

Abrupt progression of ventricular septal perforation after primary angioplasty for acute myocardial infarction

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Abstract: A 61-year-old-man was transferred to our hospital because of progressive heart failure after reperfusion for acute myocardial infarction (MI). When he visited the local hospital with severe chest pain associated with inferior MI, transthoracic echocardiography revealed small ventricular septal perforation (VSP). The patient had emergent coronary angiography, which revealed total occlusion of the mid-portion of the right coronary artery. Primary angioplasty was successful for reperfusion. However, because of hemodynamic instability the patient was transferred to our hospital. Under these conditions, transthoracic echocardiography which was undertaken 3 hours after primary angioplasty, demonstrated progressive enlargement of the VSP probably due to reperfusion injury. The rupture site, which was further enlarged at the time of operation, was repaired using the patch exclusion technique. The patient could discharge without complications. We suggest that primary angioplasty may potentially induce late reperfusion injury in patients with VSP complicating MI.

Keywords: Echocardiography; mechanical complication; myocardial infarction (MI)

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Introduction

Although the occurrence of mechanical complication after acute myocardial infarction (MI) has decreased with the introduction of the primary coronary angioplasty, ventricular septal perforation (VSP) is still the leading cause of in-hospital mortality (1,2). Despite the recommendation of aggressive early surgical repair before the onset of hemodynamic deterioration for patients with VSP, mortality remains extremely high (3-7). Herein, we report an acute inferior MI showing immediate hemodynamic instability caused by progressive enlargement of VSP that was observed in serial echocardiography and was treated successfully through emergent surgical conversion.

Case report

A 61-year-old man with a history of diabetes and chronic

kidney disease was referred to his local hospital with chest discomfort and dyspnea. His symptoms had started 18 hours earlier. ECG showed ST segment elevation and abnormal Q waves in the inferior leads. Urgent transthoracic echocardiography revealed akinesis at the inferior wall with small VSP measuring 6 mm of the maximal diameter (*Figure 1A,B*). At this time, elevated creatine phosphokinase of 750 IU/L and serum creatinine of 3.0 mg/dL were observed. The patient underwent emergent coronary angiography, which revealed total occlusion of the mid-portion of the right coronary artery without collateral vessels (*Figure 1C*). Primary angioplasty was indicated for the occluded lesion due to persistence of the patient's symptoms and ST-T changes. Thrombus aspiration and zotarolimus-eluting stent implantation led to partial reperfusion. Importantly, flow of the right ventricular branch and acute marginal branch were preserved after

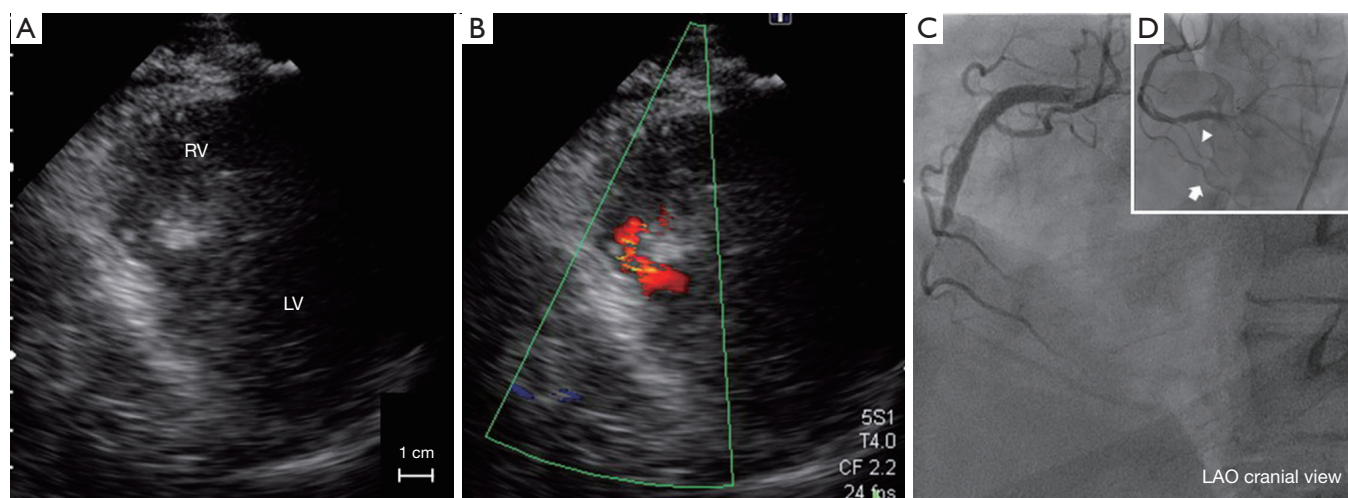


Figure 1 Echocardiography and primary angioplasty. (A) Transthoracic echocardiography in the parasternal short axis view showed ventricular septal perforation measuring 6 mm of the maximum diameter; (B) color flow image showed continuous left to right shunt flow through the ventricular septum; (C) right coronary artery was occluded in the mid portion; (D) final post-stenting angiography resulted in recanalization. Note that flow was preserved in the right ventricular branch (arrowhead) and the acute marginal branch (arrow) after the procedure. LAO, left anterior oblique; LV, left ventricle; RV, right ventricle.

stenting (Figure 1D).

