


Exertional dyspnea after myocardial infarction: thinking beyond the diagnosis of heart failure

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Abstract

Introduction: We herein present an unusual case of a pseudoaneurysm of the left ventricular myocardium, which is a rare and fatal complication of myocardial infarction.

Case report: A 64-year-old man with a history of bipolar disorder and arterial hypertension was hospitalized for delayed presentation ST-elevation myocardial infarction. He was admitted to our hospital 24 hours after symptom onset. Diagnostic coronary angiography revealed 95% stenosis at the distal third of the right coronary artery, and he underwent a primary percutaneous coronary intervention to the culprit lesion. Despite administration of a diuretic and optimization of other pharmaceutical treatment, his heart failure deteriorated. Electrocardiography showed a sinus rhythm with Q-wave formation in the inferior wall leads (II, III, aVF), T-wave inversion in the same leads, and borderline QT prolongation (QTc of 490 ms). No ST elevation suggestive of left ventricular aneurysm formation was noticed. Forty days later, cardiac ultrasound revealed a dyskinetic cavity (pseudoaneurysm) in continuity with the posterior–inferior wall of the myocardium, resulting in severe mitral valve regurgitation. Unfortunately, the patient died while awaiting surgical treatment.

Conclusion: Although most patients with left ventricular pseudoaneurysm have a relatively benign outcome, those with symptoms of heart failure must be urgently diagnosed and treated.

Keywords

Pseudoaneurysm, myocardial infarction, heart failure, left ventricle, dyskinesia, percutaneous coronary intervention

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Introduction

Left ventricular pseudoaneurysm is a rare and fatal complication of myocardial infarction.¹ After an acute myocardial infarction, patients frequently develop symptoms and signs of heart failure and are treated in accordance with guidelines for heart failure; however, emergency surgery should be performed in patients with serious mechanical complications.² We herein present a rare case involving a 64-year-old man with a ruptured myocardial wall and a nonruptured left ventricular pseudoaneurysm who presented with exertional dyspnea and worsening shortness of breath 40 days after having experienced an inferior wall transmural myocardial infarction. This case is being reported to inform clinicians that patients with symptoms of heart failure that is not improved with medical treatment require urgent diagnosis and treatment for other potential underlying abnormalities.

Case report

The case report was approved by the Ethics Committee of the General Hospital of Veroia (decision No. 11, 10/12/2017). The patient's family provided verbal informed consent. A 64-year-old man with a medical history of bipolar disorder and arterial hypertension was hospitalized for a delayed presentation ST-elevation myocardial infarction. He was admitted to our hospital 24 hours after symptom onset. Diagnostic coronary angiography revealed 95% stenosis at the distal third of the right coronary artery, and he underwent a primary percutaneous coronary intervention to the culprit lesion. Loading doses of aspirin and ticagrelor were given. During his hospitalization, he reported palpitations and shortness of breath. Clinical examination revealed normal heart sounds and a grade III systolic murmur at the cardiac apex.

The patient's blood pressure was 100/65 mmHg, pulse rate was 95 bpm, and respiratory rate was 14 breaths/minute. He had functional class II heart failure according to the New York Heart Association. Electrocardiography showed a sinus rhythm with Q-wave formation in the inferior wall leads (II, III, aVF), T-wave inversion in the same leads, and borderline QT prolongation (QTc of 490 ms). No ST elevation suggestive of left ventricular aneurysm formation was noticed. The P2Y12 inhibitor was switched from ticagrelor to prasugrel, and 24-hour ambulatory electrocardiography revealed frequent premature atrial and ventricular complexes. Transthoracic echocardiography showed a hypokinetic posteroinferior wall with a mildly reduced ejection fraction of 45% (Simpson's method), moderate mitral regurgitation (vena contracta, 0.6 cm; proximal isovelocity surface area, 0.7 cm; effective regurgitant orifice area, 34 mm²), and mild pericardial effusion.

