Case Report

Successful surgical treatment of left ventricular free wall rupture

Hakan Kara

Abstract

Left ventricular free wall rupture (LVFWR) is a rare mechanical complication of acute myocardial infarction. The clinical course of LVFWR is very poor. Direct or patch closure of the rupture area and sutureless procedures constitute the treatment for LVFWR. We present the surgical treatment of a patient who developed LVFWR after high lateral myocardial infarction, and its successful outcome. Successful salvage of LVFWR remains relatively rare. Transthoracic echocardiography, myocardial contrast echocardiography and thoracic computed tomography are important diagnostic tools for LVFWR. These patients usually present with acute cardiac tamponade symptoms requiring immediate treatment.

Keywords: left ventricular free wall rupture, myocardial infarction, cardiac tamponade

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Left ventricular free wall rupture (LVFWR) is a mechanical complication of myocardial infarction with an exceedingly high mortality rate. The overall incidence of LVFWR ranges from 4–6% but it accounts for up to 26% of mortalities associated with myocardial infarction.[1,2] Although its incidence has decreased in parallel with the development of reperfusion techniques in recent years, it remains a fatal complication. It usually occurs within a few days of a myocardial infarction.

The most important diagnostic tool for LVFWR is echocardiography. Early cardiac surgery should be performed in these patients. Traditional techniques are linear closure of the ventricular wall defect and infarctectomy with subsequent closure of the created defect by a prosthetic patch or pericardium. Surgical techniques can sometimes be ineffective in cases of poor myocardial tissue quality. We present the surgical treatment of a patient who developed LVFWR after high lateral myocardial infarction, and its successful outcome.

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Case report

A 51-year-old male patient presented to our emergency department with severe chest pain, dyspnoea, palpitations, significant chest discomfort and confusion. His medical history revealed that he had undergone right coronary artery stenting 10 years earlier, and he had systemic hypertension, alcohol dependence and excessive cigarette smoking. His physical examination revealed an undetectable systolic blood pressure, his heart rate was 130 bpm, and he had dyspnoea, tachycardia and severe jugular venous distension associated with cyanosis of the face and extremities.

His electrocardiogram (ECG) showed an acute high lateral ST-segment elevation myocardial infarction (Fig. 1). Furthermore, his laboratory tests revealed the following: white blood cell count 16.31 cells/µl, haemoglobin 15.3 g/dl, haematocrit 45%, platelets 238.00 cells/µl, glucose 189 mg/dl (10.49 mmol/l), sodium 138 mEq/l, potassium 3.80 mEq/l, blood urea nitrogen 22 mg/dl, creatinine 1.22 mg/dl, aspartate aminotransferase 56 IU/l, alanine transaminase 46 IU/l, C-reactive protein 4.37 mg/dl, lactate dehydrogenase 306 IU/l, total cholesterol 146 mg/dl (3.78 mmol/l), low-density lipoprotein cholesterol 98 mg/dl (2.54 mmol/l) and triglycerides 109 mg/dl (1.23 mmol/l). Cardiac markers showed an elevated creatine kinase mass of 6.70 ng/ml and cardiac troponin level of 8.50 ng/ml.

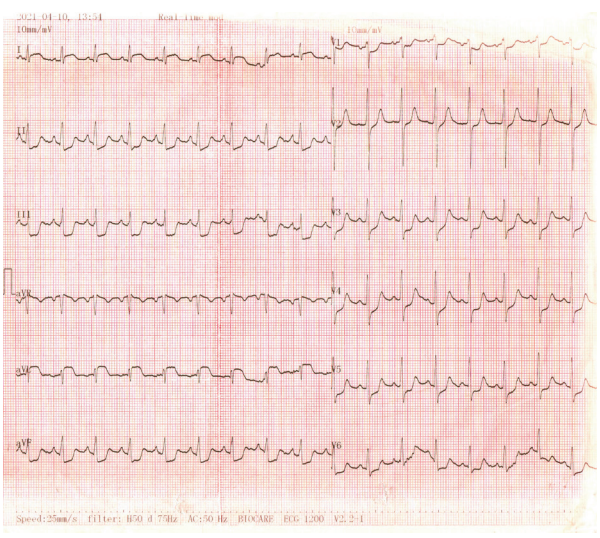

Fig. 1. ECG showing high lateral ST-segment elevation myocardial infarction.

