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Melioidosis in northern Australia, 2001–02

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Abstract

Melioidosis, caused by the Gram negative bacterium *Burkholderia pseudomallei*, is endemic in northern Australia. Using data collated from centres in Western Australia, the Northern Territory and Queensland, this report describes the epidemiology of this disease between 1 November, 2001 and 31 October, 2002. There were 47 cases seen during this period with an average annual incidence of 5.8 cases per 100,000 population. In Indigenous Australians, an incidence of 25.5 cases per 100,000 population was seen. The timing and location of cases was generally correlated with rainfall across northern Australia. A case-cluster in a Queensland community was associated with post-cyclonic flooding. Risk factors included diabetes, alcohol-related problems and renal disease. Pneumonia (51%) was the most common clinical diagnosis. The mortality rate attributable to melioidosis was 21 per cent, although a number of other patients died of underlying disease. Despite improvements in recognition and treatment, melioidosis is still associated with a high morbidity and mortality, particularly in Indigenous Australians. *Commun Dis Intell* 2003;27:272–277.

Keywords: melioidosis, Burkholderia pseudomallei, epidemiology

Introduction

Melioidosis is caused by the organism *Burkholderia pseudomallei*, a Gram negative bacterium present in soil and surface water. The disease is endemic in northern Australia and South East Asia.¹

There is a spectrum of presentations from acute sepsis to more chronic disease; infection may involve any organ but primarily involves the lungs and intra-abdominal organs. Risk factors for infection include diabetes, hazardous alcohol intake and chronic renal disease.²

The majority of cases are associated with the wet season and exposure to surface water and mud, implying that acute infection most commonly occurs soon after exposure. The incubation period of acute disease is between 1 and 21 days.³ However, latent infections with presentation delayed for months or years have also been described.³ Two outbreaks in the endemic region of Australia have been attributed to contamination of the community water supply with *B. pseudomallei*.^{4,5}

This report, using notification data, describes the epidemiology of melioidosis in the 2001–02 season in northern Australia.

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Methods

Data were collated data from the following sources: the Menzies School of Health Research, Darwin; the Tropical Public Health Unit, Queensland Health; and the Department of Health and PathCentre, Perth. Rainfall data were obtained from the Bureau of Meteorology.6 Population statistics, derived from the 2001 national census, were obtained from the Australia Bureau of Statistics.7 Locations of towns were taken from the Gazetteer of Australia, 2001.8 Cases within the northern region of Australia were included. The endemic region is generally regarded as the area north of 20°S. All cases were from within this region, except one north Queensland case from Mackay (21°10'S) where autochthonous cases had been seen in previous years.

Melioidosis is a notifiable disease in Queensland, the Northern Territory and Western Australia. A case was included if cultures from any body site were positive for *B. pseudomallei* and the patient presented with a illness

consistent with melioidosis during the period between 1 November 2001 and 31 October 2002. Location was taken from the patient's place of residence; for travellers, the place of presentation. Serological diagnoses were not included as previous work has suggested that positive serology is neither sensitive or specific in the diagnosis of melioidosis.^{9,10}

The timing of the wet season varies in northern Australia; in Western Australia and the Northern Territory, it is defined as the six month period between 1 November and 30 April, in north Queensland it is defined as from 1 December to 31 May.

Results

Epidemiology

In the 12 months to 31 October 2002 there were 47 cases of melioidosis in the northern areas of Australia: Western Australia (1 case); Northern Territory (23 cases); and Queensland (23 cases). Epidemiological features of these cases are summarised in the Table.

Table. Cases of melioidosis, northern Australia, 1 November 2001 to 31 October 2002, by state or territory

	State or territory			
	NT	Qld	WA	Total
Number of cases	23	23	1	47
Mortality	4 (17%)	7 (30%)	1	12 (26)
Attributed to illness	3	6		10
Other cause	1	1	0	2
Wet season cases*	18 (78%)	21 (95%)	1	40 (87%)
Median age (range) years	51 (3–79)	56 (30–87)	22	52 (3–87)
Male	15 (65%)	17 (74%)	1	33 (70%)
Total population	148,641	596,498	41,969	809,334
Rate (per 100,000 population per year)	15.5	3.9	2.4	5.8
Indigenous	13 (57%)	12 (52%)	0	25 (53%)
Indigenous rate (per 100,000 population per year)	42.0	22.3	0	25.5
Paediatric (<15 years)	1	0	0	1

Wet season: Northern Territory and Western Australia: November-April, Queensland: December-May; date of onset not evident for one Queensland case

In addition, a number of patients were notified but excluded; this included three patients who had been notified previously and re-presented with relapsed disease. *B. pseudomallei* was also isolated from the sputum of a 14-year-old boy with cystic fibrosis; because he was otherwise asymptomatic, it was considered that the isolation indicated colonisation, and the case was not included. One patient presented to a hospital in Perth (31°S) with an exposure history from an overseas endemic area. There were also two cases elsewhere in southern Australia where culture-confirmed melioidosis was epidemiologically linked to travel to the Northern Territory.

