

Fig. 1 - Two-dimensional echocardiogram. Cross section of four chambers evincing the presence of a suggestive image of left ventricular (LV) pseudoaneurysm (pseudo AN); LA = left atrium; RA = right atrium; RV = right ventricle.

without significant residual regurgitation, which made the annuloplasty unnecessary. The operation was completed with the myocardial revascularization through of grafts of anterior aorta-descending saphena vein and aorta – 1st diagonal branch. The anatomopathologic findings confirmed the left ventricular free wall rupture, with keeping of ventricular cavity through thrombi and pericardium. That confirmed the diagnosis of left ventricular pseudoaneurysm (fig. 4). Important congestive clinical features complicated the immediate post-operation period. A mild mitral regurgitating murmur was verified at the cardiac auscultation. The echocardiogram performed on the 10th post-operation day showed a normal sized left ventricle and moderate systolic dysfunction (ejection fraction = 35%), at the expense of an inferior and posterior akinesia and hypokinesia on the other walls. A discreet mitral insufficiency and moderate pericardial effusion, which was circumscribed to the left ventricular inferoposterior wall, were detected. The patient was discharged after clinical compensation.

During the in-hospital follow-up, the patient was under congestive cardiac insufficiency of functional class II (NYHA) with a mild mitral regurgitation. The tardive echocardiographic assessment confirmed the post-operation findings and showed a regression of the pericardial effusion.

Discussion

The ventricular pseudoaneurysm is suggested by the bulging of the cardiac silhouette detected in image examinations. Its formation comprises the gradual expansion of the intrapericardial hematoma through blood overflowing, from the ventricular cavity to the pericardial sac, after a myocardial rupture partially restrained through thrombi, fibrosis, and intrapericardial adhesions. The definite diagnosis is exclusively done through an anatomopathologic exam, which demonstrates a complete absence of myocardial fibers in a certain extension of the left ventricular walls, which are replaced for organized hematoma and thickened pericardium^{1,2}.

The real incidence of left ventricular pseudoaneurysm is unknown due to its extreme rarity and diagnostic difficulty, which makes it even more unusual after the fibrinolytic era. Despite that, it is known that the posterior topography of those fake aneurysms is at least two times more usual than the previous one. Such fact

