

# PSEUDOMONAS PSEUDOMALLEI PNEUMONIA WITH SEPTICEMIA – CASE REPORT

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## ABSTRACT

A case of *Pseudomonas pseudomallei* pneumonia with septicemia is described. The onset was insidious with paucity of systemic symptoms except fever. Diabetes mellitus and alcoholism were associated problems. Initially blood cultures were negative but subsequently *P. pseudomallei* was isolated. The outcome was fatal. Unless diagnosed early and treated appropriately, patients often succumb to septicemic shock.

**Key words:** pseudomonas, pseudomallei, pneumonia, diabetes, alcoholism.

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## INTRODUCTION

Since the first description by Whitmore in 1912 of infection in man by the organism *Pseudomonas pseudomallei*, the disease melioidosis has been reported from many parts of the world especially Asia and Australasia. From Jan 1987 to Aug 1988, 27 cases of culture-positive melioidosis have been identified by the Bacteriology Department, Singapore General Hospital. Most patients died before the culture results were available. The presentation as a septicemia with foci of infection in any organ is very variable and has no consistent features to enable a definite clinical diagnosis to be made. Associated illnesses like diabetes mellitus and alcoholism are common especially in cases reported from Australia (1).

The case is reported with a view to arousing awareness of this disease in diabetic and possibly alcoholic patients presenting either with PUO or pneumonia.

## CASE REPORT

A 55-year old male presented in June 1988 with 10 days history of intermittent fever associated with chills and rigors. He gave no history of cough. He was a known diabetic on oral medication for years. He was also known to consume approximately two 750 ml – bottles of beer a day over the past 20 years. He had a history of allergy to ampicillin.

Clinical examination revealed an ill man with temperature recordings revealing an irregular fever with bimodal distribution at times. The highest temperature recorded was 40.5°C. The liver edge was 2 cm below the right costal margin with stigmata of chronic liver disease. A soft haemic murmur was detected over the precordial region. There were no chest signs. Rest of the systems were essentially normal. A chest x-ray showed a right upper zone opacity consistent with a pneumonia (fig. 1).

Total white blood cell count was 9,500/uL (polymorph 90%); liver function studies: total protein 5.7 gm/dl, albumin 2.8 gm/dl, bilirubin 1.1 mg/dl, alkaline phosphatase 347 u/l, SGPT 41 u/l (normal 9-36 u/l) and SGOT 31 u/l (normal 15-33 u/l). Abdomen ultrasound showed no significant abnormality. Blood urea, electrolytes, haemoglobin, platelets, cerebrospinal fluid examination, urine microscopy and echocardiogram were normal. Gram stain and Ziehl Neelsen stain of sputum were non-contributory. Anaerobic and aerobic blood cultures, sputum and urine cultures were negative. He was put on parenteral ceftazidime and amikacin. Despite the treatment, his fever continued to swing. On the 5th hospitalisation day, anti-tuberculosis treatment was instituted with rifampicin 450 mg, INH 300 mg and ethambutol 1500 mg daily.

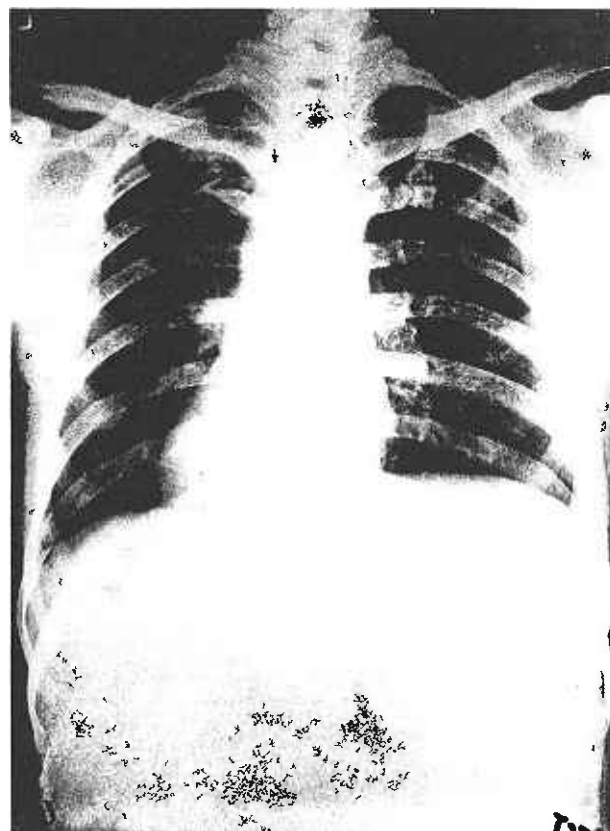


Fig. 1

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Three days after the anti-tuberculosis therapy, he became jaundiced and developed signs of early liver failure. Anti-tuberculosis therapy was discontinued and he responded to liver failure therapy. His diabetes was stabilized with insulin. He had intermittent attacks of retention of urine and had to be catheterized. No obvious cause for this could be found. Rectal examination was normal.

