Left Ventricular Free Wall Rupture During Acute Myocardial Infarction. Early Diagnosis and Treatment

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We are describing a case of a 63 year old patient who came to the emergency department seriously suffering. His clinical condition was characterized by severe paleness, hypotension and confusion. The patient was intubated and supported with mechanical ventilation. The ECG showed a slight ST elevation in leads II,III,avF,V_5,V_6. The transthoracic echocardiogram showed pericardial effusion and signs of cardiac tamponade. The patient was subjected immediately to cardiac surgery with the diagnosis of cardiac rupture. During the operation the rift was sutured with a suitable technique. After one week, the patient underwent coronary angiography which showed marginal significant stenosis of the anterior descending artery. We review the literature of this significant mechanical complication of acute myocardial infarction.

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Ventricular free wall rupture occurs in 1 to 3% of patients with acute myocardial infarction (AMI) and is the third most common cause of death, caused by AMI, ten times more common than the post-infarct rupture of the interventricular septum and the rupture of the papillary muscles. This most severe mechanical complication of the AMI often remains undiagnosed and constitutes a necropsy finding[1,2,3].

We describe the case of a patient with left ventricular free wall rupture, timely diagnosed and followed by immediate and successful therapeutic treatment.

Case description

The patient was male, 63 years old, smoker, hyperlipidemic (blood cholesterol levels 240 mg/dl), without hypertension or diabetes mellitus and without personal or family history of cardiovascular disease. Approximately thirty minutes before hospital admission, the patient complained of retrosternal pain, headache and faintness. The symptoms and his general condition presented rapid deterioration and he was transferred to the neurology clinic of the emergency department of our hospital on suspicion of cerebral vascular episode.

The patient manifested severe precordial pain, excitation, confusion, dyspnea and severe paleness of the skin and mucosa. He was unable to provide information on his condition and presented transient loss of consciousness that alternated with intense excitation during which his immobilization was very difficult. The patient presented diaphoresis, loss of urine and feces, while his arterial blood pressure could not be accurately assessed, despite the fact that in order to immobilize him on the examination table we were assisted by the total of the personnel of the clinic. It should be noted that the patient was initially transferred to the neurology clinic since his symptoms began with sudden headache, dyspnea and precordial pain.

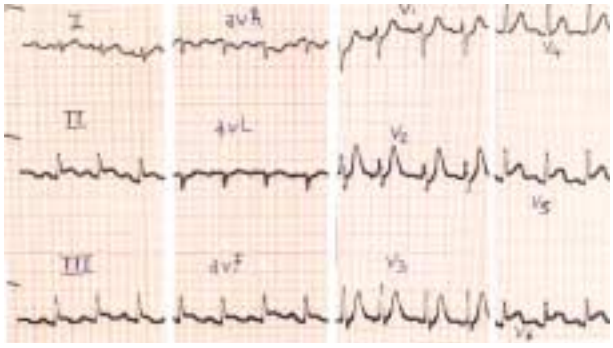

Figure 1. Electrocardiogram. ST elevation in leads II, III, avF, V_5,V_6.

Further diagnostic and treatment efforts were facilitated following the patient's intubation and mechanical ventilation, after which we were able to accurately measure his arterial pressure (70/40 mmHg) as well as to perform surface electrocardiogram that contributed to the diagnosis of acute myocardial infarction and more specifically, infarction of the lateral and inferior wall (mild ST elevation in leads II, III, avF, V_5, V_6, Figure 1). Upon clinical examination, no specific pathological findings were observed concerning cardiovascular system, nervous system, respiratory system and venous system of the lower limbs.

The patient was transferred to the coronary care unit, under rapid i.v. administration of sodium chloride saline 0.9%. We continued the effort of stabilization his hemodynamic condition in the intensive care unit where a central vein was catheterized (subclavicular vein) that was used for monitoring and rapid administration of saline. The patient presented hemodynamic improvement while blood gases and the acid-base balance were within normal limits 10 - 15 minutes following admission in the coronary care unit.

