Focal Nodular Hyperplasia with Retraction of Liver Capsule: A Case Report

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Focal nodular hyperplasia (FNH) is characterized by the presence a central scar with radiating fibrous septa. Our case had a capsular retraction, which was the result of an extension of the central scar to the surface. In addition, a hypointense scar on the T2-weighted image and a minimal enhancing central scar on the enhanced T1-weighted image, which was due to dense, sclerotic collagenous tissue, were observed. We report the first case of FNH with a capsular retraction.

Focal nodular hyperplasia (FNH) is the second most common benign hepatic tumor next to hemangioma (1). It is mainly found in young women. Generally, FNH presents as a solitary nodule < 5-cm in diameter near the liver surface (1, 2). The mass is usually lobulated and well circumscribed. The characteristic macroscopic features is the presence of a central scar with radiating fibrous septa, dividing the lesion into numerous nodules of normal hepatocytes, which are abnormally arranged (2).

A retraction of the liver capsule adjacent to the tumor is an unusual finding. The image findings of the peripheral hepatic masses with a capsular retraction can be observed in some malignant hepatic tumors, such as a hepatocellular carcinoma, a peripheral cholangiocarcinoma, a metastasis (3, 4), and a hepatic epithelioid hemangioendothelioma (5). However, this finding has seldom been associated with a benign tumor.

Several atypical imaging findings exist in FNH (1), but there is no report showing a capsular retraction. We report the atypical imaging findings of FNH, which showed a capsular retraction.

CASE REPORT

The patient was a 28-year-old woman who was admitted to hospital because of a hepatic mass found incidentally on screening sonography. She had no previous surgical and medical problems. A physical examination and the laboratory findings on admission revealed no abnormalities. The serum alpha-fetoprotein level was 1.2 ng/mL and the carcinoembryonic antigen level was 1.3 ng/mL. Test for the hepatitis-B antigen was negative and the test for hepatitis-B antibodies was positive.

CT of the abdomen showed a mass in the segment 6 measuring approximately 4.0 × 3.8-cm with a capsular retraction. The hepatic helical CT scan during the hepatic arterial phase showed a heterogeneously enhancing mass with a lobulated margin. A central scar was shown as a stellate hypodensity. The hepatic helical CT scan during the portal phase showed an isodense mass with a hypodense central scar (Fig. 1A).
These findings were compatible with the focal nodular hyperplasia except for a central scar extension to the hepatic capsule causing a retraction. The T1-weighted axial MR image of the liver demonstrated a slightly hypointense mass demarcated by a thin hypointense rim with a central hypointense scar. The T2-weighted axial MR image showed a slightly hyperintense mass with a hypointense central scar. The contrast-enhanced T1-weighted axial MR image revealed a marked enhancement of the tumor with a nonenhancing central scar (Fig. 1B).

Segment 6 of the liver was resected. The gross specimen showed a nodular configuration with a dense fibrotic central scar. On the cut surface, a central dense fibrotic scar with radiating fibrous septa dividing the lesion into smaller nodules was noted (Fig. 1C). The central scar extended to the surface, and the surface of the liver parenchyma adjacent to the mass was retracted. The capsular retraction of the liver surface was attributed to the collagenous scar. The resected specimen showed a depressed thickened stellate scar, slightly eccentrically positioned, with tapering fibrous septa, which radiated through the mass, dividing it into multiple lobules. The patient took an uneventful postoperative course and was discharged.

**DISCUSSION**

Focal nodular hyperplasia (FNH) is found predominantly in women during the third to fifth decade of life. However, it has been reported to occur in both sexes and in all age groups. Most commonly, it is found incidentally in asymptomatic patients. The cause of FNH is not well understood, but a congenital vascular malformation or vascular injury has been suggested to be an underlying mechanism for the hepatocellular hyperplasia (2).

FNH is classically seen as a solitary, homogeneous, and slightly hypoattenuating or isoattenuating area compared to the normal liver on unenhanced CT (1). In approximately 20% of patients, a central low-attenuating scar may be observed (6). On enhanced CT, FNH shows an immediate and intense enhancement, with the exception of a central
scar with a delayed enhancement, possibly due to abundant fibrous stroma (6). The atypical imaging findings of FNH include hemorrhage, necrosis, fat accumulation on an unenhanced CT, rapid contrast washout, delayed contrast accumulation, or the absence of a central scar (1, 2).

