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Case Report

A young man with an atypical liver mass: Focal nodular hyperplasia

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Abstract

With the increase in imaging methods, incidence of incidental masses has been increased. Focal nodular hyperplasia is the most common benign liver mass after hepatic adenomas and is often diagnosed with typical images in contrast enhanced sectional imaging methods. In atypical imaging, liver biopsy may be required for a definitive diagnosis. We present an atypical case that we diagnosed as focal nodular hyperplasia with clinical decision, although it does not show any typical radiologic and pathologic findings.

Keywords: Benign liver tumors; focal nodular hyperplasia; diagnosis

INTRODUCTION

Focal nodular hyperplasia (FNH) is the second most common benign tumor of the liver after hepatic hemangiomas (1). It is usually asymptomatic and discovered incidentally. It is mostly seen as a solitary mass (80%) smaller than 5 cm and 80% of cases is seen in women of reproductive age (2). Although oral contraceptive use has been assumed in the etiology, studies on the prevalence of these tumors in young women have shown that FNH is not hormonal dependent or affected by oral contraceptives or pregnancy (3).

Most of FNHs are asymptomatic and detected incidentally in an imaging process, however 25% of patients may be symtomatic and represent with epigastric pain, abdominal fulness, early satiety, or jaundice (1,4). All laboratory tests, except gamma glutamyl transpeptidase (may elevate in 50% of cases), including alpha-fetoprotein (AFP) and tumor markers are normal in FNH (1). These masses are mostly diagnosed with their typical features in contrasted enhaced sectional imaging modalities. In cases where there are no typical findings in advanced imagings, pathological diagnosis may be required (5).

With increasing access to imaging methods in recent years, liver benign lesions, which are actually quite rare, are frequently encountered. We present a young to middle-aged male case who did not show typical clinical, radiological and patholocical features of a FNH.

CASE REPORT

A 31-year-old man who had no known disease or drug usage during past 3 months, had an alanine aminotransferase (ALT) value of 52 U/L (normal range; 0-50 U/L) in blood tests performed for checkup. Patient's hepatitis B, C and all other laboratory tests were normal. Hepatobiliary ultrasonography (USG) showed an isoechoic solid lesion on liver segment 4A with an approximate size of 73x68 mm and magnetic resonance imaging (MRI) was performed to understand the nature of the mass.

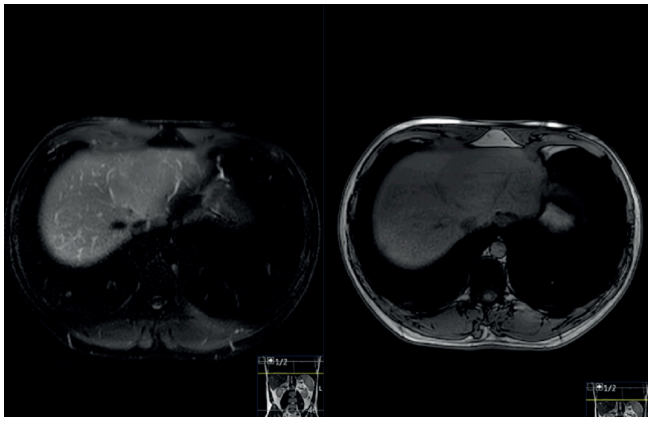

Figure 1. T1-T2A sequences of MRI show isoechoic and mild hyperintense lesion with a size of 73x76 mm in the liver segment 4A-8

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On MRI, the patient had an isoechoic and mild hyperintense lesion in liver segment 4A-8 localization with a size of 73x76 mm in T1-T2A sequences (Figure 1). After intravenous contrast material, the lesion showed diffuse contrast enhancement in the early arterial phase, the contrast enhancement decreased towards the late phase and only capsular enhancement persisted. The lesion with its irregular lobulated contour, which also showed significant diffusion restriction, was primarily evaluated as hepatocellular carcinoma (HCC) (Figure 2).

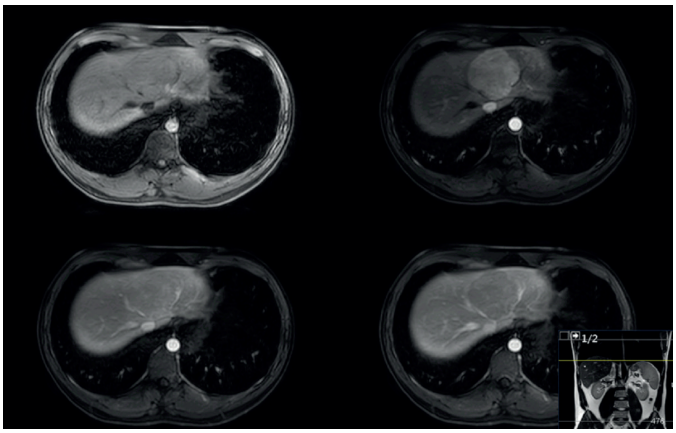

Figure 2. The contrast MRI images show a mass with diffuse contrast enhancement in the early arterial phase and capsular enhancement in the late phase, which also have significant diffusion restriction

Although the AFP value was normal and the patient had no clinical findings, the patient underwent liver biopsy with the decision of the gastroenterology council due to the strong malignant evaluation of the radiological image. Positron emission tomography (PET) showed a weakly hypodense, approximately 70 mm sized area with normal fluorodeoxyglucose (FDG) uptake similar to liver parenchyma while waiting for the pathology result (Figure 3). Pathological evaluation revealed liver parenchyma with fibrous septa. Within this septa, several vessels were seen (asterisks). Although there were ductules near the liver parenchyma (arrows), no true bile ducts adjacent to arteries were seen (Figure 4).

