Case Report

An interesting case of back pain

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ABSTRACT

Meliodosis is a disease caused by gram negative bacterium Burkholderia pseudomallei. Among its various clinical presentations, involvement of spine is rare phenomenon. On presentation they mimic tuberculosis and malignancy. We present a case of an elderly male with known case of diabetes mellitus presented with fever of unknown origin associated with back pain. On evaluation, all tests for tuberculosis and malignancy remained negative. Multiple visceral abscesses with hyper intense foci in T4 vertebrae were seen. Pleural fluid culture grew Burkholderia pseudomallei. Patient was treated with parenteral meropenem for 2 weeks, oral doxycycline and trimethoprim/sulphamethazone for 12 weeks. Dramatic decrease in back pain with improvement of constitutional symptoms was seen within 2 days of initiation of appropriate antibiotics. Meliodosis should be considered in differential diagnosis of back pain with constitutional symptoms. Dedicated team of microbiologists and physicians is required to identify and treat the disease.

Keywords: Burkholderia pseudomallei, Meliodosis, Back pain, Visceral abscesses

INTRODUCTION

Meliodosis is a bacterial infection caused by the soil dwelling gram negative bacteria Burkholderia pseudomallei. It is acquired by inhalation, percutaneous inoculation or ingestion. It is predominantly seen in northern Australia, south Asia including India and China.1 Several cases have been reported from various parts of India.2-8 But it is under diagnosed and under reported in Indian subcontinent. It is often a cause of pyrexia of unknown origin posing diagnostic challenge to treating physician. Early diagnosis and timely management is necessary for better outcome. Its clinical manifestations range from asymptomatic infection to overwhelming sepsis, high index of suspicion along with isolation and identification of B. pseudomallei is required for diagnosis. If duly considered by microbiologists and clinicians, the diagnosis can be made easily even in non endemic areas.

This case highlights the importance of awareness of this infection and its different modes of presentation among clinicians unfamiliar with the condition.

CASE REPORT

An 62 year old male from Southern Kerala, Construction worker who is a known case of COPD since 20 years on irregular medication and diabetic since 20 years on oral hypoglycaemic drug, admitted with a history of low grade intermittent fever not associated with chills, cough with mucoid sputum since 1 month with no history of chest pain or breathlessness.

He had low back pain since 1 month associated with generalised weakness, decreased appetite and weight loss of about 6 kgs over a period of 1 month. He was a smoker since 40 years, 15 cigarettes/day stopped 1 month back and alcoholic, drinks 1½ litres toddy/day stopped 1 month back. There was no history of abdominal pain,
vomiting, high risk behaviour, bowel and bladder irregularity. There was no significant family history.

He was treated in the local hospital for same symptomatically with IV antibiotics for 1 week without success. He presented to us with worsening symptoms. Examination revealed an ill looking, poorly nourished febrile patient with stable vitals. No pallor, icterus, cyanosis, clubbing, lymphadenopathy. JVP was not raised. Respiratory system examination revealed decreased breath sounds bilaterally, other systems were normal. Blood reports showed elevated ESR with uncontrolled sugars (Table 1).

**Table 1: Laboratory investigations during first admission.**

| Investigations                  | Level                  |
|--------------------------------|------------------------|
| Hemoglobin                     | 13.5g/dl               |
| Total count                    | 9400cu.mm (neutrophils-75%, lymphocytes-25%) |
| Erythrocyte sedimentation rate | 120mm/hr               |
| Platelet count                 | 180000cu.mm            |
| RBC count                      | 437 million/cu.mm      |
| Peripheral smear               | Normocytic normochromic anemia |
| Urine routine                  | Leucocytes- numerous, Protein -1+ |
| Urine culture                  | No growth              |
| Liver function test            | Total protein-7.9, Albumin-3.2, Globulin-4.7, Albumin globulin ratio-0.7, Total bilirubin-1.2, Direct bilirubin-0.5, Indirect bilirubin-0.7, SGPT-42, SGOT-32, ALP-217 |
| Fasting blood glucose, Glyco Hb| 176mg/dl, 9%           |
| Urea, Creatinine, Uric acid   | 26mg/dl, 0.8mg/dl, 3.1mg/dl |
| Calcium, Phosphorous           | 9.1mmol/L, 2.8mmol/L   |
| Sodium, Potassium              | 133mmol/L, 3.9mmol/L   |
| HIV, HBsAg, HCV spot           | Negative               |

Chest x-ray showed hyper inflated lung fields. Sputum C/S and Sputum AFB-Negative. USG Abdomen- mild hepatosplenomegaly with GB calculus of 4.3cm. ECG, 2D Echo-Normal. Bence jones protein in urine-Negative. Serum electrolytes- Normal. He was treated with IV ceftriaxone for UTI. His sugars were managed with insulin. Orthopaedics reference was sought for low back pain; tenderness was present in L3, L4, L5 spine system. Lumbarcal and hip X-ray was unremarkable. In view of high ESR and low back pain, bone marrow aspiration was done. Patient was discharged and advised to review with bone marrow biopsy report.

Patient came after 2 weeks with persisting and worsening back pain, other symptoms had improved. He was treated symptomatically with IV tramadol and NSAIDs with no much relief. Bone marrow biopsy was suggestive of normoblastic marrow with plasmacytosis. Respiratory system revealed bilateral creps. Repeat blood reports showed persistent high ESR, leucocytosis with left sided pleural effusion (Table 2).

