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Porta Hepatis Mass

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A 64-year-old man from the Philippines presented with 5 months of progressive fatigue, worsening jaundice, and a 13-kg weight loss. He denied fever, chills, and abdominal pain. Laboratory test results were significant for a total bilirubin level of 14.4mg/dL (to convert to micromoles per liter, multiply by 17.104), mildly elevated transaminases, and a white blood cell count of 13 300/mL (to convert to $\times 10^9/L$, multiply by 0.001) with a normal differential. Computed tomography (CT) of the abdomen and pelvis (Figure, A) demonstrated a heterogeneously enhancing hepatic hilar mass with extensive intrahepatic biliary ductal dilatation. The hilar mass measured $4.4 \times 4.3 \times 3.5$ cm; the gallbladder was calcified. ACT scan of the chest demonstrated a 7-mm left upper lobe partially calcified nodule and cardiophrenic and gastroesophageal lymphadenopathy. Endoscopic retrograde cholangiopancreatography demonstrated focal biliary strictures involving the right and left hepatic ducts. Cytological examination of bile duct brushings showed reactive ductal epithelial cells but no malignancy. Endoscopic ultrasonography with fine-needle aspirates revealed inflammatory cells with granulomatous changes. Serum carcinoembryonic antigen and cancer antigen 19-9 levels were 1.9 ng/mL (to convert to micrograms per liter, multiply by 1) and 293 U/mL, respectively. QuantiFERON (interferon- γ release assay; Qiagen), serum cryptococcal antigen, and serum *Histoplasma capsulatum* antigen results were all negative. Aerobic, anaerobic, mycobacterial, and fungal cultures from the CT-guided liver biopsy were negative. Laparoscopic biopsy (Figure, B) of several hepatic lesions demonstrated necrotizing granulomatous inflammation with giant cells and central necrosis. Grocott methenamine silver and acid-fast bacilli staining were negative for fungal or mycobacterial organisms, respectively.

Diagnosis

B. Hepatic tuberculosis

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Discussion

Histopathological examination of the patient's mass demonstrated necrotizing granulomatous inflammation suggestive of *Mycobacterium tuberculosis* infection. Although acid-fast bacilli stains and mycobacterial cultures of the lesions were negative, positive results are only present in 0% to 45% and 10% to 60% of cases, respectively.¹ Diagnosis using polymerase chain reaction analysis of fine-needle aspirates has been suggested; however, this has a positive predictive value of only 57%.² Primary hepatic tuberculosis represents a rare presentation, because the hepatic system is hypothesized to be unfavorable for mycobacterial growth owing to lower oxygen tension as compared with the lungs.³ Moreover, hepatic tuberculosis is typically due to reactivation of an old pulmonary tuberculous focus. The epidemiologic history of the patient's country of origin combined with evidence of a remote pulmonary infection seen on CT scan support this diagnosis.

Serum biochemical studies, the clinical presentation of obstructive jaundice, and radiographic evidence of a mass involving the confluence of the right and left hepatic ducts were suggestive of a hilar cholangiocarcinoma, also known as a Klatskin tumor. Fine-needle aspirate examination, however, showed no evidence of malignancy, and the laparoscopic liver biopsies demonstrated necrotizing granulomatous inflammation without evidence of neoplastic transformation. The differential diagnosis for necrotizing granulomatous hepatitis includes infection, autoimmune disease, and drug reaction. Included on the differential is lymphomatoid granulomatosis, an Epstein-Barr virus-associated B-cell lymphoma. Lung involvement is always observed, while hepatic involvement is only seen in 29% of cases.⁴ Finally, Langerhans cell histiocytosis is typically characterized by granulomatous inflammation of the lungs, bone, lymph nodes, and skin, and only 10% of cases are diagnosed in patients older than 55 years.⁵

Combining the clinical, radiographic, and pathologic findings to rule out neoplastic, vasculitic, and immunologic processes, a suspected diagnosis of hepatic tuberculosis was made with a positive treatment response as clinical confirmation. Clinical response to antituberculosis therapy is typically seen within 2 to 3 months after initiating treatment.¹ While this patient did not require any further surgical interventions and continues to do well since completing therapy, surgical management of hepatic tuberculosis is generally indicated if there is tuberculosis-related biliary tract compression resulting in jaundice, portal hypertension, biliary tract bleeding, or diagnostic uncertainty as was the original situation in this case.⁶ In summary, despite the difficulty in diagnosing this severe, chronic infection, hepatic tuberculosis is a treatable disease that should be included on the differential diagnosis for hepatic and biliary tract masses.

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WHAT IS YOUR DIAGNOSIS?

- A.** Hilar cholangiocarcinoma (Klatskin tumor)
- B.** Hepatic tuberculosis
- C.** Lymphomatoid granulomatosis
- D.** Langerhans cell histiocytosis of the liver

A Computed tomography of the abdomen and pelvis **B** Photomicrograph of hepatic lesions **Figure.**

A, Computed tomography of the abdomen demonstrates a $4.4 \times 4.3 \times 3.5$ -cm heterogeneous mass in the hepatic hilum (white arrowhead). There is a hepatic stent extending from the right hepatic duct into the second portion of the duodenum. Extensive intrahepatic biliary ductal dilatation is noted. Additional focal peripheral hypodensities (black arrowhead) are visualized in the liver parenchyma. B, Photomicrograph (hematoxylin-eosin, original magnification $\times 40$) demonstrates normal liver parenchyma surrounding multiple caseating granulomas with central necrosis (black arrowheads). Sparse giant cells (white arrowhead) are visualized surrounding the granulomas.