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ASSESSMENT OF LATE GADOLINIUM ENHANCEMENT IN CARDIAC MRI

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Abstract

The aim of this study was to evaluate MRI characteristics of ischemic and non-ischemic cardiomyopathies with late dadolinium enhancement analysis that can provide differentiation between these two cardiomyopathies.

Eligible 96 patients, age range from 26 to 71 years, who showed different and overlapping clinical symptoms, ECG andtransthoracic echocardiography findings that needed further evaluation were included in our study for further evaluation with cardiac MRI. Of the evaluated patients, 47 were females and 49 were males.

The examinations were performed with MRI Scanner 1,5T Siemens Avanto by using 3 channeledSiemens ECG electrodes with retrospective triggering. With the help of PSIR sequence for late gadolinium enhancement evaluation we differentiated ischemic cardiomyopathy from non-ischemic cardiomyopathy, which is crucial for management of patients with cardiac dysfunction. Of the examined 96 patients, 42 patients were diagnosed with ischemic cardiomyopathy, 51 with non-ischemic cardiomyopathy, and 3 patients had non-conclusive diagnosis.

It was found that late gadolinium images in the setting of cardiac MRI were capable of detecting myocardial scars and fibrosis. Moreover, they helped in differentiation between ischemic and non-ischemic cardiomyopathieson the basis of myocardial scar enhancement pattern.

Keywords: cardiac magnetic resonance, late gadolinium enhancement, cardiac function, prognosis

Introduction

The clinical aim of late gadolinium enhancement (LGE) is evaluation of myocardium at risk and conclusion of treatment options. During gadolinium enhanced scans there are different patterns of early and delayed contrast accumulation in normal and injured myocardial tissue, which can be differentiated by myocardial perfusion imaging and delayed imaging for the diagnosis of myocardial scar [1].

Delayed myocardial enhancement MR imaging was introduced in 1989 [2] for the identification of infarcted myocardial tissue. The technique was described with more information in 1993 [3] and since then became a routine sequence in cardiac magnetic resonance (CMR). Imaging with CMR is non-invasive, uses no ionizing radiation, and has high spatial resolution [4].

In case of ischemic cardiomyopathy cardiac MRI is used for detection of a scar. When there are acute changes on T2 Fat Sat edema sequences, but on LGE images no scar is detected, then the ischemic myocardium can be salvable by reperfusion therapy [1].

At T2 Fat Sat sequence in this ischemic myocardium there is reversible T2 accumulation of extra-cellular fluid, but in the presence of a scar that resembles an irreversible tissue with fibrosis, T2 is negative. LGE in this case can determine between acute and chronic myocardial injury.LGE in non-ischemic cardiomyopathies further can distribute distinction between dilated cardiomyopathy, hypertrophic cardiomyopathy, cardiac sarcoidosis, myocarditis, cardiac amyloidosis.

The aim of this study was to evaluate MRI characteristics of ischemic and non-ischemic cardiomyopathies with LGE analysis that can provide differentiation between these two cardiomyopathies.

Methods and Material

We evaluated 96 patients with age range from 26 to 71 years in which 47 patients were females and 49 were males. The examinations were performed with MRI Scanner 1,5T Siemens Avanto using 3 channeledSiemens ECG electrodes with retrospective triggering.Because of the administration of macrocyclicgadolinium contrast, patients had their last meal at least 3 hours before examination.Depending on patient's weight (0.2ml/kg), 10 to 20ml gadolinium intravenously was administered followed with 10ml saline. The contrast media and saline were injected using MRI contrast injector with a flow rate of 2ml/kg. We used 2-chamber, 4-chamber, short-axis, 3-chamber TRUFI-localizers for further evaluation. Short axis T2-TSE FS (Turbo Spin-Echo) was performed to access myocardial edema.Cine sequences were performed for contractile evaluation of the myocardium, and first-pass contrast enhanced perfusion for assessment of myocardial perfusion reserve.

