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Acute myocardial infarction with multiple mechanical complications

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ABSTRACT
The rupture of the inter-ventricular septum is a rare, but extremely severe mechanical complication of the acute myocardial infarction, which in most cases of no immediate surgery is fatal (30 days death rate – 80%). The incidence of the septum ventricular defect post-acute myocardial infarction was 1-3% in the period before crash injury therapy, dropping to 0.2% after introducing the thrombolytic treatment.
The acute mitral failure and the septum ventricular defect are two mechanical complications of the acute myocardial infarction associated with high death rate, regardless of the progress made in medical and surgery therapeutic approach.
This paper highlights the peculiarities of a clinical case like the acute myocardial infarction as the first manifestation of heart disease in a patient with no history of cardiovascular disease but with risk factors (obesity, dyslipidemia, hypertension), the ECG evident changes in the anterior territory and more discrete in the inferior territory, which established the initial diagnosis to be myocardial infarction and the presence of two mechanical threatening complications: ventricular septal flaw and acute mitral failure through posterior papillary muscle incomplete rupture.

Keywords: myocardial infarction, acute mitral failure, mechanical complications, interventricular septum.

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Introduction

The rupture of the inter-ventricular septum is a rare, but extremely severe mechanical complication of the acute myocardial infarction, which in most cases of no immediate surgery is fatal (30 days death rate – 80%)[1]. The incidence of the septum ventricular defect post-acute myocardial infarction was 1-3% in the period before crash injury therapy, dropping to 0.2% after introducing the thrombolytic treatment[2].

Case presentation:

The acute mitral failure and the septum ventricular defect are two mechanical complications of the acute myocardial infarction associated with high death rate, regardless of the progress made in medical and surgery therapeutic approach[3].

In 20% of the cases of acute inferior myocardial infarction complicated with septum ventricular defect (SVD), the SVD is complex and is associated with acute mitral failure through partial or total rupture of the posterior papillary muscles, imposing an immediate and aggressive therapeutic standing[4].

We present the case of a 58 year old patient, with cardiovascular risk factors (obesity, dyslipidemia and moderate hypertension values), without angina

history, who is hospitalized in the cardiology clinic for chest pain, persistent for approximately 5 days and at the moment presents clinical signs of sever pulmonary edema.

Clinically when hospitalized, the patient had a poor general condition, wet teguments and mucous, cold ends, dyspnea at rest, polipeea (respiratory rate 30 breaths / minute), spontaneously SpO2 80%, SpO2 under O2-terapy 94%, dullness and abolished left basal ventricular hum, bilateral cracling bruits, blod pressure 150/90 mmHg, 115/min, rhythmic cardiac noise, holosystolic blast rank IV/VI on the entire cardiac area, with maximum outbreak of the left parasternal auscultation, turgid jugulars, the liver with the lower edge at 2 cm under costal rebord, lack of ankle edemas and lack of neurological outbreak signs.

The electrocardiogram made when hospitalized indicates: sinus tachycardia, 115/min, ax QRS +20 degrees, QS in V1-V4, ST elevation of 0,5 mm in DIII with negative T wave, biphasic T wave, ST depression of 1 mm in DI, a VL.

When hospitalized, blood biochemical examinations showed biological inflammatory syndrome (leukocytes 16410/mmc, fibrinogen 435 mg/dl, CRP 23 mg/l), hyperglycemia (221mg/dl), slight nitrogen retention (creatinine 1,21 mg/dl, urea 67 mg/dl, NTproBNP 11280 pg/ml and troponin 905 pg/ml with CK, CK-MB in normal limits.

Transthoracic echocardiogram highlighted : LV in normal limits, EF VS 40%, posterior inferior wall aneurysm, posterior IVS rupture (12 mm size), with significant left-right hemodynamic shunt; the inferior edge of the SVD was at the insertion of the posterior papillary muscle, which represents incomplete rupture (Image 1); severe ischemic mitral regurgitation with dynamic character (at the beginning of the examination it was of 2nd degree, while at the end of the examination it was of 3rd degree); tricuspid regurgitation of 2nd degree, right ventricle moderate systolic dysfunction, PAPs 50 mmHg.

