The value of MR imaging in the characterization of small (≤20 mm) enhancing lesions seen only during hepatic arterial phase at contrast-enhanced CT of the cirrhotic liver

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Purpose

To assess the value of gadobenate dimeglumine-enhanced MR imaging during the hepatobiliary phase in the characterization of small (<20 mm) enhancing lesions previously seen only during the arterial phase at contrast-enhanced CT in cirrhotic liver.
Methods and Materials

We analysed each cirrhotic patient with or without concomitant histology-proven HCC, who revealed non-wedge-shaped small nodular lesions (<20 mm), subcapsularly or centrally located, that enhanced only during the arterial phase at contrast-enhanced CT. All patients had a definite diagnosis of liver cirrhosis (Child-Turcotte-Pugh class A or B) related to viral infection (hepatitis B [n = 85 patients], hepatitis C [n = 52] or both [n = 3]) or alcohol abuse (n = 40), obtained by means of biopsy or unequivocal imaging findings including irregular liver margins and nodulations.

Patients underwent contrast-enhanced CT due to doubtful findings or dominant nodule detection on unenhanced US and/or alpha-fetoprotein increase (>20 ng/mL). All patients underwent MR imaging according to the following protocol (Table 1).

<table>
<thead>
<tr>
<th>Sequence</th>
<th>T2-w HASTE*</th>
<th>T2-w SPAIR#</th>
<th>T1-w FFE§</th>
<th>T2-w FFE§</th>
<th>Contrast-enhanced Imaging</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fat saturated</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Respiratory triggered</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Breath-hold</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Acquisition time (sec)</td>
<td>120-180</td>
<td>120-180</td>
<td>18</td>
<td>54</td>
<td>18</td>
</tr>
<tr>
<td>Repetition time (msec)</td>
<td>593</td>
<td>448</td>
<td>332</td>
<td>350</td>
<td>3.2</td>
</tr>
<tr>
<td>Echo time (msec)</td>
<td>80</td>
<td>80</td>
<td>(4.6 out-of-phase / 2.3 in-phase)</td>
<td>71</td>
<td>1.54</td>
</tr>
<tr>
<td>Flip angle (degrees)</td>
<td>90</td>
<td>90</td>
<td>80</td>
<td>20</td>
<td>10</td>
</tr>
<tr>
<td>Echo-train length</td>
<td>97</td>
<td>70</td>
<td>/</td>
<td>/</td>
<td>/</td>
</tr>
<tr>
<td>Parallel Imaging factor</td>
<td>1.75</td>
<td>1.75</td>
<td>1.5</td>
<td>1.5</td>
<td>1.5</td>
</tr>
<tr>
<td>No. of signals averages</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Field of view (mm)</td>
<td>(300-420) x (300-420)</td>
<td>(300-420) x (300-420)</td>
<td>(300-420) x (300-420)</td>
<td>(300-420) x (300-420)</td>
<td></td>
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<tr>
<td>Matrix</td>
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<td>384 x 384</td>
<td>164 x 132</td>
<td>256 x 256</td>
<td>432 x 432</td>
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<tr>
<td>Section thickness (mm)</td>
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<td>5</td>
<td>5</td>
<td>7</td>
<td>6</td>
</tr>
<tr>
<td>Intersection gap (mm)</td>
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<td>1</td>
<td>1</td>
<td>-3</td>
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<tr>
<td>Slice number</td>
<td>30</td>
<td>30</td>
<td>30</td>
<td>30</td>
<td>30</td>
</tr>
</tbody>
</table>

Fig.: Table 1. MR imaging protocol. *T2-weighted half-Fourier single-shot Turbo Spin-Echo [TSE] # T2-weighted half-Fourier single-shot Spectral Presaturation with Inversion Recovery § T1 and T2-weighted fast field echo [FFE] sequence # T1-weighted High Resolution Isotropic Volume Examination [THRIVE] sequence before and after Gd-BOPTA injection (0.1 mmol/kg; 2 mL/sec) and contrast injection starting after contrast visualization in abdominal aorta at MR fluoroscopy during arterial phase with bolus chase, and during portal (70 seconds), late (3 minutes), and hepatospecific
Inclusion criteria were: (a) patients with at least one and no more than ten small (≤2 mm in the largest dimension) subcapsularly or centrally located round nodular enhancing lesions at the hepatic arterial phase of liver CT that showed isoattenuation compared with the background liver on both portal venous and equilibrium phase images without typical features of portal shunt, that is, wedge-shaped lesion based on the liver capsule with a centrally directed apex that could be associated with a small portal venous branch; (b) patients who underwent gadobenate dimeglumine-enhanced MR imaging within 1 month from CT; (c) patients in whom follow-up dynamic CT was performed 3–6 months after lesion identification. We excluded 55 patients who did not undergo MR examination or underwent MR imaging after 1 month from CT. Therefore, we included 125 cirrhotic patients (67 male, and 58 female; age: 55±12 years) with (n=50) or without (n=75) concomitant HCC, who revealed up to 3 small lesions (≤2 cm in diameter) that enhanced only during the arterial phase at contrast-enhanced CT.

MR images were analysed in consensus by two readers.

Each nodules underwent 3–6 months follow-up by CT, and lesions unmodified in diameter and vascularity for 2 years were considered benign.

The criteria for pseudolesion were (a) pathologic proof showing that there was no evidence of tumor in the area showing hypervascularity at CT; (b) the lesion disappeared on the follow-up CT images; (c) the lesion remained unchanged in the imaging findings for more than 6 months.

The criteria for HCCs were (a) pathologic proof for the presence of a tumor, (b) the presence of a corona sign at the second phase of CT - that is, a ringlike enhancement surrounding the lesion - and the accumulation of iodized oil, which was administered during transarterial chemoembolization following CT, as confirmed by means of the follow-up CT, and (c) typical findings at dynamic CT of arterial hypervascularity with subsequent washout. If the lesion met one of the four criteria, the final diagnosis of HCC was made. Sizes of the HCCs and the pseudolesions were measured by using CT.

Within 15 days after CT the percutaneous US-guided biopsy was performed with 18 - 20-gauge modified Menghini needles and stained with hematoxylin/eosin and the Masson trichrome method. A senior pathologist for each center made the diagnosis according to
the diagnostic criteria established by the International Working Party on the terminology of nodular hepatocellular lesions.

Fisher exact test was performed to assess relationship between HCC and presence of neoplastic enhancing lesions.
Results

One-hundred-fifty-one arterially enhancing nodular lesions were identified, including 115 benign nodules and 36 HCC nodules.

Benign nodules (Figure 1 on page , and Figure 2 on page ) appeared prevalently iso- (n=64) (Figure 3 on page ) or hyperintense (n=25) or also hypointense (n=26) during the hepatospecific phase.

HCCs (Figure 4 on page ) were found in 30 patients with concomitant dominant HCC, who also had additional benign enhancing lesions, and appeared hypointense during the hepatospecific phase (Figure 5 on page ).

HCCs were found in four patients with concomitant dominant HCC, who also had additional benign enhancing lesions (n=2). In patients without HCC no neoplastic small arterially enhancing lesion was found.

Presence of dominant HCC nodules increases the malignancy probability of small arterially enhancing nodules (P <.001).
Conclusion

Small arterially enhancing nodular lesions in cirrhotic patients are mainly non-neoplastic and may be characterized by gadobenate dimeglumine-enhanced MR imaging during the hepatobiliary phase with the possibility to avoid long term imaging follow-up or biopsy. Presence of dominant HCC nodules increases the malignancy probability of small arterially enhancing nodules.
References


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