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Left Ventricular Free Wall Rupture Following Acute Myocardial Infarction: A Case Report and Discussion

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ABSTRACT

We report a case of left ventricular free wall rupture (LVFWR) in a 57-year-old female who developed an abrupt myocardial infarction. The diagnosis was confirmed using echocardiography. The patient underwent a successful surgical repair involving suturing, patch insertion, and coronary artery bypass grafting (CABG). The present case demonstrates that, while LVFWR is generally deadly, it can be adequately controlled with timely diagnosis and surgical intervention.

Keywords: left ventricular free wall rupture (LVFWR), Acute myocardial infarction (AMI), coronary artery bypass grafting (CABG).

INTRODUCTION

Acute myocardial infarction (AMI) remains the leading cause of morbidity and mortality, with myocardial rupture being a relatively rare but devastating consequence. Left ventricular free wall rupture (LVFWR) is one such manifestation, with a high mortality rate and difficult management. Risk factors for LVFWR include advanced age, female gender, hypertension, and specific MI sites.[1,2] Recognizing prodromal symptoms and using early diagnostic methods such as echocardiography is critical for timely intervention.[3,4] Surgical repair remains the most common choice, emphasizing the importance of prompt recognition and intervention in improving outcomes.[5]

CASE REPORT

The patient, a 57-year-old female, presented to the emergency room with severe chest pain accompanied by hypotension. She had a history of hypertension but no prior cardiac events. Initial assessment suggested a non-ST-segment elevation myocardial infarction (NSTEMI).

Clinical Presentation:

Upon arrival, the patient was in a state of hemodynamic collapse, demonstrating signs of systemic hypoperfusion and cardiogenic shock. Inotropic support was initiated, and supportive measures were provided. Echocardiography revealed a large pericardial effusion with clots and moderate left ventricular (LV) dysfunction.

Diagnostic Workup:

Troponin levels were elevated, confirming the diagnosis of NSTEMI. Coronary angiogram showed double vessel disease with left main involvement, necessitating early coronary artery bypass grafting (CABG). A CT aortogram was performed to rule out dissection, revealing a large

hemorrhagic effusion with organized clots over the LV apex and lateral wall.

Surgical Procedure:

The patient underwent median sternotomy, revealing a significant clot and hemorrhagic fluid in the pericardial cavity and on the LV apex and lateral wall. The left internal mammary artery (LIMA) and saphenous vein graft (SVG) were harvested for CABG. CABG was performed using the beating heart technique, with temporary intracoronary shunt placement. Additionally, a pericardial patch was applied to the LV wall using glue and hemostats.

Postoperative Course:

The immediate postoperative period was uneventful. The patient remained hemodynamically stable and was fully ambulatory. She was discharged from the hospital after recovery.

DISCUSSION

Myocardial rupture, a consequence of acute myocardial infarction (AMI), causes death in 8% of patients.[2] Left ventricular free wall rupture (LVFWR) is one of its most severe forms, however its occurrence can be reduced with immediate primary percutaneous intervention.[1] LVFWR has several risk factors, including advanced age, female gender, hypertension history, and involvement of the lateral or anterior walls during AMI.[1,5]

The classic expression of LVFWR, known as ADAM Classic LVFWR, usually exhibits symptoms within the first 24 hours after AMI and always by the end of the first week.[4] The severity and rate of pericardial bleeding determine the clinical presentation, which frequently results in abrupt hemodynamic collapse, electromechanical dissociation, and death. However, in certain circumstances, clot development may temporarily plug pericardial leakage, causing the creation of a left ventricular pseudoaneurysm. Furthermore, a chronic type of LVFWR characterized by slow recurrent bleeding is seen in roughly one-third of cases, allowing patients to live until emergency surgical intervention.[6]

A number of studies have sought to identify premonitory signs and symptoms of fatal LVFWR, such as chronic chest pain, intractable vomiting, restlessness, and prolonged ST elevation and positive T wave deflection more than 72 hours after AMI onset. Notably, typical symptoms of cardiac tamponade such pulsus paradoxus and diastolic pressure equalization are frequently absent. Although electromechanical dissociation is possible, its diagnostic use is limited.[3]

Echocardiography emerges as the gold standard for diagnosing LVFWR, with distinctive findings such as pericardial effusion, intrapericardial echoes, and occasional right heart chamber collapse. Its diagnostic sensitivity and specificity are listed as 100% and 93%, respectively.[3,7] LVFWR should be regarded the primary diagnostic in hypotensive AMI patients with pericardial effusion.

Emergency surgical repair remains the definitive treatment for LVFWR, often involving pericardial patch placement with biological glue or epicardial sutures. Alternative techniques such as infarctectomy patch placement and ventricular wall reconstruction may be employed. Therapeutic measures including rapid fluid infusion, positive inotropic agents, and pericardiocentesis are adjunctive in managing LVFWR-related hemodynamic instability.[4,5]

In the reported case, the patient exhibited symptoms suggestive of subacute LVFWR, despite experiencing slow blood leakage causing hemodynamic instability. Rapid surgical intervention combining repair and

coronary artery bypass grafting effectively managed the condition. The decision to perform emergency coronary angiography preoperatively to determine bypass targets or proceed directly to surgery remains a clinical dilemma.[5]

CONCLUSION

In conclusion, the case underscores that LVFWR, although ominous, is not invariably fatal. Early diagnosis through echocardiography combined with prompt surgical repair significantly reduces mortality rates. In instances of hemodynamic instability, instituting intra-aortic balloon pump (IABP) support facilitates successful bridging to definitive emergency surgical therapy.

Declaration by Authors

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