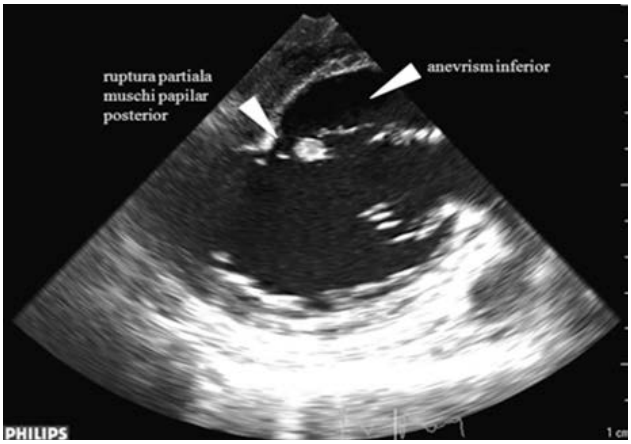

Figure 1 Transthoracic echocardiogram, 2 chambers transgastric section, in the systole: aneurysm in 2/3 basals of the inferior wall, partial rupture of the posterior papillary muscle.

The positive diagnosis in that moment was severe myocardial infarction, with complications in the 5th day of evolution: ventricular septal defect with left-right shunt and severe mitral failure moderated through incomplete rupture of the posterior papillary muscle, acute cardiogenic pulmonary edema.

It was urgently made an coronarography and a left cardiac catheterization which showed bicoronary damages (Figure 2): occlusion LAD 1, stenosis 90% Mg II that load the LAD as far as segment III through collateral circulation, board 40% LAD I, muscular SVD through which the RV is loaded, mitral regurgitation II-III degree, EF LV 40%.

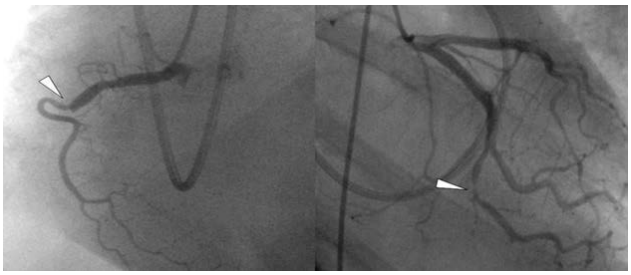

Figure 2 Coronarography: bicoronary damages: LAD I occlusion (left), stenosis 80% Mg 2.

In the presence of an unstable hemodynamic patient with acute myocardial infarction with two mechanic complications urgent surgery was decided.

Surgery treatment of the SVD with pericardial patch was conducted, aortocoronary bypass x2 (venous graft on LAD III and Mg II) and the replacement of the mitral valve with ATS 27 prosthesis.

The post-surgery evolution was slow, burdened by multiple complications:

• low cardiac output syndrome, with increased need of vasopressor support and positive inotropic in the first post-surgery 5 days.

• acute renal failure for which hemodialysis sessions were conducted;

Discussions

In the pre thrombolytic period, most cases of SVD post acute myocardial infarction emerged between the 5th and the 7th day; recent studies highlighted a tendency of decrease of the occurrence time to 2-5 days from acute myocardial infarction start, thus 2 explanation are possible: the precocious diagnostic of the SVD due to the improved echocardiographic technics and increasing access to this imaging technique, the thrombolytic therapy decreases the heart attack scale, but causes small areas of bleeding and the dissection of the myocardial level, fact that can increase the risk of IVS rupture.

The scale of the SVD may vary from few millimeters to few centimeters. Morphologically, the IVS rupture can be simple or complex. The simple IVS rupture is, in general, a small size report, the 2 ends of the continuity solution being at the same level. The extensive bleeding, with irregular path, with necrotic tissue characterizes the complex rupture. In 20% of the cases the IVS rupture comes with acute mitral failure thorough the ischemic damage of the papillary muscles.

The affection of the posteromedial papillary muscle is approximately 3 times more frequent than the one of the anterolateral papillary muscle, due to the different vascularization of the two papillary muscles. Thus, the anterolateral papillary muscle has double irrigation, both from the descending anterior artery level but also from the circumflex artery level, while the posterior papillary muscle is vascularized only from the posterior interventricular artery level, ram being either from the right coronary artery, either from the circumflex artery, depending on the right or left dominance of the coronary pattern. Another particularity of the papillary muscle vascularization is that the central artery is an end artery.