In the mean time and within 20 minutes from the patient's admittance, a transthoracic echocardiographic examination was conducted by an experienced physician. This examination, although technically difficult, provided the following information: Absence of clear segmental motility disorder of the left ventricle and good overall systolic function of the left ventricle (Ejection fraction 60%). The characteristic echocardiogram finding was the presence of small quantity of pericardial fluid and suspicion of thrombus in the pericardial space (Figure 2). Moreover, there was tamponade characterized by compression of the right cardiac cavities. The inferior vena cava had increased diameter that did not change during respiration. On the basis of clinical and echocardiogram findings, the heart surgeons on shift were called and it was decided to immediately transfer the patient in the operating room.

Surgical treatment

In order to successfully treat the tamponade, the patient underwent general anesthesia and following antisepsis of the thoracoabdominal region, mid sternotomy was performed accompanied by incision of the pericardium as well as removal of thrombotic material. Systolic arterial pressure immediately increased from 50 to 140 mmHg. A small size (approximately 5mm diameter) rift at the inferior wall of the left ventricle was then observed. The myocardium surrounding the rift was brittle, indicating recent necrosis. We then proceeded to attach a bovine pericardium patch in the area of rupture with the use of glue. The patch's free flaps were sutured on the epicardium, with prolene continuous sutures.

The afternoon blood tests revealed increased myocardial enzymes values (CK: 1784 U/l, SGOT: 149 U/l, SGPT: 147 U/l) except for the CK-MB isoenzyme which was slightly increased: 28 U/l (laboratory normal value < 23 U/l). These myocardial enzymes values presented gradual decrease. One week later, the patient underwent coronary angiography that revealed as a unique pathological finding, 60% stenosis of the proximal part of the anterior descending branch of the left coronary artery.

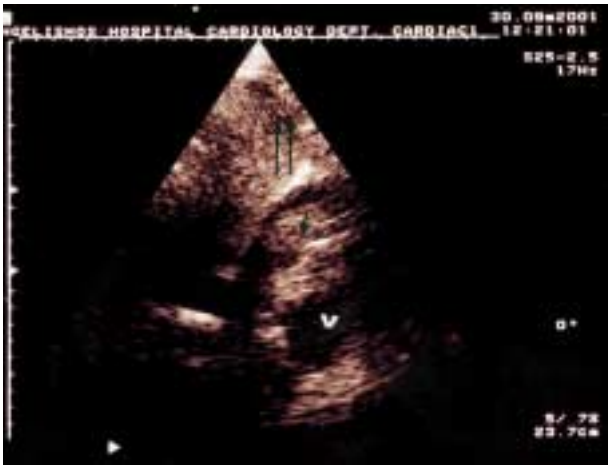

Figure 2. Echocardiographic subcostal view. Pericardial effusion with thrombus (arrow). Liver (two arrows). V: left ventricle.

Discussion

The first clinical reference of post-infarction left ventricular wall rupture was reported by William Harvey in 1647[4]. In 1765, G. Morgagni reported as necrotomy findings, 11 cases of free wall cardiac rupture, ironically, however, he too died later from cardiac rupture. Later, Hatcher et al in 1970 reported the first successful operation to treat right ventricular wall rupture[5] while Fitz Gibbon and Montegut in 1972 conducted the first successful operations for the correction of left ventricular wall rupture due to ischemic heart disease[6,7].

Rupture of the ventricular free wall and cardiogenic shock are the major causes of death following AMI, contributing to 66% of deaths due to first AMI[8]. It is more common in women, hypertensive individuals without hypertrophy of the left ventricle, in individuals without sufficient collateral network and in cases of delayed thrombolysis. It appears in the first week following the AMI, usually on the 4th or 5th day post-AMI, although the time limits of the manifestation may range from a few minutes to more than one month after the acute MI[9,10,11,12]. Aspects differ concerning the most common localization of the left ventricular free wall rupture. Previous literature studies report that the anterior wall is more often susceptible to rupture[13] and the more recent studies indicate that the rupture is more common on the lateral or posterior wall. However, it seems, as David et al[14] report, that the lateral wall ruptures are theoretically more probable, but the fact that anterior infarcts are more common than lateral infarcts, renders anterior wall ruptures more common.

