Add value of Diffusion Weighted Imaging to the Gadoxetic Acid-enhanced MRI in the study of hepatocellular carcinoma and in the detection of hypovascular nodule

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Authors: C. Briani¹, M. Di Pietropaolo¹, A. Saponi², G. F. Federici¹, C. Fantini³, A. Bucciarelli¹, E. Iannicelli², Rome/IT, Roma/IT, Caprarola (VT)/IT
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Purpose

Hepatocellular carcinoma is the fifth most common malignant tumors in the world and early detection and accurate characterization of lesion at an early stage is crucial to improve the patient’s outcome [1].

Gadoxetic acid disodium (Gd-EOB-DTPA) is a gadolinium-based contrast agent that combines the properties of both extracellular contrast media during the dynamic phases, and agents that specifically target hepatocytes due to the uptake of 50% by hepatocytes during the hepatobiliary phase [2].

Diffusion weighted imaging (DWI) provides functional information about Brownian motion of water molecules within tissue and seems to show a role in the detection of focal liver lesions based on microstructural tissue changes of the malignant lesions [3].

The purpose of this study was to evaluate the additive value of Diffusion Weighted imaging to Gd-EOB-DTPA-enhanced MRI in the study of HCC and to assess if DWI may help improve lesion characterization at an early stage.

Therefore, this paper will aim to determine if hypointensity of hepatobiliary phase could be considered as the best markers of premalignancy by itself or combined with hyperintensity on DWI.
Methods and Materials

We retrospectively reviewed our database MR imaging as part of a 3 months surveillance program of patients with liver cirrhosis.

Fifty-two patients with 80 nodules were enrolled in this study.

MRI examinations were performed using a 1.5 Tesla scanner (Sonata Siemens, Erlangen, Germany).

The MRI studies were conducted according to our routine liver protocol that included DWI by using a respiratory-triggered single-shot echo planar imaging with three increasing b-values (50-400-800 s/mm²).

Respiratory triggered T2WI and T1WI 3D-GRE (VIBE) were obtained as pre-contrast images, subsequently a bolus of 0,1mL/kg body weight GD-EOB.DTPA was injected and consisting of arterial (30-35 s), portal (70-80 s), delayed phases (180 s) and hepatobiliary phase images, acquired 15-20 minutes after intravenous injection of the contrast agent.

The baseline MRI included a T1-weighted Gradient echo in-phase and out-of-phase and T2 Turbo spin-echo Fat-Saturated (TSE-FS) sequences.

Images were evaluated in consensus by two experienced abdominal radiologists that analysed nodules size, vascular pattern on gadoxetic acid-enhanced dynamic MRI, signal intensity on hepatobiliary phase images and on DWI.

On DWimages, a lesion was considered as HCC when the hyperintensity of the lesion showed progressive signal preservation from lower b-value images to higher b-value images (b= 800 s/ mm²), and with an lower ADC compared to the background parenchyma on the ADC map.

On dynamic MRI, the final diagnosis of HCC was made according to diagnostic criteria defined by the American Association for the Study of Liver Disease (ASSLD) [4].

Nodules at risk of malignant transformation were considered as hypointenselesions on hepatobiliary phase images without hypervascularity on arterial phase images.

The accuracies of Gd-EOB-DTPA and DWI in the detection of HCC according to the size were analysed and differences between the two modalities were compared by the McNemar test. Fisher’s exact test was performed to compare proportions. A p-value < 0,05 indicated statistical significance.
Results

Eighty nodules were detected with Gd-EOB-DTPA-enhanced MRI.

Forty-eight/80 nodules (range 13-45 mm, mean size 19.7 mm) show the typical vascular pattern of HCC on dynamic imaging (Fig. 1,2).

On hepatobiliary phase images 46/48 lesions (95.8%) appeared hypointense (Fig. 3), 26/48 (54%) were hyperintense on DWI at b=800 s/mm² (Fig. 4). Among the 2 HCCs that were no hypointense in hepatobiliary phase, 1 was considered as a HCC on DWI at high b-value.

At the lesion's size increased, the prevalence of hyperintensity on DWI increased too, while there was no change in the detection of HCCs on hepatobiliary phase.

Thirty-two/80 lesions appeared hypointense in hepatobiliary phase with no signs of hypervascularization (Fig. 5,9); 25/32 (78.1%) were isointense on DWI (Fig. 6), 7/32 (21.8%) showed hyperintensity on DWI (Fig. 10).

Fifteen/32 (mean size 15 mm), showed hypervascularization and a typical vascular pattern of HCC during the 3-12 months follow-up (Fig. 7,8,11,12); the risk increased for the nodules #10 mm (p=0.017).

On DWI 6/15 nodules (40%) were hyperintense at high b-value at the first exam.
Fig. 1: Dynamic contrast-enhanced MR images show a focal liver lesion (arrow), with early arterial enhancement (Fig.1) and wash out in the equilibrium phase image (Fig.2). The lesion appears hypointense in hepatobiliary phase image (Fig.3). On diffusion weighted image at $b = 800 \text{ s/mm}^2$ (Fig.4) lesion is hyperintense compared to the background parenchyma.

