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Infero-Posterior Myocardial Infarction with RV Infarction Complicated by Post Infarction Ventricular Septal Defect – A Case Report

Dr. Mahmood Hasan Khan[1], Dr. A M Shafique[1], Dr. N A M Momenuzzaman[1], Dr. Reazur Rahman[1], Dr. Mohammad Mahabub Ul Amin[1], Dr. S M Ziaul Haque[2], Dr. Aparajita Karim[3], Dr. Samsun Nahar[1], Dr. Poppy Bala[3], Dr. Azfar H Bhuiyan[3], Dr. Nighat Islam[3]

[1]Department of Cardiology, United Hospital Limited, Dhaka, Bangladesh.

[2]Department of Cardiology, Salalah Heart Center, Salalah, Oman.

[3]Department of Clinical & Interventional Cardiology, Evercare Hospital, Dhaka, Bangladesh.

Corresponding Author: Dr. Mahmood Hasan Khan, Junior Consultant, Department of Cardiology, United Hospital Limited, Dhaka, Bangladesh.

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Abstract

Background *Post-infarction ventricular septal defects (VSD) are rare (1-2%) but often fatal complications of acute myocardial infarction. These post infarction defects require urgent surgical treatment. The management of VSR is challenging, and its surgical correction is associated with the highest mortality among all cardiac surgery procedures. We report a case in one patient with a ventricular septal perforation as a result of an acute infero-posterior myocardial infarction with RV infarction is presented.*

Case Summary: *A 55-year-old diabetic & dyslipidaemic man presented with AMI (Inferior with Posterior extension & RV infarction) complicated with a large muscular VSR. His left ventricular ejection fraction was 30%, and normal right ventricular systolic function. Cardiac catheterization revealed that the right artery was total proximal occlusion, whereas other coronary arteries had non-obstructive disease. On the course of stabilization & preparing for the surgical correction, the patient went to cardiogenic shock & ultimately expired despite all the recovery efforts.*

Discussion: *Post-myocardial infarction VSR is a rare but fatal complication following acute myocardial infarction. Although these post infarction defects require surgical treatment but they are associated with the highest mortality among the cardiac surgical procedures.*

Keywords: *• Myocardial infarction • Ventricular septal rupture • Cardiogenic shock • Case report*

Learning Points:

• Post-myocardial ventricular septal rupture (VSR) is rare but potentially fatal complication.

• Surgical correction carries highest mortality among all cardiac surgical procedures.

• Clinical presentation predicts the outcomes.

Introduction:

Ventricular septal rupture (VSR) is an uncommon but potentially fatal complication of acute myocardial infarction (AMI). Historically the incidence of VSR was about 1–2% but recent data suggest that it complicates 0.17–0.31%[1,2]. Medical management of VSR is associated with poor outcomes, and its surgical correction carries mortality of 42.9%, which is the highest among all cardiac surgery procedures[3]. Predictors of poor outcomes include old age, female sex, haemodynamics at presentation, and the timing of surgery. Peri- operative haemodynamic shock and incomplete revascularization are the strongest predictors of poor survival[4].

Recent reports have demonstrated the usefulness of echocardiographic studies in patients with coronary artery disease. Echocardiographically, coronary artery disease of the left anterior descending coronary artery has been correlated with abnormal movement of the interventricular septum. Of late,

echocardiographic descriptions of three cases of ventricular septal defect complicating myocardial infarction have been detailed. The purpose of this case report is to describe a patient with an acute infero-posterior myocardial infarction with RV infarction complicated by a ventricular septal defect, which echocardiographically produced a discontinuity of the septal echoes corresponding to the site of the perforation.

