REVIEW ARTICLE

Left Ventricular Free Wall Rupture After Acute Myocardial Infarction

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SUMMARY

Thrombolytic therapy and primary angioplasty have modified the management, evolution and prognosis of acute myocardial infarction; however, mortality from left ventricular free-wall rupture still remains extremely high.
The occurrence of this complication is sudden and catastrophic in most patients, and is characterized by cardiac tamponade, electromechanical dissociation and immediate death; however, approximately one third of patients present subacute cardiac rupture with sustained hypotension and pericardial effusion of diverse sizes that allow the implementation of therapeutic measures as a bridge to surgery with repair of the myocardial rupture.
In this paper, we provide an update on the clinical and echocardiographic features of patients with left ventricular free-wall rupture complicating an acute myocardial infarction in order to highlight the key diagnostic points and increase the clinical suspect of a severe condition that is not always fatal.

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Key words > Myocardial Infarction - Heart Rupture - Heart Ventricles

BACKGROUND

Although cardiac rupture is a rare complication of acute myocardial infarction with an overall incidence of about 6.2%, it represents the second cause of death after cardiogenic shock, and accounts for as much as 15% of in-hospital mortality. (1-3) Most patients with cardiac rupture may succumb almost instantaneously due to cardiac tamponade with rapid, irreversible, electromechanical dissociation; however, approximately 30% of patients present subacute cardiac rupture, survive several hours after the event, allowing the implementation of therapeutic measures and imaging tests to confirm the diagnosis. (4-6)
An appropriate clinical and echocardiographic evaluation performed early may identify this subgroup of patients that require rapid hemodynamic stabilization as a bridge to surgery with repair of the myocardial rupture. (7, 8)

In this paper, we provide an update on the clinical and echocardiographic features of patients with left ventricular free wall rupture in order to highlight the key diagnostic points and increase the clinical suspicion of a severe condition that is not always fatal.

EPIDEMIOLOGY

Risk factors

The following risk factors either predispose or increase the risk of cardiac rupture complicating an acute myocardial infarction: female gender, older age (> 65 years), first myocardial infarction (frequently transmural), severe one-vessel coronary artery disease with a lack of collateral formation, and absence of previous angina. (9, 10) On the contrary, the presence of multivessel disease and a history of previous myocardial infarction may exert a protective effect, probably linked to development of greater collateral circulation and a better tolerance to wall traction, respectively. (11) There is no evidence that chronic hypertension and diabetes mellitus (12) modify the incidence of cardiac rupture; yet hypertension during the acute event is an important risk factor. Some studies have suggested that the use of corticosteroids and non-steroidal anti-inflammatory drugs (13, 14) interfere in the mechanisms of tissue repair, thus increasing the risk of cardiac rupture. However, this association could not be demonstrated either by the Multicenter Investigation of Limitation of Infarct Size (MILIS) (9) or the recent meta-analysis by Giugliano et al. that included more than 3000 patients. (15)

The association between the use of thrombolytic agents and cardiac rupture has been a long-term matter of debate, probably due to data disparities emerging from the different studies or to the way they are interpreted. The initial studies have suggested an increased risk of cardiac rupture with the use of thrombolytic therapy. The GISSI-1 (16) and ISIS-2 studies (17) showed a significant reduction in overall

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mortality (20%); yet early mortality was greater (<24 hours). Honan et al. (18) and Bueno et al. (19) found similar results regarding greater risk, yet associated with late administration of thrombolytic therapy and with patient's advanced age (< 75 years), respectively. In contrast, other investigators did not find significant differences. Data from the United States National Registry of Myocardial Infarction revealed an overall incidence of cardiac rupture < 1% both in the thrombolytic and placebo groups. However, overall mortality was significantly lower in the thrombolytic group (5.9% versus 12.9%) but the number of deaths due to cardiac rupture was greater compared to the placebo group. (2) According to data from the TIMI-9A and TIMI-9B studies, there is no evidence that the type of thrombolytic agent or the intensity of anticoagulation associated with heparin or hirudin administration influence the occurrence of rupture. (20) Finally, early administration (< 4 hours) of rTPA in patients with acute myocardial infarction has been associated with a significant reduction in the incidence of cardiac rupture , (21), while late administration (> 12 hours) has not proved to be associated with an increased risk of this complication. (22) The paradoxical effect of thrombolysis on the risk of cardiac rupture has been related to the type of agent used and the time interval between symptoms onset and treatment, among other factors. Several possible mechanisms have been suggested: extension of myocardial hemorrhage, weakening and dissection of the necrotizing zone, diminishing of the myocardial collagen content, and activation of collagenases and plasmin. (23-25) In conclusion, although thrombolytic therapy reduces the infarct size and prevents the expansion of the necrotic area, it also increases myocardial hemorrhage, produces digestion of collagen by proteolytic enzymes (plasmin) and interferes with the mechanisms of tissue repair.

