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Case report

Diagnostic Dilemma of Hepatic Tuberculosis in Presence of Portal Hypertension: A Rare and Relevant Case

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Abstract

Tuberculosis (TB) is and remains a growing public health problem worldwide, especially in developing countries. Hepatic tuberculosis is an extremely rare form of extra pulmonary TB. Our case report describes hepatic tuberculosis revealed by an atypical sign: portal hypertension (PHT) in a Moroccan immuno competent young man. ***Case Report:*** A 28-year-old male with no medical history, was admitted with an abundant ascites, and a chronic right hypochondrium pain. The biology data found a low protein concentration ascitic fluid with signs of portal hypertension in the abdominal ultra sound (US). An abdominal CT scan demonstrated a multi nodular liver with profound multiple lymph nodes some with a necrotic center. The diagnosis of multifocal tuberculosis was clinched on by the positivity of Xpert MTB/RIF assay on sputum and on hepatic needle biopsy. ***Conclusion:*** Portal hypertension secondary to hepatic tuberculosis is exceptional and often causes a diagnostic dilemma. It should be considered in the differential diagnosis of non-cirrhotic portal hypertension especially in endemic areas.

Keywords: ***portal hypertension, hepatic tuberculosis, immuno competent, Xpert MTB/RIF***

Introduction

Tuberculosis is a global health problem. It can affect any organ or tissue in the abdomen such as gastrointestinal tract, peritoneum, lymphatic, system and solid organs. Hepatic TB represents less than 1% of all cases of TB, It is a rare entity and poorly described in literature even in endemic areas.

TB is a great mimicker that has a myriad of unexpected presentations, such as portal hypertension. There are a number of causes for portal hypertension, but there were a few reports that are associated with hepatic tuberculosis.

We report a rare case of hepatic tuberculosis revealed by portal hypertension in a Moroccan immune competent young man.

Case

A 28-years-old Moroccan man was admitted to our department after presenting with a one month history of abdominal swelling, right upper quadrant chronic pain and altered general condition. He had no personal or familial past and took no treatment. He was neither smoker nor alcohol consumer. He had been previously vaccinated against tuberculosis (BCG) and he denied any medical history of hepatitis, liver cirrhosis, tuberculosis or pancreatitis.

The patient described a one-month history of progressive weakness, anorexia and weight loss of 8 kilograms, and also complained of night sweats.

On arrival, the patient was stable and his body temperature was 37°C. The physical examination revealed a mild abdominal tenderness with an abundant ascites. Hepatomegaly and splenomegaly were difficult to examine. There was no jaundice and no other signs of hepatocellular insufficiency. No peripheral lymph node was palpable and there was no peripheral edema. Heart and chest sounds were normal. The rest of the physical examination was normal.

Laboratory data revealed high white blood count at 15870/µL (NV: 4000-10000µ/L) and a high C reactive protein (CRP) at 427mg/L (NV: <6) with a normal prothrombine time at 80% (NV: 70-100%) and albumin at 11,8g/L(NV: 32-45g/L). Renal function and liver enzymes tests were normal.

The ascites tap found a lymphocytic transudative fluid (protein level = 4 g/L) with 266 elements/mm3 (70% lymphocytes).

The patient underwent an abdominal US, which demonstrated a multinodular hepatomegaly, with signs of portal hypertension (splenomegaly, collateral veinous circulation, dilated portal trunk).

Consequently, the abdominal CT scan confirmed the presence of hepatomegaly with several nodular lesions and profound lymph nodes some with a necrotic center (**Fig.1**). The nodules were spontaneously hypodense, slightly enhanced after contrast agent administration, the most voluminous measured 65mm x 39mm. (**Fig.2**)

The esophagogastroduodenoscopy (EGD) was completely normal without any existence of varices. Furthermore, HIV, HBV and HCV, syphilis serologies and autoimmune antibodies were negative. Tumor markers including alpha-fetoprotein (AFP), carcino embryonic antigen (CEA), and contrast angiography (CA) 19-9 were also normal.

The patient underwent a liver needle biopsy that was tested for Xpert MTB/RIF and turned out positive. No resistance for rifampicine was detected.

The chest radiography showed no evidence of pulmonary lesions. Three days research of BK in sputum was negative, the culture and AFB stains were also negative however an Xpert MTB/RIF assay on sputum turned out positive.

