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Impact of cardiac magnetic resonance imaging in the management of post myocardial infarction ventricular septal rupture- a case report

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Abstract : A 61-year old man, a known diabetic and hypertensive, presented to us with worsening dyspnea of 25 days duration with pan-systolic murmur at left parasternal region without thrill. One month ago, he suffered from ST-segment elevation anterior wall myocardial infarction with window period of 24 hours. He was taken up for coronary angiography and subsequently, balloon angioplasty with thrombosuction of the left anterior descending coronary artery was done. He developed sudden onset of dyspnea on the 5th day of admission which progressed to NYHA class III dyspnea when he presented to us. Echocardiogram showed 8 mm ventricular septal defect near apex with left to right shunt with moderate tricuspid regurgitation and pulmonary artery hypertension. Cardiac magnet resonance imaging was performed, which provided detailed information on size and localization of the ruptured septum as well as viability of myocardium. It showed thinning of the myocardium and ballooning in the left ventricular apical region with moderate left ventricular systolic dysfunction. There was defect in the interventricular septum at the apical region of 18 10 mm. Moreover, MRI revealed that the ventricular septal rupture was within the myocardial infarction area, which was substantially larger than the rupture. Severe hypokinesia and greater than 75 late gadolinium enhancement was present in the LAD territory (mid-distal septum and apical regions) suggestive of non-viable myocardium. Subsequently, patient underwent successful surgical closure of the defect (a Gore-Tex patch closure) along with saphenous venous grafting to LAD. He was completely asymptomatic and in NYHA class I at 1 month of follow-up. Our case emphasizes the impact of cardiac magnetic resonance imaging in the management of such rare complication of acute myocardial infarction and its advantages over other imaging modalities.

Keyword : Ventricular septal rupture, Cardiac magnetic resonance imaging

INTRODUCTION:

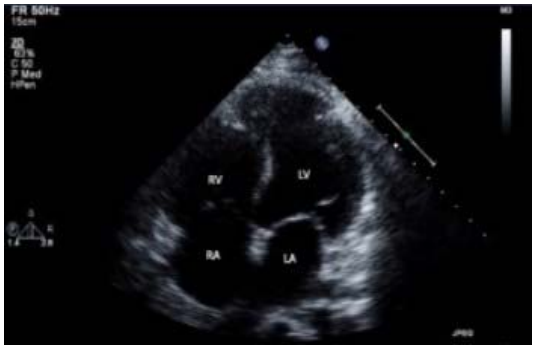
Ventricular septal rupture (VSR) is a rare mechanical complication of acute myocardial infarction and associated with high mortality. Although the gold standard test for the diagnosis of VSR remains invasive ventriculography, echocardiography with color flow Doppler and cardiac magnetic resonance (CMR) are reliable non-invasive tests for the diagnosis. Our case illustrates the role of CMR for the complete delineation of this mechanical complication prior to surgical repair.

CASE DETAILS

A 61-year old man presented with complaints of dyspnea with exertion of 25 days duration. He denied any associated angina, palpitation or syncope. He was a known diabetic since past 10 years and known hypertensive since past 1 year. He was a non-smoker. On presentation to our hospital he was in NYHA class III symptoms. Clinically, harsh pansystolic murmur (grade 3/6) at left parasternal region was present without any thrill. 1 month ago, he suffered from acute severe compressive chest pain with profuse sweating and subsequently admitted at local hospital after almost 24 hours of symptoms onset.

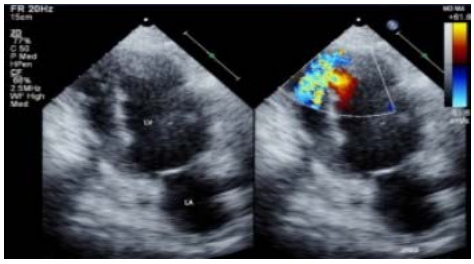
He was diagnosed as acute ST-segment elevation anterior wall myocardial infarction. Subsequently, he underwent coronary angiography which showed thrombotic occlusion of mid left anterior descending coronary artery (LAD) with normal other epicardial coronaries. Balloon angioplasty with mechanical thrombosuction of mid LAD was done. He was started on optimal medical management. On 5th day after that episode, he developed shortness of breath which was rapidly progressive. His electrocardiogram showed poor R wave progression in chest leads with T wave inversion suggestive of fully evolved anterior wall myocardial infarction (Figure 1). Chest x-ray showed mild cardiomegaly with increased pulmonary plethora. There was

MULTIMODALITY CARDIAC IMAGING: ECHOCARDIOGRAPHY AND CMR



Echocardiogram showed thinned out and akinetic mid to distal part of anterior septum with dyskinetic left ventricular apical area. 8 mm ventricular septal defect was seen near the apex with left to right shunt. The peak gradient was 47 mmHg across the defect. Left ventricular ejection fraction was 40%. There was moderate tricuspid regurgitation (TR) with pulmonary artery hypertension. TR peak gradient was 50 mmHg with pulmonary artery acceleration time of 80 millisecond. There was mildly dilated right ventricle and right atrium (Figure 3a and 3b). As septal rupture was near to apical region, it was difficult to estimate the correct size and extent of the defect by echocardiogram; subsequently, cardiac MRI was done.