The MR imaging of typical FNH is iso- or slightly hypointense mass with hypointense scar on the T1-weighted MR imaging and a slightly hyperintense mass with a hyperintense scar on the T2-weighted image. Intensely and immediately enhancing mass with a delayed enhancing central scar is a classic finding on the Gadolinium enhanced MR (1, 2). According to Vilgrain et al (7), when the fibrosis, edema, vessels, and inflammation of the central scars were carefully studied, obliterator vascular changes were observed in both hyperintense and hypointense scars on the T2-weighted images. They reported that the distribution of fibrosis, inflammation, and vessels were not different in the hyper- and hypointense scars, but most of the hyperintense scars had predominant edema, whereas the hypointense scars showed an absence of or a lower degree of edema (7). A similar correlation between the pathology findings and MR imaging was done to evaluate the central scars of the primary liver tumor (8). Rummeny et al (8) described three pathologic types of the central scars: (a) vascular scars composed mainly of vascular channels penetrating the collagenous tissue; (b) inflammatory scars with edema, necrosis, hepatocellularity and loose connective tissue; and (c) collagenous scars composed predominantly of dense, sclerotic collagen. Vascular and inflammatory scars appeared hypointense relative to the liver parenchyma on the T1-weighted images, and hyperintense on the T2-weighted images, while collagenous scars were hypointense on both the T1- and T2-weighted images. In our case, the microscopic findings showed a collagenous scar composed mainly of dense, sclerotic collagen, which explains the cause of the hypointensity on the T2-weighted image and a minimal enhancing central scar on the enhanced T1-weighted image.

The cut surface of a typical FNH revealed a central stellate scar with radiating fibrous septa dividing the lesion into smaller nodules. The central scar contained thick-walled vessels that provided arterial blood supply to the lesion (2). Grossly, the resected specimen showed a depressed thickened stellate scar, slightly eccentrically positioned, with tapering fibrous septa that radiated through the mass, dividing it into multiple lobules. Microscopically, numerous fibrous bands of varying thickness divided the lesion, surrounding the nodules of the hyperplastic hepatocytes and converging at the central scar. Several large blood vessels, which exhibited varying degrees of myointimal hyperplasia, mural thickening and intimal narrowing, were observed within the dense collagenous scar. These radiating septa contained numerous thin-walled vessels, lymphocyte infiltrates and numerous proliferated walled bile ducts, which were closely apposed to the periportal bile ducts. The hepatocytes nodules were composed of normal parenchymal elements, which were irregularly arranged with plates of one or two cells’ thickness but the normal lobular architecture was not seen.

A capsular retraction of the liver adjacent to the hepatic mass is an uncommon finding. Soyer et al. (3) reported that the prevalence of the capsular retraction adjacent to the hepatic tumor was 2% and all tumors were malignant. Seo et al (4) suggested that the prevalence was approximately 12% in malignant hepatic masses. These findings have been observed in cases of hepatic epitheloid hemangioendothelioma (HEH), hepatocellular carcinoma, intrahepatic cholangiocarcinoma, and metastases from colon and breast cancers (3–5, 10). The capsular retraction of HEH was most likely due to the tumor fibrous reaction, which distorted the tumor margin and the adjacent liver capsule (5). The main factors causing the capsular retraction in malignant hepatic tumors are a portal venous obstruction in the hepatocellular carcinoma, and hepatic atrophy followed by a bile duct obstruction in a cholangiocarcinoma (4). Soyer et al. (3) reported that this finding was never associated with a benign tumor, supporting the hypothesis that a retraction of the liver capsule adjacent to a hepatic tumor is a finding specific to malignant tumors. Recently, Yang et al. (9) reported a capsular retraction in hepatic giant hemangioma. A capsular retraction with hemangiomas in cirrhotic livers has also been described (10).

Although Blachar (10) reported that the hemangioma was the only benign neoplasm associated with a capsular retraction, this study found another benign hepatic mass with a capsular retraction. This is the first report showing a capsular retraction in focal nodular hyperplasia. In this case, the central fibrotic scar within the tumor was extended to the surface, and a retraction of the liver capsule adjacent to the tumor was made.

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