

Case Report

Recognizing Septic Cutaneous Pustules as a Manifestation of Disseminated Melioidosis

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Abstract

Melioidosis is an infection caused by the gram-negative bacterium *Burkholderia pseudomallei*. This disease is endemic in Southeast Asia and North Australia with sporadic occurrence in temperate countries. Any organ can be involved in melioidosis it causes an acute inflammatory reaction with rapid development of small abscesses which tend to coalesce to form larger abscesses. Cutaneous manifestations vary greatly. We report a man with disseminated melioidosis who presented with cutaneous lesions.

INTRODUCTION

Melioidosis caused by *Burkholderia pseudomallei* is endemic in South East Asia and Northern Australia [1]. It can present in myriad of ways ranging from localised infection to overwhelming sepsis and death [2]. This case report serves to highlight the importance of recognizing multiple septic skin pustules as one of the presentations of disseminated melioidosis which carries a high mortality rate. Appreciating secondary skin melioidosis in the form of septic pustules may allow early provision of effective antibiotics in bacteraemic patients and prompt the search for occult intra-abdominal abscesses that may require surgical drainage.

CASE PRESENTATION

A 47 year old farmer with underlying history of poorly controlled Type 2 diabetes presented with deterioration in general health for 2 weeks prior to admission. He complained of lethargy, poor oral intake, reduced effort tolerance along with fever, chills and rigors five days before seeking medical attention. Family members also reported that he was having altered sensorium characterized by incoherent speech and fluctuating level of consciousness.

Upon presentation, he was ill with a temperature of 40 degrees celcius. He was delirious with laboured breathing effort. Hypoxemia with PaO₂ of 79 mmHg was present while receiving high flow mask oxygen 15L/min. Physical examination revealed multiple pus filled blisters located over the face, neck, anterior chest and upper limbs. Lung auscultation revealed generalised crepitation with reduced air entry bilaterally. Abdomen was soft with no peritoneal irritation but liver was 2 finger breaths palpable below the right costal margin. Biochemical results revealed that he was in diabetic ketoacidosis with random blood sugar(RBS) of 55.3, pH of 7.13 bicarbonate level of 9.7 and urine

ketone 3+ complicated with acute kidney injury. Total white blood cell count was raised with thrombocytopenia. In view that melioidosis is endemic among farmers in the state of Kedah, intravenous Ceftazidime 2g 6 hourly was promptly commenced after cultures were taken and he was put on standard treatment for diabetic ketoacidosis with aggressive hydration and insulin infusion. In the ward, he developed septicaemia shock requiring intubation for airway protection and subsequently underwent dialysis for worsening renal function with severe metabolic acidosis.

On day 3 of admission, blood culture and pus aspirated from lesions over the chest wall grew *Burkholderia pseudomallei*. An urgent ultrasound of the abdomen also revealed a multiseptated hypochoic lesion located at segment IVb, IVa and V of the liver measuring 10.1cm X 5.6cm X 9.4cm in keeping with multiseptated liver abscess.

Unfortunately his condition deteriorated further with refractory shock unresponsive to fluid boluses and vasopressor support. He succumbed to the disease 4 days after admission despite appropriate antibiotics.

DISCUSSION

Melioidosis is an infection caused by the facultative intracellular gram negative bacterium, *Burkholderia pseudomallei*. This organism is widely distributed in the soil and fresh water in endemic regions like Northern Australia and South East Asia [3]. Melioidosis is a great mimic whereby bacteraemic spread of the organism can result in clinical manifestations involving virtually any sites. Pneumonia is the most common clinical presentation of melioidosis in all studies. In the Darwin Study (540 cases of cultured confirmed melioidosis prospectively followed over 20 years in Northern Australia), disseminated melioidosis and bacteremia carries a high mortality rate at 20%. Over half

of all patients are bacteraemic and up to a quarter can present with septic shock [4]. Cutaneous manifestation is however less common accounting for only 12% of the subjects in the Darwin Study [4]. Recognizing important cutaneous findings of disseminated melioidosis allows early administration of effective antibiotics and prompts the search for occult abscesses in the setting of high disease burden.

