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Review

Primary benign liver lesions

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ABSTRACT

Benign focal liver lesions can origin from all kind of liver cells: hepatocytes, mesenchymal and cholangiocellular line. Their features at imaging may sometimes pose difficulties in differential diagnosis with malignant primary and secondary lesions. In particular, the use of MDCT and MRI with extracellular and hepatobiliary Contrast Agents may non invasively help in correct interpretation and definition of hepatocellular or mesenchymal and inflammatory nature, allowing to choose the best treatment option. The peculiarities of main benign liver lesions at US, CT and MRI are described, with special attention to differential diagnosis and diagnostic clues.

1. Hepatocellular origin

1.1. Hepatocellular adenoma

Hepatocellular adenoma (HCA) is a rare benign liver lesion, with an incidence of 1 case for 1,000,000 people: the incidence increases to 1–3 cases for 100,000 in females which use or have used oral contraceptive for long-term [1]. Although the precise pathogenic mechanism leading to hepatic adenomas is still unknown, the use of oral contraceptive or anabolic steroids, some congenital diseases such as glycogen storage diseases, but also related metabolic syndrome manifestations such as diabetes mellitus, insulin resistance, dyslipidemia and obesity are considered as risk factors for development and progression of HCA. Men with metabolic syndrome are at a much higher risk (10 times more likely than females) for malignant degeneration of liver adenomas (anyway rare: <5%). Other risk factors for degeneration are: androgen use, large tumors (>5 cm) and histological subtype (β-catenin-mutated) [2,3]. More than ten adenomas widespread into liver parenchyma configure "adenomatosis". HCA can be classified in four histological types [2,4,5]: 1) hepatocyte-nuclear-factor-1α mutated type (H-HCA): they represent 25–45% of adenomas, characterized by predominant intralesional fat component; 2) β-catenin-mutated type with upregulation of glutamine synthetase (β-HCA): approximately 5–10% of adenomas. They are considered borderline lesions between HCA and HCC β-Catenin–mutated, occur more frequently in men and are associated with male hormone administration, glycogen storage disease, and familial adenomatosis polyposis; 3) inflammatory type (IHCA) with serum-amyloid-A overexpression: they represent 45–55% of adenomas, initially described as telangiectatic FNH, characterized by inflammatory infiltrates and frequent sinusoidal dilatation (intratumoral steatosis can be found); 4) unclassified type: <5–10% of cases [6–8].

Small HCAs (<5 cm) are generally asymptomatic; large lesions (6–30 cm) can determine right upper discomfort or pain due to liver capsule strain; acute and dangerous outset is possible if a large peripheral or exophitic HCA breaks and bleeds into abdominal cavity [2].

It is important to correctly characterize HCAs and possibly their subtype, because of the different therapeutic options: liver biopsy is the gold standard but it represents an invasive procedure, not devoid of risks. Imaging techniques (ultrasound, CT, MR) can rightly define different HCAs in a good part of cases because they show different characteristics at basal acquisitions and different patterns of enhancement after contrast media administration, reflecting their histological subtype.

On ultrasound examination H-HCAs typically appear as very homogeneous hyperechoic lesions because of marked and diffuse fat within the lesions; rare signal can be detected at color-Doppler examination [4]. On non-enhanced CT they are strongly hypo attenuating. MR plays a conclusive role in characterizing H-HCAs demonstrating the presence of intralesional fat: they show, in fact, homogeneous and in-

