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Case Series

Management and Prognosis of Post-Myocardial Infarction Ventricular Septal Defect: A Case Series.

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Abstract

Background: The mortality rate of ventricular septal defect post-myocardial infarction remains considerable, even if the mortality of acute myocardial infarction is decreasing in the recent decades. Ischemic ventricular septal defect is responsible for a considerable number of deaths in the immediate aftermath of a myocardial infarction.

Cases presentation: We will illustrate this deadly complication of myocardial infarction through tree cases. Their treatment was difficult and the prognosis was bad, two out of three patients died.

Conclusion: the choice of the means of closing the ventricular septal defect and the delay remains very difficult. Even if surgical repair techniques have evolved over time, the prognosis still very poor.

Keywords: Myocardial Infarction, Ventricular Septal Defect, Prognosis.

Introduction

Ventricular septal defect in acute Myocardial Infarction (MI) is a rare but lethal complication, with a very high mortality rate (97% at 30 days of MI) [1]. It is the most common mechanical complication of myocardial infarction and usually occurs in the following week [2]. The diagnosis is made by echocardiography and the First-line treatment remains surgical. The choice of the surgical repair technique depends on the seat and the size of the septal rupture. Through the observation of three patients admitted for myocardial infarction in different localities and all complicated by septal rupture; we will focus on this entity, which remains fatal until today.

Case presentation

Case 1

We report the case of a 58-year-old man, chronic smoker and diabetic, admitted for an inferior myocardial infarction non-revascularized.

The patient had a pain of epigastric region for 11 days, associated with a dyspnea class IV of the NYHA.

Clinical examination showed regular pulse at 128 bpm at rest, a blood pressure at 130/80 mmHg, cracking rales in killip II with distention of jugular veins. The cardiac auscultation revealed the presence of a pan systolic murmur along the left sternal border of 4/6 intensity.

The electrocardiogram showed sinus tachycardia with Q waves in the inferior territory. The Echocardiography demonstrated a 29-30 mm high posterior muscular septal defect (Figures 1, 2), partially clogged by trabeculations of the right ventricle with a longitudinal septal dissection and a gradient at 44 mmHg. The left ventricle was not dilated, not hypertrophied, with a discrete apical hypokinesia (EF = 68%), the right ventricle was dilated with systolic dysfunction. The pulmonary artery pression was at 86 mmHg.

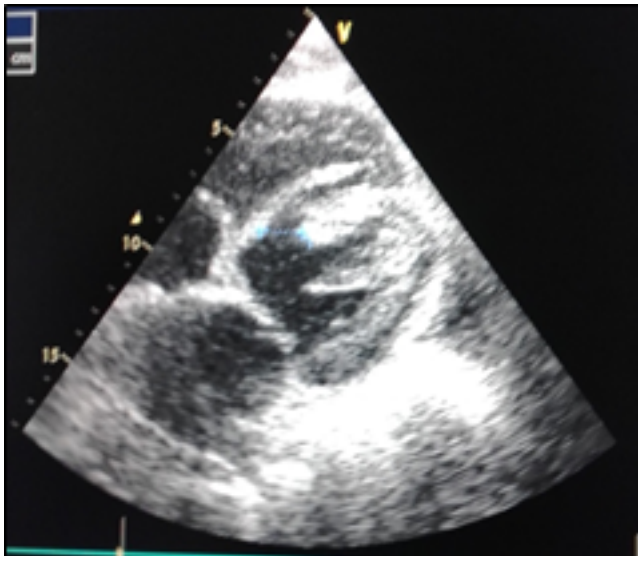


Figure 1: The echocardiographic aspect of the interventricular communication.

