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Case Report

Delayed Surgical Repair of Ventricular Septal Rupture with CABG following Acute Myocardial Infraction- A Case Report

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Key Words : *Ventricular septal rupture, Myocardial infraction, Surgical repair.*

Abstract:

Ventricular septal rupture (VSR) is a rare but often life-threatening mechanical complication after acute myocardial infarction. Although surgical intervention is challenging and associated with high mortality and morbidity, but it is still now the gold standard treatment. Percutaneous device closure of VSR is an option in selected patients with suitable anatomy and when surgery is contraindicated. Optimally medically managed patients who survive at least 4 weeks before elective surgery to generate scar formation at the edges of the defect in a viable infarcted tissue have the greatest outcomes. Here we report a case of VSR following acute myocardial infarction. About 3 weeks after the occurrence of ventricular septal rupture following acute myocardial infraction, surgery was successfully performed with good outcome.

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Introduction:

Ventricular septal rupture is a rare but potentially fatal complication of acute myocardial infarction (AMI), recent literature reported an incidence of 0.17% to 0.21%.[1,2] In 1847, Latham was first to describe ventricular septal rupture at autopsy.[3] However, Brunn made the first antemortem diagnosis of acquired VSR in 1923 and in 1934, Sager established the clinical association between myocardial infraction and VSR.[4]

The advanced age, female sex, stroke history, chronic kidney disease (CKD), single vessel coronary artery disease, extensive MI, ST-segment elevation myocardial infarction, high GRACE risk score and heart failure are the independent risks factors for rupture in patients presenting with AMI.[5] VSR is usually concomitant with ST segment elevation, initially positive cardiac biomarkers, cardiogenic shock, excessive time spent till ballooning or thrombolytic application.[6]

Ventricular septal rupture usually occurs after transmural infarction; it can involve any part of the ventricular septum. Its incidence usually shows a bimodal peak, which is within the first 24 hours or 3-5 days post MI.[7] Rupture develops in any anatomical locations of the ventricular septum and at similar frequencies after anterior, inferior and lateral transmural infractions.[6] VSR most often occurs in apical region of the interventricular septum (Anterior defect).[8]

Post-myocardial infraction VSR occurs in 1% to 3 % of patients with STEMI in the absence of reperfusion therapy and in 0.2 % to 0.34 % of patients who receive fibrinolytic therapy.[9] The incidence of post-MI VSR reduced because of primary percutaneous coronary intervention (PCI).[10] The incidence of VSR increases to 3-9 % in patients with cardiogenic shock as a complication of STEMI.[11]

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Transthoracic echocardiogram is the mainstay for the diagnosis and evaluation of VSR; its sensitivity and specificity have been reported to be as high as 100%. Echocardiography reveals the location and size of the ventricular septal rupture by color Doppler, shows left and right ventricular functions and estimates the right ventricular systolic pressure and left to right shunt. Transesophageal echocardiography is indicated in patients in whom it is difficult to get an adequate view of the myocardium via transthoracic echocardiogram. The key to the management of VSR is a prompt diagnosis and as aggressive approach to hemodynamic stabilization, angiography and surgery.

The ultimate treatment of ventricular septal rupture is a surgical repair.[12] Before surgical repair, it is necessary to restore the circulation in the diseased artery to decrease the hypoxic burden in the infracted area, especially in cases of right ventricular involvement and achieving hemodynamic stability is very beneficial, because 2-3 weeks or more after rupture allow the VSR edges to become firmer and fibrotic resulting in VSR repair that is more secure and easily accomplished. We hereby presenting a case of successful outcome after delayed surgical repair concomitant with CABG of an anterior VSR.

Case Report:

A 60 years old woman got admitted to our hospital on May 2017 with the complaints of orthopnoea for the last few days. She had complaints of sudden severe chest tightness with shortness of breath followed by unconsciousness about 20 days back prior to admission. Then she was diagnosed as acute MI and was treated by medical management for about 12 days. At 5th day of treatment at coronary care unit VSR was diagnosed. Patient's condition improved with no revascularization strategy, she was referred to cardiac center for further management.

She was hypertensive and diabetic. On examination her blood pressure was 105/80 mmHg. Pulse was 92/min, respiratory rate was 26/min and oxygen saturation was 97% without O2 support, there was a systolic murmur was found in apex of the heart but no radiation was found, vesicular breath sound was found in both the lung fields with fine crepitation at base of the left lung but no wheezing. Her laboratory findings showed normal.

