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Case Report

Catastrophic Event Following Percutaneus Coronary Intervention Developing In-Stent Thrombosis Leading Massive Pericardial Effusion and Free Wall Rupture

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Abstract

Background : One extremely unusual but serious side effect of an acute myocardial infarction is left ventricular free wall rupture. It was reported to happen either during the sub-acute phase with overt cardiac remodeling (type III, 45%) or early after the beginning of Myocardial Infarction (MI) (type I or II, about 55%). Large infarct sizes, female gender, and advanced age have all been linked to an increased risk of free wall rupture. Clinicians continue to face significant challenges in diagnosing and treating this condition because of the diverse clinical manifestations linked to elevated death rates. This case report aims to highlight a rare occurrence of mechanical complication of acute myocardial infarction
Case : A 69-year-old male patient was referred because of chest pain and dyspneu. He had a primary Percutaneous Coronary Intervention (PCI) and was diagnosed with posterior ST-Evelation Myocardial Infarction (STEMI). The patient had a stent inserted into his ostial-distal Left Circumflex (LCx) artery. Three weeks later, a reangiography revealed a left ventricle (LV) aneurysm and stent thrombosis. Massive pericardial effusion with free wall rupture was seen on the echo. He was breathing heavily while in our emergency room. His blood pressure was 125/74 (94) heart rate was 94 bpm respiratory rate 24 times/minute, SpO2 was 98%, there were no rales, and his ankles had pitting edema. By the bedside, Echo revealed an LV aneurysm, a large, localized pericardial effusion without tamponade, and a possible free wall rupture. Later, he was taken to the intensive care unit and had heart surgery
Discussion : Complications from an acute myocardial infarction may be ischemic, mechanical, arrhythmic, embolic, or inflammatory. Significant short-term clinical improvement and long-term survival are linked to the emergence of mechanical problems following acute myocardial infarction.
Conclusion : the fact that primary Percutaneous Coronary Intervention (PCI) has significantly reduced the prevalence of this deadly event. Our results indicate that one of the key predictors and primary causes of this problem is a longer symptom of angiography time.

Keywords : Percutaneus Coronary Intervention, In-Stent Thrombosis, Massive Pericardial

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INTRODUCTION

An uncommon but serious side effect of acute myocardial infarction (AMI) is left ventricular free wall rupture (LVFWR). It was observed to happen either during the sub-acute phase with overt cardiac remodeling (type III, 45%) or early after the beginning of Myocardial Infarction (type I or II, about 55%). Large infarct sizes, female gender, and advanced age have all been linked to an increased risk of free wall rupture. Especially, the diverse clinical manifestations linked to elevated mortality rates continue to provide a significant diagnostic and treatment obstacle for medical professionals.[1]

The aim of this study is to highlight a rare occurrence of a mechanical complication of acute myocardial infarction, specifically focusing on left ventricular free-wall rupture. Provide insights into the clinical manifestations, diagnostic challenges, and treatment strategies associated with this serious complication. Additionally, the study discusses the factors associated with an increased risk of left ventricular free wall rupture, such as large infarct sizes, gender, advanced age, and interval time between symptom onset and angiography. The overall goal is to contribute to the understanding and management of this life-threatening condition, ultimately improving patient outcomes and reducing mortality rates associated with acute myocardial infarction complications.[2,3]

CASE REPORT

A 69-year-old man with dyspnea and chest symptoms was referred to our department. He had diaphoresis and substernal severe chest pain a month ago. He was treated at the closest hospital, where a stent was implanted in the ostial-distal Left Circumflex (LCx) artery, primary PCI was performed and posterior STEMI was discovered. Three weeks later, he experienced dyspnea and chest pain. Physical examination revealed pansystolic murmur grade III/VI at apex then re-angiography revealed LV aneurysm and stent thrombosis in the LCx artery. Massive pericardial effusion with free wall rupture was seen on the echo. He was later sent to our hospital for additional care. He was breathing heavily while in our emergency room. His blood pressure was 125/74 (94) Heart rate was 94 bpm, Respiratory rate 24 times/minute, SpO2 was 98%, there were no rales, and his ankles had pitting edema. Bedside Echo, revealed a Left Ventricle (LV) aneurysm and a large, localized pericardial effusion without tamponade, LV aneurysm with possible rupture of the free wall. Following his admission to the intensive care unit, he had heart surgery. The patient's vital signs include a weak overall appearance, blood pressure of 125/74 (94) mmHg, heart rate of 95 bpm, Respiratory rate of 24 x/minute, SpO2 100% with 3 lpm nasal, and conjunctiva anemia observed on physical examination. There were grade 4/6 pansystolic murmur in the apex of the heart, and rales one-third of the lung's bottom. Medical findings from the Angiography indicate coronary artery disease (CAD) with one-vessel involvement (CAD1VD), a total in-stent thrombosis at the proximal LCx, and failed wiring in the LCx. An LV graph also reveals an LV aneurysm, but no contrast extravasation from the LV.

