Infectious diseases of tropical Australia

In the north of Australia, there are two additional medical issues to consider – Aboriginal health and the specific diseases of the tropics. With global warming, infections that are generally restricted to the tropics and have, to date, been more common in tropical Australia may be seen further south in the continent.

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Tropical Australia lies north of the Tropic of Capricorn (23.5°S), and has a great diversity of climates and terrains. The region has a number of infections that are mostly restricted to the north (e.g. dengue, melioidosis, scrub typhus) and infections that usually have been relatively more common in the north than in the rest of Australia (e.g. Ross River and Barmah Forest virus, Murray Valley encephalitis, leptospirosis and nocardiosis [Figure 1]). There are also increased common infection rates in Aboriginal communities.

Infectious diseases in Aboriginal communities

There is continuing disparity in morbidity and mortality between Aboriginal people and other Australians. The differential mortality rates are still highest for various infectious diseases, but noncommunicable diseases such as circulatory and chronic respiratory illnesses now cause more deaths in Aboriginal people than infectious diseases. Mortality in the 20- to 40-year-old age group is up to 10 times that for other Australians, and life expectancy is 10 to 20 years lower.

The current burden of infectious diseases in Aboriginal communities can be attributed primarily to socioeconomic disadvantage. Rates of various communicable diseases are similar to those previously seen in the urban slums of industrialised countries, including Australia, a century ago. Living conditions in many remote Aboriginal communities remain harsh, with

IN SUMMARY

- **There is continuing disparity in morbidity and mortality between Aboriginal people and other Australians. Aboriginal mortality in the 20- to 40-year-old age group is up to 10 times that for other Australians, and life expectancy is 10 to 20 years lower.**
- **Outbreaks of Ross River and Barmah Forest viruses occur throughout Australia, but are more common in the tropical north. The infection can be asymptomatic.**
- **The Murray Valley encephalitis virus is endemic in the Kimberley region of Western Australia and adjacent Northern Territory. Possibly only 1 in 1,000 infected have clinical illness, but mortality is 20% in these patients and residual neurological disease occurs in up to 40%.**
- **Melioidosis is the most common cause of fatal community-acquired bacteraemic pneumonia in the top end of the Northern Territory.**
- **Pulmonary haemorrhage from leptospirosis is increasingly recognised in north Queensland.**
- **The reintroduction of dengue continues to cause outbreaks in north Queensland.**
- **Scrub typhus is transmitted by bites from infected larval mites. A skin ulcer with a black crust (eschar) may develop at the bite site; delay in treatment may lead to multi-organ failure.**
- **Global warming may extend the endemic range of tropical diseases in Australia.**

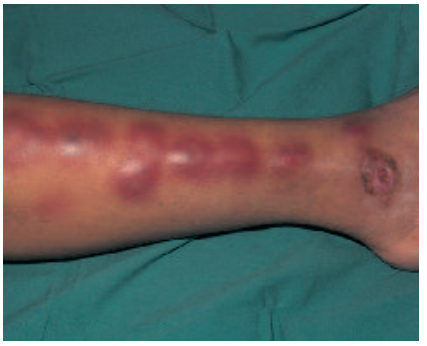

Figure 1. Cutaneous nocardiosis from *Nocardia caviae*, an environmental pathogen more common in northern Australia. The ulcer at the inoculation site is over the ankle.

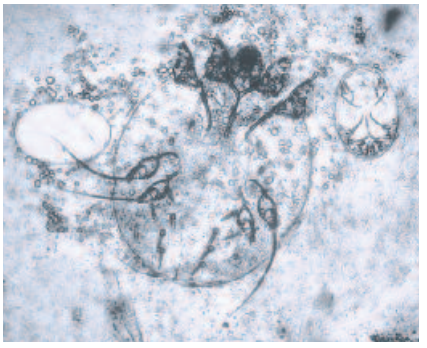

Figure 2. Scabies mite plus eggs in a skin scraping.

