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CASE REPORTS

Healing a Broken Heart: a Case Report of Left Ventricular Free Wall Rupture and Review of the Literature

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ABSTRACT

The occurrence of left ventricular free wall rupture in acute myocardial infarction decreased with the extent of interventional procedures of reperfusion, but it is still encountered in 1-2% of these patients. We are presenting the case of a 58 years old male with left ventricular free wall rupture occurred as a late complication of an inferior-lateral ST-elevated myocardial infarction. The aim of this case report is to underline the main clinical features, the diagnostic value of the echocardiographic exam and the importance of early surgical intervention in a rare, but very dangerous condition, with persistent high mortality rates. In this context, we will review the current prevalence, clinical forms and prediction factors of left ventricular free wall rupture.

Keywords: Free wall rupture of the left ventricle, acute myocardial infarction, cardiogenic shock

INTRODUCTION

It has been reported that the occurrence of left ventricular free wall rupture (LVFWR) decreased with the extension of interventional procedures of reperfusion (1, 2). However, it is still encountered in less than 2% of cases with ST-elevated acute myocardial infarction (3-7), even after urgent reperfusion therapy (8-11), and remains associated with high mortality rates (3,6). With this case report we aim to remind the importance of early recognition and prompt surgical intervention in this life-threatening condition. ❑

CASE REPORT

A 58-year-old male was admitted to the hospital with intense chest pain, collapse, and diaphoresis, one month after a prolonged episode of angina for which he did not required medical assistance. He had a history of smoking with a total load of 57 pack-years, known with untreated type 2 diabetes mellitus and dyslipidemia, symptomatic for peripheral arterial disease in the last two years. ECG showed Q waves in DII, DIII, aVF with 1 mm ST-segment elevation and negative T waves in DII, DIII, aVF, V5 and V6 leads. High levels of myocardial necrosis biomarkers

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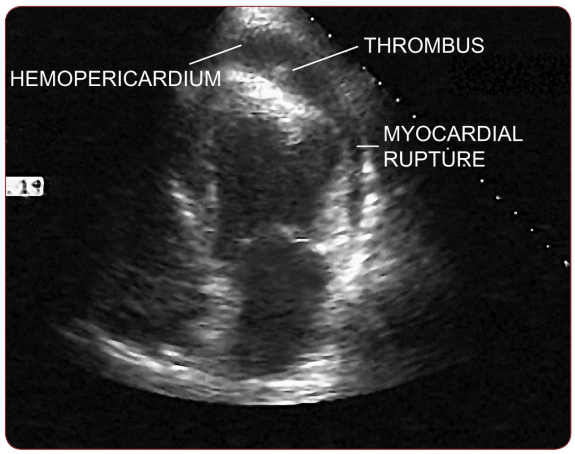

FIGURE 1. Fissured pseudoaneurysm of the inferior-lateral wall with hemopericardium and intrapericardial thrombus

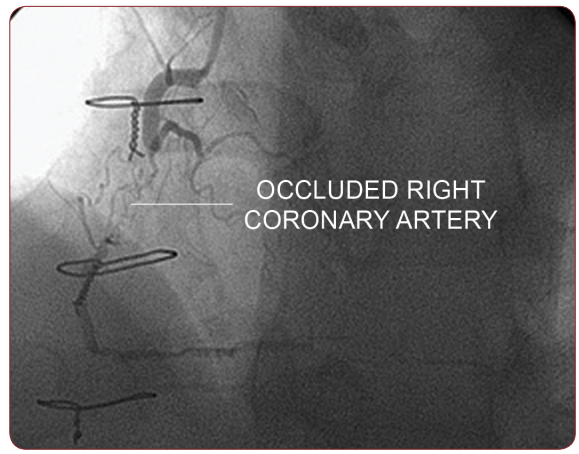

FIGURE 2. Occlusion of the right coronary artery revealed at an angiography performed three month after left ventricular free wall rupture repair

(TnI and CK-MB) have been recorded in the emergency unit. Since the patient was unstable, an echocardiographic study was immediately performed, showing a fissured pseudo-aneurysm of the inferior-lateral wall, with hemopericardium and intrapericardial thrombus (Figure 1). The maximum width of the pericardial effusion was 30 mm.

