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Case Report

A case of disseminated melioidosis: clinical insights and management

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ABSTRACT

Melioidosis is an infectious disease of humans and animals resulting from infection with the gram-negative organism Burkholderia pseudomallei. Known as the "great mimicker", melioidosis presents with a wide spectrum of disease including isolated cutaneous/pulmonary disease, primary bacteremia, visceral abscesses or fulminant fatal septicaemia. The mean incubation period is nine days (1-21 days) but symptoms can evolve more quickly (<24 h) following inhalational and/or presumed aspiration events. In acute disease, sepsis syndrome is common; >50% of patients are bacteremic at presentation and 20% develop septic shock. Pulmonary involvement is the most common acute infection in adults responsible for >50% of presentations, while pneumonia is seen in approximately 20% of paediatric cases. Presentations in children more frequently have skin involvement (60%), compared with 13% in adults. In our case report, patient was a 41year-old male, resident of Ratnagiri district, farmer by occupation, presented with fever for 1 month, cough and breathlessness for same period. He was successfully treated with meropenem and doxycycline and put on maintenance therapy for duration of 12 weeks.

Keywords: Melioidosis, Burkholderia pseudomallei, Pneumonia, Sepsis, Fatal, Diabetes mellitus

INTRODUCTION

Melioidosis, also known as Whitmore's disease, is an infectious disease caused by a bacterium, Burkholderia pseudomallei (*B.pseudomallei*, previously known as Pseudomonas pseudomallei).[1] Melioidosis, as a disease, has strong associations with climatic conditions and is found in tropical and subtropical regions globally, occurring predominantly in Southeast Asia, Northern Australia, China and Taiwan.[2,3] South Asia is estimated to have 44% of the global disease burden. Among South Asian countries, Bangladesh and Sri Lanka are considered endemic for melioidosis; a few cases have been reported from Nepal.[4] India has experienced an increase in numbers of melioidosis cases in the recent years.[4] The predisposition to melioidosis in those with diabetes, hazardous alcohol use or chronic renal disease likely reflects impairment of their innate immune function. The disease mainly affects adults (median age-5th decade) with poorly controlled diabetes in the Indian subcontinent-the diabetic capital of the world. Up to 80% of melioidosis cases in India, Sri Lanka and Bangladesh are diabetics.[4] Additionally, a study on genetic susceptibility to pneumonia during COVID-19 categorized India as having a moderate genetic risk. Taking this into consideration, it could be ascertained that environmental factors and lack of awareness constitute risk factors for melioidosis. Genetic studies related to melioidosis, similar to those in Thailand, could be useful for India.[5] The signs and symptoms are non-specific and overlap with several diseases, including tuberculosis. Thus melioidosis, 'the great mimicker' of many diseases, is grossly underdiagnosed and underreported across the tropics, including India. In South

Asia, where tuberculosis is widely prevalent, any disease like tuberculosis is misdiagnosed, leading to a delay in proper management. The organism is easily mistaken as Pseudomonas species in microbiology laboratories and may be dismissed as a common laboratory contaminant. The poor diagnostic sensitivity of blood culture also leads to missed diagnosis. Hence, both clinical ignorance and missed laboratory diagnosis have misrepresented melioidosis as a rare entity.[4] The clinical presentation is varied and the infection may be acute or chronic, localized or disseminated. It is mainly characterized by formation of abscesses, especially in the lungs, liver, spleen, skeletal muscle and prostate.[6]

Melioidosis is a considerable cause of fatal community-acquired pneumonia and septicemia in endemic regions, with mortality rates as high as 44%. Acute pulmonary infection is the most commonly diagnosed form of melioidosis. Pneumonia may be asymptomatic or may present as a severe necrotizing disease.[6] Here we discuss a case of 41-year-old male, resident of Ratnagiri district, a known case of type 2 diabetes mellitus who came with complains of fever, breathlessness, cough for one month.

CASE REPORT

A 41-year-old male, resident of Ratnagiri district, a known case of type 2 diabetes mellitus for 12 years on oral anti-diabetic agents, presented with fever of one month duration associated with episodes of chills which was associated with cough with breathlessness for same duration. He also complained of dull aching generalized abdominal pain which started along with fever. It was also associated with generalized weakness with decreased appetite.