Multifocal tuberculosis was therefore confirmed and an antituberculosis treatment was introduced including rifampicin 10 mg/kg/d, Isoniazid 5 mg/kg/d, pyrazinamide 40 mg/kg/d and ethambutol 30 mg/kg/d. The patient was re-evaluated after one month and showed signs of favorable clinical and biological evolution during treatment (restored appetite, weight gain, ascites control, normalization of albumin).

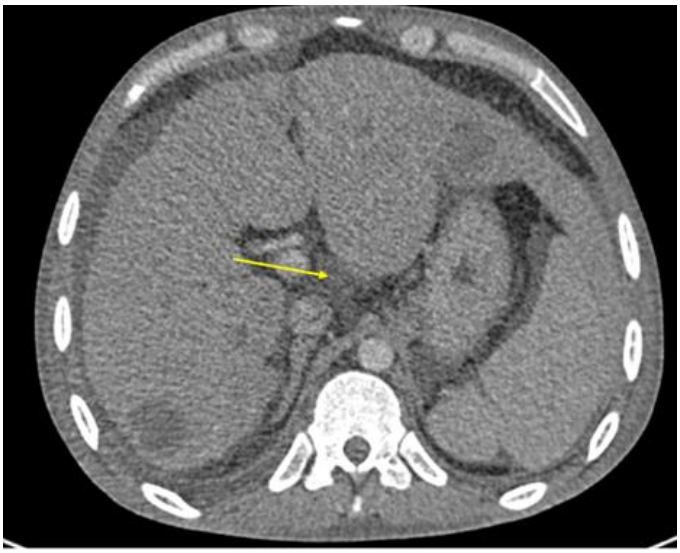

Fig 1: Abdominal enlarged lymph node with a necrotic center.

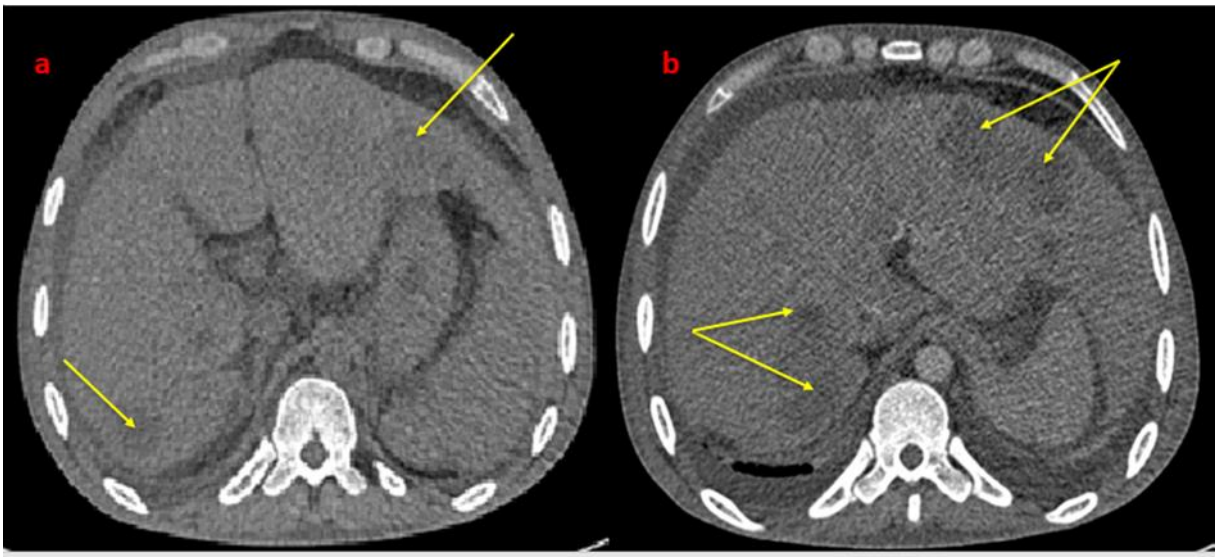

Fig 2: a: Non-contrast-enhanced abdominal CT scan shows multiples hypodense hepatic lesions. b: After the intravenous administration of iodinated contrast the lesion showing moderate enhancement in the portal-vein phase.

