Hypervascular Liver Lesions on MRI

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the superb liver-to-lesion contrast of MRI and its ability to display the same lesion enhancement patterns as CT combined with its lack of ionizing radiation have led to this modality being widely accepted for assessing the broad spectrum of hepatic abnormalities. Moreover, hepatocyte-specific contrast agents are now available.

The differential diagnosis of hypervascular hepatic lesions depends on the status of the remainder of the organ. If the liver is normal, the most common causes of hypervascular liver lesions are hemangioma, focal nodular hyperplasia (FNH), adenoma, and hypervascular metastasis. In the presence of chronic liver disease, the likely causes include vascular shunts (transient hepatic enhancement difference [THED]), regenerative nodules, dysplastic nodules, and hepatocellular carcinoma (HCC) (Table 1).

**TABLE 1: Hypervascular Liver Lesions on MRI**

<table>
<thead>
<tr>
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<th>MRI Techniques</th>
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<tbody>
<tr>
<td>Normal background liver</td>
<td>1. Coronal ultrafast spin-echo sequence (single breath-hold), which serves as a localizer and provides an overview of the anatomy. T2-weighted images help characterize lesions (e.g., cyst, hemangioma, FNH, HCC).</td>
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<tr>
<td>Hemangioma</td>
<td>2. Axial fast spin-echo (T2-weighted) images through the liver. Fat-saturated (frequency selective) images increase the conspicuity of liver lesions.</td>
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<td>Focal nodular hyperplasia</td>
<td>3. Axial 2D dual spoiled gradient-recalled echo sequence (SPGR) (both out-of-phase and in-phase imaging during a single breath-hold). This provides T1 information and helps detect focal or diffuse fatty infiltration (loss of signal on out-of-phase sequence) (Fig. 1). This sequence also can assess for presence of fat within a liver lesion (e.g., adenoma, HCC, metastases from a clear cell cancer) and detect iron deposition in hemochromatosis or hemosiderosis (loss of signal on in-phase or longer echo sequence) (Fig. 2).</td>
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<td>Adenoma</td>
<td>4. Volume interpolated gradient-echo with fat saturation. This sequence is valuable for the detection and characterization of lesions on the basis of their enhancement patterns. In addition to an unenhanced image, repeat images are acquired at multiple phases after the injection of gadolinium—early arterial and arterial (time determined by test bolus), portal venous (40–60 seconds), and delayed (varies from 90 seconds to several minutes). Additional delayed imaging is performed if gadoxetate disodium (Eovist, Bayer HealthCare) or gadobenate dimeglumine is administered. These agents are used for further characterization of hypervascular liver lesions in general and in particular to confirm that the lesion represents focal nodular hyperplasia (discussed later). The unenhanced images can be subtracted from...</td>
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those obtained after contrast administration to better assess enhancement characteristics.
Limitations to this technique are that subtraction can only be performed if there has been
no recalibration or motion during the acquisition of different phases (i.e., the images are
well registered). In addition, there is still no objective assessment of what constitutes sig-
nificant enhancement on a subtracted image, so the evaluation remains qualitative.

Contrast Agents

Gadolinium chelates without specific distribution in biologic tissues and primarily con-
fined to extracellular spaces have been commercially available since 1986. Paramagnetic
gadolinium shortens the tissue-specific relaxation times, leading to an increase in hepatic
tissue signal intensities, particularly on T1-weighted images. Rapid redistribution of gado-
linium chelates from intravascular to extracellular spaces requires IV injection of this con-
trast agent in a small-volume bolus (up to 2 mL/s) with an individually calculated dose of
0.1–0.2 mmol/kg of body weight.

