is an extremely dangerous, corrosive material. On ingestion this will react immediately with saliva to cause serious burns and possible local combustion and even explosion of hydrogen in the mouth or esophagus. The metal's low melting point can cause further complications'.[2,4] They also issue clear instructions regarding the immediate first aid measures to be adopted. All the websites give special emphasis tothe warning: 'Do not induce vomiting'. If the victim is conscious and alert, they advocate administration of large quantities of milk or water. Besides, they uniformly exhort not to give anything by mouth if the person is unconscious.[2-5] However, despite these descriptions regarding the toxic effects of sodium and the first aid measures needed, we could not find a single case report on the subject. Unfortunately in the present case, in the absence of any published literature on the subject, the patient was subjected to induced vomiting by the treating physician.

As there were extensive deep burns with complete mucosal denudation and stricture formation, the patient needed dilatation sessions with Savary Gillard dilators every week in order to maintain nutrition. After 15 sessions, he was administered corticosteroid injections into the esophagus over multiple sites along the entire length. Following corticosteroid therapy, the patient felt better and the intervals between the dilatation sessions lengthened from one week to a fortnight. We plan to insert an expandable plastic stent after a few more dilatations, after the upper and lower ends of the esophagus become healthier with restoration of the mucosa.

This case of corrosive esophageal injury due to elemental sodium ingestion has been reported because of its rarity, with the hope that in future, textbooks of toxicology would make some mention of the measures to be adopted in a case of elemental sodium poisoning.

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A rare case of atypical hemangioma showing centrifugal enhancement

Introduction

Hepatic hemangiomas are the most common benign hepatic masses with a prevalence of 1-20%.[1] They are more commonly seen in females than males with a ratio varying from 2:1 to 5:1.[1] Typical radiographic characteristics of hepatic hemangiomas are well described and extremely specific. However, intra-tumoral structural variation may cause unusual imaging features. This can lead to misleading diagnosis. Hence, knowledge of imaging features of atypical hemangiomas is imperative to avoid unnecessary interventions.

Case report

A 46year-old female patient presented with history of pain in right hypochondrium since four months. The pain was associated with two episodes of vomiting over last three days. There was no history of fever. On examination, mild hepatomegaly was noted with tenderness in right

hypochondrium. Liver function tests were normal. Ultrasound abdomen revealed a large calculus impacted in the gall bladder neck. However, there was no evidence of any wall thickening or pericholecystic collection. The liver showed a heterogeneous hypoechoic lesion with geographic distribution in the left lobe with few similar smaller areas in the right lobe also. Small peripheral echogenic foci were noted which did not cast a distal acoustic shadow (**Figure 1**). These lesions did not show any abnormal vascularity on color Doppler scan, neither within the lesion nor at their periphery (**Figure 2**). In view of the heterogeneous multifocal lesions in the liver, a CT scan was advised. The plain scan revealed near complete replacement of the left lobe of liver with a hypodense lesion showing small peripheral calcific nodules along with few similar low density round lesions in the right lobe (**Figure 3**).

Few peripheral globular enhancing areas were seen in the left lobe lesion in the arterial phase along with the central enhancing area (**Figure 4**). Few right lobe lesions also showed central enhancing nodular areas. The venous phase showed centrifugal enhancement in all the lesions (**Figure 5**). A diagnosis of atypical hemangioma was considered at this stage and an MRI was done. The MRI showed the lesions to be hypointense on T1WI (**Figure 6**) and as hyperintense as CSF on T2WI (**Figure 7**). Dynamic MR contrast study showed the lesions to follow a centrifugal enhancement pattern with

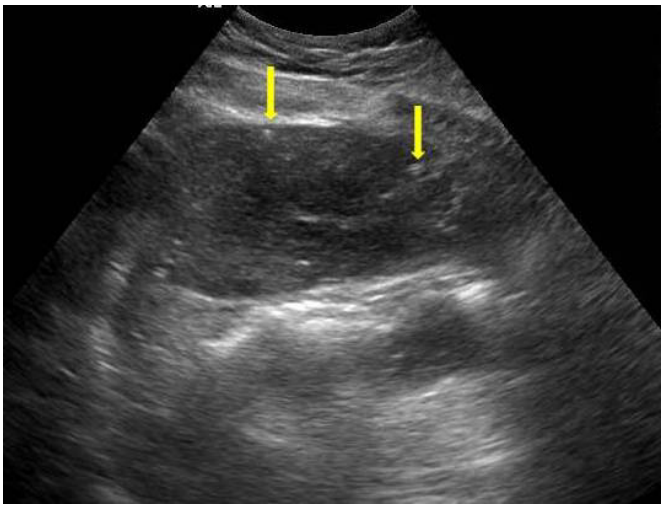

Figure 1: Transverse ultrasound image of the abdomen showing a heterogeneous hypoechoic lesion (arrow) with geographic distribution in the left lobe of liver with small peripheral echogenic foci

