Focal Nodular Hyperplasia of the Liver with Angioma-like Features in the Center

Key words: liver, FNH, hemangioma, histopathology

A 73-year-old woman was incidentally found to have a hypervascular nodule in the liver (S5) with imaging. She was subsequently followed under a diagnosis of suspected hepatic hemangioma because of continued enhancement in portal venous phase CT scan (Fig. 1A). Liver function test and tumor marker were within normal limits. Both HBs antigen and HCV antibody were negative. She had no history of taking oral contraceptives. Two years after the initial detection, the lesion appeared to be slightly enlarged, but imaging and biopsy studies failed to establish the diagnosis, and to exclude malignant tumors such as hepatocellular carcinoma. Thus, hepatic subsegmentectomy was performed. Grossly, the resected lesion was a hepatic subcapsular, well-demarcated, light yellow-whitish, solid nodule, 2 cm in diameter, with a central, dark-red, congestive area (Fig. 1B). Histologically, the nodular lesion showed mild hyperplasia of liver cells in contrast to the surrounding almost normal liver parenchyma; it contained fibrotic change with hyperplasia of arterioles, vessels, and cholangioles (Fig. 1C). The central congestive area contained abundant angioma-like, CD34-positive small vascular spaces (Fig. 1D). These findings led to a diagnosis of focal nodular hyperplasia (FNH) of the liver with angioma-like features.

Figure 1. (A) Abdominal CT findings (portal venous phase). (B) Gross findings of the liver nodule. (C) Histological findings (HE stain, ×200). Compatible with focal nodular hyperplasia (FNH) of the liver. (D) Angioma-like features in the center (CD34 immunostain, ×100).
The lesion of our case had no pathologically evident central scar often seen in FNH, but was morphologically characterized by angioma-like features in the center. Since the contrast medium was pooled at this site and was not washed out, the lesion showed continued enhancement instead of isodensity on the portal venous phase scan, which is a general finding on CT in the FNH (1). According to Nguyen et al, there are two major differences in histologic features between classical and telangiectatic FNH (2). We considered the condition of this case to be classical FNH, because neither arteries with hypertrophied muscular media nor abnormal vessels connecting with sinusoids, which are characteristic of telangiectatic FNH, were observed.

Various theories have been proposed as to the pathogenesis of FNH, and none has been established. As one of them, particularly in Western countries, the oral contraceptive theory is frequently indicated such as reports on the spontaneous regression of FNH with the discontinuation of oral contraceptives and the use of oral contraceptives for median 10 years by 70.8% (92/130) of women with FNH (2). On the other hand, FNH associated with hemangioma has been found by imaging frequently, in 6 of 26 cases (23.1%) (3). However, a histopathological analysis of many cases found FNH with hemangioma in only 3 of 130 cases (2.3%) (4), and a recent study reported that only 2 of 168 patients (1.2%) had FNH immediately adjacent to hemangiomas (2). Ndimbie et al first reported a hemangioma surrounded by focal hyperplasia of liver cells (5). Like the present case, FNH containing a wide, hemangioma-like area in center is thought to be rare. The possible mechanisms of pathogenesis of the FNH reported here are: 1) that increased blood flow in the abundant, small, angioma-like vascular spaces resulted in secondary hyperplasia of the surrounding liver cells, and 2) that many abnormal vessels caused ischemic injury to the local liver parenchyma, leading to compensatory regeneration of the surrounding liver cells. In summary, we reported a case of FNH of the liver in which the peculiar histopathological features, involving angioma-like vascular abnormalities, suggested an influence on its histogenesis.

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