



Case report

Cavitory lung lesions: Melioidosis and pulmonary embolism causing necrotizing pneumonia



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A B S T R A C T

Cavitory lung lesions of various etiologies may be encountered in patients with respiratory symptoms associated with fever. Non-malignant cavitory lesions may mimic malignant lung lesions on most of radiographic modalities including chest radiographs or thoracic computed tomography (CT). Primary lung malignancy can be detected in as high as one-fifths of CT thorax as cavitory lesions and the remaining aetiologies may be due to bacterial, parasitic, and invasive fungal infections, as well as Granulomatosis with polyangiitis (GPA), sarcoidosis, septic thrombo-embolism, and lung metastasis from extra-pulmonary primaries. We report an interesting case of melioidosis infection complicated with pulmonary embolism, both of which can lead to cavitory lung lesions and subsequently cause a clinical conundrum.

Introduction

Burkholderia pseudomallei is a Gram negative soil saprophyte that causes melioidosis which is an endemic disease in Southeast Asia and Northern Australia [1]. Sporadic case reports elsewhere in the world suggest that existing but yet unrecognised foci of infection. Melioidosis is associated with high case-fatality rates in human and animals despite the introduction of intravenous ceftazidime and carbapenem. The mortality is mainly attributable to severe sepsis and its complications. Diabetes has been found to be the single most common predisposing factor in a review [2]

Melioidosis can present with various clinical manifestations like pneumonia, septicemia, arthritis and abscesses; particularly hepatic, splenic and brain abscesses.

Virchows' triad suggest that the pathogenesis of venous thrombosis are damage to vessel wall, disorders of coagulation and stagnant flow. Damage to vessel wall can be due to infection, infiltration or trauma. However, more common problems are coagulation defects that cause hypercoagulable state. The patient presented here had thrombotic events and initially the differential diagnoses included lung malignancy and pulmonary tuberculosis. However, later melioidosis was confirmed by bacteriological evidence in the blood. Suchada Niyasom et al. previously described dural venous thrombosis in a patient with systemic melioidosis [3]. To the best of our knowledge, this is the first reported case of pulmonary embolism related to melioidosis without the presence of any other risk factors.

Case presentation

A diabetic 64-year-old man presented with one month history of productive cough and poor appetite. There was no history of fever, hemoptysis or tuberculosis contact prior to admission.

On examination he was lethargic, icteric, tachypneic and dehydrated. He was afebrile but his blood pressure was low (100/60 mm Hg), he was tachycardic (152/min), and tachypneic with respiratory rate of 30/min. Bronchial breath sounds and coarse crepitation on left lower zone were heard. Spleen was slightly enlarged without any free fluid in the abdomen. No cardiovascular or neurological abnormality was noted. Chest radiograph showed right upper lobe cavity and surrounding consolidation. The initial working diagnosis on admission was pulmonary tuberculosis (TB) as we are in the endemic, high TB prevalent area. We also considered pulmonary aspergillosis and squamous cell carcinoma as differential diagnosis because of cavitory lung lesions in a diabetic patient.

Blood tests on admission showed a raised white cell count (22,590/uL) with predominant neutrophils (89%). Otherwise the hemoglobin and platelet levels were normal. Full blood picture revealed toxic granulation and left shift which were suggestive of infection. C reactive protein was markedly elevated (3.0 mg/L). His baseline coagulation profile on admission was within normal limits. There were also impairment of renal function with raised urea (144 mg/dl) and creatinine (2.71 mg/dl) and liver functions (AST 690 U/L, ALT 234 U/L, albumin 21 g/L, total bilirubin 3.51 mg/dl (direct 2.63 mg/dl)). His diabetes was poorly controlled as reflected by HbA1c of 8.0%. The sputum cytology

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Fig. 1. Thinned wall cavity on right upper lobe with surrounding consolidation.



Fig. 2. Truncated and filling defect seen on bilateral descending pulmonary arteries suggestive of pulmonary embolism.

showed no malignant cells. Sputum AFB direct smear and TB PCR were both negative. Abdominal ultrasound revealed no evidence of intra-abdominal abscess.

He was started on high dose ceftazidime 1 g every 8 h in view of the underlying diabetes covering for pseudomonas infection in the lungs. On day 3 of admission, blood and sputum culture revealed *Burkholderia pseudomallei* which was sensitive to Ceftazidime. Hence, we increased the ceftazidime monotherapy dose to 2grams every 8 h for the treatment of melioidosis. He had persistent hypoxemia despite improving infective parameters hence CT pulmonary angiogram was carried out. CTPA showed right upper lobe cavitary lung lesion with necrotizing pneumonia and acute pulmonary embolism (PE) involving both descending pulmonary arteries (Figs. 1 and 2).

He was started on IV heparin infusion with 6-hourly APTT monitoring.

