Case Report: A Rare Case of Hepatocellular Carcinoma with Extensive Peliotic Change Mimicking Hemangioma

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Abstract: Peliosis hepatis, is a hepatic lesion characterized by blood - filled parenchymal cavities randomly scattered throughout the liver, was first described by Wagner (1). Although peliosis has been observed in liver parenchyma in association with several diseases and medications, peliosis hepatis - like blood - filled cavities are also observed with hepatocellular carcinoma (HCC). HCCs with extensive peliotic change do not have typical enhancement pattern of HCC and may mimic Hemangioma. This is a case report of 81 year old man with history of weight loss, reduced appetite and weakness and a large space occupying lesion in left lobe of liver on Ultrasound abdomen. Triphasic CECT and MRI showed atypical filling pattern and was similar to Cavernous Hemangioma. Laparoscopic left lateral sectionectomy was done and histopathology of the specimen revealed well differentiated HCC with extensive peliotic change.

Keywords: HCC, Cirrhosis, Hepatoma, Alpha - fetoprotein, Liver SOL

1. Introduction

Peliosis hepatis, a hepatic lesion characterized by blood filled parenchymal cavities randomly scattered throughout the liver, was first described by Wagner (1). Although peliosis has been observed in liver parenchyma in association with several diseases and medications, peliosis hepatis - like blood - filled cavities is also observed in the hepatocellular carcinoma (HCC) (2 - 4). This finding is generally referred to as 'peliotic change 'in HCC. HCCs are typically diagnosed by the radiological features of early arterial enhancement and wash out in the venous phase. There are varied differences in enhancement patterns with subtle changes. HCCs with extensive peliotic change do not have typical enhancement pattern of HCC and may mimic Hemangioma. This poses a challenge in the diagnostic evaluation of a patient with space occupying lesion (SOL) of liver especially when the lesion is of significant size. Here, we present one such case of Liver SOL which posed a diagnostic challenge.

2. Case Report

81 years old man, hypertensive, previous smoker (COPD), presented with complaints of decreased appetite, constipation, weakness and weight loss. Abdominal examination revealed a lump in the epigastric region moving with respiration.

Ultrasound Abdomen showed fatty liver and a large well defined circumscribed heterogenous hyperechoic mass lesion in left lobe of liver (reported as - ? atypical hemangioma/? neoplastic).

Triphasic CECT Abdomen showed 'Large multiloculated hypodense lesion involving the left lobe of liver with no evidence of calcifications. In Arterial Phase, the lesion showed continuous peripheral rim enhancement. In Portal Phase, the lesion showed continuous peripheral rim enhancement, enhancement of the septations and central non - enhancing component. In delayed phase, the periphery of the lesion was isodense to the liver parenchyma with central non enhancing component. The lesion was abutting the left portal vein. No filling defects observed within the left portal vein. Impression on CT Imaging was large multiloculated peripherally enhancing lesion involving the left lobe of liver. Possibilities were Atypical Hemangioma, HCC.

MRI Triphasic showed Large well encapsulated exophytic heterointense nodular mass like lesion in segments 2&3 measuring 8.1cm (cc) x 8.8cm (TR) x 8.3cm (AP) with no evidence of any capsular breach. Signal characteristics: T1WI - heterogeneous hypointense in relation to adjacent liver parenchyma. Few small central isointense areas were seen. T2WI showed Heterogenous hyperintense, with multiple small central iso & hypointense signals; DWI - peripheral marked hypertense signal on high B value images with corresponding intermediate ADC values; Central core showed high ADC values; In/Opposed phase - No signal drop; Arterial phase - mild peripheral hyper vascular enhancement; Portal phase - persistent moderate, progressive enhancement of peripheral rim and central septae. No evidence of any thrombosis in portal vein. Compression of distal LHD with associated mild upstream intrahepatic biliary dilatation in segments 2 & 3; Equilibrium and Parenchymal phases: Persistent progressive heterogenous enhancement of thick irregular peripheral rim and central septae; no evidence of any washout; rest of the central core was hypointense in relation to adjacent enhanced liver parenchyma. Impression was a well encapsulated mass lesion in segments 2&3 with signal characteristics and enhancement pattern suggesting a possibility of collision tumour. Enhancement pattern was

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more in favor of Cholangiocarcinoma. Other possibilities were Neuroendocrine tumor, Atypical HCC.

Alpha feto protein (AFP) was 3.26 ng/mL; CA 19 - 9 was 23.6 U/mL. Gastroduodenoscopy showed no varices. Colonoscopy was normal, excluding any unsuspected primary. ECOG performance status was 1, Pulmonary Function Test (PFT) showed mild obstructive pattern

After considering patient's symptoms and imaging findings and a strong suspicion of malignancy, surgery was planned. Laparoscopic Left lateral sectionectomy was done. Specimen removed in endobag through a small supra - pubic transverse incision. Operative time was 6 hours and blood loss was around 200mL.

Post - operative course - Required O2 support for 48 hours; mild rise in liver enzymes on POD - 1 and then showed decreasing trend. Two episodes of acute exacerbation of COPD were managed with nebulizations and O2 support. Minimal bile tinge was noted in the drain for 2 days. There was no Post - hepatectomy bleeding. Patient was discharged on 6th POD.

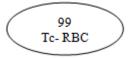
Histopathology multiple sections studied from the tumor showed predominantly dilated sinusoids filled with hemorrhage along with infarctoid changes. Peripheral rim of lesion showed hepatocytes arranged in sheets and acinar pattern. Individual cells were polygonal with vesicular nuclei showing anisonucleosis and granular eosinophilic cytoplasm. Lymphovascular invasion present. Parenchymal resection margin and capsule was free of tumor. Adjacent liver was unremarkable. A diagnosis of well differentiated Hepato cellular carcinoma with extensive peliotic change (pT2) was made.

