Case Reports

Obstructive jaundice secondary to paracoccidioidomycosis

Introduction

Paracoccidioidomycosis (PCM) is a chronic granulomatous disease caused by Paracoccidioidesbrasiliensis,endemic in Latin America, primarily involving the lungs, but also the skin, mucous membranes, lymph nodes, and internal organs.[1,2,3] Contagion occurs often by inhalation of conidia and mycelial fragments, producing an infection that can spread to other tissues via lymphatic and hematogenous means.[1,2,3,4] We report a rare case of involvement of lymph nodes in the liver hilum by P. brasiliensis.

Case Report

A 56-year-old man, with past history of rural work, between 7 to 18 years of age, presentedwith abdominal pain in the upper right quadrant for 6 months duration, associated with slowly progressive jaundice, choluria and fecal acholia. He also suffereda weight loss of 18 kg in this period. On clinical examination, he wasjaundiced, with tenderness inupperright abdominal quadrant. Laboratory studies revealed high levels of serum transaminases (AST: 148 mg/dL; ALT: 170 mg/dL), canalicular enzymes (ALP: 627 mg/dL; GGT: 750 mg/dL) and bilirubins (direct: 13.4 mg/dL; indirect: 10.7 mg/dL). Abdominal ultrasonography revealed an enlarged liver and dilatation of intra and extra-hepatic biliary ducts. Computed tomography showed a solid and infiltrative lesion of 8.8 cm x 4.5 cm involving the celiac trunk, head and body of pancreas and distal choledocus, whose caliber was increased, and wasassociated withenlarged lymph nodes in the spleen hilum and around the abdominal aorta (**Figure 1**). Magnetic resonance cholangiography showed the lesioncircumferentially involving medial to distal common biliary duct (**Figure 2**).The main diagnostic hypothesis was lymphoproliferative disease. An ultrasonography-guided percutaneous biopsy was carried out and revealed a chronic nonspecific inflammatory process. The patient underwent an endoscopic retrogradecholangiopancreatography (ERCP), which showed biliary obstruction from medial to distal choledocus (**Figure 3**). A stent was placed beyond the obstruction. Biliary