Primary angioplasty produces greater reduction in the incidence of cardiac rupture compared to thrombolysis, yet the advantages of therapeutic catheter-based interventions are not clear in old patients. (26, 27) Recently, the SENIOR-PAMI study (28) compared primary angioplasty with thrombolysis in patients > 65 years and reported that the incidence of early mortality was similar with both procedures (13% versus 11%, respectively). Reperfusion-induced hemorrhagic infarction may be related to thrombolytic therapy and primary angioplasty, therefore these outcomes may be more associated with early treatment than with the type of reperfusion therapy used.

Timing of occurrence

It has been stated that most cases of cardiac rupture occurred in the prethrombolytic therapy era during the first week after myocardial infarction, with a peak incidence in days 5 to 7. Among 100 consecutive autopsied cases of postinfarction rupture, 3 % occurred during the first day, 58% within 5 days and 80% within 7 days. (29, 30)

Coronary reperfusion, especially with thrombolytic agents, has modified the natural history of this complication, as it accelerates cardiac rupture and the number of fatal events typically to within 24 hours of treatment. These conclusions emerge from the study by Becker et al. including over 350000 patients with myocardial infarction. (2) In patients treated with primary angioplasty, the risk of cardiac rupture is lower and occurs within 1 to 5 days after infarction. (31)

PATHOGENESIS AND HISTOPATHOLOGY

Physiopathology

The physiopathological mechanisms of cardiac rupture depend on the timing of occurrence. Leukocyte infiltration and signs of expansion of the necrotic area usually absent in early ruptures (< 24 hours); (32) on the contrary, cardiomyocyte apoptosis might be the main mechanism involved in wall weakening and dissection. (33) On the other hand, cardiac ruptures that occur 24 hours after infarction are related to the expansion of the necrotic area and are due to a abnormal remodeling process, (34) probably linked to increased activity of matrix metalloproteinases that regulate the structure and function of myocardial matrix. (35) Studies in animal models have suggested that inhibition of matrix metalloproteinases attenuates left ventricular remodeling in experimental myocardial infarction. (36)

These findings might have important implications in the development of therapeutic strategies based on the inhibition of these enzymes to prevent cardiac rupture secondary to myocardial ischemia.

Rupture sites and types and associated coronary artery anatomy

Cardiac rupture associated with acute myocardial infarction may involve the left ventricular free wall, ventricular septum and papillary muscles; rupture of the left atrium is less frequent. Left ventricular free wall rupture is 3 to 10 times more frequent that rupture of the papillary muscle or ventricular septum. (37) Left ventricular free wall rupture secondary to myocardial infarction has been reported to occur in the anterior and lateral wall. (38) However, there is evidence that most cases of subacute cardiac rupture are associated with inferior myocardial infarctions probable due to the fact that blood tends to form adherent cloths in this region. (3, 39, 40) In turn, most endocardial tears are located in the middle third of the ventricle within 1 cm of the papillary muscles as they insert in left ventricular free wall. This site is particularly prone to rupture due to the arrangement of muscle fibers and to wall stress. (41, 42)