Discussion

Hepatic TB is an uncommon extra pulmonary location of tuberculosis that is generally silent or manifesting as a number of non-specific clinical and imaging findings [1]. It's a rare entity that represents 3% of all extra-pulmonary locations and 9% of abdominal locations [2]. Even though the liver tissue is not suitable for mycobacterial growth due to the low oxygen levels, it is thought to be favorable for granuloma formation, usually located near the portal tract [1]. The disease may present at any age but is often seen in young adults [3].

As far as clinical findings are concerned, TB is most frequently associated with hepatomegaly (90%), fever (70%), weight loss (60%), abdominal pain (50%) night sweats, anorexia and rarely jaundice [4]. Our patient was admitted with all of the above-mentioned signs.

Portal hypertension (PHT) is an unusual and exceptional revelation mode of tuberculosis.

There are multiple suggested mechanisms of PHT in TB such as: portal vein thrombosis secondary to lymph nodes, splenic vein thrombosis due to pancreatic tuberculosis or retroperitoneal tubercular abscess, hepatic TB causing sinusoidal compression, and hepatic outflow obstruction due to constrictive pericarditis [5].

The CT scan of our patient showed enlarged lymph nodes with no signs of portal compression. We excluded all other potential, extra hepatic, vascular causes of portal hypertension such as portal or splenic vein thrombosis and Budd-Chiari syndrome. Therefore, the portal hypertension in our case was caused by the sinusoidal compression by the hepatic nodules.

The imaging, including US, CT, and MRI, is not always so specific and has little role in the diagnosis of hepatic TB, but it could be helpful in the differential diagnosis and for detecting associated complications [2].

Hepatic lesions can mimick a number of neoplastic and non-neoplastic conditions. It is a great mimicker of which the differential diagnosis should include: primary hepatic tumors such as

cholangiocarcinoma and hepatocellular carcinoma, pyogenic abscess and metastases [6].

There are two types of hepatic TB on cross sectional imaging; the micronodular and macronodular form. The micronodular form is frequently seen and is often associated with hematogenous dissemination of TB bacilli. The macronodular form, on the other side, is less frequent and thought to be the result of conglomeration of miliary granulomas. A number of names can refer to this type of lesion, such as tuberculoma, tuberculous abscess and pseudo tumoral TB. Mixed type of hepatic TB can occur, it associates macronodular and micronodular lesions [3].

In order to confirm hepatic tuberculosis, the diagnosis approach should include: the identification of caseating granuloma or a non-caseating granuloma alongside with tuberculi bacilli elsewhere or the finding of acidophilic bacilli in liver tissue or remission on tuberculosis therapy [7].

The diagnosis in our case was difficult due to various symptoms and nonspecific imaging findings. The low protein ascites tap, the presence of US signs of portal hypertension and multinodular hepatomegaly were likely to lead to a diagnosis of hepatocellular carcinoma. However the patient had no history of hepatitis or signs of liver cirrhosis. Also, the serum tumor markers (alpha foeto protein, carcinoembryonic antigen, CA-19.9) were completely normal.

Furthermore, the positivity of an Xpert MTB/RIF assay on liver biopsy alongside the fast response to anti-mycobacterium treatment were strong positive arguments to assess and confirm the diagnosis of hepatic tuberculosis.

The diagnosis of Tuberculosis has been revolutionized by the Xpert MTB/RIF, especially pulmonary TB and some cases of extra pulmonary tuberculosis [8,9].

Although not many cases were reported about the role of X pert MTB/RIF in the diagnosis of hepatic tuberculosis [10,11], it proved to be particularly useful when the clinical suspicion is high and is the key to a precocious management and a better evolution.

Subsequently our patient showed several signs of clinical recovery after quadruple therapy introduction: ascites regression, restored appetite, weight gain and albumin normalization within one month.

Conclusion

Hepatic tuberculosis is a rare etiology of portal hypertension that often causes a diagnostic dilemma. It can be mistaken as a liver tumor such as intra hepatic carcinoma due to nonspecific and numerous imaging manifestations. Molecular techniques such as Xpert MTB/RIF are the gold standard for the diagnosis of this rare location. Medical management is key to an early treatment of hepatic TB and is correlated to an excellent prognosis.

Author Contributions

FA wrote the manuscript, SB wrote the manuscript with her support, AA supervised the findings of this work, ZS supervised the findings of this work, SO supervised the findings of this work, KK supervised the findings of this work.

Patient's Consent

Informed consent has been obtained from the patient for publication of the case report.

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Declaration of conflicting interests

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