The late gadolinium enhancement sequence was performed with PSIR sequence (Phase Sensitive Inversion Recovery) following 10-15 minutes after contrast administration by using TI (Inversion Time) for nulling the normal myocardium.This sequence helps in differentiation of injured from normal myocardiumwith a very high signal intensity difference between diseased and normal myocardial tissue [1]. We also used Inversion-recovery single shot sequences for data acquisition of the entire left ventricle.LGE-CMR is based on delivery of intravenous gadolinium chelate to the myocardium. In intact myocardium, the contrast agent cannot interact and cannot cross the intact cell membrane.

In case of increased extracellular volume and slower kinetics, there is a possibility of accumulation of gadolinium in the processes of necrosis, fibrosis, infiltration, and inflammation in the late washout phase [5].

With the physical characteristics of shortening T1 relaxation time when using gadolinium, there is a higher signal intensity which represents a sensitive tool of detecting scar both in ICM and NICM [6].The MRI reports were analyzed by two independent examiners.

Results

In ischemic cardiomyopathy with LGE an evaluation of subendocardial or transmural LGE can be made, as fibrosis caused by coronary events and in this situation the ischemic wave fronts start from subendocardium. When there is non-ischemic cardiomyopathy, LGE mostly does not correspond to any coronary artery territory and is often located in the mid-wall.

The differential diagnosis of ICM and NICM is crucial for management of patients with cardiac dysfunction. Of the examined 96 patients, 42 patients were diagnosed with ischemic cardiomyopathy, 51 with non-ischemic cardiomyopathy, 3 patients were with non-conclusive diagnosis**.** 26% of the examined patients with ischemic cardiomyopathy showed extensive LGE in the segments of coronary arteries supply with transmural scar in more than 50% of the myocardium. In 11% of the diagnosed patients with ischemic cardiomyopathy showed subendocardial hypersignal changes or myocardial edema on T2-TSE-FS sequence and on PSIR-late gadolinium sequences these patients showed no late gadolinium enhancement.

This represents reversible ischemic zone and reperfusion therapy should be administered. Figure 1 shows long axis PSIR sequence, late gadolinium enhancement as segmental subendocardial LGE at basal inferoseptal and mid-inferoseptal with less than 20% mural scar. Also, in this case on mid-inferolateral and apical lateral transmural LGE was distributed in more than 70% of the myocardial wall. These patterns of late gadolinium enhancement correspond to the coronary arteries distribution.

Figure 1. Long axis late gadolinium enhancement-cardiac magnetic resonance image in a patient with ischemic cardiomyopathy. Localized subendocardial LGE at basal inferoseptal and mid-inferoseptal with less than 20% mural scar. Mid-inferolateral and apical lateral transmural LGE distributed more than 70% of the myocardial wall

In 37% of the examined patients with NCIM, MRI characteristics of dilated cardiomyopathy (DCM) were seen, and in four or 23% patients there was an ischemic dilated cardiomyopathy with subendocardial LGE. Thirty percents of patients with DCM showed mid-wall enhancement and in 12 patients or 61% of cases no LGE was detected. Cine sequences in DCM can appreciate wall motion disability as global hypokinesia (Figure2) with decreased EF and enlargement of the heart cavities.

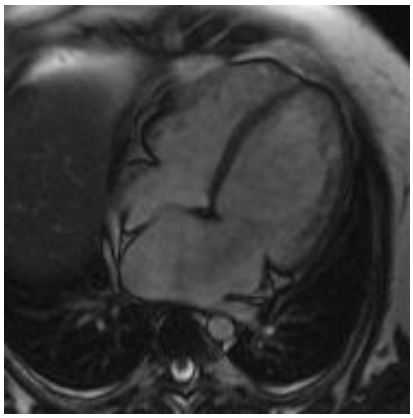

Figure 2. 4-chamber view cine-cardiac magnetic resonance image in a patient with dilated cardiomyopathy shows global hypokinesia with enlargement of the cardiac chambers

With PSIR sequence we evaluated the eventual presence of late gadolinium enhancement. Figure 3 shows mid-wall hypersignal change, zone of late gadolinium enhancement on short axis at mid-anterolateral and mid inferior segment consistent of idiopathic DCM.

