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Ventricular Septal Rupture Complicating Acute Myocardial Infarction: Interesting Case and Review

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Clinical Case

Man of 72 years old, smoking, who presented acute myocardial infarction with ST-segment elevation (STEMI) localized in anterior wall (Figure 1) and included into the code infarction protocol at emergency room of cardiology hospital in Mexico City-IMSS. The patient was successful re-perfused by percutaneous coronary intervention of left anterior descending artery, documenting several other non-critical coronary artery disease. During its evolution in the first twelve hours, it was identified an apical ventricular septal rupture documented by echocardiogram. (Figure 2) Medical management was implemented. After stabilization patient ask voluntary discharge of hospital, 10 days later he come back due to severe heart failure. He was stabilized again and submitted to surgical reparation (Figure 3 and Figure 4) later and his heart failure was successfully resolved (Figure 4) and he was discharged in an excellent condition. We present a case and review of the literature as well as the position of management of this group of patients in the Hospital's cardiology of the National Medical Center SXXI, IMSS-México, since it is currently still controversy.

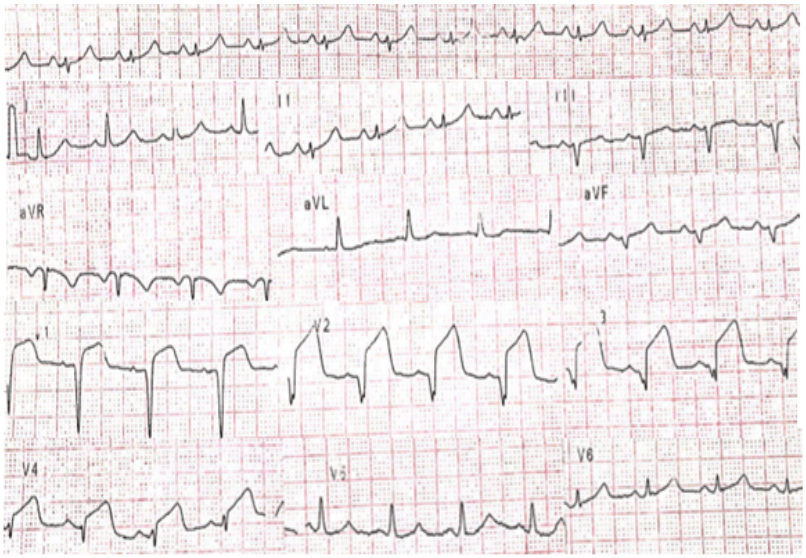

Figure 1:EKG, Acute Myocardial infarction in anterior-wall

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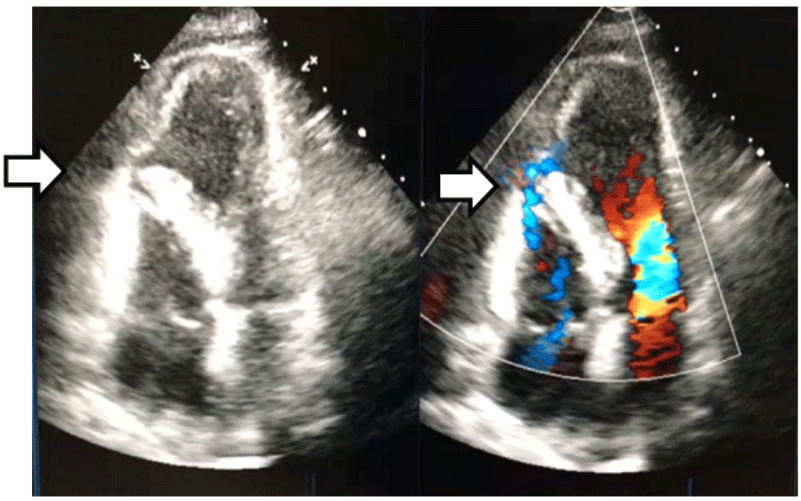

Figure 2: Four-chamber echocardiogram view. The arrows indicate the site of septal rupture

