

MELIOIDOSIS IN NORTH QUEENSLAND, 2000–2009

Jeffrey N Hanna, Jan L Humphreys, Dianne L Brookes, Terrianne Messina, Alexandra Rauli

Abstract

There were 176 culture-confirmed cases of melioidosis in north Queensland over the 10 years, 2000–2009. Most (nearly 80%) occurred in the first 4 months of the year. The overall case fatality was 21%, but was 14% in 2005–2009. Of the 173 adult cases, 45% were in Indigenous adults. Both diabetes and alcohol abuse were more prevalent among Indigenous adults with melioidosis than among non-Indigenous adults. The incidences in Indigenous adults were particularly high in the Torres Strait and Northern Peninsula Area, Cape York and Mornington Island, whereas for non-Indigenous adults there appears to be a higher risk within Townsville city. *Commun Dis Intell* 2010;34(4):444–447.

Keywords: *Burkholderia pseudomallei*, Indigenous, melioidosis, Queensland, septicaemic pneumonia

Introduction

Melioidosis is an infection caused by the soil-dwelling bacterium *Burkholderia pseudomallei*, which it causes a wide range of clinical syndromes ranging from mild superficial skin infections to fulminant septicaemic pneumonia.¹ Endemic melioidosis has long been recognised in north Queensland² and it was a notifiable disease in Queensland in the 1980s and the first half of the 1990s. Although it was not a notifiable disease in the latter half of the 1990s, it became gazetted as a notifiable disease again in mid-1999. The objective of this study is to describe the salient features of the melioidosis cases that occurred in north Queensland over 10 years, 2000–2009.

Methods

Cases of melioidosis were defined by the isolation of *B. pseudomallei* from any clinical sample; serological diagnoses were not considered valid.¹ Relapses of the disease in those known to have had a previous culture-confirmed episode were not included, nor were cases known to have been acquired outside north Queensland. (There were several ‘imported’ cases from the Northern Territory and Papua New Guinea during the 10 years.)

Upon notification, a standard questionnaire was used to collect details about each case, including indigenous status, occupation, clinical details, risk factors and any apparent exposures to soil and sur-

face waters. Retirees and pensioners were included in a single occupation category, as were manual and outdoor workers. As well as the recognised risk factors for melioidosis (i.e. diabetes, alcohol abuse, chronic renal and lung diseases, malignancy and immunosuppression¹), ‘aged’ was also included. This was defined as ≥ 50 years and ≥ 65 years of age in Indigenous and non-Indigenous adults, respectively.

Acute disease was defined by duration of illness of less than 2 months but if there was uncertainty about the onset date of the more chronic cases, the first day of the apparent month of onset was used. It was assumed that the place of onset of the acute cases was likely to have been in the Health Service District that included the place of residence, with the only exception being a miner who worked remotely from his home address.

Incidence rates were calculated using the Experimental Estimated Resident Populations (ERPs) based on national census data; these ERPs have been specifically developed to define Queensland Health Service District (HSD) populations. The ERPs of Indigenous and non-Indigenous adults ≥ 15 years of age in north Queensland in 2006 were approximately 46,610 and approximately 463,970 respectively.

One- and two-sample tests of proportion were used as appropriate.

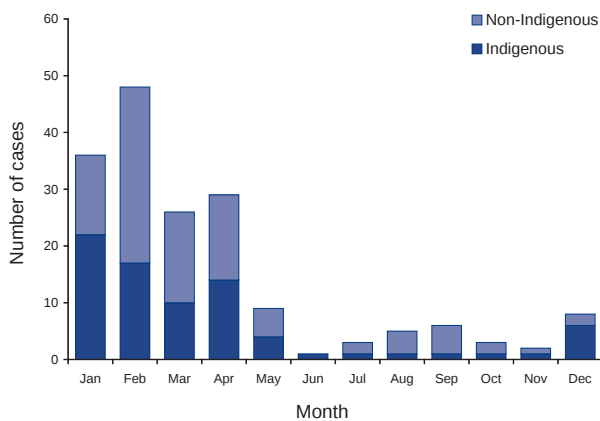
Results

There were 176 culture-confirmed cases of melioidosis in north Queensland over the 10 years, ranging from 8 (in 2003) to 38 (2000) cases per year. Of the 176, 139 (79%) had an onset in the first 4 months of the year (Figure). There were 36 melioidosis-related deaths; a case fatality of 20%. The case fatality rate in the first 5 years, 2000–2004, was 26%, compared with 14% in 2005–2009 ($P > 0.05$).

There were only 3 cases of melioidosis in children aged less than 15 years over the 10 years. An Indigenous infant, who possibly acquired melioidosis through breastfeeding, died from overwhelming sepsis at 5 days of age.³ An 11-year-old non-Indigenous boy, in otherwise good health, developed a superficial melioidosis abscess, and a 12-year-old non-Indigenous girl developed severe neurological

meliodosis with a catastrophic outcome (i.e. quadriplegia). These paediatric cases will not be considered further.

Figure: Monthly distribution of the melioidosis cases, north Queensland, 2000 to 2009



The clinical presentations of the 173 cases in those aged ≥ 15 years are given in Table 1, with many cases involving multiple organs. There were 120 (69%) patients with pneumonia and/or septicaemia, which are combined here as it was often clinically difficult to distinguish between the two.

Of the 173 adult cases, 78 (45%) were in Indigenous people. Of these, 47 (60%) occurred in 2000–2004, compared with 31 (40%) in 2005–2009 ($P < 0.01$). The melioidosis related case fatality rate in Indigenous adults was 21%.

Many patients had more than one risk factor (Table 2). The distribution of ‘aged’ persons was the

Table 1: Clinical presentations of melioidosis in adults, north Queensland, 2000 to 2009

Clinical presentation	Cases with the presentation*	
	n	%
Pneumonia	76	44
Septicaemia	76	44
Superficial tissue infection	28	16
Internal organ abscess	25	14
Urinary infection	15	9
Septic arthritis	11	6
Neurological disease	6	3
Other	6	3

* Numbers do not add up to 100% as many cases included multiple organs.

same among Indigenous (41%) and non-Indigenous cases (41%), but there were more diabetics among the Indigenous (71%) compared with the non-Indigenous cases (33%) ($P < 0.01$). Alcohol abuse was more prevalent among Indigenous adults with melioidosis (41%) than among non-Indigenous adults (25%) ($P < 0.05$).

The occupations of the adult cases are given in Table 3. A number of the retirees and pensioners volunteered that they were recreational gardeners, and seven of the manual and outdoor workers were miners.

One hundred and sixty (92%) of the adult cases were acute. The distribution of these acute cases throughout the HSDs is shown in Table 4. The average annual incidence in adults in the Townsville HSD (3.3 cases (95% CI, 2.6–4.3 cases) per 100,000 adults) was greater than that in the Cairns HSD (1.4 cases (95% CI, 0.9–2.1 cases) per 100,000 adults) ($P < 0.05$). Furthermore, whereas only 62% of the acute adult cases in the Cairns HSD were apparently acquired within Cairns city (and adjacent suburbs), 92% of the Townsville HSD cases were acquired

Table 2: Risk factors for melioidosis in adults, north Queensland, 2000 to 2009

Risk factor	Cases with the risk factor*	
	n	%
Diabetes	87	50
Aged	71	41
Alcohol abuse	56	32
Chronic respiratory disease	28	16
Chronic renal disease	21	12
Immunosuppression/transplant	19	11
No risk factors	18	10
Other	16	9
Malignancy	12	7

* Numbers do not add up to 100% as many cases included multiple risk factors.

Table 3: The occupations of the adults with melioidosis, north Queensland, 2000 to 2009

Occupation	Number of cases	%
Retiree/pensioner	68	39
Manual/outdoor worker	35	21
Unemployed	34	20
Other occupations	20	12
Home duties (females)	15	9

within Townsville city ($P < 0.01$). Seventeen (81%) of the 21 acute cases among Indigenous adults in the Mt Isa HSD were from Mornington Island.

