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Contrast enhanced cardiac MRI findings of myocardial infarction in different infarction duration

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Abstract: Background: Cardiac MRIis an important tool in the diagnosis of myocardial infarction (MI), and in differentiating acute from chronic cases. Studying the myocardial viability of infracted myocardium is very important for decision making regarding coronary revascularization. Objective: The aim of this work was to study contrast enhanced MRI criteria of acute, subacute and chronic MI , and to evaluate the ability of MRI to differentiate between them. Patients and methods: Sixty patients (54 males and 6 females) with MI of different duration were included in the study. All patients were subjected to c MRI using magnetom Sonata 1.5 T Siemens machine. Ten ml gadolinium was given to every patient. Image analysis was performed, then statistical analysis was done using SPSS program 16. P value was considered significant if > 0.05.Results: Left ventricular dilatation and thin infarction wall were seen more in chronic and subacuteMI. Most cases of MI showed hypokinesia or akinesia regardless the infarction duration. In post contrast images, microvascular obstruction (MVO) was seen more in acute MI, while delayed contrast enhancement was more with chronic MI (due to scar tissue). Conclusion: cMRI could diagnose anatomical and functional abnormalities that associate MI. some criteria were more with acute MI and others were more with chronic MI. however, some degree of overlap was seen between both.

Keywords: Cardiac MRI, Myocardial Infarction, Delayed Enhancement

1. Introduction and Aim of Work

Cardiac magnetic resonant imaging (c MRI) has been used for decades to diagnose cases of myocardial infarction (MI) and to assess the viability of the myocardial tissue prior to coronary revascularization or surgical ventricular restoration.

During the early phase of a coronary occlusion, lack of oxygen supply leads to myocardial ischaemia. If ischaemia persists, irreversible myocardial necrosis and death occurs[1]Viable myocardium is the myocardium that may regain function following coronary re-vascularization, while non viable -scaror fibrosis-that occurs as a result of chronic infarction will not regain function. After myocardial infarction, accurate assessment of myocardial viability is important for optimal clinical decision making[2]

The signal intensity (SI) of T1 and T2WI is caused by the edema that occurs in acute MI and the myocardial fibrosis that occurs as a sequel of chronic MI[3]In the study of Choi BW,2006, edema of acute MI is responsible for the high T2WI signal intensity, while fibrosis gives low T2WI signal intensity[3]However, the low SI that is seen in chronic MI is in disagreement with those findings of Ab-del-Aty et al, 2004, in which chronic MI showsT2WI isosignal intensity compared to the normalmyocar-dium[4]Apossible explanation for the low SI of infarcted myocardium in chronic MI is the fat tissue related signal from the lipomatous metaplasia of chronic scar[5]

Different pulse sequences have been tried to improve the accuracy of MRI in diagnosing acute and chronic myocardial infarction and to differentiate between them. Delayed enhancement (DE) cMRIis one of those techniques. Itis rapidly becoming the standard technique for evaluation of myocardial viability in both conditions.Retention of contrast material results in T1 shortening and thus increased signal intensity on T1-weighted images relative to that of the normal myocardium [2]

In addition, there is loss of integrity of the cellular membranes of the cardiac cells in acute myocardial infarction and also, large myocardial infarction may be associated with capillary occlusion and plugging with

cellular debris, named microvascular obstruction (MVO). After acute myocardial infarction, immediate post contrast injection images may demonstrate lack of enhancement at the region of MVO, which sometimes persists on DE images. This is typically representing the "core" of myocardial necrosis.MVO is related to poor patient prognosis as documented by Wu et al, 1998andMather AN et al, 2011 with increased incidence of congestive heart failure and recurrent infarction.[6,7]

MVO is inversely related to the eventual left ventricular ejection fraction (LVEF) and predicts the possibility of recovery of regional wall motion.[8]

DE images in acute myocardial infarction typically show diffuse enhancement of the infractedarea representing the zone of myocardial necrosis.[2]MVO may coexist as a dark hypoenhanced areas within the enhanced infracted myocardium indicating persistent dysfunction and damage.[6,9, 10]

