Rare Presentation of Pseudoaneurysm Obstructing the Left Ventricular Inflow Tract in a Patient With Acute Myocardial Infarction

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Introduction

Left ventricular (LV) pseudoaneurysm is nowadays a rare but still potentially fatal complication of myocardial infarction (MI). Recent studies have estimated that following ST-elevation MI, 0.27 to 0.91% of patients develop mechanical complications.1 LV free wall rupture (VFWR) is estimated to occur in 0.01 to 0.52% of patients, showing a clear decrease in incidence associated with the widespread adoption of reperfusion therapy.2 Nonetheless, there has been no significant decrease in associated mortality rates and this complication remains an important determinant of outcomes after MI.¹

A pseudoaneurysm is formed when a cardiac rupture is contained by adherent pericardium or scar tissue, with no myocardial tissue.³ Transmural MI is the most common cause, usually following occlusion of the right coronary artery or the circumflex artery, therefore more often occurring on the posterior or lateral wall.⁴

The clinical presentation is vast and patients with pseudoaneurysms can present with chest pain, dyspnea, cardiac tamponade, or the condition may be even incidentally detected. The risk of sudden death due to pseudoaneurysm rupture is 30-45%, and a significant proportion of patients are probably not diagnosed in the acute phase due to early and fatal rupture.⁵

Transthoracic echocardiogram (TTE) should be the first test to be performed, however, establishing the definitive diagnosis frequently poses a substantial challenge. Further assessment with cardiac computed tomography (CT) or magnetic resonance imaging (MRI) may be helpful in patients without hemodynamic instability.⁶

We describe a very rare presentation of LV pseudoaneurysm after MI producing severe extrinsic compression with obliteration of the LV inflow tract diagnosed with TTE in an acute presentation.

Case Report

A 60-year-old male with a history of smoking and no other comorbidities was admitted with typical chest pain lasting 20 hours. The initial examination at emergency department showed a regular heart rate of 80 beats per minute, a systolic blood pressure of 110 mmHg, wheezing on lung auscultation in addition to rales in both lungs bases, with 85% of peripheral saturation of oxygen (class II of Killip classification), and no other remarkable findings at physical examination.

The electrocardiogram showed ST-elevation in I, aVL, V5 and V6 leads with a small Q wave (Figure 1), suggestive of a lateral MI. He received dual antiplatelet therapy and was immediately transferred to the cardiac catheterization laboratory. The coronary angiography displayed occlusion of the mid-circumflex coronary artery, a severe proximal stenosis in the anterior descending artery and no significant lesions in the right coronary artery.

During the exam, he presented acute deterioration of hemodynamic state requiring the use of vasoactive drugs. A TTE was requested urgently to evaluate mechanical complications and decide whether to perform primary angioplasty. The TTE was performed with the patient in supine position and at regular and tachycardic heart rhythm. Pericardial effusion was immediately ruled out on subcostal view (Figure 2, panel A). At the apical four-chamber-view, a mild segmental systolic dysfunction affecting the lateral wall of the left ventricle was observed. At this view, the most impressive finding was that the mitral valve was not visualized and where seems to be its topography there was a muscular band-like image (Figure 2, panel B). It was possible to observe a continuity solution between the lateral wall and that muscular image, with bidirectional flow at color Doppler acquisition (Figure 2, panel C/D). This image can suggest rupture of LV free wall, with a pericardial hematoma potentially exerting mass effect through the left atrium (asterisk, Figure 2, panel B). Finally, at the apical three-chamber-view this inference became more evident by the visualization of a posterior pericardial hematoma compressing the left atrium (figure 2, panel E/F). This observation substantiates the main hypothesis of a LV pseudoaneurysm. At this moment, a left ventriculography was performed (Figure 3) confirming a rupture of lateral LV wall and showing an image suggestive of lateral pseudoaneurysm.

We theorize that as the pseudoaneurysm progressed, it obstructed the LV inflow, as seen by the limited mitral valve diastolic flow at color Doppler (Figure 2, panel F). The patient suffered a pulseless arrest a few minutes later. Multiple resuscitation attempts failed.

Discussion

VFWR is an important cause of death from acute MI and the short-term mortality remains very high even with rapid diagnosis and timely surgery.⁷ The clinical manifestation is contingent upon the rate at which the tear unfolds. An abrupt
Figure 1 – ECG at presentation showing ST elevation and small Q waves in lateral leads.

Figure 2 – Transthoracic echocardiogram showing (A) subcostal-view with no evident pericardial effusion, (B) apical 4-chamber-view, (C and D) apical 4-chamber-view with a bidirectional flow at color Doppler; (E) apical 3-chamber-view; (F) mitral valve color Doppler at apical 3-chamber-view. LV = left ventricle; RV = right ventricle; PH = pericardial hematoma; * = left atrium compressed by the pericardial hematoma.
Pseudoaneurysm obstructing left ventricular inflow

Case Report

tear would likely lead to sudden cardiac tamponade whereas a smaller and more gradual tear may be limited by thrombus formation or a compliant pericardium, eventually developing a pseudoaneurysm. In a comprehensive analysis of 290 patients by a systematic literature review, symptoms among patients with pseudoaneurysm included congestive heart failure, chest pain and dyspnea, with sudden death identified as the initial symptom in merely 3% of patients. In a separate case series involving 52 patients, acute presentation occurred in only 8% of cases. As highlighted in a recent review, LVFWR typically manifests within seven days post-MI, with an average time to diagnosis of 2.6 days.

In this case, the patient presented acutely and probably had already developed a tear tamponaded by a clot. Possibly the use of heparin and antiplatelet therapy may have contributed to the expansion of the pseudoaneurysm. The peculiarity of our case is the location of pseudoaneurysm producing severe extrinsic compression of the mitral valve complex and obstructing the LV inflow tract, with a very restrictive flow registered by the color Doppler through the mitral valve, leading to a progressive obstructive shock. In the literature review, there was no previous report of this exotic condition.

The diagnosis of a LV pseudoaneurysm can be made by several imaging modalities, including the TTE, cardiac ventriculography, transesophageal echocardiography, cardiac MRI and the cardiac CT scan, but none is 100% accurate. Ventriculography demonstrates notable diagnostic precision and, in this case, was useful to corroborate the diagnosis given the constraints in accessing alternative imaging modalities due to patient hemodynamic instability.

The findings that suggest pseudoaneurysm at the TTE are: (1) narrow neck, (2) lack of the normal structural elements found in an intact cardiac wall, (3) bidirectional flow with a to-and-fro pattern, consisting of systolic flow entering the pseudoaneurysm and diastolic flow exiting back into the left ventricle, and (4) the presence of turbulent flow by pulsed Doppler at the neck of a cavity or within the cavity itself.

Although the TTE is a readily available non-invasive method and should be the first imaging modality to use, it is not so effective in making a definitive diagnosis. Several factors contribute to this: (1) poor transthoracic acoustic window, (2) unstable patients, (3) posterior location of the pseudoaneurysm cavity in most cases, (4) rare condition, with low suspicion and expertise for this particular images.

Figure 3 – Left ventriculogram showing presence of left ventricular pseudoaneurysm
The present case is unique in a sense that it provided diagnostic images by TTE of a rare condition with an acute presentation. This case emphasizes the need for a high clinical index of suspicion and the prompt recognition of these non-usual images for the successful diagnosis and treatment of this potentially lethal condition.

Author Contributions
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