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Case Report

Hepatic Tuberculoma: Diagnosis of a Rare Case

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ABSTRACT

Hepatic tuberculomas are rare presentations of gastrointestinal Tuberculosis (TB) caused by Mycobacterium tuberculosis. They usually occur after dissemination of TB from a primary site like the lungs or gut. Our case report is about a 62-year-old gentleman who had disseminated TB and abnormal Liver Function Tests (LFTs). He had a history of alcohol abuse, hepatitis C and liver cirrhosis. On Computed Tomography (CT) scan, a mass 2.3 x 1.9 cm in diameter was noted. A Magnetic Resonance Imaging (MRI) revealed right portal vein occlusion, so hepatocellular carcinoma was suspected. CT guided microwave ablation and a biopsy was done for the mass and it was found to be a tuberculoma and no malignant cells were found. Anti-tubercular therapy was continued for the patient.

Keywords: Liver, Tuberculosis; Hepatocellular carcinoma; Hepatic tuberculoma; Mycobacterium Tuberculosis

INTRODUCTION

Hepatic Tuberculosis is a rare disease (1% of all TB cases) which usually occurs in adults with disseminated Tuberculosis (TB), in whom the disease spreads via hepatic artery, portal vein or lymphatics and reaches the liver.[1] The incidence of the disease has increased due to a rise in the cases of human immunodeficiency virus/acquired immunodeficiency syndrome (HIV/AIDS).[2] The presentation of the disease lacks specific clinical manifestations and imaging features which is why it can easily be misdiagnosed for other illnesses. Image guided fine needle aspiration biopsy is the best method to diagnose the disease.[3]

The type of Hepatic TB our patient had was Macronodular TB, subtype: Pseudotumoral TB. These are less common and are seen as 1-3cm large tumor-like calcified masses on imaging. They exhibit heterogeneous enhancement with areas of necrosis on CT and MRI.[1] Anti-TB treatment is effective in most cases. However, if there are indications for surgery or are difficult to diagnose, surgical procedures along with anti-tubercular drug therapy could be adopted.[3]

CASE PRESENTATION

A 62 years old Hispanic man originally from Mexico with Hepatitis C, liver cirrhosis and pulmonary TB (Tuberculosis) was transferred from Laredo Medical Center to Texas Center of Infectious Diseases (TCID) because of elevated Liver Function Tests (LFTs) and was off TB treatment due to history of drug induced acute liver failure. He had a history of Chronic Obstructive Pulmonary Disease (COPD), Gastroesophageal Reflux Disease (GERD), chronic cholelithiasis and prostate disease. The patient was a smoker since he was 14 years old and smoked one pack per day. He drank several beers per week.

On general physical examination, the patient was afebrile and pleasant. His abdomen was moderately distended and tense with caput medusae, mildly tender to palpation on the right side and hepatomegaly was noted. He had normal reflexes with a Glasgow Coma Scale (GCS) score of 15. Systemic examinations of the cardiovascular, gastrointestinal, respiratory systems and all other systems were unremarkable.

Baseline investigations were conducted, which showed a low hemoglobin level (10.5 gm/dL) and platelet count ($47\times10^3/\mu L$). His Liver Function Tests (LFTs) were also abnormal. The severity of liver damage can be determined by his CHILD score of 8 which is class B which is shown in detail in Table 1. The detailed baseline investigations are summarized in Table 2.

Table 1: Child-Pugh score of the patient.

Factor	Patient's report	Score
Total Bilirubin (mmol/L)	13.6	1
Serum Albumin (g/L)	19.3	3
INR	2.03	2
Ascites	None	1
Hepatic Encephalopathy	None	1

Table 2: Baseline investigations of the patient, including complete blood count, renal, and liver function tests.

Investigations	Normal range	Patient's report	Comments
Complete blood count (CBC)			
Hemoglobin	13-18 gm/dL	3.8	Decreased
Hematocrit	40%-50%	29.80%	Decreased
MCV	80-100 fL	87.9	Normal
WBC	4.5-11.0 × 10^3/L	1.4	Normal
Platelets	150-450×10^3/µL	169	Decreased
Electrolytes			
Na	135-145mEq/L	123	Decreased
K	3.6-5.2 mmol/L	3.7	Normal
Cl	98–107 mEq/L	93	Decreased
CO2	23-29 mEq/L	24	Normal
Ca	8.6-10.3 mg/dL	7.4	Decreased

Renal function tests			
BUN	6-20 mg/dL	26	Raised
Creatinine	0.7-1.2 mg/dL	0.98	Raised
Liver function tests			
Bleeding time	2-9 minutes	10.56	Raised
Albumin	3.4 to 5.4 g/dL	1.93	Decreased
ALT	<50 U/L	52	Raised
AST	5-40 U/L	108	Raised
ALP	50-136 U/L	151	Raised

MCV- Mean Corpuscular Volume; BUN- Blood Urea Nitrogen; ALT- Alanine Aminotransferase; ALP- Alkaline Phosphatase

Hepatocellular carcinoma was suspected due to the presence of liver cirrhosis and right portal vein thrombosis seen on the MRI (Figure 1). The MRI also showed perihepatic ascites, recanalization of umbilical veins and portosystemic shunt and periportal edema. The portal vein and its left branch, SMV, and splenic vein were patent. Peri-esophageal varices were present. There were signs of cholelithiasis with gallbladder wall edema. Few subcentimeter hyperintense cysts were seen in the pancreatic body. There were patchy airspace opacities seen at the base of the right lung and small amounts of pleural effusion.

