CASE REPORT

Ventricular free wall rupture–a rare under-diagnosed fatal complication of myocardial infarction

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Abstract

Ventricular free wall rupture is a rare complication of acute myocardial infarction (MI) i.e. <1.7% of cases, although it occurs more frequently than might be expected. Without imaging evidence or autopsy, sudden cardiac death in acute MI setting is commonly attributed to fatal dysrhythmias, high degree atrio-ventricular blocks or pulmonary embolism resulting in underdiagnosis of free wall rupture. Modern therapies have probably reduced the incidence but not eliminated the problem.

We present a case of left ventricular free wall rupture in acute MI setting and syncopal presentation. The diagnosis was established during left heart catheterisation and further confirmed on transthoracic echocardiography. The patient deteriorated abruptly, pericardial window was made to bridge for surgical repair. Unfortunately, the patient died before surgical intervention.

Keywords: Left ventricular free wall rupture; Myocardial infarction; Coronary angiogram; Transthoracic Echocardiogram

DOI: https://doi.org/10.5455/JPMA.51034

Introduction

[1]The incidence of ventricular free wall rupture in myocardial infarction setting has declined from 3.3% in 1977 to 1.7%, in 2011.[1] It may occur from the first day to three weeks after infarction, but most ruptures occur three to five days after MI. The clinical presentations include chest pain, acute dyspnoea, shock, diaphoresis and syncope.[2] Approximately 50% of patients die within five days, and 82% die within two weeks of index infarction. Prompt early diagnosis and urgent surgical intervention may improve survival rate upto 75%,but, unfortunately, in most of the cases it remains undiagnosed.

Case Report

A 65-years-old hypertensive male with a history of typical chest pain since 48 hours but pain-free since nine hours presented on March 2019 at Tabba Heart Institute, Karachi, with an episode of syncope four hours earlier. The patient initially presented to another hospital where he was diagnosed as inferior wall myocardial infarction with late arrival with Mobitz I Atrioventricular (AV) block and was referred to our hospital for further management after loading with Aspirin 300 mg, Clopidogrel 300 mg statalong with weight-based low molecular weight heparin. Upon arrival to Tabba Heart Institute, Karachi, he was completely pain free with BP 130/90 mmHg and Pulse 110 beats/minute. Chest was clear on auscultation and no murmur or added sounds were appreciated on cardiovascular exam. Follow-up ECG showed ST segment elevation with Q waves in inferior leads with no AV block. In view of electrical instability, immediate coronary angiogram was done, which showed severe multi-vessel disease with pericardial effusion. (Figure 1) Palm echocardiography revealed moderate to severe non-tamponade pericardial effusion with coagulum which was later on confirmed by bedside echocardiography, which showed mild to moderate left ventricular dysfunction, normal right ventricular function, normal valves and tear on basal inferior wall of left ventricle, consistent with ventricular free wall rupture. (Figure 2) Emergent surgical repair with coronary artery bypass grafting was planned. While the patient was being prepared for urgent surgery, he became sweaty and developed progressive hypotension and required resuscitation within intravenous fluids. The patient's condition deteriorated abruptly and he was taken on maximal dual ionotropic support. The patient had pulseless electrical activity (PEA) arrest with the need of continued CPR for 16 minutes, meanwhile pericardial window was made as a bridging procedure to surgery. Return of spontaneous circulation was achieved for two minutes and the patient underwent second episode of PEA

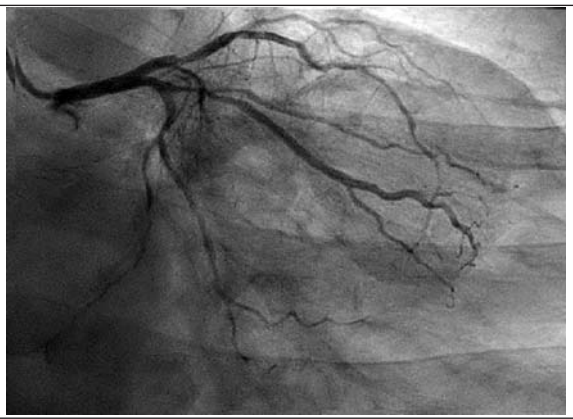

Figure-1: Coronary angiogram showing pericardial effusion.

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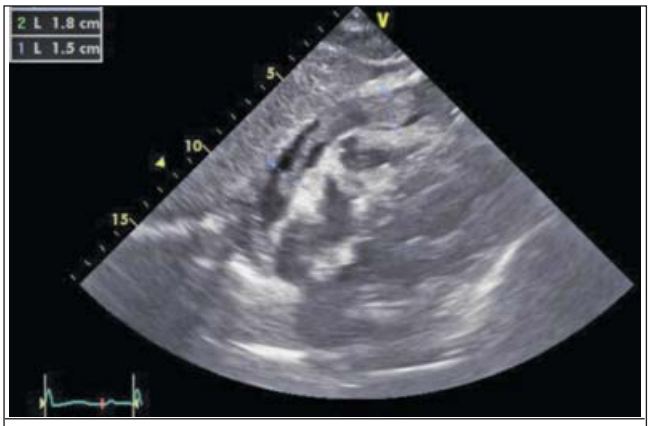

Figure-2: Echocardiogram showing pericardial effusion with co-agulum.

arrest, CPR was resumed and continued for 30 minutes, but he could not be revived this time.

Discussion

Left ventricular free wall rupture is an under-diagnosed fatal complication of acute myocardial infarction.[3] It is more commonly seen in women with first MI. Additional risk factors include hypertension and advanced age.[4] It frequently develops between three to six days after an MI. However, left ventricular free wall rupture (LVFWR) may also develop within a few minutes following an AMI or even one month later.[5] The mechanism of underlying rupture of heart muscle is still controversial but it has been suggested to be due to the dissociation of the non-contractile infracted region from surrounding healthy and contractile region.[6] Based on clinical course, LVFWR is further subdivided into three types as acute, sub-acute and chronic.

Acute rupture is life-threatening due to massive bleeding in pericardial cavity and cardiac temponade. In sub-acute rupture, leakage to pericardial cavity is limited by pericardial adhesions or clots and present clinically with stable haemodynamics, but rarely present with cardiogenic shock. In chronic rupture, formation of pseudoaneurysm prevents leakage of blood flow into the pericardial cavity.[7] Our case of LVFWR occurred in acute setting. Diagnosis of LVFWR requires high clinical suspicion.[8] Transthoracic echocardiography is usually required for diagnosis. Other diagnosing modalities include cardiac MRI and computed tomography and can be considered in haemodynamically stable patients. Urgent surgical repair is the definitive treatment,[9] for acute or sub-acute left ventricular free wall rupture, though the mortality risk is high.[10] Pericardiocentesis or formation of pericardial window may be used as bridging intervention while awaiting surgical repair.

Conclusion

Compared to the thrombolytic era, the current incidence of LVFWR with acute myocardial infarction, who reach the hospital alive, is significantly lower. However, 30-day mortality continues to be high. Early diagnosis is the key and prompt management is required to reduce its mortality.

Consent: Hospital admitting informed consent taken at the time of admission will be considered as consent for publishing this study.

Disclaimer: None.

Conflict of interest: None.

Funding Sources: None.

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