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Left Ventricle Free Wall Rupture in a Young Lady with No Cardiovascular Risk Factors

Ilaria Battistoni, MD*; Michela Brambatti, MD; Marco Marini, MD; Luca Angelini, MD; Matteo Francioni, MD; Mauro Borioni, MD; Stefano Moretti, MD; GianPiero Perna, MD

***Ilaria Battistoni, MD**

Cardiology division, Ospedali Riuniti di Ancona, Via Conca 71, 60100Ancona, Italy.

Tel: +39-071-5965018, Fax: +39-071-5965620; Email: i.battistoni@alice.it

Abstract

Left ventricle free wall rupture (LVFWR) is one of the most catastrophic complication after acute myocardial infarction. The LVFWR is often fatal and difficult to recognize in its subacute variant. We present the case of a 42-years-old woman, with no cardiovascular risk factors, who experienced a syncopal event during sexual intercourse. She reported an episode of vomiting digested food accompanied with epigastric pain seven days before. On arrival, her abdominal symptoms were predominant. No clinically significant laboratory data were found. An intensive diagnostic work-up including thoracic and abdominal CT scan did not reveal the cause of the abdominal pain. A transthoracic echocardiogram revealed a a small circumferential pericardial effusion with no signs of cardiac tamponade. For a better understanding of the pericardial effusion, we decided to perform a transesophageal echocardiogram. Echoes were suggestive of hemopericardium which was confirmed by an exploratory pericardiocentesis. Urgent median sternotomy revealed left ventricular posterolateral free wall rupture partially covered by a thrombus. A pericardial patch was sutured and glued in place over the tear. This case demonstrates that LVFWR is not always fatal and prompt recognition of the variety of its presentation may be life-saving.

Keywords

Left ventricle rupture; Myocardial infarction; Diagnosis; Cardiac surgery

Introduction

Left ventricle free wall rupture (LVFWR) is one of the most catastrophic complication after acute myocardial infarction. LVFWR is characterized by rapid development of mechanical arrest with cardiac tamponade being the leading cause of death. Early identification of clinical deterioration is generally possible; however, some patients develop a subacute variant of LVFWR which is difficult to recognize.

Case Report

A 42-years-old woman with no cardiovascular risk factors presented after a syncopal event occurred during sexual intercourse preceded by thoracic and epigastric pain. She reported an episode of vomiting digested food accompanied with epigastric pain seven days before. On arrival, the patient was lethargic. Her systolic blood pressure was 80 mmHg, heart rate 109 beats/min, oxygen saturation 93% on

on oxygen flow of 6 l/min. Electrocardiogram (ECG) showed sinus tachycardia (Figure 1A). Chest radiograph showed diffuse pulmonary congestion. Laboratory tests revealed slight anemia and raised cardiac troponin I (cTnI) value (0.82 ng/mL; normal < 0.08 ng/mL) with normal serum creatine kinase MB (CK-MB) levels. On echocardiography we saw a small circumferential pericardial effusion with diastolic right atrium collapse, a poorly collapsing inferior vena cava during inspiration with no others abnormalities**.** After administration of one liter of colloid and noradrenaline infusion, blood pressure raised to 150/70 mmHg and a normal state of consciousness was restored.

Due to the abdominal pain persistence, a thoracic and abdominal computed tomography (CT) scan was obtained which revealed the presence of endoabdominal fluid surrounding the urinary bladder with gas bubbles within the anterior-superior mediastinum and mild pericardial effusion (Figure 2A). A gastroscopy ruled out signs of perforation. Two hours later a new episode of epigastric pain with hypotension occurred. A second abdominal CT scan revealed increased endoabdominal free fluid and laboratory test showed metabolic acidosis, increased total serum bilirubin, elevated transaminases with slight raise of cTnI (1,06 ng/mL). The patient was intubated and an exploratory laparoscopy ruled out organic lesions. For a better understanding of the pericardial effusion, we performed a transesophageal echocardiogram. Echoes were suggestive of hemopericardium (Figure 1C) which was confirmed by an exploratory pericardiocentesis. Urgent median sternotomy revealed (Figure 2B) left ventricular posterolateral free wall rupture at the portion between the first diagonal and the marginal branch, partially covered by a thrombus (Figure 2C). A pericardial patch was sutured and glued in place over the tear. Coronary angiography showed myocardial bridging to the mid left anterior descending artery, a subcritical stenosis of the marginal branch and a moderate stenosis of circumflex (Figura 2A). Further investigations revealed a homozygous MTHFR C677T mutation. Ten days after admission, the patient fully recovered and was discharged on antithrombotic (100 mg acetyl salicylic acid/day), betablocker (bisoprolol) and statin therapy.

