Imaging of cirrhosis and portal hypertension

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Cirrhosis is a chronic response to repeated insults of the liver and is the most common cause of portal hypertension. Common causes of cirrhosis include alcoholic abuse and viral hepatitis.

Ultrasound (US) and computer-aided tomography (CT) findings of cirrhosis include fatty infiltration, nodular hepatic contour, enlargement of the caudate lobe and left lobe, atrophy of the right lobe, evidence of portal hypertension and regenerating nodules.

US and CT findings of portal hypertension include dilatation of the portal vein, portosystemic collateral circulation, ascites and splenomegaly. Doppler US provides more precise identification and characterization of vessels and flow direction than conventional US and should be used for all patients preoperatively to determine portal vein patency and direction of flow.

MRI has little primary role in the diagnosis of cirrhosis and portal hypertension. However, it is useful for differentiating regenerating nodules from hepatocellular carcinoma which is a common complication of cirrhosis. Regenerating nodules presents as low signal intensity on T2-weighted images, whereas hepatocellular carcinoma is hyperintense. Hemosiderin deposits within the regenerating nodules are believed to account for the low MR signal.

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โรคตับแข็งเป็นการตอบสนองแบบเรื้อรังต่อภาวะต่าง ๆ ที่ทำอันตรายต่อตับ สาเหตุสำคัญ
ของโรคตับแข็งคือ โรคพิษสุราเรื้อรัง และโรคไวรัสตับอักเสบ โรคแทรกซ้อนที่สำคัญของตับแข็งคือ
ภาวะความดันเลือดในตับสูง

อัลตร้าซาวนด์ และ CT มีบทบาทสำคัญ ในการวินิจฉัยโรคตับแข็ง โดยในระยะแรกจะพบ
ลักษณะของไขมันในตับ ต่อมาตับจะมีขนาดเล็กลง ขอบเขตไม่เรียบ มีก้อนตะปุ่มตะป่าบนผิวตับ
และในระยะสุดท้ายจะเกิดภาวะความดันเลือดสูงในตับ โดยจะพบลักษณะของ portal vein ที่มี
ขนาดใหญ่ มีการเชื่อมต่อของระบบเลือด portal กับระบบเลือดดำในร่างกาย การมีน้ำในช่องท้อง
และมีขนาดของม้ามโตขึ้น อัลตร้าซาวนด์ชนิด Doppler สามารถที่จะใช้ตรวจดูทิศทางการไหลของ
เลือดใน portal vein ได้เป็นอย่างดี

MRI มีบทบาทสำคัญในการช่วยแยกชนิดของก้อนภายในตับแข็งว่าเป็นเนื้อร้าย (hepato-
cellular carcinoma) หรือเนื้อดี (regenerating nodule) โดยเฉพาะการใช้สัญญาณแม่เหล็กแบบ
T2-weighted ซึ่งเนื้อร้ายจะมีสัญญาณภาพที่ขาวขึ้น ส่วนเนื้อดีจะมีสัญญาณภาพที่ดำลง เนื่องจาก
มีสาร hemosiderin สะสมอยู่ภายในเซลล์

Cirrhosis: Definition, Pathophysiology, and Etiology

Cirrhosis is a generic term used to describe chronic liver disease. It is a chronic response to repeated hepatocellular insults, characterized by cyclical episodes of impaired circulation, injury, inflammation, regeneration and fibrosis.[1]

Pathologically, cirrhosis may be classified into two large categories, micronodular and macronodular.[2] The micronodular variety, found commonly in alcoholic cirrhosis, has diffuse nodules of less than 3 mm size with thin fibrous septa. The macronodular type, found commonly as a sequelae of viral hepatitis, is characterized by nodules greater than 3 mm in size with thick fibrous septa. However, it is often difficult to place a cirrhotic liver into either category because the nodules vary in size and precise dimensions are difficult to determine. Furthermore, it is well recognized that as the disease progresses, micronodular cirrhosis may develop into the macronodular pattern. Therefore, a classification of cirrhosis based on etiology, when it can be determined, is often used.

Common etiologies of cirrhosis include alcoholic cirrhosis (60-70%), viral hepatitis (10%), biliary cirrhosis (5-10%), and hemochromatosis (5%). Rare etiologies include cardiac failure, constrictive pericarditis, hepatic vein obstruction, malnutrition, and hereditary and drug-induced cirrhosis.[3]

Portal hypertension: Definition, Pathophysiology, and Etiology

Portal hypertension is the presence of high blood pressure within the portal circulation. The pathophysiology of portal hypertension is complex and not entirely understood. The major factor is increased resistance in portal blood flow. In some cases, increased flow to the portal system, so-called hyperkinetic portal hypertension, is also a contributing factor.[4]

Etiologies of portal hypertension are divided into three major groups, prehepatic, intrahepatic and post-hepatic. Prehepatic portal hypertension is caused by obstruction of the splenic or portal veins before entering the liver. Causes include splenic or portal vein thrombosis and extrinsic compression by tumors or lymph nodes.

