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Role of Cardiac MRI in Assessment of Myocardial Viability

Neelima Katukuri

Abstract

Coronary artery disease accounts for a major cause of left ventricular systolic dysfunction. Left ventricular systolic dysfunction is reversible with revascularization in cases of hibernation and stunned myocardium. Revascularization is dependent on not only the presence but also the extent of viability, and a viable myocardium is necessary for functional recovery. For the detection of viability, non-invasive imaging techniques depend on cell membrane integrity, preserved myocardial metabolism or the absence of scar tissue (gadolinium-enhanced magnetic resonance imaging) in areas of dysfunctional myocardium. The late enhancement allows for direct visualization of necrotic or scarred tissue. By measuring the transmural extent of late enhancement, the probability of mechanical improvement can precisely be given. Cardiac MR with LGE can predict recovery of left ventricular systolic function after revascularization.

Keywords: viability, myocardial ischemia, cardiac MR, coronary heart disease, late gadolinium enhancement

1. Introduction

Coronary heart disease (CHD) is the major cause of heart failure. Among patients with ischemic cardiomyopathy, the left ventricular (LV) systolic dysfunction can result from myocardial necrosis and remodeling, myocardial hibernation, or repetitive myocardial stunning. While myocardial necrosis is irreversible, systolic dysfunction resulting from hibernation and stunning are potentially reversible states of ventricular dysfunction. An estimated 20–40% of patients with chronic ischemic LV dysfunction have the potential for significant improvement in LV function after revascularization. Revascularization is dependent not only on the presence but also the extent of viability, and a critical threshold mass of viable myocardium may be necessary for functional recovery and prognostic benefit to occur from revascularization. Assessment of myocardial viability can be done by different methods cardiac MRI, PET metabolism and perfusion, Thallium 201/Tc-sestamibi SPECT imaging and low dose dobutamine echocardiogram.

Previous studies: Studies in laboratory animals have found that, independent of wall motion or infarct age, regions exhibiting gadolinium contrast enhancement at least 10 min after the infusion of gadolinium-based contrast agents coincide with regions of myocardial necrosis and irreversible myocardial injury; regions that fail to enhance are viable [1, 2].

Clinical studies have confirmed that a normal LGE pattern occurs in dysfunctional myocardium that is viable and displays improved contractile function in response to low dose (5–10 mcg/kg/min) dobutamine infusion, while central regions

with enhancement where the infarct is transmural display no contractile activity in response to the dobutamine infusion. Territories that have nontransmural necrosis display a diminished contractile response to dobutamine [3]. LGE as a marker of scar closely agrees with the finding of matching defects on PET viability scanning [4].

Assessment of myocardial viability can be done by different methods cardiac MRI, PET metabolism and perfusion, Thallium 201/Tc-sestamibi SPECT imaging and low dose dobutamine echocardiogram. Each imaging modality has its own sensitivity and specificity as shown in **Table 1**.

Further support for these findings comes from a clinical study of 32 patients with a proven MI who underwent coronary angiography; LGE, performed 3 or 14 months after the MI, accurately established the presence, location, and transmural extent of healed Q wave and non-Q wave MI [5]. Large infarcts were predominantly transmural, while small infarcts were non transmural (**Table 2**). The transmural extent of infarction predicts improvement in left ventricular function. In one study of 24 patients, the extent of dysfunctional myocardium that was not infarcted or had necrosis comprising <25% of left ventricular wall thickness, as established by LGE performed within 1 week of the MI, was the best predictor of global improvement in contractility at 3 months [6].

The extent of enhancement with LGE can predict recovery of left ventricular systolic function after revascularization [7, 8]. As an example, one study of 50 patients with coronary artery disease who had left ventricular dysfunction prior to surgical or percutaneous revascularization found that 33% of myocardial segments in 80% of patients had evidence of LGE; 38% of segments had abnormal contractility [7]. After revascularization, more dysfunctional segments without LGE improved (78 versus 17% with enhancement of more than 75% of the tissue). The likelihood of improvement in regional contractility after revascularization decreased progressively as the transmural extent of LGE increased. The percentage of the left ventricle that was dysfunctional and not enhanced was significantly related to the degree of improvement in left ventricular ejection fraction.

Comparing Modalities				
	Sensitivity	Specificity	PPV	NPV
CMR				
Contrast enhanced	97	68	73	93
Dobutamine stress	94	90	86	92
Total	94	87	84	87
Conventional nuclear				
^{99m} Tc-sestamibi	96	55	74	80
SPECT FDG	89	86	---	---
²⁰¹ Tl rest, reinjection	86	63	69	85
Total	89	68	73	84
Echocardiography				
DSE	76	81	66	89
DSE SRI	82	80	---	---
End-diastolic wall thickness	94	48	53	93
Total	78	78	64	90
PET				
PET-FDG ^{67,70,75,79-81}	89	57	73	90
Total	89	57	73	90

Table 1.
Comparing sensitivity and specificity of various imaging modalities used for assessing myocardial viability.