O'Rourke first classified[15] free ventricular wall rupture as acute, subacute and chronic with pseudoaneurysm. The acute rupture is characterized by sudden and persisting thoracic pain, electromechanical dissociation, shock and death within a few minutes that it is caused by bleeding in the pericardial space and tamponade. This type of rupture does not allow us time for any kind of treatment. The subacute rupture is caused by a smaller "rift" on the wall that may temporarily be limited by a clot of blood or by pericardial synechiae. Usually this type of rupture is presented with symptoms of cardiac tamponade and cardiogenic shock and may mimic reinfarction or right ventricular infarction. We consider as subacute cardiac rupture the clinical condition, that the time between the onset of the typical symptoms and the non-reversible shock exceeds 30 minutes. Finally, the chronic rupture accompanied by the creation of pseudoaneurysm occurs when bleeding is very slightly and is limited from the peripheral pressure.

An effort has been made to further discriminate the ruptures of the free ventricular wall, depending on the clinical picture that they present. "Blow-out" ruptures are manifested with cardiogenic shock (arterial pressure < 70 mmHg for at least 10 minutes), while "stuttering" ruptures, had less noisy clinical picture and varying severity of symptoms, without hemodynamic instability. This latter category is clinically characterized by small rupture that is assessed during the operation and that most probably could be tamponaded spontaneously[15,17].

Electrocardiographic findings in ventricular rupture, are also directly correlated with its severity. Electromechanical dissociation (with diagnostic accuracy that reaches 97.6%) and bradycardia constitute the characteristics of acute rupture. The subacute rupture might manifest with increase of ST elevation by at least 1mV in affected leads, ST elevation in aVL as well as non-inversion of the T wave[9]. Without the echocardiographic examination, it is difficult to proceed to differential diagnosis between rupture and reinfarction, even following CPK, CK-MB values analysis[9]. C reacting protein values could constitute another indicator since quickly increase from the second day and remain increased (>20 mg/dl) in patients with AMI and rupture, compared to patients with AMI only, where levels increase much slower and remain lower (<10mg/dl)[15,19]. Pericardial effusion is the most common echocardiographic[10] finding in patients with subacute rupture of the ventricular wall. Echogenic masses in the pericardial fluid or evident lesions of the ventricular wall lead to a more accurate diagnosis. Such findings can be further reinforced with pericardiocentesis and aspiration of bloody pericardial fluid, something that could temporarily improve the condition of the patient[20,21].

The evolution of the events in acute free wall rupture rarely provides the adequate time to treat the patient surgically[14]. Patients usually die within a few minutes. However, when there is strong suspicion of cardiac rupture, biological glue can be administered intrapericardially following pericardiocentesis, ensuring thus valuable time until the patient is led to the operating room. On the contrary, subacute rupture allows us to surgically treat the patient. The goals of surgery include avoiding cardiac tam-

ponade and performing closure of the ventricular deficit[22,23].

Our case concerned acute left ventricular free wall rupture, where the rift was small and allowed us time for diagnosis and treatment of the patient. The 12 lead surface electrocardiogram presented signs of subacute rupture[9,23,24]. The heart ultrasound that verified the diagnosis reinforced the view that in the appropriate hands, the contribution of Doppler – ultrasound in the diagnosis of mechanical complications of myocardial infarction is enormous. Aside from the echocardiographical characteristics of the left ventricular free wall rupture that have been mentioned above, it has also been reported that with two dimensional ultrasound the rupture may appear as a break of the continuity of the myocardium and it has been found that the placement of the sample volume of the pulse Doppler at the point of rupture, shows bidirectional flow[10]. These characteristics were not observed in our case because the rupture was small in size.

In our case, since there was no delay in surgical treatment, we did not use an intra-aortic balloon counterpulsation. Its placement may be useful, if the surgical treatment is to be delayed. However its beneficial effect has not yet been proven, since the main problem with these patients is tamponade[14,15]. Following mid-sternotomy and incision of the pericardium, we proceeded very carefully and cautiously because the upcoming increase of arterial pressure could possibly increase the rupture. The rift in our case was at the inferior wall of the left ventricle, while the coronary angiography showed marginally significant stenosis at the anterior descending branch (proximal part) of the left coronary artery. This coronary angiography finding was not the expected one. However, it has been recently reported that cardiac rupture may occur even in the presence of intracoronary thrombus, followed by spasm without significant stenosis[16].

