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# Left ventricular free wall rupture after myocardial infarction: a case report with rare live diagnostic record

Ruptura de parede livre do ventrículo esquerdo pós-infarto agudo do miocárdio: relato de caso com raro registro diagnóstico em vida

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**ABSTRACT** – Left ventricular free wall rupture is one of the most catastrophic and lethal mechanical complications associated with myocardial infarction. Its diagnosis in a patient still alive is rare, and its only therapeutic option is open surgical treatment. We report the case of an 82-year-old male patient who presented with an acute ST-segment elevation myocardial infarction, within more than 24 hours of onset, in which it was possible to record, in real time, the rupture of the left ventricular free wall by means of ventriculography.

**Keywords:** Heart rupture, post-infarction; Myocardial infarction; Cardiac tamponade; Heart diseases/diagnostic imaging

**RESUMO** – A ruptura da parede livre do ventrículo esquerdo é uma das complicações mecânicas mais catastróficas e letais associadas ao infarto agudo do miocárdio. Seu diagnóstico no paciente ainda vivo é raro, e a única saída terapêutica é o tratamento cirúrgico aberto. Relata-se o caso de um idoso, de 82 anos, que apresentou um infarto agudo do miocárdio com supradesnivelamento do segmento ST, com mais de 24 horas de evolução, no qual foi possível registrar, em tempo real, a ruptura da parede livre do ventrículo esquerdo, por meio da ventriculografia.

**Descritores:** Ruptura cardíaca pós-infarto; Infarto do miocárdio; Tamponamento cardíaco; Cardiopatias/diagnóstico por imagem

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## INTRODUCTION

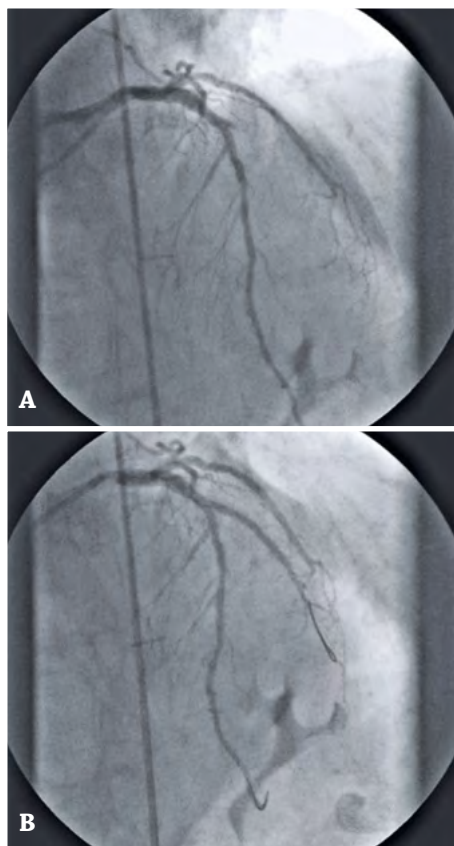
Left ventricular free wall rupture (LVFWR) is one of the most catastrophic and lethal mechanical complications associated with myocardial infarction (MI). Rupture occurs due to weakness of the heart wall caused by tissue necrosis, and its form of occurrence varies according to the time elapsed after the onset of MI. In early cases (less than 72 hours), there is a predominance of abrupt total wall rupture, while in late cases (more than 4 days), the occurrence of dilation of the infarcted area is observed.<sup>1</sup>

Left ventricular free wall rupture can be divided into three distinct categories: acute, subacute, and chronic. The acute rupture is characterized by sudden chest pain, electromechanical dissociation, shock, and death, within minutes due to massive hemorrhage in the pericardial sac. This is the most severe form, with a mortality rate of 98%.<sup>2</sup> The subacute rupture is characterized by a lesion of smaller proportion, which initially may be self-limited, with subsequent reabsorption. It presents with manifestations of cardiac tamponade and cardiogenic shock, mimicking other complications of MI. Chronic rupture, on the other hand, with pseudoaneurysm formation, occurs due to expansion mechanisms of the infarcted area, with slow and constant blood exchange between the ventricle and a limited space in the pericardium.<sup>1</sup>

