

## Ventricular Septal Rupture After Acute Myocardial Infarction

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### Abstract

Ventricular septal rupture is still a rare but often fatal complication of acute myocardial infarction. Emergent surgical closure of postinfarction ventricular septal rupture irrespective of the clinical status has been the standard treatment so far. A percutaneous approach using an occluder device is a less invasive option and allows immediate complete closure after initial hemodynamic stabilization. Furthermore immediate reduction of the left-to-right shunt, even if the ventricular septal rupture is not completely closed, may stabilize the patient enough to function as a bridge to surgery. We present two similar cases of patients which highlight the multiple features of acute myocardial infarction-related ventricular septal rupture treated with two alternative techniques.

### Introduction

Despite significant improvements in overall mortality for patients with acute myocardial infarction (AMI) the outcome of patients who develop ventricular septal rupture (VSR) remains poor [1-3]. Cardiogenic shock and severe left ventricular failure are the most important factors determining the outcome of individuals with VSR [4]. Until recently, traditional surgical repair was the only definite and highly recommended therapeutic strategy for postinfarction VSR, unfortunately associated with fairly high postoperative morbidity and mortality. Considering very high mortality rate when untreated as well as high risk of surgical procedures, attention has been drawn to other alternative therapeutic options. In this manner, percutaneous closure of VSR with occluder devices gradually appears to be mainstay therapy in the acute stage of postinfarction VSR. This regards both definite treatment and a bridge to surgical repair after patient's hemodynamic stabilization. Therefore we present two similar cases of our patients who experienced postinfarction VSR but underwent different treatment strategy.

### Case Reports

#### Patient 1

72-year-old female without a previous history of coronary artery disease (CAD), smoker, hypertensive with a body mass index (BMI) of 20.3 kg/m<sup>2</sup>. She was admitted to our intensive care unit with a ten-hour history of chest pain. The ECG revealed ST segment elevation in anterior leads. The patient underwent emergency coronary angiography, which showed complete occlusion of the left anterior descending coronary artery (LAD). Subsequently, primary percutaneous coronary intervention (pPCI) with simultaneous drug eluting stent implantation was performed. Twenty four hours after AMI onset, the patient developed respiratory distress with a concomitant cardiogenic shock. An echocardiogram was performed and revealed mild left ventricle (LV) wall hypertrophy, a moderately

decreased LV ejection fraction (EF=47%), mild mitral regurgitation and a left to right shunt through an 8 mm defect located in the mid-anterior segment of the interventricular septum. Due to the patient's haemodynamic instability, intraortic balloon counterpulsation (IABP) and inotropic agents infusion were commenced.

After 48 hours stabilization she underwent percutaneous closure of the VSR with an Amplatzer occluder device. Post-procedural transthoracic echocardiogram (TTE) showed proper positioning of device with only minimal shunting through the device. Patient remained hemodynamically stable directly after the procedure.

Unfortunately, despite this management, she died three weeks after the procedure due to multiple organ failure.

#### Patient 2

70-year-old male without a previous history of CAD, smoker, non-treated hypertensive, with BMI of 28 kg/m<sup>2</sup>. He was admitted to our intensive care unit on second day after AMI of LV anterior wall treated with pPCI of isolated complete occlusion of LAD with simultaneous drug eluting stent implantation.

Upon admission patient was found in clinical evidence of cardiogenic shock, with developed respiratory distress. An echocardiogram was performed and revealed akinetic segments of LV anterior wall with slightly decreased LV ejection fraction (EF=45%) and a left to right shunt through an 9-10 mm defect located in anterior-middle segment of the interventricular septum with coexistent enlargement and overload of right ventricle. An IABP and inotropic agents infusion were commenced. After initial stabilization he was transferred immediately to cardiosurgical ward. The VSR was resected so that the mitral subvalvular apparatus was not affected. A Dacron patch sized 2 × 3 cm was attached to left ventricular aspect of the remaining non-infarcted septum using interrupted pledgeted horizontal mattress sutures. After operation the patient remained in stable hemodynamic condition till discharge to outside hospital.

## Discussion

Post- infarction VSR is an extremely serious complication of AMI and mostly occurs within the first week after the acute event [2]. In our patients' cases VSR was detected 24 and 48 hours respectively after AMI onset which indicates the trend towards an earlier occurrence of the VSR [1]. Since the introduction of myocardial reperfusion therapies the incidence of AMI- related VSR has been reduced to 0,25-0,7% [3].

