

Functional MRI of the Bowel: Detection of Perfusion Defects and Changes in the Water Diffusion

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Introduction

Mesenteric ischemia is an acute disease, with fatal outcome in about 70% of the cases [1]. Since the interval between the onset of symptoms and irreversible damage of the bowel is app. 120-180 min [2], there is need for an effective and reliable diagnostic procedure which can detect early changes in the mesenteric perfusion. Since mesenteric ischemia can be induced by (i) arterial emboli, (ii) venous occlusion or even (iii) a non-occlusive event MR-angiography might not be sufficient for a timely diagnosis. Furthermore, MRA of the main mesenteric arteries and the intermediate vessels is an indirect approach while the early detection of bowel ischemia requires imaging methods which are (i) sensitive to changes in the blood supply or (ii) susceptible to the net water shift associated with the development of cytotoxic edema. For this reason this work studies (i) the detection of perfusion deficits in the mesenteric end-arteries using high spatial resolution contrast enhanced MRA and (ii) the detection of cytotoxic edema of the bowel wall by means of diffusion weighted (DWI) imaging.

Methods

The mesenteric blood supply was studied in six healthy female pigs (weight=50kg) using a 1.5 T MR-system (Achieva 1.5, Philips, Best, The Netherlands) and a conventional X-ray fluoroscopy (DSA) located inside of the magnet room. On a floating table the pigs could be swayed between MR-scanner and fluoroscopy without changing the table. The mesenteric ischemia was induced under X-ray fluoroscopy, where a 5F Cobra-catheter was placed in the superior mesenteric artery (SMA). Through a 3F microcatheter a smaller branch of the SMA was embolized either with histoacryl to mimic an arterial embolus (3 pigs) or with particles (40-120 μ m) to mimic a non-occlusive ischemia (3 pigs). Before and after (30 min, 1 h, 2 h) embolisation the pigs were examined with the same protocol: To display the mesenteric perfusion a 3D-MRA sequence (FOV = 35 cm x 30 cm x 8.5 cm, TE = 1.8 ms, TR = 5.5 ms, flip angle = 40°) with a spatial resolution of (1x1x1) mm³ was performed after i.v. injection of 0.2 mmol/kg body weight gadopentetate dimeglumine in coronal planes. To detect the cytotoxic edema of the bowel wall, axial diffusion weighted SE-EPI imaging (FOV = (36 x 36x13) cm³, TE = 88 ms, TR = 5513 ms, flip angle = 90°) was conducted using b-values of 0, 250, 500 and 750 s/mm². Additionally ADC-maps were calculated. For all imaging techniques used data acquisition was performed in breath-hold mode. I.v. administration of 1.0 mg/kg body weight butylscopolamine was applied to avoid motion artefacts.

Results

After embolisation with histoacryl high spatial resolution contrast enhanced 3D-MRA facilitated a direct visualisation of mesenteric perfusion defects as illustrated in Fig. 1. After embolisation with particles no filling defect of the mesenteric branches was observed but a lack of contrast-uptake in the bowel-wall in the delayed scans. DWI yielded a reduction in the water diffusion coefficient as illustrated by the ADC map shown in Fig. 2. This observation was independent from the type of embolisation.

Conclusion

Functional MRI comprising high spatial resolution contrast enhanced 3D MRA and diffusion weighted imaging holds the promise to be an appropriate method for early detection of mesenteric ischemia. The perfusion defects can be directly visualized by means of MRA. DWI affords the visualisation of cytotoxic edema of the bowel wall as an early sign for an imminent infarction of the bowel.

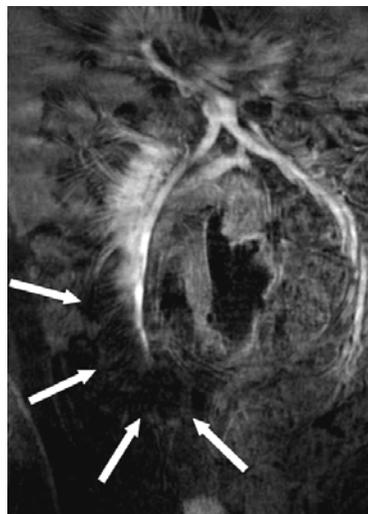
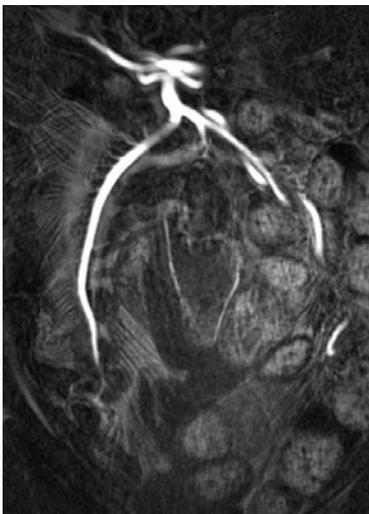


Fig 1: Coronal MR angiograms acquired prior (left) and after (right) embolisation with histoacryl. The perfusion defect is clearly visible (arrows).

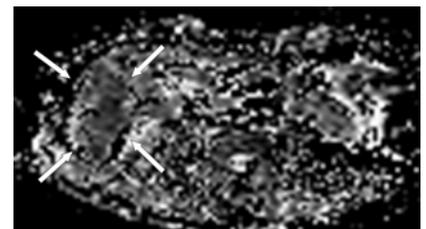
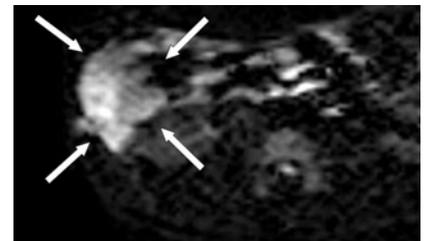


Fig 2: Axial DW- and T2-weighted images obtained 1 h after embolisation showing a distinct cytotoxic edema (arrows) after embolisation (top). The cytotoxic edema resulted in a reduction of the water diffusion coefficient as illustrated by the ADC-map (bottom).

References

[1] American Gastroenterologic Association, GASTROENTEROLOGY 2000;118:951-953[2] Bottger T, Schafer W, Weber W, Junginger T. Langenbecks Arch Chir. 1990;375(5):278-82.