Chronic Left Ventricular Pseudoaneurism of Undetermined Etiology

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Pseudoaneurysms occur as a rare complication of the myocardial infarction, of which hemorrhagic process is contained by adherences of the visceral or parietal pericardium – or of both – preventing the development of cardiac tamponade. We report the case of a 55-year-old patient, with a chronic left ventricular pseudoaneurysm of undetermined etiology, who was submitted to a conservative treatment.

Introduction

The left ventricular (LV) false aneurysms or LV pseudoaneurysms represent rare cases of myocardial rupture, of which hemorrhagic process is contained by pericardial adherences, which prevents the development of cardiac tamponade, generating saccular structures without muscle tissue, with organized thrombi within them[1]. Most of them result from complications of the acute myocardial infarction[1,2].

We present a case of LV pseudoaneurysm of undetermined cause, with chronic evolution, which resulted in heart failure and probable cerebral embolism, who received conservative treatment.

Case Report

A 55-year-old male patient, Brazilian mulatto, retired rural worker, presenting progressive dyspnea during physical exertion for the previous 5 months, associated with coughing with yellow sputum, hyporexia and weight loss was evaluated. He denied chest pain, palpitations, syncope or fever. He had present prior episodes of cerebrovascular accidents 1 and 10 years before, with convulsive crises and memory loss, without significant motor sequelae. He used phenytoin and ASA, was sedentary, and denied smoking, diabetes, arterial hypertension, dyslipidemia, infarction or other heart diseases. Due to the clinical and radiological suspicion of bronchopneumonia, he received amoxicillin for 14 days, without improvement. Therefore, a chest tomography was requested and the patient was referred to assessment by a cardiologist.

At physical examination, the patient present good general health status, was slightly dyspneic, with blood pressure of 100 x 80 mmHg and pulse of 72 bpm. The heart auscultation showed 2 rhythmic normal heart sounds, with a slight systolic murmur in mitral focus. There was a decreased vesicular murmur at the left pulmonary basis and fine rales at the right basis. There was no jugular stasis or hepatomegaly. The patient presents mild lower-limb edema.

Levels of glycemia, electrolytes, total cholesterol and fractions and triglycerides were normal, with a minimal increase in creatinine. The electrocardiogram showed a sinus rhythm with left anterior-superior division block, left atrial overload and a probably inactive anterior zone.

The chest X-ray showed cardiomegaly with a left radio-dense paracardiac image. The chest computed tomography (CT) showed an enlarged cardiac area, with a saccular image in the LV apical region with parietal calcification, measuring 13 x 7 x 6 cm, in addition to LV filling failures, compatible with the presence of thrombi.

The echocardiogram, in addition to the significant LV dilatation, disclosed diffuse and significant systolic dysfunction, associated with moderate diastolic dysfunction. There was moderate mitral reflux; a mobile thrombus was identified in the left ventricle and the presence of a pseudoaneurysm was identified in the apical region, with partial thrombosis (Figures 1A and 1B).

Considering these findings, a coronary angiography was performed, which revealed normal coronary arteries, in addition to important LV dysfunction and apical pseudoaneurysm. A nuclear magnetic resonance (NMR) was carried out, which also disclosed the large apical pseudoaneurysm extending upward. There were mobile thrombi within the dilated and diffusely hypokinetic LV, with narrowing and akinesis of the lower wall (Figure 2).

The patient received angiotensin-converting enzyme inhibitor, beta-blocker, diuretic and oral anticoagulant agents, with improvement of the cough and dyspnea symptoms. Although a surgical proposal was discussed, we chose to follow the patient at ambulatory level with clinical monitoring and through imaging techniques, due to the patient's social limitations, as well as the presence of similar images at the radiography and the chest X-ray obtained years before, which were subsequently brought by family members.

Six months after the initial diagnostic investigation, the patient came to the hospital due to decompensated heart failure symptoms. At this time he was submitted to a new

Key Words

Aneurysm, false; ventricular dysfunction, left; myocardial infarction / complications.

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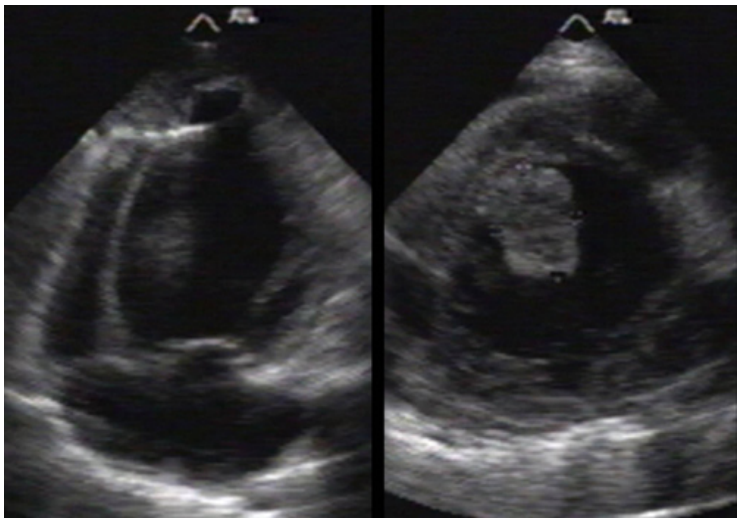

Figure 1A and 1B - Transthoracic echocardiogram. Pseudoaneurysm in the LV apical region with thrombus inside it and thrombus in the ventricular cavity.

