Case 5528

Hepatic Adenoma (HA) and Focal Nodular Hyperplasia (FNH)

Published on 17.01.2007

DOI: 10.1594/EURORAD/CASE.5528
ISSN: 1563-4086
Section: Abdominal imaging
Case Type: Clinical Cases
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Patient: 30 years, female

Clinical History:

Use of oral contraceptives for 10 years. For five months the patient had slight upper right abdominal pain and therefore underwent ultrasound examination which showed a huge hypoechoic nodule in the right lobe of the liver.

Imaging Findings:

Use of oral contraceptives for 10 years. For five months the patient had slight upper right abdominal pain and therefore underwent ultrasound examination which showed a huge hypoechoic nodule in the right lobe of the liver. CT and MR examinations were performed, and two contiguous nodules with different enhancement patterns were found. The final diagnosis was hepatic adenoma and focal nodular hyperplasia.

Discussion:

Hepatocellular adenoma (HA) is a rare benign tumor of hepatocellular origin and most commonly seen in middle age women. Estrogen- or androgen-containing steroid medication increases the prevalence, number, and size, while withdrawal of estrogen derivates may result in regression of the HA. There is an association with congenital or acquired abnormalities of the hepatic vasculature, i.e. portal vein absence or occlusion of portal venous shunts, particularly in patients with liver adenomatosis (LA). Focal nodular hyperplasia (FNH) is a benign tumorlike lesion of the liver considered to be the result of a hyperplastic response of hepatocytes to the presence of a preexisting vascular malformation. It is thought that locally increased arterial flow of the parenchyma induces a secondary hepatocellular hyperplasia. FNH is usually seen in women of childbearing age. Oral contraceptives are not causative, however, estrogens could have a trophic effect on FNH by increasing the size of the nodule and contributing to the vascular changes. The typical HA is composed of hepatocytes arranged in cords that occasionally form bile. It lacks portal tracts and terminal hepatic veins. Kupffer cells are often found but can be small in number. Bile ductules are notably absent, a key histologic feature that helps to distinguish HA from FNH. On US examination, HA can appear as a mixed echogenic lesion, mainly hypoechoic, FNH as a homogeneous hypo-, iso- or slightly hyperechoic nodule. In the present case the two nodules have the same echogenicity and appear as a single one (Fig.1). On unenhanced CT scans, HA and FNH may appear as iso- or hypodense (Fig.2). In the arterial phase of contrast-enhanced CT, HA may enhance homogeneously; FNH enhances rapidly and becomes hyperdense relative to normal liver (Fig.3). In the portal venous phase HA appears slightly hyper- or isodense, the difference in attenuation between FNH and normal liver decreases, and FNH becomes iso- or slightly hypodense (Fig.4). On precontrast T2-w MRI, HA is slightly hyperintense and is FNH isointense compared to the liver (Fig.5), both nodules are isointense on T1-w images (Fig.6). Dynamic MRI is able to demonstrate in HA the early arterial enhancement, FNH is characterized by marked and homogeneous enhancement (Fig.7). On the portal venous and equilibrium phase HA generally appears isointense or slightly hyperintense, FNH demonstrates wash-out and appears isointense (Fig.8). On delayed liver specific phase images after Gd-BOPTA administration, HA is hypointense, which is one on the main features in differentiating HA from FNH. The hypointensity reflects the lack of biliary ducts. This
enhancement pattern of Gd-BOPTA is opposite to FNH which appers as an iso- or hyperintense lesion (Fig.9). After Mn-DPDP HA is iso- or slightly hyperintense, similar to FNH; this feature limits the capability to make a correct differential diagnosis (Fig.10). After SPIO administration the accumulation depends on the amount of Kupffer cells in the tumor as well as on the boundary of the lesion. Some HA may take up SPIO, resulting in a decreased signal on T2-w images. However the uptake of SPIO in HA is usually relatively poor compared to FNH (Fig.11).

**Differential Diagnosis List:** HEPATIC ADENOMA (HA) AND FOCAL NODULAR HYPERPLASIA (FNH)

**Final Diagnosis:** HEPATIC ADENOMA (HA) AND FOCAL NODULAR HYPERPLASIA (FNH)

**References:**


Fig. 1: US reveals a huge homogenous hypoechoic lesion.
Figure 2

Description: Fig.2: On precontrast CT, a hypoechoic nodule is visible. Origin:
Figure 3

Description: Fig.3: On post contrast CT, in the arterial phase, FNH shows a discrete and homogenous enhancement, HA doesn’t show a significant enhancement compared to the surrounding parenchyma.

Origin:
Figure 4

**Description:** Fig. 4: In the portal venous phase FNH appears homogeneously and slightly hypodense, HA is isodense to the liver. **Origin:**
Description: Fig. 5: On precontrast T2 weighted image, HA appears homogenously hyperintense, and FNH homogenously isointense to the liver. Origin:
Figure 6

Description: Fig.6: On precontrast T1 weighted image, HA and FNH appear homogenously isointense to the liver. Origin:
Figure 7

Description: Fig.7: In the arterial phase after Gd-BOPTA administration, HA shows a heterogenous enhancement, conversely FNH appears markedly homogeneous and hyperintense compared to the normal liver. Origin:
Description: Fig. 8: In the equilibrium phase FNH appears isointense, while HA is slightly hyperintense.
Origin:
Description: Fig.9: In the hepatobiliary phase after Gd-BOPTA administration, HA is hypointense and FNH hyperintense to the liver. Origin:
Description: Fig. 10: In the hepatobiliary phase after Mn-DPDP administration, both nodules appear isointense to the liver. Origin:
Figure 11

Description: Fig.11: In the reticuloendothelial phase after SPIO administration, both nodules show a significant signal drop, which, however, is more evident in the HA. Origin: