MELIOIDOSIS IN SOUTHERN INDIA: EPIDEMIOLOGICAL AND CLINICAL PROFILE

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Abstract. Melioidosis, which is mainly prevalent in Thailand and Australia, has shown an increasing trend in India in the last few years. We carried out a retrospective study of 25 culture-proven adult cases of melioidosis who were admitted to a tertiary care hospital in southern India during June 2001 to September 2007. There was a six-fold increase in the number of cases in 2006 and 2007 as compared to 2001. Diabetes mellitus was the predisposing factor in 68% of cases, followed by alcoholism (28%). The clinical presentations were fever (80%), pneumonia and/or pleural effusion (48%), hepatomegaly (56%), joint involvement, and/or osteomyelitis (48%), splenomegaly (40%), splenic abscess (24%) and septicemia (28%). The organism, *Burkholderia pseudomallei*, was sensitive to co-amoxiclav, cotrimoxazole, ceftazidime, and carbapenem. The study suggests that melioidosis is an emerging infectious disease in the southwestern coastal belt of India, and it is likely to happen at much higher incidence.

Key words: melioidosis, epidemiology, clinical profile, India

INTRODUCTION

Melioidosis is endemic in Southeast Asia and northern Australia (Chaowagul *et al*, 1989; Hsueh *et al*, 2001; Currie *et al*, 2008). Melioidosis is an emerging infectious disease in India as is evident from case reports from different parts of India (John *et al*, 1996; Anuradha *et al*, 1997; Sengupta *et al*, 1998; Mathew *et al*, 1999; Rao and Shivananda, 1999; Jesudasan *et al*, 2003; Navaneethan *et al*, 2006; Vidyalakshmi *et al*, 2007; Saravu *et al*, 2008). *Burkholderia pseudomallei*, an environmental pathogen present in soil, infects individuals with predisposing factors like diabetes mellitus and alcoholism (Suputtamongkol *et al*, 1999; Currie *et al*, 2000b; Currie, 2003; White, 2003; Currie *et al*, 2004). It has a spectrum of manifestations that range from superficial skin abscesses to multiple visceral abscesses and presentations that vary from chronic debilitating illness to overwhelming septicemia.

The objective of this study was to identify the epidemiological and clinical profile of cases of melioidosis presenting to a tertiary care teaching hospital in Southern India. The research questions were as follows:

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1) What are the risk factors associated with melioidosis, including occupation, 2) is there any seasonal trend in presentation of clinical cases, 3) what are the clinical and laboratory features at the time of presentation, 4) what is the antibiotic susceptibility pattern of *B. pseudomallei*, and 5) what are the outcomes of treatment?

MATERIALS AND METHODS

This study was carried out in a tertiary care institute in southern India. Twenty-five individuals with culture proven melioidosis in the previous seven years were studied retrospectively with special reference to clinical features, occupation, epidemiology, and treatment. Specific information was collected from patient's medical records using a standardized format that included baseline demographic, laboratory, and microbiological data. Risk factors for melioidosis, such as occupation, chronic alcohol consumption, history of steroid intake, and concomitant diseases, were identified. In all individuals, the culture isolate was identified by the API ID 32 GN® commercial kit. Histopathology was done in a few patients by embedding the tissues in paraffin wax, and 5 μm sections were stained with hematoxylin and eosin.

Disseminated disease was defined as the one that affected more than one organ system. Septicemic melioidosis was defined as blood culture proven melioidosis with sepsis. Septic shock was described as sepsis with hypotension. Data analysis was done using Statistical Package for Social Sciences (SPSS®, version 10).

Ethical considerations

Ethical clearance was obtained from the University Ethics Committee, Manipal University (Ref No. UEC/30/2008, 2008/Oct/17).

RESULTS

Of the 25 patients, 80% were males, with an age range of 18-67 years (median: 45 years, IQR: 32-54). Fourteen (56%) were ≥45 years old. All patients were from the coastal regions of Kerala (3), Karnataka (18), and Goa (4). Seventeen (68%) patients presented during the monsoon season (Jun-Sep, Fig 1), and 14 (56%) patients had a history of heavy exposure to wet soil or surface water. In all, 3 cases were detected in 2001 and in 2005, but from 2006 to 2007; however, the number rose to 11 cases each year (Fig 2), which was approximately a six-fold increase by the end of the seven-year period.

Predisposing factors (Table 1) were present in 21 (84%) patients, which included diabetes mellitus (17, 68%) and alcoholism (7, 28%). Other predisposing factors included AIDS (1), chronic lung disease (1), chronic kidney disease (1), and on chemotherapy for malignancy (1). Four patients (16%) had no predisposing factors. By occupation, 4 were farmers, 7 were doing office jobs, and others were a manual laborer (1), civil contractor (1), electrician (1) and fisherman (1) who also had exposure to soil/surface water.

Patients had varying presentations (Table 2). Seventeen (68%) patients had disseminated melioidosis, 7 (28%) patients had septicemic melioidosis, and 4 (16%) had only skin abscesses. Fever was the most common complaint and was present in 20 (80%) patients. Six patients (24%) had chronic presentation with fever of >60 days, and 7(28%) patients presented with fever of 15 days or less. Seven (28%) patients with septicemic melioidosis had fever ranging from 1 to 60 days. Three of them had skin and soft tissue involvement; one had chronic osteomyelitis, and another, mediastinal lymphadenopathy.

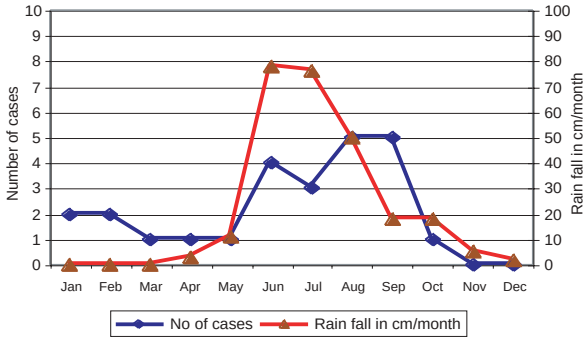

Fig 1–Seasonal distribution of melioidosis and rain fall.

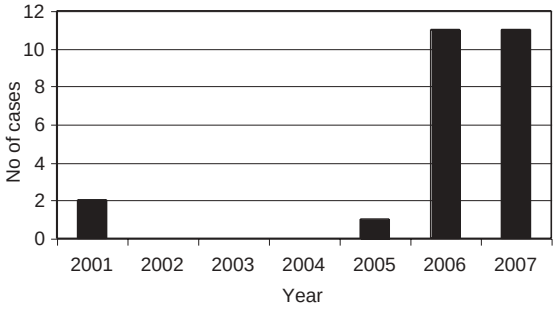

Fig 2–Annual distribution of cases, 2001-2007.

Table 1
Predisposing factors for melioidosis.

Predisposing factor	% (*n* = 25)
Diabetes mellitus	68
Alcoholism	28
Chronic lung disease	4
HIV	4
Chronic kidney disease	8
Chemotherapy for malignancy	4
Exposure to soil/farming	32
No predisposing factor	16

Table 2
Different clinical features in the study subjects.

Clinical features	% (*n* = 25)
Fever	80
Pneumonia and/or pleural effusion	48
Hepatomegaly	56
Splenomegaly	40
Skin and soft tissue involvement	32
Joint involvement	28
Septicemia	28
Splenic abscess	24
Osteomyelitis	20
Ascitis	16
Prostatic abscess	8
Pericardial effusion	4
Subdural collection	4
Mediastinal lymphadenopathy	4
No clinical focus found (only septicemia)	16

Five (20%) patients had pneumonia, and 9 (36%) patients had mild pleural effusion, one of which was loculated empyema. Four patients (16%) with hepatomegaly had multiple liver abscesses. and 6 (24%) patients with splenomegaly had splenic abscesses. Four (16%) had soft tissue swellings as the only clinical manifestation. We had patients with rarer presentations, such as ascites (16%), prostatic abscess (8%), pericardial effusion (4%), subdural collection (4%), and mediastinal lymphadenopathy (4%). No clinical focus was found in 4 (16%) patients, and they presented only with septicemia. A CT scan of the abdomen and pelvis to look for hidden abscesses was not done in all patients because of financial considerations. Seven (28%) patients had septic shock. Three (12%) patients had received empirical antitubercular therapy either in our hospital or elsewhere prior to admission. Mantoux test was positive in 2 (8%) out of 5 patients who had the procedure. Chest x-rays

Table 3
Previous reports of Melioidosis from India.

Author	Location	Year	Cases (*n*)
Shenoy *et al*	Mangalore, (Karnataka)	2009	1
Saravu *et al*	Manipal, (Karnataka)	2008	7
Valsalan *et al*	Manipal, (Karnataka)	2008	1
Krishnan *et al*	Bangalore, (Karnataka)	2008	1
Rajinikanth *et al*	Vellore (Tamil Nadu)	2008	1
Dhodapkar *et al*	Pondicherry	2008	1
Thomas	Hyderabad (Andhra Pradesh)	2008	1
Kumar *et al*	Vellore (Tamil Nadu)	2008	6 (1998-2005)
Mukhyopadhyay	Manipal (Karnataka)	2007	2
Vidyalakshmi *et al*	Mangalore (Karnataka)	2007	25 (2005-2006)
Navaneethan *et al*	Orissa	2006	1
Jesudason *et al*	Vellore (Tamil Nadu)	2003	28 (1993-2002)
Kanungo	Pondicherry (Tamil Nadu)	2002	1
Mathew *et al*	Vellore, (Tamil Nadu), Belgaum (Karnataka), Nagaon (Assam), Midnapur (West Bengal), Bihar	1999	5 (1995-1998). All the cases were detected at one center (Vellore)
Rao *et al*	Manipal (Karnataka)	1999	1
Sengupta *et al*	Manipal (Karnataka)	1998	1
Anuradha *et al*	Hyderabad, (Andhra Pradesh)	1997	1
John *et al*	Kerala (1), Orissa(1), Tripura(2), Tamil Nadu(1), Maharashtra(1)	1996	6 (1993-1995). All the cases were detected at one center (Vellore)
Paneth *et al*	Trichur, (Kerala)	1995	1
Raghavan *et al*	Maharashtra	1991	1

showed pneumonia in 5 (20%) patients, pleural effusion in 9 (36%) patients, and bilateral nodular opacity and mediastinal widening in one (4%) patient each, and was normal in 13 (52%) patients. In 3 (12%) patients, it presented like tuberculosis and empirically ATT was administered.

Hemoglobin less than 10g/dl and ESR more than 100 mm at the end of 1 hour were seen in 16 (64%) patients, and leukocytosis was seen in 14 (56%) patients. Serum CRP and ferritin were elevated in 6 (24%) and 8 (32%) patients, respectively. Liver functions revealed significantly elevated alkaline phosphatase of more than 300U/l in 12 (48%) patients, and only 2 (8%) patients had transaminases of more than 150 U/l. Acute onset renal function abnormality was present in 5 (20%) patients.

B. pseudomallei was grown in all patients in pus cultures aspirated from the liver, spleen, mediastinal lymph node, ascites, subcutaneous swellings, or joint effusions. Blood cultures were positive in 9 (36%) patients. Median duration prior to diagnosis was 10 days (IQR 5-17) after presenting to us. Bone marrow showed granuloma in one (4%) patient. Aspirated material from liver abscess showed caseating granuloma in another (4%).

