Focal nodular hyperplasia

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INTRODUCTION

Focal nodular hyperplasia (FNH) is a benign liver lesion that is composed of a proliferation of hyperplastic hepatocytes surrounding a central stellate scar. Typically, FNH is a solitary lesion that is more commonly seen in women.

This topic will discuss the pathologic features, clinical features, diagnosis, and management of FNH. The approach to patients with other benign, solid liver lesions is discussed separately. (See "Hepatic hemangioma" and "Hepatocellular adenoma".)

The approach to patients with cystic liver lesions is discussed separately. (See "Diagnosis and management of cystic lesions of the liver".)

The clinical features, diagnosis, and management of hepatocellular carcinoma are discussed separately:

- (See "Clinical features and diagnosis of hepatocellular carcinoma".)
- (See "Overview of treatment approaches for hepatocellular carcinoma".)
- (See "Surveillance for hepatocellular carcinoma in adults".)

EPIDEMIOLOGY

Prevalence — FNH is the second most commonly encountered benign liver lesion, while hepatic hemangioma is the most common. The estimated prevalence of FNH based on autopsy

series is 0.3 to 3 percent; however the prevalence of FNH based on clinical series is lower at 0.03 percent [1-3].

There is increasing recognition of FNH among patients undergoing abdominal imaging for other reasons. In a large observational series including patients referred for ultrasound or contrast-enhanced computed tomography, the prevalence of FNH was 0.2 percent and 1.6 percent, respectively [4,5].

Age and sex — FNH occurs in patients of any age, while most patients present between the ages of 35 and 50 years [6]. FNH is uncommon in children and comprises approximately 2 percent of pediatric liver tumors [7,8].

FNH is seen in both males and females, although it is found predominantly in females (up to 90 percent) [9,10].

Possible risk factors — Most data suggest that female sex hormones (eg, oral contraceptive pills) do not appear to be a risk factor for the initial development or subsequent growth of FNH [11-15].

PATHOGENESIS

FNH is a proliferation of hepatocytes described as a hyperplastic (regenerative) reaction to an aberrant dystrophic artery [16], to portal tract injury resulting in arterial to venous shunts [17], or to a congenital vascular malformation [18]. The hepatic stellate cells respond to oxidative stress caused by arterial hyperperfusion, and this results in the characteristic fibrous scar found in the center of the lesion (ie, central scar) [19-22].

FNH may occur in association with other vascular diseases including hereditary hemorrhagic telangiectasia [23] and hepatic hemangiomas [24,25].

PATHOLOGIC FEATURES

Typical form — FNH is typically a firm, solitary lesion with a well-defined margin (but without a capsule), composed of benign hyperplastic hepatocytes surrounding a central stellate scar [2]. Lesion size is usually <5 cm, but measurement up to 19 cm has been reported [24]. FNH is often in a subcapsular location and may be pedunculated. The characteristic finding is the presence of a central stellate scar (picture 1) containing a large artery with multiple branches radiating through the fibrous septa to the periphery of the lesion.

Microscopically, FNH is composed of normal-appearing hepatocytes grouped in nodules that are usually divided by fibrous septa radiating from the central scar (picture 2 and picture 3) [9]. The fibrous septa are composed of varying degrees of enlarged portal tracts including feeding arteries, portal veins, and bile ductules [20]. Kupffer cells are typically present, a feature that distinguishes FNH from hepatocellular adenoma, which usually lacks bile ducts and Kupffer cells [1,24,26-28]. (See 'Differential diagnosis' below.)

Atypical forms — Atypical forms of FNH include [9]:

- FNH without a central scar – This variant lacks the characteristic central scar, and most of these lesions are <3 cm [24].
- FNH with steatosis – FNH with steatosis is a recognized variant that is typically seen in patients with underlying hepatic steatosis [29].

CLINICAL FEATURES

Patterns of clinical presentation — The spectrum of presentation of FNH ranges from asymptomatic individuals with incidental findings on imaging (or intraoperatively) to patients with nonspecific symptoms [24,30,31].

When symptoms are present, the most commonly reported symptom is abdominal pain [24].

Physical examination is often normal but infrequently may demonstrate hepatomegaly or an abdominal mass [24].

Laboratory findings — Liver biochemical and function tests are typically normal in patients with FNH [24,30]. Alpha-fetoprotein is normal.