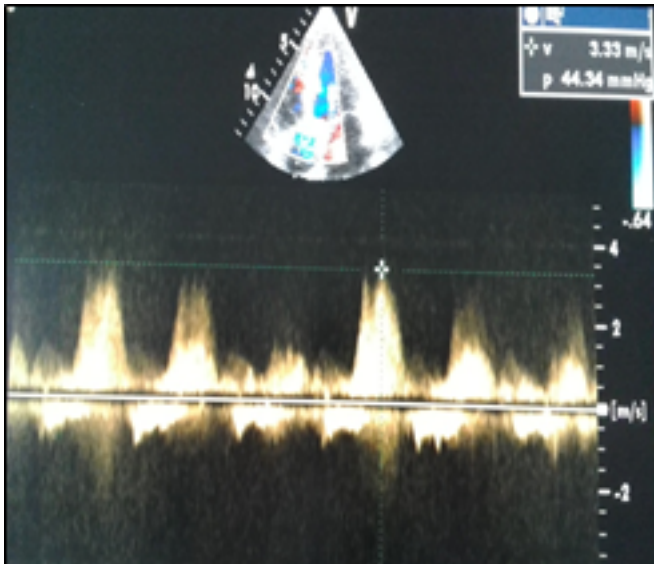


Figure 2: The aspect of the interventricular communication flow.

The coronary angiography showed significant stenosis of the middle anterior interventricular artery, the first marginal, and an occlusion of the right middle coronary artery with a thrombotic aspect (Figures 3, 4). After stabilization by medical treatment, the patient benefited from a Coronary aortic bypass with surgical closure of his ventricular septal defect. At the 7th day of post-operative, the patient died by a septic shock due to pneumonia.

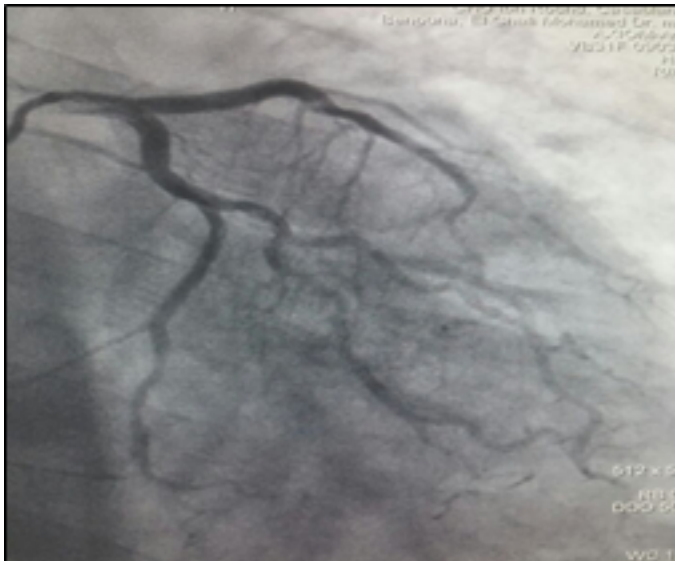


Figure 3: Significant stenosis of the middle of the anterior interventricular artery.

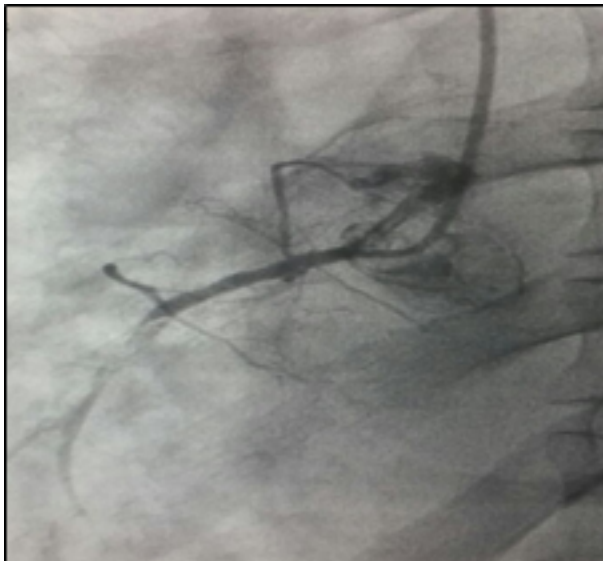


Figure 4: Thrombotic occlusion of the right middle coronary

Case 2

A 55-year-old man, hypertensive and diabetic for 10 years under oral antidiabetic, hospitalized for a myocardial infarction in antero-septo apical and inferior territory (Figure 5). Upon admission, the patient reports anginal pain that has been evolving for 4 days, as well as crescendo dyspnea.