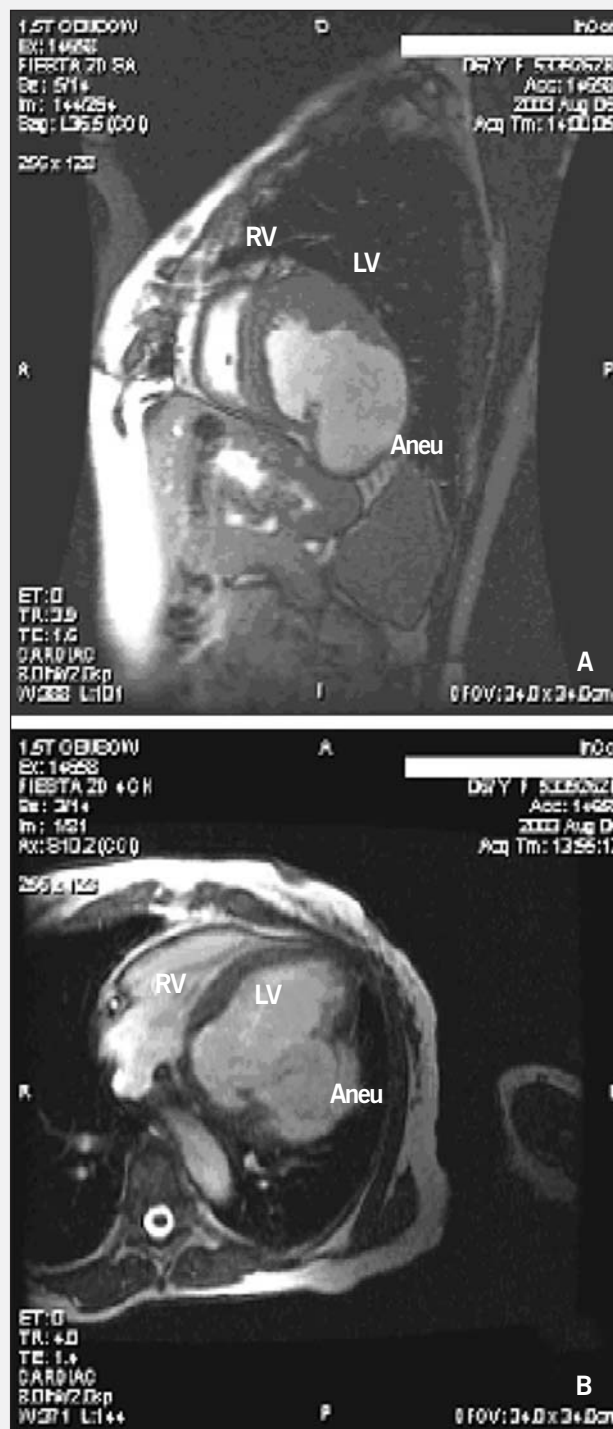


Fig. 2 - Cardiac magnetic nuclear resonance evincing the relation among the right ventricular cavity (RV), left ventricular cavity (LV), and ventricular aneurysm (Aneu). A. short axis; B. long axis.

possibly originates from the support of the inferoposterior face of the heart on the diaphragm. The support of the diaphragm favors the formation of intrapericardial adhesions during the evolution of the local fibrinous pericarditis after the lower infarction. Such fact permits that the heart does not break abruptly, despite the necrosis of the whole thickness of the cardiac muscle of the ventricular wall, facilitating the restraint of ventricular cavity by the adjacent pericardium. From there, the local inflammatory reaction that thickens the pericardium stresses, which makes it even more rupture-resistant^{1,3-5}.