The isolate from the patients was sensitive to amoxiclav, cotrimoxazole, chloramphenicol, piperacillin, cefotaxime, ceftazidime and meropenem and resistant to gentamicin. Patients were treated according to their sensitivity patterns. Fifteen (60%) patients were given multidrug therapy. Empirical therapy usually included parenteral co-amoxiclav. However, once the diagnosis was confirmed, patients received parenteral ceftazidime or carbapenem initially, followed by oral combination of co-amoxiclav and cotrimoxazole, or cotrimoxazole and doxycycline. Parenteral antibiotics were given for a mean duration of 14 days (SD ± 4days). Mean time for defervescence was 8.5 days (SD ± 2.5days). Mean duration of treatment was 5 months (SD ± 1.2 months). One patient underwent splenectomy because of persisting splenic abscesses, following which the patient was cured. Histopathology of the spleen revealed stellate granulomas. *B. pseudomallei* was cultured from the splenic tissue. Two (8%) patients died; one with HIV infection who succumbed to septicemic melioidosis and the other to candidal sepsis. Three patients were discharged against medical advice, and at the time of discharge, they were ill and could not afford further treatment. Twenty patients were discharged on oral medications, out of which, 16 were available for follow-up. Nine (36%) patients have completely improved with the treatment and are well with mean duration of follow-up of 8 months. Seven (28%) patients were receiving eradication phase treatment.

DISCUSSION

The hospital where the study was conducted is located on the western coast of Karnataka, a South Indian state. The hospital is a 1,400-bed tertiary-care teaching hospital that caters to a population of over 1 million and receives referred cases from the adjoining states of Kerala and Goa. The center is a tertiary referral center, and hence there may be a referral bias. Most of the secondary hospitals do not have the facilities to culture nor the expertise to identify *B. pseudomallei*. Melioidosis has been increasingly detected in our hospital since 2006. Cases have come from three southern states of India, mainly from the western coast. There was a male preponderance as was found in other studies, probably related to outdoor work (Puthucheary *et al*, 1992; Heng *et al*, 1998; Currie *et al*, 2000b). An age of >45 years was present in majority and has been documented as an independent risk factor (Currie *et al*, 2004). In our study, the cases were clustered during the rainy season, from May to September. *B. pseudomallei* is present in soil and surface water in endemic regions. Humans are infected by percutaneous inoculation, inhalation, and ingestion (White, 2003). In Thailand and northern Australia, 75-85% of the cases occur in rainy season (Currie *et al*, 2000b). Bacteria that move to the surface with the rising water table during the rainy season are responsible for the increase in the transmission rate (Thomas *et al*, 1979). In our study, 32% of the patients with melioidosis were farmers or had exposure to soil. Diabetes and excessive alcohol consumption were documented as the main predisposing factors that corroborate with other studies (Currie *et al*, 2004; Chan *et al*, 2005). The rate of diabetes in our study (68%) was higher when compared to the rates of other studies, which range from 23 to 60% (Chaowagul *et al*, 1989; Punyagupta *et al*, 1989; Suputtamongkol *et al*, 1999; Currie *et al*, 2004). Excess alcohol consumption has

been documented in 12-37% of melioidosis cases as compared to 28% in our study (Suputtamongkol *et al*, 1999; Currie *et al*, 2004). Although melioidosis does not seem to be associated with HIV infection (Dance, 2000; Chierakul *et al*, 2004), we had one individual presenting with the septicemic form. In an Australian study, chronic renal disease, chronic lung disease, and age >45 were independent risk factors for melioidosis (Currie *et al*, 2004). In our study, chronic lung disease and chronic renal disease were predisposing factors in 4% and 8%, respectively, which is less when compared to findings of 27% and 10%, respectively, in another study (Currie *et al*, 2000b). No predisposing conditions were identified in 13-36% cases in other studies (Suputtamongkol *et al*, 1999; Currie *et al*, 2004) as compared to 16% in the our study.