Timeline

Days	Events
Day 1	Acute ST-segment elevation, anterior wall myocardial infarction. Initial management with thrombolysis and anticoagulation. Transthoracic echocardiography showed muscular ventricular septal rupture (VSR).
Day 2	Prepared for revascularization due to heart failure and persistent chest pain. Initial stabilization. Repeated Transthoracic echocardiography showed muscular ventricular septal rupture (VSR).
Day 3	Coronary angiogram revealed single vessel coronary artery disease. Intra-aortic balloon pump inserted for better haemodynamic support. Preparing for emergency surgical correction but patient suddenly developed cardiac arrest & ultimately expired despite all the recovery measurements.

Case presentation:

Clinical Course

A 55-year-old diabetic & dyslipidaemic gentleman presented to United Hospital emergency department with a 12-hours history of left sided compressive chest pain with acute shortness of breath for 01 hour on the day of admission. On examination, he looked quite unwell and diaphoretic. His BP was 105/60 with high inotropic supports and was tachycardic with a pulse of 101. His JVP was distended. On cardiac auscultation, he had a grade 5 pansystolic murmur in the left lower parasternal area along with an LVS3. Chest auscultation revealed fine bibasilar crackles.

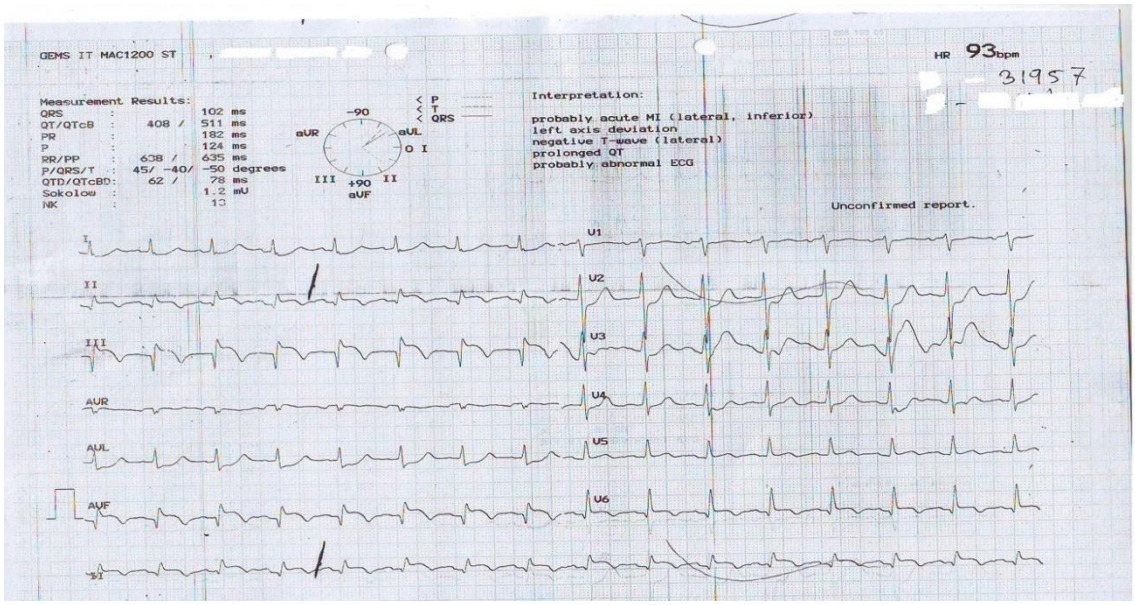

Figure 1: 12-lead electrocardiogram showing the acute inferior myocardial infarction with posterior extension

He was diagnosed as a case of AMI inferior with posterior extension with RV infarction with acute left ventricular failure with cardiogenic shock & was immediately transferred to coronary care unit. A decision of thrombolysis was taken considering the financial condition of the patient. Then he was thrombolyzed with Inj. Streptokinase. But despite thrombolysis, the patient's clinical condition did not improve. He complained of persistent shortness of breath. His haemodynamics were maintained by high inotropic supports. His echocardiographic examination revealed the underlying complication.

