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# Cardiac Rupture Sutureless Repair

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## Abstract

We retrospectively observed the long-term outcome of 40 patients with post-infarction cardiac ruptures repaired with a patch-and-glue technique, treated as an emergency between January 1997 and January 2019. In 32 patients, the cause of the rupture was late percutaneous revascularization. In patients with a poor ejection fraction, extracorporeal membrane oxygenation (ECMO) was used to unload the ventricle for a few days. A follow-up was made at 30 days, 6 months and long term with a mean follow-up of  $11.5 \pm 10.8$  years. Kaplan–Meier curves were used to evaluate the long-term survival. The 30-day mortality was 12.5% (5/40 patients), in patients with very low ejection fraction and was not related to the cardiac rupture. There were no postoperative complications except for bleeding (7.5%), due to ECMO haematologic disorders. No bleeding through the rupture site was recorded. Overall mortality was 15% (6/40 patients) at a 6-month control, at late follow-up ( $11.5 \pm 10.8$  years) was 80% (32 patients), and the causes of death were not related to the rupture. We can affirm that a patch-and-glue technique is a safe and life-saving operation to treat cardiac ruptures. An early diagnosis and surgical treatment are crucial for a successful outcome.

**Keywords:** cardiac ruptures, myocardial infarct, patch, glue

## 1. Introduction

### 1.1 History

William Harvey in 1647 was the first who described a left ventricle rupture [1]. Giambattista Morgagni, between 1707 and 1709, reported 11 cases of myocardial rupture at autopsy; he himself died of myocardial rupture [2]. He described left ventricle rupture as “an ulcer on the myocardial surface, with a lot of blood effusion in pericardium, with sudden death occurring after strong crisis of chest pain”. Lancisi, in 1728, in the *De Motu Cordis*, observed that sudden death was frequently related to “a hole on the heart apex” which he thought to be related to a congenital predisposition. He described in fact a myocardial fibre defect with an “integrity of internal and external membrane, but with a lack of tissue inside”.

### 1.2 Epidemiology

Cardiac ruptures are still a dramatic event complicating a myocardial infarction. According to some authors [3], ventricular rupture occurs in 10% of acute myocardial infarctions, and it is associated with significant mortality.

If the myocardial infarction is transmural, and the myocardial wall is damaged on its entire thickness, the rupture onset is dramatic. Pericardial blood effusion leads to an acute cardiac tamponade (**Figure 1**). In most cases, the rapid onset of symptoms leaves no chance for the patient to survive.

