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Original contribution

Inflammatory hepatic adenomas: Characterization with hepatobiliary MRI contrast agents

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ABSTRACT

Purpose: To characterize the MRI appearance of inflammatory hepatic adenomas using hepatobiliary contrast agents.

Materials and methods: MRI was performed using hepatobiliary contrast agents (3 with gadobenate dimeglumine and 24 with gadoxetic acid) in 27 patients with immunohistochemistry-confirmed diagnosis of inflammatory hepatic adenoma. The appearance of the lesions on T2 and diffusion-weighted images, pre-gadolinium T1-weighted images, dynamic post-gadolinium images, and hepatobiliary phase images was assessed.

Results: Seven lesions (26%) showed predominant hyperenhancement on hepatobiliary phase images in comparison with adjacent hepatic parenchyma: 1 lesion showed diffuse, mildly heterogeneous hyperenhancement, and the remaining 6 lesions showed peripheral hyperenhancement and central hypoenhancement. Twenty lesions (74%) were predominantly hypoenhancing compared to adjacent liver on hepatobiliary phase images. Nine lesions showed a pattern of peripheral hyperenhancement and central hypoenhancement on hepatobiliary phase images; in 6 of these lesions a majority of the mass appeared hyperenhancing, while the remaining 3 lesions showed predominant hypoenhancement.

Conclusions: This investigation shows that a significant percentage of inflammatory hepatic adenomas appear isointense or hyperintense in comparison to adjacent normal liver on hepatobiliary phase images, and therefore this feature should not be used to distinguish hepatic adenomas from focal nodular hyperplasia without additional supporting evidence.

1. Introduction

Hepatic adenomas (HAs) are relatively uncommon benign lesions that typically occur in young women and are often associated with oral contraceptive use. HAs can also occur in men, usually in association with anabolic steroid use, and in patients with glycogen storage disease [1–4]. Although most HAs occur as solitary lesions, multiple lesions can occur, and the presence of > 10 lesions in a single patient is termed hepatic adenomatosis.

Most HAs are asymptomatic and are discovered incidentally during cross-sectional imaging; however, complications occur in a small subset of patients, and the risk of hemorrhage and rupture as well as the risk of malignant transformation distinguish HAs from the more common benign hepatic lesions, hemangiomas and focal nodular hyperplasia (FNH) [2,3–4].

The need to distinguish between FNH and HA prompted several investigations of MRI performed with hepatobiliary contrast agents, and these generally found that a high percentage of FNHs showed isointense or hyperintense signal intensity relative to adjacent normal liver on hepatobiliary phase post-gadolinium images, whereas HAs appeared hypointense, with no uptake or reduced uptake of the hepatobiliary contrast agent in comparison to normal liver [6–11].

The French collaborative group proposed a subclassification system for HAs based on both histologic and immunohistochemical characteristics [12–14], with a recent modification including additional subgroups [15]. Four HA subtypes were initially identified: HAs with inactivating mutations of hepatocyte nuclear factor 1α (HA-H); HAs with activating mutations of the β-catenin gene (HA-B); HAs without mutations of the HNF1A or β-catenin genes and with inflammatory features (HA-I); and unclassified HAs that have no specific gene mutations or unique morphologic features (HA-U). The modified classification includes separate categories for β-catenin exon 3 mutated HA (b^{ex3} HA) and β-catenin exon 7/8 mutated HA ($b^{ex7,8}$ HA), as well as a new category for sonic hedgehog HA, characterized by activation of sonic hedgehog signaling due to focal deletions that fuse the promoter of inhibin β E with glioma-associated oncogene 1 (sh HA). In addition,

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there are two sub-categories representing mixed forms between inflammatory HA and β-catenin exon 3 mutated HA and between inflammatory HA and β-catenin exon 7/8 mutated HA. Studies performed using MRI with standard extracellular gadolinium contrast agents were able to successfully distinguish HA subtypes based on imaging characteristics on pre-contrast T1 and T2-weighted images and dynamic gadolinium-enhanced 3D spoiled gradient echo (SPGR) T1-weighted images [16–19].

Several authors have noted that some inflammatory HAs (HA-I) show signal intensity that is isointense or hyperintense relative to adjacent normal liver on hepatobiliary phase images obtained following injection of hepatobiliary contrast agents, an appearance more typical of FNH [7,20–23]. This is a potentially confusing imaging feature when seen in HAs, particularly when trying to distinguish between HA and FNH. The purpose of this retrospective study is to confirm these findings and to evaluate the imaging characteristics of pathologically proven inflammatory hepatic adenomas in patients who underwent MRI performed using hepatobiliary MR contrast agents.

2. Materials and methods

2.1. Patients

For this retrospective study approved by the institutional review board, pathology and body MRI records were searched for patients with a pathologic diagnosis of inflammatory or telangiectatic hepatic adenoma who had had MRIs performed with a hepatobiliary contrast agent. Age and gender of patients were noted, as well as the indication for MRI.

2.2. MRI

MRI was performed on 1.5 T systems (GE Signa EXCITE) in 17 patients and 3 T systems (GE HDx) in 10 patients. Dynamic imaging using gadolinium-based hepatobiliary contrast agents was performed in all patients: 3 patients received 0.1 mmol/kg gadobenate dimeglumine (MultiHance®, Bracco Diagnostics, Inc., Princeton, NJ), and 24 patients received 0.025 mmol/kg gadoxetic acid (Eovist®, Bayer Health Care Pharmaceuticals Inc., Wayne NJ). The examination protocol included coronal single shot fast spin echo images, 2D (1.5 T) or 3D (3 T) in-phase/out-of-phase spoiled gradient echo images, and diffusion-weighted images (DWI) performed prior to contrast administration. For patients receiving gadobenate dimeglumine, fat-suppressed respiratory-triggered FSE T2-weighted images were also acquired before contrast administration. This series was acquired after contrast injection in patients receiving gadoxetic acid.