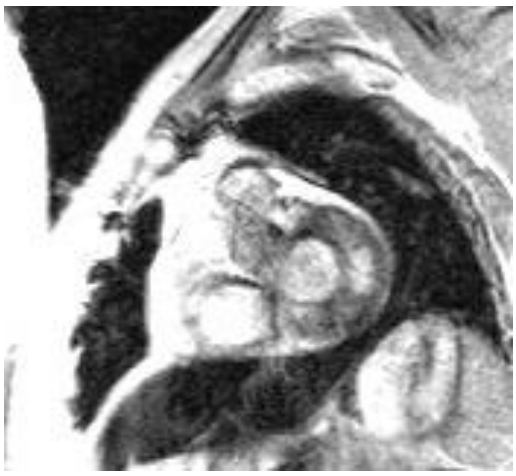

Figure 3. Short axis late gadolinium enhancement-cardiac magnetic resonance in a patient of dilated cardiomyopathy shows mid-wall LGE at mid-anterolateral and mid-inferior segment consistent of idiopathic DCM

Fourteen patientsor 27% of patients had MRI characteristics of hypertrophic cardiomyopathy (HCM) with presence of left ventricular hypertrophy (Figure 4). In 5 patients on cine sequences there were signal voids from turbulence jet across the left ventricular outflow tract (LVOT) with asymmetrical septal hypertrophy due to LVOT obstruction associated with HCM.

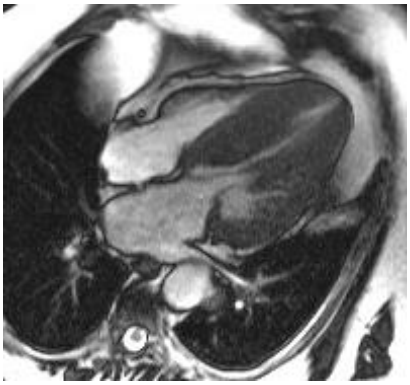

Figure 4. Cine 4-chamber sequence shows asymmetric hypertrophy with dominant septal thickening in a patient with HCM

In 5% of patients diffuse edema was recognized showing presence of inflammatory changes in the myocardium (Figure 5). In 85% of patients with HCM, nodular, focal or diffuse LGE representing fibrosis was shown (Figure 6), especially in those with end-stage HCM. In 63% of examined patients with phenotype of HCM dominantly asymmetric septal hypertrophy was observed.

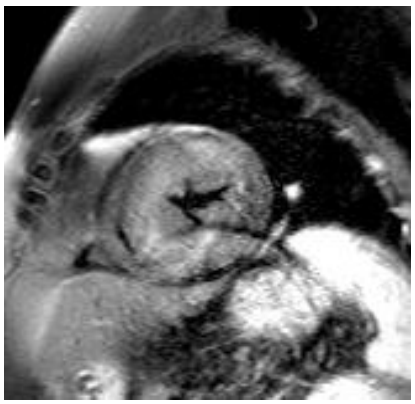

Figure 5. Edema sequence short axis view - diffuse circumferential inflammatory changes in a patient with HCM

One female patient aged 59 years showed symptoms of acute coronary syndrome.Coronarography was performed and no significant stenosis of the coronary arteries was detected. MRI was performed where on cine sequences the middle and apical part of the left ventricle were hypokinetic (Figure 7) and on the systolic phase this was better appreciated as ballooning segment.