Hepatocyte-targeted contrast agents are characterized by active transport of the chelates
into the hepatocytes, where they are further metabolized and partly eliminated through the
biliary system. Thus, they permit assessment of both the liver parenchyma and the biliary
system on T1-weighted images. The most commonly used hepatocyte-selective contrast ma-
terials are gadolinium-based agents such as gadoxetate disodium and gadobenate dimegl-
mine. Gadobenate dimeglumine has 5% excretion via the biliary tract, and delayed imaging
is performed in 1 hour. Gadoxetate disodium has 50% excretion via the biliary tract, and de-
layed imaging is performed after 20 minutes. In addition to allowing imaging during the arte-
rial phase, the redistribution or portal venous phase, and the equilibrium or hepatic venous
phase (a feature comparable with those of the other gadolinium chelates), these newer agents
also contribute a hepatocyte phase to the protocol for contrast-enhanced liver imaging.
Hemangioma

A hemangioma is a well-circumscribed mass of blood-filled spaces lined by endothelium on a thin fibrous stroma. Hemangiomas occur more frequently in women, are generally asymptomatic, and are discovered incidentally. MRI is the most sensitive and specific diagnostic study for hemangiomas, with one series reporting specificity of 100% in differentiating hemangiomas from metastases. On T2-weighted images, they are markedly hyperintense and have cystlike signal intensity (Figs. 3A, 3B, 4A, 4B, 5A, and 5B). In addition, hemangiomas retain their high signal intensity on heavily T2-weighted images with TE greater than 112 ms, whereas malignant neoplasms lose their high signal on these sequences. On T1-weighted images, hemangiomas are hypointense relative to the liver (Fig. 3C, 4C, and 5C).

The three distinct enhancement patterns of hemangiomas after contrast injection generally follow those of the blood pool. The first pattern, which is characterized by immediate uniform enhancement, is typical of small capillary hemangiomas (< 1.5 cm) (Figs. 3D–3F). In the second and most common pattern (77% of cases), the hemangioma generally appears as a well-circumscribed hepatic mass with peripheral, nodular, and interrupted enhancement that can be greater than or equal to that of the blood pool and progresses centripetally to uniform enhancement (Figs. 4D–4G). In the third pattern, there is also peripheral nodular enhancement with centripetal progression but also persistent hypointensity of the central portion of the lesion (giant hemangiomas > 5 cm) (Figs. 5D–5G).
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Fig. 4—Hemangioma type 2.
A and B, Axial non-fat-suppressed (A) and fat-suppressed (B) T2-weighted images show lesion in segment VIII of liver (circle), which shows high signal intensity compared with background liver (fat-suppression increases conspicuity of lesion).
C–G, Unenhanced (C) and dynamic contrast-enhanced (D–G) volume interpolated gradient-echo with fat saturation images after administration of gadoxetate disodium (Eovist, Bayer HealthCare) show lesion (circle) as well-circumscribed hepatic mass with peripheral, nodular, and interrupted enhancement that progresses centripetally to uniform complete enhancement on arterial (D), portal venous (E), and delayed dynamic (F) phase images. On 20-minute delayed dynamic image, lesion is hypointense to background liver (G) (i.e., does not show uptake of Eovist).
Hemangiomas type 2 and 3.

A and B, Axial non-fat-suppressed (A) and fat-suppressed (B) T2-weighted images show two lesions in segments V (circle) and VI (arrows) of liver, with high signal intensity compared with background liver. Fat-suppression increases conspicuity of lesions in image B.

C–G, Unenhanced (C) and dynamic contrast-enhanced (D–F) volume interpolated gradient-echo with fat saturation images after administration of gadoxetate disodium (Eovist, Bayer HealthCare) show that smaller lesion (circle) appears as well-circumscribed hepatic mass with peripheral, nodular, and interrupted enhancement that progresses centripetally to uniform complete enhancement. This is consistent with type 2 hemangioma. Larger lesion in segment V (arrows) shows peripheral nodular enhancement with centripetal progression but also persistent hypointensity of central portion of lesion, appearance consistent with type 3 hemangioma. On 20-minute delayed image (G), both lesions are hypointense to background liver. Gallbladder containing Eovist is seen as hyperintense linear area between two liver hemangiomas.

Fig. 5—Hemangiomas type 2 and 3.
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**Focal Nodular Hyperplasia**

FNH is a benign tumor that is thought to represent a hyperplastic response of the hepatic parenchyma to a preexisting arterial malformation. It is most common in women of reproductive age but can occur in men and children. After hemangiomas, most incidental hypervascular liver lesions in noncirrhotic livers represent FNH and not adenoma. Histologically, FNH contains hyperplastic hepatocytes and small bile ductules surrounding a central fibrous scar. The Kupffer cells are present in relatively high numbers in FNH compared with hepatic adenoma and HCC. A helpful morphologic feature for distinguishing FNH from adenoma is the margin of the lesion, which is typically ill-defined or lobulated in FNH whereas the margin is usually smooth and well circumscribed in adenomas.