Echocardiographic Studies

The septum, inferior & posterior walls were hypokinetic. The mitral valvular motion was normal. No prolapse of the mitral valve leaflets was demonstrated. An M-mode echocardiographic scan from base to apex revealed a normal aortic-root dimension of 3.28 cm/m^2 and a normal aortic valve opening of 2.26 cm. The left atrium was moderately dilated, measuring 4.41 cm/m^2. The septum revealed a discontinuity in the muscular septum close to the atrioventricular junction. This finding was reproduced by repeated echocardiographic scans. His echocardiographic findings are consistent with AMI inferior & posterior wall. The echocardiography study also showed discontinuity at posterior- septum in basal segment measuring 17 mm in LV side & 7 mm in RV side with PPG: 78 mm Hg, severe LV systolic dysfunction with LVEF: 30%. There were features of pulmonary artery hypertension suggested by tricuspid regurgitation (PASP: 51 mmHg).

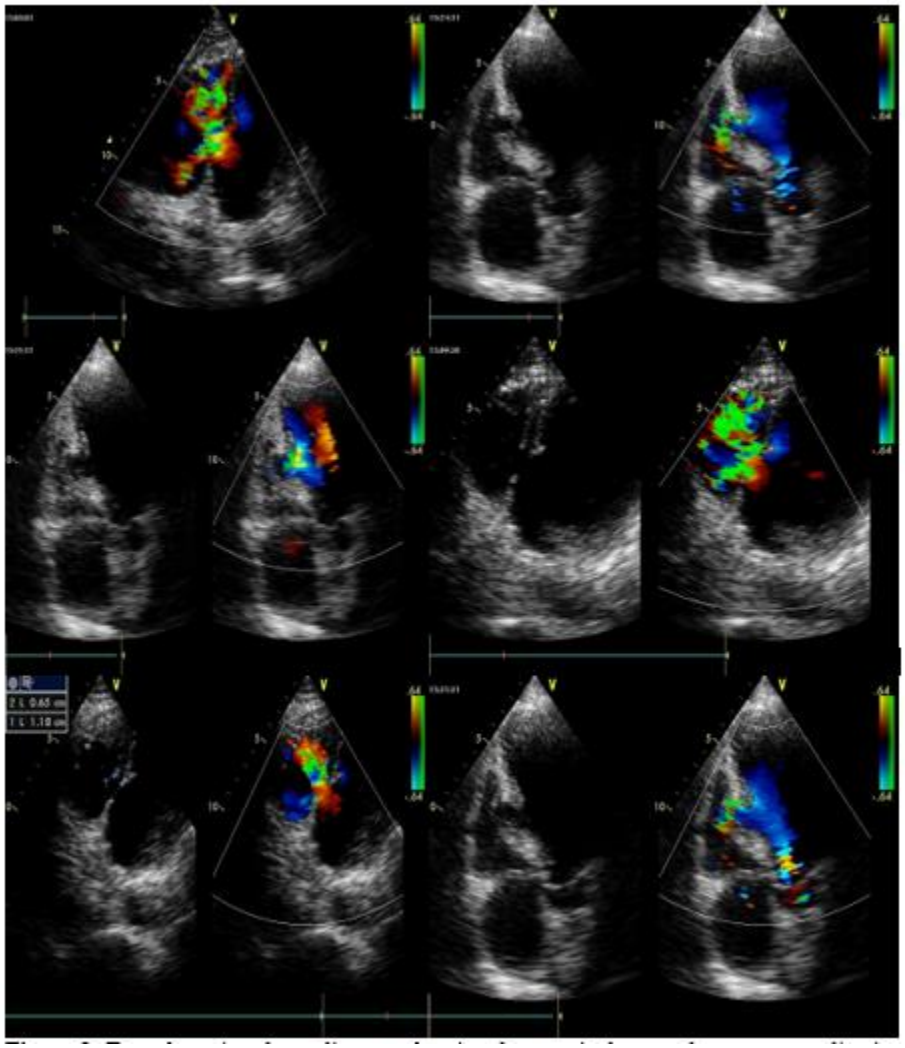

Figure 2: Transthoracic echocardiogram showing the ventricular septal rupture complicating acute inferior myocardial infarction with posterior extension.

