TROPICAL HEALTH IN THE TOP END

an introduction for health practitioners
“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......
TROPICAL HEALTH IN THE TOP END
AN INTRODUCTION FOR HEALTH PRACTITIONERS

Produced by the Tropical Health Working Group of the Top End Division of General Practice.
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This guide was developed by the Top End Division of General Practice (TEDGP) as an introduction to tropical health in the Top End and not as a source of management guidelines for the treatment of individual patients. Neither TEDGP nor any individual contributor to this guide shall be liable to users of the guide or other persons for any loss or harm arising from errors or omissions that this guide may contain.

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FOREWORD

This guide was developed by the Top End Division of General Practice in response to a need identified by remote Top End GPs. It aims to provide orientation and advice for doctors and other health practitioners new to the Top End about a range of locally important conditions that are less frequently encountered in clinical practice in southern parts of Australia.

The Top End has many unique features which affect our local medical practice. The warm, seasonally wet environment is home to many insects and micro-organisms that are uncommon in southern parts of the country. The population is young and culturally diverse with a high proportion of Aboriginal people. Many doctors work in isolated communities where the social and environmental contributors to ill health are glaring daily realities. The responsibilities of health practitioners then broaden from individual patients and families to entire communities. All these factors make medical practice in the Top End unique and challenging.

This guide contains summaries of 50 conditions and includes some clinical scenarios which highlight important local issues. It identifies the available sources of further advice such as local Centres for Disease Control, and relevant publications such as the CARPA manual. While there are a great many issues highly relevant to the health conditions that are included, it would be impossible to discuss them comprehensively and the content of this guide is intentionally brief, with a clinical focus.

Acknowledgments are due to many people and organisations. Thanks especially to Bart Currie and Vicki Krause who provided specialist editorial advice. Thanks also to the following GPs and other health practitioners who contributed: Paul Armstrong, Paul Burgess, Alan Clough, Christine Connors, Keith Edwards, Dan Ewald, Dana Fitzsimmons, Chris Harrison, Paul Kelly, Sue Lenthal, Justine Mayer, Jeanette McGregor, Jacki Mein, Peter Morris, Hung The Nguyen, Patrick O’Brien, Barbara Paterson, Brett Ritchie, Jan Savage, Steven Skov, Tania Wallace, Kate Walker, Jane Walters, Annie Whybourne, Jo Wright and Nathan Zweck.

The Commonwealth Department of Health and Ageing, as the funding agency of the Top End Division of General Practice, the Northern Territory Department of Health and Community Services, the Menzies School of Health Research and the Centre for Remote Health are thanked for their invaluable support of this project.

Finally I would like to thank my colleagues in the Tropical Health Working Group, Meredith Arnold, Simon Morgan, Wendy Page and Jan Schmitzer who guided the project with their vision, expertise and practical experience and Nilva Egana for her exceptional organisational skills, efficiency and dedication to the task.

We trust you find this a helpful resource. It is built upon the collective experience of practitioners and researchers in Northern Australia and its content will continue to be refined and improved with your feedback. Comments should be directed to the Top End Division of General Practice, GPO Box 757, Darwin, NT 0801.

Fay Johnston  BMBS MAE FACRRM FAFPHM
Editor
HOW TO USE THIS GUIDE

This guide contains an introduction to many conditions frequently encountered in the Top End. It is intended to be used as a starting point for information about particular 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 the Top End** - 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 the Top End.

An electronic version of this guide is available on the TEDGP website http://www.tedgp.asn.au
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Group A
Streptococcal Infections

Up to 70% of Aboriginal children may have streptococcal skin sores at any one time.

GROUP A
STREPTOCOCCAL INFECTIONS IN THE TOP END

Invasive streptococcal infections cause a great deal of morbidity and mortality in Northern Australia. Streptococcus pyogenes (group A streptococcus) causes skin and throat infections and also invasive disease such as bacteraemia, necrotising fasciitis, toxic shock syndrome and septic arthritis. Certain strains are linked with acute post-streptococcal glomerulonephritis and rheumatic fever, both of which have extremely high incidence rates in the Top End. Community surveys demonstrate that up to 70% of Aboriginal children may have streptococcal skin sores at any one time. Diseases due to S. pneumoniae (pneumococcal disease) are discussed separately.

AETIOLOGY AND PATHOGENESIS

Streptococcal species are part of normal skin and throat flora. Newly introduced and invasive strains spread quickly particularly in conditions of overcrowding and poor hygiene. Streptococci cause infection by adherence, respiratory tract seeding (blood borne to more distant sites) and escaping phagocytosis. S pyogenes also produces a number of toxins.

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 and tend to be rapid in onset. Locally acquired skin infections in tropical Australia (skin sores, pyoderma, impetigo) are usually due to streptococci and are often secondary to scabies. Cellulitis may follow the initial skin sore. Patients with streptococcal infections should be checked for signs of post streptococcal syndromes such as the presence of a heart murmur or oedema.

Investigations. As streptococcal skin infections are so common in Aboriginal children, anti streptococcal
antibodies (ASOT or Anti-DNase B) are often elevated and not helpful in distinguishing current from previous infection. They are important to confirm the diagnosis of acute post streptococcal syndromes such as acute rheumatic fever and post-streptococcal glomerulonephritis, where the presence of an acute process is confirmed by a concurrently reduced complement (C₃) level. Culture from a sterile site such as cerebral spinal fluid or blood is diagnostic of invasive infection. Sputum, throat and wound cultures need to be interpreted in the context of the clinical picture.

DIFFERENTIAL DIAGNOSIS

Streptococcal skin infections look like typical impetigo although they are less commonly found on the face. Differential diagnosis depends on the clinical presentation, and other major bacterial pathogens should be sought on culture of specific specimens mentioned above. Bullous or pustular impetigo is more frequently due to *Staphylococcus aureus*.

PRINCIPLES OF MANAGEMENT

The treatment of choice for widespread streptococcal skin infection is a single dose of benzathine penicillin. The aim 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 the importance of washing to reduce the risk of streptococcal skin infection. Identify any problems with their house that may prevent washing on a daily basis including health hardware problems. These should be reported to the Community Council and Environmental Health Officer. The family should also be advised about washing linen and airing out mattresses to minimise the risk of reinfection.
A NOTE ABOUT PENICILLIN

Penicillin remains the mainstay of treatment for streptococcal diseases. It is commonly prescribed in remote areas of the Northern Territory and several preparations are available. Prescribers should always refer to the most recent edition of the Therapeutic Guidelines: Antibiotic, or the CARPA manual for guidelines 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 six hourly. It is the treatment of choice for many infections.

Procaine penicillin
This is an intramuscular preparation of penicillin that was designed to extend the half-life of benzyl penicillin. It provides 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 penicillin
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 regular monthly injection to prevent recurrent rheumatic fever following an episode of acute rheumatic fever.
FURTHER INFORMATION

TELEPHONE ADVICE

Infectious diseases or general physician
Royal Darwin Hospital 8922 8888

MANAGEMENT GUIDELINES

CARPA-Standard Treatment Manual
Central Australian Division of Primary Health Care 8950 4800

Healthy Skin Program-Guidelines for Community Control of Scabies, Skin Sores and Crusted Scabies 2002
Centre for Disease Control-Darwin 8922 8044

Guidelines for the Control of Acute Post-Streptococcal Glomerulonephritis 1997
Centre for Disease Control-Darwin 8922 8044

Communicable Disease Surveillance in the NT-Guidelines for the Reporting of Notifiable Conditions
Centre for Disease Control-Darwin 8922 8089

Therapeutic Guidelines: Antibiotic
Therapeutic Guidelines LTD 1800 061 260
Or http://www.tg.com.au

EDUCATIONAL RESOURCES

Flip Chart & Video-Healthy Skin Story
Ngalkanbuy Health Service Galiwinku Aboriginal Services Inc 8987 9031

The Public Health Bush Book
Dept. of Health & Community Services-Wellness promotion 8999 2691

A Handbook of Skin Conditions in Aboriginal Population of Australia.
Dr Allen Green

Video-Australian Aborigines and Their Skin Conditions. Dr Allen Green
Royal Australian College of General Practitioners (03) 9214 1414

FURTHER READING

**Staphylococcal Infections**

**Infections tend to be purulent, acute in onset and to localise rapidly.**

**STAPHYLOCOCCAL INFECTIONS IN THE TOP END**

Infections caused by *Staphylococcus aureus* are common in the Top End, 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 of sterile sites are less common but do occur, e.g., sepsicaemia, endocarditis and pneumonia associated with aspiration. Staphylococcal septicaemia has a high case fatality rate. *S. aureus* is the most common cause of septic arthritis, osteomyelitis and pyomyositis (muscle abscess).

**AETIOLOGY AND PATHOGENESIS**

Staphylococci, particularly *S. aureus*, are ubiquitous normal flora on skin and mucous membranes. Their pathogenicity comes from toxin production, ability to breach epithelial surfaces and resist immune defence mechanisms to cause focal infections (abscesses) in sterile sites. Resistant organisms have caused major infection control problems in hospitals (e.g., methicillin-resistant *Staphylococcus aureus* (MRSA) or 'golden staph'). The Top End has a particular community acquired strain of MRSA which is identified on antibiotic susceptibilities. It is thought that widespread use of antibiotics in remote communities has selected and favoured the emergence and dissemination of community acquired MRSA. Restriction of ceftriaxone use is recommended to avoid increasing antibiotic resistance.

**CLINICAL PICTURE**

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

**Symptoms and signs.** These depend on the site of infection. For example superficial skin infections present as weeping or crusted sores, while deeper infections present as cellulitis or hot tender abscesses. Infections tend to be purulent, relatively acute in onset and to localise rapidly. They can be recurrent.

*S. aureus* is the most common cause of mastitis and breast abscesses.

**Investigations.** As staphylococci can be cultured from normal skin, the diagnosis of *S. aureus* as a pathogen is supported by the presence of neutrophils in diagnostic specimens from the affected area, and is more significant if cultured from a sterile site. It is a hardy organism that is easily cultured on routine media.

**DIFFERENTIAL DIAGNOSIS**

Staphylococcal infection should be differentiated from other causes of bacterial skin or respiratory infection. Look for signs of scabies. Infections of the occipital scalp are usually associated with infestations of head lice. Differential diagnosis for pneumonia is described in the case study of community acquired pneumonia.

**PRINCIPLES OF MANAGEMENT**

Treatment depends on antibiotic sensitivity of the organism and the seriousness of infection. Drainage of focal infection may be required. Deep infections in body cavities (e.g., pneumonia, osteomyelitis and endocarditis) require hospitalisation and long-term high dose antibiotics. Recurrent staphylococcal abscesses may warrant a staphylococcal eradication program including skin washes and nasal mupirocin (see the CARPA manual for details). Underlying conditions (e.g., scabies, diabetes) should be treated. Superficial skin sores are predominantly streptococcal and therefore usually respond to intramuscular benzathine penicillin alone.
FURTHER INFORMATION

TELEPHONE ADVICE

| Infectious diseases or general physician | Royal Darwin Hospital | 8922 8888 |

MANAGEMENT GUIDELINES

| CARPA-Standard Treatment Manual | Central Australian Division of Primary Health Care | 8950 4800 |
| Healthy Skin Program-Guidelines for Community Control of Scabies, Skin Sores and Crusted Scabies 2002 | Centre for Disease Control-Darwin | 8922 8044 |
| Guidelines for the Control of Acute Post-Streptococcal Glomerulonephritis 1997 | Centre for Disease Control-Darwin | 8922 8044 |
| Therapeutic Guidelines: Antibiotic. Latest Version | Therapeutic Guidelines LTD | 1800 061 260 |

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| Flip Chart & Video-Healthy Skin Story | Ngalkanbuy Health Service Galiwinku Aboriginal Services Inc | 8987 9031 |
| The Public Health Bush Book | Dept. of Health & Community Services-Wellness promotion | 8999 2691 |
| A Handbook of Skin Conditions in Aboriginal Population of Australia. Dr Allen Green | Office of Aboriginal & Torres Strait Islander Health (OATSIH) | 89463481 |
| Video-Australian Aborigines and Their Skin Conditions. Dr Allen Green | Royal Australian College of General Practitioners | (03) 9214 1414 |

FURTHER READING

Miranda, a two year old child is referred to you by the baby clinic health worker because of growth failure. You note from her chart that her growth has been poor and she has gained little weight over the last three months. She is often seen at the clinic for skin sores. Today she has several pustules around her wrists, some crusty lesions on her buttocks and some oozing sores on her feet.

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

Her poor growth and her skin condition both require attention. You investigate and work out an Action Plan according to the Growth Assessment and Action (GAA) guidelines. The plan includes weekly weighs at the clinic and ongoing education, support and medical reviews. You list her for review by the visiting paediatrician. For more information about GAA contact the community paediatrician in CDC-Darwin.

You treat her skin infection with single dose IM benzathine penicillin and advise on ‘washing kids’ 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?

- Not washing due to poor ‘health hardware’.
- Recurrent scabies.
- Overcrowding.
- Undiagnosed case of crusted scabies in the house- assess all household contacts for this.

What are some of the associated diseases that might occur 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).

What are the public health issues that you might consider after seeing this child and a number of other children with similar presentations?

- Health hardware and overcrowding.
- Running a ‘Healthy Skin’ program to reduce scabies and Streptococcal infection prevalence.
- Community based growth promotion initiatives such as the ‘Strong Women Strong Babies Strong Culture’ (SWSBSC) program.
FURTHER INFORMATION

TELEPHONE ADVICE

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<th>Community paediatrician</th>
<th>Centre for Disease Control-Darwin</th>
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Acute Post Streptococcal Glomerulonephritis

ACUTE POST STREPTOCOCCAL GLOMERULONEPHRITIS IN THE TOP END

Acute post streptococcal glomerulonephritis (APSGN) occurs in the NT both as sporadic cases in individuals and in epidemics that can involve numerous children within one or several related communities. In non-epidemic years there are usually less than 5 notified cases per year, while in epidemic years there may be 50-100 total notifications from many communities across the Top End. APSGN is associated with scabies, skin sores and crowded living conditions and is a risk factor for chronic renal disease in Aboriginal adults.

AETIOLOGY AND PATHOGENESIS

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. It is thought to be immunologically mediated but the exact mechanism is unclear. In the NT it is usually associated with skin rather than throat infections. As infection with a newly introduced streptococcal strain spreads rapidly, it is common to have geographic and temporal clustering of cases, often with multiple cases in one household.

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, 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 >80 if 13 years or younger, or >90 if older) and visible haematuria can occur.

Investigations. Urine dipstick may show protein and >2+ haemoglobin or blood. Microscopy may show dysmorphic red blood cells (RBC) >10/mm³, white blood cells (WBC), and hyaline, granular and red blood cell casts. Blood tests usually demonstrate elevated erythrocyte sedimentation rate (ESR), mild normochromic normocytic anaemia, and elevated creatinine. Anti-streptococcal antibodies (ASOT or Anti-DNase B) titres are usually high, consistent with recent streptococcal infection. However, as streptococcal infections are common in Aboriginal 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

APSGN should be differentiated from other infectious processes involving the kidney. An exacerbation of chronic glomerulonephritis (GN) may occur post streptococcal infection but the latent period is usually very short (1-4 days). Bacterial endocarditis may lead to high levels of circulating immune complexes and subsequent development of glomerulonephritis. Other infections are occasionally associated with nephritis eg hepatitis B virus (HBV), hepatitis C virus (HCV), glandular fever, pneumococcal pneumonia, typhoid, syphilis, leptospirosis, scrub typhus and P. falciparum malaria. Other entities which may mimic APSGN are systemic lupus erythematosus (SLE), polyarteritis nodosa, acute tubular necrosis and Henoch-Schönlein disease. Haematuria may be caused by renal calculi or urinary tract infections. The important differentiation is usually the setting of recent streptococcal infection or contact with other cases.

PRINCIPLES OF MANAGEMENT

Treatment. Most cases are either subclinical or mild and are managed in the community with close observation of weight and blood pressure. More severe cases require hospital management of circulatory overload and hypertension. Admit any case with hypertension or oedema. Mortality is rare (<1%) and unlike rheumatic fever, recurrences are rare.

APSGN is a notifiable condition to be reported by all CLINICIANS in the Northern Territory. Cases should be reported to the Centre for Disease Control in your district by phone or fax.
Prevention of further cases is important: All suspected cases should be notified to CDC and the guidelines for management and prevention of further cases should be followed. All close contacts aged 3-15 years and all other contacts with skin sores should receive a single IM dose of benzathine penicillin to eradicate the virulent streptococcal strain. Promote washing, especially of children and treat scabies. If more than one case is suspected, liaise with the Centre for Disease Control. Two clinical cases in a week or three in a month usually constitutes an outbreak and community wide treatment may be indicated. CDC staff can visit your community to assist with case and contact treatment, testing and follow-up.

FURTHER INFORMATION

TELEPHONE ADVICE

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MANAGEMENT GUIDELINES

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<td>Poster-Deadly Kids</td>
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FURTHER READING

ACUTE RHEUMATIC FEVER AND RHEUMATIC HEART DISEASE IN THE TOP END

Over the past few 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 heart disease. The highest published incidence of acute rheumatic fever in the world is in Aboriginal people living in the Top End. Many cases of acute rheumatic fever are not detected resulting in missed opportunities for prevention and a relatively high proportion of children who have established valvular heart disease at the time of first diagnosis.

AETIOLOGY AND PATHOGENESIS

Rheumatic Fever (RF) is an inflammatory illness of the heart, joints, central nervous system and subcutaneous tissues that is mediated by the immune response to infection with group A beta-haemolytic streptococci (Streptococcus pyogenes). It has traditionally been believed that rheumatic fever follows S.pyogenes infection of the throat only. The paradox in Aboriginal communities is that throat carriage rates of S.pyogenes are usually very low (<5%) and that symptomatic pharyngitis is uncommon, whereas streptococcal pyoderma is endemic—up to 70% of children in some communities are affected at any time. Much of the streptococcal pyoderma is secondary to scabies infestation.

Rheumatic heart disease (RHD) is a complication of rheumatic fever which involves dysfunction of the heart valves leading to incompetence and stenosis, and may result in early heart failure and death. It is more likely to develop with each recurrence of ARF.

CLINICAL PICTURE

Risk factors include socio-economic disadvantage and repeated streptococcal infections.

Symptoms and signs. Diagnosis is usually made using the revised Jones Criteria of 1984 (see table). A new case requires two major criterion, or one major and two minor; AND evidence of recent group A streptococcal infection. That is, a positive throat culture or anti-streptolysin O antibody titre (ASOT) > 256 IU or antideoxyribonuclease B (Anti-DNase B) >300 IU.

Diagnosis of a recurrence of RF can be made if there is documented past history of RF or established rheumatic heart disease; AND one major criterion or two minor criteria; AND evidence of recent group A streptococcal infection.

Investigations should include full blood count (FBC), erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), throat swab culture, electrocardiogram (ECG) and echocardiogram.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of ARF depends upon its mode of presentation. Polyarthritis must be distinguished from the arthritis of arboviral infections (Ross River and Barmah Forest viruses), acute febrile episodes of systemic lupus erythematosus (SLE), juvenile chronic arthritis, Henoch-Schönlein
disease, serum sickness and septic arthritis including disseminated gonococcal infection. Subacute disease with carditis but little or no joint involvement may be confused with cardiac conditions such as a viral myocarditis, tuberculous pericarditis or acute myocarditis with cardiac failure. Subclinical ARF is probably quite common. Vague illness in Aboriginal people, associated with aches and pains, pallor and tachycardia, should always be investigated for acute rheumatic fever.

**PRINCIPLES OF MANAGEMENT**

It is recommended that all cases of ARF or probable ARF are admitted to hospital for investigation and commencement of a management plan and education. It is important to fully investigate all potential cases of rheumatic fever because of the seriousness of the cardiac sequelae and the high chance of recurrence. If there is doubt about the diagnosis, commence intramuscular (IM) benzathine penicillin before waiting for investigations results, or specialist review. All cases should be notified to CDC and included on the rheumatic fever and heart disease register. This register is maintained to facilitate prophylaxis and clinical follow up. Secondary prophylaxis aims to decrease recurrences of ARF and consists of monthly Benzathine penicillin injections (See Figure 6).

Long term management of people with rheumatic heart disease needs to be developed in consultation with the specialist physician and/or paediatrician. People should be encouraged to seek treatment if they have a sore throat or scabies/skin sores, or symptoms of rheumatic fever recurrence. People with RHD need antibiotics prior to certain procedures to prevent endocarditis. Refer to the *Therapeutic Guidelines: Antibiotic*, or the CARPA Manual.

**Rheumatic fever (including recurrent episodes)** is a notifiable condition to be reported by all CLINICIANS in the Northern Territory. Cases should be reported to the Centre for Disease Control in your district by phone.

<table>
<thead>
<tr>
<th>Major</th>
<th>Minor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carditis—eg new heart murmur</td>
<td>Arthralgia</td>
</tr>
<tr>
<td>Polyarthritis—pain, swelling, loss of function in several joints, often fleeting</td>
<td>Fever (&gt; 37.5°C)</td>
</tr>
<tr>
<td>Chorea—involuntary, jerky movements, especially of the arms, legs and face.</td>
<td>Raised ESR or CRP</td>
</tr>
<tr>
<td>Erythema marginatum—pink patches often on the trunk which rapidly enlarge to form irregular crescent shapes with slightly elevated margins and may coalesce with each other.</td>
<td>Prolonged PR interval</td>
</tr>
<tr>
<td>Subcutaneous nodules</td>
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</tr>
</tbody>
</table>

Table 1 Revised Jones Criteria for the diagnosis of acute rheumatic fever.

Disease, serum sickness and septic arthritis including disseminated gonococcal infection. Subacute disease with carditis but little or no joint involvement may be confused with cardiac conditions such as a viral myocarditis, tuberculous pericarditis or acute myocarditis with cardiac failure. Subclinical ARF is probably quite common. Vague illness in Aboriginal people, associated with aches and pains, pallor and tachycardia, should always be investigated for acute rheumatic fever.
### Acute Rheumatic Fever & Rheumatic Heart Disease

#### Figure 6 A guide to secondary prophylaxis regimen following acute rheumatic fever.

Source: Rheumatic Fever and Rheumatic Heart Disease Information Folder November 1999, Centre for Disease Control-Darwin.

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Age When ARF Episode Occurs</th>
<th>Prophylaxis Duration</th>
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<tr>
<td>No Rheumatic Heart Disease No Carditis</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>0 - 16 years</td>
<td>Give until 21 years old</td>
</tr>
<tr>
<td></td>
<td>17 &amp; older</td>
<td>Give for 5 years</td>
</tr>
<tr>
<td>Mild Rheumatic Heart Disease Mild - Moderate Carditis</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>0 - 25 years</td>
<td>Give until 35 years old</td>
</tr>
<tr>
<td></td>
<td>26 &amp; older</td>
<td>Give for 10 years</td>
</tr>
<tr>
<td>Moderate - Severe RHD Severe Carditis</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>All ages</td>
<td>Give for life</td>
</tr>
</tbody>
</table>
FURTHER INFORMATION

TELEPHONE ADVICE

Specialist physicians
Community paediatrician
NT Cardiac Services
Rheumatic Heart Disease Register
Healthy school aged kids program
Public Health Officer-on call
Public Health Officer
Public Health Officer
Royal Darwin Hospital
Centre for Disease Control-Darwin
Darwin Private Hospital
Centre for Disease Control- Darwin
DHCS Community Health Team
Centre for Disease Control-Darwin
Centre for Disease Control-Gove
Centre for Disease Control-Katherine
8922 8888
8922 8044
8920 6250
8922 8044
8922 8044
8922 8044
8987 0359
8973 9049

MANAGEMENT GUIDELINES

CARPA-Standard Treatment Manual
Rheumatic Fever and Rheumatic Heart Disease Information Folder November 1999.
Communicable Disease Surveillance in the NT-Guidelines for the Reporting of Notifiable Conditions
Central Australian Division of Primary Health Care
Centre for Disease Control-Darwin
Centre for Disease Control-Darwin
950 4800
8922 8044
8922 8089

EDUCATIONAL RESOURCES

Video-Caring for People with Rheumatic Fever
Rheumatic Fever and Rheumatic Heart Disease Information Folder November 1999,
Sara Noonan
Booklet-The Rheumatic Fever Story
Booklet-Rheumatic Fever: Questions and Answers
Flip Chart-Heart Story. Ed. by Jo Scheppingen
Menzies School of Health Research
Menzies School of Health Research
Menzies School of Health Research
National Heart Foundation
8922 8196
8922 8196
8922 8196
8981 1966

FURTHER READING

PNEUMOCOCCAL DISEASE IN THE TOP END

*Streptococcus pneumoniae* causes significant morbidity and mortality in the NT. It causes approximately 50% of all cases of community acquired bacterial pneumonia in adults living in the Top End and is also the primary cause of otitis media and meningitis. The burden of disease from this organism is higher in Central Australia where the yearly incidence of invasive pneumococcal diseases (IPD) amongst children under the age of two years is the highest documented in the world. The high incidence of IPD coupled with rising rates of penicillin-resistant and multi-resistant strains of *S. pneumoniae* make this one of the most important pathogens in the NT. IPD has been notifiable in the NT since 1995 and nationally since January 2001. Enhanced surveillance of IPD commenced in all States and Territories in July 2001 coinciding with the addition of a conjugate pneumococcal vaccine (Prevenar®) to the childhood immunisation schedule for those infants and children at increased risk.

AETIOLOGY AND PATHOGENESIS

*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 approximately 90 distinct serotypes have been identified. The predominant serotypes vary in their distribution between different populations, disease types, age groups and geographic areas. The pneumococci bind to human nasopharyngeal cells and spread to anatomically contiguous sites such as the Eustachian tubes or nasal sinuses. If they are not cleared when inhaled into the lungs, pneumonia may ensue. A respiratory tract focus can also result in the spread of the bacterium via the bloodstream to the meninges, bones, joint spaces and/or the peritoneal cavity.

CLINICAL PICTURE

Risk factors:
- Aboriginal children under 2 years have the highest rate of disease.
- Aboriginal people 15 years and over have high rates of disease (the age group 15-49 years has a rate 15 times that of same age non-Aboriginal people).
- Non-Aboriginal people 65 years and over are at increased risk.
- People with chronic conditions such as
  - Cardiac, pulmonary and renal disease, diabetes or cerebrospinal (CSF) leaks.
  - Anatomic or functional asplenia.
  - Immunological compromise such as HIV infection, lymphoma or organ transplant recipient.
  - Excessive alcohol intake.