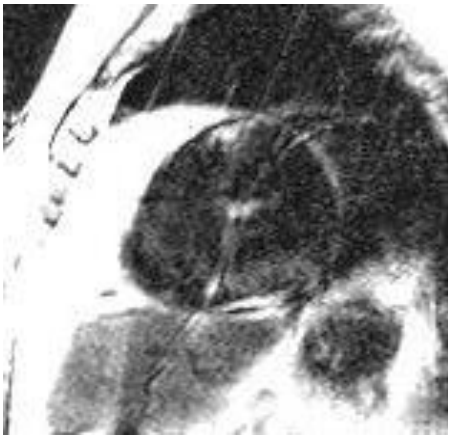

Figure 6.PSIR short axis LGE sequence shows multifocal nodular areas of fibrosis at mid-anterior and mid-inferior segment

On edema sequence a myocardial wall hypersignal was detected on the corresponding mid and apical parts of the left ventricle. On PSIR sequences, no LGE was detectedafter 15 minutes.

With the onset of clinical symptoms, the MRI characteristics of ballooning of the whole mid and apical part of the left ventricle, the edematous changes on T2 TSE-FS and on PSIR sequences no present LGE, a radiological diagnosis of Takotsubo cardiomyopathy was established. After prescribed therapy, control examinations with chest sonography were performed and normalization of the wall hypokinesia was detected.

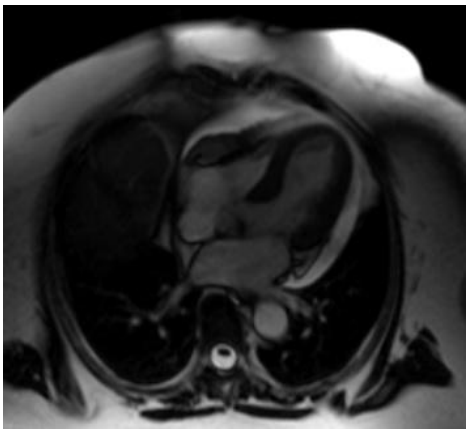

Figure 7. 4-chamber cine-cardiac magnetic resonance image in a case of stress (Takotsubo) cardiomyopathy. There is mid-anteroseptal and apical septal dyskinesis

In 17 patients (33%) non-ischemic cardiomyopathy showed nonspecific symptoms as elevation of hearth markers, clinical symptoms of acute chest pain, ECG abnormalities as AV block, ventricular or supraventricular arrhythmia, and ST changes including severe elevation mimicking acute myocardial infarction.In most cases, echocardiography mostly showed normal systolic wall motion or just mild regional dysfunction and on performed coronarography no significant stenosis was detected or it did not correspond with the myocardial segments of the wall abnormalities. In 23% of these patients cardiac MRI was performed in the acute phase 4 to 7 days after the onset of symptoms where subepicardial and circumferential edema was detected (Figure 8). This corresponded to acute edema pattern present in myocarditis.

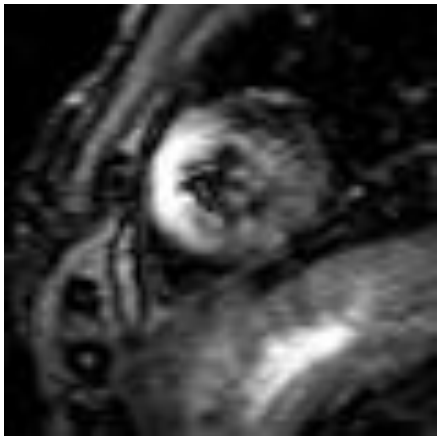

Figure 8. Short axis T2 dark blood-edema sequence image in a patient with myocarditis showed acute edema at subepicardial part in apical lateral and apical inferior myocardium

In 65% of examined patents with myocarditis, LGE was detected as focal subepicardial and mid-wall enhancement andin more severe cases as circumferential and diffuse subepicardial enhancement, accompanied with pericardial changes as part of the inflammatory reaction in the presence of definitive scar/necrosis (Figure 9).