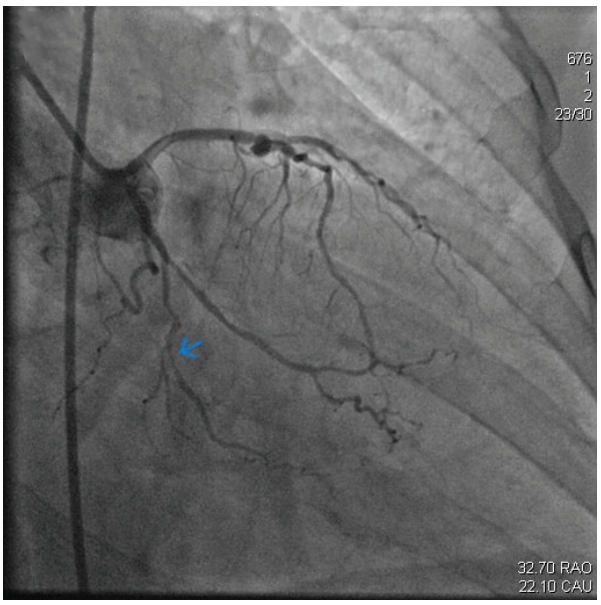

Fig. 2. Left coronary angiogram showing 95% stenosis in the second marginal branch of the circumflex coronary artery (blue arrow).

Coronary angiography was then performed, which revealed 40% stenosis in the left anterior descending coronary artery mid and proximal portion, 50% stenosis in the first diagonalis, 70% stenosis in the first marginal branch and 95% stenosis in the second marginal branch of the circumflex coronary artery (Fig. 2).The stent in the right coronary artery was open.

Percutaneous coronary balloon angioplasty was performed in the second marginal branch of the circumflex coronary artery and resulted in thrombolysis in myocardial infarction (TIMI) flow III. Bedside transthoracic echocardiography (TTE) was performed immediately in the patient whose clinical condition did not improve. TTE disclosed an infero-lateral hypokinetic left ventricle, massive pericardial effusion and suspected rupture area of the lateral left ventricle wall, ejection fraction of 45%, ascending aortic diameter of 30 mm, mild mitral regurgitation, and mild left ventricular hypertrophy. Thoracic computed tomography revealed 28-mm thick haemoragic fluid collection in the pericardial cavity (Fig. 3).

The patient was rapidly transported to the operating room for emergency cardiac surgery with a diagnosis of massive haemopericardium and cardiac tamponade. The pericardium was opened. Much blood and a massive clot were removed from the pericardial space. On direct inspection, a linear rupture of approximately 2 cm in length was observed in the upper lateral part of the left ventricle (Fig. 4). At this point, the patient had a cardiac arrest. A short period of internal cardiac massage was performed.

Cardiopulmonary bypass was initiated by rapid cannulation. A cross-clamp was placed and cardioplegia was given from the aortic root. The patient was cooled to 28°C. The ventricular rupture was closed with interrupted polyproplene horizontal mattress sutures buttressed by twoTeflon felt (Fig. 5). The patient was weaned off cardiopulmonary bypass with moderate doses of inotropes.

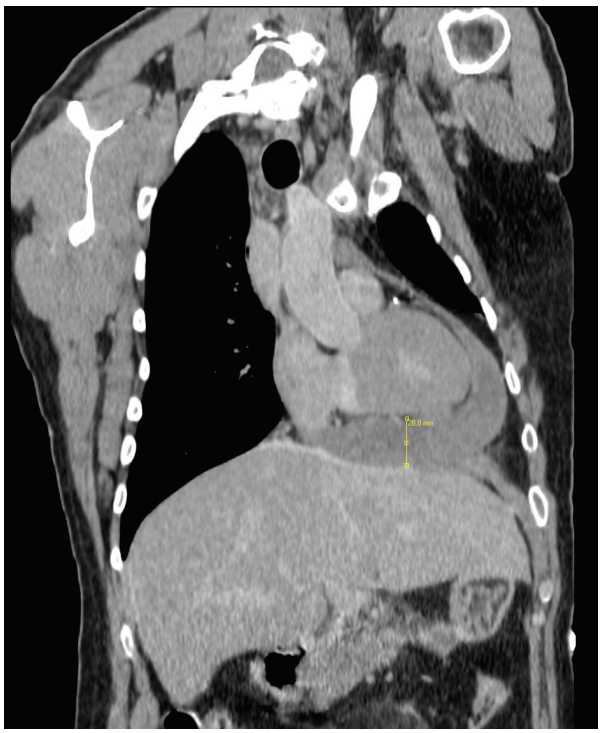

Fig. 3. Thoracic CT scan showing intrapericardial haemorrhagic fluid and clot.

