

Melioidosis or Tuberculosis?: A Diagnostic Quandary

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ABSTRACT

Introduction: Melioidosis is a disease of humans and animals, caused by *Burkholderia pseudomallei*. It is seen mostly in endemic areas and in patients with risk factors like elderly, those with comorbidities and immunosuppression. The disease has a wide variety of presentations and mimics many other diseases, especially Tuberculosis (TB). Overlapping symptoms leads to delay in diagnosis and treatment of melioidosis which in turn can lead to increase in severity and mortality.

Case report: Here we report a case of an elderly male who presented with chronic history of cough, dyspnoea, loss of weight and appetite, with generalized weakness of more than two months with clinicoradiological findings mimicking TB but without microbiological evidence of TB. Patient subsequently developed a left knee joint effusion, the aspirate of which grew *Burkholderia pseudomallei*. Meanwhile patient went in sepsis and expired.

Conclusion: A thorough evaluation and detailed history and examination will help to diagnose the most common mimickers like disseminated TB and malignancy due to very similar presentations.

Keywords: Melioidosis, Septic Arthritis, *Burkholderia Pseudomallei*

INTRODUCTION

Melioidosis is a disease of humans and animals caused by gram negative bacilli called *Burkholderia pseudomallei*. This bacillus is found in muddy water and humid soil and cultivates even under harsh conditions and spreads via ingestion, inhalation and inoculation. Rare cases of acquiring the disease by nosocomial infections, sexual route, laboratory accidents and vertical transmission at childbirth are also known.¹ It is not contagious and human to human spread is not known. It is endemic in south east Asia and northern Australia, but lately found in many other tropical and subtropical locations.²

It occurs in patients aged 40-60 years. Risk factors include diabetes mellitus, occupational exposure^{3,4}, male sex (due to higher environmental exposure, and more inhalation of contaminated dust)⁴, exposure to soils and water, chronic lung, liver and kidney disease, alcohol consumption, prolonged immunosuppression due to long term steroids and other immunosuppressants and thalassemia (which causes neutrophil dysfunction due to higher iron load).⁴ Incubation period is one to twenty-one days, with fulminant sepsis syndrome being common in acute disease.⁵

CASE REPORT

A 71 year old elderly male presented to pulmonary medicine OPD, with dysuria, burning micturition and acute inability

to pass urine for two days. He was a native of Bihar staying in Goa for last 10 years. He was unwell for last two months with breathlessness, fever, cough, loss of appetite and weight, and generalized weakness. He was a farmer by occupation, who stopped working since last 10 years, and had no addictions, no history of diabetes or other obvious causes of immunosuppression. He was treated in Bihar for Tuberculosis 20 years ago. He had a history of travel to his native place three months ago for a few days.

On examination patient was mildly dehydrated. He was febrile with pulse rate of 104 /minute, respiratory rate of 20/minute and blood pressure of 110/70 mm of Hg in right upper arm. Systemic examination was grossly normal. Hemogram showed normal counts, and liver and renal functions were also normal. Chest X-Ray at admission showed areas of fibrosis with volume loss on left hemithorax suggestive of sequelae to old Tuberculosis (Figure 1). A provisional diagnosis of Pulmonary TB was kept in mind. Urine examination showed 25-30 pus cells with albuminuria. Blood, urine and sputum cultures were sterile. Sputum for AFB smear and CBNAAT were negative. Abdominal and renal ultrasound were normal. Patient was initiated on Cefoperazone sulbactam in the dose of 1.5gms 12hrly without any response. Computed tomography of the thorax (figure 2) showed left upper lobe irregular lesion, 2.4x 1.4 cms in size with peripheral calcification and a necrotic intrapulmonary lymph node in post segment of left lower lobe with patchy nodular densities in left upper and lower lobes. Patient also had minimal left pleural effusion, with mild pericardial effusion. Abdominal CT scan was within normal limits, except for prostatomegaly.

Antibiotic was later changed to Piperacillin- tazobactam but patient continued to get high temperature spikes. On the 10th day of admission patient complained of pain and swelling in the anterior aspect of left knee. On examination, it measured 4.5x3.x2cms, which was fluctuant and tender on palpation. This swelling was aspirated. The aspirate was turbid with numerous pus cells and predominant polymorphs.