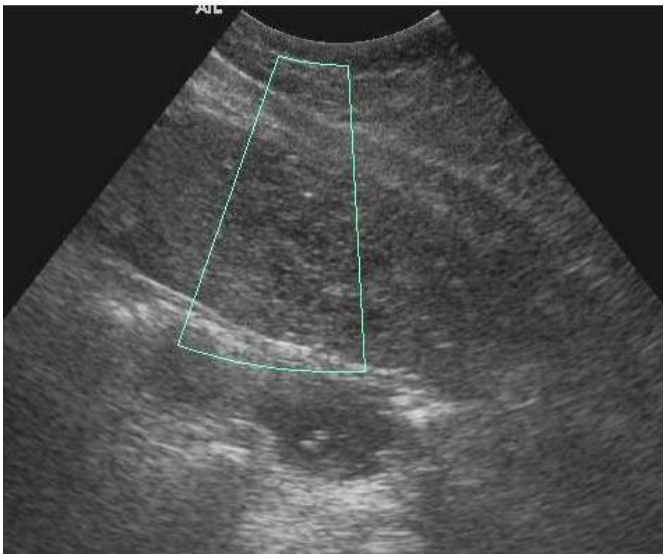

Figure 2: Color Doppler study did not show any abnormal vascularity within the lesion shown in figure 1

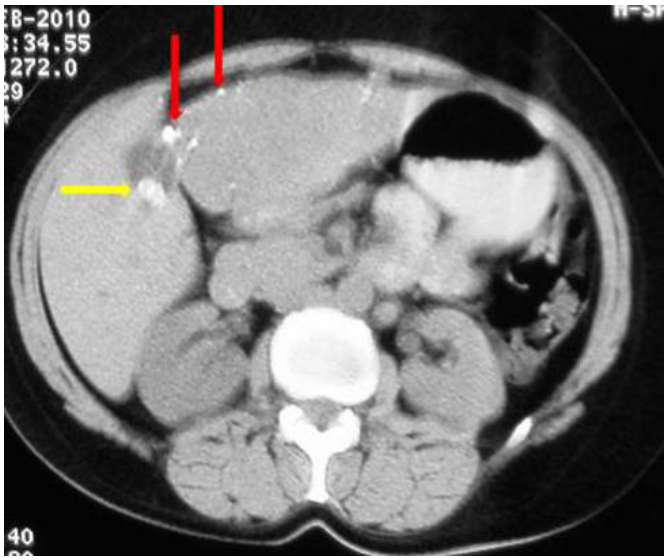

Figure 3: NCCT abdomen showing hyperdense gall bladder calculus (yellow arrow) with near complete replacement of the left lobe of liver with hypodense lesion showing small peripheral calcific nodules (red arrows)

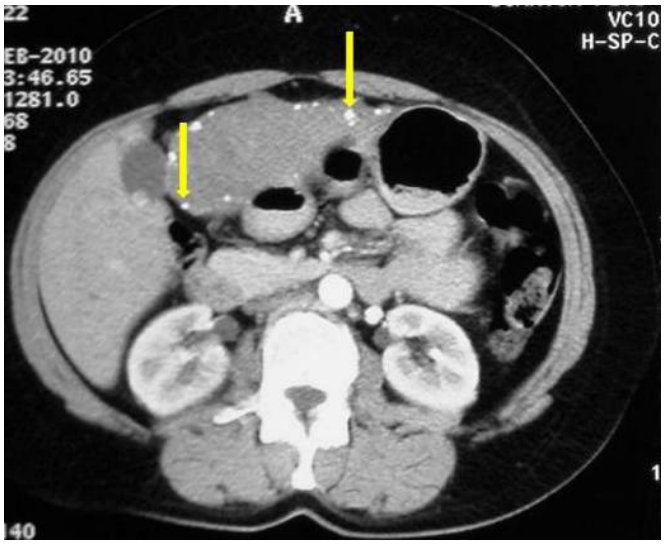

Figure 4: Few peripheral globular enhancing areas seen in the left lobe lesion in the arterial phase