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. About 75% will have pleuritic chest pain with cough productive of pink or rusty sputum.

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

DIFFERENTIAL DIAGNOSIS

Pneumococcal pneumonia is the most common cause of adult community acquired pneumonia. The differential diagnosis of a patient with clinical pneumonia includes melioidosis and other causes of bacterial pneumonia such as staphylococcal and klebsiella 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**

**Prevention.** The mainstay of control of pneumococcal disease is prevention through the immunisation of high risk individuals. There are two vaccines.

- The polysaccharide vaccine (Pneumovax®) protects against 23 serotypes and is effective in adults, but poorly immunogenic in children under 2 years. It is universally recommended for Aboriginal people 15 years of age and older and others 65 years and older and in those at increased risk due to diabetes, chronic renal, lung, liver or heart disease, alcoholism, asplenia or immuno-suppression.
- The conjugated vaccine (Prevenar®) is immunogenic in those under 2 years of age and protects against seven serotypes which account for 58% of Aboriginal and 72% of non-Aboriginal serotype isolates in the NT-in this age group.

**Treatment.** Specific treatment will depend on the clinical condition. While the mainstay of treatment of pneumococcal diseases has usually been penicillin, detailed empirical protocols have been developed as a strategy for appropriate management in the face of rising antibiotic resistance and to cover other causes of potentially fatal causes of pneumonia such as melioidosis. Clinicians should refer to the current edition of the CARPA manual or the ‘Therapeutic Guidelines: Antibiotic’ for the treatment of community acquired pneumonia (see case study of ‘Community-Acquired Pneumonia’).

Invasive Pneumococcal Disease is a notifiable condition to be reported by all LABORATORIES in the NT. All NT IPD isolates are sent for serotype testing. Adverse vaccine reactions are notifiable by all CLINICIANS in the NT and should be reported to the CDC in your district.

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**Figure 7** Age-specific incidence of invasive pneumococcal disease (cases per 100 000 person-years), in different regions, 1994-1998.

Pneumococcal Disease

FURTHER INFORMATION

TELEPHONE ADVICE

| DMO on call for remote areas | Royal Darwin Hospital 8922 8888 |
| DMO on call for remote areas | Katherine District Hospital 8973 9211 |
| DMO on call for remote areas | Gove District Hospital 8987 0211 |
| Infectious diseases or general physician | Royal Darwin Hospital 8922 8888 |
| Public Health Officer-on call | Centre for Disease Control-Darwin 8922 8044 |
| Public Health Officer | Centre for Disease Control-Gove 8987 0359 |
| Public Health Officer | Centre for Disease Control-Katherine 8973 9049 |

MANAGEMENT GUIDELINES

| CARPA-Standard Treatment Manual | Central Australian Division of Primary Health Care 8950 4800 |
| The Australian Immunisation Handbook Current Edition | Department of Health and Ageing (02) 6289 1555 |
| Communicable Disease Surveillance in the NT-Guidelines for the Reporting of Notifiable Conditions | Centre for Disease Control-Darwin 8922 8089 |
| Therapeutic Guidelines: Antibiotic, latest version | Therapeutic Guidelines LTD 1800 061 260 |

EDUCATIONAL RESOURCES

| Pamphlet-Prevent Pneumococcal Disease-Vaccinate Those at Risk | Centre for Disease Control-Darwin 8922 8089 |
| Pamphlet-Pneumococcal Pneumonia-Are You at Risk? | Centre for Disease Control-Darwin 8922 8089 |
| Poster-Pneumococcal Disease | Centre for Disease Control-Darwin 8922 8089 |

FURTHER READING

MELIOIDOSIS IN THE TOP END

Melioidosis is caused by infection with the bacillus *Burkholderia pseudomallei* (formerly known as *Pseudomonas pseudomallei*). Approximately 30-40 cases are diagnosed each year in the NT, almost all are acquired in the Top End following heavy wet season rains. Until recently it was the most common cause of fatal community-acquired bacteraemic pneumonia. However with the availability of newer therapies outcomes have recently improved. In a long-term study of adults admitted to RDH with pneumonia, *B. pseudomallei* accounted for 24% of culture positive cases of community-acquired pneumonia and 36% of deaths. *Streptococcus pneumoniae* was most commonly isolated pathogen overall, accounting for 39% of cases and 20% of fatalities.

AETIOLOGY AND PATHOGENESIS

*B. pseudomallei* is a small, free living bacillus found in soils and water across the Top End. Humans are exposed to the organism by percutaneous inoculation, inhalation or ingestion. Haemotogenous 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. Histologically, acute abscesses display necrosis with multinucleated histiocytes, polymorphonuclear leukocyte infiltration, and surrounding haemorrhage while subacute lesions exhibit caseation necrosis with mononuclear and plasma cell infiltration.

CLINICAL PICTURE

**Risk Factors.** Diabetes is the most important risk factor being present in approximately 40% of cases. Other risk factors include chronic renal disease, chronic lung disease, excessive alcohol and kava intake, malignancy, immunosuppression, skin contact with wet soil and age>45 years.

**Symptoms and signs** will depend on the site of infection. Pneumonia is the most common manifestation of the disease and has a case fatality rate of about 17%.

Melioidosis can also cause septicaemia, neurological infections, multiple organ abscesses, genito-urinary infection (especially prostate abscesses), osteomyelitis, septic arthritis, pustular skin lesions or ulcers. Two important presentations of melioidosis are (i) prostate melioidosis: males 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 reactivate 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.** 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, throat, rectal and ulcer swabs and collection of blood cultures. Culture specimens should specifically request examination for melioid. Sputum should be sent separately for microscopy and culture for other organisms including acid fast bacilli. Blood tests should include melioidosis serology. Chest Xray may show cavitation and consolidation. CT scans may identify other sites of infection, and all cases should have CT scans of the abdomen.

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. Progressive upper lobe disease mimics tuberculosis and testing to exclude this is important.

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.
These measures are particularly important for people with diabetes or other risk factors.

**Treatment.** Early diagnosis and appropriate antibiotic therapy decrease mortality. It is important to follow the protocol in the *Therapeutic Guidelines: Antibiotic or CARPA Manual* for community-acquired pneumonia, which cover the most important pathogens in the Top End (refer to the case study about 'Community Acquired Pneumonia'). 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 are managed and followed up in consultation with the RDH Infectious Diseases Unit. Subclinical infection or seropositivity alone does not warrant treatment in most instances.

**Melioidosis is a notifiable condition to be reported by all CLINICIANS and LABORATORIES in the Northern Territory. Cases should be reported to the Centre for Disease Control in your district by phone.**
Melioidosis

Figure 11 Melioidosis-acute pneumonia

Figure 12 Fatal melioidosis pneumonia-lung autopsy

Figure 13 Melioidosis-skin abscess
FURTHER INFORMATION

TELEPHONE ADVICE

| Infectious Diseases Registrar or Physician | Royal Darwin Hospital | 8922 8888 |
| Public Health Officer-on call | Centre for Disease Control-Darwin | 8922 8044 |
| Public Health Officer | Centre for Disease Control-Gove | 8987 0359 |
| Public Health Officer | Centre for Disease Control-Katherine | 8973 9049 |

MANAGEMENT GUIDELINES

| Communicable Disease Surveillance in the NT-Guidelines for the Reporting of Notifiable Conditions | Centre for Disease Control-Darwin | 8922 8089 |
| Antibiotic Protocol for Adult Community Acquired Pneumonia | Centre for Disease Control-Darwin | 8922 8089 |
| CARPA-Standard Treatment Manual | Central Australian Division of Primary Health Care | 8950 4800 |
| Therapeutic Guidelines: Antibiotic. Latest Version | Therapeutic Guidelines LTD | 1800 061 260 |

EDUCATIONAL RESOURCES

| Melioidosis Folder | Royal Darwin Hospital library | 8922 8961 |
| Fact sheet-Melioidosis | Centre for Disease Control-Darwin | 8922 8089 |

FURTHER READING

Too Many Risk Factors -
Case Study of Pneumonia

Sandra is a 51 year old local Aboriginal woman who is brought in by a female relative to the remote Top End community health centre clinic where you are working. She has been ill for five days with fever and a productive cough, and is now having difficulty breathing. You note from her records that she is a non insulin dependent diabetic who has high blood sugar readings when she presents to clinic. She has also smoked tobacco for 30-40 years and has had several previous admissions to Royal Darwin Hospital for 'bronchitis'. The Wet Season has come in properly in the last couple of weeks and you had been wondering before she came in whether the community airstrip is up to a medical evacuation.

On examination Sandra is an obese woman in respiratory distress. She is febrile 38.5°C, with a HR of 90, RR of 32, and BP of 115/80. Her fingerprick glucose reading is 23 mmol/L. She is breathless at rest. On auscultation of her chest she has an area of left sided crackles and what you think might be bronchial breathing. Her sputum is copious and yellow/green. It is not bloodstained. Sandra clearly has an acute community acquired pneumonia.

What causative 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 Hemophilus influenzae, and Cryptococcus neoformans particularly in Arnhemland.

Given the short history of illness in this case causes of atypical pneumonias such as Mycoplasma pneumoniae, Legionella pneumophila and Chlamydia pneumoniae, and infection due to Mycobacterium tuberculosis are less likely. Atypical pneumonia appears to be a less common occurrence in the Top End than in southern states.

The important causes of death in high risk groups which require specific antibiotics are Burkholderia pseudomallei 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 two fold:

- Clinical picture.
- Risk factors.

General clinical examination parameters to grade pneumonia include:

- Respiratory rate (>30 indicates severe pneumonia).
- BP (systolic < 90, diastolic < 60 indicates severe pneumonia and a high risk for developing septic shock).
- Others include evidence of shock, confusion, and O2 saturation <90% on room air. Parameters are listed in more detail in the Therapeutic Guidelines: Antibiotic and the excellent review by Johnson et al 2002 (see further reading).

In this setting, local risk factors are all important to help determine the level of risk of severe, life-threatening pneumonia caused by Acinetobacter baumannii or melioidosis. Sandra is Aboriginal, has diabetes, is a smoker, has a history of respiratory infections and it is the early wet season. Even if the pneumonia was not severe, on these factors alone broad antibiotic cover and transfer to hospital should be immediately considered and discussed with the District Medical Officer (DMO) 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, alcohol excess, chronic lung disease, chronic renal disease, steroid treatment, kava use and poor nutrition.

How would you grade this pneumonia? (mild, moderate or severe)

By clinical criteria this woman’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.
What are the organisms most likely to cause death in this woman?

*S. pneumoniae, B. pseudomallei* (melioidosis), *S. aureus*, and *A. baumannii*. All have a mortality risk of approximately 33%. Of these organisms, melioidosis is responsible for 36% of deaths from bacteraemic community acquired pneumonia. 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 O2 saturation of >90%).
- Establish intravenous (IV) access.
- Two sets of blood cultures (to be sent with the patient) immediately prior to the administration of antibiotics.
- Consider IV fluids if BP falls <90 systolic.
- Consider collecting a sputum for culture specifically for melioid if the purple Ashdown’s medium is available.
- Prompt IV antibiotics-as per protocols (CARPA/Therapeutic Guidelines: Antibiotic or community acquired pneumonia protocol). The current recommendation is IV gentamicin 4-6mg/kg and IV ceftriaxone 2g stat.

Note that penicillin is not recommended as a first line agent for moderate pneumonia in those with risk factors in tropical Australia as it does not adequately cover the organisms most likely to result in death, although elsewhere in Australia the use of third generation cephalosporins for community acquired pneumonia is being discouraged. In hospital once the causative organism is identified the antibiotic regimen can be refined. Atypical pneumonia cover is not usually recommended unless the clinical presentation is suggestive of this (eg insidious onset, > 1 week of illness, dry cough).

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 ceftriaxone via a peripherally inserted central catheter (PICC). Her hospital discharge letter notes that her blood sugar levels were poorly controlled throughout her stay.

What longer term issues need consideration?

Several issues are important to address with Sandra when she returns to the community:

- Regardless of causative organism, you should discuss pneumococcal and yearly influenza vaccines with Sandra.
- Smoking cessation, diabetes control and healthy nutrition are all priorities for Sandra’s wellbeing. 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.
- Melioid eradication therapy in consultation with RDH infectious diseases unit.

FURTHER READING

TUBERCULOSIS IN THE TOP END

Australia has one of the lowest rates of tuberculosis (TB) in the world (incidence rate has been between 5-6 per 100,000 for the past 10 years) and overseas-born people account for up to 80% of cases. In the NT annual rates vary from 17-30 per 100,000 with Aboriginal Australians accounting for just over 60% of cases, overseas-born people 30% and non-Aboriginal Australian-born people representing less than 10%. There have been several outbreaks as well as ongoing epidemics in remote and urban Aboriginal communities. Regardless of ethnicity, the three most common risk factors for disease are being a contact of a case of active TB, over-using alcohol and being malnourished. Our near neighbours, Indonesia, East Timor and Papua New Guinea have very high rates of TB as do other countries in the region eg Papua New Guinea, Vietnam and the Philippines and people from these countries have an increased risk of infection and disease.

The increase in TB cases in 1999 and 2000 is partially explained by an increase in imported cases from East Timor, unauthorised entrants from a high TB prevalence country and a large TB outbreak in a Top End community.

Though HIV is the most potent risk factor for TB infection progressing to disease there is very little TB-HIV co-infection at this time in the NT.

Though multi-drug resistant TB (MDR-TB) is a worrying concern in much of the rest of the world, in Australia and the NT it has not emerged as a problem. Only one case of MDR-TB has been notified in the NT. Preventing the emergence of MDR-TB relies on having a well co-ordinated TB control program.

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 for information), Aboriginal people, some migrant groups, alcoholics, diabetics and those with chronic renal disease and some malignancies. Patients who have been infected with M. tuberculosis and become immunosuppressed due to HIV infection, cancer chemotherapy, long term steroids, or other immunosuppressive therapy are at increased risk of developing active TB.

Symptoms and Signs. TB illness is characterised by:

- Prolonged cough for more than 3 weeks (normally productive of sputum).
- Unexplained weight loss.
- Fevers and sweating.
- Haemoptysis.

While pulmonary TB is the most common form of the disease, accounting for 70% of NT cases, TB can affect any part of the body. Other sites include (in decreasing frequency):

- Nodal: chronic, slowly growing, asymmetrical lymphadenopathy, most common in cervical area and axillary region. Matted, rubbery, nontender nodes (chronic discharging sinuses in late presentation);
- Pleural: chest pain, shortness of breath. Typical signs of pleural effusion;
- Pericardial: symptoms and signs of pericardial effusion (early) and constrictive pericarditis (late);
- Spinal: chronic back pain, symptoms of cord compression, psoas abscess causing lumbar or groin swelling.

AETIOLOGY AND PATHOGENESIS

TB is an infectious bacterial disease spread by coughing. The causative organism, Mycobacterium tuberculosis, 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 in a 12 month period. The number infected however is dependent on other factors such as overcrowding and susceptibility of contacts eg diabetes, HIV infection. Once infected, a person will have a positive tuberculin skin (Mantoux) test and may have an abnormal chest X-ray, but will not be symptomatic and is unlikely to be infectious. A person infected with M. tuberculosis has a 35% lifetime risk of developing active TB which is symptomatic and may, if present in lungs, transmit TB to others. The first 5 years after infection are the highest risk for progression to disease.
Investigations: Whenever possible, microbiological confirmation of the diagnosis of TB should be obtained. For pulmonary TB, sputum smear examination for acid fast bacillus (AFB) and culture is the gold standard. Sputum specimens should be collected in the early morning and light protected, eg use a stool specimen container or clear specimen jar and place in a paper bag. Sputum should also be considered for cytology, melioid (Burkholderia pseudomallei) culture and normal microbiological examination. If a patient cannot produce sputum, alternative methods may be considered (gastric aspiration, induced sputum or bronchoscopy). For TB in other sites, a fine needle or formal biopsy for culture is recommended (don’t put it into formalin!). Radiological (chest X-ray, computed tomography scan, magnetic resonance imaging), haematological (haemoglobin, white cell count and differential, erythrocyte sedimentation rate and C-Reactive protein) and histological tests may support the diagnosis but are not specific.

The Mantoux test is helpful in determining previous TB exposure and latent TB infection (LTBI). Use of correct technique in performing and reading Mantoux tests is essential. For detailed information about indications for and interpretation of Mantoux tests see the Guidelines for the treatment of tuberculosis in the Northern Territory.

Differential Diagnosis

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

- Other common infections in the same risk groups: melioidosis, non tuberculosis (NTM) mycobacteria, staphylococcal pneumonia;
- Carcinoma (primary bronchogenic or secondary);
- Other less common infections: cryptococcus, nocardiasis, actinomycosis, aspergillosis; pulmonary strongyloidiasis.
- Other: bronchiectasis, sarcoidosis, pneumoconiosis, lymphoma.

Principles of Management

Directly observed therapy (DOT) with anti-TB medications and adequate nutrition are the hallmarks of the clinical management of TB. Patients require close monitoring for side-effects of treatment and for signs of clinical improvement. The most effective public health interventions are the prompt diagnosis and adequate treatment of TB cases and the treatment of LTBI (formerly called preventive treatment) 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 4 drugs (usually rifampicin, isoniazid, pyrazinamide, ethambutol) is given for 2 months followed by 4 months treatment with 2 drugs (usually rifampicin and isoniazid) to complete a 6 month course of treatment. Treatment for LTBI (disease has been ruled out-patient is well and not infectious) is mainly 6 to 9 months of isoniazid.

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

The TB Control Units in Darwin, Katherine and Nhulunbuy are there to provide expert advice and clinical services. They provide management and follow up for all TB patients. They have educational materials to share with doctors and information sheets and videos for patients.

TB is a notifiable condition to be reported by all CLINICIANS and LABORATORIES in the Northern Territory. Cases should be reported to the Centre for Disease Control in your district by phone.
### FURTHER INFORMATION

#### TELEPHONE ADVICE

<table>
<thead>
<tr>
<th>Service</th>
<th>Contact</th>
<th>Phone Number</th>
</tr>
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<tbody>
<tr>
<td>TB/Leprosy Medical Officer or clinic staff</td>
<td>Centre for Disease Control-Darwin</td>
<td>8922 8804</td>
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<tr>
<td>TB/Leprosy Medical Officer or clinic staff</td>
<td>Centre for Disease Control-Gove</td>
<td>8987 0359</td>
</tr>
<tr>
<td>TB/Leprosy Medical Officer or clinic staff</td>
<td>Centre for Disease Control-Katherine</td>
<td>8973 9049</td>
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#### MANAGEMENT GUIDELINES

<table>
<thead>
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<th>Guideline</th>
<th>Contact</th>
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</thead>
<tbody>
<tr>
<td>Guidelines for the Treatment of Tuberculosis in the Northern Territory</td>
<td>Centre for Disease Control-Darwin</td>
<td>8922 8804</td>
</tr>
<tr>
<td>Communicable Disease Surveillance in the NT</td>
<td>Centre for Disease Control-Darwin</td>
<td>8922 8089</td>
</tr>
<tr>
<td>Guidelines for the Reporting of Notifiable Conditions</td>
<td>Central Australian Division of Primary Health Care</td>
<td></td>
</tr>
</tbody>
</table>

#### EDUCATIONAL RESOURCES

<table>
<thead>
<tr>
<th>Resource</th>
<th>Contact</th>
<th>Website</th>
</tr>
</thead>
<tbody>
<tr>
<td>TB CD-ROM</td>
<td>Centre for Disease Control-Darwin</td>
<td>8922 8804</td>
</tr>
</tbody>
</table>

#### FURTHER READING

LEPROSY IN THE TOP END

Although leprosy has become a rare disease in the NT, continued vigilance is required as the incubation period can be as long as 30 years. At the time of writing, the last newly identified case occurred in Darwin in 2000. There are many people in the NT who are bacteriologically cured but continue to require lifelong support because of related disabilities such as reduced visual acuity, amputations, and injury-prone anaesthetic hands and feet. Their close contacts require ongoing surveillance.

AETIOLOGY AND PATHOGENESIS

The leprosy bacillus, *Mycobacterium leprae*, is thought to be transmitted by inhalation of aerosols generated from infection in the nasal mucosa of lepromatous patients. *M. leprae* primarily attacks 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 ability of the body to mount an immune response to the invading bacilli. It is important to accurately classify cases, because their position on this continuum determines their infectivity, prognosis, likely complications and treatment regimens.

CLINICAL PICTURE

Risk factors. These include being Aboriginal or from a high prevalence country, having family or household contact with multibacillary leprosy and overcrowding.

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 most commonly affected nerves are ulnar, median, radial, common peroneal (lateral popliteal), posterior tibial nerves and the sural nerve. The TB/Leprosy unit has devised forms to be used as a guide when examining 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.

DIFFERENTIAL DIAGNOSIS

Differential diagnosis includes: tinea versicolor, systemic lupus erythematosus (SLE), lupus vulgaris, 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 coexist.

PRINCIPLES OF MANAGEMENT

The main objective of treatment is the prevention of disability. Management is complex and all cases must be notified to CDC 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 becomes irreversible after 6 months. The key monitoring activity is a monthly standardised motor and sensory test to detect silent decrements in neurologic function. High dose prednisolone is then 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, chest X-ray and clinical review as the ‘at risk’ groups overlap and treatment of leprosy may jeopardise treatment of TB.

**FURTHER INFORMATION**

**TELEPHONE ADVICE**

<table>
<thead>
<tr>
<th>Role</th>
<th>Contact Information</th>
</tr>
</thead>
<tbody>
<tr>
<td>TB/Leprosy Medical Officer or clinic staff</td>
<td>Centre for Disease Control-Darwin 8922 8804</td>
</tr>
<tr>
<td>TB/Leprosy Medical Officer or clinic staff</td>
<td>Centre for Disease Control-Gove 8987 0359</td>
</tr>
<tr>
<td>Infectious Diseases Physicians staff</td>
<td>Centre for Disease Control-Katherine 8973 9049</td>
</tr>
<tr>
<td></td>
<td>Royal Darwin Hospital 8922 8888</td>
</tr>
</tbody>
</table>

**MANAGEMENT GUIDELINES**

- Guidelines for Leprosy Control in the NT 2002
  - Centre for Disease Control-Darwin 8922 8804

- Communicable Disease Surveillance in the NT-Guidelines for the Reporting of Notifiable Conditions
  - Centre for Disease Control-Darwin 8922 8089

  - Centre for Disease Control-Darwin 8922 8804
  - Or Royal Darwin Hospital Library 8922 8961

- Leprosy CD-ROM-Wellcome Foundation
  - Centre for Disease Control-Darwin 8922 8804

- Video-Skin Smear
  - Centre for Disease Control-Darwin 8922 8804

**EDUCATIONAL RESOURCES**

  - Centre for Disease Control-Darwin 8922 8804

- Leprosy for Field staff-Alison Summers
  - Centre for Disease Control-Darwin 8922 8804

**FURTHER READING**

Viruses, Spirochetes, Chlamydial and Vector Borne Diseases
Mosquito Borne Diseases

MOSQUITO BORNE DISEASES IN THE TOP END

Mosquito borne diseases have a prominent role in clinical and public health practice in the Top End. Mosquito vectors are present in most of the Top End for the Murray Valley Encephalitis (MVE) and Kunjin viruses, malaria, Ross River virus (RRV), Barmah Forest virus (BFV) and Japanese Encephalitis (JE). Currently there are no NT mosquito vectors for dengue fever but importations have become more frequent with increased ship movements between Darwin and Timor since September 1999 and dengue mosquitos and disease are present in East Timor, Indonesia, Papua New Guinea (PNG) and North Queensland.

Since the 1960s all cases of malaria and dengue fever have been imported in the NT and there have been no cases of JE. The last case of malaria acquired in the NT was in Roper River in 1962 but the Top End remains receptive for re-introduction of the parasite. It is also receptive for entry of exotic arboviruses such as JE as the vector mosquitos are here and disease is present in surrounding East Timor, Indonesia, PNG and Far North Queensland. Re-introduction of Aedes aegypti, or the introduction of Aedes albopictus, both mosquito vectors for dengue, is a constant concern. Remaining dengue vector-free and disease-free is a priority for the NT medical entomology branch.

Ross River Fever and Barmah Forest are relatively common and occur in outbreaks each wet season. Outbreaks of encephalitis due to MVE and Kunjin viruses occur occasionally, the last being in 1993. Most cases have occurred in the Top End and northern parts of Western Australia, however in the last 2 years, cases have also occurred in Central Australia.

AETIOLOGY AND PATHOGENESIS

Malaria is caused by one of four Plasmodium parasites. Plasmodium falciparum can cause the more serious forms of the disease (for example, cerebral malaria) whilst P. vivax can cause relapse many years after a person has been infected with the organism. P. malariae and P. ovale are much less common. The parasites cause fever and chills when they rupture out of red cells, and organ-specific pathology by microvascular occlusion.

Arboviruses fall into two main groups:

Alphaviruses (RRV and BFV) which can also infect other species such as macropods. Both the arthritis and the rash characteristic of alphaviruses are caused by immunological responses to infection.

Flaviviruses (dengue, MVE, Kunjin and JE) which apart from dengue can be carried and amplified in pig populations.

Dengue is solely a disease of humans. There are four viral serotypes. Infection with a particular serotype incurs lifelong immunity to that serotype. Macrophage and monocyte infection are the most important aspects of the pathogenesis of dengue fever and the more severe dengue haemorrhagic fever/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. These cause the characteristic increased vascular permeability and bleeding.

MVE and Kunjin also infect chickens and certain water-birds and the NT has sentinel chicken surveillance to monitor for their viral presence and distribution. The encephalitis caused by these viruses in humans follow 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 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.

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Ross River Fever and Barmah Forest are relatively common and occur in outbreaks each wet season.
### Table 2 Aetiology, vector and immunity in mosquito-borne diseases of importance in the Top End.

