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Case Study

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"FLIP- FLOP SIGN AND NUTMEG LIVER" IN BUDD-CHIARI SYNDROME

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ABSTRACT

Budd-Chiari syndrome is a clinical spectrum characterized by the manifestations that arise from hepatic venous flow obstruction at any level from the hepatic venules to inferior vena cava (IVC) and the right atrium. Evaluation and diagnosis by imaging is the corner stone in early diagnosis. Computed tomography scans show various typical findings in Budd-Chiari syndrome. We present a case of Budd- Chiari syndrome with the typical "Flip-Flop Sign" in CT scan.

KEYWORDS: Hepatic, IVC, CT scan.

INTRODUCTION

The liver has a dual blood supply, coming from the hepatic artery (25%) and portal vein (75%). These two systems have a compensatory relationship as far as flow to the liver is concerned. Vascular lesions of the hepatic and portal system are characteristically evaluated by rapid bolus-infusion multidetector CT technology. Various imaging findings in CT may suggest acute, suacute and chronic Budd-Chiari syndrome.

CASE REPORT

A 19 years old female, 4 months post full term normal delivery, presented to us with complaints of painful abdominal distention since 3 months. There was no history of fever, jaundice, vomiting, hematemesis, malena, hematochezia, breathlessness, orthopnoea.

On examination : pulse was 90 beats/min, blood pressure was 120/80 mmHg. There was no icterus, pallor, clubbing, cyanosis or lymphadenopathy. JVP was normal. Per abdomen examination revealed abdominal tenderness and features of ascites. There was tender, firm hepatomegaly 4 centimetres below right costal margin. Other system examination was normal.

USG abdomen and pelvis of patient was done which was suggestive of congested hepatomegaly, chronic stenosis of right hepatic vein (cord appearance) with ostial narrowing of middle and left hepatic vein with ascites suggestive of hepatic veno-occlusion (Budd chiari syndrome). A triple phase CECT showed characteristic "Flip-Flop sign." (peripheral enhancement of liver with central nonenhancement in arterial phase (Figure-1) and reversal of enhancement during delayed phase ie; central nodular enhancement of caudate lobe and inferior venae cava and peripheral non enhancement; (figure-2)and non enhancement of left hepatic vein suggestive of thrombotic occlusion (Figure-3). Investigations revealed hyperhomocystinemia. Patient was treated with paracentesis, diuretics, vitaminB12, folic acid and pyridoxine (tab. Homochek) and transjugular intrahepatic poatosystemic stent shunt.

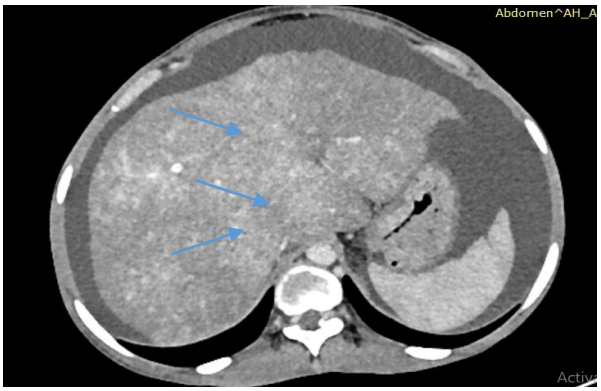

Fig. 1: Triple phase CECT abdomen in arterial phase showing.

Peripheral nodular nonenhancement of liver parenchyma with nutmeg appearance of liver with central enhancement (blue arrows).

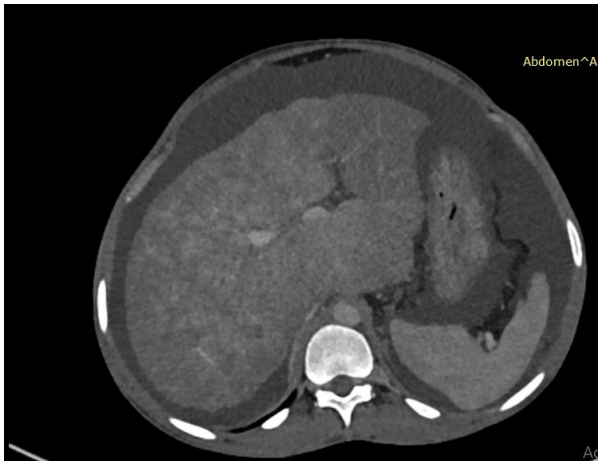

Fig. 2: CT study during delayed phase reveals delayed central nonenhancement of liver parenchyma with nodular surface with peripheral enhancement; Flip flop sign.

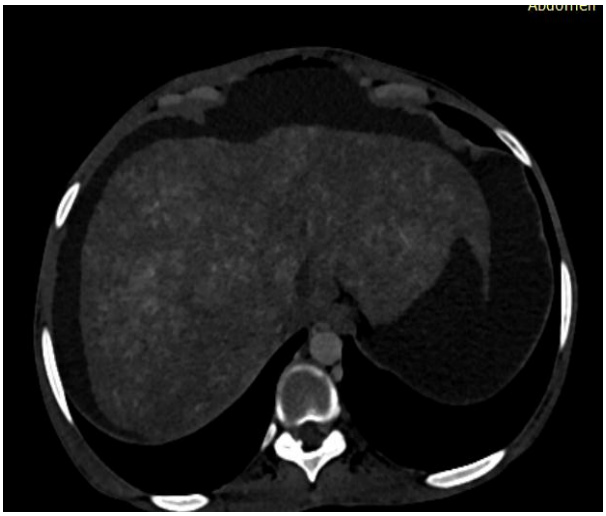

Fig. 3: Shows no enhancement of left hepatic vein suggestive of thrombotic occlusion.

DISCUSSION

Budd-Chiari syndrome is defined as lobar or segmental hepatic venous outflow obstruction at the level of the hepatic veins or inferior vena cava (IVC)..[1,2] Hepatic venous out flow obstruction leads to elevation of sinusoidal pressure which results in decreased portal venous flow, that leads to characteristic centrilobular congestion. In the acute Budd- chiari syndrome the collateral veins have not formed, so hepatocellular necrosis develops rapidly. In the subacute and chronic form, hepatic venous outflow obstruction is incomplete and various accessory hepatic veins drain the central region of the liver into the IVC. Obstruction of hepatic venules leads to shunting of blood from hepatic arteries to portal veins, which are usually at the periphery leading to reduce hepatocellular congestion but does not prevent peripheral atrophy. However, the central region of the liver usually hypertrophies because of accessory venous drainage and preservation of the portal venous supply to the caudate lobe.[1–4]

At contrast-enhanced CT of acute phase disease, early enhancement of the caudate lobe and central portion of the liver around the IVC is seen in the arterial phase, with associated non enhancement of the peripheral liver because of portal and sinusoidal stasis.[4-6] In the portal venous phase, a "flip- flop" pattern ensues, with low attenuation or enhancement of the central part of the liver because of washout, while attenuation in the peripheral part of the liver gradually increases with the accumulation of contrast material from capsular veins.

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