Cirrhosis is among the leading causes of death in the western world. Cirrhosis and its associated complications have characteristic appearances on CT and MRI that are briefly reviewed. A variety of other disease entities can mimic cirrhosis. These are discussed and differentiating features emphasized.

**Cirrhosis**

Cirrhosis is most commonly caused by chronic hepatitis infection or alcohol abuse, although a number of other diseases causing hepatic injury can lead to cirrhosis. It is pathologically defined by three main characteristics: fibrosis, nodular transformation, and distortion of hepatic architecture. Subtle morphologic changes of the liver may be among the earliest detectable with imaging including enlargement of the hilar periporal space, enlargement of the major interlobar fissure, and expansion of pericholecystic space or gallbladder fossa. Typically, the anterior segment of the right lobe and medial segment of the left lobe atrophy, whereas the

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Fig. 1.—33-year-old man with viral cirrhosis.
A, Axial breath-hold gradient-echo T1-weighted MR image shows diffuse nodules with distinct larger nodules that are hyperintense to background parenchyma (arrows). Patient also had evidence of portal hypertension: splenomegaly and esophageal varices (not shown).
B, Axial breath-hold T2-weighted turbo STIR MR image shows larger nodules (arrows) are hypointense. Linear areas of fibrosis (arrowheads) are present.
(Fig. 1 continues on next page)
caudate lobe and left lateral segment hypertrophy [1].

The nodular changes in cirrhosis yield characteristic radiologic findings (Fig. 1). The nodularity is best seen affecting the liver margin, especially on the left lateral segment. Micronodular cirrhosis, common in alcoholic liver disease, gives rise to a fine cobblestone appearance resulting from nodules typically smaller than 3 mm. A grossly nodular liver margin with 3- to 15-mm regenerative nodules is characteristic of macronodular cirrhosis, more commonly associated with viral hepatitis.

Other changes in cirrhosis include diffuse heterogeneity of the organ on CT and T1- and T2-weighted MRI. Fibrosis is the predominant cause for hepatic heterogeneity and appears high in signal intensity on T2-weighted MRI [1] (Fig. 1). Also, sequelae of portal hypertension commonly are found and include esophageal varices, ascites, splenomegaly, hepatofugal portal venous flow, enlargement and tortuosity of the hepatic artery, and portosystemic vascular shunts. The most significant complication of cirrhosis is hepatocellular carcinoma (Fig. 2). Distinction of benign regenerative from premalignant dysplastic nodules can be challenging on both CT and MRI when primarily relying on arterial phase contrast enhancement for diagnosis. Both regenerative and dysplastic nodules may show homogeneous hyperenhancement and mimic hepatocellular carcinoma. On MRI, regenerative nodules appear hypointense on T2-weighted spin-echo images and isointense on T1-weighted images, although less frequently they can be hypo- or hyperintense on T1-weighted images. Most dysplastic nodules are not visualized on CT or MRI. They may appear hyperintense on T1-weighted imaging, but this does not distinguish them from regenerative nodules.

**Pseudocirrhosis**

In patients with cancer metastases to the liver, treatment with chemotherapy can result in areas of retracted tumor tissue and scarring.

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**Fig. 1.** (continued)—33-year-old man with viral cirrhosis. C, Axial breath-hold contrast-enhanced fat-suppressed 3D MR image obtained in portal venous phase shows nodules are now slightly hyperintense to background parenchyma (arrows). Arterial phase MR images (not shown) failed to show enhancement of nodules, consistent with diagnosis of either large regenerative or dysplastic nodules.

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**Fig. 2.**—53-year-old woman with cirrhosis and hepatocellular carcinoma. A, Axial breath-hold T2-weighted turbo STIR MR image shows enlargement of caudate (arrows). Hepatocellular carcinoma is visible as mildly hyperintense mass in right lobe (arrowhead). B, Axial breath-hold contrast-enhanced fat-suppressed 3D MR image obtained in hepatic arterial dominant phase shows enhancement of hepatocellular carcinoma (arrowhead).
Fig. 3.—74-year-old woman with metastatic B-cell lymphoma.