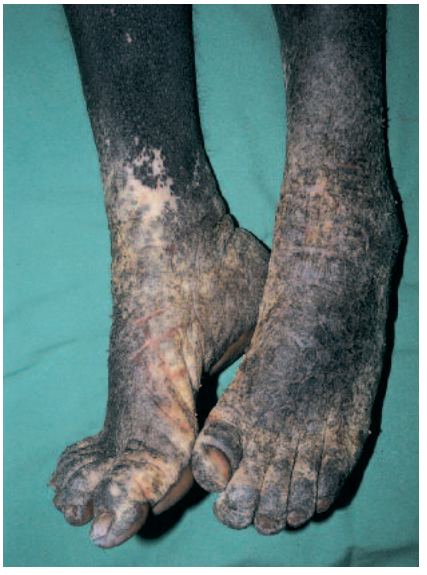

Figure 4. Scabies hyperinfestation, or crusted ('Norwegian') scabies.

continuing overcrowding and difficulties with water supplies and sanitation.

Infections that are seen in Aboriginal communities are listed in the Table, and examples are shown in Figures 2 to 5. Specific infections that are now uncommon in Australia outside of Aboriginal communities include:

- hookworm *(Ancylostoma duodenale)*, which causes iron deficiency anaemia and retards growth
- whipworm *(Trichuris trichiura)*, which causes diarrhoea and can cause rectal prolapse
- strongyloides *(Strongyloides stercoralis)*, which can cause swollen belly syndrome in children
- acute rheumatic fever following streptococcal pharyngitis and possibly impetigo
- post-streptococcal glomerulonephritis, which occurs mostly in children with impetigo and can be epidemic
- trachoma *(Chlamydia trachomatis)*, causing chronic conjunctivitis with scarring and blindness
- epidemic gonococcal conjunctivitis, which occurs mostly in children and is non-sexually transmitted
- syphilis (see Figure 6)
- donovanosis *(Calymmatobacterium granulomatis)*, causing progressive genital ulceration (see Figure 7)
- leprosy.

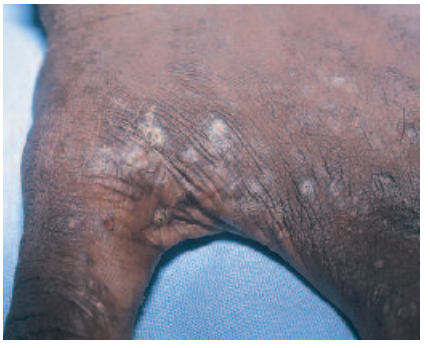

Figure 3. A common presentation of scabies.

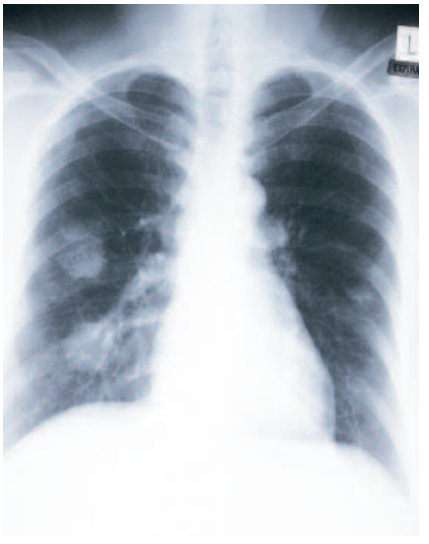

Figure 5. Pulmonary cryptococcomas from *Cryptococcus neoformans* var. gattii in a patient from Arnhem Land.

Ear, nose and throat infections

Research from the Northern Territory has shown that virtually all children in some remote communities are colonised in the nasopharynx by multiple strains of *Streptococcus pneumoniae* and non-typeable *Haemophilus influenzae* from soon after birth. Chronic middle ear effusions are common and up to half of all children in some communities have had at least one perforated eardrum by their first birthday. Rates of invasive *S. pneumoniae* reported in Aboriginal children from central Australia are among the highest documented in the world – this was also the case for invasive disease from *H. influenzae* serotype b (Hib) until its virtual elimination by vaccination.

