

The Sonographic "Scar Sign" in Focal Nodular Hyperplasia of the Liver

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The sonographic features of focal nodular hyperplasia of the liver as reported in the literature are nonspecific. However, a linear cluster of bright echoes was detected in the nodules of two of our patients with surgically proven focal nodular hyperplasia. Pathologically this finding correlated closely with the gross appearance of the characteristic fibrotic scar of focal nodular hyperplasia. When present, this "scar sign" should suggest the diagnosis of focal nodular hyperplasia, particularly in the typical clinical setting. (Key words: focal nodular hyperplasia, hepatic sonography, fibrotic scar.)

Focal nodular hyperplasia of the liver is a rare, benign hepatocellular tumor of unknown pathogenesis.¹⁻³ In the past, radiologic diagnosis of focal nodular hyperplasia depended primarily on radio-nuclide liver scanning and angiography.¹⁴ Within the past seven years, the sonographic appearance of focal nodular hyperplasia has been described in numerous case reports.⁵⁻¹⁶ More recently, computed tomography (CT) has rekindled interest in the noninvasive imaging of focal nodular hyperplasia.^{13,16,17}

A variable and nonspecific sonographic pattern in focal nodular hyperplasia has been reported in recent review articles.^{15,16} Yet, the gross pathologic findings in focal nodular hyperplasia are usually distinctive and characteristic: a central, stellate fibrotic scar with connective tissue septa radiates into a nodular, tannish mass.^{3,16,18} The diagnostic significance of identifying this pathologic signature by CT has recently been stressed by Fishman et al.¹⁷ However, the sonographic demonstration of this stellate scar has not been described in the ultrasound literature.

The following report represents two cases of patients with surgically proven focal nodular hyperplasia, and provides sonographic and gross pathologic correlation in each case. The CT findings in Case 1 have been reported by Fishman et al.¹⁷

MATERIALS AND METHODS

The ultrasound examinations were performed with commercially available B-mode units using a 3.5-MHz medium-focus transducer. Studies of the two patients with focal nodular hyperplasia were reviewed and compared with photographs of the cut surgical specimens provided by the Department of Surgical Pathology at The Johns Hopkins Hospital.

CASE REPORTS

Case 1. A 30-year-old woman sought medical treatment for epigastric pain. Slight hepatomegaly had been noticed a year prior to admission. Two months before admission, the patient had noticed left upper quadrant and epigastric pressure associated with early satiety. No weight loss was documented, however. The pain had become much worse on the day prior to admission.

The patient denied a history of hepatitis, jaundice, or pruritus. She had taken oral contraceptives for six years, but had discontinued them seven years prior to admission, when tubal ligation had been performed. She denied any current medications.

Physical examination revealed a large, firm, slightly tender mid-epigastric mass, over which a bruit was easily heard. The hematocrit was 29 per cent, but all other laboratory test results were normal.

Sonography (fig. 1, A and B) demonstrated a solid mass $14 \times 5 \times 8$ cm containing a linear cluster of bright echoes in the left hepatic lobe. CT revealed a central, low-density stellate cleft within the mass on unenhanced scans, and diffuse enhancement of the mass following intravenous bolus contrast medium injection. A ^{99m}Tc-sulfur colloid liver scan showed decreased tracer uptake in the left lobe mass. Selective hepatic angiography demonstrated a highly vascular mass. At surgery, a left hepatic lobectomy was performed after biopsy revealed focal nodular hyperplasia. The cut section of the gross specimen is shown in figure 1C.