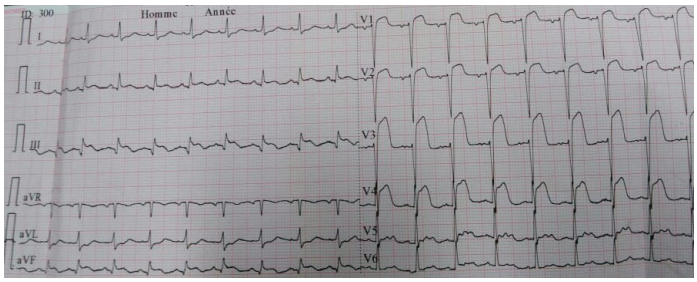
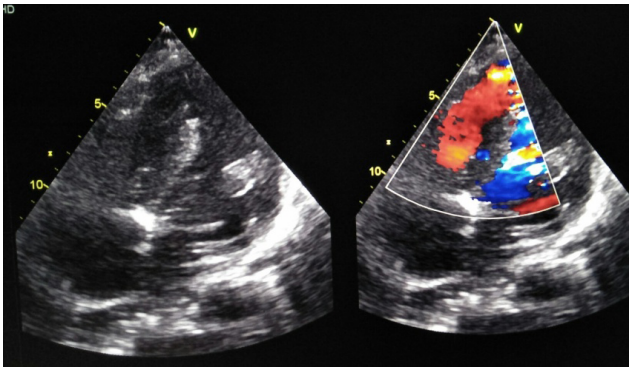
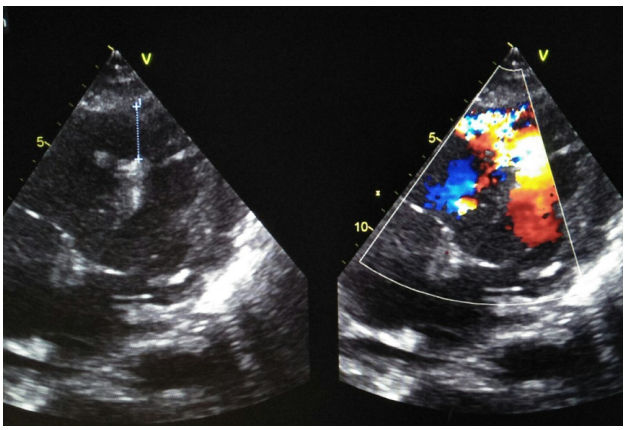


Figure 5: electrocardiogram of patient number 2.

His blood pressure was 120/70 mmHg, his heart rate was 110 bpm. Cardiopulmonary auscultation revealed a left para-sternal systolic murmur associated with crackles at the pulmonary bases. There was no sign of right heart failure.

His echocardiography showed an apical muscular septal defect of 23 mm with left-to-right shunting and a gradient at 90 mmHg (Figure 6, 7, 8) with systolic pulmonary artery pressure at 30mmHg, associated with hypokinesia of the anterior, antero-septal and anterolateral walls with a large apical akinesia and a left ejection fraction at 40%. The right ventricle was not dilated with a good systolic function.





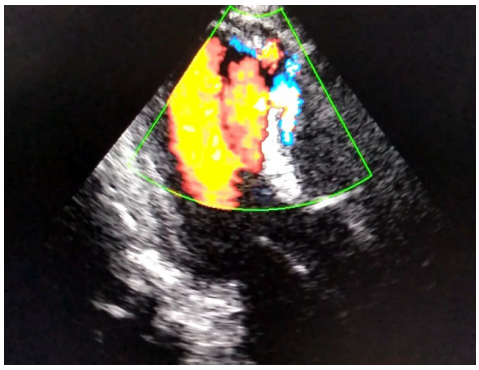


Figure 6, 7: The echocardiographic aspect of the interventricular communication.