Following the identification of the site of the rift, we proceeded to the surgical operation described above. In general, four surgical techniques have been applied by most heart surgery centers for the correction of free ventricular wall rupture. In the first technique, continuous horizontal sutures are used, reinforced with Teflon patches. This technique tends to be abandoned because the suture line lies on necrosed tissue and there is a risk of recurrence. The second technique combines resection of the infarction region and closure of the deficit with separate Teflon reinforced sutures. This technique requires occlusion of the aorta and is indicated for patients who also present ventricular septal defect. The third technique requires horizontal continuous suture of the lesion, reinforced with double Teflon layer. On the surface of the sutures and the whole necrosed surface, a Teflon patch that is sutured on healthy epicardium is placed. The fourth technique includes simple gluing of Teflon patch or of bovine pericardium processed in glutaraldeyde (Baxter Healthcare Corp., Edwards CVS Division, Irving, CA) on the lesion and the area with necrosis with the use of biocompatible glue or fibrin. This technique does not presuppose the placement of the patient in extracorporeal circulation device and is preferable in case there is no active bleeding of the ventricle. The gluing takes place in less than one minute. With the method of covering the rupture with a patch that covers the whole infarction region, it seems that we avoid the risk of immediate post-operative repeat rupture, that exists when we apply the direct closure method. As far as the use of biological glue is concerned, its infusion on the infarction region on which the patch will be placed should take place quickly in order to be infused on a as dry as possible surface, thus maximizing the action of the biological glue in insulating the specific region. This technique is also indicated in case of lateral wall ruptures.

A problem for the surgeons, is the choice of the appropriate surgical technique in patients who during the pericardium incision an extensive epicardial hematoma is observed, without a visible rupture point. Some believe that the surgeon must incise and examine the hematoma to identify the infarction region and the rupture point, in order to better assess the size of the infarction region and to resect it and close the rupture. There are also those who believe that the whole area of the epicardial hematoma should be covered by a patch, without any incision nor examination of the hematoma. Arguments in favor of the first view include the fact that in this way, the surgeon can identify the rupture and the dissection of the myocardial walls and at the same time by resecting the infarction area, restore the geometry of the left ventricle, which is important for its better function. Of course, this type of surgery must be performed on extracorporeal circulation and ischemia under cardioplegia, elements that significantly increase intra-operative mortality. On the contrary, the hematoma covering technique may be performed on a beating heart, without extra-cor-

poreal circulation. However, the risk of myocardial walls dissection still remains, increasing the probabilities of post-operative cardiac rupture. The intraoperative mortality rates with this technique are clearly much lower. In our case, no aorto-coronary bypass was performed. However, many surgeons bypass the underlying coronary disease, empirically. This could be indicated for relatively hemodynamically stable patients, leading to avoidance of reinfarction and unstable angina in the immediate postoperative period. The combination of rupture closure with concurrent aorto-coronary bypass does not seem to yield better results than the simple rupture closure. The problem lies in the administration of cardioplegia to the recently ischemic and highly sensitive myocardium[25-34]. In our case, we selected a described technique, appropriate for the specific patient[35] that contributed to saving the patient's life. The strategy to be followed in case of left ventricle free wall rupture is presented in a diagram (Diagram 1).

In conclusion, we described a case of acute left ventricular free wall rupture, where the suspicion of diagnosis, its establishment with cardiac ultrasound and the prompt surgical treatment contributed to

