

CMR in Myocardial Viability

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Citation

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Abstract

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 revascularization. Reversal of myocardial dysfunction is particularly relevant in patients with depressed ventricular function because revascularization improves long-term survival.

There are many methods available to assess viability, including

Low-dose dobutamine stress echocardiography

Thallium-SPECT

FDG-PET

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 as 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 it indicates the presence of hibernating myocardium, which is likely to improve after revascularization 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 stenoses in the LAD and LCX.

Fig. 1 (A,B). Viability study. The angiogram (A) shows

high-grade stenoses of the LAD (arrow) and circumflex (arrowhead) arteries. A diastolic frame from a mid-cavitary short axis cine study (B) shows thinning of the antero-septal (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).

Figure 1

Figure 1A:

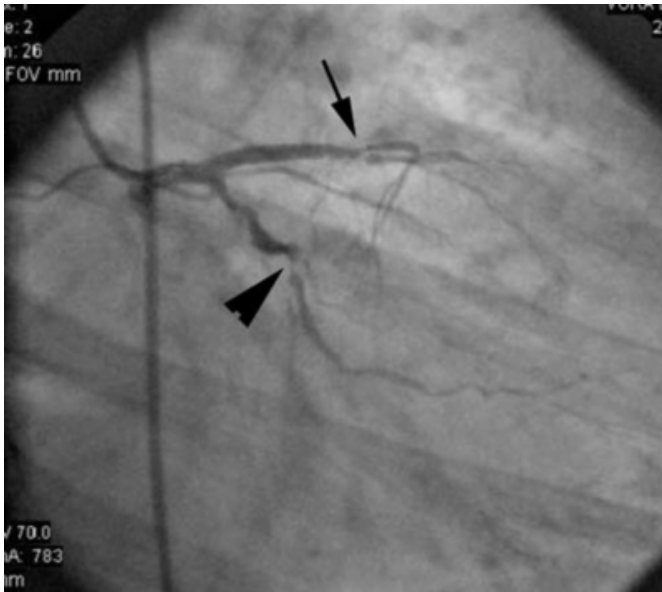


Figure 2

Figure 1 B:

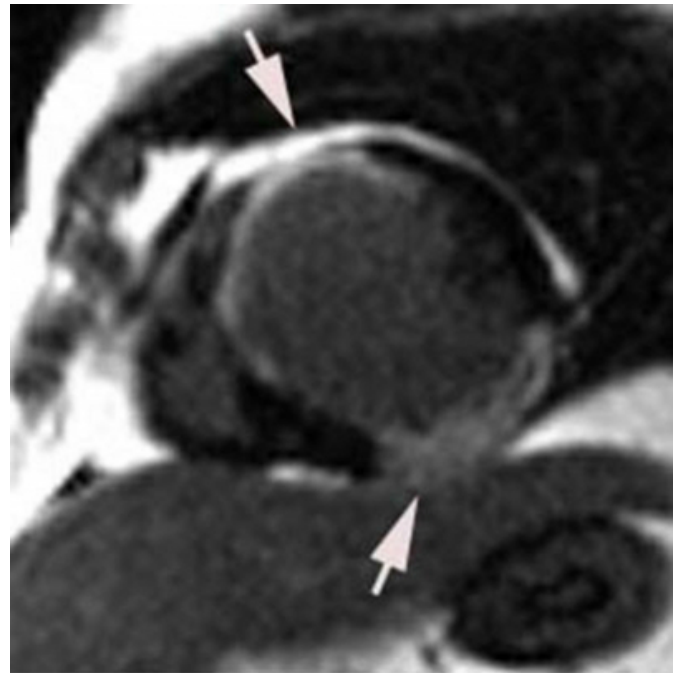


A cardiac MRI was performed for assessing viability

The cine images show marked hypokinesia and thinning of the antero-septal and infero-lateral 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).

Figure 3

Figure 1C:



In view of the full-thickness, transmural involvement, and absence of any viable myocardium, a decision was taken not to revascularize the lesions in this patient.

