Open Journal of
Clinical & Medical
Case Reports

Volume 2 (2016)
Issue 19

ISSN 2379-1039

Disseminated Melioidosis in a Patient Presenting with an Unusual Neurological Manifestation; Acute Areflexic Flaccid Paraparesis Mimicking Guillain-Barre Syndrome

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Abstract

Melioidosis is an emerging infection in Sri Lanka with a variety of clinical presentations, involving almost all the organ systems of the body. Neurological manifestations of Melioidosis include peripheral motor weakness, brainstem encephalitis, aseptic meningitis, and respiratory failure. We report a case of disseminated melioidosis in a 51 year old Sri Lankan lady who presented with an unusual acute lower limb weakness which clinically resembled the features of Guillain-Barre syndrome. She responded to the treatment with IV Meropenum followed by IV Ceftazidime during intensive phase and oral Doxycillin in eradication phase and made a good clinical improvement.

Keywords

melioidosis; *burkholderia pseudomallei;* areflexic flaccid paraparesis; Guillain-Barre syndrome

Introduction

Melioidosis is a soil associated bacterial infection caused by *Burkholderia pseudomallei,* a Gram-negative bacillus [1]. It is endemic in tropical and sub tropical zones of South East Asia and northern Australia [1, 2]. Although Sri Lanka is not considered as a country where melioidosis is endemic, an increasing number of cases have been reported recently. According to recent survey Sri Lanka is considered as a country where melioidosis is predicted to be endemic and located among the priority countries where microbiological diagnostic facilities and disease reporting systems for melioidosis should be strengthened [3].

It is commonly seen in people who have frequent contact with soil and water such as in farmers and soldiers. It spreads from percutaneous inoculation, inhalation or ingestion. Diabetes mellitus, chronic lung and renal disease and alcoholism are common predisposing factors for Meliodosis [4]. It has a wide spectrum of clinical manifestations ranging from asymptomatic disease, localized skin ulcers or abscesses to fulminate disease with disseminated infection [2]. It involves almost any organ of the body while lungs being the commonest. Skin, subcutaneous tissue involvement and infections in the urogenital tract, musculoskeletal system, liver and spleen have been reported [5]. The variety of clinical manifestations of infection makes melioidosis difficult to diagnose clinically [6]. Neurological manifestations of melioidosis are less common. This could be due to exotoxin-induced damage, rather than direct invasion of the Central nervous system with *B. Pseudomallei* [6]. We report a case of

disseminated melioidosis with an unusual neurological manifestation of areflexic flaccid paraplegia which mimicked Guillain-Barre syndrome. According to our knowledge there are no published data in literature of Sri Lankan patients with melioidosis presenting clinically as Guillain–Barre´ syndrome.

Case Presentation

A 51 year old lady who was engaged in paddy cultivation admitted with 3 months history of high grade intermittent fever with loss of appetite and loss of weight (about 5 kg during the course of illness). Since 3 weeks time she had productive cough with purulent sputum with Medical research council grade 2 dyspnoea to which she had taken inpatient treatment for 4 days at the local hospital. She was managed with intravenous antibiotics and discharged with oral antibiotics. While on treatment at home she had developed loose stools for one week with a frequency of 4-5 bowel movements per day with ongoing fever. For 3 days she had developed gradual onset bilateral lower limb weakness which caused inability to walk and also noticed to have intermittent confusion, which made her to admit to the tertiary care unit.

On examination she looked ill, febrile (101 ^{0}F) dehydrated and moderately pale with an oral thrush. Pulse rate was 120 beats/ min and blood pressure was 80/50 mm Hg. She was tachypnoic (respiratory rate 28/min) and oxygen saturation was 89% in room air, with diffuse coarse crepitations in bilateral lung fields, more in right upper zone. Neurological examination revealed flaccid paraparesis with MRC grade 3 power and absent reflexes, impaired proprioception with normal plantar response and intact other sensory modalities. Her upper limb and cranial nerve examinations were normal and there was intermittent confusional states (mini mental score 15/30). Other system examinations were unremarkable.

