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Interventricular Septal Rupture: An Uncommon Complication of an Acute Myocardial Infarction

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Abstract

Ventricular septal rupture is an uncommon but serious complication of acute myocardial infarction that requires prompt and appropriate management. In this case, we present a typical example of a patient diagnosed with a ventricular septal rupture.

Keywords

Myocardial Infarction, Ventricular Septal Rupture, Echocardiography, Case Report

Case Presentation

This case underscores the significance of close monitoring and early diagnosis and management of ventricular septal rupture. The patient, a 57-year-old female smoker with a history of type 2 diabetes, heart failure, and recent anteroseptal ST-elevation myocardial infarction (STEMI) treated with a stent, was referred to our institution due to oppressive bilateral shoulder pain, productive cough, dyspnea of exertion, and an electrocardiogram (EKG) with ST-segment elevation in V2-V4. The physical examination, laboratory evaluation, and elevated NT-proBNP (4751) levels, as well as the results of the Transthoracic echocardiography (TTE) and magnetic resonance imaging (MRI), confirmed the diagnosis of an acute anteroseptal STEMI with a post-infarction ventricular septal rupture (**Fig-1** and **Fig-2**).

The patient was successfully treated with surgical placement of a bovine patch to seal the rupture and stenting of the anterior descending artery in the cath lab. She showed steady recovery in the intensive care unit and was discharged three weeks later with a follow-up plan through an outpatient clinic and routine

EKG and TTE. This case highlights the importance of prompt and appropriate management of ventricular septal rupture to improve outcomes and prevent serious complications.

Ventricular Septal Rupture

According to the Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries (GUSTO-I) trial, ventricular septal rupture (VSR) is a rare but fatal complication of acute myocardial infarction (MI). It affects only 0.2% of patients with MI who received thrombolytic therapy and 3% of those who did not receive any treatment [1]. VSR typically develops within the first 2 weeks after a myocardial infarction, and it has a bimodal distribution, with events tending to occur in the first 24 hours or 3-5 days [2]. According to the trial, VSR is more likely to occur in patients who receive fibrinolytic therapy compared to those who undergo percutaneous coronary intervention.

Being the most common mechanical complication of MI [3], VSR is also one of the most lethal if left untreated, with a 50% survival rate after one week, 20% within one month and 10% within one year [1,3]. VSR is a consequence of loss of septal blood supply by branches derived from the left anterior descending artery from the left coronary and posterior interventricular branch from the right coronary (albeit in right dominant patients). Most commonly, an extensive transmural infarction induces a dry denaturation of proteins due to diminished oxygen supply. This event triggers septal thinning 5 days after the primary MI or within only 24 hours if there is a dissection of an intramural hematoma or a hemorrhage in the ischemic area [4].