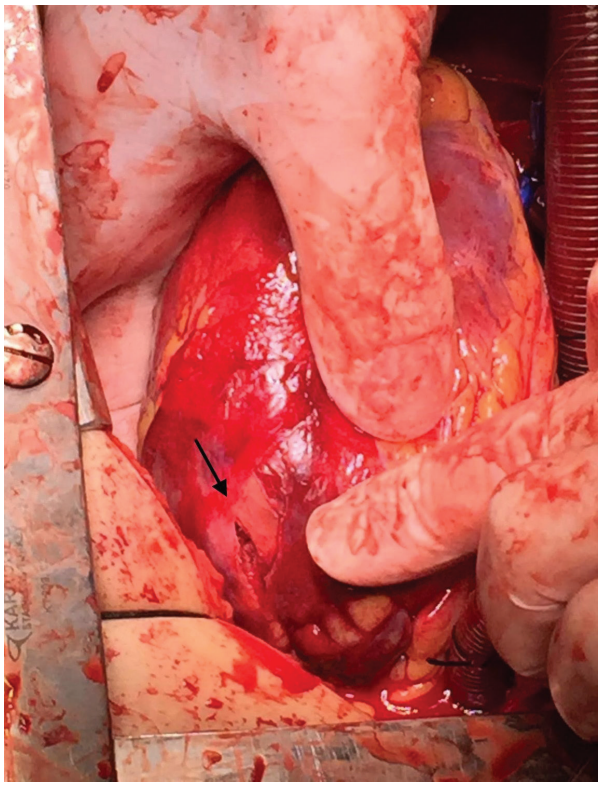

Fig. 4. Intra-operative photograph showing myocardial rupture in the high lateral wall of the left ventricle (black arrow).

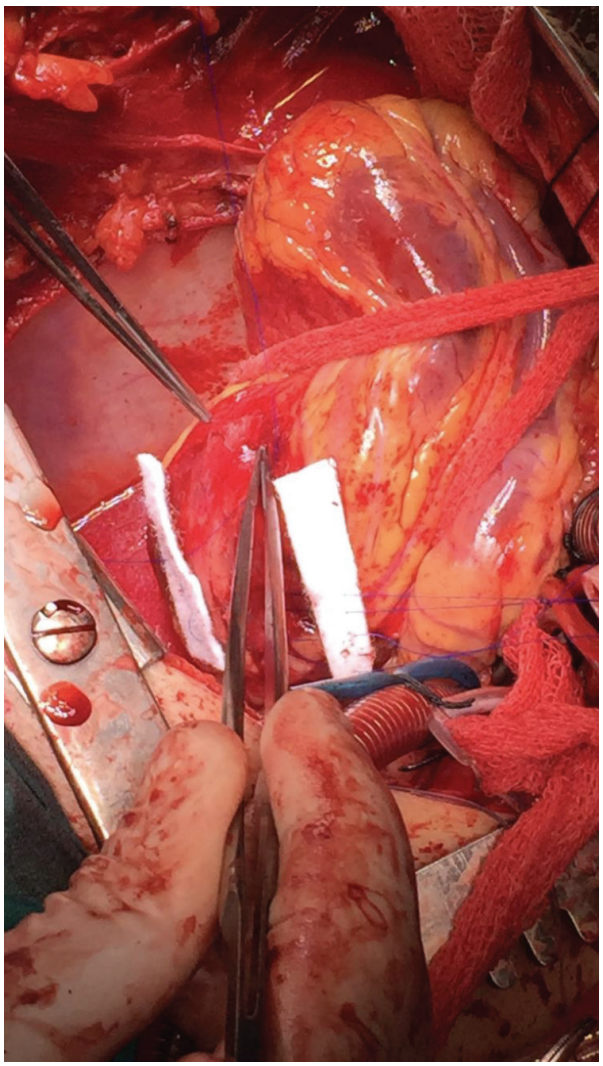

Fig. 5. Intra-operative photograph showing rupture is closed with interrupted polyproplene horizontal mattress sutures buttressed by twoTeflon felt.

He was discharged on the 13th postoperative day. No complications were observed in the postoperative follow ups with TTE.