Intrahepatic portal hypertension, which is subdivided into presinusoidal, sinusoidal and post-sinusoidal forms, accounts for more than 90% of cases of portal hypertension.[5] Alcoholic cirrhosis and viral hepatitis are the two most common causes of sinusoidal and postsinusoidal intrahepatic portal hypertension.[5] Schistosomiasis is a leading cause of presinusoidal intrahepatic block worldwide, particularly in eastern Africa, the Middle East, and southwest Asia.[6]

Causes of post hepatic portal hypertension include cardiac or pericardial diseases, the Budd-Chiari Syndrome, and hepatic veno-

occlusive disease.[5] Whatever the cause of the portal hypertension, the pathophysiologic consequences are the same. The four most important features are ascites, the formation of the portosystemic collateral circulation, splenomegaly and occasionally hepatic encephalopathy.

The following discussion on imaging findings will emphasize cirrhosis as a prototype of portal hypertension.

Imaging Findings: Cirrhosis

In the early stage of cirrhosis, parenchymal changes may be minimal and may not be visible on any imaging modality. Fatty infiltration, a nonspecific finding, may be the initial feature of alcoholic liver disease.[7] Findings with US include increased hepatic parenchymal echogenicity, decreased beam penetration through the liver, and poor demonstration of intrahepatic vessels. On plain CT, attenuation of the liver will be lower than that of the spleen. Sometimes, the density of the liver is lower than that of the intrahepatic vessels, giving the appearance of "pseudo-enhancement" on plain CT. In the late stage of cirrhosis, the change is quite dramatic. Findings with both US and CT include nodularity of the hepatic contour, atrophy of the right lobe and hypertrophy of the left and caudate lobes of the liver (caudate/right lobes ratio, >0.65), ascites, and evidence of portal hypertension [8-10] (Fig.1).
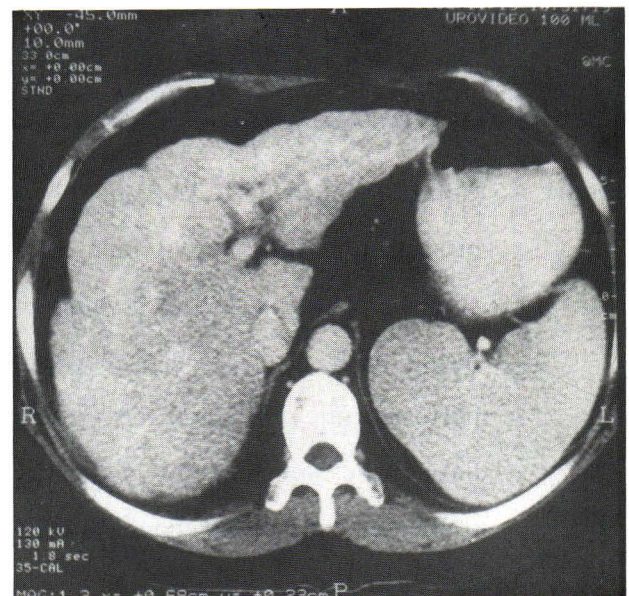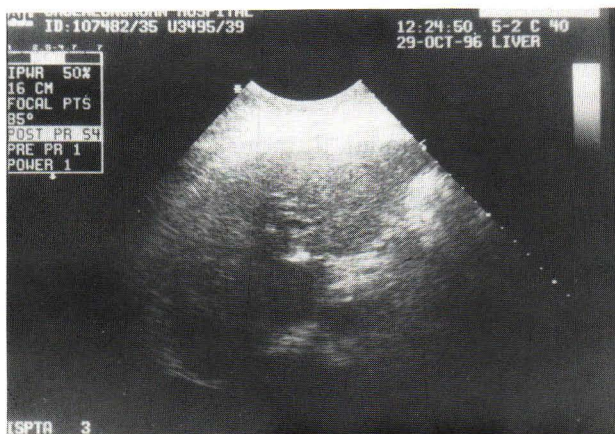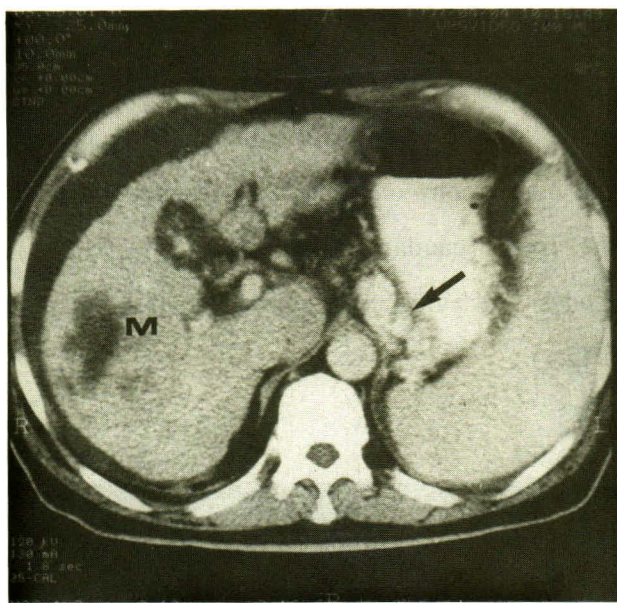

