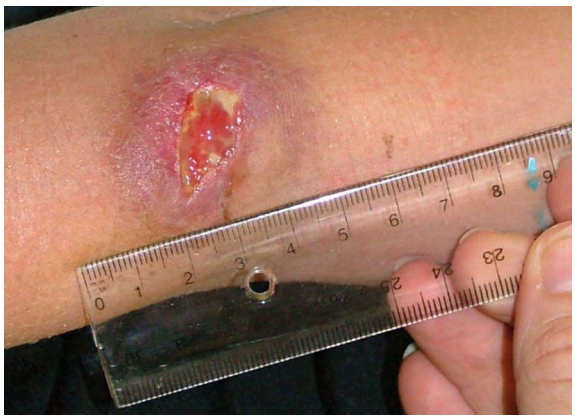

Figure 53: *Mycobacterium fortuitum* ulcer
Source: Bart Currie — Menzies School of Health Research

Investigations. Any suspected lesion should have swabs taken for a Ziehl-Neelsen stain and be sent for histology and specific PCR testing. A skin biopsy from the edge of an ulcer will demonstrate characteristic histological changes. The organism can be cultured from a swab but because of the slow growth of the organism, culture results are often not obtained in a clinically useful timeframe.

DIFFERENTIAL DIAGNOSIS

There are a range of non-infectious causes of chronic ulcers, including venous stasis, diabetic ulcers, pyoderma gangrenosum, and arthropod bites. Infectious causes which could be confused include other atypical mycobacteria, fungal infections, melioidosis and yaws.

PRINCIPLES OF MANAGEMENT

Treatment may either be surgical, medical or combined. Medical therapy comprises a combination of rifampicin with either ciprofloxacin or clarithromycin and is now recommended in all cases and usually for some weeks before surgery. The usual treatment duration is 8 weeks although shorter courses of 4 weeks may be suitable when used as an adjunct to surgical excision of early lesions. Extensive lesions may require skin grafts.

Antibiotics may be associated with an initial paradoxical worsening of an ulcer associated with cessation of the local immune suppression caused by mycolactone.

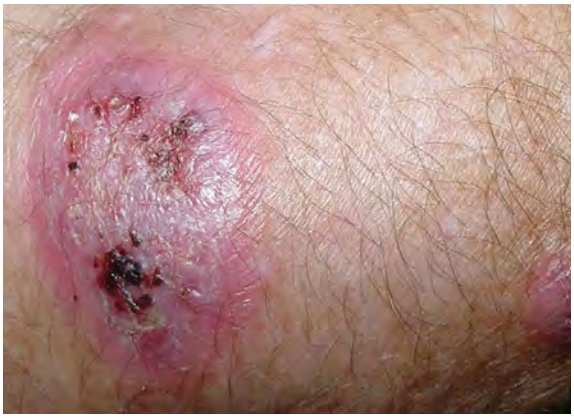

Figure 54: *Mycobacterium marinum* ulcer from fish tank exposure
Source: Bart Currie — Menzies School of Health Research

Treatment should always be discussed with the local infectious diseases services and surgical advice sought for complex lesions.

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

FURTHER INFORMATION

TELEPHONE ADVICE

Contact infectious disease physician, CDC/PHU in your local jurisdiction.

MANAGEMENT GUIDELINES

Northern Territory		
Centre for Disease Control (CDC)	■ Non-tuberculous mycobacteria (NTM) Guidelines for Health Professionals in the Northern Territory ■ Notifiable diseases	Available online
North Queensland		
Queensland Health	Communicable disease control guidance — Non-tuberculous mycobacterial	Available online

EDUCATIONAL RESOURCES

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

KEY REFERENCES AND FURTHER READING

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Acinetobacter baumannii pneumonia

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

ACINETOBACTER BAUMANNII PNEUMONIA IN NORTHERN AUSTRALIA

***A. baumannii* is a gram-negative coccobacillus that is ubiquitous in fresh water and soil, and is a frequent human skin and throat commensal. It is well-known because of the development of multiple drug resistance and causing hospital acquired pneumonia.**

Important in Northern Australia is its ability to cause community-acquired infections in those with hazardous alcohol use and diabetes, with particularly 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 found in soil, water, and in other living organisms, where they may or may not be pathogenic. 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.

CLINICAL PICTURE

Risk factors. The majority of cases in tropical Australia occur during the warmer, wetter months of October to April. Other risk factors are hazardous alcohol use, chronic lung disease, smoking, diabetes and chronic renal failure. The distribution is tropical and sub-tropical regions.

Symptoms and signs. Patients present with cough, dyspnoea and fever in most cases. The illness develops rapidly and most patients seen have met the criteria for severe sepsis. The common chest X-ray findings are lobar consolidation.

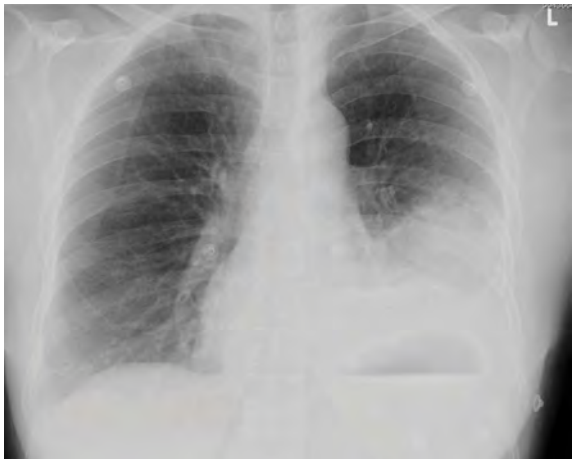

Figure 55: Severe *Acinetobacter baumannii* community-acquired pneumonia
Source: Bart Currie — Menzies School of Health Research

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* species that cause community-acquired pneumonia are often still sensitive to antibiotics including gentamicin, meropenem, and ciprofloxacin. Since the standard NT treatment for patients with severe tropical wet season community-acquired pneumonia and sepsis included an initial dose of gentamicin and meropenem has subsequently been added in those patients sick enough to be managed in ICU, mortality has fallen from more than 60% to 11%.

FURTHER INFORMATION

TELEPHONE ADVICE

Contact infectious disease physician, CDC/PHU in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines see *Appendix p200*.

EDUCATIONAL RESOURCES

Sydney Adventist Hospital	Patient and Visitor Fact Sheet — Multi-resistant Acinetobacter baumannii (MRAB)	Available online

KEY REFERENCES AND FURTHER READING

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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 the Aboriginal and Torres Strait Islander population is about 5–10 times that of non-Indigenous populations. Bronchiectasis has a reported incidence of 14.7 per 1000 in Aboriginal children under 15 years of age in Central Australia. 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

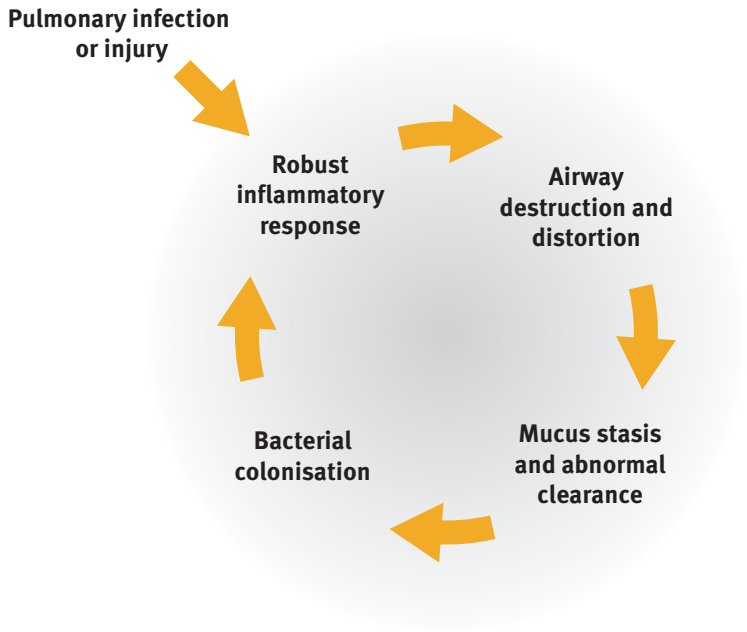

Figure 56: Cole's vicious cycle diagram
Source: Inflammation: a two-edged sword - the model of bronchiectasis. European Journal of Respiratory Diseases. Supplement 1986; 147:6-15

CLINICAL PICTURE

Early signs are a prolonged cough and recurrent respiratory infections. Chronic wet cough (more than 4 weeks) 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.

Experience has shown that Aboriginal and Torres Strait Islander 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.

PRINCIPLES OF MANAGEMENT

Management aims to improve symptoms and quality of life, while preserving lung function and reducing exacerbation frequency. 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.

FURTHER INFORMATION

TELEPHONE ADVICE

Contact respiratory or general physician in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines see *Appendix p200*.

Northern Territory		
Remote Primary Health Care Manuals (RPHCM)	CARPA Standard Treatment Manual ■ Chronic suppurative lung disease and bronchiectasis in children ■ Chronic lung disease in adults	Available online
Kimberley		
Kimberley Aboriginal Medical Services (KAMS)	Clinical Protocols/Guidelines ■ Adult Chronic Lung Disease — COPD and Bronchiectasis ■ Respiratory Disease in Children	Available online

EDUCATIONAL RESOURCES

Menzies School of Health Research	Chronic Suppurative Lung Disease/Bronchiectasis (Chronic Lung Sickness — Information sheet for Indigenous Australians)	Available online

KEY REFERENCES AND FURTHER READING

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Steinfort DP, Brady S, Weisinger HS, Einsiedel L. Bronchiectasis in Central Australia: a young face to an old disease. Respiratory Medicine. 2008 Apr;102(4):574-8. DOI: 10.1016/j.rmed.2007.11.007

Case study — Pneumonia

Sandra is a 51 year old Aboriginal woman who is brought in by a female 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 non-insulin dependent diabetes and frequently has high blood sugar readings. She has smoked tobacco for 30–40 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 90, RR of 32, and BP of 115/80. Her fingerprick glucose reading is 23mmol/L. She is breathless at rest.

On auscultation of her chest she has left sided crackles and bronchial breathing. Her yellow/green sputum is copious but is not bloodstained. You diagnose Sandra with an acute community-acquired pneumonia.

What organisms are likely to be responsible?

The organisms most likely to be responsible for the pneumonia include *Streptococcus pneumoniae, Burkholderia pseudomallei* (melioidosis), *Staphylococcus aureus*, and *Acinetobacter baumannii*. Other possibilities include *Heamophilus influenzae*, and *Cryptococcus neoformans* particularly in Arnhem Land.

Given the short history of illness, infection due to *Mycobacterium tuberculosis* is 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 uncommon in remote areas.

Influenza occurs in clusters in remote locations, where it usually has higher morbidity and mortality. 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 in adults indicates severe pneumonia
- BP — systolic less than 90, diastolic less than 60 indicates severe pneumonia and a high risk for developing septic shock
- Others include evidence of shock, confusion, and oxygen saturation less than 90% on room air. Parameters are listed in more detail in the *Therapeutic Guidelines*.

In this setting, **local risk factors are all important** to determine the risk of severe, life-threatening pneumonia caused by *Acinetobacter baumannii* or melioidosis. Sandra is Aboriginal, obese, has diabetes, is a smoker, has a history of respiratory infections and it is the early wet season. Even if the pneumonia was moderate and not severe, on these factors alone 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: Aboriginality, chronic medical conditions such as diabetes, hazardous alcohol use, chronic lung disease, chronic renal disease, steroid treatment, kava use, and poor nutrition.

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

By clinical criteria Sandra's pneumonia is severe: she has borderline high respiratory rate and is mildly tachycardic. With her many risk factors she is at high risk of life threatening pneumonia and requires prompt evaluation, treatment and transfer. See the various pneumonia severity scores discussed in *Therapeutic Guidelines*.

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

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

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%)
- Establish 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
- Prompt IV antibiotics — as per *Therapeutic Guidelines* or regional protocols (eg *CARPA Standard Treatment Manual*)
- Urine MC&S and an ECG as part of the initial management.

Penicillin is **not** recommended as a first line agent for moderate or severe pneumonia, in those with risk factors in tropical Australia, as it does not adequately cover potentially fatal organisms. In hospital once the causative organism is identified the antibiotic regimen can be rationalised. Atypical pneumonia cover is not usually recommended as primary therapy in remote communities.

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.

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 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
- Support for individuals and encouragement and support of community health promotion initiatives in these areas are important roles of the health team
- 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
- What has the diabetes educator suggested for her diabetes care and treatment
- Melioidosis eradication therapy in consultation with Royal Darwin Hospital Infectious Diseases Department.

Case study — Pneumonia

KEY REFERENCES AND FURTHER READING

Phung DT, Wang Z. Risk of pneumonia in relation to body mass index in Australian Aboriginal people. Epidemiology and Infection. 2013 Dec;141(12):2497-502. DOI: 10.1017/S0950268813000605

Deek H, Abbott P, Moore L, Davison J, Cameron S, Digiacomo M, et al. Pneumococcus in Aboriginal and Torres Strait Islander peoples: the role of Aboriginal health workers and implications for nursing practice. Contemporary Nurse. 2013 Dec;46(1):54-8. DOI: 10.5172/conu.2013.46.1.54

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.

Sparham S, Charles PGP. Controversies in diagnosis and management of community-acquired pneumonia. Medical Journal of Australia. 2017 17 April;206(7):316-319.

Ranzani OT, Prina E, Menendez R, Ceccato A, Cilloniz C, Mendez 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 Nov 15;196(10):1287-97. DOI: 10.1164/rccm.201611-2262OC

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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 has occasionally occurred in the Torres Strait Islands and adjacent Cape York (probably from wind-blown mosquitoes), and dengue outbreaks 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 for the Murray Valley encephalitis (MVE) and West Nile (Kunjin strain) viruses, malaria, Ross River virus (RRV), Barmah Forest virus (BFV) and Japanese encephalitis (JE) throughout the region.

***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 NT or WA. The re-introduction or spread of *Aedes* mosquito vectors for dengue into the NT or WA 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 virus are present in East Timor, Indonesia, Papua New Guinea and North Queensland and imported cases of these arboviruses and of malaria can occur anywhere in Australia.

MVE occurs sporadically throughout Northern Australia with the highest incidence in the northern parts of WA, occasional cases in central Australia, and exceptional outbreaks in southern Australia. JE transmission occurs 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.

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.

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 NT 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 carried out at airports and wharf areas.

AETIOLOGY AND PATHOGENESIS

Malaria in humans is caused by one of 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, Kunjin, JE and Zika Dengue only occurs in humans. 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 water-birds. WA and the NT have a viral monitoring program through sentinel chicken flocks. 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*.

Table 4: 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, mild to moderate encephalitis
Murray Valley encephalitis(MVE)	*C. annulirostris*	Common throughout Northern Australia	Probable	Moderate to severe encephalitis, death
Japanese encephalitis (JE)	*C. annulirostris, Culex jelidus*	Common throughout Northern Australia	Probable	Moderate to severe encephalitis, death
Ross River virus (RRV)	*Aedes vigilax, C. annulirostris*	Common throughout Northern Australia	Probable	Ross River fever (RRF), 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

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. Symptomatic infections are manifested by fever, headache, seizures especially in children and sometimes nausea and vomiting, rapidly followed by the features of encephalitis. **West Nile (Kunjin strain) infections** are almost invariably less severe, with encephalitis being rare. 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.

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%).

Serological investigation of arboviral infections

In the early symptomatic stage of most arboviral infections direct detection of virus from blood and/or CSF is the most appropriate method of diagnosis. Virus isolation for diagnosis of arbovirus infection is available in reference laboratories. RT-PCR, however, is much more sensitive. 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. Serology responses to flaviviruses cross react with each other — making a specific diagnosis difficult.

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.

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 following notification of the Centre for Disease Control/ Public Health Unit and liaison with an infectious disease physician (refer to relevant malaria protocol).

Treatment of severe dengue, or dengue with warning signs (refer to dengue treatment 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. 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 p56*.

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
- Serological monitoring of sentinel chicken flocks located throughout WA and the NT. Seroconversion in these animals alerts public health authorities to potential for outbreaks of arboviruses
- New technologies with virus nucleic acid detection by PCR from mosquito traps may replace the sentinel chickens
- Public health warnings are given when mosquito numbers and/or clinical cases increase above the expected levels.

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

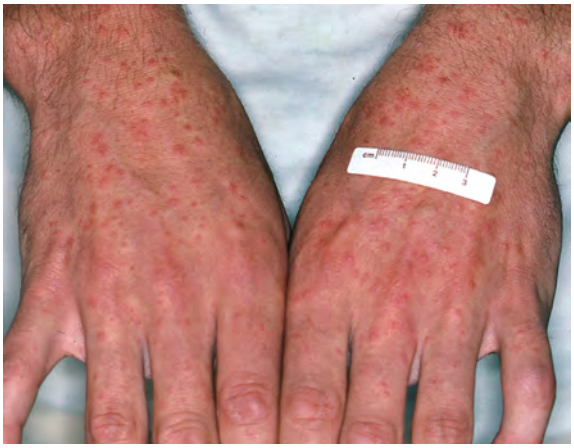

Figure 57: Barmah Forest virus arthritis and rash
Source: Bart Currie — Menzies School of Health Research

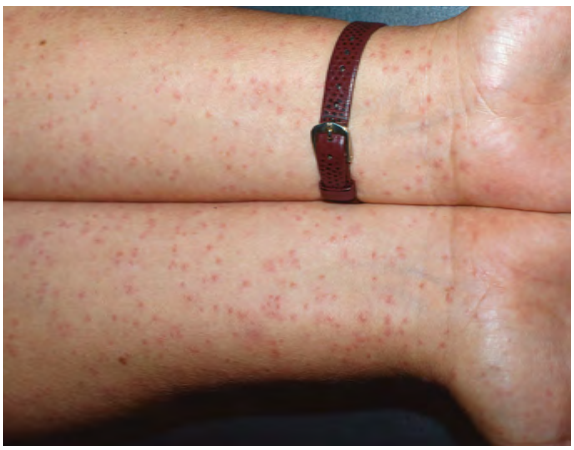

Figure 58: Barmah Forest virus rash
Source: Bart Currie — Menzies School of Health Research

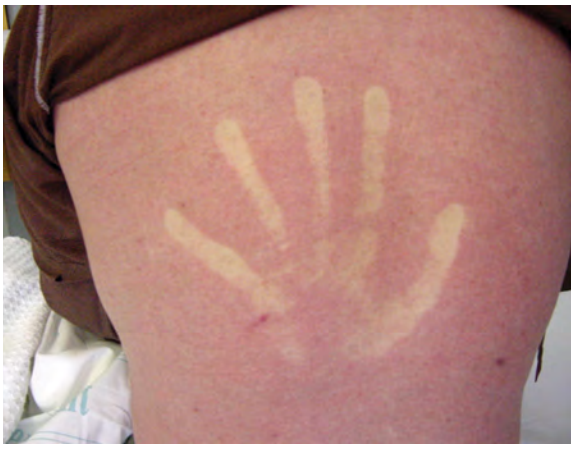

Figure 59: Dengue blanching rash — acquired in Timor Leste
Source: Bart Currie — Menzies School of Health Research

Mosquito borne diseases

Table 5: Summary of clinical and diagnosis of vector borne diseases

Illness	Fever	Rash	Other symptoms	Diagnostic test
Malaria	+	-	Headache, chills, rigors	Blood film, antigen test
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.

FURTHER INFORMATION

TELEPHONE ADVICE

Contact infectious disease physician, medical entomologist, CDC/PHU in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines see *Appendix p200*.

National		
Communicable Diseases Network Australia (CDNA)	■ National Guidelines for Public Health Units: > Dengue > Murray Valley encephalitis > Zika ■ Australian notifiable diseases case definitions	Available online
Therapeutic Guidelines	Antibiotics: Malaria	Available online
Northern Territory		
Centre for Disease Control (CDC)	Guidelines for Malaria	Available online
North Queensland		
Queensland Health	■ Communicable disease and control guidance > Malaria > Japanese encephalitis ■ Primary Clinical Care Manual > Ross River Fever and Barmah Forest Virus > Dengue fever	Available online

EDUCATIONAL RESOURCES

NT Centre for Disease Control (CDC)	Fact sheets — Ross River virus, Mosquitoes, Murray Valley encephalitis, Japanese encephalitis, Dengue, Chikungunya	Available online
Queensland Health	Fact sheets — Malaria, Japanese encephalitis	Available online
Western Australia Department of Health	■ Diseases transmitted by mosquitoes in Western Australia ■ Fact sheet — Ross River virus and Barmah Forest virus	Available online

KEY REFERENCES AND FURTHER READING

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Scrub typhus

Scrub typhus is from a bacterial infection transmitted via mite bite in Far North Queensland (FNQ), in Litchfield Park south west of Darwin, and several other remote Top End locations.

SCRUB TYPHUS IN NORTHERN AUSTRALIA

Cases of scrub typhus have been reported throughout FNQ and more recently, from some of the Torres Strait Islands. In the Top End there has been one fatality and several severe cases. Most NT cases have occurred in workers or visitors to rainforest areas within Litchfield Park, south west of Darwin, but other foci of scrub typhus have now been found in the NT. There has also been a single Kimberley case.

Scrub typhus has a wide distribution from eastern Asia to the western Pacific, and is characteristically patchy in distribution (mirroring the mite vector habitats).

AETIOLOGY AND PATHOGENESIS

Scrub typhus is a zoonotic infection in which humans are accidental hosts. The causative organism is *Orientia tsutsugamushi* (previously known as *Rickettsia tsutsugamushi*) which has several different serotypes. The bacterium is transmitted to humans by the trombiculid mite, *Leptotrombidium deliense*.

The infection is maintained in the mite population transovarially which leads to well described foci of transmission such as Litchfield Park in the NT, areas around Mission Beach, Cairns, Mossman, and certain islands in the Torres Strait.

The mites depend on small rodents for blood meals. Bacteria proliferate at the site of the mite bite before disseminating throughout the body. The period from the mite bite to development of symptoms is 5–18 days.

CLINICAL PICTURE

Risk factors include time in endemic areas, particularly walking and camping. Clusters of cases have occurred in military personnel undertaking training in coastal sites close to Innisfail, North Queensland.

Symptoms and signs include an inoculation eschar (black scab) of approximately 1cm in diameter, at the bite site which is often found on the buttocks, genitalia, lower trunk or armpit. The eschar may go unnoticed by the patient or in moist areas may look like an ulcer. Occasionally no eschar can be found, even on full body inspection. Fever, regional lymphadenopathy, myalgias, rash and severe headache follow.

In a small number of patients the disease may be severe with delirium, tremors, slurred speech and multi-organ failure. A skin rash can occur on the trunk on around day 5 of illness and may spread to the extremities. It is usually maculopapular but may be fleeting.

Investigations. Serology (indirect microimmunofluorescence test for *O. tsutsugamushi*) should be performed on serum taken at presentation and two weeks later. Initial serum is often negative but a fourfold rise in titre is diagnostic. PCR is sensitive when performed on eschar material, and less sensitive when performed on whole blood

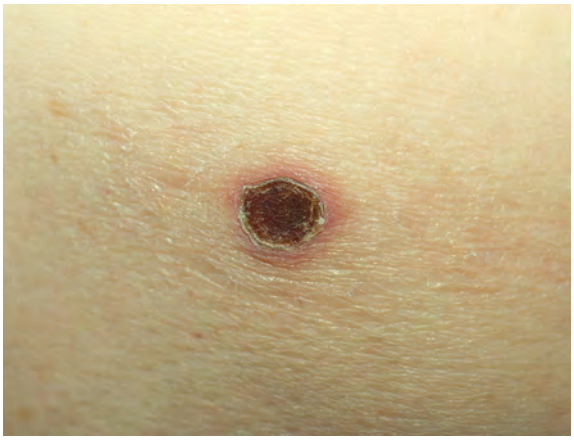

Figure 60: Scrub typhus eschar on buttock
Source: Bart Currie — Menzies School of Health Research

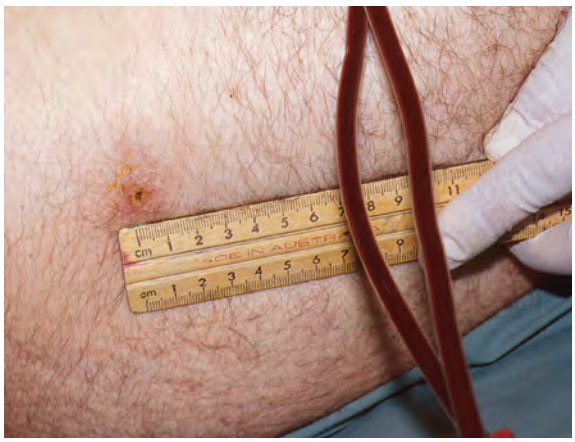

Figure 61: Scrub typhus eschar and multi-organ failure requiring dialysis
Source: Bart Currie — Menzies School of Health Research

DIFFERENTIAL DIAGNOSIS

The differential diagnosis is broad. The non-specific symptoms of fever, myalgia and headache could be influenza, toxoplasmosis, infectious mononucleosis, 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. Queensland tick typhus caused by *Rickettsia australis* should be considered in North Queensland.

PRINCIPLES OF MANAGEMENT

Management depends on the severity of infection. In mild disease oral tetracyclines, such as doxycycline, are rapidly effective and reduce the risk of relapse. In more severe disease with multi-organ failure hospital-based supportive therapy may be required and recovery is sometimes delayed. Prompt therapy may be lifesaving, so consider and test for scrub typhus in patients with flu-like illnesses and a history of forest exposure from an endemic area.

Prevention includes wearing stout footwear and long trousers in high risk areas. Use N,N-diethyl-meta-toluamide (DEET) containing repellents on skin and socks/trousers and a groundsheet for sitting. Doxycycline has been evaluated for prophylaxis for those at particularly high risk, eg soldiers.

> Scrub typhus is a notifiable condition to be reported by LABORATORIES in the Northern Territory. Report cases to the local Centre for Disease Control. It is NOT a notifiable disease in Queensland or Western Australia.

FURTHER INFORMATION

TELEPHONE ADVICE

Contact infectious disease physician, medical entomologist, CDC/PHU in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines see *Appendix p200*.

EDUCATIONAL RESOURCES

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

KEY REFERENCES AND FURTHER READING

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Faa AG, McBride WJ, Garstone G, Thompson RE, Holt P. Scrub typhus in the Torres Strait islands of north Queensland, Australia. Emerging Infectious Diseases. 2003 Apr;9(4):480-2. DOI: 10.3201/eid0904.020509

Frances SP. Rickettsial diseases of military importance: an Australian perspective. Journal of Military and Veterans' Health. 2011 Oct 1;19(4).

Likeman RK. Scrub typhus: a recent outbreak among military personnel in North Queensland. ADF Health. 2006;7(1):10-3.

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Leptospirosis

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

LEPTOSPIROSIS IN NORTHERN AUSTRALIA

The average notification rate of leptospirosis is 0.79/100,000 population/year in Australia (1991–2015), with Queensland having the highest average notification rate of 2.3/100,000 population. The NT has the second highest average notification rate of 1.08/100,000 population.

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

AETIOLOGY AND PATHOGENESIS

Leptospirosis occurs worldwide, but is more common in tropical/subtropical areas with high rainfall. It is primarily a disease of animals — reported vectors include domestic animals (especially dogs), rodents, marsupials and bats.

There are nine species pathogenic to humans comprising more than 250 serovars. The predominant serovar causing disease in the NT is *Leptospira australis*. Humans are infected through contact of abraded skin or of mucous membranes with urine/body fluids of infected animals or soil contaminated by infected animals. Human cases are not usually infectious to others if standard precautions are followed.

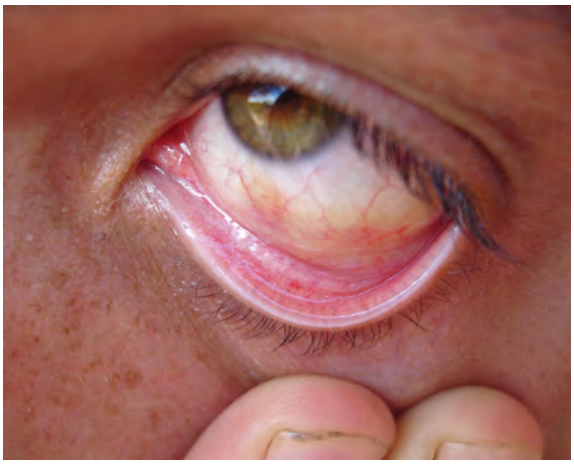

Figure 62: Leptospirosis conjunctival injection
Source: Bart Currie — Menzies School of Health Research

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.

Conjunctival suffusion (redness without exudate) may occur in over 50% of cases and its presence in a patient with a nonspecific febrile illness should raise the possibility of leptospirosis.

CLINICAL PICTURE

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

Symptoms. The severity of illness varies. Approximately 90% of cases are self-limiting systemic illness, with 10% exhibiting a more severe potentially fatal illness (Weil's disease). 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 can be biphasic with defervescence for a day or two between phases. Some patients present in the second phase of illness and the two phases may not be distinct in severe illness. The first phase may last 5–7 days and the second phase may last up to 30 days after onset of illness.

Early symptoms of mild illness are flu-like with marked fever and myalgia. Headache, abdominal pain, 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 and muscle tenderness are the most common signs. Less common signs include lymphadenopathy, splenomegaly, hepatomegaly and rarely a maculopapular pretibial rash. In the second phase of the illness any of the following may be present: conjunctival suffusion, muscle tenderness, adenopathy, signs of meningitis, signs of pneumonitis and possibly pulmonary haemorrhage, signs of myocarditis, hepatomegaly and jaundice, splenomegaly, and signs of renal failure.

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 may show a moderate elevation in transaminases and bilirubin. Serum creatine kinase is usually high, and elevated creatine kinase is an important indicator to suspect leptospirosis. Renal function tests may show renal impairment. Hypokalemia and hyponatraemia are common. Cerebrospinal fluid may show an aseptic meningitis picture with a lymphocytic pleocytosis. In severe disease chest X-ray changes may be evident and progressive. Nodular densities may progress to confluent consolidation or a ground glass appearance consistent with pulmonary haemorrhage.

Appropriate leptospirosis directed testing depends on the timing of the infection (Figure 65). During the first week leptospires are present in the blood and may 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 may also be detected in blood by nucleic amplification method performed on serum (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 may be detectable within 5–10 days of illness onset but the IgM test may be false positive for a number of reasons and 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. Not uncommonly 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.

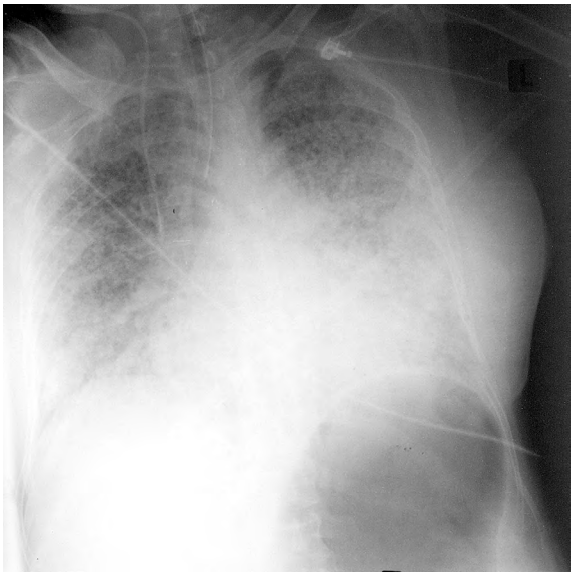

Figure 63: Severe leptospirosis with pulmonary haemorrhage
Source: Bart Currie — Menzies School of Health Research

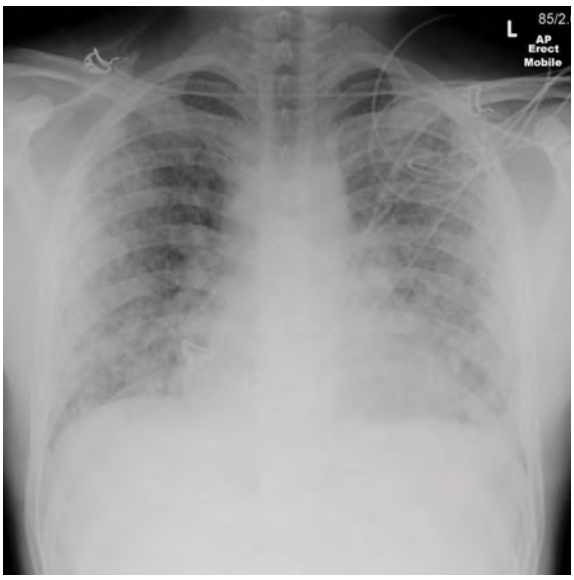

Figure 64: Nineteen year old with leptospirosis severe pneumonia
Source: Bart Currie — Menzies School of Health Research

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 (including travel to North Queensland during a dengue outbreak), Hantavirus infections, and other rickettsial diseases. Finally 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/or 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.

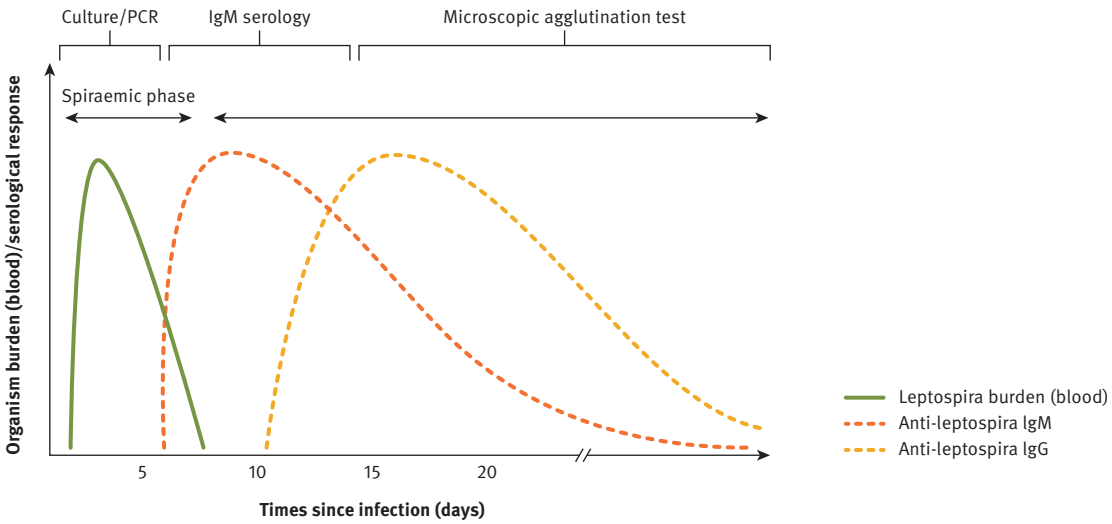

Figure 65: Prototypic antibody response in Leptospirosis against time. Appropriate diagnostic testing for leptospirosis is dependent on accurate timing of infection.

Source: The Royal Australian College of General Practitioners from Slack A. Leptospirosis. Australian Family Physician 2010;39(7):495–98. Reproduced with permission.

Treatment includes supportive treatment and antibiotic therapy. Refer to *Therapeutic Guidelines: Antibiotic* for details. Patients who recover before the diagnosis is made do not require antibiotic treatment. If leptospirosis is suspected start treatment before the diagnosis is confirmed. 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 (qid) 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 have been reported in patients treated with penicillin.

> 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

TELEPHONE ADVICE

Contact infectious disease physician, CDC/PHU in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines see *Appendix p200*.

Northern Territory		
Centre for Disease Control (CDC)	Notifiable diseases	Available online
Kimberley		
WA Department of Health	Notification of Infectious Diseases and related conditions	Available online

EDUCATIONAL RESOURCES

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

ACKNOWLEDGEMENTS

For serovar specific data: WHO Leptospirosis Laboratory, Public and Environmental Health, Health Support Queensland, Department of Health.

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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 Aboriginal and Torres Strait Islander people.

Mass 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 compliance. In the 1990s trachoma control programs ceased in many regions. By 2008 the NT wide mean prevalence in remote communities was 29%. East Arnhem Land was an exception with a prevalence of less than 5%.

In 2006 the Communicable Diseases Network Australia (CDNA) published guidelines with prevalence thresholds for recommending community wide treatments with oral azithromycin. The current iteration of the National Trachoma Elimination Project is part of the 'WHO Global Elimination Trachoma by 2020' project. In 2012 the NT average prevalence had fallen to 4%. These initial gains have been attenuated with the average prevalence rising to 5% and 5.9% in 2013 and 2014 respectively.

Overall the majority of communities have demonstrated a reduction in trachoma prevalence to non and meso endemic levels, however several communities in Central Australia, the Barkly Region, and the western side of the Katherine Region continue to demonstrate hyperendemic levels of trachoma and receive community wide treatment with oral azithromycin.

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 trichiasis is present, 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 (following).

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.

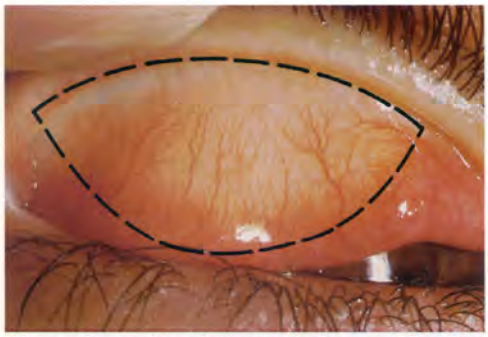
Table 6: WHO Trachoma simplified grading card

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).

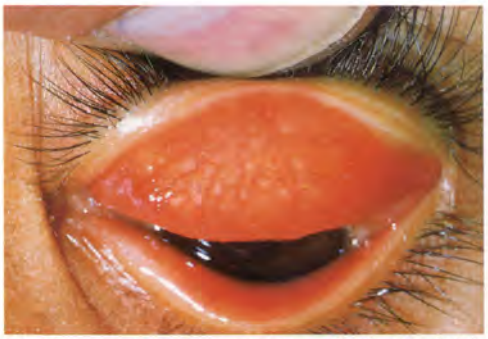
The normal conjunctiva is pink, smooth, thin and transparent. Over the whole area of the tarsal conjunctiva there are normally large deep-lying blood vessels that run vertically.



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.

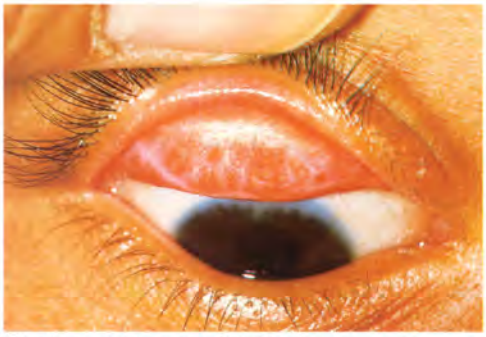
Trachomatous inflammation – follicular and intense (TF + TI).

WHO Trachoma simplified grading card. SAFE documents — reproduced with permission
http://www.who.int/neglected_diseases/resources/SAFE_documents/en

Table 6: WHO Trachoma simplified grading card *(continued)*

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

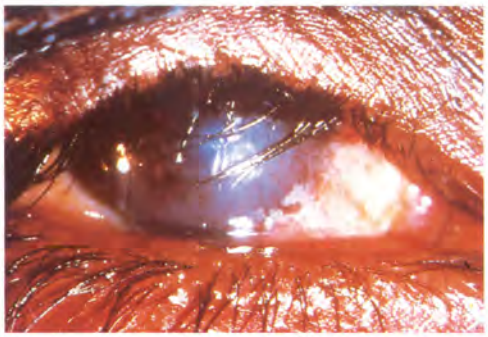
Scars are easily visible as white lines, bands, or sheets in the tarsal conjunctiva. They are glistening and fibrous in appearance. Scarring, especially diffuse fibrosis, may obscure the tarsal blood vessels.



Trachomatous scarring (TS)

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

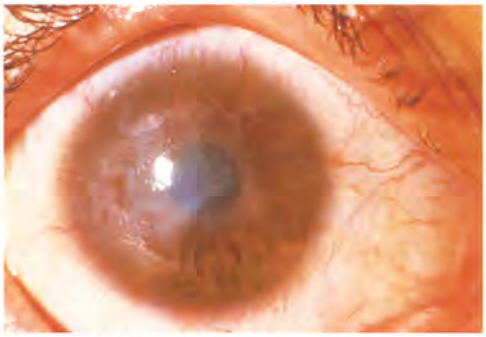
Evidence of recent removal of inturned eyelashes should also be graded as trichiasis.



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.

WHO Trachoma simplified grading card. SAFE documents — reproduced with permission http://www.who.int/neglected_diseases/resources/SAFE_documents/en

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.

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. The approach to treatment of individuals depends upon the prevalence of trachoma among 5–9 year olds in the community. In small populations the inclusion of children 1–4 years old will enhance the validity of findings. The coverage goal for screening is 85% of the target population and the treatment goal is 85% of the household contacts and 100% of known cases.

Community wide treatment.

1. If prevalence is ≥20% (hyperendemic) there should be community wide education and treatment with azithromycin. The aim is to decrease the reservoir of active trachoma by treating all members of every household where a child under 15 years spends one night or more a week. Treatment must be completed within 10 days. Five 6 monthly iterations of community wide treatments will be required.

2. If prevalence is ≥5 to <20% and there is ***no obvious clustering*** of cases treat all people more than 3kg living in households with children less than 15 years of age. Three annual iterations of community wide treatments will be required

   OR

   If prevalence is ≥5 to <20% and cases are ***obviously clustered*** within several households and health staff can easily identify ***all*** household contacts of ***all*** cases: single-dose azithromycin to all people more than 3kg living in households with an active trachoma case. Rescreen 12 months after the treatment.

3. If prevalence is <5% (non-endemic) treat the individual cases and all household contacts. Screen at 1, 3 and 5 years then cease if prevalence <5% at each screen.

4. If the prevalence is unknown discuss with the community paediatrician CDC or trachoma team.

The medication of choice is azithromycin orally as a single dose. Refer to the *Therapeutic Guidelines* or regional guidelines (eg *CARPA Standard Treatment Manual*).

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

Trachoma

FURTHER INFORMATION

TELEPHONE ADVICE

Contact infectious disease physician, paediatrician, ophthalmologist, CDC/PHU in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines see *Appendix p200*.

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
North Queensland		
Queensland Health	■ Communicable disease and control guidance — Trachoma ■ Primary Clinical Care Manual — Trachoma	Available online

EDUCATIONAL RESOURCES

University of Melbourne — Indigenous Eye Health Unit	Trachoma Includes story kits, posters, audio-visual content, trachoma grading self-directed package	Available online
World Health Organization	SAFE documents	Available online
Fred Hollows Foundation	Indigenous Australia	Available online

KEY REFERENCES AND FURTHER READING

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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 (ABL) is a rabies-like virus carried by several species of bats and flying foxes. 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, with infected bats identified in 2014 and 2015 in the NT.

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 immunoglobulin.

Those at occupational risk of handling bats should be immunised against ABL with the rabies vaccine. NT CDC data shows that between 2011 and 2015 an average of 29 exposures potentially requiring rabies immunoglobulin occur annually and that three quarters of those exposed had not been vaccinated prior to the exposure.

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 is introduced to 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). It 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 may be non-specific, eg fever. Paraesthesias and cranial nerve palsies may be present later in the disease.

Investigations. If possible the bat should be sent for testing to specialised veterinary laboratories. Infected people are usually diagnosed on clinical and historical grounds, confirmed by post-mortem PCR testing of brain tissue.

DIFFERENTIAL DIAGNOSIS

Unusual neurologic illnesses that may require differentiation from lyssavirus infection include Guillain-Barré syndrome and Murray Valley encephalitis. Australia is rabies free, however rabies should be considered in people with a suspicious clinical illness and a history of animal bite in endemic areas (notably South East Asia, including Bali).

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 human rabies immunoglobulin (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 for a minimum of 5 minutes with soap under running water as soon as possible. Proper cleaning of the wound is the most effective way to reduce transmission of the virus
- Apply an antiseptic solution after washing if possible (eg povidone-iodine)
- Cover the wound and seek medical attention immediately. Vaccination is protective against ABL if given promptly.

First Aid if bat saliva in your mouth eyes or nose:

- Flush the area with water
- If already vaccinated, medical attention should be sought as soon as possible as a further 2 doses of vaccine are required.

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.

Bat borne viruses

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 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.

CLINICAL PICTURE

Risk factors. Exposure to sick horses, particularly 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 at state-based reference laboratories using blood and urine. 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 and are only stocked in Queensland.

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

FURTHER INFORMATION

TELEPHONE ADVICE

Contact infectious disease physician, general physician, PHU/CDC in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines (eg *The Australian Immunisation Handbook*) see *Appendix p200*.

National		
Communicable Disease Network Australia (CDNA)	National Guidelines for Public Health Units: ■ Rabies and other lyssavirus ■ Hendra virus	Available online
Northern Territory		
Centre for Disease Control (CDC)	Australian Bat Lyssavirus Post-Exposure Prophylaxis (PEP) 2013	Available online
North Queensland		
Queensland Health	■ Communicable Disease Control Guidance: > Lyssavirus > Hendra virus ■ Primary Clinical Care Manual — Bat bite/scratch	Available online

EDUCATIONAL RESOURCES

NT Centre for Disease Control (CDC)	Fact Sheet — Australian Bat Lyssavirus	Available online
Queensland Health	Bats and human health — fact sheets, other resources	Available online

KEY REFERENCES AND FURTHER READING

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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.

As part of a national immunisation program free hepatitis A vaccine is offered to all Aboriginal and Torres Strait Islander infants born on or after 1 May 2004. In the NT this vaccine is offered routinely at 12 and 18 months of age. Since the introduction of this vaccine for Aboriginal and Torres Strait Islander infants there have been very few cases of hepatitis A acquired in the Northern Territory (Figure 66).

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 child care centres; being intellectually disabled; anal sex; injecting drug use; travel to developing countries or areas of poor sanitation within Australia.

Symptoms and signs. The infectious period extends from 2 weeks before symptoms appear, and until one week after. 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 some days to about a month. Signs include jaundice, tender enlarged liver, dark urine from bilirubinuria, and pale stools.

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

DIFFERENTIAL DIAGNOSIS

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

PRINCIPLES OF MANAGEMENT

Treatment of acute infection is supportive: rest, maintaining hydration and avoiding alcohol. Once positive, there is no need for repeat serology.

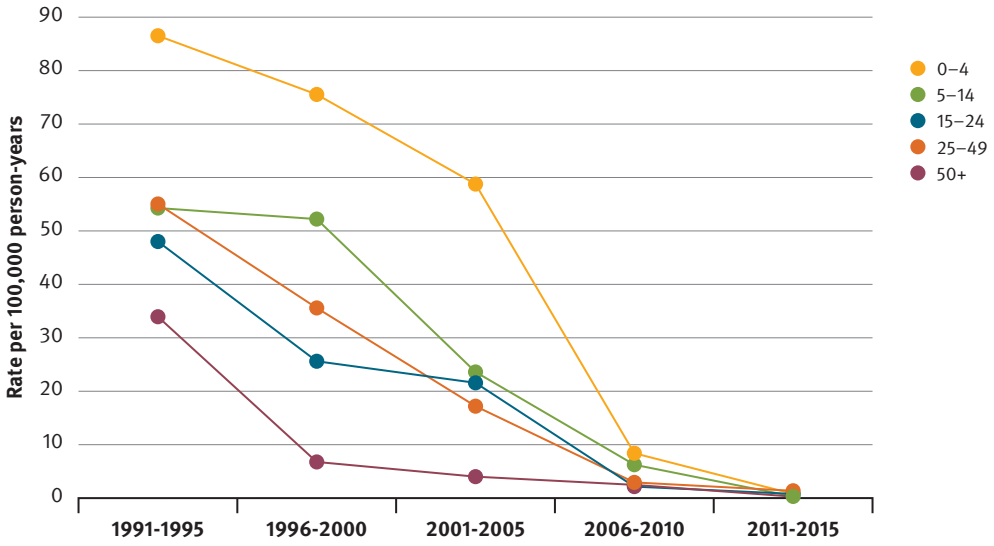

Figure 66: Age-specific incidence rates of hepatitis A in the NT by 5 year time period
Note: There were no cases of locally acquired hepatitis A 2011–2015. Source: Data from NT Notifiable Diseases System (Courtesy P Markey)

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 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 (eg disability or child care 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 Aboriginal and Torres Strait Islander 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 paediatric and rural health professionals, people raised in low prevalence communities who move to live in remote NT communities, travellers to developing 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

TELEPHONE ADVICE

Contact infectious disease physician, specialist physician, CDC/PHU in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines see *Appendix p200*.

National		
Communicable Disease Network Australia (CDNA)	■ National Guidelines for Public Health Units — Hepatitis A ■ Australian notifiable diseases case definitions	Available Onine
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
North Queensland		
Queensland Health	■ Communicable Disease Control Guidance — Hepatitis A ■ Primary Clinical Care Manual — Acute hepatitis A	Available online

EDUCATIONAL RESOURCES

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

KEY REFERENCES AND FURTHER READING

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Poulos RG, Ferson MJ, Orr KJ, McCarthy MA, Botham SJ, Stern JM, et al. Vaccination against hepatitis A and B in persons subject to homelessness in inner Sydney: vaccine acceptance, completion rates and immunogenicity. Australian and New Zealand Journal of Public Health. 2010 Apr;34(2):130-5. DOI: 10.1111/j.1753-6405.2010.00496.x

Mayer CA, Neilson AA. Hepatitis A - prevention in travellers. Australian Family Physician. 2010 Dec;39(12):924-8.

Miller N, Reid S. Factors affecting hepatitis A vaccination uptake among Childcare workers in the Northern Territory. The Northern Territory Disease Control Bulletin. 2001; 8(3):p10-12.

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

Without treatment 15–25% of people living with chronic hepatitis B (CHB) will die from liver cirrhosis or hepatocellular carcinoma — direct consequences of their infection.

Hepatitis B surface antigen was first identified in an Aboriginal patient and was initially known as the Australian antigen. Although CHB sero-prevalence in Australia overall is low at 1%, in high risk groups such as Aboriginal and Torres Strait Islander people and culturally and linguistically diverse communities (CALD), the seroprevalence of CHB can be up to 10%.

The NT has the highest overall percentage of people (1.9%) living with chronic HBV of any primary healthcare network in Australia. The prevalence in Aboriginal people who make up 67% of those living with chronic HBV in the NT is 6.08%. The majority of the remaining 33% are from CALD communities.

Aboriginal people living with chronic HBV in the NT 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.

AETIOLOGY AND PATHOGENESIS

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

CLINICAL PICTURE

Risk factors. HBV is transmitted by body fluids: being born to a CHB infected mother; blood to blood contact particularly in early life; unprotected sex; 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 >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 hepatocellular carcinoma. These outcomes can be prevented with publicly funded treatments.

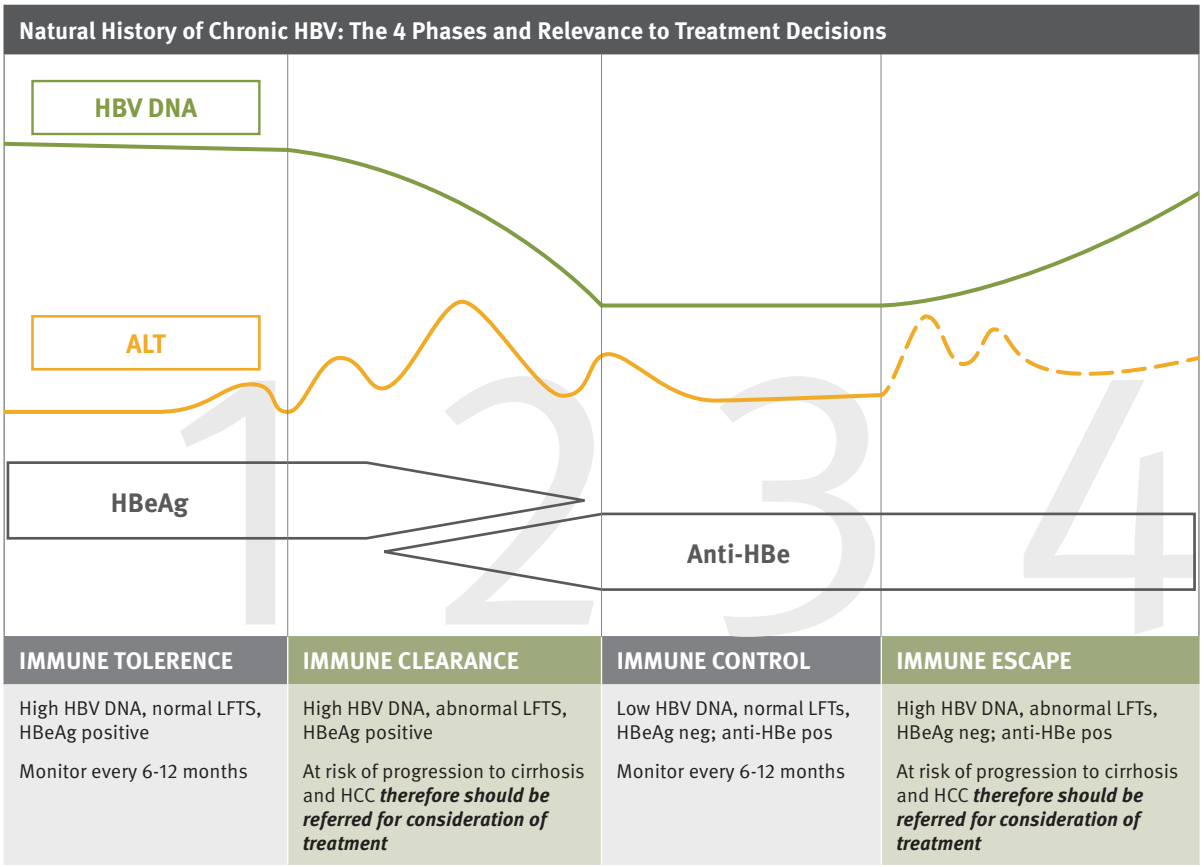
In acute infection and during flares in chronic infection symptoms can include: fever, malaise, nausea and vomiting, abdominal pain, myalgias, rash or arthritis. The acute illness may last for weeks and tiredness can persist for months. The signs of HBV infection include jaundice, tender enlarged liver, dark urine, pale stools. Individuals with chronic HBV may present with signs and symptoms of cirrhosis, either compensated or decompensated, or hepatocellular carcinoma, 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 C IgG, Hepatitis D antigen* and antibody and HIV serology. A baseline ultrasound and non-invasive assessment of fibrosis (Fibroscan® if available otherwise APRI or hepascore) is recommended.

* HDV testing is not necessary in ATSI people as it has never been identified in the NT, but should be considered in CALD communities.

DIFFERENTIAL DIAGNOSIS

Acute hepatitis may result from infection (hepatitis A, B, C, D, E, arboviruses, EBV, CMV, leptospirosis), many toxins (eg alcohol), medications (eg rifampicin, flucloxacillin), or autoimmune disease (eg 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 6/12) may be detected incidentally by screening, or by follow-up after an acute illness.



ALT, alanine transaminase; anti-HBe, antibody to e antigen; HBeAg, hepatitis B e antigen; HBV, hepatitis B virus; HCC, hepatocellular carinoma; LFT, liver function test

Figure 67: Phases of disease in chronic hepatitis B infection and when to consider treatment
Source: ASHM. Decision making in HBV. Sydney: Australasian Society for HIV, Viral Hepatitis and Sexual Health Medicine; 2013. www.ashm.org.au

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, renal function tests and INR and annual HBV viral load as well as a baseline ultrasound and fibroscan.

Establishing the phase of chronic HBV infection 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.

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

Hepatitis B

Six monthly hepatocellular carcinoma screening is recommended for those individuals outlined in Figure 68 below.

Hepatocellular carcinoma (HCC) surveillance (6 monthly ultrasound and AFP) is recommended in these HBsAg + groups:	
■ Aboriginal and Torres Strait Inslander people >50 years ■ Africans >20 years	■ Asian men >40 years ■ Asian women >50 years ■ Patients with cirrhosis ■ HCC family history

Figure 68: HCC recommendations for the Northern Territory
Source: http://courses.ashm.org.au/products/product/1-920773-26-6

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 grant is establishing region and clinic specific core clinical care groups (including streamlined access to a prescriber, trained AHP, ultrasound and fibroscan®) for Aboriginal and Torres Strait Islander people living with CHB in the NT — 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)
- Anyone you are concerned about.

Prevention. Aboriginal and Torres Strait Islander peoples are identified as a high risk group in the National Hepatitis B Strategy (see *Key references and further reading p78*) 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.

See CDC *NT Hepatitis B vaccination and public health guidelines* (under *Management Guidelines p78*) 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 also includes routine immunisation, 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

TELEPHONE ADVICE

Contact specialist physician, CDC/PHU in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines see *Appendix p200*.

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	Available online
American Association for the Study of Liver Diseases	Practice Guidelines	Available online
European Association for the Study of the Liver	EASL Clinical Practices Guidelines on the Management of Hepatitis B Virus Infection	Available online
Asia Pacific Association for the Study of the Liver	Asian-Pacific clinical practice guidelines on the management of hepatitis B	Available online
Northern Territory		
Centre for Disease Control (CDC)	■ Northern Territory Hepatitis B Vaccination and Public Health Guidelines ■ 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 — Hepatitis ■ Women's Business Manual — Hepatitis in pregnancy	Available online
NT Public Health Network (NT PHN)	Northern Territory HealthPathways — Hepatitis B	Available online
Kimberley		
WA Department of Health	Notification of Infectious Diseases and related conditions	Available online
Kimberley Aboriginal Medical Services (KAMS)	Clinical Protocols/Guidelines — Hepatitis B	Available online
North Queensland		
Queensland Health	■ Communicable disease control guidance — Hepatitis B ■ Primary Clinical Care Manual — Acute Hepatitis B	Available online

EDUCATIONAL 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

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

HTLV-1 causes haematological malignancies, inflammatory diseases and predisposes people to other infections.

HUMAN T-CELL LYMPHOTROPIC VIRUS-1 IN NORTHERN AUSTRALIA

Human T-cell leukaemia virus type 1 (HTLV-1) is present in confined populations, particularly in Japan, the Caribbean, South America and central Africa. High rates of HTLV-1 infection have been reported in a community in the Kimberley region of WA and among adults admitted to Alice Springs Hospital from communities in WA and SA. HTLV-1 is highly endemic in parts of Central Australia where seropositivity rates among hospitalised adults exceed 30% and more than 40% of adults were infected in one remote community surveyed.

Infection rates are lower to the north of central Australia and the virus is uncommon in Arnhem Land and Darwin rural communities. Prevalence in the adjacent areas of Queensland is extremely rare.

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. HTLV-1 infection is often asymptomatic but predisposes to other infections including crusted scabies, symptomatic strongyloidiasis, and mycobacterial infections. Recent studies in the NT suggest a link to bronchiectasis and progressive lung disease. After many years of infection it can (rarely) cause:

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

CLINICAL PICTURE

Risk factors include exposure to the transmission routes described above, and belonging to particular ethnic 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. The true burden of HTLV-1 disease in a community setting has not been determined. However, data derived from hospitals in Japan and the Caribbean suggest that the life-time risk of developing adult T-cell leukaemia/lymphoma and HTLV-1 associated myelopathy is 1–5% and 0.3–4%, respectively. No data are available for other HTLV-1 associated diseases.

Adult T-cell leukaemia/lymphoma is typically highly aggressive, resulting in a median survival of less than ten months in 75% of cases. Lymphadenopathy is common and many patients also have hepatosplenomegaly and skin lesions. Individuals with adult T-cell leukaemia/lymphoma are immunosuppressed and subject to opportunistic infections. Hypercalcaemia is a common complication.

HTLV-1 associated myelopathy typically presents as a slowly progressive, symmetrical, spastic paresis involving the lower limbs. Difficulty walking and bladder dysfunction with repeated urinary tract infections are often the initial symptoms. 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, eyelids, 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 NT suggest a link between HTLV-1 and bronchiectasis and progressive lung disease. In these circumstances management plans for the bronchiectasis need emphasis, including ensuring all vaccinations are up to date, when to use antibiotics and access to physiotherapy support.

Investigations. Diagnosis is made by positive serology. All patients with crusted scabies and strongyloidiasis should be tested for HTLV-1.

DIFFERENTIAL DIAGNOSIS

Differential diagnosis depends on the presenting symptoms. The malignancy can be confused with other malignancies (eg cutaneous T-cell lymphomas, mycosis fungoides) or infectious diseases, and the myelopathy with various genetic syndromes, or infectious diseases such as late syphilis. An important differentiating question is whether the patient is from a known endemic group with the above symptoms. HTLV-1 is also associated with other malignant and inflammatory conditions.

PRINCIPLES OF MANAGEMENT

Adult T-cell leukaemia/lymphoma may respond to chemotherapy and TB and strongyloidiasis should be excluded **before** treatment commences. Steroids and plasmapheresis may induce a transient response in the myelopathy. Infection control measures should be stressed to avoid further infections eg safe sex, 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. Although there is no current treatment for HTLV-1 infection, treating complications such as scabies, strongyloidiasis, skin and urinary tract infections make it safer to live with HTLV-1.

HTLV-1 must be requested in screening tests following needlestick injuries involving an Aboriginal person from Katherine or the southern region of the NT. The Royal Darwin Hospital Infection Control Biohazard Injury Management manual offers more information.

> HTLV-1 is a notifiable condition to be reported by CLINICIANS and LABORATORIES in the Northern Territory. Report cases to the local Centre for Disease Control.

FURTHER INFORMATION

TELEPHONE ADVICE

Contact infectious disease physician, CDC/PHU in your local jurisdiction.

MANAGEMENT GUIDELINES

Northern Territory		
Centre for Disease Control (CDC)	Notifiable diseases	Available online

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Influenza

Remote communities are often among the first geographic locations within Australia to experience influenza outbreaks.

INFLUENZA IN NORTHERN AUSTRALIA

Influenza occurs throughout the year in the Top End but there are usually 2 peaks of activity (Figure 69). This pattern is different from that seen in temperate parts of Australia and the timing of epidemics is less predictable. 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 are often 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 CDC and collect appropriate specimens for viral culture. The World Health Organisation Collaborating Centre for Influenza Reference and Research in Melbourne provides free influenza culture for specimens from the Top End.

There are a number of online national surveillance systems. 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.

PRINCIPLES OF MANAGEMENT

Treatment is usually supportive with early use of Oseltamivir recommended for patients at risk of severe disease. Refer to the *Influenza infection, CDNA National Guidelines for Public Health Units* (following) for further information.

> Influenza is a nationally notifiable condition to be reported by LABORATORIES. Report cases to the local Centre for Disease Control/Public Health Unit. CLINICIANS should report possible outbreaks to the local Centre for Disease Control/Public Health Unit.

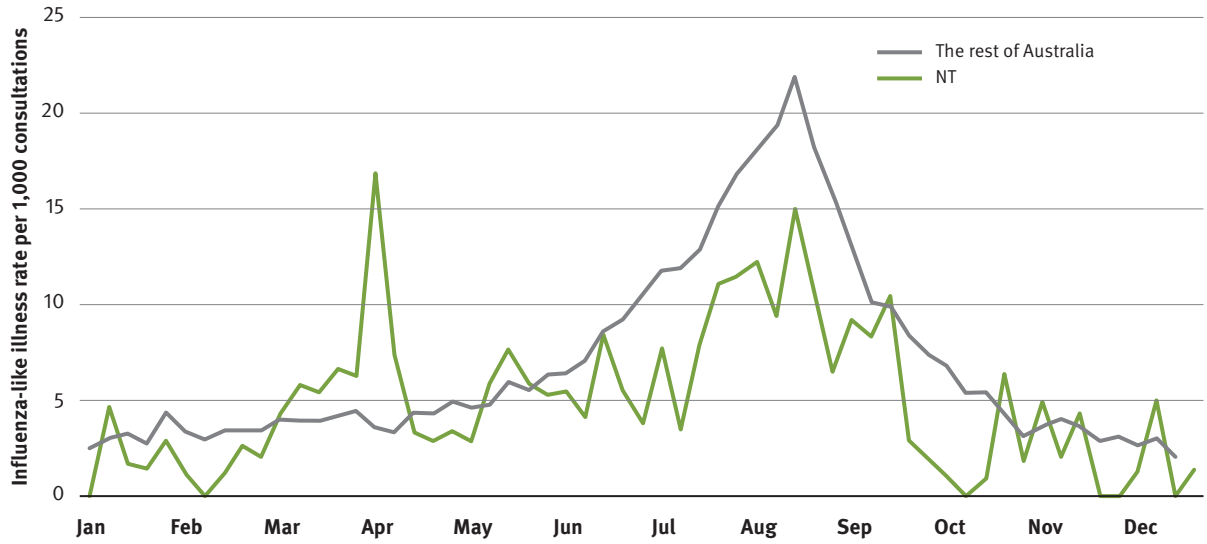

Figure 69: Influenza activity in the Top End compared to Australia as a whole, based on GP consultations for influenza-like illnesses.
Source: The Australian Sentinel Practices Research Network (ASPREN) 2016. http://www.aspren.com.au/

FURTHER INFORMATION

TELEPHONE ADVICE

Contact specialist physician, CDC/PHU in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines see *Appendix p200*.

National		
Australian Government, Department of Health	■ Flu (influenza) immunisation service ■ 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		
Centre for Disease Control (CDC)	Influenza	Available online
Kimberley		
WA Department of Health	Influenza — Statutory notification alert, Public health management	Available online
North Queensland		
Queensland Health	Communicable disease control guidance — Influenza	Available online

EDUCATIONAL RESOURCES

NT Centre for Disease Control (CDC)	Fact sheet — Influenza	Available online
Queensland Health	Fact sheet	Available online

KEY REFERENCES AND FURTHER READING

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SECTION 3 — INFESTATIONS, PARASITIC AND FUNGAL INFECTIONS

Cryptococcal meningitis and pneumonia

Cryptococcus neoformans variant gattii is a yeast like fungus that causes respiratory and neurological illness.

CRYPTOCOCCAL MENINGITIS AND PNEUMONIA IN NORTHERN AUSTRALIA

***Cryptococcus neoformans* is known for causing disease in immunosuppressed people. However, where *Cryptococcus neoformans var. gattii* is endemic in Northern Australia, particularly western Arnhem Land, the disease can occur in apparently immunocompetent people.**

In other parts of Australia its distribution is associated with that of the River Redgum (*Eucalyptus camaldulensis*) and is thought to be transmitted to humans via contact with these trees. While River Redgums are not common in the Top End, the fungus has been grown from various eucalypts (including the Northern Woollybutt, *Eucalyptus miniata*) and other trees in Arnhem Land.

AETIOLOGY AND PATHOGENESIS

Cryptococcus neoformans causes infection via inhalation, then dissemination, commonly to the central nervous system. The *Cryptococcus neoformans var gattii* variety is mostly restricted to tropical and subtropical locations. Disease incidence Australia wide is 0.61 per million but in the Northern Territory it is 6.5 per million.

CLINICAL PICTURE

Symptoms and signs.

- Early symptoms — non-specific, can include headache and subtle changes to activity. Fever is an unreliable sign
- Respiratory — can be asymptomatic or present with symptoms of lower respiratory tract infection. Pulmonary lesions are visible on chest X-ray
- Neurological — headache, neck stiffness, impaired consciousness, cerebellar defects, limb weakness, seizures, cranial nerve defects, papilloedema.

Symptoms are often indolent. The most common presentation of recent NT 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 86 cases in Australia between 2000 and 2007, showed that the median time to diagnosis from the first symptoms was 45 days. Most cases (85%) presented with central nervous system involvement, 52% had both neurological and lung symptoms, and 12% presented with lung involvement only. Of the central nervous system infections meningoencephalitis (89%) and brain involvement with cryptococommas (62%) were common. The Northern Territory cases have clustered around the western Arnhem Land communities of Maningrida and Gunbalanya.

Investigations. Cryptococcal antigen in cerebrospinal fluid and serum is both sensitive and specific. The antigen is positive in serum in nearly all cases of cryptococcal meningitis and two thirds of those with cryptococcal pneumonia. Chest X-ray will usually show the solid mass lesion of pulmonary cryptococcoma. Lumbar puncture is almost always diagnostic in central nervous system disease. On cerebrospinal fluid microscopy, encapsulated yeasts may be seen with an India ink stain.

DIFFERENTIAL DIAGNOSIS

Consider other causes of subacute meningitis especially tuberculous meningitis. CT and lumbar puncture are mandatory to exclude cryptococcal meningitis in suspected cases.

PRINCIPLES OF MANAGEMENT

Management focuses on antimicrobial therapy, intracranial pressure monitoring and management of hydrocephalus, identification of cerebral cryptococcomas, and management of pulmonary lesions. Initial treatment is with IV amphotericin and 5-flucytosine for 4–6 weeks. Patients need continuing 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. Treatment in the setting of HIV infection is slightly different and cure is less certain. Regular clinical follow-up is critical.

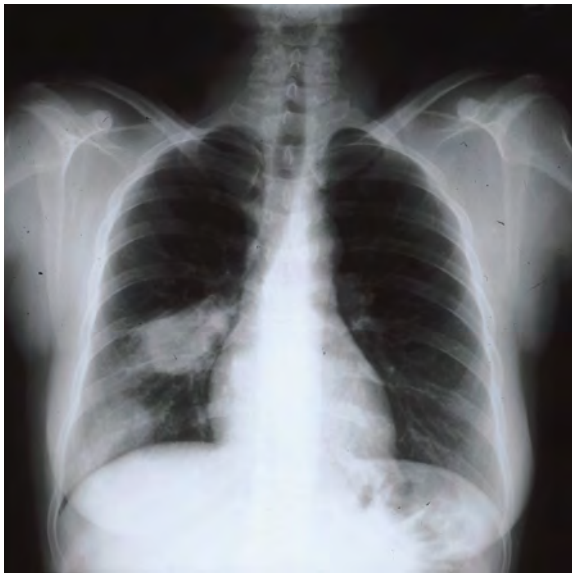

Figure 70: Cryptococcoma in lung
Source: Bart Currie — Menzies School of Health Research

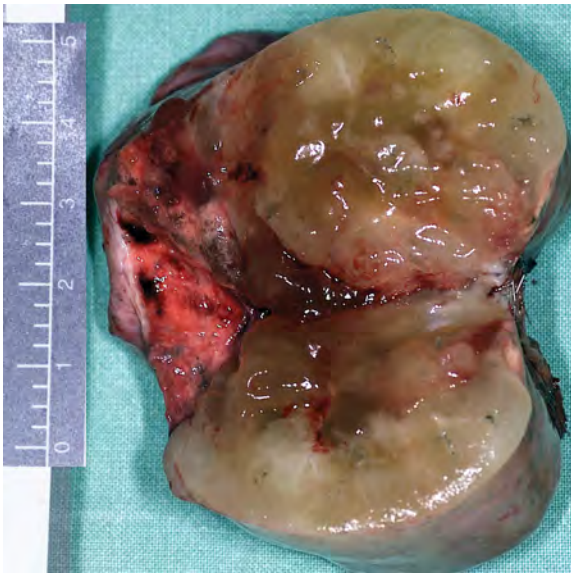

Figure 71: Cryptococcoma surgically excised from lung
Source: Bart Currie — Menzies School of Health Research

FURTHER INFORMATION

TELEPHONE ADVICE

Contact infectious disease physician, CDC/PHU in your local jurisdiction.

KEY REFERENCES AND FURTHER READING

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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 Aboriginal and Torres Strait Islander 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.

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. In pale skin the advancing edge is often erythematous, while in dark skin there is usually just a silvery scale. 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. Superficial bleeding is common when scraping as the *Trichophyton rubrum* scales are very adherent.

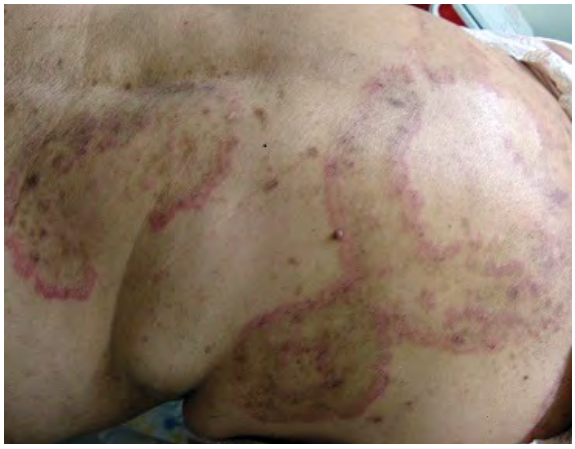

Figure 72: Tinea corporis
Source: Ian McCrossin

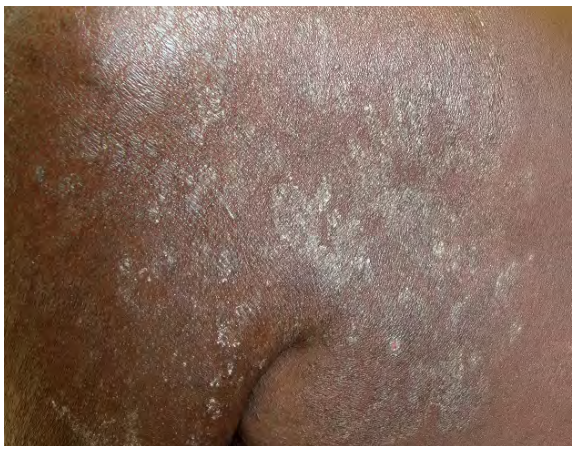

Figure 73: Tinea corporis
Source: Ian McCrossin

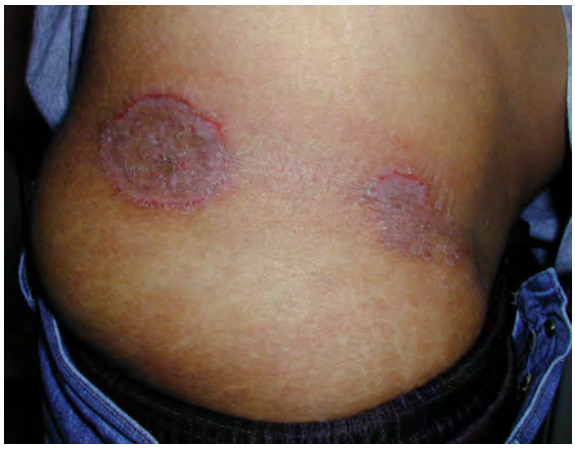

Figure 74: Tinea corporis discrete lesions
Source: Bart Currie — Menzies School of Health Research

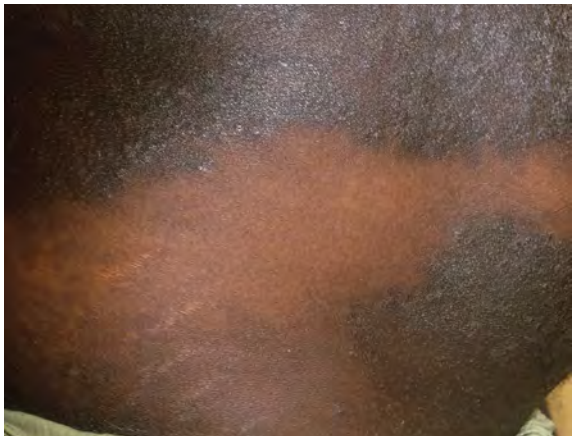

Figure 75: Tinea corporis extensive pigmentation
Source: Bart Currie — Menzies School of Health Research

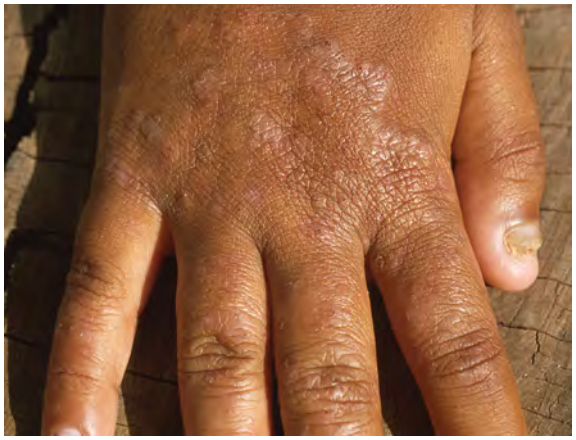

Figure 76: Tinea – hand and nails
Source: Bart Currie — Menzies School of Health Research

Tinea corporis

DIFFERENTIAL DIAGNOSIS

The main differential diagnosis is pityriasis versicolor caused by *Malassezia furfur*, otherwise known as the 'lace handkerchief'. Other possibilities include eczema, psoriasis, kava affected skin, extensive scabies or crusted scabies and leprosy.

PRINCIPLES OF MANAGEMENT

Smaller lesions respond to topical anti-fungals such as terbinafine or miconazole applied over lesions and to 3cm beyond the active margin.

Extensive lesions require oral anti-fungals. While griseofulvin was the standard therapy for many years, this drug is only fungistatic 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 LFTs and FBC before terbinafine treatment. Abnormal liver function may require dose modification and monitoring.

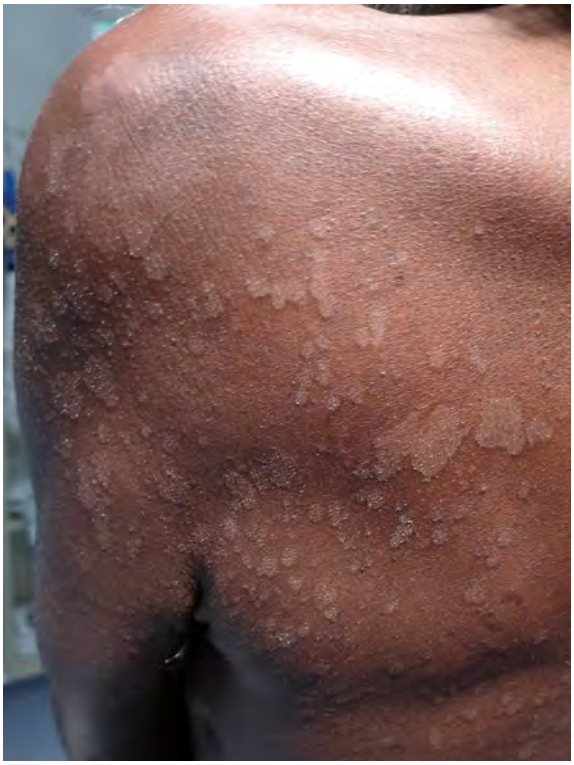

Figure 77: Tinea (pityriasis) versicolor — not *T. rubrum* (differential diagnosis)
Source: Bart Currie — Menzies School of Health Research

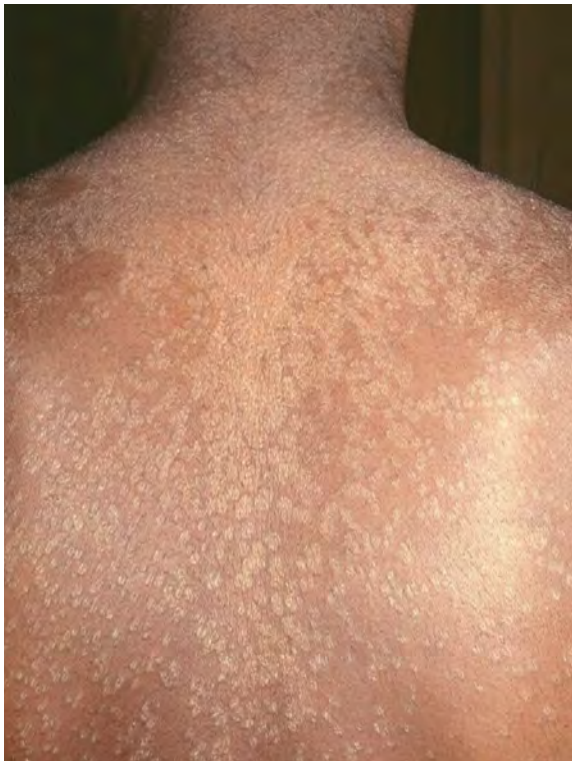

Figure 78: Tinea (pityriasis) versicolor — not *T. rubrum* (differential diagnosis)
Source: Bart Currie — Menzies School of Health Research

FURTHER INFORMATION

TELEPHONE ADVICE

Contact dermatologist, infectious disease physician in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines see *Appendix p200*.

Northern Territory		
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
North Queensland		
Queensland Health	Primary Clinical Care Manual — Tinea/ringworm	Available online

EDUCATIONAL 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

KEY REFERENCES AND FURTHER READING

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Onychomycosis

Fungal infections of the finger and toe nails are common in tropical climates.

ONYCHOMYCOSIS IN NORTHERN AUSTRALIA

Tinea unguium or onychomycosis is a dermatophyte infection of the finger or toe nails and are usually secondary to other fungal infections, such as tinea corporis (*p87*). 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. 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/or scrape material from under the distal nail 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.

PRINCIPLES OF MANAGEMENT

Explain to patients that three months (fingernails) to six months (toenails) oral treatment is often required. Oral terbinafine is the treatment of choice. As treatment is needed for more than 2 weeks, liver function and FBC should be checked before and during treatment.

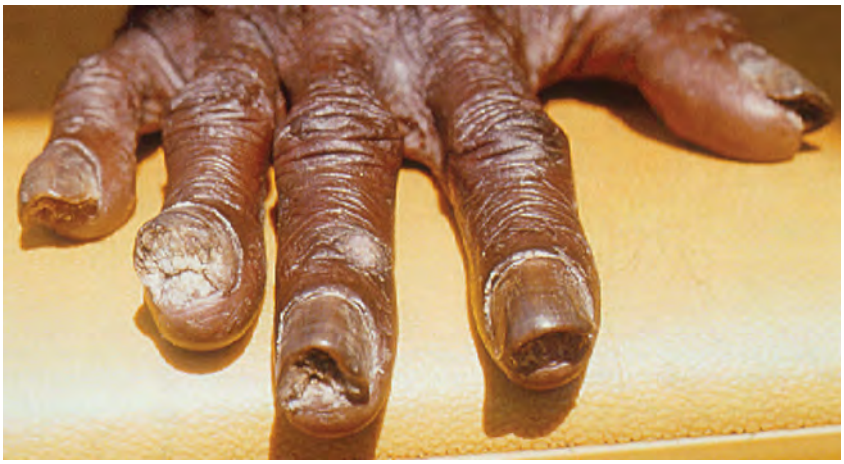

Figure 79: Onychomycosis — nail tinea with *Trichophyton rubrum*
Source: Bart Currie — Menzies School of Health Research

FURTHER INFORMATION

TELEPHONE ADVICE

Contact dermatologist, infectious disease physician in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines see *Appendix p200*.

KEY REFERENCES AND FURTHER READING

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Scabies

Skin infection with the scabies mite is associated with secondary bacterial skin infections that can lead to life-long kidney and heart disease.

SCABIES IN NORTHERN AUSTRALIA

Scabies and associated streptococcal skin infections are the two most common and important skin conditions in Aboriginal and Torres Strait Islander people in Northern Australia. The high prevalence of streptococcal skin infection contributes to the extremely high rates of acute post-streptococcal glomerulonephritis and rheumatic fever.

Currently community surveys show high but variable prevalence rates ranging from 30–50% in children under the age of 15, with approximately 70% of infants being affected in the first year of life. Adults have levels of at least 30% at any one time, although they generally have milder clinical manifestations.

One of the important features of scabies is that it provides an ideal niche for bacterial skin infections, particularly *Streptococcus pyogenes* and *Staphylococcus aureus*. Community surveys have demonstrated that up to 45% of children have streptococcal skin sores. However, the contribution that scabies makes to skin sores varies in different seasonal, environmental and geographical circumstances.

AETIOLOGY AND PATHOGENESIS

Scabies is caused by a parasitic mite, *Sarcoptes scabiei,* which is transmitted from person to person through close contact. The gravid female mite burrows and deposits about two to three eggs a day in the stratum corneum of the skin. The nymphs emerge as adults on the surface of the skin after a series of moults which takes about 2 weeks. The mature mites then mate and reinvade the skin of the same or another host.

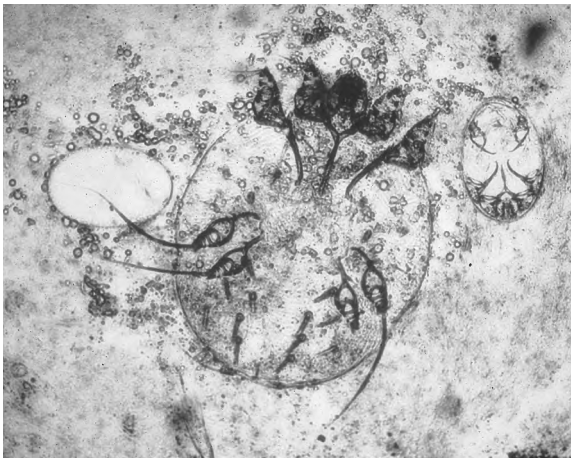

Figure 80: Scabies female with eggs
Source: Bart Currie — Menzies School of Health Research

Initial infestation is asymptomatic. After 4–6 weeks the host becomes sensitised to the excreta of the mites and an itch and rash develops. With subsequent reinfestation the host will immediately develop a hypersensitivity reaction and become symptomatic.

Crusted scabies (also known as Norwegian scabies) is a severe form of the disease in people who usually have some form of immune deficiency, including the elderly. In international studies the cause of the deficiency is usually well documented, such as HIV infection. However, in the NT the underlying immune problems may be more complex and subtle as they are rarely identified, except in Central Australia where HTLV-1 (*p79*) is a common link. People with crusted scabies cannot contain mite replication and become infested with thousands of them, developing initially scaly skin, then a thick, crusted skin in response. They are highly infectious to others and highly susceptible to reinfestation.

CLINICAL PICTURE

Risk factors. Scabies is transmitted through direct contact with an infected person. Poverty and overcrowded living conditions are risk factors for scabies. Young children and people with crusted scabies are ‘key transmitters’ as they carry large numbers of mites. Always consider whether scabies is the underlying cause of skin sores.

Symptoms and signs. The distribution and severity of scabies infection 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 lesions. Older children and adults often only have a small number of lesions.

The most common symptom is an itchy rash. In young children, the lesions may be from head to toe particularly including pustular blisters on the palms of their hands and soles of their feet. These are not always infected with bacteria but are caused by the immune response to the mite. Older children and adults usually have lesions at the wrists, in the inter-digital space between fingers and toes, the buttocks and around the ankles. Scabies lesions may also be found on the head in older children and adults, although this is much less common. Lesions are often crusted, pustular or weeping due to secondary streptococcal infection.

The rash from crusted scabies can vary. Mild cases may have localised patches on the buttocks, upper thighs, upper arms and on the dorsum of the hands and the feet. Severe cases will literally be covered from head to toe with thick, elevated crusted lesions, which may also have fissures. Crusted scabies is often not itchy and many people are not diagnosed or are misdiagnosed as dermatitis. Clinicians may be alerted to a case by seeing recurrent presentations of scabies in a child from the same household.

Investigations. Investigations are not routinely required because the clinical picture is usually pathognomonic. Scabies mites can be identified from burrows, often between fingers, and demonstrated under a microscope. However, burrows are hard to see on dark skin. A swab of associated skin sores will usually grow streptococcus and often staphylococcus.

It is important to confirm the diagnosis in crusted scabies. Put on gloves and scrape some crusted skin lesions into a pathology jar, and send for microscopy. Wash your hands afterwards as crusted scabies is very infectious. Investigate to exclude underlying immune deficiencies, such as renal failure, systemic lupus erythematosus (SLE) and Human T-cell lymphotropic virus type 1(HTLV-I). Recommended tests are in the CDC *Guidelines for Community Control of Scabies, Skin Sores, Tinea and Crusted Scabies in the Northern Territory* — see *Management guidelines p97*.

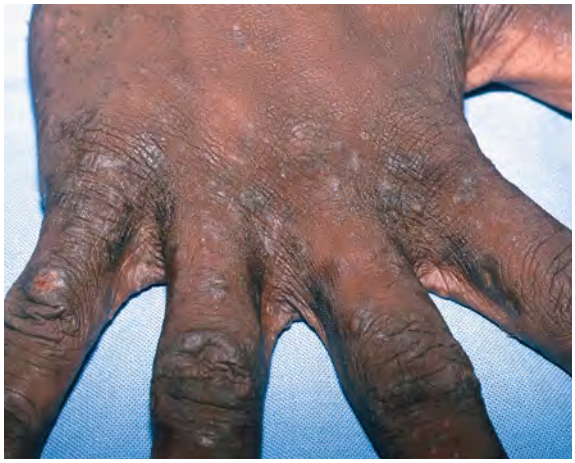

Figure 81: Scabies
Source: Bart Currie — Menzies School of Health Research

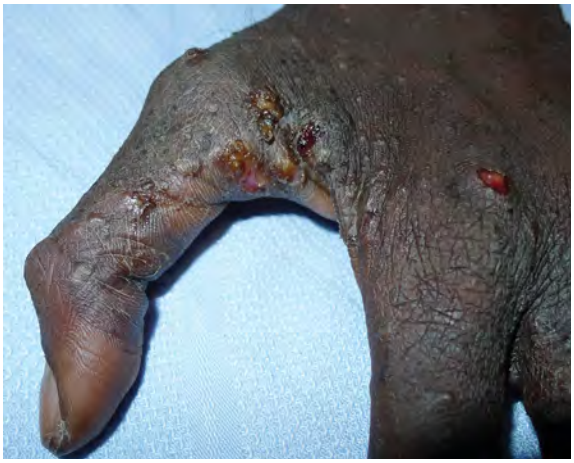

Figure 83: Sixteen year old with infected scabies
Source: Bart Currie — Menzies School of Health Research

Figure 82: Scabies with streptococcal pyoderma
Source: Bart Currie — Menzies School of Health Research

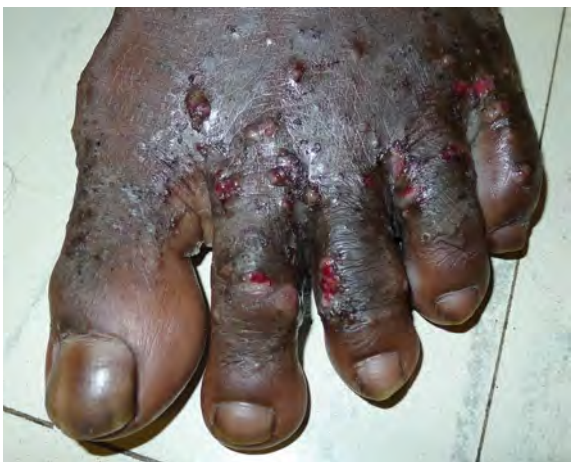

Figure 84: Sixteen year old with infected scabies
Source: Bart Currie — Menzies School of Health Research

Scabies

DIFFERENTIAL DIAGNOSIS

The differential diagnosis includes eczema, mild cases of psoriasis and contact dermatitis. For crusted scabies, particularly milder cases, the differential diagnosis is broader and it is frequently misdiagnosed. The conditions to consider include eczema, psoriasis, fungal infection and kava dermatitis.

