CMR in Myocardial Viability

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The concept of myocardial viability is based on the fact that even severely dysfunctional myocardium in patients with coronary artery disease may show functional improvement after revascularisation . Reversal of myocardial dysfunction is particularly relevant in patients with depressed ventricular function because revascularisation improves long-term survival.

There are many methods available to assess viability including

1. Low-dose dobutamine stress echocardiography.
2. Thallium-SPECT
3. FDG-PET
4. CMR-cardiac MRI

All recent studies (1) have shown that CMR is superior to low-dose dobutamine echocardiography and thallium –SPECT for viability imaging. CMR is good as FDG-PET(2) for picking up the infarcts with the added advantage of better spatial resolution which allows evaluation of the exact transmural involvement of the myocardium. The whole concept of viability on CMR is based on the fact that all infarcts enhance vividly 10-15 minutes after intravenous contrast administration. This phenomenon of delayed hyperenhancement has been proven to correlate with the actual extent of the infarct in numerous animal and human studies (3). CMR shows the transmural extent of the infarct very reliably. In infarct imaging on contrast –enhanced CMR it is now said that "white is dead". In areas of hypokinesia if there is a rim of "black" or non-infarcted myocardium that is not contracting well if indicates the presence of hibernating myocardium which is likely to improve after revascularisation of the artery supplying that particular territory. Viability imaging reliably allows identification of areas of hibernation and viable or non-viable myocardium.

Case 1 :
A 64-years old man presented with cardiac failure and an ejection fraction of 20 % on echocardiography. Coronary angiography (Fig 1A) showed high-grade stenosis in the LAD and LCX.
A cardiac MRI was performed for assessing viability.
The cine images show marked hypokinesia and thinning of the anteroseptal and inferolateral walls of the myocardium, areas supplied by the LAD and LCX respectively (Fig. 1B). Full thickness delayed hyperenhancement is seen in both these areas, suggesting scar tissue (Fig 1C).
In view of the full-thickness, transumural involvement, and absence of any viable myocardium, a decision was taken not to revascularize the lesions in this patients

Case 2 :
A 53 years old lady presented with an LAD occlusion and a stenotic lesion in the OM1 (Fig. 2A). Ejection fraction was 24%. A decision had to be made about further treatment. A cardiac MRI was performed for assessing viability. The CMR study shows thinning and hypokinesia of the anterior wall of the myocardium on the short axis image (Fig. 2B) with approximately 50% delayed hyperenhancement of an LAD territory infarct (Fig. 2C). there is at least 50% viable myocardium in the infarct region. In the apical region seen best in the vertical long axis (VLA) view (Fig. 2d), marked wall thinning is seen with hypokinesia noted on the cine images. Most of the anterior wall shows sub-endocardial enhancement of approximately 50% of the myocardial thickness with full thickness, transmural enhancement at the apex itself (fig.2E). Except at the apex, the rest of the anterior wall shows viable (black) myocardium of atleast 50% thickness. As a result a decision to revascularisation the patient was taken.

INFARCT IMAGING AND HEART FAILURE.
As a corollary, CMR is also very useful in the evaluation of patients with cardiac failure to distinguish between dilated cardiomyopathy (DCM) and chronic LV dysfunction due to coronary artery disease (Fig. 4). CMR can reliably differentiate between these two entities, thus obviating the need for initial coronary angiography to differentiate between these two conditions in patients with LV failure

References:
1. Wagner A, Mahrgoldt H, Holly TA et al. Contrast-enhanced MRI and routine SPECT perfusion imaging for detection of subendocardial infarcts: an imaging study. Lancet 2003;361:359-360.

2. Kuhl Hp, Beek AM, Van der Weerdt AP et al. Myocardial viability in chronic ischemic heart disease: comparison of contrast-enhanced magnetic resonance imaging with (18) FDG PET, JACC 2003;16: 1341-1348.
3. Kim RJ, Wu E, Rafael A et al. The use of contrast enhanced magnetic resonance imaging to identify reversible myocardial dysfunction. NEJN 2000;343:1488-1490.
4. McCrohon JA, Moon JCC, Prasad SK et al. Differentiation of heart failure related to dilated cardiomyopathy and coronary artery diseases using gadolinium enhanced cardiovascular magnetic resonance. Circulation 2003;108:0054-0058.

LEGENDS

Fig1. (A,B). Viability study. The angiogram (A) shows high grade stenoses of LAD (arrow) and circumflex (arrowhead) arteries. A diastolic frame from a mid-cavitary short axis cine study (B) shows thinning of the anteroseptal (arrow) and infero-lateral (arrowhead) regions- marked hypokinesia was seen on the cine images. The corresponding areas on the contrast-enhanced viability study (C) show full-thickness, transmural infarcts (arrows).