The median age was 52 (range 3 to 87) years and 33 (70%) were male. There was one child (3 years of age) with melioidosis during this time. There were 25 infections involving Indigenous Australians. The rate of melioidosis was 5.8 cases per 100,000 population overall and 25.5 cases per 100,000 population in Indigenous Australians.

In the Northern Territory, most cases were seen around Darwin. Ten cases were from the Darwin urban region and four from the rural areas surrounding Darwin. One patient presented to Tennant Creek Hospital (19°39'S); although locally acquired cases have been seen there previously, this patient had recently travelled from further north in the endemic area. Six patients developed their illness in remote Aboriginal communities and were transferred to Royal Darwin Hospital for further management.

In Queensland, there were three main geographical foci of cases. Seven cases (30% of the Queensland total) were from the one Gulf community, six (26%) were from Townsville and adjacent suburbs, four (17%) were from the Torres Strait and the Northern Peninsula Area, and the remaining six (26%) were from other areas. Of note, only one case was acquired in Cairns.

Rainfall and incident cases

The majority (87%) of cases were seen during the wet season months (Table). During this time, rainfall across the Top End of the Northern Territory was between 150–600 mm less than the 1961–1990, 30-year median rainfall. On the western side of Cape York, rainfall was between 75–300 mm greater than the median rainfall. The location of cases together with average annual rainfall is detailed in Figure 1.

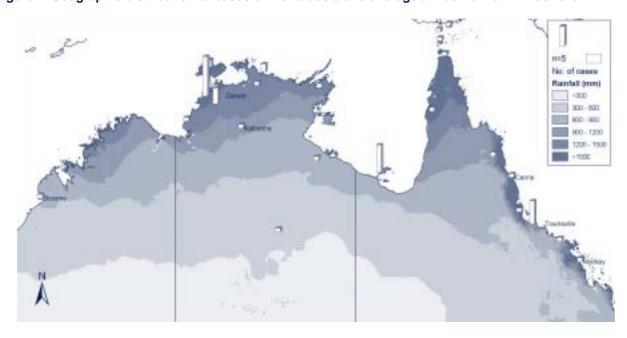
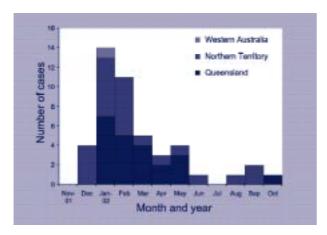


Figure 1. Geographic distribution of cases of melioidosis and average annual rainfall in Australia

In the Northern Territory, the number of cases was low compared to previous years; in the preceding 12 months there were 33 cases and in the 1997–98 season there were 48 cases. In addition, there were no cases in November for the first time since 1989; this was attributed to the lower than average rainfall. Most cases occurred during the Northern Territory wet season (n=18), with 12 cases in January and February, coinciding with the relatively late onset of the monsoon rain (see Figure 2).

Figure 2.Monthly number of cases of melioidosis, northern Australia, November 2001 to October 2002*



^{*} Date of onset not evident for one Queensland case

In Queensland, the seasonal peak started in January (see Figure 2). However, five of the seven cases in January, and seven (30%) of the total number of cases, were from the same community in the Gulf country north of Mt Isa. The first two cases at the community became unwell within three days of a tropical cyclone that passed over the community, leading to extensive flooding, in early January. Similarly, two of the three cases in Townsville in February occurred very soon after heavy rainfall in the city.

Clinical features

Risk factors for infection included diabetes (n=20; 43%), alcohol-related problems (n=14; 30%), renal disease (n=9: 19%), chronic obstructive airway disease (n=8, 17%), immunosuppression (n=4; 8.5%) and malignancy (n=2; 4.2%). Only seven patients (15%) did not have obvious medical risk factors; only two of these did not have a history of occupational or recreational exposure to mud/water.

Most cases had pneumonia (n=24; 51%) with other infections involving bone/joint (n=3; 6.3%), prostate (n=5; 11%), skin/soft tissue (n=3; 6.3%), gastrointestinal tract (n=1), spleen (n=1) and the central nervous system (n=1).