Several morphologic types of rupture have been described based on findings in the operating room or in necropsies. Perdigao et al. (43) described four types of rupture in 42 patients: type I rupture (13 cases) with an almost direct trajectory with little dissection and bloody infiltration of the myocardium; type II (13 cases) with a

multicanalicular trajectory and widespread myocardial dissection and bloody infiltration; type III (9 cases) in which the orifice of rupture is protected by an intraventricular thrombus or pericardial symphysis; type IV (7 cases) with incomplete non-transmural rupture. More recently, Pucaro et al. (3) observed 6 morphologic types in a series of 28 patients: transmural rupture of the infarcted area in the presence of normal thickness of the wall (type 1) (n = 7); expansion of a softened necrotic zone with rupture of the wall (type 2) (n = 14); numerous small perforations within an area of myomalacia (type 3) (n = 1); incomplete (transmural) rupture of the infarcted area in the presence of normal thickness of the wall (type 4) (n = 3); large epicardial hematoma under pressure, covert rupture (type 5) (n = 1); hemorrhagic infarct with grossly intact epicardial surface (type 6) (n = 2).(Figure 1). Although each type of rupture has its own physiopathological behavior and clinical presentation, in general transmural ruptures present active bleeding and acute cardiac tamponade, multicanalicular tears have a subacute course with different degrees of pericardial effusion and covert ruptures constitute the most stable group and frequently develop chronic pseudoaneurysms.

The distribution of coronary artery anatomy associated with left ventricular free wall rupture is interesting. Figueras et al. have identified that the left anterior descending coronary artery and the left circumflex are most frequently affected (42% and 41%, respectively), while the right coronary artery is compromised in only 18% of cases. (44)

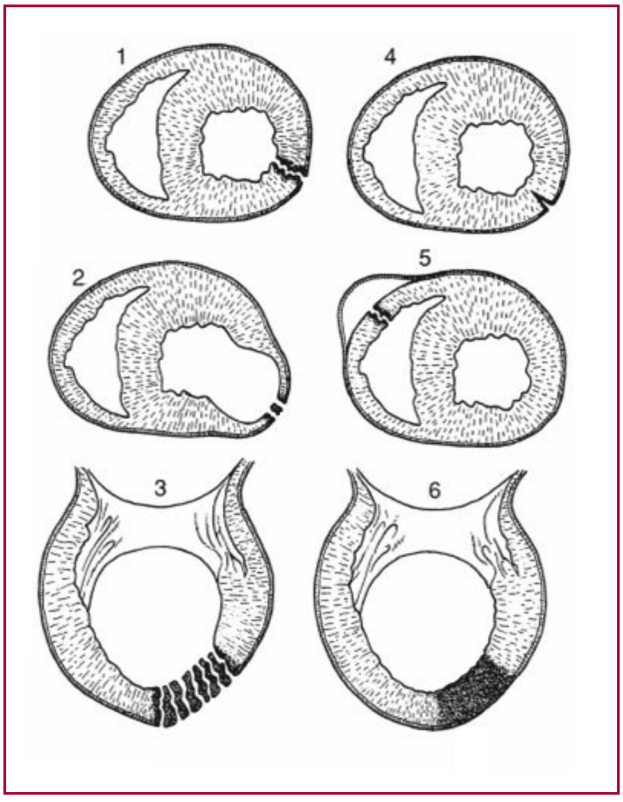

Fig. 1. Morphologic types of free-wall rupture: transmural unidirectional rupture (1); expansion of a softened necrotic zone with rupture of the wall (2); transmural multicanalicular rupture (3); non-transmural, incomplete rupture (4); covert rupture (5); hemorrhagic infarction (6).

DIAGNOSIS

Clinical Presentation

During the '70s, O'Rourke described the first three types of left ventricular free wall rupture (acute, subacute and chronic) in the basis of the clinical presentation and outcomes (6) (Figure 2). Other investigators have developed other classifications based on O'Rourke's description which is currently valid (Table 1).

The clinical presentation of patients with **acute rupture** is catastrophic and generally irreversible, with sudden chest pain, electromechanical dissociation, shock and death shortly after onset of rupture (< 30 minutes) The anatomical substrate of this group of patients is a transmural tear with sudden blood loss into the pericardial sac and rapid development of pericardial tamponade, which is fatal in most cases. (1, 3, 6, 7) On the contrary, **subacute** left ventricular free wall rupture leads to cardiac tamponade or cardiogenic shock with transient response to hemodynamic support. This is due to a covert or incomplete tear with slow or intermittent bleeding and progressive hemopericardium. The hallmark of this presentation is that the time from symptoms onset to hemodynamic collapse is > 30-60 minutes, usually allowing the diagnosis and surgical approach of the condition. (1, 3, 6, 7) The clinical presentation of these patients includes recurrent chest pain, pleuritic pain, sustained hypotension, syncope, restlessness, nausea and repetitive emesis. (45) These patients present different degrees of cardiac tamponade. However, the traditional signs as jugular vein distension and pulsus paradoxus are found in about 30% and 45% of these patients, respectively. (1, 3, 7) In rare occasions, a sealed rupture with a pseudoaneurysm, the anatomical substrate of **chronic** left ventricular free wall rupture, develops. These patients may remain stable and asymptomatic, or develop progressive dyspnea or ventricular arrhythmias depending on the severity of myocardial involvement. The recognition of this condition is based on the history of myocardial infarction, the clinical suspicion and echocardiographic findings. (46, 47)

