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An Overview of Liver cirrhosis Radiology

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Abstract

Cirrhosis is caused by repeated cycles of injury and repair, which can be due to metabolic (alcohol, steato-hepatitis, hemochromatosis, or Wilson disease), infectious (chronic hepatitis B or C), or inflammatory (primary biliary cirrhosis or primary sclerosing cholangitis) etiologies. The hallmarks of cirrhosis are fibrosis and attempted, disorganized regeneration. The micronodular form of cirrhosis is most often due to metabolic causes. While the macronodular form of cirrhosis is most often post-viral (hepatitis B or C). There are early signs of cirrhosis such as expansion of the periportal space, atrophy of the medial segment of the left hepatic lobe and enlargement of the caudate lobe which is a specific sign of cirrhosis. If caudate to right lobe size ratio reaches >0.65, it highly suggests cirrhosis. (the caudate lobe drains directly to the IVC, not via the hepatic veins, which results in compensatory caudate hypertrophy). There are secondary manifestations of cirrhosis: Portal hypertension which causes splenomegaly, portosystemic collaterals, and varices. Various imaging modalities are used in detection of cirrhosis and its various sequalae such as portal hypertensions with its various manifestations and HCC.

Keywords: Liver Cirrhosis, Hepatocellular Carcinoma, Ultra sonography

Introduction

The classical role of many imaging modalities in liver cirrhosis diagnosis is the detection of morphological changes in the liver. Cirrhotic liver shows nodular hepatic contour,

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changes in **volume distribution**, including an enlarged caudate lobe and left lobe lateral segment, atrophy of the right and left lobe medial segments, **widening of the fissures and the porta hepatis**, and **regenerative nodules**, Secondary findings related to portal hypertension may present, including varices, ascites, splenomegaly, fatty infiltration in the omentum and mesentery, edematous wall thickening of gastrointestinal tracts due to venous congestion, and intrahepatic arterioportal or arteriovenous shunts[1]

Ultrasound:

Ultrasound (US) has a major role in the diagnosis and management of chronic liver diseases by providing diagnostic and prognostic information as well as detecting complications such as HCC and portal hypertension.

Liver parenchymal texture is a characteristic that is somewhat subjective and has low sensitivity for the detection of cirrhosis. A recent retrospective study on the accuracy of conventional US in the staging of fibrosis found that routine US is not an accurate predictor for either early or significant fibrosis in chronic viral hepatitis[2] **.** However in a study of 103 patients with chronic liver disease it has been shown that liver parenchymal texture (graded as fine echotexture, mildly coarse, coarse and highly coarse) has a statistically significant correlation ($rs = 0.8853$) with the degree of fibrosis.[3] When combined with two more features (liver surface nodularity and liver edge), correlation with the degree of fibrosis increased to ($rs = 0.9524$).(4) When compared to echotexture, liver surface nodularity has better accuracy for the presence of cirrhosis.[4]