<table>
<thead>
<tr>
<th>Organism</th>
<th>Main vector or potential vector</th>
<th>Vector present in NT</th>
<th>Immunity after infection</th>
<th>Illness</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasmodium sp</td>
<td>Anopheles farauti</td>
<td>Yes</td>
<td>No</td>
<td>Malaria</td>
</tr>
<tr>
<td>Dengue virus (serotypes I-IV)</td>
<td>Aedes aegypti</td>
<td>Yes</td>
<td>No</td>
<td>Dengue fever, dengue haemorrhagic fever</td>
</tr>
<tr>
<td></td>
<td>Aedes albopictus</td>
<td>No</td>
<td>Yes (serotype specific), but risk of DHF if infected with another serotype</td>
<td></td>
</tr>
<tr>
<td>Kunjin virus</td>
<td>Culex annulirostris</td>
<td>Yes</td>
<td>Probable</td>
<td>Mild to moderate encephalitis</td>
</tr>
<tr>
<td>Murray Valley Encephalitis virus</td>
<td>Culex annulirostris</td>
<td>Yes</td>
<td>Probable</td>
<td>Moderate to severe encephalitis, death</td>
</tr>
<tr>
<td>Japanese encephalitis</td>
<td>Culex annulirostris Culex jelidus</td>
<td>Yes</td>
<td>Probable</td>
<td>Moderate to severe encephalitis, death</td>
</tr>
<tr>
<td>Ross River virus</td>
<td>Aedes vigilax Culex annulirostris</td>
<td>Yes</td>
<td>Probable</td>
<td>Ross River Fever (RRF), rash and polyarthritis, fatigue</td>
</tr>
<tr>
<td>Barmah Forest virus</td>
<td>Aedes vigilax Culex annulirostris</td>
<td>Yes</td>
<td>Probable</td>
<td>Barmah Forest fever, rash often more prominent and itchy than RRF and arthritis less prominent</td>
</tr>
</tbody>
</table>

**Barmah Forest and Ross River Fever** have similar clinical pictures, although rash is more common in the former and arthralgia 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 “back break fever” commonly presents with backpain, 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 and blanches with pressure. In persons who have previously been infected with another serotype of dengue, the more severe dengue haemorrhagic fever/dengue shock syndrome may occur. In contrast to simple dengue, myalgia and bone pains are unusual. Dengue haemorrhagic fever has an abrupt onset characterised by fever, lymphadenopathy, hepatomegaly, scattered petechiae and rapid deterioration.

**Murray Valley Encephalitis virus, Kunjin virus and Japanese Encephalitis virus.** Symptomatic infections are manifested by fever, headache and sometimes nausea and vomiting, rapidly followed in the majority of cases by the features of encephalitis. Kunjin infections tend to be less severe. It has been estimated from serological studies that only 1 in 1000 people infected with MVE, become symptomatic for the disease. 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%). JE has not been diagnosed in the NT but has a spectrum of disease similar to MVE.
Serological Investigation of arboviral infections

The gold standard (virus isolation) for diagnosis in arbovirus infection is not normally used in practice conditions. The cross-reactivity of flavivirus serology (including dengue, yellow fever, Murray Valley Encephalitis, Kokobera, Japanese Encephalitis and Kunjin) often makes a definitive diagnosis difficult. Serological diagnosis of alphaviruses (BFV and RRV) does not have the same problem of cross reactivity, though false positive IgM results can occur. For both flaviviruses and alphaviruses, confirmation of the diagnosis needs a careful history and clinical examination and the collection of convalescent serum to check for a rising titre of antibodies.

PRINCIPLES OF MANAGEMENT

All the diseases described in this section, apart from those caused by RRV and BF, require expert advice from an infectious disease physician. Malaria is the only disease which has specific treatment. Antimalarial drug resistance is common worldwide and it is important that current protocols are consulted prior to commencing treatment. Hospitalisation is required for all cases of *P. falciparum* to ensure effective treatment and to minimise the risk of transmission of the parasite to local mosquitoes. *P. vivax* malaria can be managed as an outpatient following notification of CDC and liaison with RDH Infectious Diseases Unit if specific criteria are able to be met (refer to CDC Malaria protocol). The other diseases have no specific treatment although analgesia, antipyretics, fluids and rest offer some relief of symptoms. One should be aware of fluid overload in dengue and aspirin and non steroidal anti-inflammatories should also be avoided in treatment of that illness. Complicated dengue, MVE and JE require admission to hospital for close monitoring and supportive therapy as required. Patient advice is important for RRV and BF. Queensland Health in addition to CDC Darwin have excellent fact sheets which may be downloaded from their website (see educational resource list).

Public health surveillance and management are coordinated by DHCS through:

- Rapid notification of suspected cases of dengue fever, malaria, MVE, Kunjin and JE infection to CDC. This leads to prompt investigation including identification of the place the infection was acquired, mosquito control measures, and if necessary, trapping and testing of mosquitos close to the location of the case.
- Serological monitoring of sentinel chickens flocks located throughout the NT. Seroconversions in these animals alert public health authorities to potential outbreaks of arboviruses.
- Public health warnings are given when mosquito numbers and/or clinical cases increase above the expected levels.

Remember: In most cases of fever in the Top End, always take a travel history..."Where have you been and when?"

All suspected cases of MVE, Kunjin virus, JE, malaria, and dengue fever should be urgently notified to CDC by CLINICIANS and LABORATORIES. Cases of Barmah Forrest and Ross River Fever, are notifiable by LABORATORIES only.

<table>
<thead>
<tr>
<th>Illness</th>
<th>Fever</th>
<th>Rash</th>
<th>Other symptoms</th>
<th>Diagnostic test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Malaria</td>
<td>+</td>
<td>-</td>
<td>Headache, chills, rigors</td>
<td>Blood film</td>
</tr>
<tr>
<td>Dengue</td>
<td>+</td>
<td>+</td>
<td>Retro orbital headache, myalgias</td>
<td>Serology, FBC</td>
</tr>
<tr>
<td>RRV, BFV</td>
<td>+/-</td>
<td>+/-</td>
<td>Arthralgia</td>
<td>(↑ WCC, platelets)</td>
</tr>
<tr>
<td>MVE, Kunjin, JE</td>
<td>+</td>
<td>-</td>
<td>Altered mental state; coma</td>
<td>Serology</td>
</tr>
</tbody>
</table>

Table 3 Summary of clinical and diagnosis of vector borne diseases
**FURTHER INFORMATION**

**TELEPHONE ADVICE**

| Infectious diseases or general physicians | Royal Darwin Hospital | 8922 8888 |
| Public Health Officer-on call | Centre for Disease Control-Darwin | 8922 8044 |
| Public Health Officer | Centre for Disease Control-Gove | 8987 0359 |
| Public Health Officer | Centre for Disease Control-Katherine | 8973 9049 |
| Medical Entomology | Department of Health and Community Services | 8922 8901 |

**MANAGEMENT GUIDELINES**

- **Communicable Disease Surveillance**
  - Centre for Disease Control-Darwin
- **Therapeutic Guidelines: Antibiotic. Latest Version**
  - Therapeutic Guidelines LTD
- **Malaria protocol-1999 with updates**
  - Centre for Disease Control-Darwin

**EDUCATIONAL RESOURCES**

- **Fact sheets-**
  - Ross River Fever
  - Mosquito
  - Murray Valley Encephalitis
  - Japanese Encephalitis
  - Dengue
  - Pamphlet- Malaria and You
  - Brochures- Barmah Forest Virus, Dengue
  - Ross River Fever, Queensland Health, QLD Government

**FURTHER READING**

**SCRUB TYPHUS IN THE TOP END**

Ten cases of scrub typhus have been described in the Top End since the early 1990’s including one fatality and several severe illnesses with multi-organ failure. All occurred in workers or visitors to rainforest areas within Litchfield Park south west of Darwin. Scrub typhus elsewhere has a wide distribution from eastern Asia to the western Pacific, and is characteristically patchy in distribution (mirroring the insect vector habitats). In Queensland *Rickettsia australis* causes a similar illness.

**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. In the Top End it is transmitted from native rats to humans by the trombiculid mite, *Leptotrombidium deliense*. While the same species of rat are found in many rainforest areas of the Top End, the disease has so far only been associated with Litchfield Park. The distribution of the mite vector has not been studied in detail. The period from the mite bite to development of symptoms is 8-12 days (range 6-18 days).

**CLINICAL PICTURE**

**Risk factors** include time spent in Litchfield Park and surrounds, particularly walking and sitting in areas of rainforest in or near the park.

**Symptoms and signs** include an inoculation eschar (black scab) of approximately 1 cm 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. 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. The skin rash starts on the trunk on around day 5 and may spread to the extremities. It is usually maculopapular but may be fleeting or absent.

**Investigations.** Serology (indirect microimmunofluorescence test for *Orientia tsutsugamushi*) should be performed on presentation and convalescent serum two weeks after presentation will be positive.

**DIFFERENTIAL DIAGNOSIS**

Differential diagnosis is broad unless the characteristic eschar and regional lymphadenopathy are present in someone with a history of visiting Litchfield Park or surrounds. If only the more nonspecific symptoms of fever, myalgia and headache are present differentials include influenza, toxoplasmosis, infectious mononucleosis, leptospirosis, Ross River fever, Murray Valley Encephalitis and bacterial sepsis. Other infections that may need to be considered depending on the patient’s history, especially of travel, include dengue and typhoid. Travel to tropical Queensland makes *Rickettsia australis* infection possible.

**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 it is important to consider and test for scrub typhus in the setting of locally acquired flu-like illness particularly with a history of rainforest...
exposure in or near Litchfield Park.

Prevention includes wearing stout footwear and long trousers when in high risk areas. Use of N, N-diethyl-meta-toluamide (DEET) containing repellents on skin and socks/trousers. The use of a groundsheet for sitting is also recommended. Doxycycline has been evaluated for prophylaxis for those at risk but is not recommended in the NT.

Scrub Typhus is a notifiable condition to be reported by all LABORATORIES in the Northern Territory

FURTHER INFORMATION

TELEPHONE ADVICE

Infectious diseases registrar or physician  Royal Darwin Hospital  8922 8888
Public Health Officer-on call  Centre for Disease Control-Darwin  8922 8044
Public Health Officer  Centre for Disease Control-Gove  8987 0359
Public Health Officer  Centre for Disease Control-Katherine  8973 9049

MANAGEMENT GUIDELINES

Therapeutic Guidelines: Antibiotic. Latest Version  Therapeutic Guidelines LTD  1800 061 260

Communicable Disease Surveillance in the NT-Guidelines for the Reporting of Notifiable Conditions  Centre for Disease Control-Darwin  8922 8089

EDUCATIONAL RESOURCES

Fact Sheet-Scrub Typhus  Centre for Disease Control- Darwin  8922 8044

FURTHER READING

• Currie B, O’Connor, Rhodes F, Whelan P, Pritchard D, Bell P, Dwyer B. Scrub typhus focus in the Northern Territory. CDI. 1991; p15-156
Leptospirosis

LEPTOSPIROSIS IN THE TOP END

Between 1992 and 2000, 17 cases of leptospirosis were notified in the Top End. (See figure 17) Activities associated with the cases included: working in abattoirs; hunting of animals such as duck, goose and turtle; living on rural blocks; and water-based recreational activities.

AETIOLOGY AND PATHOGENESIS

Leptospirosis is an acute generalised infectious disease caused by spirochaetes of the genus Leptospira. It has a worldwide distribution and is primarily a disease of animals, especially rats. The serotype causing disease in the NT is Leptospira australis. Humans are infected through contact of abraded skin by urine of infected animals and are not usually infectious to others. After penetration of the skin or mucous membrane, leptospires migrate throughout the body, including to the cerebral spinal fluid (CSF), muscle, liver and kidneys, causing inflammation, vasculitis and immune mediated damage. Incubation period is usually 10 days (range 4-19).

CLINICAL PICTURE

Risk factors include occupational/recreational exposure to infected animals, such as abattoir workers, farmers, veterinarians, and hunters. In the Top End it may also include tourists camping and walking through rough country (particularly with sharp grass) after heavy rains.

Symptoms. Infection is often asymptomatic. Approximately 90% of cases are mild, with 10% exhibiting a more severe icteric illness (Weil’s disease). The illness is often biphasic. Incubation is 7-12 days; first stage is 4-7 days and the final phase is 0-30 days. Early symptoms of mild illness are flu-like with marked myalgias. Headache, stomach-ache, vomiting and fever may also be present. Defervescence may then occur for a day or two followed by meningitis, uveitis, rash and return of fever. In the more severe form of the illness, hepatitis, myocarditis, haemorrhage and renal failure may develop. There is a 10% mortality rate with severe disease.

Signs. Conjunctival suffusion is common and may be a particular clue to the illness. Adenopathy and splenomegaly may be present. Jaundice, hepatomegaly, haemorrhage and signs of renal failure may be present in severe cases.

Investigations include an elevated C-Reactive protein/erythrocyte sedimentation rate, and neutropenia. Liver function tests may show a moderate elevation in transaminases but creatine kinase is usually high. CSF may show an aseptic meningitis picture. In severe disease, chest X-ray changes may be evident.

Specific testing is usually microagglutination serology which becomes positive 6-12 days after the onset of illness, so serology repeated at two and if necessary four weeks may be useful. Leptospires can remain in anticoagulated blood for up to 10 days. Blood can therefore be sent to a reference laboratory for culture if taken during leptospiraemia, i.e. within 7 days of start of illness.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis is large as 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, viral hepatitis or scrub typhus. In travellers also consider malaria, typhoid, dengue, Hantavirus infections, and other rickettsial diseases. For severe disease, the differential includes other causes of acute hepatitis (eg viruses specifically Hepatitis A or E or toxins such as alcohol), and systemic conditions causing vasculitis. The latter might include immune mediated conditions such as SLE, or infections such as syphilis.

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.** Wearing closed footwear and covering the skin likely to come into contact with infected urine is recommended for those at risk, including health staff who may handle urine of infected patients. Vaccination has been trialed and doxycycline has been shown to prevent infection in army recruits in a highly endemic area, but these measures are not generally appropriate for the NT. As a large pool of wild animals may be infected and animal vaccines may only be partially protective, control via reduction of animal disease is unlikely.

**Treatment.** For mild disease, tetracyclines such as doxycycline are currently recommended. See the ‘Therapeutic Guidelines: Antibiotic’. Fever and myalgias can be treated with paracetamol. For more severe disease referral to hospital is required for intravenous antibiotics and supportive therapy for renal failure, haemorrhage or hypotension. Renal failure is usually reversible even if therapy commences relatively late in the illness. The disease is notifiable to the Centre for Disease Control.
TRACHOMA IN THE TOP END

Trachoma is the world’s leading cause of blindness with 500 million people affected and approximately 7 million blind. In Australia, trachoma remains an important cause of blindness in the Aboriginal population. In a review of surveys of the prevalence of trachoma in the Northern Territory from 1940 to 1986, it was found that there had been a decrease in the prevalence of trachoma in the coastal communities of the Top End, however, this was not the case for the more inland arid regions with a prevalence rate in the Katherine Region in children up to 10 years of age of 38%. Results from school screening in the Top End in the 1990’s showed continued high rates in some communities.

Mass treatment programs were introduced by the National Trachoma and Eye Health Programme (oral sulphamethoxazole-trimethoprim), and by the NT Department of Health & Community Services (oily tetracycline eye drops) in the mid 1980’s. These programs were time consuming, unpleasant for children, and had poor compliance. In the 1990’s trachoma control programs fragmented and ceased in many regions. With the introduction of azithromycin for treatment of trachoma and the development of the NT Healthy School Aged Kids policy and program there is now a more systematic approach to evaluation and treatment of trachoma.

AETIOLOGY AND PATHOGENESIS

Trachoma is a chronic kerato-conjunctivitis caused by infection with the bacteria 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, which if it persists, can lead to chronic complications later in life requiring surgery or causing visual impairment. Children are the chief reservoir of infection and the main transmission is within families. The severity of scarring and risk of subsequent blindness is a function of the intensity and duration of the previous inflammatory phase. 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 have been proven to 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 entropion and trichiasis are present, pain and irritation from corneal abrasions may occur. Blindness can result and is intractable.

Signs. The World Health Organization (WHO) has developed a simple five sign grading system for diagnosis and assessment of trachoma.

- **Trachomatous inflammation-follicular (TF)**: Five or more follicles in the upper tarsal conjunctiva. Follicles must be more than 0.5mm in diameter.
- **Trachomatous inflammation-intense (TI)**: Pronounced inflammatory thickening of the tarsal conjunctiva which obscures half of the normal deep tarsal vessels.
- **Trachomatous conjunctival-scarring (TS)**: Visible scars in the upper tarsal conjunctiva.
- **Trachomatous trichiasis (TT)**: At least one eyelash rubs on the eyeball, or evidence of recent removal of in-turned lashes.
- **Corneal opacity (CO)**: Visible corneal opacity over the pupil which is so dense that at least part of the pupil margin is blurred through the opacity.

Investigations. Trachoma is predominantly a clinical diagnosis and microbiological testing for diagnosis is not usually necessary. Community and individual screening require assessment by people trained in making the clinical diagnosis and grading of trachoma. However, there are newer laboratory methods for diagnosis of chlamydia. There is the classical direct microscopy, immunofluorescence, ELISA tests and, most 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 and binocular loupes (x 2.5) and adequate lighting are necessary. Signs must be clearly seen to be considered present. Examine for trichiasis, either in-turned eyelashes or previously removed lashes. To check for this the upper lid needs to be pushed upwards slightly, to expose the lid margins. Examine the cornea for opacities. Examine the inside of the everted upper eyelid (tarsal conjunctiva) for follicles, inflammation and scarring. Refer to the WHO trachoma grading card for a coloured pictorial guide to trachoma grading (available from CDC).
DIFFERENTIAL DIAGNOSIS

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

Trachomatous inflammation-intense: chronic blepharitis, allergic conjunctivitis, bacterial infection, 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 acknowledged as the most important factors in preventing trachoma. Community based approaches have had some success in addressing these issues.

Treatment. The approach to treatment of individuals depends upon the prevalence of trachoma among school-aged children in the community.

• If the 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 children, and if possible women who are caregivers of children. Treatment of the groups must be completed within 14 days. Re-treatment of all children, and if possible women who are caregivers of children should occur at 6 months.
  • If the prevalence is 5%-20% (endemic) treat the case and all household contacts.
  • If the prevalence is <5% (non-endemic) treat the individual case only.
  • If the prevalence is unknown discuss with the community paediatrician CDC. They may have information about your community as this is collected as part of the Healthy School-aged Kids Program.

Detailed guidelines are available from CDC. The medication of choice is azithromycin orally as a single dose. Refer to the Therapeutic Guidelines: Antibiotic or the CARPA Manual for doses.

The chronic sequelae of trachoma (trachomatous conjunctival-scarring, trichiasis, corneal opacity and blindness) occur in adults. This should be looked for whenever examining an eye in an adult. Ophthalmological follow up is required.

FURTHER INFORMATION

TELEPHONE ADVICE

Community Paediatrician
Infectious diseases registrar or physician.
Opthalmology registrar or specialist
Public Health Officer on call
Public Health Officer
For chronic sequelae contact the ophthalmologists

Centre for Disease Control-Darwin
Royal Darwin Hospital
Royal Darwin Hospital
Centre for Disease Control-Darwin
Centre for Disease Control-Gove
Royal Darwin Hospital
8922 8044
8922 8888
8922 8888
8922 8044
8987 0359
8973 9049
8922 8888

MANAGEMENT GUIDELINES

Guidelines for the Treatment of Trachoma in the NT,1998
CARPA-Standard Treatment Manual
Specialist Eye Health Guidelines for use in Aboriginal and Torres Strait Islander Populations

Centre for Disease Control-Darwin
Central Australian Division of Primary Health Care
8922 8044
8950 4800
8946 3481
Or OATSIH Office NT

EDUCATIONAL RESOURCES

WHO Trachoma Grading Cards
WHO Slides of Trachoma
Video Called “Jabbys friend. A Story About Trachoma”

Centre for Disease Control-Darwin
Centre for Disease Control-Darwin
Available for purchase from Desert Pictures in WA
8922 8044
8922 8044
(08) 9193 7455

FURTHER READING

• Laming AC, Currie BJ, DiFrancesco M, Taylor HR, Mathews JD. A targeted, single-dose azithromycin strategy for trachoma.
• Lietman T, Porco T, Dawson C, Blower S. Global elimination of trachoma: how frequently should we administer mass chemotherapy?
Australian Bat Lyssavirus

**LYSSAVIRUS IN THE TOP END**

Australian Bat Lyssavirus (ABL) is a rabies-like virus carried by several species of bats and flying foxes. Only two human cases have ever been described, both from Queensland and each resulting in death. Serological testing of bats however indicates that lyssavirus is widely distributed throughout Australia including 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. Approximately 10 people each year have received post exposure prophylaxis for lyssavirus in the NT since 1996.

**AETIOLOGY AND PATHOGENESIS**

ABL infection is caused by a lyssavirus, genotype 7, which is very similar to the lyssavirus that causes rabies (genotype 1). 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 the bat is 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 (CNS). The incubation period is variable, ranging from 7 days to over 1 year (mean, 1 to 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 CNS. 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. A bat exhibiting unusually aggressive behaviour is more likely to be infected.

**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 dysfunction of brainstem centers, and death. The prodromal period (1 to 4 days) is generally marked by fever, headache, malaise, myalgias, anorexia, nausea and vomiting, sore throat, and dry cough. The one specific symptom is paresthesia 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 apnea after involvement of the respiratory centre.

**Signs.** Early on the signs may be nonspecific, 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, which is confirmed by post mortem PCR testing of brain tissue.

**DIFFERENTIAL DIAGNOSIS**

Unusual neurologic illnesses that may require differentiation from Lyssavirus infection include Guillain Barre Syndrome, Murray Valley encephalitis. Australia is rabies free: however in persons with a suspicious clinical illness and with a history of animal bite in endemic areas (notably Southeast Asia) rabies should be suspected. Polio may also present as a paralytic illness and should be kept in mind in persons presenting from those few areas where polio still exists.

**PRINCIPLES OF MANAGEMENT**

**Prevention** is the cornerstone of management as lyssavirus infections including rabies are universally fatal. This involves pre exposure vaccination and boosters in those at risk of occupational exposure such
as Parks and Wildlife staff who may handle bats. Post exposure prophylaxis is recommended as soon as possible after bat bites or scratches and consists of a five dose course of rabies vaccine and importantly, passive cover with rabies immune globulin if not more than 7 days has elapsed since the start of vaccination. The decision to give vaccine, timing and completion of vaccine, 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 testing of the bat is really warranted, the Centre for Disease Control or the on call DMO 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.

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

Australian bat lyssavirus infection is a notifiable condition to be reported by all LABORATORIES in the Northern Territory. Cases should be reported to the Centre for Disease Control in your district.

FURTHER INFORMATION

TELEPHONE ADVICE

Immunisation Senior Project Officer
Infectious diseases or general physicians
Public Health Officer-on call
Public Health Officer
Public Health Officer
On call DMO-for remote areas
On call DMO-for remote areas
On call DMO-for remote areas
Centre for Disease Control-Darwin
Royal Darwin Hospital
Centre for Disease Control-Darwin
Centre for Disease Control-Gove
Centre for Disease Control-Katherine
Royal Darwin Hospital
Katherine District Hospital
Gove District Hospital
8922 8564
8922 8888
8922 8044
8987 0359
8973 9049
8922 8888
8973 9211
8987 0211

MANAGEMENT GUIDELINES

Australian Bat Lyssavirus Post-Exposure Prophylaxis (PEP) 2000
Centre for Disease Control-Darwin
8922 8044

The Australian Immunisation Handbook Current Edition
Centre for Disease Control-Darwin
8922 8044
Or Department of Health and Ageing
(02) 6289 1555

Australian Bat Lyssavirus Guidelines
Communicable Disease Network Australia
(02) 6289 7983

Communicable Disease Surveillance in the NT- Guidelines for the Reporting of Notifiable Conditions
Centre for Disease Control-Darwin
8922 8089

EDUCATIONAL RESOURCES

Poster-‘Don’t Touch or Handle Bats’
Fact Sheet-Australian Bat Lyssavirus (ABL)
Centre for Disease Control-Darwin
8922 8564
Centre for Disease Control-Darwin
8922 8564

FURTHER READING

HEPATITIS A IN THE TOP END

Hepatitis A (HAV) is a self-limiting viral infection of the liver. Where there is poor sanitation and hygiene, cumulative rates approach 80% by the age of two years. Although Australia has a low overall incidence of hepatitis A, the rates are very high in Aboriginal communities but epidemics of hepatitis A are rarely seen in this setting. This is because most Aboriginal people are infected as infants when the disease is usually subclinical and therefore not noticed and subsequent lifelong immunity is established. As hygiene improves the pool of susceptible people will increase and localised epidemics will become more common.

AETIOLOGY AND PATHOGENESIS

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 signs and symptoms listed below are due to acute hepatitis.

CLINICAL PICTURE

Risk factors. Risks include ingestion of sewage via contaminated water or shellfish; being in preschools, intellectually disabled or child care centres; anal sex; injecting drug use; and travel to developing countries or areas of poor sanitation within Australia.

Symptoms and signs. Infectiousness extends from two weeks prior to one week after symptom onset. No carrier state exists and lifelong immunity results. Fulminant HAV occurs in <0.5% of cases. Infected children under the age of five do not usually become jaundiced and cases are 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 hepatomegaly, dark urine from bilirubinuria, and pale stools. Investigations include urine dipstick for bilirubinuria, liver function tests, and hepatitis serology. HAV serology includes HepA IgM (positive in acute infection) and HepA IgG (remains positive after infection).

DIFFERENTIAL DIAGNOSIS

Acute hepatitis may result from infection (eg hepatitis A,B,C,D,E,G, arboviruses or leptospirosis), many toxins (eg alcohol), many medications (eg isoniazid, rifampicin, flucloxacinil) 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. 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. If non-immune, they should be offered immuneoglobulin (Normal Human Immunoglobulin) if within 14 days of exposure. All non-immune household and family contacts should be offered hepatitis A vaccine. Infections in areas of high transmission risk to the non-immune (disability or child care centres, preschools) may require treatment of more than household and family contacts. Phone CDC for advice.

Prevention of HAV infection includes good sanitation and vaccination. Many people over 40 years of age and most Aboriginal people have immunity. In some cases it may be worth checking this prior to offering the vaccine. HAV vaccination is 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, gay men, and plumbers. Boosters or serology post vaccination are not necessary. The priority and need for routine child HAV vaccination is being evaluated and not currently recommended.