On MRI, FNH is generally isointense relative to the liver on T1-weighted images (Fig. 6C) and isointense to slightly hyperintense on T2-weighted images (Figs. 6A and 6B). The classic central scar is T1 hypointense and T2 hyperintense because of the presence of blood vessels, bile ductules, and edema within myxomatous tissue and typically shows delayed enhancement. On dynamic contrast-enhanced images (Figs. 6D–6G), FNH shows markedly homogeneous arterial phase enhancement that becomes isointense during the portal venous phase. MRI with a hepatocyte-specific contrast agent (Eovist or gadobenate dimeglumine) helps confirm the hepatocellular origin of the mass. On 20-minute delayed contrast-enhanced images, FNH appears isointense to the background liver because of the presence of bile ducts (Fig. 6G).

**Hepatic Adenoma**

Hepatic adenoma is a very rare benign neoplasm that is most commonly seen in women taking oral contraceptives. The prevalence of this tumor increases with the duration of oral contraceptive use and the size of the estrogen dose. Although adenomas are typically solitary, they are multiple in about 20% of cases, especially in patients with glycogen storage disease or those who have used anabolic steroids.

Pathologically, a hepatic adenoma is composed of benign hepatocytes that are arranged in large plates or cords without acinar architecture. The hepatocytes are separated by dilated sinusoids, which may cause the hypervascularity in this tumor. The absence of bile ducts is an important histologic means of distinguishing an adenoma from FNH. An accurate diagnosis of hepatic adenoma is essential for clinical management because of the tendency of the lesion to spontaneously rupture or hemorrhage as well as the potential for malignant transformation.

On MRI, adenomas have variable signal intensity but can show hyperintense foci on unenhanced T1-weighted images secondary to hemorrhage or intracellular lipid. The visualization of fat within the lesion on fat-suppressed or opposed-phase T1-weighted images helps distinguish an adenoma from FNH (Figs. 7A and 7B). On T2-weighted images, hepatic adenomas can have variable signal intensity, but they are often mildly hyperintense relative to the liver (Fig. 7C). On dynamic contrast-enhanced images, adenomas show heterogeneous hypervascularity during the arterial phase. These masses often show no washout of contrast material, becoming isointense to the liver parenchyma on portal venous and subsequent dynamic series (Figs. 7D–7F), and they may have a delayed-enhancing pseudocapsule. Adenomas do not take up hepatocyte-specific contrast material and therefore appear hypointense to liver on delayed images when these agents are used (Fig. 7G).

**Hepatocellular Carcinoma**

HCC is the most common primary malignancy of the liver. It is often associated with underlying hepatic cirrhosis, which may be secondary to infectious (hepatitis), toxic (alcohol), or metabolic (e.g., hemochromatosis, Wilson disease, α-antitrypsin deficiency syndrome) processes. It is essential to accurately differentiate frankly malignant HCC from cirrhotic nodules ranging from benign regenerative to premalignant dysplasia, although this can be challenging because of considerable overlap in imaging and histologic features.

On T2-weighted images, HCC is usually hyperintense (Figs. 8A, 8B, and 9A). On unenhanced T1-weighted images, HCC is often isointense to liver, whereas larger lesions may be hyperintense secondary to their lipid, copper, or glycogen contents (Fig. 8C and 9D). High-grade dysplastic nodules and small HCC may have a nodule-within-a-nodule appearance, especially if a focus of HCC originates in a larger dysplastic nodule. Fatty metamorphosis in a cirrhotic nodule is highly suspicious for HCC (Figs. 9B and 9C). In cirrhotic patients, any
Fig. 6—Focal nodular hyperplasia.

A and B, Non-fat-suppressed (A) and fat-suppressed (B) T2-weighted images show lesion in caudate lobe–right lobe of liver (arrows), which is isointense to background liver and contains high-signal-intensity central scar. Fat-suppression increases conspicuity of lesion.

C, On unenhanced axial image, lesion (arrows) is isointense to background liver.