Challenge in this case was pre - operative inconclusive diagnosis based on imaging. But, combining clinical features and imaging, there was a strong suspicion of malignancy. Another challenge was to perform a major surgery in an octogenarian patient with COPD.

3. Discussion

HCCs are typically diagnosed by radiological features of early arterial enhancement and wash out in the venous phase (5). There are varied differences in enhancement patterns with subtle changes. Peliosis hepatis - like blood - filled cavities are also observed in the HCC (2 - 4). Various mechanisms have been proposed such as obstruction of hepatic outflow at the sinusoidal level, direct breakdown of sinusoidal borders, dilatation of the central vein of the hepatic lobule, or hepatocellular necrosis leading to cavity formation (6). In general, degeneration and/or necrosis of tumor tissue can cause hemorrhage in HCC. On the other hand, tumor tissue around peliotic changes is not degenerative or necrotic and blood is localized within the spaces. Thus, it is suggested that peliotic changes are different from hemorrhage. However, when peliotic changes become extensive, they may rupture and hemorrhage may develop (7). HCCs with extensive peliotic change do not have typical enhancement pattern of HCC and pose diagnostic challenge inspite of advancements in imaging techniques.

In the present case, multiphasic imaging showed centripetal progression of enhancement, similar to the case in hepatic hemangioma. These imaging findings in our present case made it difficult to arrive at correct diagnosis preoperatively. Similar findings were noted in other such reported cases [8, 9, 10].



Ji et al (11) first described the radiological findings in their report on the scintigraphic findings in a patient with pelioid HCC. Kim et al (8) were the first to report radiological findings based on dynamic phase CT in a pelioid HCC. Fujimoto et al (7) in their clinicopathological study found hepatocellular carcinoma with peliotic change in 116 (39.5%) out of 294 cases in patients with ages ranged from 41 to 78 years. In majority of patients who underwent dynamic CT, typical HCC patterns were observed, such as high attenuation in the early enhanced phase and wash - out in the delayed enhanced phase. No specific difference in CT findings was noted in

Peliotic HCCs compared to HCCs without peliosis. No significant difference was noted in the background liver between the two groups. No significant difference was noted in recurrence rate after resection. However, the tumor size in their series was less than 5cm.

Kim et al (8), reported pelioid HCC in a HBsAg +ve patient without cirrhotic changes. In the case reported by Hashimoto et al (9), patient had chronic hepatitis C and alcohol abuse. They have done FNAB and confirmed it as HCC before proceeding for surgery. They proposed that the pathophysiological mechanism of peliotic change in HCC consists of increased sinusoidal pressure and difficulty in the outflow of blood from the tumor because of immaturity of the sinusoid - like structures and central lobular veins in the cancer nodule. In the case reported by Aumpansub et al (10), patient of HBV infection with pelioid HCC showed typical enhancement pattern in a portion of the tumor.

Huang et al (12) have reported a case of atypical liver lesion and was found to have peliosis without HCC. On the contrary, misdiagnosis of a malignancy as a hemangioma is a widely acknowledged but most probably underreported issue (13). So, it has been suggested if radiological findings are not suggestive of cancer, percutaneous liver biopsy can be used to confirm the diagnosis (12).

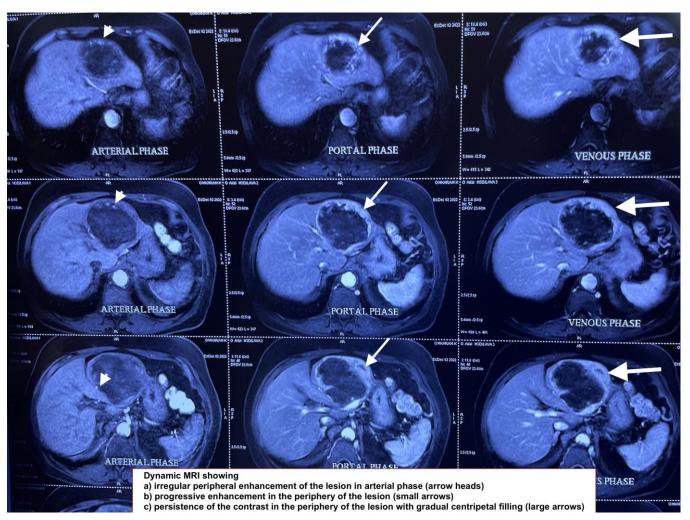
In our case, the patient was negative for viral hepatitis and there was no history of alcohol abuse. However he had fatty liver but the LFT was normal. Tumor markers were within normal limits. It was a real challenge to decide on 'Surgery vs No surgery' in an octogenarian patient with COPD.

However, we had a strong clinical suspicion of malignancy based on patient's history and atypical imaging pattern which led us to go ahead with surgical resection. Cut specimen showed large blood filled spaces with intact capsule. These multifocal blood filled spaces in the lesion might have been responsible for the gradual fill - in pattern of enhancement, similar to cavernous hemangioma.

4. Conclusion

Inspite of advancements in imaging techniques, certain space occupying lesions in the liver pose a diagnostic chal-

lenge, more so in a patient without any underlying viral infection and normal levels of the tumor marker. As HCC with extensive peliotic change poses a diagnostic challenge, there is a need to be aware of this entity and correlation of imaging features with the clinical findings.



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