Furosemide was added to lisinopril and metoprolol, but only mild to moderate improvement of the patient's symptoms was noticed before he deteriorated. Furthermore, up-titration of these medications was difficult because of the borderline arterial pressure. The addition of ivabradine and the switch from ticagrelor to prasugrel did not change his clinical status. Ticagrelor may cause transient dyspnea in some patients, which reverses with discontinuation.³

Forty days after the myocardial infarction, the patient was readmitted because of exertional dyspnea and worsening shortness of breath. New transthoracic echocardiography showed a dyskinctic cavity that was connected to the posteroinferior wall and communicated with the left ventricle, causing damage to the posterior mitral valve leaflet (P1 scallop) and resulting in severe mitral regurgitation. A color Doppler study confirmed passage of blood from the left ventricle into the cavity through

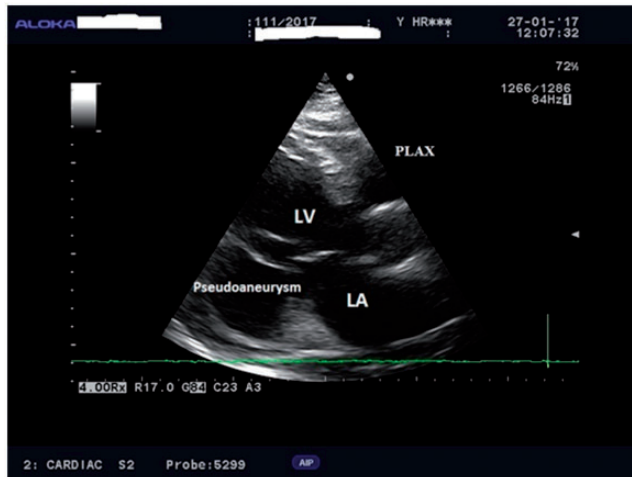


Figure 1. Parasternal long-axis (PLAX) view showing a pseudoaneurysm as a dyskinetic cavity located in the posteroinferior left ventricular wall. Note the presence of mild pericardial effusion. LV: left ventricle, LA: left atrium.

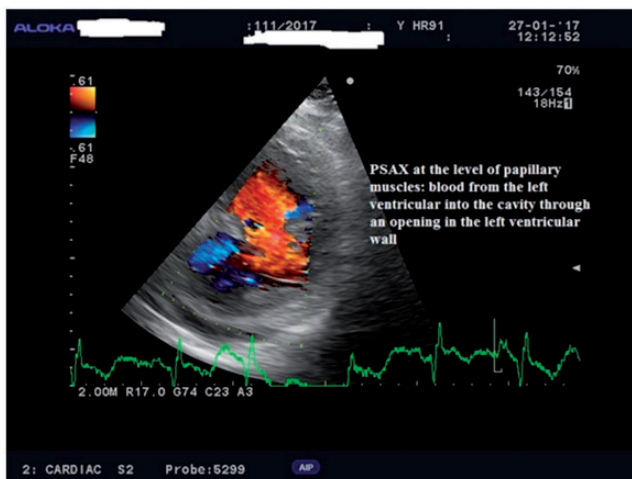


Figure 2. Parasternal short-axis (PSAX) view at the level of the papillary muscles. A color Doppler study confirmed the passage of blood from the left ventricle into the cavity through an opening in the left ventricular wall.

an opening in the left ventricular wall (Figures 1–4). No thrombus formation was detected. The diagnosis of left ventricular pseudoaneurysm was made.

Unfortunately, the patient died while waiting to transfer for surgical treatment.

Discussion

Left ventricular pseudoaneurysms develop after left ventricular free wall rupture.¹ Left ventricular free wall rupture is a lethal complication after transmural

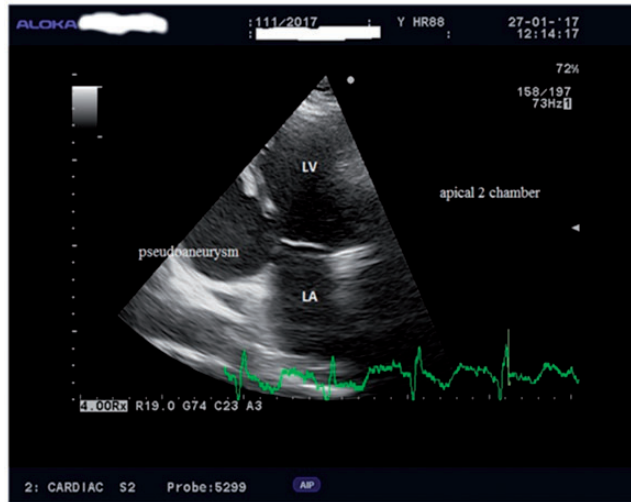


Figure 3. Apical two-chamber view. Pseudoaneurysms have a neck that is narrower than the diameter of the aneurysm. LV: left ventricle, LA: left Atrium.