Overall, 12 patients (25%) died; 5 (41%) from the overwhelming acute infection. Two deaths were felt to be attributable to other causes (underlying end stage renal disease and malignancy). Two patients died prior to or on admission to hospital. The case fatality rate in Indigenous Australians was higher than in other patients, but this difference was not statistically significant (33% vs 17%, Fisher's exact: p=0.3).

Excluding two patients who died prior to or during admission in Queensland, and two patients with mild pneumonia who did not require admission, the remaining 43 patients spent a total of 1,182 days in hospital. The median duration of hospital stay was 18 days (range 1 to 114).

Discussion

Melioidosis is endemic in northern Australia. The average annual rate in the Top End of the Northern Territory is 16.5 cases per 100,000 population with a rate of 34.5 cases per 100,000 population in the 1997/98 season. In the Torres Strait communities in northern Queensland between 1995 and 2000, the annual rate was 42.7 cases per 100,000 population. These rates are much higher than those documented in northeast Thailand (3.5–5.5 cases per 100,000 population) and Singapore (1.7 cases per 100,000 population).

Rainfall during the 2001-02 wet season varied from previous seasons, with later and lower than average precipitation in the Top End of the Northern Territory. This was reflected in the lower number of cases in the Northern Territory, with no cases seen in November for the first time since records commenced in 1989. However, the higher than average rainfall around Cape York was not associated with increased numbers of cases in this area possibly indicating the influence of as yet undefined factors other than rainfall per se. Ongoing studies are examining the role of other environmental factors, such as rainfall rate, soil type and physical properties of drinking and surface water, in the epidemiology of melioidosis.

As previously noted,⁹ Indigenous Australians are over-represented in the melioidosis cases. In the defined area of northern Australia, it was estimated that 12.4 per cent of the total 2001 population at risk were Aboriginal and Torres Strait Islander people, whereas 53 per cent of the cases in this report occurred in Indigenous people. Although this may partly be related to exposure, risk factors such as diabetes and renal disease are also more common in this population. Other important risk factors in this and the wider population include high alcohol intake, and occupational and recreational exposures.

The prevalence of risk factors, namely diabetes, alcohol-related problems, chronic lung disease and chronic renal disease is similar to that described previously in Australia. Similarly, the clinical features of this disease, with pneumonia present at presentation in half the cases, with smaller percentages of patients with skin and soft tissue infections, osteomyelitis and genitourinary infection reflect patterns noted previously. The clinical pattern of disease varies from the Thai series, where many of the cases have no obvious clinical focus. In addition, paediatric disease is much less common in Australia in comparison to Thailand.

The diversity of presentations with melioidosis is illustrated by a number of the cases during this year. A 51-year-old man, presented to his local medical officer with impotence following a flulike illness, and a prostatic abscess was subsequently diagnosed. A 3-year-old child and presented with ataxia brainstem encephalitis following a culture-positive scalp boil. Two patients identified as having had previous mycobacterial infections, one with M. leprae and another with M. terrae; isolated case reports have noted this association that may reflect a common host susceptibility to these intracellular pathogens. 17,18,19 Additionally, the presentation of non-acute melioidosis may mimic that of tuberculosis. A number of patients in previous years had been treated for presumed tuberculosis, but subsequent cultures were negative for M. tuberculosis and positive for *B. pseudomallei* (unpublished data).

The only culture-confirmed case of melioidosis presenting in the endemic region of Western Australia during the 12 month period was a tourist, who presented following recent travel

from the Northern Territory. Despite appropriate antibiotics and intensive supportive therapy he had a rapidly fatal septic course. The occurrence of melioidosis in this and other travellers, although uncommon, reinforces the need for clinicians throughout Australia to be mindful of this disease in patients that have been in endemic areas.

The mortality from melioidosis in the Northern Territory has halved over the past decade. Historically, most deaths have been attributable to the complications of severe sepsis due to overwhelming infection.² With improvements in of melioidosis, recognition the commencement of therapy, and improved intensive care management of patients with severe sepsis, an increasing proportion of deaths are attributable to causes other than the sepsis syndrome, such as the complications of the prolonged treatment course and underlying disease.

There is considerable economic cost associated with melioidosis. Hospital admission, including the need for intensive care, is likely to represent only a fraction of the cost associated with this disease. Treatment of melioidosis often requires outpatient administration of expensive antibiotics and extensive follow-up, and may involve patients in remote settings.

Ongoing studies are aimed at determining the environmental factors important in the development of this disease, such as contamination of potable water supplies, and exploring better therapeutic strategies. Efforts are also continuing to improve the awareness of melioidosis in communities to reduce exposure to this organism in high-risk individuals during the wet season.

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