The coexistence of risk factors plays the important role in the appearance of this complication and the higher incidence of its fatal outcome. The first presented patient was an elderly female with a low BMI, all of which are important independent predictors of AMI-related VSR [4,5].

In addition, in keeping with other studies, our patients had also other risk factors of VSR including arterial hypertension, lack of previous history of CAD, anterior localization of AMI, single-vessel CAD and delayed treatment after the onset of AMI symptoms [2-6]. This suggests, that pathophysiology of AMI- related VSR involves sudden, severe ischemia, which in turn leads to extensive myocardial necrosis. Moreover, patients with single vessel CAD and short history of ischemia rarely develop small natural vessel bypass and collateral circulation. Therefore, AMI in these patients leads to larger heart injury, promoting post- infarction wall weakness with no collateral flow protection.

As reported in our observation, patients with AMI- related VSR present rapid clinical state deterioration and the development of cardiogenic shock. The principal treatment of VSR consists of emergency rapid stabilization with inotropic agents and, if necessary, the use of intra- aortic balloon counterpulsation. Early surgery is considered the gold standard for postinfarction VSR, with the high surgical risk being acceptable in the face of the even higher risk of death without surgery [5-7]. More recently an additional option of percutaneous closure of the rupture has become available. Percutaneous closure of the VSR was initially reported in patients at too high risk for surgical repair due to their recent postinfarction status, advanced age, severe coronary artery disease, haemodynamic instability, and added comorbidity (such as renal failure and diabetes mellitus). Therefore in selected patients, percutaneous closure of VSR can be considered either as an alternative or a bridge to surgical repair [8,9].

In case of our first patient's, her condition was firstly stabilized with inotropic support and intra- aortic balloon counterpulsation, for 48-hours. After restoration of patient's hemodynamic stability, the percutaneous closure of VSR with Amplatzer occluder device was performed. Unfortunately she failed to improve her general clinical state, consequently developing multi- organ failure leading to death.

Although it appears to be the attractive option, percutaneous closure with occluder device carries some limitations possibly influencing the unfortunate outcome of our first patient. Firstly, interventional reports are mainly restricted to VSR closure in the chronic and subacute setting, or for residual shunts after initial surgical closure [8,9]. It has not been sufficient data regarding performance of percutaneous closure almost straight after the myocardial infarction acute phase. Therefore more clinical experience needs to be accumulated from different centers before a consensus can be reached on this issue. Additionally, in most cases the devices used for postinfarction VSR closure are dedicated originally for congenital defects and therefore have some technologic limitations to effectively

occlude the anatomically complex postinfarction VSRs [9]. Not only are the postinfarction VSRs larger in size and more complex in their geometry, the rims consist of unhealthy infarcted tissue that may cause some difficulties in positioning the occluder. To make matters even worse, these devices are prone to degenerate over time leading to further increase in VSR size. It may provoke occluding device displacements and consequent secondary trans septal shunts [8,9].

Emergent or early surgical intervention remains main definitive treatment for AMI- related VSR. On the other hand as regards classic surgery of VSR, many surgeons recommend the procedure after a 3-4 week delay to allow scarring of the surrounding tissue, which allows firmer anchoring of suture and patch material [6-10]. In case of our second patient the decision of immediate surgical repair of VSR was strictly determined by patient severe state due to cardiogenic shock. Fortunately the periprocedural and early recovery outcome was fairly satisfactory. In the meta analysis by Arnaoutakis et al. [10] of patients surgically operated due to postinfarction VSR the overall operative mortality was 42.9% (n=1,235). Interestingly, when the procedure was performed within 7 days from AMI, operative mortality was 54.1%, whereas 18.4% when this period was exceeded to more than 7 days from AMI.

In accordance with above findings percutaneous closure as a bridge to surgery can help improve prognosis of unstable patients with AMI-related VSR by allowing hemodynamic stabilization and delaying surgical intervention. Therefore this hybrid approach should be considered in all individuals with marked hemodynamic instability.

## Conclusion

Despite significant improvements in diagnosis, therapy and development of new techniques and devices, VSR is still a rare but catastrophic mechanical complication after AMI and carries an extremely high mortality rate.

Mechanical closure should be a definite treatment strategy in each case. Irrespective of whether it is performed by means of classic open surgery or percutaneous closure, both options are connected with high risk as well as the patient's clinical status seems to be the most important determinant of final outcome. Percutaneous closure with occluder devices carries some advantages over surgical repair of being less invasive and leaving open option for eventual definite surgical management after patient's stabilization. Undoubtedly, timing and type of treatment for patients with AMI- related VSR still need to be evaluated.

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