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MYOCARDIAL VIABILITY ASSESSMENT USING CARDIAC MRI IN ACUTE CORONARY SYNDROME (ACS) WITH LATE PRESENTATION

Cardiology

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ABSTRACT

Late presenters after acute coronary syndrome pose a significant challenge to the physician. Revascularization without myocardial viability assessment can give angiographic success with uncertain long-term outcomes. Cardiac MRI has evolved significantly in the last decade as a noninvasive tool for viability assessment, and clinical decision-making, provides unique information on chest pain syndrome, and improves risk stratification after an acute coronary event.

KEYWORDS

INTRODUCTION

The prevalence of heart failure has gradually increased in India over the last decade and ischaemic etiology is the leading cause. Ischaemic cardiomyopathy imposes a substantial burden on our healthcare facility. Coronary artery disease in the Indian population has a premature onset, many patients have extensive and aggressive atherosclerosis. The majority of patients, after acute coronary syndrome present late in the health care facility. Even after successful revascularization with angioplasty or coronary artery bypass grafting (CABG), they continue to suffer from Left ventricular (LV) systolic dysfunction.

The role of noninvasive imaging is of paramount importance in assessing this subset of patients before they are taken to the catheterization laboratory. Pathophysiologically myocardial viability refers to those cardiomyocytes that are alive defined by a presence of cellular and microscopic contractile function. In a clinical setting viability means dysfunctional myocardium having potential of recovery after establishment of complete blood flow. Myocardial viability not only guides the revascularization strategy but also helps to prognosticate. Discrimination of hibernating and stunned myocardium should be done properly.

Traditionally Echocardiography is being used for myocardial viability assessment by evaluating left ventricular wall thickness, distribution of scar, etc. However Nuclear imaging and Cardiac MRI have reformed the scenario and should be considered for viability assessment whenever applicable, particularly in patients presented late after an acute coronary syndrome. In addition, cardiac magnetic resonance (CMR) can provide information regarding anatomy, function, tissue characterization, and viability with excellent spatial resolution.

MRI Protocol

Cardiac MRI examination for myocardial viability assessment can be done in both 1.5 as well as 3 tesla. The majority of examinations are still conducted on 1.5 Tesla as 3 Tesla MRI can generate dark banding artifacts, particularly during steady-state free precession(SSFP) cine imaging. Multiphasic array coil is recommended to have an anterior surface coil, posterior coil, and cardiac coil. In-plane spatial resolution will vary with the sequence, field of view (FOV) should be<320 mm, and slice thickness of 6-10 mm.

MRI Sequences

- SSFP or T2 black blood – overview of cardiac surroundings along with evaluation of the great vessel
- Cine imaging- left ventricular wall motion and geometry
- Resting perfusion- assessment of myocardial ischemia
- Late gadolinium enhancement (LGE) for evaluation of myocardial necrosis and scar.
- T2 weighted imaging- evaluation of myocardial edema, hemorrhage
- T1 mapping – myocardial scar tissue
- Early gadolinium enhancement- microvascular obstruction (MVO), intracardiac thrombi.

Cardiac MRI Imaging

CMR characterizes suspected hibernating myocardium by a combination of techniques: LV end-diastolic wall thickness (EDWT), an inotropic reserve of segmental contractile function, and transmural extent of MI with LGE imaging. Wall thinning after myocardial infarction is due to infarct resorption and fibrotic contracture but it can also seen in extensive ischemia. End diastolic wall thickness (EDWT) of less than 5.5 mm has 94% sensitivity and only 52% specificity for functional recovery of the myocardium after successful revascularization. (1)

The low specificity of LV wall thinning can be attributed to the fact that EDWT as a structural variable does not assess the physiological response of the myocardium and that there is a wide variation of EDWT between individuals and even between different myocardial segments in the same individual. (2)

Shaw et al. demonstrated that 20% of dysfunctional and thinned myocardium have limited scar burden and recover after successful revascularization. (3)

Gadolinium-based contrast agents (GBCAs) shorten the T1 relaxation time of the surrounding tissues proportional to the local gadolinium concentration in comparison to the normal state.

Gadolinium, a large, high-density element made metabolically inert by chelation, enters the extracellular space readily after intravascular injection, but it is unable to cross the cell membrane of a normal myocyte. However, when the myocyte cell membrane is damaged (eg, infarction) or if there is an increase in the extracellular space between myocytes (eg, acute interstitial edema or chronic fibrosis or MI), GBCA accumulates in the extracellular space, and its washout is delayed after the injection. Thus, areas of abnormal myocardium (eg, infarcted, fibrotic, inflamed, or infiltrated myocardium) will have elevated per-voxel GBCA concentration and therefore will have a bright signal on T1-weighted images relative to the surrounding normal myocardium.

