International Journal of Research in Medical Sciences
*Manimaran R et al. Int J Res Med Sci. 2018 Aug;6(8):2872-2874*
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: http://dx.doi.org/10.18203/2320-6012.ijrms20183287

**Case Report**

# Melioidosis: a multiple disease imposter

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**Received:** 31 May 2018
**Accepted:** 28 June 2018

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## ABSTRACT

Melioidosis, an endemic disease in South East Asia and Australia is emerging a potential infectious threat off late in India. Though not highly infectious it can be extremely fatal. It has various manifestations which can mimic a wide array of disease including tuberculosis, malignancy, etc., Though not an untreatable disease it can certainly worsen with treatment. An interesting fact is that the fast deteriorating and fatality has made Burkholderia pseudomaleii, the causative organism qualified for biological warfare.

**Keywords:** Burkholderia, B. psedomallei, Fatal infection, Melioidosis, Septicaemia

## INTRODUCTION

Burkholderia pseudomalei which was a potential threat in South East Asia and Australian countries has started to spread in India. It can be treated and cured if diagnosed correctly but most of the times it is mistaken for its mimicking symptoms as various diseases including tuberculosis for its cavitating lesion and malignancy for its rapid spread and easy multisystem involvement.[1] Our patient had many of the predisposing factors for Burkholderia pseudomallei infection including diabetes, male gender, soil exposure being a farmer, COPD and renal disease. But he had even more manifestations to mimic various other common prevailing disease which lead other small hospitals he went to start on ATT.

According to Royal Darwin hospital study the various clinical manifestation including pneumonia 58%, 19% genito-urinary infection, 4% neurological or brain stem involvement, 17% skin and soft tissue involvement, 4% Splenic abscess, 2% liver infection, 18% prostatic, 3% other abdominal infection, 20% septic shock, 46% Bacteremia.[2] In this patient he had pneumonia, neurological involvement, involvement of the vocal cord for which it could be a secondary infection, weakness of left arm probably due to Multi focal vertebral lesions involving cervico-dorso-lumbo sacral vertebrae, osteolytic enhancing lesion involving C4, C5and C6 vertebrae with left paravertebral soft tissue encasing the adjacent left VA and involving the left C5 and C6 exiting nerve roots and proximal brachial plexus, infective spondylitis with left paravertebral granulation tissue with abscess which authors doubted to be malignancy, a cavitating lesion in the left lung probably we mistook for Koch. His loss of weight, loss of appetite, and a mal nourished sick look made us think in terms of malignancy which is common in this age and tuberculosis which is endemic in the country and he is an old case of treated tuberculosis.

## CASE REPORT

This case is a 64 years old gentleman who is a known case of Hypertension, Old TB, COPD, recently diagnosed as Type II diabetes mellitus and CKD on treatment. Admitted with the complaints of increased breathlessness for 7 days, cough with yellow coloured sputum present, and decreased appetite. History of mild blood streaks in the sputum +. History of? Reactivation of TB and started on ATT on 01/02/2014 outside. Took ATT for 7 days and

then stopped. History of recent hospitalization for COPD exacerbation from 26/01/2014 to 01/02/2014.

Evaluated the patient in detail by Critical Care Physician, Pulmonologist and Neurologist. On examination patient conscious, oriented, audible wheeze +, RR - 26/mt, BP - 130/80mmHg, $SPO_2$ - 95%, RS - Bilateral rhonchi and crepts +, CVS - S1S2 +, Left upper limb paresis +, weakness of left deltoid, biceps, rhomboids, brachioradialis present. C5 –C6 innervation, DTR's decreased left side.

Initial X-ray showing cavitating like lesion mimicking pulmonary tuberculosis.

