

Post-reperfusion LV Free Wall Rupture: Early Diagnosis and Surgical Repair Can Be Life Saving - A Rare Case Report

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PRESENTATION OF CASE

A 51-year-old male patient was admitted to the hospital with sudden onset chest pain and diaphoresis. He had a history of smoking with a total load of 40 pack-years, was non-diabetic and non-hypertensive. ECG showed ST elevation in leads II, III and aVF s/o acute inferior wall myocardial infarction. High levels of cardiac biomarkers (TnI and CK-MB) were recorded. He was thrombolysed with intravenous Streptokinase. Coronary angiogram showed 99% stenosis in mid RCA for which he was advised percutaneous angioplasty to RCA but the patient denied further intervention and took discharge against medical advice. 5 days later he again presented with an episode of acute onset chest pain. ECG was suggestive of reinfarction in inferior wall (as shown in figure 1) with high levels of CK-MB. Coronary angiography revealed 100% stenosis in mid RCA for which he underwent percutaneous angioplasty with drug eluting stent with a post procedure TIMI flow of grade 3 and the patient was transferred to the CCU for routine monitoring. However, 6 hours later patient developed diaphoresis and persistent hypotension. So further evaluation was done.

2D echo revealed akinetic inferior wall of left ventricle and moderate pericardial effusion. Cardiac CT showed 27 x 24 mm pseudoaneurysm of postinfarct inferior wall of left ventricle at the level of mid-cavity with moderate pericardial effusion (as shown in figure 2). The patient was transferred in department of cardiovascular surgery for the intervention of LV free wall rupture repair. The pericardium was opened via a median sternotomy. A significant amount of blood and multiple clots were removed. The heart was arrested with antegrade cold blood cardioplegia. LV free wall rupture (as shown in figure 3) was repaired using a composite Dacron and autologous pericardium patch (as shown in figure 4). The postoperative evolution was uneventful, and patient was discharged after 10 days of hospitalization.

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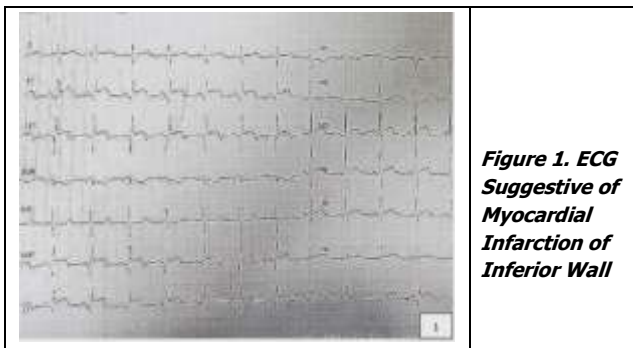


Figure 1. ECG Suggestive of Myocardial Infarction of Inferior Wall

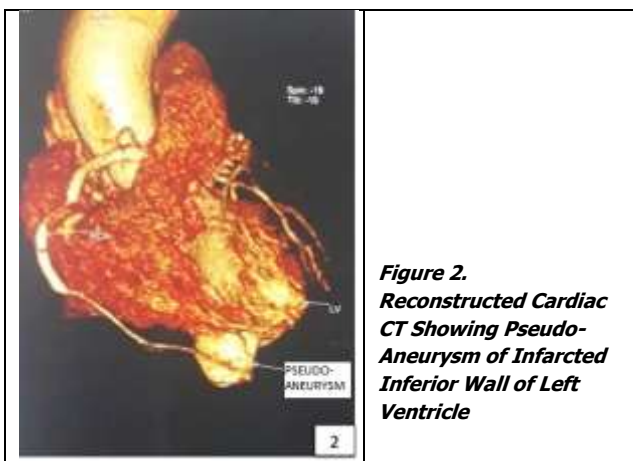


Figure 2. Reconstructed Cardiac CT Showing Pseudo-Aneurysm of Infarcted Inferior Wall of Left Ventricle

