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Meeting abstract

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222 Cardiac magnetic resonance assessment of induced myocardial infarction and reverse remodelling early after alcohol septal ablation

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Objectives and background

Magnetic resonance imaging (MRI) has been used to delineate the extent of acute myocardial infarction in coronary artery disease, but its role after ethanol induced septal infarction has not been largely assessed. We report the evaluation of septal infarction induced by alcohol septal ablation (ASA) in patients with symptomatic hypertrophic obstructive cardiomyopathy using contrast enhanced MRI.

Methods

Of the 103 consecutive patients who underwent ASA from 1999 to April 2007, MRI was performed systematically before and early after ASA in 48 patients. The main contraindication to MRI was permanent pacemaker or per or post procedure temporary PM implantation. Size and location of the induced infarction, assessed by MRI were compared to left ventricular (LV) mass reduction, enzyme release, volume of ethanol administered and LV outflow gradient reduction.

Results

CE MRI evaluation was performed 3.9 \pm 2.5 days after the procedure and revealed regional hyper enhancement in the basal interventricular septum in all patients. Only one pt had a remote site signals in inferior wall. The mean infarct size was 17.2 \pm 6.3% (delayed enhanced images).

Total LV mass decreased from 277.2 \pm 84.9 g to 234.7 \pm 82.6 g (p = 0.008) and ejection fraction (EF) from 70.3 \pm 09.9 to 64.9 \pm 8.7% (p = 0.001).

On first pass imaging, the observed Early hypoenhancement correlated with maximum gradient reduction, LV mass reduction, Post procedure EF reduction, total quantity of alcohol injected, diameter of septal arteries treated, number of septal treated and Troponin peak (all p < 0.01). For delayed enhanced images, induced infarct area correlated with all the above parameters (all p < 0.001) and coefficient factors were respectively: 0.46 for gradient reduction, 0.47 for myocardial mass reduction, 0.04 for EF% reduction, 0.21 for ethanol dose, 0.30 for diameter of septal arteries treated, and 0.50 for peak Troponin. By linear regression analysis, a good correlation was found between early hypo enhancement and late hyper enhancement measures (r = 0.66). There was significant reduction in MR grade (2.1 to 0.96, p < 0.001) and occurrence of SAM (0.99 to 0.33, p < 0.001) post procedure.

Conclusion

Contrast enhanced MRI allows detailed evaluation of size and location of septal myocardial infarction induced by ASA. Infarction size as assessed by MRI is better correlated with Peak troponin, gradient reduction and myocardial mass reduction.

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