Table 4: The distribution of acute adult melioidosis, north Queensland, 2000 to 2009, by Health Service District

Health service district	All acute cases	Indigenous acute cases
Bowen	4	0
Cairns	21	3
Cape York	17	15
Charters Towers	0	0
Innisfail	0	0
Mackay	2	0
Moranbah	1	0
Mt Isa	24	21
Tablelands	7	1
Torres Strait and Northern Peninsula Area	24	23
Townsville	60	13
Total	160	76

The average annual incidence in Indigenous adults was the same in both the Torres Strait and Northern Peninsula Area and Mt Isa HSDs: 42 cases (95% CI, 26–63 cases) per 100,000 Indigenous adults aged ≥ 15 years. The incidence in Cape York HSD was similar: 40 cases (95% CI, 22–66 cases) per 100,000 Indigenous adults.

Discussion

This 10-year prospective study has shown that many of the features of melioidosis in north Queensland are similar to those documented elsewhere from the tropical north of the Northern Territory.¹

Most (nearly 80%) of the cases are compressed into the first 4 months of the year coinciding with the height of the annual monsoonal wet season throughout the region. These months also coincide with the tropical cyclone season, and several tropical cyclones were soon followed (i.e. within a week) by ‘clusters’ of melioidosis, for example in Townsville city in 2000 (10 cases) and Mornington Island (4 cases) in 2002. However, Cape York was affected by cyclones in 2005 and 2006, but neither cyclone was followed by such clusters.

Some wet seasons were associated with a relatively small number of cases, for example in 2001 (9 cases) and 2003 (8 cases). Furthermore, there was a relative paucity of cases throughout much of the wet tropics

bioregion, from Ingham via Innisfail, Cairns and Mossman/Port Douglas north to Daintree. Indeed, no cases were reported from the Innisfail HSD (which has the highest rainfall in north Queensland) over the 10 years, even following severe Cyclone Larry in 2006.

The study has identified two very localised geographic foci, both apparently associated with increased risk of melioidosis: Townsville city (where the cases occur predominantly in non-Indigenous adults) and Mornington Island (where cases occur exclusively among Indigenous adults). The ‘high-risk’ focus of Townsville city has been recognised previously, and it has been suggested that some specific soil characteristics may contribute to this local risk.⁴

The study has also identified very high incidences among Indigenous adults in both the Torres Strait and Northern Peninsula Area and Cape York HSDs. However, in both there was no focal distribution of the cases, which were scattered throughout the islands and communities in these HSDs. Presumably, the high risk for Indigenous adults within these HSDs is more a reflection upon the high prevalence of co-morbidities, particularly diabetes, in these adults rather than any specific soil characteristics.

Similar to those documented in the Northern Territory,¹ the most prevalent risk factors for melioidosis in adults in north Queensland are diabetes, alcohol abuse and chronic respiratory disease. Diabetes was particularly prevalent among the Indigenous adult cases, and this may have contributed to the considerably higher overall prevalence of diabetes in melioidosis cases (50%) compared with that documented in the Northern Territory (37%).¹ Another potential risk factor is being ‘aged’; although the definitions differed, the percentages of aged Indigenous and non-Indigenous adults were very similar.

Clearly, the relatively high percentage of cases that were either retirees or pensioners reflects the number of cases that were either aged or had significant co-morbidities. However, it is of concern that approximately 20% of the cases were either manual or outdoor workers, suggesting that melioidosis may be a significant occupation-related infection in north Queensland, particularly in those with underlying conditions such as diabetes.

Many of the clinical features seen in north Queensland have been described previously from the Northern Territory.¹ Paediatric melioidosis is rare in north Queensland. The 12-year-old non-Indigenous girl with neurological melioidosis was very unusual for several reasons: her young age, severe disease despite the absence of underlying risk

factors, onset during a 'low risk' month (September), and she was the only case from the farming areas near Atherton during the 10 years.

