Original Research Article

Patterns of late gadolinium enhancement in injured myocardial tissue – A descriptive single centre study

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Abstract

Background: CMR and LGE MR imaging is now been increasing used in diagnosis of various cardiac diseases and the characterization of different patterns of scarring / fibrosis of myocardial tissue. Different spectrum of enhancement can be seen in many pathophysiologic conditions including ischemic and non-ischemic causes**. Methods:** Late gadolinium enhancement MRI was performed in 28 patients in the age group of 13-70 years (mean age group of 42) and the patterns of myocardial delayed enhancement was analyzed in the year 2020 - 2021**. Results:** 14 out of 28 (50%) patients demonstrated late gadolinium enhancement. 3 out 14 (21.4 %) cases were due to ischemic causes and 11 cases (79.6%) due to non ischemic causes. **Conclusion:** Imaging of myocardial disorders encompasses a large variety of conditions including both ischemic and nonischemic diseases. Cardiac MRI sequences like LGE play a critical role in establishing diagnosis, determining prognosis, and guiding therapeutic management Categorization of abnormal delayed myocardial enhancement according to location (subendocardial, transmural, subepicardial, or mesocardial) allows differentiation between ischemic (infarct-related) and nonischemic cardiomyopathies and, in cases of nonischemic cardiomyopathy, narrowing of the differential diagnosis

Keywords: Late gadolinium enhancement**,** Ischemic / non ischemic, Scarring / fibrosis of myocardium

Introduction

Delayed CM imaging traditionally for the evaluation of ischemic heart disease with respect to infarction and viability assessment has been described .However, its increasingly being used in evaluation of nonischemic cardiomyopathies as well. A myriad of causes exist including infections, inflammatory processes, infiltrative processes such as sarcoidosis and amyloidosis and ischemic heart disease Delayed contrast-enhanced cardiac MR imaging can assist in ischemic from nonischemic causes, thus narrowing the differential diagnosis on the basis of various enhancement patterns[1].

Ischemic myocardium usually shows delayed time to peak enhancement , also lower maximal peak enhancement when compared to normal myocardium. Myocardial scar shows increased accumulation of contrast agent along with delayed washout over time, that manifests as abnormal myocardial contrast enhancement[2].

Cardiac MRI has emerged as an imaging modality that allows precise assessment of ventricular function and comprehensive tissue characterization in a single examination without the use of ionizing radiation.

Methods

Study population

Our study included 28 patients who was referred to our department of Radiodiagnosis for cardiac MRI workup with various cardiac conditions from 2020 to 2021. Mean age group was 42 years

Out of 28 patients, 9 patients were male and 19 were female.

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CMR imaging acquisition

Cardiac MRI was performed using 1.5 T MRI machine (GE healthcare 1.5 T HDxt- HD 28). Routine dedicated CMR protocol consists of T1, T2, Cine, double IR , triple IR , perfusion and late gadolinium enhancement sequences. Cine images were acquired using true fast imaging with steady state free precession in cardiac short axis and standard long axis views.

Late gadolinium enhancement was acquired after administration of 10 ml of intravenous gadovist (gadobutrol). Imaging is after 8 minutes after administration of contrast agent by using the following parameters as listed below

Parameter	Description
Type of acquisition	Fast GRE
Timing of acquisition	8 minutes after contrast administration
Repetition time	7.5 msec
Echo time	3.5 msec
Flip angle	25 degrees
Matrix	192 x 192
Section thickness	8
Section spacing	0
Inversion time	250 (adjusted to suppress normal myocardium)
Imaging planes	Short axis , 4 CH and 3 CH views

Results

All datasets with LGE was reviewed and the type of pattern and extend of delayed enhancement was described. Left ventricular LGE was described according to the 17-segment model.