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Figure-1: Chest Radiograph depicting non homogenous opacity in the left upper zone



Figure-2c: Minimal Pleural Effusion on Left side

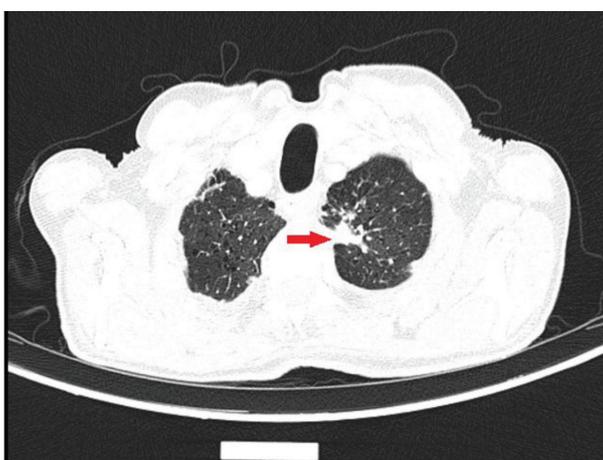


Figure-2a: IV Contrast CT study showing Irregular lesion 2.4*1.4cm with calcification in periphery in Left Lung Apex.

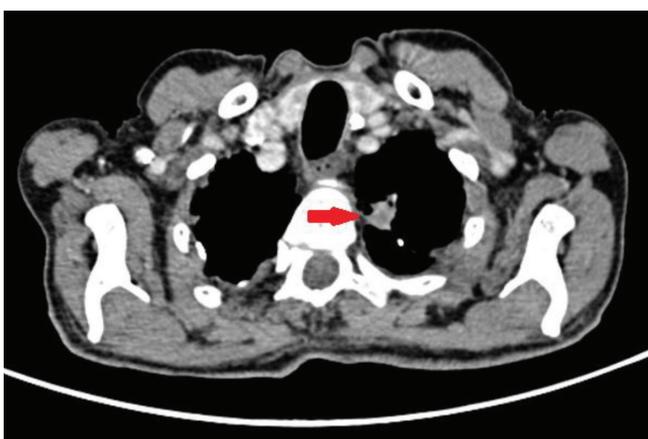


Figure-2b: Apical irregular lesion delineated on mediastinal window

CBNAAT and AFB smear of the aspirate was negative, but culture showed *Burkholderia pseudomallei*. Meanwhile he developed hypotension, and started deteriorating. Serum procalcitonin rose from 1.3 at admission to 57.2. Antibiotics were changed to injection meropenem and linezolid according to sensitivity report along with other supportive measures. However patient deteriorated and expired.

DISCUSSION

Melioidosis is a disease caused by a gram negative, oxidase positive, vacuolated, bipolar stained organism with rounded ends ("safety pin" appearance) *Burkholderia Pseudomallei* that assimilates arabinose & forms smooth colonies on incubation.¹ Diagnosis is established by culture of the clinical specimens with slow growth of the organism on standard blood and Mac Conkey Agar. Selective media like Ashdown's Agar, Matrix assisted laser desorption and ionization time-of-flight mass spectrometry also help in accurate diagnosis wherever available.⁵

The disease has a wide variety of manifestations ranging from localized cutaneous abscesses; visceral abscesses (in liver, adrenals, kidney, prostate, and spleen); intracerebral brain abscess and encephalomyelitis; osteomyelitis & septic arthritis; pulmonary involvement as infiltrates, cavitation, pleural effusion, empyema, nodules, and diffuse parenchymal disease; mediastinal masses, pericarditis, mycotic aneurysms and scrotal abscesses.^{2,6} The acute disease has symptom duration of less than two months, chronic of more than two months with symptoms mimicking pulmonary tuberculosis or non healing cutaneous lesion. Nearly 50% have respiratory involvement, ranging from consolidation to massive pleural effusion.⁷ Septic arthritis and osteomyelitis are very rare presentations with large joints especially knee joint most commonly involved, followed by ankle, foot, shoulder and hip.⁷ Usually septic arthritis and osteomyelitis are due to bacterial dissemination from infection elsewhere in the body, rarely can be a primary manifestation of melioidosis. Usually septic arthritis is due to hematogenous spread, but can spread via direct inoculation from contiguous sites. Lungs, soft tissues and osteoarticular tissues show longer persistence of the organisms and may act as a reservoir thus spreading the organisms to other organs.⁴ Treatment is divided into an initial Intensive/Induction phase for 10-14 days with carbapenem or ceftazidime or Trimethoprim-Sulphamethoxazole (TMP-SMX) followed by an Eradication phase for 3-6 months with TMP-SMX or doxycycline monotherapy depending on clinical response.⁸