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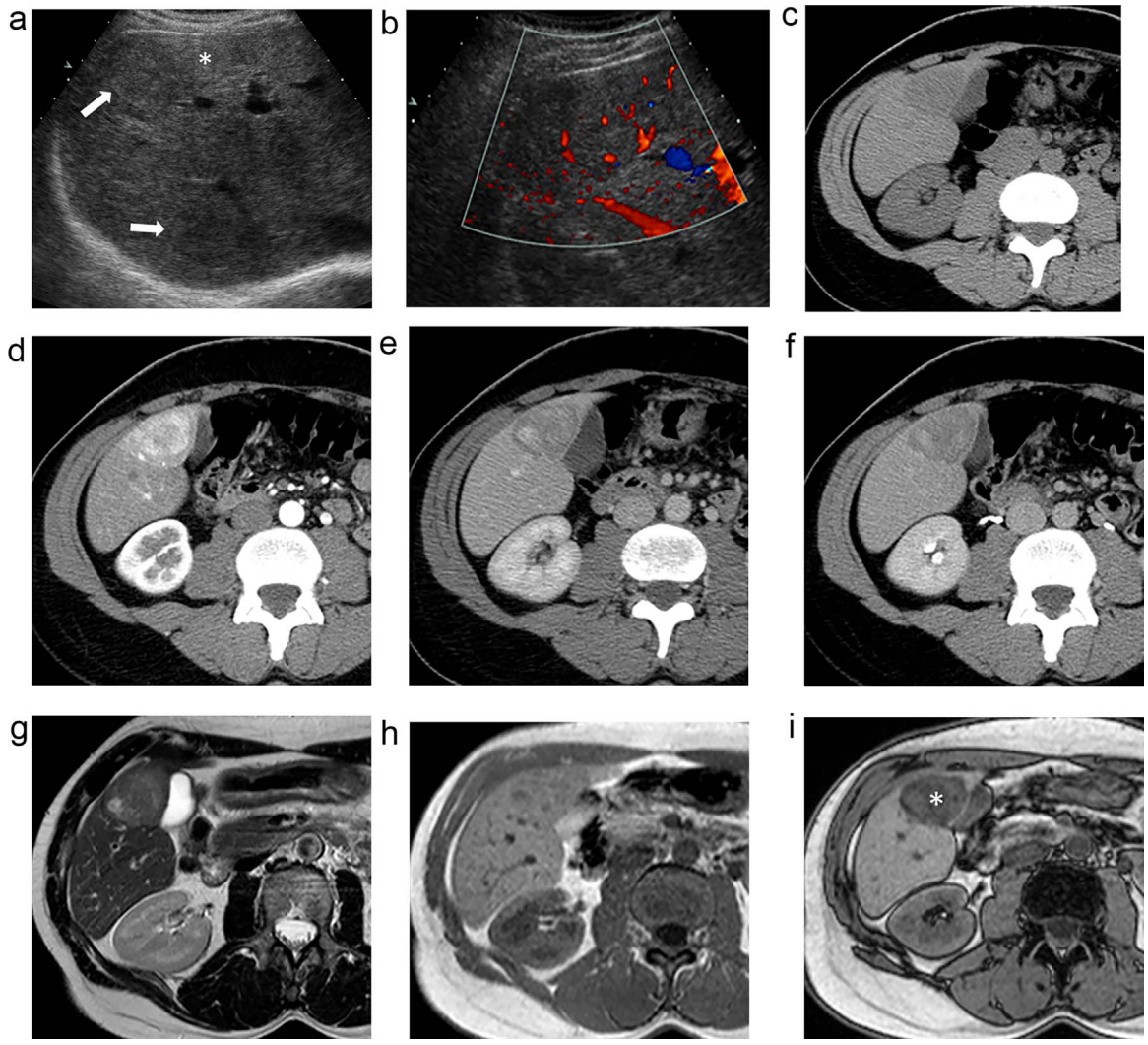

Fig. 1. 45 years old female, asymptomatic; incidental finding on US examination (a) of multiple isohypoechoic nodules (* and arrows), vascularized at color-Doppler evaluation (b). At NECT (c) a big nodule in Vs appears hypodense. During CECT it shows strong disomogeneous enhancement during arterial phase (d) with rapid wash-out on PVP (e); evidence of thin hyperdense pseudocapsule on delayed phase (f). At MR examination the lesion in Vs is slighltly hyperintense on T2 w images (g); it appears isointense on T1 in-phase acquisition (h) and shows intense drop of signal on T1 opp-phase acquisition (* in i) because of intralesional steatosis. No signs of restriction of the signal at DWI study (b: 50–400-800) (j-l). The lesion appears hypointense on T1-w with fat saturation (m) and shows disomogeneous enhancement after injection of gadoxetate disodium during arterial phase (n). The nodule has rapid wash-out and appears disomogenously hypointense in portal and delayed phases (o,p). On HPB, 15 min after contrast medium injection (q) the lesion is markedly hypointense: hepatocyte-nuclear-factor-1α mutated adenoma ("steatotic" adenoma) at biopsy.

tense signal dropout on opposed-phaseT1-weighted sequences [5,9]. Hyper intensity on T1 images can be seen if glycogen component is present (or less commonly haemorrhage) [4,5,10]. On T2w images H-HCAs appear generally iso- or hypo intense without significant restriction on DWI [11]. At real-time CEUS, on CECT and after hepatobiliary Gd-chelates administration they show variable grade of enhancement during arterial phase, in same cases not very intense, and rapid wash-out during portal and late dynamic phases. During hepatobiliary phase they appear generally homogeneously hypo intense [9,11] (Fig. 1).

I-HCAs appear as well-delineated, often hyperechoic and heterogeneous nodules on ultrasound. Doppler signals are commonly seen and may mimic central arteries [1,9]. On CT HCAs are heterogeneously hypo attenuating and may contain spontaneously hyper attenuating areas in relation to recent intralesional bleeding [5,9]. At real-time CEUS they show rapid centripetal filling in the arterial phase and persistent peripheral rim enhancement with central wash-out during portal and late phases. On CECT their characteristic pattern is strong arterial enhancement and a persistent enhancement in the delayed phase [4,5,9]. On MR, HCAs show discrete hyper intense signal on T2-weighted images and iso to hyper intense signal on T1-weighted sequences with and without fat suppression. Some lesions may contain small amount of fat, visible as signal dropout opposed-phaseT1-weighted sequences [4,5,10]. Most I-HCAs show diffusion restriction on DWI [11]. After hepatobiliary Gd-chelates administration the pattern of enhancement is similar as on CECT, with arterial enhancement which

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