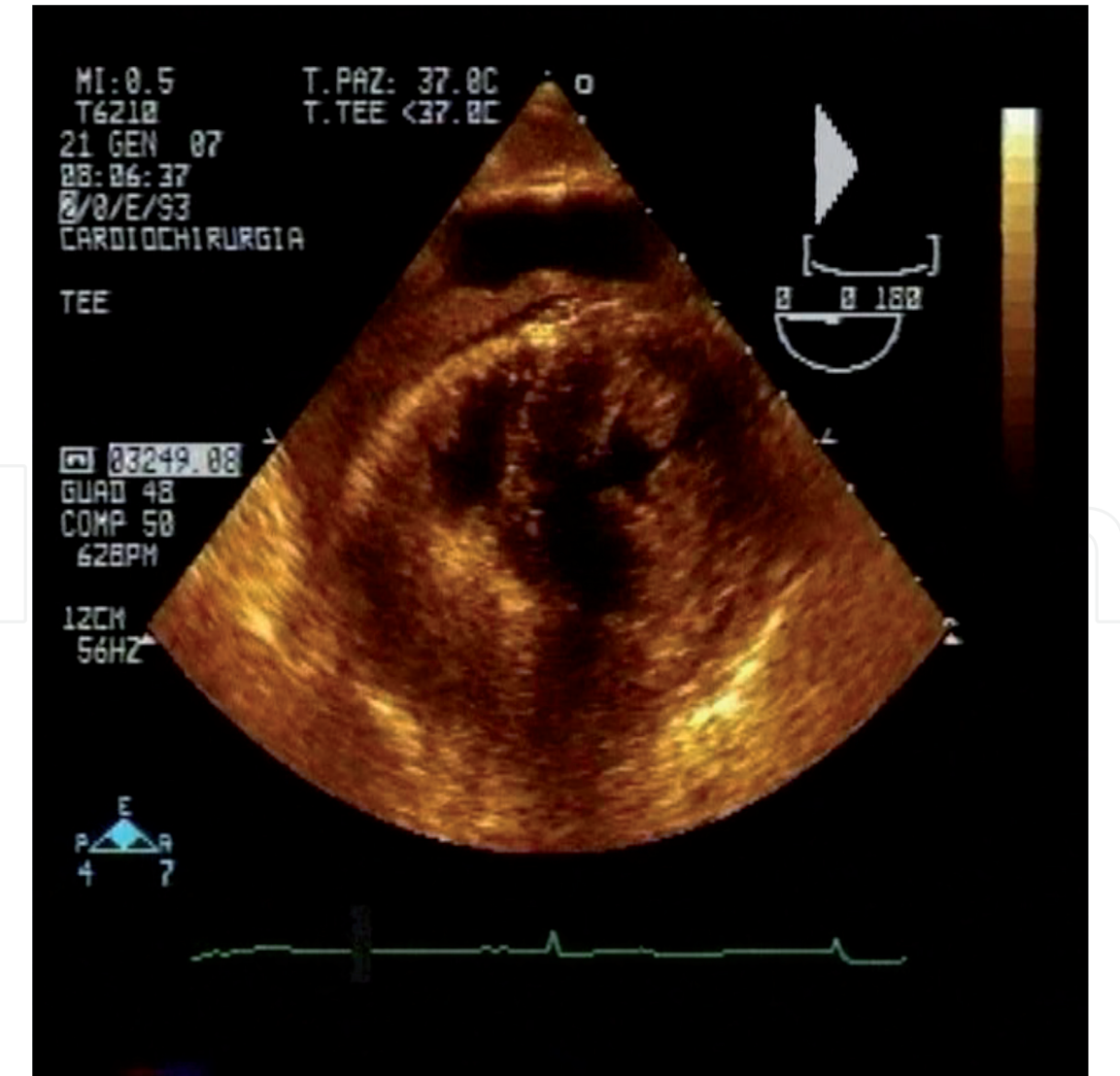
Transmural myocardial infarction diagnosed or treated with a certain delay is another dramatic though rare condition that affects 5 patients on 100,000 inhabitants per year. Even if the patient reaches the hospital, there are few chances for him to survive.

Symptoms, especially in diabetic patients, may occur later (hours or days) from the infarct onset. All of these conditions have a common characteristic: a transmural lesion that leads to ventricular wall rupture.

