Meliodosis is an endemic communicable disease caused by *Burkholderia pseudomallei* and a prevalent amphixenosis in tropical and subtropical regions. The main routes of infection are thought to be through compromised surface tissues and through inhalation of contaminated soil and water. *B. pseudomallei* can affect all organs, but pulmonary infection is the most common symptom, which could easily lead to sepsis. Here, we reported what we believe is a rare case of acute pericarditis caused by *B. pseudomallei* infection.

A 15-year-old male resident within a rural area in Dongfang County of Hainan Province in China was admitted on April 30, 2014, complaining of chills, fever lasting for more than 10 days, and difficulty in breathing due to chest congestion. The patient reported walking several times through a pond created by a typhoon 1 week prior in his residential area. Dr. Yi-Jiang Huang, Department of Respiratory, Hainan General Hospital, Haikou, Hainan 570311, China

Key words: *Burkholderia pseudomallei*; Melioidosis; Pericarditis; Tuberculosis

The patient was febrile and still had a continuous outflow of slightly yellowish fluid from the pericardium. Reexamination of routine blood tests revealed that the patient’s WBC count had increased to 18.3 × 10^9/L with 83% neutrophils. Blood and pericardial drainage fluid cultures were carried out over several days until a growth of *B. pseudomallei* was observed in the pericardial drainage fluid on day 13 while blood cultures were negative. Antitubercular treatment was stopped and an anti-infective combination therapy of cefazidime and compound sulfamethoxazole was administered. The patient’s body temperature gradually normalized after 3 days, and routine blood tests conducted 1 week later showed a WBC count of 8.4 × 10^9/L with 78% neutrophils, a CRP level of 38 mg/L, and a PCT level of 0.22 ng/ml. Ultrasonography imaging 1 month later indicated an adhesion in the pericardium at the apex. The patient was discharged and oral administration of compound sulfamethoxazole was continued as maintenance treatment. Adhesion and thickening in the pericardium were found in 2 months after hospital discharge.

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discharge [Figure 1b], and the patient often complained about mild shortness of breath when activity.

The clinical manifestations associated with melioidosis are diverse and can present as acute, chronic, and latent disease. It is difficult to distinguish melioidosis from tuberculosis when considering the physical signs and symptoms, and chest radiological tests. Patients presenting with risk factors, such as diabetes mellitus, or who are taking steroid therapy, are predisposed to both bacterial diseases. Histological examination of tissue taken from patients with melioidosis might reveal granulomas with central necrosis, which is also found in cases of tuberculosis. *B. pseudomallei* and *Mycobacterium tuberculosis* are both intracellular pathogens. The main host response to intracellular pathogens is mediated by cellular immune responses through activated lymphocytes and interferon-mediated signaling pathways.[2]

In the present case, the continuous increase in ADA levels in the pericardial fluid resulted in an initial misdiagnosis. It has been reported that the ADA value is > 25 U/L in tuberculous exudates with 89.66% sensitivity and 91.01% specificity.[3] The measurement of ADA is an important auxiliary method that might serve as a crucial diagnostic test of tuberculous exudates in the absence of direct evidence of infection because ADA is an enzyme biomarker indicating the activation status of the cellular immune response.[4] There is a great deal of confusion in the diagnosis of melioidosis and tuberculosis due to the similar presentation of the infections.

The patient in this case report was a resident of a rural area and, therefore, was more likely to be exposed to contaminated soil and water. Moreover, the patient was infected during the summer typhoon season, when the soil that might breed bacteria was washed by rainwater, leading to contamination of water sources. Melioidosis probably resulted from infection through compromised surface tissues by *B. pseudomallei* or through the inhalation of aerosol into the lungs. Pathogen invasion of the lungs might induce pneumonia and pulmonary cavity, causing the rapid development of sepsis. Although *B. pseudomallei* infection can cause abscesses at unusual sites such as brain, liver, and spleen, infective pericarditis is extremely rare. The pathogen might spread directly from the lungs or from the bloodstream and infect the pericardium, the route of infection in this case is a possible, but not definite. The current report indicates that the possibility of *B. pseudomallei* infection should not be excluded when considering a diagnosis of tuberculosis in patients living in melioidosis endemic areas. A delay in treatment would probably affect prognosis as discussed in this case where adhesion and thickening in the pericardium were found.

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There are no conflicts of interest.

**REFERENCES**

1. Cheng AC, Currie BJ, Dance DA, Funnell SG, Linmathurotsakul D, Simpson AJ, et al. Clinical definitions of melioidosis. Am J Trop Med Hyg 2013;88:411-3. doi: 10.4269/ajtmh.12-0555.
2. Koh GC, Schreiber MF, Bautista R, Maude RR, Dunachie S, Linmathurotsakul D, et al. Host responses to melioidosis and tuberculosis are both dominated by interferon-mediated signaling. PLoS One 2013;8:e54961. doi: 10.1371/journal.pone.0054961.
3. Barrios Barreto D, Rodríguez EP, Gotera C, Meneses PL, Narvaez PA, Mirambeaux Villalona R, et al. Lymphocytic pleural effusion and change of the adenosine deaminase cut-off level in tuberculosis diagnosis. Chest 2014;145:274A. doi: 10.1378/chest.1821853.
4. Arroyo M, Soberman JE. Adenosine deaminase in the diagnosis of tuberculous pericardial effusion. Am J Med Sci 2008;335:227-9. doi: 10.1097/MAJ.0b013e3180cab71a.