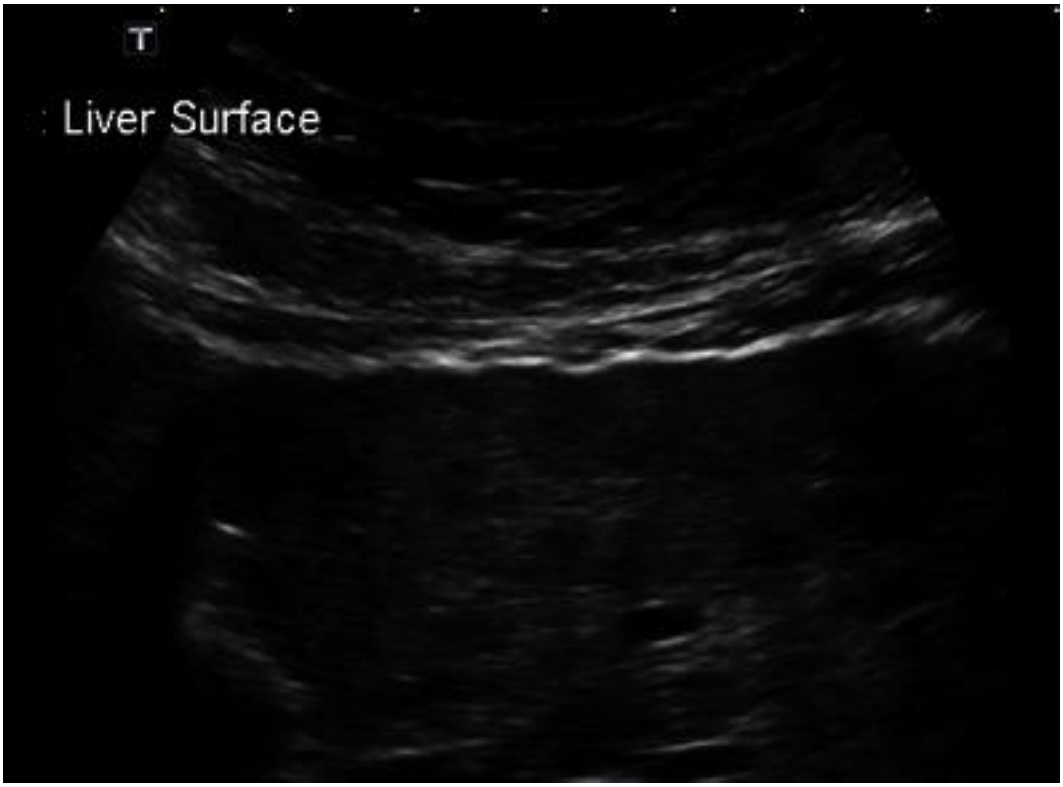

Figure (1): liver surface nodularity [5]

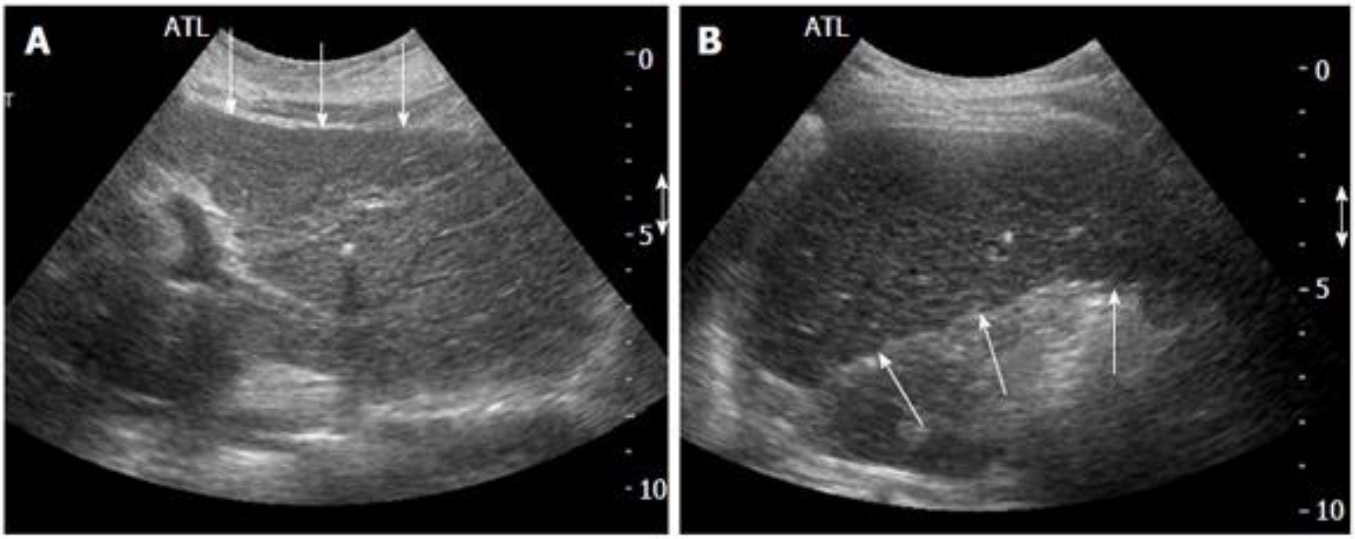

figure (2): Transaxially scan. A: Transaxial epigastric scan shows the left lobe of the liver with surface irregularity (arrows), and coarse parenchyma echotexture; B: Subcostal transaxial scan shows the inferior margin of right hepatic lobe with irregular surface (arrows) [1]

