Rupture of Left Ventricular Pseudoaneurysm: A Tragedy that Must Be Avoided

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Introduction

Even patients with favorable evolution in the first days after acute myocardial infarction (AMI) may evolve with severe mechanical complications, which may be sudden and lethal, such as a rupture of the left ventricular (LV) free wall.¹

Another severe mechanical complication of AMI patients is LV pseudoaneurysm, which develops due to rupture of the LV musculature contained by pericardial adhesions.¹,²

When not diagnosed and treated early, pseudoaneurysm may also have a catastrophic evolution.¹ This case report aims to stress the importance of recognizing the main risk factors associated with mechanical complications after AMI, emphasize the importance of careful analysis of echocardiograms and angiographic ventriculography scans in search of signs of mechanical complications, and to emphasize the need for early diagnosis and immediate surgery in patients with pseudoaneurysm detected.

We report the case of a patient who had LV pseudoaneurysm as a complication of lower AMI, whose diagnosis was not delivered while in hospital, despite suggestive clinical and angiographic imaging, which prevented the surgical correction of LV rupture in a timely manner.

Case Report

A 65-year-old male presented non-thrombolytic inferolateral wall AMI with good initial clinical evolution. Coronary angiography was performed on the fourth day of hospitalization, which evidenced triarterial obstructive pattern with major proximal stenosis in the anterior descending and circumflex arteries, and total occlusion of the right coronary artery (not receiving the collateral arteries). The ventriculography revealed large inferior wall akinesia and “image suggesting LV inferior wall aneurysm” (Figure 1).

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The patient maintained favorable clinical course and was discharged on the sixth day after the initial event. He was instructed to seek a cardiac surgeon to schedule coronary artery bypass grafting surgery (and possibly LV aneurysmectomy) in an outpatient setting. On hospital discharge, he received aspirin (100 mg/day), clopidogrel (75 mg/day), metoprolol (50 mg/day), ramipril (10 mg/day) and atorvastatin (80 mg/day).

The patient saw a cardiovascular surgeon in the outpatient setting, who requested transthoracic echocardiogram, performed on the tenth day after AMI, which revealed LV inferior wall pseudoaneurysm (Figure 2). The echocardiographer immediately contacted the surgeon who requested the examination, which, in turn, recommended immediate hospital readmission to schedule early surgery. However, on leaving the echocardiography room, the patient had a non-reversed cardiorespiratory arrest after cardiopulmonary resuscitation (CPR), as well as pericardiocentesis by Marfan puncture.

During the CPR, a quick transthoracic echocardiogram was performed, which confirmed an image suggesting severe pericardial effusion, not identified in the examination performed minutes before (Figure 3).

Discussion

The mechanical complications post-AMI are still a challenge in clinical practice. They include LV free wall rupture, rupture of septal wall or papillary muscles, and the formation of pseudoaneurysms and true aneurysms.¹ The ruptures occur more frequently between the fifth and seventh day after the coronary event, when the friability of necrotic tissue infarcted and infiltrated by inflammatory cells makes the ventricular wall significantly weak. However, they can occur between 1 and 30 days after the acute event.¹

The main risk factors for the occurrence of mechanical complications include: failure to perform (or delayed) reperfusion therapy, fibrinolytic therapy (compared to the gold standard of percutaneous mechanical recanalization), concomitant use of anticoagulants and multiple antiplatelet agents, chronic use of nonsteroidal anti-inflammatory drugs (NSAID) or corticosteroids and old age.

Patient’s evolution with mechanical complications after AMI varies according to the type of rupture. Some cases present acute and dramatic evolution, generating sudden death in a few minutes (as in the LV free wall rupture). Those with interventricular septal rupture, papillary muscle rupture, or with pseudoaneurysm, may present from abrupt hypotension, precordial pain and dyspnea to less typical symptoms, such as malaise, nausea and vomiting.¹ Thus, considering the clinical evolution of the patient and the degree of reperfusion obtained with treatment, hospital discharge should be delayed, allowing...
Figure 1 – Ventriculography showing bulging left ventricular inferior wall.

Figure 2 – Echocardiogram showing left ventricular (LV) pseudoaneurysm. AO: aorta; LA: left atrial.
Case Report

Differentiating true aneurysm from pseudoaneurysm is still a major challenge. The basic difference between them is that in true aneurysm, the ventricular wall is intact and there is bulging of all portions of the unbroken myocardium and the adjacent scar tissue (myocardial fibrosis). The pseudoaneurysm is formed by a rupture of the ventricular wall in the area of transmural infarction, which is contained by pericardial adhesions, leading to the formation of a localized hemopericardium. Therefore, the wall of the false aneurysm is fragile, as it is only formed by layers of fibrous tissue collagen of the pericardium, associated or not with thrombi.

While true aneurysm can hardly break, pseudoaneurysm is prone to expansion and rupture, so early corrective surgery should be recommended for all patients. Surgery can also be recommended for true aneurysms, but this depends on the patient’s unfavorable clinical outcome (ventricular arrhythmia, heart failure, and intracardiac thrombi embolization). However, when surgery is recommended, it should not be performed within the first 30 days. In general, before performing the surgery, the true aneurysm extent and colla should be defined, ventricular wall friability is to be improved and a potential response to optimized clinical treatment should be awaited.

The occurrence of pseudoaneurysm is rarer and most commonly affects the lower wall, whereas true aneurysms are more common and often located on the anterolateral-apical wall. A pseudoaneurysm is usually first suspected on transthoracic echocardiography. This is because the method is accessible and affordable. Thus, Doppler echocardiogram has good sensitivity, specificity and low cost, and can offer an early diagnosis of the problem. On echocardiogram, the LV contained cavity is characterized by a narrow “neck,” which freely communicates with the left ventricle. Nevertheless, a definitive echocardiogram diagnosis is performed in only 26% of the patients. The method most commonly used to confirm the diagnosis of a pseudoaneurysm is angiographic ventriculography (in which definitive diagnosis can be offered in more than 85% of the patients).

When doubt persists regarding differential diagnosis, transesophageal echocardiography, computed tomography and cardiac magnetic resonance are excellent alternatives to classical methods to distinguish a pseudoaneurysm from a true aneurysm, although they are still hardly accessible at many services.

Rapid and accurate diagnosis of LV pseudoaneurysm is mandatory. Due to the high risk of rupture, recommendation of early surgery is still the main therapeutic option, with perioperative mortality of less than 10%. Strict control of anxiety, blood pressure and heart rate until surgery are also key therapeutic measures.
Conclusion

Left ventricular aneurysms and pseudoaneurysms are mechanical complications associated with acute myocardial infarction, and differential diagnosis between the two is still a challenge in clinical practice. Unlike true aneurysms, pseudoaneurysms have a high tendency for rupture and should be operated soon after diagnosis. Transthoracic echocardiography is the most widely used method to suggest diagnosis, which can be confirmed by other methods (such as angiographic ventriculography). This case report stresses the importance of care in the analysis of risk factors for the occurrence of mechanical complications after acute myocardial infarction (especially left ventricular pseudoaneurysm), as well as the careful analysis of the scans that allow early diagnosis and treatment of this potentially fatal clinical situation.

Authors’ contributions

Research creation and design: Faria LAO, Santos PC, O’Connell JL; data acquisition: Faria LAO, Vitor EHC, Cecílio J; manuscript writing: Faria LAO, Vitor EHC, Santos PC, Kalill R, O’Connell JL; critical revision of the manuscript for important intellectual content: Faria LAO, Vitor EHC, Santos PC, Cecílio J, Kalill R, O’Connell JL.

Potential Conflicts of Interest

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References