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Hepatocellular carcinoma presenting as liver abcess

Introduction

Hepatocellular carcinoma (HCC) is the commonest primary malignant tumor of the liver.HCC presenting as a pyogenic liver abscess is reported but is uncommon and rarely addressed in literature[1]. It is challenging to diagnose HCC presenting as a pyogenic abscess.[2]

Case Report

A 38 year old male patient presented with the complaint of continuous, dull aching pain in the right hypochondrium for 2 months. The pain was of moderate intensity,non radiating and associated with high grade fever with chills. There was history of loss of appetite and weight loss of approximately 3 kilograms over two months.On examination he was febrile and hemodynamically stable. He had tender hepatomegaly 6 cm below the costal margin in the mid-clavicular line. The liver was firm in consistency, had rounded margins and a smooth surface.He had pallor and the rest of the general and systemic examinations were normal. Lab investigations revealed a TLC of 24,600 /cu.mm with 83 percent polymorphs,hemoglobin was 9.7 g/dl and platelets were 3.99lacs/cu.mm.His liver function tests were normal except for a raised alkaline phosphatase (ALP), with total bilirubin of 1.2 mg/dl, Direct:0.8 mg/dl,Indirect:0.4 mg/dl,aspartate aminotransferase:35 IU/L,alanine aminostransferase:12 IU/L, ALP:260 IU/L (Normal upper limit:110), total protein:7.6 g/dl,albumin: 4.6 g/dl,globulin:3.0 g/dl, INR: 1.06.Malaria antigen, Widal test,urine R/M,blood culture and urine cultures were all negative.He was positive for HBsAg and negative for HBeAg with HBV DNA titer of 1180 IU/mL.HIV and antiHCV antibodies were negative. Ultrasound abdomen revealed hepatomegaly (18.1 cm),spleen 10.1 cm.The gallbladder,portal vein and common bile duct were normal while minimal free fluid was found in the abdomen.A 9.9 x 6.8 x 9.4 cm sized mixed echogenic, predominantly hypoechoic collection was noted in seg V and VI of the liver, abutting the right kidney.Impression of a liver abscess was given. Intravenous antibiotics were started and ultrasound guided aspiration of the lesion was done. The cellular smears revealed innumerable neutrophils admixed with several foamy macrophages,bare nuclei and degenerated cells over a necrotic background. No amoebea, scolices or hooklets, epitheloid cell clusters or multinucleated giant cells were seen.No atypical or malignant cells were detected. Diagnosis of pyogenic liver abscess was suggested.Aspirate culture showed no growth,Indirect hemagglitination test for anti amoebaantibodies was negative and serum alfa fetoprotein was within normal limits (4.11 IU/ml).

An ultrasound guided liver biopsy findings showed fibrin plugs enmeshing few cords and ribbons of partly degenerated cuboidal cells. The cells possess granular acidophilic cytoplasm and rather monotonous round nuclei with minimal anisonucleosis. At places, "ghost outlines "of similar cells were seen. The polyhedral cells could represent a necrotic neoplasm however,the accompanying smears were entirely inflammatory. To further characterize the lesion dynamic C.T. abdomen **(Figure 1)** was done. It showed 12 x 7 x 7.5 cm

heterogeneously enhancing soft tissue mass noted in segment VII of the liver and in the sub hepatic region. It showed multiple non-enhancing areas within. Rest of the liver was normal and no typical pattern of enhancement was noted. Thepatient's fever responded and the TLC normalized after a course of antibiotics but his right hypochondrium pain persisted. In view of HBsAg positive status and persistent symptoms, MRI abdomen and repeat biopsy of the lesion were done.

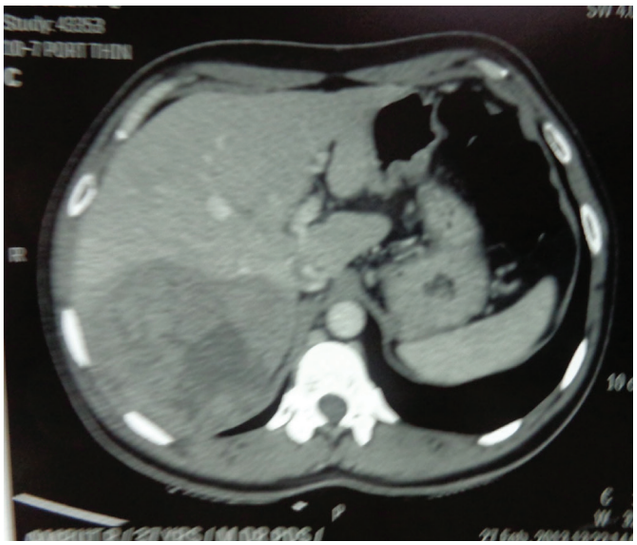

Figure 1: 12 x 7 x 7.5 cm heterogeneously enhancing soft tissue mass noted in segment VII of the liver and in the sub hepatic region on CT scan

MRI findings **(Figure 2)** showed a large,well defined 13 x 9 x 10 cm heterogeneous exophytic,bilobed, irregular mass lesion involving segments VI and VII of the liver. It showed hemorrhagic changes within, especially in the inferomedialexophytic component. On T2,hyperintense edema in the adjoining liver parenchyma was found. There wasno significant arterial phase enhancement,no evidence of contrast washout and the rest of the liver parenchyma appeared normal with no other focal lesions.The second imaging also did not show a typical pattern of enhancement.