In order to provide a fluid-tissue interface, ascites needs to be present for optimal evaluation. Once ascites is present, cirrhosis is generally more advanced and less of a diagnostic challenge.

A different approach is the use of the hepatic vein lumen as an internal fluid-tissue interface when ascites is absent.

Assuming that internal nodularity in cirrhosis would cause architectural distortion, the hepatic vein morphology would also be altered. In a prospective pilot study comprising 38 patients with cirrhosis and 50 patients without liver disease, the following features were evaluated: hepatic vein straightness, uniformity of hepatic vein echogenicity and visualisation of a 1-cm segment of hepatic vein[6] Hepatic vein straightness, stratified into three categories (straight, slightly wavy and very wavy) yielded the highest sensitivity and specificity of 0.97 and 0.91 respectively using real-time compound imaging (RTCI) with a 5–2 MHz transducer.[7]

Doppler ultrasound:

The main role of Doppler ultrasound is the assessment of portal venous hypertension as a complication of cirrhosis.

Doppler ultrasound of the ligamentum teres showing hepatofugal venous signal (i.e. a patent paraumbilical vein) and hepatofugal flow in the portal vein are both specific signs and have a high positive predictive value for the presence of portal hypertension[8]

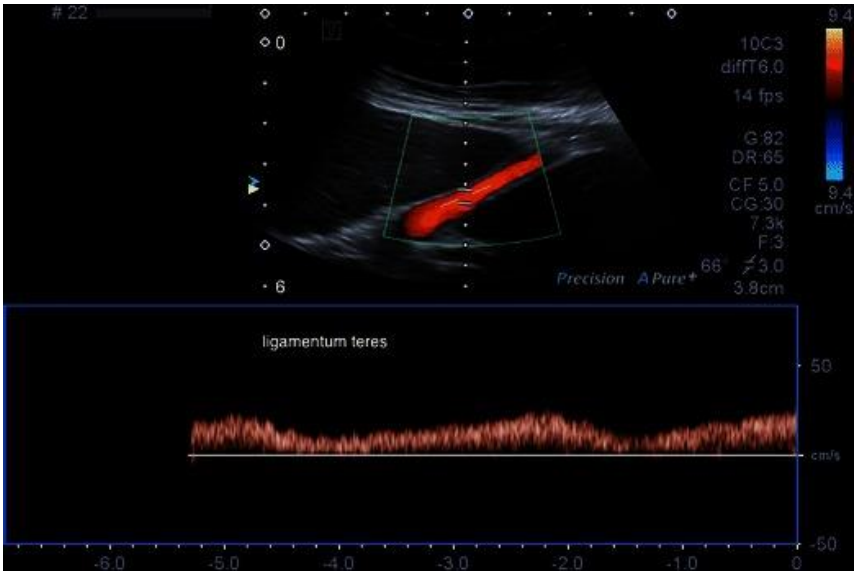

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Figure (3): Recanalisation of the paraumbilical vein as shown by venous Doppler signal in the ligamentum teres. This is sensitive and specific for the presence of portal venous hypertension[5]

CT and MRI of liver cirrhosis:

CT is the most sensitive diagnostic tool for evaluating hepatic morphological changes[9] CT readily shows alterations in hepatic morphology and extra-hepatic manifestations related to portal hypertension. With liver cirrhosis progression, the nodularity of the liver surface and generalized heterogeneity of the hepatic parenchyma are visible. The porta hepatis and interlobar fissure frequently appear wider due to shrinkage of the right lobe and the medial segment of the left lobe with concomitant enlargement of the caudate lobe and the lateral segment of the left lobe. Changes in size and volume distribution are easily visible in a CT scan.[10]