Melioidosis can present acutely or may follow chronic indolent course; it may present in disseminated or localized forms with or without septicemia. In our study, pneumonia, pleural effusion, skin and soft tissue swellings, and liver and splenic abscesses were the common presentations. Various studies have shown that pneumonia is the commonest clinical presentation (Chan *et al*, 2005). Neurological involvement, though less common, can be seen in 4% of cases. They can have brain stem encephalitis, cerebral abscess, cranial nerve palsies, and paraparesis (White, 2003). One (4%) of our patients had subdural collection and presented with hemiplegia and altered sensorium.

Melioidosis is a great masquerader, and it is often confused with staphylococcal abscesses in acute form or tuberculosis in chronic presentations. Mantoux test results may be positive, as it was in two of our patients. Aspirated pus may be confused with caseous material of tuberculosis. Tissue biopsy may show granuloma. In our series, granuloma was demonstrated in bone marrow, liver aspirate, and the spleen. Three of our patients had received empiric anti-tubercular therapy (ATT). In the present series, 36% were bacteremic. The incidence of bacteremic melioidosis has been 46% to 60% in different series (Currie *et al*, 2000b).

Mortality rate in our series was 8%, and it was 22% in patients with bacteremic melioidosis. In previously reported studies, overall mortality ranged from 19 to 46% (Suputtamongkol *et al*, 1999; Currie, 2003), while in septicemic form it went up to 65% (Puthucheary *et al*, 1992). Rate of relapse is around 10% and can be more if antibiotic therapy is shortened to eight weeks or less (Currie *et al*, 2000a).

The sensitivity to third generation cephalosporins, especially ceftazidime, doxycycline, piperacillin, cotrimoxazole, and co-amoxiclav and resistance to aminoglycosides, first- and second-generation cephalosporins, macrolides, and rifampicin is unusual and unique to this bacillus. Fluoroquinolones are also weakly sensitive. Growth of pseudomonas species with sensitivity to ceftazidime, amoxicillin clavulanic acid, and co-trimoxazole, and resistant to gentamicin should prompt us to specifically look for this species.

The recommended antibiotic regimens are a) co-trimoxazole at 8/40 mg/kg (≤320/1,600 mg) every 12 hours and ceftazidime at 50 mg/kg (≤2 g) intravenously every 6 hours, or b) meropenem at 25mg/kg (≤1 g) intravenously every 8 hours. The duration of therapy for intensive phase is at least 14 days, and a longer duration of 4-8 weeks for deep-seated infection, osteomyelitis, or septic arthritis

(Cheng and Currie, 2005). The other recommended regimen are imipenem at 20 mg/kg intravenously every 8 hours or ampicillin-clavulanate at 20/4 mg/kg intravenously every 4 hours (White, 2003).

The subsequent eradication phase antibiotic regimens are a) co-trimoxazole at 8/40 mg/kg, every 12 hours for 3-6 months (Cheng and Currie, 2005); b) chloramphenicol 10 mg/kg orally every 6 hourly for 8 weeks, doxycycline at 2 mg/kg orally twice a day for at least 20 weeks, and co-trimoxazole at 5/25 mg/kg orally twice a day for at least 20 weeks, or c)amoxicillin-clavulanate at 30/15 mg/kg orally (tds) for 20 weeks and amoxicillin at 30 mg/kg orally (tds) for 20 weeks (White, 2003).

Melioidosis in India has been reported since 1991 (Table 3), when it was detected in a child (Raghavan *et al*, 1991). A series of 28 patients of septicemic melioidosis over 10 years has been reported from Vellore (Jesudasan *et al*, 2003). The same center had reported this infection in 6 patients in 1996, where patients were from Tamil Nadu, Maharashtra, Kerala, Orissa, West Bengal, and Tripura (John *et al*, 1996). Recently 25 cases of melioidosis were reported from Karnataka, which included patients from Kerala (Vidyalakshmi *et al*, 2007). Thus, melioidosis and the causative organism are being recognized gradually in various centers in India. However, it is mostly under-recognized and under-reported. In the present study, it is interesting to note that there has been a drastic increase in number of positive cases since 2006, which may be partly attributed to the increasing detection rate due to a higher-index of suspicion.