Investigations showed total count - 36740cell/cu mm, ESR - 90, Hb - 11gm/dl, Urea - 112mg/dl, Creatinine - 2.2mg/dl, Sodium - 127, Potassium - 5, $HCO_3$ - 24. Chest x-ray showed bilateral infiltrates. C-reactive protein is raised.[3] HRCT chest showed thick walled cavitatory lesion in left lower lobe, Multifocal nodular lesions in bilateral lungs with adjacent ground glass attenuation, bilateral Bronchiectasis, multifocal centrilobular nodules in bilateral lungs, findings suggestive of pulmonary infection with trans bronchial spread. Echo heart showed Normal sized cardiac chambers, no RWMA, Normal LV systolic function with EF - 64%, Grade I LV diastolic dysfunction, Trivial MR, Calcified AOV, Mild to moderate AS / Trivial AR, Trivial TR, No PAH. USG abdomen showed bilateral chronic medical renal disease.

Started on IV antibiotics (Inj. Cefaperazone / Sulbactum and Inj. Levoflox), bronchodilators, steroids, Nasal $O_2$ and other supportive measures.[4]

Serial chest x-ray showed persistent patchy infiltrates and raised total count to 34150. Sputum grew gram negative bacilli. No AFB seen. Antibiotics were stepped up to Carbepenem along with Inj. Levoflox.[5] Peripheral blood smear showed neutrophilic Leukocytosis. Shift to left, toxic granules was suggestive of infection. For worsening paresis and left upper limb weakness, MRI cervical spine with screening of left brachial plexuses and screening brain done which showed Multi focal vertebral lesions involving cervico-dorso-lumbo sacral vertebrae, osteolytic enhancing lesion involving C4, C5 and C6 vertebrae with left paravertebral soft tissue encasing the adjacent left VA and involving the left C5 and C6 exiting nerve roots and proximal brachial plexuses, infective spondylitis with left paravertebral granulation tissue with abscess,? metastasis.

Sputum culture grew Burkloderia pseudomallei, antibiotics were changed to Inj. Ceftazidime + Doxy.[6] In spite of all these patients had persistent high total count (Total count - 33600 - 38390 - 51650) and chest x-ray showed bilateral patchy infiltrates.

CT abdomen showed Chronic medical renal disease, bilateral renal corticosteroids cyst, well defined hypodense lesion in uncinate process and body of pancreas. Small peripheral enhancing lesion in segment VIII of liver. In view of change of voice and muffled speech, videolaryngoscopy was done by the ENT surgeon and diagnosed to have left vocal cord paralysis.

Gradually his breathlessness decreased and was maintained with Nasal $O_2$.

On 10/03/2014 morning patient had worsening of breathlessness with hemoptysis. Chest x-ray showed worsening opacities, developed desaturation on NRBM O2, hence intubated and given mechanical ventilatory support. Had sudden cardiac arrest and revived with short CPR. Repeat chest x-ray showed acute left lung collapse.

Fibro optic bronchoscopy was done showed thick blood clot and active bleeding seen in entire left bronchial tree blood clot was removed, followed by which O2 saturation was improved. Also required inotropes for hypotension.

The patient and the attenders' were not affordable for the tertiary care anymore and had to take the patient back on against medical advice discharge.

The second case was a 74 year old male patient from Gudiyatham, Tamilnadu came with C/O Bronchorrhea (>50ml/day), Breathlessnes and cough for the past 3 weeks. The patient vitals were BP - 130/90mm of Hg, pulse rate - 76/min, $SPO_2$ - 98%, RR - 28/min. The patient was admitted in Respiratory medicine ward and the following incestigations were taken. Hb - 11.2g, TC - 18,000cells/cu.mm., DC - N - 61%, L-20%, E-1%, B-0%, M - 3%, ESR 1/2hr - 20mm 1hr - 40mm, Sputum AFB - Negative, Mantoux - Positive. Spirometry Volumes Were recorded. FRC <1.5 L, TLC <4.0L. Chest X- Ray revealed Patchy Consolidation in all parts of Lung predominantly over the Right Lower lobe. Sputum sample was sent for culture sensitivity. Patient was started on with Broad Spectrum Antibiotics and Proton pump inhibitors to prevent micro aspirations. HRCT was ordered By Pulmonologist. CT report showed Lung parenchyma shows diffuse Bilateral cavitary Lesions With areas of Honeycombing in both the lung. Lymphadenopathy around the HILUS and para tracheal region. After 3 days sputum culture report showed Burkholderia pseudomallei. Then the patient was given Intra venous ceftazidime and oral Doxycycline following which patient symptoms resolved over 14 days.[7]