D and E, On volume interpolated gradient-echo with fat saturation images after administration of gadoxetate disodium (Eovist, Bayer HealthCare), lesion (arrows) enhances avidly during arterial phase (D) and becomes isointense to liver during portal venous phase (E).

F, Central scar (arrows), which was hypoenhancing on early contrast-enhanced images, enhances on delayed image.

G, On 20-minute delayed image, lesion (arrows) becomes isointense to background liver (i.e., takes up Eovist), consistent with intralesional bile ducts.
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Fig. 7—Hepatic adenoma. A and B, Out-of-phase (A) and in-phase (B) unenhanced axial T1-weighted images show segment III lesion (circle) with loss of signal on out-of-phase image compared with in-phase image, consistent with intralesional fat. C–G, On axial non-fat-suppressed T2-weighted image (C), lesion shows subtle high signal intensity compared with background liver (circle). Unenhanced (D) and contrast-enhanced volume interpolated gradient-echo with fat saturation (E) images after administration of gadoxetate disodium (Eovist, Bayer HealthCare) show that lesion has heterogeneous avid enhancement during arterial phase (E) and becomes isointense to mildly hypointense to liver on portal venous phase (F). On 20-minute delayed image (G), lesion is hypointense to background liver (i.e., no retention of contrast material in adenoma), feature that differentiates it from focal nodular hyperplasia.
Fig. 8—Hepatocellular carcinoma. A and B, Non-fat-suppressed (A) and fat-suppressed (B) axial T2-weighted images show lesion in segment VIII of liver (circle) that is mildly hyperintense compared with background liver. Note increased conspicuity of lesion on fat-suppressed T2-weighted image (B). C–G, On unenhanced volume interpolated gradient-echo with fat saturation image (C), lesion is isointense to background liver. After administration of gadoxetate disodium (Eovist, Bayer HealthCare), lesion enhances avidly during arterial phase (D) and becomes hypointense to liver on dynamic images (E and F [washout]). On 20-minute delayed image (G), lesion does not take up Eovist, consistent with lack of intralesional bile ducts. Circle indicates lesion in segment VIII of liver.
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hypervascular liver mass other than a hemangioma, which is larger than 2 cm and shows increased T2 signal similar to that of the spleen, is highly suspicious for HCC.

With dynamic contrast-enhanced imaging, small lesions (< 2 cm) may show homogeneous intense enhancement during the arterial phase. Larger lesions more often show heterogeneous enhancement. During the portal venous and equilibrium phases, HCC shows a rapid washout.
(loss of enhancement), becoming hypointense relative to the adjacent normal liver (Figs. 8D–8G and 9E–9G). A surrounding tumor capsule may be seen in lesions of any size. However, because the thickness of the capsule increases with tumor size, capsules are better seen with larger HCCs. The capsule, if present, is typically thin and discontinuous, hypointense to liver on both T1-weighted images and T2-weighted images, and shows progressive delayed enhancement.

