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Acute melioidosis outbreak in Western Australia

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SUMMARY

A cluster of acute melioidosis cases occurred in a remote, coastal community in tropical Western Australia. Molecular typing of *Burkholderia pseudomallei* isolates from culture-confirmed cases and suspected environmental sources by pulsed-field gel electrophoresis (PFGE) of *Xba*I chromosomal DNA digests showed that a single PFGE type was responsible for five cases of acute infection in a community of around 300 during a 5 week period. This temporal and geographical clustering of acute melioidosis cases provided a unique opportunity to investigate the environmental factors contributing to this disease. *B. pseudomallei* isolated from a domestic tap at the home of an asymptomatic seroconverter was indistinguishable by PFGE. Possible contributing environmental factors included an unusually acid communal water supply, unrecordable chlorine levels during the probable exposure period, a nearby earth tremor, and gusting winds during the installation of new water and electricity supplies. The possible role of the potable water supply as a source of *B. pseudomallei* was investigated further.

INTRODUCTION

Melioidosis is recognized with increasing frequency in Northern Australia, where the disease is endemic. The incidence of melioidosis is higher during the wet season, at the beginning of the calendar year [1]. A range of clinical presentations is seen, including acute septicaemic, subacute and chronic disease, occurring more commonly in those with a history of diabetes mellitus, chronic renal failure or high-risk alcohol intake [2].

Although the causal role of *Burkholderia* (previously *Pseudomonas*) *pseudomallei* has been recognized for decades and exposure to contaminated soil or water has been inferred on epidemiological grounds [3–5], the precise means of exposure is still the subject of debate. In spite of the presence of *B. pseudomallei* in soil and water across the north of the Australian continent [6], and as far south as Toodyay, in Western Australia [7], only one Australian outbreak has been reported previously, involving the Northern Territory and spreading to North Queensland, during the 1990–1 wet season [8, 9]. A cluster of cases is thought to have taken place in India during 1995, though the description of this as an outbreak was subsequently questioned [10, 11]. The only other cluster of human cases reported occurred in Northeastern Thailand [12].

Melioidosis is not notifiable in Western Australia. Initial recognition of cases is therefore by informal voluntary reporting via a diagnostic laboratory net-

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work. Between two and five sporadic cases occur per year. An unusual clustering of culture-confirmed cases of acute melioidosis in a remote coastal community during the dry season prompted an urgent outbreak investigation. This report describes the detailed investigation of that cluster which was initially reported elsewhere [13], and the disease control measures that were introduced.

METHODS

A diagnostic microbiology laboratory in Western Australia confirmed a diagnosis of acute melioidosis in three patients from the same community in quick succession. An urgent investigation was planned when it became clear that major construction work had been undertaken in the community recently and in view of the imminent onset of the rainy season. The chronology of events is outlined in Table 1.

Specimen collection

The outbreak investigation team identified the homes of known cases, permanent collections of surface water, recent excavation or construction work, and water supply installations during their first visit. Soil samples (approx. 2–5 g) were taken at these locations from the moist surface or from below the surface crust if the surface had dried out. Water samples were obtained from standing water with a sterile, disposable pipette. Samples from taps and water supply pipes were run directly into the specimen container. Environmental specimens were packed in an insulated container and flown out on the day of collection for processing the next day. Large volume water (5 l), soil (1 kg) and rammed earth specimens were obtained during three subsequent visits to the community. A section of asbestos cement piping from the common water supply was also obtained for culture.

Laboratory methods

All specimens collected during the initial site visit were plated directly onto Ashdown's medium and blood agar and incubated at 37 °C in air. Selective broth media were not in stock at the time. Five litre water samples were filtered through a sterile 0·22 μm pore membrane filter, and cultured on Ashdown's medium.

Five gram aliquots of soil and a crushed portion of rammed earth block were each added to 5 ml sterile, distilled water and shaken vigorously. These were left to settle, the supernatant was added to Ashdown's broth and incubated at 37 °C. Dried biofilm from the asbestos cement pipe was scraped clear for culture.

Plates were examined after overnight incubation, daily for a further 3 days and finally, after 1 week. Suspect colonies were picked for isolation in pure culture on blood agar. Biochemical confirmation was performed using a substrate-utilization test panel (API 20NE). *B. pseudomallei* isolates from all cases were collected in the central reference laboratory for confirmation of identity and molecular typing. Genotypic confirmation was performed using a nested polymerase chain reaction protocol to amplify a *B. pseudomallei*-specific sequence as described previously [14]. Molecular typing was performed by pulsed-field gel electrophoresis (PFGE) of *Xba*I digests of chromosomal DNA from all clinical and environmental isolates by an independent reference laboratory [15]. Agarose blocks were prepared using a modification Haase and colleagues [16], and PFGE was performed with a pulse time and ramp of 5·5–52 s for 20 h at 200 V.

Epidemiological follow-up

The preliminary descriptive epidemiology was based on information obtained by the diagnostic laboratory network and the regional Public Health Unit. Active case-finding was undertaken. Further information on risk and behavioural factors (including time lived in the community, house occupied, houses visited, occupational soil exposure, footwear usage, drinking tap water and recent wounds or abrasions) was sought through a series of hypothesis-generating interviews with structured questionnaire with members of the community. Relatives were used as substitutes for deceased subjects and one survivor. Other respondents were sought among the adult members of the community. Community and cultural constraints prevented a formal case-control study from being completed. One new acute infection and one relapse were diagnosed in community residents after an interval of > 6 months.

Serological investigation

Community residents, especially those with chronic underlying disease, were offered a *B. pseudomallei* serological test in the January and February following

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Table 1. *Chronology of Western Australian melioidosis outbreak and subsequent investigations*

Date	Patient age and sex	*B. pseudomallei* culture site	Risk factors, comments	Outcome
13/11/97	52 M	Blood	NIDDM,	Recovered
27/11/97	56 F	Blood	Alcohol abuse, arrived in community 11/97	Deceased
8/12/97	40 M	Sputum	Chronic renal failure	Deceased
19/12/97	Central public health laboratory alerted by branch laboratory manager, Public Health Unit informed			
22/12/97	First outbreak investigation field trip. Soil movement, excavations and surface water located, environmental sampling. Community meetings, control measures and educational materials planned and initiated.			
23/12/97	Environmental samples set up for culture			
27/12/97	55 F	Sputum	NIDDM, wheelchair bound	Recovered
27/12/97	34 F	Blood, Bronchial washings	Rheumatic heart disease	Deceased
1/98–2/98	Sero-survey and hypothesis building interviews			
1/98–7/98	Multiple field visits, community meetings and education campaign			
7/98	Relapse of unrelated case from 2/97 one month after stopping maintenance doxycycline			
13/7/98	55 F	Blood	NIDDM, visited community in 11/97, moved away before investigation	Developed abscesses after leaving community, late onset septicaemia, recovered

the outbreak. Fortuitously, two sets of serum had been collected from some community members in August 1995 and August 1997 as part of an Australian encephalitis (AE) study and stored at −20 °C. Written consent was obtained from each individual patient to examine stored sera for the presence of antibodies to *B. pseudomallei*. An indirect haemagglutination assay for IgG antibodies to *B. pseudomallei* crude antigen was used. A titre of 40 was regarded as borderline and > 40, positive [17].

Disease control measures

A community education programme was commenced consisting of meetings, mail-outs, posters and individual education advising residents to wear footwear when outside and to attend to wounds promptly. Residents with chronic disease risk factors for infection were advised to avoid soil contact and to present early if they had symptoms of pneumonia. Immediate measures were taken to repair leaking taps and pipes, fill in puddles and prevent splash-back under outdoor taps with shell grit. Many community members left to stay with relatives elsewhere. All construction work was stopped until after the Christmas holiday. Excavated soil was allowed to grass over with the arrival of the wet season. Clinic staff were advised to obtain sputum and blood cultures and to commence intravenous antibiotic therapy early in the course of suspected acute melioidosis, for which purpose a stock of ceftazidime was provided.

