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The Place of Conservative Treatment for Right Ventricular Free Wall Rupture after Acute Myocardial Infarction

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Abstract **Case Report**

Cardiac rupture is a rare but fatal complication of acute myocardial infarction (AMI), often with devastating hemodynamic consequences. Although the incidence has been reduced with the practice of reperfusion therapy, it still carries a high mortality rate of over 50%. Isolated right ventricular free wall rupture (RVFWR) is a rare entity with very few previously reported cases, and survival after RVFWR due to ST-segment elevation myocardial infarction (STEMI) treated with a conservative therapeutic strategy is extremely rare. Here, we presented two cases of RVFWR secondary to AMI, treated conservatively and survived.

Keywords: Cardiac rupture, acute myocardial infarction (AMI), (RVFWR).

INTRODUCTION

Cardiac rupture is a rare but fatal complication of acute myocardial infarction (AMI), often with devastating hemodynamic consequences. Although the incidence has been reduced with the practice of reperfusion therapy, it still carries a high mortality rate of over 50%.

Cardiac rupture can involve the free wall of the ventricles, the interventricular septum and atrium, or the papillary muscles, in which cases of free wall rupture (FWR) are approximately ten times less frequent compared to septal and papillary muscle rupture [1]. Left ventricular rupture accounts for the majority of reported ventricular rupture cases, whereas isolated right ventricular free wall rupture (RVFWR) is a rare entity with very few previously reported cases [1]. Here, we presented two cases of RVFWR secondary to AMI, treated conservatively and survived.

Case 1

Patient aged 70 years, with dyslipidemia as a CVDF, who presented with a symptomatology dating back 15 days made of: typical chest pain associated with NYHA stage II dyspnea. The evolution was marked by the persistence of chest pain with a worsening of the dyspnea becoming a dyspnea stage III of NYHA, which led the patient to consult the emergency room. Clinical examination found a conscious, hemodynamically and respiratory stable patient with blood pressure at 110/65 mmHg, heart rate at 80 beats per minute and respiratory rate at 20 cpm. The cardiovascular examination was unremarkable. The ECG showed a regular sinus rhythm at 89 bpm with negative apical-lateral and inferior T waves (Figure 1). Chest X-ray showed cardiomegaly without signs of pulmonary edema. On biological workup, troponins were 736 ng/ml (N <0.5), white blood cells were 15,090/mm3, CRP was 117mg/l and SV was 29mm. Transthoracic echocardiography objectified inferior, inferoseptal, and inferolateral hypokinesis in the basal and medial stages and inferior hypokenesis in the apical stage. A non-dilated, non-hypertrophic left ventricle in moderate dysfunction with LVEF at 47%, a circumferential pericardial effusion of great abundance with echogenic material in the pericardial space adjacent to the right ventricular free wall and apex (Figure 2), without respiratory variations, the ICV dilated, uncomplicated. The patient was put on colchicine 0.5mg/d, aspirin 75mg/d, furosemide 40 mgx2/d and PPI 20mg/d. Given the clinical stability of the patient and the stationary pericardial effusion, a coronary angiography was performed one week later which showed a subocclusion of the middle circumflex artery and a subocclusion of the middle right coronary artery. Following the presence of the pericardial effusion, the ALT was referred until the effusion was

evacuated. Surgical drainage was performed bringing back 750 ml of hematic fluid. Within 24 hours, the drain brought back 80 ml. After removal of the pericardial drainage, the echocardiographic control showed a stationary medium-abundance effusion, without respiratory variations, with a non-dilated, compliant IVC. Subsequently an angioplasty of the middle circumflex artery and the middle right coronary artery was performed and the patient was put on aspirin 75mg /d, clopidogrel 75 mg/d, bisoprolol 1.25mg/d, atorvastatin 40 mg/d, colchicine 0. 5mg/d, moxifloxacin 400 mg/d, furosemide 40 mgx2/d and PPI 20mg/d, d. An echocardiographic control was carried out in 15 days, 1 month, 2 months and 3 months which showed a regression of the pericardial effusion with a good clinical evolution.