Dynamic fat-suppressed T1-weighted 3D spoiled gradient-recalled echo (SPGR) images were acquired before and after injection of 0.025 mmol/kg gadoxetic acid at 1 ml/s or 0.1 mmol/kg gadobenate dimeglumine at 2 ml/s using an automatic injector (Medrad® Spectris, Bayer HealthCare LLC, Whippany, NJ). Timing of the arterial phase acquisition was determined using a 1 ml test bolus for all gadobenate dimeglumine injections, with additional portal venous and equilibrium phase images acquired at 70 s and 3 min following contrast injection. Hepatobiliary phase 3D SPGR images were acquired 60–110 min after injection, with average delay 84 min. Following gadoxetic acid administration, a single breath-hold triple arterial phase acquisition without bolus timing was acquired 15 s following injection in 20 patients, and a fluoro-triggered single arterial phase acquisition followed immediately by 2 additional acquisitions in 4 patients. Additional images were acquired 1–2 min following contrast injection, and hepatobiliary phase images were obtained 13–30 min after injection, with average delay 18 min. In 16 patients, hepatobiliary phase images were acquired using a high flip angle, typically 35°, versus 12° for the dynamic acquisitions.

Imaging parameters for the triple arterial phase gadoxetic acid acquisition include: TR/TE 3.4/1.2 ms, flip angle 15°, receiver bandwidth 82–100 kHz, section thickness 6–7 mm, field of view (FOV) 36–45 cm, partial phase field of view 0.8–1.0, matrix 256 × 128. Acquisition times ranged from 25 to 35 s with average 29 s. Single arterial phase fluoro-triggered acquisitions were obtained with 4 mm section thickness, 256 × 160 image matrix, and acquisition time of 10–12 s. Pre-contrast, hepatobiliary phase, and remaining dynamic 3D SPGR acquisitions were acquired with the following parameters: TR/TE 3.4/1.2 ms, flip angle 12° (or 35° in 16 patients for hepatobiliary phase acquisitions), receiver bandwidth 82–100 kHz, section thickness 3–4 mm, FOV 36–45 cm, partial phase FOV 0.8–1.0, matrix 256 × 224.

Diffusion-weighted images were obtained in 26 patients, consisting of a respiratory-triggered pulse sequence with b values of 0, 100, and 600 s/mm^2 acquired with 1, 4, and 10 numbers of signals averaged (NSAs) respectively.

2.3. Image analysis

Images were read by 2 authors in consensus (JG and CL). In patients with multiple lesions, analysis was performed on the largest lesion for which pathologic confirmation was available (correlation was based on images and reports from ultrasound or CT-guided biopsies, or from operative reports in patients who had surgical resection).

Analysis of in-phase/out-of-phase SPGR images included assessment for diffuse hepatic steatosis, defined as signal loss from in-phase to out-of-phase images of greater than one standard deviation of average signal intensity for a region of interest (ROI) placed in the same lobe as the measured lesion, avoiding vessels and additional lesions. When present, hepatic steatosis was subjectively graded as mild, moderate, or severe. Hepatic steatosis was also quantitatively assessed by measuring signal intensity (SI) on in-phase and corresponding out-of-phase images and estimating the fat fraction (FF) with the formula: $FF = (S_{IP} - S_{OP}) / 2S_{IP}$ [24]. A region of interest with area approximately 10 cm^2 was placed on the in-phase image on the same slice or on a slice adjacent to the hepatic adenoma, and then copied to the out-of-phase image.

The presence of lipid within the inflammatory adenomas was similarly assessed by placing an ROI encompassing the entire lesion except for regions of hemorrhage or cystic/necrotic degeneration and measuring the signal intensity on in-phase and out-of-phase images, with a signal intensity loss of > 1 standard deviation of measured signal intensity on the out-of-phase image defining the presence of lipid. Lesions with focal rather than diffuse lipid were also noted. When present, the extent of lipid within the lesion was subjectively graded as mild, moderate, or severe.

Dynamic contrast-enhanced images were assessed visually for the following criteria: presence and degree of enhancement on various phases. Hyperenhancement relative to adjacent hepatic parenchyma was classified as mild, moderate, or marked.

Signal intensity on DWI b = 600 s/mm^2 images was assessed subjectively in comparison to adjacent liver and graded as isointense, mildly hyperintense, moderately hyperintense, or markedly hyperintense.

Signal intensity on fat-suppressed respiratory-triggered FSE T2-weighted images was subjectively graded as isointense relative to liver, mildly hyperintense, moderately hyperintense, or markedly hyperintense. In addition, the presence or absence of a rim of high signal intensity (atoll sign) was noted.

2.4. Histopathologic analysis

All Hematoxylin and Eosin (H&E) stained slides were evaluated histologically by a liver pathologist (TM). A reticulin stain was performed on all lesions. All tumors were also subject to immunohistochemical (IHC) evaluation with liver fatty acid binding protein (LFABP), β-catenin, glutamine synthetase (GS), C-reactive

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