Hepatic focal nodular hyperplasia: contrast-enhanced ultrasound findings with emphasis on lesion size, depth and liver echogenicity

Poster No.: C-1464
Congress: ECR 2011
Type: Scientific Paper
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Keywords: Ultrasound, Contrast agent-intravenous, Liver
DOI: 10.1594/ecr2011/C-1464

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Focal nodular hyperplasia (FNH) is the second most common benign tumour of the liver after haemangioma, accounting for approximately 8% of all primary hepatic tumours and it is being increasingly discovered, mostly in young women, because of the widespread use of cross-sectional imaging, in particular abdominal ultrasound [1,2]. Differentiation of FNH from other focal liver lesions is of clinical relevance, as surgery is not recommended for asymptomatic patients. Grey-scale ultrasound (US) is not a specific technique in the diagnosis of FNH, because of the lack of a peculiar echo pattern. In large FNHs colour, power and pulsed Doppler US (US/CD) may show a characteristic spoke-wheel arterial pattern of vessels, thus providing further clues to the diagnosis [3]. Nevertheless, Doppler examination may be unsatisfactory because of motion artefacts, or when small or deeply located lesions are evaluated.

The introduction of contrast-enhanced ultrasound (CEUS) represents a significant breakthrough in liver ultrasound [4,5]. FNH is a highly hypervascular tumour and it is predicted at CEUS on the basis of arterial phase centrifugal filling and stellate vascularity [6]. The presence of a hypoechoic non-enhancing central scar and an arterial feeding vessel are other features suggestive of FNH on CEUS [4]. Hence, the aim of this study was to assess the role of CEUS in detecting the spoke-wheel sign, central scar and feeding vessel in FNH and to correlate CEUS findings with lesion size, depth and liver echogenicity.
Methods and Materials

Two radiologists, blinded to the final diagnosis, evaluated baseline US and CEUS examinations of 92 FNHs (mean size: 3.1±1.7 cm) in 71 patients (59 women and 12 men, mean age: 38.9 years), for detecting the spoke-wheel sign, central scar and feeding vessel.

US and CEUS examinations were performed by means of either an HDI 5000 (ATL, Bothell, WA, USA) (n = 35) or an iU22 unit (Philips Ultrasound, Bothell, WA, USA) (n = 57), both of them provided with C5-2/C5-1 convex array probes and Pulse Inversion imaging software. A baseline survey examination, including a colour/power Doppler (CD/PD) and spectral analysis, was performed. Once set, the US imaging parameters - such as the focal zone and time gain compensation - were not changed throughout the study. The US contrast agent used in the present study was SonoVue (Bracco, Milan Italy), which was injected intravenously as a 2.4 mL bolus followed by 10 mL of normal sterile saline flush by using a 20- or 22-gauge peripheral intravenous cannula. A low frame-rate (5 Hz) and a very low mechanical index (MI), ranging from 0.05 to 0.08, were used for real-time imaging. One focus was positioned below the level of the lesion. Each examination lasted about 5 minutes after bolus injection.

Digital cineloops were registered during both baseline and post-contrast US in the arterial, portal venous, and extended portal venous or late phase - (i.e. 5-40 s, 55-90 s and until 200-300 s from the beginning of injection respectively).

The FNHs were grouped and analyzed by dimension (a. # 3 cm; b. < 3 cm), depth (a. equal to or less than 5 cm, b. 5.1 cm to 10 cm, c. more than 10 cm) and liver echogenicity (a. echogenicity increased more than the renal cortex; b. poor visualisation of the portal veins or the posterior portion of the right lobe and c. non-visualisation of the right lobe and the portal veins) [7].

The final diagnosis was obtained by core-biopsy performed with an 18 G needle (n = 1) and/or typical helical CT (n = 30), MRI findings (n = 56) or both (n = 6).
Results

After contrast agent injection, 91 out of 92 (98.9%) FNHs showed hyperenhancement to various degrees in comparison with adjacent liver parenchyma in the arterial phase. In the portal-venous and late phases all these 91 FNHs were either isoenhancing (n = 66) or slightly hyperenhancing (n = 25) in comparison with surrounding liver parenchyma. One FNH (sized 3.6 cm and located in the subcapsular region of segment VII in a "bright" echogenic liver) remained hypoenhanced throughout the whole vascular phase.

At least one sign - among spoke-wheel, central scar, and/or feeding vessel - could be detected at CEUS in 44 out of 92 (47.8%) FNHs (mean size 3.9 cm ± 1.8 cm), whereas 48 of 92 (52.2%) FNHs (mean size 2.4 cm ± 1.3 cm) showed none of these signs at CEUS (p < 0.0001). A spoke-wheel sign, a central scar, and/or a feeding vessel could be detected at CEUS in 27 out of 36 (75%) FNHs larger than 3 cm (Fig. 1a on page , 1b on page , 1c on page , 1d on page ) and in 17 out of 56 (30%) FNH measuring 3 cm or less (Fig. 2a on page , 2b on page , 2c on page , 2d on page ) (p < 0.0001).

No statistically significant differences were noted between lesion depth or liver echogenicity and detection rate of these sign at CEUS (p>0.05) as well as between CEUS or baseline US/CD with regards of lesion size, depth or liver echogenicity (p>0.05).
Conclusion

In conclusion, our results show that the detection rate of the central scar and spoke-wheel sign in FNH at CEUS is strongly dependent on lesion size and CEUS can confidently diagnose most FNHs larger than 3 cm. On the contrary, small FNHs (< 3 cm) may not show those signs at CEUS, but in the appropriate clinical setting, a specific diagnosis may equally be achieved by combining colour Doppler and CEUS findings. Eventually, MR with hepato-specific contrast agent may provide a reliable tool for the ultimate characterisation of those FNH still undiagnosed at CEUS.
References