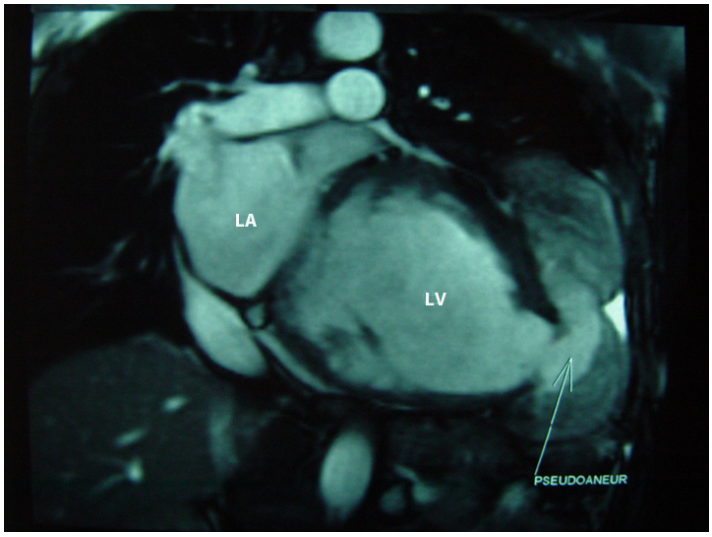

Figure2 - Nuclear magnetic resonance of the heart. Pseudoaneurysm in the LV apex, with thrombus inside it and in the left ventricular cavity.

echocardiogram, which showed to be similar to the previous one, except for the absence of thrombi within the LV.

Discussion

The present case describes a patient with LV failure and probable previous embolic event due to a LV pseudoaneurysm, with at least 15 months of evolution, of undetermined etiology.

The most common cause of LV pseudoaneurysm is a mechanical complication after a transmural acute myocardial infarction[1,2]. Other less frequent causes include: manipulation of the heart cavities after cardiac surgical procedure; penetrating or closed chest trauma; endocarditis; and more rarely, after suppurative pericarditis or due to tumor infiltration[2-4]. None of these conditions were present in this case, who presented a global LV dysfunction associated to the pseudoaneurysm. Even with the parietal narrowing in this situation, it becomes difficult to understand the occurrence of spontaneous myocardial rupture. It can be speculated the remote possibility of a coronary embolism from intraventricular thrombi caused by a pre-existing dilated myocardiopathy, with a myocardial infarction complicated by a pseudoaneurysm.

The patients with LV pseudoaneurysms can be completely asymptomatic or present symptomatology that is similar to that of true aneurysms: dyspnea, arrhythmias, angina or consequences of the systemic embolism[2]. In the present case, the embolic phenomena preceded the heart failure

manifestations, which might have occurred due to the existence of thrombi in the dilated and hypocontractile LV or even originated from within the pseudoaneurysm.

The pseudoaneurysm can be detected at the chest X-ray as an area adjacent to the heart that presents clear expansion at seriated images, which can even reach dimensions similar to that of the ventricle. The electrocardiogram can disclose unspecific repolarization alterations. Imaging assessments such as the echocardiogram with or without contrast, computed tomography, nuclear magnetic resonance and angiography can help define the diagnosis, differentiating it from the true aneurysm, from the pericardial cyst and the localized pericardial effusion[1,5]. In the present case, it was necessary to use several modalities of imaging diagnosis, both invasive and noninvasive, given the exceptionality of the absence of obstructive coronariopathy at the coronary angiography assessment.

In spite of the possibility of an unfavorable outcome of the pseudoaneurysm when the patient is submitted a conservative treatment, we chose, in the present case, the optimization of the drug therapy as the initial strategy, as this was a chronic case, among other factors.

Differently from the true aneurysm, the pseudoaneurysm presents a high risk of rupture, with progression to hemopericardium and death, and therefore, it has an indication of emergency surgical resection[2,6]. However, similarly to the present case, there are many reports of patients with chronic pseudoaneurysms with high survival and no rupture-related mortality[7,8], which leaves the question on the best conduct (surgical or conservative approach) open to discussion for each particular case. In 2000, Pretre et al[9] reported a relatively high surgical mortality for a pseudoaneurysm formed soon after an acute infarction or in cardiac reoperations. However, as the rate of fatal rupture in acute or chronic expanding pseudoaneurysm is high, these authors concluded for the potential benefit of the surgical treatment in comparison to the clinical one, in these conditions. Moreno et al[10], in turn, presented a series of 9 patients with pseudoaneurysm secondary to myocardial infarction, treated conservatively, with a mean follow-up of 3.8 years, with no evidence of death due to myocardial rupture in any of the cases. Nonetheless, three patients presented ischemic cerebrovascular accident, resulting in the recommendation for treatment with oral anticoagulation in these cases, in spite of the inhibition of local thrombus formation as a way to tamponade the process.

Therefore, we conclude that the choice of the conservative *versus* invasive treatment strategy in cases of LV pseudoaneurysm must take into account the etiological, morphological and evolution characteristics and needs to be individualized for each case.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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Study Association

This study is not associated with any post-graduation program.

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