

## Left Ventricular Pseudoaneurysm Associated to Severe Mitral Insufficiency, Complicating Inferolaterodorsal Acute Myocardial Infarction

João Luiz de A. A. Falcão, Sandra Nívea R. S. Falcão, Maria Fernanda M. A. Garcia, Ana Lúcia M. Arruda, Alexadre C. Hueb, Fábio B. Jatene, Paulo S. Gutierrez, José Carlos Nicolau, José Antônio F. Ramires, Roberto Rocha C. V. Giraldez Instituto do Coração do Hospital das Clínicas - FMUSP - São Paulo, SP - Brazil

We described a case of left ventricular pseudoaneurysm associated to a severe mitral regurgitation, complicating a inferolaterodorsal acute myocardial infarction. The lesion was found in a routine echocardiogram during the in-hospital follow-up. The well-succeeded surgical strategy and the good clinical evolution of the patient were distinguished.

The left ventricular pseudoaneurysm after acute myocardial infarction is a very rare complication and it is not frequently diagnosed. However, its appearance gives rise to a bleak prognostic to its carriers, who can evolve quickly to death. Opposing to the rupture of the free ventricular wall, the pericardial adherences of pseudoaneurysm have the intraventricular blood, which gives a less acute nature to such condition and allows for the therapeutic intervention to be performed before a casual fatal outcome. So, it is important to be aware about the risk of appearance of postinfarction pseudoaneurysm, in order to not to resort to more suitable diagnostic methods and correct the condition rapidly after its confirmation.

## Case report

A 67-year-old female patient, hypertensive, carrier of diabetes mellitus and irregular control dyslipidemia, was admitted to the Emergency Room of Instituto do Coração with a severe pain on the left shoulder for 10 hours. The clinical features started at rest, associating to nausea, vomit and cold sudoresis. The echocardiogram indicated the presence of an ongoing inferolaterodorsal acute myocardial infarction, with affection of the right ventricle. The coronary angiography of urgency revealed a complete occlusion of the left coronary artery in its proximal portion, in addition to luminal obstructions of 70% in the mid-third of the anterior intraventricular artery (anterior descendent), 90% in the mid-portion of its first diagonal branch and 90% in the first marginal branch of the circumflex artery. The patient was submitted to a percutaneous transluminal angioplasty of the left coronary with a stent implant,

Mailing address: João Luiz de A. A. Falcão - Rua Alves Guimarães

408/121 - 05410-000 - São Paulo, SP - Brazil

E-mail: jlfalcao@uol.com.br

Received for publishing on 07/10/2004

Accepted on 12/16/2004

from which a distal coronary flow was obtained, and classified as TIMI II. The procedure was complicated by thrombosis and distal embolization with occlusion of the posterior ventricular branch of the right coronary. The patient evolved during the initial stage of hospitalization in Killip Class II, keeping herself dyspneic to little strains. The echocardiographic assessment on the 2<sup>nd</sup> day postacute myocardial infarction showed akinesia of the left ventricular lower and posterior walls, associated to side and septal hypokinesia, which led to an important left ventricular systolic dysfunction (ejection fraction through the method of Simpson=35%). The right ventricle showed normal systolic function. At the Doppler, the mitral insufficiency was moderate. Progressively, the patient improved her clinical condition, with the use of conversion enzyme inhibitor, hydralazine, nitrate and diuretics. There was a reduction of the tiredness, which still persisted to moderate strains, and the pulmonary rales disappeared. The transthoracic echocardiogram, performed on the 7th day post-myocardial infarction, indicated a clear reduction of mitral insufficiency, with appearance of discreet pericardial effusion. The myocardial perfusion photoscan, performed on the 10th day post-myocardial infarction showed an underscored persisting hypocaptation of the left ventricular lower, side, apical and dorsal walls. The radioisotopic ventriculography, conjoined to the photoscan study, estimated the left ventricular fraction of ejection in 20%. The discharge took place on the day after.