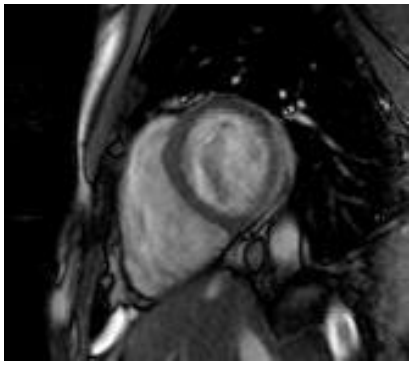

Figure 9. LGE images in short axis showed enhancement, definitive scar presented as circumferential subepicardial LGE with pericardial enhancement on the lateral wall

Discussion

Cardiac MRI (CMR) has been recently considered as an important imaging modality for assessment of patients with cardiomyopathy with up to 94% sensitivity [7].

In the diagnosis of ischemic cardiomyopathyin coronary artery disease,LGE has a sensitivity if 85.7% and specificity of 92.4% [8].

Delayed enhancement shows infarction or fibrotic tissue as an area of high signal intensity, while absence of enhancement indicates viable myocardium likely to improve following revascularization [9].

The pattern of the delayed enhancement is in correlation with coronary vascular territory distribution. The location of delayed enhancement is with subendocardial or transmural pathway [10]. In our presented cases, LGE corresponded with the coronary artery territories and also on cine sequences LGE zones were in synchronization with the wall movement abnormalities. Myocardial scar or fibrosis involving more than 50% of myocardial wall thickness is unlikely to recover contractile function after coronary revascularization [6].

Dilated cardiomyopathy is the most common type of NICM and its characteristics are dilatation of left ventricular chamber and systolic dysfunction, which is predictive factor of progressive heart failure, high risk for fatal arrhythmias and high mortality rate [11].

In cine-CMR, all cardiac chambers are enlarged and decrease in LC ejection fraction (EF) is seen. LV wall thickness is normal or decreased. The results obtained in the larger number of our examined patients corresponded to non-ischemic cardiomyopathy, showed enlargement of the heart cavities, thinned myocardium and ventricular hypokinesia. In LGE, DCM mostly has shown lack of late gadolinium enhancement or eventually presence of mid-wall enhancement or in rarer cases have shown patchy or diffuse striated LGE. The distribution of LGE is not related to any particular coronary artery territory, and pathohistological findings are consistent for fibrosis. With PSIR sequences on the delayed scans we estimated LGE in subendocardial and transmural zones which corresponded to ischemic dilated cardiomyopathy and it helped us in differentiating between ischemic and non-ischemic cardiomyopathy. In most cases with morphological changes of DCM, no LGE was shown and in one third of the patents there was mid-wall LGE. The mechanisms of myocardial fibrosis in DCM are complex and include inflammation, genetic predisposition, micro-vascular ischemia, and neurohumoral changes [12].

LGE-CMR technique may miss a diffuse type of fibrosis, and a certain part of DCM patients may not show LGE [13].

A recent development of T1 mapping technique may display a diffuse type of fibrosis [14]. The mid-wall LGE in DCM correlates with intra-ventricular conduction disturbance, which is predictive of sudden cardiac death (SCD) or ventricular tachycardias (VTs) [15-17].

Hypertrophic cardiomyopathyis a genetic myocardial disorder with an autosomal dominant transmission and is characterized by focal or diffuse left ventricular wall thickening in the absence of dilatation. The most often type of presentation of this cardiomyopathy is asymmetric septal hypertrophy, but it also can be presented as hypertrophy involving the mid-ventricle and apex. Transthoracic echocardiography has been a standard tool for assessment of HCM, but because of spatial resolution incompatibility for visualization of the whole ventricle and quantification of hypertrophy, CMR has become an important tool for these two parameters especially for apical hypertrophy and apical aneurysm [18-20].

Our evaluated patients with HCM showed different types of phenotypic expression, but there was a higher prevalence of asymmetrical septal hypertrophy. Late gadolinium enhancement is a common characteristic of HCM, and can be focal or diffuse into any areas in LV and is significantly related to ventricular tachyarrhythmia and sudden cardiac death (SCD) associated with HCM [18,21].