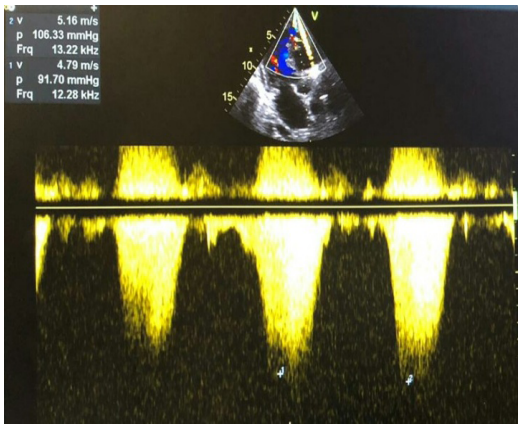
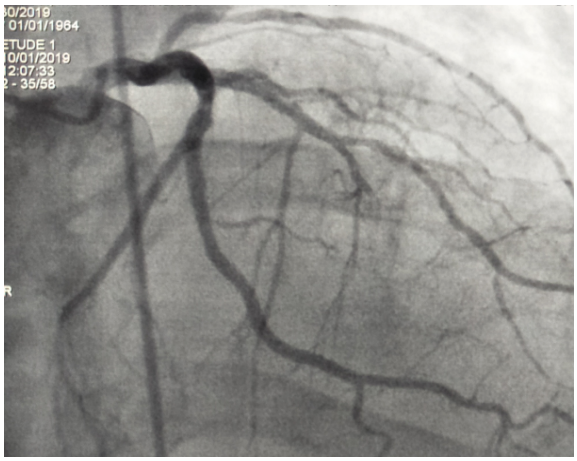


Figure 8: The aspect of the interventricular communication flow

Coronary angiography showed an occlusion of the mean anterior interventricular artery without a collateral circulation, a non-significant lesion of the first marginal and an occlusion of the right middle coronary artery (Figure 9, 10, 11).



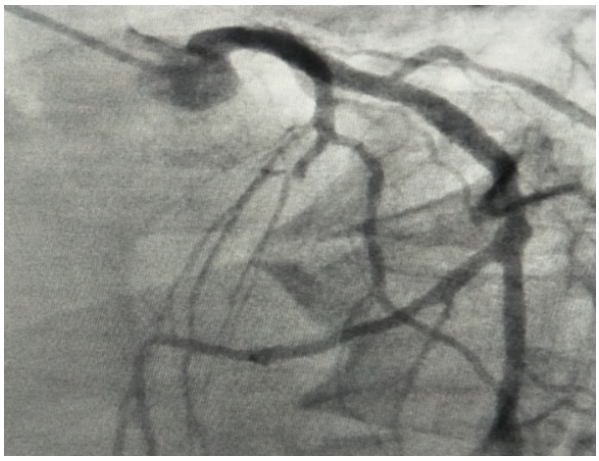


Figure 9, 10: occlusion of anterior interventricular artery

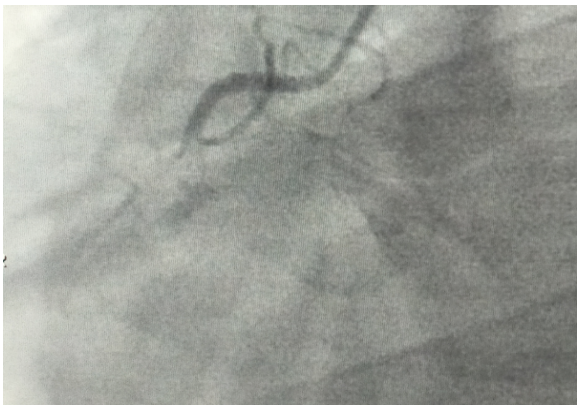


Figure 11: occlusion of the right middle coronary artery

The decision of the heart team was to monitor the patient for 3 weeks before proposing surgery, but the evolution was marked by the occurrence of cardiogenic shock in the third week of hospitalization and the patient died.

Case 3

A 61 year-old man, chronic smoker, admitted for an anterior myocardial infarction. The patient reports a chest pain for 6 days associated with exacerbating exercise dyspnea becoming at rest.

The clinical examination found a blood pressure at 90/70 mmHg, a heart rate at 114 bpm, a left parasternal systolic murmur with crackles at the pulmonary bases on auscultation, with distension of jugular veins.

The electrocardiogram showed Q waves in anterior and inferior territory (Figure 12).