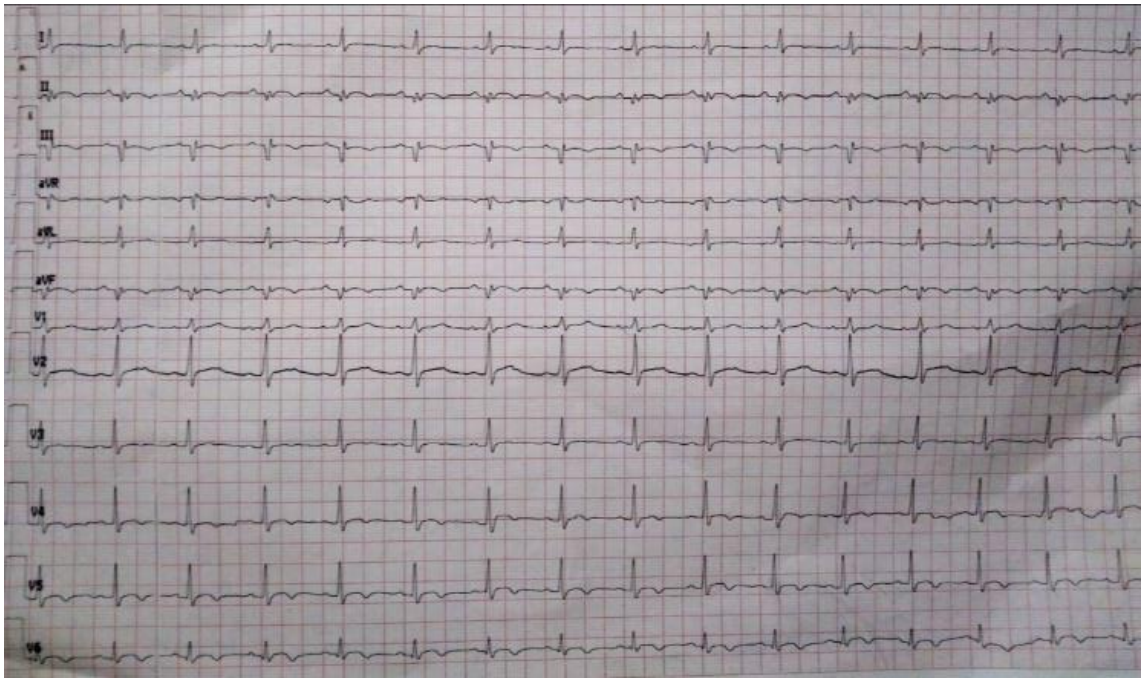

Figure 1: ECG showing negative T waves in apico-lateral and inferior

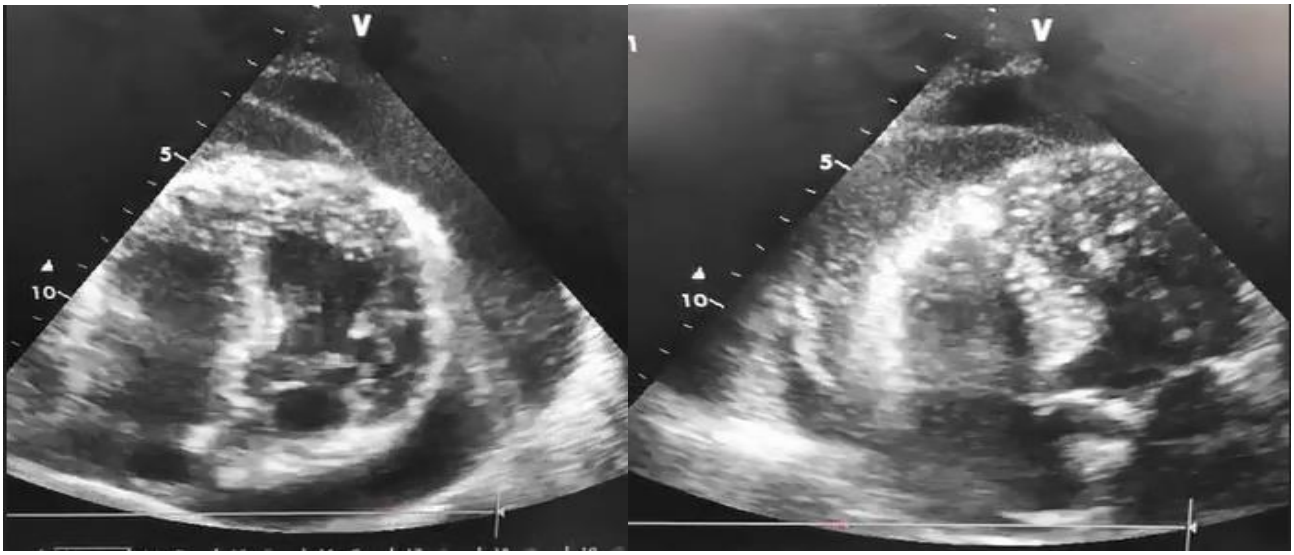

Figure 2: TTE that objectifies a large circumferential pericardial effusion with echogenic material in the pericardial space adjacent to the right ventricular free wall and apex

Case 2

Patient aged 73 years, with type 2 diabetes, chronic smoking weaned 3 months ago and dyslipidemia, who presented for a symptomatology that goes back to 4 days made of: the aggravation of an effort angina becoming an intense and prolonged rest angina associated with a dyspnea stage II of NYHA. The evolution was marked by the persistence of chest pain, which led the patient to consult the emergency room. Clinical examination found a conscious, hemodynamically and respiratory stable patient with a blood pressure of 149/95 mmHg, a heart rate of 64 beats per minute. The cardiovascular examination was unremarkable. The ECG showed a Mobitz I 2nd degree

BAV with ST-segment elevation in the inferior and right leads with Q waves and inverted T waves (Figure 3). Chest radiography showed cardiomegaly without signs of pulmonary edema. On biological workup, troponins were 5.2 ng/ml (N < 0.5), white blood cells 17940/mm3, CRP 139 mg/l and SV 95 mm. Transthoracic echocardiography objectified inferior, inferoseptal, and inferolateral hypokinesis in the basal and medial stages and inferior and septal hypokenesis in the apical stage. A non-dilated, enlarged left ventricle in