

# Ventricular Septal Rupture – A Critical Condition as a Complication of Acute Myocardial Infarction

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## ABSTRACT

Ventricular septal rupture is a potentially fatal complication of acute myocardial infarction. The key to management of this critical condition is an aggressive approach to haemodynamic stabilization and surgical closure of the rupture. Where there is a small rupture and the patient is in a haemodynamically stable condition, surgery can be delayed with the prospect of achieving better perioperative results. However, in unstable critically ill patients either immediate surgery or extracorporeal membranous oxygenation support and delayed surgery is indicated. In some patients, trans-catheter closure may be considered as an alternative to surgery.

**Keywords:** ventricular septal rupture, acute myocardial infarction, mechanical complications, timing of surgery, extracorporeal membraneous oxygenation, transcatheter closure

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Myocardial rupture is a critical condition responsible for 10-15% of all deaths in patients with acute myocardial infarction. The most common type of myocardial rupture is that of the ventricular septum (85% of cases) while less frequent types include the rupture of the free left ventricular wall with the development of cardiac tamponade (10%), or papillary muscle rupture with the development of acute mitral regurgitation (5%) [1]. Due to the progressive development and availability of reperfusion therapy, the incidence of these complications in the last decades has steadily declined [2]. However, the mortality in these critically ill patients remains high, despite a downward trend in incidence.

The incidence of post-infarction ventricular septal rupture (VSR) associated with acute myocardial infarction (AMI) has also significantly decreased with the introduction of reperfusion therapy. In the GUSTO-I trial, the incidence of post-infarction VSR was 0.2% in patients treated with thrombolysis [3]. A similar incidence was reported in patients treated by primary percutaneous coronary intervention [4]. In the SHOCK

Trial Registry, post-infarction VSR was present in 3.9% of patients, with cardiogenic shock complicating the AMI [5].

The time interval from the onset of an AMI to the manifestation of VSR has a bimodal distribution, with a higher incidence in the first twenty four hours after the onset of infarction and later between the third and fifth day. It develops only rarely at more than two weeks after an AMI [6]. In the early stages of rupture formation, the characteristic clinical manifestation of VSR is the sudden development of cardiogenic shock. In the SHOCK Trial Registry, the median time interval from AMI to the manifestation of VSR was sixteen hours [5].

Risk factors for VSR include age, female gender, hypertension, first myocardial infarction and absence of collaterals in the coronary circulation. In most of the patients, VSR is associated with a lesion in a single coronary artery and often, with its complete occlusion [3,4]. A minority of patients presents with multiple coronary artery stenosis.

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In 60% of patients, septal rupture complicates an AMI of the anterior left ventricular wall. Ruptures are typically localized in the apical portion of the septum and are simple.

In 40% of patients, septal rupture complicates an AMI of the inferior wall of the left ventricle. Localisation of these ruptures is typically in the basal septum and commonly these are complex with extensive intramural rupture propagation in different directions, often with significant damage of the right ventricle and mitral regurgitation due to dysfunction of papillary muscle. These ruptures have a worse prognosis [7].

The first three to five days after the occurrence of a VSR are characterized by development of coagulation necrosis of the myocardium with neutrophilic infiltration. VSR occurring in the first twenty four hours after an AMI onset is most likely caused by the dissection of an intramural haematoma or haemorrhage into ischemic myocardial tissue. This occurs due to physical shear stress at the border of an infarcted zone, combined with a hyper-contractile surrounding myocardium.

Subsequent retraction of surrounding tissue may lead to enlargement of the defect, followed by a progressive fibrosis and mechanical reinforcement of tissue in the following weeks.

From a haemodynamic point of view, the left-to-right shunt in a VSR leads to pressure and volume overload of the right heart chambers and volume overload of the left ventricle. The size of the shunt is determined by the actual size of the VSR and the ratio of systemic and pulmonary vascular resistances. The decrease in cardiac output leads to a compensatory increase in systemic vascular resistance and further progression of the left-to-right shunt.

