Interventricular septal rupture after myocardial infarction despite early percutaneous coronary intervention

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Abstract

Ventricular septal rupture (VSR) complicating acute myocardial infarction (AMI) is a serious clinical problem with high mortality rate due to cardiogenic shock or prolonged hemodynamic compromise. Despite multiple improvements in medical, interventional and surgical techniques, early and long-term prognosis after AMI related VSR still remain unpromising. We report a patient in whom an acute VSR was diagnosed 7 days after an anterior myocardial infarction treated with early primary percutaneous coronary intervention (pPCI).

Introduction

Ventricular septal rupture (VSR) is a serious complication of acute myocardial infarction (AMI) that generally causes progressive circulatory failure, rapid clinical state deterioration, and death, despite advances on the treatment of it.[1] The incidence of VSR after AMI was found to be about 0.71 % with using primary percutaneous coronary intervention (pPCI).[1] Although this decrease is promising, the mortality rate of this complication compared to AMI without VSR has remained high.[2] We present the case of a 59 year-old patient with acute VSR related to AMI who underwent a surgical correction, and died shortly after the surgery.

Case Report

The patient was a 59-year-old Caucasian female with no record of hypertension, diabetes, hypercholesterolemia, tobacco use or previously known coronary artery disease. She was not taking any cardiovascular medication. The patient first presented to the emergency department of a hospital complaining of chest pain. She was diagnosed with acute anterior myocardial infarction and referred to our hospital after 2nd hour of symptoms onset. She was admitted to the coronary care unit (CCU) and continued on her ordered medications. Transthoracic echocardiography showed akinesia in the apex and apical septum and mid septum walls, with a calculated ejection fraction of 56%. A CAG showed a 98 % stenosis in the proximal LAD and PCI was performed successfully without any complication (Figure 1 A,B). However, despite full anti ischemic therapies, the patient's condition became worse, with decline of the cognitive state, on the 7th day of AMI. Physical examination revealed a regular pulse of 120 beats/min. She had progressive hypotension and dyspnoea and there was a harsh grade IV/VI systolic murmur best heard at the apex, radiating to the axilla. Pulmonary crackles were heard through lungs field and there was no peripheral edema. Ongoing tachycardia, patient's unstable condition and persistent ST segment elevation and led to an urgent bedside echocardiogram which showed apex, apical septum and mid septum-walls akinesis, a large echodense area at the side of apical septum (Figure 2), through which continuous wave-Doppler examination suggested blood crossing between right and left ventricle (Figure 3). An intra-aortic balloon pump (IABP) was inserted. Early surgical repair was planned. Exploration of the ventricular septum showed an apical rupture (Figure 4). Inspection showed no other aneurysm formation, necrosis or ischaemic region. Unfortunately the postoperative course didn't go well. The patient could not be weaned from the IABP because of severe hemodynamic compromise on the first postoperative day and inotropic drugs were increased to maximum dose. In spite of all efforts she died on the same day of the operation.

Discussion

The contemporary strategy of the treatment of acute coronary syndromes is now focused on primary PCI of culprit lesion using balloon angioplasty and stent implantation, but VSR as the most feared complication of AMI still caries extremely high mortality rate.[1] Clinical features associated with an increased risk of rupture of the interventricular septum include lack of development of a collateral network, advanced age, single vessel coronary artery disease (CAD), hypertension, anterior location of infarction, no current smoking, high Killip class, lower Body mass index (BMI), and possibly thrombolysis.[1,3] Post-infarction VSD is usually located in the anterior or apical portion of the ventricular septum (about 60% of cases)

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Written informed consent was obtained from the kin of the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Contributions: SG, DC, designed the study, carried out subject recruitment, performed echocardiography, analysed the data, and wrote the manuscript; HM, assisted recruitment and manuscript revision. All authors read and approved the final manuscript.