In clinical CMR, the most common current protocols use a GBCA at 0.1 mmol/kg patient weight and then perform LGE imaging 10 minutes after GBCA injection.

Native T1 Mapping

Recent work suggests that native T1 mapping has value in detecting myocardial viability. T1 time increases both in acute infarct as well as fibrotic scar tissue as compared to normal myocardium. Abnormally

high T1 values can be seen in cardiac amyloidosis as well as in acute myocardial edema. The main advantage of T1 sequencing is the non-requirement of contrast agents.

Late Gadolinium Enhancement (LGE)

LGE can reveal acute myocardial infarction as well as chronic infarction. Many animal studies have shown that LGE corresponds well with histopathology. It can accurately estimate myocardial scar indicative of non-viability of the myocardium. However, in the presence of myocardial edema LGE can overestimate scar burden.

Assessment of transmurality of myocardial scar also predicts viability. The absence of a segmental scar or small subendocardial scar (<25% transmurality) indicates a dysfunctional viable myocardium. The high degree of transmural scar (>75% transmurality) indicates the near absence of viable myocardium. Recovery of myocardial function depends on the amount of viable myocardium. Only the degree of transmurality of LGE (a marker of non-viable myocardium) provides incomplete information rather it should be expressed as absolute or percent relative thickness of myocardium free of LGE.

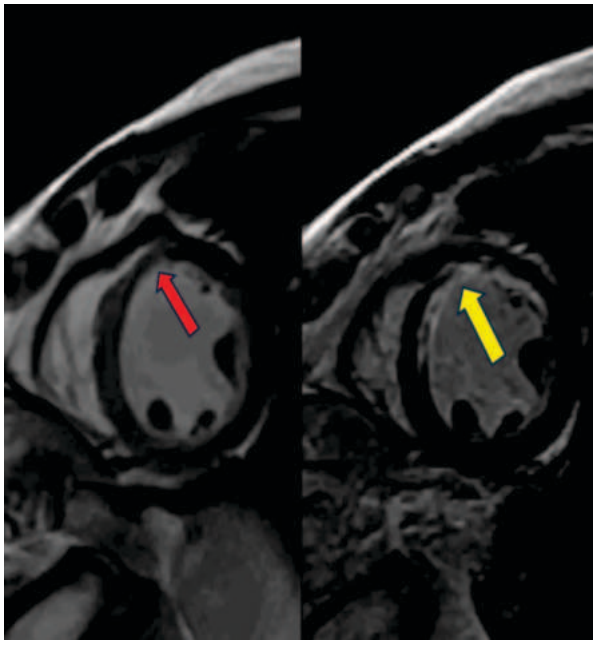

Figure 1- Chronic Anterior Wall MI (Left Picture-Red Arrow), Late Gadolinium Enhancement(LGE) more Than 50% in the Anterolateral and Anteroseptal Wall (Right Picture-Yellow Arrow)

MVO Assessment with Cardiac MRI

Microvascular obstruction (MVO) is one of the dreaded complications following acute coronary syndrome, particularly after revascularization of an occluded artery. It is damage and dysfunction of the myocardial microvasculature along with no re-flow phenomenon in the infarct zone following prolonged ischemia.(4)
MVO typically starts increasing 48-72 hours after myocardial infarction and generally resolves within 6 weeks of an acute event.

MVO typically appears as a central dark focus within an area of early enhancing myocardium signifying a focal absence of contrast enhancement within a site of myocardial infarct. Early (1-3 mins after gadolinium injection) and late (8-10 mins after Gadolinium) gadolinium enhancement (EGE and LGE) can demonstrate MVO. Longer inversion time (T1 around 440 ms) is required for EGE sequences whereas T1 is shorter (around 240 ms) for LGE sequences. (5)

MVO in LGE sequences appears to be smaller and prognostically more significant than early gadolinium enhancement (EGE). MVO is commonly seen after myocardial infarction in an occluded coronary segment but it can also seen after myocardial ablation for ventricular tachyarrhythmia.

Focal myocardial hemorrhage is revealed by a central dark zone on either T_2 or T_2* weighted MRI sequences owing to the paramagnetic properties of hemoglobin breakdown products in the subacute phase following hemorrhage.(6)

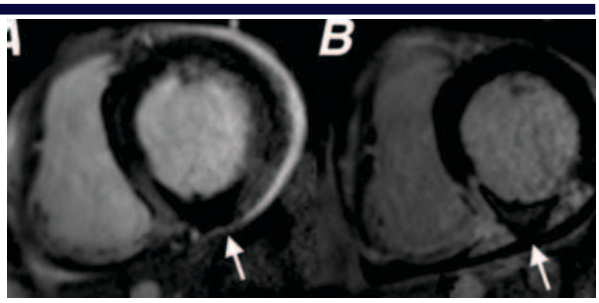

Figure 2- MVO in the Inferior Wall of the Left Ventricle (White Arrow), Along With the Involvement of Some Area of the Right Ventricle (Black Arrow).