moderate dysfunction with LVEF at 48%, a circumferential pericardial effusion of moderate abundance with echogenic material in the pericardial space adjacent to the right ventricular free wall (Figure 4), with no respiratory variations, the IVC dilated to 23 mm, poorly compliant. The patient was put on aspirin 75mg/d, clopidogrel 75 mg/d, atorvastatin 40 mg/d, ACEI 3.75 mg/d, colchicine 0.5mg/d, moxifloxacin 400 mg/d and PPI 20mg/d. A coronary angiography was performed which showed a tight stenosis of the middle circumflex artery and a total occlusion of the proximal right coronary artery. The echocardiographic controls carried out showed a progressive increase in the volume of the pericardial effusion, with the presence of respiratory variations, the IVC dilated to 23 mm, not very compliant. All antithrombotic drugs were stopped and the decision was made in view of the high operative risk to evacuate the effusion by pericardial puncture with monitoring. A pericardiocentesis was performed bringing back 1400 ml of serum fluid the first 24 hours and then 450 cc the following 48 hours. After removal of the pericardial drainage, the echocardiographic control showed a stationary medium-sized effusion. The patient was put on ivabradine 5mg x2/d, atorvastatin 40 mg/d, colchicine 0.5mg x2/d, moxifloxacin 400 mg/d and PPI 20mg/d. An echocardiographic check-up was carried out in 15 days, 1 month, 2 months and 3 months which showed a regression of the pericardial effusion with a good clinical evolution.