Figure 1. Characteristic imaging findings of cirrhosis.

1A) US shows nodularity of the hepatic surface, atrophy of the right lobe, and hypertrophy of the left lobe of the liver.

1B) Contrast-enhanced CT scan shows a small liver with nodularity of its contour. The spleen is enlarged, secondary to portal hypertension.
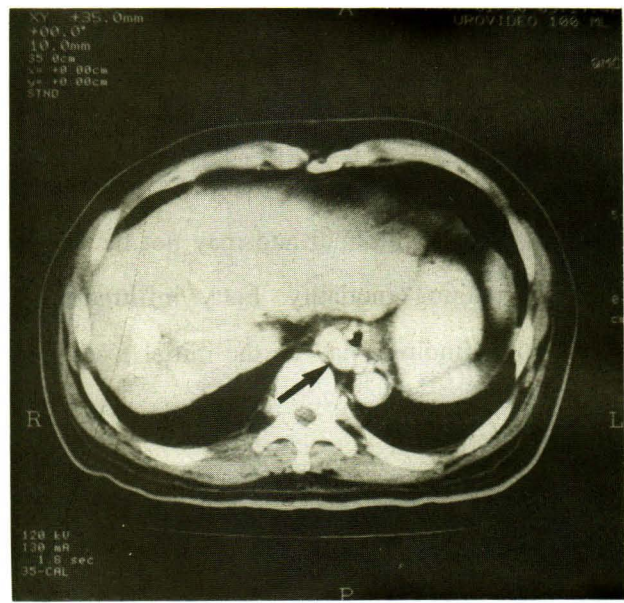
Imaging Findings: Portal hypertension

Imaging findings of portal hypertension include ascites, splenomegaly, enlarged portal vein (>13 mm), and evidence of portosystemic collateral circulation.[11-14]

The most common collateral circulation involves the coronary veins which help shunt blood from the main portal vein to gastroesophageal varices. These varices can be identified as circular and tubular, worm-like structures on both US and CT in the region of the gastroesophageal junction and the lesser curve of the stomach (Fig. 2, 3).

Figure 2. Contrast-enhanced CT scan shows enhancing tubular, worm-like structures along the lesser curvature of the stomach (arrow), which represent portosystemic collateral circulation through the coronary veins. Nodularity of the liver surface, splenomegaly and ascites indicate cirrhosis with portal hypertension. Hepatocellular carcinoma (M) is also noted within the right lobe of the liver.
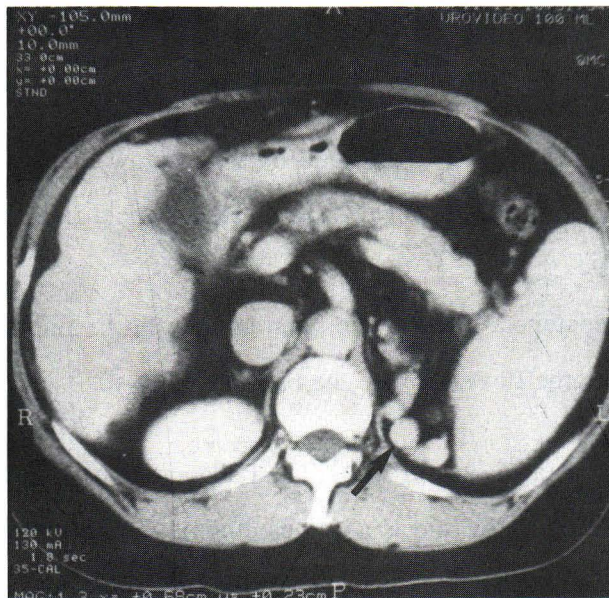

Figure 3. Contrast-enhanced CT scan shows enhancing vessels (arrow) behind the esophageal lumen. These vessels represent esophageal varices.