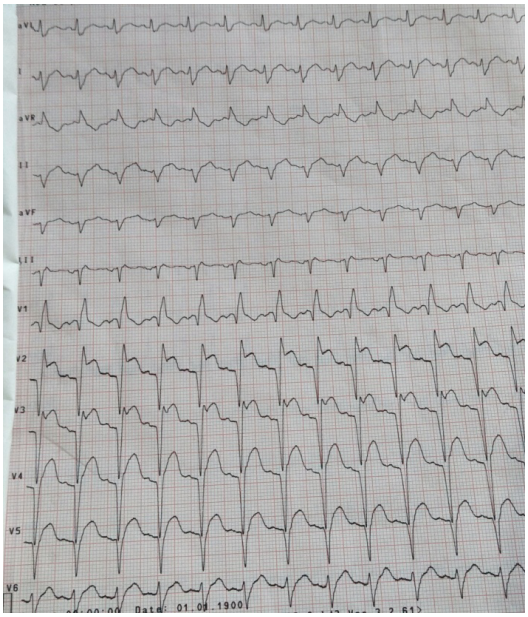
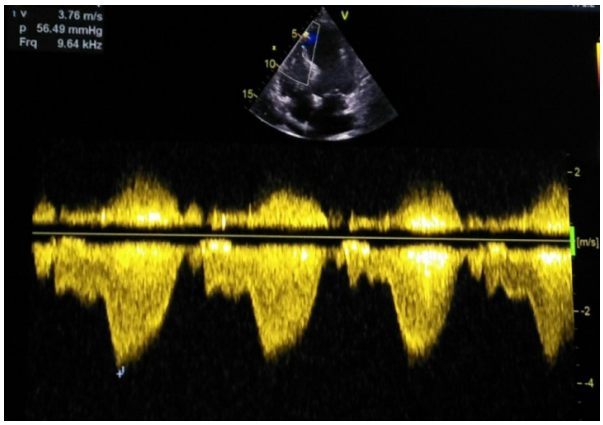


Figure 12: electrocardiogram with Q waves in anterior and inferior territory

Trans-thoracic echocardiography shows segmental kinetic abnormalities in the anterior, antero-septal and anterolateral walls as well as an apical muscular interventricular communication of 9 mm with left-to-right shunting, a trans-septal defect gradient at 35 mmHg and pulmonary hypertension at 55 mmHg. The ejection fraction was at 30% (Figures 13, 14). The right ventricle was of normal size with a mild systolic dysfunction.



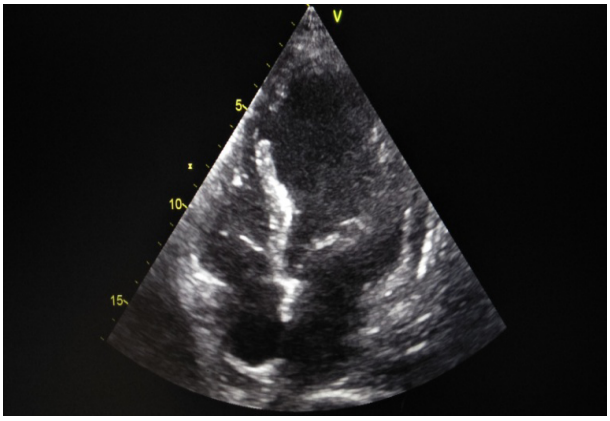


Figure13, 14: appearance of the ventricular septal defect in two-dimensional and continuous Doppler flow

The patient received anti-ischemic treatment with diuretics. His coronary angiography (Figure 15) showed an occlusion of the middle anterior interventricular artery. The therapeutic decision was to close the septal defect surgically after clinical stabilization of the patient.

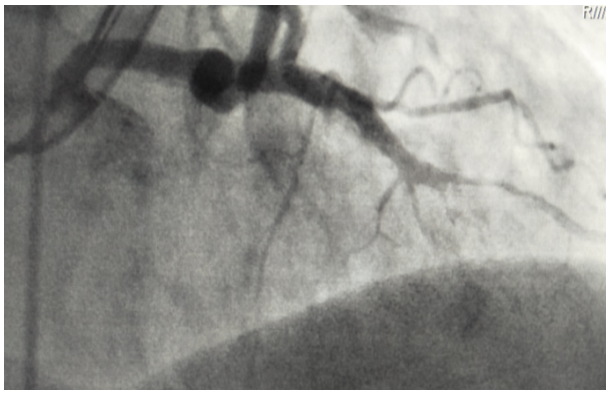


Figure 15: occlusion of the middle anterior interventricular artery in the coronary angiography.