Myocardial fibrosis and scar tissue formation is the end result of chronic MI. Within 8 weeks of the insult, the scar is fully mature[11]My fibroblasts in the cardiac infarct scar can persist for many years[12] This leads to impairment of the cardiac function and contractility. To identify viable versus nonviable myocardium in chronic MI, DEMR imaging is used where scar or fibrosis is depicted as an area of high signal intensity in a coronary artery distribution[2]Hyper enhancement of infracted myocardium on the DE MRI give information about theinfarct's location and size. However, there is the same degree of hyper enhancement of the infracted myocardium on the DE MRI regardless of the infarct's age.[3]Hyperenhancment in acute MI is explained by the diffusion of gadoliniuminto myocytes through the ruptured membrane, whilein the setting ofchronic MI, scar has replaced necrotic tissue and the interstitial space is expanded which is the leading cause ofincreased gadolinium concentration and enhancement.[13]

Both acute and chronic MI show regional wall motion abnormality in echocardiography, and although some studies mentioned that wall thinning is a feature of chronic infarcts due to remodeling of the infracted myocardium [14], this finding is not observed in nontransmural infarcts. [15]So, wall thinning hadbeen used as an indication for the determination of viability,and not for the differentiation of acute MI from chronicMI[3]

MRI provides information about myocardial contractility, but it is not sufficient to assessthe myocardial viability. While area displaying wall thinning or impaired regional myocardial contraction can be clearly identified, this functional information does not indicate whether the myocardium is viable or not.[16]Segmental wall-motion analysis using cine MRI performed in conjunction with DE images has helped to identify the myocardial viability of both acute and chronic MI[16]Kim et al, 2000 have reported that the CE-cine-SSFP sequence could be used for differentiating the acutely infarcted myocardium from chronic myocardial scar with a sensitivity of 95.8%. [8] Absence of delay enhancement in the presence of normal wall motion generally indicates that the segment is viable and will recover contractile function after coronary artery revascularization [16] so,distinguishing betweeninfracted non viablemyocardium from dysfunctional but viable myocardium is clinically important because contractile abnormalities in viable tissue can often be reversed by revascularization procedures with good patient prognosis [17]

Patients with viable cardiac muscle are more likely to have an increased left ventricular ejection fraction and improved survival after revascularization.[18]

One of the complications of MI is thrombosis within the ventricular cavity which mayoccur secondary to poor ventricular function and contractility. Acute thrombi may appear mass like, whereas chronic thrombi often conform to the contour of the cardiac chamber. Usually, thrombi do not enhance after gadolinium administration due to poor blood supply. Rarely, thrombi may demonstrate DEif organized.[19]

The aim of this work is to study contrast enhanced MRI criteria ofacute, sub acute and chronic MI, and to evaluate the ability of MRI to differentiate between them.

2. Patients and Methods

2.1. Type of the Study

This was a cross section observation study.

2.2. Patients

This study included 60 patients (54 male and 6 females) ranging in age from 32 to84years with the median age 58years. Diagnosis of MI depended on clinical signs and symptoms of myocardial infarctionin addition totypical ECG changes.

MI was classified according to its duration into acute (< 2 weeks duration), subacute (from 2:4 weeks duration) and chronic (>4 weeks duration). Twelvepatients presented with acute MI, 10 patients presented withsubacute MI and 38 patients presented with chronic MI.

2.3. Methods

Ethical approval was obtained. cMRI was performed using magnetom Sonata 1.5 T Siemens machine with a dedicated cardiac four element phased-array receiver coil. Post contrast study was performed usingOmniscan (Magnevist) 10 ml.

Protocol used for myocardial infarction:

1. Scout imaging – axial, coronal, and sagittal.
2. Chamber - long axis also called the vertical,horizontal long axis.Steady state free precession short axis cine images to detect wall motion abnormalities.
3. Optional: T2 weighted black blood imaging (at least areas with wall motion abnormalities).

4. Late gadolinium enhancement module: Post contrast images –after 10 minutes- with an inversion pulse set to minimize signal from normal myocardium.
   - 2D segmented inversion recovery GRE imaging during diastolic stand-still.
   - Inversion time set to null normal myocardium.