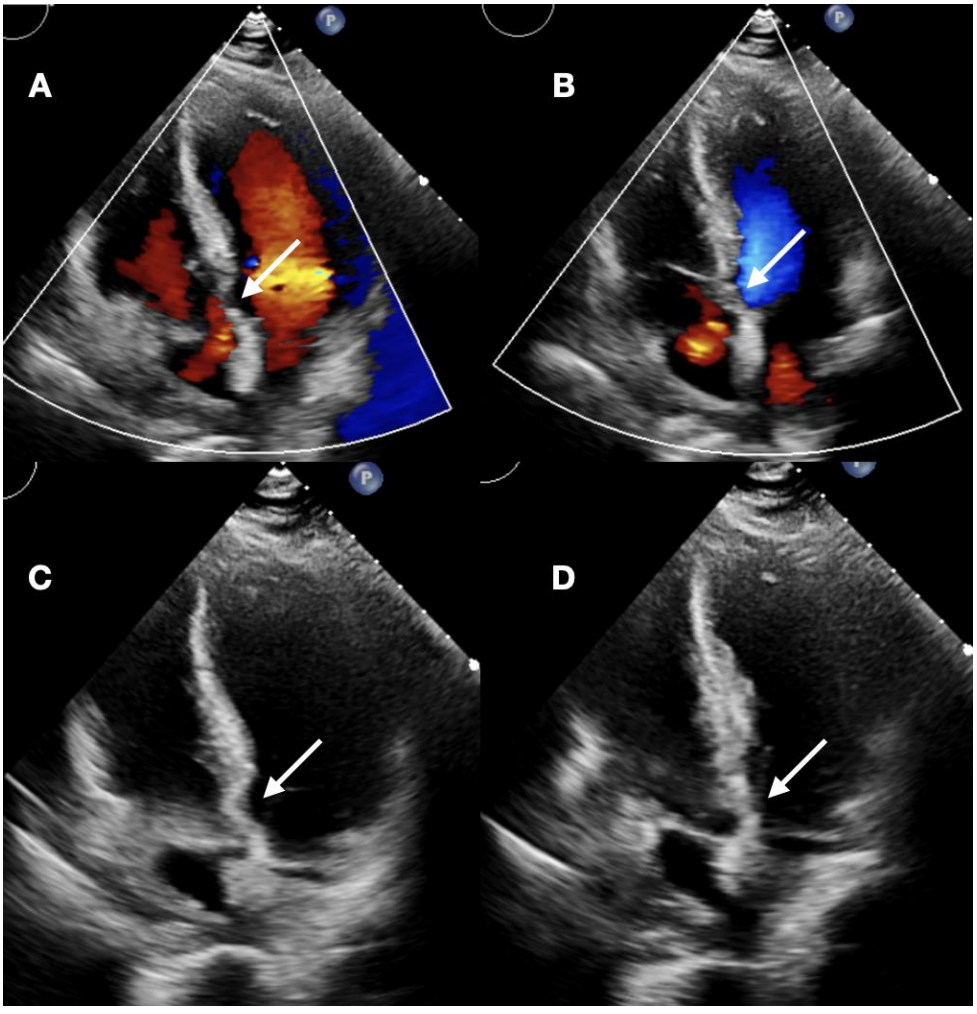

Fig-1: TTE showing a ventricular defect from (apical 4-chamber view)

A and B: *Doppler color showing slight flow abnormalities because of the defect.* ***C and D:*** *Traditional 2D TTE showing the defect in the basal portion of the interventricular septum.*

