TUBERCULOSIS OR MELIOIDOSIS? - LOOK TWICE IN SOUTHWESTERN COASTAL INDIA

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ABSTRACT

Melioidosis is known endemic in many Southeast Asian countries, especially Thailand, and in Northern Australia. The disease was long considered under-recognized in India but has now gained the status of emerging infectious disease here. Increasing isolation of the causitive agent *B. pseudomallei* is encountered in the recent years from this part of western coastal India. We report one such case of pulmonary melioidosis in an elderly patient misdiagnosed as tuberculosis ending fatally. Prompt microbiological diagnosis prevents the unnecessary Anti-TB treatment or prophylaxis. This was also our first of isolation of *B. pseudomallei* from endotracheal aspirate.

Key words: Burkholderia pseudomallei, Melioidosis, Pneumonia, Tuberculosis

INTRODUCTION

Melioidosis is an endemic disease in Southeast Asia, especially in Thailand and also in Northern Australia. Humans acquire infection by exposure to *Burkholderia pseudomallei* present in soil and surface water.[1] Clinical presentations range from localized infections to fulminant septicaemia. Lung is the ommonest organ involved in nearly 48% of cases.[2.] Pulmonary melioidosis can present as mild undifferentiated pneumonia, which can be acute or subacute in nature to fulminant septic shock with high mortality. Subacute and chronic presentations mimic pulmonary tuberculosis clinically and radiologically making differentiation difficult.[3] There has been increasing reporting of cases from south-western coastal India, majority of which are from tertiary care centers.[4–6.] A combined effort of microbiologists in prompt identification of the bacilli, testing with the right antibiotics and a high index of suspicion from the clinicians leading to appropriate diagnosis and management is required in TB endemic areas such as India.[10]

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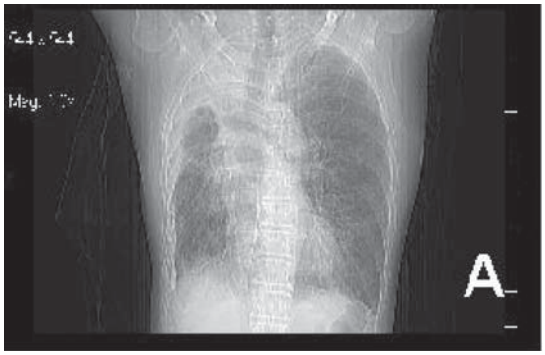
Case report

An 80-year-old retired school teacher diagnosed with ischemic heart disease (IHD), hypertension and multi-infarct dementia for 2 years, on treatment, presented with sudden onset of fever and severe breathlessness for 3 days. At presentation, he was semi-conscious, cyanosed with bilateral crepitations all over the chest. He was provisionally diagnosed as a case of pulmonary tuberculosis 2 years ago with cough, breathlessness, elevated ESR and suggestive radiological findings on chest x-ray (Figure 1 A) and CT scan (Figure 1 B) though microbiologically there was no evidence of mycobacteria in sputum microscopy, culture and



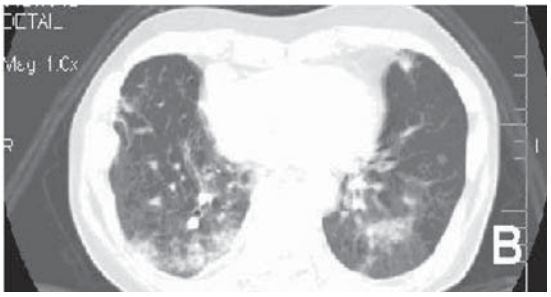


Figure 1 A, B: Chest X ray and CT scan in 2006 at the time of 1st presentation.

PCR. The patient had refused the planned anti-tubercular therapy. Further interrogation revealed that he had similar intermittent breathlessness with chest discomfort for last 4 years. He was non diabetic, did not smoke or consume alcohol. He was a bare foot walker and a snuff (a finely powdered tobacco used for inhalation by rural folk in India) abuser for 30 years.