However, half an hour after the interventional procedure, hemodynamic instability occurred. The patient was then transferred to our hospital with an intra-aortic balloon pump in place. At that time, transthoracic echocardiography which was undertaken 3 hours after primary angioplasty, demonstrated progressive enlargement of the VSP (Figure 2). The ratio of pulmonary to systemic flow was 2.8. Thus, emergent surgical repair was indicated. While waiting for surgery, additional mechanical support using percutaneous cardiopulmonary support was necessary to maintain hemodynamics. Intraoperative transesophageal echocardiography, which was performed 6 hours after primary angioplasty, showed further development of the ventricular defect (Figures 3,4).

During operation, the large rupture site (Figure 5A) was repaired using the patch exclusion technique, as described by David *et al.* (10). Concomitant coronary artery bypass grafting (CABG) was not applied to the right coronary artery. Although there remained small residual VSP (Figure 5B,C), he was discharged on the 86th day after admission without complication except hemodialysis.

Discussion

Mechanical complications are relatively uncommon

after acute MI. A previous study has shown that VSP with inferior infarction tended to have a worse outcome compared with anterior infarcts, due to the difficulty of surgical approach and the more frequent association with right ventricular infarction (7). One possible explanation for the successful recovery in this case is that the patient did not suffer from right ventricular infarction, which is compatible with preservation of the right ventricular branch flow that was detected with angiography.

The timing of surgery in patients with VSP remains controversial. This patient initially presented in a stable hemodynamic state that motivated the decision to not repair the VSP emergently; thus, primary angioplasty was performed for the recent inferior MI. After angioplasty, significant hemodynamic alterations occurred and urgent VSP repair was considered in addition to mechanical support. One of the largest studies of VSP cases showed that early repair was an independent predictor of 30-day mortality; however, this should not be interpreted as indicating that surgery should be delayed to reduce mortality (5). Delay of operation could allow the fragile myocardium to transform into firm tissue. On the other hand, the beneficial effects of a delay for a surgical indication are offset by the increases risk of multiple organ failure. Nishida *et al.* reported that preoperative percutaneous cardiopulmonary support was associated with