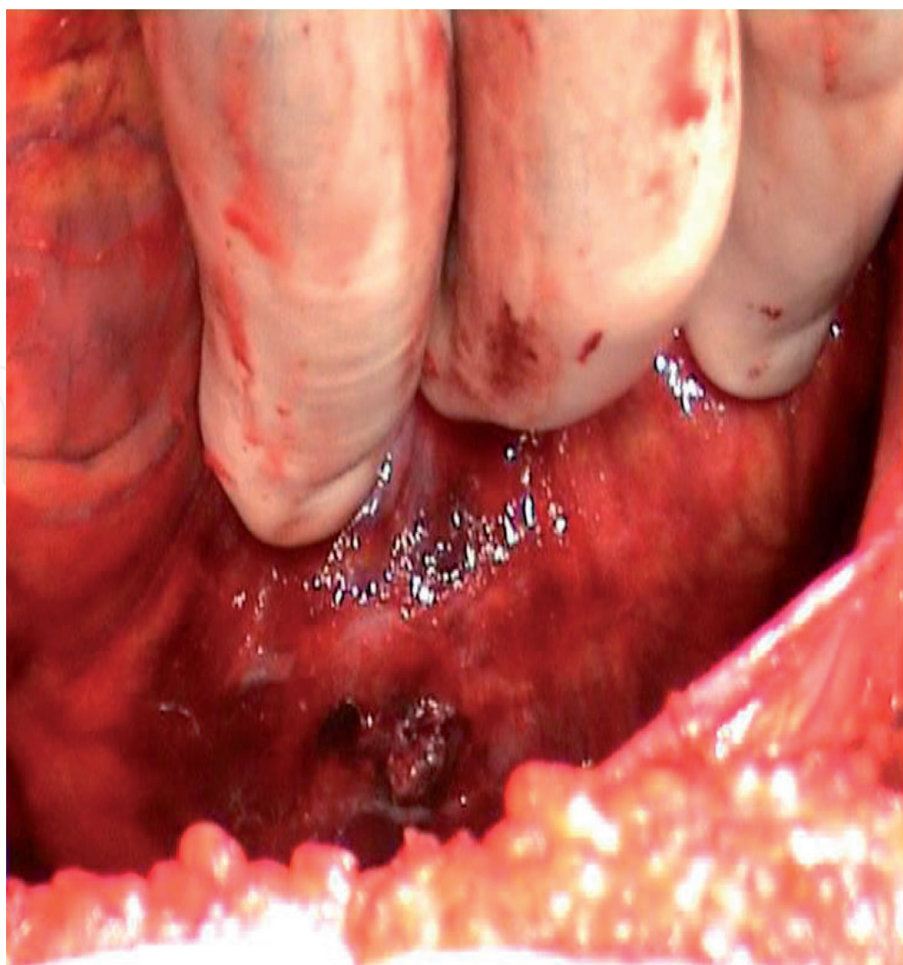
1.3 Definition

Wall rupture can be better defined as a “locus minori resistentiae” rather than a real hole in the myocardial wall. Myocardial wall changes its structure: it becomes similar to a sponge, through which the blood leaks into the pericardium (**Figure 2**).

We can identify two kinds of cardiac ruptures: one in which no macroscopic tear of the left ventricle free wall (LVFW) can be detected, with blood oozing from the infarcted zone (**oozing-type left ventricular free wall rupture (LVFWR)**),



**Figure 1.**  
*TEE image of a pericardial blood effusion with acute cardiac tamponade.*



**Figure 2.**  
*Postmyocardial infarction inferior rupture.*

and another in which a macroscopic defect of the epicardium is identified, with a free communication between the left ventricular cavity and pericardial space (**blowout-type LVFWR**).

#### 1.4 Pathophysiology

In both types of ruptures, the blood leaks into the pericardium: the haemorrhagic event starts as a gradual lowering of systemic pressure. When blood compresses the venous return to the heart, there's a significant hypotension due to cardiac tamponade. At that point the patient could die in a few minutes. On the other hand, in those conditions, wall rupture is temporarily auto-sealed, because the pericardium is a close cavity with a limited volume capability. In those cases the question is: drain it or not?

Pericardial drainage provides a short-term improvement of the haemodynamics but, at the same time, exposes the patient to a dangerous condition. In fact during cardiac tamponade, the pressure inside the pericardium is similar or higher than the systemic blood pressure, and clots could temporarily plug the rupture. Percutaneous approach can be very dangerous because pericardial pressure will fast slow down with a high risk of further ventricular wall rupture.

Percutaneous drainage could only be helpful to stabilise the patient while preparing the operating theatre. **The real need for the patient in those conditions is emergent cardiac surgery.**

Our conviction [4] is that the operation has to be as minimally invasive as possible. We usually perform it through median sternotomy, to better control possible



complications, off-pump and sutureless, with sealant and patch apposition. This will be better described later.

According to the literature, another debated topic is coronary angiography, whether to perform it or not in order to save time. Some authors suggest to perform coronary bypass graft without angiography; in our opinion angiography is mandatory. Proper revascularization has a positive impact on survival and on symptoms after revascularization; we generally bypass major vessels with significant stenosis supplying areas not involved in the infarct.