RESULTS

The population of the affected community fluctuates between about 200 and 300 persons. No record has been found of any cases of melioidosis in the community before 1997. In a typical year there would be around 3–5 cases of melioidosis confirmed in Western Australia.

There have been seven culture-confirmed cases of acute melioidosis in the affected community to date (chronology, Table 1). The first of these occurred in February 1997 and relapsed in July 1998 shortly after completing a 1 year course of suppressive doxycycline therapy. A further five culture-confirmed cases occurred during a 6-week period at the end of 1997. All five had septicaemia and/or pneumonia, the dates of onset suggesting exposure to *B. pseudomallei* during November or, at latest, December 1997. Three of the five died. The seven patients had an age range of 34–57 years, four were female and three were male. All had at least one of the following chronic disease or co-morbidity risk factors: diabetes, high-risk alcohol

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Table 2. *Water quality measurements on potable water supplied to affected community prior, to, and during melioidosis outbreak*

Potable water sample date	Free chlorine (ppm)	Total coliforms	Thermotolerant coliforms	Heterotrophic plate count	Thermophilic amoebae
9/7/97	0·4	0	0	—	0
13/8/97	0·8	0	0	—	0
10/9/97	0·5	0	0	—	0
7/10/97	0·5	0	0	—	0
4/11/97	0·0	0	0	—	0
19/11/97	0·7	0	0	±	0
30/12/97	0·0	0	0	±	0
Water pH sample date	pH bore 1	pH bore 2	pH bore 3	Reticulation pH	
13/8/97	4·36	4·48	4·32	4·88	

consumption, chronic renal failure or rheumatic heart disease. The seventh case presented in August 1998 and gave a history of a brief febrile illness in December 1997, followed by multiple soft tissue lesions and finally culture-positive septicaemic melioidosis in August 1998.

Environmental and geophysical factors

The apparent clustering of acute cases in a small coastal community during the dry season suggested an unusual disturbance of soil or water in the settlement. Construction work had taken place throughout the community shortly before the case cluster occurred. The work included fabrication of rammed earth homes and extensive trenching for underground power cables and water pipes. Recently turned soil was found where the new cables and water pipes had been laid. New homes were made with red soil from a large borrow pit near the airstrip, and formed by mixing with concrete at the construction site. House building had taken place for 1 year up to the time when the cases presented. Heaps of rammed earth debris were left outside new homes.

There were permanent puddles in the main borrow pit and below taps and pipes outside most homes despite hot, dry weather for weeks prior to the first site visit. The water supply to the settlement came from a series of capped bore-holes located 2 km to the east, feeding via a common underground pipe into collecting tanks, before passing through a chlorination system to the water main. Available chlorine levels fell to zero during November, around the beginning of the presumed exposure period (Table 2). A pH of 4·36–4·88 was recorded in routine bore water analysis in August 1997. The main water pipes were noted to leak at several points due to the corrosive effects of acid water. During November a contractor had accidentally punctured the prechlorination main pipe at one point while digging a trench for a replacement pipe. Rainfall was twice the average during the 1996/7 wet season and there was 380 mm rain in May 1997, which is usually a dry month (source: Australian Meteorological Bureau). As a result the water table was unusually high. The land crossed by the main water pipes was low-lying with mangrove encroachment. Winds greater than 30 km/h in 1·3 standard recordings reaching up to 47 km/h were noted during November 1997 at a weather station 5 km from the community. In August 1997 an earthquake occurred measuring 6·3 on the Richter scale with an epicentre 120 km north of the affected community (source: Australian Geological Survey Organisation). Residents felt buildings shake at the time.

Microbiology

B. pseudomallei was isolated from all seven patients. *B. pseudomallei* was also isolated from water dripping from a back yard tap and from the 5 l pre-chlorination bore water sample. No other environmental specimens (including 14 soil specimens from sites around homes of cases, 16 other soils, 4 water specimens from sites around homes of cases, 9 other surface water samples, 10×5L water specimens [4 from capped bores], 2 samples of rammed earth block and 1 water pipe) contained detectable *B. pseudomallei*.