B, Axial portal venous phase CT scan obtained 5 months after chemotherapy with Cytotoxan (cyclophosphamide, Bristol-Myers Squibb), Adriamycin (doxorubicin hydrochloride, Pharmacia), and vincristine shows near-complete resolution of previously seen masses but new appearance of volume loss and nodularity of contour, mimicking cirrhosis.

C, Axial breath-hold contrast-enhanced fat-suppressed 3D MR image obtained in portal venous phase shows lobulated contour and areas of linear enhancing fibrosis (arrows), mimicking cirrhosis.

Fig. 4.—51-year-old woman with metastatic breast carcinoma.

A, Axial breath-hold gradient-echo T1-weighted MR image shows innumerable hepatic metastases.

B, Axial breath-hold T2-weighted turbo STIR MR image shows innumerable hepatic metastases.

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Between areas of scarring, the liver parenchyma is regenerative. This entity is referred to as pseudocirrhosis because it resembles macronodular cirrhosis. Common imaging findings include a lobular margin, volume loss caudate hypertrophy, and portal hypertension and can be observed within a few weeks or months after therapy [2, 3] (Figs. 3 and 4). Unlike cirrhosis, at pathology patients do not have bridging portal fibrosis, but can manifest nodular regenerative hyperplasia. Frequently, patients have a residual tumor that is difficult to detect given the morphologic abnormalities. The findings of pseudocirrhosis typically have been described in chemotherapy-treated patients with metastatic disease in the liver. However, these changes also can occur without coexistent liver disease, secondary to the hepatotoxic effects of chemotherapy [3].

**Metastatic Disease**

Typically, hepatic metastases appear on MRI as focal lesions that are hypointense on T1-weighted images and hyperintense on T2-weighted images. Contrast-enhanced MR

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**Fig. 4. (continued)—** 51-year-old woman with metastatic breast carcinoma.
C, Axial breath-hold gradient-echo T1-weighted MR image obtained 7 months after chemotherapy shows marked regression of masses with morphologic changes including lobular contour (arrows) and areas of capsular retraction, mimicking cirrhosis.
D, Axial breath-hold T2-weighted turbo STIR MR image shows extensive fibrosis (arrow) and ascites, mimicking cirrhosis.

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**Fig. 5.—** 70-year-old woman with metastatic breast carcinoma.
A, Axial breath-hold T2-weighted turbo STIR MR image shows lobular contour (arrows) and innumerable hyperintense metastases (arrowheads). Morphology of liver resembles that seen in cirrhosis. Note marked decreased signal intensity of spleen.
B, Axial breath-hold contrast-enhanced fat-suppressed 3D MR image obtained in hepatic arterial dominant phase shows innumerable enhancing liver metastases (arrowheads) and bone metastasis (arrow).
Fig. 6.—59-year-old woman with subacute hepatic necrosis of unknown cause.
A, Axial breath-hold gradient-echo T1-weighted MR image shows lobular contour (small arrows) and fibrosis (large arrow), mimicking cirrhosis.
B, Axial breath-hold T2-weighted turbo STIR MR image shows fibrosis is hyperintense with respect to background hepatic parenchyma (arrow).