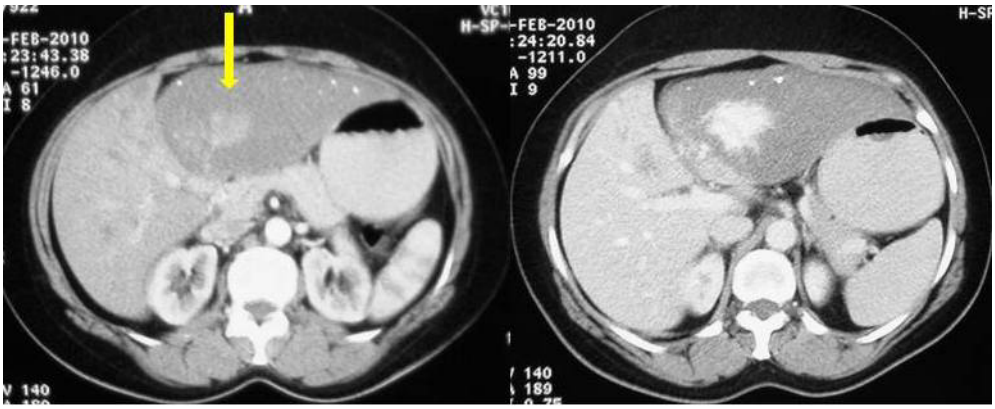

Figure 5: Central enhancing area seen in the left lobe lesion showing centrifugal filling in the venous phase

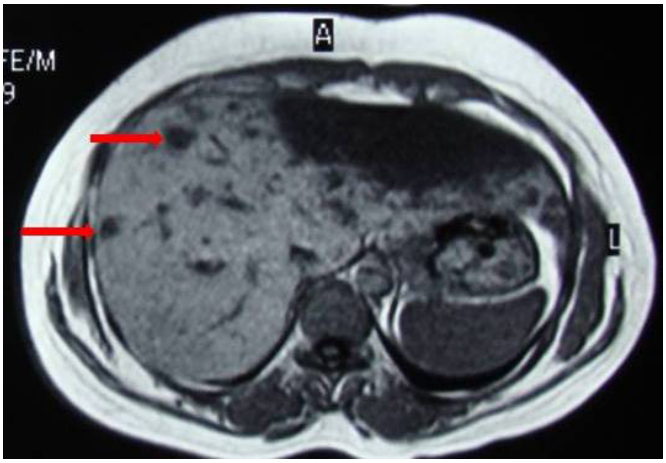

Figure 6: T1WI showing multifocal well defined hypointense lesions in both lobes of liver (red arrows)

complete filling of the lesions in delayed study (**Figure 8**). An FNAC from the lesion confirmed the diagnosis of hemangioma.

Discussion

Hepatic hemangiomas can have varied imaging appearances making it difficult at times to differentiate them from malignant masses. Imaging is fairly accurate in diagnosing typical hemangiomas with MRI being the modality of choice with a sensitivity and specificity above 90%.[1]

The varied imaging appearances are due to different structural composition of the lesions. Hemangiomas showing rapid enhancement have relatively large vascular spaces with

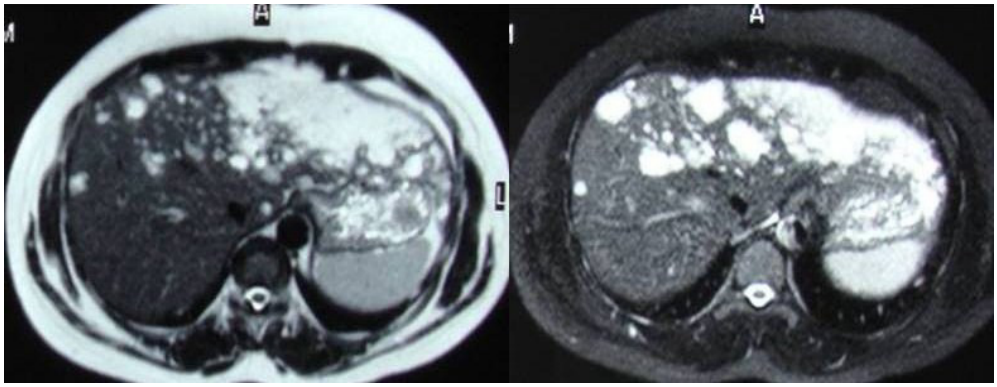

Figure 7: The lesions seen in figure 6 are seen as hyperintense in SENSE & SPAIR images