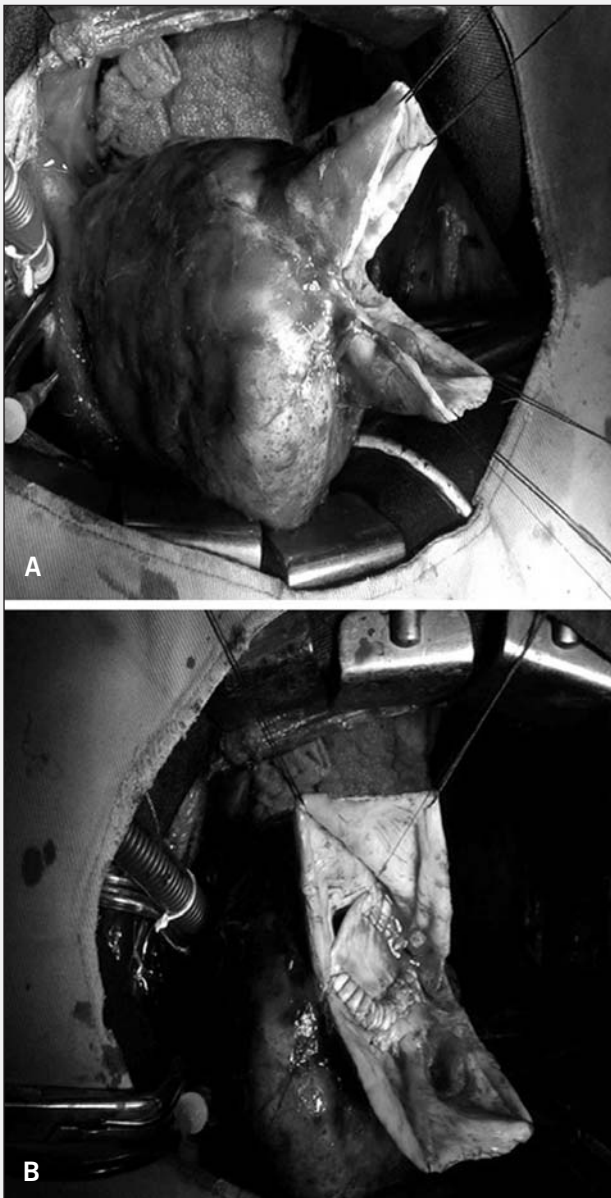


Fig. 3 - Intra-operative findings. A) anterior face of the heart with the pseudoaneurysm opened, repaired through surgical threads. Note the thin thickness of its wall; B) detail of the suture of bovine pericardium repair on the pseudoaneurysm orifice. That closing remodeled the ventricular cavity and approximated the papillary muscles.

In the reported case, besides the delay in the myocardial reperfusion resulting from the tardive admission of the patient, it was not possible to obtain a complete TIMI III distal flow, through distal embolization to the posterior ventricular branch of the right coronary and for the microflow (a *no-reflow* phenomenon), contributing to a more extensive myocardial damage and predisposing to the formation of ventricular pseudoaneurysm.

The clinical diagnosis of that condition is difficult. In almost two decades of follow-up, between 1980 and 1996, only 22 antemortem cases of postinfarction ventricular pseudoaneurysm were diagnosed at Mayo Clinic. In that series, most of patients were males and old aged (average age of 68 years old). The average interval between the myocardial infarction and the diagnosis of ventricular pseudoaneurysm was 3.9 months, varying from 0 days to 15 years. The main clinical manifestations associated to the formation of pseudoaneurysm were cardiac insufficiency, precor-

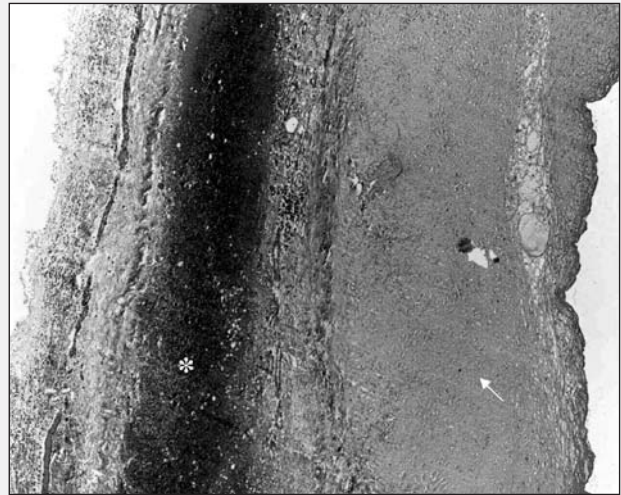


Fig. 4 - Anatomopathologic findings. Cavitary lesion wall constituted by a thick layer of fibrosis (arrow), re-covering a hematoma characterized by erythrocytes (especially present in the asterisk area, but also out of it, including among collagen). Note the absence of myocardial fibers.

dialgia, syncope, arrhythmia and systemic thromboembolism. Almost a quarter of the cases was asymptomatic and was accidentally discovered. Half of the patients had previous hypertension, and most of them (87%) were victims of first infarction. The physical examination was non-specific, but most of them showed systolic murmur in mitral focus at auscultation, which suggested simultaneous affection of mitral valve in that situation⁵. The above-mentioned case lies within such epidemiological profile. The diagnosis of left ventricle posterolateral pseudoaneurysm was provided by the echocardiogram performed for the assessment of cardiac insufficiency in the 4th month after first infarction.

The image examinations are the most important part in the investigation, as the electrocardiogram and the simple thoracic radiography are of little use.

The angiography was the main image method used, showing high sensitivity in the diagnosis of complication. The presence of a narrow orifice leading to a saccular dilatation and the absence of coronary arteries externally bordering the aneurismal wall, are angiographic findings that suggest the presence of pseudoaneurysm, as opposed to the real ventricular aneurysm³.

