Magnetic Resonance Imaging of Liver Lesions: Exceptions and Atypical Lesions

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On state-of-the-art magnetic resonance imaging, most lesions can be detected and characterized with confidence according to well-known criteria. However, atypical characteristics in some common lesions and the incidental encounter with rare lesions may pose diagnostic difficulties. In this article, six challenging hepatic lesions will be discussed and evaluated on the most important magnetic resonance imaging sequences, with histological correlation when available. In addition, the background information concerning these lesions will be described based on the most recent available literature. By reading this article, the reader will be able to (1) categorize the lesion in solid and fluid-containing lesions, based on the T2 signal intensity; and (2) define the benign or malignant nature of the lesion, in relation to the signal intensity and dynamic enhancement pattern, despite the presence of atypical characteristics of some lesions.

On state-of-the-art magnetic resonance (MR) imaging, most primary hepatocellular tumors can be detected and characterized accurately. For this purpose, careful evaluation of the information obtained in the four most important MR sequences is essential, including T1, T2, arterial, and delayed contrast-enhanced images. For a thorough description of the classic MR imaging findings of primary hepatocellular lesions and the most important lesions that need to be considered in the differential diagnosis, the reader is kindly referred to the article presented earlier in this issue by the same authors.

However, atypical characteristics in some common lesions and the incidental encounter with rare lesions may pose diagnostic difficulty. This includes atypical morphologic features, atypical location, or lesions that may mimic other primary hepatocellular tumors. To address this, we describe and illustrate six benign and malignant pathologically proven challenging cases that include the following: (1) giant hemangioma; (2) exophytic focal nodular hyperplasia; (3) hepatocellular carcinoma with a central scar; (4) fibrolamellar hepatocellular carcinoma; (5) epithelioid hemangioendothelioma; and (6) peripheral intrahepatic cholangiocarcinoma. Following the description of these lesions, relevant background information is provided based on the most recent available literature.

Giant Hemangioma

Hemangiomas are the most common benign hepatic lesions (incidence, 0.4 to 20%).[1] The origin of a hepatic hemangioma is a vascular malformation. Giant hemangiomas (>6 cm) may cause compensatory hypertrophy of the unaffected liver and may compress adjacent vessels.[2] It may present with abdominal discomfort, gastric outlet obstruction, or rarely, even spontaneous life-threatening rupture.[3,4] In addition, Kasabach–Merrit syndrome (disseminated intravascular coagulopathy) is rarely observed, in which thrombocytopenia occurs because of fibrinolysis within the hemangioma.[4] Some giant hemangiomas may present

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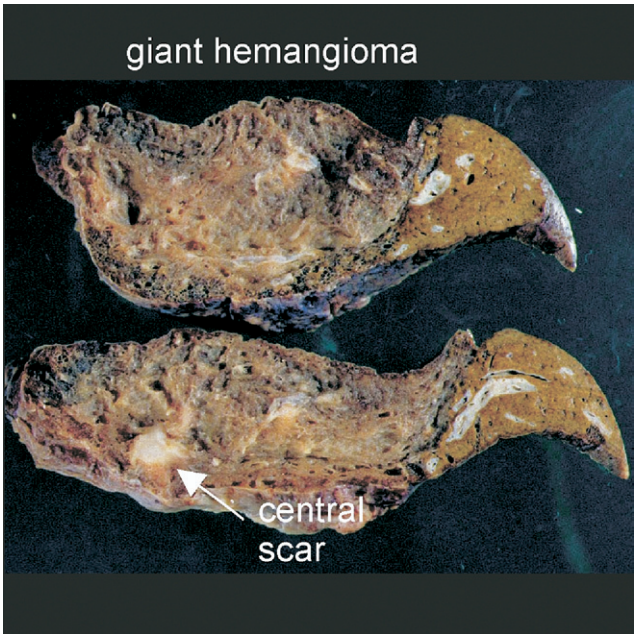

FIG 1. Macroscopic image of resected giant hemangioma. Note the areas of whitish central scar-like tissue, which is composed of myxoid tissue. (Color version of figure is available online.)