As his clinical & haemodynamical condition did not improve a decision of emergency cardiac catheterization was made. Consulted with the patient's family, briefed about the clinical condition, they also agreed to proceed. His coronary angiogram revealed single vessel disease (totally occluded right coronary artery from the proximal segment).

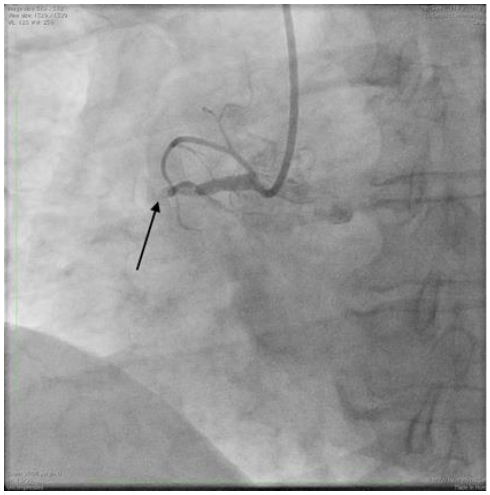

Figure 3: Coronary angiography showing the totally occluded right coronary artery form proximal segment.

After coronary angiogram, the family was briefed in details about the condition, treatment modalities & possible outcomes of the patient. He was advised for emergency CABG with repair of the VSR. Family agreed & the process for emergency surgical procedure was initiated. Intra-aortic balloon pump was inserted to maintain his haemodynamics in addition to inotropes. During this preparation for emergency surgical correction the patient suddenly developed cardiac arrest & ultimately expired despite all the recovery measurements.

Discussion:

In 1845 Latham described a post-infarction ventricular septal rupture at autopsy, but it was not until 1923 that Brunn made the first ante-mortem diagnosis. Sager in 1934 established specific clinical criteria for diagnosis, stressing the association of post-infarction septal rupture with coronary artery disease. Ventricular septal defects rupture can occur from a few hours to a few weeks post MI but occurs most commonly by the end of the first week. It is usually followed by low cardiac output and multi-organ failure[5]. Reviews[6] reveal that nearly 25% of patients with post-infarction septal rupture and no surgical intervention died within the first 24 hours, 50% died within 1 week, and 80% died within 4 weeks; only 7% lived longer than one year. Of 25 patients with post-infarction ventricular septal defects treated medically, 19 died within one month[7]. Thus, the risk of death following post-infarction ventricular septal defect (VSD) is highest immediately after infarction and septal rupture, and then gradually declines. Interestingly, there are reports of spontaneous closure of small defects, though this is so rare that it would be unreasonable to manage a patient with the expectation of closure. Recently, the SHOCK Trial (SHould we emergently revascularize Occluded Coronaries in cardiogenic shocK) provided intriguing data on the outcome of medically managed patients with shock and post-infarction VSD. The multi-

institutional study tracked 55 patients in cardiogenic shock from post-infarction VSD. Rupture occurred a median of 16 hours after infarction, and the median time to the onset of shock was 7.3 hours. Twenty-four patients were managed medically; the remaining 31 patients comprised a high-risk surgical group. There were only 7 survivors, of whom 6 had surgery to repair the defect.