After 3 weeks, the patient had coronary artery bypass grafting with closure of the ventricular septal defect, with a satisfactory evolution.

Discussion

Mechanical complications of acute myocardial infarction are rare and responsible for 40-80% mortality due to the cardiogenic shock, the heart failure and the difficulty of surgical repair [2].

However, their incidence tends to decrease thanks to the progress of the early revascularization. It is in the order of 0.17%-0.31% [3, 4].

We distinguish three forms of mechanical complications, occurring classically 3 to 5 days after the necrosis: free wall rupture of the left ventricle, septal rupture and mitral papillary muscle rupture [5, 6].

The main risk factors for the ischemic interventricular communication are advanced age, absence of revascularization, female sex, the transmural character of the infarct and the territory of the anterior interventricular artery (Table 1). It was also noticed

that smoking is a protective factor against this complication [7]. The limited number of cases in our serie may explain that 100% of patients (3 cases) were men.

GUSTO-I	STROCK	Anna et all
Advanced age	Advanced age	Advanced age > 70 years
Female Sex	Female Sex	female sex
Anterior Infarct	Inaugural infarct	Anterior Infarct
Absence of smoking	Tight stenosis TIMI 0/1	Tight Single Vessel Lesion
		Low Body Mass Index
		Left ventricular hypertrophy

Table 1: predictive factors of septal rupture after myocardial infarct found in the literature [7].

More than 70 % of the patients with ventricular septal defect die at the hospital, and more than 90 % after one year [1].

Myocardial rupture typically occurs 3 to 5 days after infarction, and usually occurs at the zone between the healthy and the infarcted myocardium [1]. The underlying mechanism is an intramural hematoma, which dissects the tissues and leads to rupture.

The interventricular septum has a double vascularization, which explains the two types of localization of the Ventricular Septal Defect (VSD): anterior VSD due to the occlusion of the anterior interventricular artery, which is the most frequent (60%), and the posterior VSD due to the occlusion of a dominant right coronary artery or more rarely of a dominant circumflex artery [1].

The lesion of the basal portion of the septum is rare (20-40%), and due to the inferior infarcts. It is associated with a poor prognosis, given the difficulties of surgical repair, the frequent extension to the right ventricle and the possible association with papillary muscle rupture [8, 9].

Septal rupture is more frequent in cases of acute infarction, as no collateral circulation is developed in the concerned area [10].

The diagnosis is made firstly by the clinical context: it is a recent infarct, usually with a late revascularization. Clinical examination may reveal a systolic murmur with or without hemodynamic disorders.

The final diagnosis is based on cardiac ultrasound [11], which shows the defect in two-dimensional mode and the shunt with the color Doppler.

Transthoracic echocardiography allows diagnosis in about 80% of cases [7]. The Trans esophageal echocardiography may be useful in patients with a bad echogenicity.

The main prognostic factors of septal rupture are: delayed diagnosis, the importance of systolic dysfunction, the extent of septal defect and the hemodynamic status [12, 13, 14].

Preoperative hemodynamic status and shunt size are major determinants of postoperative results [15, 16].

Closure of Post-Infarction Ventricular Septal Defects (PIVSD) decreases mortality compared to medical treatment alone and should be attempted as soon as possible after diagnosis [17].

The choice between interventional and surgical treatment should be based on factors such as the complexity of the defect, the availability of closure devices, operator skills, and the clinical condition of the patient.

Studies have shown that mortality by simple medical management was significantly higher than with surgical closure (92 ± 6.3% vs 61 ± 22%, respectively). Similarly, early closure of ventricular septal defect by transcatheter was associated with significantly lower mortality than medical treatment [17].

Although surgical closure and transcatheter have advantages and disadvantages, transcatheter can be performed in patients who are too unstable to undergo surgical repair. However, it is also limited due to complex anatomic difficulties associated with PIVSD.