During the current admission, the patient was ventilated with falling saturation. Investigations revealed elevated blood urea, creatinine and alkaline phosphatase levels. The chest X-ray showed air space opacities with radiolucent areas within the left paracardiac region suggestive of consolidation with breakdown signifying active disease (Figure 1 C). The unconfirmed working diagnosis at this stage included pneumonia with adult respiratory distress syndrome (ARDS) or reactivation of tuberculosis. He was empirically started on azithromycin and piperacillin-tazobactum along with other supportive therapy. However bilateral opacity in the serial chest X-ray (Figure1 D) showed progression and clinical condition worsened which were also thought to be result of ventilator associated pneumonia (VAP) and antimicrobial therapy was switched over to meropenem, teicoplanin and metronidazole on 5th day of ventilation. Meanwhile, the first 2 samples of ET aspirate had no growth after 48 hours; 3rd sample on 5th day of ventilation grew B. pseudomallei, sensitive to ceftazidime, piperacillin, co-trimoxazole, doxycyclin, meropenem, and resistant to amoxicillin-clavulanic acid, ciprofloxacin and gentamicin. The isolate had the typical safety-pin appearance on gram stain, and was biochemically identified and confirmed by API (biomeruix, France) as B. pseudomallei. Fungal culture of ET aspirate, AFB and blood culture were sterile. Although the patient showed a very brief clinical improvement with meropenem initially, he developed hypotension with falling platelets and rising serum creatinine levels despite aggressive therapy and succumbed to multi-organ dysfunction/ septic shock secondary to pulmonary melioidosis on the 13th day of hospitalization.

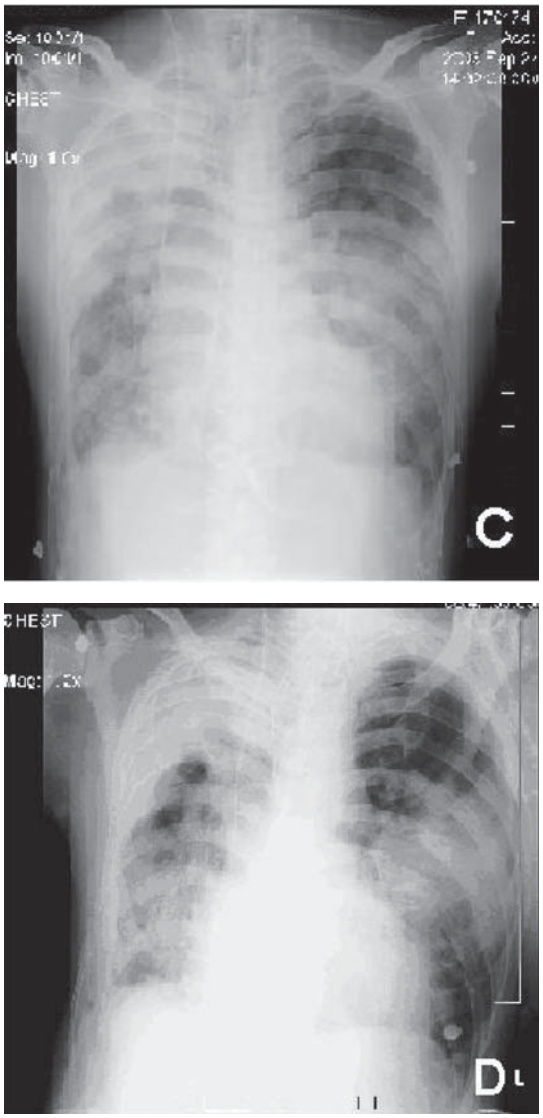


Figure 1 C, D: Chest X ray in 2008 at the time of last presentation at admission and day of expiry