Despite the many advances in the non-operative treatment of congestive heart failure and cardiogenic shock, including the intra-aortic balloon pump and a multitude of new inotropic agents and vasodilators, these do not supplant the need for operative intervention in these critically ill patients[8]. The culprit artery in the setting of VSD is usually the LAD. Several reports have questioned the role of concomitant CABG along with VSD repair, there being no difference in early or late outcomes[5]. The tissue in the LAD territory becomes obliterated by the sutures that close the LV. Therefore, nothing is gained by grafting the LAD. Commonly, after the 2nd or 3rd day of post MI VSD repair, the patch blows up and the shunt re-opens, as happened in our case too, in spite of using an exclusion technique. The reason is that although the surgeon stitches onto tissue that appears solid enough to hold the sutures at the time of operation, surgery is performed in the setting of an acute infarct. Over the next few days additional myocardium may become necrotic and friable as a result of developing inflammation at the infarct margin. This results in stitches pulling out of the septal tissue, allowing the shunt to re-open. The technical problems are largely different when the necrotic muscle is replaced by fibrous tissue[9]. Risk factors for mortality include preoperative and postoperative evolution of clinical status and right ventricular functions. Two patches repair have a better prognosis than 1 patch repair[10]. A shorter interval between perforation of the septum and surgical repair has been found to be an increased risk factor for mortality; however, it is ethically unacceptable to delay surgery in these brittle patients[10].

In spite of, the high preponderance of septal involvement in myocardial infarction, septal perforation occurs in only 1-5.5% of all infarcts. Unlike most congenital septal defects, which are located within the membranous portion of the septum, the acquired ones are in the muscular part. Congenital septal defects tend to have sharply circumscribed circular or ovoid margins, while the post-infarctional type usually tends to be smaller and has ragged borders and a fissure-like Acquired ventricular septal defect is an unusual but catastrophic event that generally occurs within the first two weeks after infarction and accounts for 1-2% of the mortality following acute myocardial infarction. The diagnosis should be suspected when a loud, harsh pansystolic murmur accompanied by a thrill appears in the fourth left intercostal space between the sternum and the apex. The presence of the thrill is most helpful in the diagnosis because it is present in 40-65% of patients with acquired ventricular septal defects and is infrequently found in patients with acute mitral insufficiency. Although the murmur of an acquired ventricular septal defect is often characteristic, the differentiation from acute mitral insufficiency complicating myocardial infarction may present a quandary. The broad radiation of the murmur in the case presented and its high-pitched quality and lack of thrill obscured the bedside diagnosis. Echocardiography has proved to be a valuable noninvasive tool in the diagnosis of mitral valvular

prolapse. There was no evidence of prolapse or flail mitral leaflets in our patient. A recent report pointed out some indirect echocardiographic signs as clues to the diagnosis of acquired ventricular septal defect; these included dilation of the right ventricle and complete diastolic closure of the mitral valve after its initial opening, followed by almost complete reopening. In the present case the right ventricular dimension was normal, and complete early closure of the mitral valve was not present. Our patient had a low stroke index and severe left ventricular failure, perhaps explaining the absence of this pattern of mitral valvular movement. In our case, analysis of the septal echoes demonstrated the presence of the ventricular septal defect. Normally, by tilting the transducer from the aorta toward the mitral valve and left ventricle, the continuity between the anterior aortic root and septum is seen. Below the atrio-ventricular junction, we found a septal discontinuity, indicating the site of the ventricular septal defect, as later corroborated by angiographic studies and surgery. The relatively large size and unusual location of the ventricular septal defect made possible its detection. This septal dropout was reproduced at will. The left ventricular "cone-down" effect was never appreciated. This finding was probably due to dilation of the left ventricle and the flaccid aneurysmal septum, which simply did not approach the posterior wall in the parasternal area that we were able to reach in our scan. It should be noted that M-mode.