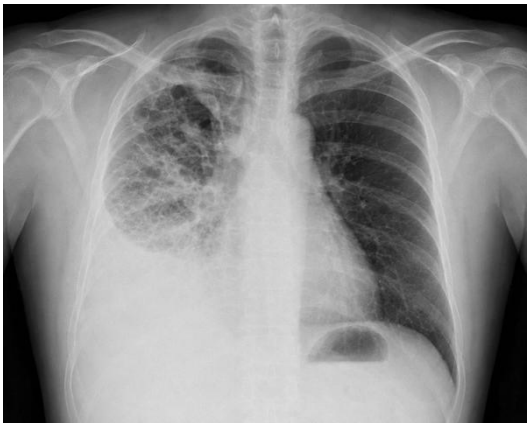

Figure 1: Chest radiograph (PA view) showing a right upper lung cavitary lesion with surrounding consolidation, associated bronchiectatic changes and right pleural effusion.

Notably, he had a recent hospitalization for same complaints. Symptoms temporarily receded but relapsed and then patient came to our hospital for further management. On detailed history taking, patient had a past history of pulmonary tuberculosis 8years ago, for which he had completed full course of anti-tubercular treatment for 6 months. There was no history of hypertension, bronchial asthma or ischemic heart disease. He had no history of addiction to any substances.

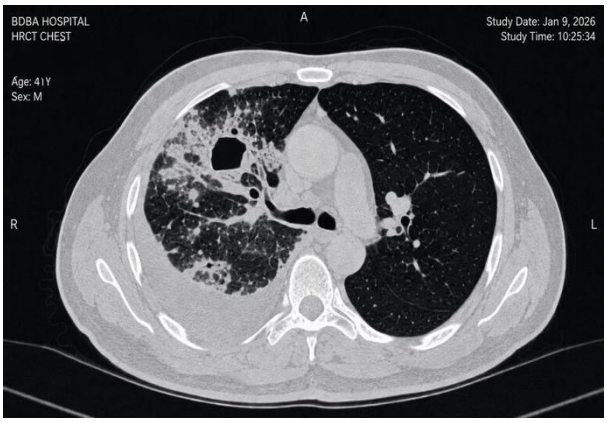

Figure 2: Axial HRCT chest image (lung window) showing a right upper lobe cavitary lesion with surrounding patchy consolidative opacities and associated right pleural effusion.

On examination, the patient was conscious and oriented, with tachycardia (pulse rate 125/min) and blood pressure of 122/92 mm Hg. Random blood glucose was markedly elevated (321 mg/dl) and HbA1C was 10.3 indicating poor glycemic control. Oxygen saturation was 92% on room air and maintained at 98% on oxygen by nasal prongs. On systemic examination revealed right sided crepitations with bronchial breath sounds, normal heart sounds, soft non-tender abdomen and no focal neurological deficits. Laboratory investigations revealed moderate anemia with hemoglobin levels around 8 g/dl, and peripheral smear showed anisocytosis. Total leukocyte count was elevated (15800/cu.mm), with neutrophilia and relative lymphopenia, with normal platelet counts. Inflammatory markers were significantly elevated, with C-reactive protein of 128.1 mg/l and D-dimer of 956 ng/ml, suggesting ongoing systemic inflammation. Renal function tests were within normal limits. Screening for tropical infections, including dengue, leptospirosis, malaria and enteric fever were negative. Sputum studies for Acid Fast Bacilli (AFB) and GeneXpert was negative.

Urine analysis demonstrated glycosuria (3+), proteinuria (2+) and microscopic hematuria (10-15 RBCs per high power field (HPF) and 10-15 pus cell/HPF. Chest radiography revealed homogeneous opacity in the right lower zone with obliteration of the costophrenic angle, consistent with right-sided pleural effusion (Figure 1). High-resolution computed tomography of the thorax demonstrated right-sided pleural effusion. A cavitatory lesion measuring approximately 22.2×34.9×24.2 mm was identified in the right apicoposterior segment, with adjacent tractional bronchiectasis and air–fluid level. Additionally, multifocal patchy consolidations and

ground-glass opacities were noted on right lung, along with mediastinal lymphadenopathy (Figure 2).