14 out of 28 (62 %) patients demonstrated late gadolinium enhancement. 3 out 14 (21.4 %) cases were due to ischemic causes and 11 cases (79.6%) due to non ischemic causes

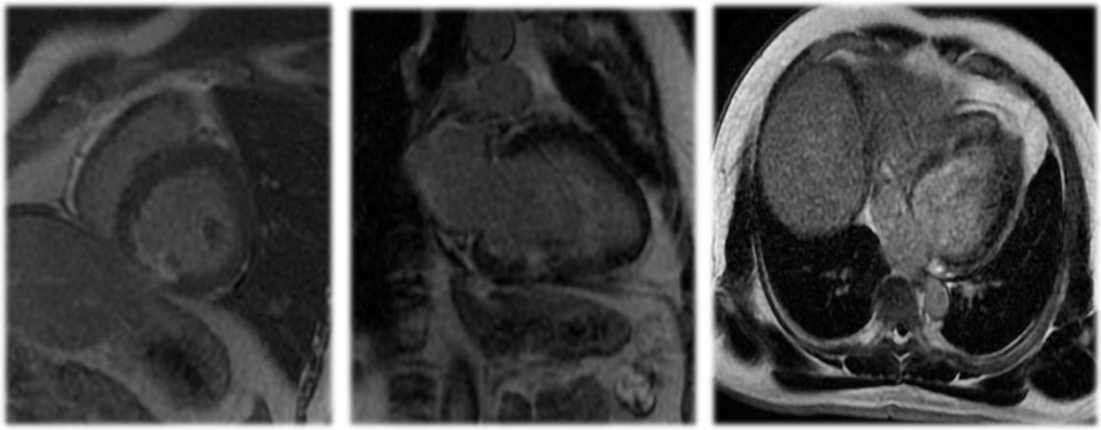
Patterns of delayed enhancement observed were subendocardial, endocardial, subepicardial, epicardial and transmural. Few patients showed more than one type of delayed enhancement.the percentage of involment was estimated in ischemic causes which prognosticated the viability of the myocardial tissue. Similarly the fibrotic burden of myocardial tissue was also calculated in certain non ischemic etiologies.

Type of enhancement	No. of patients
Epicardial	1
Endocardial	1
Subendocardial	5
Subepicardial	3
Transmural	3

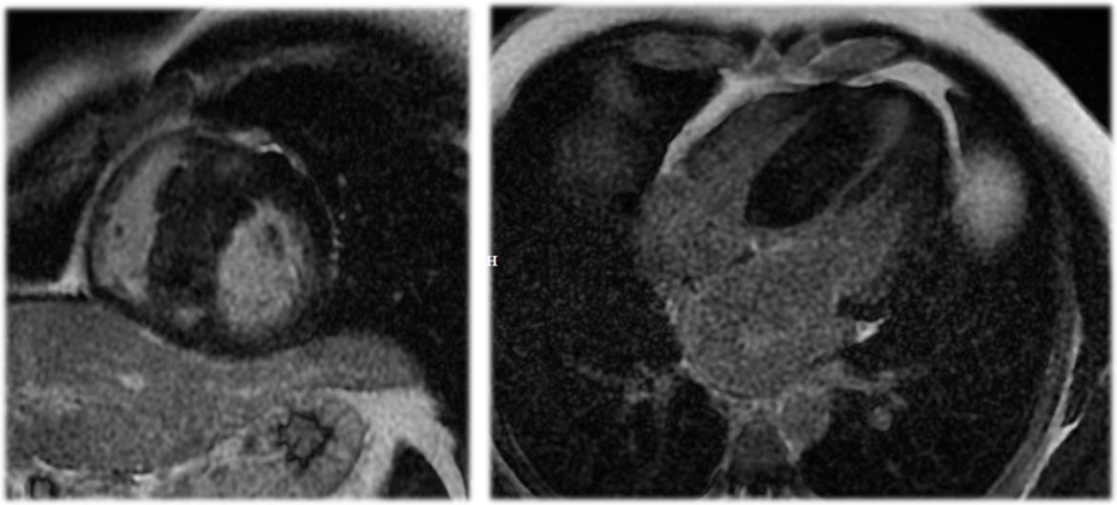
Among the Non ischemic group causes for LGE were the following

Cause of LGE	No. of patients
HOCM	3
Sarcoidosis	2
Amyloidosis	2
Myocarditis	2
Endocarditis	1
Non specific	1