Hepatitis A is a notifiable condition to be reported by all CLINICIANS and LABORATORIES in the Northern Territory. Cases should be reported to the Centre for Disease Control in your district by phone.
FURTHER INFORMATION

TELEPHONE ADVICE

Specialist physician
Public Health Officer-On call
Public Health Officer
Public Health Officer
Immunisation Senior Project Officer

Royal Darwin Hospital
Centre for Disease Control-Darwin
Centre for Disease Control-Gove
Centre for Disease Control-Katherine
Centre for Disease Control-Darwin

8922 8888
8922 8044
8987 0359
8973 9049
8922 8564

MANAGEMENT GUIDELINES

CARPA-Standard Treatment Manual
The Australian Immunisation Handbook Current Edition
NT Hepatitis A Vaccination Policy and Public Health Management Guidelines 2000
Communicable Disease Surveillance in the NT-Guidelines for the Reporting of Notifiable Conditions

Central Australian Division of Primary Health Care
Centre for Disease Control-Darwin
Centre for Disease Control-Darwin
Centre for Disease Control-Darwin

8950 4800
8922 8044
Or
Or

Department of Health and Ageing
Or

(02) 6289 1555

EDUCATIONAL RESOURCES

Fact Sheet- Hepatitis A

Centre for Disease Control-Darwin

8922 8044


FURTHER READING


Figure 18 Distribution of Hepatitis A notifications by State/Territory 1991 to 1997.
Hepatitis B (HBV) is a viral infection of the liver. Hepatitis B surface antigen was first identified in an Aboriginal patient and was initially known as the Australian antigen. Although carrier rates in developed Australia vary between 0.1 and 0.2%, in high risk groups such as Aboriginal Australians and immigrants from developing countries, the carrier rate is over 10%. NT rates are therefore higher than those in southern Australia.

**AETIOLOGY AND PATHOGENESIS**

Hepatitis B virus is transmitted via blood and secretions. Infection causes an immune mediated acute hepatitis.

**CLINICAL PICTURE**

**Risk factors.** HBV is transmitted by body fluids: unprotected sex; sharing injecting equipment, toothbrushes or razors; needlestick injury; and being born to or breastfed by a HBV carrier mother.

**Symptoms and signs.** The incubation period is 45 to 180 days. Infectiousness extends from several weeks before symptoms appear, until the resolution of the illness unless the carrier state develops. The infection is cleared in 90-99% of adults and lifelong immunity results. In 90% of neonatal and <10% of adult cases, an infectious chronic carrier state develops. Chronic active hepatitis develops in over 25% of carriers and of these 15-25% will die prematurely of cirrhosis or hepatocellular carcinoma. In adults 50% of HBV is asymptomatic; children under five are usually asymptomatic. Symptoms include: fever, malaise, nausea and vomiting, abdominal pain, myalgias, rash or arthritis. The acute illness may last for weeks; tiredness can persist for months. The signs of HBV infection include jaundice, tender hepatomegaly, dark urine, pale stools.

**Investigations.** Includes urine dipstick for bilirubinuria, liver function tests, and hepatitis serology for Hepatitis A, B and C. HBV serology changes are shown by Figures 19 and 20.

Hepatitis B is a notifiable condition to be reported by all CLINICIANS and LABORATORIES in the Northern Territory. Cases should be reported to the Centre for Disease Control in your district.
the patient is a carrier. HBV carriers should be followed with 6 monthly liver function tests (LFTs), alpha foetoprotein (AFP) and second yearly liver ultrasound. Referral to a specialist liver clinic should be considered.

**Prevention.** See CDC public health guidelines for detailed information about contact tracing and immunisation. Household and family contacts of people with acute infection or HBV carriers 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 14 days of exposure. Prevention of HBV infection also includes, safe sex practices, and needle exchange and routine immunisation. In the NT all neonates have been offered vaccination against hepatitis B since 1990 and in Aboriginal infants since 1988, and all Year 7 school children were targeted for vaccination in 1998/9. HBV vaccination is recommended for high risk groups such as health professionals, disability carers, gay men, and sex industry workers. Boosters or serology post vaccination are not necessary.

**FURTHER INFORMATION**

**TELEPHONE ADVICE**

| Immmunisation Senior Project Officer | Centre for Disease Control-Darwin | 8922 8564 |
| Public Health Officer | Centre for Disease Control-Gove | 8987 0359 |
| Public Health Officer | Centre for Disease Control-Katherine | 8973 9049 |
| Medical Officer or clinic staff | Clinic 34-Darwin | 8922 8007 |
| Specialist physician | Royal Darwin Hospital | 8922 8888 |
| Liver Clinic | Darwin Private Hospital | 8920 6181 |

**MANAGEMENT GUIDELINES**

| Communicable Disease Surveillance in the NT- Guidelines for the reporting of notifiable conditions | Centre for Disease Control-Darwin | 8922 8089 |

| CARPA-Standard Treatment Manual | Central Australian Division of Primary Health Care | 8950 4800 |
| The Australian Immunisation Handbook Current Edition | Centre for Disease Control-Darwin | 8922 8044 |
| Or | Department of Health and Ageing |
| Or | (02) 6289 1555 |

| NT Hepatitis B Vaccination Policy and Public Health Management Guidelines 2000 | Centre for Disease Control-Darwin | 8922 8044 |

**EDUCATIONAL RESOURCES**

| Fact Sheet-Hepatitis B | Centre for Disease Control-Darwin | 8922 8044 |

| Education Officer | AIDS/STD Programme | 8922 8007 |
| Or | http://www.sma.org.au/ |

| Pamphlet and Video-Blood Rules, OK | Sports Medicine of Australia | (02) 6230 4650 |

**FURTHER READING**

HTLV-I IN THE TOP END

HTLV-I, or Human T-cell Lymphotropic Virus-I, is present in confined populations, particularly in Japan and the Caribbean. It is endemic in Central Australia, with prevalence in remote communities up to 14%. In the Top End it is found predominantly in certain language groups of Walpiri peoples and other Aboriginal groups from Central Australia. It is not endemic in Arnhem Land or Darwin rural communities.

AETIOLOGY AND PATHOGENESIS

HTLV-I is a human retrovirus (distantly related to HIV) transmissible via breastfeeding, from mother to infant at birth, and through sexual intercourse. It can also be transmitted by blood (eg needlestick, transfusion). It causes illness through low-grade immunological compromise.

CLINICAL PICTURE

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

Symptoms and Signs: HTLV-I infection is often asymptomatic. It is sometimes found when investigating for underlying immunological problems in patients with crusted scabies. There are two major clinical syndromes associated with HTLV-I: adult T-cell leukaemia or lymphoma, and myelopathy. The malignancy may be indolent or aggressive: skin lesions, lymphadenopathy and hepatosplenomegaly may be present.

The myelopathy is a spastic paresis usually of lower limbs, with mild sensory involvement and occasionally incontinence. These syndromes are uncommon in Aboriginal people infected with HTLV-I. HTLV-I may worsen chronic infections such as scabies, TB, pyoderma, scabies and strongyloidiasis.

Investigations: Diagnosis is made by positive serology. All patients with crusted scabies should be tested for HTLV-I.

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 (eg malaria), 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-I is also associated with other malignant and inflammatory conditions.

PRINCIPLES OF MANAGEMENT

Management depends on the symptoms. The malignancy may initially respond to chemotherapy. TB and strongyloidiasis should be excluded prior to starting chemotherapy. 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. No cure is known.

In the instance where a needle stick injury has occurred and the source is an Aboriginal person from Katherine or the southern region of the NT then HTLV-I will need to be added to the standard blood tests. The Royal Darwin Hospital Infection Control Biohazard Injury Management manual offers more information.

HTLV-I is a CLINICIAN and LABORATORY notifiable condition in the Northern Territory and all cases should be notified to the Centre for Disease Control in your district.
FURTHER INFORMATION

TELEPHONE ADVICE

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<th>Role</th>
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<tr>
<td>Infectious diseases registrar or</td>
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<td>8922 8888</td>
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<td>Public Health Officer-on call</td>
<td>Centre for Disease</td>
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<td>Public Health Officer</td>
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<td>Public Health Officer</td>
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MANAGEMENT GUIDELINES

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<td>Reporting of Notifiable Conditions</td>
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<td>Darwin Hospital Infection Control</td>
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EDUCATIONAL RESOURCES

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<tr>
<td>The Public Health Bush Book</td>
<td>Dept. of Health &amp;</td>
<td>8999 2691</td>
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<td>Community Services-Wellness promotion</td>
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FURTHER READING

INFLUENZA IN THE TOP END

Influenza occurs throughout the year in the Top End but there are 2 peaks of activity (shown in the graph below). This pattern is different from that seen in temperate parts of Australia and the timing of epidemics is less predictable. It remains an important disease because of the rapidity with which epidemics evolve, the widespread morbidity and the seriousness of 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 early March however the vaccine should be promoted to unimmunised people in these groups throughout the year until the following year’s vaccine becomes available. There are Territory and national programs for providing free vaccines to some “high risk” individuals. Information about these programs is available from CDC-Darwin and from the immunisation support nurse of the Top End Division of General Practice.

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. For this reason laboratories in the NT must notify CDC of all positive diagnoses of influenza and 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.

CDC-Darwin co-ordinates the Tropical Influenza Surveillance scheme through GPs in the Top End. Participation in the scheme requires only minimal input from GPs who also receive 16 QA/CME points per year. Involvement of rural and remote GPs in addition to the urban GPs presently participating would greatly improve representativeness and usefulness of the scheme. For more information contact CDC-Darwin on 8922 8044.

Influenza is a notifiable condition to be reported by all sentinel surveillance GPs and LABORATORIES in the NT. Possible outbreaks should be reported by clinicians to the Centre for Disease Control in your district.
FURTHER INFORMATION

TELEPHONE ADVICE

Specialist Physician & Registrar
Immunisation support nurse
Public Health Officer-on call

Royal Darwin Hospital
Top End Division of General Practice
Centre for Disease Control-Darwin

8922 8888
8962 1000
8922 8044

MANAGEMENT GUIDELINES

Communicable Disease Surveillance in the NT-Guidelines for the reporting of notifiable conditions
The Australian Immunisation Handbook Current Edition

Centre for Disease Control-Darwin
Department of Health and Ageing

8922 8089
(02) 6289 1555

EDUCATIONAL RESOURCES

Influenza-Are you on the list for flu protection?
Fact Sheet-National Indigenous Pneumococcal and Influenza Health Worker
National Indigenous Pneumococcal and Influenza program poster
Various information sheets

Centre for Disease Control-Darwin

8922 8089


FURTHER READING

• Johnston F, Connors C, Krause V. Early influenza in the Northern Territory.CDI. 1995; May; 19(9):p 208-10
• Johnston F. Flu shots for health staff-three good reasons to be immunised. The Northern Territory Disease Control Bulletin. 1997, 4 (2): p4
Infestations and Fungal Infections
Cryptococcal Meningitis & Pneumonia

CRYPTOCOCCAL MENINGITIS AND PNEUMONIA IN THE TOP END

Cryptococcus neoformans can cause severe meningitis, which in the developed world, usually occurs in the setting of immune compromise. However in the Top End infection with C. neoformans var. gattii usually occurs in immunocompetent people, particularly in geographic areas such as Arnhemland. In other parts of Australia its distribution is associated with that of the River Redgum, and it is thought to be transmitted to humans via contact with these trees. River Redgums are not present in much of the Top End and the local sources of human cryptococcal infection are unknown, although the fungus has recently been grown from various eucalypts in Arnhemland.

AETIOLOGY AND PATHOGENESIS

C. neoformans is a yeast-like fungus. This ubiquitous environmental organism probably causes infection via inhalation. The C. neoformans var. gattii variety is mostly restricted to tropical and subtropical locations.

CLINICAL PICTURE

Symptoms and signs. Although the organism can also cause systemic infection and pneumonia, the most common clinical syndrome is meningitis. Symptoms may be similar to acute bacterial meningitis but are often insidious, and include headache, fever, irritability and confusion. Fever is usually mild, fluctuating up to 39°C. Memory loss or clouding of consciousness may be present. Cranial nerve palsies and papilloedema/papillitis sometimes occur accompanied by visual changes. Lung cryptococcomas are common and chest X-ray shows mass lesion(s).

Investigations are usually done in hospital. On CSF microscopy, diagnostic encapsulated yeasts may be seen best with an India ink stain. A mixed picture of lymphocytes and neutrophils are usually present. Cryptococcal antigen in CSF is both a sensitive and specific test. The antigen is positive in serum in nearly all cases of cryptococcal meningitis and two thirds of those with cryptococcal pneumonia.

DIFFERENTIAL DIAGNOSIS

Other causes of subacute meningitis should be considered, especially tuberculous meningitis. CT and lumbar puncture are mandatory to exclude cryptococcal meningitis in suspected cases.

PRINCIPLES OF MANAGEMENT

Initial treatment is hospitalisation and IV amphotericin for 4 to 6 weeks. Eradication therapy with fluconazole for some months is required, but long term follow up of Top End cases shows cure is the expected outcome. Treatment in the setting of HIV infection is slightly different and cure is less certain. Regular clinical follow-up after infection is important to exclude relapse.
FURTHER INFORMATION

TELEPHONE ADVICE

Infectious diseases registrar or physician Royal Darwin Hospital 8922 8888
Public Health Officer-on call Centre for Disease Control-Darwin 8922 8044
Public Health Officer Centre for Disease Control-Gove 8987 0359
Public Health Officer Centre for Disease Control-Katherine 8973 9049

MANAGEMENT GUIDELINES

CARPA-Standard Treatment Manual Central Australian Division of Primary Health Care 8950 4800
Therapeutic Guidelines: Antibiotic, Latest Version Therapeutic Guidelines LTD 1800 061 260

FURTHER READING

Tinea corporis is endemic in tropical areas and is ubiquitous in many Aboriginal communities. The predominant species in the Top End is *Trichophyton rubrum*. High levels of anthropophilic infections such as this 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 excluding groin, hands or feet (Tinea cruris, Tinea manuum and Tinea pedis). It is due to a related group of filamentous fungi also known as ringworm fungi. While *T. rubrum* is by far the commonest dermatophyte in the Top End, *T. tonsurans*, Epidermophyton and Mycosporum species also occur.

**CLINICAL PICTURE**

**Risk factors.** The risk factors include direct contact with skin or nail lesions of infected people. Children, teenagers and young adolescents are commonly affected.

**Symptoms and signs.** The clinical presentation depends upon several factors. These include the site of infection, immunological response of host and species of infecting fungus. There are usually no symptoms although mild pruritis may be present. The lesions are dry and scaly with an active border of advancing infection. They are often extensive, covering up to 50% of the body with exposed areas affected more commonly. Secondary bacterial infection is common.

**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. Clean 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.

**DIFFERENTIAL DIAGNOSIS**

The main differential diagnosis is pityriasis versicolor caused by *Malassezia furfur*, otherwise known as the ‘white handkerchief’ or ‘tissue’. Other possibilities include eczema, psoriasis, kava affected skin, extensive scabies and leprosy.
PRINCIPLES OF MANAGEMENT

Smaller lesions usually respond to topical anti-fungals applied over lesions and to 3cm beyond the active margin. Treat for one week beyond clinical improvement.

Extensive lesions usually require oral anti-fungals while griseofulvin has been the standard therapy for many years, this drug is only fungistatic and treatment for many weeks is required. 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. Abnormal liver function may require dose modification and monitoring. If greater than 2 weeks of terbinafine is planned, liver function should be checked before starting or during treatment.

FURTHER INFORMATION

TELEPHONE ADVICE

Dermatology registrar or dermatologist
Infectious diseases registrar or physician
Royal Darwin Hospital
Royal Darwin Hospital
8922 8888
8922 8888

MANAGEMENT GUIDELINES

CARPA-Standard Treatment Manual
Central Australian Division of Primary Health Care
8950 4800

Therapeutic Guidelines: Antibiotic or Dermatology-Latest Version
Therapeutic Guidelines LTD
1800 061 260

EDUCATIONAL RESOURCES

A Handbook of Skin Conditions in Aboriginal Population of Australia. Dr Allen Green
Office of Aboriginal & Torres Strait Islander Health (OATSIH)
8946 3481

Video-Australian Aborigines and Their Skin Conditions. Dr Allen Green
Royal Australian College of General Practitioners
(03) 9214 1414

FURTHER READING

**ONYCHOMYCOSIS IN THE TOP END**

*Tinea unguium or onychomycosis is a dermatophyte infection of the finger or toe nails. This is a common infection in the tropical climate of the Top End.*

**AETIOLOGY AND PATHOGENESIS**

Nail infection is usually secondary to *Tinea pedis*, *Tinea capitus* or *Tinea corporis*. *Trichophyton rubrum* is responsible for nearly all cases.

**CLINICAL PICTURE**

**Risk factors.** The risk factors include direct contact with skin or nail lesions of infected people or from contact with contaminated surface eg shower recess.

**Symptoms and signs.** Toenails are more commonly affected than fingernails. The nails become usually white or yellow in colour and the nail cracks and has irregular edges. Changes first appears at the free distal edge of the nail. Subungual hyperkeratosis may cause the nail to become detached from the nail bed. Paronychial inflammation is absent.

**Investigations.** Use curette or spatula to obtain material from under the nail for microscopic examination and culture. It is important to scrape the pulp under the nail to obtain live fungal elements for culture. This may be painful.

Laboratory diagnosis is important for the following reasons:

- High cost of treatment.
- Potential side effects (monitor liver function) of treatment.
- Eligibility for the Pharmaceutical Benefit Scheme (PBS) subsidy.

**DIFFERENTIAL DIAGNOSIS**

- Candida albicans (usually begins in the proximal nail plate and paronychial inflammation is present
- Bacterial infections.

**PRINCIPLES OF MANAGEMENT**

Oral antifungals are required. Treatment with griseofulvin is required for 4-12 months and relapse rates are high. Terbinafine has resulted in major improvements in fungal skin and nail disease in Aboriginal communities. Daily therapy for three months (fingernails) to six months (toe nails) is required. Pulsed terbinafine therapy and regimens with pulsed fluconazole and itraconazole are also being studied. Liver function should be checked prior to and during treatment when > 2 weeks terbinafine is planned.
FURTHER INFORMATION

TELEPHONE ADVICE

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MANAGEMENT GUIDELINES

CARPA-Standard Treatment Manual  Central Australian Division of Primary Health Care  8950 4800
Therapeutic Guidelines; Antibiotic or Dermatology-Latest Version  Therapeutic Guidelines LTD  1800 061 260

EDUCATIONAL RESOURCES

A Handbook of Skin Conditions in Aboriginal Population of Australia. Dr Allen Green  Office of Aboriginal & Torres Strait Islander Health (OATSIH)  8946 3481
Video-Australian Aborigines and Their Skin Conditions. Dr Allen Green  Royal Australian College of General Practitioners  (03) 9214 1414

FURTHER READING

SCABIES IN THE TOP END

Scabies and associated streptococcal skin infections are the two most common and important skin conditions that are seen in Aboriginal people in the Top End. The high prevalence of streptococcal skin infection contributes to the extremely high rates of acute post-streptococcal glomerulonephritis and rheumatic fever. Scabies has been documented to occur in 30 year cycles and it is thought that this relates to the level of population immunity.

Historical documentation about the prevalence of scabies in the Territory is patchy. There were reports in 1815 of scabies being problematic on missions but not amongst tribal people. In Central Australia in 1957 it was reported to be common however in 1960 a scientific expedition in Arnhemland demonstrated that scabies was quite rare. This is consistent with the experience of leprosy nurses who worked throughout the NT and recalled that scabies and skin sores were rare around that time. Long term health professionals in the Top End believe the rates of scabies and skin infections have been increasing since the 1970’s.

Currently community surveys show high but variable prevalence rates ranging from 30-50% in children under the age of 15. Surveys of adults have shown similar levels of at least 30% at any one time, although adults generally have milder clinical manifestations. One of the important features of scabies is that it provides an ideal niche for streptococcal skin infections. Community surveys have demonstrated that up to 70% of children have streptococcal skin sores. However, the contribution that scabies makes to skin sores may vary 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 mate and reinvade the skin of the same or another host.

Initial infestation is asymptomatic. After 4 to 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 occurring in people who usually have some form of immune deficiency. 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. In crusted scabies the affected person cannot contain the mite replication and becomes infested with thousands of them, developing a thick, crusted skin in response. They are highly infectious to others and also highly susceptible to reinfection.

CLINICAL PICTURE

Risk Factors. The major risk factor is close contact with an infected person. In a situation of overcrowding the risk of infection is high. Young children and people with crusted scabies are ‘key transmitters’ as they carry large numbers of mites.

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 wide distribution of lesions. Older children and adults usually only have a small number of lesions.

The most common symptom is an itchy rash, which usually has a classic distribution. 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 interdigital 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 occasionally 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 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 rarely performed because the clinical picture is usually clear. Ideally the scabies mites are identified from burrows, often between fingers, and demonstrated under a microscope. However burrows are much less commonly found in the Top End as they are hard to see on dark skin. A swab of associated skin sores will usually grow streptococcus and often staphylococcus.

For crusted scabies it is important to confirm the diagnosis. This is done by scraping the crusted skin lesions into a yellow top pathology jar and sending it with a request for Microscopy for scabies. Wear gloves and wash hands afterwards as crusted scabies is very infectious. Investigations should be done to exclude underlying immune deficiencies, including renal failure, systemic lupus erythematosus (SLE) and Human T cell lymphotropic virus (HTLV-I). Crusted scabies is one of several conditions that may occur in people with immune disorders, others include melioidosis, tuberculosis and persistent strongyloidiasis. Recommended investigations are in the CDC treatment protocol for crusted scabies.

**DIFFERENTIAL DIAGNOSIS**

Scabies should always be considered as the underlying cause of skin sores. The differential diagnosis should include eczema, mild cases of psoriasis and possibly 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 possibly kava dermatitis. Ingestion of kava can cause a flat, fine, grey dermatitis, which people often describe as having black skin because the shininess of healthy skin is lost.
PRINCIPLES OF MANAGEMENT

For treatment details refer to the scabies and skin sore guidelines from CDC and the CARPA Manual. The mainstay of treatment is permethrin cream, two treatments separated by two weeks. The individual and all their closely related contacts need to be treated. In Aboriginal communities this includes everyone within the household, in particularly adults who share a bed 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 the Benzathine penicillin for streptococcal infection.

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.

Crusted scabies involves both topical treatment with permethrin and a keratolytic cream to soften the crust and allow permethrin to penetrate. They 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 CDC crusted scabies protocol or discuss with an infectious diseases physician. Severe cases usually need treatment in hospital. This provides a good opportunity for the family to be adequately treated and the house to be cleaned.

The protocol for crusted scabies also includes treatment of the entire household. Additionally, Environmental Health Officers work with family members to clean the house and use insecticide bombs to kill all scabies mites. The main complication of crusted scabies is septicaemia. A study done at RDH 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 causes of death were related to septicaemia due to the fissuring that occurs in the crusted skin allowing direct access for infecting. Aggressive antibiotic therapy has reduced this mortality.

Healthy skin programs. Community programs aiming to reduce the prevalence of scabies and streptococcal skin disease have been run in a number of communities across the Top End. Guidelines are available from CDC. The programs involve an education campaign, developing local resources and a one off treatment of the entire community with scabies cream. Regular surveillance of young children both pre and post the community treatment day is important.
FURTHER INFORMATION

TELEPHONE ADVICE

Infectious Diseases Registrar or Physician  
Public Health Officer-on call  
Public Health Officer  
Public Health Officer  
Dermatology Registrar or Dermatologist  
Healthy Skin Team  
Royal Darwin Hospital  
Centre for Disease Control-Darwin  
Centre for Disease Control-Gove  
Centre for Disease Control-Katherine  
Centre for Disease Control-Darwin  
Centre for Disease Control-Darwin  
Co operative Research Centre for Aboriginal and Tropical Health, Menzies School of Health Research  
8922 8888  
8922 8044  
8987 0359  
8973 9049  
8922 8888  
8922 8196

MANAGEMENT GUIDELINES

CARPA—Standard Treatment Manual  
Therapeutic Guidelines: Antibiotic. Latest Version  
Healthy Skin Program—Guidelines for Community Control of Scabies, Skin Sores and Crusted Scabies 2002  
Guidelines for the Control of Acute Post-Streptococcal Glomerulonephritis 1997  
Infection Control Standards  
Central Australian Division of Primary Health Care  
Therapeutic Guidelines LTD  
Centre for Disease Control-Darwin  
Centre for Disease Control-Darwin  
Centre for Disease Control-Darwin  
DHCS-Royal Darwin Hospital Infection Control  
8950 4800  
1800 061 260  
8922 8044  
8922 8044  
8922 8888

EDUCATIONAL RESOURCES

Flip Chart & Video—Healthy Skin Story  
A Handbook of Skin Conditions in Aboriginal Population of Australia. Dr Allen Green  
The Public Health Bush Book  
Video—Australian Aborigines and their Skin Conditions. Dr Allen Green  
Pamphlet—Scabies  
Scabies/Treatment Poster & Scabies Kidney Skin Sores Poster  
Deadly Kids Poster & Scabies Fact Sheet  
CD ROM—Scabies  
CRCATH Health Skin Team Education Package  
Nalkanbuy Health Service Galiwinku  
Office of Aboriginal & Torres Strait Islander Health (OATSIH)  
Dept. of Health & Community Services—Wellness promotion  
Royal Australian College of General Practitioners  
Centre for Disease Control-Darwin  
Australian Kidney Foundation  
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FURTHER READING

HEAD LICE IN THE TOP END

The prevalence of head lice in the Top End is high, particularly in remote 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 wingless insects, 2-3mm in length and opaque to brown in colour. Nits are the small eggs laid by the lice and bonded onto the hair shaft close to the scalp. They are yellow to white in colour and are difficult to remove from the hair shaft. 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 >1cm from the scalp are dead or empty.

CLINICAL PICTURE

Infestation may be asymptomatic, although it often causes an itchy scalp. Direct inspection of the scalp/hair in the areas behind the ears with a strong light will detect the nits. Live lice are difficult to see as they quickly move away from disturbed areas. To find them you can 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 abscess are often associated with head lice infestation.

PRINCIPLES OF MANAGEMENT

The mainstay of management is 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 is becoming an increasing problem. Recently it has been shown that treatment with thick white hair conditioner applied to slightly moistened hair and combed with a “nit” comb is as effective as commercial anti lice treatments. The best results are probably obtained by using both the combing AND insecticide regimens, ie insecticide to get on top of heavy infestations and combing for ongoing maintenance therapy. In all cases you should:

- 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 reinfestation.

Conditioner/combing method. Use a thick white conditioner, apply to slightly moistened hair and rub through to scalp. Leave for ten minutes and then work through hair with a nit comb. Wipe comb on to white tissue. Both the adult lice and nits will be removed. Comb in three directions, especially the occiput and around the ears, before rinsing out with water. Repeat every 2 days until no further lice are removed. This method does not cause local irritation and is well tolerated.

Using insecticides. The types of insecticides available are pyrethrins, synthetic pyrethroids (permethrin and bioallethrin) and organophosphates (malathion or maldisone). Natural products such as ti-tree and eucalyptus oil have been advocated but have been shown to be ineffective and have high toxicity, especially if ingested by children. However it should be noted that all insecticides can be toxic and should be used strictly according to the instructions on the bottle.