2.4. Image Interpretation

The images produced were independently evaluated using PACS workstations by a qualified radiologist with good experience in CMRI and cardiologist .The report included; size of the left ventricle whether it was normal or dilated, intraventricular blood clot, ventricular function and contractility. Cine images were used to detect hypokinetic, akineticor normal wall contractility. Significant abnormality of the valves for the presence of any regurgitation .Delayed contrast enhanced images were used to evaluate the extent of the non viable myocardial tissue which appeared as hyper intense in relation to the normal dark myocardium. The percent of LVEF was determined - with the EF of 50% was used as normal value. Then, after reviewing the images and by combining the degree of hyperintensity and myocardial contractility, the best way for treating patients whether SVR or revascularization of the coronary artery affected was recommended.

3. Data Analysis

Statistical analyses were performed using the SPSS for Windows, version 16(SPSS, Inc.Chicago, USA) software package. Demographic, co-morbidities and cardiac abnormality variables were analyzed using Fischer's exact test to assess the significance of the correlation between the categorical variables. In all tests, the values $p < 0.05$ were regarded statistically significant.

4. Results

Males were more affected with MI than females with the ratio 9:1. It was noticed that most females presented with acute infarction while most males presented late after 4 weeks.The duration of MI varied from one week to 520 weeks, 20% patients presented acutely (within 2 weeks), and 16.7% presented from 2:4 weeks and the majority (63.3%) presented after 4 weeks.

4.1. MRI Findings

4.1.1. Size of Left Ventricle

The size of the left ventricle was dilated in 45 patientsand was normalin 15 patients (75%and25% respectively) with the P value= 0.08.

Dilatation was seen in chronic and subacute cases more than in acute cases. (Table 1)

Table 1. *incidence of left ventricular dilatation.*

	Dilated LV	Normal LV
Acute MI	50%	50%
Subacute MI	90%	10%
Chronic MI	78.9%	21.1%

4.1.2. Left Ventricle Wall Hypertrophy

Fifty-seven cases showednormal left ventricle wall thickness (95%), and only3 cases showed wallhypertrophy (5%). Out of those, 2 hadchronic MI and 1 hadacute MI with the P value = 1

Lt Ventricle contractility:

Only one case was missed.Using cine MRI to study wall contractility, 23 cases (38.3%) showedakinesia,14 cases (23.3%) showedhypokinesia,20 cases (33.3%)had mixedakinesia/ hypokinesia and only2 cases (3.3%) werenormal.Akinesia was predominant in both acute and subacute MI (50% and 40% respectively, while most chronic MI showed mixed akinesia/hypokinesia (43.2%) with the P value =0.25

4.1.3. Infarction Wall Thickness

Thin wall of the area ofmyocardial infarction was depicted in39 cases(65%). Most cases of chronic and subacute MI showed thin wall (78.9% and 70% respectively). On the other side, most cases of acute MI(83.3%) showednormal wallthickness with the P value =0.

Mitral valve regurgitation was found in 53 patients (88.6%); 35of them had chronic MI, 10patients had subacute MI and8 patients had acute MI.P value = 0.04

Blood clot within the left ventricle cavity was seen inonly 11 cases (18.3%), most of them (8) had chronic MI with theP=0.89. (fig.1)

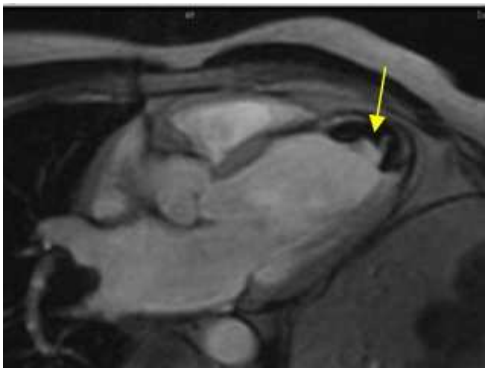

Fig 1. *Axial MRI image.large LV apical crescent shaped blood clot appear as a filling defect (arrow).*

4.1.4. EarlyPost Contrast Images

After I.V. contrast injection, immediate images were taken to detect abnormal enhancement.MVO-detected by non enhanced area of infarction -was seen in 15 cases (25%). More than eighty-three (83.3%) of acute MI showed MVO, 40% ofsub acute cases had MVO, while in chronic MI this was uncommonly seen (only 2.6%) with the P value = 0.

Persistent DCE was seen in 42cases(70% of all cases); 37 in chronic MI (due to scar tissue), 4 in subacute MI and only one in acute MI with the p value=0 (fig.2).