Patients with HCM very often have LGE at the anterior and posterior RV insertion points. In our examined patients LGE was shown in 85% with multifocal, patchy hypersignal zones in most hypertrophied myocardial regions.LGE represents area of increased interstitial fibrosis, but it can also come as a result of necrosis, scarring because of myocardial disorder [22].

Since 15% to 20% of HCM patients have progressive heart failure [23], LGE in HCM is very important prognostic tool,especiallyin determination of high-risk patients who can benefit from early aggressive therapies.

Stress cardiomyopathy is an acute, severe, but reversible LV dysfunction without a significant coronary artery disease [24].

The major symptom is similar to acute coronary syndrome [24]. Stress cardiomyopathy (SC) is with high prevalence in elderly women, and is possibly triggered by physical and emotional stress [25,26].

The mechanism of Takotsubo cardiomyopathy is not yet established but increased sympathetic activity might play a pathogenic role in the transient myocardial dysfunction in which myocardial edema is shown as a sign of acute and reversible injury in absence of significant necrosis or fibrosis [27,28]. Other histological analyses of the heart in SC showed focal parts of myocardial necrosis with contraction bands in the akinetic area [27, 28].

In the acute phase, the most accurate time for diagnostic evaluation with CMR is after five days. CMR can differentiate reversible from irreversible changes, which is very importantsince it helps in distinguishing stress cardiomyopathy from acute coronary syndrome and myocarditis [24,29,30].

We presented one case where on cine sequence there were mid and apical wall motion abnormalities with no presented area of LGE on the delayed images.

The most often cause of myocarditis is viral infection in which there is myocardial inflammation and immune-mediated damage in myocytes. Symptoms of acute myocarditis are chest pain, ECG changes and elevated cardiac enzymes with similar appearance as acute coronary syndrome. Chronic myocarditis is often a cause of NCIM, and sometimes misdiagnosed as DCM [31].

Myocarditis in CMR is usually presented with myocardial edema, diffuse wall dyskinesia, subepicardial patchy myocardial LGE, and often accompanied with pericardial LGE [32, 33]. T2 dark blood sequence plays an important role in edema imaging when suspicion of myocarditis is established. There is early gadolinium enhancement and prolonged native T1 also suggestive of myocardial edema [33, 34].

On LGE subepicardial enhancement can be shown, mostly at postero-lateral wall, patchy but in severe cases, LGE may be more diffuse and circumferential [35].

We evaluated patients where on edema sequence a significant hypersignal was detected in a subepicardial pattern in one zone or in more segments representing acute changes in which on LGE

sequences these changes corresponded in the same or small matter representing definitive scar. Two patients had edematous changes but not on LGE scan representing reversible changes and 9 cases had no edematous changes but on delayed scans LGE with subepicardial and mid wall-pattern was detected representing chronic changes.

Since the beginning of Covid-19 pandemic the latest studies have showna higher prevalence of heart involvement with complications like myocarditis, acute coronary syndrome, thromboembolic events. With cardiac MRI an assessment of heart involvement can be evaluated when there is elevated troponin especially in the set of multiorgan dysfunction.There is initiative that myocardial injury is a result of the immune-response from the infection rather than of direct viral damaging [36].

Conclusion

Late gadolinium enhancement is a highly important tool for detecting presence of a scar, fibrosis or nonviable myocardium, whether the full thickness of the myocardial segment is involved or one of its layers. In our study LGE scans helped in differentiating between ischemic and non-ischemic cardiomyopathy especially in the setting of dilated cardiomyopathy.

Furthermore, it helped in separating hypertrophic cardiomyopathy fromother conditions of wall hypertrophy where multifocal hypersignal changes can be detected in the RV insertion points or in the most hypertrophied myocardium. It helped in differentiating myocarditis from acute myocardial infarct with the help of T2 dark blood sequence and on LGE images there were changes consistent of those of myocarditis with pericardial involvement. With the definition of the type, pattern and extend of the delayed enhancement in CMR images, prognosis and therapeutic management decision can be made.

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