Treatment needs to be done twice, seven days apart. The first to kill adult lice and the second to kill newly hatched juvenile lice.

To test for resistance, comb the hair after the preparation has been on for 20 minutes. If all lice removed are dead, then the lice are sensitive to the
product used. If some lice are inactive but alive, partial resistance is present. If some lice are active, infestation is resistant and treatment should be repeated with an alternative product. If lice are found after a second treatment, a third treatment is required after 7 days.

**Figure 31 A flow chart for the treatment of head lice.** Source: http://www.nt.gov.au/health/cdc/protocols.shtml

**FURTHER INFORMATION**

**TELEPHONE ADVICE**

DHCS Community Health Team
Community Paediatrician
Centre for Disease Control-Darwin
Centre for Disease Control
8922 8044
8922 8044

**MANAGEMENT GUIDELINES**

*Nits Not*. The Northern Territory Head Lice Action Pack-Information for NT parents, Schools and Childcare Centres, Current Edition

DHCS Community Health Team
8922 8044
Or

**EDUCATIONAL RESOURCES**

Fact Sheet-Head Lice and Nits
Head Lice Information Sheet
Fact Sheet-Head Lice
Dept of Health, Victoria, Public Health Division


**FURTHER READING**

Paederus Australis (the ‘Acid Beetle’)

PAEDERUS AUSTRALIS IN THE TOP END

*Paederus australis*, (‘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. It is 5 -10 mm 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. Skin damage takes several hours to occur and the patient may not associate the symptoms with the beetle. In the Top End, communities close to flood plains can experience plagues of beetles, especially following heavy rainfall.

Clinically, *Paederus* causes an irritant contact dermatitis characterised by linear streaks and ‘kissing lesions’ which occur where two skin surfaces are in contact with each other. Initial contact with the beetle often occurs at night and is painless. The rash appears after about 24 hours and subsequently blisters, becoming very painful. After about a week the lesions dry and become itchy and 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 diagnoses include herpes simplex or zoster, impetigo, other forms of irritant or contact dermatitis, conjunctivitis and the periorbital swelling of acute glomerulonephritis. 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. Outbreaks can be so severe that evacuation of an entire outstation may be required.
FURTHER INFORMATION

TELEPHONE ADVICE

Public Health Officer-on call
Medical Entomology
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8922 8044
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FURTHER READING

Hookworm

HOOKWORM IN THE TOP END

Hookworm is a common faecal/soil transmitted helminth and one quarter of the world’s population is thought to be infected with hookworm at any given time. It is particularly common in tropical areas, such as remote communities of the Top End. While it is an important cause of anaemia on a global scale, regular deworming programs in the Top End have dramatically reduced its importance as a contributor to iron deficiency in this setting.

In a recent study of Aboriginal children admitted to RDH, Hookworm was only detected in one of 275 children with diarrhoea and was not detected at all in a comparison group of 87 Aboriginal children admitted for other reasons.

AETIOLOGY AND PATHOGENESIS

There are two species, *Ancylostoma duodenale* (Old World hookworm) and *Necator americanus* (New World hookworm). *A. duodenale* is thought to be the hookworm present in the Top End. 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 0.2 mls of blood per day. Adult worms live for 2-8 years and produce thousands of eggs. The period from skin invasion to appearance of eggs in the faeces is about 6 to 8 weeks. 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.

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 sensitized 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 symptomatic with weakness, shortness of breath and skin depigmentation.

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*. *A. caninum*, the dog hookworm, has been identified as a cause of human eosinophilic enteritis, especially in northeastern Australia. It has not been diagnosed in Aboriginal communities. Cutaneous larva migrans, from larvae of the cat and dog hookworm (*A. braziliense*) migrating through the skin, is seen almost exclusively in non-Aboriginal people in the Top End.

PRINCIPLES OF MANAGEMENT

Treatment: The treatment of hookworm infection is effective with several safe and highly effective anthelmintic drugs. Regular treatment of children with anthelmintics is part of the child health programs in the Top End. Severe hookworm disease with protein loss and malabsorption is rare but requires additional nutritional support. In the absence of
reinfection, the majority of worms is eliminated spontaneously within 1-2 years.

Prevention includes community education, avoiding skin contact with contaminated soil, and improved sanitation infrastructure and practices. For more information about the prevention and management of anaemia refer to the case study 'Keeping The School Aged Kids Healthy'.

Figure 34 The life cycle of hookworm. Source: http://www.dpd.cdc.gov/dpdx/HTML/ImageLibrary/Hookworm_il.htm

Figure 35 Ancylostoma duodenale egg. Source: www.dpd.cdc.gov/dpdx/html/ImageLibrary/Hookworm_il.asp?body=G-

Figure 36 Cutaneous Larva migrans from Ancylostoma braziliense (cat - dog Hookworm).

Figure 37 Hookworm. Source: www.bigwavetv.com/BodySnatchers/Hookworm.jpg
FURTHER INFORMATION

TELEPHONE ADVICE

DHCS Community Child Health Team Centre for Disease Control-Darwin Paediatrician 8922 8044 Royal Darwin Hospital 8922 8888

MANAGEMENT GUIDELINES

CARPA-Standard Treatment Manual Central Australian Division of Primary Health Care 8950 4800
Therapeutic Guidelines: Gastrointestinal Therapeutic Guidelines LTD 1800 061 260

Healthy School Aged Kids Program DHCS Community Child Health Team 8922 8044
Growth Assessment and Action Program DHCS Community Child Health Team 8922 8044
Community Based Diagnosis and Treatment of Anaemia in NT Aboriginal Children Discussion Paper. January 2001 DHCS Community Child Health Team 8922 8044

EDUCATIONAL RESOURCES

Intestinal Parasite Prevention & Education Project Video Top End Division of General Practice 8982 1000

FURTHER READING

• Kukuruzovic R, Brewer D. Diarrhoeal Diseases in Top End Aboriginal Children. Occasional Papers Series Issue 1, 2002. Centre for Clinical Excellence. (Available at DHCS medical libraries)
STRONGYLOIDIASIS IN THE TOP END

Strongyloidiasis is caused by infestation with *Strongyloides stercoralis*. It 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 returned travellers from endemic areas. Worldwide, an estimated 80 million are thought to be infected and prevalence rates of above 5% have been defined as hyperendemic. In the NT, some eastern Arnhemland communities have demonstrated positive isolation rates from stools in selected people ranging from 15% to 25% and sero-positivity in up to 60% of people tested. Strongyloidiasis in the Top End warrants further study particularly in relation to epidemiology, diagnostic testing and optimal management protocols.

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 although some infections may result from ingestion of contaminated food and drink.

The 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 worms (2mm long) and penetrate the mucosa of the proximal small bowel (jejunum) where they lay up to 40 eggs a day. This occurs 17 to 28 days after the initial infection. The eggs hatch in the intestinal mucosa to release rhabditiform larvae that migrate to the lumen of the bowel. Three cycles are possible.

**Direct host-soil-host cycle.** The rhabditiform larvae in the faeces enter the soil and become filariform larvae that can then infect the host.

**Indirect cycle.** The rhabditiform larvae in the faeces enter the soil and develop into free-living male and female adults which reside and reproduce in the soil, thus creating a reservoir of infection independent of the human host.

**Autoinfection or hyperinfection cycle:** The rhabditiform larvae develop in filariform larvae before they are passed in the stool. The filariform larvae can penetrate the colonic wall or perianal skin and enter the circulation to repeat the migration that establishes ongoing internal reinfection. Replication occurs through a process called parthenogenesis and 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 Indigenous communities in northern Australia and many tropical countries. This includes immigrants, returned travellers and Vietnam war veterans.

**Symptoms and signs.** Infection may be asymptomatic. However, most organs of the body can be affected including:

- **Skin:** Larva currens, lesions over lower back and buttocks, recurrent urticaria.
- **Respiratory tract:** Dyspnoea, bronchospasm, gross haemoptysis.
• Gastrointestinal tract: 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.
• Genitourinary tract: Urinary tract infection.
• CNS: Gram negative meningitis, focal or general CNS
• Systemic: Gram negative bacteraemia, disseminated strongyloidiasis.

In the Top End there are three important clinical manifestations of infection with *S. stercoralis* in Aboriginal communities;

i. Acute gastrointestinal infection in children, often with diarrhoea, hypokalaemia and abdominal distension. Pseudo-intestinal obstruction can occur. The diarrhoea has a distinctive odour.

ii. Gram-negative meningitis/septicaemia, as the parasite facilitates penetration of gut bacteria into the circulation.

iii. Disseminated strongyloidiasis in patients on immunosuppressive therapy. There have been several deaths in the Top End over the last 15 years in patients on high dose immunosuppressive therapy.

**Investigations.**

**Microscopy and culture of faeces.** To increase the sensitivity, three (or more) specimens should be sent. Agar plate is considered by many to be the most sensitive culture technique but is not offered by all laboratories in the NT. Fresh faeces may demonstrate larvae of *S. stercoralis* on microscopy. However, if faeces are examined a few days later, any hookworm eggs that may be present may also have hatched into larvae. Larvae should therefore be accurately identified, especially if specimens have been sent from a remote site. Placing faeces into formalin prior to transport may improve detection rates in specimens from remote locations.

**Blood:** Eosinophilia is present in 10 to 50% of cases. Strongyloides serology is used in diagnosing chronic strongyloidiasis in individuals, particularly adults, and for individual follow-up as titres may drop after 6 months with successful treatment. Serology results in an endemic area such as the Top End need to be interpreted in the light of the clinical picture.

In disseminated disease, sputum wet mount examination may be useful. Strongyloides 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 aim of treatment of infected individuals is to prevent the risk of severe invasive disease. The drugs of choice are ivermectin and albendazole. Thiabendazole is rarely used now because of side effects. Refer to the current ‘Therapeutic Guidelines: Antibiotic’ or ‘Therapeutic Guidelines: Gastrointestinal’.

Follow-up is important because the parasite is not easily eradicated and retreatment may be necessary. The diagnosis is worth considering in household contacts. New cases may be stool positive and seronegative because seroconversion has not occurred.

Prognosis is usually good except in severe cases of hyperinfection. Since the occurrence of hyperinfection is unpredictable, every effort should be made to eradicate infection in each case. 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 prolonged high dose steroid or immunosuppressive therapy. In some cases, this may require prophylactic treatment prior to receiving results. Protocols are being developed for investigation and
prophylaxis/treatment of Aboriginal patients on high dose steroids (eg prednisolone ≥ 0.5mg/kg/day in children or 25mg/day in adults for over 2 weeks) or other immunosuppressive therapy (eg for renal transplant).

**Prevention.** Infection may be transmitted by direct contact with faeces or by ingestion of contaminated food or contaminated drinking water. Prevention therefore includes community education about disease transmission, avoiding skin contact with contaminated soil and improved sanitation practices.
FURTHER INFORMATION

TELEPHONE ADVICE

DHCS Community Health Team
Community paediatrician
Paediatrician
Infectious diseases registrar or physician
Centre for Disease Control-Darwin
Centre for Disease Control-Darwin
Royal Darwin Hospital
Royal Darwin Hospital
8922 8044
8922 8044
8922 8888
8922 8888

MANAGEMENT GUIDELINES

CARPA-Standard Treatment Manual
Therapeutic Guidelines: Gastrointestinal
Therapeutic Guidelines: Antibiotic
Healthy School Aged Kids Program
Growth Assessment and Action Program
Community Based Diagnosis and Treatment of Anaemia in NT Aboriginal Children-Discussion Paper, January 2001
Central Australian Division of Primary Health Care
Therapeutic Guidelines LTD
DHCS Community Health Team
DHCS Community Health Team
8950 4800
1800 061 260
8922 8044
8922 8044

EDUCATIONAL RESOURCES

Video-Intestinal Parasite Prevention & Education Project
Strongyloidiasis Clinical Audit Report
Flipchart- Strongyloides Story
Top End Division of General Practice
Top End Division of General Practice
Top End Division of General Practice
8982 1000
8982 1000
8982 1000

FURTHER READING

TRICHURIASIS IN THE TOP END

Trichuriasis is an intestinal infection of humans caused by the helminth Trichuris trichiura. Along with other intestinal parasites it is common in many remote communities. It has a global distribution especially in the tropics and areas with poor sanitation. In one community in the East Arnhem region, it was identified in the stools of 80% of those examined. Infection may contribute to anaemia and growth retardation.

AETIOLOGY AND PATHOGENESIS

The adult worm is 30-50 mm in length and has a characteristic whiplike shape. Trichuris has a faecal-soil-oral transmission cycle. The anterior section embeds into the superficial mucosa of the colon and caecum, lays 3000 to 7000 eggs per day and may live for 5 years. The eggs are passed with the faeces and incubate for at least 3 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 entire cycle takes about 3 months.

CLINICAL PICTURE

Risk factors. Living in conditions of poor sanitation 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 burdens may also contribute to growth retardation and anaemia. It has been estimated that infected patients lose 0.005ml blood per worm per day.

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

PRINCIPLES OF MANAGEMENT

Treatment of individuals is with anthelmintics. However even 3 doses of albendazole will not eradicate all Trichuris infections, although the worm burden will be substantially decreased. Refer to the current Antibiotic Guidelines. Regular antihelminthic treatment is part of child health programs in the Top End. Contact the community paediatric team in CDC for information.

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

Figure 39 The life cycle of T. trichiura

Source: http://www.dpd.cdc.gov/dpdx/HTML/ImageLibrary/Trichuriasis_il.htm
Figure 40 Egg of *Trichuris trichuria* as seen on wet mount.

FURTHER INFORMATION

**TELEPHONE ADVICE**

<table>
<thead>
<tr>
<th>Service</th>
<th>Contact Information</th>
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**EDUCATIONAL RESOURCES**

<table>
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<tr>
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<tr>
<td>Intestinal Parasite Prevention &amp; Education Project Video.</td>
<td>Top End Division of General Practice</td>
</tr>
<tr>
<td></td>
<td>8982 1000</td>
</tr>
</tbody>
</table>

**FURTHER READING**

Sexually Transmitted Infections
STIs IN THE TOP END

The Northern Territory has disproportionately high notification rates of all STIs. NT data demonstrate a higher prevalence in both Aboriginal and non-Aboriginal populations compared with other parts of Australia. The burden of disease is greatest among Aboriginal groups, with the highest notification rates occurring in the 15-19 year age group. Notification rates are a crude measure, reflecting community testing patterns, screening programs, laboratory testing and reporting practices of practitioners. They are therefore more likely to be an underestimate of the true prevalence of many STIs.

CLINICAL PICTURE

Risk factors. There are many reasons for the NT having such high notification rates for STIs. These include a younger and highly mobile population, high rates of substance misuse (alcohol, marijuana, petrol), barriers to access and delivery of health services including, cultural and language barriers, relatively good detection with regular screening programs and a lower threshold for testing by health care practitioners.

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 pelvic inflammatory disease, infertility, tubal pregnancy, adverse pregnancy outcomes and psychological distress. There is also now strong evidence that the presence of other STIs significantly increases the risk of transmission of HIV.

Infection with an STI is frequently asymptomatic (see Table 4). As a result, only a minority of patients with a STI may present to a medical service for treatment. This proportion may be further reduced by ignorance of the significance of symptoms, lack of access to services, fear of unpleasant or embarrassing tests and the stigma attached to a diagnosis of an STI. This is often compounded in the remote Aboriginal 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, suggests a STI or PID and requires appropriate further history, examination and testing. A high degree of clinical suspicion for PID should be maintained for Aboriginal women presenting with low abdominal pain and/or dysuria and a diagnosis of urinary tract infection made with caution.

PRINCIPLES OF MANAGEMENT

Syndromic approach

It is well documented that diagnosis of the aetiology of STIs on clinical features alone is highly inaccurate, even by 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 well-defined aetiologies. Its chief advantage lies in the use of empirical therapy, or treatment at the first visit in the absence of a microbiological diagnosis, resulting 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. The WHO has recommended that national STI control programs in developing countries incorporate syndrome based diagnostic and therapeutic flowcharts in their management guidelines. The use of syndromic flow charts standardises diagnosis, treatment, referral and reporting, leading to improved surveillance and program management.

The NT uses the syndromic approach in STI management, for example in such presentations as urethritis, cervicitis and genital ulcer disease. Unlike developing countries, however, this strategy is supported by laboratory investigation in almost all cases.

Population Approach
The traditional way that health services deliver STI services is to wait for symptomatic people to present to the clinic for treatment. With high rates of asymptomatic infection and significant barriers to service utilisation, this will clearly miss a large proportion of people in the community with an STI.

A population based approach is therefore necessary for effective STI control, including strategies such as opportunistic and community screening, education programs, provision of condoms, specific staff training and increased accessibility of health services. For a comprehensive discussion of the population health approach to STIs, see ‘STI Control in Remote Aboriginal Communities. A manual for clinic workers’ (see management guidelines).

Contact tracing
Contact tracing has long been regarded as an essential control strategy in STI management. It is recognized that 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 partners.

In remote Aboriginal communities, contact tracing is often viewed as resource and time intensive. Nonetheless, it continues to be an integral part of STI control activities in the NT and an important component of comprehensive care and needs to be carried out. Your local CDC AIDS/STD Unit should be called on to assist and give direction in contact tracing.

Notifications
In the NT, notification of diseases may be made by both laboratories and primary care practitioners, including doctors and nurses. However, unlike other jurisdictions, there is no requirement for GP’s to notify many infections already notified by laboratories. These include gonorrhoea, chlamydia, trichomoniasis and HIV. The STIs that doctors must report are chancroid and lymphogranuloma venereum (rare ulcerative STIs not endemic in Australia), donovanosis, syphilis and AIDS.

<table>
<thead>
<tr>
<th>STIs</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gonorrhoea</td>
<td>10-15%</td>
<td>50%</td>
</tr>
<tr>
<td>Chlamydia</td>
<td>30-40%</td>
<td>75%</td>
</tr>
<tr>
<td>HSV</td>
<td>25-30%</td>
<td>25-30%</td>
</tr>
<tr>
<td>Trichomonas</td>
<td>50%</td>
<td>50%</td>
</tr>
</tbody>
</table>

Table 4 Asymptomatic Rates for Men and Women for Common STIs. Source: AIDS/STD Unit, Darwin.
An Overview of Sexually Transmitted Infections (STIs)

FURTHER INFORMATION

TELEPHONE ADVICE

Medical Officer or clinic staff    Clinic 34-Darwin    8922 8007
Medical Officer or clinic staff    Clinic 34-Gove    8987 0359
Medical Officer or clinic staff    Clinic 34-Katherine    8973 9049

MANAGEMENT GUIDELINES

Standard Treatment Protocols for Sexually Transmitted Infections (Pelvic Inflammatory Disease). October 2000    AIDS/STD Program-Darwin    8922 8007
CARPA-Standard Treatment Manual    Central Australian Division of Primary Health Care    8950 4800
Women’s Business Manual    Ngnampa Health Council Inc.    8950 5435
Communicable Disease Surveillance in the NT-Guidelines for the Reporting of Notifiable Conditions    Centre for Disease Control-Darwin    8922 8089
STI Control in Remote Aboriginal Communities A Manual for Clinic Workers, Commonwealth Department of Health and Aged Care, Canberra, 1999.
Investigation and Treatment of Infants at Risk of Congenital Syphilis 1998
Guidelines for the Control of Gonococcal Conjunctivitis 1997

FURTHER READING

Gonorrhoea

In the Top End, genital tract infection with gonorrhoea is common and a cause of significant morbidity.

**GONORRHOEA IN THE TOP END**

In the Top End, genital tract infection with gonorrhoea is common and a cause of significant morbidity, particularly in women. Notification rates for gonorrhoea in the NT (621/100,000) were about twenty times higher than the overall Australian rate (29/100,000) in 1998, with higher rates in both Aboriginal and non-Aboriginal Territorians. As with other sexually transmitted infections (STIs), the vast majority of this excess burden of disease is in Aboriginal people, with rates (1603/100,000) about 50 times that of the national rate. The highest rates of infection were in the age group 15-19 years.

**AETIOLOGY AND PATHOGENESIS**

*Neisseria gonorrhoeae*, the causative organism of gonorrhoea, is a gram negative diplococcus and was identified over 120 years ago. 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 STI overview.

**Symptoms and Signs.** Infection with *Neisseria gonorrhoeae* manifests as a wide spectrum of clinical presentations, including asymptomatic infection, 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 epididymitis and prostatitis.

**FEMALES** Gonococcal infection in women is symptomatic in only about half of cases. The most common symptomatic presentation is of lower genital tract infection, manifesting as vaginal discharge, dysuria and/or intermenstrual bleeding. A “friable” cervix with contact bleeding or frank mucopurulent cervicitis are signs commonly found on speculum examination.

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 variably present with any combination of pelvic pain, fever, dyspareunia or menstrual irregularities, but is also often 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 tubal infertility and ectopic pregnancy.

**GENERAL** Pharyngeal infection is not uncommon in both sexes and though also usually asymptomatic, may present as an acute sore throat. Rectal gonorrhoea may be asymptomatic or cause a rectal discharge. Gonococcal conjunctivitis is rare in adults but should always be considered in the neonate.

Disseminated gonococcal infection (DGI) is rare (0.5 - 3% gonorrhoea cases), and manifests most commonly as the arthritis-dermatitis syndrome, with joint pain and skin lesions. Endocarditis and meningitis are less common.

**DIFFERENTIAL DIAGNOSIS**

See the urethritis case study ‘Case Study of a Common Syndrome’ for discussion of the differential diagnosis of urethritis in men. In women, the presence of vaginal discharge could represent cervicitis, upper genital tract infection (PID) or vaginitis from candidiasis or trichomoniasis, as well as non infective causes. Similarly, dysuria or positive leukocytes on urinalysis could be a manifestation of a STI (with or without PID), vaginitis or a urinary tract infection.

**PRINCIPLES OF MANAGEMENT**

For discussion of investigations and management see
the case studies about urethritis 'Case Study of a Common Syndrome' and pelvic pain 'More Than Just Pain' in this guide, the Clinic 34 Protocols, the CARPA manual and the Women's Business Manual.

Treatment should be syndromic and empirical. Always send a specimen for culture to determine antibiotic susceptibilities.

PID is notoriously difficult to diagnose and a high degree of clinical suspicion must be maintained at all times. All women with possible PID, and particularly those with confirmed gonorrhoea or endocervicitis, should be assessed for PID by appropriate history and examination. There should be a low threshold for treatment of suspected PID.

As with all STIs, comprehensive contact tracing should be undertaken for partners of patients infected with gonorrhoea.

Gonococcal infection is a notifiable condition to be reported by all LABORATORIES in the Northern Territory. Gonococcal conjunctivitis is a notifiable condition to be reported by all CLINICIANS in the Northern Territory and all cases should be notified to the Centre for Disease Control in your district by phone.

FURTHER INFORMATION

TELEPHONE ADVICE

| Obstetrician/gynaecologist | Royal Darwin Hospital | 8922 8888 |
| Medical Officer or clinic staff | Clinic 34-Darwin | 8922 8007 |
| Medical Officer or clinic staff | Clinic 34-Gove | 8987 0359 |
| Medical Officer or clinic staff | Clinic 34-Katherine | 8973 9049 |

MANAGEMENT GUIDELINES


Standard Treatment Protocols for Sexually Transmitted Infections (Pelvic Inflammatory Disease). October 2000 | AIDS/STD Program-Darwin | 8922 8007 |

CARPA-Standard Treatment Manual | Central Australian Division of Primary Health Care | 8950 4800 |

Women's Business Manual | Nganampa Health Council Inc. | 8950 5435 |

Communicable Disease Surveillance in the NT-Guidelines for the Reporting of Notifiable Conditions | Centre for Disease Control- Darwin | 8922 8089 |

Or OATSIH Office NT

EDUCATIONAL RESOURCES

Fact Sheet-Gonorrhoea | Clinic 34-Darwin | 8922 8007 |

FURTHER READING

A Common Syndrome-
Case Study of 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 an episode of unprotected sex about a week previously. 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 other possible causes of urethral discharge?

<table>
<thead>
<tr>
<th>Sexually Transmitted</th>
<th>Non Sexually Transmitted</th>
</tr>
</thead>
</table>
| Gonococcal urethritis  
*Neisseria gonorrhoeae* | Prostatitis |
| Non-gonococcal urethritis (NGU)  
*Chlamydia trachomatis*  
*Ureaplasma urealyticum*  
*Mycoplasma genitalium*  
*Trichomonas vaginalis*  
Yeasts  
HSV  
others | Stricture  
Phimosis  
Chemical Irritation  
Trauma |

Table 5 Aetiology of urethritis in males

Sexually acquired urethritis can be divided into gonococcal (GU) and non-gonococcal (NGU) aetiologies. Roughly half of NGU is caused by chlamydia, and the other half from other organisms. *Ureaplasma urealyticum* probably accounts for a significant part of *C. trachomatis*-negative NGU, though it is not an infection routinely sought by current laboratory methods. *Trichomonas vaginalis*, candidiasis and covert urethral herpes infection should also be considered in the male patient with urethral discharge not responding to routine therapy.

In investigating Victor, what specimens would you collect and what would you test them for?

This patient should have the following specimens collected.
- Pus swab from urethra-slide and swab for micro culture & susceptibility (MCS).
- Pus swab from urethra-Polymerase chain reaction (PCR) for gonorrhoea and chlamydia.
- Blood - serology for HIV and syphilis +/- HBV (and repeat in 3 months).
When testing for gonorrhoea, a specimen should always be sent for culture. If there is a urethral discharge as in this patient, a urethral swab should be sent for MCS. Otherwise, urine should be sent for culture. The importance of continuing to culture in the era of highly sensitive PCR tests is to maintain surveillance for antibiotic sensitivities. Even in the asymptomatic patient presenting for a routine screen, a culture should be requested as well as the PCR test.

In the presence of a urethral discharge, PCR tests should be requested from a urethral swab and urine PCR is not necessary. In the absence of a discharge, PCR should be requested on urine. The best urine specimen for PCR testing is a first void urine (FVU) and not a mid stream urine (MSU). Ideally, this is collected after the patient has not voided for a period of four hours. There is almost no role for invasive and painful endo-urethral swabs in current practice.

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 definitely 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 local complications and transmission respectively. The most common complication of gonococcal urethritis is epididymitis, which has been reported to occur in about 20% of untreated patients.

**What treatment would you give?**

Victor should receive immediate "syndromic management". As stated above, despite differences in the typical presentations of GU and NGU, it is impossible to make an absolute distinction on clinical grounds. Moreover, co-infection is common, with many studies showing rates of isolation of *C. trachomatis* from the urethras of men with GU to be about 15-25%.