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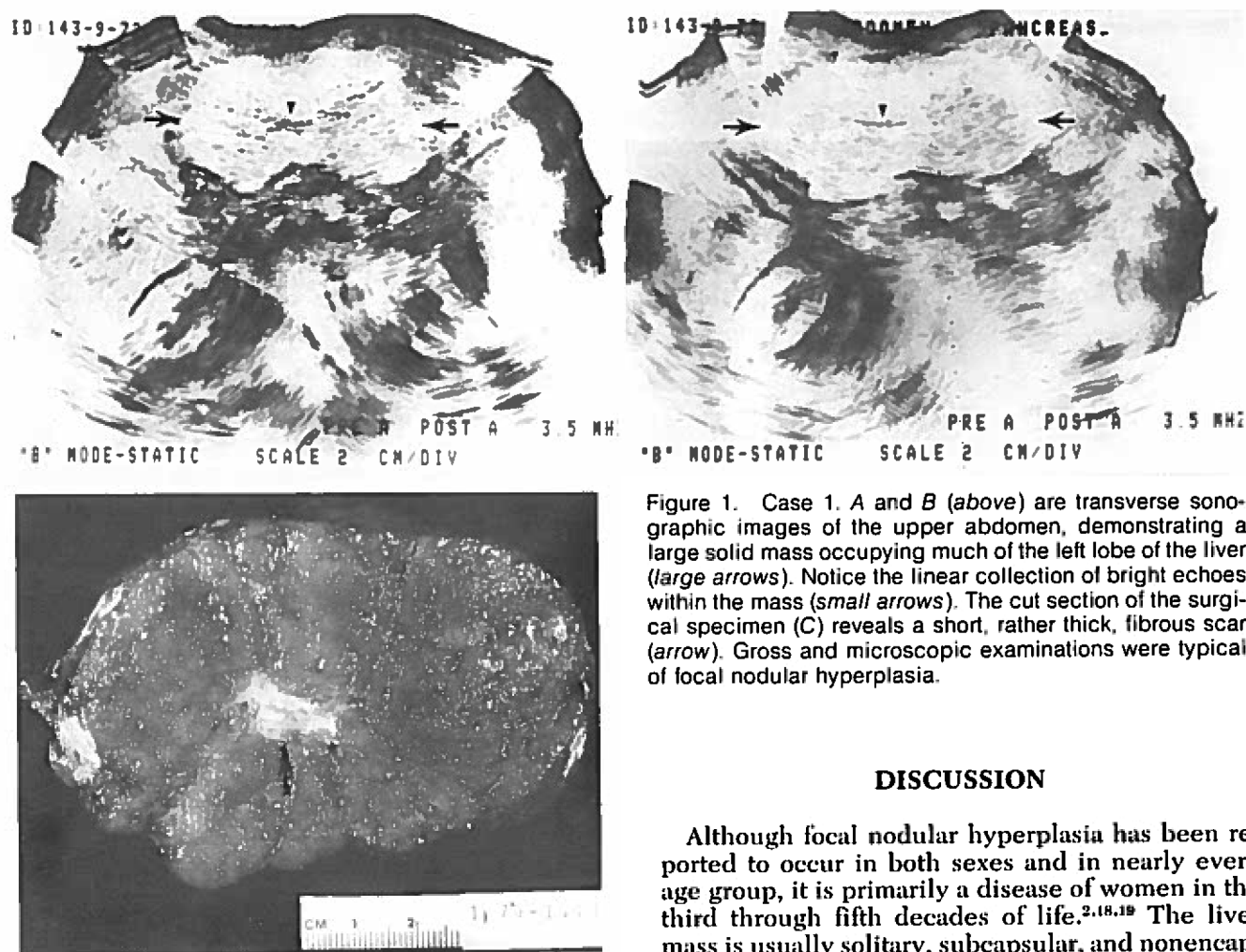


Figure 1. Case 1. A and B (above) are transverse sonographic images of the upper abdomen, demonstrating a large solid mass occupying much of the left lobe of the liver (large arrows). Notice the linear collection of bright echoes within the mass (small arrows). The cut section of the surgical specimen (C) reveals a short, rather thick, fibrous scar (arrow). Gross and microscopic examinations were typical of focal nodular hyperplasia.

DISCUSSION

Although focal nodular hyperplasia has been reported to occur in both sexes and in nearly every age group, it is primarily a disease of women in the third through fifth decades of life.^{2,18,19} The liver mass is usually solitary, subcapsular, and nonencapsulated, averaging 5 cm in diameter. However, masses as large as 24 cm have been documented.^{3,18} Incidences of multiple nodules range from 7 to 15 per cent.^{1-3,18} The intrahepatic distribution of focal nodular hyperplasia masses has been disputed. Sorensen and Baden¹ reported a higher incidence in the left hepatic lobe, while Fechner¹⁹ cited an equal right-to-left distribution, and Ishak and Rabin² found a 2-to-1 right lobe-to-left lobe bias.