The majority of the Indian population eats rice, and consequently, there are vast paddy fields. Being a tropical country, one-third of a year will have monsoons with heavy rains in most places. However, melioidosis is diagnosed increasingly only from the southern coastal part of India. The diagnosis of melioidosis requires clinical vigilance and an intensive microbiological workup. With this study, we hope to raise the awareness of clinicians to the existence of melioidosis in India. To recommend prophylactic measures, environmental studies are required to know the prevalence of the etiological organism in the environment.

REFERENCES

Anuradha K, Meena AK, Lakshmi V. Isolation of *Burkholderia pseudomallei*: an emerging pathogen in India. *Indian J Med Microbiol* 1997; 15: 1-2.

Chan KP, Low JG, Raghuram J, Fook-Chong SM, Kurup A. Clinical characteristics and outcome of severe melioidosis requiring intensive care. *Chest* 2005; 128: 3674-9.

Chaowagul W, White NJ, Dance DA, *et al*. Melioidosis: a major cause of community acquired septicemia in northeastern Thailand. *J Infect Dis* 1989; 159: 890-9.

Cheng AC, Jacups SP, Gal D, Mayo M, Currie BJ. Melioidosis epidemiology and risk factors from a prospective whole population study in northern Australia. *Trop Med Inter Health* 2004; 9: 1167-74.

Cheng AC, Currie BJ. Melioidosis: epidemiology, pathophysiology, and management. *Clin Microbiol Rev* 2005; 18: 383-416.

Chierakul W, Rajanuwong A, Wuthiekanun V, *et al*. The changing pattern of bloodstream infections associated with the rise in HIV prevalence in northeastern Thailand. *Trans R Soc Trop Med Hyg* 2004; 98: 678-86.

Currie BJ, Fisher DA, Anstey NM, Jacups SP. Melioidosis: acute and chronic disease, relapse and reactivation. *Trans R Soc Trop Med Hyg* 2000a; 94: 302-4.

Currie BJ, Fisher DA, Howard DM, *et al*. En-

demic melioidosis in tropical northern Australia: a 10-year prospective study. *Clin Infect Dis* 2000b; 31: 981-6.

Currie BJ. Melioidosis: an important cause of pneumonia in residents of and travelers returned from endemic regions. *Eur Respir J* 2003; 22: 542-50.

Currie BJ, Jacups SP, Cheng AC, Fisher DA, Anstey NM, Huffam SE, *et al.* Melioidosis epidemiology and risk factors from a propective whole population study in northern Australia. *Trop Med Int Health* 2004; 9: 1167-74.

Currie BJ, Dance DAB, Cheng AC. The global distribution of *Burkholderia pseudomallei* and melioidosis: an update. *Trans R Soc Trop Med Hyg* 2008; 102 (suppl 1): 1-4.

Dance DA. Melioidosis as an emerging global problem. *Acta Trop* 2000; 74: 115-9.

Dhodapkar R, Sujatha S, Sivasangeetha K, Prasanth G, Parija SC. *Burkholderia pseudomallei* infection in a patient with diabetes presenting with multiple splenic abscesses and abscess in the foot: a case report. *Cases J* 2008; 1: 224.

Heng BH, Goh KT, Yap EH, Loh H, Yeo M. Epidemiological surveillance of melioidosis in Singapore. *Ann Acad med Singapore* 1998; 27: 478-84.

Hsueh PR, Teng LJ, Lee LN, *et al.* Melioidosis: an emerging infection in Taiwan? *Emerg Inf Dis* 2001; 7: 428-33.

Jesudasan MV, Ambarasu A, John TJ. Septicaemic melioidosis in a tertiary care hospital in South India. *Indian J Med Res* 2003; 117: 119-21.

John TJ, Jesudason MV, Lalitha MK, *et al.* Melioidosis in India: the tip of the iceberg? *Indian J Med Res* 1996; 103: 62-5.