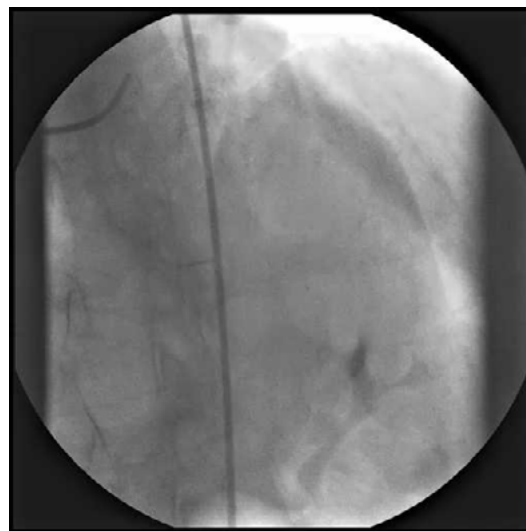
The objective of this study was to report the case of an 82-year-old male patient, who presented with ST-segment elevation MI within more than 24 hours of onset, in which it was possible to record, in real time, the rupture of the left ventricular free wall using ventriculography. This study was approved by the Research Ethics Committee of the *Faculdade de Medicina de Barbacena* (protocol 4.770.487, CAAE 47965421.0.0000.8307).

## CASE REPORT

An 82-year-old male hypertensive patient was admitted to the emergency department with signs and symptoms suggestive of MI. The electrocardiogram (ECG) confirmed the diagnosis of large anterior wall ST-segment elevation MI, with more than 24 hours of onset of angina. Emergency coronary angiography was requested, and during the procedure, a severe lesion in the left anterior descending artery, and acute occlusion in the diagonal artery were demonstrated. Primary percutaneous coronary intervention with bare metal stent implantation (Figure 1 and Videos 1 to 3) was performed in both arteries with no intercurrent events.



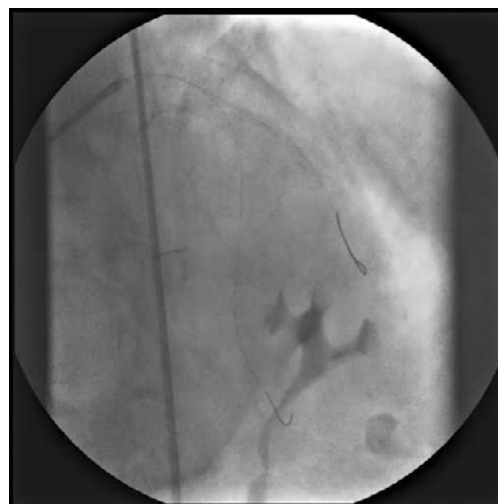
**Figure 1.** Left coronary angiography. (A) First large diagonal branch occluded at the origin, and severe lesion in the middle portion of the left anterior descending coronary artery. (B) Patent diagonal branch after primary percutaneous coronary intervention with stent implantation.



**Video 1.** Left coronary angiography showing occluded diagonal branch.



**Video 2.** Ventriculography with still intact wall performed during diagnostic catheterization, before angioplasty and before free wall rupture.