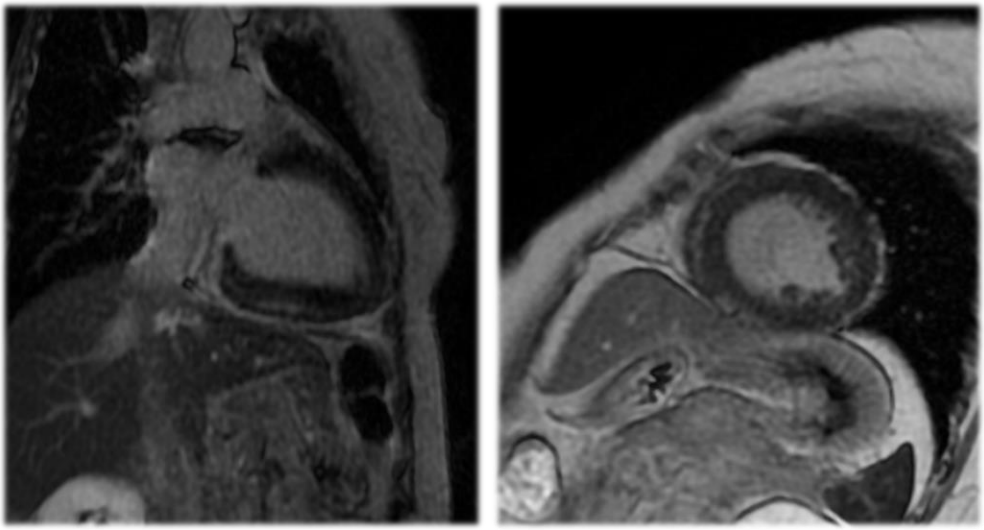
Cases



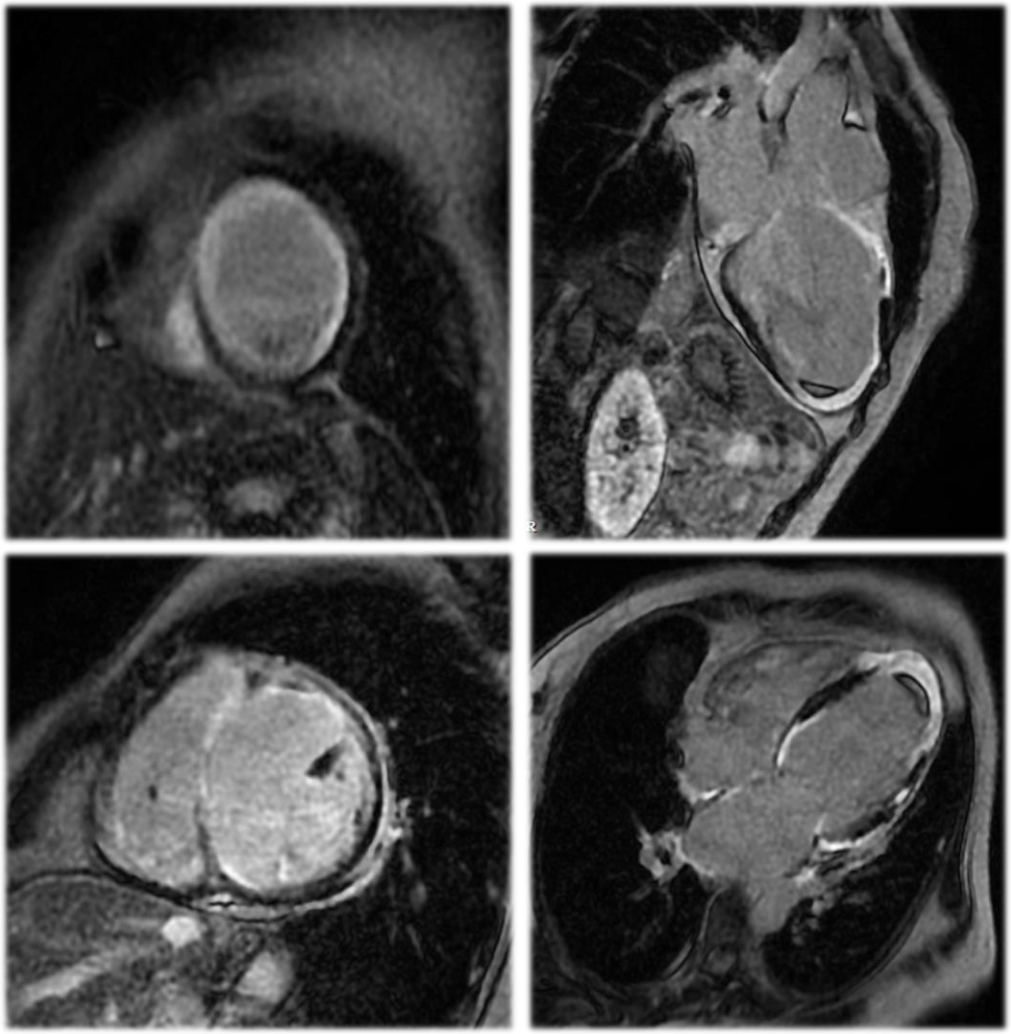
Case 1 - Subendocardial infarction in a 40year-old patient who presented with chest pain. Delayed contrast-enhanced short-axis , 3 CH and 4 CH cardiac MR images obtained show subendocardial enhancement in the inferoseptal and mid inferior segments of left lateral ventricle in RCA territory .Perfusion defect was also noted , features were suggestive of non transmural infarct.