In the in-hospital follow-up, the patient persisted with tiredness and dyspnea to moderate strains, despite therapeutic optimization. The follow-up echocardiogram (4 months after the acute myocardial infarction) confirmed an important left ventricular dysfunction (ejection fraction=27%), at the expense of lower akinesia and moderate mitral insufficiency, when the presence of an extensive left ventricular posterolateral pseudoaneurysm, with a 3.5 cm diameter collum, associated to a moderate pericardial effusion, which was related to the left ventricular inferoposterior wall (fig. 1).

The patient was hospitalized for clinical reassessment and clinical approach. She was submitted to a heart magnetic nuclear resonance that confirmed the diagnosis of ventricular pseudoaneurysm of left ventricular inferoposterior wall and serious mitral regurgitation (fig. 2) and the surgical correction was indicated. During the procedure, the aneurysm-free wall was exsiccated and the myocardial imperfection was corrected with a bovine pericardium patch (fig. 3). The mitral valve was explored through left atriotomy. The hemodynamic trials showed a good coaptation of valval leaflets,

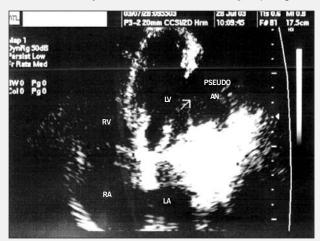


Fig. 1 - Two-dimensional echocardiogram. Cross section of four chambers evincing the presence of a suggestive image of left ventricular (LV) pseudoaneurysm (pseudo AN); LA = left atriun; RA = righ atriun; RV = righ ventricle.

without significant residual regurgitation, which made the annuloplasty unnecessary. The operation was completed with the myocardial revascularization through of grafts of anterior aortadescending saphena vein and aorta -1<sup>st</sup> 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 10<sup>th</sup> 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<sup>1,2</sup>.

The real incidence of left ventricular pseudoaneurysm in 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

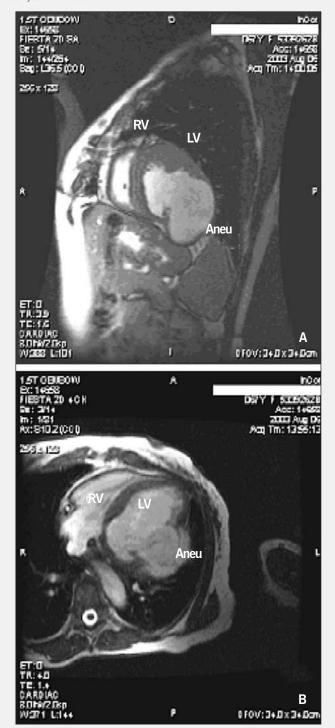


Fig. 2 - Cardiac magnetic nuclear resonance evincing the relation among the right ventricular cavity (RV), left ventricular cavity (LV), and ventricular aneurysm (Aneu). A. short axis; B. long axis.

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<sup>1,3-5</sup>.

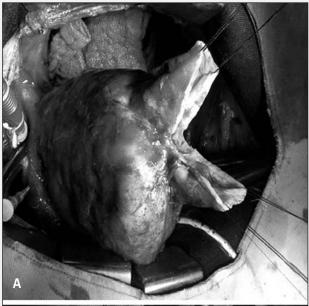




Fig. 3 - Intra-operative findings. A) anterior face of the heart with the pseudoaneurysm opened, repaired through surgical threads. Note the thin thickness of its wall; B) detail of the suture of bovine pericardium repair on the pseudoaneurysm orifice. That closing remodeled the ventricular cavity and 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 situation<sup>5</sup>. The abovementioned 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 4<sup>th</sup> 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 aneurysm<sup>3</sup>.

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 cases<sup>3</sup>.

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 myocardium<sup>4</sup>.

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<sup>1,3,6</sup>. 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<sup>4,5</sup>. The frequent association of serious mitral regurgitation to the left ventricular posterior wall fake aneurysm is related to high operation mortality rate<sup>7</sup>. However, regardless of the modality of therapy used, the pseudoaneurysm mortality seems to primarily relate to the seriousness of ventricular dysfunction and subjacent ischemic myocardiopathy. 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 antegrograde 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 performance<sup>7</sup>.

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 insufficiency<sup>7</sup>.

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