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Meeting abstract

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1037 T2-STIR signal in acute reperfused myocardial infarction is dependent on microvascular flow

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Introduction

T2-weighted cardiovascular magnetic resonance imaging is widely used to detect myocardial edema, and differentiate acute from chronic myocardial infarction.

The development of edema, however, may depend on reperfusion blood flow, and the role of T2-weighted imaging in the presence of microvascular obstruction (MO) has not been investigated. Microvascular obstruction may influence the development of edema, and therefore T2 signal.

Hypothesis

We hypothesize that the signal increase in T2-weighted CMR images in acute reperfused myocardial infarction is dependent on microvascular blood flow, and is not present in areas of microvascular obstruction.

Methods

Out of a cohort of 21 patients with acutely reperfused ST-elevation myocardial infarction, 12 patients with microvascular obstruction were identified based on late enhancement findings. On a clinical 1.5 T system (Avanto, 6-channel cardiac phased-array coil, Siemens Medical Solutions) we acquired T2-weighted STIR images (TR 2RR intervals, TE 61 msec) to identify myocardial edema, and at 10 minutes after injection of 0.2 mmol/l Gd-DTPA (Magnevist, Bayer Health Care, Canada), late enhancement images (IR-GE, inversion time adjusted manually between 280–460 msec), covering the entire left ventricle in contiguous 10 mm slices. Using validated software, infarcted myocardium was identified using threshold detection as the region with more than 10 contiguous pixels >2 standard deviations above the mean of remote myocardium. The area of microvascular obstruction was defined as the subendocardial non-enhanced zone surrounded by late enhancement. Corresponding regions of interest of a. Late enhancement, excluding microvascular obstruction (infarct-reflow), b. Microvascular obstruction zone (infarct-no-reflow) c. Complete infarction (reflow plus no-reflow) were copied to the T2-STIR images, and the T2 signal intensities were measured in these different injury zones. The mean values for signal intensities were compared using a t-test.

Results

Image quality was diagnostic in all slices. The mean T2-STIR signal intensity in the remote myocardium was 65.8 ± 17.3, in the infarct zone excluding microvascular obstruction (reflow zone) it was 94.8 ± 28.8, in the microvascular obstruction zone (no-reflow zone) was 77.2 ± 21.1, and the entire infarction comprising tissue with and without reflow was 91.0 ± 29.2 (see Figure 1).

The mean T2 signal intensity in the microvascular obstruction zone was significantly lower than in the infarction zone without microvascular obstruction ($p < 0.01$).

Conclusion

In acute myocardial infarction, the T2 signal in areas of MO is significantly lower than in areas of acute infarction without MO. This may suggest that the development of tissue edema depends at least in part on microvascular perfusion. Importantly, a lack of T2-signal increase caused

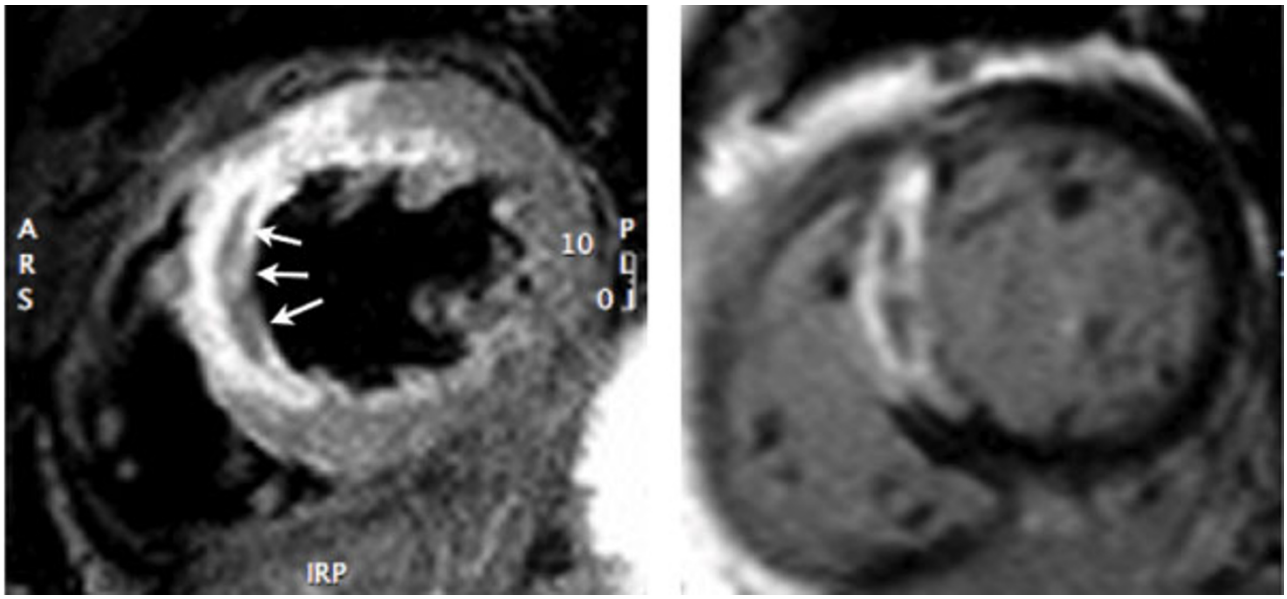

Figure 1
T2-STIR image (left) and late enhancement image (right). Note the signal drop in the microvascular obstruction zone in the STIR image (arrows).

by large areas of MO may be a source of error in the identification of acute myocardial infarction.