PRINCIPLES OF MANAGEMENT

For treatment details refer to scabies and skin sore guidelines in your region (eg *CDC*, *CARPA Standard Treatment Manual*). The mainstay of treatment is permethrin cream, two treatments separated by one to two weeks. The individual *and all their closely related contacts* need to be treated. In Aboriginal and Torres Strait Islander communities this includes everyone within the household, in particular adults who share a bed with or have other close contact with an affected child. As many children have lesions on their head and behind their ears, the recommendations for treatment include the head and neck. Permethrin can be given at the same time as benzathine benzylpenicillin or cotrimoxazole for streptococcal infection. Oral ivermectin can be used if topical scabies treatment fails.

Community wide treatment of mangy dogs is unnecessary to prevent reinfection as the dogs have different types of mites that are unlikely to infect people. However, dog mites can cause an irritating dermatitis on close human contacts. The NGO *One Disease*, has developed guidelines for support of households and families of patients with crusted scabies — see *Educational resources p97*.

Crusted scabies involves both topical treatment with permethrin and a keratolytic cream to soften the crust and allow permethrin to penetrate. Patients also require oral ivermectin. The number of doses and the time between doses is determined by the severity of the crusted scabies and clinicians should refer to the Centre for Disease Control (CDC) and Royal Darwin Hospital crusted scabies protocols or discuss with an infectious diseases physician. Cases usually need treatment in hospital.

The protocol for crusted scabies also includes treatment of the entire household. Additionally, environmental health officers and *One Disease* can work with family members to provide education about household cleaning and f acilitate the treatment of contacts. The main complication of crusted scabies is septicaemia. A study done 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 fissures in the cracked skin. Aggressive antibiotic therapy has 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 with scabies cream. Mass drug administration with ivermectin has been attempted in one remote NT community, with initial benefits but the return to prior infection rates. Regular surveillance of young children before and after such community treatment days is important.

> 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.

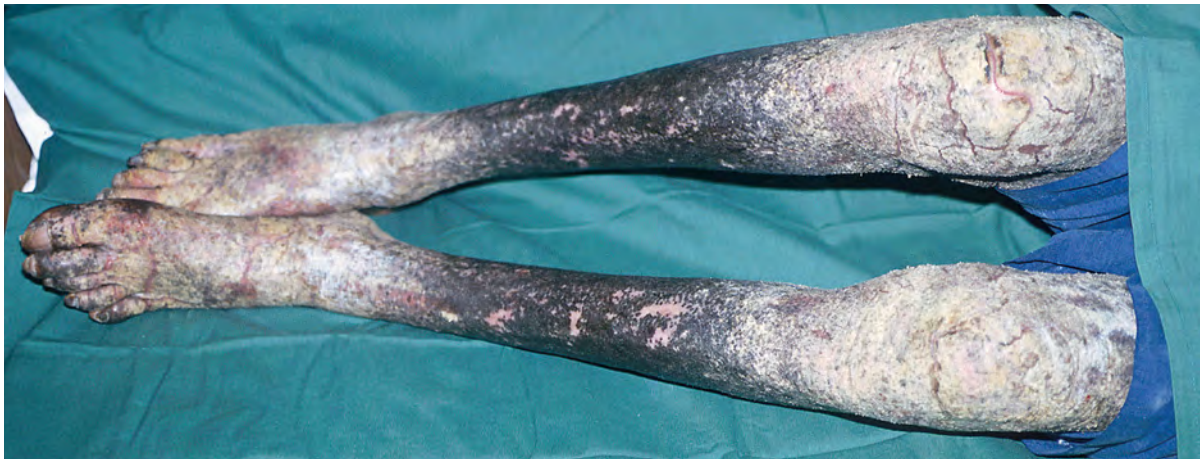

Figure 85: Crusted scabies
Source: Bart Currie — Menzies School of Health Research

Figure 86: Crusted scabies
Source: Bart Currie — Menzies School of Health Research

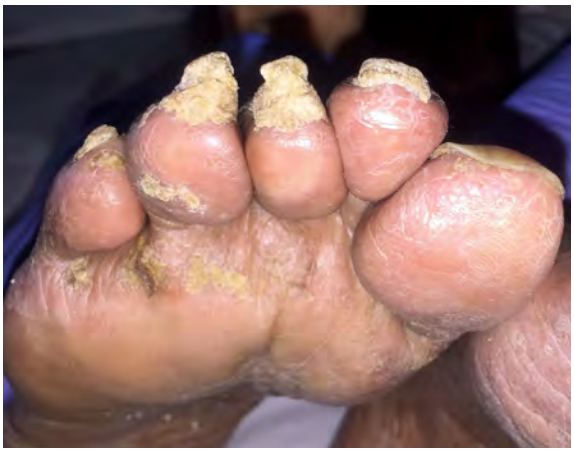

Figure 88: Crusted scabies with residual disease under nails
Source: Bart Currie — Menzies School of Health Research

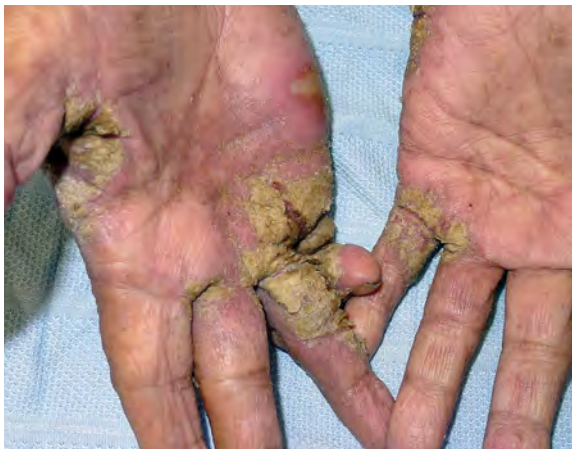

Figure 87: Crusted Scabies with fissures
Source: Bart Currie — Menzies School of Health Research

Scabies

FURTHER INFORMATION

TELEPHONE ADVICE

Contact infectious disease physician, dermatologist, CDC/PHU in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines see *Appendix p200*.

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		
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 ■ Notifiable diseases	Available online
Remote Primary Health Care Manuals (RPHCM)	CARPA Standard Treatment Manual — Scabies	Available online
Kimberley		
Kimberley Aboriginal Medical Services (KAMS)	Clinical Protocols/Guidelines — Skin infections/Scabies	Available online
North Queensland		
Queensland Health	Primary Clinical Care Manual — Scabies	Available online

EDUCATIONAL RESOURCES

One Disease	Resources	Available online

KEY REFERENCES AND FURTHER READING

May PJ, Tong SYC, Steer AC, Currie BJ, Andrews RM, Carapetis JR, 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 Mar;24(3):280-93. DOI: 10.1111/tmi.13198

Kearns TM, Speare R, Cheng AC, McCarthy J, Carapetis JR, Holt DC, Currie BJ, Page W, Shield J, Gundjirryirr R, Bundhala L. Impact of anilvermectin mass drug administration on scabies prevalence in a remote Australian Aboriginal community. PLoS Neglected Tropical Diseases. 2015 Oct;9(10):e0004151. DOI: 10.1371/journal.pntd.0004151

Hildebrand F, Pretty H, Saroukos C, Thompson S, Ganambarr R, Maymuru B, Bray M, Gunasekara D, Wills A, Prince S. One Disease update: tackling scabies and crusted scabies across the Top End. The Northern Territory Disease Control Bulletin. 2015;22(3)15-17.

Lokuge B, Kopczynski A, Woltmann A, Alvoen F, Connors C, Guyula T, Mulholland E, Cran S, Foster T, Lokuge K, Burke T. 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 Jun 16;200(11):644-8.

Kearns TM, Andrews R, Speare R, Cheng A, McCarthy J, Carapetis J, Currie B, Page W, Shield J. Prevalence of scabies and strongyloidiasis before and after MDA in a remote Aboriginal community in Northern Territory, Australia. International Journal of Infectious Diseases. 2014 Apr 1;21:252. DOI: https://doi.org/10.1016/j.ijid.2014.03.944

Einsiedel LJ, Pepperill C, Wilson K. Crusted scabies: a clinical marker of human T-lymphotropic virus type 1 infection in central Australia. The Medical Journal of Australia. 2014 Jun 16;200(11):633-4.

Whitehall J, Kuzulugil D, Sheldrick K, Wood A. Burden of paediatric pyoderma and scabies in North West Queensland. Journal of Paediatrics and Child Health. 2013 Feb;49(2):141-3. DOI: 10.1111/jpc.12095

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 Aboriginal and Torres Strait Islander communities. In 1994–1995 it was estimated that 50% of all primary school children had 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 and bonded onto the hair shaft close to the scalp. They are yellow to white and are difficult to remove. The eggs hatch in 7–10 days. They need high temperature and humidity and die quickly on brushes, hats, pillows or furniture. The lice are highly contagious and usually transferred by head to head contact. Nits that are more than 1cm from the scalp are dead or empty.

CLINICAL PICTURE

Infestation may be asymptomatic, although it often causes an itchy scalp. Look at the scalp/ hair behind the ears with a strong light to detect the nits. Live lice are difficult to see as they quickly move away when disturbed. To find them apply thick white hair conditioner to dry hair and repeatedly comb each section of hair with a fine toothed 'nit' comb. The conditioner stuns the lice for about 20 minutes allowing them to be combed out onto tissue paper.

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

The mainstay of management has been treatment of the scalp with an insecticide to kill the lice, followed by manual removal of live lice with a nit comb. However insecticides are relatively toxic and resistance has become an increasing problem.

Current recommended treatment is with an occlusive product containing dimeticone that coats the lice and smothers them. Newer products that contain nerolidol kill the lice. The product is applied to clean dry hair covering each hair from root to tip. Comb in sections with a fine-tooth comb, wiping on a tissue to check for lice and nits. Check in one week using a fine-tooth comb with either conditioner or the occlusive product. The treatment can be repeated every 7 days.

In all cases:

- 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

TELEPHONE ADVICE

Contact paediatrician, CDC/PHU in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines see *Appendix p200*.

Northern Territory		
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
Kimberley		
Kimberley Aboriginal Medical Services (KAMS)	Clinical Protocols/Guidelines — Skin infections/Head lice	Available online
North Queensland		
Queensland Health	Primary Clinical Care Manual — Head lice/nits	Available online

EDUCATIONAL RESOURCES

NT Centre for Disease Control (CDC)	■ Fact Sheet — Head Lice and Nits ■ Kid's nit booklet ■ Head lice advice posters	Available online
Queensland Health	Fact Sheet — Head Lice	Available online

KEY REFERENCES AND FURTHER READING

Woolcock K. Head lice and treatment failure. Australian Pharmacist. 2014 Feb;33(2):46.

Maxwell M, Crawford B, Rose V. Nitbusters: lessons from a school-based intervention study to reduce head lice in a disadvantaged community. Health Promotion Journal of Australia : Official Journal of Australian Association of Health Promotion Professionals. 2014 Apr;25(1):67-8. DOI: 10.1071/HE13092

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 Nov;15(4):181.

Paederus australis (the 'acid beetle') and other stings

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 river banks, 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 5–10mm long with brightly coloured sections of blue/black and orange. It produces a toxic alkaline substance, perderin, which is released by 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 to occur and patients may not associate the symptoms with the beetle.

CLINICAL PICTURE

Symptoms and signs. *Paederus australis* causes an irritant contact dermatitis characterised by linear streaks and 'kissing lesions' where two skin surfaces are in contact with each other.

The rash appears after about 24 hours and subsequently blisters, becoming very painful. After about a week the lesions become dry and itchy. Healing occurs at 10–12 days leaving a dark pigmented area that may persist for 2–3 weeks. It has also been reported to cause acute conjunctivitis with marked oedema of the eyelids.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include herpes simplex or zoster, impetigo, other forms of irritant or contact dermatitis, conjunctivitis, and the periorbital swelling of acute glomerulonephritis.

Other common stings can be seen following.

Figure 89: Acid or whiplash rove beetle *Paederus australis*
Source: Bart Currie — Menzies School of Health Research

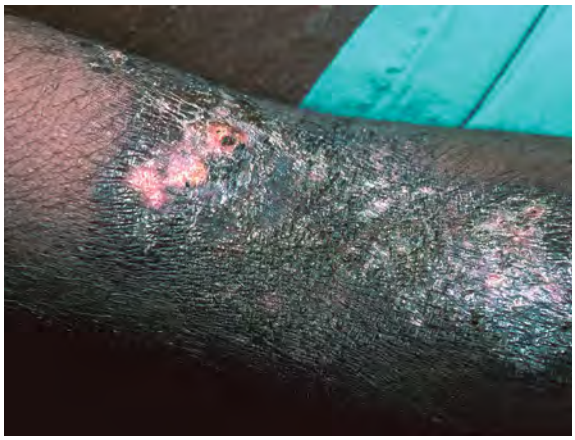

Figure 90: Acid beetle burn — kissing lesion
Source: Bart Currie — Menzies School of Health Research

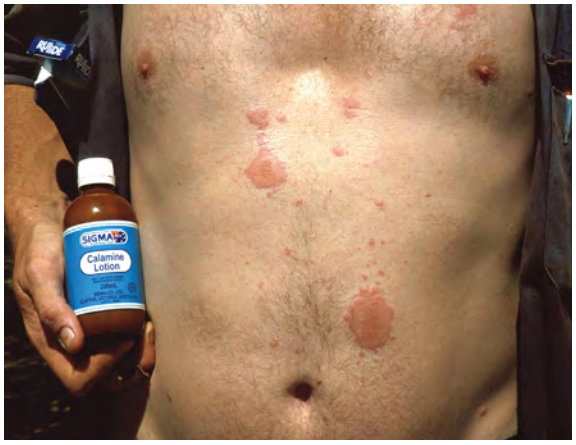

Figure 91: Mango sap rash
Source: Bart Currie — Menzies School of Health Research

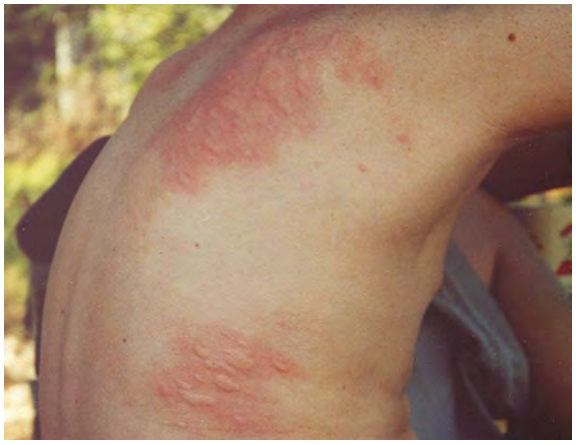

Figure 92: Stings from Freshwater mangrove itchy caterpillar *Euproctis lutea*
Source: Bart Currie — Menzies School of Health Research

Figure 93: Caterpillar *Euproctis lutea*
Source: Bart Currie — Menzies School of Health Research

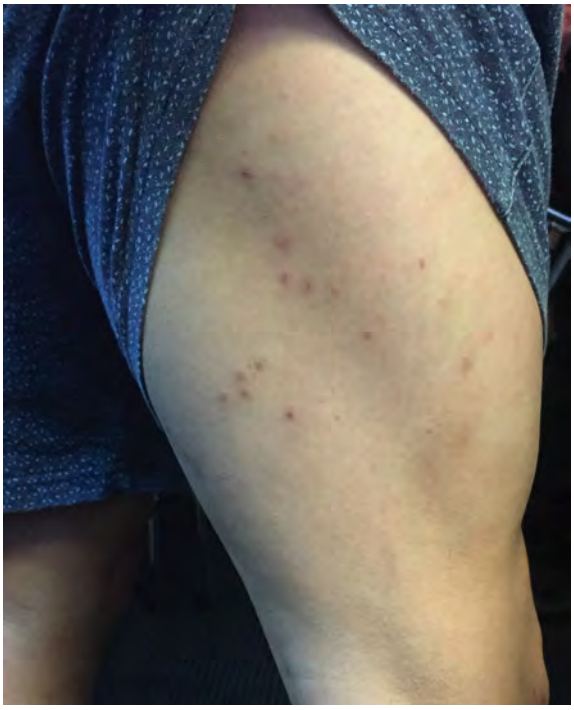

Figure 94: Biting midges (Culicoides) in a fishing doctor new to the Top End
Source: Bart Currie — Menzies School of Health Research

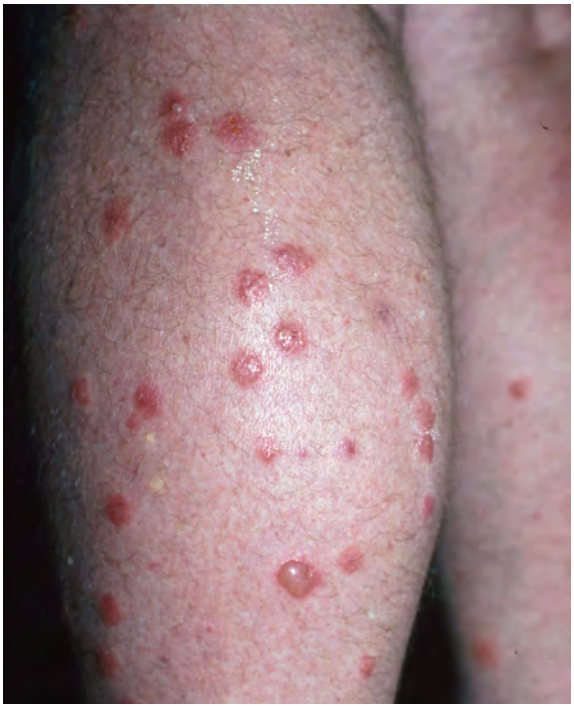

Figure 95: Biting midges (Culicoides) in a fishing scientist new to the Top End
Source: Bart Currie — Menzies School of Health Research

Paederus australis (the 'acid beetle') and other stings

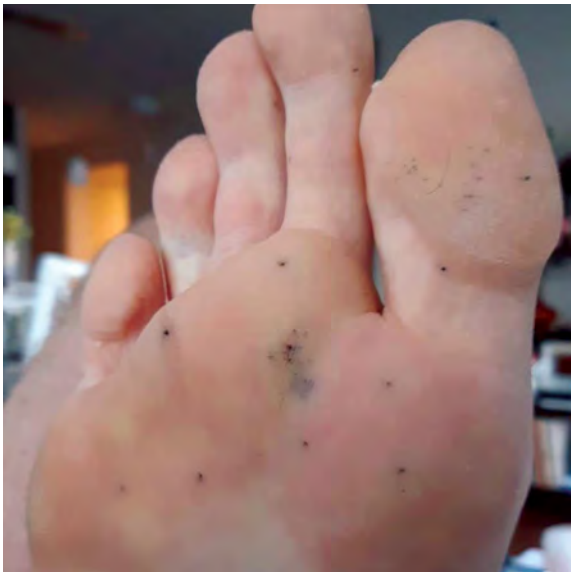

Figure 96: Sea urchin spines
Source: Bart Currie — Menzies School of Health Research

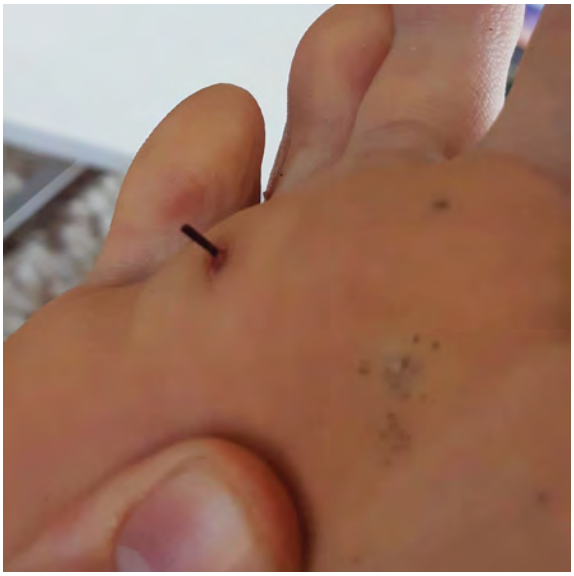

Figure 97: Sea urchin spines extracted from skin after 17 days
Source: Bart Currie — Menzies School of Health Research

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.

FURTHER INFORMATION

TELEPHONE ADVICE

Contact medical entomologist, CDC/PHU in your local jurisdiction.

KEY REFERENCES AND FURTHER READING

Todd RE, Guthridge SL, Montgomery BL. Evacuation of an Aboriginal community in response to an outbreak of blistering dermatitis induced by a beetle (Paederus australis). The Medical Journal of Australia. 1996 Feb 19;164(4):238-40.

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.

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 one quarter of the world's population at any given time. It is particularly common in tropical areas and used to be a major health issue in remote Aboriginal and Torres Strait Islander 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). *A. duodenale* is the dominant hookworm present in Northern Australia. The usual mode of transmission is by penetration of the skin by 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 and suck approximately 0.2mL of blood per day. Adult *A. duoendale* 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 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. The eggs are deposited with faeces in soil where they hatch into rhabditiform larvae which develop over one week into the filariform larvae which are able to invade the host.

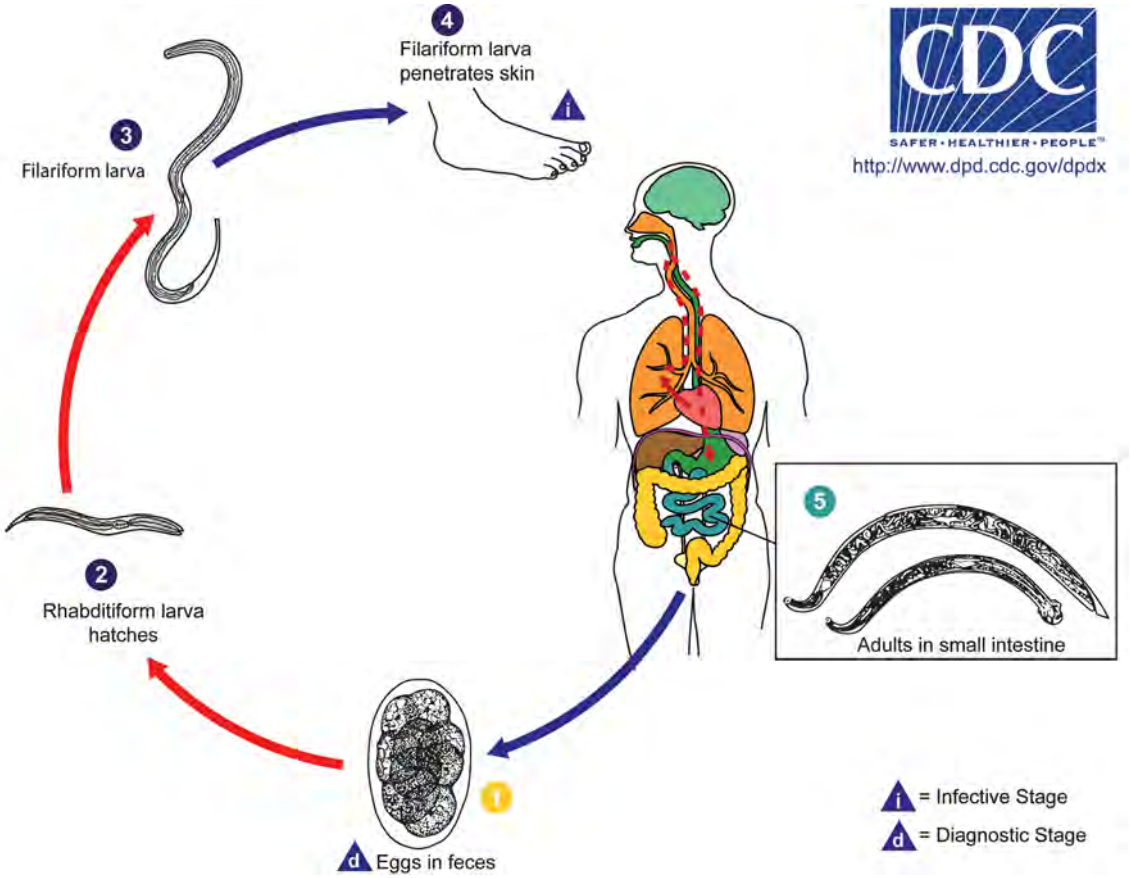

Figure 98: The life cycle of hookworm.
Source: https://www.cdc.gov/parasites/hookworm/biology.html

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 (eg 1,000 worms) 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.

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 north eastern Australia and diagnosed in a case at Numbulwar in the NT. 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. *Ancylostoma ceylanicum* has been detected in north-east Arnhem Land.

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, the majority of 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 p183*.

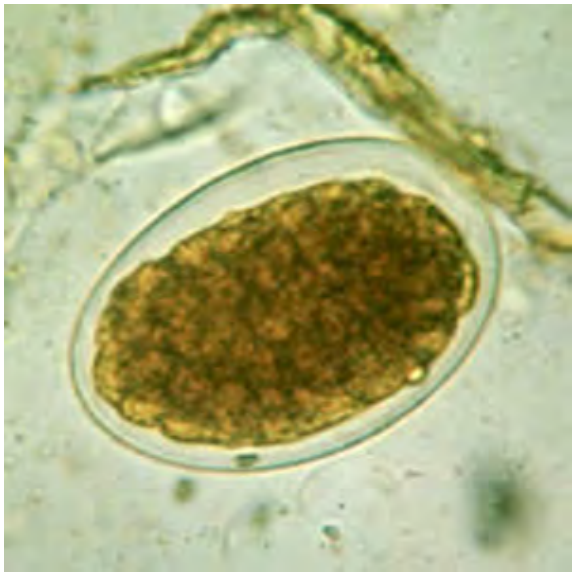

Figure 99: *Ancylostoma duodenale* egg.
https://www.cdc.gov/dpdx/hookworm/index.html

Figure 100: Hookworm (*ancylostoma duodenale*)
Source: http://slideplayer.com/slide/6366037

Hookworm

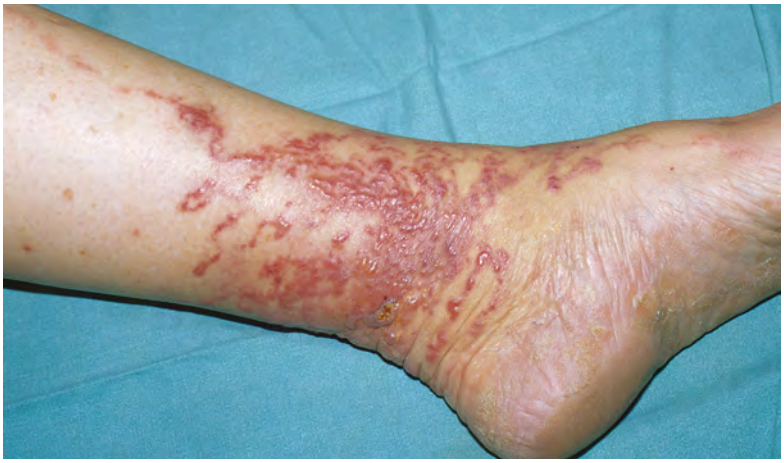

Figure 101: Cutaneous larva migrans from *Ancylostoma braziliense* (cat–dog Hookworm)
Source: Bart Currie — Menzies School of Health Research

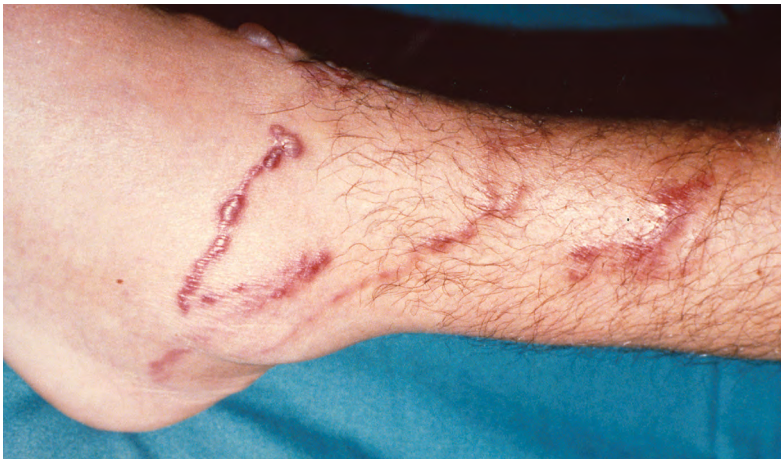

Figure 102: Cutaneous larva migrans from *Ancylostoma braziliense* (cat–dog Hookworm)
Source: Bart Currie — Menzies School of Health Research

FURTHER INFORMATION

TELEPHONE ADVICE

Contact paediatrician, CDC/PHU in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines see *Appendix p200*.

Northern Territory		
Remote Primary Health Care Manuals (RPHCM)	CARPA Standard Treatment Manual — Worms	Available online
Kimberley		
Kimberley Aboriginal Medical Services (KAMS)	Clinical Protocols/Guidelines — Parasites	Available online
North Queensland		
Queensland Health	Primary Clinical Care Manual — Intestinal worms	Available online

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Strongyloidiasis

Strongyloidiasis is a potentially fatal roundworm infection that can affect any organ of the body.

