Journal of Cardiovascular Magnetic Resonance

Meeting abstract

2044 Can delayed hyperenhancement shed insight into the mechanism of mitral regurgitation?

Robert WW Biederman*, Ronald Williams, June Yamrozik, Ketheswaram Caruppannan, Vikas K Rathi, Geetha Rayarao, Diane A Vido, Saundra Grant and Mark Doyle

Address: Allegheny General Hospital, The Gerald McGinnis Cardiovascular Institute, Pittsburgh, PA, USA
* Corresponding author

from 11th Annual SCMR Scientific Sessions
Los Angeles, CA, USA. 1–3 February 2008

Published: 22 October 2008

Journal of Cardiovascular Magnetic Resonance 2008, **10**(Suppl 1):A313 doi:10.1186/1532-429X-10-S1-A313

This abstract is available from: http://jcmr-online.com/content/10/S1/A313

Introduction

A myriad of explanations underpin the mechanisms proposed for the LV annular dilatation following a myocardial infarction that eventually lead to mitral regurgitation. Most believe an *active* process drives remodeling of adjacent myocardium with subsequent secondary mitral annular dilation. However, non-geometric, *passive* mechanisms have not been considered as potential contributers to either the mitral valve or the annular patholgy. Classic CMR delayed hyperenhancement (DHE) describes a myriad of LV myocardial histopathology but may also be sensitive to non-myocardial pathology such as the annulus or the mitral leaflets.

Hypothesis

We hypothesize that DHE may detect occult LV annular and/or mitral valvular enhancement in post MI patients.

Methods

220 patients; 140 s/p MI (55 F, 50 acute, 90 chronic) underwent CMR (1.5 T GE) with 0.2 mmol/kg of Magnevist (Berlex, Montville, New Jersey or 0.1 mmol/kg MultiHance (Bracco Diagnostics, Princeton, New Jersey). Notation of presence or absence of DHE involving the mitral annulus and/or mitral valve was made. Non-MI patientss (80) served as controls.

Results

All post-MI pts demonstrated infarct by DHE. Additional DHE was present involving the mitral valve in 93 (67%) but representing 86% of chronic MI's but only 11% of acute MI's. Exactly 56 (40%) pts also had mitral annular DHE. Lesser amounts of DHE signal was also seen in adjacent valves: aortic 31 (22%), tricuspid 43 (31%) while virtually no DHE signal was seen along the tricuspid annulus 11 (7%). Only 7 (9%) of controls demonstrated any degree of valvular DHE and 3 (4%) had annular DHE (mostly myocarditis patients). Figure 1.

Conclusion

CMR DHE depicts focal annular and/or valvar enhancement in a large number of post MI patients, suggesting a specific, as yet unknown reactive process may contribute to annular dilatation and/or mitral leaflet pathology. This passive phenomena is currently not a suspected contributor to the post MI phenotype but may portend late LV dilatation and either primary or secondary mitral regurgitation.

The Delayed Hyperenhancement phenomenon has been well described for improved myocardial tissue characterization for infarct and non-infarct related pathologies. We demonstrate that this same property of DHE appears to delineate previously unrecognized properties within the mitral valve in post-MI patients.

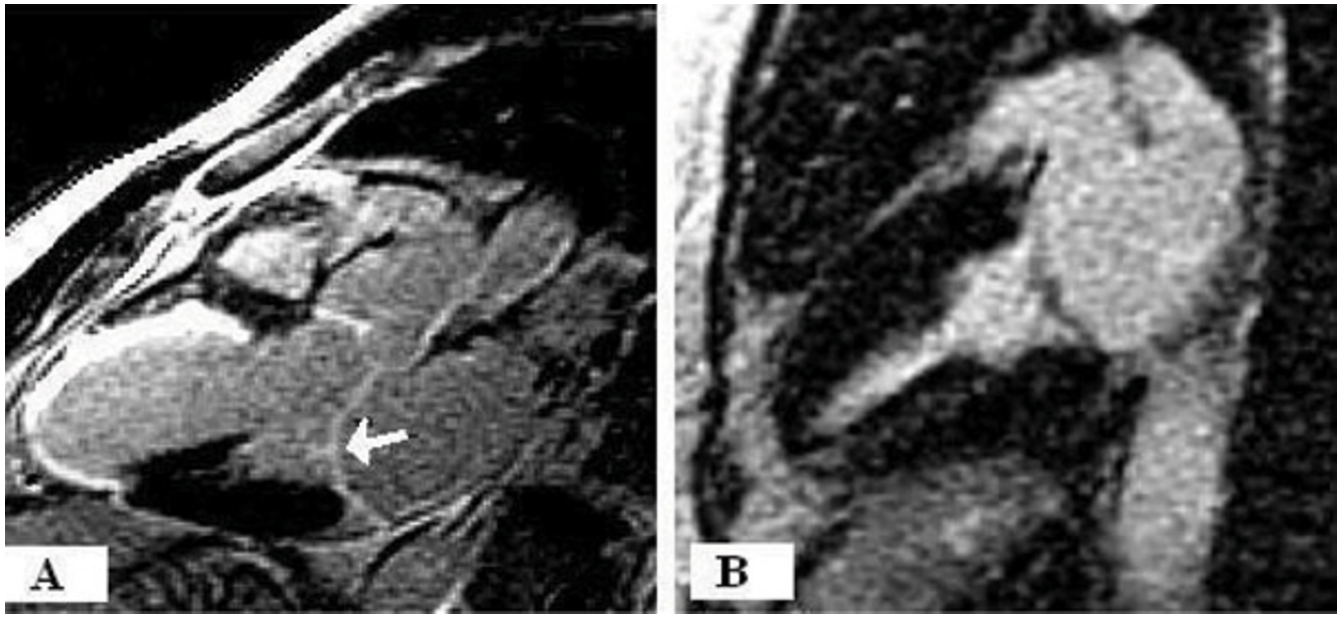

Figure 1