MR Characterization of Focal Liver Lesions
Pearls and Pitfalls

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KEYWORDS

• Focal nodular hyperplasia • Hepatic adenoma • Hepatic steatosis • Hepatocellular carcinoma • Magnetic resonance imaging • Siderotic regenerative nodules

KEY POINTS

- Focal liver lesions that are isointense to hyperintense to liver on in-phase T1-weighted images are usually hepatocellular in origin.
- Focal liver lesions that lose signal intensity on an opposed-phase image compared with the matched in-phase image contain lipid and are usually hepatocellular in origin.
- Focal liver lesions that lose signal intensity on an in-phase image compared with an opposed-phase image are most often iron-containing siderotic nodules.
- Focal liver lesions that are isointense to spleen on T2-weighted images are solid and often malignant, whereas focal liver lesions that are hyperintense to spleen on heavily T2-weighted images are usually nonsolid benign cysts or hemangiomas.

PEARL 1: THE T1 PEARL: A FOCAL LESION THAT IS ISOINTENSE TO HYPERINTENSE TO LIVER ON T1-WEIGHTED IMAGES IS HEPATOCELLULAR IN ORIGIN

The first 3 imaging pearls discussed in this review are the 3 instances when focal liver lesion characterization is possible with T1-weighted gradient echo images.

The 3 most common focal liver lesions encountered in clinical practice are cysts, hemangiomas, and metastatic disease. Nonsolid benign hepatic lesions (cysts and hemangiomas) and almost all metastatic lesions are hypointense relative to liver on T1-weighted images (**Fig. 1**). Normal liver has relative high signal intensity on T1-weighted images that has been attributed to high concentrations of protein, rough endoplasmic reticulum, and paramagnetic substances, such as manganese and copper.[1–3] One study calculated the T1 relaxation times of liver at 1.5 T as 547 ms and that of solid lesions, hemangiomas, and cysts to be 1004, 1337, and 3143 ms, respectively. Thus, most liver lesions are initially detected on T1-weighted images as being hypointense to liver.[4] The authors use other pulse sequences besides T1-weighted images in order to differentiate among cysts, hemangiomas, and solid liver lesions.

If a focal liver lesion is isointense to hyperintense to liver on a T1-weighted image, then it is most commonly hepatocellular in origin. The 5 most common focal hepatocellular lesions encountered in clinical practice are regenerative nodules (RN), hepatocellular carcinoma (HCC), focal nodular hyperplasia (FNH), hepatocellular adenoma (HCA), and focal steatosis. In this section, the authors discuss FNH and the inflammatory subtype of HCA (IHCA). The reader is referred to the articles by Barr and Hussain and Sirlin in this issue of the

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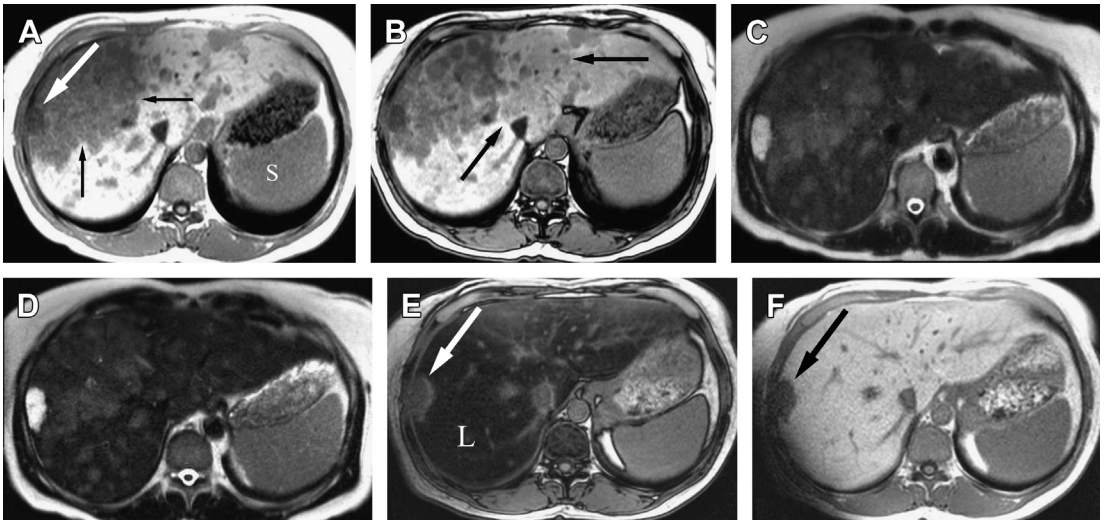