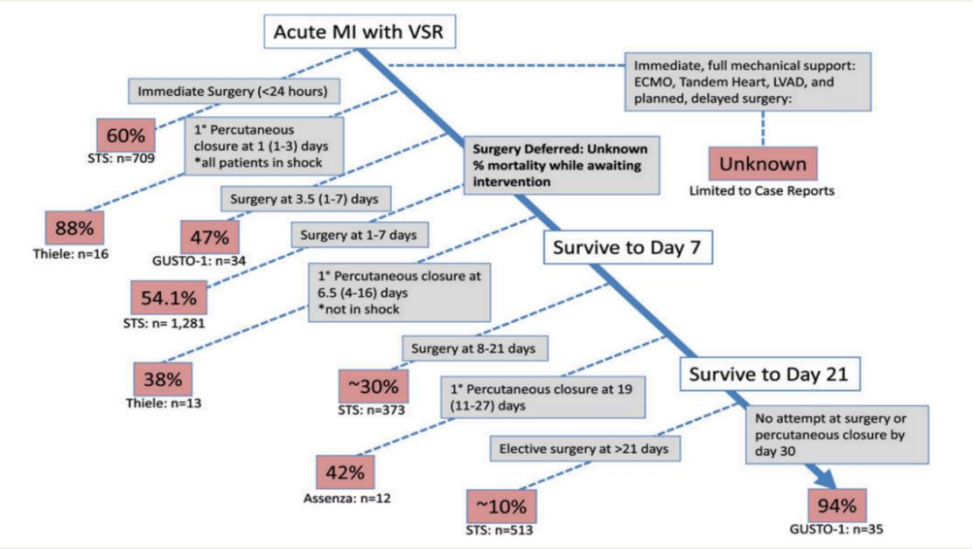

Figure 4[11]: Reported 30-day mortality of VSR with immediate surgical management or at various time points of delayed intervention.

echocardiographic scanning using a strip-chart recording facilitated the discovery of the localized absence of ventricular septal echoes. The rest of the septum showed a marked decrease in motion. After the resection of the septal aneurysm and the repair of the ventricular septal defect, the echocardiographic recordings showed an intact ventricular septum, which displayed paradoxical motion. It is obvious from the findings presented here that this noninvasive technique can enable one to identify an acquired ventricular septal defect and to distinguish it from acute mitral regurgitation.

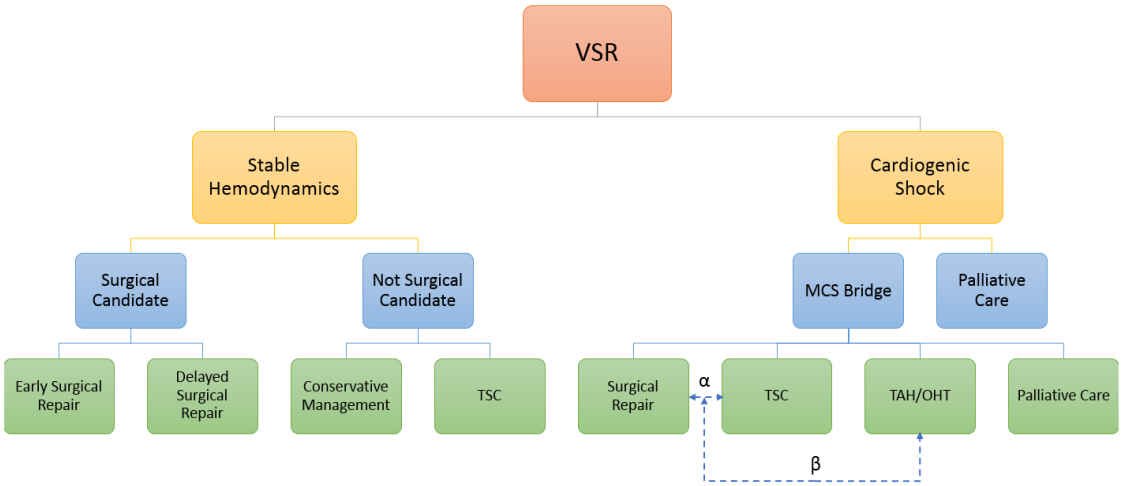

Figure 5[11]: VSR management algorithm (α: if deemed suitable for percutaneous repair, β: candidacy for total artificial heart and/or cardiac transplantation, TSC: trans-catheter septal closure, MCS: mechanical circulatory support, OHT: orthotopic heart transplant, TAH: total artificial heart).

Conclusion:

In summary, the treatment strategy should be individualized according to the patient's clinical status and haemodynamics. We inferred that the successful surgical outcome depended on the prompt recognition and simultaneous repair of the lesion.

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