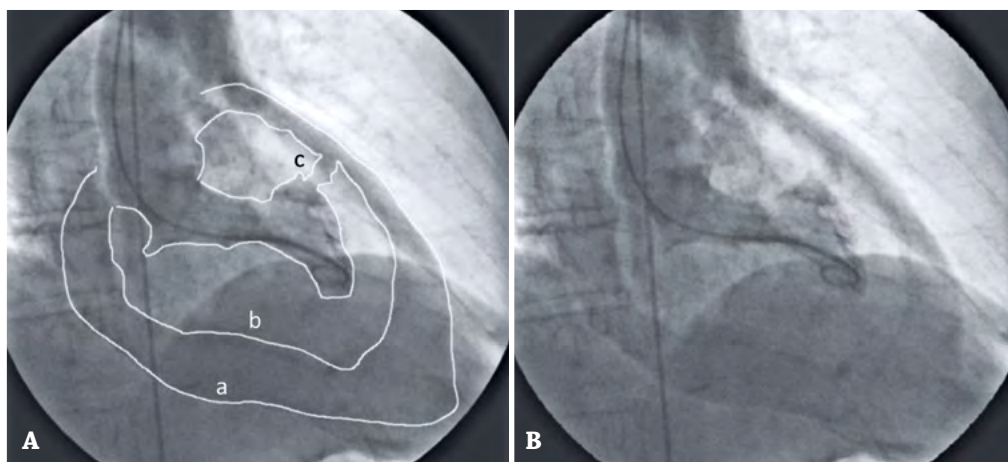
**Video 3.** Left coronary angiography showing a patent diagonal branch after primary angioplasty with stenting of this vessel.

Thirty minutes after the end of the procedure, the patient progressed, still in the catheterization laboratory, with abrupt cardiogenic shock accompanied by jugular vein engorgement, and decreased level of consciousness. Emergency treatment was initiated, with administration of noradrenaline, with no response. It was then decided to return the patient to the cathlab, considering the possibility of coronary dissection/perforation or acute stent thrombosis. Initial fluoroscopy already suggested an image of pericardial effusion (Video 4). The coronary arteries were evaluated with contrast injection and no arterial or implanted stent changes were evident.

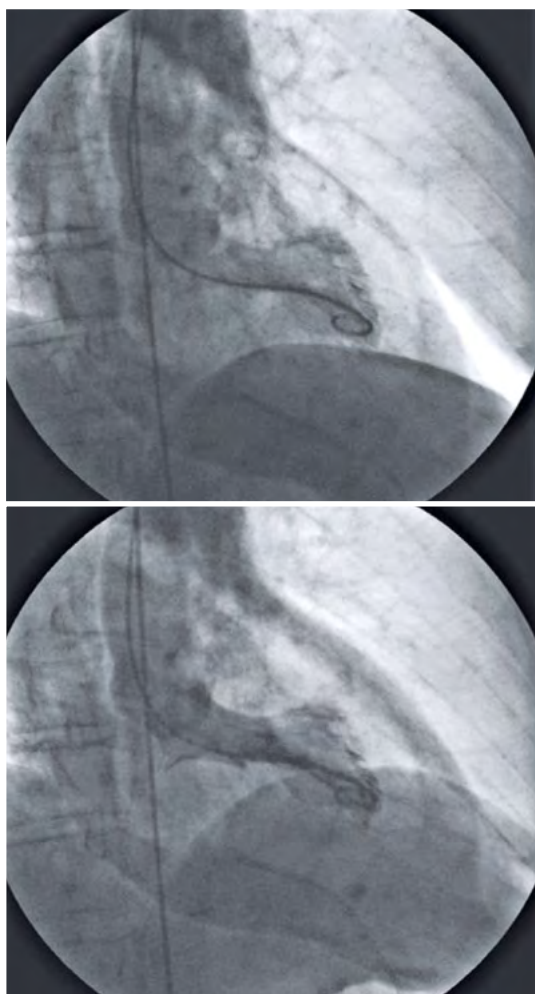
After ruling out coronary complications, a left ventriculography was performed, which showed LVFWR (Figures 2 and 3, and Videos 5 and 6). For hemodynamic improvement, we chose to perform pericardiocentesis, followed by passage of monolumen 14 (Video 4), and, when removing the effusion volume, the patient presented with some momentary improvement, but the blood quickly formed a new collection, leading to a new tamponade and clinical instability. In total, approximately 2L of blood were withdrawn, requiring replacement of packed red blood cells. Given the severity of the condition, the cardiac surgery team was called, but there was no time for open surgical intervention, since the patient rapidly progressed to electrical instability and death.