Other authors suggest to perform a complete myocardial revascularization associated with the ventricle wall repair [5]. This is an important point to focus on, because, in most cases, as described before, ventricular rupture follows a late diagnosis and late percutaneous transluminal coronary angioplasty (PTCA). So, frequently ventricular rupture occurs in patients with reperfused coronary arteries. Should the patient need an emergent treatment before a coronary angiography, the goal is always to treat the ventricle rupture more rapidly and safely as possible.

In case of cardiogenic shock, a cardiac mechanical support is needed in order to unload the left ventricle and let cardiac muscle reinforce. In those cases, before surgery, some authors [6] suggest to implant an extracorporeal membrane oxygenation (ECMO) at the moment of the diagnosis of left ventricle rupture. In some selected cases presenting with postischemic very poor left ventricular ejection fraction (LVEF), we implanted a ventricular assist device (VAD), a biopump to support circulation, as a bridge to recovery.

## **2. BioGlue**

BioGlue is a serum albumin and glutaraldehyde sealant.

### **2.1 Our clinical experience with BioGlue in cardiac ruptures**

#### *2.1.1 Methods*

Since the foundation of the Venice Cardiac Surgery in 1997, we treated as an emergency 40 consecutive patients affected by post-infarction cardiac rupture. In 32 patients the cause of the rupture was late percutaneous revascularization. Patients' mean age was  $69 \pm 9.4$  years. The mean interval time between the acute myocardial infarction and left ventricle free wall rupture was  $93.5 \pm 35.6$  h. All patients presented with cardiac tamponade at echocardiogram. No one received intra-aortic balloon pump (IABP) preoperatively. Even if some authors [5] suggest to insert the IABP before, we think that this device could stress too much the ventricle wall. In patients with poor left ventricular ejection fraction, extracorporeal membrane oxygenation was used to unload the ventricle for a few days and promote the reparation process. ECMO device was implanted in five (12.5%) cases.

A follow-up was made at 30 days, 6 months and long term with a mean follow-up of  $11.5 \pm 10.8$  years.

#### *2.1.2 The technique*

In all patients we used a double-layer solution, also called a "patch-and-glue" technique.

We open the patient with median sternotomy, in most cases without a cardiopulmonary bypass assistance. In some cases it is necessary to use a circulatory support to better expose the heart and the infarction site, especially if it is inferior, but, in

every case, without clamping the aorta or stopping the heart. Those patients have often a poor ejection fraction due to the acute infarct.