Laboratory studies showed the following values: haemoglobin 7.l g/dl with normal red cell indices, platelet 69x10^{3} / µl, white cell count 9x10^{3}/µl (mild neutrophil leukocytosis). Peripheral blood film showed normochromic normocytic anaemia with moderate rouloux formation, neutrophil vacuolations with thrombocytopaenia, compatible with severe bacterial infection with sepsis induced marrow suppression. Erythrocyte sedimentation rate was 108 mm in 1st hour. C reactive protein was 162mg/dl. Activated partial thromboplastin time was mildly elevated (52 seconds, normal range 26-36 seconds) and rest of the clotting profile was normal. Random blood sugar level on admission was 134mg/dl and arterial blood gas analysis showed mild arterial hypoxia of 68.9mm Hg and urine ketone bodies were negative. Chest radiograph showed bilateral inflammatory shadows with right upper zone opacification. Spinal x rays and Contrast enhanced computed tomography brain were normal and nerve conduction study showed severe sensory motor axonal polyneuropathy in both lower limbs. Alanin transaminase was 111U/L and Aspartate transaminase was 52U/L with serum albumin of 2.3g/dl and serum globulin 3.5g/dl with normal bilirubin levels. USS abdomen and 2D echocardiography were normal. Serum creatinine, blood urea, serum electrolytes were normal. Sputum Acid fast bacilli (3 samples), mantoux test and retroviral screening were negative. Two peripheral blood samples were positive for Pseudomonas spp. within 13- 14 hours of incubation and *Burkholderia pseudomallei* were isolated in further analysis. Meliodosis antibody was strongly positive with the titers of > 1/10240. She was treated during intensive phase with intravenous Meropenum 1g 8 hourly for 10 days and stepped down to intravenous Ceftazidime 2g 8 hourly for 11 days. Since the isolate was resistant to oral Cotrimaxazole, oral coamoxiclav and doxycycline combination were started during eradication phase.

But oral coamoxiclav was omitted in few days as patient developed diarrhoea. She was continued with oral Doxycycline 100 mg 12 hourly for 3 months. Meanwhile she was given supportive care with face mask oxygen, intravenous fluid, inotropic support, blood transfusion and limb and chest physiotherapy. Optimal nutritional support was given with high protein diet.

She improved dramatically during the 3 weeks course of intensive phase antibiotic treatment with resolution of fever, improved appetite and improved lower limb weakness achieving ability to walk on her own. Her haematological and biochemical parameters were normalized. Chest x ray got cleared and inflammatory markers dropped down. She was discharged with oral Doxycillin after 1 month of hospital stay. She was followed weekly in the medical clinic and no complications were encountered.

Discussion

Melioidosis is an emerging infection in Sri Lanka with an increasing number of cases reporting throughout the country. Since the clinical presentation of meliodosis is not definitive, high index of clinical suspicion is needed for the diagnosis [7]. Since our patient's presentation was acute onset areflexic flaccid paraparesis, clinically it mimicked Gullien barre syndrome. But nerve conduction studies revealed sensory motor axonal polyneuropathy. Even though there were few reported cases of melioidosis presenting as flaccid lower limb weakness, they were either due to demyelinating neuropathy or transverse myelitis with spinal shock but not due to axonal neuropathy.

However it has been suggested the possibility of toxin-mediated demyelination or nuclear damage for severe peripheral motor weakness [6, 8]. Bacterial spread within nervous tissue, including the possibility of bacterial travel along nerves is another suggestion [6].

In this patient lumbar puncture was not done initially due to low platelets, later due to lack of patient's consent which would have given more information on Central nervous system involvement.

High degree of clinical suspicion is needed to make a proper and a timely diagnosis to prevent adverse outcomes of the illness especially in a country like Sri Lanka, which is situated in the endemic belt of melioidosis. Timely antibiotic management with good supportive care help to save the patients.

Low endemicity of Melioidosis in Sri lanka could be due to under diagnosis. It may be due its unfamiliarity for the clinical and laboratory personals of the healthcare centres. Strategies to improve the awareness and diagnostic facilities need to be implemented in order to minimize the morbidity and mortality associated with the disease condition.

Conclusion

Meliodosis shows a variety of clinical presentations including neurological involvement. To our knowledge there are no published data in literature of melioidosis presenting clinically as Guillain–Barre´ syndrome in Sri Lankan patients. Pathogenesis of this type of presentation has to be studied further. High degree of clinical suspicion is needed to establish the diagnosis and prevention of fatal outcome of the disease.

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Manuscript Information: Received: June 23, 2016; Accepted: October 04, 2016; Published: October 07, 2016

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Citation: Dharmadasa DSP, Basnayake BMDB, Kannangara T, Kothalawala M, Hemachandra T. Disseminated melioidosis in a patient presenting with an unusual neurological manifestation; acute areflexic flaccid paraparesis mimicking Guillain-Barre Syndrome. Open J Clin Med Case Rep. 2016; 1170

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