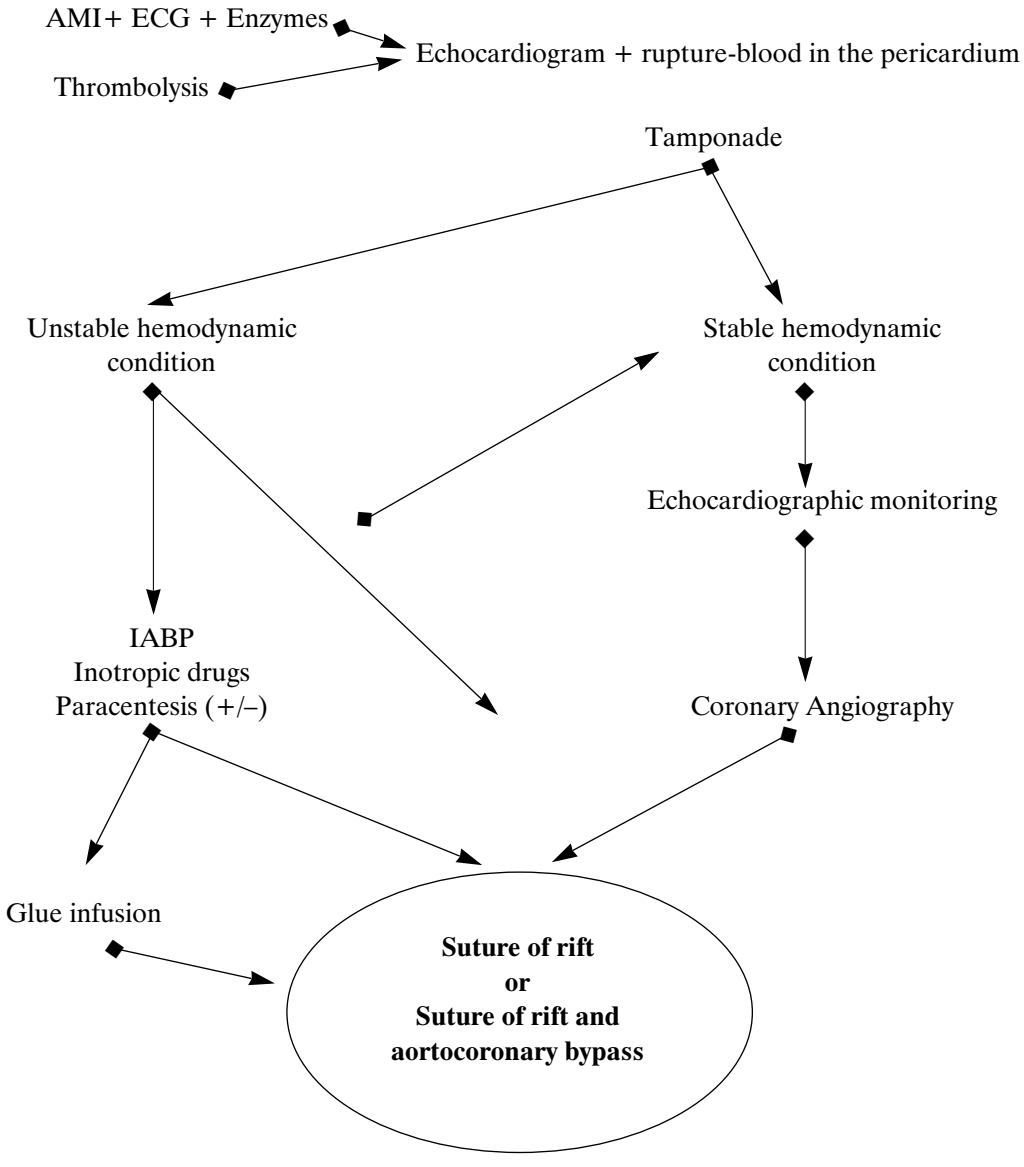

Diagram 1. Ventricular free wall rupture.
AMI: acute myocardial infarction,
ECG: electrocardiogram, IABP: intaaortic balloon pump

saving the patient's life. An additional element that played a significant part was the co-ordination of efforts. No other similar report has been published in available, recent, Greek literature. The good outcome of our patient is of value if one considers the recent reported mortality rates of 83.6% due to this mechanical complication[36], as well as the intra-operative mortality rates of 11.8% - 50% with several other techniques[37]. Based on this case, it would be worth mentioning certain points that played an important role and could be taken into consideration in similar cases:

1) Despite the fact that the out-patient cardiology departments in all the hospitals host a plethora of non-cardiological symptoms, patients with severe and in particular rare cardiological problems may be admitted to other hospital departments due to non-specific symptoms. Such departments should be provided with immediate support by cardiologists and anesthesiologists.
2) The prompt sedation, intubation and mechanical ventilation saves lives, where indicated of course, since it helps rapid diagnostic and therapeutic approach.
3) Prompt transfer to the intensive care unit decisively facilitates hemodynamic support and diagnostic approach.
4) Transthoracic echocardiogram is of fundamental importance in treating mechanical complications of acute MI.
5) The presence of a heart surgeon on shift and his/her good co-operation with the rest of the staff is required in order to save the life of patients who are in a similar, severe condition. In our case, the intervention of heart surgeons was immediate and contributed to the good outcome of the patient. However, it is known that similar support is not provided in all tertiary care hospitals in our country.

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