From echocardiography, the LVH (Left Ventricle Hypertrophy) is concentric, with a visualization of an LV aneurysm in the posterior LV. Pericardial effusion has a size of 50 mm with a visualization of fibrin. Free wall rupture has a size of 30 mm. The systolic function of the LV has decreased, with an Ejection Fraction (EF) of 37% by Sympson and 32% by Teichz. Diastolic function of the LV is mildly impaired, with a grade I dysfunction (E/A 0.7). Systolic function of the Right Ventricle (RV) has also decreased, with a Tricuspid Annular Plane Systolic Excursion (TAPSE) of 7 mm. There is mild mitral regurgitation (MR), and there is an 18% variation in mitral

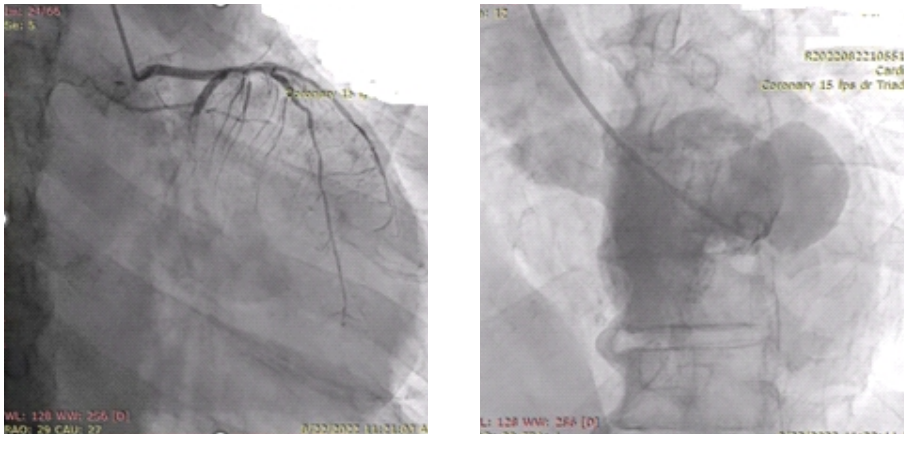

Figure 1. Angiography showed a total instent thrombosis at the proximal LCx, and failed wiring in the LCx. An LV graph also reveals an LV aneurysm, but no contrast extravasation from the LV.

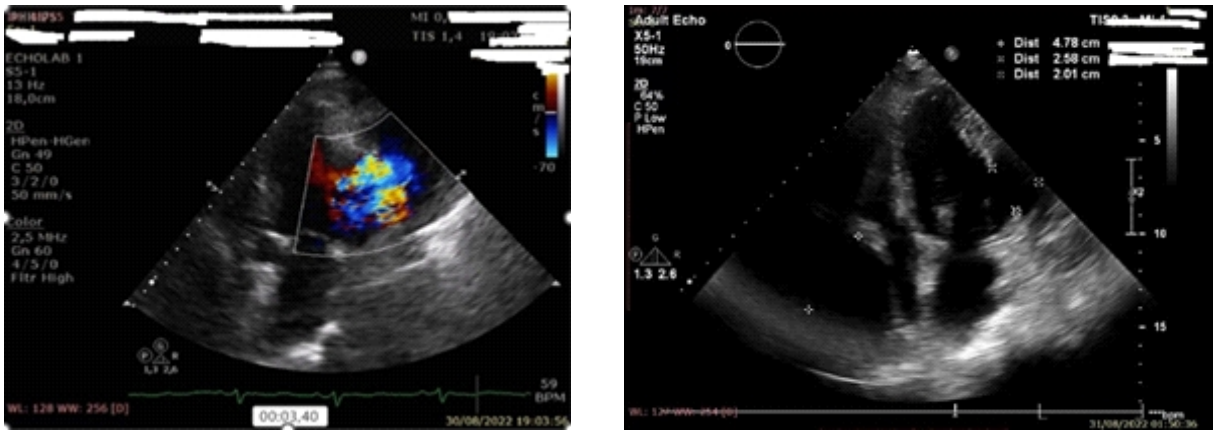

Figure 2. Echocardiography of Pseudoaneurysm (left) and Massive Pericardial Effusion (right)