Imaging

Noncontrast ultrasound — FNH has a generally isoechoic appearance on noncontrast ultrasound. Doppler studies show the central arteries having a spoke-wheel pattern [32].

Contrast-enhanced imaging — Characteristics of FNH seen on contrast-enhanced imaging (eg, magnetic resonance imaging [MRI], computed tomography [CT] scan, or contrast enhanced ultrasound [CEUS]) include [9,33,34]:

- Central scar is surrounded by a homogenous lesion.
- Lesion appearance differs from the surrounding liver.

- Homogenous enhancement is seen during arterial phase, then the lesion returns to precontrast density during portal phase [35-37].

- The lesion lacks a capsule, but its edges often appear lobulated.

FNH features by specific imaging modality include:

- Magnetic resonance imaging – Administration of a liver-specific gadolinium-based magnetic resonance contrast agent produces rapid enhancement of FNH due to its arterial blood supply, producing a hyperintense lesion on early films (image 1). On delayed images it becomes more isointense with respect to normal liver. The central scar enhances on delayed imaging as contrast gradually diffuses into the fibrous center of the mass [38-41].

  On precontrast MRI, FNH may appear similar to the adjacent liver parenchyma, because FNH is composed of hepatocytes. An isointense lesion is noted on T1-weighted images, while an isointense to slightly hyperintense mass appears on T2-weighted images [42]. The scar typically shows high-signal intensity on T2-weighted images due to vessels within the scar (image 2) [33].

  Patient preparation and indications for intravenous administration of a gadolinium-based contrast agent are discussed separately. (See "Patient evaluation before gadolinium contrast administration for magnetic resonance imaging".)

- CT scan – On multiphasic CT scan, FNH may appear hypo- or isodense on precontrast images. The arterial phase shows a homogenous hyperdense lesion that generally becomes isodense during the portal venous phase [43]. On delayed imaging, the central scar often becomes hyperdense as contrast diffuses into the fibrous scar.

- Contrast-enhanced ultrasonography – On CEUS, FNH typically demonstrates arterial and (early) portal venous phase enhancement, while the central arteries display a spoke-wheel pattern in the arterial phase [36,37,44,45]. Use of CEUS in the United States has been limited, but it is more widely available in other countries. (See "Contrast-enhanced ultrasound for the evaluation of liver lesions".)

DIAGNOSTIC APPROACH

The diagnosis of FNH may be suspected in a patient without cirrhosis who is found to have a solid liver lesion on imaging. For patients with suspected FNH based on ultrasound or computed tomography (CT) findings, the diagnosis is made with cross-sectional, contrast-

enhanced multiphase magnetic resonance imaging [46]. (See 'Contrast-enhanced imaging' above.)

The general approach to incidental liver lesions identified by ultrasound or CT is discussed separately. (See "Approach to the adult patient with an incidental solid liver lesion".)

A biopsy of the lesion is not routinely indicated to confirm the diagnosis, but may be necessary if the contrast-enhanced imaging features are not typical of FNH. (See 'Differential diagnosis' below.) Alternatively, patients who are symptomatic may undergo surgical resection, and the diagnosis is then confirmed with histology. (See 'Symptomatic lesions' below.)

DIFFERENTIAL DIAGNOSIS

If cross–sectional imaging of the liver lesion is not specific, additional evaluation for an alternative diagnosis may be required. The differential diagnosis includes:

- **Hepatocellular adenoma** – Although contrast-enhanced multiphasic imaging (ie, magnetic resonance imaging [MRI]) often differentiates between FNH and hepatocellular adenoma (HCA), further evaluation (eg, biopsy) may be required for definitive diagnosis. In a systematic review of six studies including over 300 patients who had multiphase MRI with hepatobiliary contrast, the sensitivity and specificity to differentiate FNH from HCA were high, with ranges from 91 to 100 percent and from 87 to 100 percent, respectively [47]. In addition, HCA may present with acute bleeding, while bleeding is not typically seen with FNH. (See "Hepatocellular adenoma".)

- **Hepatocellular carcinoma (HCC)** – In contrast to patients with FNH, patients with HCC often have history of cirrhosis. The risk factors for and diagnosis of HCC are discussed in detail separately. (See "Clinical features and diagnosis of hepatocellular carcinoma" and "Surveillance for hepatocellular carcinoma in adults", section on 'High-risk groups'.)

- **Fibrolamellar carcinoma** – While more characteristic of FNH, a central scar may be present in the fibrolamellar variant of HCC. (See "Epidemiology, clinical manifestations, diagnosis, and treatment of fibrolamellar carcinoma", section on 'Imaging'.)