Fig.2 (A-E): Viability study. The angiogram (A) shows an LAD occlusion (arrow) with a stenotic lesion of the OM1 (arrowhead). A diastolic frame from a mid cavitary short axis cine study (B) shows thinning of the anterior and antero-septal walls (arrow) with hypokinesia noted on the cine study. The corresponding viability image shows a sub-endocardial infarct (C) involving approximately 50% of the myocardial thickness (arrow). A diastolic frame from a vertical long –axis (VLA) cine study. The corresponding viability image (E) shows a transmural infarct (arrow) involving the apex (arrow) with a sub-endocardial infarct involving 50% of the myocardium (arrowhead) in the anterior wall.

Fig.3(A,B) : Dilated cardiomyopathy, A diastolic frame from a horizontal long-axis (HLA) cine study shows LV dilatation (A). The corresponding contrast-enhanced image (B) shows no enhancement.

Fig.4 (A,B) : Ischemic cardiomyopathy. A diast
olic frame from an HLA cine study (A) shows LV dilatation with thinning of the apex (arrow). The corresponding contrast –enhanced study (B) shows a full-thickness transmural infarct involving the apex (arrow).

Fig. 1A

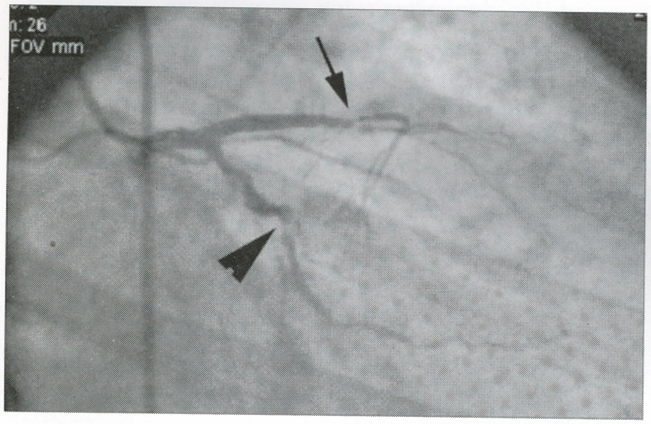

Fig. 1B

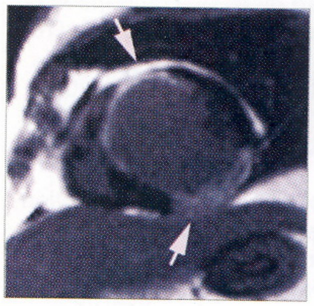

Fig. 1C

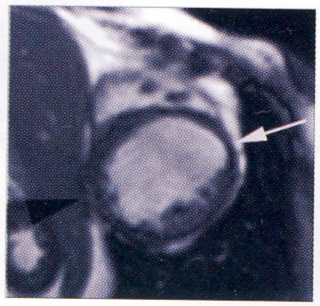

Fig. 2A

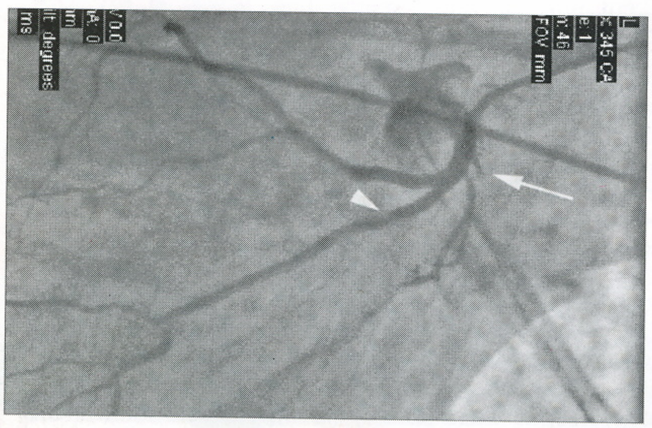

Fig. 2B

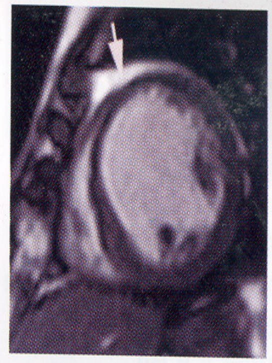

Fig. 2C

Fig. 2D

Fig. 2E

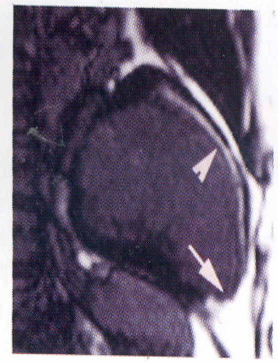

Fig. 3A

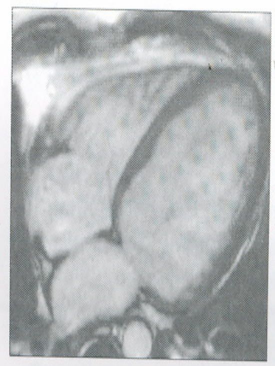

Fig. 3B

Fig. 4A

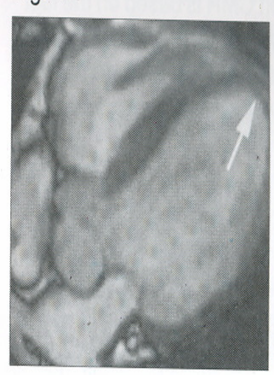

Fig. 4B