Kanungo R, Pradhan P, Battacharya S, Srimannarayana J, Jayanthi S, Swaminathan RP. Melioidosis: a report from Pondicherry, South India. *J Assoc Physicians India* 2002; 50: 1438-9.

Krishnan P, Fernandes S, Savio J, *et al.* Melioidosis. *J Assoc Physicians India* 2008; 56: 636-9.

Kumar GS, Raj PM, Chacko G, Lalitha MK, Chako AG, Rajshekhar V. Cranial melioidosis presenting as a mass lesion or osteomyelitis. *J Neurosurg* 2008; 108: 243-7.

Mathew S, Perakath B, Mathew G, *et al.* Surgical presentation of melioidosis in India. *Natl Med J India* 1999; 12: 59-61.

Mukhyopadhyay C, Dey A, Bairy I. Atypical presentations of melioidosis as emerging threat: a case report. *Indian J Pathol Microbiol.*2007; 50: 933-6.

Navaneethan U, Ramesh AC, Ravi G. Multiple visceral abscess in a case of melioidosis. *Indian J Med Sci* 2006; 60: 68-70.

Paneth N, Twigg G, Cherian T, *et al.* Plague in India [Letter to Editor]. *Lancet* 1995; 345: 258-9.

Punyagupta dÍ Melioidosis: review of 686 cases and presentation of a new clinical classification: In: Punyagupta S, Sirisanthana T, Stapatayavong B, eds. Melioidosis. Bangkok: Bangkok Medical, 1989: 217-29.

Puthucheary SD, Parasakthi N, Lee MK. Septicaemic melioidosis: a review of 50 cases from Malaysia. *Trans R Soc Trop Med Hyg* 1992; 86: 6835.

Raghavan KR, Shenoi RP, Zaer F, Aiyar R, Ramamorthy R, Mehta MN. Melioidosis in India. *Indian Pediatr* 1991; 28: 184-8.

Rajinikanth J, Balaji V, Gaikwad P, Muthusami JC. Melioidosis of the parotid: the tip of the iceberg. *ORL Head Neck Surg* 2008; 139: 731-2.

Rao PS, Shivananda PG. *Burkholderia pseudomallei:* abscess in an unusual site. *Indian J Pathol Microbiol* 1999; 42: 493-4.

Saravu K, Vishwanath S, Kumar RS, *et al.* Melioidosis-a case series from South India. *Trans R Soc Trop Med Hyg* 2008; 102 (suppl 1): 18-20.

Sengupta S, Murthy R, Kumari GR, *et al.* *Burkholderia pseudomallei* in a case of hepatic abscess. *Indian J Med Microbiol* 1998; 16: 88-9.

Shenoy V, Kamath MP, Hegde MC, D'Souza T, Mammen SS. Melioidosis and tuberculo-

sis: dual pathogens in a neck abscess. *J Laryngol Otol*.2009; 21: 1-3.

Suputtamongkol V, Chaowagul W, Chetchotisakd P, *et al*. Risk factors for melioidosis and bacteremic melioidosis. *Clin Infect Dis* 1999; 29: 408-13.

Thomas A, Forbes-Faulkner J, Parker M. Isolation of *Pseudomonas pseudomallei* from clay layers at defined depths. *Am J Epidemiol* 1979; 110: 515-21.

Thomas J, Jayachandran NV, Shenoy C, Lakshmi V, Narasimulu G. Melioidosis: an unusual cause of septic arthritis. *Clin Rheumatol* 2008; 27 (suppl 2): 59-61.

Valsalan R, Sheshadri, S, Pandit V. Melioidosis masquerading as enteric fever. *Trans R Soc Trop Med Hyg* 2008; 102 (suppl 1): 117-8.

Vidyalakshmi K, Shrikala B, Bharathi B, Suchitra U. Melioidosis: an under-diagnosed entity in western coastal India: a clinico-microbiological analysis. *Indian J Med Microbiol* 2007; 25: 245-8.

White NJ. Melioidosis. *Lancet 2*003; 361: 1715-22.