Case Report

Inflammatory pseudo-tumoral primary hepatic tuberculosis: a surgeon's dilemma

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ABSTRACT

Pseudo-tumor hepatic tuberculosis is a rare entity. Tuberculosis involving the liver in the absence of active pulmonary tuberculosis is very uncommon. It is characterized by non-specific symptoms and radiological polymorphism. This case report illustrates the difficulty in reaching the correct diagnosis in case of hepatic masses, which are most often confused with carcinoma of the liver, primary or metastatic and hence, in the past referred to as pseudo-tumoral hepatic tuberculosis. We report a case of an inflammatory pseudo-tumor of the liver due to tuberculosis in a 6 years child. Computer tomography showed evidence of a large heterogeneously enhancing predominantly cystic multiseptated mass in left lobe of liver with peripheral nodular enhancement and persistent enhancement of internal septations and nodular solid components. Hence malignant tumor was initially considered in view of the radiological findings. Alpha fetoprotein was within normal limits. The patient was treated surgically by debridement of left lobe of liver.

Keywords: Primary hepatic tuberculosis, Inflammatory pseudo-tumor, Hepatic tuberculosis, Mimicking malignancy

INTRODUCTION

Hepatic tuberculosis presenting primarily in the absence of pleuro-pulmonary involvement is infrequent.¹ Usually, tubercular liver abscesses are associated with an immune-compromised state, with a focus of infection in the lungs or in the gastrointestinal tract.² Localization to the liver without active pulmonary, peritoneal or miliary tuberculosis (TB) presenting as pseudo-tumoral masses (macro nodular) form of hepatic tuberculosis, characterized by clinical and radio-logical polymorphism, is rare.³ Most of the cases reported in the literature are in the form of a localized mass and are usually diagnosed as a primary or secondary liver tumor.³,⁴ Final diagnosis is made after multidisciplinary concentration regarding imaging techniques like ultrasonography, computed tomography (CT), magnetic resonance imaging (MRI) along with pathological examination of percutaneous needle biopsy or resected specimen.⁵ The difficulty in making a proper diagnosis leads to surgical intervention. In most cases, treatment of the disease provides a positive outcome. This article presents a case of inflammatory hepatic pseudo-tumor due to tuberculosis that was difficult to diagnose pre-operatively. Definitive diagnosis could be made only after surgery.

CASE REPORT

A 6 years male child was brought to the hospital with complaints of high-grade fever, vomiting and pain in the epigastric region of moderate intensity. On examination, the child was less than 3rd percentile for height and weight and body mass index (BMI) was 14.5 kg/m². He was pale with no icterus. A 7×5 cms tender, hard lump was palpable
in the epigastrum with local rise of temperature. The liver edge was 6 cms below the costal margin and liver span was 16 cm. A provisional clinical diagnosis of a liver abscess was made and investigated. Blood tests showed hemoglobin 7.7gms, total leucocyte count (TLC) 41.1x1000/cumm, peripheral blood film (PBF) - mild anaemia with leucocytosis, neutrophilia, liver function test (LFT) showing total bilirubin as 0.8 mgm/dl, aspartate aminotransferase (AST) 45 IU/l, alanine transaminase (ALT) 31 IU/l and alkaline phosphatase (ALP) 102 IU/l. Ultrasonography showed a large heterogeneously hypo to iso-echoic soft tissue mass lesion measuring nearly 9.2x7.3 cm seen in the segment 4 of liver with solid and cystic components and internal calcification. Doppler study showed increased central as well as peripheral vascularity, ill-defined margins with areas of internal haemorrhage likely suggestive of? Hepatoblastoma? Hepatocellular carcinoma in segment 4 of liver. Contrast enhanced CT (CECT) scan abdomen showed a large heterogeneously enhancing predominantly cystic multi-septated lesion in the left lobe of liver suggestive of? malignant Undifferentiated embryonal sarcoma (Figure 1).

**Figure 1:** Mass measuring 9.2x8x7.3 cm which appeared hypo dense to liver parenchyma in non-contrast images. Mass showed peripheral nodular enhancement and persistent enhancement of internal septations and nodular solid components in all phases with splenomegaly.

USG guided fine needle aspiration cytology (FNAC) left lobe of liver showed intact and degenerated neutrophils in the background of necrotic material. No malignant cells were seen. Opinionated as acute suppurative pathology. Aspirate for culture showed no growth, Alfa fetoprotein (AFP) was 0.01 ng/ml (normal <20 ng/ml). After appropriate antibiotic therapy and packed red blood cell transfusion, he was taken up for surgery.