DISCUSSION

Pulmonary melioidosis mimics tuberculosis both clinically and radiologically and is often misdiagnosed as tuberculosis, more so in endemic countries like India where both diseases coexist.[2] Patients with chronic pulmonary melioidosis also have fever, weight loss and productive cough, sometimes with haemoptysis like TB. Pleuritic

chest pain occurs in half of the patients.[8] Disease may often slowly progressive over months like TB. It can also be remitting and relapsing over many years, and acute deterioration with septicaemia may also occur**.** On chest radiography, they often have diffuse nodular infiltrates throughout both lungs, which coalesce, cavitate and progress rapidly, consistent with caseous necrosis and multiple metastatic abscess formation. Acute pneumonia with upper lobe consolidation in endemic regions warrants consideration of melioidosis**.**[3,8,9] The loss of volume on the right lung due to fibrothorax, pleural calcification and fibrocavitatory changes with consolidatory changes in right upper lobe and basal segments of lower lobes and areas of patchy opacity in bilateral posterior segments in CT scan (Figure 1 B) and on chest X-ray (Figure 1 A) support the earlier reports that pulmonary melioidosis can have varied radiological findings, which make it more difficult in arriving at the right diagnosis and often mistaken for TB.[3]

B. pseudomallai is a natural inhabitant of soil and water in tropics and subtropics. Inoculation through skin abrasions from contaminated soil, inhalation, aspiration are the various modes of transmission[7]. The mode of acquisition in this case might have been through inhalation or through an unnoticed inoculation attributed to bare foot walking for many years.

B. pseudomallai is isolated from wide range of specimens like blood, body fluids, sputum, pleural fluid, pus, wound swab in the laboratory, and our first time from ET aspirate.[9] This case marks out the disease as a true ticking 'Vietnamese time bomb' probably from many years where it followed a chronic course with a rapid fulminant outcome. Since the course of community-acquired pneumonia by *B.* pseudomallei is associated with high mortality rate, it becomes life saving to arrive at an accurate faster diagnosis. The co-morbid conditions of the patient like the advanced age, pre-existing hypertension, IHD and chronic obstructive pulmonary disease (COPD) collectively might have contributed to the fatal outcome.

The laboratory diagnosis of *B.pseudomallei* is not difficult if promptly looked for. It grows in 24-48hrs on the routine culture media used in laboratories like the Blood and Macconkey agar. Simple biochemical manual tests for non-fermenting gram-negative bacilli like a positive oxidase test, motility, polymixin –B resistance and dihydrolysis of arginine are sufficient for the lead and can be further confirmed by automated instruments if available. Molecular confirmation is not routinely necessary, more so in developing countries.

Therapy for Melioidosis too is also long term like TB. The initial intensive therapy consists of Ceftazidime, 50 mg/kg up to 2g, every 6 h or Meropenem, 25 mg/kg up to 1 g, every 8h or Imipenem, 25 mg/kg up to 1g, every 6h with or without Trimethoprim/sulfamethoxazole, 8/40mg/kg up to 320/1600 mg, every 12 h for a minimum of 14 days. The recommended eradication therapy includes Trimethoprim/sulfamethoxazole, 8/40 mg/kg up to 320/1600 mg, every 12 h with or without Doxycycline, 2 mg/kg up to 100 mg, 12 h for a minimum of 3 months. Accurate microbiological and clinical diagnosis is important in pulmonary melioidosis, which may be rapidly fatal in patients, especially with the existing pre-morbid conditions. Respiratory care practitioners may well be aware of the same that B.pseudomallei is always a potential pathogen and not to disregard it as a colonizer of the respiratory tract. TB in India many times stands over-diagnosed leading to unnecessary administration of Anti TB therapy. Rifampicin, an Anti-TB drug, is a known sterilizer and false clinical improvement in pulmonary melioidosis may sometimes be encountered. The burden, endemicity and public health importance of melioidosis may not be at present comparable to the magnanimity of TB for the formulation of national programs in our country for its diagnosis and prevention. Nevertheless, judicious judgment from the clinicians is required to look beyond TB in South Western Coastal India, an increasing place for isolation of *B*. pseudomallei.

ACKNOWLEDGMENT

Our special thanks to the deceased patient and his family, Indira Bairy, Professor, Department of Microbiology and Dr. Smithi, Dept of Radiology, Kasturba Medical College and Hospital, Manipal.

The publication of this case report has been approved by the relatives of the deceased patient.

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