Burkholderia pseudomallei: Rare pathogen causing urosepsis in a diabetic patient - A case report

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ABSTRACT

Burkholderia pseudomallei is soil-dwelling bacterium which causes melioidosis, a disease of tropical climates. Very few cases have been reported from the Indian subcontinent despite similarities in environmental conditions with Southeast Asian countries. Here, we report a case of acute bloodstream infection in a diabetic patient with bilateral renal calculi with bilateral hydroureteronephrosis and left gluteal cellulitis. Blood and urine samples were processed in the microbiology laboratory for culture and sensitivity. Gram stain showed short, straight Gram-negative bacilli with a "safety-pin" appearance which were motile. Blood agar yielded smooth, creamy white translucent colonies and MacConkey agar yielded wrinkled, pink-colored colonies with metallic sheen. Isolate was identified as *B. pseudomallei* based on biochemical reactions which were also confirmed by Vitek 2 compact. The conventional, automated blood culture and urine culture yielded the same organism which was susceptible to ceftazidime, cotrimoxazole, carbapenems, and minocycline. Diabetes is an important risk factor for the development of bacteremic melioidosis and delayed diagnosis may lead to fatal sepsis and eventually death. Our aim is to emphasize the importance of early diagnosis and treatment with appropriate antibiotics and thereby prevent mortality.

Key words: *Burkholderia, Septicemia, Urosepsis*

Burkholderia pseudomallei is soil-dwelling bacteria which causes melioidosis. It is predominantly a disease of tropical climates, especially in Southeast Asia and Northern Australia [1]. Very few cases have been reported so far from Indian subcontinent despite similarities in environmental conditions with Southeast Asian countries and are often underreported. Urinary tract infections (UTIs) are more common in diabetic patients, but *B. pseudomallei* is a rare pathogen causing UTI [2]. Increasing awareness about this pathogen will enable the diagnosis of more cases in India [3,4]. Here, we report a case of acute bloodstream infection in a diabetic patient with bilateral renal calculi with bilateral hydroureteronephrosis and left gluteal cellulitis.

CASE REPORT

A 60-year-old female, who was a known diabetic and hypertensive with coronary artery disease on medication for the past 8 years, was admitted in Intensive Care Unit with a history of breathlessness, abdominal discomfort, and oliguria of 1-day duration. She had one episode of hematuria and gave a history of fever, joint pains, and cough with expectoration for the past 20 days for which she had been treated symptomatically elsewhere. She also complained of the pain and swelling over the left gluteal region.

On examination, the patient was dyspneic, febrile, and pale with bilateral pitting pedal edema. Her blood pressure was 160/100 mmHg, pulse was 118/min, and SpO2 was 97% with 4 L of oxygen. Swelling, redness, and tenderness were present over the left gluteal region. Respiratory examination revealed bilateral wheeze and crepitations. Based on the patient's clinical symptoms and other vital parameters, the condition was provisionally diagnosed as acute pulmonary edema with the left gluteal cellulitis and acute renal failure. The patient was managed with third-generation cephalosporins, bronchodilators, diuretics, and 4 L/min of oxygen through oxygen mask.

Hematological and biochemical parameters were deranged with total WBC count - 27,000/mm^3, Hb - 10.9 g/dl, urea - 76 mg/dl, creatinine - 4.75 mg/dl, sodium - 125 mEq/L, and potassium - 6.2 mEq/L. Her glycemic status was not under control as evidenced by the capillary blood glucose of 182 mg/dl on admission. Echocardiogram showed a left ventricular ejection fraction of 45%. Ultrasonogram study of the abdomen revealed fatty liver, multiple hypoechoic foci with posterior shadow in renal pelvis, largest measuring 13 mm in the right kidney, and a distal ureteric calculus of 13 mm in the left ureter with bilateral hydroureteronephrosis. Computed tomography kidneys, ureters, and bladder (Fig. 1) revealed the same findings. Serum procalcitonin levels were assessed to confirm sepsis and the levels were elevated

at 18.04 ng/ml. The patient's blood and urine samples were sent to microbiology laboratory for culture and sensitivity.

Blood culture was performed by both conventional and automated (BACTEC) methods. Blood sample was inoculated into brain heart infusion (BHI) broth and Bactec aerobic blood culture bottles. Routine subcultures from the BHI broth were done on blood agar (BA), MacConkey agar, and chocolate agar, and urine sample was inoculated on cysteine lactose electrolyte deficient agar and incubated at 37°C for 24 h. Gram staining (Fig. 2) of colonies showed short, straight Gram-negative bacilli with a "safety-pin" appearance. The organism was motile by hanging drop method.