Figure 3a- Echocardiogram shows apical 4 chamber view with mid-distal septal thinned out and mild right ventricular and right atrial dilatation



right-sided pleural effusion also (Figure 2).

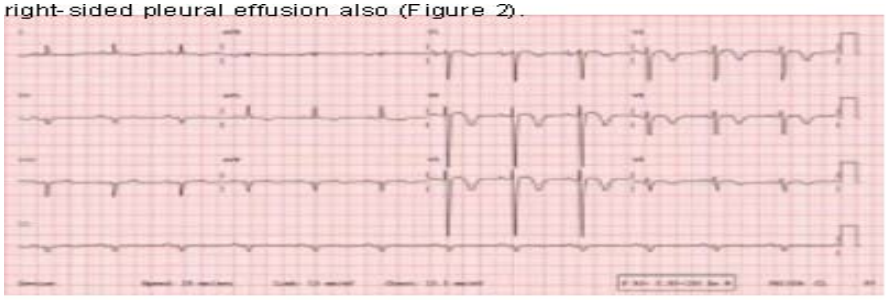
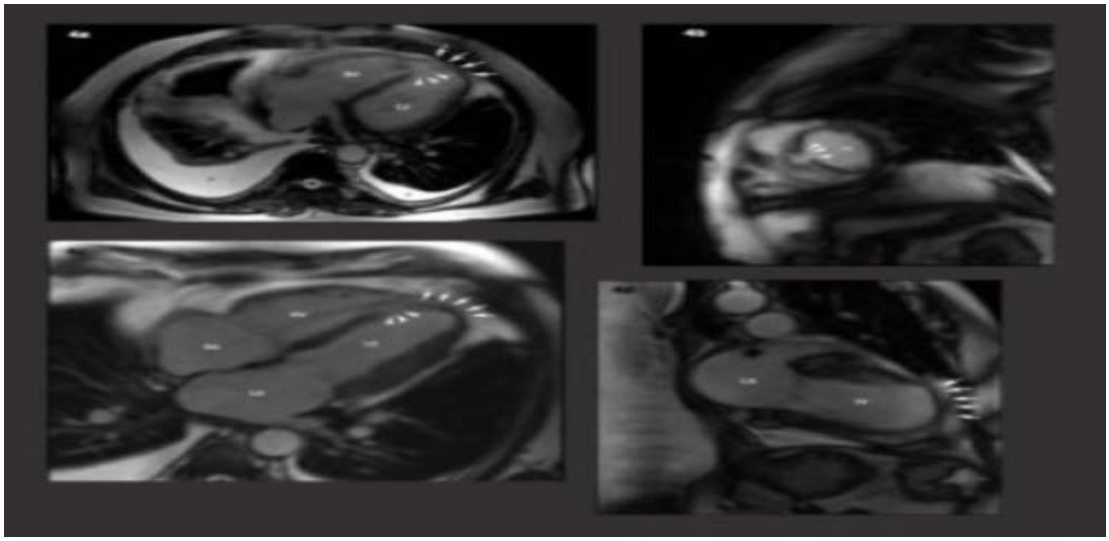


Figure 1- Standard 12-lead ECG shows fully evolved anterior wall myocardial infarction



Cardiac MRI (1.5 Tesla) in transverse, short axis as well as two- and four-chamber view was performed specifically to look for localization and extent of septal rupture (see Figure 4). MRI showed moderate dilatation of both the atria and ventricles. There was thin ning of the myocardium and ballooning in the left ventricular apical region. There was defect in the interventricular septum near the apical region with left to right shunt. The maximum size of the defect measured was 18 × 10 mm. There was moderate left ventricular dysfunction.**Figure 4- Ventricular septal rupture (arrowheads) seen in different views of cardiac MRI. Also seen is thinning and ballooning of left ventricular apical region (arrows). Bilateral pleural effusion is seen in figure 4a (asterisk). [4a- Transverse view, 4b- Short axis view, 4c- Four chamber view, 4d- Two chamber view of LV]**