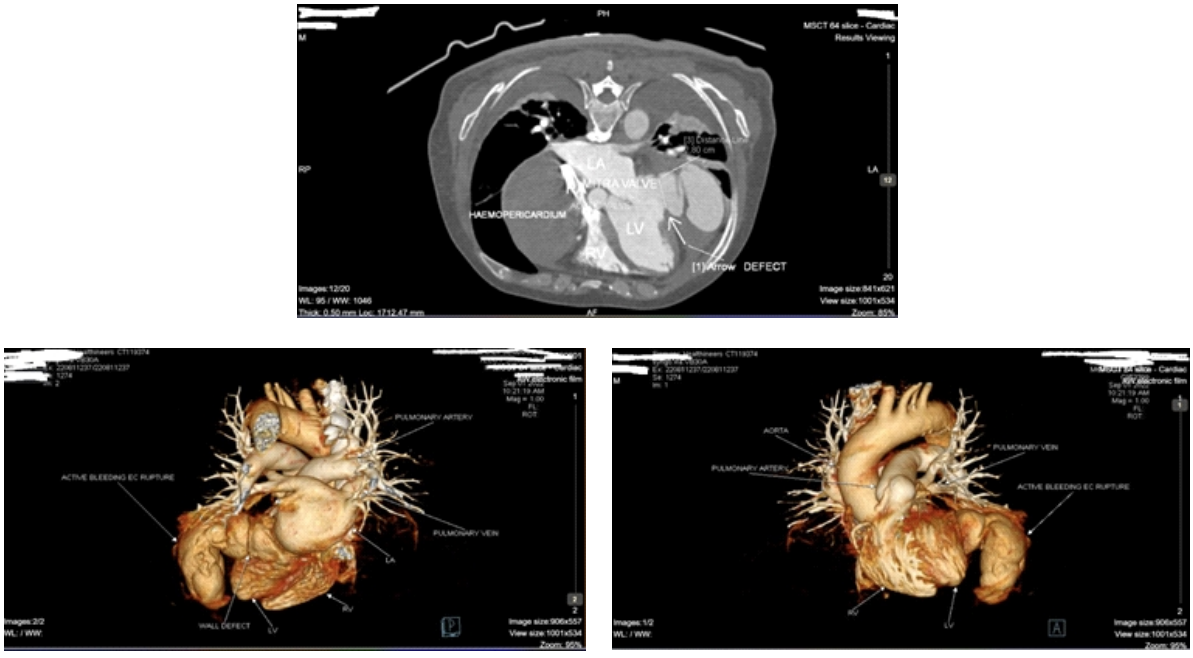

Figure 3. MSCT Cardiac of LV Pseudoaneurysm and Massive Pericardial Effusion

inflow. From Multi slice Computerized Tomography (MSCT) Cardiac the patient has an extravasation of contrast through a defect on the lateral ventricle of the left heart, with a diameter of approximately 2.8 cm and a distance of approximately 1.7 cm from the mitral valve. This has resulted in hemopericardium, which is accompanied by active bleeding, suggesting a possible free wall rupture of the left ventricle. Right atrium appears to have decreased in size, with a long axis of approximately 4.92 cm and a short axis of approximately 1.66 cm.

During the surgery, a clot was found behind the right atrium, and a massive pericardial effusion was present in the pseudoaneurysm of the left ventricle, amounting to approximately 150 ml. A closure was performed on the free wall rupture using gortex, and a delayed sternal closure was carried out. Then, he performed sternal closure 3 days after surgery. During monitoring in cardiac intensive care, he was well recovered. Evaluation by transthoracic echocardiography and chest X-ray there was no expansion of pericardial effusion, no shunt of LV free wall, and improved LV function. The substernal drain itself showed improving significantly from 300 to 5 ml, on the nine days. later he showed improving in all parameters. The patient was stepdown to the cardiac ward.

DISCUSSION

Left ventricular free wall rupture and LV aneurysm formation are serious, yet rare, structural complications

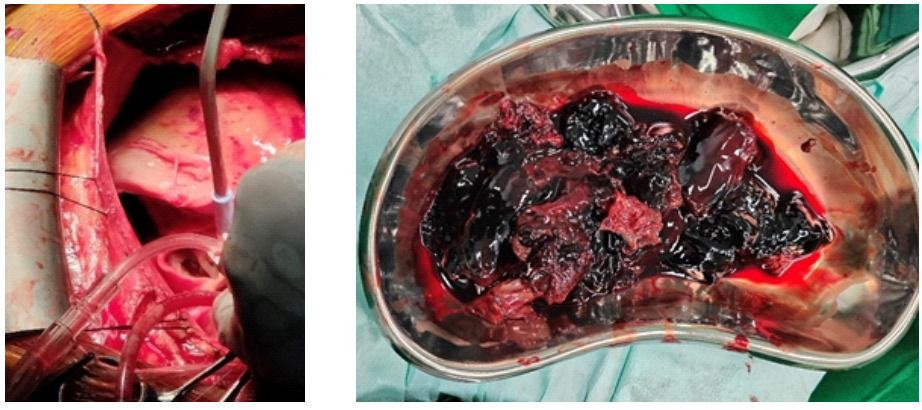

Figure 4. Result of Repair LV Free Wall Rupture + Pericardial Effusion Evacuation

