Tubercular Abscess: An Uncommon Manifestation Of Hepatic Tuberculosis- A Case Report

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Citation

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Abstract

CASE HISTORY

A 34 year old young male presented to the medical emergency with complaints of abdominal pain and distension for 10 days. There was an associated history of jaundice of 2 weeks duration. Further history and clinical examination were not suggestive of and underlying chronic liver disease.

Lab investigations revealed hemoglobin of 9.7 gm/dl, TLC of 10,000 cells/cu mm with a differential of P70 L28 E2. Total bilirubin was 7.2 with a direct fraction of 6. Hepatic enzymes were also mildly elevated. ESR was 35. The patient was HIV negative. Renal functions were normal.

Sonographic evaluation revealed multiple conglomerate hypo echoic lesions in periportal area consistent with hepatic abscesses (figure 1 and 2). Few enlarged periportal lymph nodes were also seen.

Figure 1

Figure 1: Ultrasounds scan through liver showing multiple thick walled hepatic abscesses.

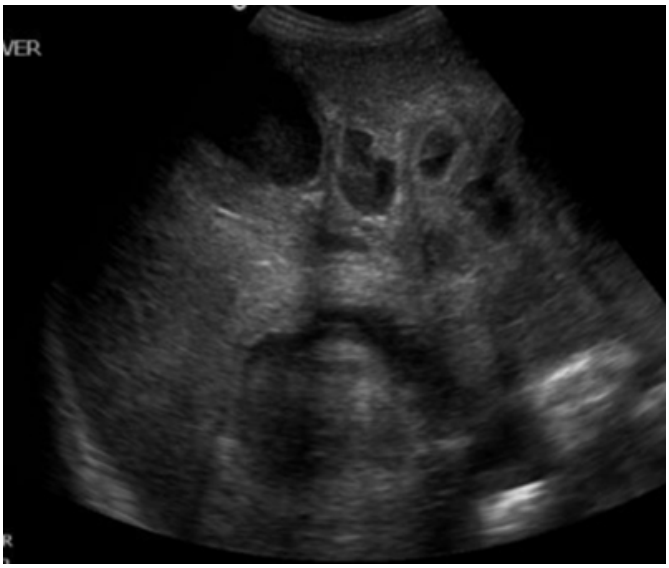
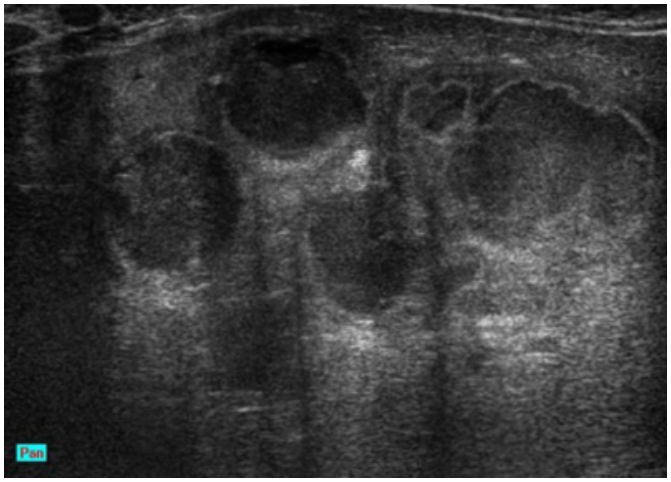


Figure 2

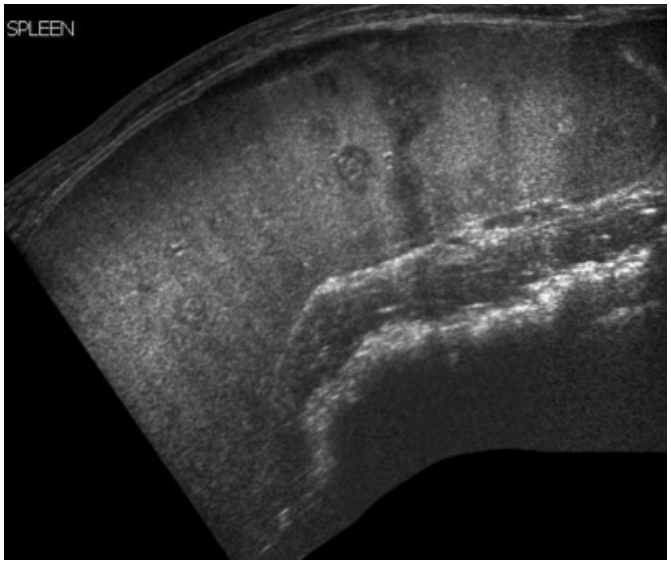
Figure 2: EFOV image with a high frequency probe well demonstrates the conglomerate nature of the abscesses with a typical "cluster sign"



Associated splenomegaly was noted with multiple hypoechoic lesions in the splenic parenchyma (figure 3). Small amount of ascites was also seen.

Figure 3

Figure 3: EFOV image of spleen showing multiple hypo and hyper echoic lesions.



CT scanning revealed similar findings with conglomerate hepatic abscesses showing "cluster sign" with mild peripheral enhancement (figure 4). Splenomegaly with hypodense splenic granulomas and associated ascites were also seen (figure 5).