Need of Viability Assessment in Indian Patients Before Revascularization

With the increasing number of cardiac catheterization laboratories (cath lab) many Indian patients are now undergoing angioplasty after acute coronary syndrome. Despite successful revascularization, many patients fail to recover from left ventricular dysfunction. Subsequently, they develop heart failure, and heart failure-related hospitalization which eventually increases cardiovascular morbidity and mortality. This increasing prevalence of ischaemic LV dysfunction imposes enormous stress on the healthcare facility of our country.

One of the main reasons for this short-term and long-term failure of revascularization is ignorance and a casual approach toward myocardial viability assessment. Traditionally echocardiography is being used to evaluate LV function and viability following myocardial infarction. It is dependent on the operator and has limited sensitivity. End diastolic wall thickness of less than 6 mm in echocardiography was considered to be a marker of non-viable myocardium but it was recently challenged by Shah et al,(3) who showed that about one-fifth of segments with regional wall thinning caused by ischemic heart disease without evidence of LGE demonstrate LV function improvement after revascularization with reversal of wall thinning.

Increased LV end-systolic volume (> 130 ml) due to abnormal LV remodeling is associated with worse clinical outcomes after Revascularization despite the presence of myocardial viability. (7)

Nuclear imaging (SPECT and PET scan), cardiac computed tomography (cardiac CT), and cardiac MRI have evolved in a gigantic way for the assessment of myocardial viability. These investigations are costly, not available in every cardiac center, and require expertise for interpretation of the findings. Such investigations should be ordered before planned revascularization whenever indicated.

CONCLUSION

A substantial proportion of patients with acute coronary syndrome develop clinical heart failure, which remains a common and major healthcare burden in developing countries like India. Imaging of myocardial viability is an essential tool for the proper use of invasive treatment strategies. However, this notion has been challenged by large-scale clinical trials saying that imaging of myocardial viability failed to deliver effective guidance of surgical revascularization to a reduction of adverse cardiac outcomes. Among the numerous technologies available cardiac MRI has shown greater sensitivity and specificity for viability assessment, identification of complications following ACS, planning for revascularization, and prognostication.

REFERENCES

1. Baer FM, Theissen P, Schneider CA, Voth E, Sechtem U, Schicha H, Erdmann E. Dobutamine magnetic resonance imaging predicts contractile recovery of chronically dysfunctional myocardium after successful revascularization. J Am Coll Cardiol. 1998;31:1040–1048. doi:10.1016/s0735-1097(98)00032-1.
2. Kim RJ, Manning WJ. Viability assessment by delayed enhancement cardiovascular magnetic resonance: will low-dose dobutamine dull the shine? Circulation. 2004;109:2476–2479. doi:10.1161/01.CIR.0000130730.63776.69.
3. Shah DJ, Kim HW, James O, Parker M, Wu E, Bonow RO, Judd RM, Kim RJ. Prevalence of regional myocardial thinning and relationship with myocardial scarring in patients with coronary artery disease. JAMA.2013;309:909–918. doi: 10.1001/jama.2013.1381.
4. Nijveldt R, Hofman MB, Hirsch A, Beek AM, Umans VA, Algra PR, et al. Assessment of microvascular obstruction and prediction of short-term remodeling after acute myocardial infarction: cardiac MR imaging study. Radiology 2009; 250: 363–70. doi: 10.1148/radiol.2502080739.
5. Mather AN, Lockie T, Nagel E, Marber M, Perera D, Redwood S, et al. The appearance of microvascular obstruction on high-resolution first-pass perfusion, early and late gadolinium enhancement CMR in patients with acute myocardial infarction. J Cardiovasc Magn Reson 2009; 11: 33. doi: 10.1186/1532-429X-11-33.

6. Wu KC. CMR of microvascular obstruction and hemorrhage in myocardial infarction. J Cardiovasc Magn Reson 2012; 14: 68. doi: 10.1186/1532-429X-14-68.
7. Bax JJ, Schinkel AF, Boersma E, Elhendy A, Rizzello V, Maat A, Roelandt JR, van der Wall EE, Poldermans D. Extensive left ventricular remodeling does not allow viable myocardium to improve in left ventricular ejection fraction after revascularization and is associated with worse long-term prognosis. Circulation. 2004;110(suppl 1):II18–II22.