Rheumatic fever

In the Aboriginal communities of northern and central Australia, the incidence of acute rheumatic fever and the prevalence of established rheumatic heart disease are amongst the highest documented in the world (see Figure 8). Acute rheumatic fever is an immune-mediated response to infection with group A streptococcus *(Streptococcus pyogenes)*.

It has 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.

Models for Aboriginal healthcare

In community clinics, immediate needs of sick patients often make it difficult for health staff to maintain preventive programme initiatives and perform other essential public health functions. To balance the needs of acute clinical care against preventive and public health medicine, a number of healthcare delivery models have emerged, with common themes of strong community involvement, co-ordination of programmes and a multi-disciplinary approach. Recently, the increased resources for placing GPs in remote communities has had strong support from community members and appears to be achieving improved standards of care.

Aboriginal healthcare programmes and treatment protocols are increasingly

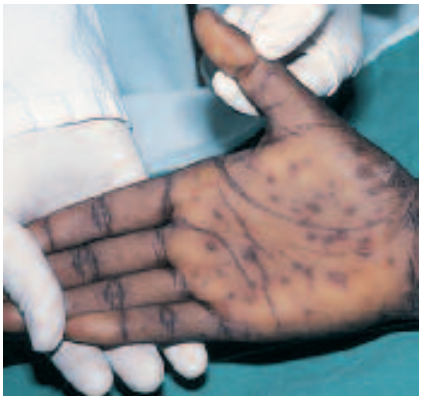

Figure 6. Secondary syphilis.

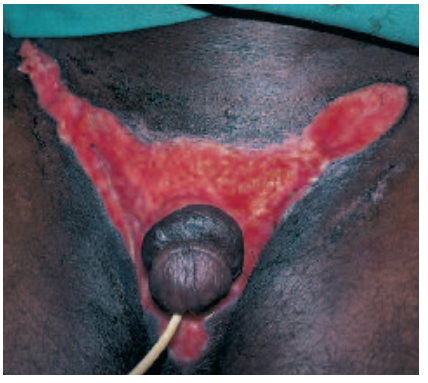

Figure 7. Extensive donovanosis.

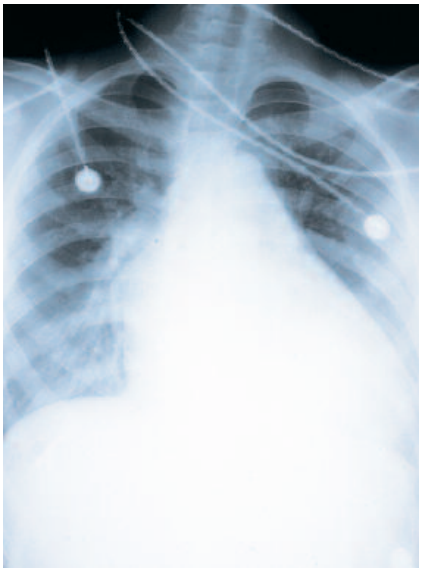

Figure 8. Chest x-ray of fatal recurrent rheumatic fever in a 16-year-old boy from a remote Aboriginal community.

Table. Infections seen more commonly in Aboriginal communities

Skin infections

- Impetigo (skin sores – especially in children, usually *Streptococcus pyogenes*)
- Boils (in all ages, usually *Staphylococcus aureus*)
- Scabies (in all ages), see Figures 2 to 4
- Tinea corporis (in all ages, usually *Trichophyton rubrum*, often with nail involvement)

Respiratory infections

- Otitis media (especially chronic suppurative otitis media in children)
- Bacterial pneumonia (in all ages)
- Infectious exacerbations of chronic airways disease (mostly in adults)
- Tuberculosis (in all ages)

Gastrointestinal infections

- Infectious diarrhoea caused by *Salmonella* and *Shigella* (in all ages, especially children), *Rotavirus* (in children), *Campylobacter* (in all ages), *Cryptosporidium* (in children) and *Giardia* (in all ages)
- Intestinal nematodes (roundworms) such as hookworm, whipworm and strongyloidiasis