Isolates from all seven patients and both environmental specimens were confirmed by PCR as *B.*

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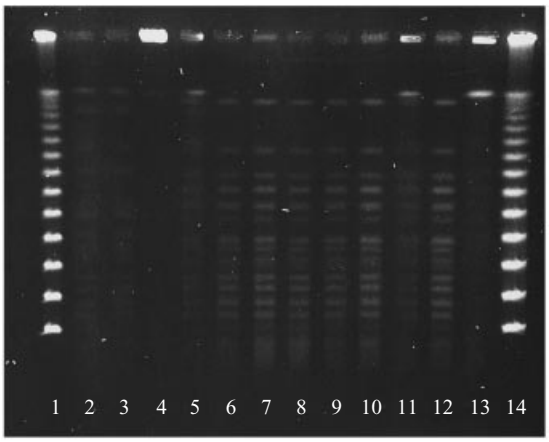

Fig. 1. PFGE of *Xba*I digests of *B. pseudomallei* DNA. Lanes 1 and 14 contain molecular marker ladders. Lanes 2 – 13 correspond to isolates as follows: 2 and 3, patient presenting in February 1997 and relapsing in July 1998; 4–11, six subsequent patients; 12, water from back yard tap; 13, bore water pre-chlorination.

pseudomallei. The isolates from the patient who first presented in February 1997 and relapsed in July 1998 were indistinguishable by PFGE, but distinct from the other isolates (Fig. 1). All isolates from the remaining six patients and the back-yard tap were indistinguishable by PFGE. The single isolate from the pre-chlorination bore water belonged to a distinct type that was different from all other clinical and environmental isolates tested.

Epidemiological follow-up

The principal cluster of five cases lived in four different groups of homes. Four were present in the community between September and December 1997. One arrived in mid-November. As four of the five cases had pneumonia, respiratory exposure was considered possible. None of the substitute interviewees reported recent penetrating skin injury or slow healing sores in their infected relative. Only one case went about barefoot and one was a double amputee who had to use a wheelchair. None of the cases had a history of occupational soil exposure including manufacture of rammed earth blocks, but 14 healthy interviewees reported such exposure. Three cases gardened for at least 1 h per day. All five cases drank tap water without boiling it first, while some community members boiled tap water prior to drinking. Around 1000 people attended a cultural festival at the community in October 1997. No new cases of acute melioidosis have occurred in neighbouring communities since the present outbreak, other than the one who visited the affected community during the presumed exposure period then moved away before investigations began. This one late onset case (number seven) was the only patient to develop features of soft tissue infection.

Serology

Ninety-five individuals provided serum samples between November 1997 and February 1998. Twelve of these, including the five cases, were positive (12·6%). There were two positives among the 19 pairs of sera from 1997–8; one with a titre of 80 for both 1997 and 1998 samples and one with a seroconversion from 20 in 1997 to 320 in 1998. Neither developed symptoms of acute melioidosis. The patient with asymptomatic seroconversion during the outbreak period was resident in the house from whose tap water *B. pseudomallei* was isolated.

Disease control

The fifth case was diagnosed as the outbreak investigation began. There were no further cases of either acute or subacute, culture-confirmed melioidosis during the ensuing wet season. Only two additional cases have been diagnosed in the region since the introduction of control measures. One of these was a relapse of an unrelated infection, and the other appears on clinical data to have been a late onset septicaemia complicating chronic soft tissue infection contracted around the presumed exposure period.

DISCUSSION

The present cluster of acute melioidosis cases from a small, isolated community in Western Australia has the epidemiological and bacteriological features of a discrete outbreak. PFGE established the presence of a single *B. pseudomallei* genotype in six acute cases and one geographically-related environmental sample. The outbreak was remarkable on several counts. No such temporal or geographical clustering of acute melioidosis had previously occurred in Western Australia. These features implied the presence of novel precipitating or contributory factors. Furthermore, the dry season timing of this outbreak appeared to contradict the putative role of bacteria in soil moistened by heavy rain.

