

Septicemic melioidosis in a young adult with transfusion-dependent β -thalassemia major

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Abstract

Melioidosis, a neglected infection in Bangladesh, is caused by *Burkholderia pseudomallei* and carries high mortality, if not diagnosed or treated timely. Individuals with transfusion-dependent β -thalassemia major make a person especially vulnerable to *Burkholderia pseudomallei* infection owing to iron overload and immune dysfunction. Here, we report a fatal case of septicemic melioidosis in a 24-year-old man with Hb E β -thalassemia major who presented with fever, dyspnea, a cervical abscess, and septicemia. This case highlights the threat of melioidosis in thalassemia patients and emphasizes the importance of timely recognition and targeted therapy in endemic settings.

Introduction

Melioidosis, caused by *Burkholderia pseudomallei*, is a potentially life-threatening infectious disease that is endemic in South-East Asia and northern Australia and is increasingly recognized in South Asia, including Bangladesh [1]. The bacterium is widely present in soil and surface water, and human infection typically occurs through percutaneous inoculation, inhalation, or ingestion [2]. The disease manifests as pneumonia, septicemia, localized abscesses, or chronic disseminated infection, with mortality rates reported between 14–40% despite treatment [1,3]. In Bangladesh, the true incidence of melioidosis remains unclear due to underdiagnosis owing to limited laboratory capacity and lack of clinical awareness [1,4].

Underlying conditions that compromise immune function markedly increase susceptibility to *B. pseudomallei* infection. Transfusion-dependent β -thalassemia major represents one such high-risk state, with immune dysfunction attributed to impaired neutrophil activity, chronic iron overload from repeated transfusions, and hyposplenism [3,5]. These factors significantly reduce host defense against intracellular pathogens such as *B. pseudomallei*. Pediatric and young adult patients with β -thalassemia major may develop severe, atypical, or disseminated forms of melioidosis, due to delayed diagnosis and resulting in adverse outcomes [6,7]. Although sporadic reports from South and South-East Asia have described this comorbidity in melioidosis cases, data from Bangladesh are scarce despite the concurrent

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endemicity of both conditions [8]. In Bangladesh, an estimated 6–12% of the population, approximately 10 to 19 million people carry the β -thalassemia trait [9]. A 2005 study on school children from six districts of Bangladesh, using hemoglobin electrophoresis, reported an overall β -thalassemia carrier rate to be 4.1%, with the highest prevalence recorded in Barishal division at 8.1% [10]. On the contrary, till now around 150 cases of melioidosis have been reported from this country, and only a single case of septicemic melioidosis with β -thalassemia minor in a Bangladeshi farmer was reported till today [4,8]. Therefore, if a thalassemia major patient presents with refractory infection despite antibiotic treatment, melioidosis should be suspected.

We present here a rare case of septicemic melioidosis in a young adult with transfusion-dependent β -thalassemia major from coastal area of Bangladesh, emphasizing diagnostic delay, therapeutic challenges, and the need for heightened awareness among clinicians in endemic settings.

Case presentation

A 24-year-old male student hailing from Barguna, a coastal area of Barishal district of Bangladesh, with a known history of Hb-E β -thalassemia major was admitted to a tertiary care hospital in the capital Dhaka with the chief complaints of fever for 20 days (maximum temperature recorded 104°C), dyspnoea for 5 days along with right-sided neck swelling for 3 days. The patient was diagnosed as a case of Hb E β -thalassemia major at 3 years of age based on complete blood count, Hb-electrophoresis and iron profile. He had splenectomy in 2008, following which regular blood transfusions were required every 3-4 weeks. On general examination, temperature was 103.8°C, blood pressure was 90/60 mmHg, SpO₂ was found 92% (room air). A swelling was present on the right side of the neck along with pallor and hepatomegaly. Respiratory findings revealed bilateral crepitations. The patient's blood analysis demonstrated hemoglobin: 6.8 g/dL, total WBC: 23,170/ μ L (neutrophil count: 16,682/ μ L), platelets: 120,000/ μ L. Serum bilirubin was 4.6 mg/dL, while SGPT was found 130 U/L. Iron

