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Case Report

Left ventricular free-wall rupture: an unusual and deadly complication after iodinated contrast medium exposure during primary percutaneous coronary intervention

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ABSTRACT

Acute left ventricular free-wall rupture (LVFWR) is a fatal mechanical complication that occurs in about 1-3% of acute myocardial infarction patients. Amongst, the iodinated contrast medium induced LVFWR is a very rare complication. Yet, it cannot be disregarded as the iodinated contrast medium is commonly used in contrast-enhanced imaging techniques. Here we have presented a rare case of iodinated contrast medium induced LVFWR after primary percutaneous coronary intervention. The patient's LV angiogram was suggestive of contrast extravasation from LV into the pericardial cavity. A small rent in the inferior LV wall was sutured, and the repaired area was covered with glutaraldehyde treated autologous pericardial patch. Post-operative complication of heavy bleeding and acute kidney injury was managed by blood components transfusion and hemodialysis. In conclusion, the contrast medium induced LVFWR has infrequent and unpreventable nature with acute hemodynamic alterations hence, should be treated aggressively to prevent life-threatening complication.

Keywords: Acute left ventricular free-wall rupture, Acute myocardial infarction, Percutaneous coronary intervention

INTRODUCTION

Acute left ventricular free-wall rupture (LVFWR) is invariably fatal mechanical complication that occurs in about 1-3% of acute myocardial infarction (AMI) patients.[1] The primary percutaneous coronary intervention (PCI) was found to be protective against LVFWR as it lowers the incidence rate.[2]

In completely occluded infarct-related artery in STEMI patients, the incidence of LVFWR is highest in the first 24 hours or may occur within the first week of MI, most probably due to the infarct extension in incompletely vascularized myocardium.[3,4] Unlike traditional risk factors induced LVFWR, the iodinated contrast medium induced LVFWR is a very rare complication.[5] Yet, it cannot be disregarded as the use of iodinated contrast medium is common in contrast-enhanced imaging techniques. Here we have presented a rare case of iodinated contrast medium induced LVFWR after primary PCI in rest angina patient.

CASE REPORT

A 58-year-old, smoker, alcoholic, non-diabetic, and normotensive male presented with rest angina for five days. Electrocardiogram showed deep Q waves with elevated ST-segment in inferior leads. Echocardiography was suggestive of hypokinesia of inferior wall and moderate left ventricular (LV) systolic dysfunction. The

patient was taken for cardiac catheterization. Left coronary artery angiogram showed type III left anterior descending (LAD) with proximal to mid aneurysmal dilatation, and proximal total occlusion in left circumflex (LCX). Right coronary artery (RCA) angiogram showed 90% stenosis with thrombus in the proximal portion (Figure 1). The patient was hemodynamically stable. The PCI of infarct-related RCA was done with a second-generation drug-eluting stent (Figure 2). The patient was shifted to the critical coronary care unit (CCCU) post-procedure, where, he developed transient hypertension with shivering which was managed by nitroglycerin infusion. After four hours of the procedure, the patient developed sudden hypotensive cardiac arrest with ST-elevation. He was revived after cardiopulmonary resuscitation (CPR) along with norepinephrine and epinephrine infusion. Then, he was rushed to the catheterization laboratory and the angiogram was done that showed a patent stent without any new lesion in the left coronary artery. The diagnostic catheter pushed in the LV cavity during the coronary angiography showed considerably elevated diastolic pressure. Subsequently, the echocardiography was performed, which confirmed the presence of large circumferential effusion with tamponade. Hence, prompt pericardiocentesis was done. The pigtail catheter was placed in the pericardial cavity to aspire the effusion and auto transfuse through the right femoral venous sheath. The patient became hemodynamically stable; however, the effusion didn't stop and kept accumulating. The LV angiogram showed contrast extravasation from LV into the pericardial cavity (Figure 3). Surgical consultation was taken and the patient was shifted immediately to the cardiothoracic vascular surgery-operation theater (CTVS-OT), where, crash mid-sternotomy was done. A small rent in the inferior LV wall noted, which was sutured with 3.0 running suture buttressed with Teflon on either side of the suture line. The repaired area was covered with glutaraldehyde treated autologous pericardial patch using continuous 6-0 polypropylene suture (Figure 4). Post-operative course was complicated by heavy bleeding from the drain and acute kidney injury, which was managed by blood components transfusion and hemodialysis.

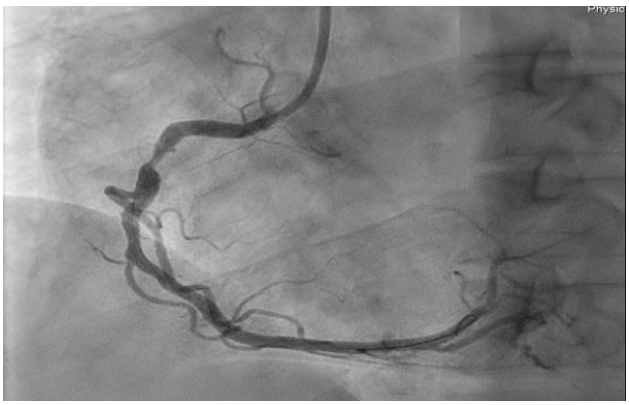

Figure 1: Thrombotic lesion in the proximal artery.