Case 2 -Young 34 year-old patient with HOCM (asymmetrical septal hypertrophy type). Delayed contrast-enhanced short-axis and 4 CH cardiac MR images obtained show Patchy near transmural late gadolinium enhancement seen in the hypertrophied septal wall predominantly in right ventricular insertion sites at anteroseptal and inferoseptal walls basal and mid regions. Fibrotic burden was approximately 5.07 % of LV myocardial mass.



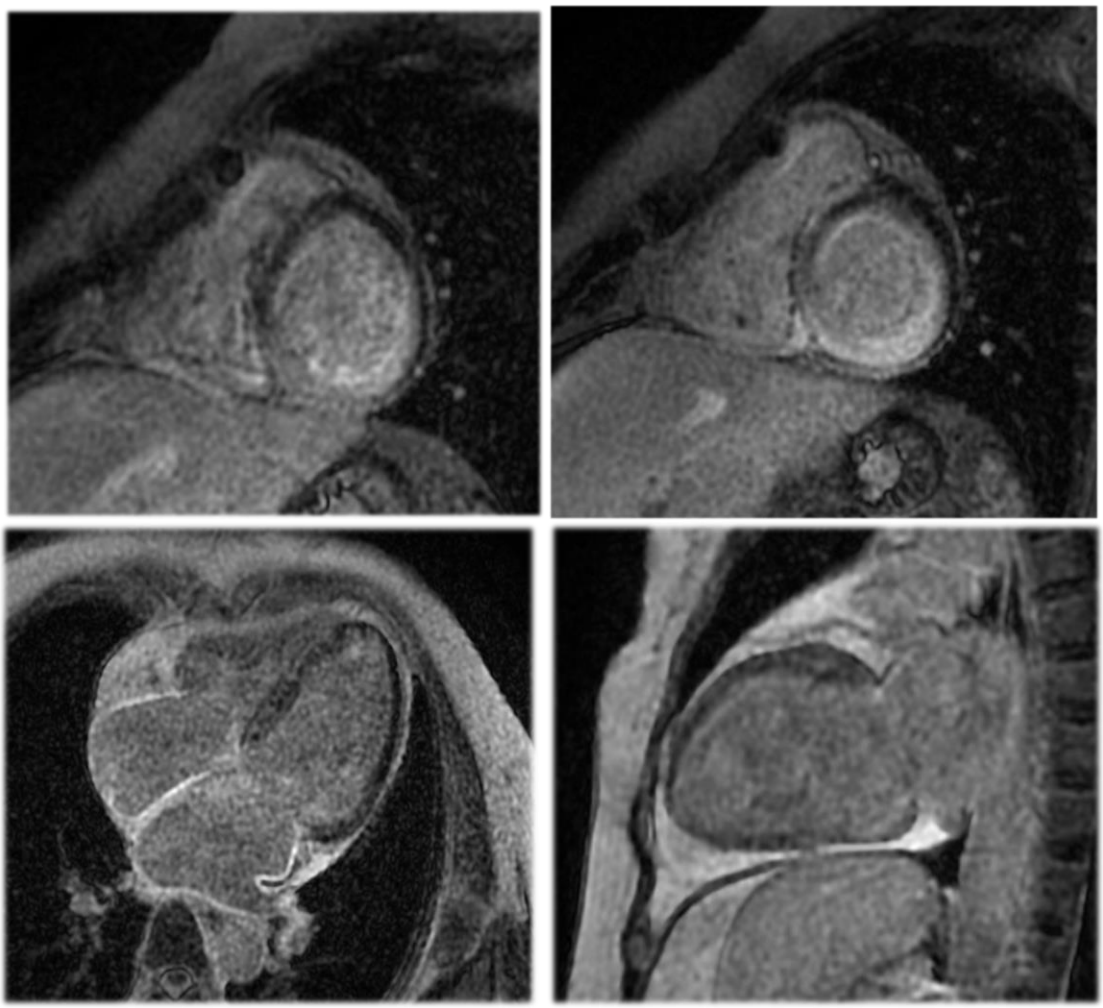
Case 3 – 57 year old patient with rheumatoid arthritis Delayed contrast-enhanced 3 CH and short axis cardiac MR images obtained show Subepicardial delayed enhancement in mid inferoseptal and mid inferior wall of left ventricle – represent Myocarditis.