IVS rupture determines the apparition of a left-right shunt, which leads to the flow increase in the pulmonary artery, systemic hypotension, right ventricle systolic dysfunction and systemic venous congestion. Starting with left ventricular systolic function deterioration, the compensatory vasoconstriction phenomena appears, which causes an increase of the systemic vascular resistance and, as a consequence, the grade of the left-right shunt increases. The grade is determined by the IVS rupture dimension, pulmonary and systemic vascular resistance and the relation between them, left and right ventricle function. Most of the studies showed that in 50% of the cases, most of the patients had angiography vessel damage, with total occlusion of the incriminated vessel. Collateral circulation was poor represented, which sustains the hypothesis that the collateral circulation reduces the risk of ventricle free wall rupture or of IVS. Our patient showed bicoronary lesion, with damage of the two possible arterial sources of the posterior papillary muscle, the right coronary artery and circumflex artery. In the absence of reperfusion, the IVS rupture usually appears in the first week post acute myocardial infarction. The spontaneous closure of the SVD is extremely rare.

The clinical manifestations usually depend on the septal defect`s size and myocardial infarction extension. The symptoms associated with the post myocardial infarction SVD include: chest pain, dyspnea, low cardiac output syndrome and shock (alterated state of consciousness, cold extremities, cyanotic, cutaneous vasoconstriction, anuria). Objective, you hear a rough holosystolic souffle, intense, left parasternal, with irradiation towards the apex, base and right parasternal, and at palpation a left parasternal murmur on approximately half of the patients.

At the patients in cardiogenic shock, due to the

decrease of the left-right shunt, the murmur is missing and the shunt has reduced intensity.

The echocardiography is useful for determining the position, size and relations of the septal flow with the nearby anatomic structures, the left and right ventricle functions and estimate the pulmonary artery pressure. The sensitivity and the specificity of the Doppler color echocardiography was reported to be 100%. The coronarography was urgently performed because the patient presented changes in ECG in two different areas.

The treatment recommended to such patients consists in:

1.Medical treatment which has the purpose to lowering the filling pressure of the left ventricle

• positive inotropic and vasopressor medication (left-right shunt increases → pulmonary artery flow increased right ventricular dysfunction);

• vasodilators (side effects: hypotension, reversal of the shunt → arterial desaturation marked);

• loop diuretic intravenous administrated in continuous infusion;

• additional intake of O2: O2 administrated through nasal mask, CPAP,

• dialysis for oligoanuric patients;

2. Surgery treatment - Surgical correction is indicated for the patients with IVS rupture after acute myocardial infarction. At the moment there is no agreement regarding the best surgery moment. There are studies which showed that the patients who have had surgery in the first 2 days since the diagnostic, had a more difficult post-surgery evolution compared with these after. A possible explanation could be that this patients with early surgery had more advanced stages of cardiac failure with an artery pressure significantly smaller. Another explanation could be that necrotic myocardium tissue is fragile, so in terms of surgery technic, the best moment for performing the surgery is after the emergence of the fibrosis in the infarction area. Histological studies showed that the proliferation of connective tissue in the necrosis area is present only in week 3 post-acute myocardial infarction. In most patients, postponing the surgery so long is not possible because they develop severe cardiac failure with multiple organ dysfunctions.

The negative prognostic factors for the patients with IVS rupture after myocardial infarction: cardiogenic shock, right ventricular dysfunction, age> 75 years, history of hypertension, heart rate, Killip class, diabetes, acute renal failure.

The occurrence of residual or recurrent rupture shunt was reported at 24% of patients with surgical correction, and is associated with high mortality. At the patients with small residual shunt, asymptomatic, the medical therapy is the first aim, while in those with clinical evidence of cardiac failure or pulmonary-systemic shunt fraction greater than 2.0 follow-up is indicated.

The particularities of the case presented

• acute myocardial infarction as the first manifestation of heart disease in a patient with no history of cardiovascular disease but with risk factors (obesity, dyslipidemia, hypertension);

• ECG evident changes in the anterior territory and more discrete in the inferior territory, which established the initial diagnosis to be myocardial infarction;

• the presence of two mechanical threatening complications: ventricular septal flaw and acute mitral failure through posterior papillary muscle incomplete rupture.

• the complexity of the surgery (fixing the IVS with pericardium patch, bypass and mitral valve prosthesis) urgently performed at a patient hemodynamic unstable.

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