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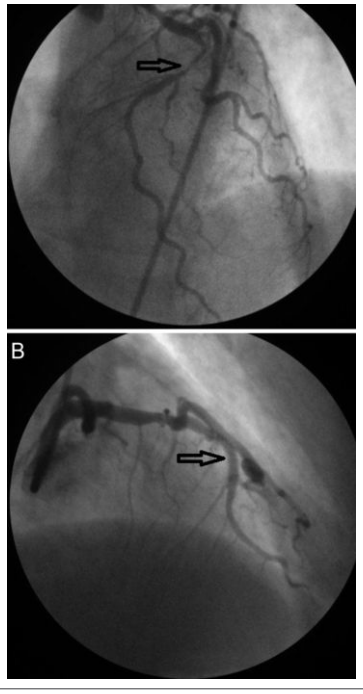

Figure 1. A) Coronary angiography showing 98% stenosis in the proximal LAD. LAD, left anterior descending artery. B) Coronary angiogram revealing successful stenting to the LAD.

as a result of an anterior MI.[4] Patients with VSD had relatively longer time from the AMI onset to primary PCI compared to the non-VSD group and female gender was the strongest independent predictor of AMI related VSD.[1]

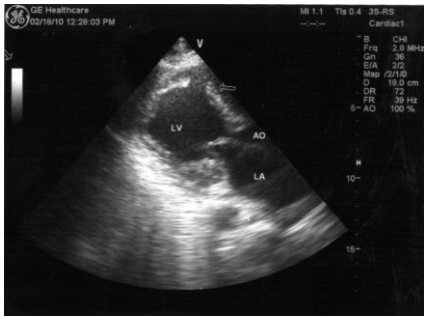

Figure 2. A large echodense area at the side of apical septum.

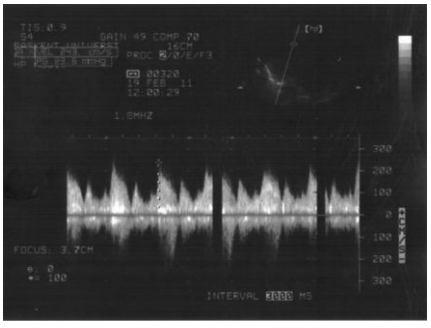

Figure 3. Continuous wave doppler recordings from the parasternal position of an infarct related ventricular septal defect. The peak systolic velocity is 2.4 cm/s, corresponding to a 24 mmHg pressure gradient between the LV and RV. Systolic blood pressure was 100 mmHg; hence, RV systolic pressure=100-24=76 mmHg. There is a continuous shunt through the VSD except during early diastole. LV, left ventricle, RV, right ventricle, VSD, ventricular septal defect.

One hypothesis is based on a concept that, in women, the rupture is attributable to more susceptible collagen framework and to differences in the collagen matrix within the infarcted myocardium.[5] Our patient's demographic characteristics were consistent with these poor outcomes including an anterior infarct with single CAD except for having the chance of early intervention which is about 2nd hour from the onset of AMI symptoms. Although many surgeons recommend surgical VSD closure after a 3-4 week delay to allow scarring of the surrounding tissue to occur, which allows for firmer anchoring of suture and patch material,[6] unfortunately, in this case we were not able to postpone the operation because the patient did express cardiogenic shock despite an IABP. In the ward, the patient was not in a good condition. After three hours of operation, the patient's condition became worse and she died shortly after it.

Conclusions

This case is an example of acute VSR causing catastrophic death, despite percutaneous coronary intervention after myocardial infarction and surgical correction. Awareness of the clinical signs and early use of echocardiography may be life-saving, but the outcomes related to interventricular septum rupture even in the advanced interventional era are bad. More effective ways to predict, prevent, and treat this devastating complication are needed.

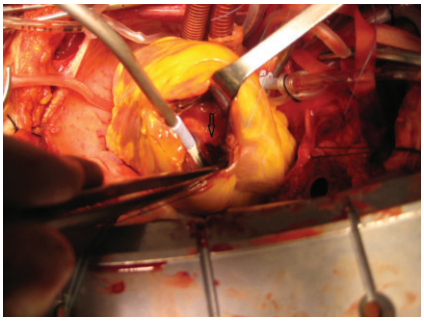

Figure 4. Exploration of the ventricular septum showing an apical rupture.

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