Melioidosis with endocarditis and massive cerebral infarct

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ABSTRACT

Endocarditis due to melioidosis is rare. A 60-year-old male was admitted with upper abdominal pain and vomiting for one month. Contrast enhancing computed tomography (CT) of the abdomen showed multiple hepatic, splenic and pancreatic non-enhancing cystic lesions. Culture of CT guided aspirate from the liver lesion showed growth of *Burkholderia pseudomallei*. He was started on ceftazidime and cotrimoxazole. Four days after admission patient developed decreased speech and response. Magnetic resonance imaging (MRI) and MR angiogram brain showed massive infarct in the middle cerebral artery territory and occlusion of the middle cerebral artery. Echocardiogram showed vegetation in the aortic valve. He was diagnosed to have disseminated melioidosis with endocarditis resulting in massive infarct in the middle cerebral artery territory.

Introduction

Melioidosis, caused by gram-negative soil saprophyte *Burkholderia pseudomallei*, is endemic in Southeastern Asia and Northern Australia. Clinical manifestations can vary from acute disseminated infection to subacute or chronic. Despite adequate treatment, melioidosis still carries a significant mortality attributable to severe sepsis and its complications. Cardiac involvement in melioidosis is found in only 1% of patients. Endocarditis due to melioidosis resulting in massive infarct in the middle cerebral artery territory was not previously reported.

Case Report

A 60-year-old male was admitted with upper abdominal pain and vomiting for one month. He stopped smoking 8 years before and had an uneventful past. He had epigastric tenderness and rest of the systemic examination did not reveal any abnormalities. Hemoglobin was 13.8 g/dL, total white blood cell count 10,200/µL with predominant neutrophils, platelet count 1.8 × 10⁹/L, and erythrocyte sedimentation rate 18 mm in 1 h. Urinalysis was normal. Biochemical parameters revealed random blood sugar 151 mg%, urea 15 mg/dL, creatinine 1.1 mg/dL, sodium 148 mmol/L, potassium 4.1 mmol/L. Liver function test was normal. Chest X-ray and electrocardiogram were normal. Ultrasonography abdomen showed multiple hypoechoic lesions in both lobes of liver. Contrast enhancing computed tomography (CT) of the abdomen showed multiple hepatic, splenic and pancreatic non-enhancing cystic lesions (Figure 1). Culture of CT guided aspirate from the liver lesion showed growth of *B. pseudomallei*. Acid fast bacilli and fungal cultures of CT guided aspirate were negative. His blood cultures were sterile. He was started on ceftazidime and cotrimoxazole.

On the sixth day of admission patient had decreased speech and response. Magnetic resonance imaging (MRI) and MR angiogram brain showed massive infarct in the middle cerebral artery territory and occlusion of the middle cerebral artery (Figure 2). Echocardiogram showed vegetation in the aortic valve. He was diagnosed to have disseminated melioidosis with endocarditis resulting in massive infarct in the middle cerebral artery territory.

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sudden cardio respiratory arrest and despite all efforts he succumbed to his illness.

Discussion

Stanton and Fletcher in 1932 termed melioidosis from the Greek words *melis* (distemper of asses) and *eidos* (resemblance). *B. pseudomallei*, the causative pathogen is visualized as a gram-negative bacillus with bipolar staining and is often described as having a *safety pin* appearance. In the latter half of the 20th century, melioidosis emerged as an infectious disease of major public health importance in southeastern Asia and northern Australia. Acute form can mimic community acquired bacterial sepsis, pneumonia or abscess and in its chronic form, can mimic tuberculosis or malignancy. Melioidosis can present with subcutaneous abscesses and visceral abscesses in the liver, spleen, prostate, parotid, and lymph nodal mass. Risk factors for melioidosis include diabetes mellitus, pre-existing renal disease, thalassemia, and occupational

![Contrast enhancing computed tomography of the abdomen showed multiple hepatic, splenic and pancreatic non-enhancing cystic lesions.](image1)

![Magnetic resonance (MR) imaging and MR angiogram brain showed massive infarct in the middle cerebral artery territory and occlusion of the middle cerebral artery.](image2)
Our patient did not have any of the aforementioned risk factors. Cardiac involvement in melioidosis is rare. Pericarditis is probably the most common manifestation, but myocardial abscesses and endocarditis have been reported. Pericardial effusion occurs in 1-3% of patients with melioidosis, confined to endemic regions. In the Darwin prospective melioidosis study from Australia only four out of 540 documented cases had pericarditis. Most of the cases with melioidosis pericarditis have subacute to chronic manifestations with moderate to massive pericardial effusion, similar to tuberculosis. Mycotic aneurysm or pseudoaneurysm is another rarer vascular manifestation of B. pseudomallei.

Neurological melioidosis is unusual, but both macroscopic brain abscesses and encephalitis occur. The prospective melioidosis study at Royal Darwin Hospital has documented 12 cases of neurological melioidosis over 9 years out of a total of 232 cases of melioidosis manifesting as meningo-encephalitis, myelitis and cerebral abscesses. Peripheral motor weakness (mimicking Guillain-Barré syndrome), brain-stem encephalitis, aseptic meningitis were previously reported as a part of neurological melioidosis.

Conclusions

We describe a patient with endocarditis due to melioidosis resulting in massive infarct in the middle cerebral artery territory which to our knowledge is the first such report. We hope this case may help the readers to be aware of such unusual presentations of an emerging infection.

References