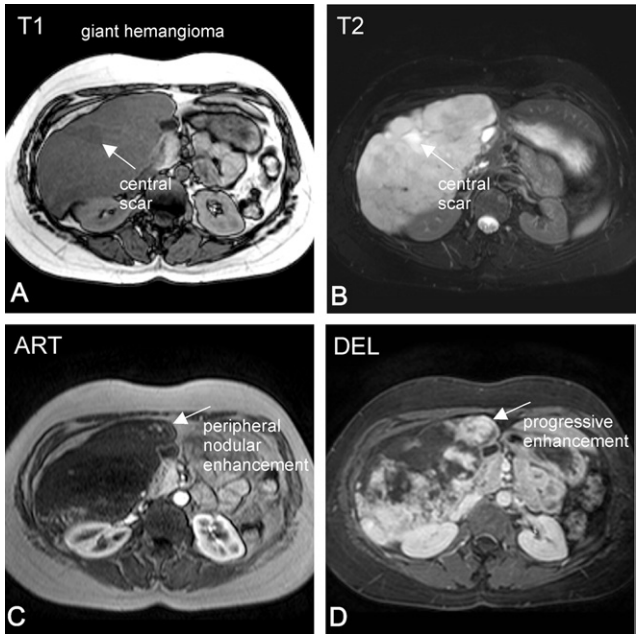

FIG 2. Giant hemangioma in a 47-year-old female patient. (A) Opposed-phase T1-weighted gradient-recalled echo (GRE), showing hypointensity of the lesion, with a hypointense central scar (arrow). (B) Fat-suppressed T2-weighted fast spin-echo (FSE), showing very high fluid-like signal intensity of the entire lesion. Note the even higher signal intensity of the central scar (arrow). (C) Arterial-phase T1-weighted GRE, illustrating peripheral nodular enhancement in a discontinuous ring. (D) Delayed-phase T1-weighted GRE, showing progressive enhancement toward the center of the lesion.

with fever of unknown origin.[5] Symptomatic lesions may be treated surgically; however, as demonstrated in a recent study, size is not a criterion for resection of the lesion, but the degree of complaints should be considered in the evaluation of possible surgical intervention in these patients.[3]

At histology, giant hemangiomas do not differ much from their smaller variants. However, a central scar may be observed within the lesion, which often consists of myxoid tissue components[6] (Fig 1). In addition, areas with either thrombosis or fibrosis may be observed.

At MR imaging, giant hemangiomas typically show low signal intensity on T1, and homogeneous high signal intensity on the T2-weighted images (Fig 2), which remains high or even increases on the T2-weighted images with longer echo times. In the dynamic gadolinium-enhanced sequences, giant hemangioma typically shows a discontinuous ring-like enhancement in the periphery of the lesion in the arterial phase, with slow filling-in toward the center of the lesion over time[4] (Fig 2). Most giant hemangiomas are associated with a central scar within the lesion, which frequently depicts high signal intensity on the T2-weighted images, although this may vary. Areas of distinctly higher signal intensity may be observed within the scar, which correspond to myxoid tissue, while areas of lower signal intensity correspond to fibrous septa.[2,6] Usually, the central scar does not enhance after gadolinium administration.

The differential diagnosis of a large solitary liver lesion with a central scar includes focal nodular hyperplasia (FNH), hepatocellular carcinoma (HCC), or rarely, hepatocellular adenoma (HCA). Giant hemangioma shows very high T2-signal intensity compared with these (solid) lesions, indicating the nonsolid origin with fluid-like hyperintense signal. In addition, the enhancement pattern of giant hemangioma is quite characteristic, with peripheral nodular or sometimes flame-like enhancement which persists over time. Particularly, the central scar in giant hemangiomas has very high signal intensity on T2-weighted images (higher than the remainder of the lesion) and remains unenhanced after injection of gadolinium. FNH, on the other hand, shows homogeneous flash-like arterial enhancement with fading to isointensity in the delayed phase. The central scar in FNH usually shows enhancement in the delayed phase after gadolinium injection. HCC is characterized by