More recently, the two-dimensional echocardiogram has offered some diagnostic gains, allowing for detecting the discontinuity of ventricular endocardium, as well as checking the presence of a narrow orifice (in relation to the diameter of the aneurysm) communicating between the ventricular and aneurismal cavities. The pulse and color Doppler have been very useful, by permitting the identification of intercavity systolic and diastolic flows, i.e., between the left ventricle and the aneurysm. Despite detecting abnormalities in up to 90% of the patients, it allows for the definite diagnosis in only 25 to 33% of the cases³.

Many recent works have been reported the usefulness of the magnetic nuclear resonance for identification of the presence of left ventricular pseudoaneurysm. The advantages of the method are the excellent spatial definition and the capacity of examine the heart globally, which allows for the detection of the size and location of the pseudoaneurysm, besides differentiating the many adjacent structures as the pericardium, thrombi and myocardium⁴.

For being a little invasive, low cost and easily available method,

the echocardiogram must be preferred for the initial diagnostic investigation. The magnetic nuclear resonance should be performed as a supplementary method, as the definite diagnosis normally raises doubts and the detection of a residual layer of myocardium on the ventricular wall can make the prognostic more favorable. The cardiac catheterization can be used in case diagnostic doubts persist or aiming at defining the coronary anatomy, with the objective of a better surgical planning.

The surgical treatment is the most suitable for left ventricular pseudoaneurysm due to the risk of rupture and sudden death, which occurs in 30 to 45% of the cases^{1,3,6}. However, some authors propose the possibility of clinical treatment for pseudoaneurysms tardively diagnosed, after 3 months from the infarction, for considering that the spontaneous rupture is less usual at that stage^{4,5}. The frequent association of serious mitral regurgitation to the left ventricular posterior wall fake aneurysm is related to high operation mortality rate⁷. However, regardless of the modality of therapy used, the pseudoaneurysm mortality seems to primarily relate to the seriousness of ventricular dysfunction and subjacent ischemic cardiomyopathy. In the case under matter, the surgical approach was adopted, aiming at improving the clinical condition of the patient, who, despite the optimization of the clinical therapy, was still dyspneic to little strains.

The pseudoaneurysm reduces the cardiac debt when it dilates during the left ventricular systole, by taking part of the blood volume to be ejected by the heart. So, it impairs the antegrade ventricular ejection during systole, which generates cardiac insufficiency.

Such hemodynamic change leads to progressive ventricular dilatation. In the coexistence of mitral insufficiency and left ventricular pseudoaneurysm, the hemodynamic conditions are even more unfavorable and the evolution to ventricular failure can be much faster. In those cases, despite the urgent surgical indication, the exchange of mitral valve, simultaneously to the repair of pseudoaneurysm, may produce a sudden increase of left ventricular post-load, with a possible failure of pump in the post-operation period. For that reason, the option for the repair of mitral valve, preserving the functioning of valve system (cordages and papillary muscles) seems to be the earnest option, which favors the ventricular performance⁷.

Mitral insufficiency associated to pseudoaneurysm is originated from three main factors: dilatation of the mitral valvar ring, ventricular dilatation and restriction of the motility of posterior valvar leaflet, due to the loss of contraction of ventricular wall and/or papillary muscles. In the reported case, the repair of myocardial wall, with approximation of the rims and the use of bovine pericardium patch, allowed for the restoration of the left ventricle geometry, reestablishing the mitral valvar function. Such fact can be explained by the reduction of the dyskinetic area, ventricular reconstruction and adjustment of tension of posterior papillary muscle, allowing for a better functioning of mitral system. Despite the annuloplasty is usually performed in the repair of mitral valve that follows the correction of ventricular pseudoaneurysm, the exclusion of the akinetic area, with ventricular reconstruction and adjustment of tension of posterior papillary muscle, seems to be the main determinant for an appropriate surgical correction of mitral insufficiency⁷.

References

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