Electrocardiographic signs

A certain correlation exists between the electrocardiographic findings and the severity of cardiac rupture. Electromechanical dissociation and extreme bradycardia are typical signs of acute cardiac rupture, the former with an accuracy of 97.6%. (1, 48) On the contrary, the electrocardiographic signs of subacute rupture are suggestive of this condition, yet unspecific, and include intraventricular conduction abnor-

Table 1. Comparison between the main clinical characteristics of each type of left ventricular free wall rupture secondary to acute myocardial infarction

	Acute	Subacute	Chronic
Anatomical substrate	Transmural tear with active bleeding	Incomplete rupture with progressive or intermittent hemorrhage	Covert rupture due to pericardial symphysis
Clinical presentation	Hemodynamic collapse and/or sudden death	Chest pain, sustained hypotension, repetitive emesis	Variable (absence of symptoms or symptoms due to heart failure depending on the severity of myocar dial involvement)
Electrocardiogram	Electromechanical dissociation	Unspecific ST-segment abnormalities	Unspecific
Echocardiogram	Hemopericardium with cardiac tamponade	Moderate pericardial effusion with *echogenic masses* within the pericardial fluid (with or without cardiac tamponade)	Pseudoaneurysm
Resuscitation and hemodynamic support	Hemodynamic instability generally refractory to refractory to therapy (death < 30 min)	Satisfactory response to hemodynamic support beyond 30-60 minutes	Satisfactory response to hemody namic support beyond 30-60 minutes
Treatment	Surgery	Surgery	Surgery (recent and expansive rup ture) Expectant magement (chronic ansstable rupture)

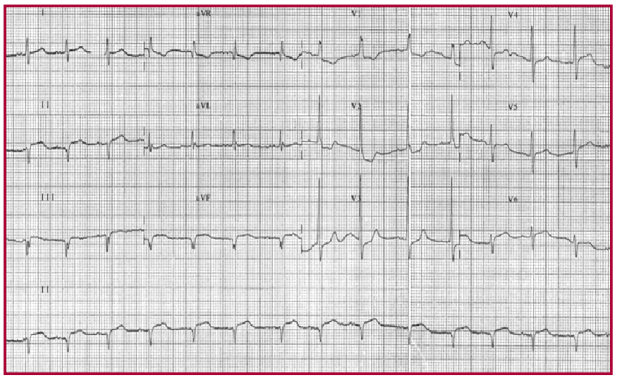

Fig. 2. Electrocardiographic changes during an episode of chest pain and hypotension in a patient with a recent inferior infarction and ongoing free-wall rupture. See the presence of QS waves in leads I, II and III and aVF, intraventricular conduction abnormalities and ST-segment elevation (1.5 mm) within the same leads.

malities (particularly, wide R wave in aVR), evidence of transmural compromise (new Q waves), (1) or infarct expansion (persistent ST-segment elevation or pseudonormalization of inverted T waves), (3) (see Figure 2). Oliva et al. found that persistent, progressive or recurrent ST-segment elevation > 0.3mV developed in 61% of patients with left ventricular free wall rupture (sensitivity 61%, specificity 72%). (45) Similar data were reported by Pollak et al., who observed that 56% of patients showed new ST segment elevations of 0.2 to 1 mV in the infarct-related leads. (7) Interestingly, in patients with acute anteroseptal myocardial infarction, ST segment elevation in lead aVL is a significant risk factor for left ventricular free wall rupture (odds ratio: 5.4). (49)

Unfortunately, none of these ECG signs are sensitive and specific enough to identify patients with an impending or ongoing rupture.