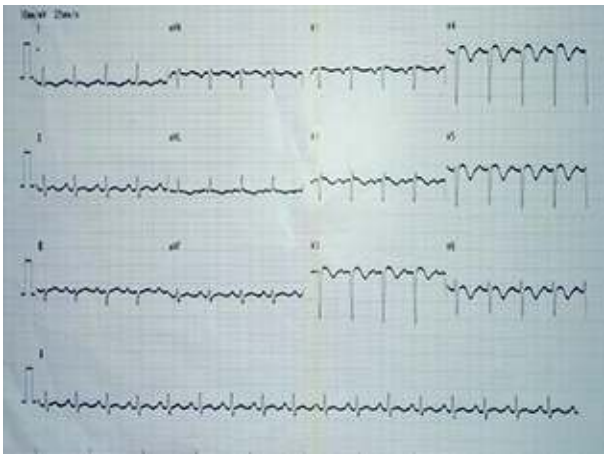

Fig.-1: *ECG showed sinus tachycardia with old antero-lateral infraction.*

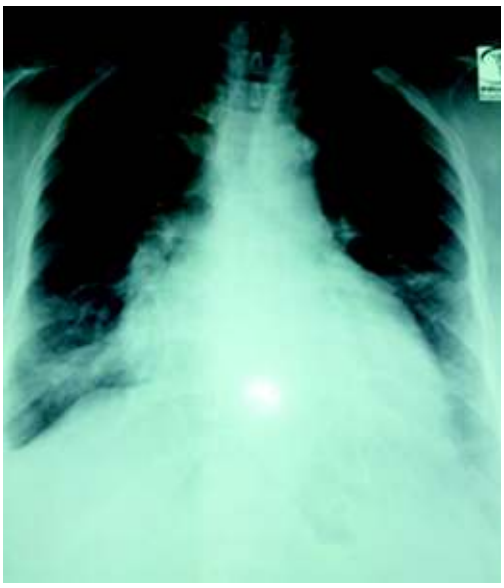

Fig.-2: *Chest X-ray represents Cardiomegaly.*

Echocardiography revealed VSR seen at near apical septal portion with diameter (10×08 mm) with left to right ventricle flow. Apex, mid antero-septum and mid anterior wall of left ventricle was hypokinetic. Moderate LV systolic dysfunction (LVEF-40%) with normal RV systolic function, mild MR, mild TR, PASP-5O mm Hg with mild pericardial effusion. CAG was done day after admission and diagnosed with CAD- single vessel coronary artery disease (mid LAD 100% occluded after D2). In the next morning, patient was sent to operation theatre for surgery.

Per-operative Findings:

Surgery was performed with endotracheal intubation under G/A and with median sternotomy using cardio-pulmonary bypass under moderate hypothermia. Heart was arrested with antegrade cold blood cardioplegia (del Nido). During the procedure, we found RA-Dilated, RV-Dilated, LV-Apex was thin & aneurysmal. LVtomy done, excised aneurysmal part of LV, VSR was identified and VSR margin showed scar formation. VSR repair was performed with Dacron patch through LVtomy.