Focal nodular hyperplasia is usually discovered as an incidental finding at laparotomy, laparoscopy, or necropsy.^{1,2} Of patients with focal nodular hyperplasia, 20 to 35 per cent have symptoms or signs, usually vague epigastric pain, a palpable mass, hepatomegaly, or, rarely, intrahepatic or intraperitoneal hemorrhage.^{1,2,18} Although a relationship between focal nodular hyperplasia and oral contraceptive use has been postulated,^{6,8} several authors dispute this claim.^{2,19} Focal nodular hyperplasia carries no apparent malignant potential.¹⁻³

On gross pathologic examination focal nodular hyperplasia is a well-circumscribed, nodular cirrhotic-like mass of tan tissue within otherwise normal hepatic parenchyma.^{3,18} The characteristic

Case 2. A 40-year-old woman was admitted for evaluation of a left hepatic lobe mass discovered incidentally during gynecologic surgery. An intraoperative biopsy of the liver mass was inconclusive. Therefore, following recovery from the pelvic surgery, the patient was transferred to The Johns Hopkins Hospital. No history of exposure to hepatotoxins or use of oral contraceptives could be elicited.

Physical examination revealed a smooth, nontender epigastric mass. All results of laboratory studies were normal except for very slight elevations of the serum glutamic pyruvic transaminase and serum alkaline phosphatase levels.

Gray-scale hepatic sonography confirmed a hypoechoic solid mass 8 × 10 cm with a linear cluster of echoes in the left lobe (fig. 2, A and B). Unenhanced CT revealed a mass of decreased attenuation and an equivocal central cleft; the mass became isodense following an intravenous bolus injection of contrast medium. Selective hepatic angiography demonstrated a hypervascular mass consistent with focal nodular hyperplasia or hepatoma. At surgery, left hepatic lobectomy confirmed focal nodular hyperplasia. A cut section of the gross surgical specimen is shown in figure 2C.

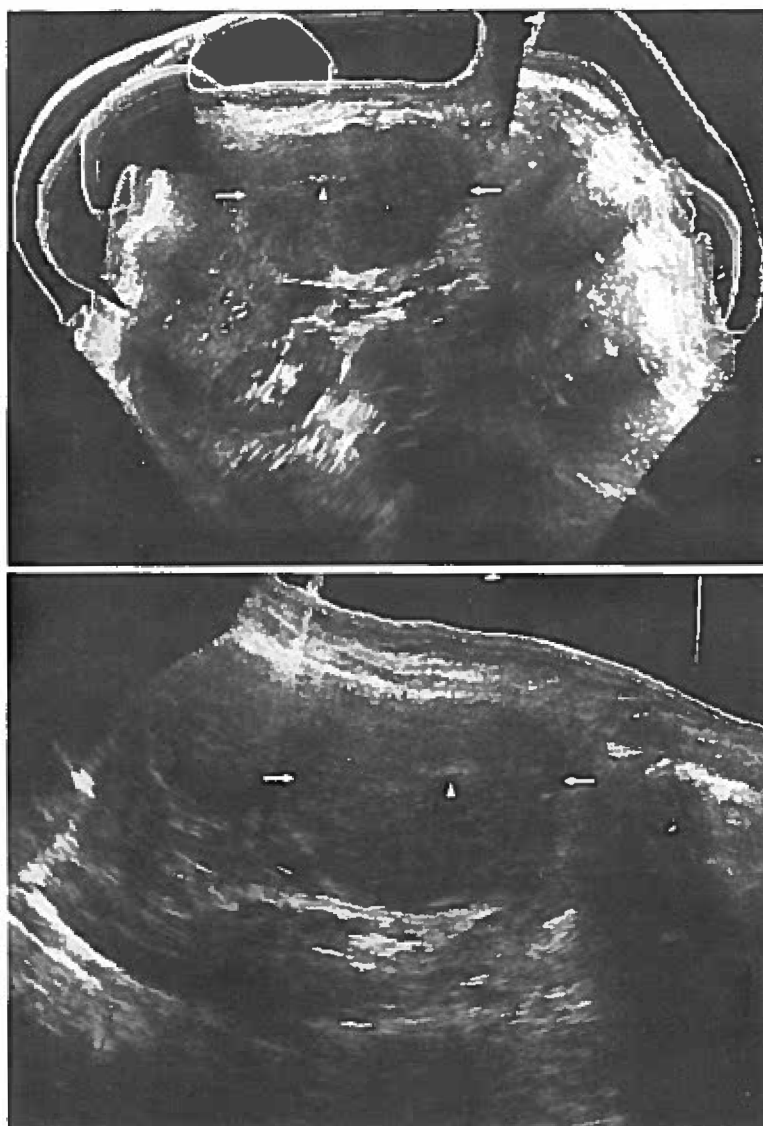
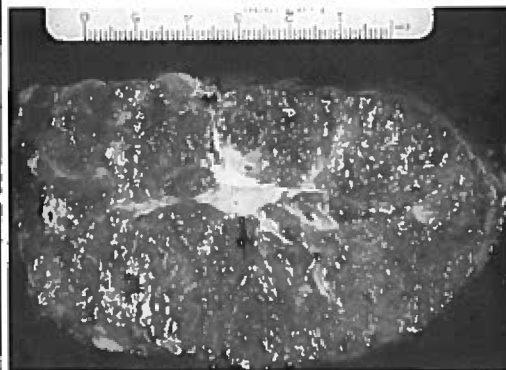