Sexually transmitted diseases

- Gonorrhoea
- Syphilis
- Chlamydial infection
- Trichomoniasis
- Donovanosis

Urinary tract infections

- Cystitis (including children, usually *Escherichia coli*)
- Pyelonephritis (in adults, usually *E. coli*)

Bone and joint infections

- Septic arthritis (in all ages, often *Staphylococcus aureus*)
- Osteomyelitis (in all ages, usually *S. aureus*)

Other infections

- Hepatitis B
- Rheumatic fever
- Post-streptococcal glomerulonephritis
- Trachoma
- Cryptococcal meningitis and pneumonia (*Cryptococcus neoformans* var. *gattii*), see Figure 5

being refined and documented. Although there are some regional differences, there is much that is common across central and northern Australia. Public health programmes for co-ordinating control of specific infections in Aboriginal communities are listed in the box on this page. For treatments of specific conditions and for immunisation and preventive programmes, readers are referred to local State and Territory guidelines and the further information listed at the end of this article.

Specific diseases in the tropics

Malaria

The decline of malaria in Australia reflects a combination of case treatment, surveillance and drug eradication programmes, together with vector mosquito control measures around population centres. The last indigenous (not imported) infection occurred in the Northern Territory in 1962, but the continuing presence of certain indigenous *Anopheles* mosquito species and potential local transmission from imported cases means that the area north of latitude 19°S (i.e. north of Broome and Townsville) is still considered to be receptive for the re-establishment of malaria.

Occasional local cycles of transmission called 'introduced malaria' (involving local mosquitoes infected from an imported case) have occurred in north Queensland, especially in the Torres Strait islands with close geographical and cultural links to Papua New Guinea. A case of 'airport malaria' presumed to be caused by importation of an infected mosquito was reported in Cairns in 1996.

There is no infallible chemoprophylaxis for malaria. Significant protection is conferred by measures to prevent mosquito bites, such as avoiding outside activities between dusk and dawn, using insect repellents, and wearing long, loose, light-coloured clothing.

Malaria must be considered in any visitor to a malarious area who presents with a febrile illness. Thick and thin blood films, together with blood collected in an EDTA tube, should be sent to an appropriate laboratory. A single negative blood film does not exclude the diagnosis, particularly if antimalarials or antibiotics have been taken recently.

Immediate treatment with intravenous quinine is required if the patient has altered consciousness, jaundice, oliguria, severe anaemia or hypoglycaemia, a parasite count greater than 100,000 per mm^3, or is vomiting or clinically acidotic. Expert advice should be sought for management of severe malaria.[1]

Typhoid

The prolonged, unremittent fever of typhoid had a mortality of approximately 15% before antibacterial chemotherapy, but there was a steady decline from the late 1800s with increasing understanding of sanitation and the development of piped water supplies and sewerage ser-

Public health programmes in Aboriginal communities

Ongoing programmes

- Immunisation (including pneumococcal vaccination)
- Routine deworming (often linked to school screening)
- Programmes for sexually transmitted diseases (curative treatment, contact tracing and community screening)
- Rheumatic fever programme (secondary prophylaxis)
- Programme for tuberculosis (curative treatment, contact tracing and community screening)

Responses to specific outbreaks or cases

- Community-based scabies programme
- Community-based trachoma programme
- Meningococcal meningitis response protocol
- Gonococcal conjunctivitis response protocol
- Post-streptococcal glomerulonephritis response protocol

Leptospirosis

Organism

Spirochaetes of the *Leptospira interrogans* complex.

Location

The organism is widespread: infections have occurred in workers on banana and cane plantations, rangers and north Queensland river rafters.

Transmission

The organism is transmitted by direct contact of broken skin (?or mucous membranes) with urine, blood or tissue of infected animals, often via contaminated rivers, pools or surface water. The rat is the major animal vector, but cattle, pigs, goats, dogs and marsupials are also infected.

Prevention

Reduce exposure to the organism (with footwear and rodent control).

Clinical description

The incubation period is 4 to 19 days (usually 10). Infection is often asymptomatic. Symptoms and signs include:

- fever
- chills
- headache
- myalgia
- conjunctivitis.