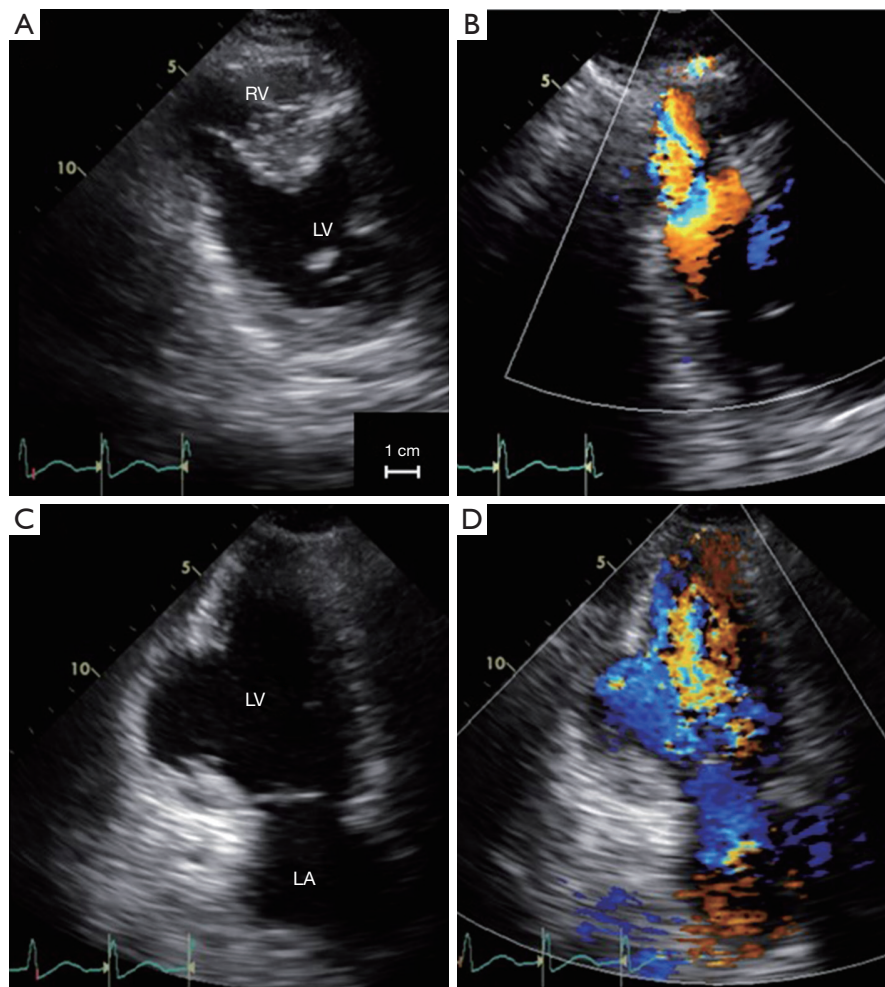


Figure 2 Echocardiography after primary angioplasty. (A) Transthoracic echocardiography in the parasternal short axis view, after transferred to our hospital, showed progressive ventricular septal perforation measuring 18 mm of the diameter; (B) color flow imaging also showed massive shunt flow in comparison with previous examination; (C) echocardiography in the apical two-chamber view showed clear separation of the basal interventricular septum; (D) color flow imaging demonstrated further increases in shunt flow. LV, left ventricle; RV, right ventricle; LA, left atrium.

in-hospital death (3). Under these conditions, the use of transcatheter closure device and stent graft to treat VSP complicating acute MI can be considered as an alternative therapy to open heart surgery (11-13).

As reported in the series of Takahashi *et al.* (6), concomitant CABG and preoperative acute percutaneous coronary intervention accounted for 63.5% and 13.5% of such cases, respectively. However, it remains controversial whether concomitant coronary revascularization improves outcome in patients with VSP. In this case, hemodynamic instability occurred suddenly in the catheterization laboratory while the patient was undergoing angioplasty

for the culprit lesion. Cardiac magnetic resonance imaging suggested that late reperfusion appeared to increase the infarction area compared with conservative therapy for patients with ST-elevation MI (14). Among these patients, the supply of late coronary flow into the infarction area might influence the microvascular blood flow and induce reperfusion injury, thus resulting in enlargement of the infarction area. Even in the previous large-scale clinical investigators, it was hard to prove the clinical efficacy of percutaneous coronary intervention for patients with recent MI (15,16).

From this case, we suggest primary angioplasty may



Figure 3 Intraoperative transesophageal echocardiography from the four-chamber view showed further enlargement of the VSP (8). VSP, ventricular septal perforation.

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Figure 4 Intraoperative transesophageal echocardiography from the four-chamber view in color flow imaging (9).

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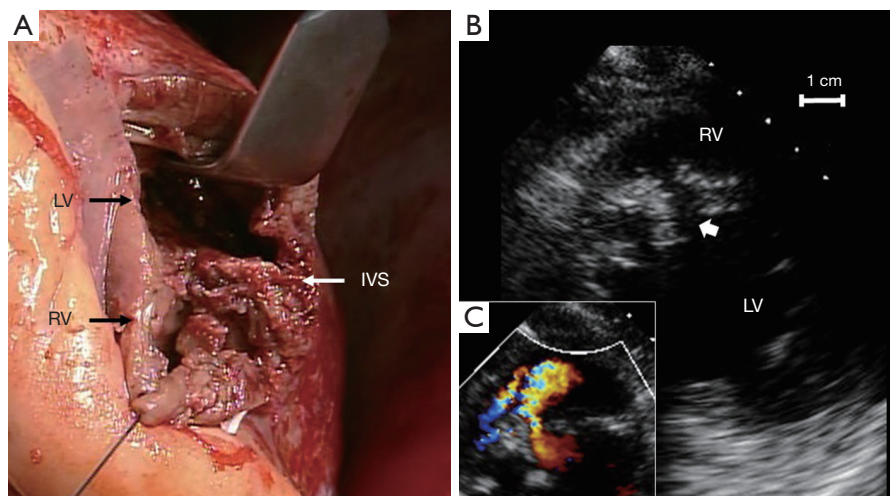


Figure 5 Intraoperative imaging and echocardiography after surgery. (A) During surgery, a large defect within the necrotic interventricular septum (IVS) was observed; (B) transthoracic echocardiography in the parasternal short axis view after surgery; (C) color flow imaging showed small shunt flow. LV, left ventricle; RV, right ventricle.

potentially induce late reperfusion injury in patients with VSP complicating MI.

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None.

Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

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