Figure 2. Case 2. Transverse (A, above) and longitudinal (B, below) sonographic images of the liver reveal a mass (large arrows) occupying most of the left lobe. The echogenicity of this mass is slightly less than that of normal liver. A short linear horizontal cluster of bright echoes (small arrows) is visible within the mass on both projections. Section of the surgically resected mass (C) demonstrates a prominent central fibrous scar with radiating septa (arrow), the typical gross appearance of focal nodular hyperplasia. Microscopic study confirmed that diagnosis.



depressed, central, or eccentric stellate scar is composed of dense fibrous connective tissue, proliferating bile ducts, and thin-walled blood vessels.^{2,18} Connective tissue septa radiate from this fibrous cleft, dividing the cytologically normal hepatocytes and Kupffer cells into lobules. Hemorrhage within focal nodular hyperplasia is unusual.

The sonographic features of focal nodular hyperplasia, recently reviewed by Sandler et al. and Rogers et al., have been variable.^{15,16} The mass, frequently subcapsular, is usually sharply defined and can show echogenicity either greater or less than that of the adjacent uninvolved liver tissue. In the unusual case of hemorrhage within focal nodular hyperplasia, a complex pattern of mixed echogenicity has been reported.^{7,10,16}

The two patients described in this report had large, solid, left hepatic lobe masses with echo-

genicity equal to or slightly less than that of the nearby uninvolved parenchyma (figs. 1 and 2). Of crucial significance, however, is the discrete linear cluster of echoes within each mass (arrows, figs. 1A, 1B, 2A, and 2B), which corresponds closely to the central fibrotic scar visible on the cut gross pathologic specimen (arrows, figs. 1C and 2C). Although in vitro ultrasonic imaging was not performed on either resected surgical specimen, we believe that the interface between histologically normal liver tissue and the central fibrotic scar with radiating septa accounted for the prominent linear echoes observed in each case.

Gas bubbles and calcification can occasionally develop within hepatic masses. Sonography of such a mass will show an aggregate of very bright echoes with an associated acoustical shadow.^{20,21} The absence of acoustical shadowing in the sonograms of

the two patients in this report is an important differential diagnostic clue in support of focal nodular hyperplasia.

Review of the sonographic literature relating to focal nodular hyperplasia did disclose one case report of a large hepatic mass that contained an irregularly shaped, eccentric concentration of bright echoes.¹² Although no pathologic correlation was presented in that article, perhaps the echoes were produced by a large fibrous scar.

Hence, sonographic visualization of a dense, non-shadowing linear or stellate group of echoes in a solitary, solid hepatic mass may be specific for or at least highly suggestive of focal nodular hyperplasia. However, several factors may limit the usefulness of this sonographic "scar sign." First, the characteristic central scar of focal nodular hyperplasia may be small or absent altogether, thereby eliminating the "scar sign." The pathology literature describing focal nodular hyperplasia does not clearly state how frequently the cleft is lacking, although the scar's absence is probably uncommon. Second, careful scanning technique with a higher-frequency transducer may be necessary to display optimally the characteristic internal anatomy of focal nodular hyperplasia. While masses along the caudal margin of the left or right lobe would present no technical problem, focal nodular hyperplasia along the diaphragmatic surface might be technically more challenging. Third, a linear or stellate cluster of bright echoes within a hepatic mass may occur in diseases other than focal nodular hyperplasia. Conceivably, linear or stellate zones of fibrosis or organized hematoma within a liver cell adenoma or cavernous hemangioma might mimic the "scar sign." However, no such report exists in the current literature. Finally, hemorrhage within focal nodular hyperplasia, although rare, might distort the internal structures sufficiently to render even a prominent central scar sonographically invisible.

CONCLUSION

We believe that the linear or stellate sonographic "scar sign" should suggest focal nodular hyperplasia, particularly when observed in the typical clinical setting. However, until further experience

with this rare tumor is gained, absence of this sign does not exclude focal nodular hyperplasia.

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