Severe cases (Weil's disease) have jaundice and acute renal failure with haemorrhage caused by endothelial damage and thrombocytopaenia. Pulmonary haemorrhage is increasingly recognised. The illness may be biphasic.

Diagnosis

Diagnosis is by special blood cultures, urine special microscopy and serology.

Treatment

Penicillin or doxycycline.

vices. The last major outbreak occurred in north Queensland in 1928; typhoid is now uncommon in Australia, with some cases occurring in or linked to overseas visitors or returned travellers.

The risk of typhoid to travellers is low but is higher in the Indian subcontinent and Papua New Guinea. In areas where sanitation is poor, immunisation is no substitute for careful selection and handling of food and water. Vaccination will reduce risk, but does not give complete protection against ingestion of heavily contaminated foods. Patients with suspected typhoid should be hospitalised and isolated.

North Queensland coastal fevers

Leptospirosis and scrub typhus were confirmed in the 1930s to be the major infections responsible for fever in north Queensland other than malaria and typhoid. A focus of scrub typhus has recently been identified in Litchfield Park (140 km south of Darwin) and clusters of cases still occur in coastal north Queensland.[2] In far north Queensland, cases of severe pulmonary haemorrhage as a manifestation of leptospirosis have recently been documented.[3]

Management is described in the boxes on pages 76 and this page).

It was subsequently established that other fevers occur in north Queensland, including:

- endemic typhus *(Rickettsia typhi)*
- Australian tick typhus *(Rickettsia australis)*
- Q fever *(Coxiella burnetii)*.

Dengue

Dengue and its main vector mosquito, *Aedes aegypti*, have invaded north Australia periodically since the 1880s, and initial epidemics spread south as far as northern New South Wales. *A. aegypti* breeds in water receptacles, and the replacement of house rainwater tanks with reticulated water is thought to account (at least partly) for the dramatic decline of *A. aegypti* that resulted in eradication of dengue in 1955.

In the 1980s, however, a resurgence of dengue occurred in north Queensland and continues, with seven outbreaks documented between 1990 and 1998.[4] *A. aegypti* and hence dengue remain eradicated from the Northern Territory and Western Australia, but surveillance is essential because imported mosquitoes or larvae are occasionally detected, and travellers enter with dengue acquired from the increasingly endemic countries to the north.

As yet, there is no vaccine for dengue but the disease is usually self-limiting. Effective treatment includes bedrest, fluids and paracetamol. Expert advice should be sought if any haemorrhagic complications or shock occur.

Epidemic polyarthritis

Outbreaks of illness with fever, polyarthritis or polyarthralgia and rash were described from 1927. The alphaviruses Ross River virus and Barmah Forest (Figure 9) virus account for the majority of cases, but a number of other viruses are occasionally implicated and new viruses are being sought.

Although more common in the tropical north, Ross River and Barmah Forest viruses cause outbreaks throughout Australia. Infection can be asymptomatic, and it is likely that infection in childhood accounts for the very low incidence of clinical disease in Aboriginal communities in northern Australia despite high rates of seropositivity.

Australian encephalitis

The Murray Valley encephalitis virus, a mosquito-borne flavivirus, is endemic in the Kimberley region of Western Australia and adjacent Northern Territory.[5] Since the last encephalitis epidemic in 1974, which occurred in the region of the Murray and Darling rivers, all confirmed cases until January 2000 have been in northern Australia.

Scrub typhus

Organism

Orientia tsutsugamushi.

Location

The organism occurs in coastal north Queensland and Litchfield Park, 140 km south of Darwin. There are possibly other discrete rainforest patches across tropical Australia. It is widespread from South-East Asia to Pakistan.

Transmission

The organism is transmitted by bites from infected larval mites, which normally live on native rodents. It is not transmitted from person to person.

Prevention

Avoid mite exposure (with clothing and insect repellent).

Clinical description

Incubation period is 6 to 21 days (usually 9 to 12). Symptoms and signs include:

- skin ulcer with black crust (eschar) often at the bite site, usually the genitals, armpits or buttocks (Figure A)
- fever
- headache
- sweating
- myalgia
- lymphadenopathy
- truncal maculopapular dull red rash.

