

Mechanical Complications of MI: Diagnosis, Management and Review of the Literature

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Introduction

Cardiovascular diseases are the leading cause of death worldwide. They represent a public health problem due to the cost of hospital care, the enormous burden on social security, the significant morbidity caused and therefore the consequences that follow such as incapacity for work and the decline of the productivity.

In Tunisia, cardiovascular diseases represent the major cause of mortality (30%) [1]. Among the pathologies encompassed under the term “cardiovascular diseases”, myocardial infarction occupies an important place due to its incidence and its mortality rate.

This entity has been the subject of several definitions over the years. The latest definition, which is the 4th published in 2018, states that the diagnosis of IDM arises when there is evidence of an acute ischemic lesion associated with clinical signs of infarction with elevation or fall in troponins [2].

In addition to the management, which must be extremely rapid, conditioning the vital prognosis of the patient, the particularity of this pathology lies in the diversity of these complications. Indeed, complications can be mechanical, embolic, ischemic, rhythmic and inflammatory [3]. In this work, we are interested in the mechanical complications of IDM through the study of three cases.

Case 1

This is a 70-year-old patient admitted for CIV complicating the immediate aftermath of a myocardial infarction of the lower territory. The clinical picture is dominated by dyspnea, edema in both lower limbs and crackles on pulmonary auscultation. Cardiac

ultrasound revealed a 33mm wide muscle CIV in the major axis responsible for a left-right shunt and PAH and segmental kinetics disorders. The coronarography objectified a tight stenosis of the posterior interventricular artery (IVP). Assistance by intra-aortic counter pulsation balloon was put in place and the patient was operated on. Under CEC, he had aorto-coronary bypass by a segment of the right internal saphenous vein on the IVP then the closure of the CIV was performed through a vertical left ventriculotomy parallel to the IVP followed by placement of a Dacron prosthetic patch. Exit from CEC required the introduction of low-dose adrenaline with continued BCPIA support.

Case 2

We report the case of a 60-year-old man who smoked 60 packs a year and presented with chest pain evolving for 1 day related to an acute coronary syndrome with ST segment elevation.

The patient benefited from fibrinolysis. The evolution was marked by the persistence of chest pain and worsening dyspnea. An ultrasound (ETT) objectified the presence of a very abundant pericardial effusion with basal ventricular hypokinesia. The diagnosis of RPLVG was retained and the patient benefited, without extracorporeal circulation (ECC), from drainage of the pericardial effusion with reinforcement of the ventricular wall by affixing a pericardial patch fixed by a suture at the healthy muscle level with biological glue. Postoperatively, the patient was extubated after 24 hours and the postoperative course was uneventful.

Case 3

This is a male patient, 65 years old, with a history of diabetes on insulin, non-weaned smoking. Hospitalized in the cardiology department for management of a lower MI. After management of his ischemia, the patient presented with dyspnea and

tachyarrhythmia. An ETT was performed on him demonstrating ischemic heart disease with massive MI. The origin was a broken pillar. The indication for surgery was asked and the patient was transferred to the surgery department for surgical management, but the patient died before the intervention.

Discussion

Complications of MI are grouped under 5 main headings: Electrical, inflammatory, embolic, ischemic and mechanical complications.

Mechanical complications post IDM are not common but constitute a true evolutionary turning point by the increased rate of mortality [4].

These are cardiogenic shock, rupture of the LV free wall, rupture of the interventricular septum, acute mitral insufficiency and ventricular aneurysm. With the advent of different methods of revascularization, these injuries have become increasingly rare and the associated mortality rate has been declining for about thirty years [5].

Among the complications found, cardiogenic shock is the most common mechanical damage [6]. It is often the consequence of an extension of the infarction causing RV dysfunction. The other causes are none other than complications such as rupture of the free wall of the LV, septal rupture that we will study in what follows. Rupture of the free wall of the LV has an incidence of 2% in the acute phase of MI [7].

Its occurrence is fatal except in the rare cases where the rupture is sealed. In this case, surgery is an extreme emergency [8]. Brown and Coll have described the risk factors for myocardial rupture, including advanced age, female gender, and the transmural character of the infarction and the territory of the anterior descending muscle [9].

The rupture occurs at the level of: the inferolateral wall (43%), the lateral wall (28%) and the apical wall (24%) [9]. The clinical picture varies from typical angina pain to the picture of tamponade [10].

In our patient, the symptomatology was made up of chest pain associated with dyspnea. The ECG usually shows electromechanical dissociation. The diagnosis can be confirmed by echocardiography; its sensitivity is 100% and its specificity 93% [11], which demonstrates the existence of pericardial effusion, signs of tamponade such as diastolic collapse of the RV, systolic collapse of the OD [12]. Such was the case in our patient, in whom the ETT had objectified a very abundant effusion. Management begins with hemodynamic stabilization of the patient [13].