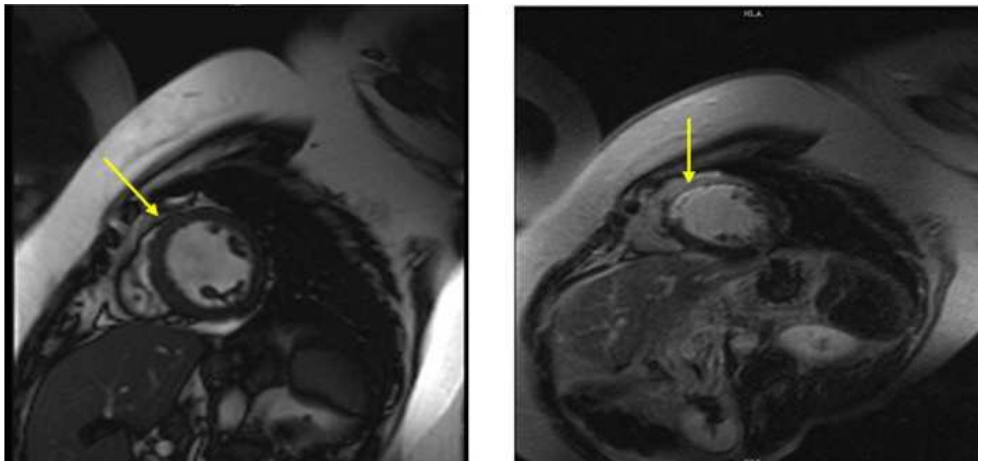

Fig 2. *Coronal oblique MRI:early(a) and late Gad enhancement(b) images show scarring of the apex and distal parts of both anterior wall and anterior septum as hyper intense areas in the late images compared to the early one (arrows).*

Detection of the obstructed coronary artery was inferred from the distribution of the infracted myocardium. Left anterior descending artery (LAD) was the cause of MI in 90% of cases. Right coronary artery (RCA) andcircumflex artery (CX)obstruction represented 5% for each.

Each coronary artery has its own territory. The myocardial tissue supplied by each artery was divided into 8 segments, so the amount of non viable tissue was expressed as a number out of 8 e.g. 2/8, 3/8…etc

Non viable tissue was seen in 55 cases including; acute, sub acute and chronic MI with the p value = 0.31. Table2 shows the percentage of non viable segments in relation to each coronary artery.

Table 2. *Percentage of non-viable myocardial segments*

			infarction_duration			Total
			acute	subacute	chronic	
Non viable_tissue	2/8	Count	0	0	2	2
		% within infarction_duration	.0%	.0%	5.4%	3.6%
	3/8	Count	0	1	5	6
		% within infarction_duration	.0%	11.1%	13.5%	10.9%
	4/8	Count	2	0	10	12
		% within infarction_duration	22.2%	.0%	27.0%	21.8%
	*5/8	Count	2	3	11	16
		% within infarction_duration	22.2%	33.3%	29.7%	29.1%
	6/8	Count	3	5	6	14
		% within infarction_duration	33.3%	55.6%	16.2%	25.5%
	7/8	Count	2	0	3	5
		% within infarction_duration	22.2%	.0%	8.1%	9.1%
Total		Count	9	9	37	55
		% within infarction_duration	100.0%	100.0%	100.0%	100.0%

*Most non viable tissue was 5/8 segments (29.1%)

Recommendation of coronary artery revascularization depended on the amount of remaining viable tissue, the integrity of the left ventricular function and wall thickness. Revascularization was found to be likely in 19 patients, unlikely in 9 patients, of limited benefit in 9 patients and of marginal benefit 2 patients.

Surgical ventricular restoration was recommended in 5 cases of chronic infarction with scar tissue and dilated left ventricle.

5. Discussion

There are different MRI criteria for diagnosing MI. some criteria are more in favor with acute MI, and others are more with chronic MI.Microvascular obstruction has been initially defined as hypoenhancement at 1–2 min after gadolinium injectionand it indicates greater myocardial damage, and poor functional recovery and

prognosis.[6,9,10]In the current study, 25% of patients show MVO which appear as an area of low signal intensity due to perfusion defect in the infracted tissue. MVO is related to acute necrosis and infarction with the p value = 0. Although MVO is seen mainly in acute cases (83.3% of acute MI showed MVO), but it is also seen in subacute and toa much lesser extent chronic cases meaning that it is not exclusively found in acute MI.