**Video 4.** Right oblique view showing voluminous pericardial effusion, and catheter already positioned in the pericardial space, through thoracic percutaneous puncture, to drain the blood.



**Figure 2.** Left ventriculography with left ventricular free wall rupture. (A) Pericardial sac with voluminous effusion (a); myocardium (b) and ventricular rupture (c). (B) Same image, with no delineation.



**Figure 3.** Left ventriculography in systole with presence of free wall rupture and cardiac tamponade.



**Video 5.** Left ventriculography after free wall rupture.



**Video 6.** Left ventriculography showing contrast leakage from the ruptured wall.

## DISCUSSION

The acute form of LVFWR, as in the reported case, initially presents with signs of rapid cardiac tamponade, with muffled heart sounds, jugular engorgement, and paradoxical pulse; it may even manifest as sudden death.<sup>3</sup> The most reliable clinical sign of LVFWR is electromechanical dissociation, which has 97.6% accuracy.<sup>4</sup> It is one of the most dramatic, severe, and rare complications of MI today. Its prevalence has been greatly influenced by myocardial reperfusion therapies, going from values between 6% and 8% in the 1970s and 1980s to a rate of less than 1% today.<sup>5,6</sup> Early diagnosis is essential for a better clinical outcome, but this does not always guarantee a favorable outcome, since open surgical treatment must be performed in a hemodynamically unstable patient.

Left ventricular free wall rupture can occur in any wall of the heart; however, it is more frequent in the anterior wall, besides being more common after occlusion of the left main coronary artery or proximal occlusions of the left anterior descending artery, with extensive infarctions.<sup>3</sup> These data are consistent with the report of this case. Although it is a rare complication, one should always be aware of its risk factors, such as no past history of angina or MI, ST-segment elevation, or presence of Q waves early in the electrocardiogram, plus a creatine kinase MB (CK-MB) peak above 150IU/L. Other factors, such as MI with a large area of tissue necrosis, age over 70 years, female sex, heart valve disease, chronic kidney disease, and white race, in some studies, have been shown to increase the prevalence of this condition.<sup>7,8</sup>

The first measure to be adopted in suspected LVFWR is pericardiocentesis, to reduce the repercussions of cardiac tamponade, aiming at hemodynamic stabilization. The definitive treatment is essentially based on emergency



open surgery, a measure that, despite high mortality, is the modality with the greatest possibility of saving patients in this scenario. In some selected cases, in whom hemodynamic stability is achieved after pericardiocentesis, and bleeding is controlled, clinical measures can be adopted, but surgical therapy is still necessary as early as possible.<sup>1</sup> As shown in the clinical case, even with early diagnosis, the progression of the condition is rapid, and there is no guarantee that there will be a timely surgical intervention. It is also worth remembering that many patients die suddenly, still undiagnosed, with LVFWR being discovered only during necropsy.

Since it is an infrequent and highly severe mechanical complication, imaging registration of LVFWR is quite rare, and there is no time available to perform any imaging method after onset of the complication. The rapid progression to death and the difficulty of early diagnosis mean that this type of complication is reported in the literature, in most cases through autopsies. The report in this article is important, not only because of the rarity of LVFWR today, but also because early diagnosis does not guarantee a favorable outcome, and there are no effective ways to predict it. It can also be inferred, even with the lack of robust evidence, that delayed diagnosis and treatment of MI, especially in cases of extensive involvement of the myocardial wall, are factors that may be related to increased risk of occurrence of this severe event.

#### SOURCE OF FINANCING

None.

#### DECLARATION OF CONFLICTS OF INTEREST

The authors declare having no conflicts of interest.

#### CONTRIBUTION OF AUTHORS

Conception and design of the study: FDR and HABC;

data collection: FDR, GCS, LMBF and LPBD; data interpretation: FDR, GCS, LMBF and LPBD; text writing: FDR, GCS, LMBF and LPBD; approval of the final version to be published: FDR and HABC.

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