Clinically, the rupture is manifest by the sudden development of dyspnoea, hypotension, and bilateral cardiac insufficiency, predominantly on the right side. A newly formed holosystolic rough murmur, at maximum at the lower sternum, can also be detected. Half of the patients have also a palpable thrill, though both murmurs and thrills may be difficult to detect in patients with a low cardiac output state. Other physical examination findings result from augmented right-sided flow, including a loud pulmonic component of the second heart sound, S3 gallop or a murmur from tricuspid regurgitation. In one third of the patients a complete atrioventricular block may be present. Unless the defect is closed, most of the patients with increasing

left-to-right shunt develop progressive shock with multiple organ failure, leading to death. The prognosis of the conservatively treated patients is very poor and the mortality rate among patients who are treated conservatively without mechanical closure is approximately 24% in the first 24 hours, 46% at one week, and 67-82% at two months [6].

Echocardiography is essential in order to confirm a diagnosis. The left-to-right shunt is usually evaluated by colour Doppler (Figure 1), which is able to estimate the size of the defect, the function of both ventricles, the dilatation of the right ventricle and the pressure in the right ventricle. When the patient has a poor acoustic window, due to mechanical ventilation or body habitus, transoesophageal examination should be considered.

The left-to-right shunt can also be confirmed by right heart catheterization with oximetry, a technique that identifies a step-up in blood oxygenation level in the right ventricle and quantifies the ratio between pulmonary and systemic blood flow ( $Q_p/Q_s$ ).

All patients with STEMI should undergo urgent coronary angiography. Left ventriculography confirms a leakage of the contrast agent from the left ventricle into the right ventricle and pulmonary artery (Figure 2). When a diagnosis of VSR is established prior to primary PCI, the prompt restoration of coronary flow must be a primary consideration, taking into account the competing needs of possible emergent surgery and the associated risks of bleeding after dual-antiplatelet therapy. Limitation of the ischaemic burden in the infarct-related artery remains crucial, especially if there is right-ventricular involvement, therefore immediate collaboration between the interventional cardiologist and cardiac surgeon is critical in developing a case-

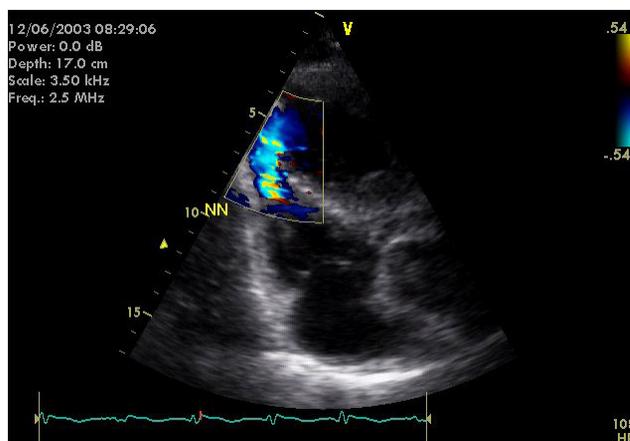
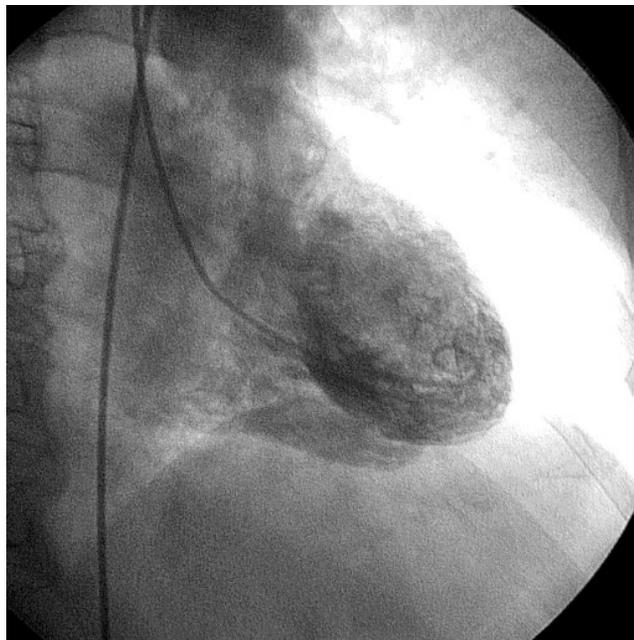


Fig. 1. Left-to-right shunt in colour Doppler imaging



**Fig. 2. Left ventriculography, leakage of contrast agent to the right ventricle**

specific approach. Flow may be restored in the infarct-related artery with aspiration thrombectomy and/or balloon angioplasty, and preparations made for immediate VSR repair with coronary artery bypass grafting [8].

