

Ventricular Septal Rupture with Acute Myocardial Infarction: Primary Percutaneous Coronary Intervention and Delayed Transcatheter Closure of the Rupture

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Abstract

Postinfarction ventricular septum rupture is a lethal mechanical complication of acute myocardial infarction. Due to the fragility of necrotic septum, an early operation is associated with residual shunt and an extremely high mortality. We report an 80-year-old woman with ST-segment elevation myocardial infarction developed ventricular septum rupture occurring in the early postinfarction. In order to save the dying myocardium, she underwent primary percutaneous coronary intervention at first, but sudden hemodynamic deterioration occurred in the fourth day after the infarction. An intra-aortic balloon pump was inserted to reduce systemic vascular resistance and decrease the magnitude of left-to-right shunt. The patient's condition was stabilized under the intra-aortic balloon pump support and a delayed transcatheter closure of the ventricular septum rupture was performed three weeks later. The patient was discharged with complete recovery.

Keywords: Acute myocardial infarction; Ventricular septum rupture; Primary percutaneous coronary intervention; Intra-aortic balloon pump

Introduction

Postinfarction ventricular septum rupture (VSR) is a rare but potentially fatal mechanical complication of acute myocardial infarction. Before the era of reperfusion therapy, the event occurs in approximately 1% to 3% of all infarcts [1]. With the use of aggressive antithrombotic medication and primary percutaneous coronary intervention (PCI), the incidence has been declining and is less than 1% of individuals with myocardial infarction [2]. In current practice, postinfarction VSR is recognized as a surgical emergency, and the presence of severely symptomatic is an indication for emergency surgical treatment. However, an aggressive approach may be associated with high perioperative mortality [3]. Recently, percutaneous closure of postinfarction VSR has become an accepted alternative to surgical repair, but an early closure is associated with residual shunt. Here, we report a case with VSR complicating acute myocardial infarction after primary PCI underwent a successful delayed transcatheter closure of the VSR under intra-aortic balloon pump (IABP) support.

Case Report

This is the case of an 80-year-old previously healthy woman who was admitted to the emergency department with chest pain ongoing for about 10 hours. On admission, her blood pressure was 102/76 mmHg with a heart rate of 104 beats/min. A new loud pansystolic murmur was detected in the left parasternal area. The initial electrocardiogram showed anterior ST-segment elevation, suggestive of acute anterior

wall myocardial infarction (Figure 1). Bedside transthoracic echocardiography revealed a ventricular septal defect of 7mm in diameter at the apical septum, an akinetic wall motion at the anterior wall, mild mitral valve regurgitation, and left ventricular ejection fraction of 42%. The patient was diagnosed as acute ST-elevation myocardial infarction complicated with ventricular septum rupture. Considering the calculated shunt by echocardiography was not large and there was ongoing ischemia, we decided to perform primary PCI at first. The patient was initially managed medically with oral 300mg aspirin, 180mg ticagrelor, and then emergent coronary angiography was performed which revealed normal the right coronary artery (RCA) and left circumflex coronary artery (LCX), total occlusion of the proximal left anterior descending (LAD) thrombus (Figure 2A). We performed PCI in the LAD and coronary blood flow was successfully restored with aspiration thrombectomy and balloon angioplasty followed by placement of a drug eluting stent (Figure 2B).

Four days after PCI, the patient suddenly experienced respiratory distress and tachycardia, which occurred after physical exertion. The size of the VSR was gradually increasing and approximately 10mm in diameter causing a significant left-to-right shunt. The patient subsequently developed hemodynamic instability due to low cardiac output and required placement of an IABP for hemodynamic support. Under IABP and medical supports, the patient's condition was relatively stabilized and did not show any serious symptoms during the following weeks. A delayed transcatheter closure approach was decided and cardiac catheterization was performed at three weeks after the infarction, which confirmed a 10mm muscular VSR (Figure 3A). Transcatheter closure of the VSR was performed and an amplatzer septal occluder was released at the apical septum. After the device deployment, the patient showed an increase in arterial blood pressure and left ventricular angiography revealed successful reduction of the left-to-right shunt (Figure 3B). Cardiac murmur in the left parasternal area disappeared and transthoracic echocardiography showed only a small residual shunt.

Due to the hemodynamic parameters improved significantly after closure of the VSR, IABP was weaned off. The patient survived to discharge from the hospital without other complications and with close follow-up.