Echocardiographic signs

Echocardiography if the diagnostic tool of choice to establish the different types of cardiac rupture, as this method is mostly available, accurate and safe. (12, 42, 50, 51) The most frequent echocardiographic finding is pericardial effusion in patients with left ventricular free wall rupture; in fact, the absence of pericardial effusion has a high negative predictive value and excludes cardiac rupture in patients with myocardial infarction (1) (Figure 3). The presence of pericardial effusion has a low positive predictive value for left ventricular free wall rupture as it may be present in 28% of patients with acute myocardial infarction without cardiac rupture. (1, 52) The presence of echogenic masses in the effusion fluid is a relevant sign, particularly in patients with subacute free rupture (sensitivity: 97% - specificity: 93%); however, these signs may be found in fibrinous pericarditis associated with acute myocardial infarction (53) (Figure 4). Direct visualization of the myocardial tear confirms the diagnosis, yet is only

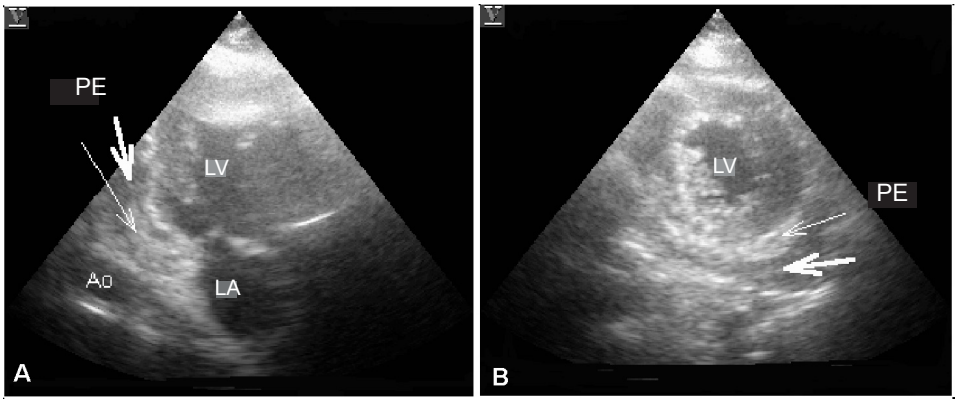

Fig. 3. Transthoracic echocardiogram of a patient with subacute rupture of left ventricular free wall. **A.** Two-chambers view showing mild dyskinesia of the basal inferior segment and moderate pericardial effusion *(thick arrow)* with echogenic masses in the fluid *(thin arrow)*. **B.** Basal short axis view showing a large semilunar pericardial hematoma with scarce residual hemopericardium.

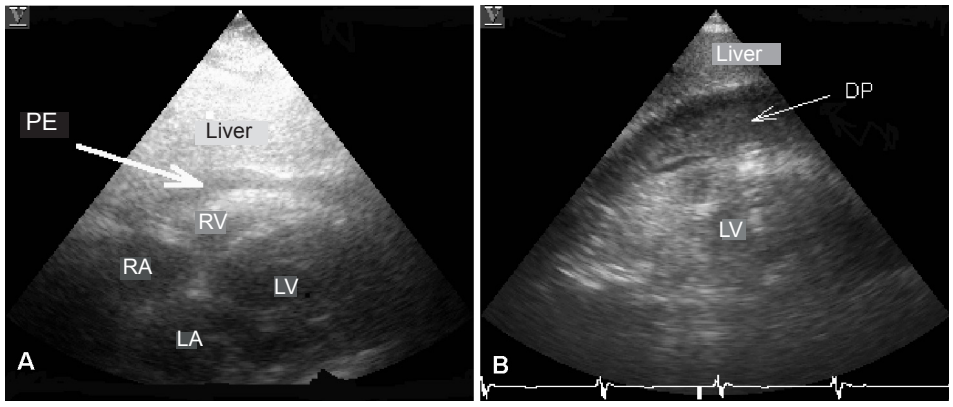

Fig. 4. Echocardiogram of a patient with subacute rupture of left ventricular free wall. **A.** Subcostal view showing moderate pericardial effusion with echogenic masses in contact with the right ventricular free wall. Despite the patient was hemodinamically unstable, there was no collapse of the right-sided chambers. **B.** Transesophageal echocardiogram (transgastric short axis view) of the same patient. A pericardial hematoma is visible with scarce residual hemopericardium.

possible in one third of patients with cardiac rupture. (1, 7, 54)