Discussion

B. pseudomallei is an environmental inhabitant and is widely disseminated in soil, water or paddy fields. It is geographically restricted to tropical and subtropical areas of Australia and Southeast Asian countries. In Malaysia quite a number of cases were reported albeit, many are still under-reported due to its enigmatic manifestations.

Pneumonia is the most common presentation and the clinical manifestations range from acute fulminant sepsis to chronic infection mimicking tuberculosis. Pneumonia may be the primary presenting feature, or it can develop secondary to initial disease at a distant focus. Like our patient, the cavitating pneumonia is perhaps due to manifestation of melioidosis as evidence by temporal relationship with his prolonged respiratory symptoms prior to admission. The other

possibility is that pulmonary embolism could be responsible for necrotizing pneumonia as reported previously by Y Bashir et al. [4]. Meumann et al. reported majority of patient out of 319 had acute/subacute presentations accounted for the majority of primary pneumonia cases (91%) whereas chronic disease was seen less commonly (9%) [5]. In other multiple case series, pneumonia is the most frequent presentation of melioidosis and is involved in approximately half of all cases [6].

Isolation of *B. pseudomallei* from patients' body fluids remains the gold standard in the diagnosis and requires the use of selective media for non-sterile specimens [7,8]. Cavitory lung lesion may represent subacute or chronic pneumonia in this patient and it was confirmed by the isolation of *Burkholderia pseudomallei* from the sputum culture. There was no reported cases so far of acute pulmonary embolism related to the melioidosis that cause the cavitating lung lesion.

The presented patient was diagnosed with melioidosis septicemia, with an atypical presentation with venous thrombo-embolism along with cavitory pneumonia initially mimicking pulmonary tuberculosis and squamous cell lung carcinoma. Our hypothesis of this condition of the presented patient was sepsis induced acquired hypercoagulable state. We could not find a previous report of venous thrombosis associated with melioidosis septicemia without other predisposing factor such as immobilization and underlying malignancy. Hence the presented patient might be the first case of venous thrombosis in melioidosis septicemia.

Although melioidosis is such a common infection in our country, yet it is still underdiagnosed due to wide array of manifestations and disease entity. The authors' hypothesis of pathogenesis of venous thrombosis is sepsis induced acquired hypercoagulable state. Prompt treatment with intravenous heparin and appropriate antibiotics on a timely manner is potentially curative. Our patient had presented with PE from unrecognised *Burkholderia pseudomallei* bacteremia. Diabetes is a single risk factor for melioidosis recognised in this patient.

Conclusion

Cavitary pneumonia may be caused by melioidosis however it may also complicate as secondary infection due to pulmonary embolism. Diabetes mellitus is a recognised risk factor for melioidosis, hence requires high index of suspicions especially in an endemic area. Melioidosis and venous thromboembolism is rarely reported but an important association. We think the PE was most likely due to hypercoagulable state in view of absence of other risk factors. A prospective population-based cohort study suggested that raised C-reactive protein (CRP) levels were associated with an increased risk of VTE [9]. Early recognition and treatment of this association should be embarked to avoid fatal complications.

Conflicts of interest

None.

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References

- [1] Cheng AC, Currie BJ. Melioidosis: epidemiology, pathophysiology, and management. *Clin Microbiol Rev* 2005;18(2):383–416. Apr 1.
- [2] Niyasom S, Sithinamsuwan P, Udommongkol C, Suwantamee J. Dural sinus thrombosis in melioidosis: the first case report. *J-Med Assoc Thailand* 2006;89(2):242. Feb 4.
- [3] 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(1):1. Oct 7.

- [4] Bashir Y, Benson MK. Necrotising pneumonia and empyema due to *Clostridium perfringens* complicating pulmonary embolus. *Thorax* 1990;45(January (1)):72.
- [5] Meumann EM, Cheng AC, Ward L, Currie BJ. Clinical features and epidemiology of melioidosis pneumonia: results from a 21-year study and review of the literature. *Clin Infect Dis* 2012;54(3):362–9. Feb 1.
- [6] Douglas MW, Lum G, Roy J, Fisher DA, Anstey NM, Currie BJ. Epidemiology of community-acquired and nosocomial bloodstream infections in tropical Australia: a 12-month prospective study. *Trop Med Int Health* 2004;9(7):795–804. Jul 1.
- [7] Dance DA. Melioidosis. *Curr Opin Infect Dis* 2002;15:127–32.
- [8] Limmathurotsakul D, Wongratanacheewin S, Teerawattanasook N, Wongsuvan G, Chaisuksant S, Chetchotisakd P, et al. Increasing incidence of human melioidosis in Northeast Thailand. *Am J Trop Med Hyg* 2010;82(6):1113–7. Jun 1.
- [9] Quist-Paulsen P, Næss IA, Cannegieter SC, Romundstad PR, Christiansen SC, Rosendaal FR, et al. Arterial cardiovascular risk factors and venous thrombosis: results from a population-based, prospective study (the HUNT 2). *Haematologica* 2010;95(1):119–25. Jan 1.