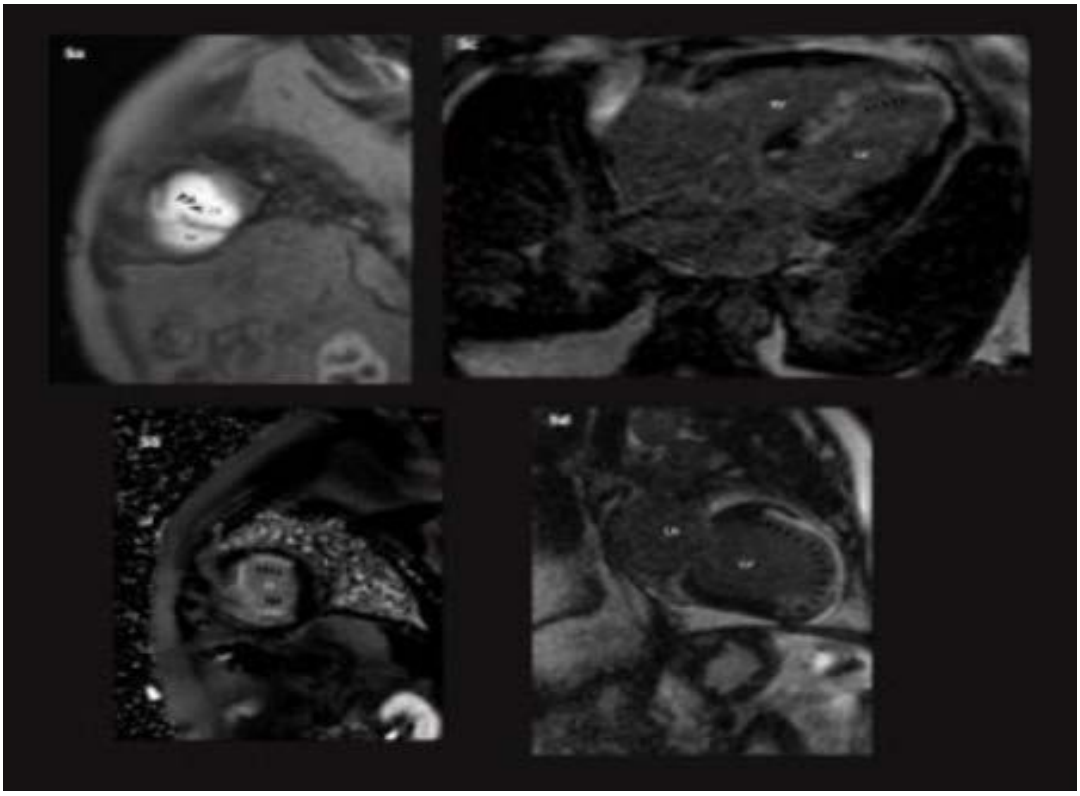
Subsequently, myocardial viability images were taken to look for perfusion defect and myocardial delayed enhancement by PSIR (Phase Sensitive Inversion Recovery) technique. Late Gadolinium enhancement (LGE) was checked about 10 minutes after injection of the contrast agent. Delayed enhancement was suggestive of presence of gadolinium in the pathological tissues. Rest perfusion defect was noted in the anteroseptal and inferoseptal regions from mid-level to apex as well as entire apical area. Severe hypokinesia and greater than 75% delayed enhancem ent was present in the LAD territory (mid-distal septum and apical regions) suggestive of non-viable myocardium (see Figure 5).

Figure 5- Perfusion imaging with late gadolinium enhancement (LGE) in different views shows mid to distal septum and left ventricular apical region involvement (arrows). Figure 5a and 5c show septal defect near apical area (arrowheads).

IMPACT OF CMR ON MANAGEMENT: As the size of the defect assessed by Echocardiogram was small (8 mm), initially it was planned for percutaneous device closure of VSR. But CMR showed the exact size (18 × 10 mm) of the defect along with the morphology of surrounding myocardium. MRI revealed that the ventricular septal rupture was within myocardial infarction area, which was substantially larger than the rupture. In view of these findings, percutaneous intervention was cancelled and patient was referred for surgical closure of VSR. Patient underwent successful surgical closure of the defect (a Gore-Tex patch closure) along with saphenous venous grafting to LAD. Operative findings showed anterior septal defect (25 × 15 mm in size) close to the apex with fibrosed and scarred margins, which were correlating with hat of MRI findings. Patient needed prolonged inotropic supports in the immediate postoperative period from which he was gradually weaned off. Follow-up echocardiography at 1 month showed no residual shunt through interventricular septum with only mild tricuspid regurgitation. He was completely asymptomatic and in NYHA class I at 1 month of follow-up.