Although the severity of pericardial effusion is directly related to the amount of hemorrhage in the pericardial sac, in clinical situations as cardiac rupture the hemodynamic compromise depends more on the velocity blood collection accumulates than in the absolute volume of blood contained. Small and moderate rapidly accumulating collections may have catastrophic consequences, while subacute or chronic large effusions may be better tolerated. Echocardiography allows quantification of the severity of pericardial effusion; in addition, serial determinations of the distance between the endocardium and epicardium can help to infer the rate of bleeding. Doppler echocardiography provides useful information about the hemodynamic repercussion of pericardial effusion by assessing left ventricular diastolic flow. An exaggerated reduction in the amplitude of mitral E-wave during inspiration is a sign of cardiac tamponade that is independent of the severity of the effusion, even in the absence of clinical signs of cardiac tamponade (preclinical tamponade). (55)

Diastolic collapse of the right-sided chambers may be the echocardiographic sign of cardiac tamponade most recognized; however the presence of this sign does not necessarily imply cardiac tamponade and its absence does not exclude it. Although the traditional triad of cardiac tamponade is constituted by severe pericardial effusion, diastolic collapse of the right-sided chambers and clinical manifestations of cardiac tamponade, some patients present clinical manifestations without diastolic collapse and, conversely, other patients have severe effusions and collapse without clinical signs of cardiac tamponade. Mercé et al. have recently assessed the correlation between the hemodynamic profile and Doppler echocardiographic signs in 110 patients with moderate to severe cardiac tamponade. In patients with cardiac tamponade, 11% had no evidence of diastolic collapse of the right-sided chambers, while 51% of patients without clinical signs of cardiac tamponade had different degrees of collapse. Diastolic collapse may be absent in patients with cardiac tamponade and pericardial symphysis or with conditions that increase right-sided chambers pressure (COPD, tricuspid valve regurgitation, pulmonary hypertension, etc.). On the other hand, hypovolemia or low cardiac output may induce collapse in patients without clinical cardiac tamponade. (56)

Finally, patients with small pericardial effusions may present signs of hemodynamic compromise (cardiac tamponade) due to direct compression of one chamber, as seen in localized effusions (radiotherapy, malignancies) or pericardial hematomas (as in the postoperative period of cardiovascular surgery). (57, 58)

A pseudoaneurysm is a cardiac rupture sealed by the pericardium that generally develops in the infero-

lateral area of the left ventricle. It is visualized as a saccular structure with a narrow neck; the wall of this communication is formed by pericardium with organized thrombus and fresh blood. Color-Doppler echocardiography confirms the communication between this structure and the left ventricle, and spectral Doppler shows a characteristic bidirectional systolic and diastolic flow. (59, 60) Subsequently, the thrombotic material becomes organized, with a density similar to the surrounding soft tissues. In these cases, echocardiography fails to detect the pseudoaneurysm, while computed tomography scan and particularly magnetic resonance imaging which provide images based on different tissue characteristics are useful to identify the presence of a pseudoaneurysm and to exclude other differential diagnoses (60) (Figure 5). Intravenous injections of echocardiographic contrast agents are useful to identify wall defects and to evaluate the configuration of pseudoaneurysms. (61) Transesophageal echocardiography is especially useful in patients with poor acoustic window and is the method of choice in patients under mechanical ventilation or during the postoperative period of cardiovascular surgery, when transthoracic echocardiogram is usually limited in obtaining adequate images. (62)

Finally, echocardiography is an essential tool to guide emergent pericardiocentesis in patients with cardiac tamponade and hemodynamic instability. However, pericardiocentesis may give only temporary relief in acute ruptures with active bleeding or may not be complete if blood clotting inside the drainage tubing prevents further evacuation of pericardial fluid. (63)