Repeat chest x-ray after 14 days of treatment showed radiological clearance of the pneumonia (fig. 2). However, he continued to have a swinging fever. Ceftazidime and amikacin were stopped. Blood cultures were repeated. Twenty-four hours following stoppage of antibiotics, he went into septicemic shock and perished on the 21st day of hospitalisation. The blood culture results available only after death reported the isolation of *Pseudomonas pseudomallei* which was sensitive to ceftazidime (Fortum) and equivocally sensitive to amikacin.



Fig. 2

## DISCUSSION

The clinical manifestations of melioidosis are variable. The illness can present as an acute, subacute or chronic process. In this case, he presented as an acute illness with symptoms of fever and intermittent acute retention of urine. It has been reported that pyrexia can be the main clinical presentation on admission. The fever is often in excess of 38.9°C and may be associated with rigors.

The extensive clinical reviews of human melioidosis cases from Australia and overseas (2, 3, 4) have noted many common features and all stress the importance of associated pathology in these patients. Diabetes mellitus

and alcoholism are the most frequent associations. Others are malignancies, immuno-suppression and inter-current infective illnesses. Our patient had diabetes mellitus and alcoholism with liver impairment. The review by Rode and Webling showed that 49% of their patients had a recent history of a penetrating or slowly healing wound or an inflammatory skin lesion (4). We could not find any evidence of this in our patient.

The pulmonary manifestation of the disease is the most common form encountered in the review by Webling (4) and other investigators in Southeast Asia (5, 6). The portal of entry of the bacterium in this form of the disease is unknown. Direct inhalation or hematogeneous spread from the gut (4, 7) have been postulated. Most patients with pulmonary melioidosis complain of cough and productive sputum (2). The upper lobes are commonly involved with the radiographic appearance of consolidation. There are no distinguishing clinical or radiological features in these patients to differentiate from other forms of pneumonia. Pulmonary melioidosis may mimic pulmonary tuberculosis in lesions in the upper lobe (6) (fig 1). Acute retention of urine may occur in acute melioidosis with urinary tract involvement and prostatitis (8). The urinary symptoms of our patient could have been due to involvement of the genito-urinary tract. Primary involvement of the genital tract is an important form of this disease in Aborigines in Northern territory of Australia (4). Hepatomegaly and jaundice are well documented in acute septicemic melioidosis (4, 9). Drug induced hepatitis from INH and rifampicin occurs in the early weeks of treatment and consists mainly of elevated serum transaminases (10); clinical jaundice is unusual following 3 days of anti-tuberculosis chemotherapy in this case.

It has been reported that blood cultures are often negative in *Pseudomonas pseudomallei* septicaemia, yet blood culture was the only means of making a definite diagnosis in this patient and this was also the experience of Guard et al (1). Serology is useful confirmatory technique (7). However, delayed seroconversion and the interpretation of elevated IFA-IgM titres in the absence of appropriate clinical findings remain unresolved problems.

As *P. pseudomallei* is resistant to most antibiotics commonly used in the initial treatment of undiagnosed septicemic illness, a high index of clinical suspicion is required in dealing with patients presenting with septicaemia in endemic areas. Even when a powerful anti-pseudomonal cephalosporin, ceftazidime, was administered, the patient still succumbed to his illness. This may suggest that a cephalosporin — aminoglycoside combination used commonly in undetermined septicaemia — is inadequate to treat *P. pseudomallei* septicaemia. Based on clinical suspicion treatment should be with tetracycline in high dosage in combination with chloramphenicol and kanamycin. More recently cotrimoxazole has been shown to be effective. *P. pseudomallei* is usually sensitive in vitro to tetracycline, chloramphenicol, kanamycin, amikacin, trimethoprim — sulfamethoxazole, novobiocin and the third — generation anti-pseudomonal cephalosporins, especially ceftazidime (Fortum). It is in most instances resistant to penicillin, ampicillin, vancomycin, gentamicin and second generation cephalosporins. Current recommendations are that treatment should continue for a prolonged period, but the optimal length of time has not been defined. The majority of such patients receive treatment for about 3 to 6 months.

The overall mortality rate for septicemic patients is about 75% (4, 12, 13).

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