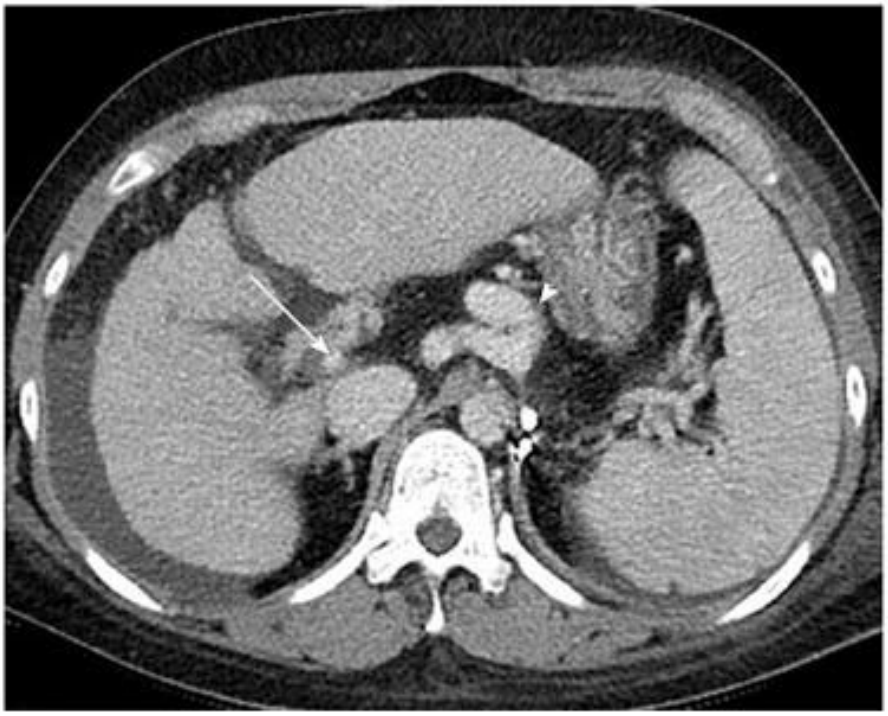

Figure (4): Image of liver cirrhosis caused by chronic hepatitis B. Contrast enhanced computed tomography portal phase image shows the liver with irregular surface and heterogeneous enhancement of parenchyma with reticular pattern suggesting confluent

fibrosis. The image shows decreased diameter of portal vein (arrow) due to large collateral vessels (arrowhead) and also shows large amount of ascites.[1]

Liver morphology:

At an early stage of cirrhosis, the liver may appear normal on cross sectional imaging. With disease progression, heterogeneity of liver parenchyma and surface nodularity are observed.

Caudate lobe hypertrophy is the most characteristic morphologic feature of liver cirrhosis. A ratio of transverse caudate lobe width to right lobe width greater than or equal to 0.65 constitutes a positive indicator for the diagnosis of cirrhosis with high level of accuracy**.**[11]

Other regional changes in hepatic morphology typically seen in advanced cirrhosis are segmental hypertrophy involving the lateral segments (II, III) of the left lobe, and segmental atrophy affecting both the posterior segments (VI, VII) of the right lobe and medial segment (IV) of the left lobe.[12] Alteration of blood flow is the likely explanation for these morphologic abnormalities. Enlargement of hilar periportal space, the notch-sign[13], an expanded gallbladder fossa[14] and generalized widening of the interlobar fissures are also considered typical findings of cirrhosis.