Fig. 1. Magnetic resonance demonstration of hepatic hemangioma and metastatic breast cancer in a 51-year-old woman. Metastases, cysts, and hemangiomas are almost all hypointense relative to liver on T1-weighted images. (*A*) Axial in-phase T1-weighted image shows low signal intensity hemangioma (*white arrow*) and infiltrative multifocal metastases (*smaller black arrows*). The metastases are isointense and the hemangioma is hypointense to spleen (S). However, the authors prefer to use T2-weighted images, diffusion-weighted images, and/or enhanced imaging to differentiate solid masses, such as metastatic disease, from nonsolid hepatic cysts and hemangiomas. (*B*) Corresponding opposed-phase image shows similar relative signal intensities of the hemangioma and metastatic disease. Geographic regions of lower signal intensity within the left lobe of liver (*black arrows*) represent steatosis. (*C, D*) T2-weighted fast-spin-echo images obtained with effective echo times of 90 (*C*) and 180 ms (*D*). The hemangioma is hyperintense to spleen, whereas the metastases are isointense. As the echo time increases, the contrast between the hemangioma and the adjacent metastases improve. This improvement is not because the hemangioma lights up or enhances; rather, the improved image contrast is because the hemangioma loses less signal as the echo time increases compared with liver, spleen, and metastases. (*E*) Opposed-phase T1-weighted imaging performed 2 years prior shows that the hemangioma (*arrow*) is hyperintense to the surrounding liver (L). When trying to establish the hepatocellular origin of a focal liver lesion by showing isointensity or hyperintensity to liver, one should use an in-phase image as steatotic liver can be of low signal intensity on opposed-phase imaging and can confound relative signal assessment. (*F*) Corresponding in-phase T1-weighted image shows that the hemangioma (*black arrow*) is hypointense to the steatotic liver.

Magnetic Resonance Imaging Clinics of North America concerning the evaluation of the cirrhotic liver and how to differentiate RN from HCC. Lipid and fat-containing liver lesions are discussed in "Pearl 2."

FNH

FNH is the second most common benign hepatic tumor in adults after hemangioma. FNH composes approximately 8% of all primary liver tumors and has an estimated prevalence between 0.3% and 3.0%.[5,6] FNH is not considered a neoplasm but instead is hypothesized to develop as a hyperplastic response of hepatic parenchyma around a central developmental vascular malformation.[7] Individuals with FNH are more likely to have coexistent hepatic hemangiomas (20%) than would be expected by chance[8]; both hemangioma and FNH involve focal abnormalities in the hepatic blood supply.

FNH is typically detected in women aged 20 to 50 years and is uncommon in men (female-to-male ratio = 10:1).[5] Unlike hepatic adenomas, there is no proven association of oral contraceptive use or pregnancy with the development or growth of FNH.[9,10] Although up to 15% of FNH lesions can grow when followed longitudinally,[11] this should not cause clinical concern. The authors are skeptical of reports of malignant transformation to fibrolamellar hepatoma[12] or HCC,[13] as most investigators think that malignant transformation of FNH does not occur.[14]

The magnetic resonance (MR) features of FNH can be considered in 2 parts (**Fig. 2**). The first component is the vascular nidus that forms the central scar of FNH, and the second is the surrounding hyperplastic response of adjacent liver. The vascular scar is hypointense to liver on both in-phase and opposed-phase imaging and is hyperintense to liver on T2-weighted imaging. The higher T2-weighted signal intensity is