Therefore, the indications for percutaneous closure remain controversial considering the complications of this technique: embolization of the umbrella, residual shunt, ventricular rupture or malignant arrhythmia. However, the recognized indications are: [18]

- A small VSD in acute period less than 15 mm.
- Residual VSD after conventional surgery.
- VSD during the subacute period more than the fourth week after the infarction

Immediate surgical repair is the recommended strategy for PIVSD and offers a better outcome than medical management alone [19, 20]; However, it can be difficult because of the clinical instability and friable heart tissue surrounding the infarct area due to the necrosis of the myocardium, then delayed surgery in hemodynamically stable patients may be considered when surgical anatomy is complex [21]

The Society of Thoracic Surgeons' national database revealed that operative mortality was 54.1% in patients who underwent early surgical repair of VSD (less than 7 days after MI), compared to 18.4% after delayed surgery 7 days later [22].

The techniques of closing the VSD differ according to its location. When the VSD is anterior, the best approach is a left ventriculotomy through the infarct, parallel to the Left Anterior Descending Artery (LAD), since it allows a complete view of the septal defect [16].

For the posterior VSD, the preferred approach is through the lower face of the left ventricular, parallel to the Posterior Descending Interventricular Artery (PDA). In that technique, there is a risk of injury to the mitral subvalvular apparatus [16].

For the surgical management of VSD, two techniques are used: Daggett and David techniques.

The Daggett procedure consists in the closure of the septal defect by using a patch [16].

The David technique is the exclusion of the infarct area and placing a patch to close the VSD [23].

At the end of the procedure, the closure of the ventriculotomy is done with a patch of Dacron.

In several studies, the David procedure is the preferred one [24].

At the end, the management of the acute ventricular septal rupture and the decision of the timing and the surgical technique require a multidisciplinary approach (Figure 16) [25].

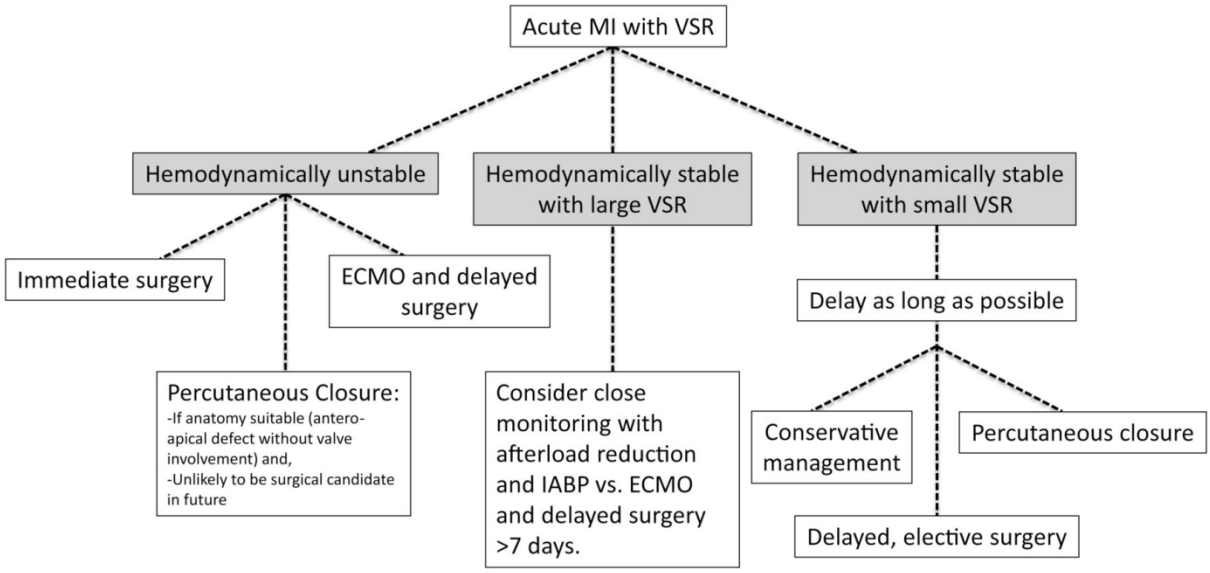


Figure 16: diagram illustrating the therapeutic management of post-infarction ventricular Septal defect.

Conclusion

Myocardial rupture is a major mechanical complication of myocardial infarction, which has become rarer with the advent of a more invasive and early coronary reperfusion strategy; however, the prognosis of this complication remains dark.

It remains the consequence of a too late reperfusion and whose treatment of choice is the surgery, which is burdened with a heavy morbidity and mortality.

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