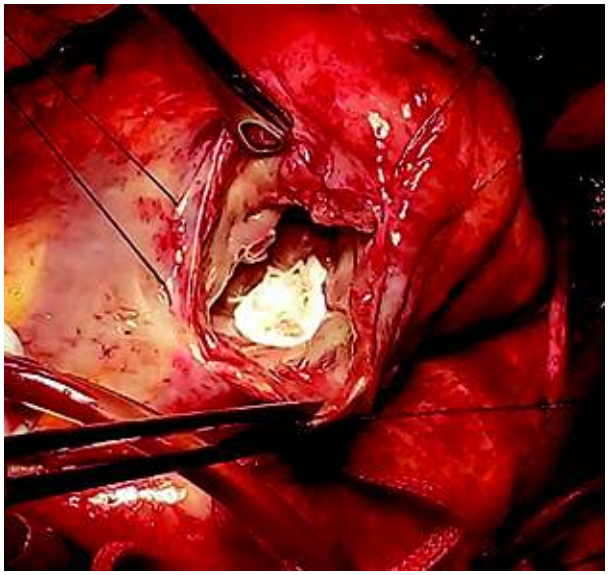

Fig.-3: *VSR closed with Dacron patch.*

Fig.-4: *Closure of LVtomy with PTFE felt strips.*

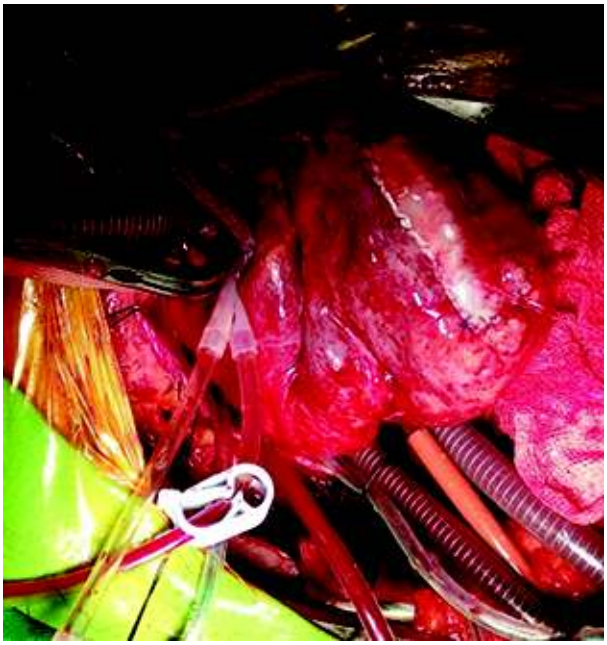

Fig.-5: *LVtomy closure site (PTFE felt stips) covered with pericardial patch.*

LVtomy was closed by direct 4-0 proline sutures buttressed on PTFE felt strips which was covered with pericardial patch. After that, a single bypass was performed by saphenous vein grafting to the distal LAD artery. Proximal anastomosis done with aorta using side clamp.

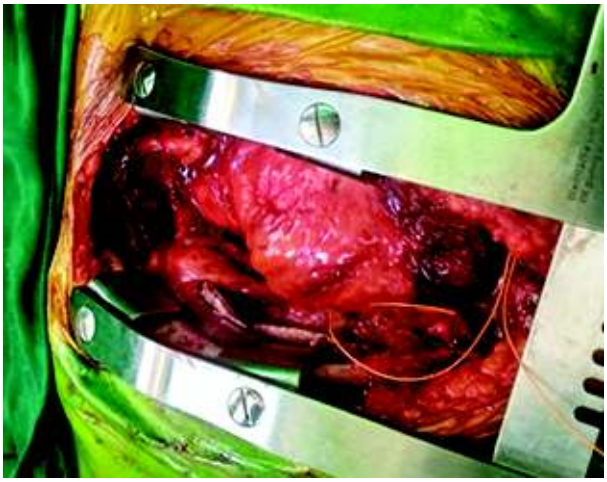

Fig.-6: *RSVG to LAD graft (Distal anastomosis).*

Heart was deaired and gradually weaned from CPB. Decannulation done. Heparin reversed with protamine sulphate. After protaminization hemostasis achieved and wound closed in layers leaving chest drain tubes and RV pacing wire in situ. Patient was shifted to ICU with stable hemodynamics with dobutamine 5 mic/kg/min.

Post-operative status

Patient was extubated on 1st post-operative day and subsequent post-operative period was uneventful. Post-operative biochemical investigations were within normal limits. The post-operative medical management included intravenous inotropes, intravenous diuretics, ACE inhibitors, small doses of beta-blocker, aspirin. Post-operative echocardiography showed IVS patch closure found intact with no residual shunt, no pericardial effusion, PASP reduced to 35 mm Hg. LV systolic function (LVEF) was 45%. Good RV systolic function. Dobutamine was tapered off on 4th POD. Patient was discharged from hospital on the 10th day after surgery with good condition.

Follow Up

At the 5-year follow-up, the patient was doing well and the echocardiography revealed an intact VSR patch and 50% LVEF.