The surgical procedure consists of closing the parietal defect either directly or by inserting a patch. Recently, "Sutureless" techniques have been introduced, which consist of using a prosthetic patch, which is inserted into the area of healthy myocardium and adheres

to it with biological surgical glue [7]. The advantage of this technique is the non-use of CEC and the absence of the use of heparin, which therefore reduces the risk of bleeding [14].

In our case, the patient had recourse to this surgical technique using a Teflon patch and without extracorporeal circulation. The suites were simple. The other complication found in connection with IDM is the rupture of the interventricular septum, which complicates 1 to 2% of IDM [15] and is responsible for 5% of mortality. Septal rupture most often occurs at the transition from healthy to infarcted tissue [16]. The risk factors found are [17]: advanced age, arterial hypertension, female sex, a history of IDM. In addition, involvement of the anterior territory was found to be more likely to cause this complication [18].

In our case, the myocardial infarction is located in the lower territory with extension to the right ventricle and the only risk factor in the patient was his advanced age (70 years). Hayashi has shown that subjects with VIA occlusion are more at risk of septal rupture [19].

This attack usually occurs within a week of MI [20]. In our case, it was immediately after the acute episode of infarction.

Arterial hypertension seems to play an important role in the genesis of septal rupture. Indeed, Roberts has shown in his work that patients who died by septal rupture had, in their majority, ventricular hypertrophy with a hypertensive appearance [21]. Symptoms include chest pain, dyspnea, and even cardiogenic shock [22]. For our patient, the picture of heart failure dominated the clinical presentation with crackles on pulmonary auscultation and edema of both lower limbs. Diagnosis can be made by ETT showing CIV. Similarly, intraoperative TEE can be used, which will show the septal defect [23]. Coronary angiography, if the patient's condition allows it, determines an assessment of coronary lesions [16].

Management consists of hemodynamic and respiratory stabilization, followed by correction of the defect. The intra-aortic counter pulsation balloon is the best hemodynamic support contributing not only to reducing afterload but also to improving coronary perfusion and reducing oxygen consumption [22]. Our patient benefited from the establishment of the BCPIA before recourse to surgery, which made it possible to improve his hemodynamic instability.

This correction can be done surgically or percutaneously. Surgical management consists of closing the communication via a left ventriculotomy [24] by placing a patch, which will therefore be located between RV and LV. In our case, the patient was operated under CEC. Initially, he had coronary artery bypass grafting via the internal saphenous vein on the IVP followed by closure of the CIV with a Dacron prosthetic patch. The surgery is performed after at least 7 days post-infarction so as not to work on a myocardium weakened by ischemia [25]. The percutaneous alternative consists

of inserting an “Amplatzer” device. This technique is done under ultrasound control [26].

The other pathology less complicating MI but still current is ischemic MI. The incidence of mitral leakage by pillar rupture is estimated at 0.26% in the era of percutaneous intervention [27]. In the absence of urgent treatment, mortality can reach 80% within 24 hours [28].

The posterior pillars seem to be more affected by the rupture than the anterior pillars [29]. A better network of replacements for the anterior pillars could explain this. The rupture of pillars would be more frequent in the event of infarction in the lower territory [30]. Symptoms generally appear 2 to 7 days after the ischemic event [17]. It is generally well tolerated and asymptomatic [3] but can lead to cardiogenic shock.

The ECG may show tachycardia or confirm the diagnosis of MI.

Chest x-ray shows evidence of pulmonary edema suggesting increased pressure in the pulmonary veins [31]. Diagnosis is made by ETT but TEE retains a greater specificity estimated at 95 to 100% [32]. Minami has studied in his work the ventricular function in patients who have had a myocardial infarction complicated by pillar rupture. He found that LVEF was retained in most patients [33]. This was the case in our patient. Post-mortem studies have shown that the territory affected by ischemia was limited to the endocardium surrounding the papillary muscle [34]. The rupture would occur in patients with normal ventricular function and would be explained by the pulling forces on the weakened endocardium.

The management begins with the stabilization of the hemodynamic state by using drugs (inotropes, vasopressors) or even the use of an intra-aortic counter pulsation balloon. However, the latter should not delay surgical management because the evolution is fatal. The surgery is either radical: valve replacement or conservative: mitral plasty. Valve replacement can be conservative, i.e. preserving the sub valvular apparatus. Studies have shown that preservation of the sub valvular apparatus is associated with preservation of adequate contractile function and increased survival [35]. Postoperative mortality would be due to impaired contractile function or recurrence of the infarction [36].

Conclusion

Complications of MI are diverse. They can be electrical, inflammatory, embolic, ischemic and mechanical. Mechanical complications are infrequent but are fatal. Their treatment of choice remains surgery despite the advent of percutaneous measures. The diagnosis can be easy in front of the installation of hemodynamic instability going to the table of cardiogenic shock but sometimes the table is limited to chest pain or electrical changes. The management must be urgent starting with the stabilization of the vital functions then we will intervene specifically according to the pathology.

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