The coronary angiogram was postponed in the context of surgical emergency. The patient was transferred in the department of cardiovascular surgery for the intervention of LVFWR repair. The pericardium was opened via a median sternotomy. A significant amount of clot and blood was removed. The posterior left ventricular fissured pseudoaneurysm was confirmed and carefully dissected only after the heart was discharged on cardiopulmonary bypass. The pseudoaneurysm was partially adherent to the diaphragm and it should be considered that this feature is the main reason for the avoidance of a "blow-out" rupture with sudden death. The heart was arrested with ante-grade cold blood cardioplegia. A composite Dacron with autologous pericardium patch was used for LVFWR repair. The geometry of the left ventricle was restored. The postoperative evolution was free of side effects and the patient was discharged after eight days of hospitalization.

The patient stopped smoking, started to keep appropriate diet, and followed a pharmacological treatment with a beta-blocker, an ACE-inhibitor, a statin, aspirin, and oral anti-diabetics.

Three month later a coronary and peripheral arteries angiography were performed indicating

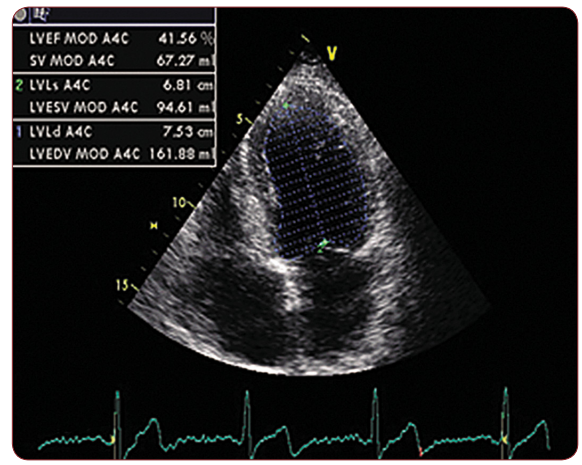

FIGURE 3. Echocardiographic evaluation of left ventricular ejection fraction at eight years after the surgical cure of the free wall rupture

one-vessel heart disease with occlusion of the right coronary artery (Figure 2) and a segmental occlusion of the right common iliac artery. Decision was to not proceed to any further interventions.

After eight years the patient is free of cardiac symptoms, with a residual mild left ventricular systolic dysfunction (Figure 3). ❑

DISCUSSION

It is considered that the first report about myocardial rupture was recorded by William Harvey in 1649. Richard Quain is the one who correlated this condition with the obstruction of the correspondent coronary artery (12).

Evolution of LVFWR. Usually LVFWR occurs between 24 hours to one week from the acute coronary event (13). In one of the largest report-

ed randomized trials in which primary percutaneous coronary intervention (PCI) was the reperfusion therapy mechanical complications occurred at a median of 23.5 hours after symptoms onset (6). In the presented case we have considered the LVFWR as a late complication of a previous ignored myocardial necrosis which evolved with inferior-lateral pseudoaneurysm that finally cracked and generated pericardial tamponade. This scenario is corresponding to type III free wall rupture in the classification of Becker and van Mantgem, the so-called "concealed rupture" associated with an older and severely expanded infarct. The other two forms of LVFWR are the abrupt fissure (type I) - occurring usually early after infarction and the erosion in the area of necrosis (type II) (14,15). We have initially diagnosed the thin, dilated and fissured segment of the left ventricular wall as a pseudoaneurysm, due to its topography on the lateral wall and evolution to rupture – features which are not characteristic for true aneurysms (16,17). The patologic exam has confirmed this assumption.