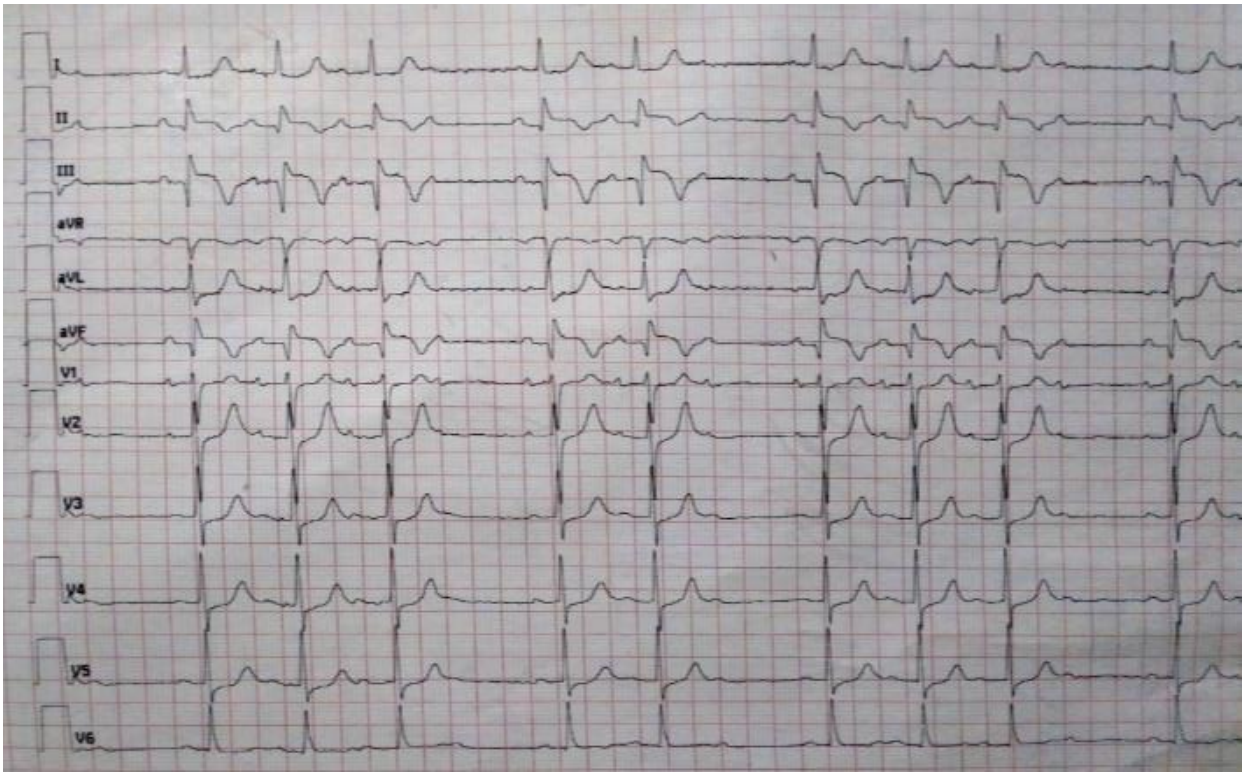

Figure 3: ECG showing inferior ST segment shift with Q waves and inverted T waves