DISCUSSION:

Ventricular septal rupture (VSR) is a rare but lethal complication of acute myocardial infarction. Septal rupture occurs more frequently with anterior wall myocardial infarction. It occurs 2-8 days after an infarction and often precipitates cardiogenic shock.(1) In the era before reperfusion therapy, septal rupture complicated 1 to 3 percent of acute myocardial infarctions. Risk factors for septal rupture in the era before thrombolytic therapy were hypertension, advanced age, female sex, and the absence of a history of angina or myocardial infarction.(2) Reperfusion therapy has decreased the incidence of septal rupture. In patients undergoing thrombolysis, advanced age (60 to 69 years), female sex, and the absence of smoking are often associated with an increased risk of septal rupture. In the GUSTO-I trial, there was a nonlinear relation between the systolic and diastolic blood pressures at enrollment and septal rupture. (3) Our patients had various risk factors for VSR like, advanced age, hypertension and absence of smoking. In addition, he had anterior wall infarction with late presentation followed by revascularization, which is more commonly associated with VSR. Our patient developed VSR on 5th day after onset of AMI based on his onset of symptoms. Without reperfusion, septal rupture generally occurs within the first week after infarction.(4) There is a bimodal distribution of septal rupture, with a high incidence on the first day and on days 3 through 5



and rarely more than two weeks after infarction. The median time from the onset of symptoms of acute myocardial infarction to rupture is generally 24 hours or less in patients who receive thrombolysis.(5)The median time from the onset of infarction to septal rupture was 1 day in the GUSTO-I trial (3) and 16 hours in the SHOCK trial.(6)

Symptoms of septal rupture include chest pain, shortness of breath, and those associated with low cardiac output and shock. Acute septal rupture produces a harsh, loud holo-systolic murmur along the left sternal border and a palpable parasternal thrill in half of patients.(7) But with cardiac failure and a low-output state complicating septal rupture, thrill can be absent like in this case. In comparison to acute mitral regurgitation, septal rupture has a loud murmur, a thrill, and right ventricular failure (RV S3 gallop) This patient developed septal rupture in apical region which is common in patients with anterior wall myocardial infarction. Conversely, in patients with inferior myocardial infarction, septal ruptures involve the basal inferoposterior septum and are often complex. Ventricular septal rupture is likely to be associated with total occlusion of the infarct-related artery. In the GUSTO-I study, total occlusion of the infarct-related artery was documented in 57 percent of patients with ventricular septal rupture, as compared with 18 percent of those without ventricular septal rupture.(3) Collaterals are less often evident in patients with ventricular septal rupture.(9) Multimodality cardiac imaging using ventriculography, echocardiography and cardiac magnetic resonance (CMR) imaging can provide complementary information for accurate and complete delineation of the VSR. Although the gold standard test to confirm left-to-right shunting between ventricular cavities remains invasive ventriculography,

it is difficult to define the exact size and morphology of the VSR.(10) Similarly, color Doppler echocardiography accurately localizes the positionbut is less often characterized by severe pulmonary edema. In patients with a low cardiac output, distinguishing between these two entities can be difficult. In addition, severe mitral regurgitation may occur in 20 percent of patients with septal rupture.(8)
of the septal defect. It also demonstrates the presence of multiple defects and coexisting mitral regurgitation.(11) Though small defect, particularly in apical septal rupture can be easily missed by echocardiography. In addition, exact size of apical septal defect is often underestimated by echocardiography, like in this patient. Cardiac magnetic resonance (CMR) with its higher spatial resolution can accurately delineate the anatomy, location, and size of the apical VSR. Late gadolinium enhancement (LGE) provides information of surrounding tissue whether the rupture is located inside nonviable tissue or surrounded by viable tissue for feasibility of device or surgical closure of the myocardial defect. LGE imaging is currently the most accurate and precise noninvasive method to quantify infarct size and morphology in acute myocardial infarction.(12) In addition, cardiac MRI provides complementary prognostic information by correct estimation of the left ventricular (LV) ejection fraction. Also, cardiac MRI offers the potential advantage to assess for myocardial viability that helps in decision towards revascularization in the affected territories. The cutoff of 50% for late gadolinium enhancement is sensitive in predicting segmental contractile recovery after revascularization.(13) Thus, cardiac MRI is superior to other imaging modalities and provides much more information which is extremely helpful to decide further management plan in VSR. Though, cardiac MRI may not be applicable in all patients; as several limitations such as heart rate or circulatory stability exist for performing cardiac MRI in critically ill patients.

CONCLUSION:

In patients with post myocardial infarction ventricular septal rupture, cardiac MRI can provide precise information on localization and size of the defect with respect to the myocardial infarction zone, which is of particular interest before planning for further intervention. We would therefore recommend that cardiac MRI should be considered as an essential imaging tool in such patients. Early diagnosis and prompt percutaneous or surgical management is crucial for survival in VSR, which is highly influenced by selection of appropriate imaging modality.

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