Case Report

Double ventricular rupture after acute myocardial infarction: A rare case report

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A B S T R A C T

A previously asymptomatic 53-year-old male presented 5 days after an acute anterior wall myocardial infarction, who was fibrinolytic naive, with worsening dyspnea. Transthoracic echocardiographic evaluation revealed rupture of the interventricular septum and pseudoneurysm of the left ventricle, confirmed by angiography. Coronary angiogram revealed multivessel disease. The patient underwent successful closure of ventricular septal rupture with repair of pseudoneurysm and saphenous vein grafts to posterior descending branch of right coronary artery and obtuse marginal branch of left circumflex artery. Double ventricular ruptures following acute myocardial infarction are very rare with a reported incidence of 0.3% from various series in the revascularization era. They are also associated with exceedingly high mortality rates reaching up to 50%, even when intervened emergently.

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1. Introduction

Cardiac ruptures following acute myocardial infarction (AMI) consists of ventricular free-wall rupture (FWR), ventricular septal rupture (VSR), and papillary muscle rupture. The incidence of these complications has dramatically reduced in the revascularization era (0.8% for rupture of FWR, 0.2% for VSR, and 0.7% for papillary muscle rupture) in contrast to the pre-fibrinolytic era in which VSR was observed in 1–3% of AMI and accounted for almost 5% of deaths in the peri-infarction period.1,2 The treatment of these complications requires emergent surgical intervention, which is associated with a mortality rate ranging between 20% and 40% for each procedure.3

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2. Case report

A previously asymptomatic 53-year-old male presented in the emergency department with a history of chest discomfort for 5 days and NYHA class IV dyspnea. Examination revealed a heart rate of 90/min and a blood pressure of 110/80 mmHg with a harsh systolic murmur at the lower left sternal border and bilateral fine basal crepitations over the lung fields. Electrocardiogram showed ST segment elevation in anterior precordial leads and right bundle branch block suggestive of proximal left anterior descending artery occlusion (Fig. 1A). He was not on any medication and was fibrinolytic naïve. Chest skiagram showed cardiomegaly with features of pulmonary venous hypertension (Fig. 1B) and transthoracic echocardiogram revealed a VSR in the mid-septum with a gradient of 40 mmHg (Fig. 1C–E; supplementary video 1). Significant collection was noted in the pericardial space with a contained rupture of the ventricular free wall (pseudoaneurysm), which was confirmed in left ventricular angiography (Fig. 2A; supplementary video 2). Coronary angiography revealed total occlusion of right coronary artery and left anterior descending

![Fig. 1](image1.png)

Fig. 1 – (A) 12-lead-electrocardiogram showing right bundle branch block, ST segment elevation in precordial leads, and q waves in leads III, AVF. (B) Chest skiagram showing cardiomegaly and features of pulmonary edema. (C) Transthoracic Color Doppler echocardiogram demonstrating the defect in mid to distal IVS (thick arrow). (D) Echo picture demonstrating the size of defect (ventricular septal rupture). (E) Echo showing gradient across the ventricular septal rupture.

![Fig. 2](image2.png)

Fig. 2 – (A) Left ventricular angiogram demonstrating pseudoaneurysm. (B) Coronary angiogram demonstrating three-vessel disease. (C) Intraoperative demonstration of pseudoaneurysm.
coronary artery and tight stenosis of left circumflex artery (Fig. 2B; supplementary video 3). The patient was taken up for an emergency surgery with intra-aortic balloon pump support. Intraoperatively, the patient was found to have a VSR in mid-septum and rupture at the junction between interventricular septum (IVS) and free wall that was contained by a pseudoaneurysm (Fig. 2C; supplementary video 4). The patient underwent successful closure of VSR with a Dacron patch and the pseudoaneurysm was excised successfully (supplementary video 5). Saphenous vein grafts to posterior descending branch of right coronary artery and obtuse marginal branch of left circumflex artery were performed. The left anterior descending artery was diffusely diseased and was found unsuitable for grafting. The postoperative period was uneventful, and on 5 months follow-up, the patient is doing well and is in a stable NYHA functional class II.

3. Discussion

Ventricular rupture in AMI is a serious mechanical complication and is associated with a bimodal peak. Ventricular FWR is responsible for up to 10–15% of deaths in early AMI. Double ventricular rupture, which is defined as the combination of any two of the three forms of cardiac rupture described and commonly presents as a combination of FWR and VSR. Double ventricular rupture can either present as (1) rupture of both IVS and LV free wall, or (2) rupture at the junction between IVS and free wall. Sometimes, the coexistence of FWR is frequently established only at the time of surgery for the correction of VSR.

Becker and van Mantgem classified the morphology of FWR into three types, which include: abrupt tear in the wall without thinning (type-I), infarcted myocardium erodes before rupture occurs and is covered by a thrombus (type II), and marked thinning of the myocardium with secondary formation of an aneurysm and perforation into the central portion of the aneurysm (type III). This classification can be extended to VSRs also.

The most important factors that increase the likelihood of rupture and affect prognosis are increasing age, female gender, large area of myocardial necrosis, decreased thickness of myocardium at the site of infarction, poorly collateralized coronaries, fibrinolytic use, and time to surgery from symptom onset. Fibrinolytic use is associated with earlier (within 24 h) cardiac rupture when compared to fibrinolytic-naïve patients who present in the 3–5 days of AMI as in our case.

The management of patients presenting with ventricular rupture is surgical and is associated with better outcomes when compared to medical therapy (47% mortality for surgical repair vs. 94% mortality for medical therapy at 30 days post-MI). Our case was a double ventricular rupture, along with a pseudoaneurysm, which developed within a very short time frame, post-AMI. A case like this may have 100% mortality if not intervened emergently.

4. Conclusion

Increasing use of echocardiography in the present era contributes significantly to the prompt diagnosis of cardiac rupture and determining outcomes. Invasive measures like LV angiogram increase the sensitivity of detecting additional complications like pseudoaneurysm as in our case. Tailoring the surgical repair to the extent and site of tear at the time of operation is critical in predicting outcomes. Our case despite its rarity of presentation underwent tailored surgical therapy and had the best possible outcome for the patient.

Conflicts of interest

The authors have none to declare.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.ijhj.2015.06.020.

REFERENCES

1. Crenshaw BS, Granger CB, Birnbaum Y, et al. Risk factors, angiographic patterns, and outcomes in patients with ventricular septal defect complicating acute myocardial infarction GUSTO-I (Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries) Trial Investigators. Circulation. 2000;101:27–32.

2. Edwards BS, Edwards WD, Edwards JE. Ventricular septal rupture complicating acute myocardial infarction: identification of complex and simple types in 53 autopsied hearts. Am J Cardiol. 1984;54:1201–1205.

3. Tanaka K, Sato N, Yasutake M, et al. Clinicopathological characteristics of 10 patients with rupture of both ventricular free wall and septum (double rupture) after acute myocardial infarction. J Nippon Med Sch. 2003;70:21–27.

4. Mann JM, Roberts WC. Fatal rupture of both left ventricular free wall and ventricular septum (double rupture) during acute myocardial infarction: analysis of seven patients studied at necropsy. Am J Cardiol. 1987;60:722–724.

5. Ide H, Ino T, Mizuara A, Yamaguchi A. Successful repair of combined ventricular septal rupture and free wall rupture. Ann Thorac Surg. 1993;55:762–763.

6. Becker AE, van Mantgem JP. Cardiac tamponade: a study of 50 hearts. Eur J Cardiol. 1975;3:349–358.