Operative findings showed the mass involved the whole of the left lobe. On dissection, it had cheesy white tissue with necrotic and hemorrhagic areas within it (Figure 2).

Maximum debridement of the mass was done. Post operatively he had an uneventful recovery.

**Figure 2:** Intra-operative image of the liver mass.

Histopathology impression was multiple micro abscessess with extensive proliferation of myofibroblasts with lymphoplasmacytic infiltrate around the necrotic areas (Figure 3). Tumor of muscle origin localized to myofibroblasts in the stroma of tumors was suspected. Immunohistochemistry (IHC) marker for ALK-1 was negative. Acid fast bacilli (AFB) stain was done on strong clinical suspicion was positive. The patient was started on Anti Tuberculous treatment.

**DISCUSSION**

TB is challenging health problem in developing countries. Abdominal tuberculosis primarily involves peritoneum and gastrointestinal tract. PHT is relatively rare and accounts for 0.5% of primary TB. PHT commonly presenting as micro-nodular form (<2 mm). Levine classified hepatic involvement in TB as hepatic involvement into 4 types - military TB, pulmonary TB, PHT as focal tuberculoma/abscess, TB cholangitis. Reed classified into 3 types: hepatic TB with generalized military TB, primary military TB of the liver, and primary hepatic tuberculoma/abscess. Hepatic TB lacks typical clinical features of fever, right upper quadrant pain, weight loss and hepatomegaly as they are less specific. The description of the radiological features are also variable. PHT rarely presents as macro-nodular, pseudo tumoral form or as
single or multiple tuberculoma lesions > 2 cms. TB bacilli disseminate through hepatic artery in miliary TB. Milary TB accounts for 80% cases of HT. Macro nodular HT requiring resection is confusing and a difficult question. IPT mimics hypo vascular tumors secondary to the plentiful fibrosis and there is delayed enhancement on CT similar to liver metastatic lesions. CT contrast enhancement varies from none to heterogeneous or homogenous. In endemic areas of TB with a clinical presentation of malignant like liver mass, pain abdomen, loss of weight and fever, HT should be considered as differential diagnosis along with associated inflammatory syndrome. The trouble over seeing patients of PHT lies in arriving at the right diagnosis. Rarely PHT is considered in DD of liver lesions because of its low prevalence. Therefore, a high degree of clinical suspicion is required for diagnosis. The oxygen content of hepatic blood is rather low and does not favor survival of TB bacilli. Hepatic TB commonly includes elevated ALP and GGT with occasional elevation of ALT and AST. Therefore intense clinical doubt is necessary for diagnosing PHT. Proposed theory for pathogenesis of PHT is that the bacilli gain entry into the portal system through a breech in the intestinal mucosa in a form of an ulcer which heals later leaving behind no trace of initial focus. The bacilli ascend into the liver parenchyma and immune complexes in the liver result in the formation of a granuloma. Clumping of granuloma forms tuberculoma. Doubt of neoplasm remains a significant worry for the specialist and serves to legitimize surgery on minimal suspicion. Laboratory investigation may show cholestasis, an inflammatory syndrome and elevated liver enzymes. CT varies with the stage of evolution of the disease. Initially isodense then hypodense lesions may be found due to caseating necrosis mimicking primary/secondary liver tumors with calcification at a later stage. Information on the wide spectrum of CT findings of abdominal tuberculosis should make the clinician think about PHT diagnosis in high risk patients. Histology usually has giant cells with caseating necrosis. Sensitivity of Ziehl-Neelsen stain on the tissue is less than 20%. Sensitivity of culture for TB bacilli is less than 33%. Affirmation of mycobacterium in the tissue test is uncommon. In this patient ZN stain was positive revealing a tubercular bacillus. Surgical debridement or resection is usually performed due to lack of a prior proper diagnosis. Extrahepatic TB is not usually found in these cases. The treatment of choice is Anti tubercular drugs.

CONCLUSION

Primary pseudo-tumoral hepatic tuberculosis is rare. A definitive diagnosis can generally be made only on the basis of surgical evidence. Pre op diagnosis of hepatic tuberculosis is challenging mimicking malignancy. Although rare, HT should be kept in mind as a differential diagnosis in hepatic mass lesions in endemic areas. We have a follow up of the patient for 8 months on ATT with complete resolution of symptoms. The child is feeding well and has gained weight.

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REFERENCES


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