9 Focal Nodular Hyperplasia

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9.1 Introduction

Focal nodular hyperplasia (FNH) is the second most common benign hepatic tumor after hemangioma and has a prevalence that in different studies ranges from 1% to 3% (Karhunen 1986).

It can occur in both sexes but it is most frequently found in young and middle aged women during the third to fifth decade of life (Mathieu et al. 1997). Although a relationship between the use of oral contraceptives and FNH has never been proven, endogenous or exogenous estrogens play a role in the growth of the lesions by increasing the size of the nodule and inducing vascular change thanks to a trophic effect on FNH. Approximately 20% of the patients have multiple FNH lesions (Fig. 9.1) (Nguyen et al. 1999).

FNH is always asymptomatic, incidentally discovered during imaging studies performed for other reasons. When present, the clinical symptoms are due to the large diameter of the lesion which may expand the Glisson capsule and/or may compress adjacent structures (Fig. 9.2).

The absence of a malignant potential, the unlikely modifications of internal structure due to hemorrhage or necrosis and the reduction in size of these nodules, make it possible to conservatively manage FNH (Di Stasi et al. 1996). Only in symptomatic cases, surgical resection of the nodule can be considered.

9.2 Histopathological Findings

FNH is considered a hyperplastic response of the liver to a pre-existing arteriovenous malformation (Wanless et al. 1985). The arteries of FNH arise from the hepatic artery and the vein drains into the hepatic vein. Therefore, FNH does not contain portal vessels.

There are two types of FNH: typical (80%) and atypical (20%).

The gross appearance of typical FNH is characterized by lobulated contours and by nodules surrounded by radiating fibrous septa originating from a central scar that contains a large artery from which blood flows centrifugally to the periphery of the lesion. Histologically, the typical FNH is characterized by a proliferation of hepatocytes, Kupffer cells, bile ductules and blood vessels arranged in abnormal pattern.

Differently from typical FNH which is a lobulated, well-circumscribed solid, hypervascular mass, with a central scar and peripheral fibrous septa, atypical FNH is more heterogeneous and the central scar is always absent at gross appearance.

Both typical and atypical FNH contains hepatocytes and Kupffer cells; however, in atypical FNH there is only bile ductular proliferation with a lack of malformed vessels or nodular architecture that are always present in typical FNH. Wanless et al. (1989) and Nguyen et al. (1999) reported the pathologic features of a form of atypical FNH, called telangiectatic FNH. According to these authors, there are two differences between classic and telangiectatic FNH:
Fig. 9.1a–d. Multiple focal nodular hyperplasia. a Arterial phase image acquired after Gd-BOPTA bolus injection shows strong hypervascularization of all lesions (arrows) and a hypointense central scar in the biggest of them. b In the portal venous phase the lesions are slightly hyperintense. c,d T1-weighted images acquired during the hepatobiliary phase show enhancement of the liver lesions which appear slightly hyperintense with a clear evidence of hypointense central scar in all lesions.

Fig. 9.2a–b. Chronic right upper abdominal pain in a 25 year-old woman. a Conventional transverse US scan through the lateral segment of the left hepatic lobe shows a 10-cm hypoechoic solid lesion. A subtle hyperechoic central scar is seen (arrows). b Power Doppler US scan shows the central stellate aspect of the scar. c Contrast-enhanced US scan with low MI pulse inversion and a second generation contrast agent: in the arterial phase the macrocirculation of the FNH is very well depicted. d The portal phase scan highlights the microcirculation of the lesion that enhances homogeneously apart from the central scan which is hypoechoic. e Non-enhanced CT scan shows a homogeneously hypodense lesion. f Enhanced dynamic CT scans reveal intense and homogeneous enhancement during the arterial phase.