STRONGYLOIDIASIS IN NORTHERN AUSTRALIA

Strongyloidiasis is caused by the parasitic roundworm *Strongyloides stercoralis*. This neglected disease is common throughout tropical regions of the world where warmth, moisture and poor sanitation favour its spread. In temperate areas it is recognised in immigrants, war veterans and travellers returning from endemic areas. 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. Some East Arnhem Land communities have demonstrated positive isolation rates from stools ranging from 15% to 41% and sero-positivity in up to 60% of people tested. In Central Australia, it also occurs in conjunction with HTLV-1. Strongyloidiasis in Australia warrants further study particularly in relation to epidemiology, diagnostic testing, optimal management protocols and control programs for endemic communities.

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.

The majority of 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 proximal small bowel (jejunum) where they lay up to 40 eggs a day. The parasitic females 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
- *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. All the offspring of the free-living females become infective filariform larvae, resulting in an increase in the number of filariform larvae that can infect the host. *Strongyloides stercoralis* has only one generation of free-living adults. The total life span in the soil is considered to be up to 3 weeks
- *Autoinfection or hyperinfection* cycle. Some of the rhabditiform larvae develop into filariform larvae in the lower intestine. The filariform larvae can penetrate the colonic wall or perianal skin and enter the circulation to repeat the migration to the jejunum. This establishes ongoing internal reinfection. Random migratory routes involving organs other than the lungs may predominate. This autoinfection cycle allows strongyloidiasis to persist for decades after the host has left an endemic area.

CLINICAL PICTURE

Risk factors. A history of living in areas with a high prevalence of strongyloides such as Aboriginal and Torres Strait Islander communities in Northern Australia and many tropical countries. This includes workers in Aboriginal and Torres Strait Islander communities, immigrants, returned travellers and service personnel. Infection can be lifelong without effective treatment.

Symptoms and signs. Infection is mostly asymptomatic, but any organ of the body can be affected:

- Respiratory: dyspnoea, bronchospasm, gross haemoptysis
- Gastrointestinal: subacute obstruction or segmental ileus, ulcerative colitis with intestinal perforation and peritonitis, vague abdominal complaints, epigastric pain and tenderness (simulates peptic ulcer), impaired absorption/ malnutrition
- Renal, hepatic and cardiac systems
- Neurological: gram-negative meningitis, focal or general central nervous system
- Skin: larva currens, lesions over lower back and buttocks, recurrent urticaria
- Systemic: gram-negative bacteraemia, disseminated strongyloidiasis.

There are three important clinical patterns of severe infection with *S. stercoralis* in Northern Australian Aboriginal and Torres Strait Islander communities:

1. Acute gastrointestinal infection in children, often with diarrhoea, hypokalaemia and wasting. Pseudo-intestinal obstruction can occur. The diarrhoea may have a distinctive odour

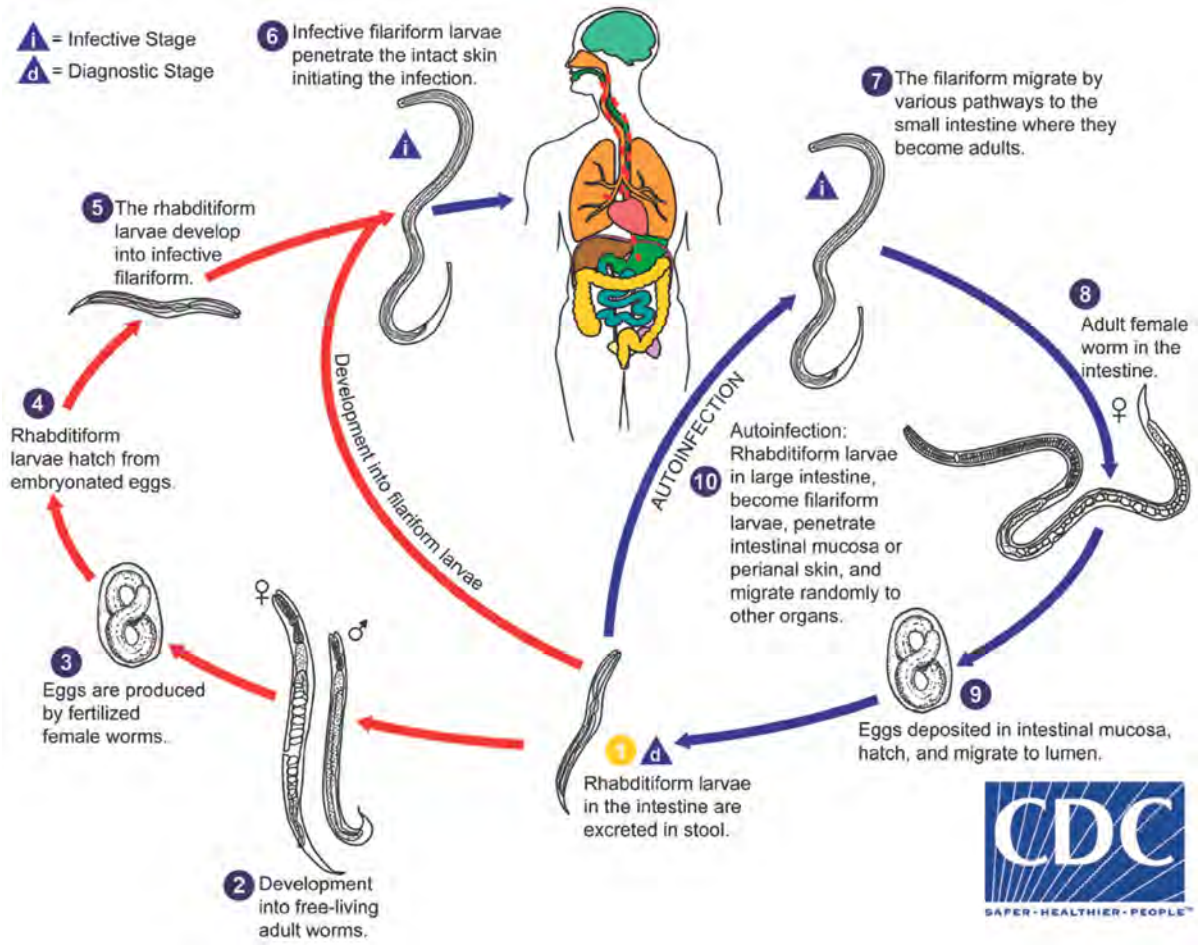

Figure 103: The life cycle of *S. stercoralis*. April 2015.
Source: https://www.cdc.gov/parasites/strongyloides/

2. 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 *Strongyloides stercoralis* infection, some with HTLV-1 comorbidity
3. Disseminated strongyloidiasis in patients on immunosuppressive therapy. 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* — see *Key references p112*.

Investigations. Faecal testing has poor sensitivity in chronic strongyloidiasis due to intermittent shedding of low numbers of larvae. To increase the sensitivity, three (or more) specimens should be sent. Agar plate is the most sensitive culture technique but requires fresh faeces with live larvae. *S. stercoralis* larvae die at low and high temperatures. 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. Westmead/ICPMR laboratory's test 'Strongy NAT' (nuclear acid test) is accredited and has a Medicare rebate.

Strongyloidiasis

Eosinophilia is present in 10–50% of cases. *Strongyloides* serology is used in diagnosing chronic strongyloidiasis in individuals, particularly adults, and for individual follow-up as titres have been shown to decrease to negative 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).

In disseminated disease, sputum wet mount examination may be useful, although positive results are usually associated with a poor outcome. Strongyloidiasis may also cause an abnormal chest X-ray.

DIFFERENTIAL DIAGNOSIS

Consider strongyloidiasis as an underlying cause of recurrent gram-negative septicaemia or meningitis. Clinical deterioration in a patient started on immunosuppressant therapy for another condition may also indicate underlying strongyloidiasis. Patients in this group are at high risk of disseminated disease, which is commonly fatal.

PRINCIPLES OF MANAGEMENT

Treatment. The most important principle is to prevent hyperinfection and potentially fatal disseminated disease in all patients requiring high dose steroids and immunosuppressive therapy. All infected individuals should be treated, including the asymptomatic. The first drug of choice is ivermectin, and albendazole as second line therapy, is less effective. Refer to the current *Therapeutic Guidelines: Antibiotic* for 'Strongyloidiasis' and '*Strongyloides stercoralis*'.

Follow-up is important because the parasite is not easily eradicated and retreatment may be necessary. It is worth considering testing household contacts. New cases may be stool positive and sero-negative because seroconversion has not occurred.

Prognosis is usually good except in severe cases of hyperinfection. Patients who have a history of residence in an endemic area or eosinophilia should be carefully checked for the presence of the parasite prior to the initiation of corticosteroid or immunosuppressive therapy. In some cases, this may require prophylactic treatment prior to receiving results. Protocols have been developed and recently revised for investigation and prophylaxis/treatment of Aboriginal and Torres Strait Islander patients on high dose steroids or other immunosuppressive therapy. Disseminated strongyloidiasis requires urgent specialist management.

Prevention. Infection may be transmitted by direct contact with faeces or soil contaminated by faeces from an infected person. 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.

FURTHER INFORMATION

TELEPHONE ADVICE

Contact infectious disease physician, paediatrician, CDC/PHU in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines see *Appendix p200*.

Northern Territory		
Remote Primary Health Care Manuals (RPHCM)	CARPA Standard Treatment Manual — Worms	Available online
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Kimberley Aboriginal Medical Services (KAMS)	Clinical Protocols/Guidelines — Parasites	Available online
North Queensland		
Queensland Health	Primary Clinical Care Manual — Intestinal worms	Available online

EDUCATIONAL RESOURCES

Miwatj Health Aboriginal Corporation	Flipchart — Strongyloides Story	Available online
ARDS Aboriginal Corporation	Strongyloides Information Resource	Available online

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Trichuriasis (whipworm)

Whipworm infection occurs globally and may cause anaemia, Trichuris dysentry 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 Aboriginal and Torres Strait Islander communities, particularly in the Northern Territory and Queensland. It has a global distribution especially 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.

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. The anterior section embeds into the superficial mucosa of the colon and caecum, lays 3000–7000 eggs per day and may live for five years. The eggs are passed with the faeces and incubate for at least three weeks in the soil before they become infective. After ingestion, infective eggs hatch in the duodenum, releasing larvae that mature before migrating to the large bowel. The cycle from ingestion to egg-laying takes about three months.

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 rectal prolapse can occur. Moderately heavy worm loads may contribute to growth retardation and anaemia. Infected patients lose approximately 0.005mL blood per worm per day.

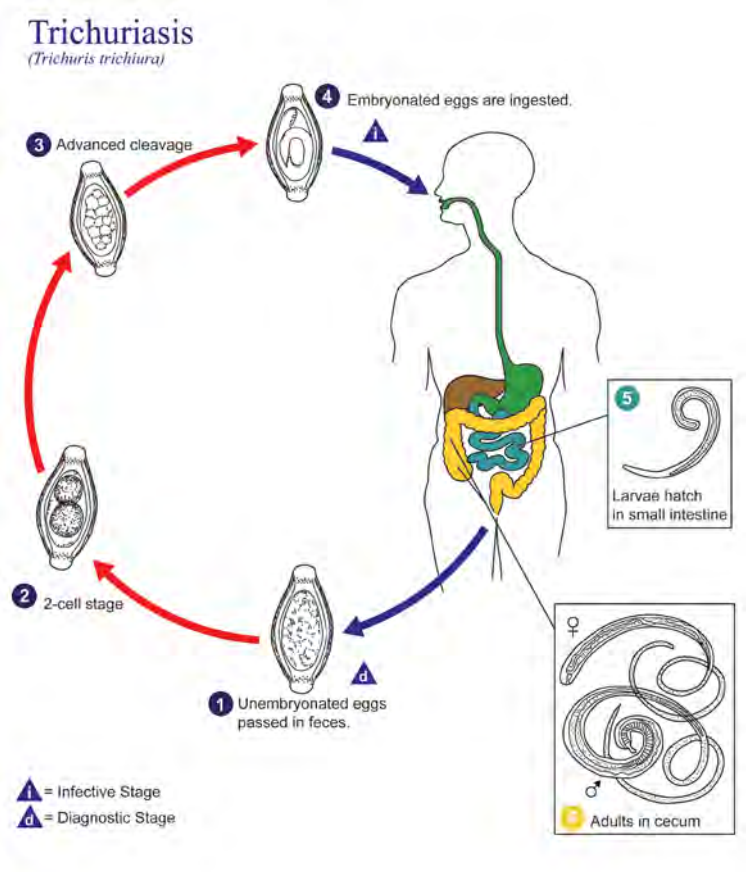

Figure 104: The life cycle of *T. trichiura*
Source: https://www.cdc.gov/parasites/whipworm/biology.html

Investigations. Faecal microscopy may demonstrate characteristic 50 by 20µm football or lemon-shaped eggs. Proctoscopy may reveal adult worms, 3–5cm in length. A blood film often shows eosinophilia.

PRINCIPLES OF MANAGEMENT

Treatment of individuals is with anthelmintics. In spite of regular dosing with single-dose albendazole, prevalence remains high. Even three doses of albendazole will not eradicate all *T. trichiura* infections, although the worm burden will be substantially decreased. Refer to the current *Therapeutic Guidelines: Antibiotic*. Regular anthelminthic treatment is part of child health programs in the Top End. Contact the community paediatric team in CDC/PHU for information.

Prevention. Community health education, improved hygiene, particularly washing hands and food handling practices, and improved infrastructure for sanitation.

Figure 105: Egg of *T. trichiura* as seen in an unstained wet mount.
https://www.cdc.gov/dpdx/trichuriasis/index.html

FURTHER INFORMATION

TELEPHONE ADVICE

Contact paediatrician, CDC/PHU in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines see *Appendix p200*.

Northern Territory		
Remote Primary Health Care Manuals (RPHCM)	CARPA Standard Treatment Manual — Worms	Available online
Kimberley		
Kimberley Aboriginal Medical Services (KAMS)	Clinical Protocols/Guidelines — Parasites	Available online
North Queensland		
Queensland Health	Primary Clinical Care Manual — Intestinal worms	Available online

KEY REFERENCES AND FURTHER READING

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SECTION 4 — SEXUALLY TRANSMITTED INFECTIONS

An overview of sexually transmitted infections (STIs)

Sexually transmitted infections are treated according to their presenting syndrome as clinical diagnosis is often inaccurate.

STIS IN NORTHERN AUSTRALIA

Remote Aboriginal and Torres Strait Islander communities in Northern Australia have disproportionately high notification rates of STIs. Northern Territory data demonstrate a higher prevalence in both Aboriginal and Torres Strait Islander and non-Indigenous populations compared with other parts of Australia.

Among Aboriginal and Torres Strait Islander groups the highest notification rates occur in people aged 15–35 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 cultural and language barriers.

Clinical manifestations. STIs are a significant clinical and public health problem. 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, infant morbidity and mortality, and psychological distress. The presence of other STIs also significantly increases the risk of transmission 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 Aboriginal and Torres Strait Islander 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 on urinalysis, in the absence of nitrites, could be due to an STI or PID and requires appropriate further history,

Figure 106: Notification Rate of Sexually Transmitted Infection, received from State and Territory health authorities, 2017.
Source: National Notifiable Diseases Surveillance System, 2018; http://www9.health.gov.au/cda/source/rpt_2.cfm.

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 made with caution.

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.

In the developing world, where laboratory tests are often not performed, it is also considerably less costly with less constraints on time, resources and access to treatment. The WHO has recommended that national STI control programs in developing countries incorporate syndrome based diagnostic and therapeutic flowcharts in their management guidelines. This standardises diagnosis, treatment, referral and reporting, leading to improved surveillance and program management.

Guidelines in Northern Australia use the syndromic approach in STI management, for example, in urethritis, cervicitis and genital ulcers. Unlike developing countries, however, 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 infection 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
- Large numbers of contacts
- Anonymous contacts.

In remote Aboriginal and Torres Strait Islander communities, 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. Your local CDC sexual health unit should be called on to assist and give direction in contact tracing.

Chlamydia, gonorrhoea, syphilis, donovanosis, chancroid, HIV and lymphogranuloma venereum (LGV) are LABORATORY notifiable. In Western Australia they are also notifiable by CLINICIANS.

Trichomonas is notifiable by LABORATORIES in the Northern Territory.

Gonococcal conjunctivitis, chancroid, donovanosis, syphilis, AIDS, lymphogranuloma venereum and hepatitis B are notifiable by CLINICIANS in the Northern Territory.

AIDS is notifiable by CLINICIANS in Queensland.

All above cases are reported to the local Centre for Disease Control/Public Health Unit.

An overview of sexually transmitted infections (STIs)

FURTHER INFORMATION

TELEPHONE ADVICE

Contact sexual health unit, CDC/PHU in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines and sexual health guidelines see *Appendix p200*.

Northern Territory		
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 — Sexual Health ■ Women's Business Manual — Sexual Health	Available online
Kimberley		
Kimberley Aboriginal Medical Services (KAMS)	Silver book — Guidelines for managing sexually transmitted infections and blood-borne viruses	Available online
North Queensland		
Queensland Health	Primary Clinical Care Manual — Sexually transmitted infections	Available online

KEY REFERENCES AND FURTHER READING

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Gonorrhoea

Neisseria gonorrhoea causes gonorrhoea which can be asymptomatic, have acute symptoms and cause long-term complications.

GONORRHOEA IN NORTHERN AUSTRALIA

Gonorrhoeal genital tract infection is common in Northern Australian remote Aboriginal and Torres Strait Islander 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 NT than other states. People aged 15–35 have the highest rates of infection.

AETIOLOGY AND PATHOGENESIS

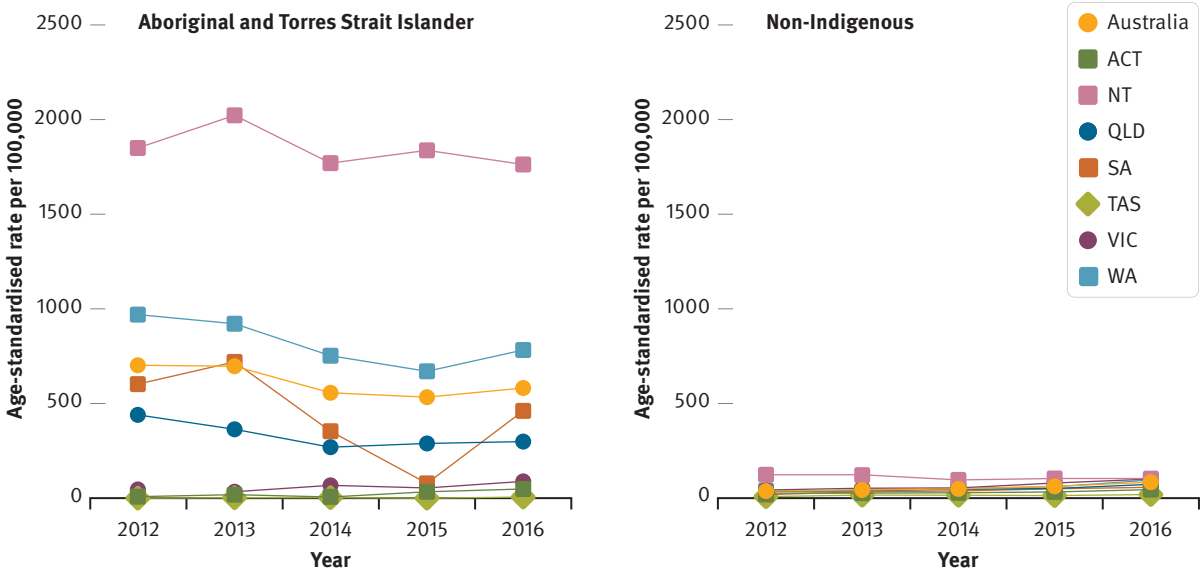
Neisseria gonorrhoeae is the gram-negative diplococcus that causes gonorrhoea. Mucous membranes lined by columnar or cuboidal cells are susceptible to gonococcal infection, including the urethra, endocervix, fallopian tube, and rectum. Genital gonorrhoea is exclusively a sexually transmitted infection.

CLINICAL PICTURE

Risk factors. See *An overview of Sexually Transmitted Infections p117*.

Symptoms and signs. Clinical features of *N. gonorrhoea* infection range from none to local genital symptoms and systemic illness.

- **Males.** In men, infection with gonorrhoea usually produces a frankly purulent urethral discharge and dysuria, after an incubation period of about 2–5 days. However, in about a quarter of cases the discharge may be scant and mucoid, indistinguishable from chlamydial urethritis, or may even be absent. Local complications in men include epididymo-orchitis and prostatitis. In 10% of cases it is asymptomatic. Co-infection with chlamydia is common.
- **Females.** Gonococcal infection in women is symptomatic in only about half of cases. The most common symptomatic presentation is of urethritis and cervicitis (lower genital tract infection), manifesting as 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. The most common complication of gonorrhoea in women, and also the most clinically important, is pelvic inflammatory disease (PID). This is reported to occur in 10–20% of women with untreated lower genital tract gonorrhoea. Gonococcal PID may present with any combination of pelvic pain, fever, deep dyspareunia or menstrual irregularities,



Includes jurisdictions with Aboriginal and Torres Strait Islander status completeness ≥50% (ACT, NT, QLD, SA, TAS, VIC and WA) for each of the five years 2012–2016.

Figure 107: Gonorrhoea notification rate per 100 000 population, 2012–2016, by Aboriginal and Torres Strait Islander status and state/territory

Source: Kirby Institute. Bloodborne viral and sexually transmissible infections in Aboriginal and Torres Strait Islander people: annual surveillance report 2017. Sydney: Kirby Institute, UNSW Sydney; 2017.

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 +/- rupture, tubal infertility and ectopic pregnancy.

- **Infections in other sites.** Pharyngeal infection is not uncommon in both sexes and is usually asymptomatic. Most rectal gonococcal infections are asymptomatic or subclinical. If present, symptoms include tenesmus, discharge, irritation, painful defecation, disturbed bowel function and bleeding. Rectal and throat swabs for gonococcal nucleic acid amplification test (NAAT) and culture are routine in the sexual health work up of men who have sex with men.

  Gonococcal conjunctivitis, although uncommon, 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 conjunctivitis that persists for weeks or months. It requires systemic treatment and may occasionally occur as part of an epidemic. It is an important diagnosis to make as untreated infection may lead to corneal ulceration, perforation and blindness. A sporadic case in a child should raise the suspicion of sexual abuse, although non-sexual transmission is possible.

  Disseminated gonococcal infection is rare (0.5–3% of gonorrhoea cases), and manifests most commonly as the arthritis-dermatitis syndrome, with joint swelling 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.

  Gonorrhoea should be considered in monoarthritis and tested for by culture and NAAT of joint aspirates.

  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 usually unremarkable, but diagnostic sampling is usually the same as for PID.

  Endocarditis and meningitis and prostatitis can occur, although rare.

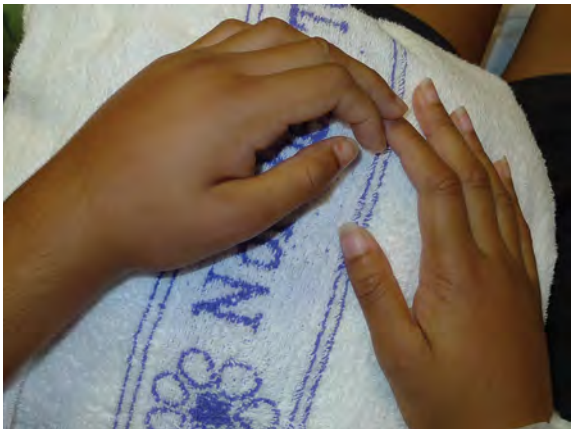


Figure 108: Disseminated gonococcal infection — septic arthritis
Source: Bart Currie — Menzies School of Health Research

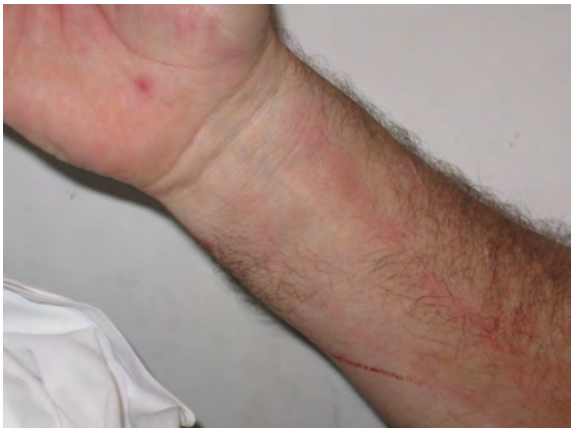


Figure 109: Disseminated gonococcal infection — tenosynovitis, arthritis, skin lesion
Source: Bart Currie — Menzies School of Health Research

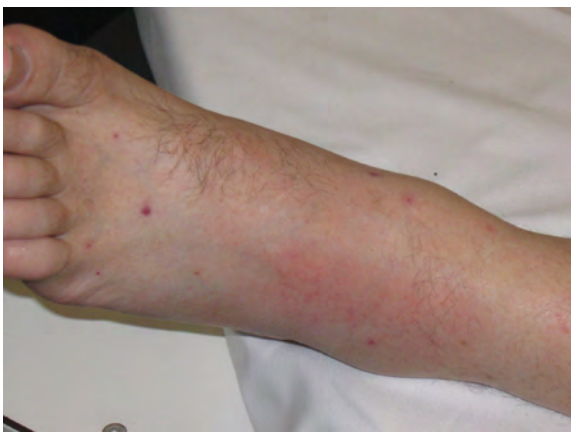


Figure 110: Disseminated gonococcal infection — ankle joint arthritis and skin lesions
Source: Bart Currie — Menzies School of Health Research

Gonorrhoea

DIFFERENTIAL DIAGNOSIS

The *Case study — Urethral discharge* (*p143*) discusses the differential diagnosis of urethritis in men. CARPA guidelines recommend empiric treatment for chlamydia and gonorrhoea in men from remote Aboriginal and Torres Strait Islander communities, aged 15–35, who present with urethral discharge or dysuria.

In women, the presence of vaginal discharge could represent cervicitis, upper genital tract infection (pelvic inflammatory disease), or vaginitis from candidiasis or 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.

PRINCIPLES OF MANAGEMENT

Gonorrhoea in the setting of the STI syndromes is discussed further in the urethral discharge (*p143*) and pelvic pain (*p147*) case studies. A few specific points about the investigations and treatment of gonorrhoea are worth making.

Investigations

Due to issues with false positives in low risk populations, screening for gonorrhoea is not recommended in low risk heterosexual populations in Queensland or Western Australia. The incidence is sufficiently high that screening is recommended in men who have sex with men, people from remote Aboriginal and Torres Strait Islander communities and the main stream 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.

Otherwise testing is recommended in the settings of STI syndromes/clinical illnesses suggestive of gonorrhoea, contact tracing, sterile pyuria and leukocytes in the urine of 15–35 year old sexually active males living in Aboriginal and Torres Strait Islander communities of the Northern Territory.

All guidelines in Australia recommend NAAT as the main screening test for gonorrhoea. This is because of the increased sensitivity and reasonable specificity compared to culture, and the ability to test multiple sites including urine, genital secretions, throat, rectum, and joints.

However, NAAT is unable to provide sensitivities, has occasional false positives and in certain sites (throat and rectal), validity of NAAT is sufficiently questioned as to always warrant a culture as well.

Culture is less sensitive but is important due to its increased specificity and ability to detect antibiotic resistance.

Knowing antibiotic sensitivities is important as certain parts of Northern Australia are considered penicillin sensitive zones, meaning that oral therapy is possible. These regions have a more than 95% sensitivity to penicillin on culture isolates. This relies completely on widespread use of culture and sensitivity to detect gonorrhoea.

Balancing this is the reduced yield of gonorrhoea from culture compared to NAAT, which becomes even more marginal when dealing with urine samples and time delays to the laboratory.

Each state varies in its testing guidelines, except for pharyngeal and rectal swabs, vaginal and urethral discharge samples, where culture in addition to NAAT is routine.

In the Northern Territory, screening is with both culture and NAAT in remote communities even if the sample is urine, whereas in city clinics, NAAT is the screening test and culture is performed in STI syndromes, endocervical samples or in the setting of positive NAATs.

In WA and Queensland, NAAT is the principal screening test and culture is only performed for urethral or vaginal discharge, endocervical samples, and throat and rectal swabs. Cultures are encouraged in the penicillin sensitive zones, although following the same guidelines of testing.

Be familiar with your 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 usually favours transport at fridge temperatures. Delays in transport reduce the yield of culture.

Management

Treatment requires knowledge of the place of origin of the partner and the syndrome the patient presents with.

As a general rule all patients with presentations more complicated than cervicitis, urethritis or positive asymptomatic screens from genital or urinary samples require parenteral ceftriaxone and oral azithromycin as part of the gonorrhoea treatment.

For cervicitis, urethritis or asymptomatic positive isolates from genital or urinary samples, the treatment decision is based on the place of origin of the partner.

If the partner is 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 three 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 culture or NAAT is recommended in some sexual health guidelines in Northern Australia but not in others.

***Note*:** At the time of writing the penicillin sensitive zone of Northern Australia was all of NT outside of Darwin, the Kimberley, Pilbara and Goldfields of WA.

> Gonorrhoea is a nationally notifiable condition by LABORATORIES. Cases are reported to the local Centre for Disease Control/Public Health Unit. CLINICIANS are also required to notify gonorrhoea in Western Australia and Gonococcal conjunctivitis in the Northern Territory.

FURTHER INFORMATION

TELEPHONE ADVICE

Contact sexual health clinics, obstetrician/gynaecologist, CDC/PHU in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines and sexual health guidelines see *Appendix p200*.

