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CASE REPORT

Disseminated tuberculosis presenting as tuberculous peritonitis and sepsis tuberculosa gravissima in a patient with cirrhosis of the liver: A diagnosis of challenge



Chun-Yuan Lee^a, Hung-Chin Tsai^{a,b}, Susan Shin-Jung Lee^{a,b},
ChengLen Sy^a, Yao-Shen Chen^{a,b,*}

^a Division of Infectious Diseases, Department of Medicine, Kaohsiung Veterans General Hospital, Kaohsiung, Taiwan

^b Faculty of Medicine, School of Medicine, National Yang-Ming University, Taipei, Taiwan

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We report the case of an 81-year-old man diagnosed with liver cirrhosis complicated by spontaneous bacterial peritonitis and septic shock. *Mycobacterium tuberculosis* complex was isolated from the ascites, sputum, and blood culture 1 month after the patient died. Clinicians should be aware of the unusual diagnosis of sepsis tuberculosa gravissima presenting with tuberculous peritonitis, which is easily misdiagnosed as spontaneous bacterial peritonitis and Gram-negative bacillus sepsis in patients with cirrhosis. Clinicians should cautiously evaluate the patient's sputum, gastric contents, urine, cerebrospinal fluid, and bone marrow for early diagnosis of disseminated tuberculosis in patients with a high degree of suspicion of this diagnosis.

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Introduction

Tuberculous peritonitis is a rare presentation of extrapulmonary tuberculosis (TB). Although some reports have discussed the epidemiology and clinical manifestations of tuberculous peritonitis in Taiwan, it continues to pose a significant challenge due to its protean manifestations and

* Corresponding author. Section of Infectious Diseases, Department of Medicine, Kaohsiung Veterans General Hospital, 386 Ta-Chung First Road, Kaohsiung 813, Taiwan.

E-mail address: hctsai1011@yahoo.com.tw (Y.-S. Chen).

overlapping presentations with other diseases.^{1–4} The mean duration from presentation to diagnosis is about 1 month and the mortality rate is 60% among patients for whom treatment is not started within 30 days of presentation.⁵ Infection with *Mycobacterium tuberculosis* rarely presents as septic shock with multiple organ failure, i.e., sepsis tuberculosa gravissima, the behavior of which is similar to that of Gram-negative bacillus (GNB) sepsis.^{6–10}

We report here a case of disseminated TB presenting with tuberculous peritonitis and sepsis tuberculosa gravissima in a patient with cirrhosis of the liver, which was initially misdiagnosed as spontaneous bacterial peritonitis (SBP) with GNB sepsis.

Case report

An 81-year-old man with diabetes mellitus, hypertension, and previous cerebrovascular accident was brought to our emergency department with stupor for 1 day. He reported progressive abdominal distension, anorexia, and limb edema for 2 weeks prior to presentation. There was neither fever nor cough with sputum. One day prior to admission, he developed stupor.

In the emergency department, his initial physical examination showed a blood pressure of 138/61 mmHg, body temperature 37.3°C, heart rate 123 beats/minute, and respiratory rate 21 breaths/minute. The initial Glasgow Coma Scale score was E3V2M3. Pale conjunctiva, abdominal distension with shifting dullness, spider angioma over the chest wall, and palmar erythema were also found. There was no focal neurological sign such as eye deviation or limb weakness. A computed tomography scan of his brain disclosed several tiny hypodense lesions involving bilateral basal ganglia and left periventricular white matter in favor of old lacunar infarction.

His laboratory tests showed a white blood cell count of $15.19 \times 10^9/L$ with 84% granulocytes, 12% lymphocytes, and 4% monocytes. Hemoglobin was 10.1 g/dL, the mean corpuscular volume was 88.4 fL/red cell, and the platelet count was 332,000/mm³. Serum electrolyte levels were 139 mmol/L Na (normal range 135–147 mmol/L), 3.8 mmol/L K (normal range 3.4–4.7 mmol/L), and 8.3 mg/dL Ca (normal range 8.4–10.6 mg/dL). The renal function test showed 34 mg/dL blood urea nitrogen (normal range 7–20 mg/dL) and 2.1 mg/dL serum creatinine (normal range 0.7–1.5 mg/dL). Liver function tests showed 10 U/L glutamate pyruvate transaminase (normal range 0–40), 91 U/L γ -glutamyl transpeptidase (normal range 8–60 U/L), 89 U/L alkaline phosphatase (normal range 42–128 U/L), and 57 μ g/dL serum ammonia (normal range 12–66 μ g/dL). The viral hepatitis test gave a negative result for anti-HCV antibody and HBs antigen and a positive result for anti-HBs antibody. The family did not report a history of alcoholism in the patient.

A chest radiograph showed a band-like lesion over the right lower lung field (Fig. 1) which was not seen on previous chest radiographs. The family reported an episode of choking while the patient was in stupor. Cefoxitin (2 g every 12 hours) was initially administered intravenously to treat suspected aspiration pneumonia based on the case history and findings of the chest radiograph. Kidney, ureter, and



Figure 1. Chest radiograph taken in the emergency department showing a band-like lesion over the right lower lung field.

bladder radiographs showed floating bowel gas, indicating ascites formation. An abdominal computed tomography scan (Fig. 2) showed findings compatible with liver cirrhosis. Diagnostic abdominal paracentesis showed turbid fluid with a white blood cell count of 3510/mm³ with 65% granulocytes, 30% lymphocytes, 4% monocytes, 1% eosinophils, 2 g/L total protein, and 388 g/L lactate dehydrogenase. Gram staining and acid-fast staining of ascites were both negative. The serum–ascites albumin gradient (SAAG) was >1.1 g/dL.

Shock with acute respiratory failure developed on Day 1 of hospitalization and the patient was intubated, ventilated mechanically, and given fluid resuscitation and inotropic drugs. An initial sputum smear showed a high polymorphonuclear (PMN) cell count with a predominance of GNB. With a diagnosis of liver cirrhosis complicated with SBP, multiple organ failure, and GNB pneumonia, antibiotic treatment was changed to cefotaxime 2 g every 8 hours.

Table 1 gives the serial analysis of the ascites fluid. Initially, the patient's vital signs and level of consciousness improved gradually and the repeat analysis of ascites also showed a reduction in the PMN cell count. On Day 4 of hospitalization, however, his clinical condition deteriorated again and shock with multiple organ failure was exacerbated despite fluid hydration and the use of inotropic drugs. The sputum culture grew *Enterobacter cloacae*. Four sets of ascites bacterial cultures showed negative results. Based on culture-negative neutrocytic ascites, tuberculous peritonitis and malignancy could not be excluded. An abdominal computed tomography scan showed no evidence of a primary tumor with peritoneal carcinomatosis. We looked for other evidence of disseminated TB and collected TB culture from sputum, ascites fluid, urine, and blood, but

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