Cardiac Pathologies demonstrated by CMR	
	Ischemic cardiomyopathy typically demonstrates a subendocardial rim of enhancement which is consistent with a known coronary territory, otherwise known as an infarct. All other enhancement patterns are independent of coronary territories.
	Dilated cardiomyopathy demonstrates enhancement which is typically focal and located deep within the middle layer of the myocardium, and in conjunction with a dilated ventricular cavity.
	Hypertrophic cardiomyopathy also demonstrates enhancement which is typically focal, particularly near the right ventricular insertion into the septum, and in conjunction with a thickened ventricular wall.
	Amyloidosis can demonstrate a variety of patterns, including a rim of circumferential subendocardial enhancement, or overall diffuse patchy enhancement throughout the entire myocardium.
	Myocarditis typically demonstrates enhancement near the epicardial surface or deep within the myocardium, but rarely at the endocardial surface.
	Arrhythmogenic Right Ventricular Dysplasia (ARVD) are demonstrated by fatty infiltration and delayed enhancement in the right ventricular walls and septum.

Table 2.
Various patterns of cardiac pathology demonstrated on cardiac MRI.

Because the size of LGE enhancement may decrease with time, there may be predictive value in assessing non enhanced regions of the ventricle. One study demonstrated the value of measuring the nonenhancing wall thickness to predict improvement in systolic wall thickening [9].

CMR myocardial tagging is another noninvasive method that quantifies local myocardial segment shortening throughout the left ventricular myocardium at sites across the left ventricular wall thickness [10].

1.1 Cardiomyopathy

The high spatial resolution of CMR enables accurate assessment of ventricular volumes, ventricular systolic function (ejection fraction), and myocardial mass and wall thickness. Such analysis is useful in the assessment of patients with heart failure, for the diagnostic evaluation of cardiomyopathy, for prediction of outcomes, and may frequently be the preferred diagnostic test.

1.2 Ischemic versus nonischemic cardiomyopathy

High-resolution evaluation of regional ventricular systolic function can help differentiate between ischemic and nonischemic cardiomyopathy. LGE, which identifies myocardial scar/fibrosis, can also be used to make this distinction.

LGE is present in most patients with ischemic cardiomyopathy (81–100%) compared with 12–41% in patients without significant obstructive coronary disease [10–12]. Although LGE can be seen in ischemic and nonischemic cardiomyopathies, the patterns of LGE tend to be different in the two disorders [11–13]:

Ischemic cardiomyopathy is characterized by subendocardial and/or transmural LGE.

In comparison, isolated mid-wall or epicardial enhancement is strongly suggestive of a nonischemic cardiomyopathy. Mid-wall involvement in ischemic cardiomyopathy involved segments different from those showing subendocardial LGE [11]. **Tables 2 and 3** as shown below:

In two studies, LGE similar to that in ischemic cardiomyopathy was seen in 9–13% of patients with unobstructed coronary arteries [11, 12]. A possible explanation for this finding is recanalization after an MI [12].

LGE also may be seen in hypertrophic cardiomyopathy, myocarditis, sarcoidosis, and infiltrative cardiomyopathies such as amyloidosis [13].

Although, several powerful imaging techniques can be used clinically to identify viable tissue (and to distinguish it from scar) within dysfunctional LV segments

Table 3.
Differentiation of ischemic and nonischemic patterns on cardiac MRI.

CMR Imaging Versus SPECT in Subendocardial Myocardial Infarction

Figure 1.

Short-axis view of a patient who had an occluded left circumflex artery demonstrating normal appearing SPECT, but subendocardial myocardial infarction is easily identified on the CMR image as an area of late gadolinium enhancement in the myocardium. CMR = cardiovascular magnetic resonance; SPECT = single-photon emission computed tomography. Adapted from [14].

subtended by diseased coronary arteries. CMR has a much higher spatial resolution in detecting MI than single photon emission computed tomography (SPECT), which can miss small or localized sub endocardial infarctions (**Figure 1**). The incidental finding of late gadolinium enhancement (LGE), which reflects an area of infarcted myocardium, is independently associated with poor prognosis compared with absence of LGE. The ability to better define myocardial infarctions has led to studies to evaluate myocardial viability and recovery of wall motion after revascularization (myocardial viability). Wall segments with >50% transmural extent of infarction, the likelihood that the segment will regain function after revascularization is <8%. Wall segments with <50% transmural extent of infarction, the likelihood of the segment regaining function is much higher.

This information can be used to stratify patients more effectively and to guide their subsequent treatment. Although we still lack data from ad hoc randomized trials to prove this point unequivocally, a great number of studies in thousands of cases have provided compelling evidence that revascularization of dysfunctional but viable myocardium may lead to reverse LV remodeling and confer prognostic benefits in patients with post ischemic heart failure.

2. Conclusions

The identification of viable myocardium following an MI has important implications with regard to potential benefits following revascularization. Cardiovascular MRI provides a unique tool to assess viability as it offers superior spatial resolution and has emerged as the gold standard for the quantification of myocardial scar via LGE. The presence of viability was associated with survival benefit from coronary artery bypass graft compared with medical therapy alone in patients with severe

LV dysfunction. In the setting of complex coronary disease and concomitant LV dysfunction, a viability assessment via cardiac MRI can provide important diagnostic and prognostic information. Considering the greater spatial resolution compared with PET and the wealth of correlative pathological data, DE-MRI represents the gold standard in the detection of irreversibly damaged myocardium.

Conflict of interest

I have no 'conflict of interest' declaration.

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