Risk factors of LVFWR. Many conditions have been evaluated in relation with free wall rupture. There are consistent data indicating that primary PCI in acute myocardial infarction is the most important protective factor against cardiac rupture (1,2,4,7). In their series of 5699 patients with acute myocardial infarction Honda et al. have analyzed the clinical and pathological trends of cardiac rupture on three cohorts characteristic for the following periods of time: 1977-1989, 1990-2001 and 2001-2011. The incidence of this complication decreased concomitantly with the widespread application of reperfusion therapy, from 3.3% in the first cohort to 1.7% in the last one (2). Recently, similar conclusions have been revealed by Chang RY et al. In their study, conducted between 1999 and 2013, they have reported that LVFWR was significantly lower in patients with ST-elevated myocardial infarction submitted to primary PCI than in patients who have received thrombolysis or pharmacologic treatment alone (7).

There is a large agreement that advanced age (>70 years old) or female sex are predictors of risk for LVFWR (2-4,6). Other factors have been associated with the risk of LVFWR in different studies: first myocardial infarction (2,18), Killip class ≥ 2 at presentation (4,6-7), initial high levels of myocardial biomarkers (3,6) or poor coronary collateralization (18). In the reperfusion era it looks that, very probably, arterial hypertension has no longer a significant association with the appearance of LVFWR (2).

In our case report we should consider that the lack of early revascularization, due to a neglected acute coronary syndrome, was the main contributing factor to LVFWR.

Topography of LVFWR. In the presented case the localization of LVFWR was on the inferior and lateral walls. Data from early studies, reconfirmed by large series in the reperfusion era, have indicated anterior wall as the most common topography of LVFWR (2). In the last years some authors have emphasized the involvement of other segments as well, that can reach 28% of cases for the lateral wall (5).

Mechanisms of LVFWR. In our patient LVFWR has occurred in the context of one-vessel coronary artery disease. However, it has been already shown that 80% of the patients with left ventricular free wall rupture have 3-vessels coronary artery disease (19).

The pathologic substrate of LVFWR consists in intramyocardial hemorrhage and microvascular obstruction (11). Beyond ischemia, the occurrence of these lesions can be influenced by two other factors: the quality of the collateral circulation (20) and the balance between extracellular matrix and the expression of metalloproteinases (MMPs) (15). Accordingly, the sex influence on myocardial rupture could be explained by a less developed collaterals in women than in men (21), while the age risk could derive from an inflammatory status prone to the activation of MMPs (15). It is considered that all these conditions are contributing to the decrease in tensile strength of the infarcted myocardium.

Diagnostic work-up. In the presented case the diagnosis of myocardial rupture was realized promptly based on echocardiographic examination, for which was reported a 100% sensibility and a 93% specificity for the detection of this complication (22). The amount of pericardial effusion was responsible for collapse. In a study conducted by Figueras et al. the mean SD of pericardial width of 22.2 (4.7) mm has been associated with a higher probability of hemodynamic instability *versus* 16.3 (5.7) mm (23).

Prognostic of patients with LVFWR. LVFWR is still associated with high mortality rates (50-60%) (2,6), accounting for 95% of electromechanical

dissociation in patients with acute myocardial infarction (24). Prompt surgical intervention can be life-saving, ensuring survival for a long period of time, as it was emphasized in our report. ❑

CONCLUSION

The incidence of LVFWR has decreased with the widespread of reperfusion therapy in acute myocardial infarction. However, it can be still encountered, even after primary PCI, but more probably in patients who do not benefit from urgent interventional procedures of reperfusion. LVFWR occurs usually in the first 24 hours after acute myocardial infarction, in patients with advanced age, more frequently in females and on the anterior wall of the left ventricle, in the context of three-vessel coronary artery disease. We aimed to increase awareness for this life-threatening condition even in less expected situations, like that of the presented patient: male, younger than 70 years old, with one-vessel coronary artery disease and with the rupture localised in the inferior and lateral wall, evolving very late after the onset of the acute episode of myocardial ischemia and in the absence of primary reperfusion. The other two reasons for which we have reported this case were to remind the echocardiographic features of "concealed rupture" with fissured pseudoaneurysm, less illustrated in the last years than the acute forms of LVFWR (the abrupt fissure or the erosion) and to underline the importance of prompt surgical intervention for the survival of patients with LVFWR. ❑

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