Management of patients with VSR includes an aggressive approach to haemodynamic stabilization and closure of the rupture. Insertion of an intra-aortic balloon pump is recommended in all the patients to reduce the afterload of the left ventricle, and thus to reduce the significance of a left-to-right shunt [9]. Extracorporeal membrane oxygenation (ECMO) is indicated for patients in a critical condition [10].

The timing of closure of the defect, either interventional or by surgery, still remains controversial. Achieving patient stabilisation reduces the risk associated with any subsequent procedure. However a prolonged effort to stabilize patients delays the closure of the defect with potentially dangerous consequences. Shearing forces applied to the myocardium can lead to continued septal rupture and sudden circulatory collapse. Acute worsening of the patient's condition before the surgery, is then a predictor of high perioperative mortality, and a high number of patients die before surgery.

Many studies agree that early surgery, compared to delayed surgery, is associated with higher perioperative mortality [11]. However, non-surgical mortality is certainly higher. Thus, the clinician must weigh the

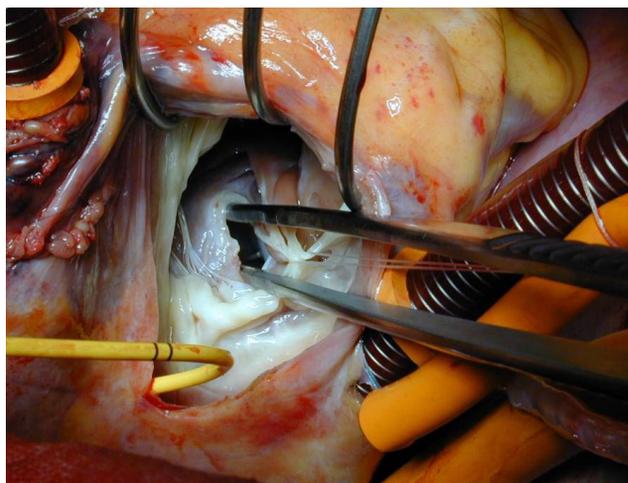
known risk of expedient surgery against the unknown risk of advanced clinical deterioration associated with the postponement of surgery [8].

The American Heart Association recommends an urgent closure of the rupture in all patients [12]. In contrast, the European Society of Cardiology guidelines point to the lack of expert consensus on the optimal timing of the closure [13].

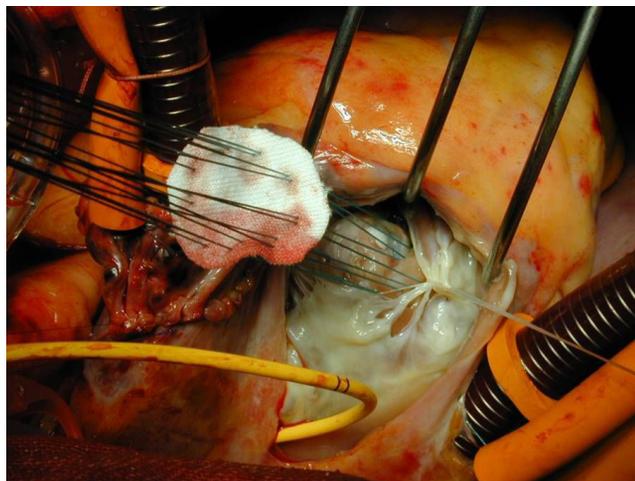
The preferred approach in critically ill patients in an unstable condition may be the use of ECMO and subsequently delayed closure of the rupture [10].

The surgical closure of a VSR in the acute phase of myocardial infarction is technically extremely difficult, due to the necrotic tissue surrounding the rupture. It consists of gaining access through the infarcted zone of the free wall of the left ventricle in order to minimize the damage of functional myocardium, resection of necrotic tissue around the VSR and suturing of the defect using a patch from the pericardium or Dacron®. A sandwich technique using two Dacron® patches from both sides of the VSR, using a tissue glue, is the preferred technique [14,15]. For ruptures located in the upper part of the ventricular septum, the least invasive approach is through the right atrium and tricuspid valve (Figures 3,4). Revascularization of the myocardium, with coronary artery bypass grafting, is performed at the same time. In cases with a left ventricular aneurysm, part of the procedure is to undertake aneurysmectomy, while in case of significant mitral regurgitation, the mitral valve is replaced with a prosthesis or by mitral annuloplasty.