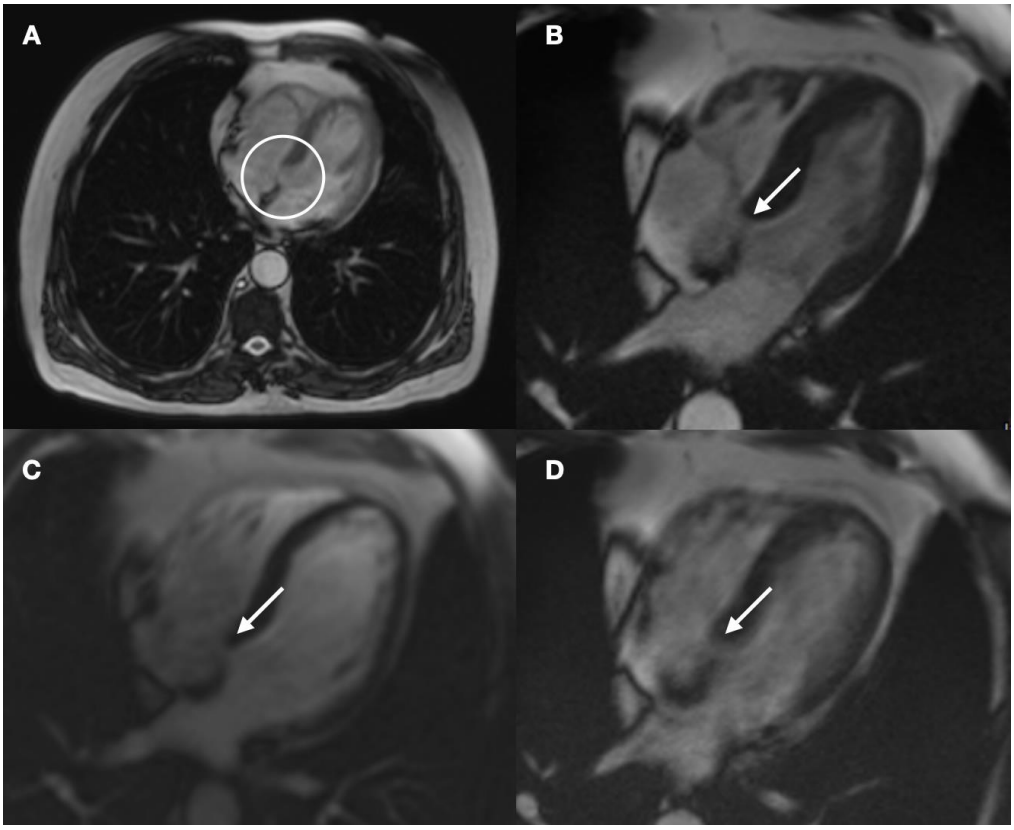

Fig-2: MRI showing the ventricular defect in the basal portion of the interventricular septum

Becker and Mantgem created a classification for ventricular free wall rupture, which is similar in pathophysiology to VSR. Because of the overlap between these two conditions, this classification system is useful for VSR as well.

1) Abrupt, slit like tear, <24 hr
2) Erosion in the infarcted zone, subacute presentation
3) Due to a concomitant aneurysm which leads to a rupture, older infarcts

VSR may also be classified as simple or complex. Simple ruptures are direct connections between the right and left ventricles, while complex ruptures have a fluctuating course and are caused by several shears in the necrotic septum [5].

Constrictive Pericarditis

Constrictive pericarditis (CP) is the name given to severe diastolic dysfunction of the heart due to an inelastic pericardium, usually as a consequence of an uncontrolled pericardial effusion. Normally, the pericardium is a fibroelastic sac filled with 30-50 mL of pericardial fluid that protects the heart as a mechanical barrier while allowing it to contract and relax freely. However, overfilling of the pericardial sac by other fluids can lead to an inelastic pericardium, which will ultimately lead to a rise in filling pressures and a decrease in stroke volume [6]. Both of these events will force an increase in heart rate to maintain cardiac output.

11% of hemorrhagic pericardial effusions are complications of MI, and in 1-2% it occurs as a complication of the surgery itself. However, because guidelines of the American Heart Association suggest urgent surgical intervention in VSR, presentation of CP should be warned [7,8]. CP manifests as cardiac tamponade, followed by cardiogenic shock and pulseless electrical activity if left untreated. Therefore, if CP is presented, emergency pericardiocentesis is the procedure of choice; however, if patient status does not improve and/or bleeding cannot be stopped; emergent cardiac surgery should be performed [7].

Clinical Presentation

As a mechanical cause of acute heart failure, clinical manifestations associated with hemodynamic

compromise are characteristic of VSR. The most common is the appearance of flash pulmonary edema, angina and hypotension, and, in severe cases, cardiogenic shock [2,5,9].

Post-MI patients with a VSR present with a characteristic strong and rough holosystolic murmur, heard loudly in the lower left sternal border. In 50% of cases, this murmur is accompanied by a thrill [2,5]. In some cases, due to the increased heart flow, a loud pulmonic component of S2, a tricuspid regurgitation murmur or a third heart sound may be heard. In patients with large VSR and severe heart failure or cardiogenic shock, the murmur may be low in intensity or inaudible [9].

Diagnosis

All patients with an acute MI should be evaluated for possible mechanical complications before primary PCI is performed. Findings that raise suspicion for VSR (such as the systolic murmur described earlier) should elicit the need for a left ventriculogram, which may show a contrast shunt that goes from left to right [10,11].