Our third case was 32yrs old chronic smoker (10 yrs.) from Thiruchirapalli was diagnosed to be Tuberculosis and was started with Anti-Tuberculosis drugs. After 2 months of Intensive phase, Patient symptoms didn't resolve, and Chest X-ray still showed pulmonary infiltrates. Patient was referred to a higher centre and sputum AFB taken again. Sputum AFB was negative. But the patient chest X-ray revealed pulmonary infiltrates with pleural effusion. Sputum sample was sent for culture

sensitivity and pleural tapping done. Pleural fluid was analysed and its shows rich in Lymphocytes and was found to be Exudative. LDH ratio is 0.7. Hb-10.9gm, TC-21,000cells/cu.mm, DC-N-61%, L-31%, E1%, B-0%, M-3.1%, ESR-1/2 hr-16mm and 1hr-34mm.Patient was started with broad spectrum antibiotics along with ATT (DOTS) CAT-II regimen was started. Patient symptoms worsened and was intubated and connected to the ventilator. Culture sensitivity report suggested Burkholderia pseudomallei sensitive to Ceftazidime. Patient was started on IV Ceftazidime and oral Doxycycline. Patient symptoms worsened day by day and tracheostomy was done. IV antibiotics Colistin and tigecycline was started. Serial Chest X-ray were taken which showed increasing number of cavitation. Tracheostomy stoma site and tracheostomy secretion were sent for culture sensitivity. Culture reports were positive again for Burfholderia pseudomallei and Klebsiella resistant to Colistin and many other antibiotics. Patient developed ventilator associated pneumonia. Despite all efforts, patient couldn't have reviewed.

## DISCUSSION

The melioidosis was a challenge to any primary care physician without appropriate diagnostic facility and for a country to have endemic tuberculosis, it is common for any doctor to think it's reactivated tuberculosis and start on an ATT.[8] It took some time for us to realize the ongoing phenomenon. Thus, we conclude that the earlier the patient comes to critical care set up and the more aggressive the critical care physician treat the melioidosis, authors can reduce the mortality with the new spreading Burkholderia pseudomalleii.

There are quite a few scoring systems for mortality of meliodosis patients but they are neither sensitive nor specific for Indian population. Using data, collected prospectively from the period October 1989 to June 2002, from patients with acute culture-confirmed melioidosis presenting at the Royal Darwin Hospital, Darwin, Australia, a number of variables were selected that were easily available at the time of admission and reflected organ dysfunction. Mortality was predicted in univariate logistic and multivariate models by the presence of pneumonia, age at diagnosis, serum urea, serum bilirubin, lymphocyte count, and serum bicarbonate.[9] A score was assigned from 0 to 2, based on the degree of abnormality. A melioidosis score was formed from the sum of these scores, with a maximum score of 11. A score of < or = 3 (n= 140) was associated with a mortality of 8.6%, whereas a score of >or = 4 (n = 112) was associated with a mortality of 44.6%. Although this scoring system requires external validation, it may help identify a suitable target group of patients for intensive intervention such as early admission to an intensive care unit, the early use of meropenem, and goal-directed resuscitation therapies.

## CONCLUSION

Thus, to conclude this interesting case of meliododsis mimicking multiple medical conditions and its course. So, it's essential for every critical care physician to have even the atypical infections and medical conditions in the back of the mind to not leave out the fatal infection exceed their safe time to react and start our management.

*Funding: No funding sources*
*Conflict of interest: None declared*
*Ethical approval: Not required*

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**Cite this article as:** Manimaran R, Anand S, Aravind K. Melioidosis: a multiple disease imposter. Int J Res Med Sci 2018;6:2872-4.