Another common collateral circulation is through recanalization of the paraumbilical vein within the ligamentum teres. It shunts blood from the left portal vein to the anterior abdominal wall venous system.

Uncommon forms of portosystemic collateral circulation include spontaneous splenorenal shunt, multiple retroperitoneal veins shunting blood from the superior mesenteric vein to the systemic lumbar veins, and shunts from the inferior mesenteric vein to the inferior and middle hemorrhoidal veins (Fig. 4).


Figure 4. Contrast-enhanced CT scan shows multiple retroperitoneal veins (arrow), another portosystemic collateral pathway in a patient with portal hypertension.

Imaging: Role of Duplex Doppler US

Doppler sonography should be performed in all cases of portal hypertension before surgical intervention. The direction of portal venous blood flow will determine the choice of operation. If the portal venous flow is reversed (hepatofugal flow, found in about 3-8% of cases of cirrhosis), the patient is no longer a candidate for a selective distal splenorenal shunt (Warren's shunt) and must undergo a total shunt (portocaval or mesocaval shunt) or transjugular intrahepatic portosystemic shunt (TIPS) procedures[15-17] (Fig. 5). Warren's shunt is the preferred method because it prevents the most serious complication, hepatic encephalopathy.[18, 19]
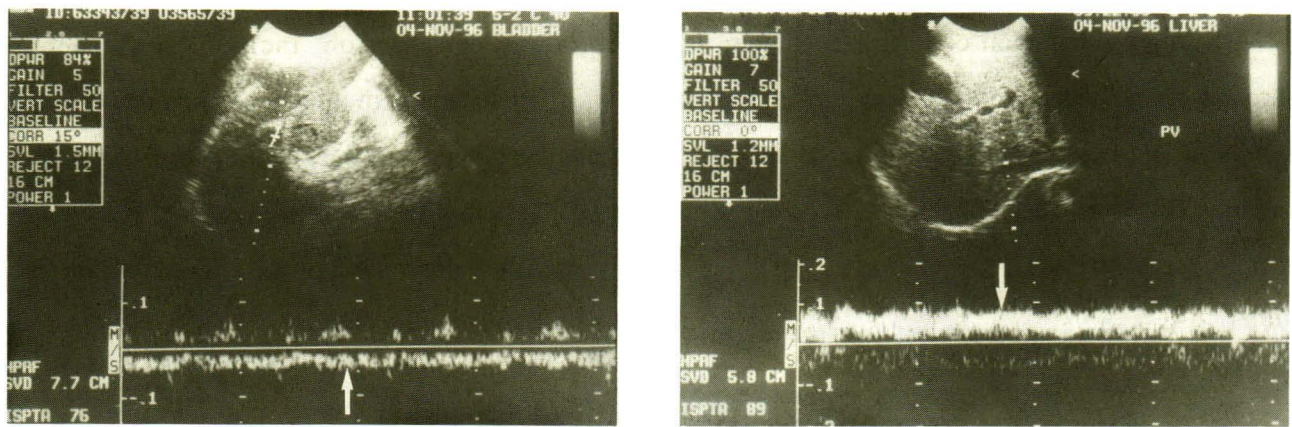
Figure 5. Doppler sonography of the main portal vein.

5A) The portal venous flow, in a cirrhotic liver, is below the baseline (arrow), suggestive of a reverse flow (hepatofugal flow). This patient is no longer a candidate for Warren's shunt.

5B) The normal hepatopetal flow of the main portal vein in a normal patient for comparison to Fig.5A. Note the flow is above the baseline (arrow).

Doppler sonography is also useful in assessing blood flow after shunt surgery and is a superb means of guiding TIPS procedures.[20]

Cirrhosis: Complications

Confusing but common complications of cirrhosis are formation of regenerating nodules, adenomatous hyperplasia, and hepatocellular carcinoma.[21] It is not clear whether these are three separate pathological entities or stages in a continuing process of the same pathology. A regenerating nodule consists of compensatory hyperplasia of hepatocytes. Adenomatous hyperplasia is the adenomatous change of such a nodule. It is usually benign but is also believed to be a premalignant lesion. Hepatocellular carcinoma is a malignant tumor of hepatocytes.