Northern Territory		
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/Gonorrhoea ■ Women's Business Manual — STI management for women/Gonorrhoea	Available online
North Queensland		
Queensland Health	Primary Clinical Care Manual — Chlamydia/gonorrhoea/trichomonas/ mycoplasma genitalium	Available online

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Syphilis

Syphilis is a chronic systemic infection caused by the spirochaete *Treponema 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 Aboriginal and Torres Strait Islander communities, in men who have sex with men, or is acquired overseas. In 2011, an outbreak of early infectious syphilis swept across from the Queensland gulf area reaching the Northern Territory in 2013 and has eventually entered the Kimberley and South Australia. This outbreak has included seven infant deaths from congenital infection. 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 infective lesions of primary and secondary syphilis in ano-genital areas or the mouth. 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 in between secondary and tertiary syphilis.

Infection in pregnancy crosses into the fetus 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 fetus 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 p117*.

Syphilis can be classified into early syphilis, late syphilis, latent 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 infection occur in the 15–30 year age group with the ensuing risk of transmitting the infection to the fetus 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 units dose (2 x 2.3mL or 1.8g) 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 fetus. 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.

- **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.

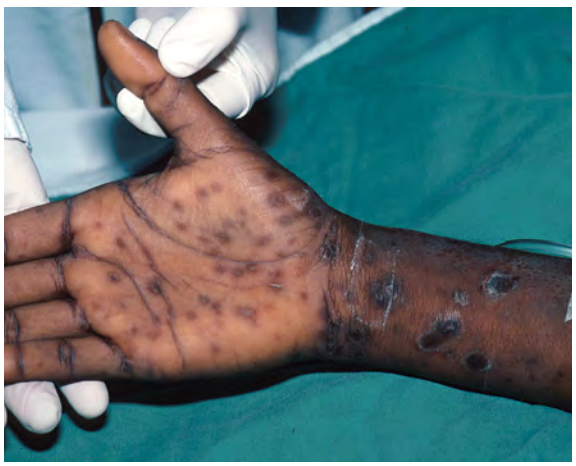

Figure 111: Secondary syphilis
Source: Bart Currie — Menzies School of Health Research

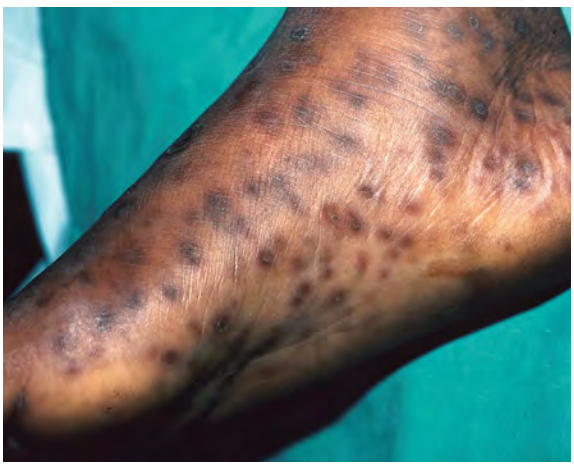

Figure 112: Secondary syphilis
Source: Bart Currie — Menzies School of Health Research

Syphilis

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 Aboriginal 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 occurs at or near puberty. Stigmata include sensorineural deafness, interstitial keratitis, Hutchison's teeth (Hutchison's triad), rhagades 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 fetus in the first trimester.

Screening and diagnosis. In low risk populations, testing for syphilis is part of a routine antenatal screen (once per pregnancy), 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 Aboriginal and Torres Strait Islander 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 Aboriginal 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).

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 antibodies (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 of public health units in Queensland, Western Australia and Northern Territory 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 2.3mL or 1.8g)
- 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 2.3mL or 1.8g) 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/4.6mL /1.8g of benzathine benzylpenicillin is two syringes not one (one syringe contains 1.2 million units/2.3mL/ 900mg).
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.

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 (ie 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* (*p145*) 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. CLINICIANS are also required to notify syphilis in Western Australia. Cases are reported to the local Centre for Disease Control/Public Health Unit.

Syphilis

FURTHER INFORMATION

TELEPHONE ADVICE

Contact syphilis register, sexual health clinic, CDC/PHU in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines and sexual health guidelines see *Appendix p200*.

Northern Territory		
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	Available online
Remote Primary Health Care Manuals (RPHCM)	■ CARPA Standard Treatment Manual — STI management/Syphilis ■ Women's Business Manual — STI management for women/Syphilis	Available online
NT Public Health Network (NT PHN)	Northern Territory HealthPathways — Syphilis	Available online
North Queensland		
Queensland Health	Primary Clinical Care Manual — Syphilis	Available online

KEY REFERENCES AND FURTHER READING

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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 Aboriginal and Torres Strait Islander people, with rates five times that of the national rate. The highest rates of infection are in 15–24 year olds.

AETIOLOGY AND PATHOGENESIS

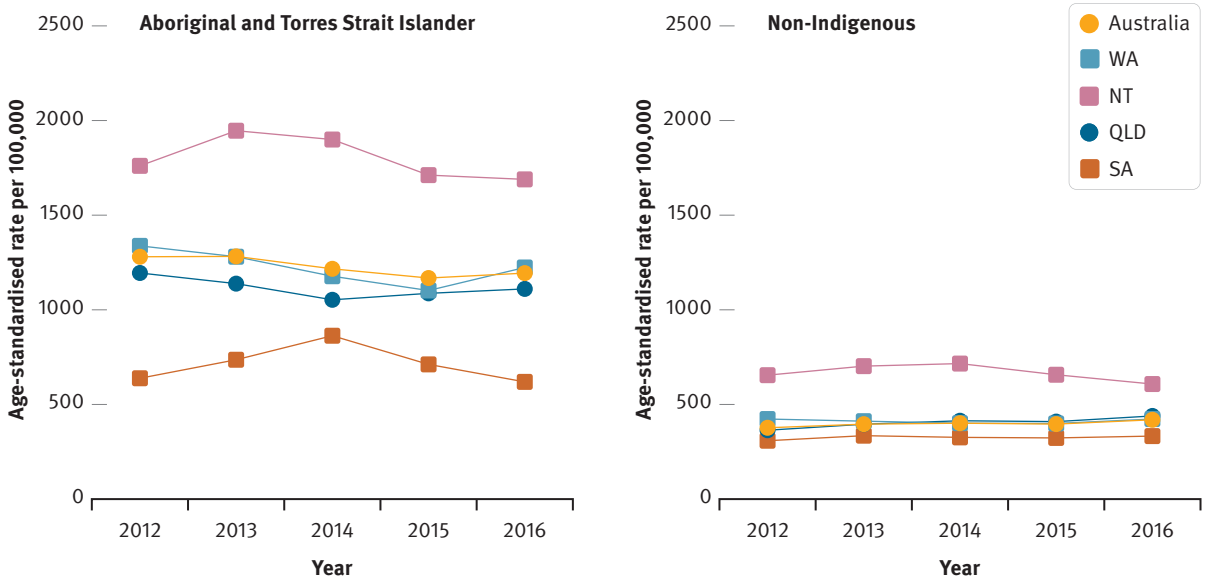
Chlamydia trachomatis is one of four species within the genus *Chlamydia*. Two other species are also pathogens of humans; *Chlamydia psittaci,* the cause of *psittacosis*, and *Chlamydia pneumoniae,* a common respiratory pathogen.

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 B–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 p117*.

Symptoms and signs. The clinical manifestations of infection with *C. trachomatis* have close parallels with those from *Neisseria gonorrhoeae,* including high rates of asymptomatic infection, predominantly genital tract symptoms, and symptoms of pharyngitis or proctitis in both men and women and occasional systemic disease. Asymptomatic chlamydial infection is very 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.



Includes jurisdictions with Aboriginal and Torres Strait Islander status completeness ≥50% (NT, QLD, SA and WA) for each of the five years 2012–2016.

Figure 113: Chlamydia notification rate per 100 000 population, 2012–2016, by Aboriginal and Torres Strait Islander status, state/territory and year.

Source: Kirby Institute. Bloodborne viral and sexually transmissible infections in Aboriginal and Torres Strait Islander people: annual surveillance report 2017. Sydney: Kirby Institute, UNSW Sydney; 2017.

- **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.
- **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 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 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* (*p143*) 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 Aboriginal and Torres Strait Islander 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 of urethritis (*p143*) and lower abdominal pain (*p147*) for more detail on the syndromes.

In the Northern Territory, the finding of leukocytes in urine of 15–35 year olds in remote Aboriginal 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 but usually involves azithromycin or doxycycline. As a minimum, abstinence from sex is recommended for six days post treatment of partners and patient.

Chlamydia

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.

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 resting three months post STI is recommended for reinfection.

> Chlamydia infection is a nationally notifiable condition by LABORATORIES. CLINICIANS are also required to notify chlamydia in Western Australia. Cases are reported to the local Centre for Disease Control/Public Health Unit.

FURTHER INFORMATION

TELEPHONE ADVICE

Contact sexual health unit, CDC/PHU in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines and sexual health guidelines see *Appendix p200*.

Northern Territory		
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
North Queensland		
Queensland Health	Primary Clinical Care Manual — Chlamydia/gonorrhoea/trichomonas/ mycoplasma genitalium	Available online

EDUCATIONAL RESOURCES

NT Centre for Disease Control (CDC)	Fact Sheet ■ Chlamydia ■ Chlamydia: directions for sexual partners	Available online

KEY REFERENCES AND FURTHER READING

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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 Aboriginal and Torres Strait Islander people. This is the result of the introduction of treatment with azithromycin and dedicated donovanosis management programs.

However, the disease has serious complications and ongoing awareness of this infection is very important. Consequently all guidelines in Northern Australia that address remote Aboriginal and Torres Strait Islander 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 and differences in race and sex distribution.

CLINICAL PICTURE

Risk factors. See *An overview of Sexually Transmitted Infections p117*.

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, that bleeds readily when touched. When complicated by secondary bacterial infection, it is often accompanied by a foul smell and may also be painful. Other variants include hypertrophic or verrucous (warty), cicatricial (scarring) and necrotic types. Donovanosis involves the genitals or inguinal lymph nodes in most cases, though extragenital lesions can occur. The sequelae of untreated donovanosis is local tissue destruction and genital deformity (eg saxophone penis) with some case reports of associated genital tract carcinoma. Very rarely, the infection may disseminate.

Figure 114: Number of notifications of newly diagnosed donovanosis infections, 2007–2016, by Aboriginal and Torres Strait Islander status.

Source: Kirby Institute. Bloodborne viral and sexually transmissible infections in Aboriginal and Torres Strait Islander people: annual surveillance report 2017. Sydney: Kirby Institute, UNSW Sydney; 2017.

Investigations. Nucleic acid amplification testing (NAAT) for donovanosis is recommended in remote Aboriginal and Torres Strait Islander 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 testing of impression smears or biopsies may be required.

PRINCIPLES OF MANAGEMENT

For discussion of the differential diagnosis, investigations and treatment, see *Case study — Genital ulcer* (*p145*), or local guidelines.

Empirical therapy for donovanosis is azithromycin 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

TELEPHONE ADVICE

Contact sexual health unit, CDC/PHU in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines and sexual health guidelines see *Appendix p200*.

Northern Territory		
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/Donovanosis ■ Women's Business Manual — STI management for women/Donovanosis	Available online
North Queensland		
Queensland Health	Primary Clinical Care Manual —Donovanosis	Available online

EDUCATIONAL RESOURCES

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

KEY REFERENCES AND FURTHER READING

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Trichomoniasis

Trichomoniasis is caused by a protozoal infection of the genitourinary tract that causes purulent vaginal or urethral discharge and dysuria.

TRICHOMONIASIS IN NORTHERN AUSTRALIA

***Trichomonas vaginalis* is transmitted almost exclusively by sexual intercourse, and is very common in Aboriginal and Torres Strait Islander 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 purulent, yellow-green vaginal discharge and dysuria. The vulva and vagina may become erythematous and oedematous. 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.

T. vaginalis causes urethritis in males, 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* (*p143*) for discussion of the differential diagnosis of urethritis in men.

Trichomonal infection can have significant complications. 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 Aboriginal and Torres Strait Islander communities of the Kimberley, Queensland and Northern Territory 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 2g or tinidazole 2g as an oral single dose. Use metronidazole 2g as a single dose in pregnant women as tinidazole's safety in pregnancy has not been well-evaluated. Metronidazole 400mg twice daily for 5 days is used for recurrent/refractory infection. This is also recommended for breastfeeding women, with doses given immediately after feeds.

As a general rule, treatment of pregnant women is not routinely recommended unless the patient is symptomatic or beyond a certain gestation or under the specific advice of a specialist. In the Northern Territory this gestation is set at 36 weeks.

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.

> Trichomoniasis is notifiable condition by LABORATORIES in the Northern Territory, but is not notifiable in Western Australia or Queensland. Cases are reported to the local Centre for Disease Control.

FURTHER INFORMATION

TELEPHONE ADVICE

Contact sexual health unit, CDC/PHU in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines and sexual health guidelines see *Appendix p200*.

Northern Territory		
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
North Queensland		
Queensland Health	Primary Clinical Care Manual — Chlamydia/gonorrhoea/trichomonas/ mycoplasma genitalium	Available online

EDUCATIONAL RESOURCES

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

KEY REFERENCES AND FURTHER READING

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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 in local HIV transmission.

HIV IN NORTHERN AUSTRALIA

The epidemiology of HIV infection demonstrates that:

- **The rate of total infection notifications has come down nationally however in 2017 there were 31 HIV notifications in Aboriginal and Torres Strait Islander people in Australia, an increase from 26 notifications in 2013 and accounting for 3% of all HIV notifications (963)**
- **In the five-year period 2013–2017 there has been a 260% increase in the notification rate of HIV for the Aboriginal and Torres Strait Islander population residing in remote areas (1.5 per 100 000 to 5.4 per 100 000). It is important to note this represents a small number of cases, and caution should be taken in interpretation. In the NT crude numbers remain small too**
- **According to the 2017 census 35% of Aboriginal and Torres Strait Islander live in major cities, 45% in inner or outer regional areas, 20% in remote or very remote areas**
- **For the period 2013–2017 82% of notifications occur in men.**

Patterns of risk behaviour show:

- **For men: male-male sex +/- injecting drug use >heterosexual sex >injecting drug use**
- **For women: heterosexual sex >injecting drug use**
- **A number of factors associated with greater risk of transmission of STI/blood borne viruses such as being intoxicated, having multiple sexual partners, lower condom use and a higher rate of needle sharing have been demonstrated in the Aboriginal and Torres Strait Islander population.**

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 NT has a higher proportion of heterosexually acquired infection (25% versus 10% nationally). This may reflect Darwin's close proximity to many countries with a high prevalence of HIV infection in the heterosexual population (including SE Asia). The index case in half of the heterosexually acquired NT cases came from a high HIV prevalence country.

Until 1991, there had been no HIV notifications in Aboriginal or Torres Strait Islander people in the NT. However, since that time, Aboriginal and Torres Strait Islander notifications have made up 25% of the total. Numbers remain low with an average of 1–2 new notifications per year, however this may change with clusters of infections reported in some neighbouring jurisdictions in the recent past. This requires that practitioners maximise both opportunistic and targeted testing as early detection and diagnosis has implications for both individual and public health.

Compared with other states and nationally, the rates of STIs in the NT are disproportionately high, with a huge ongoing syphilis outbreak. As discussed earlier, the Aboriginal and Torres Strait Islander population is over-represented in these notification rates. There is good evidence that STIs facilitate the transmission of HIV, particularly STIs associated with genital ulcers, such as syphilis.

Making the diagnosis of an STI in an individual demands that health professionals consider the presence of concurrent STIs, including HIV. HIV tests should therefore be offered to all people with an STI, including discussion of the three month window period. Analysis of testing data indicates that HIV testing of Aboriginal and Torres Strait Islander people remains sub-optimal.

CLINICAL FEATURES AND TREATMENT

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 outcomes as well as for non-HIV related outcomes with early commencement of antiretrovirals.

A standard regimen includes 3 active agents often given as a daily dose with just 1 tablet per day for most patients. Early initiation of care also has the advantage of providing 'treatment as prevention'; 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.

GPs can prescribe antiretrovirals after completing the HIV S100 prescribers course run by the Australasian Society for HIV, Viral Hepatitis and Sexual Health Medicine (ASHM).

> HIV is a nationally notifiable condition by LABORATORIES. Cases are reported to the local Centre for Disease Control/Public Health Unit. The HIV Public Health Team in Queensland and CLINICIANS in Western Australia must notify HIV.

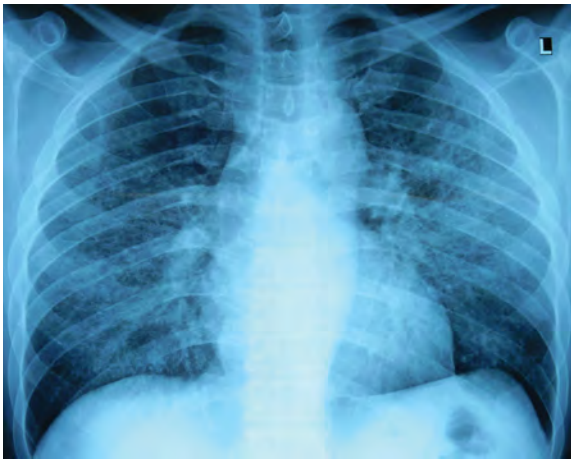

Figure 115: Pneumocystis pneumonia — undiagnosed HIV heterosexually acquired in SE Asia
Source: Bart Currie — Menzies School of Health Research

Human immunodeficiency virus (HIV)

FURTHER INFORMATION

TELEPHONE ADVICE

Contact sexual health unit, CDC/PHU in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines and sexual health guidelines see *Appendix p200*.

Kimberley		
Kimberley Aboriginal Medical Services (KAMS)	Clinical Protocols/Guidelines — HIV — Delivering a positive HIV result in the Kimberley	Available online
North Queensland		
Queensland Health	Primary Clinical Care Manual — Human immunodeficiency virus (HIV) infection	Available online

EDUCATIONAL RESOURCES

Australian Federation of AIDS Organisations (AFAO)	About HIV, living with HIV	Available online
NT Department of Health	Fact sheet — HIV	Available online
Australasian Society for HIV, Viral Hepatitis and Sexual Health Medicine (AHSM)	Prevention, testing and diagnosis, resources	Available online
Northern Territory AIDS and Hepatitis Council (NTAHC)	Resources	Available online

KEY REFERENCES AND FURTHER READING

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. http://hdl.handle.net/10137/237 (Accessed July 2019).

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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 a grossly 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 7: 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* > *Ureaplasma spp.* > *Candidiasis*	■ Prostatitis ■ Urethral strictures ■ Urinary tract infections ■ Chemical irritation ■ Trauma

Sexually acquired urethritis is divided into gonococcal and non-gonococcal aetiologies. Roughly 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, although it is not an infection routinely sought unless the syndrome persists for more than one week post treatment. *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 would you test them for?

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 NAAT.
2. Blood serology for HIV and syphilis +/- hepatitis B, if status needs clarification (and repeat HIV and syphilis serology in 3 months 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 and gonorrhoea.

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. In Queensland and Western Australia this means culturing any expressed pus found but not urine. In the Northern Territory this means culturing the urine when no pus is able to be expressed. Cultures maintain surveillance for antibiotic resistant strains.

Syphilis and HIV tests should be offered. Serology may be performed as a baseline but the three month window period needs 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 epididymo-orchitis, 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*.

The antibiotic regimen will depend on whether his partner is from a penicillin sensitive or resistant area for gonorrhoea. In a penicillin sensitive area, amoxicillin/probenecid and azithromycin are used whereas in penicillin resistant areas treatment is with ceftriaxone and azithromycin. If unsure talk with your local CDC/PHU/sexual health clinic.

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, ie using condoms, testing of both partners prior to sex when with new partner, avoiding more than one partner.

How should you follow-up Victor?

Follow-up is usually at one week and three months.

At one week Victor should be asked about his well-being, re-assessed for symptoms and signs of urethritis (ie 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 (ie 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.

Contact your local CDC/PHU for information about penicillin-sensitive and penicillin-resistant gonorrhoea areas.

KEY REFERENCES AND FURTHER READING

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Case study — Genital ulcer

Cathy, a 28 year old woman from a remote Aboriginal community, reluctantly presents to you for a 'check-up'. You offer a general 'well woman's check' which includes a pap smear 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 and prior treatment with topical or oral medication. 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 8: 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 Aboriginal and Torres Strait Islander communities in Northern Australia) *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*	■ Malignancy (a particular concern in East Arnhem land) ■ Severe candidiasis with fissuring ■ Fixed drug eruption ■ Behcet's syndrome

The prevalence of these specific aetiologies of genital ulcer varies according to the population. HSV is by far the commonest ulcerative STI in Australia, with 50% being herpes simplex virus 1 and 50% being herpes simplex virus 2. Syphilis notification rates are significantly higher in Aboriginal and Torres Strait Islander people in Northern Australia compared to non-Indigenous people. Similarly donovanosis, although very rare in recent years, is much more common in Aboriginal and Torres Strait Islander 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 inguino-femoral lymph node swelling and/or discharge (bubo), +/- overlying erythema.

What specimens would you collect and what would you test them for?

Cathy should have the following specimens collected:

- Dry swab from the ulcer for syphilis and HSV NAAT
- Screen for other STIs ie chlamydia, gonorrhoea and trichomonas on first void urine, self-collected swabs 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 in the event that 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 Aboriginal and Torres Strait Islander 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 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 probably at 3 months if this is still negative 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. It is able to distinguish herpes simplex virus 1 from herpes simplex virus 2, which is very important for subsequent patient counselling.

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 three-month window period for HIV must to be explained to the patient.

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 Aboriginal and Torres Strait Islander communities is reserved for painless lesions, whereas painful lesions are empirically treated for herpes simplex. Cathy should be given benzathine benzylpenicillin 2.4million units/4.6mL/1.8g (2 x 2.3ml syringes) 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.

KEY REFERENCES AND FURTHER READING

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More than just a pain — Case study of lower abdominal pain

Serena is a 34 year old Aboriginal woman who comes to see you at a community clinic in the NT. 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, 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 — appendicitis, gastroenteritis, acute infective colitis, diverticulitis.

What investigations would you do immediately?

Immediate investigations include:

- Urine tests for pregnancy and dipstick urinalysis +/- 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) for *C. trachomatis* (NAAT), *N. gonorrhoeae* (NAAT and MC&S) and (if available) *M. genitalium* (NAAT) ***OR***
  - (3) first void urine for *N. gonorrhoeae* (NAAT and MC&S) and *C. trachomatosis* (NAAT)
- As Serena lives in an NT remote Aboriginal community the sample/s would also be sent for *T. vaginalis* (NAAT)
- Blood for acute phase reactants: CRP/ESR, FBC and film
- 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
- Two IV lines
- Frequent observations — especially be alert for PV bleeding
- Gentle or no further abdominal examinations.

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 Aboriginal 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 signs indicate treatment for PID is needed but other conditions are possible. Appendicitis is less likely as the tenderness is across the lower pelvis and not in the right lower quadrant.

The decision to hospitalise versus outpatient therapy is determined by a number of factors.

Serena needs to be in hospital and given IV therapy as she has severe pain, a fever, low blood pressure, pelvic tenderness, and cervical excitation.

The recommended therapy would be:

- Metronidazole 500mg IV 12 hourly with intent of changing to 400mg oral twice a day (bd) when improved for total 14 days (to cover anaerobes)
- Azithromycin 500mg IV (1g orally if IV unavailable) to allow dual therapy for gonorrhoea (and therefore minimise resistance)
- Ceftriaxone 2g IV daily with intent of ceasing when improved (principal agent to cover gonorrhoea)
- Either 14 days of oral doxycycline 100mg oral twice a day (bd)
  OR a repeat dose of azithromycin seven days later (to cover chlamydia and streptococci).

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. Contact tracing matters but this needs to factor in the inherent uncertainty that Serena actually has PID, to prevent unnecessarily damaging her relationship with her partner. The extent of contact tracing depends on the organisms identified and the timing of symptoms.

After management of the acute condition, what follow-up investigation and/or management would be useful if the infection is mild or the woman does not wish to leave the community?

If a woman chooses to stay in the community and start oral antibiotics, and there is no improvement in one to three days, hospitalisation for more detailed assessment, pelvic ultrasound and admission for IV antibiotics should be considered. If the patient is already on IV therapy, a tubo-ovarian abscess or an alternative diagnosis need to be considered.

Negative STI laboratory tests do not rule out PID as organisms are only found 70% of the time.

Discuss safe sex. 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.

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 October 2018 from a sea snake bite; the first death from sea snake bite in Australia for over 80 years. Brown snakes account for the last three NT deaths before that, with the last death in 2013 from a northern (tropical) brown snake bite 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*. 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 9 shows the regional distribution of the potentially lethal terrestrial snakes, by decreasing frequency of envenoming for regions of Northern Australia.

In addition to those listed in Table 9 there are many other species of less venomous elapids in the region and several species in the black *(Pseudechis)* and brown *(Pseudonaja)* snake genera which may not have been associated with fatal human envenoming. In the Top End black whip snakes *(Demansia atra and Demansia papuensis)* account for more confirmed elapid bites than all other 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 9: The distribution of potentially lethal terrestrial snakes in tropical Australia in decreasing order of bites seen in each region.

Tropical Northern Territory and Tropical Western Australia
■ ***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]* family)
■ ***Acanthophis spp.*** Death adder
■ ***Oxyuranus scutellatus**** Taipan
Tropical Queensland
■ ***Pseudonaja textilis*** Common (Eastern) brown snake
■ ***Oxyuranus scutellatus*** Taipan
■ ***Pseudonaja nuchalis*** Northern (Tropical) brown snake
■ ***seudechis australis*** Mulga
■ ***Acanthophis spp.*** Death adder
■ ***Tropidechis carinatus*** Rough-scaled snake
■ ***Rhinoplocephalus nigrescens*** Eastern small-eyed snake

* Taipans have been found in the Top End and across to the Kimberley, but are very 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 and its progression across the NT and into the Kimberley 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. It was predicted that taipans would increase in numbers and habitat once cane toads became established. This appears to have happened and in early 2019 there was the first recorded taipan bite in the NT 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.

Figure 116: Mulga snake
Source: Bart Currie — Menzies School of Health Research

Figure 118: Slaty Grey snake Darwin (non-venomous)
Source: Bart Currie — Menzies School of Health Research

Figure 117: Western brown snake
Source: Bart Currie — Menzies School of Health Research

Figure 119: Northern Death adder
Source: Bart Currie — Menzies School of Health Research

Snakebites

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 occur. This can be a most dramatic event, especially with brown snakes *(Pseudonaja spp.)*.
- **Neuromuscular paralysis (neurotoxins).** Progressive descending paralysis of the eyes (ptosis, diplopia, blurred vision), bulbar muscles, chest and diaphragm, then limb muscles. Ptosis should be added to the neurological observations of all snake bite victims.
- **Haematological disorders —**
  - > **Venom-induced consumptive coagulopathy** 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, thrombocytopenia and rising creatinine levels).**
- **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 envenoming but can also be secondary to myoglobinuria from severe rhabdomyolysis which may result in the need for haemodialysis. The relative contributions of the various potential pathogenetic mechanisms for early hypotension and collapse are unknown and are likely to vary for different snakes from different regions of the world. **However 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.

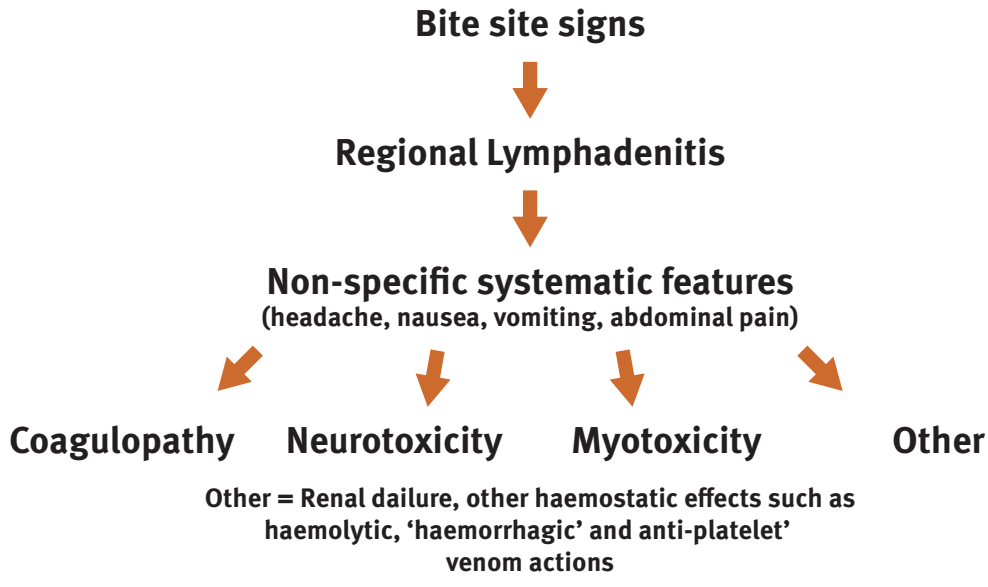

Figure 122: Australasian elapid envenoming

CLINICAL PICTURE

Figure 122 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 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 neurological observations including checking for ptosis.

THE TIME COURSE OF ENVENOMING:

Table 10 (*p156*) 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, 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 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 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.