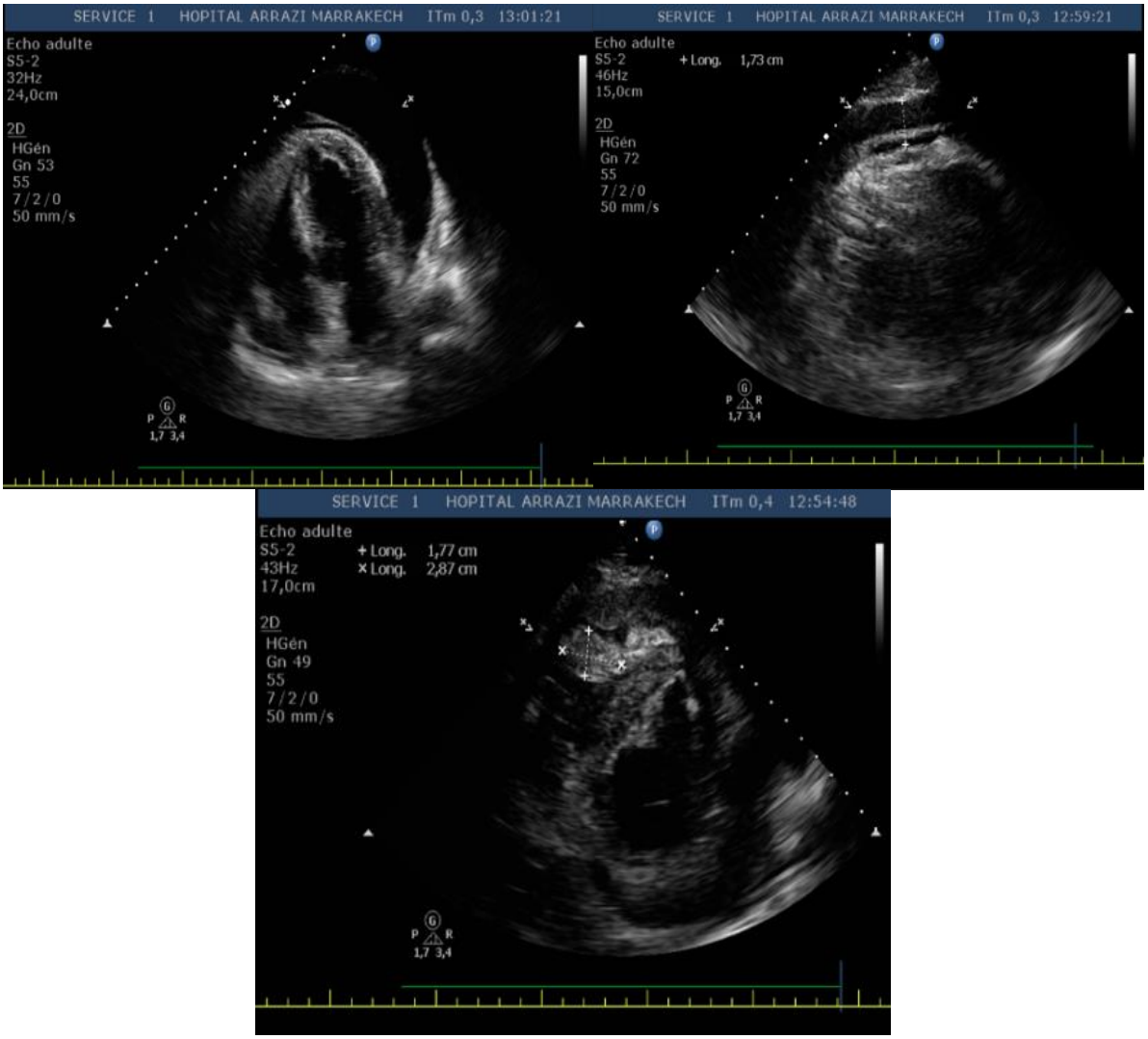

Figure 4: TTE that objectifies a large circumferential pericardial effusion with echogenic material in the pericardial space adjacent to the right ventricular free wall

DISCUSSION

Ventricular rupture is one of the most serious complications of AMI [1]. The incidence of cardiac rupture after STEMI was 6% [2]. In recent years, the incidence of cardiac rupture after AMI has decreased to less than more than 2% with the widespread use of PPCI [3]. It is a serious and more frequently fatal mechanical complication of AMI. The mortality rate for free wall rupture is 75% to 90% [4].

Most ruptures usually occur within the first 7 days after AMI, approximately 50% within the first 4 days; however, some cases have been reported up to a month or even later [5, 6].

RVFWR is primarily caused by inferolateral MI and right coronary artery occlusion. The low incidence of RVFWR was due to a lower pressure effect on the right ventricle compared to the left ventricle. This is not the only factor that protects the right ventricle. The right ventricle has a better collateral circulation and also a thinner wall, allowing for better myocardial perfusion and decreasing the likelihood of transmural infarction [1, 7].

Major risk factors for cardiac rupture include advanced age, female gender, hypertension, delayed or no reperfusion, no history of angina or myocardial infarction, massive anterior or lateral wall infarction with high ST-segment elevation, large transmural infarction, monotruncal involvement without collateral circulation, anemia, and high heart rate [8, 9, 1, 10].

Morphologically, four patterns of free wall rupture have been described by Perdigao *et al*., (Table 1) and the majority of survivors had a type III or IV rupture [8, 11,1 0, 12, 7].

Table 1: Four morphological profiles of myocardial rupture

Type I	An almost direct trajectory with small dissection and bloody infiltration of the myocardium.
Type II	A multicanalicular trajectory with myocardial dissections and bloody infiltration.
Type III	The rupture orifice is protected either by a thrombus or pericardial symphysis.
Type IV	Incomplete, with the trajectory not crossing all myocardial layers

Rapid diagnosis and early management are critical to saving patients' lives. Physicians should be aware of the signs and symptoms of ventricular rupture in patients with AMI.