Figure 4

Figure 4: Axial post contrast CT image showing multiple conglomerate hypo dense lesions giving rise to "cluster sign"

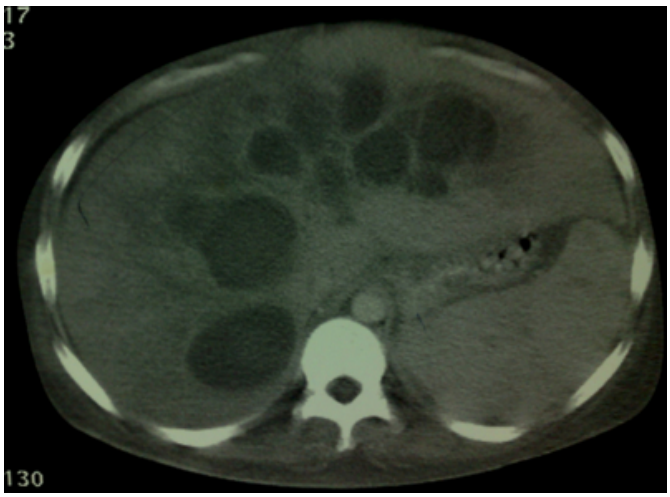
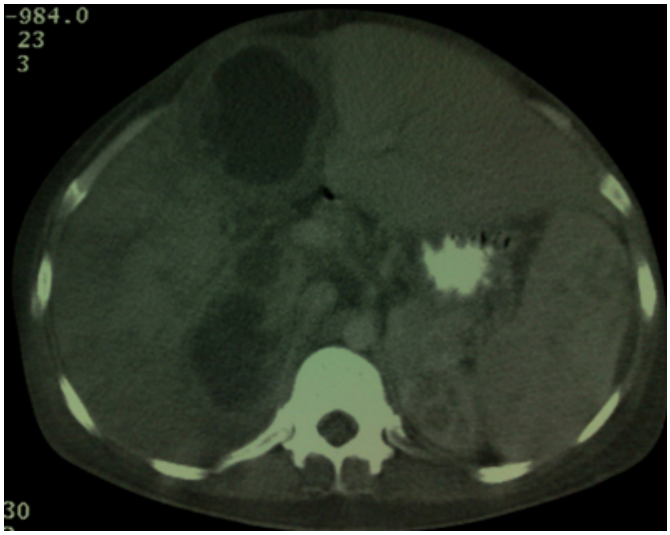


Figure 5

Figure 5: Another caudal image showing thick walled abscess cavity with multiple ill defined hypodense splenic granulomas. Also note few small hypo dense portal lymph nodes and small amount of ascites.



FNA of the hepatic abscesses revealed presence of acid fast bacilli in the pus. Subsequent PCR and Bactec results were positive for M. tuberculosis. The patient was put on anti tubercular therapy. Subsequent imaging at two weeks interval revealed mild reduction in the size of the lesion. The patient is currently taking ATT and has improved symptomatically.

DISCUSSION

Tuberculosis can involve the liver in many ways. Both hepatic parenchyma and biliary system may be affected. The

pattern of hepatic parenchymal involvement varies between military, nodular and mixed types.

The Miliary form is the most common and is thought to result from hematogenous dissemination of tubercle bacilli through the hepatic artery ([1], [2], [3], [4]). Concurrent pulmonary tuberculosis may or may not be seen.

Miliary hepatic tuberculosis is seen as multiple or diffuse miliary micro nodular lesions less than 2 cm in diameter, often as part of disseminated systemic tuberculosis and is therefore not difficult to diagnose correctly ([5], [6]).

Nodular hepatic tuberculosis includes lesions more than 2 cm in diameter and is probably secondary to conglomeration of miliary granulomas ([7]). Such lesions usually appear hypo dense on CT and may show foci of punctuate calcification.

Tubercular abscess is formed when there is subsequent liquefactive necrosis in the centre of tuberculous granuloma. These appear as minimally enhancing thick walled cystic lesions which may show conglomeration.

Such conglomerated abscesses give the appearance of a "cluster sign" on CT, as seen in our case. Such an appearance however is not pathognomonic of a tubercular abscess and is also seen in pyogenic liver abscesses.

Mixed tuberculosis is said to be present when there are both miliary and macronodular lesions. This gives rise to different density lesions on Ultrasound and CT since the various lesions are pathologically in different stages of evolution.

Tuberculous cholangitis is considered to be extra parenchymal tuberculosis by some authors. It is a rare entity and occurs mainly in children. It usually presents with obstructive jaundice with cross sectional imaging revealing dilated IHBR with miliary calcifications along the course of bile ducts ([8], [9]).

Another pattern of tuberculous hepatic involvement recently reported by Sheng yu et al involves miliary hepatic lesions in sub capsular region resulting in formation of a thickened capsule and giving the appearance of "frosted liver". This pattern has been termed as serohepatic type of tuberculosis by the authors ([10]).

LEARNING POINTS

Although miliary hepatic tubercular involvement is common, hepatic tubercular abscess, tubercular cholangitis and sero hepatic patterns of involvement are rare.
In endemic regions with high prevalence of tuberculosis, and presence of supportive imaging findings, uncommon manifestations of hepatic tuberculosis should be considered, especially in the appropriate clinical setting.

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