Vascular shunts, THED, regenerative nodules, and dysplastic nodules are the more common lesions that require differentiation from HCC in patients with chronic liver disease. Vascular shunts and THED can also be seen in the absence of cirrhosis but are much more commonly encountered in the setting of cirrhosis. The liver has a dual blood supply, with approximately 75% of its flow from the portal vein and 25% from the hepatic artery. On contrast-enhanced MRI, the conduits supplying blood to the liver parenchyma (i.e., hepatic artery and portal vein pathways) are opacified tens of seconds apart, resulting in a variety of enhancement patterns if there is not uniform flow across the liver parenchyma. The hepatic arterial flow varies inversely with portal venous flow, so that any condition that decreases portal flow locally will result in an increase in the arterial flow. THEDs are the imaging manifestation of regional variations in the balance between hepatic arterial and portal venous inflow that
result in local heterogeneity and masslike appearance in some cases on contrast-enhanced MRI (Figs. 10 and 11). They also can result from nontumorous arteriportal shunts or obstruction of parenchymal portal venous flow and may cause arterial phase enhancement that can mimic an underlying mass. These arteriportal shunts in the setting of cirrhosis are a common cause of concern for HCC on imaging. A vascular shunt such as THED is typically geographically shaped, located peripherally, and isointense to liver on all unenhanced sequences (Figs. 10A, 10B, 11A, and 11B). The lesion is hypervascular on arterial phase images (Figs. 10C, 11C, and 11D) and becomes isointense to liver on delayed dynamic images, features that help distinguish arteriportal shunts from HCC (Figs. 10D, 10E, 11E, and 11F). Regenerative nodules result from localized proliferation of hepatocytes and their supporting stroma. These are simply the background nodules of cirrhosis, are typically small and isointense to liver on all sequences, and show a degree of enhancement similar to adjacent liver.
parenchyma, If they contain iron and/or copper, the nodules may be low signal intensity on
gradient-echo sequences (siderotic nodules) (Fig. 12). Dysplastic nodules are composed of
hepatocytes that show histologic characteristics of abnormal growth, may show hypervascu-
larity without washout or a capsule (unlike HCC), and are generally T2 hypointense (Fig. 12).
Fibrolamellar carcinoma, unlike HCC, has no association with underlying chronic liver dis-
ease, and its cause is unknown. At presentation, fibrolamellar carcinoma generally presents
as a large mass (5–20 cm) with well-defined and lobulated margins. On MRI, fibrolamellar
carcinoma is usually hypo- to isointense relative to the liver parenchyma on unenhanced T1-
weighted images and slightly hyperintense on T2-weighted images; foci of necrosis may be seen
as areas of high T2 signal. The central scar of fibrolamellar carcinoma has low signal intensity
on both T1- and T2-weighted images in contrast to FNH, which has a high signal intensity scar
on T2-weighted images. In addition, the central scar of fibrolamellar carcinoma shows minimal
or no enhancement.

Hypervascular Metastases
Metastases are the most common malignant hepatic tumor. Although most commonly multi-
focal, discrete lesions may sometimes manifest as a solitary mass or as confluent masses. All
hepatic tumors are preferentially supplied by the hepatic artery. Consequently, the degree of
enhancement depends on the briskness of the underlying hepatic vascular supply. Metastases
generally are irregular with indistinct margins. On T2-weighted images, hypervascular metas-
tases are usually hypointense and may be cystic or necrotic (Figs. 13A, 14A, and 14B). On
unenhanced T1-weighted images, hypervascular metastases generally are moderately hypoint-
tense relative to normal liver (Fig. 13B). However, hemorrhagic metastases (kidney, melanoma)

Fig. 12—Dysplastic and regenerative nodules.
A, Axial T2-weighted image of liver shows innumerable low-signal-intensity regenerative nodules. Arrow
points to dominant–dysplastic nodule. Liver has nodular contour (cirrhosis) with subtle intervening linear tracts
of high signal intensity throughout liver parenchyma that are consistent with background fibrosis.
B, On unenhanced volume interpolated gradient-echo with fat saturation image, nodules are hyperintense to
intervening fibrosis. Arrow points to dominant–dysplastic nodule.
C, After administration of gadopentetate dimeglumine, enhancement of nodules is similar to that of background
liver. Arrow points to dominant–dysplastic nodule.
D, On delayed dynamic image, lesions do not show washout (as documented by measurement and comparison
of regions of interest on arterial and portal venous contrast-enhanced images). Intervening linear tracts or
fibrosis enhances progressively on delayed dynamic images. Arrow points to dominant–dysplastic nodule.
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![Images of liver sections](image)

**Fig. 13**—Carcinoid metastasis.

A. Fat-suppressed axial T2-weighted image shows lesion in segment VI of liver (circle) that has blurry appearance and shows high signal intensity compared with background liver.

B–E. On unenhanced volume interpolated gradient-echo with fat saturation image (B), lesion shows low signal intensity compared with background liver. After administration of gadopentetate dimeglumine, there is avid enhancement of lesion in arterial phase image (C). Subsequent portal venous (D) and delayed dynamic (E) phase images show washout and lesion becomes hypointense compared with background liver. Circle indicates lesion in segment VI of liver.

Hypervascular metastases may be hyperintense to liver on unenhanced and contrast-enhanced images (Figs. 14C–14F). Perilesional fat deposition, which has been described as characteristic of hepatic metastases from a primary pancreatic insulinoma, is thought to be related to the effects of insulin on inhibiting fatty acid oxidation and promoting hepatocyte triglyceride accumulation.

