Left Ventricular Rupture: Where is the Key in Management?

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Abstract

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Received on 05.04.2017, Accepted on 24.04.2017

In the modern era of reperfusion, left ventricular free-wall rupture is seen in less than 1% of myocardial infarctions. Typically, acute left ventricular free-wall rupture leads to sudden death from cardiac tamponade. Here we present a case of left ventricular rupture as a complication of inferolateral wall myocardial infarction being saved by timely action and coordination of cardiologists and cardiothoracic surgeons. By presenting this case we would like to highlight that even deadliest complications like left ventricular free wall rupture can be treated and saved if aggressively managed.

Keywords: Left Ventricular Rupture; Myocardial Infarction; Patch Closure; Cardiac Temponade.

Introduction

Free wall rupture is the most lethal complication of acute myocardial infarction (AMI), and is responsible for about 10-15% of in hospital deaths [1]. Rupture occurs most frequently in the left ventricular free wall, less frequently in the right ventricular free wall and rarely in the atrium. Patient can be saved only if early diagnosis and prompt treatment is given. But most die before getting to surgery. This article presents a case of acute myocardial infarction with cardiogenic shock detected with free wall rupture and cardiac tamponade and was surgically repaired by temporarily stabilizing on pericardiocentesis and auto transfusion.

Case

A70 yrs male non diabetic / hypertensive presented to emergency room with complaints of chest pain since 3 days and few episodes of prolonged unconsciousness since early morning. In the emergency room electrocardiogram taken showed s/o inferolateral wall myocardial infarction (Figure 1), screening echo confirmed the diagnosis and patient was started on inotropes for hypotension and

tachycardia, but loading of antithrombotics were withheld to get clearance from neurologist for prolonged unconsciousness. Meanwhile patient was shifted to CCU for further assessment, a departmental/detailed echo was done in view of persistent tachycardia and hypotension on inotropes in CCU and signs of impending cardiac temponade were confirmed (Figure 2). Patient went on to have similar episodes of loss of consciousness as explained earlier by relatives. With this information a diagnosis of

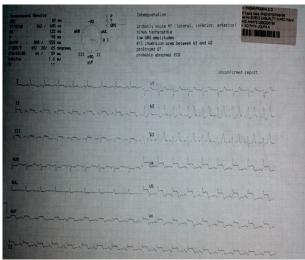


Fig. 1: Electrocardiogram showing signs of inferolateral wall myocardial infarction in a 70 yrs male

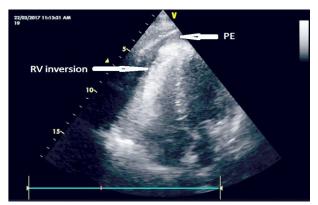


Fig. 2: 2D ECHO apical 4 chamber view showing pericardial effusion impending temponade

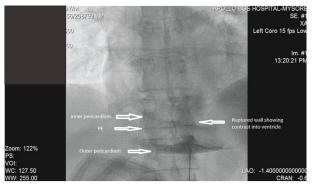


Fig. 3: Cine AP view showing ruptured ventricle, free floating contrast from pericardial cavity to ventricle

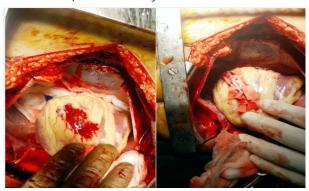


Fig. 4: Showing the site of LVR with partially occluded clot (left), patch closure of same rupture (right)

cardiac temponade probably due to left ventricle rupture was made. Patient took urgently for therapeutic pericardiocentesis and a cardiothoracic reference was taken. On table flouro pictures from pericardium confirmed rupture (Figure 3) and patient was shifted to operation theatre for patch closure meanwhile keeping the patient on auto transfusion. Patient then successfully underwent patch closure of left ventricular inferoposterior wall just across the posterior descending artery (Figure 4) on intra-aortic balloon counterpulsion (IABP) support. On post op day 2 patients vitals were stable and was out of IABP, inotropes and ventilatory support with normal vitals

and ready to shift to ward.

Discussion

Myocardial rupture as a complication of acute myocardial infarction directly causes death in approximately 8% of patients [2]. A grave form of this complication is left ventricular rupture (LVR), whose incidence appears to be decreasing in patients undergoing primary percutaneous intervention [3].

Patients with LVR often present with cardiac tamponade, acute cardiogenic shock, heart failure, or sudden death. However, some patients with contained and subacute rupture might experience a more indolent course [4]. Echocardiography is the procedure of choice for the diagnosis of LVR and usually reveals a pericardial effusion with intrapericardial echoes consistent with hematoma. Occasionally, diastolic collapse of the right ventricle can be seen. Echocardiography has a diagnostic sensitivity of 100% and a specificity of 93% [5].

Acute nonsurgical management involves aggressive volume resuscitation with inotropic and vasopressor support, pericardiocentesis, and percutaneous circulatory support, such as intra-aortic balloon counterpulsation and extracorporeal membrane oxygenation [6,7]. Surgical repair is the only definitive treatment, still successful surgery carries a high mortality rate [8].

Emergency surgery is usually the only therapeutic option available. Several different techniques have been described in the literature. These include direct closure without excision; excision of infarct segment and direct closure or closure using a prosthetic or autologous patch. Recent reports suggest that the conventional technique of infarctectomy might be avoided and a more conservative approach (as in our case) can give equally good results [8]. Simple mattress sutures buttressed with Teflon felt can achieve haemostasis. The suture line in all cases should be along the nonischemic area and deep preferably trasmural stitches are required which can lead to further deterioration in ventricular function due to damage to the nonischemic myocardium [9].

In our case patient presented late in window period for complications of acute myocardial infarction, timely reassessment played a major role as we would have missed the deadly complication if not reassessed. Timely action by cardiologists and immediate availability of cardiothoracic surgeon and operation theatre was the key for survival of patient. Inspite of

these other reason responsible for miraculous survival of patient is not giving antithrombotics, as seen in OT the rupture was partially occluded by preformed clot, which would have not formed if antithrombotic were given in emergency room.

Conclusions

Post myocardial infarction ventricular rupture is a well known complication which has become less common in recent primary angioplasty era. Still once we have a ventricular rupture the mortality is unchanged. In this regard this case report gives some important information into successful management of LVR. Firstly, coordination between cardiologist and cardiothoracic surgeon and immediate availability of Operation Theater is a basic requirement. Secondly, a frequent reassessment and looking into the causes for prolonged hypotension whenever presentation is of acute MI. Thirdly, withholding antithrombotic till the confirmation of LVR will be a tricky call but feels like shifts the curve more towards patient survival in case of LVR primarily due to auto seal and less bleeding during OT.

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