

# Is There an Alternative Explanation for Post-MI Mitral Regurgitation; Insights from CMR

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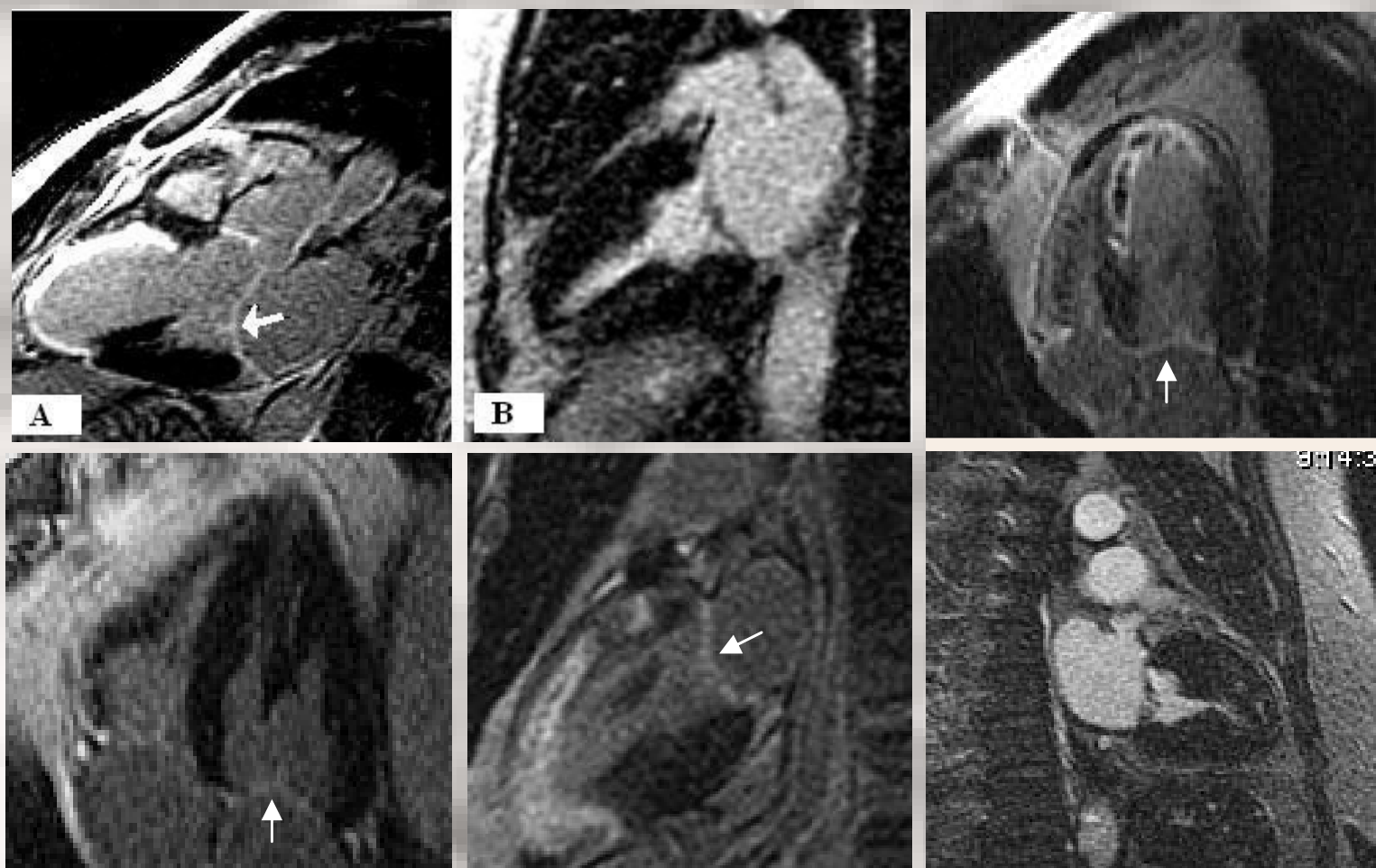
**Introduction** Multiple explanations exist for the etiology of LV annular dilatation post-myocardial infarction (MI). The current assumptions suggest an active process reflecting remodeling of adjacent myocardium. However, non-geometric, passive mechanisms have not been considered, yet are important. Cardiac MRI (CMR) delayed hyperenhancement (DHE) post-contrast techniques describe a myriad of LV myocardial histopathology such as infarct, infiltrative and inflammatory perturbations within the LV but may also be sensitive to non-myocardial pathology.

**Hypothesis** We hypothesize that DHE may detect occult LV annular and/or mitral valvar enhancement in post-MI patients and its presence might predict progression of unfavorable changes in annular geometry that lead to progression of mitral regurgitation (MR).

**Methods** One hundred sixty-four (164) patients; 111 S/P MI (43 F, 37 acute, 74 chronic) underwent CMR (1.5T GE, Milwaukee, WI) with 0.2mmol/kg of Magnevist (Berlex, Wayne, NJ) or 0.1mmol/kg MultiHance (Bracco, Princeton, NJ). Notation of presence or absence of a DHE pattern involving the mitral annulus and/or valve was made. Patients were specifically excluded if MI pattern involved basal myocardium to avoid confounding signal etiology.

**Methods, continued** Non-MI patients (53) referred for contrast CMR served as controls. A subset of patients in whom follow-up data was available was analyzed for geometric changes of the available was analyzed for geometric changes of the mitral annulus and degree of MR while related to presence or absence of DHE (+DHE, -DHE).

**Results** All post-MI patients demonstrated an area of infarct by functional analysis, confirmed by DHE (100%). Additional DHE was present involving the mitral annulus in 49/111 (44%) and in 84/111 (76%) the mitral valve. Lesser amounts of DHE signal was also seen in adjacent valves: aortic 25/111 (22%), tricuspid 33/111 (30%) while virtually no DHE signal was seen along the tricuspid annulus 5/111 (5%). Only 6/53 (11%) of controls demonstrated any degree of valvar enhancement and 3/52 (6%) had annular enhancement with the majority of these in myocarditis patients. In the subset of 12 post-MI patients available at baseline (3±2 days), 6 weeks and 6 months, there was progressive mitral annular dilation present in 10/12 (83%) +DHE but only 2/6 (33%) -DHE patients. As well, the degree of mitral annular dilation trended higher in those with +DHE vs. -DHE (3.0x2.9x4.5mm vs. 2.5x2.0x4.2mm, p=NS). Finally, the progression of MR underwent greater deterioration at a faster pace in those with +DHE vs. -DHE (p <0.05). Indeed, MR progressed in all pts except for one in whom DHE decreased and in one pt who remained unchanged. MR remained unchanged in all patients who were -DHE. See figure (arrow denote enhanced mitral valve whereas no enhancement in normal valves).



## Conclusions

CMR DHE depicts focal annular and/or valvar enhancement in large number of post MI patients, suggesting a specific, as yet unknown reactive process may contribute to annular dilatation and/or mitral leaflet pathology. Preliminary data supports a deleterious impact in the post-MI remodeling pattern in those with mitral annular or valvar delayed enhancement. 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 progressive mitral regurgitation.