

Poster presentation

Contrast enhanced magnetic resonance imaging of culprit lesions in patients with acute coronary syndrome

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Introduction

X-Ray Angiography (XRA) is the gold standard for the assessment of lumen encroaching coronary stenosis, which typically underlies stable coronary artery disease. However, XRA cannot distinguish between stable and vulnerable plaques, which are thought to be the precursor lesions of most acute myocardial infarctions (MI) and death. The development of a non-invasive technique that can detect culprit lesions immediately after MI may eventually prove useful for prospective identification of vulnerable plaque.

Purpose

The aim of the study was to evaluate whether contrast-enhanced magnetic resonance imaging (CE-MRI) could detect culprit lesions in patients with a recent ACS (troponin T >1.0).

Methods

16 patients (14 male, age 62.7 ± 8.7) were imaged within 24-72 h after presenting with ACS prior to XRA. Following coronary lumen imaging and contrast injection, CE-MRI of the coronary artery wall was performed using a T1-weighted 3D gradient echo inversion recovery sequence (3D IR TFE).

Results

In 13 patients culprit lesions (6 × LAD, 2 × LCX, 5 × RCA) were identified by XRA (Fig. 1e+j) +/- intravascular ultrasound (IVUS). CE-MRI (fused with magnetic resonance angiography, Fig. 1c+h) correctly identified culprit lesions in 11 of 13 patients (sensitivity: 85%) and correctly classified the 3 patients without culprit lesions (specificity 100%). Contrast uptake in culprit lesions (Fig. 2a) as compared to overall vessel wall contrast uptake was 4-fold increased (50.7 ± 24.2 vs. 12.1 ± 7.7 , $p < 0.01$) and 2-fold increased as compared to affected segments (19.2 ± 14.2 , $p < 0.01$). Intra pair analysis (Fig. 2b) of the culprit lesion showed a 4-fold increase of the CNR as compared to the overall vessel wall contrast uptake (Ratio: $406 \pm 123\%$), a 3-fold increase for the segments of the affected vessel ($356 \pm 155\%$) and a 2-fold increase for the affected segments ($262 \pm 92\%$). Subgroup analysis (Fig. 2c+d) revealed a significant higher contrast uptake of the culprit lesion (69.4 ± 20.5 vs. 31.2 ± 5.6 , $p = 0.01$) and adjacent segments (30.7 ± 15.1 vs. 12.8 ± 5.1 , $p = 0.04$) in the left coronary system (Fig. 2c) as compared to the right system (Fig. 2d). Non-affected segments showed no significant difference between the left and right system (3.5 ± 1.5 vs. 4.1 ± 1.3 , $p = 0.50$). These findings are in concordance with a trend towards a higher TIMI Risk Score in patients with anterior MI (median: 21.5 (Range 16.1-35.9) vs. 1.6 (0.8-23.4), $p = 0.06$).

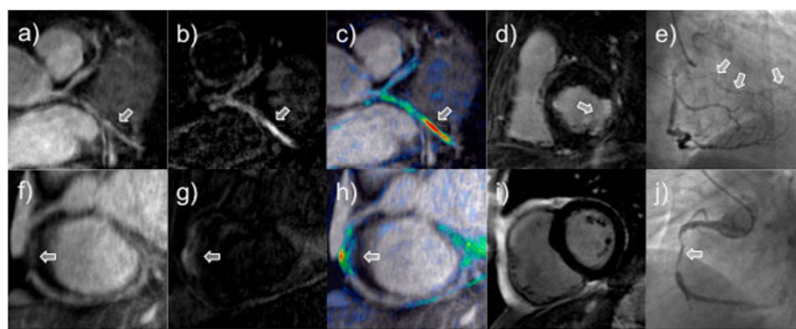


Figure 1
a-e) This 72-yr old gentleman with known CAD and previous NSTEMI was thrombolysed for an anterior STEMI (peak CK 1850). MRA (a) showed mid LAD and ostial LCX stenosis with corresponding enhancement in CE-MRI (b). Fusion (c) of MRA (a) and CE-MRI (b) revealed marginal branch as culprit lesion (arrow). LE images showed transmural scar of the lateral wall. XRA of the left system showed ostial LCX and mid LAD stenosis. XRA of the RCA confirmed CE-MRI findings with retrograde filling of the completed occluded marginal branch (e, arrows). f-g) This 48-yr old man was admitted with troponin positive ACS (1st presentation) with no significant ECG changes. Fusion (h) of MRA (g) and CE-MRI demonstrate mid RCA stenosis as culprit lesion (arrow). LE images showed no scar. XRA (j) showed proximal to distal tight RCA stenosis with normal flow (culprit lesion, arrow).

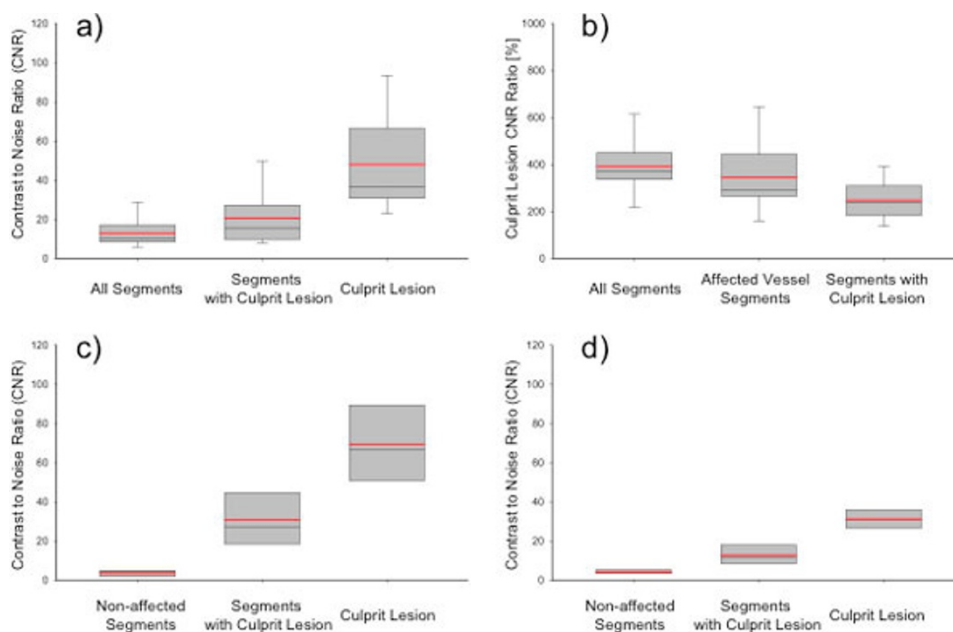


Figure 2
Box-plot (red line indicating mean), 13 patients (12 male, age 62.1 ± 8.9) with identified culprit lesion (6 × LAD, 2 × LCX, 5 × RCA) in XCA. a) Comparison of the contrast uptake in all segments, affected segments and the culprit lesion. b) Intra pair of CNR analysis between the culprit lesion and overall vessel wall contrast uptake, uptake in segments of the affected vessel and affected segments. c+d) Subgroup analysis of the left (c) and right (d) coronary system.

Conclusion

CE-MRI allows selective visualization of culprit lesions in patients after MI. This technique may be useful for prospective detection of vulnerable plaque.