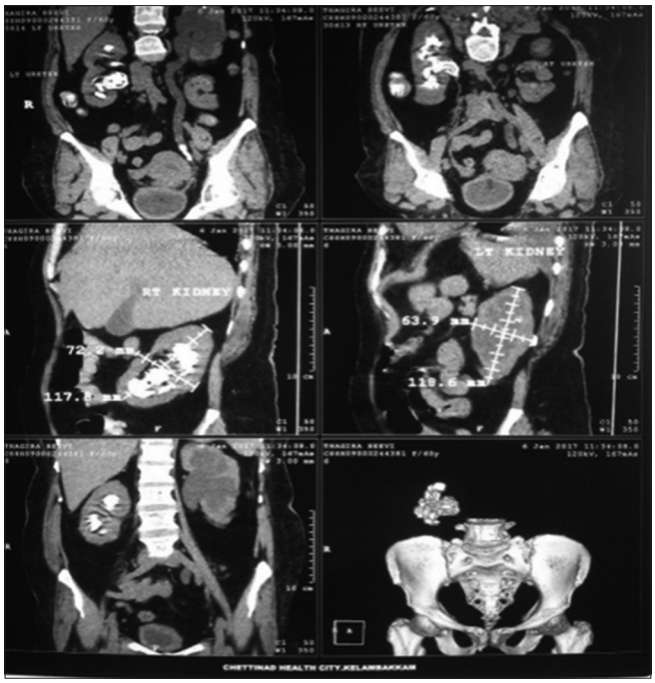

Figure 1: Computed tomography kidneys, ureters, and bladder showing the left distal ureteric calculi and bilateral hydroureteronephrosis

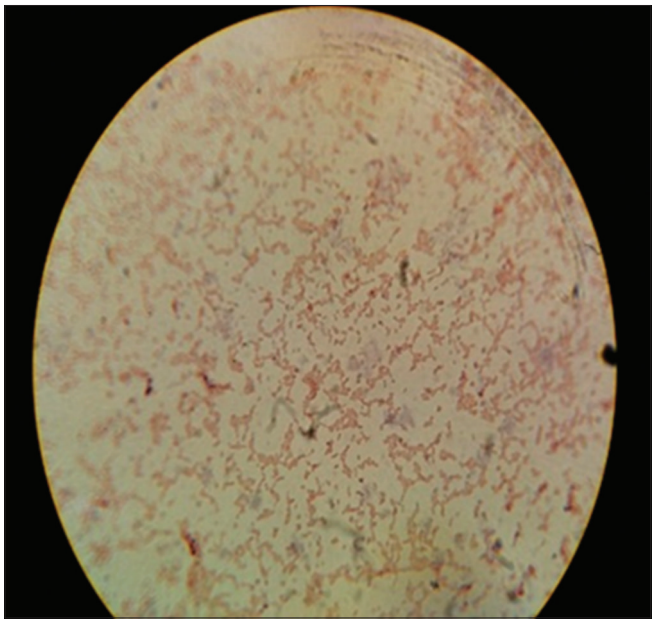

Figure 2: Gram stain showing Gram-negative bacilli with bipolar staining

Appropriate biochemical reactions were performed. BA yielded smooth, creamy white translucent, wrinkled, and umbonated colonies with no hemolysis (Fig. 3), and MacConkey agar (Fig. 4) yielded wrinkled, pink-colored colonies with metallic sheen. The organism was identified based on its biochemical reactions, which were that it was oxidase positive, oxidatively utilized glucose, did not ferment lactose, mannitol and maltose, lysine decarboxylase negative, arginine dihydrolase positive, and polymyxin B resistant. Selective Ashdown's media containing trypticase peptone, gentamicin, glycerol, crystal violet, and neutral red was inoculated as well and was incubated at 41°C which yielded wrinkled purple colored colonies (Fig. 5). The organism was identified as *B. pseudomallei* based on biochemical reactions, which was confirmed by the Vitek 2 compact automated organism identification system. Urine culture also yielded the same organism. Antibiotic sensitivity testing was performed by the Kirby–Bauer disc diffusion method as per the CLSI [4] guidelines 2017, on Muller–Hinton agar plate. Our isolate was resistant to aminoglycosides and colistin and susceptible to ceftazidime, cotrimoxazole, carbapenems, and minocycline (Fig. 6).