He should therefore be managed as a "urethritis syndrome". His empirical treatment should cover both GU and the common causative organisms of NGU (*C. trachomatis* and *U. urealyticum*). To date, the Northern Territory has been relatively free from penicillin resistant gonorrhoea. This situation is virtually unique across Australia and overseas and has allowed us to recommend single doses of amoxycillin (3g) and probenicid (1g) to treat genital gonorrhoea acquired in the NT. This should be given with a stat dose of azithromycin to treat NGU.

For more details, see the Clinic 34 protocol "Standard Treatment Protocols for Sexually Transmitted Infections" or the CARPA manual.

As with all STIs, comprehensive contact tracing should be undertaken.
SYPHILIS IN THE TOP END

Notification rates for syphilis are significantly higher in the NT, including the Top End, than the rest of Australia. Compared to the overall Australian rate of 9/100 000 (1998), the NT rate was 176/100 000, twenty times as high. The majority of this high rate of syphilis infection was found in Aboriginal people, with a notification rate in NT Aboriginal people of 583/100 000, or over sixty times the national rate. Though now almost a medical curiosity in many parts of Australia, syphilis is a common and serious disease in the Top End. A high index of suspicion for possible infection with syphilis, in both asymptomatic and symptomatic patients, should always be maintained.

AETIOLOGY AND PATHOGENESIS

The causative organism of syphilis is a spirochaete, Treponema pallidum subspecies pallidum. It is predominantly a sexually transmitted disease, with transmission occurring though direct contact with the highly infective lesions of primary and secondary syphilis. However, transmission may also be transplacental, blood borne, and via non sexual physical contact and auto inoculation. T. pallidum is very difficult to culture and serology is the mainstay of diagnosis.

Syphilis is a chronic systemic infection, manifesting as periods of active disease on a background of long periods of latency. The classical lesion of primary syphilis, the chancre, develops at the site of inoculation with the organism and occurs about 2-6 weeks after infection. However, well before the appearance of the genital ulcer, the organism disseminates to the draining lymph nodes, and also to distant organs and tissues. These include the CNS, cardiovascular system, eyes, skin and bone. The involvement of these distant sites manifests clinically as the secondary and tertiary stages of syphilis.

CLINICAL PICTURE

Risk factors. See STI overview.

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, 1-2 cm 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 variably manifests as a constitutional illness, including symptoms of malaise, headaches, fever and generalised lymphadenopathy. The classical rash of secondary syphilis is a generalised, maculopapular eruption, characteristically involving the palms and soles. Other typical features are condylomata lata, highly infectious warty lesions found in warm, moist areas like the genitals; mucous patches; and alopecia. 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.
Latency. The latent stage of syphilis represents ongoing but dormant infection with *T. pallidum*, typified by the patient with serological evidence of infection in the absence of any clinical features. Though untreated patients with latent syphilis (and tertiary syphilis) are essentially non infectious by sexual contact, congenital transmission is still possible years after initial infection. There is a distinction made between early and late latent infection (less than or more than two years after exposure respectively), which has implications for the extent of treatment required.

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 involvement, including aneurysm of the ascending aorta and aortic valve disease; neurosyphilis, including meningitis, general paresis and tabes dorsalis; and gummas, granulomatous tumours of skin, bone and viscera. Tertiary disease may occur years to decades after primary infection.

Syphilis in Pregnancy. Untreated maternal primary or latent syphilis results in foetal or perinatal death in up to 20% of pregnancies, preterm delivery in 20% and congenital syphilis in 40% of term deliveries. The features of early congenital syphilis are similar to secondary syphilis. Usually it presents 2-8 weeks after birth with failure to thrive, muco-cutaneous lesions (condylomata lata), generalized lymphadenopathy, nasal snuffles and skin rash. The onset of late congenital syphilis usually occurs at or near puberty. Well-known stigmata include nerve deafness, interstitial keratitis, Hutchinson’s teeth (Hutchison’s triad), rhagades around mouth, Clutton’s joint, osteitis & chondritis (saddle nose, frontal bossing, sabre tibia) and perforated palate.

Important investigations. Serology is the major diagnostic test in the investigation of syphilis. Interpretation of syphilis serology is notoriously difficult, the detailed discussion of which is beyond the scope of this guide. An excellent guide to this subject is listed below under ‘Management Guidelines’.

PRINCIPLES OF MANAGEMENT

While benzathine penicillin remains the mainstay of treatment of syphilis, treatment should be syndromic and empirical in most cases of genital ulcer disease (GUD). See the genital ulcer disease case study ‘A Case Study of a Private Problem’ in this guide for discussion of the differential diagnosis, investigations and management of genital ulcers. See also the Clinic 34 protocols and the CARPA manual. As with all STIs, comprehensive contact tracing should be undertaken for partners of patients infected with syphilis.

Syphilis is a notifiable condition to be reported by all CLINICIANS and LABORATORIES in the Northern Territory. Cases should be reported to the Centre for Disease Control in your district.
FURTHER INFORMATION

TELEPHONE ADVICE

| Medical Officer or clinic staff | Clinic 34-Darwin | 8922 8007 |
| Medical Officer or clinic staff | Clinic 34-Gove | 8967 0359 |
| Medical Officer or clinic staff | Clinic 34-Katherine | 8973 9049 |

MANAGEMENT GUIDELINES

| CARPA-Standard Treatment Manual | Central Australian Division of Primary Health Care | 8950 4800 |
| Or OATSIH Office NT | Nganampa Health Council Inc. | 8950 5435 |
| Women’s Business Manual | Centre for Disease Control-Darwin | 8922 8089 |

EDUCATIONAL RESOURCES

| Australian National Council on AIDS, Hepatitis C and Related Diseases | http://www.ancahrd.org/ | 8950 5435 |

FURTHER READING

- Tramont E. Syphilis in Adults: From Christopher Columbus to Sir Alexander Fleming to AIDS. CID. 1995; 21:p 1561-69
- Young H. Guidelines for serological testing for syphilis. Sex Transm Inf 2000; 76: p 403-405
A 28 year old woman, Cathy, reluctantly presents to you for a ‘check up’. Unsure about what is worrying her you offer a general ‘well woman’s check’ which includes a pap smear. 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 just a few days.

**What is her most likely diagnosis?**

The most likely diagnosis is primary syphilis. The classical chancre of primary syphilis typically presents as a single, painless, deep 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 Australia. Non tender bilateral inguinal lymphadenopathy often accompanies the primary chancre of syphilis infection. This differs from the tender a denopathy of HSV and the usual absence of lymph node (LN) involvement in donovanosis.

The classic textbook descriptions of cause specific genital ulcers, as above, 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. This emphasises the need to attempt microbiological diagnosis and offer syndromic treatment in all cases.

**What are the other possible causes of genital ulceration?**

The prevalence of these specific aetiologies of genital ulcer varies considerably according to the study population. HSV is by far the commonest ulcerative STI in Australia. However, syphilis notification rates are significantly higher in Aboriginal people in the Top End than in non-Aboriginal people. Similarly, donovanosis is much more common in Aboriginal people. The two exotic ulcerative diseases, chancroid and lymphogranuloma venereum, should be considered in a returned traveller from an endemic area like Africa or Asia.

**Table 6 Aetiology of genital ulcer disease**

<table>
<thead>
<tr>
<th>Sexually Transmitted</th>
<th>Non Sexually Transmitted</th>
</tr>
</thead>
<tbody>
<tr>
<td>Syphilis (both primary and secondary)</td>
<td>Malignancy</td>
</tr>
<tr>
<td>Treponema pallidum</td>
<td></td>
</tr>
<tr>
<td>Genital herpes</td>
<td>Severe Candidiasis with fissuring</td>
</tr>
<tr>
<td>Herpes Simplex Virus (HSV)</td>
<td>Fixed Drug Eruption</td>
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<tr>
<td>Donovanosis</td>
<td>Reiter’s Syndrome</td>
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<tr>
<td>Calymmatobacterium granulomatis</td>
<td>Behcet’s Syndrome</td>
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<tr>
<td>Chancroid (not found in Australia)</td>
<td>Trauma</td>
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<tr>
<td>Haemophilus ducreyi</td>
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<tr>
<td>Lymphogranuloma Venereum (not found in Australia)</td>
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<tr>
<td>Chlamydia trachomatis</td>
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<tr>
<td>Scabies (excoriated)</td>
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<tr>
<td>Sarcoptes scabiei</td>
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</table>

The prevalence of these specific aetiologies of genital ulcer varies considerably according to the study population. HSV is by far the commonest ulcerative STI in Australia. However, syphilis notification rates are significantly higher in Aboriginal people in the Top End than in non-Aboriginal people. Similarly, donovanosis is much more common in Aboriginal people. The two exotic ulcerative diseases, chancroid and lymphogranuloma venereum, should be considered in a returned traveller from an endemic area like Africa or Asia.

**In investigating Cathy, what specimens would you collect and what would you test them for?**

This patient should have the following specimens collected:

- Ulcer-slide and swab for microbiology, culture and sensitivity (MCS).
- Herpes Simplex virus (HSV) culture, Polymerase chain reaction (PCR) and if possible, HSV immunofluorescence (IF).
- Air dried impression slide or smear for donovanosis cytology.
• Endocervical swabs (ECS) if possible-MCS and PCR gonorrhoea and chlamydia.
• High Vaginal Swab (HVS)-slide and swab for MCS.
• Blood-serology for syphilis and HIV (and repeat in 3 months).

The genital ulcer should be swabbed for viral culture (and HSV antigen detection by IF through Westerns or RDH pathology). Though HSV ulcers are usually small (1-2 mm) and multiple, they may coalesce into larger, solitary lesions, as with this case. Specifically, the base of the lesion must be firmly swabbed to collect cells containing the intracellular virus. This is still worth doing five days into the illness, as with this case, though sensitivity of viral culture declines after 72 hours. PCR testing for HSV is increasingly available. Indications for HSV PCR testing include older lesions as this case, where the sensitivity of culture is likely to be reduced after 72 hours, and in atypical genital symptoms that are suggestive of HSV eg recurrent red patches or itch.

Cytology of an air dried impression smear remains the test of choice for donovanosis. Any slough should be cleaned away with saline and the slide pressed firmly to the base of the ulcer. Biopsy should be considered in suggestive cases which are smear negative, or if there is a concern about malignancy.

The presence of one probable STI, as with this case, demands that the practitioner consider the presence of other concurrent STI’s. 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, the cervix should be visualised and swabs collected for culture and PCR as above.

A reactive rapid plasma reagin test (RPR) is present in about 80% of patients with a syphilitic chancre, usually at a relatively low titre (1:16 or less). The treponema pallidum haemagglutination (TPHA) is positive in about 90%. Thus, while most patients with primary syphilis will have reactive serology, a negative result does not exclude the diagnosis. In patients with genital ulcers from secondary syphilis, the TPHA and RPR are almost always positive. 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.

Type specific herpes serology is also now available. However, its clinical applications are still uncertain. It has no role in diagnosis of primary HSV infection but depending on the circumstances of the sexual history, may be helpful in other situations. Such tests must be interpreted with caution and the possible implications made clear to the patient prior to testing.

**Would you offer Cathy presumptive treatment?**

Yes. She should definitely 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 would you give?**

Cathy should receive immediate “syndromic management”. As stated above, despite differences in the typical presentations of genital ulcers, it is sometimes impossible to make an absolute distinction on clinical grounds. She should therefore be managed as a “genital ulcer syndrome”. Her empirical treatment should cover both syphilis and donovanosis, and if clinically suspicious, HSV. She should therefore be given LA Bicillin 4 ml IMI and azithromycin 1 g stat, and reviewed in one week.

For more details, see the Clinic 34 protocol “Management Guidelines for Genital Ulcer Disease including donovanosis” or the CARPA manual. As with all STIs, comprehensive contact tracing should be undertaken.
CHLAMYDIA IN THE TOP END

Genital tract infection with chlamydia is common in the Top End and causes high rates of PID and infertility in women. Notification rates for chlamydia in the Northern Territory (408/100 000) were about five times higher than the overall Australian rate (88/100 000) in 1998, with higher rates in both Aboriginal and non-Aboriginal Territorians. As with other STIs, the vast majority of this excess burden of disease was Aboriginal people, with rates (882/100 000) up to ten times that of the national rate. The highest rates of infection were in women, and in the age group 15-24 years.

AETIOLOGY AND PATHOGENESIS

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

Specific *C. trachomatis* serovars are responsible for a variety of human diseases. Serovars A-C cause trachoma, L1-L3 cause LGV (lymphogranuloma venereum, an STI not endemic to Australia) 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 STI overview.

Symptoms and Signs. The clinical manifestations of infection with *Chlamydia trachomatis* have close parallels with those from *N. gonorrhoeae*, including high rates of asymptomatic infection, predominantly genital tract symptoms, pharyngitis and 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 low index of suspicion for diagnosis.

MALES After a 7-21 day incubation period, *C. trachomatis* infection in men presents typically as dysuria and a scant to moderate white or clear urethral discharge. Examination is usually otherwise unremarkable. Proctitis may occur in homosexual men and is commonly asymptomatic. Epididymitis is a well recognised local complication of *C. trachomatis* in men; the role of this infection in nonbacterial prostatitis is inconclusive. Reiter’s Syndrome (urethritis, conjunctivitis, arthritis and mucocutaneous lesions) is a systemic manifestation of *C. trachomatis* genital infection.

FEMALES In 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 a “friable” cervix, hypertrophic ectopy and mucopurulent cervicitis. 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 complication of *C. trachomatis* in women is salpingitis, or pelvic inflammatory disease (PID). Chlamydial PID is frequently subclinical, though may present with any combination of pelvic pain, fever, dyspareunia or menstrual irregularities. Cervical excitation and adnexal tenderness are typical features on clinical examination that 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.

GENERAL Culture negative endocarditis and meningoencephalitis due to *C. trachomatis* have been reported.
DIFFERENTIAL DIAGNOSIS

See the urethritis case study ‘Case Study of A Common Syndrome’ for discussion of the differential diagnosis of urethritis in men. In women, the presence of vaginal discharge could represent cervicitis, upper genital tract infection (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

For discussion of investigation and management see the case studies of pelvic pain and urethritis in this manual.

See also the Clinic 34 protocols, the CARPA manual and Women’s Business Manual.

PID is notoriously difficult to diagnose and a high degree of clinical suspicion must be maintained at all times. 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. Always send a specimen for culture. As with all STIs, comprehensive contact tracing should be undertaken for partners of patients infected with chlamydia.

Chlamydia infection is a notifiable condition to be reported by all LABORATORIES in the Northern Territory.

FURTHER INFORMATION

TELEPHONE ADVICE

Medical Officer or clinic staff
Medical Officer or clinic staff
Medical Officer or clinic staff
8922 8007
8987 0359
8973 9049

MANAGEMENT GUIDELINES

Recommended Testing Guidelines for Genital Tract Infections. October 2000
AIDS/STD Program-Darwin
8922 8007

Standard Treatment Protocols for Sexually Transmitted Infections. October 2000
AIDS/STD Program-Darwin
8922 8007

Standard Treatment Protocols for Sexually Transmitted Infections (Pelvic Inflammatory Disease). October 2000
AIDS/STD Program-Darwin
8922 8007

CARPA-Standard Treatment Manual
Central Australian Division of Primary Health Care
8950 4800

Women’s Business Manual
Nganampa Health Council Inc.
8950 5435

Communicable Disease Surveillance in the NT-Guidelines for the Reporting of Notifiable Conditions
Centre for Disease Control-Darwin
8922 8089

STD control in Remote Aboriginal Communities A Manual for Clinic Workers
8946 3481
Or OATSIH Office NT

EDUCATIONAL RESOURCES

Fact Sheet-Chlamydia
Clinic 34-Darwin
8922 8007

FURTHER READING

More Than Just a Pain-
Case Study of Lower Abdominal Pain

Serena is a 34 year old local Aboriginal woman who presents to the community clinic where you are working. She has had lower abdominal pain for four days and now presents to you doubled up in distress with the pain.

Serena has not noticed any vaginal discharge or dysuria, or symptoms of pregnancy. She is unsure about the date of her last period but thinks it is now about three to four weeks late. Her periods are usually heavy, irregular, last for 7 days and are painful for the duration. She has been with her current partner for about a year and a half and has not used any contraception in that time. She denies any partners outside this relationship. Her bowel habit is regular apart from a small amount of diarrhoea yesterday, with no blood in the stool. You note from her history she has a child aged 6 years and another that died five years ago in early infancy. She has had several urinary tract infections in the past, but no prior abdominal surgery.

The health worker tells you Serena’s partner has been seen with another woman recently. She also tells you Serena is very keen to have more children and is wondering why she has not fallen pregnant.

On examination Serena is febrile, 37.80C, HR 90, BP 90/60. She is tender over the lower abdomen and 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 pelvic digital examination she is tender in the right fornix (no mass palpable) and is acutely tender on rocking the cervix.

What are the important conditions that should be diagnosed or excluded early?

The important condition that needs exclusion in a woman not using contraception and with an uncertain last menstrual period (LMP) is ectopic pregnancy. Less urgent but still important are pelvic inflammatory disease and appendicitis.

What are the differential diagnoses in this woman?

The differential diagnoses in this woman include:

• Ectopic pregnancy.
• Pelvic inflammatory disease.
• Appendicitis-early.
• Severe urinary tract infection.
• Other gynae pathology less likely - septic abortion, ovarian cyst or torsion, fibroid infarction.
• Other bowel pathology less likely - acute infective colitis, diverticulitis.

What are the immediate investigations you would do to assist diagnosis?

Immediate investigations include:

• Urine tests for (1) pregnancy and (2) dipstick urinalysis +/- mid stream urine (MSU) for microscopy culture & Sensitivities (MCS).
• Either (in order of preference) (1) an endocervical swab (ECS) for chlamydia and gonorrhoea Polymerase chain reaction (PCR) and high vaginal swab (HVS) for MCS or, (2) a tampon test (T-Test), low vaginal swab (LVS), or self administered swab for gonorrhoeae and chlamydia PCR and MCS or (3) first void urine (FVU) for gonorrhoea and chlamydia PCR and MCS.
• Blood for acute phase reactants (C-Reactive protein [CRP]/ Erythrocyte sedimentation rate [ESR]), FBC and film.
• Blood for STI screen, including syphilis and hepatitis B serology, and HIV serology if appropriate with counselling.
undertaken and consent obtained. This may be left for follow-up if Serena is acutely unwell.

**Given that a urine pregnancy test is positive, what is your management?**

If the pregnancy test is positive, manage as for 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.

**If the urine dipstick and pregnancy test were negative, what would be your management?**

If the urine dipstick and pregnancy test is negative, then management is less urgent unless an acute abdomen develops. A little time will sort out whether this is early appendicitis. However, in a 34 year old woman with a high risk of severe PID (with risk factors, febrile, pelvic pain and cervical excitation) who is very keen to have children it is very important to discuss hospital management of PID. This involves IV antibiotics and bed rest until there is substantial clinical improvement or the diagnosis is disproved. For severe infection, the following regimen is recommended:

- Metronidazole 500mg IV 12 hourly (anaerobe cover) and
- Doxycycline 100mg orally 12 hourly (chlamydia cover).
- With either cefotaxime 1g IV 8 hourly or ceftriaxone 1g IV daily (gonorrhoea).

On discharge the antibiotics are altered to oral as below to give a 14 day total of antibiotic treatment.

If a woman has more than two risk factors for infertility, then IV therapy (even if it is only first dose) should be considered. Risk factors for infertility include: young age, prior STI infection, prior PID, late presentation (>3 days of symptoms), high likelihood of no/poor follow-up.

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

- Oral metronidazole or tinidazole may be substituted for IV metronidazole and continued for 14 days with oral doxycycline.
- A stat dose of amoxycillin and probenecid to cover gonorrhoea is included.

If the woman has chosen to stay in the community and start oral antibiotics, and there is no improvement in one to three days, pelvic ultrasound and admission for IV antibiotics as above should be considered.

Longer term issues include partner notification, screening and treatment (do not forget to investigate any previous contacts he/she may have had), and blood STIs, including HIV screening if not already performed. Discussion of safer sex practices and condom use in the setting of a relationship, and where the woman wishes to conceive, is clearly problematic. STI issues should be at least discussed, if possible with the partner also even if this needs to be on a separate occasion, and are easier where fertility is not such an important issue. Periconceptual folate and investigation of potential infertility may also require addressing once the acute illness is improved.
Donovanosis

DONOVANOSIS IN THE TOP END

Donovanosis is an uncommon infection, with only 6 cases reported in the NT in 2000 and 13 cases (8 females, 5 males) in 2001. It is a disease almost exclusively of Aboriginal people. Due to the introduction of azithromycin as treatment and dedicated donovanosis management programs, notification rates are steadily declining. However, it is a disease associated with serious complications and social stigma, and ongoing awareness of this infection is very important.

AETIOLOGY AND PATHOGENESIS

The causative organism of donovanosis is a bacteria named *Calymmatobacterium granulomatis*. Cells infected with this organism demonstrate characteristic Donovan bodies when appropriately stained. *C. granulomatis* is extremely difficult to culture.

Donovanosis is a sexually transmitted infection (STI) with many unusual epidemiological features, including a low incidence and transmission rate and differences in race and sex distribution.

CLINICAL PICTURE

Risk factors. See STI overview.

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, with some case reports of associated genital tract carcinoma. Very rarely, the infection may disseminate.

Investigations. Cytohistological examination of impression smears or biopsy specimens remains the mainstay method of diagnosis for donovanosis. Even so, these tests have a poor sensitivity of only 60-70%. There is current research into polymerase chain reaction diagnostic methods.

PRINCIPLES OF MANAGEMENT

For discussion of the differential diagnosis, investigations and treatment, see the genital ulcer disease case study in this manual, the Clinic 34 protocols or the CARPA manual.

Treatment should be syndromic and empirical in most cases of genital ulcer disease (GUD). Donovanosis is a chronic condition that requires close follow up until complete healing is observed. As with all STIs, other STI tests (including HIV) and contact tracing of partners of patients infected with donovanosis should be done.

Donovanosis is a notifiable condition to be reported by all CLINICIANS and LABORATORIES in the Northern Territory. Cases should be reported to the Centre for Disease Control in your district.
FURTHER INFORMATION

TELEPHONE ADVICE

| Medical Officer or clinic staff | Clinic 34-Darwin | 8922 8007 |
| Medical Officer or clinic staff | Clinic 34-Gove   | 8987 0359 |
| Medical Officer or clinic staff | Clinic 34-Katherine | 8973 9049 |

MANAGEMENT GUIDELINES

| CARPA-Standard Treatment Manual | Central Australian Division of Primary Health Care | 8950 4800 |
| Communicable Disease Surveillance in the NT-Guidelines for the Reporting of Notifiable Conditions | Centre for Disease Control-Darwin | 8922 8089 |

EDUCATIONAL RESOURCES

| CD-ROM-Donovanosis-An overview | Clinic 34-Darwin | 8922 8007 |

FURTHER READING

Trichomoniasis

TRICHOMONIASIS IN THE TOP END

*Trichomonas vaginalis* is a protozoal parasite of the genitourinary tract in humans. It is transmitted almost exclusively by sexual intercourse. It is very common in the Top End, particularly in the Aboriginal population.

The vaginal epithelium is the chief site of infection with *T. 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 gonorrhoeae and chlamydia; as well as many non infective causes. Similarly, dysuria could 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 and rapidly, probably a result of anti-trichomonal host immune factors. See the urethritis case study ‘Case Study of A Common Syndrome’ for discussion of the differential diagnosis of urethritis in men.

In addition, a number of significant complications of trichomonal infection have been reported. These include an association between trichomoniasis and adverse pregnancy outcomes, including low birth weight infants and preterm delivery. There is also some evidence that trichomoniasis-associated mucosal inflammation increases the risk of transmission of HIV. However, at present the results of intervention studies do not show benefit from treatment.

For the investigation of trichomoniasis, see Clinic 34 Protocol "Recommended Testing Guidelines for Genital Tract Infections". Also see the urethritis case study ‘Case Study of A Common Syndrome’ for discussion of the investigation of urethritis as well as the CARPA manual and Women’s Business Manual. Remember always to send a specimen for culture.

For treatment, see Clinic 34 protocols "Standard treatment Protocols for Sexually Transmitted Infections". Also see the Urethritis Case Study for discussion of the treatment of urethritis and the CARPA manuals. Treatment should be syndromic and empirical.

As with all STI’s, comprehensive contact tracing should be undertaken for partners of patients infected with trichomonas.

*Trichomoniasis* is a notifiable condition to be reported by all LABORATORIES in the Northern Territory.
FURTHER INFORMATION

TELEPHONE ADVICE
Medical Officer or clinic staff
Medical Officer or clinic staff
Medical Officer or clinic staff

Clinic 34-Darwin
Clinic 34-Gove
Clinic 34-Katherine

8922 8007
8987 0359
8973 9049

MANAGEMENT GUIDELINES
Recommended Testing Guidelines for Genital Tract Infections. October 2000
Standard Treatment Protocols for Sexually Transmitted Infections. October 2000
CARPA-Standard Treatment Manual
Women's Business Manual
Communicable Disease Surveillance in the NT-Guidelines for the reporting of Notifiable Conditions
STD Control in Remote Aboriginal communities - A Manual for Clinic Workers

AIDS/STD Program-Darwin
AIDS/STD Program-Darwin
Central Australian Division of Primary Health Care
Nanganampa Health Council Inc.
Centre for Disease Control-Darwin

8922 8007
8922 8007
8950 4800
8950 5435
8922 8089
8946 3481
8922 8007

EDUCATIONAL RESOURCES
Fact Sheet–Trichomoniais

Clinic 34-Darwin

8922 8007

FURTHER READING
• Cotch MF. Trichomonas vaginalis associated with low birth weight and preterm delivery. Sex Trans Dis 1997;24: p353-60
• Laga M et al. The interrelationship of sexually transmitted diseases and HIV infection: Implications for the control of both epidemics in Africa. AIDS 1991;5 Suppl 1: S55-63
HIV IN THE TOP END

Human Immunodeficiency Virus (HIV) was first identified in Paris in 1983. It is a retrovirus, containing a unique enzyme, reverse transcriptase, which copies viral ribonucleic acid into host deoxyribonucleic acid. It causes chronic immune deficiency, leading to opportunistic infection and neoplasia, and is the cause of AIDS, or the Acquired Immunodeficiency Syndrome. AIDS is a clinical diagnosis made when certain clinical conditions are present.