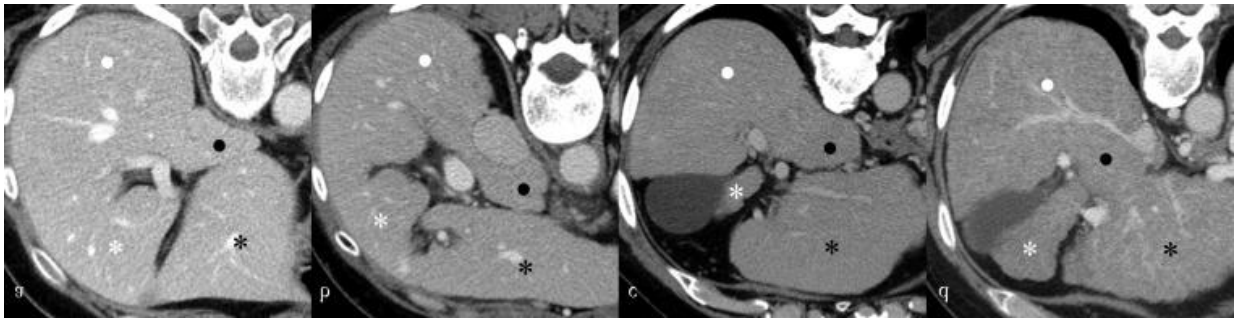

Figure (5): Comparison of portal phase of axial CT images in a 49-year-old woman with normal liver (control group) (a), in a 55-year-old man with virus-related cirrhosis (b), in a 54-year-old man with alcoholic cirrhosis (c) and in a 52-year-old woman with non-

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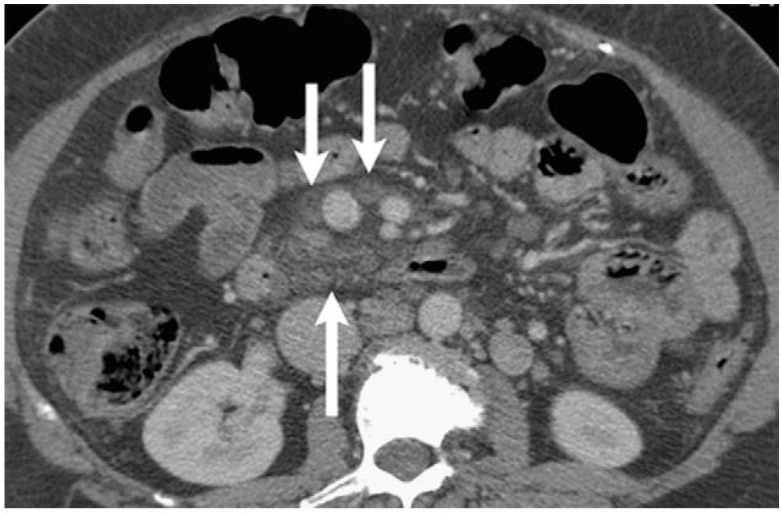
alcoholic steatohepatitis (NASH)-related cirrhosis (d) in Child–Pugh Class A. Virus-related (b), alcoholic (c) and NASH-related (d) cirrhosis show atrophy of the medial (white asterisks) and anterior segments and right lobe (white dots) and hypertrophy of the lateral segment (black asterisks) and caudate lobe (black dots) as compared with the control group by multiple comparisons. In particular, the differences in the atrophy of the medial segment and hypertrophy of the caudate lobe among the etiologies are easily understandable in axial CT images.[12]

Portal hypertension and mesenteric edema.

In chronic liver disease, progressive hepatic fibrosis leads to increased vascular resistance at the level of the hepatic sinusoids. The increased pressure gradient is defined as portal hypertension and causes complications such as ascites and the development of engorged and tortuous collateral vessels that typically develop at the lower end of the esophagus and at the gastric fundus (hypertensive gastropathy).[15]

The paraumbilical veins and the left gastric vein, both draining into the portal vein, also reopen to form portosystemic shunts. Other shunts between the portal and the systemic circulation include splenorenal collaterals, hemorrhoidal veins, abdominal wall and retroperitoneal collaterals.

Increased venous pressure is also responsible for the prominent mesenteric edema and stranding occurring in 86% of patients with cirrhosis [16]**.**It can occur in mild, moderate or severe form with pseudonodules surrounding mesenteric vessels and mimicking enlarged lymph nodes.