Discussion

LVFWR is one of the most feared complications of myocardial infarction and its overall incidence was approximately 2-4% in the pre-reperfusion era [1,2]. Although the incidence declined progressively over the time in conjunction with a better use of anti-ischemic treatment (betablockers, angiotensin-converting enzyme inhibitors and aspirin), reperfusion therapies, and better control of blood pressure, LVFWR still accounts for up to 10% of mortality associated with myocardial infarction [2,3]. Several risk factors for cardiac rupture have been identified such as older age, female sex, first myocardial infarction, anterior infarction, no history of angina, large transmural infarction and history of hypertension [3,4]. At the present time no genetic cause has been demonstrated. Cardiac tamponade and subsequently hemodynamic collapse are the most catastrophic consequences of cardiac rupture [4].

However, sometimes the clinical scenario is not fulminant and the diagnosis can be challenging as in our clinical case. According to the clinical presentation, LVFWR has been classified into two different forms: acute and subacute [5,6].The acute form generally leads to a sudden hemodynamic collapse from hemopericardium followed quickly by electromecanical dissociation and death. A subacute variant of LVFWR generally occurs when organized thrombus and the pericardium close the ventricular perforation. This circumstance can evolve to frank rupture with cardiac tamponade, to the development

of a false aneurysm communicating with the left ventricle or to formation of a left ventricular diverticulum [6-8]. The subacute form has been increasingly recognised and may account for up to 30% of all cases of in-hospital free wall rupture [4,9]. Unlike patients with acute LVFWR, those with the subacute variant have higher chances of survival until emergency surgery is performed [6]. Our case describes a very challenging example of subacute LVFWR. This patient almost certainly had a transmural myiocardial infarction 7 days before her admission to the ED that was unrecognized due to several factors such as young age, no cardiovascular risk factors, prominence of abdominal symptoms, and non clinically significant laboratory and instrumental data. In light of these data, one could speculate that the MTHFR C677T homozygous mutation may have played a role in the patient's myocardial infarction. However, previous findings demonstrate that the MTHFR C677T homozygous mutation without hyperhomocysteinemia has no clinical meaning [10].

Echocardiography usually represents the first-line diagnostic tool for suspected myocardial rupture with the advantage to be rapid and non invasive [11]. The main echocardiographic findings in patients with LVFWR are pericardial effusion and intra-pericardial echoes; these features, in our case, provided important clues for suspecting hemopericardium and perform the subsequent pericardiocentis [4]. We finally established definitive diagnosis of subacute LVFWR after an acute infero-posteral myocardial infarction on the basis of an exploratory sternotomy that brought us to perform heart surgery with subsequent restore of cardiac tissue integrity and prompt patient's recovery.

Conclusion

Subacute LVFWR is a rare mechanical complication of myocardial infarction which may be easily misdiagnosed. Since surgical correction can be life-saving, a high index of suspicion is warranted.

Figures

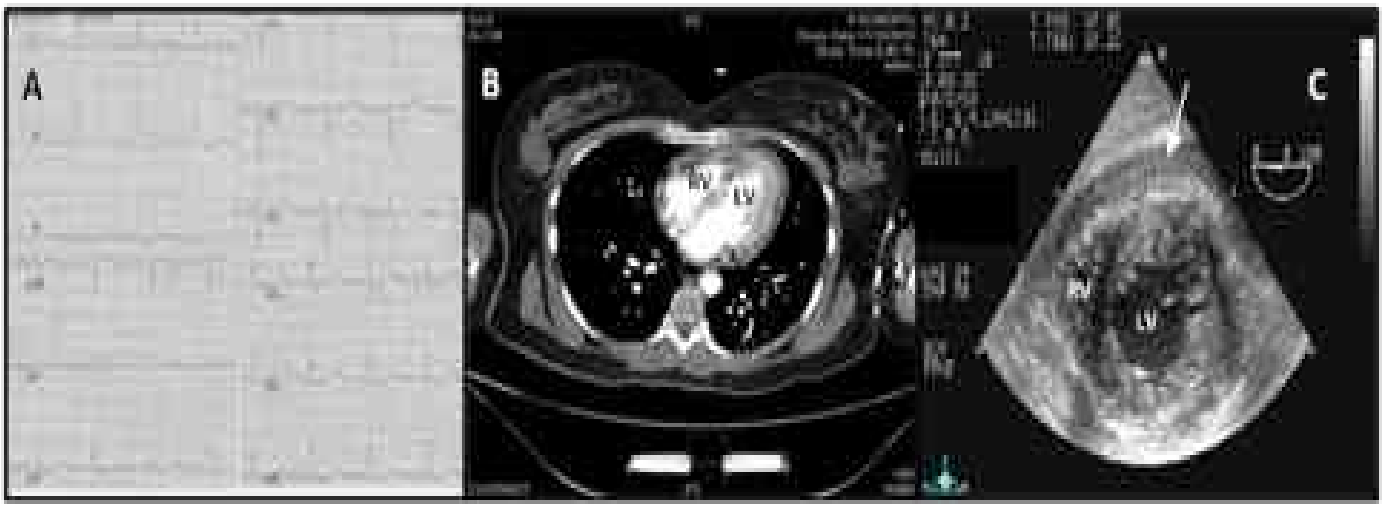

Figure 1: Panel A: Initial 12 lead ECG on admission. Panel B: Mild circumferential pericardial effusion on CT scan. Panel C: Transesophageal echocardiogram (transgastric view) showing circumferential pericardial effusion with echogenic material consistent with hemopericardium. LV-left ventricle, RV-right ventricle.