Skin and soft tissue infection comprise 12%-24% of clinical presentations with melioidosis in published case series [5]. In the Darwin study, primary skin melioidosis was considered on the basis of clinical presentation; patients whose major presenting symptoms was skin infection were considered to have primary skin melioidosis whereas those with clinical presentation suggestive of an infection at another site or evident systemic sepsis were categorized as having other melioidosis. Those with other melioidosis who had incidental skin manifestations in the form of multiple skin pustules were considered to have secondary skin melioidosis whereby if tested positive for *Burkholderia pseudomallei* were presumed to be due to hematogenous spread.

Primary skin melioidosis is often localised and less fatal than other forms of melioidosis. Majority of patients (93%) who presented with primary skin melioidosis had localised disease with only 2 percent of them documented to be bacteraemic. There were no episodes of septic shock or fatality reported among the 32 patients who had primary skin melioidosis over the 10 year period [4]. Secondary skin melioidosis in the form of septic pustules is rare as shown in the study occurring in only 2% (10 out of 428) of the study population. In contrary to primary skin melioidosis, secondary cutaneous manifestation often reflect hematogenous dissemination whereby, 9 out of 10(90%) with secondary skin manifestation in the form of multiple pustules had documented bacteremia [4].

In this case, the patient had disseminated melioidosis infection with lung, liver and cutaneous manifestations. He was hypoxic from the pulmonary infection evidenced on chest radiograph and there was also incidental skin manifestation in the form of multiple skin pustules. Blood and pus aspirated



Figure 1 Figure above showing septic pustules over the face, neck and arms. Pus aspirated from these lesions grew *Burkholderia pseudomallei*.



Figure 2 Chest radiograph taken during admission revealing pulmonary consolidation over the right upper and middle zone along with ill defined opacities with central lucency seen at left lower zone suspicious of early abscess formation.

from skin lesions were both cultured positive for *Burkholderia pseudomallei* with similar ETEST MIC further strengthening the association between septic skin pustules and bacteremia. There was also an incidental finding of a large liver abscess that require prompt surgical drainage. Diabetes mellitus and his occupation as a farmer probably predisposed this patient to acquire melioidosis.

Diagnosing disseminated melioidosis especially in endemic regions requires high index of suspicion. This case report illustrates how acute septicaemia pulmonary melioidosis may mimic other forms of pneumonia for example staphylococcal pneumonia and is associated with high mortality when there is a delay in administering appropriate antimicrobial therapy. Recognizing skin manifestation of disseminated melioidosis along with prompt gram staining of pus from skin pustules may lead to early diagnosis of disseminated melioidosis. Despite early provision of appropriate antimicrobial therapy, the patient unfortunately succumbed to overwhelming infection due to high disease burden and large liver abscess that was unable to be drained in time.

REFERENCES

1. Hassan MR, Pani SP, Peng NP, Voralu K, Vijayalakshmi N, Mehanderkar R, et al. Incidence, risk factors and clinical epidemiology of melioidosis: a complex socio-ecological emerging infectious disease in the Alor Setar region of Kedah, Malaysia. *BMC Infect Dis.* 2010; 10: 302.
2. White NJ. Melioidosis. *Lancet.* 2003; 361: 1715-1722.
3. Cheng AC, Currie BJ. Melioidosis: epidemiology, pathophysiology, and management. *Clin Microbiol Rev.* 2005; 18: 383-416..
4. Currie BJ, Ward L, Cheng AC. The epidemiology and clinical spectrum of melioidosis: 540 cases from the 20 year Darwin prospective study. *PLoS Negl Trop Dis.* 2010; 4: e900.
5. Gibney KB, Cheng AC, Currie BJ. Cutaneous melioidosis in the tropical top end of Australia: a prospective study and review of the literature. *Clin Infect Dis.* 2008; 47: 603-609.

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