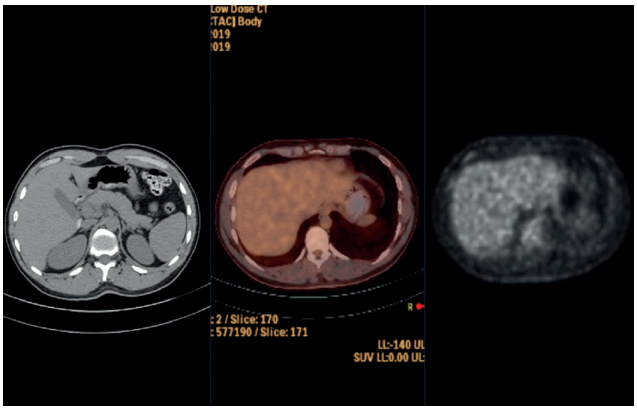

Figure 3. PET images show a weakly hypodense, approximately 70 mm sized area with normal FDG uptake similar to liver parenchyma

Contrary to what is seen in HCC, liver cell plates which were devoid of CD34 and Glypican-3 immunostains were one or two cells thickness. After laboratory, clinical, radiological and pathological evaluation, the patient was diagnosed as FNH and discharged to be followed from the outpatient clinic.

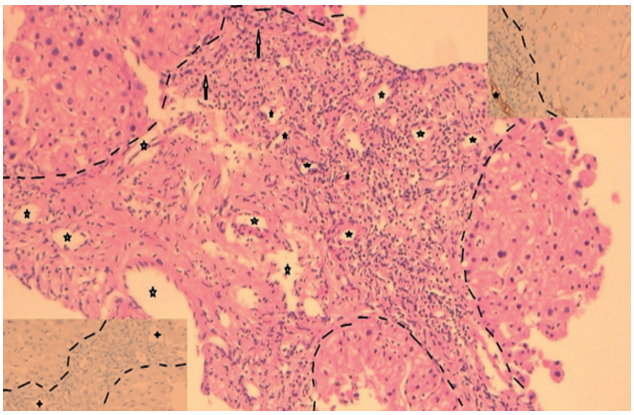

Figure 4. Portal space-like fibrous area was circled with liver parenchyma. Note that there were several vascular spaces (asterisks) without true bile duct. Two proliferated ductuli were seen at the upper left (arrows) (H&E,x10). Please also note that this area had a stellate shape. Inset on upper right highlighted vascular endothelium as brown color (asterisks). Liver parenchyma on the right would have been positive if it was a HCC (CD34 antibody, x10). Inset on bottom left showed negative immunostain with Glypican-3, which is a HCC marker (Glypican antibody, x10)

DISCUSSION

The prevalence of FNH is approximately 0.9% (1). Hemangioma, hepatic adenoma (HA), HCC, fibrolamellar carcinoma, regenerative nodule, and metastases should be considered in the differential diagnosis of liver lesions besides FNH. HAs are the most common liver mass in differential diagnosis with FNH, as they affect patients of similar age and gender. Although it is easy to distinguish a classical mass, despite advances in radiological imaging techniques, radiological methods may sometimes fail to diagnose a non-classical mass as in our case. Hepatic percutaneous biopsy also has low diagnostic sensitivity (60%–82%) and even pathology can be challenged in clear discrimination of these lesions (1).

The role of radiological examinations is very important in the diagnosis of benign lesions of the liver. On USG, FNH is usually seen as a hypoechoic or isoechoic mass with displacement of vessels and a lobulated outline. A central scar may be detected as a thin hyperechoic zone in 20% of cases (1). MRI has a high sensitivity for characterization and the detection of FNH. It shows near-isointensity on noncontrast T1 and T2 weighted images, dense arterial enhancement of cotton-wool type, uniform fading on venous phase images, and late enhancement of a central scar (6). Although USG and MRI echo patterns were similar to FNH in this case, the lesion was interpreted in favor of HCC because of lack of central scarring in both imaging modalities and the presence of diffusion restriction after contrast-enhanced MRI.

The pathogenesis of FNH is not well characterized; it is thought to occur as a result of a hyperplasic response to a regenerative non-neoplasic nodule caused by a congenital vascular malformation. Histological features include a central stellar scar of a connective tissue with a large arterial vessel and a septum. In a typical FNH, a fibrotic central zone composed of connective tissue, proliferating cells, altered biliary structures, perilesional inflammatory cells and numerous vessels can be observed in pathological examination (1). However, 20% of all FNHs do not show typical pathological features and called as non-classic FNH. These types has no blood vessels and always has biliary duct proliferation in the nodule. Central scars are rarely observed and the appearance of non-classic FNH is more heterogeneous than classic FNH (1). Our case had no central scar in imaging modalities, but histology revealed minute focus of typical vascular proliferations without bile duct in a portal space-like fibrous area.

When FNH is diagnosed, patients usually undergo a regular follow-up schedule (3). Any change in size, any symptoms or complications associated with FNH such as abdominal pain, obstructive jaundice, tumor rupture, or bleeding are conditions that may require surgical resection (1,3). Depending on the follow-up duration, the size of the lesion may increase or decrease. If feeding artery of lesion was gradually compressed between the enlarging lesion and normal hepatic parenchyma, an apoptosis process may be triggered due to ischemia and this may lead to decrease of size (7).

CONCLUSION

In conclusion, perhaps many liver masses, which will no longer show symptoms throughout life, will often appear due to increased hospital accessibility and imaging methods. In this process, we should be prepared to encounter liver masses that do not show typical characteristics, and clinical decision may be the most important factor besides laboratory, radiology and pathology.

Conflict of interest: The authors declare that they have no competing interest.
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