INTRODUCTION

Left ventricular (LV) aneurysms have long been described during autopsies. However, it was not until 1881 that they were associated with coronary artery disease. The first angiographic diagnosis of an LV aneurysm was made in 1951. In 1977, the true LV aneurysm was defined as a thinned segment of the left ventricle - a thin layer of muscle and scar tissue, as well as the pericardium, that protrudes during both systole and diastole, and has a broad neck, yet a growing number of authors favor defining it more loosely as any large area of left ventricular dyskinesia that lowers the ejection fraction. The true LV aneurysm is a thinned segment of the left ventricle - a thin layer of muscle and scar tissue, as well as the pericardium, that protrudes during both systole and diastole, and has a broad neck, yet a growing number of authors favor defining it more loosely as any large area of left ventricular dyskinesia that lowers the ejection fraction. The main cause of aneurysm formation, in up to 95% of cases, is coronary artery disease, the rest of the cases being due to congenital or traumatic causes, Chaga’s disease, sarcoidosis and, in exceptional cases, due to diverticula of the left ventricle. The main cause of aneurysm formation, in up to 95% of cases, is coronary artery disease, the rest of the cases being due to congenital or traumatic causes, Chaga’s disease, sarcoidosis and, in exceptional cases, due to diverticula of the left ventricle. 

REZUMAT

Anevrismele de ventricul stâng (VS) au drept cauză principală în până la 95% din cazuri boala coronariană: infarctul miocardic acut cu supradenivelare de segment ST la nivelul peretelui anterior al VS, în asociere cu ocluzia arterei coronare descendentante (ADA) și cu circulația colaterală săracă. În această prezentare de caz, dorim să prezentăm formarea unui anevrism gigant de VS, în ciuda reperfuziei spontane a ADA, la aproximativ o săptămână de la evenimentul acut, în ciuda tratamentului correct antiremodelare ventriculară. Această prezentare vine să sublinieze importanța ecocardiografiei pentru diagnosticul pozitiv și localizarea preciză a anevrismelor VS, precum și importanța ecocardiografiei de contrast pentru diagnosticul diferențial al acestei entități. În acest sens, cea ce părea a fi o ruptură de perete de ventricul stâng cu sângerare întrapericardică, era, de fapt, un anevrism de ventricul stâng asociat cu pericardită, aspect ce a fost ulterior confirmat în timpul intervenției chirurgicale. În plus, pacienta a prezentat insuficiență mitrală ischemică, mecanism documentat ecocardiografic tridimensional și confirmat de aspectul intraoperator.

Cuvinte cheie: anevrism de ventricul stâng, ecocardiografie de contrast, ecocardiografie tridimensională

ABSTRACT

Left ventricular (LV) aneurysms have as the main cause of formation, in up to 95% of the cases, the coronary artery disease: the ST myocardial infarction of the LV anterior wall, in association with total left anterior descending (LAD) coronary artery occlusion and poor collateral circulation. In this case report we want to present a situation where a giant LV aneurysm was formed, despite the LAD spontaneous reperfusion, 1 week after the acute event, and despite the correct anti-remodeling medication. We want to emphasize the role of echocardiography for the positive diagnosis and the precise location of the LV aneurysm, as well as the importance of contrast-echocardiography in regards to the differential diagnosis that may rise from the clinical and paraclinical context. What seemed to be a LV wall rupture was, in fact, a true LV aneurysm, independently associated with pericarditis, an aspect later confirmed during surgery. In addition to this, the ischemic mechanism of mitral regurgitation was documented by tridimensional echocardiography and also confirmed by intraoperative data.