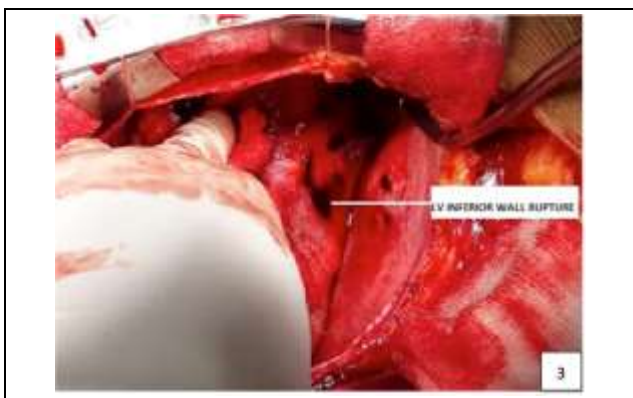


Figure 3. Rupture of Free Wall of Left Ventricle Noted at Thoracotomy

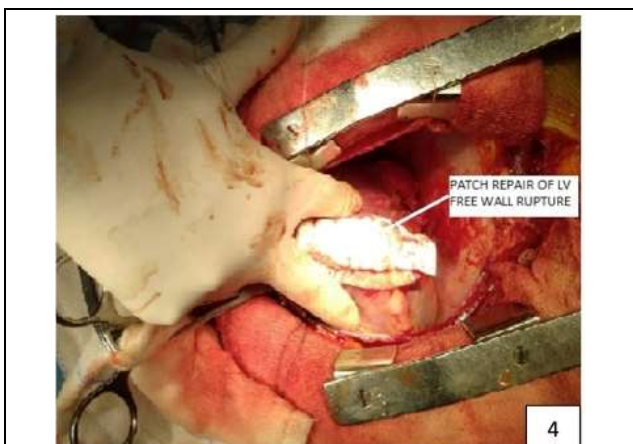


Figure 4. Patch Repair of Ruptured LV Free Wall

DIFFERENTIAL DIAGNOSIS

Differential diagnosis of LV pseudoaneurysm includes pulmonary embolism, aortic dissection, LV aneurysm, LV diverticulum, acute myocardial infarction.

PATHOLOGICAL DISCUSSION

The pathologic substrate of LVFWR consists of intramyocardial hemorrhage and microvascular obstruction.¹ Becker and colleagues identified 3 morphological types of LV free wall rupture. Type 1 rupture is characterized as an abrupt, slit-like myocardial tear and corresponds to the acute phase of MI (<24 hours). In type 2 rupture, an area of myocardial erosion is evident, indicating a slowly progressive tear. Type 3 rupture has marked thinning of the myocardium and perforation in the central portion of aneurysm, which typically occurs during the late phase of MI (>7 days). This pathological classification system can be also applied to VSR.²

DISCUSSION OF MANAGEMENT

Post-Reperfusion Therapy LV Free Wall Rupture

Reperfusion therapy appears to aid in the prevention of cardiac rupture, especially late-phase rupture. However, reperfusion therapy does not adequately prevent ruptures in patients with anterior MI, in whom ruptures are more likely to occur in the early phase.³ Historically, the first clinical reference to post infarction LV free wall rupture was reported by William Harvey in 1647. Despite being theoretically more probable in the inferolateral wall, it seems that the fact that anterior infarcts are more common, renders anterior wall ruptures more frequent. However, in the specific case of subacute LV wall rupture, inferior infarctions seem to be involved in the majority of the cases.⁴ In the presented case the localization of LVFWR was on the inferior free wall. In the LATE trial, Becker et al. found no significant differences in the incidence of LVFWR in patients treated with a rt-PA based regimen within the 6- to 12-hour period after symptom onset and those randomized to the active treatment arm between 12 and 24 hours. However, there was a significant interaction between the assignment to active treatment and the time of rupture occurrence, as thrombolysis appeared to accelerate rupture events, typically to within 24 hours after treatment.⁵

Post-Revascularisation LV Free Wall Rupture

Whether or not PCI would have similar effects has been recently examined in a retrospective analysis of a single centre registry of 1250 patients treated with PCI. In the 12 patients who had LV rupture (including free wall rupture and VSR), this complication occurred somewhat later than with thrombolysis, but earlier than that reported for non-reperused patients.⁴

Prognosis and Management

LVFWR is still associated with high mortality rates (50-60%),^{2,6} accounting for 95% of electromechanical dissociation in patients with acute Myocardial infarction.⁷ Prompt surgical intervention can be lifesaving as it was emphasized in our report.

CONCLUSIONS

We consider that the case reported here highlights the importance of reperfusion injury and partial or aborted ruptures. This case equally demonstrates that left ventricular free wall rupture is not always fatal and that early diagnosis and emergency surgical therapy may be lifesaving.

Abbreviations

AMI- Acute Myocardial Infarction, CCU- Critical Care Unit, CK-MB- Creatine Kinase- MB, CT- Computed Tomography, LV- Left Ventricle, LVFWR- LV Free Wall Rupture, PCI- Percutaneous Coronary Intervention, rTPA- Recombinant Tissue Plasminogen Activator, RCA- Right Coronary Artery, TnI- Troponin I, VSR- Ventricular Septal Rupture.

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