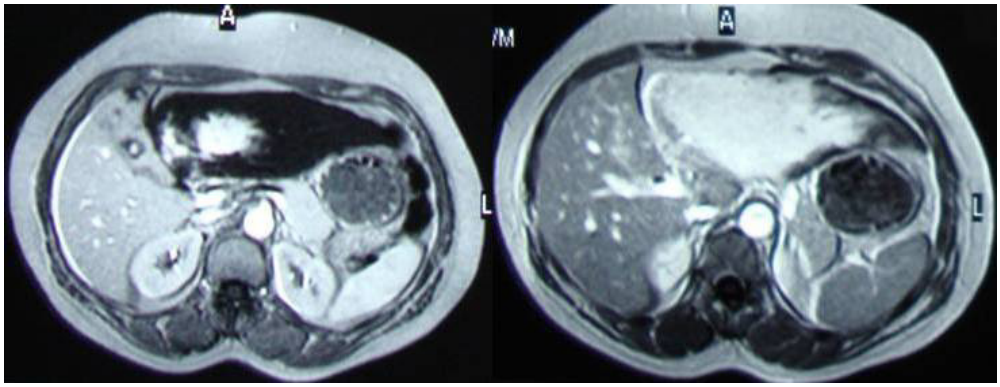

Figure 8: Dynamic MR contrast study showing the lesions to follow centrifugal enhancement pattern with complete filling of the lesions in delayed study

thin intervening septae whereas hemangiomas with slow filling pattern have small vascular spaces with a large portion of fibrous interstitial spaces.[2] The signal intensity characteristics on MRI are related to relative composition of intralesional vascular spaces, connective tissue, presence of thrombosis, calcification, hemorrhage, or fibrosis. Imaging characteristics of hemangioma have shown correlation with their size, with typical features seen in most lesions less than 3 cm in diameter.[3]

The typical hemangioma appears as an echogenic lesion on USG showing posterior acoustic enhancement. On CT scan it appears as a hypodense lesion showing peripheral globular enhancement in arterial phase with gradual complete filling in centripetal manner in delayed images. On MRI the typical hemangioma appears hypointense on T1WI and hyperintense as CSF on T2WI. The light bulb sign on T2WI has been linked to benign hepatic lesion like cysts or hemangioma. However, its absence is more specific for malignant lesion as this sign may be seen in some malignant lesions also.[4]

Atypical hepatic hemangiomas may present with an echogenic border and central hypoechoic area on USG. This appearance is noted in up to 40% of cases.[5] Hemangiomas may be large and heterogeneous when they are called giant hemangiomas. The criteria to call the lesion as giant hemangioma varies in different text. Its size should be more than 4 cm, 6 cm or 12 cm.[1] We may have rapidly filling hemangiomas. This pattern is seen more commonly in lesions smaller than 1 cm. It is seen in up to 16% of cases. These need to be differentiated from hypervascular malignancies. In a retrospective study done by Outwater et al,[6] it was seen that T2-weighted images and dynamic gadolinium-enhanced images could very accurately distinguish the two lesions with high accuracy (97-100%) as signal intensity on the heavily T2-weighted images of the hemangiomas was significantly greater than that of hypervascular malignant lesions ($p < 0.05$). Although hemangiomas may not show any enhancement but once they enhance they do not show washout as seen in malignant lesions.[7]