Discussion

Postinfarction VSR is a well recognized mechanical complication of acute myocardial infarction, which can occur from few hours to several days postinfarction, but most frequently occurs 3 to 5 days after myocardial infarction. In the SHOCK trial registry and the GUSTO-1 (The Global Use of Strategies to Open Occluded Coronary Arteries) trial, VSR occurs most often within the first 24 hours in patients with ST-segment elevation myocardial infarction treated with thrombolytic therapy [3,4]. Patients with VSR are more often older, female, with less prior myocardial infarction and comorbidity compared with those without mechanical complications of myocardial infarction [3]. Early reperfusion therapy may lead to reopening of the occluded vessels, thereby preventing extensive myocardial necrosis thus reducing the incidence of VSR. However, PCI sometimes can cause reperfusion injury and induce more damage to the infarcted myocardium, especially in cases with complete coronary artery occlusion with little or no collateral flow [4,5].

Myocardial infarction complicated by VSR usually presents as rapid-onset clinical deterioration with acute heart failure or cardiogenic shock, depending on the size of the rupture and the degree of cardiac dysfunction. The goals of initial treatment are to reduce afterload

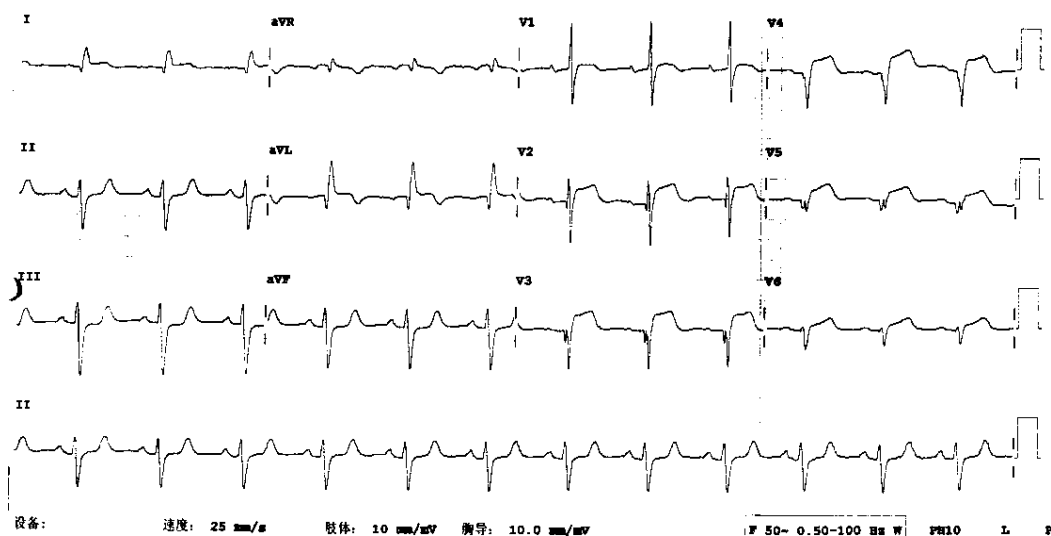


Figure 1: The 12-lead electrocardiogram results on admission which demonstrated sinus rhythm with ST segment elevation in anterior wall leads, suggestive of acute anterior wall myocardial infarction.

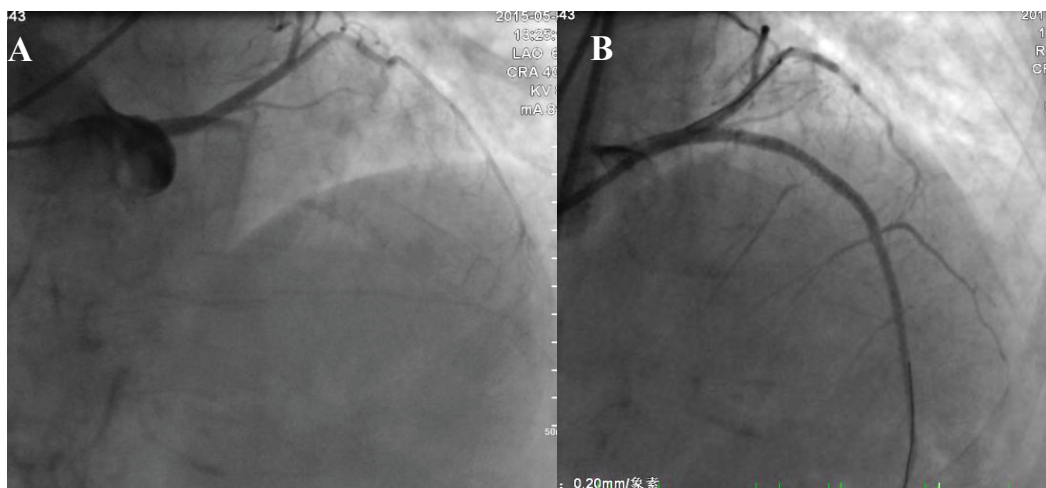


Figure 2: Coronary angiography revealed total occlusion of the proximal LAD (A). Percutaneous coronary intervention was performed in the LAD and a drug eluting stent was deployed to the LAD. (B). LAD:left anterior descending.