Figure 120: Northern Death adder fangs
Source: Bart Currie — Menzies School of Health Research

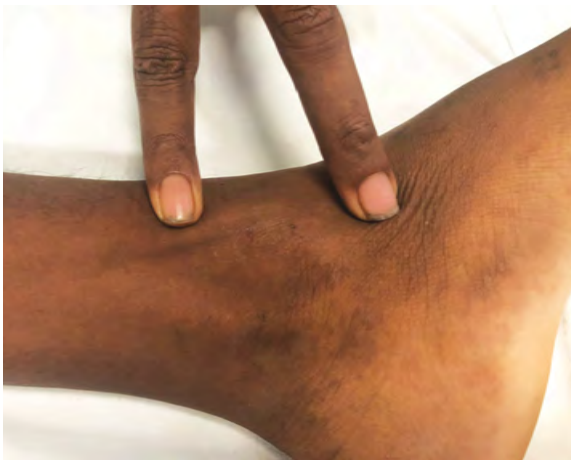

Figure 121: Death adder bite with subtle fang marks
Source: Bart Currie — Menzies School of Health Research

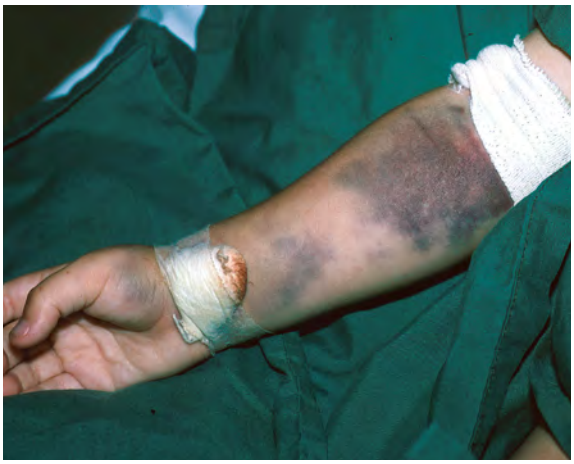

Figure 123: Brown snake coagulopathy
Source: Bart Currie — Menzies School of Health Research

Snakebites

IMPORTANT BEDSIDE TESTS

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 be forming in the glass tube by 10 minutes. An assay validated in the field is the 20 WBCT, which simply determines whether or not 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 to be used. Formal laboratory coagulation testing is required to assess for coagulopathy and confirm any WBCT abnormality.

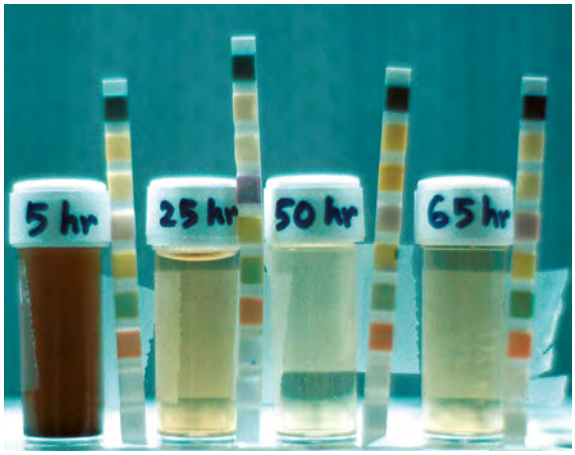

Figure 124: Mulga snake myoglobinuria
Source: Bart Currie — Menzies School of Health Research

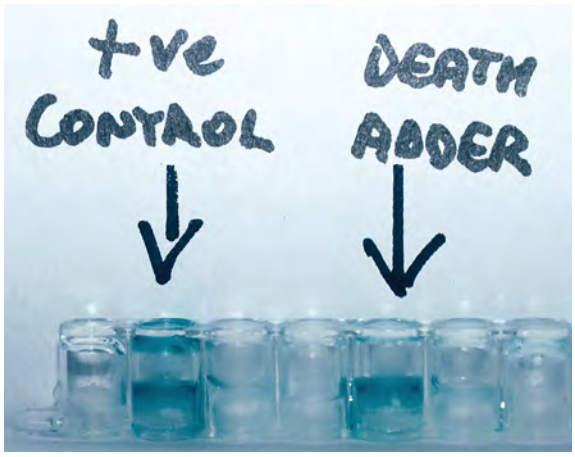

Figure 125: Death adder snakebite venom detection bite swab positive
Source: Bart Currie — Menzies School of Health Research

PRINCIPLES OF MANAGEMENT

First aid

Pressure immobilisation of the limb and strict immobilisation 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 should remain in place until the patient is transported to hospital and can be released under supervision.

Pre-hospital care

All patients should be admitted/evacuated to hospital for observation, investigation, removal of pressure immobilisation and administration of antivenom if required. Prior to transfer (if in a remote clinic), establish intravenous access with 2 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. 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. A 2013 article by Prof. Geoff Isbister provides a useful summary of current practice.

Table 10 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	+	+/-	+	+	+	+[2]	+
Eastern small-eyed snake	-	+/-	+	+	+	+/-	?
Whip snakes[6]	-	+	+/-	+/-	-	-	-
Tiger snakes[7]	+	+	+	+	++	+[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
5 Predominantly pre-synaptic
6 Not potentially lethal but common
7 Not in the tropics but included for comparison

FURTHER INFORMATION

TELEPHONE ADVICE

Contact emergency physician in your local jurisdiction.

National

Poisons information **13 11 26**

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines see *Appendix p200*.

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 — Snake bites — land and sea	Available online
North Queensland		
Queensland Health	Primary Clinical Care Manual — Snakebite including sea snake	Available online

KEY REFERENCES AND FURTHER READING

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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 is a dinoflagellate alga that adheres to dead coral. Under particular environmental conditions it produces a toxin, which is then biochemically converted to ciguatoxins. Herbivorous fish consume the algae and the toxins increase in concentration along the food chain. It 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. Ciguatoxin does not harm the fish and cannot be removed by freezing, cooking or cleaning the fish. It is colourless, odourless and tasteless and the fish does not look spoilt. Symptoms are due to the direct effects of the toxin.

CLINICAL PICTURE

Risk factors. Ingestion of large predatory reef fish in the preceding 30 hours.

Symptoms and signs. Diagnosis is based on 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, 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 may increase sensitivity to the toxin. The acute illness lasts from 1–8 days, whereas neurological symptoms can last for months.

Investigations. The toxin can be tested for 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
- 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.

FURTHER INFORMATION

TELEPHONE ADVICE

Contact specialist physician, CDC/PHU in your local jurisdiction.

National
Poisons information **13 11 26**

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines see *Appendix p200*.

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
North Queensland		
Queensland Health	Primary Clinical Care Manual — Ciguatera poisoning	Available online

EDUCATIONAL RESOURCES

NT Department of Primary Industry and Fisheries	Ciguatera poisoning	Available online
NT Centre for Disease Control (CDC)	Factsheet — Ciguatera fish poisoning	Available online
Queensland Health	Naturally occurring seafood toxins	Available online

KEY REFERENCES AND FURTHER READING

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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 60 deaths over the last 100 years in Australia. Children are most at risk because of their small body mass and the last 10 fatalities in the NT have occurred in children.

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 discharge venom through the skin on contact.

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
- Neurological — confusion, agitation, unconsciousness
- Skin — erythematous wheals where the tentacles have made contact.

Figure 126: *C. fleckeri* jellyfish at Nightcliff beach
Source: Bart Currie — Menzies School of Health Research

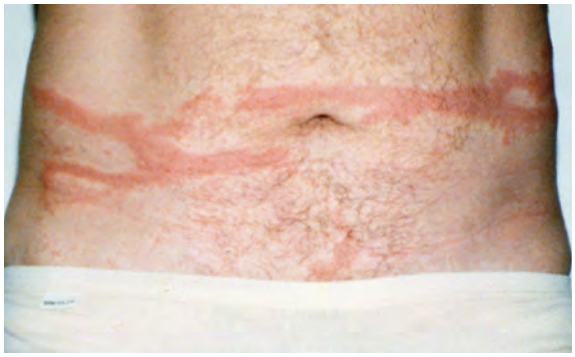

Figure 127: *C. fleckeri* stings
Source: Bart Currie — Menzies School of Health Research

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 May. Stinger suits are advised for water activities. Have vinegar available at all times when swimming off the coast of Northern Australia.

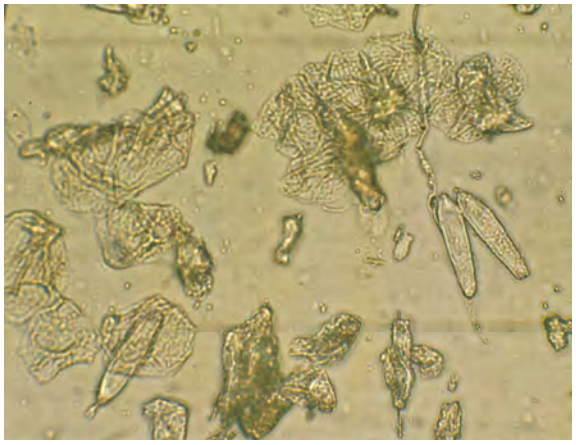

Figure 128: *C. fleckeri* nematocysts under microscope (x200 magnification) from sticky tape test
Source: Bart Currie — Menzies School of Health Research

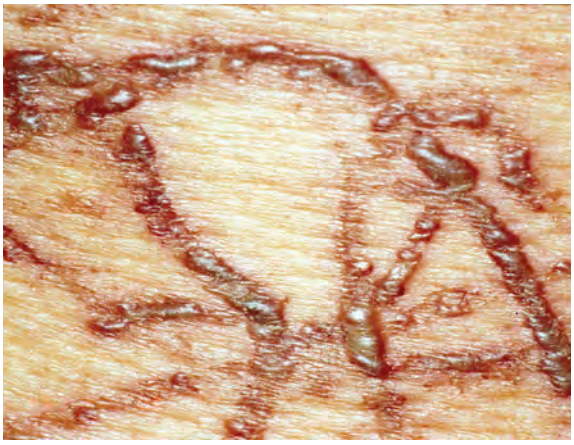

Figure 129: *C. fleckeri* stings
Source: Bart Currie — Menzies School of Health Research

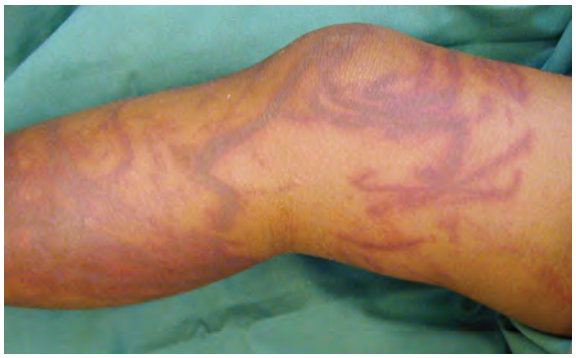

Figure 130: *C. fleckeri* sting at 5 hours
Source: Bart Currie — Menzies School of Health Research

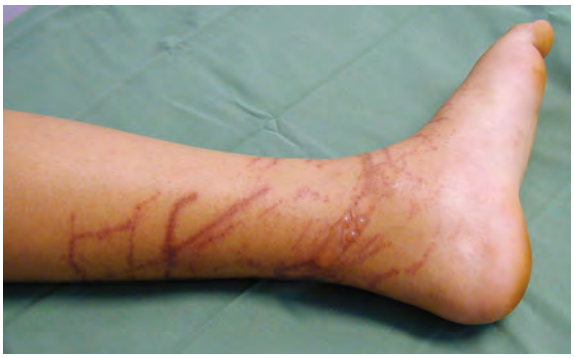

Figure 131: *C. fleckeri* sting at 48 hours
Source: Bart Currie — Menzies School of Health Research

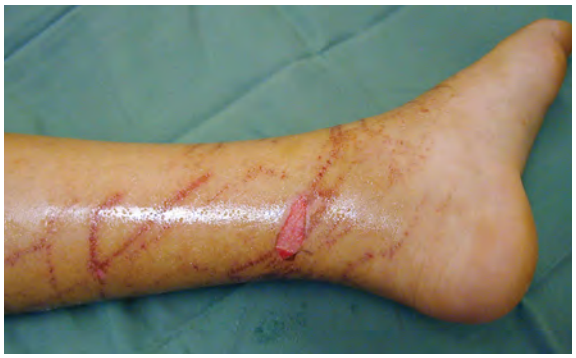

Figure 132: *C. fleckeri* sting at 5 days
Source: Bart Currie — Menzies School of Health Research

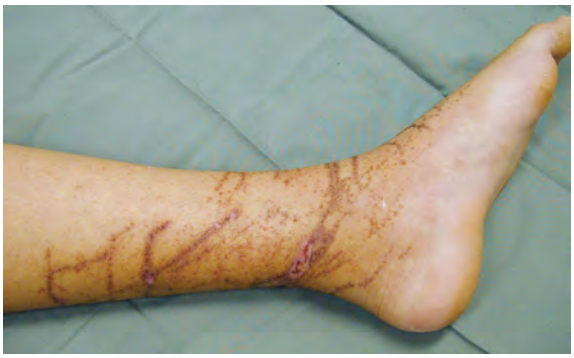

Figure 133: *C. fleckeri* sting at 13 days
Source: Bart Currie — Menzies School of Health Research

Box jellyfish

PRINCIPLES OF MANAGEMENT:

- Immediately flood the affected skin with vinegar to prevent further envenoming from nematocysts
- Remove tentacles from the skin
- 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.

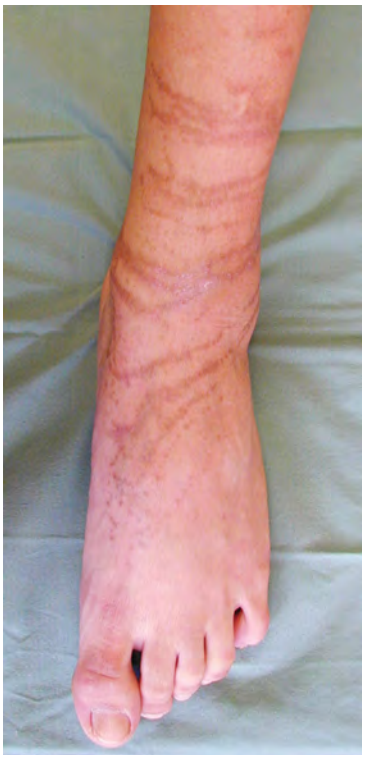

Figure 134: *C. fleckeri* sting at 10 weeks
Source: Bart Currie — Menzies School of Health Research

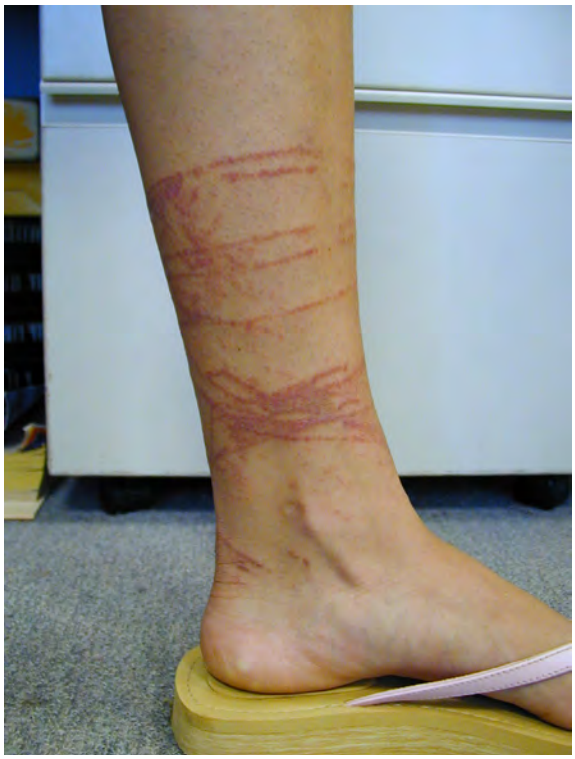

Figure 135: *C. fleckeri* delayed hypersensitivity papular uricaria at 19 days
Source: Bart Currie — Menzies School of Health Research

FURTHER INFORMATION

TELEPHONE ADVICE

Contact emergency physician in your local jurisdiction.

National
Poisons information **13 11 26**

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines see *Appendix p200*.

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
North Queensland		
Queensland Health	Primary Clinical Care Manual — Box jellyfish (Chironex fleckeri) envenomation	Available online

EDUCATIONAL RESOURCES

NT Centre for Disease Control (CDC)	Fact sheet — Box jelly fish	Available online

KEY REFERENCES AND FURTHER READING

Crowley-Cyr L. Public authority responses to marine stinger public health risks: a scenario analysis of the Irukandji health threat in controlled spaces at public beaches in Australia. Journal of Law and Medicine. 2012 Dec;20(2):363-79.

Little M. First aid for jellyfish stings: do we really know what we are doing? Emergency Medicine Australasia: EMA. 2008 Feb;20(1):78-80. DOI: 10.1111/j.1742-6723.2007.01053.x

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Currie BJ. Marine antivenoms. Journal of Toxicology: Clinical Toxicology. 2003;41(3):301-8.

Isbister GK, Palmer DJ, Weir RL, Currie BJ. Hot water immersion v icepacks for treating the pain of Chironex fleckeri stings: a randomised controlled trial. The Medical Journal of Australia. 2017 Apr 3;206(6):258-61.

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 an Aboriginal 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 WA and other locations where this species has rarely been captured. 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 Western Australia. Irukandji jellyfish are hard to see in the water as they are colourless and very small — the bell is 2.5cm or smaller and the tentacles range from a few centimetres to 35cm.

Symptoms and signs:

- Cardiovascular — tachycardia, marked hypertension. The toxin is a direct myocardial depressant. Subsequent hypotension and cardiac failure with pulmonary oedema has been described, although rare
- Gastrointestinal — abdominal pain, often with cramps and occurring in waves lasting a few minutes
- Skin — sweating and piloerection are common and can be severe
- Musculoskeletal — back and chest pain
- Psychological — restlessness, anxiety, 'impending doom'.

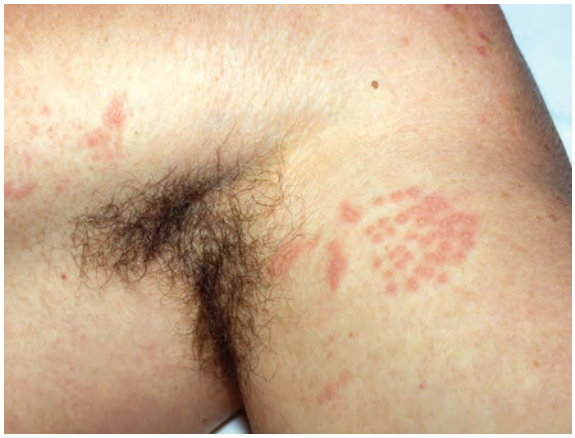

Figure 136: Irukandji syndrome post jellyfish bell contact
Source: Bart Currie — Menzies School of Health Research

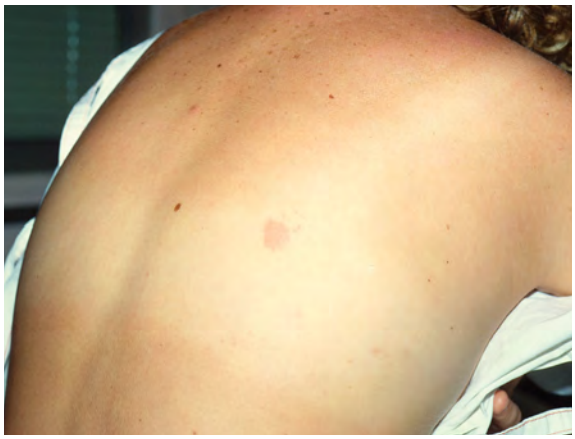

Figure 137: Irukandji syndrome severe despite minimal jellyfish bell contact
Source: Bart Currie — Menzies School of Health Research

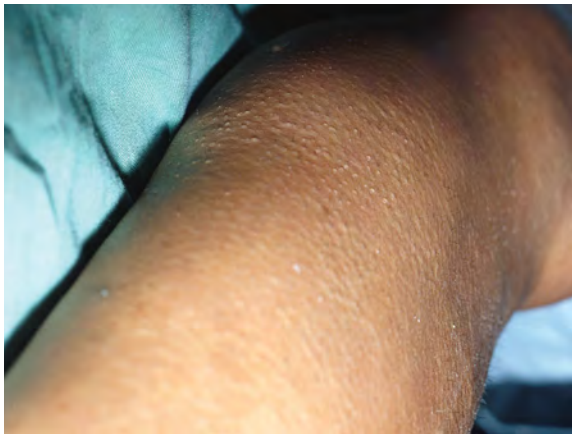

Figure 138: Irukandji syndrome with severe sweating and piloerection
Source: Bart Currie — Menzies School of Health Research

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

TELEPHONE ADVICE

Contact emergency physician in your local jurisdiction.

National

Poisons information **13 11 26**

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines see *Appendix p200*.

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
North Queensland		
Queensland Health	Primary Clinical Care Manual — Irukandji syndrome	Available online

EDUCATIONAL RESOURCES

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

KEY REFERENCES AND FURTHER READING

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Crowley-Cyr L. Public authority responses to marine stinger public health risks: a scenario analysis of the Irukandji health threat in controlled spaces at public beaches in Australia. Journal of Law and Medicine. 2012 Dec;20(2):363-79.

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 trachynus* and *Synanceia verrucosa*. The fish inflict their defensive effect through penetrating injuries caused by 13 paired dorsal spines with venomous glandular tissue 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 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 maybe required to detect retained foreign body.

PRINCIPLES OF MANAGEMENT

- Immersing limbs in hot water for up to 90 minutes relieves pain but may not denature the toxins. Make sure the unaffected limb is also immersed, to ensure water temperature is tolerable, preventing burns. Pain recurs when limb is removed from hot water.
- IV morphine 0.1mg/kg up to 5mg increments may be given

Figure 139: Stonefish
Source: Bart Currie — Menzies School of Health Research

- 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. Oral steroids can be used to minimise symptoms.

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.

FURTHER INFORMATION

TELEPHONE ADVICE

Contact emergency physician in your local jurisdiction.

National

Poisons information **13 11 26**

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines see *Appendix p200*.

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 sting	Available online
North Queensland		
Queensland Health	Primary Clinical Care Manual — Fish stings	Available online

EDUCATIONAL RESOURCES

Women's and Children's Hospital, South Australian	Toxinology	Available online

KEY REFERENCES AND FURTHER READING

Currie BJ. Marine antivenoms. Journal of Toxicology: Clinical Toxicology. 2003;41(3):301-8.

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Lyon RM. Stonefish poisoning. Wilderness & Environmental Medicine. 2004 Winter;15(4):284-8.

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 and predators 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.

In the Indo-Pacific region 35 deaths have been recorded in the past 90 years. The only recorded death from cone snail toxin in Australia was in 1935 at Hayman Island in Northern Queensland. In 2015, Care Flight rescued a man in Northern Queensland, who needed ventilation as a result of cone snail envenoming.

Figure 140: 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 may be relatively mild, but is rapidly followed by localised numbness which spreads, and may be accompanied by partial or complete paralysis of limbs and most importantly respiratory function. Difficulties with speech, swallowing and coordination may precede paralysis.

PRINCIPLES OF MANAGEMENT:

- Pressure bandage with immobilisation as for snakebite, to delay venom absorption
- All cases should be triaged to urgent assessment and management in hospital
- There is no antivenom for cone snail envenoming
- Treatment is supportive — ventilation can be life-saving
- Complete recovery may take up to 3 days
- Treat any contamination/retained foreign body
- Ensure adequate tetanus immunisation coverage.

FURTHER INFORMATION

TELEPHONE ADVICE

Contact emergency physician in your local jurisdiction.

National
Poisons information **13 11 26**

MANAGEMENT GUIDELINES

National		
White J. A	Clinician's Guide to Australian Venomous Bites and Stings, 2013.	Available online
North Queensland		
Queensland Health	Primary Clinical Care Manual — Blue-ringed octopus and cone shell envenomation	Available online

EDUCATIONAL RESOURCES

Women's and Children's Hospital, South Australian	Toxinology	Available online

KEY REFERENCES AND FURTHER READING

Halford ZA, Yu PY, Likeman RK, Hawley-Molloy JS, Thomas C, Bingham JP. Cone shell envenomation: epidemiology, pharmacology and medical care. Diving and Hyperbaric Medicine. 2015 Sep;45(3):200-7.

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Kava use

Kava is widely used in south Pacific countries particularly on ceremonial occasions.

KAVA USE IN NORTHERN AUSTRALIA

Kava is an intoxicating and sedating drink prepared from crushed roots of the pepper plant *(Piper methysticum)*. It was brought to Arnhem Land Aboriginal 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 to control kava consumption in 1990 prevented licensees from supplying more than 50g/day or 350g/week per person. In 1998, the Kava Management Act was introduced. Then in 2001, the Northern Territory Government introduced the Kava licensing regimen. In June 2007, the Australian Government as part of its intervention in the Northern Territory 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 persists.

CLINICAL PICTURE

Risk factors. The measured health effects appear to occur more frequently in those estimated to be using more than 400g/week of kava powder. While 'safe' levels of any substance are difficult to define because of variations between individuals, population health status and other factors, community kava use at average levels of more than 400g/person/week is currently considered to be harmful.

Symptoms and signs. Kava users frequently show the following:

- A characteristic dermopathy. This consists of a generalised flat, fine, grey dermatitis with no active border. It is sometimes described as 'crocodile skin' because the shine of healthy skin is lost. It may be confused with a fungal rash
- Lower mean body mass index, measures of body fat and skin-fold thickness
- Abnormal LFTs, particularly increased levels of gamma glutamyl transpeptidase (GGT) and alkaline phosphatase (ALP)
- Decreased lymphocytes.

Clinical observations suggest that the combination of bloodshot eyes and leathery skin is highly suggestive of kava use. The skin rash can be confused with tinea corporis, which is very common in the Top End. In areas where kava is used, the diagnosis of fungal skin infection should be microscopically confirmed. Kava use may also be a risk factor for sudden cardiac deaths particularly when coupled with recent heavy alcohol use. Possible mechanisms might include, enhanced thrombosis with dehydration amongst heavy users and/or arrhythmia. Other possible clinical associations that have been documented include acute neurological events and an increased risk of melioidosis.

Figure 141: *P. methysticum*

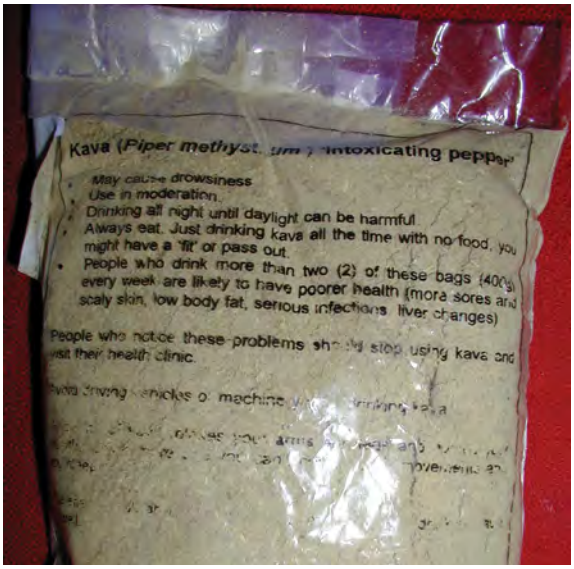

Figure 142: Commercial kava powder prior to being made illegal
Source: Bart Currie — Menzies School of Health Research

FURTHER INFORMATION

TELEPHONE ADVICE

Contact Alcohol and Other Drugs service in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines see *Appendix p200*.

Northern Territory		
Remote Primary Health Care Manuals (RPHCM)	CARPA Standard Treatment Manual — Kava	Available online

KEY REFERENCES AND FURTHER READING

Fuller T. Kava in the Northern Territory: a unique situation. Australian Police Journal. 2013;67(1):18-25. https://apjl.com.au/articles/kava_in_the_northern_territory_a_unique_situation_

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Figure 143: Kava dermopathy
Source: Bart Currie — Menzies School of Health Research

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 misuse is the intentional inhalation of volatile substances to achieve an altered mental state. In remote communities of Australia, petrol sniffing had seemed an almost intractable problem until the regional roll-out of low aromatic fuel in 2005. More recently, the inhalation of aerosol deodorants has become problematic, particularly in urban areas. 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.

AETIOLOGY AND PATHOGENESIS

There are two major components in petrol that cause toxicity. These are the volatile hydrocarbons, particularly toluene, and alkyl lead additives (in 'leaded' petrol only). The main lead additive, tetraethyl lead, is extremely neurotoxic. Although it has a half-life in blood of 3–5 days, it tends to persist in brain tissue with a much longer biological half-life. Inhalation of butane and propane based fuels (as found in aerosol deodorants) is particularly dangerous due to the risk of arrhythmias leading to sudden death, otherwise known as 'sudden sniffing death'.

Symptoms and signs.

The **immediate** effects of petrol inhalation include euphoria and intoxication. An acute encephalopathy can occur which may manifest as restlessness and excitation, impaired consciousness, delirium, fitting, acute psychosis, or death, depending upon levels of exposure. The encephalopathy occurs within minutes of inhalation and may last for 5–6 hours. An approach to managing a patient in this situation is described in the *CARPA Standard Treatment Manual* (*see Management guidelines* following). Petrol sniffing has been associated with increased accidents, trauma, burns, STIs and pneumonia.

The **chronic** effects of petrol sniffing include a variety of neurological abnormalities: behavioural disorders; cognitive impairment; impaired vision; movement disorders such as tremor, chorea and ataxia; nystagmus; pyramidal signs and convulsions. Volatile substances are also believed to cause kidney, liver, heart and lungs damage.

Prevention. Various interventions have been tried for the problem of petrol sniffing, the most successful of which have been those with community support and participation. Aim to minimise harm by:

- 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, outstation programs such as Mt Theo in Central Australia
- Harm Reduction — use of non-leaded petrol, avoiding use in enclosed spaces or covering 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.

FURTHER INFORMATION

TELEPHONE ADVICE

Contact Alcohol and Other Drugs service in your local jurisdiction.

MANAGEMENT GUIDELINES

National		
NHMRC	Consensus-Based Clinical Practice Guideline for the management of Volatile Substance Use in Australia	Available online
Northern Territory		
Remote Primary Health Care Manuals (RPHCM)	CARPA Standard Treatment Manual — Volatile substance misuse	Available online
North Queensland		
Queensland Health	Primary Clinical Care Manual — Sniffing petrol/glue/aerosol	Available online

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SECTION 6 — OTHER CONDITIONS AFFECTING CHILDREN

Otitis media

The World Health Organization advises that rates of otitis media greater than 4% represent a public health emergency.

OTITIS MEDIA IN NORTHERN AUSTRALIA

Rural and remote Aboriginal and Torres Strait Islander children have extremely high rates of severe otitis media (acute infection with perforation and chronic suppurative otitis media). Many Aboriginal and Torres Strait Islander children from remote communities have persistent otitis media and some associated hearing loss. Rates of perforation vary considerably from 5–67%. The World Health Organization considers perforation rates greater than 4% a public health emergency.