**Table 2: Laboratory investigations during second admission.**

| Investigations                  | Level                  |
|--------------------------------|------------------------|
| Hemoglobin                     | 10.4g/dl               |
| Total count                    | 11200cu.mm (neutrophils-78%, lymphocytes-22%) |
| Erythrocyte sedimentation rate | 110mm/hr               |
| Platelet count                 | 11500cu.mm             |
| Peripheral smear               | Normocytic normochromic anemia with neutrophil leucocytosis and mild thrombocytopenia |
| Urine routine                  | Leucocytes- 8-10/hpf   |
| Liver function test            | Total protein-6.5, Albumin-2.5, Globulin-4, Albumin globulin ratio-0.6, Total bilirubin-0.9, Direct bilirubin-0.3, Indirect bilirubin-0.6, SGPT-15, SGOT-25, ALP-182 |
| Fasting blood glucose, Glyco Hb| 184mg/dl, 7%           |
| Post prandial glucose          | 230mg/dl               |
| LDH                            | 254U/L                 |
| Urea, Creatinine, Uric acid   | 26mg/dl, 0.8mg/dl, 3.1mg/dl |
| Calcium, Phosphorous           | 8mmol/L, 2.4mmol/L     |
| Sodium, Potassium              | 114mmol/L, 3.8mmol/L   |
| Chest X-ray                    | Minimal left sided pleural effusion with bilateral infiltrates. |

USG abdomen-hepatosplenomegaly with focal lesion in caudate lobe of liver, enlarged heterogeneous lobulated prostate left sided mild pleural effusion, moderate splenomegaly, and few retroperitoneal lymph nodes.

Hence oncology reference was sought and was advised for CEA level, PSA and UGI scopy. PSA- 0.18 ng/ml, CEA-1ng/ml, UGI scopy-antral gastritis with oesophageal varices. Biopsy of antrum revealed no granulomas or malignancy. MRI spine showed generalised decreased marrow signal with small hyperintense foci in the T4 vertebral body and sternum possibility of lymphoma and metastasis to be considered (Figure 1).
CECT abdomen showed multiple splenic, liver and prostatic abscesses, right pyelonephritis, with infective pulmonary nodules (Figure 2). In the background of diabetes with presence of multiple visceral abscesses a clinical suspicion of Melioidosis was considered. However, we needed to rule out disseminated tuberculosis as well. Hence pleural aspiration was done. Thick brown fluid and hemorrhagic fluid was aspirated from 2 collections from the left pleural space. Blood c/s-Pseudomonas fluorescens, Pleural fluid: cell count-40cells/cumm (N-91%, L-9), no evidence of malignant cells, Pleural fluid: sugar- 182, protein-6.8, albumin-2.6, LDH-756, pleural fluid AFB was negative, pleural fluid c/s-Burkholderia pseudomallei (causative agent of Melioidosis).

Patient was treated with IV meropenem for 14 days and Tab doxycycline and Tab trimethoprim/sulphamethazone for 12 weeks. Dramatic decrease in back pain was seen with improvement in the constitutional symptoms was seen within 2 days of initiation of antibiotics. On followup after a month patient was symptomatically better with good appetite and weight gain of 4kgs.

**DISCUSSION**

Melioidosis also called as whitmores disease, is caused by gram negative bipolar, safety pin shaped bacillus- B. pseudomallei. From India, cases are reported frequently from all regions, suggesting that it is becoming endemic disease, but large number of cases is not diagnosed. Risk factors are diabetes mellitus, chronic renal failure, alcohol abuse, thalassaemia, chronic lung or liver disease, malignancy and immunosuppresion. Diabetes mellitus is found up to 60.9% of affected cases. Our patient had diabetes has a risk factor. Melioidosis can present as multiple visceral abscesses, skin ulcers. It can also present as septic arthritis, osteomyelitis, pericardial effusion, and encephalomyelitis. Sometimes only bacteraemia without any focus may be present. Diagnosis can be done by isolating organism from the blood, urine, sputum or skin lesions. Done by detecting and measuring antibodies to the bacteria in the blood. Gram stain of material from skin, sputum.

In all suspected cases, staining with wrights stain and methylene blue is done. Agglutination tests, ELISA based on monoclonal antitoxin also used for diagnosis. Treatment has 2 phases: intensive and eradication phase. Intravenous ceftazidime (50mg/kg, up to 2g, 6hourly) or meropenem (25mg/kg up to 1g, 8hourly) are agents of choice in intensive phase. Oral antimicrobial for 3-6months for maintenance phase consists of trimethoprim-sulfamethaxazole every 12 hrs (40/8mg/kg up to 1600/320mg) or doxycycline every 12 hrs. In local or mild disease, intensive phase is of 2-4weeks followed by 3 months of eradication phase. In severe infection including neurological disease, intensive phase is of 6-8 weeks and 6 months of eradication phase. Meropenem in double dose is preferred in neurological disease.
Meliodosis is associated with a range of mortality from 10% to 39%, with septic shock-86%. Mortality in meliodosis is <10% where resources for rapid diagnosis, early implementation of antibiotics and good intensive care facilities for managing severe sepsis are available.¹

CONCLUSION

This case is reported because of rarity of finding skeletal abscess in a case of meliodosis. There are 2 cases reported. One presenting as spondylodiscitis and other as psoas abscess. Case emphasizes the need to have a closer view even in symptoms such as back pain which is often ignored owing to back pain of old age. Meliodosis may present as back pain with constitutional symptoms. Recurrence of illness after treatment with routine antibiotics should raise the suspicion of meliodosis and must be investigated thoroughly.

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