Fig. 7.—37-year-old man with fulminant hepatic necrosis secondary to drug toxicity.
A, Axial breath-hold T2-weighted turbo STIR MR image shows lobulated contour (arrows) and hyperintense nodule (arrowhead) that could be confused with hepatocellular carcinoma in setting of cirrhosis. Marked ascites is present.
B, Axial breath-hold contrast-enhanced fat-suppressed 3D MR image obtained in hepatic arterial dominant phase shows relative increased enhancement of posterior right lobe that could be confused with hepatocellular carcinoma. However, note healthy arteries (arrows) traversing region.
C, Axial breath-hold contrast-enhanced fat-suppressed 3D MR image obtained in portal venous phase shows decreased enhancement of areas of presumed necrosis (arrows). No corresponding areas of focal fat or sparing were seen on opposed phase MR (not shown).
images may show peripheral rim enhancement with the “peripheral washout” sign in the delayed enhancement phases, where the peripheral rim is hypointense relative to the center of the lesion [4]. In the setting of diffusely infiltrative metastatic disease with an associated desmoplastic reaction, the liver can appear cirrhotic. Without focal lesions, imaging studies may fail to detect metastases [4]. Diffuse metastatic involvement of the liver giving rise to a cirrhotic appearance has been reported most commonly with breast cancer, although melanoma also has been reported. Findings include nodular margins, decreased volume, and enlargement of the caudate lobe [5] (Fig. 5). In addition, patients may present with signs of portal hypertension, including splenomegaly and varices. Pathologic evaluation of the liver does not show cirrhosis and instead shows dense areas of fibrosis and diffusely abnormal liver architecture [4]. Unlike patients with pseudocirrhosis, the parenchyma is diffusely replaced with viable tumor, with little healthy liver tissue [5].

**Hepatic Necrosis and Regeneration After Fulminant Hepatitis**

Fulminant hepatitis is characterized clinically by acute severe impairment of liver function resulting in hepatic coma within 8 weeks. It usually is associated with large areas of hepatic necrosis along with inflammation and hemorrhage [6]. If patients survive, necrotic areas shrink because of replacement by scarring and fibrosis, and regenerating nodules appear within weeks (Fig. 6). These nodules distort the liver margin, yielding an appearance of macronodular cirrhosis. Areas of regenerating liver appear as hypervascular masses on contrast-enhanced imaging, mimicking hepatocellular carcinoma. The combination of nodularity, fibrosis, and segmental volume changes may make distinguishing hepatic necrosis from cirrhosis difficult; however, one clue may be that in hepatic necrosis, vessels may traverse necrotic lesions without displacement [6] (Fig. 7). Intraoperatively, differentiation of hepatic necrosis with regeneration from true cirrhosis is readily made by palpation; the former results in a soft, pliable liver whereas the cirrhotic liver is firm.

**Nodular Regenerative Hyperplasia or Hepatoportal Sclerosis**

Noncirrhotic intrahepatic portal hypertension is a nonspecific term that encompasses a spectrum of disease processes including nodular regenerative hyperplasia, periportal fibrosis, and hepatoporal sclerosis [7]. Nodular regenerative hyperplasia is described histopathologically as regenerative nodules with little or no hepatic fibrosis and largely healthy hepatic architecture (Fig. 8). Without obvious clinical manifestations of portal hypertension, the diagnosis usually is detected on liver biopsy specimens. Periportal fibrosis refers to fibrous enlargement of the portal tracts with or without extending fibrous septa. Hepatoportal sclerosis describes
a pathologic diagnosis of intimal fibrous thick-
ening of the portal vein. Each of these pro-
cesses can occur in tandem and can imitate cirrhosis on CT and MRI [7, 8]. Imaging may show heterogeneous hepatic parenchyma, nod-
ular transformation, fibrosis, and sequelae of portal hypertension (Fig. 8). The nodules in nodular regenerative hyperplasia may be hypo-,
iso-, or hypointense on T1- and T2-weighted images, respectively, and may show a peripheral ring of enhancement, or a halo sign, on T2-weighted imaging, imitating metastatic tu-
mor or regenerative nodules [8].

Conclusion

MRI and CT of the liver are widely used tools for the evaluation of cirrhosis and its related complications. Among disease enti-
ties that can mimic cirrhosis are chemother-
apy-induced cirrhosis (with and without the presence of metastases), diffuse hepatic meta-
tastases, massive hepatic necrosis with regeneration, and hepatoportal sclerosis or nodular regenerative hyperplasia. These disease processes should be considered in the differential diagnosis of a cirrhotic-appear-
ing liver on CT and MRI, particularly in the absence of a cause for cirrhosis.

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