[17]

Figure (6): Contrast-enhanced CT scan shows Mesenteric edema in cirrhosis

Fibrosis

Fibrosis is an inherent part of hepatic cirrhosis, and is typically detected as patchy fibrosis, as a lacelike pattern, or as a confluent mass. The lacelike type of fibrosis is best described as thin or thick bands that surround regenerative nodules. This pattern is best visualized on non-enhanced CT, and is usually not well visualized on portal venous phase images[18]

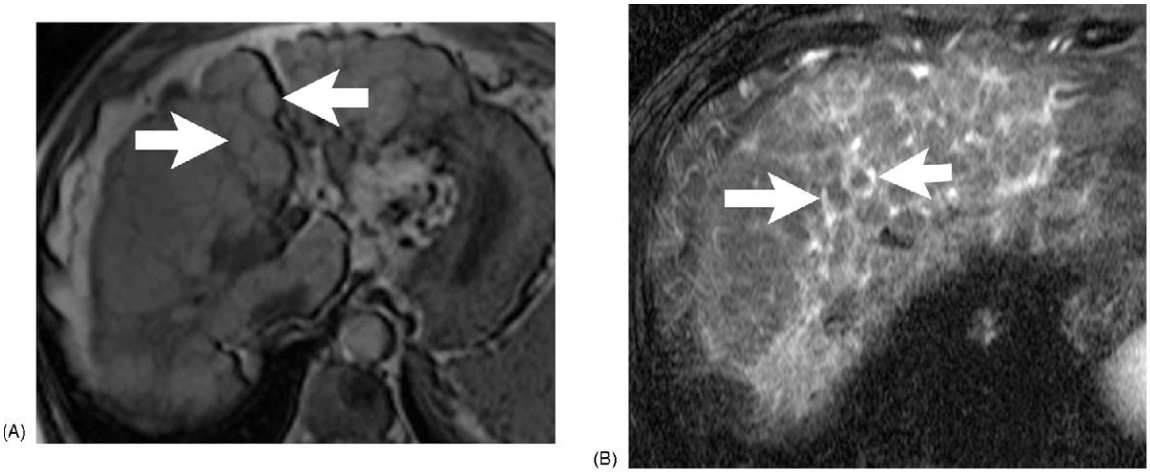


Figure (7): Cirrhosis and multiple regenerative nodules at MR imaging. Transverse T1-weighted gradient-echo MR image (A) shows multiple subcentimeter isointense nodules (arrows), surrounded by diffuse lacework of low-intensity fibrosis. On transverse T2-weighted fat-suppressed turbo spin-echo MR image (B), nodules (arrows) are still isointense and surrounded by thick hyperintense septa of lacelike fibrosis. Arterial

dominant phase (not shown) failed to show enhancement of nodules, consistent with diagnosis of regenerative nodules.[17]

Regenerative nodules

In the cirrhotic liver, regenerative nodules are macronodular
(≥9 mm), as usually seen in chronic hepatitis B, or micronodular
(3–9 mm), as seen in other causes of cirrhosis. Most regenerative nodules are difficult to detect at CT or MR because they are too small or are too similar to surrounding liver parenchyma

MR imaging demonstrates regenerative nodules with greater
sensitivity than any other imaging modality. They usually appear
isointense to hypointense on T2-weighted MR images relative to the surrounding inflammatory fibrous septa and isointense to hyperintense relative to background liver parenchyma on T1-weighted sequences [19]

Transient elastography (FibroScan):

Transient elastography (FibroScan; Echosens, Paris, France) is a fast, and non-invasive technique that measures liver stiffness, this system is equipped with a probe that consists of an ultrasound transducer mounted on the axis of a vibrator.(20) A vibration of mild amplitude and low frequency is transmitted from the vibrator to the tissue by the transducer. This vibration produces an elastic shear wave which is then propagated through the tissue. Meanwhile, pulse-echo ultrasonic acquisitions are performed to follow the propagation of the shear wave and measure its velocity, which is directly related to tissue stiffness. The harder the tissue, the faster the shear wave propagates. (21)(22)

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