post-myocardial infarction. Patients was referred from a previous hospital with an LV aneurysm found during coronary artery angiography (CAG). Physical examination and echocardiography were performed. Patient was diagnosed with LV free wall rupture differential diagnosis with LV pseudoaneurysm and aneurysm, massive pericardial effusion without cardiac tamponade signs, and previous angiography indicated total in-stent thrombosis in LCx. He was completely in a state of failure due to LV free wall rupture and massive pericardial effusion. A continued dose of furosemide and optimization of dual antiplatelet (aspirin and clopidogrel were chosen), ace-inhibitor, and mineralocorticoid receptor antagonist were administered. In this case, the process of left ventricular pseudoaneurysm caused by rupture of the left ventricular wall after myocardial infarction was clear. Then, patient was planned for urgent repair of LV free wall rupture and evacuation of pericardium effusion, and Coronary Artery Bypass Graft (CABG) as needed to improve prognosis as most of patients with pseudoaneurysms treated conservatively have poor prognosis in one series and the mortality rate at the first 2 year is almost 50%. This case report showed us the complex complications after in-stent thrombosis after acute myocardial infarction that should we avoid.[3–5]

Pseudoaneurysms in the left ventricle are typically located on its lateral or diaphragmatic surface. LV pseudoaneurysms are prone to rupture and have a tendency to expand quickly. Once the diagnosis is established, prompt surgery is required. Further testing, like as Magnetic Resonance Imaging (MRI) or MSCT cardiac, may be necessary to confirm the diagnosis of LV pseudoaneurysms, as supplementary exams like echocardiography may not be sufficient.[8]

There are two main categories into which Free Wall Rupture falls, Oozing type: this kind of bleeding is marked by sluggish, unclear bleeding that builds up in the pericardial sac.[9] Blow-out type: characterized by a large hemorrhage and a macroscopic defect that causes abrupt tamponade.[9]

The patient's initial symptoms might range from minor chest discomfort to severe chest pain or even abrupt death, depending on the patient's clinical status. The coronary arteries that are most frequently involved are LAD and LCX. Heart failure owing to AMI is a particular symptom of free wall rupture, even though the clinical signs of the rupture are less specific for cardiac tamponade than for the rupture itself. Timely referral for surgery is essential. When tamponade and hemodynamic instability are proven, pericardiocentesis can save lives, However, blood clots could be discovered in the pericardial sac.[9]

The principles of surgical treatment of LVFWR are to relieve tamponade, close the tear and/or stop the bleeding, anchor the repair on healthy tissue, and minimize distortion of heart geometry while preventing the recurrence of rupture or pseudoaneurysm formation. The chosen method for surgical repair is usually dictated by the type of rupture, its surrounding tissues, and the presence of concomitant lesions. Some authors believe that coronary angiography should be promptly performed as soon as pericardial effusion is noted in AMI patients before they deteriorate. The knowledge of coronary status is of great help in deciding where and how to place the sutures during surgery; in addition, proper revascularization of the diseased vessels supplying the non-infarcted area at the time of LVFWR repair (concomitant coronary artery bypass grafting) has been shown to exert a positive impact on survival and freedom from angina.[6,7]

One serious risk related to stent implantation is stent thrombosis. Myocardial infarction, cardiogenic shock, hematoma, pseudoaneurysm, retroperitoneal hemorrhage, arrhythmia, thrombus development, and stroke are among the outcomes that might occur from undetected or untreated problems. Following AMI, the patient's clinical and hemodynamic status remained stable, while mechanical problems such as LV pseudoaneurysm and free wall rupture were noted. Subacute stent thrombosis, which happened between

24 hours and a month following stent implantation, was identified as the root cause of these problems. Hemopericardium behind the Right Atrium caused a huge pericardial effusion with imminent tamponade, which avoided tamponade symptoms and did not require inotropic support. Following comprehensive evaluations to determine the best surgical strategy, the pericardial effusion was surgically evacuated during the intervention and healing of the free wall rupture following the patient's cessation of antiplatelet medication.[10–12]

CONCLUSION

AMI may result in problems related to embolism, arrhythmia, mechanical, ischemia, or inflammation. Significant clinical improvement after the emergence of surgical mechanical problems following acute myocardial infarction. As seen in our case, free wall rupture can develop into a Pseudoaneurysm, if it happened then precise care about free wall rupture can be saved.

CONFLICT OF INTEREST

The author has no conflicts of interest that could affect the results or interpretation in this report.

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