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 severe otitis media, such as acute otitis media with perforation and chronic suppurative otitis media. Persistent discharge for longer than 6 weeks is usually associated with secondary infection with multiple additional organisms (most commonly *pseudomonas, proteus, Escherichia coli* and *staphylococci*). 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 very early exposure to other children with persistent nasal discharge. The underlying cause of this phenomenon is socio-economic disadvantage. Similar rates of severe otitis media and persistent nasal discharge were seen in the poor neighbourhoods of all cities in the first half of the 20th century.

Other recognised risk factors from studies in Europe and the USA include: recent upper respiratory tract infection, family history of otitis media, child care attendance, large numbers of siblings, passive smoke exposure, lack of breastfeeding, and use of a dummy.

Symptoms and signs. Aboriginal and Torres Strait Islander children with otitis media should be categorised as having either severe or non-severe otitis media. Severe otitis media should be regarded as a preventable bacterial disease. 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. Technique for holding the child, holding the auroscope and straightening the canal is also very important.

- **Non-severe otitis media**
  - **Otitis media with effusion.** Presence of fluid behind an intact tympanic membrane without any of the symptoms or signs of an acute infection. Otitis media with effusion is very common in young Aboriginal and Torres Strait Islander 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.
  - **Acute otitis media.** Presence of fluid behind an intact tympanic membrane with at least one of the following symptoms or signs of an acute infection: ear pain, bulging or very red tympanic membrane. Acute otitis media is very common in young Aboriginal and Torres Strait Islander and non-Indigenous children.
- **Severe otitis media**
  - **Acute otitis media with perforation.** Acute otitis media plus perforation of the tympanic membrane within the last 6 weeks. Aboriginal and Torres Strait Islander 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 Aboriginal and Torres Strait Islander 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

Acute otitis media without perforation frequently occurs with other upper respiratory tract infections. Unless ear pain is present, these conditions cannot be reliably distinguished without careful otoscopy. Acute otitis media is confirmed by the presence of a bulging tympanic membrane. Otitis media with effusion is common in all children. Reliable diagnosis requires pneumatic otoscopy or tympanometry to confirm normal tympanic membrane mobility.

Severe otitis media

It is most important to distinguish between new perforations (acute otitis media with perforation) and chronic discharge (chronic suppurative otitis media). 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. 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 a painful canal wall 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.

PRINCIPLES OF MANAGEMENT

Most Aboriginal and Torres Strait Islander 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 compliance with recommended antibiotic therapy 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 the use of 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.

Clinical management depends on the clinical picture as classified above.

Otitis media

FURTHER INFORMATION

TELEPHONE ADVICE

Contact paediatrician in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines see *Appendix p200*.

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
Kimberley		
Kimberley Aboriginal Medical Services (KAMS)	Clinical Protocols/Guidelines — Ear problems in children	Available online
North Queensland		
Queensland Health	Primary Clinical Care Manual — Ear problems	Available online

EDUCATIONAL RESOURCES

Aussie Deaf Kids	Glue Ear: A guide for parents	Available online
Deafness Association of the NT	Support and Resources	Available online
Queensland Health	Healthy Hearing Program	Available online

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Diarrhoea in Aboriginal and Torres Strait Islander children

Acute infectious diarrhoea is a serious health problem for Aboriginal and Torres Strait Islander infants and children.

DIARRHOEA IN ABORIGINAL AND TORRES STRAIT ISLANDER CHILDREN

Historically over one third of Aboriginal and Torres Strait Islander children in the NT 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 Aboriginal and Torres Strait Islander communities is related to overcrowding, poor hygiene and sanitation all of which reflect socio-economic disadvantage. Recurrent episodes of diarrhoea can lead to Tropical Environmental Enteropathy Syndrome resulting in asymptomatic malabsorption.

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), cryptosporidium and giardia. However, many organisms including *E. coli,* giardia, salmonella and campylobacter are also found more than one pathogen is common.

Rotavirus infection is commonly associated with acidosis and osmotic diarrhoea, wasting and hypokalaemia. Infections with strongyloides and cryptosporidium are associated with prolonged diarrhoea in children admitted to hospital.

High rates of lactose intolerance (25%) in Aboriginal and Torres Strait Islander 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 has 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 fetus and severity appears related to time spent in the tropics. They also affect different races similarly. The causes are most likely environmental, with recurrent insults to the gut from infective organisms and changes in the gut microflora. An unhygienic living environment 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 underlying malnutrition
- Children with immune dysfunction
- Children with chronic disease, with renal disease, congenital heart disease, metabolic disorders, short-gut syndrome or ileostomy/colostomy.

Symptoms of gastroenteritis include 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. Aboriginal and Torres Strait Islander 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. Recent weight loss provides a good approximation of the amount of dehydration.

Investigations. Minor self-limiting episodes do not usually require laboratory investigation. Severe or prolonged cases usually require admission to hospital. Investigations might include stool samples for microscopy and culture, PCR multiplex, rotavirus antigen detection, shiga toxin (bloody diarrhoea), reducing substances for lactose intolerance, blood samples 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 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 generally require hospital admission for nasogastric or intravenous rehydration. Refer to regional rehydration guidelines (eg *CARPA Standard Treatment Manual*).

General principles 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; moderate dehydration; severe disease; chronic diseases; review 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 Aboriginal and Torres Strait Islander children

FURTHER INFORMATION

TELEPHONE ADVICE

Contact paediatrician, CDC/PHU in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines see *Appendix p200*.

Northern Territory		
NT Department of Health	Strong Women Strong Babies Strong Culture program — Information for Strong Women Workers, Government and Non-Government Professionals	Available online
Remote Primary Health Care Manuals (RPHCM)	CARPA Standard Treatment Manual — Child health/Diarrhoea	Available online
North Queensland		
Queensland Health	Primary Clinical Care Manual — Child with chronic diarrhoea	Available online

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Case study — Anaemia

It is the first term of the school year. You and the health centre staff have just completed the annual screening as part of 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 out of 51 children who live in the community. When you review the results, you notice that 14 children 4–15 years of age 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 Aboriginal communities is dietary iron deficiency. It is also increasingly recognised that infection, even mild viral infections, may transiently lower the Hb by several grams per litre. This is due to a decrease in iron utilisation and not iron deficiency, although they may co-exist.

Hookworm *(Ancylostoma duodenale)* is the main intestinal helminth in Australia, which causes anaemia due to blood loss. It is found north of the Tropic of Capricorn, ie north of Tennant Creek. It is unlikely to be a major contributor to the aetiology of anaemia due to the regular community 'deworming' programs over the past 10–20 years. 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* causes malabsorption, diarrhoea and growth faltering. Severe infestation and malabsorption may cause nutritional anaemia but only in children with diarrhoea.

Anaemia may be due to folate deficiency alone or in combination with iron deficiency. Prevalence of 0.6–9.8% has been reported in the NT for folate deficiency. These rates are lower than would be expected given the 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 Aboriginal and Torres Strait Islander Australians, although alpha thalassaemia carrier status has been identified in communities in Northern Territory and Northern Western Australia.

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 anaemia and 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 in the order of 62% or less, ie about one third with a screening result Hb less than 110g/L will not be truly anaemic.

How would you treat these children?

In your community the coverage of screening was high ie (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. Compliance to oral iron is often a problem in remote communities. Decisions about the type of iron regimen should be made in consultation with children, their carers and take into account staff resources.

The broad spectrum antihelminthic albendazole is currently recommended as a daily dose for 3 days. While a single dose is sufficient for hookworm, a 3 day course also covers 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 recur.

Would you repeat the Hb and if so when?

A repeat Hb is recommended in 1 month. If there has not been an increase in Hb, or it has fallen, then perform a FBC, film and red cell folate. If the child had been prescribed unsupervised daily iron and compliance has been poor, then offer IM or twice weekly supervised oral iron.

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 target information to suit them, which might include:

- Eat regular nutritious meals and snacks
- Encourage lots of different foods every day
- Encourage the intake of meat, fish, bush foods, iron fortified cereals (eg 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.

Are there any other preventive measures to be considered?

Anaemia is a significant public health problem. Anaemia surveillance and treatment alone will not solve the problem of iron deficiency anaemia although it may reduce the prevalence and severity. Overcrowding, poor environmental health, problems with the availability and cost of fresh food and social and family pressures, including alcohol and gambling all contribute towards the problem.

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. In the NT iron supplementation to prevent iron deficiency anaemia is rarely used.

Case study — Anaemia

FURTHER INFORMATION

TELEPHONE ADVICE

Contact paediatrician, nutritionist in your local jurisdiction.

MANAGEMENT GUIDELINES

For a list of general national and regional guidelines see *Appendix p200*.

EDUCATIONAL RESOURCES

NT Department of Health	Strong Women Strong Babies Strong Culture program — Information for Strong Women Workers, Government and Non-Government Professionals	Available online

KEY REFERENCES AND FURTHER READING

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Case study — Recurrent diarrhoea

Martin is 8 months old and lives with his extended family in a remote Aboriginal 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 2 week history of increasingly loose stools and high 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 another 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. There may be additional explanations for his loose stools such as concurrent infection with gut parasites. Another consideration is lactose intolerance secondary to recent gastroenteritis episodes.

Pathogens commonly causing persistent diarrhoea from initial infection include *Shigella*, enteropathogenic *Escherichia coli, Cryptosporidium*, *Giardia lamblia* and *Strongyloides*.

Lack of introduction of solids to supplement breastmilk from 6 months of age as well as acute infective gut insults and relative malabsorption, may have contributed to Martin's poor weight gain.

How dehydrated is Martin on the information provided?

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.

Calculate his fluid deficit

Deficit in mL	= 6.72 (wt in kg) x 4/100 (percentage dehydration) x 1000
	= 268.8mL

What is his maintenance fluid requirement?

For children up to 10kg maintenance requirement is 4mL/kg/hr

7 (wt in kg) x 4 (mLs) = 28mL/hr

Suggest a management plan for Martin

Continue to breastfeed and supplement early with oral rehydration solution

Deficit replacement over 8 hrs = 33.6mL/hr

Maintenance = 28.0mL/hr

Total = approx 62mL/hr over next 8 hrs

Martin's very patient 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. If she had not been successful, an alternative plan could have been to insert a nasogastric tube and put fluid down the tube every hour.

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 solids. 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. Some perianal excoriation is noted on examination. There are no clinical signs of dehydration. Martin's 380g weight gain = 380mL water gain, which means that he was approximately 5.3% dehydrated.

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.

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.

An Action Plan is worked out for Martin according to the Growth Assessment and Action (GAA) guidelines. The plan involves weekly weighs at the clinic and ongoing parental education and promotion of frequent nutritious foods for Martin. He will have regular reviews with the community doctor and the visiting paediatrician.

An Aboriginal and Torres Strait Islander Health Practitioner (ATSIHP) works closely with Martin's mother. She helps make sure that money is budgeted to buy foods 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.

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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 with one or more extra-articular features.

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 has a higher prevalence, morbidity and mortality amongst Aboriginal people and affects women much more frequently than men. The prevalence in Aboriginal people of the Top End has been estimated to be 1:1900, and up to three times that of non-Indigenous Australians.

The interaction between individuals and the environment appears to have a greater impact on the prevalence and outcomes of the condition than the prevalence of gene markers such as the C4A null allele. Improved living conditions and access to health services are likely to contribute to an improved prognosis for patients with SLE in Northern Australia.

AETIOLOGY AND PATHOGENESIS

The clinical manifestations of SLE are due to disturbed immune regulation. Tissue damage occurs from direct cytotoxicity of antibodies and complement, and from deposition of immune complexes.

CLINICAL PICTURE

Risk factors. In a study of 22 Aboriginal patients in the Top End, the 5 year survival was 60%. In first world settings the 5 year survival is greater than 90%. Renal involvement, active disease, and sepsis associated with immunosuppressive therapy were all identified as risk factors for poor outcomes. Infections, drugs and UV light are common trigger agents in SLE.

Symptoms and signs. Arthritis, arthralgia 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 Aboriginal people although discoid (skin) lupus may be present. However, concurrent skin conditions such as crusted scabies and bacterial infections are common. Renal involvement, particularly proteinuria, occurs in more than 50% of patients and carries the worst prognosis.

Central nervous system involvement includes mild psychiatric disturbances, frank psychosis, migraine and epilepsy. Cardiopulmonary features include pericarditis, pleurisy and fibrosing alveolitis. SLE may cause secondary Sjogren's syndrome (dry eyes and mouth) and secondary antiphospholipid antibody syndrome (miscarriages, thrombosis and occasionally chorea).

Table 11: Summary of classification criteria for systemic lupus erythematosus

Systemic Lupus International Collaborating Clinics (SLICC) Criteria	American College of Rheumatology (ACR) Criteria
Clinical criteria	**Clinical criteria**
1. Acute cutaneous lupus Flat red patches on the cheeks and nose (butterfly rash) that looks like sunburn	**1. Malar rash** Fixed erythema, flat or raised, over the cheek bones (malar eminences)
2. Chronic cutaneous lupus Thickened, red scaly patches that often appear on the cheeks, nose and ears, often not itchy or painful	**2. Discoid rash** Erythematous raised patches with adherent keratotic scaling and follicular plugging; atrophic scarring may occur in older lesions
3. Oral ulcers Palate, buccal, tongue or nasal ulcers in the absence of other causes	**3. Oral ulcers** Includes oral and nasopharyngeal, usually painless, observed by physician
4. Non-scarring alopecia Diffuse thinning or hair fragility with visible broken hairs in the absence of other causes	**4. Photosensitivity** Skin rash as a result of unusual reaction to sunlight, by patient history or physician observation

5. Synovitis ■ Involving 2 or more joints, characterized by swelling or effusion ■ OR Tenderness in 2 or more joints and at least 30 minutes of morning stiffness	**5. Non-erosive arthritis** Involving two or more peripheral joints, characterised by tenderness, swelling, or effusion
6. Serositis ■ Typical pleurisy for more than 1 day OR pleural effusions OR pleural rub ■ Typical pericardial pain (pain with reclining improved by sitting forward) for more than 1 day, OR pericardial effusion, OR pericardial rub, OR pericarditis by electrocardiography in the absence of other causes	**6. Serositis** ■ Pleuritis — convincing history of pleuritic pain or rubbing heard by a physician or evidence of pleural effusion ■ OR Pericarditis — documented by ECG or rub or evidence of pericardial effusion
7. Renal ■ Urine protein–to-creatinine ratio (or 24-hour urine protein) representing 500mg protein/24 hours ■ OR Red blood cell casts	**7. Renal disorder** ■ Proteinuria >0.5g/d or >3+ ■ OR Cellular casts
8. Neurologic ■ Seizures ■ Psychosis ■ Mononeuritis multiplex in the absence of other known causes ■ Myelitis ■ Peripheral or cranial neuropathy OR Acute confusional state in the absence of other known causes	**8. Neurologic disorder** Seizures without other cause or psychosis without other cause
9. Haemolytic anaemia	**9. Haematologic disorder** Haemolytic anaemia OR leukopenia (<4000/µL) OR lymphopenia (<1500/µL) OR thrombocytopenia (<100 000/µL) in the absence of offending drugs
10. Leukopenia ■ Leukopaenia (<4,000/mm³ at least once) inthe absence of other known causes ■ OR Lymphopaenia (<1,000/mm³ at least once)	
11. Thrombocytopenia (<100,000/mm³), At least once in the absence of other known causes	

Systemic lupus erythematosus (SLE)

Laboratory criteria	Laboratory criteria
1. Antinuclear antibody Level above laboratory reference range	**1. Antinuclear antibody** An abnormal titre of ANAs by immunofluorescence or an equivalent assay at any point in time in the absence of drugs known to induce ANAs
2. Anti-double-stranded DNA antibody Level above laboratory reference range (or >2-fold the reference range if tested by ELISA)	**2. Immunological disorders** Anti-dsDNA, anti-Sm, AND/OR anti-phospholipid
3. Anti-Sm antibody Presence of antibody to Sm nuclear antigen	
4. Antiphospholipid antibody positivity As determined by any of the following: ■ Positive test result for lupus anticoagulant ■ False-positive test result for rapid plasma reagin ■ Medium or high-titer anticardiolipin antibody level (IgA, IgG, or IgM) ■ Positive test result	
5. Low complement Low C3 OR low C4 OR low CH50	
6. Direct Coombs' test Positive test in the absence of haemolytic anaemia	
Requirement for diagnosis	**Requirement for diagnosis**
■ Must meet 4 above criteria (with at least one criterion being clinical and at least one criterion being immunological) **OR** ■ Lupus nephritis proven by biopsy and at least one immunological criterion	■ Must meet 4 of above 11 criteria

Source: Adapted from Golder V and Hoi A. Systemic lupus erythematosus: an update. Med J Aust 2017; 206 (5): 215-220. © Copyright 2017 The Medical Journal of Australia — reproduced with permission.

Investigations. Antinuclear antibodies are detected in more than 90% of patients, but are commonly found in other autoimmune diseases. AntidsDNA antibodies are more specific but only occur in about 50% of patients. The ESR is usually raised in active disease while the CRP is rarely raised in the absence of infection. FBC may demonstrate leucopoenia, anaemia or thrombocytopenia. 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 include acute rheumatic fever, rheumatoid arthritis, gonococcal arthritis, other autoimmune diseases (eg mixed connective tissue disease and systemic sclerosis), dermatitis, epilepsy, multiple sclerosis, psychiatric disorders, idiopathic thrombocytopenic purpura and vasculitis (eg polyarteritis nodosa).

PRINCIPLES OF MANAGEMENT

Systemic lupus erythematosus (SLE) is not curable and complete remission is rare, therefore the health team together with the client 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 steroids. In the longer term, antimalarial or cytotoxic treatments can be used. Patients on daily hydroxychloroquine need yearly ophthalmology reviews. Latent infections, particularly **tuberculosis, melioidosis, hepatitis B** and **strongyloidiasis** should be excluded or, if present, treated prior to immunosuppressive therapy.

FURTHER INFORMATION

TELEPHONE ADVICE

Contact specialist physician in your local jurisdiction.

Northern Territory		
Arthritis and Osteoporosis NT	http://www.aont.org.au	1800 011 041

MANAGEMENT GUIDELINES

National	
Lupus and You. A Practical Guide to Understanding, Managing and Living with SLE	Schned, E. Lupus and You, A Practical Guide to Understanding, Managing and Living with SLE. Minnesota: Health Systems Minnesota; 1997

EDUCATIONAL RESOURCES

Arthritis and Osteoporosis NT	Arthritis & Osteoporosis NT (AONT)	Online

KEY REFERENCES AND FURTHER READING

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Machado-Joseph disease (MJD)

Machdo-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 1800s and is now found in 12 remote communities across the Northern Territory and North Queensland. There are close to 50 known affected individuals, another 50 being monitored for the disease, and more than 600 first degree relatives of cases who are at risk.

The **MJD Foundation** was established in 2008 to improve the quality of life for Aboriginal Australians and their families living with Machado-Joseph disease in Arnhem Land and other affected communities in the NT and QLD.

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, resulting in multi-system degeneration affecting the cerebellum and its connections, including the basal ganglia, dorsal columns, upper and lower motor neurones and peripheral and autonomic nerves.

There are three phenotypes, determined by age of onset. The disease may present earlier and progress faster with each generation, especially when passed from father to son.

- **Type 1 —** presents in late teenage years, tends to progress faster with greater spasticity and impairment of coordination.
- **Type 2 —** presents late 20s to mid 30s and is the most common. Cases may have the full range of symptoms and signs between types 1–3 (see *Clinical picture*).
- **Type 3 —** presents in 50s and may not affect longevity. It is characterised by ataxia, distal muscle wasting, depressed reflexes and decreased pinprick, vibration and position sense.

CLINICAL PICTURE

Risk factors. Parent or grandparent with the condition.

Symptoms and signs. A complete neurological examination is required. The condition is characterised by:

- Incoordination and spasticity — a lurching unsteady gait, unsteady hand movements, slow and indistinct speech, progressive dysphagia and dysarthria. Tendon reflexes may be normal, decreased or increased
- Amyotrophy — proximal or distal wasting of limb muscles and weakness of the shoulder and hip girdles
- Ophthalmoplegia — loss of upgaze, nystagmus and possible loss of saccadic eye movement, diplopia especially to distant gaze, and bulging eyes
- Bowel and bladder dysfunction — constipation, faecal retention, pseudo-bowel obstruction, urinary incontinence and retention
- Sleep disturbances — insomnia, obstructive sleep apnoea, restless legs, rapid eye movement sleep disorder.

Investigations. Genetic testing is now available for the chromosome 14 abnormality. Pre-test counselling is essential especially for families who are requesting that their children be tested.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses may include other spinocerebellar ataxias, demyelination disorders and brainstem ischaemia or infarction.

PRINCIPLES OF MANAGEMENT

Multidisciplinary assessment and ongoing clinical management and social support are required including the following:

- **Bladder management** includes assessment of post void volumes with ultrasounds, management of infections and incontinence. Discuss with rehabilitation consultant, renal physician, and refer to the Machado-Joseph Disease 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
- **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 range of movement exercises in consultation with physiotherapists, antispasmodic agents and the treatment of underlying precipitants such as urinary tract infections or faecal impaction
- **Dysphagia and dysarthria** are managed in consultation with a speech pathologist. This may include oral feeds with thickened fluids, tongue, lip and mouth exercises, and communication using closed questions
- **Respiratory care** is managed in consultation with a physiotherapist and respiratory physician. Sleep studies should be considered
- **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
- **Carer stress and respite.** Support of carers in developing knowledge and skills to meet the needs of their family and manage problems as they arise. Refer to regional aged and disability service for community/respite supports and services
- **Depression.** May require psychological intervention and/or 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.

FURTHER INFORMATION

TELEPHONE ADVICE

Contact rehabilitation or renal physician, allied health services in your local jurisdiction.

National	
The Machado-Joseph Disease Foundation	1300 584 122

EDUCATIONAL RESOURCES

The MJD Foundation	Medical protocols, educational videos	Available online

KEY REFERENCES AND FURTHER READING

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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 doesn't take them. Recently he has had a number of 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 couldn't sleep last night, he is really short of breath and has pain in the right side of the chest on breathing. He can hardly stand up. He has been 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 can't 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 examined for.

What is your immediate management, given that the ambulance is at least an hour away?

Kelvin is hypotensive and hypoxic and needs resuscitation and early commencement of antibiotics

1. Insert two IV lines
2. Administer an initial bolus of IV fluid over the first 30 minutes. 10–15mL/kg of normal saline could be used as a guide
3. Provide oxygen via mask, adjust flow rate according to improvement in SpO2
4. Draw blood for cultures and other baseline tests (FBC, UEC, LFT, BGL, CRP, ESR)
5. Obtain urine sample for MC&S
6. Administer IV antibiotics. Use a regimen that will cover melioidosis as well as other likely organisms.

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. A HBsAg positive patient taking oral steroids for chronic obstructive pulmonary disease should have regular monitoring of their infection.

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

MANAGEMENT GUIDELINES

National		
Monash University	Spleen Australia	Available online
The Australasian Society for HIV, Viral Hepatitis and Sexual Health Medicine (ASHM)	B Positive: all you wanted to know about hepatitis B — a guide for primary care providers	Available online
Northern Territory		
Department of Health	Opportunistic infections prevention in patients with immunosuppression TEHS Guideline	Available online

KEY REFERENCES AND FURTHER READING

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APPENDIX

Acronyms

ABL	Australian bat lyssavirus
ACR	albumin creatinine ratio
AFB	acid fast bacilli
AFP	alpha foetoprotein
AIDS	acquired immunodeficiency syndrome
ALP	alkaline phosphatase
ALT	alanine aminotransferase
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
ATSIHP	Aboriginal and Torres Strait Islander health practitioner
bd	bis die — twice a day
BFV	Barmah Forest virus
BGL	blood glucose level
BP	blood pressure
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
cm	centimetre
CMV	cytomegalovirus
CNS	central nervous system
CRP	c-reactive protein
CSF	cerebrospinal fluid
CSOM	chronic suppurative otitis media
CT scan	computed tomography
dB	decibel
DEET	N,N-diethyl-m-toluamide
DNA	deoxyribonucleic acid
DOT	directly observed therapy
dsDNA	double stranded deoxyribonucleic acid
EBV	Epstein-Barr virus
ECG	electrocardiogram
ELISA	enzyme-linked immunosorbent assay
ENT	ear nose and throat
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
GP	general practitioner
GUD	genital ulcer disease
HAV	hepatitis A virus
Hb	haemoglobin
HBeAg	hepatitis B envelope antigen
HBsAg	hepatitis B surface antigen

HBV	hepatitis B virus
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 Streptococcal
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
MC&S	microscopy, culture and sensitivity
mcg/dL	micrograms per decilitre
MDRTB	Multi-drug resistant tuberculosis
mg	milligram
min	minute
MJD	Machado-Joseph disease
mL	millilitre
mm	millimetre
mmol	millimole
MRSA	methicillin-resistant *staphylococcus aureus*
MVE	Murray Valley encephalitis
NAAT	nucleic acid amplification test
NGO	non-government organisation
NGU	non-gonococcal urethritis
NS1	nonstructural protein 1
NSAID	non-steroidal ant-inflammatory drug
NT	Northern Territory
NTM	non-tuberculosis mycobacteria
PCCM	Primary Clinical Care Manual
PCR	polymerase chain reaction
PHU	Public Health Unit
PID	pelvic inflammatory disease
po	taken orally
PVC	pneumococcal conjugate vaccine
qid	quater in die — 4 times a day
QLD	Queensland
RHD	rheumatic heart disease
RPR	rapid plasma reagin test
RR	respiration rate
RRF	Ross River fever
RRV	Ross River virus
RT–PCR	reverse transcription — polymerase chain reaction
S100	highly specialised drugs program on Pharmaceutical Benefits Scheme
SE Asia	South East Asia
SLE	systemic lupus erythematosus
STI	sexually transmitted infection
T	temperature
TB	tuberculosis
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

Telephone advice

NORTHERN TERRITORY

Hospitals	
Royal Darwin Hospital	**08 8922 8888**
Tennant Creek Hospital	**08 8962 4399**
Alice Springs Hospital	**08 8951 7777**
Gove District Hospital	**08 8987 0211**
Katherine Hospital	**08 8973 9211**
Centre for Disease Control (CDC)	
Darwin	**08 8922 8044**
Katherine	**08 8973 9049**
Tennant Creek	**08 8962 4259**
Nhulunbuy	**08 8987 0357**
Alice Springs	**08 8951 7540**

Sexual Health Units	
Clinic 34	
— Darwin	**08 8999 2678**
— Katherine	**08 8973 9046**
— Alice Springs	**08 8951 7549**
NT Syphilis register	
— Alice Springs	**08 8951 7552**
— Darwin	**08 8922 7818**
Alcohol and Other Drugs	
Darwin	**08 8922 8399**
Alice Springs	**08 8952 8412**

KIMBERLEY

Hospitals	
Broome Hospital	**08 9194 2222**
Royal Perth Hospital	**08 9224 2244**
Kununurra Hospital	**08 9166 4222**
Population Health Units (PHU)	
Kimberley Population Health Unit — Broome	**08 9194 1630**
Kununurra Population Health Unit	**08 9168 2498**
Communicable Disease Control Directorate	
— Perth	**08 9388 4868**
— Notifications	**08 9388 4852**
— A/H Infectious diseases emergency	**08 9328 0553**

Sexual Health Units	
Fremantle (South Terrace)	**08 9431 2149**
Perth (Royal Perth Hospital)	**08 9224 2178**
Broome (Kimberley Population Health Unit)	**08 9194 1630**
WA syphilis register	**08 9194 1641** **08 9194 1646**
Alcohol and Other Drugs	
Kimberley Alcohol and Drug Services — Broome	**08 9194 2640**

NORTH QUEENSLAND

Hospitals	
Cairns Hospital	**07 4226 0000**
Townsville Hospital	**07 4433 1111**
Mt Isa Hospital	**07 4744 4444**
Public Health Units (PHU)	
Tropical Public Health Services — Cairns	**07 4226 5555**
Mt Isa and Gulf	**07 4744 7178**
Townsville	**07 4433 6900**

Sexual Health Units	
Cairns	**07 4226 4769**
Townsville	**07 4433 9600**
QLD syphilis register	**1800 032 238**
Alcohol and Other Drugs	
Cairns	**07 4226 3900**

Notifiable conditions

NATIONAL

Communicable Diseases Network Australia (CDNA)	Australian notifiable diseases and case definitions	Available online

NORTHERN TERRITORY

Centre for Disease Control (CDC)	Notifiable diseases	Available online

KIMBERLEY

WA Department of Health	Notification of infectious diseases and related conditions	Available online

NORTH QUEENSLAND

Queensland Health	List of notifiable conditions	Available online

General management guidelines

NATIONAL

Therapeutic Guidelines	Available online
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) Northern Territory HealthPathways	Available online
Centre for Disease Control (CDC) Resources and Publications	Available online

KIMBERLEY

Kimberley Aboriginal Medical Services (KAMS) Clinical Protocols/Guidelines	Available online
WA Department of Health Communicable disease guidelines	Available online

NORTH QUEENSLAND

Queensland Health Primary Clinical Care Manual	Available online
Queensland Health Communicable disease control guidance	Available online

Sexual health guidelines

NATIONAL

The Australasian Society for HIV, Viral Hepatitis and Sexual Health Medicine (ASHM) — Available online

- Australian STI Management Guidelines for use in Primary Care
- Australasian Contact Tracing Guidelines

Melbourne Sexual Health Clinic — Available online

Treatment Guidelines

NORTHERN TERRITORY

Remote Primary Health Care Manuals (RPHCM) — Available online

- CARPA Standard Treatment Manual
- *Minymaku Kutju Tjukurpa* — Women's Business Manual 6th Edition

Centre for Disease Control (CDC) — Available online

- NT Guidelines for the Management of Sexually Transmitted Infections in the Primary Health Care setting
- Contact Tracing for Sexually Transmitted Infections

KIMBERLEY

Kimberley Aboriginal Medical Services (KAMS) — Available online

Clinical Protocols/Guidelines

WA Department of Health — Available online

Silver book. Guidelines for managing sexually transmitted infections and blood-borne viruses

NORTH QUEENSLAND

Queensland Health — Available online

Primary Clinical Care Manual

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