Differentiation of regenerating nodules and adenomatous hyperplasia from hepatocellular carcinoma is important because of the difference in clinical treatment. Regenerating nodules and adenomatous hyperplasia are benign conditions that require no intervention. Follow-up and observation are recommended for these lesions. In contrast, hepatocellular carcinoma requires more aggressive treatment, either surgical resection or chemoembolization.[21]

Differentiation of these lesions by any imaging modality can be very difficult. Atypical adenomatous hyperplasia can manifest, both radiologically and pathologically, similar to well-differentiated hepatocellular carcinoma. However, in certain cases these can be distinguished by using MRI.[22-24] On T2-weighted or gradient echo images, benign lesions (regenerating nodule and adenomatous hyperplasia) will show

low signal intensity secondary to hemosiderin deposits in these lesions (Fig. 6). In contrast, hepatocellular carcinoma will have high signal intensity due to high water content within the tumor cells (Fig. 7). Although this difference in MRI signal is helpful, certain type of hepatocellular carcinoma, particularly one with poor glandular

formation, can manifest low signal intensity on T2-weighted which will cause confusion with the benign nodules. In this particular case, intravenous administration of Gadolinium is suggested, and hepatocellular carcinoma will usually show some degree of enhancement.[21]
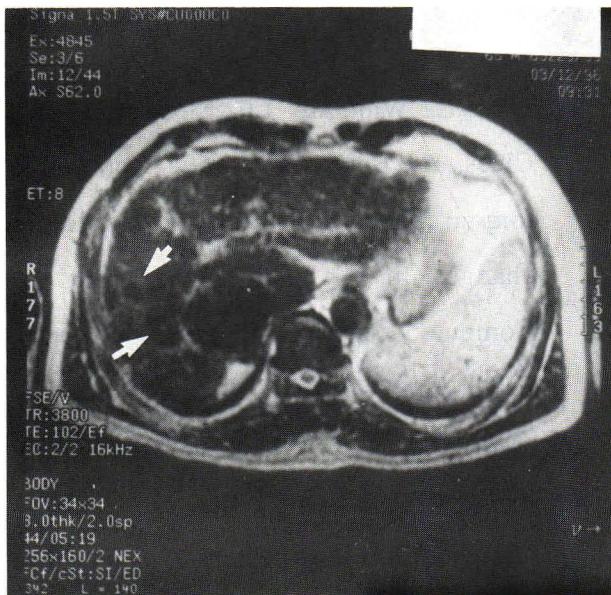
Figure 6. Fast spin echo, T2-weighted MRI (TR 3800, TE 102 Ef) of the cirrhotic liver shows multiple low signal intensity regenerating nodules (arrows). Hemosiderin deposits within these nodules account for low MR signal.
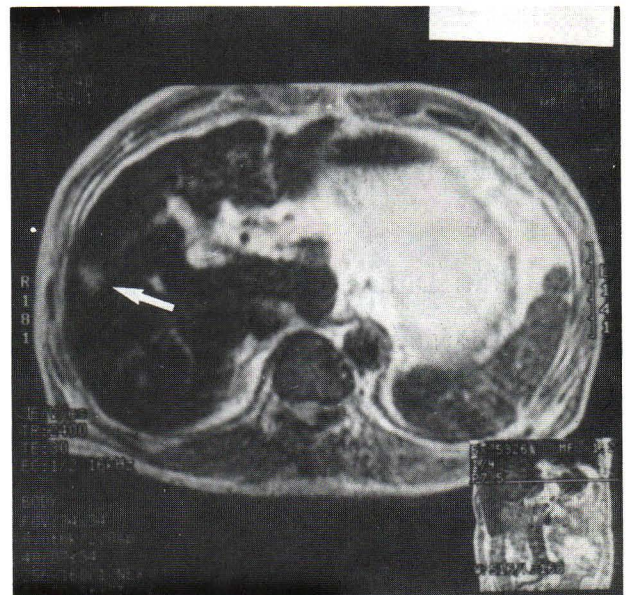
Figure 7. T2-weighted MRI (TR 2400, TE30) of the cirrhotic liver shows a high signal intensity nodule (arrow), which is proved to be a hepatocellular carcinoma.

Helical CT may also play a useful role. Since hepatocellular carcinomas are supplied mainly by hepatic arteries, lesions will enhance early, during the arterial phase of the scan.

Regenerating nodules and adenomatous hyperplasia are supplied by the portal circulation and will enhance later, during the portal venous phase. [25, 26]

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