Disease may progress to pneumonitis, encephalopathy and multi-organ failure with bleeding. Mortality is higher in elderly patients or with delayed treatment or certain strains (e.g. Litchfield Park strain).

Diagnosis

Serology.

Treatment

Doxycycline.

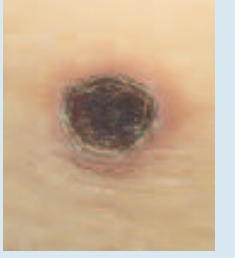

Figure A. Buttock eschar of scrub typhus, the site of a mite bite. The black area is 8 mm in diameter.

Murray Valley encephalitis

Organism

Murray Valley encephalitis virus, a mosquito-borne flavivirus of the west-Nile Japanese encephalitis antigenic complex.

Location

The virus is endemic in the Kimberley region of Western Australia and adjacent area of the Northern Territory. Occasional cases occur in Queensland and Papua New Guinea. Cases occur at the end of and just after the wet season.

Transmission

The virus is transmitted by mosquito *(Culex annulirostris),* by predominantly night biting (peak biting activity is in the first two hours after sundown). Natural hosts are birds and possibly some mammals.

Prevention

Avoid mosquito bites:

- make accommodation mosquito-proof
- wear protective light-coloured clothing
- use insect repellent containing DEET
- avoid being outdoors after dusk.

Clinical description

Patients are usually asymptomatic – possibly only 1 in 1,000 infected have clinical illness, but mortality is 20% in these patients and residual neurological disease occurs in up to 40%.

Symptoms and signs include:

- encephalitis with fever
- seizures (especially in children)
- cerebellar, spinal cord and brainstem signs (pseudo-poliomyelitis)
- tremor.

Diagnosis

Diagnosis is by serology and polymerase chain reaction (PCR) of blood and cerebrospinal fluid. EEG is abnormal but brain CT is usually normal.

Treatment

No specific drugs are used. Patients may require prolonged ventilation.

Serological studies show considerable rates of exposure in the endemic region, which is consistent with a low rate of clinical attack despite the often devastating nature of the encephalitis. Occasional cases of Australian enceph - alitis are attributed to Kunjin, a closely related flavivirus.

The management of Murray Valley encephalitis is discussed in the box on this page.

Melioidosis

Infection with *Burkholderia pseudomallei* is the most common cause of fatal community-acquired bacteraemic pneumonia in the tropical top end of the Northern Territory, where the documented incidence of melioidosis is the highest in the world.[6]

In the 1998 to 1999 monsoon season, there were 47 cases of melioidosis in the top end of the Northern Territory, including six deaths.

Burkholderia pseudomallei is also an important cause of sepsis in north Queensland and the Kimberley. The organism is an environmental pathogen, found in tropical soils and in water.

The management of melioidosis is described in the box on page 81.

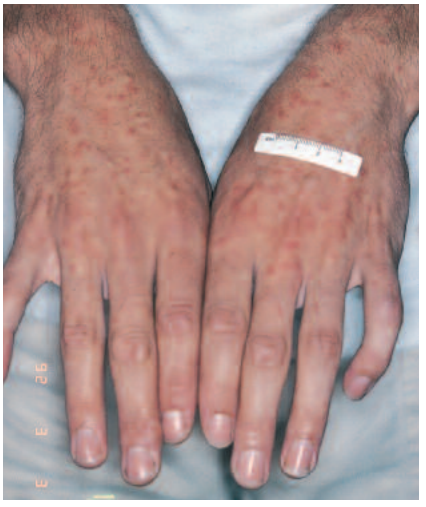

Figure 9. Maculopapular rash and small joint arthritis caused by Barmah Forest virus.

Emerging infectious diseases in Australia

Japanese encephalitis was introduced to the Torres Strait in 1995 and a case occurred on the Australian mainland in 1998.[7] Two viruses carried by bats have emerged in Queensland:

- Hendra virus in 1994 (which caused severe respiratory disease in race-horses associated with a fatal human case and another human fatality from meningoencephalitis)
- bat lyssavirus, which caused a fatal human case of rabies-like disease in 1996.