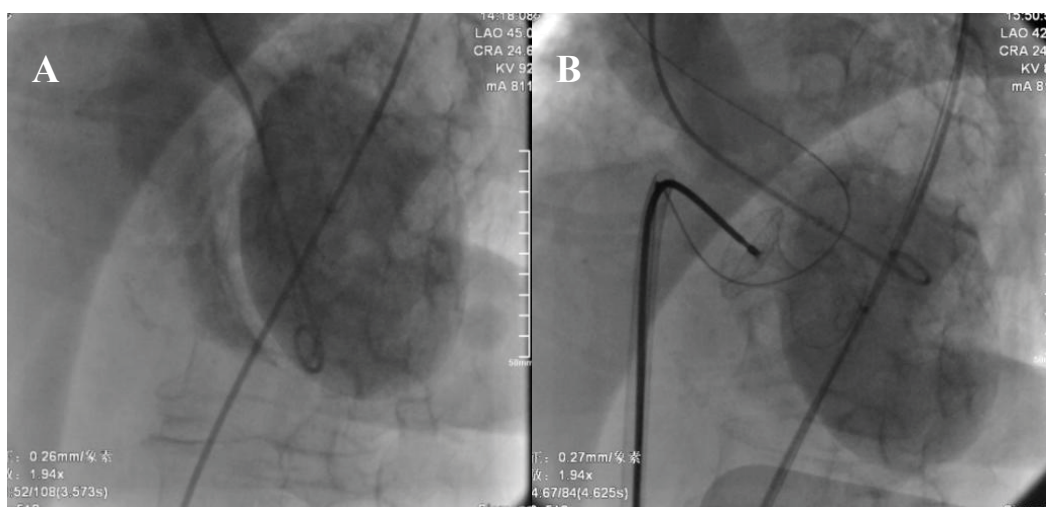


Figure 3: Left ventricular angiogram showing left-to-right shunt through VSR (A). Left ventricular angiography post-Amplatzer implantation occluding VSR (B).VSR: ventricular septum rupture.

on the heart and thus to decrease the magnitude of the left-to-right shunt. Pharmacologic therapy in the setting is usually very limited.

Vasodilators can be used in an attempt to decrease the left-to-right shunt, but the vasodilators are often associated with hypotension and

should be used with caution in hypotensive patients. On the other hand, patients with cardiogenic shock often require vasopressor support. However, vasopressors may increase systemic afterload and further increase the magnitude of the left-to-right shunt, and thus lowering cardiac output and greatly augmenting myocardial oxygen consumption. Unlike vasopressors, IABP can reduce left ventricular afterload, thus increasing cardiac output and decreasing the magnitude of the left-to-right shunt. In addition, IABP can also facilitate diastolic augmentation with a potential increase in coronary blood flow, resulting in an improved oxygen supply.

For all patients with postinfarction VSR, further surgical repair or closure is usually required. However, the optimal timing of surgical repair or closure has been a critical issue of debate. The current guidelines recommend early or urgent surgical repair, preferably before decompensation [6,7]. Due to the fragility of necrotic septum, an aggressive approach may be associated with high perioperative mortality and a high risk of recurrent ventricular rupture [3,6,8], while delayed surgery allows the margins of the infarcted muscle to develop a firmer scar, which may be easier for septal repair. Therefore, if patients could be stabilized by medical treatment or IABP support, a delayed surgical correction may lead to an improved survival [9]. Recently, transcatheter closure is a less invasive option and has become a widely accepted as an adequate bridge to a later surgery or as an alternative to surgical repair. However, percutaneous closure remains one of the most challenging procedures in interventional cardiology and residual shunts are still common [10]. Delayed transcatheter closure may decrease the incidence of residual shunts.

In our case, although the patient developed VSR occurring in the early postinfarction, the left to right shunt was only small initially and ongoing ischemia is a major contributor to the initial clinical scenario. In order to save the dying myocardium, we performed primary PCI initially. As the shunt increased over the next few days, VSR closure became necessary. Because the clinical scenario on IABP and medical supports allowing waiting for delayed VSR closure was feasible in this patient, we performed a delayed percutaneous closure after the 3th postinfarction week. In this particular patient, early revascularization therapy may prevent extensive myocardial necrosis and IABP support as a temporizing approach to bridge the unstable patient to delayed closure of the VSR is effective in stabilizing the patient, thereby allowing the VSR edges to become firmer and fibrotic, which may be beneficial for closure.

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