MANAGEMENT

General measures for all patients — We generally do not insist that oral contraceptives and other estrogen-containing preparations should be discontinued. However, it is reasonable to

obtain a follow-up imaging study in 6 to 12 months in women with FNH who continue taking these drugs. (See 'Possible risk factors' above.)

Asymptomatic lesions — We do not routinely obtain surveillance imaging for asymptomatic patients with FNH because of the low risk of lesion growth or complications. In a study including 30 patients with FNH (34 lesions) who were monitored with ultrasound, 33 lesions (97 percent) either remained stable or regressed in size during a mean follow-up of 42 months [48]. (See 'Prognosis' below.)

Symptomatic lesions — Symptoms such as abdominal pain are uncommon, but some patients with persistent pain attributed to FNH may require procedural intervention. Surgical resection may be performed, although less invasive approaches (eg, transarterial embolization, radiofrequency ablation) have also been used [49-54].

PROGNOSIS

The prognosis for patients with FNH is generally excellent because the lesion is most often stable or may regress over time [55,56], while complications (eg, bleeding) are very rarely reported [57,58]. Malignant transformation has not been reported.

PREGNANCY

Pregnancy is not contraindicated for asymptomatic female patients with FNH who wish to conceive, in addition, we do not routinely perform surveillance liver ultrasound during pregnancy [2,9,14,59].

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Focal liver lesions".)

SUMMARY AND RECOMMENDATIONS

- FNH is a benign liver lesion that is composed of hepatocytes surrounding a central stellate scar. Typically, FNH is a solitary lesion that is more commonly seen in women. (See 'Introduction' above.)

- FNH is a proliferation of hyperplastic hepatocytes that occurs in response to an aberrant dystrophic artery, to portal tract injury resulting in arterial to venous shunts, or to a congenital vascular malformation. The hepatic stellate cells respond to oxidative stress caused by arterial hyperperfusion, and this results in the characteristic fibrous scar found in the center of the lesion (ie, central scar. (See 'Pathogenesis' above.)

- The spectrum of presentation of FNH ranges from asymptomatic individuals with incidental findings on imaging to patients with nonspecific symptoms. When symptoms are present, the most commonly reported symptom is abdominal pain. (See 'Patterns of clinical presentation' above.)

- The diagnosis of FNH may be suspected in a patient without cirrhosis who is found to have a solid liver lesion on imaging. For patients with suspected FNH based on ultrasound or CT findings, the diagnosis is made with cross-sectional, contrast-enhanced, multiphase magnetic resonance imaging. (See 'Diagnostic approach' above.)

- We do not routinely obtain surveillance imaging to monitor asymptomatic patients with FNH because of the very low risk of lesion growth or complications. (See 'Asymptomatic lesions' above.)

- Symptoms such as abdominal pain are uncommon, but some patients with persistent abdominal pain may require procedural intervention. Surgical resection may be performed, although less invasive approaches (eg, transarterial embolization, radiofrequency ablation) have also been used. (See 'Symptomatic lesions' above.)

- The prognosis for patients with FNH is generally excellent because the lesion is most often stable or may regress over time, while complications (eg, bleeding) are very rarely reported. (See 'Prognosis' above.)

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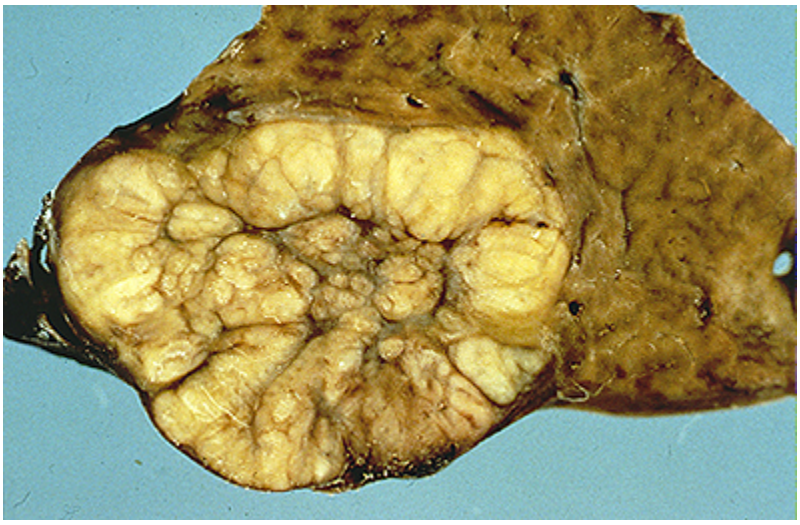
GRAPHICS