Discussion

LVFWR is a catastrophic complication of myocardial infarction and it is associated with old age, lack of collateral circulation, ischaemic preconditioning and presentation with a first myocardial infarction. It can occur in two types. An acute form, also referred to as blowout, involves sudden pulseless electrical activity and death. The subacute form of free wall rupture (also referred to as oozing) does not involve cardiac arrest, but describes cases of moderate-to-severe pericardial effusion, chest pain, hypotension, dyspnoea, cyanosis and confusion.[1] In our study it was a subacute blowout type of LVFWR.

Cardiac ruptures can occur in any portion of the heart depending on the myocardial infarction involvement area, most frequently in the end regions supplied by the left anterior descending artery, especially in the anterior and lateral walls. Factors causing rupture include thinning of the wall in the coronary end-flow area, lack of collateral circulation, and deterioration of elastic structures in the tissue after transmural myocardial infarction.

LVFWR is common during the early stages following the onset of myocardial infarction.[3] It usually leads to haemopericardium. Immediate pericardiocentesis will temporarily relieve tamponade, but pericardiosentesis cannot always be performed in cases of severe haemodymic collapse. Moreover, it is often unsuccessful because much of the pericardial space is taken up by undrainable clots. In this case, we did not perform pericardiocentesis because the patient's clinical condition allowed cardiac surgery. Intra-aortic balloon pumping (IABP) is sometimes used to reduce the afterload of the left ventricle.

In cases of circulatory collapse, rapid placement on extracorporeal membrane oxygenation (ECMO) support may provide sufficient circulation, but poor venous return in cases with tamponade may limit ECMO blood flow.[4] We did not use IABP or ECMO.

Diagnosis can be made by several imaging techniques, including echocardiography, computed tomography, contrast ventriculography and magnetic resonance imaging.[5] Echocardiography may demonstrate a pericardial effusion and typical findings of cardiac tamponade.

Køber *et al*.[6] suggested that in cases of a small amount of pericardial effusion after myocardial infarction, the possibility of wall rupture should be considered as the first stage, patients should be approached with suspicion of rupture until proven otherwise, and hourly echocardiography follow ups should be performed.

Contrast echocardiography, has the potential to diagnose LVFWR prior to the development of cardiac tamponade. Contrast material can be visualised into the myocardium before reaching the pericardial space, suggesting an impending rupture. Trindade *et al*.[7] and Okabe *et al*.[8] revealed the early diagnosis of LVFWR by contrast echocardiography.

Cardiac magnetic resonance imaging is especially useful in detecting rupture location and size in haemodynamically stable patients.[9] In this case, we confirmed the diagnosis with TTE and thoracic computed tomography.

The principles of surgical treatment of LVFWR are to relieve tamponade, close the rupture area and prevent recurrence of rupture or pseudoaneurysm formation.[5] The preferred technique is linear closure supported by Teflon felt. However, in the presence of excessive necrosis, an infarctectomy is followed by closure with materials such as dacron or pericardium.[10]

Roberts *et al*.[11] treated a LVFWR that developed nine days after myocardial infarction with a dacron patch. The ventricular wall rupture was closed with interrupted polyproplene horizontal mattress sutures buttressed by twoTeflon felt under cardiopulmonary bypass in our patient.

Tissue surrounding the injury site is usually in a poor condition and vulnerable to manipulation. Surgical techniques can sometimes be ineffective in cases of poor tissue quality, increasing the risk of enlargement of the rupture. Padró *et al*.[12] reported 13 successful cases treated by a sutureless procedure. They applied a polytetrafluoroethylene patch over the infarcted area, which was attached to the heart surface with surgical glue.

Misava *et al*.[13] reported a sutureless technique can be a

promising strategy for the treatment of ischaemic rupture, but serial echocardiographic studies should be mandatory for diagnosing a left ventricular pseudoaneurysm formation thereafter. Bergman *et al*.[14] demonstrated the feasibility of using collagen sponges and haemostatic matrix sealant for effective haemostatic closure of ventricular free-wall ruptures when the tissue quality is poor. The use of intrapericardial fibrin-glue and thrombin injection, as an alternative sealant, have also been reported.[15,16]

Conclusion

LVFWR is a severe and deadly mechanical complication of myocardial infarction. It should be suspected in any patient with cardiac tamponade symptoms following myocardial infarction. Patients should immediately be taken into cardiac surgery and the ruptured area should be repaired. Prompt diagnosis and surgical intervention can lead to successful treatment for LVFWR.

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