The microscopy sections from the liver SOL showed tumor cells which were uniform and round,with moderate cytoplasm.Tumor cells were arranged in cords and sheets and few abnormal mitotic figures were present.Diagnosis ofa malignant epithelial tumor was made.The possibility of a neuro-endocrine tumor or a well differentiated HCC was suggested.A few sections showed features of chronic hepatitis with fibrosis.On Immunohistochemistry, the tumor cells expressed cytokeratin,hepatocyte specific antigen (OCYH1E5) and Glypican3 While they wereimmunonegative for synaptophysin,chromogranin A,CK-7 and CK-20.The Mib-1 index was approximately 60 -70 %.Impression ofa well differentiated hepatocellular carcinoma was provided based on the IHC.

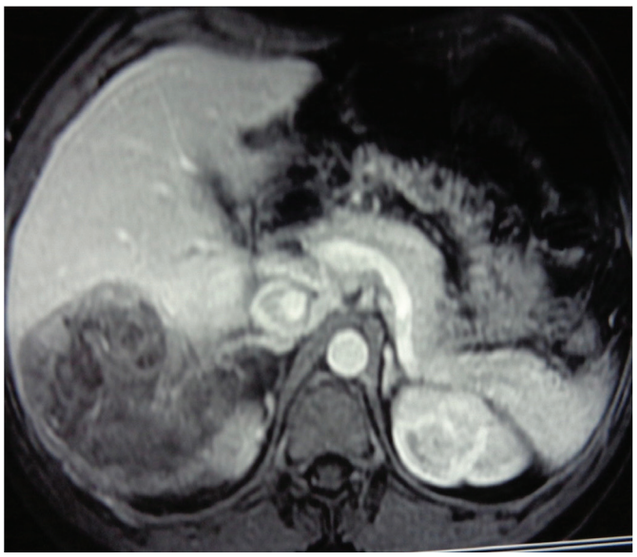

Figure 2: MRI findings showed a large,well defined 13 x 9 x 10 cm heterogeneous exophytic,bilobed, irregular mass lesion involving segments VI and VII of the liver.

So we had a patient who was positive for the HBsAg, presenting to us with what seemed to be only a pyogenic liver abscess, finally diagnosed with a well differentiated HCC following biopsy and IHC. He had atypical findings on imaging, normal serum AFP levels and showed no evidence of liver cirrhosis.The patient underwent hepatic resectionof the tumor.He tolerated the procedure well and is now under surveillance for recurrence.

Discussion

HCC may present as liver abscess because of spontaneous tumor necrosis and/or biliary obstruction caused by tumor thrombi, superimposed with bacterial infection. Elevated serum alfa feto protein levels may help in diagnosing HCC but they may not always be raised, as in our case.A high index of suspicion is required to diagnose HCC as imaging and biomarkers may not always help.

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Menetrier's Disease with Early Gastric Cancer

Introduction

Menetrier's Disease is a rare form of hypertrophic gastropathy characterized by giant gastric rugal folds and associated with enteral protein loss and hypoalbuminemia. Until now only around 200 cases have been reported, and of these only fewer than 20 cases have been associated with gastric adenocarcinoma. Our case is unique due to its rarity: the incidental detection of adenocarcinoma in a patient first detected with Menetrier's disease and the presence of characteristic clinicopathologic manifestations.

Case History

A 49 year old gentleman presented to our department with the history of loose stools, anorexia, weight loss and generalized malaise of 3 month's duration. He had no other complaints. At presentation he was anemic and had bilateral pedal edema. His abdominal examination was unremarkable. His hemogram, liver function tests and renal parameters were essentially normal except for a low serum albumin of 2.0 mg/dl. An ultrasonogram abdomen showed diffuse wall thickening involving the fundus of the stomach, and upper gastrointestinal endoscopy revealed hypertrophic rugal folds involving the fundus and body of the stomach along with hypersecretion of mucus. Endosonography showed marked thickening of the mucosa and muscularis mucosa with normal appearing submucosa, muscularis propria and serosa **(Figure 1a)**.

Subsequently, multiple snare biopsies of the stomach were taken. These showed evidence of chronic gastritis with foveolar hyperplasia, dysplastic glands, reversal of pit-gland ratio and areas of intestinal metaplasia with foci of well differentiated adenocarcinoma **(Figure 2)**. A contrast enhanced CT scan of the abdomen confirmed thickened mucosal folds involving the fundus and body of the stomach with antral sparing. It also revealed symmetric enhancement of hypertrophic gastric mucosa **(Figure 1b)**. CT did not show any abnormal enhancing mass lesion. He was planned for, and underwent a total gastrectomy with an uneventful postoperative period. The mucosal surface of the resected stomach exhibited typical cerebriform convolutions **(Figure 3)** and histopathology confirmed features of Menetrier's disease with foci of well differentiated adenocarcinoma.

Discussion

Menetrier's Disease is a hypertrophic gastropathy believed to be caused by an overexpression of tumour growth factor α (TGF-α), a ligand for the tyrosine kinase epidermal growth factor receptor, resulting in selective expansion of surface mucous cells and hypersecretion of mucus and hypochlorhydria which also predisposes to a malignancy.[1] It affects mostly adults, but can also occur in children. The average age at diagnosis in adults is 55 years, with male preponderance. Menetrier's disease should be distinguished from other hypertrophic gastropathies such as the Zollinger–Ellison syndrome, lymphocytic gastritis, and diffuse-type adenocarcinoma (linitis plastica), all of which exhibit generalized rugal hypertrophy. The hyperplastic changes seen in Menetrier's disease typically involve the body and/or the