Case 4-Cardiac sarcoidosis in a 46 -year-old woman with pulmonary sarcoidosis Delayed contrast-enhanced short-axis , 3 CH and 4 CH cardiac MR images show

Non territorial delayed enhancement noted in LV myocardium.

Basal level - Transmural enhancement noted in septal wall, near transmural enhancement in inferolateral wall. Mid cavity level - Transmural enhancement noted in anteroseptal wall, near transmural enhancement in anterior, anterolateral wall, subepicardial enhancement inferior and inferolateral walls, mid myocardial enhancement noted in lateral wall. Enhancement also noted in RV insertion point. Apical level - subepicardial enhancement noted in septal wall. Transmural enhancement is seen involving all walls near apical region.



Case 5 - Cardiac amyloidosis in a 62 year-old woman .Delayed contrat-enhanced short-axis , 4 CH and 3 CH cardiac MR images show Left ventricular wall transmural enhancement noted in basal cavity levels in inferolateral and inferoseptal walls. Diffuse subendocardial enhancement noted in basal, midcavity and apical anterior wall. Patchy enhancement predominantly involving subendocardial surfaces of both ventricles along interventricular septum.There is also enhancement of biatrial walls, interatrial septum.

Discussion

Patterns of delayed enhancement

Myocardium enhancement can be divided into subendocardial, subepicardial, endocardial,epicardial, mid wall and transmural. Ischemic cardiomyopathies tend to involve in a vascular distribution and nonischemic caused delayed enhancement in more than one vascular territory

Enhancement in ischemic causes

Myocardial infarction is related to loss of integrity of cellular membranes of myocardium, myocardial necrosis and microvascular obstruction. First pass perfusion may demonstrate lack of enhancement at site of microvascular obstruction. Delayed LGE MR may show either subendocardial or transmural pattern of enhancement in vascular territory involved. If infarct is transmural and chronic, myocardial thinning may be observed[3].

Enhancement in non ischemic causes

Myocarditis

Myocarditis is inflammation of myocardial muscles. presence of focal LGE in non coronary distribution frequently in the lateral wall along with wall motion abnormalities in correct clinical setting correlates with myocarditis. Subepicardial enhancement is typically seen in majority of cases, although focal transmural has also been reported .Differentiation from infarction is easily made as predominant enhancement in myocarditis is subepicardial .