Focal nodular hyperplasia of the liver

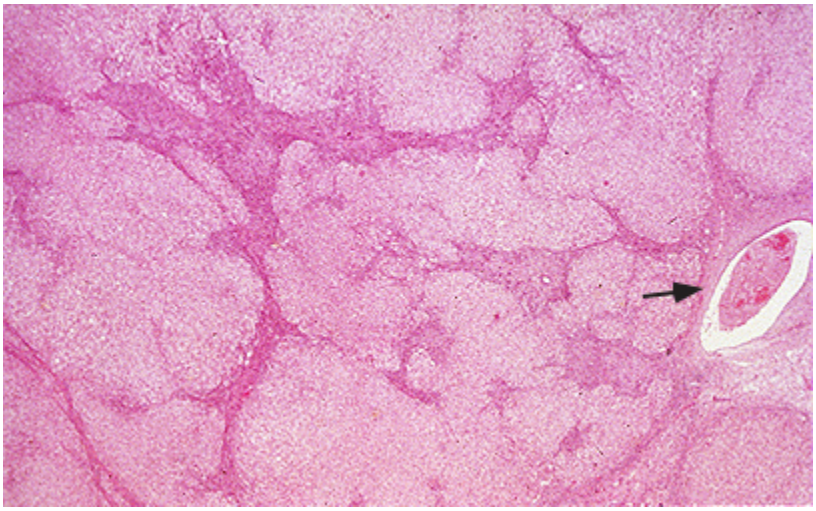


Surgical specimen showing a mass lesion within a noncirrhotic liver. Note the central stellate scar.

Courtesy of Frank A Mitros, MD.

Graphic 54209 Version 2.0

Focal nodular hyperplasia of the liver

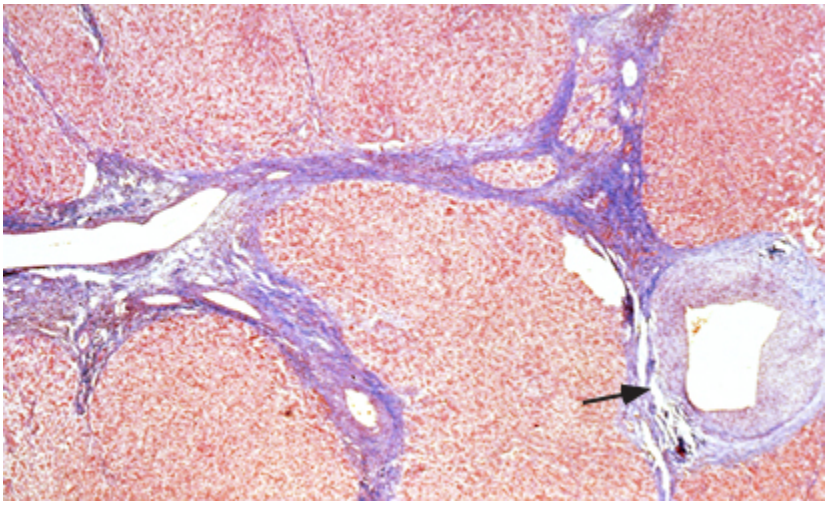


Biopsy specimen showing fibrosis closely mimicking cirrhosis. Note the large feeder artery typical of FNH (arrow).

Courtesy of Frank A Mitros, MD.

