CASE REPORT

MELIOIDOSIS MIMICKING PULMONARY TUBERCULOSIS

S. H. Pathirathne[1], G. P. Athukorala[1], H. Hussain[1], P. L. B. Nishantha[1], H. M. P. K. Senevirathne[1], E. Corea[2], A. D. Siribaddana[1]

[1]*Respiratory Unit, Teaching Hospital, Kandy, Sri Lanka*
[2]*Department of Microbiology, Faculty of Medicine, University of Colombo, Sri Lanka*
Corresponding Author: Dr. A.D. Siribaddana, Email: chamath1122@gmail.com

Introduction

Melioidosis is caused by the soil bacterium *Burkholderia pseudomallei*. The bacterium is an oxidase-positive, motile gram-negative bacillus, showing bipolar staining. While most cases are considered to be from percutaneous inoculation, inhalation is also a well-recognized mode of infection[1].

Melioidosis is endemic in Southeast Asia, Northern Australia and the Indian subcontinent[2]. Sri Lanka is situated in the endemic region for melioidosis and the incidence of the disease is increasing[3]. Melioidosis can present with a variety of clinical manifestations. Clinical course may be acute, subacute or chronic. In subacute and chronic forms involving the respiratory system, the presenting features may resemble other chronic pulmonary infections including tuberculosis. Melioidosis tends to cause suppurative visceral lesions which may accompany the pulmonary manifestations[4]. It has been referred to as the 'great mimicker' by various authors[5]. Presentations mimicking tuberculosis are important clinical considerations as a significant number of patients are diagnosed clinically as tuberculosis where the results of their bacteriological tests are negative. We report a case of melioidosis with a clinical presentation similar to tuberculosis.

Case Presentation

A 29 year old army soldier serving in Nuwara Eliya for the past 5 years, presented to our unit with intermittent fever, loss of appetite and loss of weight of 3 months duration. He also complained of a mild intermittent cough of 2 months duration. Prior to his appointment to Nuwara Eliya he was engaged in paddy farming at Mahiyangana. He had a history of IgA nephropathy, detected 6 months previously, and was on daily low dose corticosteroids.

Examination revealed a febrile patient with a temperature of 39 ºC. His pulse rate was 112/minute while the blood pressure measured was 140/90 mmHg. Upon admission the patient developed right sided flank pain and renal angle tenderness was elicited on the same side. The rest of the system examination was unremarkable.

The hemoglobin concentration was 10.5 g/dl and the white blood cell count was $32.5\times10^9/mm^3$ comprising 74% neutrophils, 22% lymphocyte and 3% eosinophils. The platelet count was $464,000/mm^3$. Urine microscopy showed pyuria (field full pus cells/HPF), however the urine culture was sterile. The renal function tests were impaired revealing a blood urea level of 16.7 mg/dL and a creatinine value of 2.13 mg/dL. The serum electrolyte levels were normal. The liver function tests were within normal limits.

HIV antibody test was negative. The ESR was 120 mm in the first hour. The CRP titre was 40 IU/L. Ultrasonography of the abdomen revealed a right side renal fullness compatible with pyelonephritis.

The initial chest radiograph (**Figure 1**) revealed a consolidation with cavity formation in the left upper lobe, for which the most common differential diagnosis is pulmonary tuberculosis.

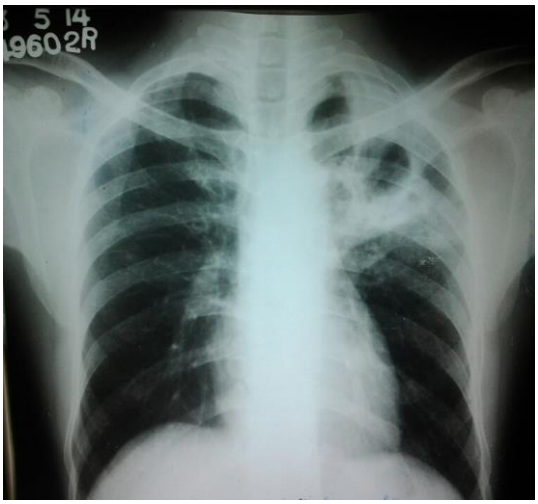

Figure 1. Chest radiograph showing a consolidation with cavity formation in the left upper lobe

Sputum for acid fast bacilli (AFB) was negative and sputum AFB culture was also negative. Tuberculin test carried with 5TU of PPD showed an induration of 13 mm diameter. Melioidosis antibody titre was highly positive with a titre of 1/5120 and blood culture yielded *Burkholderia* species, probably *B. pseudomallei*. The culture was sensitive to Meropenem and Ciprofloxacin.

The patient was commenced on initial intensive phase of the management with intravenous Meropenem 1 g every 8 hours with close monitoring of the renal functions. His fever subsided within 72 hours and there was a dramatic improvement in his renal functions, white cell count, and CRP in the subsequent two weeks following treatment. Repeat chest radiography showed significant resolution of the pulmonary lesions (**Figure 2**) while the repeat ultrasound scan of the abdomen was normal and showed resolution of pyelonephritis.