Several distinct clinical forms of ventricular free wall rupture have been identified. Acute rupture is characterized by sudden and persistent chest pain, electromechanical dissociation, shock, and sudden death within minutes. This type of rupture does not allow time for any type of treatment. Subacute rupture which is caused by a small "fault" in the wall with slow and sometimes repetitive hemorrhages in the pericardial cavity, which may be temporarily limited by a blood clot or by pericardial synechiae. Usually this type of rupture presents with cardiac symptoms of tamponade and cardiogenic shock and may mimic reinfarction or infarction of the right ventricle. In the subacute form, the presentation may evolve over hours, days, or longer. Importantly, patients with the subacute form of rupture may remain hemodynamically stable. Finally, chronic rupture accompanied by the creation of a pseudoaneurysm occurs when the bleeding is very mild and limited from the peripheral pressure. In both of our cases, it is a subacute RVFWR in hemodynamically stable patients [9, 13].

Echocardiography usually represents the first-line diagnostic tool for suspected myocardial rupture that is rapid and non-invasive. The presence of pericardial effusion and diastolic compression of the right cavities are the 2 most relevant findings. The presence of echogenic masses on the epicardium increases both sensitivity (97%) and specificity (93%). If leakage into the pericardial cavity is demonstrated, the diagnosis is confirmed without doubt [7, 5].

However, there may be limitations in the evaluation of the right ventricle due to its crescent shape, substernal location, and the presence of a large amount of artifact. Fortunately, in our case, the echocardiographic finding of pericardial effusion and evidence of right coronary artery occlusion on coronary angiography led to the diagnosis of RVFWR [1].

Magnetic resonance imaging can improve the diagnosis by identifying the contained ventricular rupture and its anatomical features. Clearly, MRI is not the investigation of choice in acute patients with hemodynamic instability; however, in subacute cases, timely MRI with contrast can help delineate the anatomic location of the RVFWR. Multidetector CT has also been shown to be effective in detecting ventricular rupture and may aid in diagnosis [5].

The definitive diagnosis is usually made during surgery. Preoperative pericardiocentesis, which confirms a hemorrhagic effusion, can further support the diagnosis [5].

In the management of ventricular rupture, available treatment options include conservative measures and salvageable surgery.

After a diagnosis of LVFWR, the first step in treatment is to restore a satisfactory hemodynamic state with pericardiocentesis, intra-aortic balloon pump (IABP), intravenous fluid, and inotropic support [9].

Although it is generally accepted that surgery is the only definitive treatment for subacute ventricular rupture, cases of patients with long-term survival after medical management have been reported. In a series reported by Figuer as et al, selected patients with left ventricular free wall rupture after infarction were treated acutely with fluids, inotropic support, and therapeutic pericardiocentesis. Patients were followed clinically without surgical intervention. Thirteen of the 19 patients survived for an average of more than 4 years. The possibility that some of these patients had a contained myocardial rupture was not pursued. It is possible that the presence of an epicardial roof may confer adequate stability to provide some protection against bleeding into the pericardial space and the development of cardiac tamponade [14].

In our opinion, conservative management can be successfully performed in elderly and high-risk surgical patients who have small infarcts with slow bleeding and no other mechanical complications.

Medical management usually includes prolonged bed rest, strict blood pressure control (preferably with beta-blockers), and pericardiocentesis if necessary. In addition to prompt diagnosis, discontinuation of all antithrombotic drugs is an important measure to ensure successful conservative management. Now that antithrombotics are mandatory for STEMI patients, it is crucial to avoid further bleeding by stopping them.

In our two cases, our patients were treated conservatively and surprisingly survived with a good clinical course. A similar case was reported by Sherer *et al*., [15], and this remote phenomenon can be explained by a rapid resolution of the thrombus with the small myocardial defect that later became impermeable to blood. However, the recurrent rate of right ventricular rupture was not previously well established; therefore,

regular follow-up with echocardiography is essential to detect any recurrence or presence of complications.

CONCLUSION

Right ventricular free wall rupture following myocardial infarction is a rare complication with a fairly high mortality despite prompt surgical treatment.

Early detection of the first signs of RVFWR is crucial for successful management. The definitive and standard treatment is emergency surgical repair.

Conservative management includes hemodynamic monitoring, prolonged bed rest, beta-blockers, and angiotensin-converting enzyme inhibitors in selected cases.

