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CLINICAL CASE

Disseminated Tuberculosis in an Immunocompetent Patient: The Answer is in the Liver

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Abstract Tuberculosis, a chronic infectious disease caused by *Mycobacterium tuberculosis*, may invade all organs but mainly affect the lungs. We report a case of disseminated tuberculosis with hepatic, pericardial and pleural involvement and a review of the relevant literature. A 64-year-old Portuguese male was admitted with epigastric and right upper quadrant pain associated with low grade fever, fatigue, nausea, anorexia, weight loss (6 kg) and mild jaundice. A chest X-ray showed cardiomegaly and a computed tomographic scan of the thorax and abdomen revealed a mild left pleural effusion, a thickened pericardium with signs of incipient calcification and hepatomegaly. The echocardiogram suggested the diagnosis of constrictive pericarditis. Liver biopsy revealed granulomatous lesions with central caseating necrosis. Tuberculosis is usually associated with atypical clinical manifestations. Imaging examination combined with histopathological features, a high index of clinical suspicion and improvement with antibacilar therapeutic are necessary to confirm a diagnosis, especially in the cases of extrapulmonary tuberculosis.
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PALAVRAS-CHAVE
Imunocompetência;
Fígado;
Tuberculose

Tuberculose Disseminada num Doente Imunocompetente: A Resposta Está no Fígado

Resumo A tuberculose, uma doença infecciosa causada pelo *Mycobacterium tuberculosis*, pode invadir todos os órgãos, afectando sobretudo os pulmões. Relatamos um caso de tuberculose hepática com envolvimento pericárdico e pleural e uma revisão da literatura relevante. Um homem de 64 anos, de nacionalidade portuguesa, foi admitido por dor no quadrante superior direito do abdómen e no epigastro associada a febre baixa, astenia, náuseas, anorexia, perda de peso (6 kg) e icterícia. A radiografia de tórax revelou cardiomegalia e a tomografia computadorizada de tórax e abdómen revelou um derrame pleural esquerdo ligeiro, um pericárdio espessado com sinais incipientes de calcificação e hepatomegalia. O ecocardiograma era sugestivo de pericardite constritiva. A biopsia hepática revelou granuloma com necrose caseosa

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central. A tuberculose geralmente está associada a manifestações clínicas atípicas. A presença de aspectos imagiológicos em conjunto com características histológicas típicas, um elevado índice de suspeita clínica e resposta à terapêutica antibacilar são necessários para confirmar o diagnóstico, especialmente nos casos de tuberculose extrapulmonar.
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1. Introduction

Tuberculosis (TB) can present with a variable clinical picture, consequently, making the diagnosis difficult. Disseminated tuberculosis (TB) is defined as having two or more noncontiguous sites resulting from lymphohematogenous dissemination of *Mycobacterium tuberculosis*.[1] Extrapulmonary involvement occurs in one-fifth of all TB cases[2] and it may occur in the absence of histological and radiological evidence of pulmonary infection.

Hepatic tuberculosis, particularly in the absence of miliary tuberculosis, is rare[3] and can occur as a primary case or due to reactivation of an old tubercular focus.[3]

Diagnosis is often difficult as clinical manifestations are nonspecific and because it can mimic several other disorders. Clinically, hepatic tuberculosis can present as fever of unknown origin, abdominal pain and jaundice, which if not timely diagnosed and properly managed can culminate in fulminant hepatic failure that may prove fatal.[4]

Pericardial involvement in tuberculosis may result in acute pericarditis, chronic pericardial effusion, cardiac tamponade or pericardial constriction.[5] The disease has insidious onset and patients may present with fever and can manifest vague precordial pain or cardiomegaly on a chest radiograph.

Definitive diagnosis of extrapulmonary TB can be very difficult; it relies on histological and/or bacteriological findings of the liver tissue obtained by biopsy.[6] Sometimes, clinical diagnosis is only confirmed after complete recovery with specific treatment.[6]

2. Case report

A previously healthy 60-year-old Portuguese male presented with intermittent sharp epigastric and right upper quadrant pain in the last 2 weeks, low grade fever, fatigue, nausea, anorexia and weight loss (6 kg) in previous 2 months. He admitted moderate alcohol consumption.

There was no previous history of similar pain and his past medical history was uneventful as well as his family history.

On physical examination at admission, he had a temperature of 37.2 °C, a heart rate of 102/min, a blood pressure of 110/79 mmHg and a respiratory rate of 24 breaths per minute with oxygen saturation of 98% on ambient air. He was alert but appeared uncomfortable, had mild jaundice and moderate tenderness in the right upper abdomen with palpable hepatomegaly. Cardiovascular examination revealed sinus rhythm; jugular veins were distended to the angle of the mandible when the patient sat upright, but no further venous engorgement was noted on inspiration and no peripheral edema was found. No enlargement of the superficial lymph nodes was found.

Initial blood tests investigations (Table 1) reported normal hemogram and renal function; mild hyponatremia 133 mmol/L; elevated C-reactive protein (CRP) 19.95 mg/dL; abnormal liver tests with alanine aminotransferase (ALT) of 282 U/L, aspartate aminotransferase (AST) of 339 U/L, alkaline phosphatase (AP) of 186 U/L, gamma-glutamyl transpeptidase (GGT) of 252 U/L, total bilirubin of 3.1 mg/L with a direct fraction of 2.0 mg/dL; and prothrombin time (PT) was 16.7 s. Amylase and lipase were within normal ranges as well as cardiac markers, other serum electrolytes and urinalysis. Electrocardiogram showed sinus tachycardia, abdominal ultrasonography revealed hepatomegaly and slightly coarse echotexture of the liver, suggesting hepatic steatosis and chest X-ray revealed only a small left pleural effusion.

After blood and urine cultures were taken, antibiotic therapy (amoxicillin/clavulanic acid) was initiated at the Emergency room because of the suspicion of an infectious process, and the patient was transferred to Gastroenterology ward to further investigation. During the first few days of hospital stay, there was improvement of right upper abdominal pain but afterwards the patient started to complain of a precordial sharp pain worsening with deep inspiration and had evening fever (up to 39.5 °C), with chills and sweating. Additional study (Table 1) showed anemia (Hb = 9.8 g/dL) of chronic illness with low iron levels of 16 μg/dL, low total iron binding capacity of 199 μg/dL and high ferritin of 987 ng/mL; leukocytosis (11×10^3 cells/mm^3) with neutrophilia (78%); his erythrocyte sedimentation rate (ESR) was elevated to 103 mm/h and the CRP peaked to 37 mg/dL. Peripheral blood immunophenotypage did not show any abnormality as well as blood smear. There was an improvement of transaminases (AST = 83 U/L and ALT = 140 U/L) and normalization of bilirubin values but the cholestasis parameters worsened, with GGT reaching up to 1371 U/L and AP to 507 U/L.

A chest X-ray showed a discrete left pleural effusion and now there was cardiomegaly (Fig. 1) and a computed tomographic (CT) scan of the thorax and abdomen revealed a mild left pleural effusion, a thickened pericardium with signs of calcification (Fig. 2) and hepatomegaly. The echocardiogram (Fig. 3) with color Doppler suggested the diagnosis of constrictive pericarditis (CP), with a thickened pericardium and incipient calcification predominantly over right heart, without signs of hemodynamic compromise.

Blood, sputum, and urine were all negative for bacteria and acid-fast bacilli on both smears and mycobacterial

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