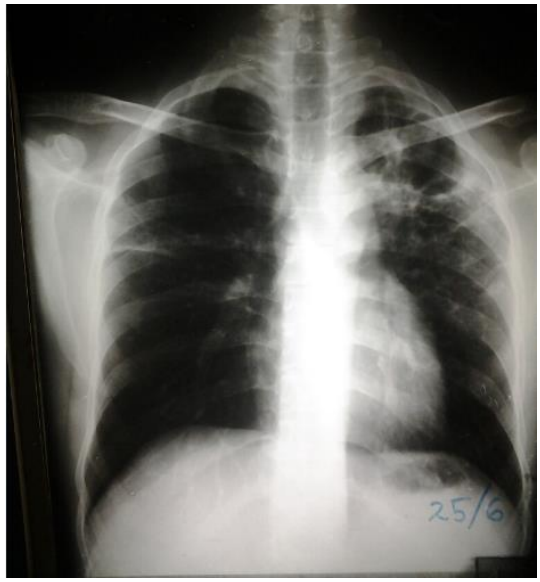

Figure 2. Chest radiography showing significant resolution of the pulmonary lesions

The intensive phase was continued over 28 days and the patient was discharged on Cotrimoxazole and Ciprofloxacin. The maintenance dose of Prednisolone was continued as the therapy for IgA nephropathy.

Following discharge the patient was monitored biweekly with regard to his clinical status. At the end of 3 months of therapy the white cell count was $10\times10^9/mm^3$ while the ESR was 42. Chest radiography at that point showed a significant improvement (**Figure 3**). The therapy was continued for 6 months. The patient remains well up to now after completion of treatment which was 9

months ago. There is no evidence of relapse of the disease.

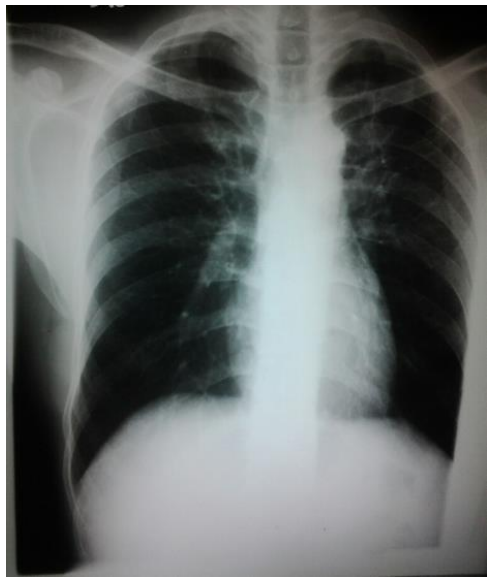

Figure 3. Chest radiography at the end of 3 months of therapy

Discussion

This patient's clinical presentation and chest radiography closely mimicked tuberculosis. Repeatedly negative bacteriological tests for tuberculosis should alert clinicians to look for alternative diagnoses. The concomitant suppuration, which was pyelonephritis in our patient, along with the pulmonary lesions should raise the possibility of melioidosis. Since the detection of melioidosis in Sri Lanka is increasing, it is an important differential diagnosis to be considered in patients suspected of tuberculosis without microbiological confirmation. Early initiation of treatment is important as rapid deterioration with fatal outcome has been reported[6].

The infection is known to have a prolonged latent period with possible reactivation into acute and fulminating infection[2]. The reactivation of the latent disease is often associated with concurrent diseases such as diabetes mellitus, chronic lung disease and chronic renal failure, which are considered as risk factors for developing Melioidosis. Use of steroids is also associated with an increased risk of Melioidosis[2]. The steroid therapy for IgA nephropathy in our patient, probably would have resulted in immune suppression leading to activation of latent *B. pseudomallei* infection, acquired previously. This case report highlights the importance of considering other differential diagnoses in patients suspected of Tuberculosis as management of them differs and delayed treatment can significantly increase morbidity and mortality.

References

1. Inglis A, Timothy JJ, Mee C, Brian J, Chang E, Barbara J. The environmental microbiology of melioidosis. *Reviews in Medical Microbiology* 2001; **12(1)**: 13-20

2. Cheng AC, Currie BJ. Melioidosis: epidemiology, pathophysiology, and management. *Clinical Microbiology Reviews* 2005; **18(2)**:383-416

3. Corea E, Thevanesam V, Perera S, I Jayasinghe, Ekanayake A, Masakorala J, Inglis TJJ. Melioidosis in Sri Lanka: an emerging infection. *Sri Lanka Journal of Infectious Diseases* 2012; **1(2)**: 2-8

4. Currie BJ, Fisher DA, Howard DM, et al. Endemic melioidosis in tropical northern Australia: a 10-year prospective study and review of the literature. *Clinical Infectious Diseases* 2000; **31**:981-6

5. Loveleena, Chaudhry R, Dhawan B. Melioidosis; the remarkable imitator: recent perspectives. *The Journal of the Association of Physicians of India*. 2004; **52**:417-20

6. Frickmann H, Neubauer H, et al. Fatal urosepsis due to delayed diagnosis of genitourinary melioidosis: *Laboratoriums Medizin* 2013; **37(4):** 4