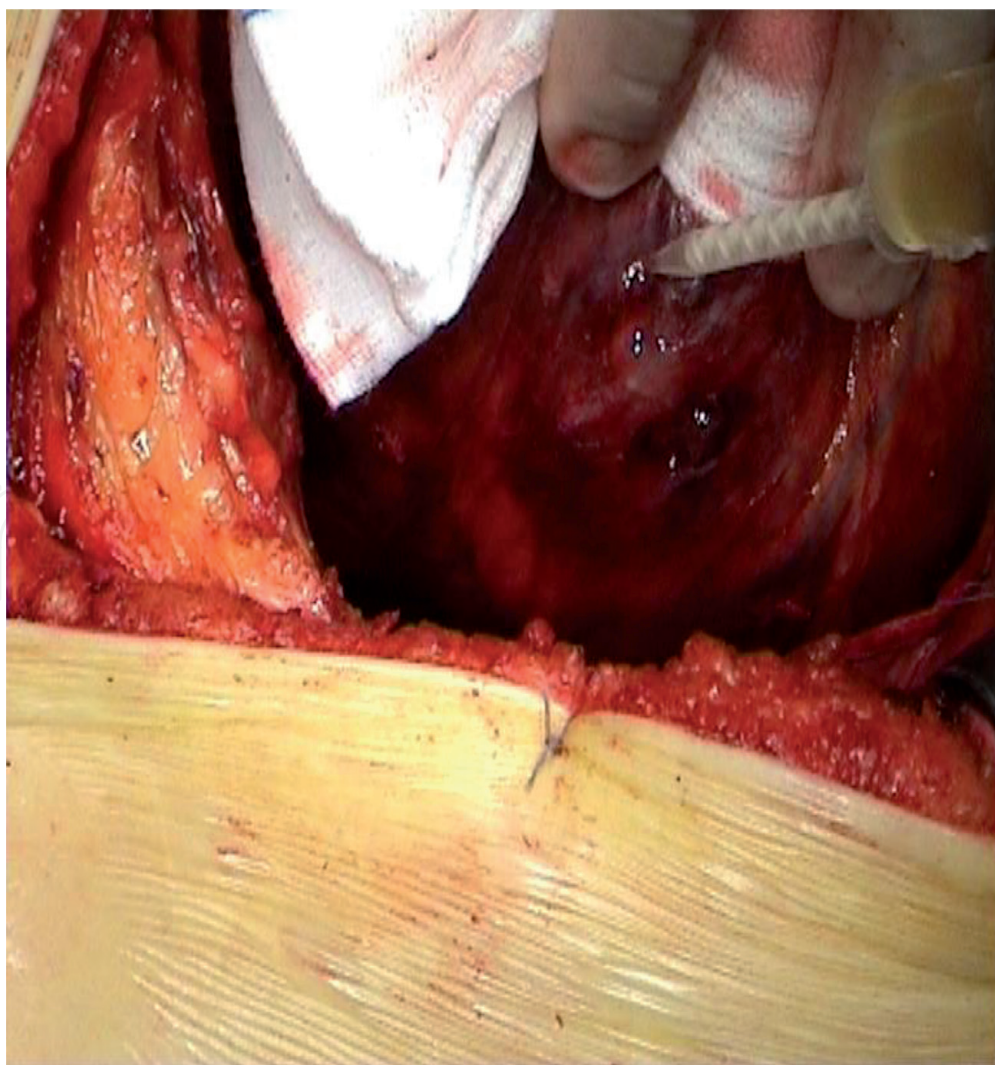
The cardiac tamponade is drained and the pericardium site is dried with some tissues. Especially the infarction site has to be as dry as possible before BioGlue application.

We first cover the infarction site with BioGlue (**Figure 3**), a sealant manufactured and distributed by CryoLife, Inc. (Kennesaw, GA). We prefer to cover a circle area, two centimetres bigger than the apparent rupture, almost corresponding to the patch size we want to put on.

Immediately after BioGlue application, we place a bovine tissue patch over the BioGlue layer, before glue polymerisation. We suggest to gently compress (from 30 s to 1 min) the “patch-and-glue” site, with a tissue.