The predominant clinical presentation in adults is pneumonia and/or septicaemia. Eighty-three per cent of the 36 melioidosis-related deaths in adults occurred in those with pneumonia and/or septicaemia, but all except one had significant underlying co-morbidities. The overall melioidosis case fatality was similar to that in the Northern Territory (19%).¹ Although not statistically significant, the recent decline in the case fatality is encouraging, and may be a reflection upon the recent decline in cases in Indigenous adults.

Internal organ abscesses (liver, splenic, prostatic and lung abscesses) are not uncommon, with some requiring extensive surgery. So as to recognise clinically unapparent visceral abscesses, abdominal–pelvic CT scanning of all adult melioidosis cases is recommended in the Northern Territory regardless of clinical presentation,^{1,5} but this does not yet seem to be a routine practice throughout north Queensland. Therefore there could have been an under-ascertainment of some abscesses—prostatic abscesses in particular.⁵

Superficial tissue infections (ulcers and abscesses) were also common. Twenty (77%) of those with superficial infections were non-Indigenous adults, and 9 (32%) had no underlying co-morbidities. Six of the 13 with chronic disease (duration of illness ≥ 2 months) had superficial tissue infections.

Although simple messages about melioidosis and measures that should be taken to reduce the risk of disease are publicised annually, the reality is that melioidosis is probably not a preventable condition at the current time. Indeed, with the aging population, the increase in chronic diseases, and an increasing population in north Queensland, it is quite plausible that melioidosis could actually increase over time. Clinicians in north Queensland need to maintain a high degree of suspicion of the disease in an acutely unwell adult with a systemic febrile illness, particularly if the adult is diabetic or aged or has alcohol-related problems, during the first 4 months of the year, and particularly if the

adult is a resident of Townsville city, Mornington Island, Cape York or the Torres Strait and Northern Peninsula Area.

Acknowledgement

We thank Rohan Pratt for assisting with the database and analyses, and also thank the other Public Health Unit personnel who have gathered the relevant information over the years.

Author details

Jeffrey N Hanna MPH, FAFPHM, Public Health Physician¹
Jan L Humphreys, Public Health Nursing Officer²
Dianne L Brookes MPH&TM, Public Health Nursing Officer¹
Terrianne Messina BHSc (Nursing), MPH, Public Health Nursing Officer³
Alexandra Rauli BSc (Hons), MPH, Epidemiologist¹

1. Cairns Population Health Unit, Tropical Regional Services, Division of the Chief Health Officer, Queensland Health, Cairns, Queensland
2. Townsville Public Health Unit, Tropical Regional Services, Division of the Chief Health Officer, Queensland Health, Townsville Queensland
3. Mackay Public Health Unit, Tropical Regional Services, Division of the Chief Health Officer, Queensland Health, Mackay MC Queensland

Corresponding author: Dr J Hanna, Cairns Population Health Unit, PO Box 1103, CAIRNS QLD 4870. Telephone: +61 7 4050 3600. Facsimile: +61 7 4031 1440. Email: Jeffrey_hanna@health.qld.gov.au

References

1. Currie BJ. *Burkholderia pseudomallei* and *Burkholderia mallei*: melioidosis and glanders. In: Mandell GL, Bennett JE, Dolin R, editors. *Mandell, Douglas and Bennett's Principles and Practice of Infectious Diseases*. 7th edn. Philadelphia: Churchill Livingstone Elsevier, 2010:2869–2885.
2. Rimington RA. Melioidosis in Northern Queensland. *Med J Aust* 1962;49:50–53.
3. Ralph A, McBride J, Currie BJ. Transmission of *Burkholderia pseudomallei* via breast milk in northern Australia. *Pediatr Infect Dis J* 2004;23(12):1169–1171.
4. Corkeron ML, Norton R, Nelson PN. Spatial distribution of melioidosis distribution in a suburban area. *Epidemiol Infect* 2010;138(9):1346–1352.
5. Morse LP, Moller CB, Harvey E, Ward L, Cheng AC, Carson PJ, et al. Prostatic abscess due to *Burkholderia pseudomallei*: 81 cases from a 19-year prospective melioidosis study. *J Urol* 2009;182(2):542–547.