Global warming and environmental change

It is predicted that northern Australia will become warmer and wetter, extending the receptive area for malaria further south, principally by extending the range of the major mosquito vector *Anopheles farauti*. Transmission of dengue, Murray Valley encephalitis, and Ross River and Barmah Forest viruses would be facilitated, and the endemic region for melioidosis would expand southward.

It is possible, however, that the major impact on Australia would occur from social disruption and upheaval in countries that are located to the north, where further environmental destruction seems to be inevitable. History shows that population movements at times of war, famine and social unrest are a major determinant of epidemics of infectious diseases, especially malaria and other tropical diseases.

In conclusion

In the north of Australia, there are two important issues in addition to those encountered elsewhere – Aboriginal health and specific diseases of the tropics. In recent years, new infections have emerged or have been introduced, and some endemic infections have re-emerged or increased.

It is predicted that global warming

Melioidosis

Organism

Burkholderia pseudomallei (Gram-negative bacillus).

Location

The organism occurs in soil and water in northern Australia, South-East Asia and other tropical locations. The majority of infections occur in the wet season, and there are occasional introduced foci in temperate locations.

Transmission

Transmission is probably mostly percutaneous. Inhalation and (possibly) occasional ingestion are also important. Person-to-person spread is considered to be very rare.

Prevention

Use footwear and wear gloves for gardening.

Clinical description

Of cases of melioidosis:

- 85% are acute disease from recent infection (incubation period is 1 to 21 days)
- 10% are chronic disease (sick for more than two months)
- 5% result from reactivation of latent focus.

The clinical spectrum ranges from asymptomatic, through local skin ulcer (Figure B), to fulminant septicaemia. Pneumonia is most common (Figure C), but bacteraemic spread can cause abscesses in any organ, especially the spleen and prostate. Patients with diabetes, alcohol dependence or chronic renal failure are at risk of severe disease (Figure D).

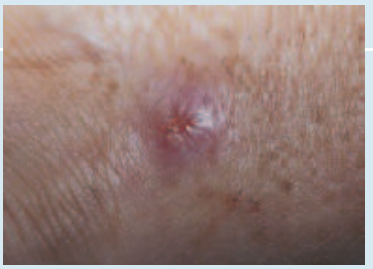

Figure B. Cutaneous melioidosis, local disease only.

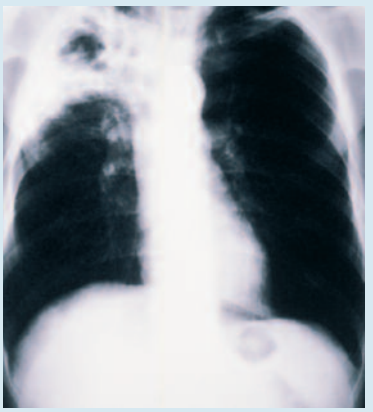

Figure C. Chest x-ray of pulmonary melioidosis mimicking tuberculosis.

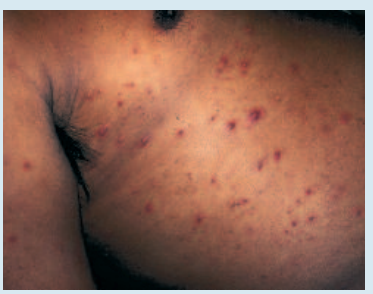

Figure D. Fatal disseminated melioidosis in a patient with undiagnosed diabetes.

Diagnosis

Diagnosis is by culture (selective media are helpful). The interpretation of serology is difficult in endemic areas.

Treatment

Initial intensive therapy (for at least two weeks) is cotrimoxazole plus one of ceftazidime (Fortum), meropenem (Merrem IV) or imipenem (in Primaxin). Subsequent eradication therapy (for at least another three months) is high dose cotrimoxazole or various combination therapies.

will lead to northern Australia becoming both hotter and wetter, the extended vector mosquito ranges and favourable environmental conditions which may well lead to a number of tropical infections being more commonly seen further south in Australia. MT

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