Signal intensity in areas of delayed LGE seen in myocarditis is often much lower when compared to that seen in myocardial infarction[1,3].

In a study by Mahrholdt et al, contrast enhancement was never seen to originate from the subendocardium, a pattern that is otherwise typical for myocardial infarction[4].

Sarcoidosis

Cardiac sarcoidosis is an infiltrative multisystemic cardiomyopathy. In acute sarcoidosis focal areas of T2 high signal intensity can be noted. LGE. Most common pattern of LGE is midmyocardial or subepicardial enhancement involving lateral and septal basal segments although patterns like transmural and subendocardial are also described. Presence of LGE is a risk factor for arrythmia and adverse cardiac events[5].

In a study by Shimada et al , eight of 16 patients with sarcoidosis and suspected cardiac involvement demonstrated gadolinium enhancement of the myocardium. Endomyocardial biopsies of all eight patients with MR imaging abnormalities were positive[6].

Amyloidosis

it is characterised by deposition of amyloid . Accumulation of various proteins in fibrillar amyloid configuration in myocardial interstitium leads to diastolic dysfunction and restrictive cardiomyopathy. LGE typically shows global subendocardial enhancement usually in a circumferential manner starting from endocardium. Null point of myocardium is reached before blood pool is nulled thereby TI of myocardium is shorter than blood. therefore TI is adjusted accordingly[2].

Hypereosinophilic syndrome

Also known as Loefflers endocarditis is a rare entity that results in eosinophil mediated damage of subendocardium, necrosis , thrombosis and late stage fibrosis of endomyocardial surface of both right and left ventricles. At LGE intense enhancement of subendocarium that is not limited to vascular distribution is observed due to necrosis and fibrosis of cells in that region.thrombus formation can result in areas of stasis and apices[1].

Hypertrophic cardiomyopathy

It is caused by mutation of genes encoding components of sarcomere, principal component of which is collagen. Delayed enhancement is most commonly mesocardial with a linear and patchy pattern often observed in the interventricular septum. Either diffuse or focal hypertrophic changes is observed and restricted diastolic filling of left ventricle.

Additional abnormalities associated with HOCM include systolic anterior motion of mitral valve that results in left ventricular outflow tract obstruction[1].

The LGE of the fibrotic myocardium involving more than 15% indicates that the patient may benefit from Implantable cardiac defribillator placement Implantable cardiac defibrillators are effective in termination of malignant ventricular arrhythmias in this group[7].

Dilated cardiomyopathy

It can be idiopathic or secondary to other diseases including myocarditis, pregnancy Duchenne or Becker muscular dystrophies . Hallmarks of the pathologic process include left ventricular end diastolic diameter more than 55mm and decreased ejection fraction. LGE patterns typically demonstrates a patchy longitudinal mid-wall pattern and usually without subendocardial involvement

Arrhythmogenic right ventricular cardiomyopathy

CMR findings of this condition includes right ventricular dilatation, hypertrophy, regional wall thinning and aneurysms. It can lead to ventricular tachycardia, also sudden death. Fatty and fibro-fatty variants exist. On LGE MR imaging can demonstrate diffuse or segmental replacement of myocardium in the right ventricular free wall[2].

Conclusion

Myocardial delayed enhancement is valuable tool in the diagnosis of fibrosis, scar or nonviable myocardium, Cardiac MRI sequences such as balanced steady-state free precession and late gadolinium enhancement play a critical role in establishing diagnosis, determining prognosis, and guiding therapeutic management.However, delayed myocardial enhancement is not specific for myocardial infarction and can be seen in many other cardiac pathologic conditions and after cardiac interventions. Therefore, clinical history is critical in the evaluation of myocardial delayed enhancement cardiac MR images.

There is emerging evidence for the use of mapping in imaging of myocardial disease. Multiple other new techniques are currently being studied. These novel techniques will likely change the way myocardial disorders are understood and diagnosed in the near future.

Study limitation

Limited number of cases studied.

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