Due to the past history of TB and the patient being elderly

and cachexic, with history of more than two months, with CT Thorax showing pleural and pericardial effusion and necrotic lymph nodes, our first differential diagnosis was disseminated tuberculosis. The pleural or pericardial effusion was too little to be aspirated and evaluated. Malignancy was also considered in view of CT thorax finding of an irregular nodular lesion with peripheral calcification (? scar carcinoma).

N Patil et al⁷ reported hip and knee joint septic arthritis with left pleural effusion in a patient without any risk factors. Our patient however was elderly, malnourished and an ex farmer with recent history of travel to Bihar, which is known to be endemic for melioidosis⁴ and these were the probable risk factors for melioidosis in our patient. But the possibility of nosocomial inoculation of the organism could not be completely ruled out as the boggy painful knee swelling developed in hospital stay. Usually just like many saprophytes, *B. pseudomallei* is intrinsically resistant to many antibiotics.¹ Our patient was diagnosed late and so went into sepsis very rapidly, deteriorated and could not be revived.

CONCLUSION

Melioidosis usually presents in patients with risk factors in endemic areas. The clinical features mimic many other diseases like sepsis/ septic shock, community acquired pneumonia and tuberculosis, therefore resulting in frequent misdiagnosis ("the great mimicker").⁵ A high degree of suspicion, along with appropriate investigations and facilities are needed to diagnose melioidosis. A thorough evaluation and detailed history and examination will help to diagnose the most common mimickers like disseminated TB and malignancy due to very similar presentations. If not diagnosed or treated on time, and if the disease is disseminated or with respiratory system involvement, the disease has a bad prognosis and high mortality.

REFERENCES

1. Redondo M, Gomez M, Landaeta M, Ríos H, Khalil R, Guevara R et al. Melioidosis presenting as sepsis syndrome: A case report. *International journal of infectious diseases*. IJID. 2011; 15: e217-18.
2. Cheng A., Currie B. Melioidosis: Epidemiology, Pathophysiology, and Management. *Clinical Microbiology Reviews*. 2005;18:383–416.
3. Khiangte HL, Vimala RL, Veerarahavan B, Yesudhasan BL, Karuppusami R. Can the imaging manifestations of melioidosis prognosticate the clinical outcome? A 6-year retrospective study. *Insights into Imaging*. 2019;10:17.
4. Zueter A.R, Yean C.Y, Abumarzouq M, Rahman Z.A, Zakuan Z., Harun A *et al*. The epidemiology and clinical spectrum of melioidosis in a teaching hospital in a North-Eastern state of Malaysia: a fifteen-year review. *BMC Infectious Diseases*. 2016; 16: 333.
5. Wiersinga W.J, Harjeet S.V, Alfredo G.T, Currie B.J, Shoron J. P, David A et al. *Nat Rev Dis Primers*. 2017; 4:17107.
6. Currie, B. J., D. A. Fisher, D. M. Howard, J. N. Burrow,

D. Lo, S. SelvaNayagam, N. M. Anstey et al. Endemic melioidosis in tropical northern Australia: a 10-year prospective study and review of the literature. *Clin. Infect. Dis*. 2000; 31:981–86.

7. Patil N, Balaji O, Rao K.N, Hande H.M, Ahmed T, Singhal S. A rare cause of septic arthritis with pleural effusion: *Burkholderia pseudomallei*. *Asian Journal of Pharmaceutical and Clinical Research*. 2017; 10: 8-9.
8. Chakravorty A, Christopher H. Melioidosis: An updated review. *Australian Journal of General Practice*. 2019; 48: 327-32.

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