Ultrasonography of abdomen and pelvis showed splenomegaly (14.3 cm) with a hypoechoic lesion suggestive of splenic abscess or cyst. Both kidneys demonstrated increased cortical echogenicity with preserved corticomedullary differentiation, along with bilateral hydroureteronephrosis and a 10 mm calculus in the left kidney. The urinary bladder showed significant post-void residual urine (~510 ml), and the prostate was borderline enlarged (~25 cc).

Blood culture yielded growth of Burkholderia pseudomallei. Antibiotic susceptibility testing showed sensitivity to ceftazidime, carbapenems, doxycycline, minocycline, piperacillin–tazobactam and amoxicillin–clavulanic acid, while resistance was noted to cotrimoxazole, polymyxin-B and tetracycline.

The patient was managed with supplemental oxygen therapy, strict glycemic control and intravenous antibiotics tailored to culture sensitivity, including meropenem and doxycycline for 4 weeks. Further then doxycycline was given for long duration as maintenance therapy for 12 weeks. Supportive care and close monitoring were provided given the extent of systemic involvement. Patient was followed up in the outpatient department and his symptoms relieved over the duration of treatment. Repeat ultrasonography showed resolution of splenic lesions and prostatic enlargement. Chest Xray showed resolution of opacity in lung fields.

DISCUSSION

B. pseudomallei are small, gram-negative, oxidase-positive, motile, aerobic bacilli that reside in soil, water and plants in endemic regions.[7,8] Transmission of *B. pseudomallei* occurs primarily through percutaneous inoculation and inhalation/aspiration. Ingestion is a common route of infection in grazing animals and a recent study suggested that for humans, ingestion of *B. pseudomallei* from unchlorinated domestic water supplies and other water sources such as rivers may be more common than previously thought.[7,8] The environmental factors contributing to melioidosis include monsoons, skin abrasions, occupational hazards and physical injuries such as thorn pricks. Studies indicate that male agricultural workers are particularly vulnerable to melioidosis.[9]

Manifestations of melioidosis vary and can include localized to multifocal infection with or without septicemia. Though the most common presentation is pneumonia with symptoms similar to pulmonary tuberculosis it can also present as encephalomyelitis, septic arthritis, osteomyelitis and skin and visceral organ abscesses involving renal, splenic, prostatic and hepatic sites.[10] The disease may also manifest as a cutaneous form, either due to a primary skin infection or dissemination from another infected organ. Skin lesions range from papules and nodules to pustules and ulcers, resembling symptoms seen in other infectious diseases such as tuberculosis, plague, anthrax, cat scratch disease and sporotrichosis.[11] Familiarity with different presentations of this infection is essential for early diagnosis as delayed diagnosis contributes to increased mortality and morbidity. Melioidosis is associated with a range of mortality from 10-39%. For those with septic shock, it can rise up to 86%.[12-14]

B. pseudomallei exhibits resistance to penicillin's, amino-glycosides and relatively insensitive to macrolides and flouroquinolones.[14,15] Ceftazidime and carbapenems remain the drugs of choice during the intensive phase therapy. Use of meropenem especially in severe sepsis is advocated. Cotrimoxazole with or without doxycycline is used for the prolonged eradication phase. Doxycycline should not be used as monotherapy as drug resistance is expected.[15] Adherence to therapy (24-week course of therapy) is the major factor that prevents relapse.

CONCLUSION

This case represents disseminated melioidosis in a poorly controlled diabetic patient, with involvement of the lungs, spleen, and genitourinary system. It underscores the importance of early microbiological diagnosis and prompt institution of targeted antimicrobial therapy to reduce morbidity and improve clinical outcomes. Modelled estimates of the global burden predict that melioidosis remains vastly under-reported and a call has been made for it to be recognized as a neglected tropical disease by the WHO.

Melioidosis should be suspected in the appropriate epidemiological setting with clinical presentation and necessary steps should be taken to confirm the diagnosis microbiologically. In the acute septicaemic and subacute types, early aggressive therapy is essential to save life and antibiotics must be commenced empirically until microbiological diagnosis is established.

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