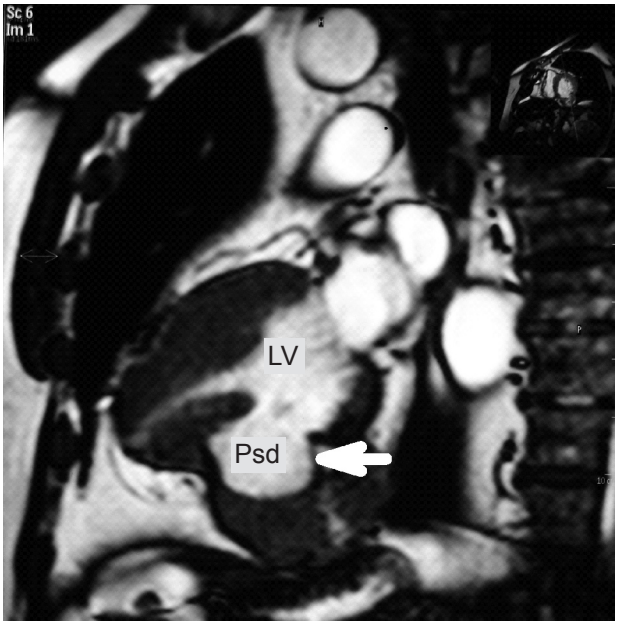

Fig. 5. Magnetic resonance imaging of the heart showing a pseudoaneurysm of the left ventricle in the mid inferolateral segment. The patient had remained asymptomatic for 10 years after a myocardial infarction. The images were taken from a control echocardiogram.

Biochemical markers

Increased plasma brain natriuretic peptide (BNP) concentrations (>250 pg/ml), a marker for increased ventricular wall tension/stress, even in the absence of symptoms or hemodynamic changes, might be a useful marker for predicting cardiac rupture after myocardial infarction. (64, 65) It has been shown that C-reactive protein, in a peak-concentration of >20 mg/dl on day 2 after acute myocardial infarction is an independent risk factor for cardiac rupture. (66, 67) Serum amyloid A protein, another marker of acute inflammatory response, has been recently proposed as an independent marker for cardiac rupture in patients undergoing primary angioplasty. (68)

Despite this evidence, the clinical usefulness of these parameters for the early detection of cardiac rupture is still unclear.

PREVENTION AND TREATMENT

Cardiac rupture should be prevented in all patients with acute myocardial infarction, especially in those at high risk of rupture. Management consists of strict control of systolic blood pressure and bed rest. Beta-blocking agents should be given to regulate the sympathetic stimulation as a response to low cardiac output. Angiotensin-converting enzyme inhibitors (ACEIs) reduce wall stress and prevent the expansion of the necrotic area. (69) Once the diagnosis of cardiac rupture has been established, the initial goal is to achieve hemodynamic stabilization as a bridge to surgery with repair of the myocardial rupture.

The administration of colloid solutions and inotropic agents improves cardiac output in patients with cardiac tamponade. (69, 70) Mechanical ventilation helps to achieve hemodynamic stabilization in these patients. In patients with cardiac rupture and hemodynamic instability, pericardiocentesis may produce only temporary relief because bleeding usually recurs and blood clotting inside the drainage tubing may prevent further evacuation of pericardial fluid. (42) In patients with acute rupture, emergency pericardiocentesis followed by the application of a patch to the epicardial surface with biological glue have been reported to lead to a successful outcome. Intra-aortic balloon pump reduces afterload, increases cardiac output and improves myocardial perfusion (71) and is indicated in unstable patients unresponsive to pharmacological therapy.

As cardiac rupture usually leads to death, emergency surgery is the treatment of choice regardless of the patient's condition. Surgical repair produces a significant reduction in mortality and provides favorable short-term outcomes and adequate prognosis during late follow-up. López-Sendón et al. reported operative and postoperative mortality of 25% and 52%, respectively. (1) Although survival without surgery has been

described in isolated cases (40, 69, 72), there is general agreement that surgery is the definite treatment in patients with cardiac rupture. Surgical treatment removes pericardial clots and repairs the myocardial defect. Concomitant myocardial revascularization does not improve the outcomes and should be delayed by three weeks after the acute episode. (42, 73) The first approach to repair myocardial rupture, described by Montgut et al. in 1972, is to perform an infarctectomy and replacement with a prosthetic patch under cardiopulmonary bypass. (74, 75)

However, recent reports suggest that more conservative interventions may be preferable. Direct mattress suture buttressed with Teflon felt or application of an autologous (pericardial) or synthetic (Dacron) patch adhered with biological glue or sutures (Figures 6 and 7) without infartectomy or cardiopulmonary bypass have been reported to lead to a successful outcome. (76-78) In general, the procedures that use direct suture and cardiopulmonary bypass are used to repair ruptures with active bleeding, while those that use biological glue are indicated in ruptures with progressive or intermittent hemorrhage.