Transthoracic echocardiography (TTE) is the test of choice to identify the presence, region, and size of the VSR. TTE is also useful for establishing whether a shunt exists and the severity of its flow, evidence of chamber enlargement and dysfunction, and pulmonary hypertension. Another alternative may be transesophageal echocardiography (TEE), which should be considered as an additional imaging technique with the coexistence of lateral or inferior MIs that may have been missed on routine transthoracic imaging [4].

Management

Surgical management of VSR can approach 100% mortality for some patients, while 90% of patients die within 2 months [7,11,12]. However, this method is the only therapeutic path with a good chance of survival. Some patients with a relatively well-preserved left ventricular function may have a lower surgical risk [7]. Recommendation for closure depends on the patient's hemodynamic status to avoid further morbidity. Even though the current literature states that surgery must be performed immediately, some surgeons suggest a 3 to 4-week delay in order to allow scarring of the surrounding tissue to occur, that allows a firmer attachment of suture and patch material [11,12].

Closure is mainly achieved using a heterologous pericardial patch, which assures the sealing of the defect. A first line of interrupted sutures and a second line of continuous sutures are applied. The first line of sutures serves to fix the patch in place, while the second line is used to position the patch to cover the defect. Afterwards, 2 lines of continuous polypropylene sutures and 4 polytetrafluoroethylene felts are applied in order to close the left ventriculotomy [13].

According to The Society of Thoracic Surgeons National Database, survivors to VSR and its surgical repair showed to be mostly 66-year-old caucasian males. It was also demonstrated that mortality rates were dramatically reduced if more than 7 days had passed after the MI [14].

Up to 20% of procedures presented with postoperative residual shunts and high mortality, which is why a percutaneous VSR closure double-umbrella device that closes the VSR bilaterally was developed. The use of percutaneous techniques is increasing in use and effectiveness, with better general outcomes for patients with VSR [12].

Invasive monitoring is imperative in the management of post-VSR patients in the intensive care unit (ICU). Measuring both ventricular filling pressures is mandatory to guide fluid administration and diuretic use. Measuring cardiac output and mean arterial pressure help to estimate systemic vascular resistance to direct vasodilator therapy. Inotropes may also be useful, as well as an intra-aortic balloon counter pulsation (IABP) if pharmacologic therapy is not fruitful [2]. IABPs have proven useful for measuring 30-day survival in patients with post-MI VSR and signs of cardiogenic shock. However, in patients with absence of cardiogenic shock, clear evidence in its favor has not been discussed [11].

Other Complications of Acute MI

Ventricular Free Wall Rupture:

Ventricular free wall rupture is a rare but often lethal mechanical complication of acute myocardial infarction (AMI). This complication is more likely to occur in the first 24 hours, and incidence decreases progressively until it becomes infrequent after the fifth day. Cardiac tamponade is the main clinical presentation, however, this condition may vary from acute to subacute, [15,16] leading to hemodynamic instability and sudden death. TTE is essential for diagnosis, being the massive pericardial effusion the most frequent finding. For treatment, emergency surgery is indicated and often, the rupture site is easily identified [17].

Papillary Muscle Rupture:

Papillary muscle rupture is rare, it occurs in 1 to 5% of the patients with an IAM due to improvements in early identification and treatment of IAM. However, when rupture does occur, it has a high mortality (50% within 24 hours) without surgical intervention. Papillary muscle dysfunction leads to the regurgitation of blood through the valves causing the backward flow of blood, which could develop heart failure. This most commonly occurs 2 to 7 days post-MI, and happens less frequently in non ST segment elevation infarctions [18].

The posterolateral papillary muscle is supplied only by the dominant right coronary artery or left circumflex artery, therefore, it is more often involved. TTE may show a leaflet of the mitral valve during systole into the atrium or the ruptured papillary muscle head with inadequate movements in the ventricle or a mobile mass attached to the chordae tendineae [19,20].

Conflict of Interest

The authors have read and approved the final version of the manuscript. The authors have no conflicts of interest to declare.

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