Clinical Image

Cystic Mass of the Hepatic Hilar Region in a 16-Year-Old Girl: Hepatic Infarction

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1. Clinical Image

A previously healthy 16-year-old girl was referred for further evaluation of intermittent epigastric pain and distention for one week. During this episode, she occasionally suffered high fever up to 38°C. Her review of systems was unremarkable, and no abdominal mass was detected on palpation. Laboratory studies including leukocyte, hepatic function and serum tumor markers indicated normal level. Her C-reactive protein and ESR were also within normal limits. A workup for underlying chronic liver disease was negative(antinuclear antibody, anti-smooth muscle antibodies, anti-mitochondrial antibodies, viral hepatitis panel, iron panel, and ceruloplasmin levels). Non-enhanced abdominal CT showed a round cystic hypodense shadow in the hepatic hilar region (**Figure1A**), which manifested as an oval hypodense mass with peripheral patchy enhancement and ill-demarcated border with the liver (**Figure1B-D**). There was no direct evidence of vascular compromise. MRI indicated high signal mass with peripheral low signal on T2-weighted image (**Figure 2**). Complete removal of the mass was achieved via laparoscope. Intraoperatively, mass was located above the gall cyst and close to the liver. A thin base connected to the liver was found and cut off. Histopathological examination showed diffuse hepatocelluar infarction with contour of hepatic trabecula and microvascular thrombosis (**Figure 3**). The diagnosis of hepatic infarction was finally made.

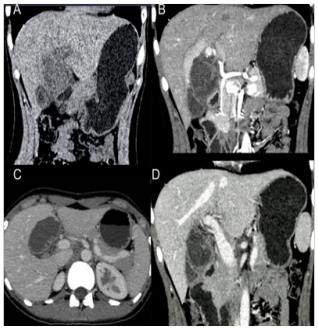

Figure1a-d: Coronary reconstructed CT image showed a round hypodense shadow in the hepatic hilum, which was difficult to define its origin (A-B). Cystic lesion with partial inner wall enhancement was further identified on delayed phase(C-D).

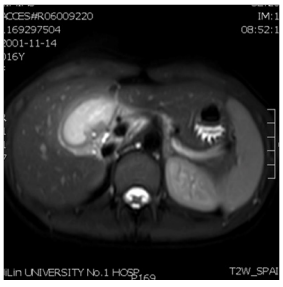

Figure 2: MRI indicated an oval cystic mass with local marginal low signal on T2-weighted image.

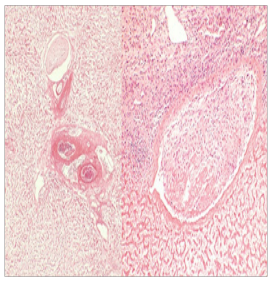

Figure 3: Histopathology showed diffuse hepatocelluar infarction with contour of hepatic trabecula and the presence of microvascular thrombosis.

The hepatic artery supplies about 35% of hepatic blood flow and 50% of the oxygen required by the liver, while the portal vein supplies the remainder. Except for the dual blood supply from the portal vein and the hepatic artery, the liver is also protected from ischemia by collateral pathways between the celiac axis and superior mesenteric artery. So, hepatic infarction is very rare condition. Hepatic infarction is usually associated with the onset of sudden upper abdominal pain, fever, elevated white blood cell count, and markedly elevated liver enzymes. However, our patient lacking chronic liver diseases only complained of abdominal pain and fever, and there was no obvious change such as infection indicators and hepatic function on admission.As known, best diagnostic clue of hepatic infarction is commonly a peripheral wedge-shaped low-attenuation areas with absent or heterogenous enhancement after intravenous administration of contrast material on imaging investigations, which become better distinguishable from the healthy surrounding parenchyma. The infarction may, however, also have oval or pseudonodular morphology and be located in the parenchymal periphery or centre. Multiple post-processing CT images are the method of choice in the diagnosis of hepatic infarction, providing useful information regarding site, morphology, and extent of the lesion. Occasionally, contrast-enhanced CT is able to suggest the possible aetiology like thrombosis and guide appropriate management. Different from commonly wedge-shaped low attenuation on contrast-enhanced CT, our young patient only demonstrated oval cystic mass with partial marginal enhancement in the hepatic hilar region, which simulated duodenal duplication, and the diagnosis of hepatic infarction was finally established by removed specimen. The cause of hepatic infarction is difficult to identified on CT imaging, but emboli was observed within the small vessels on histological study. CT appearances of hepatic infarction vary according to time of visit. Of the 16 wedge-shaped lesions seen initially in the literature about hepatic infarction caused by arterial disease, four evolved into rounded lesions, with such changes beginning as early as 48hr after presented symptoms. So, some morphological discrepancies in different reports are probably due to differences in the time of imaging investigation relative to the onset of liver infarction. Treatment of hepatic infarction depends on the extent and aetiology of the lesion, and the patient's general condition. Segmental and subsegmental infarctions may be treated conservatively whereas lobar infarctions required hepatectomy. For our patient, localized lesion resection was performed because of unknown origin and nature of disease.