HIV has been a notifiable condition in the NT since routine serological testing became available in Australia in 1985. Since that time to the end of 2001 there have been 122 cases of HIV infection notified in NT residents and 13 in non-residents for a total of 135 (or about 9 cases each year in total). The cumulative incidence of HIV in the NT (60/100,000) is similar to that of comparable geographic regions within Australia. However, it is considerably less than the national cumulative incidence (105/100,000), which includes notifications for Sydney and Melbourne, where rates are much higher than elsewhere in the country.

HIV notifications in the NT are predominantly in homosexually active men, who are non-Aboriginal, aged between 30 and 39 years and who live in Darwin. 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 people in the NT. However, since that time, Aboriginal notifications have made up 25% of the total.

Compared with other states and nationally, the rates of STIs in the NT are disproportionately high. As discussed elsewhere, the Aboriginal population is over-represented in these notification rates. There is good evidence that STIs facilitate the transmission of HIV.

Making the diagnosis of an STI in an individual demands that the practitioner considers the presence of concurrent STIs, including HIV. There is now a legal precedent for this in Australia, where a GP in NSW was found negligent for not diagnosing HIV infection in a patient with acute Hepatitis B. HIV tests should therefore be offered to all people with an STI, including discussion of the three month window period.

From a community point of view, practitioners in the NT need to remain vigilant to the possibility of a local HIV epidemic. Early detection and diagnosis has implications for both individual and public health.

For a brief discussion of clinical features and testing for HIV (including pre-test information) see the CARPA manual. Management of the HIV positive patient should involve a multidisciplinary health care team approach, usually including a shared care partnership between GP and a specialist physician (usually through the local Clinic 34). The GPs role is varied, and may include primary care, monitoring of immune function, coordination of care with other team members, provision of information, patient and family support and advocacy.

HIV is a notifiable condition to be reported by all LABORATORIES in the Northern Territory.
FURTHER INFORMATION

TELEPHONE ADVICE

Medical Officer or clinic staff
Clinic 34-Darwin 8922 8007
Clinic 34-Gove 8987 0359
Clinic 34-Katherine 8973 9049

MANAGEMENT GUIDELINES

CARPA-Standard Treatment Manual
Central Australian Division of Primary Health Care 8950 4800

Communicable Disease Surveillance in the NT-Guidelines for the Reporting of Notifiable Conditions
Centre for Disease Control-Darwin 8922 8089
Or

STD control in remote Aboriginal Communities
A Manual for Clinic Workers
Or
OATSIH Office NT

EDUCATIONAL RESOURCES

What Now? Some Questions on HIV Answered
Clinic 34-Darwin 8922 8007
HIV tests and Treatments
Clinic 34-Darwin 8922 8007
Northern Territory AIDS Council Inc.
http://www.octa4.net.au/ntac/
Australian Federation of AIDS Organisations
http://www.aftao.org.au
Where to Get Clean Injecting Equipment in the Northern Territory
Clinic 34-Darwin 8922 8007
HIV/HCV Viral Hepatitis
Australian Society for HIV Medicine (02) 9368 2700
A Guide for Primary Care
Health for Injectors in the NT
Northern Territory AIDS Council Inc. 8941 1711

FURTHER READING

**Snakebites in the Top End**

While snakebite numbers are decreasing in temperate Australia, they remain an important cause of morbidity in tropical regions. For example, there are more snakebite admissions in tropical than in sub-tropical coastal Queensland, despite this region having a much lower population. In the Top End the annual incidence in children during the 1990’s was 18.3 per 100,000. Deaths from snakebite in tropical Australia are now rare, with no confirmed death in the Northern Territory for over 10 years despite the highest incidence of bites within Australia.

In temperate Australia snakebites occur most frequently in January and are very unusual in winter months, reflecting snake activity and hibernation. 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.

**Aetiology and Pathogenesis**

Venomous snakes of the region

All potentially lethal terrestrial snakes in Australia belong to the family Elapidae. Of note, tiger snakes (Notechis spp.) are absent from tropical regions. In addition, a number of potentially lethal sea snakes (family Hydrophiidae) are present in the surrounding seas and occasionally inland up tidal rivers. Table 7 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 the table 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 (*D. atra* and *D. papuensis*) account for more confirmed elapid bites than all other species except the western brown snake and mulga snake, but life-threatening envenoming has never been documented. Whip snakes 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 collettii*) in north Queensland and *Pseudonaja guttata*, *Pseudonaja ingrami* and *Pseudonaja modesta* in various locations across northern and central Australia.

<table>
<thead>
<tr>
<th>Northern Territory and Tropical Western Australia</th>
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<tr>
<td><em>Pseudonaja nuchalis</em></td>
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<tr>
<td>Western brown snake (Gwardar)</td>
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<td>Mulga</td>
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<td><em>Acanthophis spp.</em></td>
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<td>Death Adder</td>
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<td><em>Oxyuranus scutellatus</em></td>
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<td>Taipan</td>
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<tr>
<td><em>Tropical Queensland</em></td>
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<td><em>Pseudonaja textilis</em></td>
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<td>Common (Eastern) brown snake</td>
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<td><em>Oxyuranus scutellatus</em></td>
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</tr>
<tr>
<td>Mulga</td>
</tr>
<tr>
<td><em>Acanthophis spp.</em></td>
</tr>
<tr>
<td>Death Adder</td>
</tr>
<tr>
<td><em>Tropidechis carinatus</em></td>
</tr>
<tr>
<td>Rough-scaled snake</td>
</tr>
<tr>
<td><em>Rhinoplocephalus nigrescens</em></td>
</tr>
<tr>
<td>Eastern small-eyed snake</td>
</tr>
</tbody>
</table>

Table 7 The distribution of potentially lethal terrestrial snakes in tropical Australia in decreasing order of bites seen in each region.

**Pathogenesis**

Snake venoms are a diverse and complex mixture of proteins. Elapidae venoms usually cause only minor local damage at the bite site, with systemic effects predominant. The mulga snake (*Pseudechis australis*) can be the exception, with occasionally severe local damage, especially if tight first-aid has been applied around or above the bite site. Each of the
snake species has a fairly consistent combination of venom components which results in a usually consistent clinical syndrome of envenoming. The major venom components for Australasian elapids are these which cause:

(i) **Neuromuscular paralysis (neurotoxins),** either by pre-synaptic action (affecting acetylcholine processing and release from the nerve endings), or by post-synaptic action (blocking acetylcholine receptors on the muscle fibre).

(ii) **Haemostatic abnormalities.** Most important clinically is (a) **procoagulant action** with prothrombin activators leading to fibrinogen depletion, fibrinogen degradation products and incoagulable blood. Also sometimes occurring are (b) **anticoagulant action,** (c) **haemorrhagin** directly affecting blood vessel walls (less common than in Viperidae) and (d) **thrombocytopenia** or abnormal platelet function. Haemolysins causing anaemia and hemoglobinuria may also be present.

(iii) **Rhabdomyolysis (myotoxins)** with muscle breakdown leading to myoglobinuria. In the taipan the myotoxin is actually one of the three subunits (alpha sub-unit) of taipoxin, the principal neurotoxin.

(iv) **Nephrotoxicity,** which is less common than with Viperidae. For Australasian Elapidae it is usually subsequent to myoglobinuria from severe rhabdomyolysis. It is uncertain if haemoglobinuria alone, or other haemostatic abnormalities or uncharacterised nephrotoxins cause renal failure in Australasian snake bites.

(v) **Early transient collapse (hypotension),** which may occur up to 30 minutes after the bite and often with 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.)

The relative contributions of the various potential pathogenetic mechanisms for early hypotension and collapse remain to be determined and will be likely to vary for different snakes from different regions of the world. However early collapse after a snakebite in Australia does correlate strongly with those snakes with potent procoagulant venoms. Direct myocardial depression by venom components is also possible.

**CLINICAL PICTURE**

Figure 43 summarises the clinical manifestations of envenoming from the major Australasian elapids. Of note is that 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. Such a scenario may be life-threatening, especially in a sleeping child not on neurological observations including ptosis.

<table>
<thead>
<tr>
<th>Bite site signs</th>
<th>Regional Lymphadenitis</th>
<th>Non - specific systematic features</th>
<th>Coagulopathy</th>
<th>Neurotoxicity</th>
<th>Myotoxicity</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>(headache, nausea, vomiting, abdominal pain)</td>
<td></td>
<td>Neurotoxicity</td>
<td>Myotoxicity</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Other=Other=</td>
<td></td>
<td></td>
<td>Other=</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Renal failure, other haematostatic effects such as haemolytic, “haemorrhagic” and “anti-platelet” venom actions.</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Figure 43 Australasian elapid envenoming

**The time course of envenoming:**

Table 8 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,
Snakebites

spitting blood, macroscopic or microscopic haematuria or prolonged glass tube whole blood clotting time) usually begin from 30 to 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 around 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, being observed in hospital, for 24 hours after the bite.

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.

**PRINCIPLES OF MANAGEMENT**

Refer to the detailed Acute Care Guidelines from the Top End Division of General Practice. Pressure immobilization of the limb and strict immobilization 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. 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. Seek expert advice, and obtain detailed management guidelines before administering antivenom.

<table>
<thead>
<tr>
<th>Early Collapse</th>
<th>Local swelling</th>
<th>Tender regional lymph nodes</th>
<th>Non-specific “systemic features”</th>
<th>Myotoxicity</th>
<th>Coagulopathy</th>
<th>Neurotoxicity</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Brown snakes</strong></td>
<td>++</td>
<td>+/-</td>
<td>+/-</td>
<td>+/-</td>
<td>-</td>
<td>+++^2</td>
</tr>
<tr>
<td><strong>Mulga snake</strong></td>
<td>-</td>
<td>++</td>
<td>++</td>
<td>++</td>
<td>++</td>
<td>+^3</td>
</tr>
<tr>
<td><strong>Death adder</strong></td>
<td>-</td>
<td>+/-</td>
<td>+/-</td>
<td>+/-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td><strong>Taipans</strong></td>
<td>+</td>
<td>+/-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>++^2</td>
</tr>
<tr>
<td><strong>Rough-scaled snake</strong></td>
<td>+</td>
<td>+/-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td><strong>Eastern small-eyed snake</strong></td>
<td>-</td>
<td>+/-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+/-</td>
</tr>
<tr>
<td><strong>Whip snakes</strong>^5</td>
<td>-</td>
<td>+</td>
<td>+/-</td>
<td>+/-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td><strong>Tiger Snakes</strong>^7</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>++</td>
<td>+^2</td>
</tr>
</tbody>
</table>

Table 8 Clinical syndromes of envenoming by the major Australasian snakes

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

Prof Bart Currie  
Emergency Specialists  
Royal Darwin Hospital  
Emergency Department-Royal Darwin Hospital  
8922 8888  
8922 8888

MANAGEMENT GUIDELINES

Acute Care Management Guidelines for the Resident Remote GP  
Top End Division Of General Practice  
8982 1000

CSL, Melbourne.  
1800 642 865

EDUCATIONAL RESOURCES

Clinical Toxinology Resource  
http://www.toxinology.com/

FURTHER READING


Acknowledgement The information, tables and figures presented in this section are reproduced from Prof Bart Currie’s paper Snakebite in tropical Australia, Papua New Guinea and Irian Jaya. See above for the full reference.
CIGUATERA IN THE TOP END

Ciguatera is a syndrome that results from eating tropical reef fish that have been contaminated with an algal toxin. It occurs throughout the tropical and subtropical waters of the Pacific, Indian and Atlantic Oceans and surrounding seas. Outbreaks and sporadic cases occur in residents and visitors of these areas and also in unsuspecting persons far from tropical waters who have eaten contaminated fish transported from these areas. The Gove Peninsula is the only known high-risk region for ciguatera poisoning in the NT. The most recent published outbreak occurred in 1995. Twenty people were affected, of whom six required hospitalisation. All had eaten the same fish.

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, Filefish, Moray Eel, Coral Trout, Coral Cod, Red Emperor, Parrot fish, Sweetlip, 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 signs and symptoms including:

- Gastrointestinal (vomiting, abdominal cramps, diarrhoea)
- Neurological (temperature perception reversal, tingling and numbness around the lips, hands and feet, dental pain, muscle weakness and headaches)
- Dermal (severe pruritis, skin rash)
- Cardiovascular (bradycardia or hypotension)
- Musculoskeletal (joint pain, muscle pain, neck stiffness, difficulties walking)
- Psychological (tiredness, depression, short term memory loss)
- Respiratory (dyspnoea, sore/dry throat or respiratory depression)

In severe cases death may occur from respiratory paralysis. Previous exposure does not confer immunity and may increase sensitivity to the toxin.

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

Prevention. Avoid eating fish species that are locally implicated and never eat the head, viscera or roe of reef fish. Treat all large warm water carnivorous reef fish with suspicion and eat no more than 250 grams of flesh at a first sitting.

Notify CDC of cases to help avoid further cases and assist in the collection of local information about the number of incidents and the locations of ciguatera poisoning. Ciguatera reporting forms can be obtained from CDC, Darwin on 8922 8044.

Treatment is largely supportive and symptomatic. Mannitol may relieve symptoms of ciguatera if administered within 24 hours of the onset of symptoms. Specialist advice should be sought about this. The duration of the acute illness ranges from 1 to 8 days however neurological symptoms can last for months. Neurological symptoms may be made worse by alcohol consumption, exercise and changes in dietary behaviour such as restricted diet or high-protein diet.
FURTHER INFORMATION

TELEPHONE ADVICE

Specialist Physicians
Medical or Clinical Nurse Consultant
Royal Darwin Hospital
Centre for Disease Control-Gove 8922 8888
8987 0359

MANAGEMENT GUIDELINES

Pamphlet-Ciguatera (fish poisoning)

Pamphlet-Ciguatera Poisoning-Information and Treatment Queensland Fisheries and Management Association, Queensland State Health Department, Queensland Department of Primary Industries, undated.


FURTHER READING

BOX JELLYFISH IN THE TOP END

The world’s most venomous box jellyfish species, *Chironex fleckeri*, seasonally inhabits coastal waters of the Top End. *C. fleckeri* has caused more than 60 deaths over the last 100 years in Australia. Those greatest at risk are children due to their small body mass and the last 10 fatalities in the NT have been 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. There are an estimated five stings each year occurring from June to September, the time considered to be the safest period for swimming.

AETIOLOGY AND PATHOGENESIS

*C. fleckeri* has a rounded box shape transparent bell that can measure up to 25 cm 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 cells) 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 to cardiotoxicity and possibly due to the central effect on the respiratory drive. In addition, the venom is dermo-necrotic and may result in scarring.

CLINICAL PICTURE

Risk factors. Swimming in coastal waters, particularly during the wet season. Young children are at greater risk of envenomation.

Symptoms and signs. Mild to moderate stings are much more common than ones causing severe envenomation. Symptoms and signs of envenomation include:

- Severe localised pain
- Erythematous weals on the skin where the tentacles have made contact.
- Confusion, agitation, unconsciousness
- Arrhythmias
- Collapse with respiratory failure and/or cardiac arrest.

Investigations. A baseline ECG is useful in all but minor stings. Nematocysts can be collected by putting 4-8 cm of ordinary transparent sticky tape over the sting site and then removing and taping it onto a glass slide. The Menzies School of Health Research offers microscopic identification of nematocysts. 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. Have vinegar available at all times when swimming in the Top End.

Figure 48 *Chironex fleckeri* jellyfish at Nightcliff beach

Figure 49 *Chironex fleckeri* stings
Management depends on the severity of the sting and includes washing with liberal amounts of vinegar to prevent further firing of the nematocysts, physically removing tentacles from the skin, and pain relief as required. Severe cases may require cardiopulmonary resuscitation, intravenous antivenom, and immediate transfer to hospital. Prevention of infection is important to prevent scarring. Detailed management protocols are contained in the guidelines listed below and in the article Box-jellyfish—An update from the Northern Territory and the NT Chironex fleckeri treatment protocol.

**Figure 50 Chironex fleckeri stings**

**Figure 51 Chironex fleckeri nematocysts under microscope (x200 magnification) from sticky tape test**

**FURTHER INFORMATION**

**TELEPHONE ADVICE**

| On call DMO-for remote areas | Royal Darwin Hospital | 8922 8888 |
| On call DMO-for remote areas | Katherine District Hospital | 8973 9211 |
| On call DMO-for remote areas | Gove District Hospital | 8987 0211 |
| Specialist Physician or Medical Registrar or Emergency Medicine Specialist | Royal Darwin Hospital only | 8922 8888 |

**MANAGEMENT GUIDELINES**

| Acute Care Management Guidelines for the Resident Remote GP | Top End Division Of General Practice | 8982 1000 |
| CARPA- Standard Treatment Manual | Central Australian Division Of Primary Health Care | 8950 4800 |

**EDUCATIONAL RESOURCES**

| Box Jellyfish Folder | Royal Darwin Hospital library | 8922 8961 |
| | Gove District Hospital library | 8987 0262 |
| | Katherine District Hospital library | 8973 9036 |

**FURTHER READING**

- O'Reilly GM, Isbister GK, Lawrie PM, Treston GT, Currie BJ. Prospective study of jellyfish stings from tropical Australia, including the major box jellyfish Chironex fleckeri. *Med./Aust.* 2001;175:p 652-55.
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 and other locations where this species has rarely been captured. In 2002 the first two documented fatalities 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 presynaptic 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 tropical waters of North Queensland, Northern Territory and Western Australia.

**Symptoms and signs** include back, chest, and abdominal pain, often with cramps and occurring in waves lasting a few minutes. Restlessness, anxiety, sweating and piloerection are common and may be severe. Tachycardia and marked hypertension may occur and the toxin may also have a direct myocardial depressant action. Pulmonary oedema from cardiac decompensation can occur.

**PRINCIPLES OF MANAGEMENT**

Immediate dousing with vinegar is recommended to prevent further envenomation from nematocysts. Blood pressure must be closely monitored. Large doses of narcotics are sometimes required to control the pain.
**FURTHER INFORMATION**

**TELEPHONE ADVICE**

| DMO on call-for remote areas | Royal Darwin Hospital | 8922 8888 |
| DMO on call-for remote areas | Katherine District Hospital | 8973 9211 |
| DMO on call-for remote areas | Gove District Hospital | 8987 0211 |
| Specialist Physician or Medical Registrar | Royal Darwin Hospital only | 8922 8888 |
| or Emergency medicine specialist | | |

**MANAGEMENT GUIDELINES**

Acute Care Management Guidelines for the Resident Remote GP

Top End Division Of General Practice 8982 1000

**FURTHER READING**

An Overview of Kava Use

**KAVA USE IN THE TOP END**

Kava is an intoxicating and sedating drink prepared from crushed roots of the pepper plant (*Piper methysticum*). It is widely used in south Pacific countries on ceremonial occasions and in secular drinking. 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.

The experience with kava in the NT is relatively short. There have been two community studies of associated health effects conducted by the Menzies School of Health Research. In both studies kava users frequently showed the following:

- A characteristic dermopathy. This consists of a generalised flat, fine, gray dermatitis with no active border. It is sometimes described as ‘black skin’ or ‘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 liver function tests, 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.

The measured health effects appear to occur more frequently in those estimated to be using more than 400g/week of kava powder. 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 NT Government introduced the Kava licensing regime. 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.

FURTHER INFORMATION

**TELEPHONE ADVICE**

Menzies School of Health Research
Alcohol and Other Drugs

**FURTHER READING**

PETROL SNIFFING IN THE TOP END

For over thirty years petrol sniffing has been relatively common among Aboriginal youths in some remote areas of Australia. The toxicity and social effects of petrol sniffing are substantial. Between January 1991 and 1994, for example, 70 individuals were admitted to RDH for petrol-sniffing related illnesses, seven of whom died. It has been associated with juvenile crime particularly property damage, poor school performance, and unsafe sexual practices. Petrol sniffing has appeared to be an intractable problem and continues to attract public attention and comment.

There are two major components in petrol that cause toxicity. These are the volatile hydrocarbons, particularly toluene, and alkyllead additives (in ‘leaded’ petrol only). The main lead additive, tetraethyllead, 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.

The immediate effect of petrol inhalation is an acute encephalopathy, which may manifest as restlessness and excitation, impaired consciousness, delirium, fitting, acute psychosis, or death, depending upon levels of exposure. The onset occurs within minutes and may last for 5-6 hours. An approach to managing a patient in this situation is described in the CARPA manual. Many of the acute effects of petrol sniffing have been attributed to the effects of the volatile hydrocarbons but some may also be associated with acute exposure to alkyllead.

The chronic effects of petrol sniffing include a variety of neurological abnormalities: behavioural change, cognitive impairment, movement disorders such as tremor, chorea and ataxia, nystagmus, pyramidal signs and convulsions. It is thought that lead additives rather than hydrocarbons are the major contributors to longer-term toxicity.

The use of unleaded petrol is a useful harm-minimisation strategy and the use of aviation gasoline has helped to eliminate the practice in some communities. There have been no admissions to RDH for petrol sniffing encephalopathy for more than five years. This is attributed to removal of access to leaded petrol. Lessons may be drawn from strategies implemented in two regions of Arnhemland. In one community, petrol sniffing ceased and petrol related crime fell markedly after the community introduced an intervention strategy involving employment and training programs, management of alcohol issues, sport and recreation programs, local administration of court community service orders along with the substitution of aviation gasoline in the fuel supply. Blood lead levels began to fall in those with a history of petrol sniffing and there were signs of recovery of some neurocognitive functions in former sniffers. The forerunner to this remarkable success was in a nearby community where traditional social mechanisms were used to curb petrol sniffing, an initiative of the local Aboriginal people.

While aviation gasoline was a key element in eliminating petrol sniffing, its mixed success as a single intervention elsewhere indicates the importance of widespread community resolve against petrol sniffing and the development of coordinated employment strategies in successfully eliminating the practice and reducing associated social disruption.
FURTHER INFORMATION

TELEPHONE ADVICE

Menzies School of Health Research
Alcohol and Other Drugs
CARPA-Standard Treatment Manual

FURTHER READING

OTITIS MEDIA IN THE TOP END

Rural and remote Aboriginal children have extremely high rates of severe otitis media (acute otitis media with perforation and chronic suppurative otitis media). Most Aboriginal children from remote communities will have persistent otitis media and some associated hearing loss. Rates of perforation vary considerably from 5-67%. The World Health Organization advises that rates greater than 4% represent a public health emergency.

AETIOLOGY AND PATHOGENESIS

Otitis media is defined by the presence of fluid in the middle ear space. This is usually a consequence of an infection which leads to a blockage of the eustachian tube (which prevents subsequent drainage of the fluid). Infections can be viral (most commonly RSV, 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 cases of severe otitis media. These are frequently seen in rural and remote Aboriginal children (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, E 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 breast feeding, and use of a pacifier (dummy).

Symptoms and signs. Aboriginal 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.

Non-severe Otitis Media

Otitis Media with Effusion (OME). Presence of fluid behind an intact tympanic membrane without any of the symptoms or signs of an acute infection. OME is very common in young Aboriginal and non-Aboriginal children. Accurate diagnosis of this condition requires the use of either pneumatic otoscopy or tympanometry. Bilateral disease associated with a mean hearing loss of 25dB (equivalent to sticking your fingers in your ears and the sound level of a whisper).

Acute Otitis Media (AOM). 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 tympanic membrane or very red tympanic membrane. AOM is very common in young Aboriginal and non-Aboriginal children.

Severe Otitis Media

Acute otitis media with perforation. Acute otitis media (AOM- see above) plus the perforation of the tympanic membrane within the last 6 weeks. AOM and perforation of the tympanic membrane are frequently painless in Aboriginal children. Perforations usually heal and re-perforate several times before perforations become chronic. Consequently, if there are signs of discharge in the canal, this diagnosis can be made even when the tympanic membrane is intact.

Chronic suppurative Otitis Media (CSOM). Persistent discharge through a perforation of the tympanic membrane for at least 6 weeks despite appropriate treatment for AOM 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). Prevention of CSOM is a priority in Aboriginal 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**
AOM without perforation frequently occurs with other upper respiratory tract infections. Unless ear pain is present, these conditions cannot be reliably distinguished without careful otoscopy. AOM is confirmed by the presence of a bulging tympanic membrane. OME is common in all children. Reliable diagnosis requires the use of pneumatic otoscopy or tympanometry to confirm normal tympanic membrane mobility.

**Severe Otitis Media**
It is most important to distinguish between new perforations (AOM with perforation) and chronic discharge (CSOM). Most new perforations occur in the first 2 years of life and are associated with small holes (<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. Otitis externa is another important cause of ear discharge. It is usually associated with a painful canal wall that may be identified by pain on moving the pinna prior to otoscopy. The presence of otitis externa does not exclude severe otitis media since the chronic presence of discharge in the canal may be the cause of the local skin infection.

**PRINCIPLES OF MANAGEMENT**

Most Aboriginal 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. For children with severe otitis media, early identification and compliance with recommended antibiotic therapy is the key to effective medical management. Even if severe otitis media is not present, parents should be advised about the need to bring their child to the clinic should they develop 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. Check that your school has an ear health program.

Clinical management depends on the clinical syndrome as classified above. A detailed protocol has been published by the Office of Aboriginal and Torres Strait Islander Health. See management guidelines.
FURTHER INFORMATION

TELEPHONE ADVICE

DHCS Community Health Team  Centre for Disease Control-Darwin  8922 8044
Paediatricians  Royal Darwin Hospital  8922 8888
NT Hearing Services  Dept of Health Community Services  8922 7110
Australian Hearing  Commonwealth Government-Darwin  8945 5511

MANAGEMENT GUIDELINES

Recommendations for Clinical Care  Office of Aboriginal and Torres Strait Islander Health  (02) 6289 5280
Otitis Media in Aboriginal and Torres
Strait Islander Populations.

EDUCATIONAL RESOURCES

Helping the Child with a Hearing Loss  DHCS-NT Hearing Services  8922 7110
Glue Ear-Information for Parents  DHCS-NT Hearing Services  8922 7110
Fluctuating Conductive Hearing Loss  Australian Conductive Deafness Association  (03) 9596 1896
Support and Resources  Deafness Association of the NT  8945 2016

FURTHER READING

• Couzos S Murray S, Aboriginal Primary Health Care - An evidenced based approach. 20002/3 Oxford University Press, Melbourne 2nd
Ed-in press.
Diarrhoea in Aboriginal Children

Intestinal morphology and function has been shown to vary geographically.