Coronary angioplasty improves the mortality and morbidity of AMI and, compared with thrombolytic therapy and pharmacological treatment, reduces the risk of ventricular rupture and its concomitant mortality.

REFERENCES

1. Firdaus, M. A. B. M., Rahim, N. S. A., Rusdi, N., Idris, N. S., Mohd Said, M. R., & Abidin, I. Z. (2020). Conservative Treatment for Right Ventricular Free Wall Rupture in a Patient with Acute Myocardial Infarction. *Case reports in cardiology*, 2020.
2. Figueras, J., Alcalde, O., Barrabés, J. A., Serra, V., Alguersuari, J., Cortadellas, J., & Lidón, R. M. (2008). Changes in hospital mortality rates in 425 patients with acute ST-elevation myocardial infarction and cardiac rupture over a 30-year period. *Circulation*, 118(25), 2783-2789.
3. Honda, S., Asaumi, Y., Yamane, T., Nagai, T., Miyagi, T., Noguchi, T., ... & Yasuda, S. (2014). Trends in the clinical and pathological characteristics of cardiac rupture in patients with acute myocardial infarction over 35 years. *Journal of the American Heart Association*, 3(5), e000984.
4. Bates, E. R. (2014). Reperfusion therapy reduces the risk of myocardial rupture complicating ST-elevation myocardial infarction. *Journal of the American Heart Association*, 3(5), e001368.
5. Matteucci, M., Fina, D., Jiritano, F., Blankesteijn, W. M., Raffa, G. M., Kowalewski, M., ... & Lorusso, R. (2019). Sutured and sutureless repair of postinfarction left ventricular free-wall rupture: a systematic review. *European Journal of Cardio-Thoracic Surgery*, 56(5), 840-848.
6. Shiyovich, A., & Nesher, L. (2012). Contained left ventricular free wall rupture following myocardial infarction. *Case reports in critical care*, 2012.
7. Lateef, F., & Nimbkar, N. (2003). Ventricular free wall rupture after myocardial infarction. *Hong Kong journal of emergency medicine*, 10(4), 238-246.
8. Han, C. S., Bohonis, S., Walker, J. R., Vo, M., Hussain, F., Pascoe, E., & Jassal, D. S. (2010). An unusual presentation of left ventricular free wall rupture. *Canadian Journal of Cardiology*, *26*(8), e328-e329.
9. Yan, L., Wang, H., Su, B., Fan, J., Wang, M., & Zhao, X. (2021). Survival after left ventricular free wall rupture following acute myocardial infarction by conservative treatment. *The American journal of emergency medicine*, 39, 21–23.
10. Kabaj, M., Berezowski, M., Przybylski, R., Witkowska, A., Brzostowicz, T., & Kosmala, W. (2020). Incomplete myocardial rupture following inferolateral myocardial infarction. *Kardiochirurgia Torakochirurgia Polska Polish Journal of Cardio-Thoracic Surgery*, 17(2), 105.
11. Mishra, P. K., Pathi, V., & Murday, A. (2007). Post myocardial infarction left ventricular free wall rupture. *Interactive Cardio Vascular and Thoracic Surgery*, 6(1), 39-42.
12. Che, J., Li, G., Chen, K., & Liu, T. (2016). Syndrome de rupture de la paroi libre post-IM. Rapport de cas, revue de la littérature et nouvelle terminologie. *Rapports de cas cliniques*, 4(6), 576–583.
13. Exadaktylos, N. I., Kranidis, A. I., Argyriou, M. O., Charitos, C. G., & Andrikopoulos, G. K. (2002). Left ventricular free wall rupture during acute myocardial infarction: early diagnosis and treatment. *Hellenic J Cardiol*, 43, 246-52.
14. Helmy, T. A., Nicholson, W. J., Lick, S., & Uretsky, B. F. (2005). Contained myocardial rupture: a variant linking complete and incomplete rupture. *Heart*, 91(2), e13-e13.
15. Sherer, Y., Levy, Y., Leibovich, L., Shoenfeld, Y., Shahar, A., & Konen, E. (1999). Survival without surgical repair of acute rupture of the right ventricular free wall. *Clinical cardiology*, 22(4), 319-320.