Hypovascular metastases show decreased enhancement relative to normal liver and are most conspicuous on portal venous phase images. Hypervascular metastases enhance earlier and are best seen on arterial phase images. They also show perilesional rim enhancement and irregular washout on delayed images (Fig. 13E). Hypervascular metastases typically arise from primary neuroendocrine tumors (pancreatic islet cell tumor, carcinoid tumor, and pheochromocytoma), renal cell carcinoma, thyroid carcinoma, choriocarcinoma, or melanoma.
Fig. 14—Melanoma metastasis. A and B, Non-fat-suppressed (A) and fat-suppressed (B) axial T2-weighted images show lesion in segment III of liver (arrow, A and circle, B) that is mildly hyperintense to background liver. Fat suppression increases conspicuity of lesions in image B. C, On unenhanced volume interpolated gradient-echo with fat saturation image, lesion (circle) is hyperintense to background liver (due to melanin). D–F, On dynamic series images after administration of gadopentetate dimeglumine, parts of lesion enhance and show subtle washout (arrow, D and E and circle, F). These findings were visualized by comparison of regions of interest on arterial and portal venous phase images.

Vascular Fistulas Mimicking Hypervascular Lesions

Hypervascular liver lesions can be seen in cases of congenital or acquired or iatrogenic arteriovenous or venovenous fistulas. Multiplanar reformats and maximum-intensity-projection images are extremely helpful for assessment of these lesions (Figs. 15 and 16).

Conclusions

As the role of MRI for clinical hepatic imaging continues to evolve, keeping abreast of newer pulse sequences and contrast agents can be daunting as well as confusing. Because the liver can be involved primarily or secondarily by numerous vascular, metabolic, infectious, and neoplastic processes, the patient’s clinical history can have a considerable impact on the imaging differential diagnosis. Thus, a systematic approach that makes use of a decision algorithm...
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**Fig. 15**—Venovenous fistula (after liver biopsy).

A, Unenhanced volume interpolated gradient-echo image with fat saturation shows hypointense lesion in segment VII of liver (circle).

B, On contrast-enhanced image after administration of gadopentetate dimeglumine, there is avid enhancement of lesion (circle).

C, Multiplanar reformat image shows that lesion is venovenous fistula between right hepatic vein and right branch of portal vein (circle).

**Fig. 16**—Arteriovenous fistula (after liver biopsy).

A, Unenhanced volume interpolated gradient-echo image with fat saturation shows hypointense lesion in segment VIII of liver (circle).

B, Contrast-enhanced image after administration of gadopentetate dimeglumine shows avid enhancement of lesion in arterial phase (circle).

C, Maximum-intensity-projection image shows that lesion is arteriovenous fistula (circle) between right hepatic artery (arrow) and branches of right portal vein (arrowheads).

D and E, Lesion becomes isointense to background liver (circle) on subsequent dynamic images. Cirrhosis (i.e., innumerable hepatic nodules, splenomegaly, and varices) is also present.
Indentifiable lesions on MRI

Cirrhosis

Primary malignancy

Follow-up imaging

Primary malignancy yes

Short-term follow-up vs biopsy

Follow-up imaging

Primary malignancy no

Short-term follow-up vs biopsy

Note—Arrows indicate increased (↑) or decreased (↓) signal intensity or enhancement relative to surrounding liver parenchyma. T1-weighted imaging performed with volume interpolated gradient-echo with fat saturation. Eovist (gadoxetate disodium) manufactured by Bayer HealthCare.

*Heterogeneous appearance or signal intensity.

**Lesion T1-Weighted T2-Weighted T1-Weighted Arterial Phase T1-Weighted Venous Phase T1-Weighted Delayed Phase T1-Weighted Eovist 20-Min Delayed

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<th>Lesion</th>
<th>T1-Weighted</th>
<th>T2-Weighted</th>
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<th>T1-Weighted Venous Phase</th>
<th>T1-Weighted Delayed Phase</th>
<th>T1-Weighted Eovist 20-Min Delayed</th>
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**Note**—Arrows indicate increased (↑) or decreased (↓) signal intensity or enhancement relative to surrounding liver parenchyma. T1-weighted imaging performed with volume interpolated gradient-echo with fat saturation. Eovist (gadoxetate disodium) manufactured by Bayer HealthCare.

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**Suggested Reading**