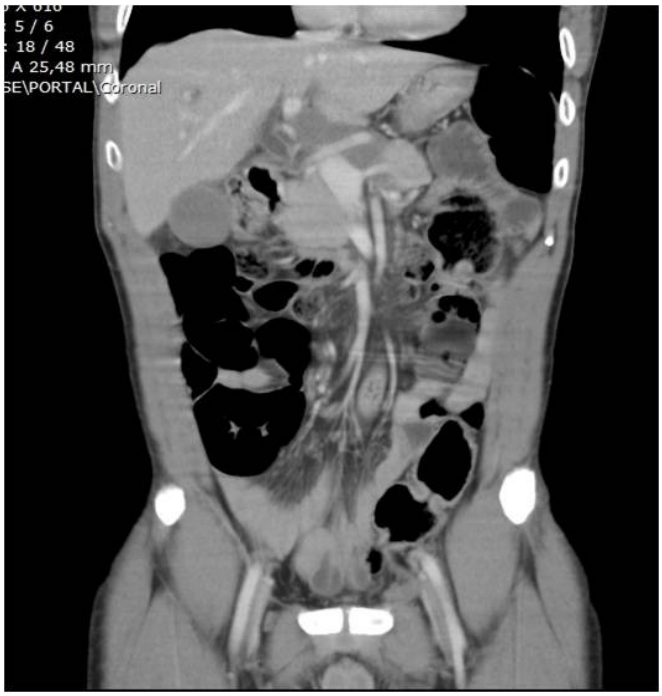

Figure 1: Computed tomography

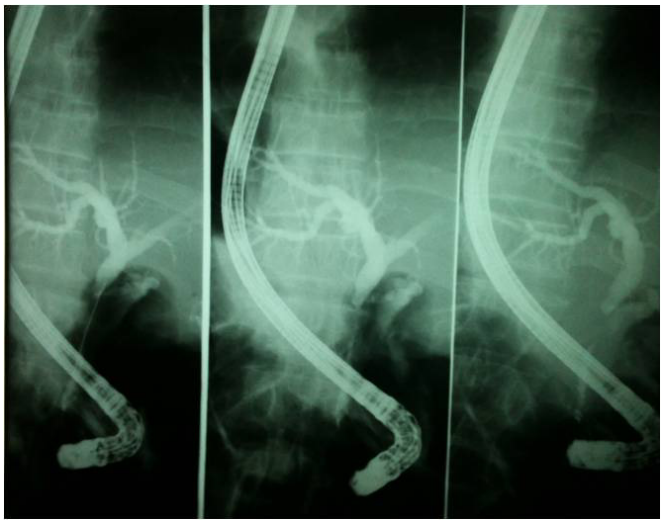

Figure 2: Endoscopic retrograde cholangio-pancreatography

brushing cytology revealed no neoplastic cells. After 30 days, the patient presented with an episode of acute cholangitis. Wide spectrum antibiotic therapy was carried out and succeeded. Surgical intervention was advised. At laparotomy several enlarged lymph nodes around distal choledocus, involving head and body of pancreas, celiac trunk and portal vessels, and without cleavage plane, was observed. An incisional biopsy of lymph nodes was carried out. A Roux-en-Y hepaticojejunostomy was created. The patient had an uneventful postoperative stayand was discharged from the hospital 5 days after surgery. Hepatic enzyme and bilirubin levels were normal after 14 days. Histological examination revealed a granulomatous caseous process with P. brasiliensis.

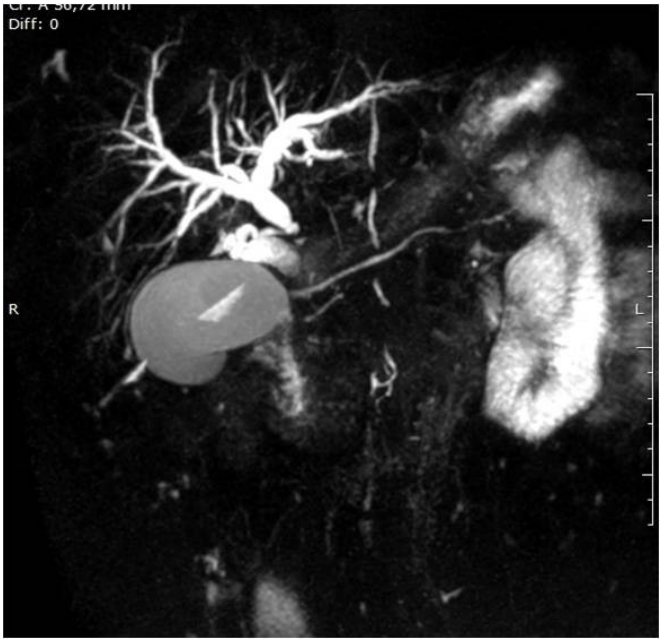

Figure 3: Magnetic resonance choloangiography

Treatment with itraconazole 200 mg daily wasinitiated and lasted 6 months. After 12 months the patient has no clinical or tomographic evidence of disease.

Discussion

PCM is classifiedin acute and subacute or chronic forms. Acute and subacute forms develop from a primary undetected pulmonary lesion that evolves quickly with lymphatic and hematogenous spread to the monocyte-macrophage system. Chronic form is the most commonly seen type in clinical practice and develops from the primary pulmonary complex or from reactivation of a quiescent pulmonary or metastatic focus, after a relatively long duration, and often expressed by pulmonary and tegumental damage.[5]GastrointestinalPCM is rarely recognized because of nonspecific clinical manifestations. It can present as part of a progressive dissemination of infection or as a result of local complications from a silent healing process.[2]Jaundice secondary to PCM may occur as a result of three independent mechanisms: 1) extrinsic compression of biliary ducts due to head of pancreas or liver hilum lymph node involvement; 2) biliary duct involvement due to granuloma (intrinsic lesion); 3) hepatitis secondary to PCM. The first is the most common mechanism. [2]

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Gastrointestinal stromal tumor mimicking a prostatic abscess

Introduction

Gastrointestinal stromal tumors (GIST) in the pelvis can cause intestinal obstruction or symptoms mimicking benign prostatic hyperplasia.[1]

Case Report

A 32-year-old man presented with rectal bleeding and passage of fleshy pieces per rectum, for 4 months. He also had constipation, distension of abdomen and occasional vomiting. 5 months ago he had presented with fever, burning micturition and urinary retention at another hospital. Sonography and computed tomography of the abdomen had suggested a diagnosis of prostatic abscess. He thenunderwent transurethral drainage of 700 ml pus; and culture had shown a growth of E.coli.He had repeated episodes of retention of urine, which was relieved only by catheterization.

On presentation at our institution, he was afebrile, pale, hypotensive and had tachycardia. Foley's catheter was in-situ