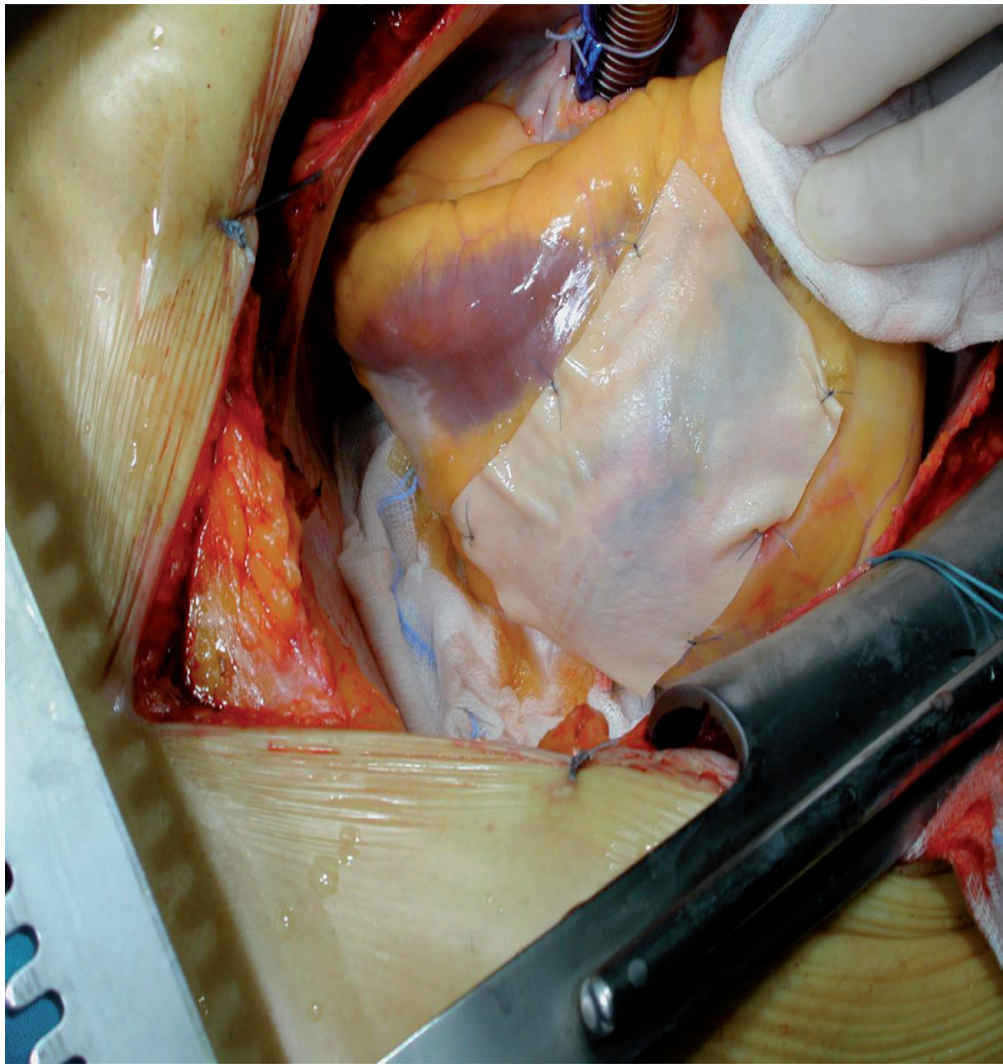
In the first four cases, we fixed the pericardium patch with four to six polypropylene stitches (**Figure 4**), but we early noticed that this fixation was not necessary (**Figure 5**), especially if the patch is positioned immediately after BioGlue application, generally within 30 s from glue application, before polymerisation.

Population characteristics and operative data were recorded. Postoperative echocardiogram at 6 days, 6 months and late follow-up was performed (average FU 11.5 years). In two cases we associated a CABG procedure. In five cases we implanted a ventricular assist device (ECMO) for about 6 days (mean 5.6 days). Kaplan–Meier curves were used to describe the long-term survival.