Figure 2: Drug eluting stent deployed in the proximal artery.

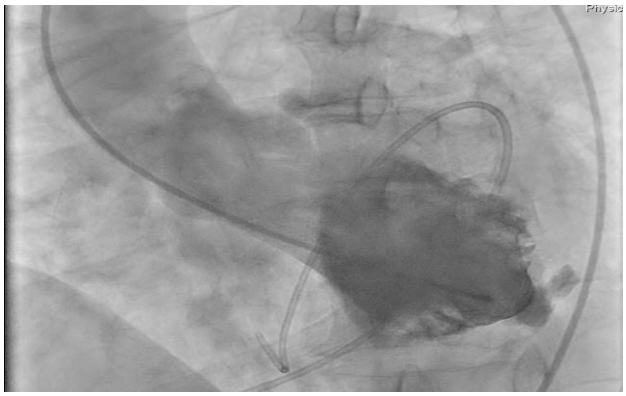

Figure 3: Left ventricular angiogram showing contrast extravasation.

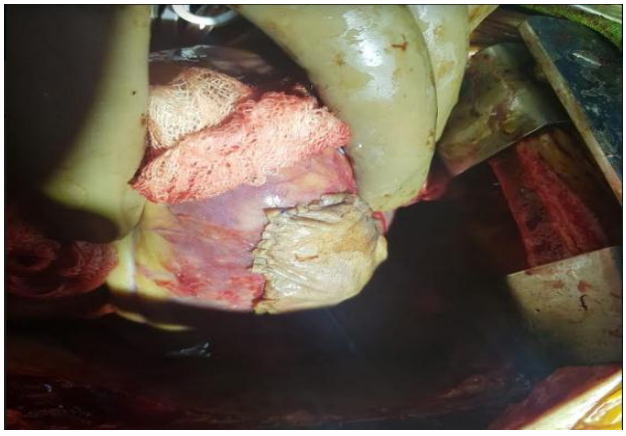

Figure 4: Pericardial patch over the left ventricular repair.

DISCUSSION

The acute LVFWR is manifested by the recurrent, continuous, sharp, and intense chest pain localized to the same area as an initial presentation but aggravated by deep inspiration. It is followed by hypotension, syncope and electromechanical dissociation after the development of hemopericardium and cardiac tamponade.[6]

A case discussed here, presented late after the AMI and underwent uneventful angioplasty of infarct-related artery. After the PCI procedure, the patient developed moderate to a severe adverse reaction to contrast agent characterized by shivering and transient hypertension followed by a blowout rupture of LV free-wall. Any hemodynamic deterioration following the coronary intervention, particularly in acute coronary set up is usually thought to be due to procedural complications like guidewire induced distal coronary perforation, ostial dissection or acute stent thrombosis.[7,8] Such complication in the patient was ruled out by the check angiogram. Also, the diagnostic catheter placed into the LV showed quite high diastolic pressure which was confirmed by the diagnosis of cardiac tamponade by echocardiography. The extravasation of dye into pericardial cavity establishing LV free-wall rupture as the cause of tamponade was confirmed by LV angiogram. Adverse reaction to iodinated contrast medium shows both anaphylactic and non-anaphylactic reactions of varying severity. However, such reactions after the therapeutic or diagnostic use of the contrast medium are rarely witnessed due to the precautions like hypersensitivity testing and routine use of premedication in the catheterization laboratory. A more frequent and less serious adverse reaction of iodinated contrast medium is characterized by shivering and transient hypertension despite premedication. Such events are routinely treated by warming up the patient and nitroglycerin infusion to control hypertension.[9] However, the acute surge of blood pressure after exposure of contrast medium leads to a sudden increase in left ventricular afterload and wall tension, which, in rare cases may not be tolerated by the infarcted ventricular wall.[5] In our case, the incidence of LVFWR occurred partly due to the late presentation of the patient after AMI, and partly due to sudden increase in afterload induced by hypertensive crisis.

Fortunately, the incidences of acute reactions to the intravascular contrast media are rare. The patients with prior reactions to contrast media and history of asthma are at high risk for developing reactions. Additionally, low-osmolar iodinated agents have low adverse reactions and are preferred over high-osmolar agents.[9] As in the treatment of aortic dissection antihypertensive agent that favorably alters LV dp/dt could be better option in patient with acute MI to control hypertensive crisis

To conclude, considering the infrequent and unpreventable nature of the contrast medium induced LVFWR, the acute hemodynamic alterations after the primary PCI should be treated aggressively to prevent life-threatening complication.

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