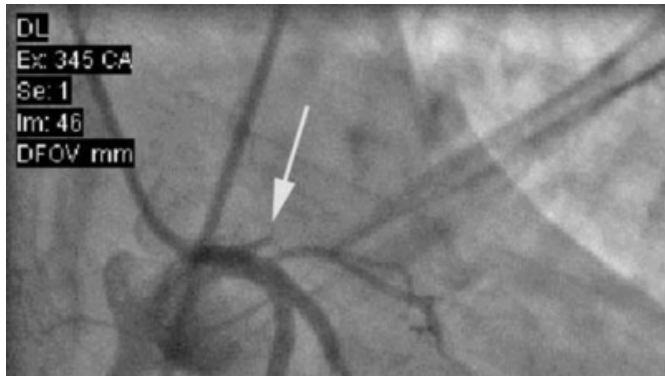
CASE 2

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.

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 they myocardial thickness (arrow). A diastolic frame from a vertical long-axis (VLA) cine study (D) shows marked apical thinning (arrow) with moderate anterior wall thinning (arrowheads) with marked hypokinesia seen at the apex on the cine study. The corresponding viability image (E) shows a transmural infarct (arrow) involving the apex (arrow) with a sub-endocardial infarct involving approximately 50% of the myocardium (arrowhead) in the anterior wall.

Figure 4

Figure 2A:



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 at least 50% thickness. As a result, a decision to revascularize the patient was taken.

Figure 5

Figure 2B:

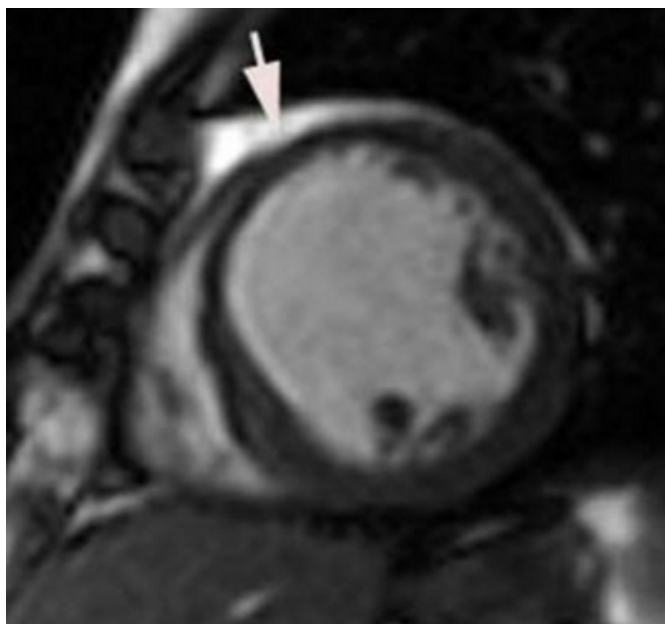


Figure 6

Figure 2C:

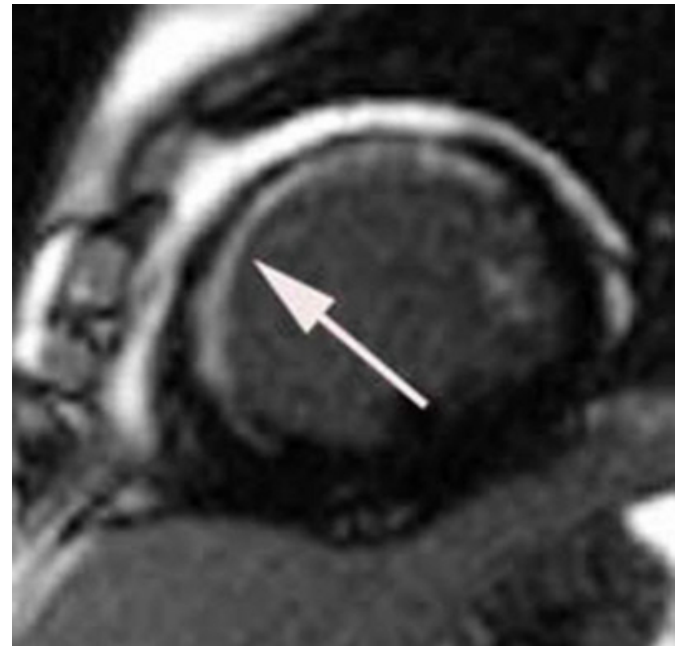


Figure 7

Figure 2D:

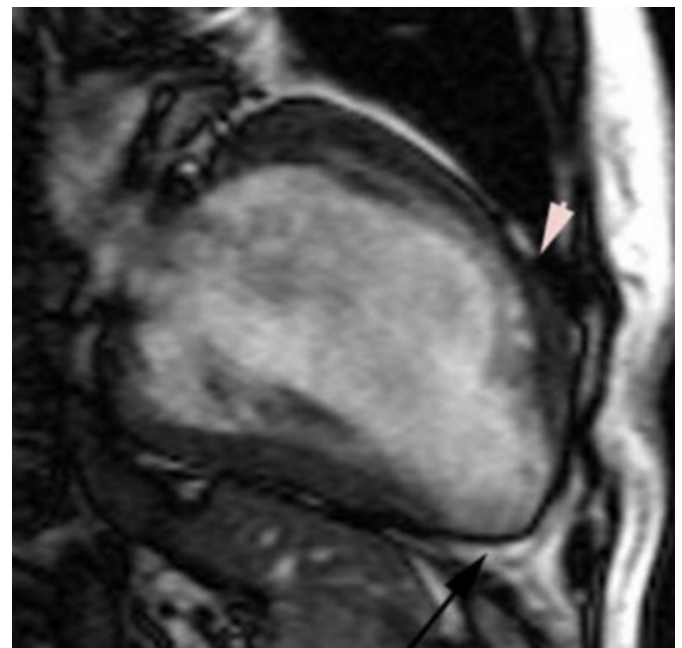


Figure 8

Figure 2E:



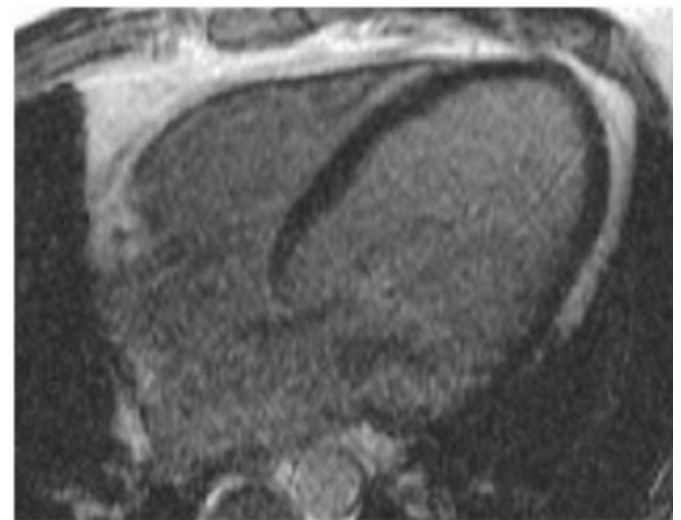
Figure 9

Figure 3A:



Figure 10

Figure 3B:



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, also called “ischemic cardiomyopathy”.

In patients with DCM, CMR either shows no enhancement or in some patients shows mid-myocardial enhancement due to fibrosis (Fig. 3). In patients with ischemic cardiomyopathy, CMR shows infarcts, either sub-endocardial or full-thickness and confirms the presence of 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.

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 diastolic 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 transmurular infarct involving the apex (arrow).

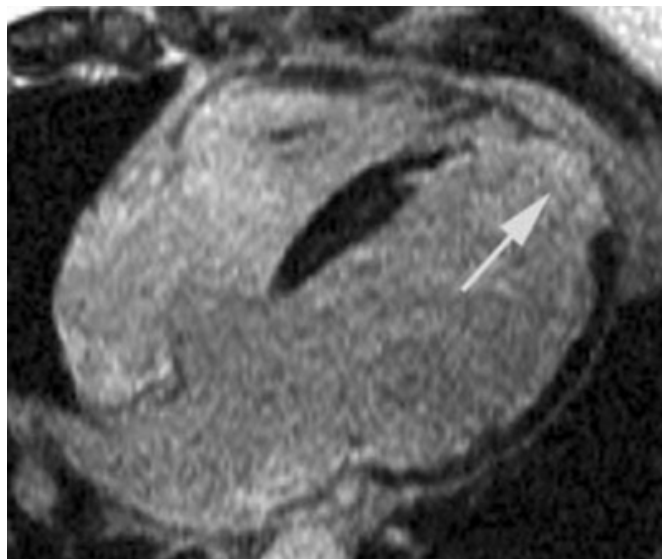
Figure 11

Figure 4A:



Figure 12

Figure 4B:



References

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