Impact of Myocardial Infarct on Regional Left Ventricular Function

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Background

Previous studies in animal models of myocardial infarct have demonstrated that myocardial contractile function is impaired not only in left ventricular (LV) regions with infarct, but also in non-infarcted regions1. We sought to measure the extent of functional impairment in the infarct, infarct-adjacent and remote myocardial regions in patients with previous myocardial infarction by using delayed contrast-enhanced (DE) MRI to detect myocardial scar and define infarct borders.

Methods

Segmented SSFP cardiac cine and multi-shot inversion recovery spoiled gradient echo DE MR images were obtained at 1.5 T, in 13 patients (11 M, 2 F; aged 40-82 years, mean 60.3 ± 12.3) with documented myocardial infarction. Cine and DE images were acquired in corresponding short-axis planes (spatial resolution ≤ 2.5x2.0x10.0mm; slice gap ≤ 2mm) from the base of the heart to the apex. 3DSlicer v2.6, free open source image analysis software (slicer.org), was used to manually segment and create a three-dimensional model of the infarct.

LV function was assessed using CIM software2, which provided LVEF as well as the following segmental data based on a 16 segment model: myocardial thickening (peak thickness – minimum thickness), EDV, ESV, and EF. Infarct and LV masses were derived from their respective three-dimensional models assuming a constant density of 1.04 g/ml. Mid-ventricular segments that visually contained greater than 50% mural hyper-enhancement on DE MRI were considered “infarct”. Mid-ventricular segments that were directly adjacent to infarct segments were considered “infarct-adjacent,” and the remaining segments were considered “remote” (Fig 1). One patient did not have any remote segments due to the large size of the infarct region. Statistical comparisons were made using Anova with Bonferroni corrections for multiple post-hoc paired, two-tailed student’s t-test.

Results

Global LV function as measured by LVEF decreased as relative infarct mass increased (R = -0.526). The regional ESV, EF, and myocardial thickening for the infarct, infarct-adjacent, and remote regions were all found to be statistically different (p < 0.05 for all comparisons; Table 1). Myocardial thickening decreased from the remote to infarct-adjacent region and from the infarct-adjacent to infarct region (remote: 9.79mm ± 2.47, infarct-adjacent: 6.99mm ± 2.19, infarct: 3.47mm ± 1.95, p < 0.01; Fig 2). Also, the percent decrease in thickening from the infarct-adjacent to infarct region was significantly greater than from the remote to infarct-adjacent region (135.9% ± 96.3 vs. 23.2% ± 9.79). EF decreased from the remote to infarct-adjacent region (78.41% ± 8.05 vs. 63.93% ± 12.88) and from the infarct-adjacent to infarct region (63.93% ± 12.88 vs. 40.34% ± 15.72). EDV was not different between regions (p > 0.05).

Conclusions

The infarct-adjacent region was found to be functionally distinct from both the infarct and remote regions as measured by segmental myocardial thickening, EF, and ESV, in agreement with previous animal studies3. Furthermore, the decline in thickening from the infarct-adjacent to infarct region was significantly greater than from the remote to infarct-adjacent region. This finding suggests that a non-linear relationship exists between post-MI myocardial function and distance from the infarct region, which has important clinical implications in revascularization therapy.

References


Table 1. Bonferroni corrected p-values from a paired, two-tailed t-test.

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Fig 1. Classification of the infarct (i), infarct-adjacent (a), and remote (r) regions.

Fig 2. (A) Myocardial thickening of individual patients (B) Mean ± SD over all patients for each region (infarct: 3.47mm ± 1.95, infarct-adjacent: 6.99mm ± 2.19, remote: 9.79mm ± 2.47).