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Fig. 2: Dynamic contrast-enhanced MR images show a focal liver lesion (arrow), with early arterial enhancement (Fig.1) and wash out in the equilibrium phase image (Fig.2). The lesion appears hypointense in hepatobiliary phase image (Fig.3). On diffusion weighted image at $b = 800 \text{ s/mm}^2$ (Fig.4) lesion is hyperintense compared to the background parenchyma.

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**Fig. 3:** Dynamic contrast-enhanced MR images show a focal liver lesion (arrow), with early arterial enhancement (Fig.1) and wash out in the equilibrium phase image (Fig.2). The lesion appears hypointense in hepatobiliary phase image (Fig.3). On diffusion weighted image at $b = 800 \text{ s/mm}^2$ (Fig.4) lesion is hyperintense compared to the background parenchyma.

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Fig. 4: Dynamic contrast-enhanced MR images show a focal liver lesion (arrow), with early arterial enhancement (Fig.1) and wash out in the equilibrium phase image (Fig.2). The lesion appears hypointense in hepatobiliary phase image (Fig.3). On diffusion weighted image at $b = 800 \text{ s/ mm}^2$ (Fig.4) lesion is hyperintense compared to the background parenchyma.

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Fig. 5: The hepatobiliary phase image (Fig.5) demonstrates a hypointense nodule that 
not show hyperintensity on DWI at $b = 800 \text{ s/ mm}^2$ (Fig.6). Gd-EOB-DTPA-enhanced-
MR image, 12 months after start of follow-up, shows typical dynamic pattern of HCC of
the lesion with a homogeneously enhancing in the arterial phase (Fig. 7) and wash out
in the equilibrium phase (Fig.8) images.

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Fig. 6: The hepatobiliary phase image (Fig.5) demonstrates a hypointense nodule that not show hyperintensity on DWI at b = 800 s/mm² (Fig.6). Gd-EOB-DTPA-enhanced-MR image, 12 months after start of follow-up, shows typical dynamic pattern of HCC of the lesion with a homogeneously enhancing in the arterial phase (Fig. 7) and wash out in the equilibrium phase (Fig.8) images.

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Fig. 7: The hepatobiliary phase image (Fig. 5) demonstrates a hypointense nodule that does not show hyperintensity on DWI at $b = 800 \text{ s/mm}^2$ (Fig. 6). Gd-EOB-DTPA-enhanced-MR image, 12 months after start of follow-up, shows typical dynamic pattern of HCC of the lesion with a homogeneously enhancing in the arterial phase (Fig. 7) and wash out in the equilibrium phase (Fig. 8) images.

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Fig. 8: The hepatobiliary phase image (Fig.5) demonstrates a hypointense nodule that does not show hyperintensity on DWI at b = 800 s/mm² (Fig.6). Gd-EOB-DTPA-enhanced-MR image, 12 months after start of follow-up, shows typical dynamic pattern of HCC of the lesion with a homogeneously enhancing in the arterial phase (Fig. 7) and wash out in the equilibrium phase (Fig.8) images.

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**Fig. 9:** The Hepatobiliary phase MR image (Fig.9) at 20 minutes after administration of gadoxetic acid, clearly shows a hypointense lesion, 20 mm in size, at segment IVb. On diffusion weighted image at $b = 800 \text{ s/mm}^2$ (Fig.10) lesion is hyperintense. Six months later, during the follow up, dynamic MR images show typical vascular HCC pattern: hypervascularity on the arterial phase image (Fig.11) and wash out in the equilibrium phase image (Fig.12).

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**Fig. 10:** The Hepatobiliary phase MR image (Fig.9) at 20 minutes after administration of gadoxetic acid, clearly shows a hypointense lesion, 20 mm in size, at segment IVb. On diffusion weighted image at $b = 800 \text{ s/mm}^2$ (Fig.10) lesion is hyperintense. Six months later, during the follow up, dynamic MR images show typical vascular HCC pattern: hypervascularity on the arterial phase image (Fig.11) and wash out in the equilibrium phase image (Fig.12).

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**Fig. 11:** The Hepatobiliary phase MR image (Fig.9) at 20 minutes after administration of gadoxetic acid, clearly shows a hypointense lesion, 20 mm in size, at segment IVb. On diffusion weighted image at $b = 800 \text{ s/mm}^2$ (Fig.10) lesion is hyperintense. Six months later, during the follow up, dynamic MR images show typical vascular HCC pattern: hypervascularity on the arterial phase image (Fig.11) and wash out in the equilibrium phase image (Fig.12).

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**Fig. 12:** The Hepatobiliary phase MR image (Fig.9) at 20 minutes after administration of gadoxetic acid, clearly shows a hypointense lesion, 20 mm in size, at segment IVb. On diffusion weighted image at $b = 800 \text{ s/mm}^2$ (Fig.10) lesion is hyperintense. Six months later, during the follow up, dynamic MR images show typical vascular HCC pattern: hypervascularity on the arterial phase image (Fig.11) and wash out in the equilibrium phase image (Fig.12).

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Conclusion

Gd-EOB-enhanced-MRI is highly accuracy in the detection of HCC and of hypovascular nodules with potential progression to HCC (high risk for nodules #10 mm).

The usefulness and the additive value of DWI in the detection of HCCs could be demonstrated with a slightly increased detection sensitivities after addition of DWI (95.8% vs 97.9%) but we found no significant additional value in the identification of hypovascular nodules at risk of malignant transformation.