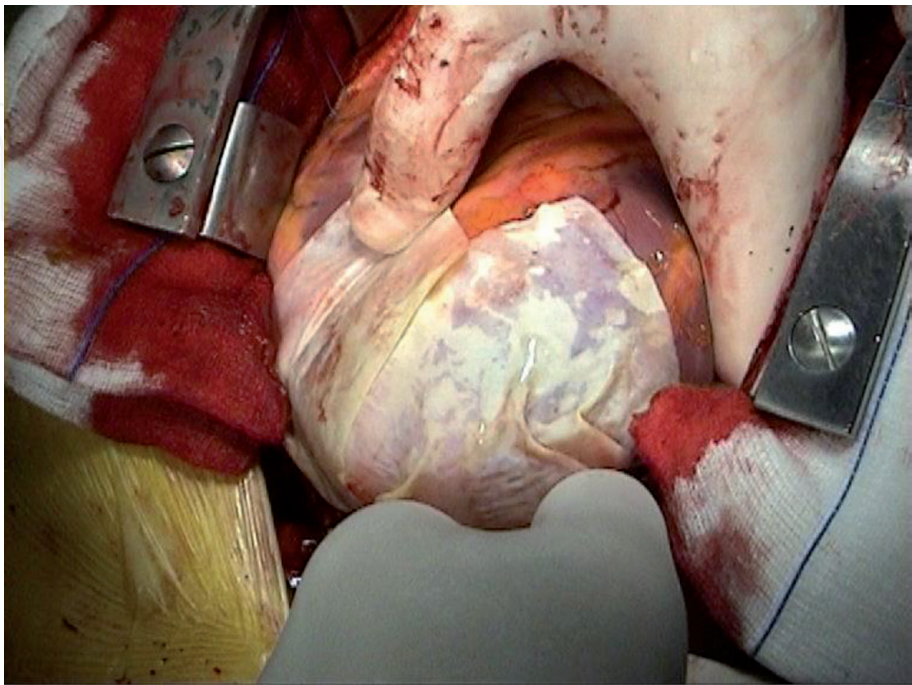


**Figure 3.**  
*BioGlue application over the infarction site.*





**Figure 4.**  
*Patch apposition over the glue (fixed with prolene stitches).*



**Figure 5.**  
*Extended patch apposition over BioGlue (no fixation with prolene stitches).*

### 3. Results

Postoperative death was 12.5% (5/40 patients), due to the infarction consequences, generally in patients with a very low ejection fraction, and was not related to the cardiac rupture. Three of those deaths were registered in patients with ECMO implant. There were no postoperative complications except for bleeding that was recorded in three patients (7.5%), with circulatory mechanical support, due to haematologic disorders, and no bleeding through the rupture site was recorded. Those three patients who received ECMO (60%, 3/5 patients) died of bleeding not related to the rupture.

One late death, 2 months later, was registered related to poor left ventricular ejection fraction, and not to the rupture.

We can affirm that at a 6-month control, the overall mortality was 6/40 patients (15%).

At late follow-up,  $11.5 \pm 10.8$  years, 32 patients were not alive (80%), and there were no death relations to the ventricle wall rupture.

### 4. Discussion

Cardiac ruptures are a dramatic event that occurs after myocardial infarction. Sometimes the rupture happens after some days (3–4 days) after late revascularization. The rupture manifests as a cardiac tamponade. In those patients who survive, there is a chance of a life-saving treatment with less invasiveness. Some authors suggest to keep patients in medical therapy [7], with very uncomfortable results. An experience of percutaneous treatment for cardiac ruptures has been reported, with blood drained from the pericardium and infusion of fibrin glue [8]. This technique is less invasive than surgery, but also very less accurate and full of risks. Moreover, since its publication in 2000, there have been no other reports about this technique. Recent advances in research suggest that there is a genetic determinant of the myocardial free wall rupture of the left ventricle in humans [9]. We do not think there are applications for this technique beyond acute ischaemic events to other areas, such as iatrogenic perforations, trauma, re-entry injuries, etc. In case of ischaemic injuries, the myocardial wall is functionally damaged, and the tissue is similar to a sponge (as mentioned before), and it is better to cover it with a patch rather than put a stitch. In other cases, such as iatrogenic myocardial trauma by a catheter, we think it is better to put a stitch to fix the problem, because the surrounding muscle is not pathologic.

We do not have any direct experience on injuries on other organs, such as liver or kidney trauma, but maybe this technique could be experimented.

### 5. Conclusions

We can affirm that the patch-and-glue technique is a safe and life-saving operation to treat cardiac ruptures. It is a very fast, simple and less invasive technique to treat a very huge complication of myocardial infarction as cardiac rupture.

In our series of 40 patients, no early or late rupture was recorded in the treated site. Kojima and colleagues report a rupture 5 days after the “patch-and-glue” technique, but the rupture was in a different site from the repair [10]. An early diagnosis and surgical treatment are crucial for a successful outcome [11] especially when excellent results can be achieved with a simple glued patch covering technique.



Very good early results and an excellent long-term follow-up support us affirming that this technique could definitely cure cardiac ruptures, although we have to better understand how to support those patients, especially ones with a very poor ventricular function.

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