profile revealed total iron: 25.84 μ g/dL, TIBC: 93 μ g/dL and serum ferritin: 12,440.45 ng/mL (normal: 20–300 ng/mL in adult males). Ultrasonography of whole abdomen showed multiple tiny cystic lesions in both lobes of the liver suggestive of micro-abscesses, along with bilateral pleural effusion. Chest X-ray was advised but could not be done because of deteriorated condition of the patient. Blood culture revealed growth of *B. pseudomallei*, sensitive to ceftazidime, meropenem, co-amoxiclav, co-trimoxazole, and doxycycline; resistant to aminoglycosides and colistin. Therefore, the case was finally diagnosed as septicemic melioidosis in a β -thalassemic major patient. The patient was started on intravenous meropenem 6 days following the onset of symptoms (2 g every 8 hours) along with supportive transfusions and oxygen supplementation. Despite initial stabilization, his fever persisted, and respiratory status worsened. After 11 days of hospitalization, and 7 days of antibiotic administration, he succumbed to septicemia and multi-organ failure.



Figure-1: Growth of *B. pseudomallei* in MacConkey agar media from blood sample.

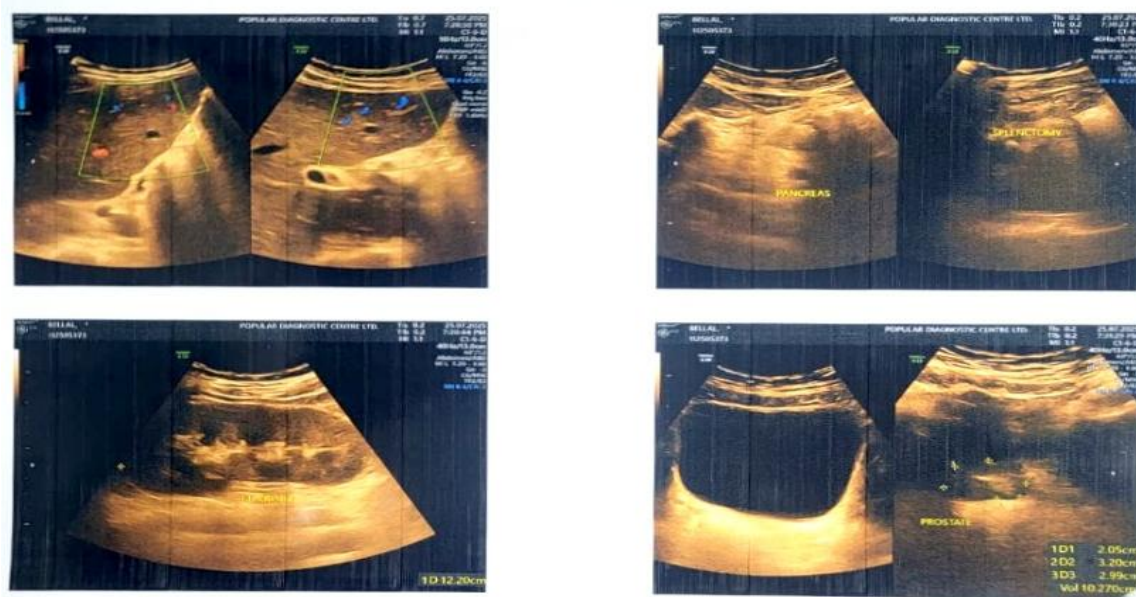


Figure-2: Ultrasonography of abdomen showing presence of multiple micro-abscesses in the liver

Discussion

Melioidosis is a fatal endemic infectious disease caused by the gram-negative bacteria *Burkholderia pseudomallei*. This case from Bangladesh highlights the impact of melioidosis in a high-risk host with transfusion dependent thalassemia major. Multiple risk factors such as iron overload, splenectomy and chronic transfusions with immune dysregulation predisposed the patient to *B. pseudomallei* infection. The patient's serum ferritin was more than 12,000 ng/mL, providing abundant free iron that facilitated the growth and virulence of *B. pseudomallei* [5]. Loss of splenic function markedly increases susceptibility to systemic bacterial infections, while regular transfusions further compromised host defenses [11].