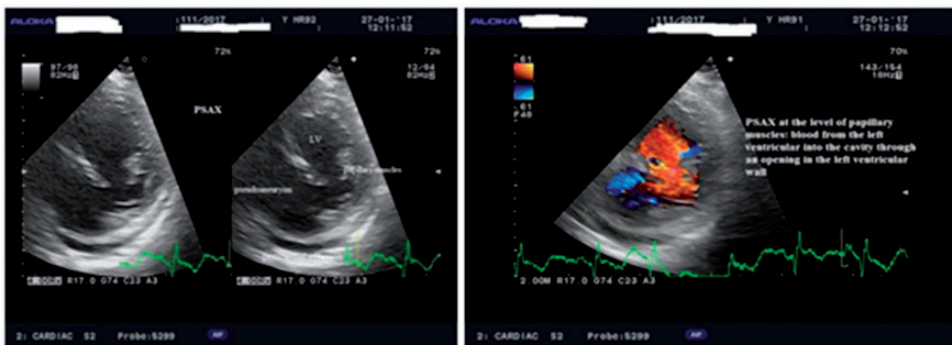


Figure 4. Parasternal short-axis (PSAX) view at the level of the papillary muscles. The image shows a dyskinetic cavity that was connected to the posteroinferior wall and communicated with the left ventricle, causing damage to the posterior mitral valve leaflet (P1 scallop) and resulting in severe mitral regurgitation. Note the presence of mild pericardial effusion. LV: left ventricle.

infarction following acute myocardial infarction.⁴ Pseudoaneurysms without treatment have a 30% to 45% risk of rupture and a 50% mortality rate when not treated surgically; the mortality rate is 23% with surgical resection.⁴ A typical diagnostic problem is the differential diagnosis between a left ventricular pseudoaneurysm and true aneurysm.⁵ Pseudoaneurysms have a neck that

is narrower than the diameter of an aneurysm. They are located in the posterior or inferior left ventricular wall. Transthoracic echocardiography shows the presence of a dyskinetic cavity, and the optimal therapy is surgery, which has a lower surgical risk than that of medical therapy.⁶ In contrast, true aneurysms are located in the posterior left ventricular wall in only 3% of cases;

transthoracic echocardiography shows a thinned myocardium with no contractility, and they can be managed either medically or surgically. Congestive heart failure, embolic events, and ventricular arrhythmias can be the first clinical manifestation of both pseudoaneurysms and true aneurysms.⁶

Mitral regurgitation may occur due to left ventricular dilatation, papillary muscle dysfunction or tip rupture, or chordae tendineae rupture.⁶ In the present case, neither the moderate mitral regurgitation nor the mildly reduced ejection fraction provided a convincing explanation of the patient's dyspnea. Frequent premature atrial and ventricular complexes may be perceived by the patient as dyspnea.⁷ Switching from metoprolol to bisoprolol reduced the arrhythmic burden but did not improve the dyspnea.

Cardiac tamponade was easily excluded. Pulsus paradoxus was not detected, and echocardiography was negative for echocardiographic signs of cardiac tamponade.⁸ Finally, psychogenic dyspnea should be a diagnosis of exclusion in such cases.⁹

In patients with delayed presentation of ST-elevation myocardial infarction, a prolonged ischemic time is a risk factor for the appearance of life-threatening cardiac complications.⁶ Especially in patients with dyspnea who remain symptomatic despite optimal medical treatment, repeated clinical examination, repeated echocardiography, transesophageal echocardiography, cardiac computed tomography or magnetic resonance imaging, radionuclide scanning, and left ventricular angiography can help to confirm or rule out specific cardiac complications that require emergency surgery.¹⁰

Conclusion

Although the outcome of most patients with pseudoaneurysm of the left ventricle appears to be relatively benign, those with symptoms of heart failure that cannot be

improved by optimal medical treatment must be urgently diagnosed and treated.

Declaration of conflicting interest

The authors declare that there is no conflict of interest.

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