In 10-40% of patients, the stitches may cut through during the post-operative period, especially if the VSR



**Fig. 3. Surgical approach to VSR through tricuspid valve**



**Fig. 4. Closure of VSR, double patch technique**

is positioned posteriorly. This leads to the recurrence of a left-to-right shunt [16]. Minor defects, in clinically stable patients, are managed conservatively [17].

The rate of mortality of operated patients varies between 20-60% and is mainly dependent on the haemodynamic status of the patient prior the surgery [11]. However, a significant amount of patients die not having undergone surgery and they are not included in published data. The main factors worsening the prognosis of patients undergoing surgery include complex forms of VSR, right ventricular dysfunction, the presence of cardiogenic shock, multiple organ failure and deterioration of the patient's condition prior to the surgery. Revascularisation of the myocardium, when possible, improves the prognosis of patients [18]. The long-term prognosis of successfully operated patients is good, the 5-year survival rate is between 40-75%, with most patients being in NYHA functional class II [19,20].

Transcatheter closure, using an Amplatzer® septal occluder, is an established method of treatment of congenital defects. In post-infarction VSR, this may be considered as a possible alternative to surgical closure in selected patients. It consists of the retrograde introduction of a wire from the femoral artery into the left ventricle and then through the VSR, into the right ventricle and then to the pulmonary artery or superior vena cava, where it is caught and pulled out with a snare wire introduced through a central venous access. This produces the formation of an arterio-venous loop. Following this, the delivery system is introduced along the wire from the venous side through the VSR and the Amplatzer® septal occluder is implanted. The whole procedure is performed under transoesophageal or intracardiac echocardiographic control [8].

A potential obstacle, preventing the transcatheter closure of VSR, is the presence of a small tissue rim of the ventricular septum which is insufficient to allow fixation of the occluder. Potential complications include the risk of the occluder catching onto the mitral or tricuspid valve subvalvular structures, the development of regurgitation, progression in the VSR size due to manipulation with the occlude, and complete atrioventricular block. Another possible major complication is associated with the occluder becoming disengaged and subsequent risk of embolization.

Closure of a post-infarction VSR with an Amplatzer® septal occluder is frequently associated with a residual shunt, though this is usually insignificant and without need of further intervention. Any significant residual or recurrent shunt can be solved with another trans-catheter closure or with delayed cardiac surgery with already low risk in a stabilised patient. Similarly, residual or recurrent significant shunt after surgical closure of VSR can be managed with Amplatzer® septal occluder.

Clinical experience with trans-catheter closure of post-infarction VSR is limited compared to surgical experience. Several case series have been published documenting single experiences with both primary percutaneous VSR closure as well as closure of residual defects after surgery. In agreement with the surgical literature, outcomes improve as patients progress from the acute to chronic phase [8,21-24]. Thiele [25] described a set of twenty nine consecutive patients when closure with an Amplatzer® occluder was performed very early, the median time from manifestation of VSR to closure being one day. Cardiogenic shock developed in 55% of patients. The closure was performed successfully in 86% of patients and led to a reduction in Qp/Qs ratio from 3.3 to 1.4. Serious complications such as significant residual shunt, dislocation of the occluder or left ventricle rupture, occurred in 41% of patients. The overall thirty-day mortality rate was 65% compared to 88% in patients with cardiogenic shock and 39% in all other patients [25]. In a follow-up of surviving patients, median 730 days, one patient died of an infectious endocarditis on the side of the occluder. All the other patients were reported to be in a good condition [25].

## ■ CONCLUSION

Rupture of the ventricular septum is a serious complication of an acute myocardial infarction. Without clo-

sure of the rupture, most patients die. Timing of surgery or transcatheter closure is still controversial. An appropriate strategy, in unstable patients, may be the use of extracorporeal membranous oxygenation and subsequently delayed closure of the rupture. Patients who survive this complication have good long-term prognosis.

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