Finally, the management of pseudoaneurysms will depend on the anatomical characteristics of the defect, its development, and patient's clinical status. Undoubtedly, surgery provides benefit for patients with symptoms of heart failure and recent (< 3 months) large, expansive pseudoaneurysms. On the contrary, expectant management is suggested on asymptomatic patients with chronic and stable pseudoaneurysms. (79)

CONCLUSIONS

Left ventricular free wall rupture complicating acute myocardial infarction is a catastrophic event; yet, this condition has a subacute course in one out of three patients, allowing a proper diagnosis and management.

An appropriate early clinical and echocardiographic evaluation may identify this subgroup of patients that require rapid hemodynamic stabilization as a bridge to surgery with repair of the myocardial rupture. As an example, a woman > 65 years old, without a history of coronary artery disease, has a first transmural infarction of the anterior and lateral wall. Reperfusion is achieved with thrombolytic therapy. The patient presents sudden chest pain within 24 hours after the episode, with ST-segment abnormalities and hemodynamic instability that initially responds to resuscitation. The presence of moderate pericardial effusion and echogenic masses within the fluid with signs of cardiac tamponade confirms the diagnosis although the defect is not visualized.

Infusion of colloid solutions and inotropic agents with or without pericardiocentesis is essential to stabilize the patient in order to perform surgery, a situation infrequent in acute ruptures. Surgery aimed at removing pericardial clots and closing the defect is the only definite treatment of patients with cardiac rupture.

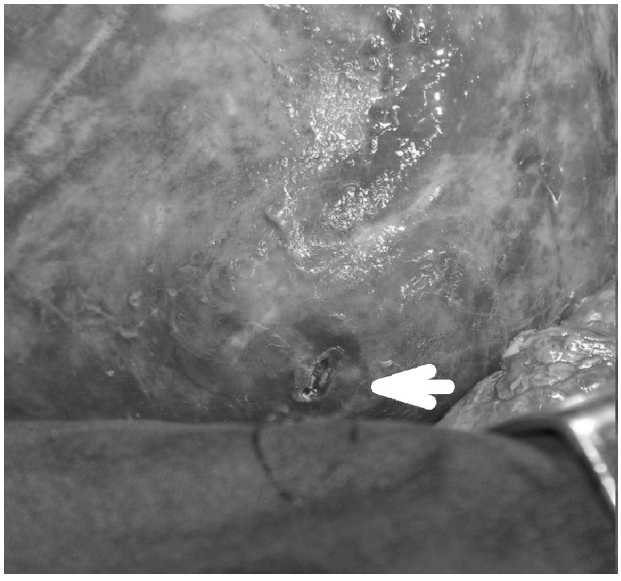

Fig. 6. Surgical repair of a subacute rupture of the left ventricular free wall secondary to myocardial infarction. A oval orifice of 1.5 mm can be seen in the area between the normal tissue and the necrotic scar (basal inferior and lateral).

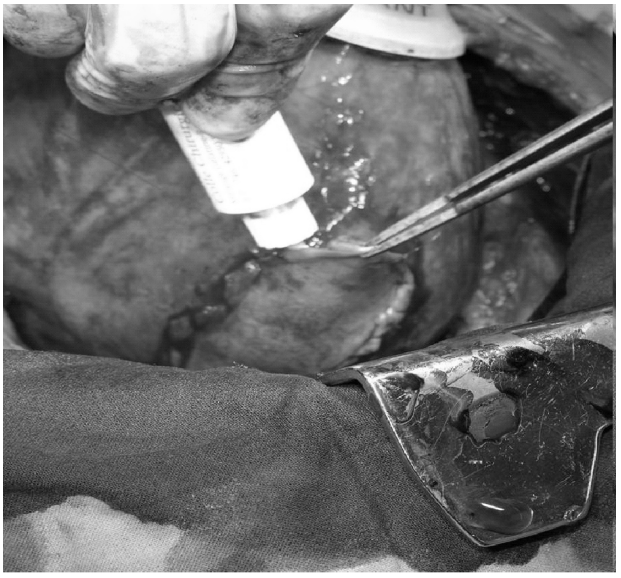

Fig. 7. Image from the same patient of Figure 6 after surgical repair of the defect with application of a Dacron patch with continuous suture.

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