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In contrast, the previous Australian acute melioidosis outbreak occurred during the 1990–1 wet season, when unusually high rainfall may have caused proliferation of *B. pseudomallei* in the soil [8]. Occupational and recreational soil exposure were epidemiologically associated with disease. *B. pseudomallei* was demonstrated in soil samples collected at the time. Ribotyping was used to type *B. pseudomallei* isolates from the 1990–1 Darwin outbreak, but no identical environmental isolate was linked by geographical location to any of the clinical cases and there was considerable diversity among the clinical isolates [18]. Although a more recent study of *B. pseudomallei* isolates from clinical and environmental sources in several countries argued that the relationship between environmental and clinical strains has not been established [19], soil, human and animal isolates have been shown to be indistinguishable in a recent Australian study [20].

The environmental sampling strategy we took was designed to identify surface sites heavily contaminated with *B. pseudomallei*; possible sources for direct contact or aerosol formation. We concentrated on places where soil was permanently wet or moist at the surface, where deep soil had been raised to the surface by recent excavation, and water outlets. The laboratory methods used were not as sensitive as recently reported enrichment methods or PCR-based gene amplification techniques [6]. Consequently, results were more specific to higher levels of environmental contamination. Our finding of the same molecular type of *B. pseudomallei* in clinical strains and a water sample collected during the course of the outbreak strengthens the case for a putative environmental exposure. Although it proved to be a different subtype of *B. pseudomallei* from the outbreak strain, the strain present in the pre-chlorination bore water highlighted a possible source of contamination of the communal water supply. Bore water was also implicated in a recent study from Thailand in which the persistence of *B. pseudomallei* near to a bore-hole supplying a rice farm during the dry season was noted [21].

One of the reasons for repair work on the affected community's water supply was corrosion of underground pipes by acid bore water. The low pH was thought unlikely to allow growth of *B. pseudomallei* whose pH survival range has been reported as 5–8 [22]. However, soil would be expected to have a buffering effect on water leaking from pipes. The wet soil surrounding leaking pipes would thus have been more suitable for proliferation of *B. pseudomallei*, creating a micro-environment similar to the proposed effect of a rising water table during the wet season [6, 12, 23]. The reason for the unusually low water pH is unclear, but it may resemble the water supply to coastal communities in the Northern Territory where low pH is caused by high concentrations of dissolved carbon dioxide from a combination of plant roots and silica-based sandstones [24]. Additional soil-water admixture may have been caused by the earth tremor, or by accidental puncture of the water main during replacement work. The protective effect of chlorine will have been negated by the chlorination failure that coincided with the outbreak. The extensive excavation going on throughout the community during the most likely period of exposure cannot be ruled out as a contributory factor.

The community water supply was thus considered a possible means of bacterial distribution. It was not possible to pursue this hypothesis as far as indoor water outlets because access was not granted to the homes of melioidosis cases. Circumstantial epidemiological findings hint at a possible role for ingestion of contaminated tap water, or inhalation of wind-driven, moist, excavated soil, but stand against direct skin inoculation. Seroepidemiology supported the presence of at least one asymptomatic exposure around the time of the outbreak but did not indicate community-wide subclinical infection. It is notable that this case was the resident of a home with culture positive water. Since a proportion of acute cases of melioidosis do not seroconvert [25], seroprevalence data are liable to underestimate the total disease burden in a community.

It is notable that there have been no further cases during a wet season that began with a cyclone passing through the area. Measures taken during the initial investigation led to prompt acute management of the fifth case and may have helped bring the outbreak to a halt. However, the much longer incubation period of chronic suppurative melioidosis leads us to treat this observation with caution. It remains possible that the seventh case, the sixth in the cluster, may have been caused by skin inoculation. Infection by local skin inoculation via cuts and abrasions may lead to further cases of late onset or chronic infection if *B. pseudomallei* is more widely distributed in the human environment than we have shown. Antibiotic intervention was avoided as a control measure owing to uncertainty over its role and the choice of agent.

The short duration and abrupt end to the recent outbreak of acute melioidosis in Western Australia

provided a clearly defined period in which to study potential exposure to *B. pseudomallei* and provided a unique opportunity to investigate the environmental determinants of infection with this organism.

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