Our patient presented with disseminated melioidosis, evident by the presence of hepatic micro-abscesses, neck abscess, pleural effusion, and sepsis. Although appropriate antibiotics were initiated within first day of diagnosis his severe iron overload and compromised immunity likely contributed to poor treatment outcome and death.

Melioidosis in individuals with thalassemia is uncommon but recently increasingly recognized. In

a retrospective study performed in Sabah, Malaysia it was found that, 41% of confirmed pediatric melioidosis patients had thalassemia major [5]. The same study reported that during the period of 2011-2012, when iron chelation therapy was initiated in thalassemic patients, out of 860 patients, none of them were infected with melioidosis [5]. In case of our patient, iron chelation therapy was earlier prescribed following blood transfusion due to iron overload. However, owing to poverty and illiteracy, the patient could not comply with the suggested management. A case control study from Thailand in adult patients showed that people with thalassemia were about four times more likely to get melioidosis than adults with diabetes [12]. This suggests that thalassemia makes people more vulnerable, beyond the usual risk factors.

Case reports specifically linking melioidosis with transfusion-dependent β -thalassemia have appeared from multiple endemic settings. A recent report from Thailand described disseminated disease in a patient with transfusion-dependent β -thalassemia major, highlighting severe multi-organ involvement and the need for early recognition and prolonged antibiotic therapy [6]. In Sri Lanka, a 7-

year-old boy with β -thalassemia major developed chronic melioidosis with multiple hepatic abscesses like our case, illustrating the organism's tropism for visceral abscesses in iron-overloaded hosts [7].

Immune dysregulation in β -thalassemia (including altered cytokine responses) and iron overload, common in transfusion-dependent and post-splenectomy patients facilitate *B. pseudomallei* growth and impaired its clearance, aligning with our patient's marked hyperferritinemia [3]. The exact role of iron causing susceptibility to *B. pseudomallei* is not fully understood. Iron is essential for bacterial growth and is normally stored in the body bound to proteins such as transferrin, lactoferrin, and ferritin [9]. Many bacteria release small molecules called siderophores to capture iron, and these have been linked to virulence in pathogens like *Vibrio parahaemolyticus* [13]. In *B. pseudomallei*, the siderophore "malleobactin" has been shown to promote bacterial growth, especially in the presence of higher iron levels. However, the role of iron in *B. pseudomallei* infection is more complex [14,15]. A *B. pseudomallei* strain that lacked malleobactin was found to be just as virulent as normal strains. Such mutant strain uses alternative pathways, including the production of proteases that release iron from ferritin, a protein stored in excess in conditions like thalassemia [16,17].

Conclusion

In Bangladesh, melioidosis remains under-recognized but is documented as endemic. National reviews summarize dozens of sporadic cases since 1960 [1,7]. There were no previously published Bangladeshi reports directly pairing thalassemia major with melioidosis. Global literature supports β -thalassemia, particularly transfusion-dependent disease with iron overload as a risk state for severe, disseminated melioidosis. Bangladesh is endemic for melioidosis; lack of local case documentation linking thalassemia major with *B. pseudomallei* infection likely reflects under-recognition rather than absence of association.

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and MSAJ were involved in diagnosis of the disease, analysis of the case and maintaining routine follow up; MSK collected and recorded patient's data, performed and supervised the laboratory investigations. MSAJ contributed to editing the manuscript and was involved in providing critical insights regarding the management of the patient.

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