Key Words: left ventricular aneurysm, contrast echocardiography, tridimensional echocardiography
basal inferior wall, in right coronary artery (RCA) occlusion.\textsuperscript{13,14} Aneurysm formation is rare in patients with STEMI who receive fibrinolytic therapy and later exhibit a patent infarct-related coronary artery. Rare as it is, still, 7.2\% of patients with patent infarct-related arteries on the angiogram will develop an aneurysm.\textsuperscript{15} Despite this, the absolute percentage of LV aneurysms has decreased over the last years, due to the early thrombolysis and revascularization techniques.\textsuperscript{16,17} The aneurysm formation consists of 2 phases: the early expansion and the late remodeling. The early expansion begins within the first 48 hours after the infarction in half of the patients that develop an aneurysm, and in the rest of the cases after a few weeks.\textsuperscript{18} The late remodeling phase begins 2-4 weeks after the acute event, when the granulation tissue will be formed, which will later be replaced by fibrous scar tissue, 6-8 weeks later, thus creating the true aneurysm.

\section*{CASE REPORT}

We want to report a case of a 67 years old female who, within a period of 4 months, developed a giant left ventricular aneurysm.

In February 2010, she was admitted in our clinic for coronarography, one week after an acute event: anterior myocardial infarction. We must underline that the patient did not undergo fibrinolytic therapy at the acute moment, due to very high, uncontrolled blood pressure values (210/125mmHg). On admission, her physical examination showed normal values for the blood pressure (under medication: beta blocker, angiotensin converting enzyme inhibitor (ACEi), diuretics), a systolic apical murmur (grade III out of VI) and normal pulmonary sounds on auscultation.

The ECG revealed irregular sinus rhythm with ventricular ectopic beats and an acute anterior STEMI aspect: QS with ST elevation of 4mm in V1-V4 and of 2mm in V5 leads.

The biological data pointed out a type II diabetes associated with dyslipidemia.

From the echocardiographic evaluation, we underline the following aspects: hypertensive cardiopathy, slight degenerative mitral and aortic lesions, moderate pulmonary hypertension with severe tricuspid regurgitation, ischemic grade II mitral regurgitation (MR). The end diastolic volume was 82ml, with apical dyskinesia and anterior akinesia. The left ventricular ejection fraction was 40\%. We performed tridimensional (3D) transthoracic echocardiography to study the etiology of MR. The normal aspect of the entire mitral apparatus associated with LV remodeling sustained the ischemic etiology. (Fig. 1)

The angiocoronarography revealed a right dominance, subocclusion of the LAD after the emergence of its first branch and no other significant lesions. Percutaneous transluminal angioplasty (PTCA) and stent implant was performed for the LAD lesion, with good angiographic result, TIMI III flow.

The patient was discharged with the following medication: beta blocker, ACEi, Spironolactone, Aspirin, Clopidogrel and statins, with an immediate favorable evolution.

Two months later, the patient was admitted again, this time with class IV NYHA symptoms: dyspnea at rest, ortopnea, paroxysmal nocturnal dyspnea. The physical examination revealed regular tachycardia (130bpm), low blood pressure (90/55mmHg), the same apical murmur, edema of the calf, painful hepatomegaly.

The ECG showed a persistent QS and ST elevation in V1-V5. There were no major changes in the biological profile, except for a high plasma fasting glucose.

The major changes from the previous investigations
came with the echocardiographic exploration. Even though the mitral regurgitation was now grade I, with grade II tricuspid regurgitation and mild pulmonary hypertension (systolic pulmonary arterial pressure – SPAP = 40mmHg), the end diastolic volume of the left ventricle was 200ml, with a giant apical aneurysm, a very low EF (10%) and a medium quantity of pericardial fluid, along with right atria collapse. (Fig. 2) At this point a suspicion of myocardial rupture and pseudoaneurysm formation arose. We performed contrast echocardiography, which offered the differential diagnosis between LV aneurysm and pseudoaneurysm. (Fig. 3)

The only therapeutic option in this case was considered to be the surgical intervention. The ACC/AHA guidelines state that patients with STEMI that develop left ventricular aneurysms associated with malignant ventricular tachyarritmias and/or pump failure unresponsive to medical or catheter-based therapy should be considered for aneurysmectomy and coronary arterial bypass graft (CABG).13

The main goal of the surgical intervention was to resect the aneurysm and to reconstruct the left ventricular geometry, using a Dacron patch. The operation was performed using cardio-pulmonary bypass, cannulation of the left atria, the inferior vena cava, the aortic root and the coronary sinus, cardioplegia, systemic normothermia. The intraoperative findings, consistent with the echocardiographic ones, were as follows: a giant aneurysm of the apex, anterior and lateral wall, 8-10cm, with an intracardiac thrombus; the only contractile region of the left ventricle remained the basal parts. (Fig. 4) The papillary muscles and the chordae of the mitral apparatus appeared to be normal.