Graphic 66952 Version 2.0

Focal nodular hyperplasia of the liver

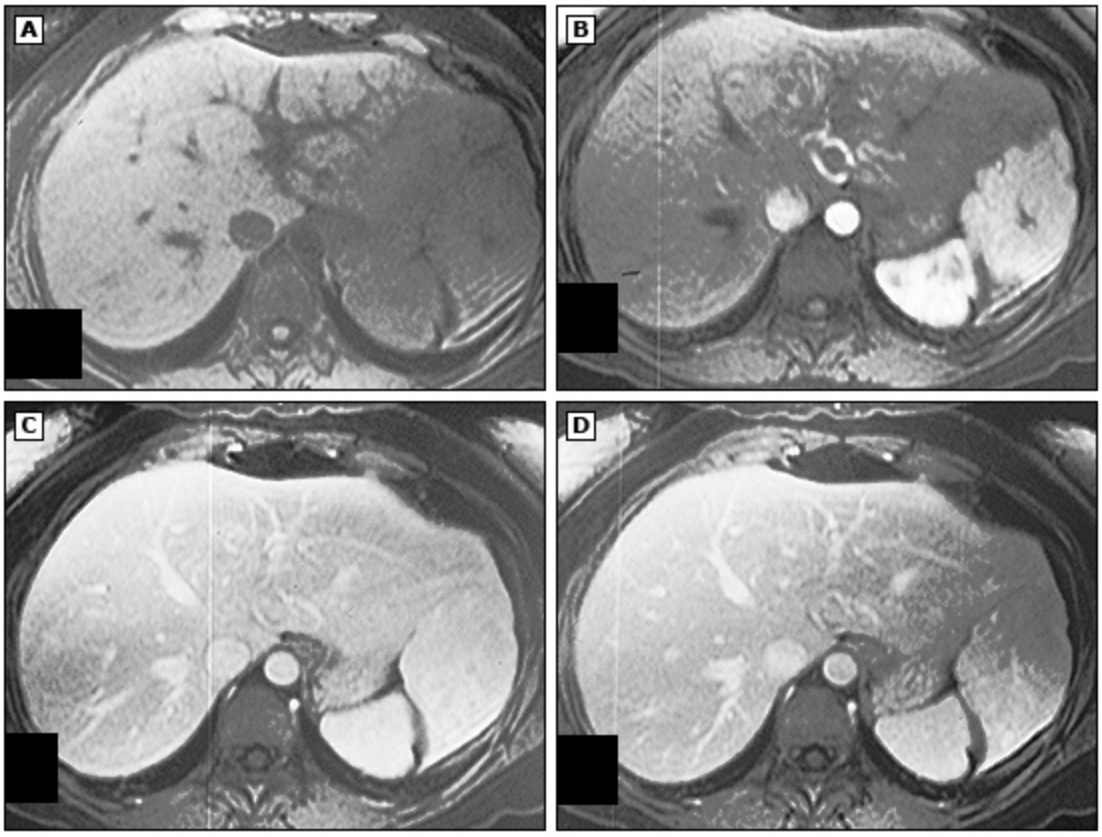


Biopsy specimen showing the fibrous septa and a large, thick-walled artery at the edge of the stellate scar (arrow). (Klatskin trichrome stain X 10).

Courtesy of Frank A Mitros, MD.

Graphic 52949 Version 3.0

Focal nodular hyperplasia of the liver seen on MRI

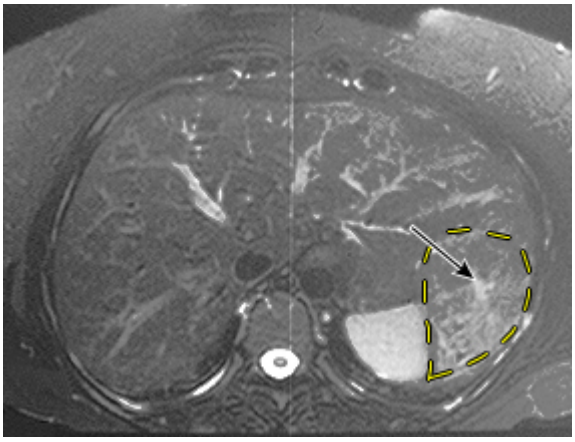


T1-weighted magnetic resonance imaging (MRI) pre-gadolinium shows a hypointense lesion in the left lobe of the liver (top left). Immediately after gadolinium infusion, the arterial phase reveals a hyperintense mass with a hypodense central scar, characteristic of focal nodular hyperplasia (top right). After two minutes, the mass appears nearly isodense with the normal liver, and the central scar is difficult to distinguish (bottom left). After three minutes, the central scar is hyperdense due to delayed accumulation of gadolinium (bottom right).

Courtesy of Douglas R LaBrecque, MD.

Graphic 51338 Version 5.0

Focal nodular hyperplasia of the liver seen on MRI



T2-weighted fat saturation magnetic resonance imaging (MRI) shows a hyperintense lesion in the left lobe of the liver (within dashed line) with a striking hyperintensive central scar (arrow).

Courtesy of Douglas R LaBrecque, MD.

Graphic 64152 Version 5.0

Contributor Disclosures

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