According to Mather AN et al, 2011, MVO may be present from day 2 up to1 month, but never after 3 months.In his study, 29 patients showed MVO between 2 days and one week ofMI, which reduced to only 11 patients at one month, and by 3 months, there were no cases identified. The extent of MVO reduced significantly between each time. [7]

In the current study, DE is seen in 70% of the study population;37with chronic, 4with subacute and1 with acute MIwith theP value = 0 denoting that DE is not specific forchronic MI. DE in chronic cases was explained by the development of scar tissue, and in the case with acute MI, it indicates complete infarction of the ischemic regions.Although in many literatures DE was proved to be the ideal imaging modality to distinguish between acute and chronic MI [4, 20], yet other studies found that DE can be detected in both acute and chronic cases(and all stages in between).[13, 21] Distinguishing acute versus chronic infarction may be difficult with these MR imaging methods.[2]In addition,wecan't rely on DE to predict the severity of acute MI. DE doesnot accurately represent the extent of myocardial damage in acute MI because-as reported by Oshinski et al, 2001 "immediately after contrast injection, the enhanced region overestimates the true infracted tissue, on histology, by20 –40%".[21] Also Saeed et al, 2001have suggested that DE does not occur exclusively in regions with myocardial necrosis but also involves the border zones of injured but viable myocardium surroundingacute MI. [22] Other clinical studies have observed an initial overestimation of myocardial necrosis on DE of patient with acute MI that gradually decreases over time on follow up. [23]

In some literature, it isshown that an approach combining DE and T2-weighted MR is a clinically reliable tool to differentiate acute from chronic MI. Although DE is a powerful marker of nonviability and therefore detects infarction at any disease stage, transmural high T2 signal accurately identifies the area of the acute event. [4]

Both patterns of injury present as a regional wall motion abnormality in echocardiography, and although wall thinning is a feature of chronic infarcts [14]this finding is not observed in nontransmural infarcts.[15] In the absence of viable myocardial cells, both acute myocardial infarction (MI) and chronic MI fail to uptake radioactive tracers in radionuclide imaging and thus appear as fixed defects.[24]Finally, although DE cardiovascular MRI accurately detects irreversible myocardial injury, both acute MI and chronic MI exhibit DE regardless of their age. [25, 26, 27]

Other significant parameters in the diagnosis of MI are; Left ventricle dilatation whichis seen more in chronic and subacute cases, Wall thinning is found in 65% mostly chronic and subacute,Mitral valve regurgitation is found in 88.6% ,Akinesia and hypokinesiawere seen in 96.7% of MI cases (P value =0.25).

Application of DE sequences for LV thrombosis detection has been reported to be superior than cine MRI and echocardiography.[28]In the current study, blood clot is seen in 18.3% with (p value =0.89) and is seen more in chronic MI.

Another advantage of MRI is its ability to measure the number of viable and non viable LV segments.Thenumber of LV segments with transmural necrosis has additional predictive value for early LV remodelling independently of microvascular damage.The presence of four LV segments with transmural necrosis represents a powerful predictor of adverse remodeling.[29]In the current study, MRI can accurately estimate the percentage of viable and non viable myocardial segments (p value= 0.31) where29.1% have 5/8 non viable segments. Coronary revascularization is suggested to be of value in 19 cases. All of them have 4/8 or more viable segments.

Cardiac MRI has a great role in evaluating cases of MI by providing -not only anatomical-but also functional information about the myocardium in a single, non stress, non invasive and non ionizing examination. Many sequences are used. The most important of them is gadolinium contrast study. Early and delayed images are taken to diagnose and differentiate acute from chronic MI. however, there is a debate regarding the ability of MRI to differentiate acute from chronic MI, and which technique is better; T2WI or DE with Cine images or both. However, this is of limited application in cases of acute MI on top of chronic, where ECG and coronary angiography cannot detect the acute lesion as mentioned by Sechtem U andMahrholdt H, 2004. [30]

However, in conclusion, MVO is highly diagnostic of acuteMIwith p value =0. DE due to scar tissue, with thin infarction wall (p value= 0for each)-are more with chronic MI.

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