Discussion:

Ventricular septal rupture is a life threatening complication of transmural acute MI. It can involve any part of the ventricular septum. Anterior MI is more likely to cause apical or anterior septal defects, and inferior or lateral MI is more likely to cause basal or posterior septal defects.[10]

From the autopsy, it was found that the anterior VSR was associated with 33% left ventricular infraction and 10% right ventricular infraction, while the posterior VSR is associated with 20% left ventricular infraction and 33% right ventricle. This is related to sudden increase in volume and pressure in right ventricle and often causes right heart failure. Becker make the VSR classification into 3 types. Type I: sudden in onset, slit-like tear within 24 hours. Type II: subacute, erosion of infracted myocardium. Type III: Late presentation, aneurysm formation and rupture, associated with older infracts.[13]

The incidence of VSR depends upon the type of myocardial infraction and is higher in patients with ST-segment elevation myocardial infraction (0.9%). Its occurrence in non ST-segment elevation myocardial infraction and unstable angina is 0.17% and 0.25% respectively.[14]

An interprofessional team, including an interventional cardiologist and cardiothoracic surgeon, is required to treat the VSR. Before surgical repair, to stabilize the patient, the afterload must be reduced with vasodilators, these agents may also decrease the left to right shunt associated with the VSR. Intravenous nitroglycerin is often used to improve myocardial blood flow and vasodilates the vessels. Ionotropic support may be required in patients with low cardiac output.

Intra-aortic balloon pump (IABP) should be considered if pharmacological therapy fails to achieve hemodynamic stability.[12,15] IABP can support these patients by lowering afterload and also decreasing the shunt and at the same time facilitating coronary perfusion.

Whenever patient becomes stable, surgery should be performed.[16] Surgery should be delayed in case of no evidence of cardiogenic shock, having good perfusion and cardiac output, minimal or no signs of congestive heart failure, minimal use of vasopressors, no retention of fluid, normal kidney function.

So surgical repair is considered the treatment of choice. However, surgical treatment remains a challenging operation, often with a complicated course. Surgical repair of VSR was first reported in 1957 by Denton A Cooley using a polyvinyl sponge to plug a posterior VSR.[17] Subsequently the technique of Winard M. Daggett, Jr with infarctectomy and direct closure of VSR from the left side, with or without a patch, became the standard procedure, until Tirone David and Colleagues introduced the infract exclusion technique without infarctectomy.[18,19] Along with repair of VSR, some additional procedure may be required e.g. a) mitral valve replacement b) mitral valve repair c) coronary artery bypass d) resection of the left ventricular aneurysm.

In our case we performed ventricular septal rupture repair with single graft coronary artery bypass with excellent outcome.

The optimal timing for surgery is still on debate. A longer interval before repair has been reported to be associated with better survival.[20] In the acute setting, infracted myocardium is weak and friable and it holds sutures poorly leading to increased risk for tearing and post-operative residual shunt. The improved outcome with delayed surgical

correction may be related to the evolution of the infarct and scar tissue formation which may facilitate the VSR repair. Surgical mortality among patients with inferior MI (70%) is higher when compared to patients with anterior infarcts (30%). This is because of greater technical difficulty and the need for concomitant mitral valve repair in patients with basal septal rupture since they often have coexisting mitral regurgitation.[11] Early mortality outcomes may be significantly better if patients with VSR survive for at least 4 weeks before operation. In the study by Coskun et.al; the mortality rate after urgent VSR surgery within 3 days due to intractable cardiogenic shock was 100%, while all the patients who underwent surgical repair later than after 36 days survived.[21] Coronary artery bypass grafting performed simultaneously improves the collateral flow to the myocardium, contributes to its better recovery and significantly improves survival.[22,23] VSR patients treated medically have mortality rate 24% at 72 hours and increases to 75% at three weeks. The mortality of surgical intervention within 24 hours of acute myocardial infraction is over 60%. The untreated ventricular septal rupture has a mortality of 40-80%. Surgical intervention within seven days of this complication has a mortality of 54.1%. On the other hand, surgery after seven days was a death rate of 18.4%. Late surgical intervention has a good prognosis with hemodynamically stable patient.[24]

Residual VSR can develop in 10-25% of cases even in the best hands. If the defect is small the patient may be observed, otherwise repeat surgery is recommended. Although surgery remains the gold standard treatment for post-MI VSR, percutaneous closure is increasingly attempted, especially in patients with high surgical risk.[25]

Conclusion:

An interprofessional team, including an interventional cardiologist, cardiac anesthesiologists and cardio-thoracic surgeon plays an important role in the management of ventricular septal rupture. The essentials to managing patients with VSR are early diagnosis and an aggressive approach to hemodynamic stabilization. The patient will require long-term cardiovascular rehabilitation after surgery and should be enrolled in a supervised exercise program.

Conflict of Interest - None.

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