The first step was to evacuate, layer by layer the intracavitary thrombus, allowing afterwards a better assessment of the aneurysm: ¾ of the lateral wall, the anterior wall, the entire interventricular septum, the apex. The next step was the aneurysmectomy, with the resection of almost the entire scar tissue. The reconstruction of the left ventricle was done using a Dacron patch, in a manner leaving more than 1 cm of extra material at the border of the suture, allowing further remodeling of the ventricle.

The early prognosis in our patient was favorable, and she was discharged 10 days after surgery, with the following medication: beta blocker, ACEi, diuretics, antiplatelet medication, statins and oral antidiabetic therapy. On the long term, her prognosis still remains uncertain, due to the major cardiovascular risk factor association and it is totally dependent on their adequate control.

**DISCUSSION**

The history of LV aneurysm begun in 1944, when Beck described fascia lata plication to treat a left ventricular aneurysm.19 Then, in 1955, Likoff
and Boyle successfully resected an aneurysm through thoracotomy, using a special clamp, without cardiopulmonary bypass.\textsuperscript{20} The modern treatment area began in 1958 when Cooley performed a linear repair, using cardiopulmonary bypass.\textsuperscript{21} Since then, all the techniques aim to achieve a good left ventricular anatomy, as close to the normal heart as possible.

In the early stages after the intervention, the mortality rate is dependent on the surgical technique; the lowest mortality (3-7\%) is associated with patch or linear closures.\textsuperscript{22-24} Other important early stage mortality risk factors include age, high class NYHA heart failure, female gender, EF<30\%, pulmonary mean pressure >53mmHg, serum creatinine >1.8mg/dl and failure to use the internal mammary artery for CABG.\textsuperscript{16,23-26}

Long term prognosis is dependent on the adequacy of the residual left ventricular function, patients with severe left ventricular dysfunction having higher mortality rates.\textsuperscript{25}

In this case the early prognosis was favorable, but on the long term, the prognosis still remains uncertain, due to the major cardiovascular risk factor association.

The particularity of this case is that we are dealing with a patient with numerous major cardiovascular risk factors (hypertension, hyperlipoproteinemia, type II diabetes mellitus, age) which led to an important coronary vascular event - an anterior myocardial infarction. In evolution, from a small apical dyskinetic area, in a few months, appeared an important alteration of LV function and hemodynamics. It is essential to emphasize that at the time of the PTCA (1 week after the acute event), the LAD was sub-occluded (probably spontaneous endogenous reperfusion, as she was given no fibrinolytic medication). Despite this and despite the optimal medication against the ventricular remodeling process, the damaged myocardium was of high proportions, probably due to lack of collateral circulation, the expansion and remodeling phase were aggressive and, in 4 months, a giant aneurysm was formed.

The echocardiographic study used multiple techniques for a correct diagnosis. For the initial diagnosis of the cause of mitral regurgitation, 3D echocardiography was used, and the ischemic etiology was confirmed intraoperatory. Then, the contrast echocardiography assured the differential diagnosis between a LV pseudoaneurysm and a true aneurysm with thrombus inside. Usually, the contrast echocardiography is useful for the identification of intracardiac shunts or unidentified structures, for endocardial border detection and for assessing the myocardial perfusion.\textsuperscript{27} However, situations have been reported when the differential diagnosis between a true and a false aneurysm of the LV can be made using a contrast agent.\textsuperscript{28}

In conclusion, a correct diagnosis was made due to a complete echocardiographic study, using multiple techniques, which led to an optimal surgical treatment.

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REFERENCES