DIARRHOEA IN ABORIGINAL CHILDREN IN THE TOP END

Acute infectious diarrhoea is a serious health problem for Aboriginal infants and children. Over one third of Aboriginal children in the NT less than one year of age are admitted to hospital with acute gastroenteritis each year. Diarrhoeal illness remains the most common cause of admission to hospital for children under 5 years of age and their admissions are usually prolonged due to multiple co-morbidities. The high prevalence of diarrhoeal diseases in Aboriginal communities is related to overcrowding, poor hygiene and sanitation all of which reflect socio-economic disadvantage. Living in such an environment contributes to recurrent episodes of diarrhoea and the development of 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 in the stools of children without diarrhoea. Isolation of 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 children have been reported. The brush border enzyme lactase is reduced from a combination of malnutrition, tropical environmental enteropathy syndrome and acute infection and the threshold of activity can be easily exceeded. This is most common in breastfeeding infants who receive high lactose loads from the milk. 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 30-50% of asymptomatic inhabitants have a different small bowel structure with leaf shaped villi and ridges that are broader and more stunted in architecture. These changes are often associated with an increased inflammatory cell infiltrate and impaired permeability with reduced absorptive capacity. The abnormalities described are not seen in the foetus and severity appears related to time spent in the tropics. The causes are most likely environmental, with recurrent insults to the gut from infective organisms and changes in the gut microflora. It is exacerbated by an unhygienic living environment. 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.

CLINICAL PICTURE

Risk factors. Children at greatest risk of complications from diarrhoeal illness include:

- Infants < 12 months.
- Children with underlying malnutrition.
- Children with immune dysfunction (e.g. from zinc deficiency).
- 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 obtundation. Aboriginal children presenting with diarrhoea should be thoroughly examined for other infective co-morbidities.
including ear, chest, central nervous system and urine infections.

A guide to the clinical assessment, grading and management of dehydration can be found in the CARPA manual. Malnourished and septic children may be less dehydrated than clinical examination would suggest and physical signs are unreliable in the obese infant. 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, rotavirus antigen detection, shiga toxin (bloody diarrhoea), reducing substances for lactose intolerance, blood samples for electrolytes, acid/base status, serum urea and creatinine. A lead II rhythm strip may be helpful in assessing severe hypokalemia associated with acute gastroenteritis.

**DIFFERENTIAL DIAGNOSIS**

It is important to consider other diagnoses, especially in younger children.

**Infective.** If a high fever of ≥ 39°C is present search for another focus of infection including septicaemia, meningitis, urinary tract infection, pneumonia, soft tissue infection and acute otitis media.

**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 is provided. Mode of replacement will depend on the degree of dehydration. Children with ≥ 5% dehydration generally require admission to hospital for nasogastric or intravenous rehydration. For rehydration protocols refer to the CARPA manual or the remote GP guidelines from TEDGP.

**General principles include:**

- Continuation of breast feeding.
- Use of oral rehydration solutions.
- Reintroduction of solids early by feeding of hungry children.
- Avoid antibiotics, antiemetics, and antidiarrhoeals. It is important however to review anti-helminth treatment especially in cases where diarrhoea is prolonged.
- Provide simple instructions for home management using oral rehydration with ORS.
- Consider admission for: babies < 6 months old; moderate dehydration; severe disease; chronic diseases; review not possible; or difficult social circumstances.
- Hand washing needs to be encouraged at every opportunity.

Public health actions will 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.
Diarrhoea in Aboriginal Children

FURTHER INFORMATION

TELEPHONE ADVICE

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<th>Gove District Hospital</th>
<th>Katherine District Hospital</th>
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MANAGEMENT GUIDELINES

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<th>Acute Care Management Guidelines for the Resident Remote GP</th>
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<tr>
<td>CARPA-Standard Treatment Manual</td>
<td>Central Australian Division of Primary Health Care</td>
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<td>SWSBSC Program</td>
<td>Dept of Health &amp; Community Services</td>
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EDUCATIONAL RESOURCES

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<tr>
<th>Flipchart-Take Care of the Outside to Take Care of the Inside</th>
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FURTHER READING

It is the first semester of the school year. Along with the health centre staff, you have just completed the annual school-aged screening as part of the Healthy School-Age Kids Program. This is a combined health and education program which aims to improve the health and learning outcomes of children by health promotion in the school setting, child health screening and integration of other programs and services for school-age children.

You saw 46 out of 51 children identified as living in the community and are very pleased with effort made by teachers and health staff, in seeing such a good proportion of the children. When you review the results that evening you notice that 14 children 4-15 years of age had a haemoglobin (Hb) of less than 110g/L on a fingerprick 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 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. Trichuris trichuria (whipworm) 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 to 9.8% have 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 due to bacterial overgrowth in the small bowel.

Inherited haemoglobinopathies are very rare in Aboriginal Australians.

Is mild iron deficiency anaemia (Hb 90-109g/L) a concern if the child is otherwise well?

Yes it is a concern. The fact that anaemia exists indicates that iron stores are depleted. Iron deficiency anaemia and even iron deficiency has been shown to 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. Mild anaemia may progress to severe anaemia if not treated and there may be 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 full blood examination (FBE) 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 of the increased inflammatory load due to chronic or recurrent infection (especially on ferritin).

The HemoCue haemoglobinometer, using fingerprick blood sample, is a simple and acceptable screening tool which has high sensitivity and specificity. A Top End study confirmed high correlation between fingerprick and laboratory Hb.
results. However, it is essential that staff are trained and follow the manufacturers 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 (PPV) of the test will be correspondingly high. When the prevalence drops to 20% or below, a FBE and film are recommended as the PPV of the fingerprick test is likely to be in the order of 62% or less, ie about one third with a screening result Hb <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 twice weekly supervised oral iron for 3 months or a short course of intramuscular iron. Compliance to oral iron is often a problem in remote communities. The decision as to the type of iron regimen should be made in consultation with the child and the carer and take into account staff resources.

The broad spectrum antihelmintic 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 trichuris and strongyloidiasis and anaemia and growth faltering often co-exist. This guideline may change pending further research into 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 FBE, 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 sort of advice would you give to the parent/carer to prevent them becoming anaemic again?

Key nutritional advice includes:

- 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 wheatbix), Milo milk drinks and green vegetables.
- Encourage fruit juice with meals.
- Avoid drinking tea with meals, as it decreases the absorption of non-haem iron by 75%. The iron added to cereals such as wheatbix is non-haem, so tea with breakfast may prevent iron absorption, while orange juice will enhance it considerably.
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.

The Healthy School-Age Kids program encourages feedback of health information. The Community Child Health Nurse will send a community report of the school-age screening results. It may be useful to discuss the results with clinic staff (Aboriginal Health Worker, Nurses and Doctors) to decide how to provide community feedback. The Council, School, the Aboriginal Student Support and Parent Awareness Committee (ASSPA) and Women’s Centre may be interested. 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 are school achievement. School meal programs in conjunction with routine “deworming” and iron supplementation are common practice in many developing countries with high prevalence of iron deficiency anaemia. In the NT iron supplementation to prevent iron deficiency anaemia is rarely used.
FURTHER INFORMATION

Community paediatrician  Centre for Disease Control-Darwin  8922 8444
DHCS Community Health Team Centre for Disease Control-Darwin  8922 8044
Nutritionists  Darwin Rural  8922 8236 or 8922 7810
                  East Arnhem  8987 0446 or 8987 0429
                  Katherine  8973 8631 or 8973 8946
Healthy School-Age Kids Program Manual for Rural/Remote Communities, 1998, DH&CS and NT Dept. of Education Each community school has a copy. If no copy available then contact the Community Health Team  8922 7816
SWSBSC Program Dept of Health & Community Services  8922 7766
Health School-Age Kids Resource Folder for Rural/Remote Communities, 1999. Each community school has a copy. If no copy available then contact the Community Health Team  8922 7816

FURTHER READING

• Sharmanov, Almaz 2000. Anaemia testing manual for population-based surveys. Calverton, Maryland USA: Macro International Inc.
Martin is an 8 month old baby in a large family who live in a remote Aboriginal community. He was born in hospital at term, after an uneventful pregnancy. His birth weight was 2.6kg. The community nurses have been concerned because Martin has not gained any weight for the previous two months. He had been admitted to hospital at 3 months of age with pneumonia and gastroenteritis and again at 6 months with gastroenteritis and iron deficiency.

Martin was referred to you after his mother and grandmother brought him to the health centre with a 2 week history of loose stools that had recently got worse and high fever. His mother said that Martin was still breast feeding 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 was crying and irritable but appeared vigorous. His temperature was 38.5°C, pulse rate 140/min, respiratory rate 38/min, BP 95/65 and he was well perfused. His lips and mucous membranes were dry but his skin turgor was normal. His eyes looked sunken. His heart sounds were normal and lung fields were clear. His abdomen was soft and non tender and bowel sounds were present. He passed another moderate sized loose offensive stool during the examination. His weight two weeks previously was 7.0kg and weight today 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 caused by rotavirus (which typically occurs in epidemics) or bacterial organisms, superimposed on gut parasites which may or may not be contributing to the looseness of the stools. He may have suffered a gut insult two weeks prior to his acute symptoms, but his chronic loose stools are probably secondary to lactose intolerance secondary to his previous gastroenteritis episodes, and possible tropical enteropathy syndrome, although he is only 8 months. Pathogens commonly causing persistent diarrhoea from initial infection include Shigella, Enteropathogenic E.coli, Cryptosporidium, Giardia lamblia and Strongyloides.

Lack of introduction of solids to breast feeding 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? See CARPA manual for guidelines on assessment of hydration.

Martin's clinical signs of dehydration are dry lips and mucus membranes. Martin also has sunken eyes, but this could be because he is chronically underweight. He is tachycardic, but this is reflective of 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 mls = 6.72 (wt in kg) x 4/100 (percentage dehydration) x 1000
= 268.8 mL

What is his maintenance fluid requirement?

For children up to 10 kg maintenance requirement is 4 mL/kg/hr
7(wt in kg) x 4 (mls) = 28 mL/hr
Suggest a management plan for Martin (the CARPA manual and the Remote GP Guidelines both contain rehydration protocols)

Continue to breast feed
Supplementation with ORS early
Deficit replacement over 8 hrs = 33.6mLs/hr
  - maintenance = 28.0 mLs/hr
  - Total = approx 62 mLs/hr over next 8 hrs

Oral hydration with ORS using a 10ml syringe is started by his very patient grandmother. Martin vomited after the second syringe but this settled as his grandmother persevered with his rehydration. She remained 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 was collected. Urinanalysis was weakly positive for protein only. The urine was sent to the lab in town, along with a stool specimen. Martin was given 3 days of albendazole, and his mother continued breast-feeding. Advice about commencing solids was provided. His mother was asked to bring him back to the clinic the following morning or earlier if vomiting and/or diarrhoea became worse.

When Martin returned the next morning his weight was 7.1kg. He looked well and was still breast feeding. His stools remained loose but less frequent. Some perianal excoriation was noted on examination. There were no clinical signs of dehydration. Martin’s 380g weight gain = 380 mL water gain which means that he was approximately 5.3% dehydrated.

When the laboratory results were available, the stool microscopy was negative for ova, cysts and parasites. No rotavirus antigens were detected and culture was negative for bacterial pathogens. Urine culture showed no significant growth.

What would you now advise Martin’s mother?

Martin should continue to be breast fed, and if the diarrhoea persists he needs ongoing supplements with ORS, which she can get 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 weigh at the clinic and ongoing parental education and promotion of frequent nutritious foods for Martin. He is listed for review and investigation with the visiting paediatrician and will continue to have regular reviews by the community doctor. One of the Aboriginal Health Workers in the clinic now works closely with Martin’s mother. She helps make sure that there is enough money budgeted in the household to buy foods for Martin. Martin’s maternal aunt is on the Strong Women’s Strong Babies Strong Culture (SWSBSC) committee and she is asked by one of the Community Health Centre nurses to assist Martin’s mother in learning how to give appropriate foods to Martin.
Other Conditions
Systemic Lupus Erythematosus (SLE)

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. It 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-Aboriginal 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 the Top End.

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.

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).

Investigations. Antinuclear antibodies are detected in more than 90% of patients, but are commonly found in other autoimmune diseases. Anti dsDNA 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 leukopenia, 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 diagnosis includes 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

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 and strongyloidiasis should be excluded or, if present, treated prior to immunosuppressive therapy.
Suggestive Symptoms
(Arthralgia/arthritis, rash, serositis, periorbital or ankle oedema, neurologic complaints, fever, fatigue)

+ Suggestive Physical Findings
(Arthritis, rash, alopecia, oral/nasal ulcers, vasculitic lesions, adenopathy, pleural/pericardial/peritoneal signs, oedema, neurologic deficits)

Laboratory investigations

- ANA*
- Haematologic*
- Renal*
- Serologic

<table>
<thead>
<tr>
<th>Haematologic</th>
<th>Renal</th>
<th>Serologic</th>
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<tr>
<td>Anaemia, WBC, Proteinuria, RBC, casts, lymphocytes, platelets</td>
<td>Antibodies to dsDNA, Sm, lymphocytes, RBC (Coombs), platelets, phospholipids</td>
<td>Complement</td>
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NEGATIVE
- Symptoms Remit or alternative Dx
- SLE not present

POSITIVE
- Symptoms persist

 Repeat ANA & order serology
- All tests neg
- SLE not present
- One or more tests positive
- SLE or other CTD
- Not SLE

Definite SLE
If ≥4 ACR criteria present (see next page)
OR
Possible SLE or other CTD if <4 ACR criteria present

ANA-antinuclear antibody;
ACR-American College of Rheumatology;
CTD-connective tissue disease;
WBC-white cell count;
RBC-red blood cell count.

* recommended in initial evaluation.

1. Malar rash  
Fixed erythema, flat or raised, over the malar eminences.

2. Discoid rash  
Erythematous raised patches with adherent keratotic scaling and follicular plugging; atrophic scarring may occur.

3. Photosensitivity  
Exposure to UV light causes rash.

4. Oral ulcers  
Includes oral and nasopharyngeal, observed by physician.

5. Arthritis  
Nonerosive arthritis involving two or more peripheral joints, characterised by tenderness, swelling, or effusion.

6. Serositis  
Pleuritis or pericarditis documented by ECG or rub or evidence of pericardial effusion.

7. Renal disorder  
Proteinuria >0.5g/d or >3+, or cellular casts.

8. Neurologic disorder  
Seizures without other cause or psychosis without other cause.

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. Immunologic disorder  
Anti-dsDNA, anti-Sm, and/or anti-phospholipid.

11. Antinuclear antibodies  
An abnormal titer of ANAs by immunofluorescence or an equivalent assay at any point in time in the absence of drugs known to induce ANAs.

If four of these criteria are present at any time during the course of disease, a diagnosis of systemic lupus can be made with 98% specificity and 97% sensitivity.

FURTHER INFORMATION

TELEPHONE ADVICE

Specialist Physician & Registrar
Arthritis & Osteoporosis NT
Royal Darwin Hospital
18 Bauhinia St, Nightcliff
8922 8888
8948 5232

MANAGEMENT GUIDELINES


EDUCATIONAL RESOURCES

Arthritis & Osteoporosis NT
18 Bauhinia St, Nightcliff
8948 5232

FURTHER READING

MACHADO-JOSEPH DISEASE IN THE TOP END

Machado-Joseph Disease (MJD) is an inherited neurological disorder found in some family groups in northeast Arnhemland. The disease is thought to have been spread around the world by Portuguese traders, eventually reaching Arnhemland via the Macassan traders. The earliest recalled cases of the disease in the Top End were in the late 1800’s. It remains isolated to four Aboriginal family groups with most affected people now living on Groote Eylandt. At present there are twelve known affected individuals and about 80 children and grandchildren of cases who are at risk of developing the disease.

AETIOLOGY AND PATHOGENESIS

MJD, 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 multisystem degeneration affecting the cerebellum and its connections, including the basal ganglia, dorsal columns, upper and lower motor neurons 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 20’s to mid 30’s and is the most common. Cases may have the full range of signs & symptoms between types 1-3 (see clinical picture).
- **Type 3** - presents in 50’s and may not affect the life span. 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 dysarthria and tremors.
- **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.

**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 diagnosis may include: Wernicke’s encephalopathy, 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 renal
physician, rehabilitation consultant and refer to the MJD Care Guidelines.

- **Bowel management** includes establishing a bowel routine in consultation with client/carers to suit needs & lifestyle including aprients 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 & mouth exercises and communication using closed questions.

- **Respiratory care** is managed in consultation with a physiotherapist.

- **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.

- **Vocational, recreational, housing and financial support.** Identify needs and refer to appropriate services such as:
  - Regional Aged & Disability Service
  - Allied Health
  - Commonwealth Rehabilitation Service
  - Centrelink
  - Community Disability Legal Service

**FURTHER INFORMATION**

**TELEPHONE ADVICE**

<table>
<thead>
<tr>
<th>Service</th>
<th>Contact Details</th>
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<tbody>
<tr>
<td>Rehabilitation Consultant, Physician, Renal Physician</td>
<td>Royal Darwin Hospital 8922 8888</td>
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<tr>
<td>Continence Advisor, Spinal Nurse</td>
<td>Specialist Allied Health Services, Darwin 8922 7283</td>
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<tr>
<td>Specialist Allied Health Services, Darwin</td>
<td>Department of Health &amp; Community Services 8922 7283</td>
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<tr>
<td>East Arnhem Aged &amp; Disability Service, Nhulunbuy</td>
<td>Department of Health &amp; Community Services 8987 0400</td>
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**MANAGEMENT GUIDELINES**

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<thead>
<tr>
<th>Guideline</th>
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<tr>
<td>Machado-Joseph Disease Care Guidelines-May 2001</td>
<td>East Arnhem Aged &amp; Disability Service 8987 0400</td>
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**FURTHER READING**

### ACRONYMS

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<tr>
<th>Acronym</th>
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<td>ABL</td>
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<td>American College of Rheumatology</td>
</tr>
<tr>
<td>AFB</td>
<td>Acid fast bacilli</td>
</tr>
<tr>
<td>α-FP</td>
<td>Alpha foetoprotein</td>
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<tr>
<td>AIDS</td>
<td>Acquired immunodeficiency syndrome</td>
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<td>ALP</td>
<td>Alkaline phosphatase</td>
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<tr>
<td>ANA</td>
<td>Antinuclear antibody</td>
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<tr>
<td>Anti-DNase B</td>
<td>Antideoxyribonuclease B</td>
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<tr>
<td>AOM</td>
<td>Acute otitis media</td>
</tr>
<tr>
<td>APSGN</td>
<td>Acute post streptococcal glomerulonephritis</td>
</tr>
<tr>
<td>ARF</td>
<td>Acute rheumatic fever</td>
</tr>
<tr>
<td>ASOT</td>
<td>Antistreptolysin 0 titres</td>
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<td>Council for Aboriginal Alcohol Program Services</td>
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<td>CARPA</td>
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<td>CHC</td>
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<td>CK</td>
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<td>CNS</td>
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<td>CRCATH</td>
<td>Co-operative Research Centre for Aboriginal and Tropical Health</td>
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<tr>
<td>CRP</td>
<td>C-Reactive protein</td>
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<td>CSF</td>
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<tr>
<td>CSOM</td>
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<td>CT scan</td>
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<td>CTD</td>
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<td>DEET</td>
<td>N,N-diethyl-m-toluamide</td>
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<td>ESR</td>
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<td>FVU</td>
<td>First void urine</td>
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<tr>
<td>GAA</td>
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<td>HSV</td>
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<td>Abbreviation</td>
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<tr>
<td>HTLV-I</td>
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<td>HVS</td>
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<td>IF</td>
<td>Immunofluorescence</td>
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<td>LN</td>
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<td>LTBI</td>
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<td>MCS</td>
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<td>MJD</td>
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<td>MRI</td>
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<td>MRS A</td>
<td>Methicillin-resistant <em>Staphylococcus aureus</em></td>
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<td>MSU</td>
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<td>Murray Valley Encephalitis</td>
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<td>PICC</td>
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<td>Pelvic inflammatory disease</td>
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<td>PNG</td>
<td>Papua New Guinea</td>
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<td>RNA</td>
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<td>SE Asia</td>
<td>South East Asia</td>
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<td>SLE</td>
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<td>STI</td>
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<td>SWSBSC</td>
<td>Strong Women Strong Babies Strong Culture</td>
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<td>TB</td>
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<td>TPHA</td>
<td>Treponema pallidum haemagglutination</td>
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<td>WBCT</td>
<td>Whole blood clotting test</td>
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<td>WCC</td>
<td>White cell count</td>
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<td>World Wide Web</td>
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## RESOURCE AGENCIES

### TOP END DIVISION OF GENERAL PRACTICE

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<tr>
<th>NT GOVERNMENT SWITCHBOARD</th>
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### EMERGENCY SERVICES

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<thead>
<tr>
<th>CRANA Bush Crisis Line</th>
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<tr>
<td>Crisis Line</td>
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<tr>
<td>Sexual Assault Referral Centre</td>
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<tr>
<th>Child/Infant Health Clinic</th>
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<th>Sports Medicine Australia</th>
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<td>Darwin Dental Clinic</td>
<td>8924 4475</td>
<td>AIDS STD Program</td>
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<td>Poisons Information Centre</td>
<td>8922 7341</td>
<td>NT AIDS Council</td>
<td>8941 1711</td>
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<td>Poisons Info Line</td>
<td>13 11 26</td>
<td>SIDS NT</td>
<td>8948 5311</td>
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<tr>
<td>National Heart Foundation</td>
<td>8981 1966</td>
<td>Mental Health Services</td>
<td>8999 4988</td>
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<tr>
<td>Australian Red Cross</td>
<td>8924 3999</td>
<td>Patient Assistance Scheme Darwin</td>
<td>8922 8134</td>
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<tr>
<td>NT Women’s Cancer Prevention</td>
<td>8999 5511</td>
<td>Rural Services Darwin</td>
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<td>Breastscreening NT</td>
<td>8922 5522 /13 20 50</td>
<td>Remote Health Clinics-Katherine</td>
<td>8973 8654</td>
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<tr>
<td>Arthritis &amp; Osteoporosis NT</td>
<td>8948 5232</td>
<td>Ashma Foundation NT</td>
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<tr>
<td>Medicines Line (Consumer Info)</td>
<td>1300 888 763</td>
<td>Therapeutic Advise &amp; Information Services</td>
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### TOP END HOSPITALS

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<td>Katherine District Hospital</td>
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<td>RDH Access Line-A&amp;E</td>
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<th>Centre for Disease Control-Nhulunbuy</th>
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### ABORIGINAL SERVICES

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<thead>
<tr>
<th>Danila Dilba Aboriginal Health Service</th>
<th>8936 1717</th>
<th>Aboriginal Resource &amp; Development Service (Language &amp; Education)</th>
<th>8984 4174</th>
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<tbody>
<tr>
<td>Aboriginal Interpreter Service</td>
<td>8999 8353</td>
<td>Darwin Aboriginal and Islander Women’s Shelter</td>
<td>8945 2284</td>
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<tr>
<td>ATSIC (including Legal Aid)</td>
<td>8944 5566</td>
<td>Dept Community Development Sport &amp; Cultural Affairs</td>
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### AGED CARE/DISABILITY SERVICES

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<thead>
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<th>Council of the Ageing</th>
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<th>Dept of Health &amp; Ageing</th>
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<tr>
<td>Aged &amp; Disability Services</td>
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<td>Disability Information Officer</td>
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### ALCOHOL & OTHER DRUG SERVICES

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<tr>
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<th>Alcohol &amp; Other Drug Services</th>
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<tr>
<td>Alcoholics Anonymous</td>
<td>8948 5202</td>
<td>Darwin Withdrawal Services</td>
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<td>Alcohol Awareness &amp; Family Recovery</td>
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<td>C.A.A.P.S</td>
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### CHILD BIRTH/FAMILY PLANNING SERVICES

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<th>Mobile Midwives</th>
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<tr>
<td><strong>YOUTH/CHILDRENS SERVICES</strong></td>
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<tr>
<td><strong>Darwin Homebirth Group</strong></td>
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<td><strong>Nursing Mothers Association</strong></td>
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<tr>
<td><strong>Kids Help Line</strong></td>
<td>1800 551 800</td>
<td><strong>School Sports Council</strong></td>
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<tr>
<td><strong>Adoption Enquiries</strong></td>
<td>1300 360 208</td>
<td><strong>YMCA-Darwin</strong></td>
<td>8981 8377</td>
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<tr>
<td><strong>Child Working Conditions</strong></td>
<td>8999 2887</td>
<td><strong>Office of Youth Affairs</strong></td>
<td>8999 6706</td>
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<tr>
<td><strong>Kidsafe</strong></td>
<td>8985 1085</td>
<td><strong>Dept of Education, Science &amp; Training</strong></td>
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<td><strong>Playgroup Association of NT</strong></td>
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<td><strong>Casy House-Youth Refuge</strong></td>
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<td><strong>Somerville Community Services</strong></td>
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<tr>
<td><strong>St Vincent De Paul Society</strong></td>
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<td><strong>Darwin Aboriginal &amp; Islander Women's Shelter</strong></td>
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<td><strong>NT Police Domestic Violence Unit</strong></td>
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<tr>
<td><strong>Domestic Violence Counsellor</strong></td>
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<td><strong>Relationships Australia (NT)</strong></td>
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<td><strong>Crisis Line</strong></td>
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<td><strong>RESOLVE Counselling Service</strong></td>
<td>1800 898 500</td>
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<td><strong>Sexual Assault Counsellor</strong></td>
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<tr>
<td><strong>Abortion &amp; Grief Counselling</strong></td>
<td>1800 777 690</td>
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<td><strong>Anglicare Family Counselling</strong></td>
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<th><strong>LEGAL ADVICE/OTHER LANGUAGES</strong></th>
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<tr>
<td><strong>Anti-Discrimination Commission</strong></td>
<td>8999 1444</td>
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<td><strong>Health &amp; Community Services Complaints</strong></td>
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<tr>
<td><strong>Commission</strong></td>
<td><strong>Translating Interpreter Services (TIS)</strong></td>
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<td><strong>NT Working Women's Centre</strong></td>
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<td><strong>Multi Cultural Council NT</strong></td>
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<td><strong>NT Interpreter and Translator Service</strong></td>
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<td><strong>Women's Advisory Council</strong></td>
<td>8924 4333</td>
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<td><strong>Office of Women's Policy</strong></td>
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<td><strong>Superannuation Consulting &amp; Advisory Service</strong></td>
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<td><strong>Family Court of Australia</strong></td>
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<td><strong>Territory Housing</strong></td>
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<td><strong>Salvation Army</strong></td>
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<td><strong>Volunteering NT</strong></td>
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<td><strong>Ombudsman for the NT</strong></td>
<td>1800 806 380</td>
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<tr>
<td><strong>Consumer Affairs Council</strong></td>
<td>1800 019 319</td>
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