In view of elevated renal parameters, nephrologist opinion was sought and was advised emergency hemodialysis, fluid, and

Figure 3: Blood agar showing gray-white wrinkled colonies

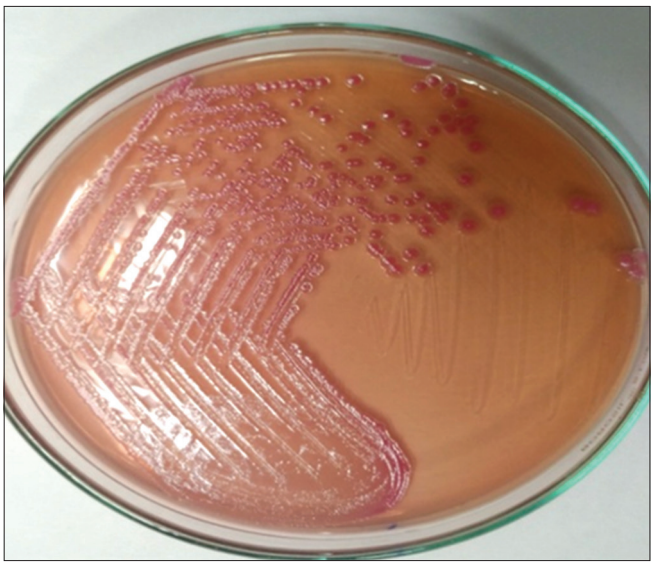

Figure 4: MacConkey agar showing pink colonies

Figure 5: Ashdown's media showing purple-pigmented colonies

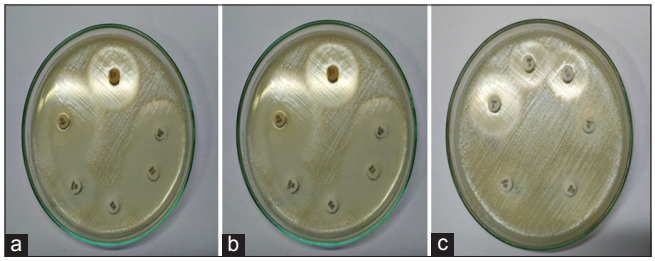

Figure 6: (a-c) Antibiotic susceptibility testing showing polymyxin and gentamicin resistance

salt-restricted diet along with potassium binders. Four units of fresh frozen plasma were transfused in view of elevated international normalized ratio. Clinical evaluation was suggestive of acute on chronic kidney disease caused by urosepsis. In view of severity of symptoms, the patient was taken up for ureteroscopy with DJ stenting procedure by the urologist. Here, the patient's vital signs were monitored regularly and she was started on parenteral Imipenem twice daily dose for 7 days, then switched over to oral cotrimoxazole, and asked to come for follow-up after 2 weeks. With the above management, the patient's condition was improved.

DISCUSSION

According to the Centers for Disease Control and Prevention, diagnosis of melioidosis is confirmed by isolating *B. pseudomallei* from blood, urine, sputum, skin lesions, or abscesses or by detecting an antibody response to bacteria [5]. It is also classified clinically based on the site of infection into acute or localized, acute pulmonary, acute bloodstream infection, and disseminated infection [5,6]. The major risk factors for developing melioidosis include diabetes, renal disease, liver disease, thalassemia, cancer, or another immune-suppressing condition not related to HIV. Patients with underlying risk factors such as diabetes [3,7] and renal insufficiency are more prone to develop this form of bloodstream infection, leading to sepsis [3]. The infection is acquired mainly through direct contact with contaminated soil and surface waters, ingestion of contaminated water or by inhalation of contaminated dust or aerosols. Contaminated soil and surface waters remain the main mode of transmission [8].

B. pseudomallei is intrinsically resistant to many antibiotics including penicillin, first- and second-generation cephalosporins, macrolides, rifamycin, colistin, and aminoglycosides and generally susceptible to chloramphenicol, tetracyclines, cotrimoxazole, ureidopenicillins, third-generation cephalosporins, carbapenems, and amoxicillin-clavulanate [9]. Our isolate was sensitive to ceftazidime, cotrimoxazole, carbapenems, and minocycline and resistant to gentamicin and colistin. This unusual antibiotic profile (i.e., gentamicin and colistin-resistant and amoxicillin-clavulanate susceptible) demonstrated by a Gram-negative oxidase positive bacillus can be used for confirming the identity of *B. pseudomallei* in the microbiology laboratory. This patient with underlying risk factor such as diabetes and acute on chronic kidney disease could have acquired infection from the environment through contaminated sources.

CONCLUSION

Diabetes is an important risk factor for the development of bacteremic melioidosis. Delayed diagnosis may lead to fatal sepsis and eventually death. It is important to treat suspected cases of melioidosis with appropriate antibiotics before any other interventions like draining lesions to avoid sepsis. Mortality rate is 95% in patients with acute disease who are not treated properly. Our aim is to emphasize the importance of early diagnosis and treatment with appropriate antibiotics and thereby prevent mortality.

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