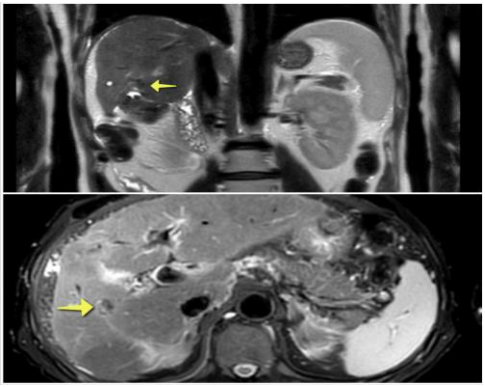

Figure 1: MRI of the abdomen.

On Computed Tomography (CT) Scan of the abdomen, hepatomegaly was seen with a focal hypodense lesion in the right lobe of liver measuring 2.3 x 1.9 cm in diameter with no further characterizable features (Figure 2). The patient underwent CT guided microwave ablation of the liver mass. Pre-operative CT and ultrasound of the abdomen showed a cirrhotic liver and a tumor thrombus in the right portal vein. The lesion was 1.4 cm in the fifth segment of the liver. The final histopathological report showed necrotizing granulomatous inflammation (Figure 3). There were neither malignant cells nor microorganisms seen. Because of the patient's history of tuberculosis and hepatitis C,

infection was not ruled out and additional tissue sampling was sent for culture and molecular microbial testing which tested AFB positive and susceptible to RIPE therapy.

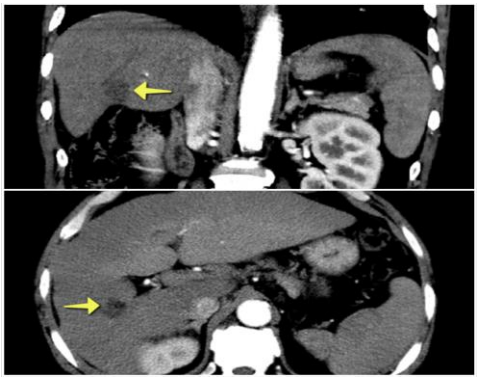

Figure 2: CT of the abdomen.

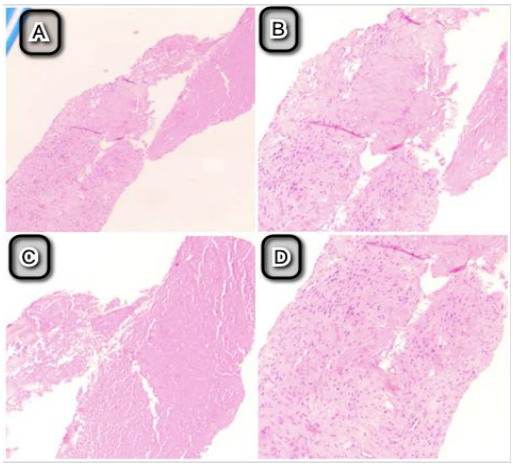

Figure 3: Histological appearance of the excised mass.
Images A and B show the granuloma and necrosis. Image C shows only the necrotic part. Image D shows only the granuloma part.

DISCUSSION

Tuberculosis is a great healthcare challenge especially in developing countries. Because of a rise in immigration from endemic regions, diseases like Acquired Immunodeficiency Syndrome (AIDS) and multi drug- and extensively drug- resistant (MDR, XDR) strains of MTB, the disease is also spreading in the developed countries.[4] Abdominal TB is rare and can occur in four ways: (a) Hematogenous or lymphatic spread of TB disease, (b) Reactivation of TB infection and its spread, (c) Ingestion of the pathogen and (d) Direct invasion of the tissue.[5]

Hepatic TB is a rare form of abdominal TB. It usually occurs after dissemination of the disease from a primary site like the lungs or gastrointestinal tract. There are three types of liver TB: miliary hepatic TB, biliary tract TB and

nodular hepatic TB. Miliary hepatic TB is the most common type.[6] Biliary tract TB can present in different ways but its most common presentation is the fever, jaundice, and hepatic calcifications triad.[7,8] Nodular hepatic TB as in our case, is the rarest form of TB and there have only been few publications related to it. It is termed as Tuberculoma and it was first described by Bristowe in 1858.[7,9]

Nodular hepatic TB appears as distinct nodules and therefore can be difficult to diagnose. It does not have a specific clinical presentation and presents with vague clinical symptoms like fever, weight loss, right upper quadrant pain and anorexia.[8,9] It can easily be treated medically but if not treated in time then it can quickly progress to liver failure and death.[9,10] The treatment includes RIPE therapy for two months which is followed by only isoniazid and rifampin for four to seven months. The total duration of the treatment is six to nine months.[7,8]

Ultrasound imaging is used to screen for this disease.[5,8] On ultrasound, hepatic tuberculomas are seen as well marginated hypoechoic lesions with or without calcifications.[10] On CT scan, it typically presents as a well-circumscribed lesion with a moderate peripheral enhancement. The canalicular form shows expanded intrahepatic bile ducts with calcifications along their walls.[8,11] A definitive diagnosis of this disease is conventionally made by acid-fast staining of clinical isolates followed by culture or histological examination of the tissue specimens for evidence of caseation with granuloma.[5,8]

CONCLUSION

Hepatic Tuberculomas are a rare and difficult diagnosis. They should be suspected in adults with disseminated TB and a liver mass. The liver mass can be seen on an ultrasound and MRI. To confirm the diagnosis a biopsy is needed which should show necrosis and granulomas. The treatment is necessary to prevent the progression of the disease into liver failure. It can be treated with RIPE therapy for TB and very large tuberculomas can be removed surgically.

DISCLOSURES: None

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