There has been one case report of hemangioma presenting with peripheral rim enhancement on dynamic CT scan mimicking hepatocellular carcinoma.[8] This may be attributed to the surrounding liver parenchymal fibrotic change caused by an active hepatitis C viral infection. There may be calcified hemangioma, hyalinized hemangioma, cystic or multilocular hemangioma. Only one case of multilocular hemangioma is reported.[1] Calcification may be seen in the lesion's periphery in a spotty nodular form representing phleboliths or they may be large and organized. Hyalinised hemangiomas are not as hyperintense as the typical hemangioma on T2WI. These do not show early enhancement in dynamic contrast study and can be difficult to differentiate from malignant lesions. Hemangiomas may present with a fluid-fluid level. Two such cases have been reported.[1] This is most likely due to bleed within the hemangioma forming a lower layer of stagnant blood and upper layer of serous unclotted blood. Pedunculated appearance of hemangioma is also known where the thin pedicle with the liver may not be discernible on imaging and the diagnosis is made on the basis of the typical enhancement pattern. Arterial–portal venous shunting is also seen in hemangiomas with a total of fourteen cases reported with this appearance.[1] Hemangiomas may present with capsular retraction and confuse with cholangiocarcinoma.

Lastly, hemangiomas may present with atypical inside-out pattern showing centrifugal enhancement pattern on dynamic contrast-enhanced CT and MR imaging. Two such cases have been reported in CT/MR imaging by Kim et al,[2] one by Hosokawa et al,[9] and one on CT scan by Matsushita et al.[10] Bartolotta et al reported a series of seven patients with this atypical pattern on contrast USG.[11] The lesion reported by Hosokawa et al was exophytic and showed centrifugal enhancement along with dilatation of the right posterior inferior branch of the hepatic artery on angiography. Matsushita et al performed dynamic computed tomography during hepatic arteriography and demonstrated a centrifugal enhancement pattern with subsequent peri-tumoral ring-shaped enhancement mimicking corona enhancement in a cavernous hemangioma of a 68-year-old man. Bartolotta et all conducted contrast enhanced USG in 92 cases of atypical appearing hemangiomas on USG and found 12 out of 158 lesions to show central enhancing focus in arterial phase followed by centrifugal enhancement in the portal venous and delayed phases.[11]

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Steatohepatitic variant of hepatocellular carcinoma: a case report of new histological variant

Introduction

Hepatocellular carcinoma (HCC) is the sixth most common malignancy and the third most frequent cause of cancer mortality worldwide with increasing incidence both in developed and developing countries.[1] Although chronic viral hepatitis (B and C) and alcoholic liver disease (ALD) are the commonest and well established risk factors for HCC, there is now sufficient evidence and literature to suggest that non-alcohalic fatty liver disease (NAFLD) is also an important risk factor for the development of cirrhosis and may develop HCC.[2,3]

In addition to the already described histological variants of HCC, recently a new histological subtype has been described by Salomao et al.[4] This new variant was termed as steatohepatitic variant of hepatocellular carcinoma (SH-HCC), identified in liver explants with chronic hepatitis C. The histomorphological features of this variant resembled that of non-neoplastic steatohepatitis. The common findings described in this new variant include large droplet steatosis, inflammation, ballooning of malignant hepatocytes, Mallory-Denk bodies and pericellular fibrosis.[4,5] The same authors have shown that SH-HCC are strongly associated with underlying steatohepatitis and metabolic syndrome.[5] We could identify the first case of this new histological subtype of HCC out of 14 explant/resected liver specimens we received in 2 years duration.

Case report

A 49-year- old female, a known case of hepatitis C related chronic liver disease was referred to the Hepatology out-patient department at our institute for evaluation and management. She was evaluated at our centre for chronic liver disease and investigated accordingly. Triple phase computerized tomography (CT) of abdomen showed changes of chronic liver disease with cirrhosis along with splenomegaly and multiple abdominal collaterals. On MRI there were three small 1 to 1.3 cm heterogeneous lobulated lesions seen in segment VIII, with arterial phase enhancement and washout in venous and delayed phase which were suggestive of HCC (**Figure 1**). Triple phase contrast enhanced (with Gadolinium BOPTA) multiplanar imaging of the upper abdomen also showed changes of chronic liver disease with cirrhosis and portal hypertension (splenomegaly and abdominal collaterals). Laboratory investigations showed total biliribin of 1.91 mg/dl, with a direct fraction of 0.68 mg/dl, aspartate aminotransferase level of 116 IU/L, alanine aminotransferase of 85 IU/L, lactate dehydrogenase of 75 IU/L, gamma glutamyl transferase of 47 IU/L, total protein of 7.1 g/dl with albumin level of 2.7 g/dl. She was detected to be diabetic with fasting glucose of 156 mg/dl