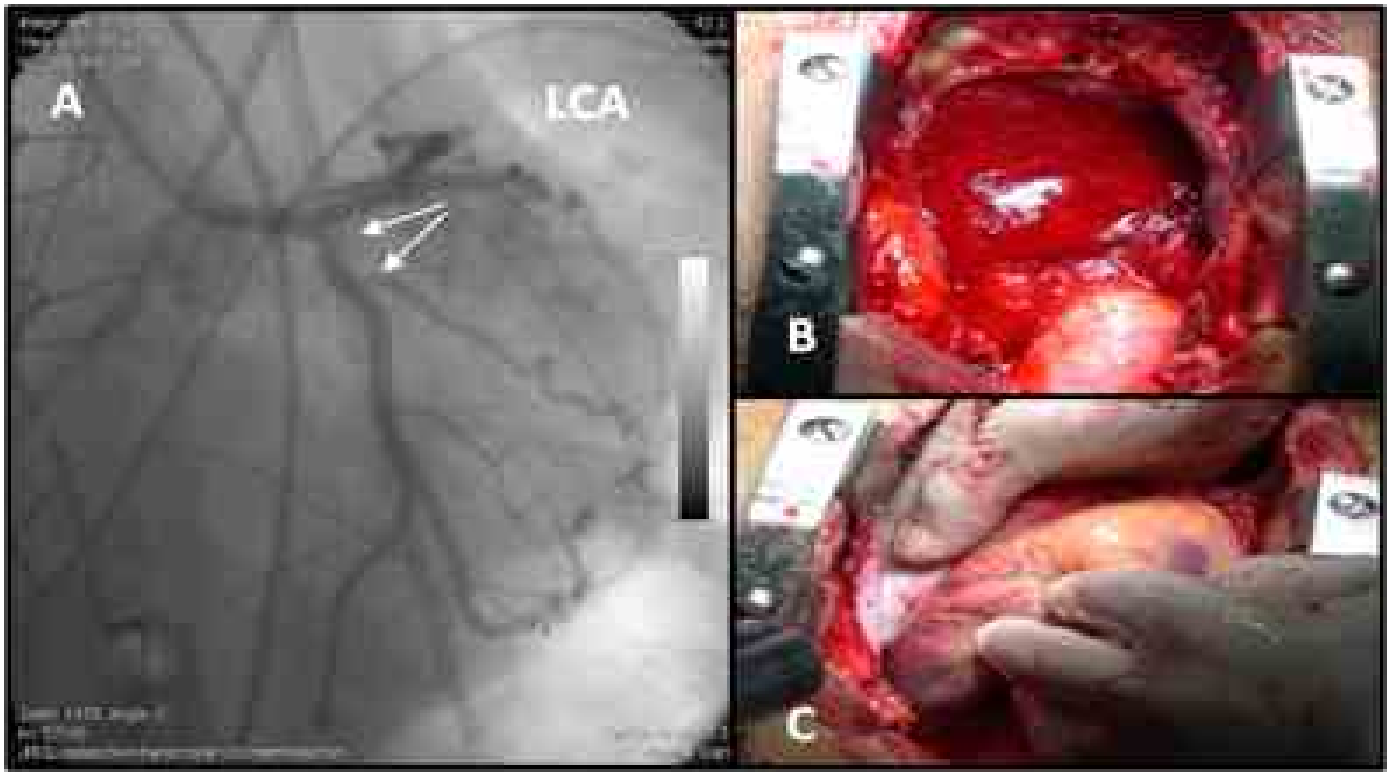

Figure 2: Panel A: Coronary angiogram showing moderate stenosis of circumflex coronary artery and subcritical stenosis of marginal branch. LCA, left coronary artery. Panel B, Panel C: Heart examination after median sternotomy showed hemopericardium (Panel B) and postero-lateral rupture (Panel C).

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Authors Information: Ilaria Battistoni, MD[*1]; Michela Brambatti, MD[1]; Marco Marini, MD[1]; Luca Angelini, MD[1]; Matteo Francioni, MD[1]; Mauro Borioni, MD[2]; Stefano Moretti, MD[1]; GianPiero Perna, MD[1]
[1]Cardiology Division, 'Ospedali Riuniti di Ancona', Ancona, Italy
[2]Division of Cardiac Surgery, 'Ospedali Riuniti di Ancona', Ancona, Italy

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