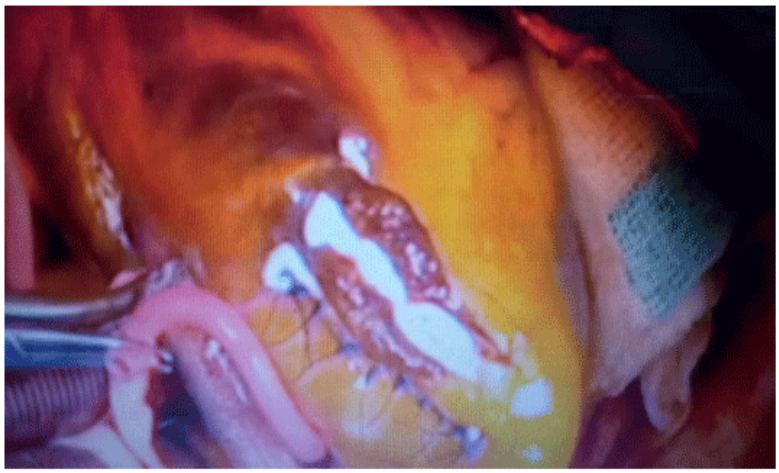

Figure 3: Ventricular geometry reconstruction

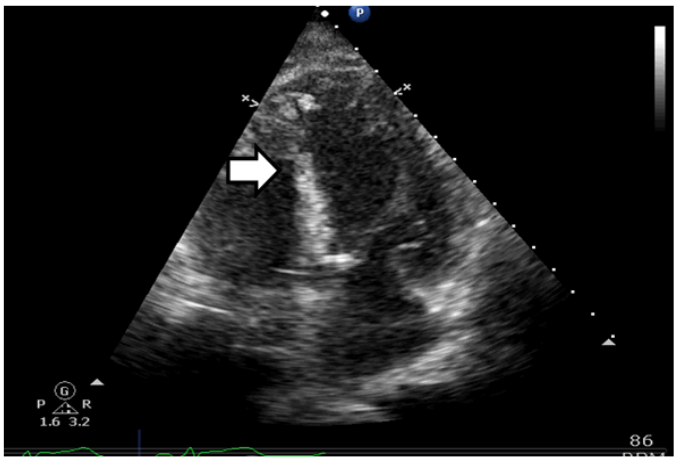

Figure 4: Four-chamber echocardiogram view. Arrow indicate the surgical reparation site

Introduction

Ventricular Septal Rupture (VSR) complicating acute myocardial infarction is a severe cardiovascular condition that can be considered similar to cardiogenic shock in terms of mortality. Although its incidence has declined in recent years, the mortality associated to this condition, remains considerably high.

The incidence in the pre-reperfusion era was reported in ~1-2%, currently with different reperfusion methods is considered a rare complication, with an incidence of 0.17% to 0.31% [1, 2]. In Mexico, the RENASICA II reported an incidence of 0.3% on ST-segment elevation myocardial infarction [3].

Mortality is described from 10% in elective surgery after 21 days to 60% with surgery in the first 24 hours, and 94% with medical treatment only [4].

Depending on the evolution of coupled with injury by reperfusion treatment as the platelet anti-aggregation and anticoagulant therapy described a trend toward earlier detection of this complication, the identification was described in 3 to 5 days the index event, in 2000 after the SHOCK study reported an average of 16 hours for the diagnosis of the same [5].

Independent risk factors for the presentation of this complication are: old age, female sex, heart failure, cerebral vascular event and nephropathy; besides the delay in time guide, door- needle, prolonged infusion of the fibrinolytic and Killip-Kimball 3 an 4 degrees are associate with increased incidence [6].

Pathophysiology

The primary cause of Ventricular Septal Defect (VSD) complication has its origin in the transmural infarction of the interventricular septum; the classical mechanism is coagulative necrosis with infiltration of Neutrophilic, in which intervenes significantly metalloproteinase matrix extracellular 9 (MMP-9), this process requires approximately 3 to 5 days. In the case of rupture occurring in the first 24 hrs, its histological behavior is from the formation of an intramural hematoma or hemorrhage of the ischemic myocardium, with subsequent tissue dissection secondary to the shear forces in the infarcted zone boundaries [1].

The location of the infarction (above or below) is not significantly associated with the risk of RSIV, although yes it associated with acute infarction regional anatomical site; previous infarctions are complicated by more frequently with apical defects and the bottom or sides with basal defects [2].

Becker and Mantgem classified the rupture of the free wall of the left ventricle which is applicable to the RSIV: type I involves defects that occur in less than 24 hours of the event index with a presentation that is abrupt in the form of cleft, type II or sub acute which s (e) generated mainly by erosion of the myocardium and type III that is usually accompanied by concomitant formation of aneurysm, with thinning of the wall and subsequent rupture. According to its complexity it is classified into simple when there is direct communication between both cameras and complex when there is a serpentine ride and irregular with indirect communication between both Chambers, is due to hemorrhage and multiple septal defects [3, 7].

Diagnosis

All patients in the context of acute coronary syndrome should be evaluated clinically and according to findings on Auscultation is a tough blow, holosystolic in mesocardio, which can be accompanied by thrill. Other finds are 3rd noise left or right and strengthening of the pulmonary component of the second noise. The low cardiac output is a condition not to find characteristic findings in the physical examination.

Chest x-ray shows both hypertension venocapilar from grade II to acute Lung edema. The electrocardiogram in evidence is important to determine the location of acute infarction, as well as possible variations in atrioventricular conduction until locks branch.

Transthoracic Echocardiogram and, in particular, the approach to Transesophageal is a manoeuvre that must be done to get better definition of the form and magnitude of the short. In addition we can in an irrefutable manner to confirm the clinical suspicion, an adequate assessment of left ventricular systolic and diastolic function and regional mobility disorders, calculate the magnitude of the short from left to right, define the type and size of the defect and evaluate the existence of other associated mechanical complications. If the patient has a pulmonary artery catheter in diagnosis with the demonstration of a oximetry jump greater than 3 volumes per cent between the right atrium and right ventricle [3, 8].

Treatment

The definitive treatment of choice is surgical closure of the defect, the first intervention in this context is reported in 1957 by Denton Cooley; There is a relationship of inversely proportional mortality reported in the literature as to the moment of intervention with respect to the index event; of the society of Thoracic Surgeons database reports that on average it has a mortality rate of 42.9%, with up to 54% of mortality in the first 7 days of the index event and 18.4% when the procedure is performed after the first week, this being the surgery card IACA with increased mortality currently [9, 10].

There are different recommendations on time optimal for the closure of the defect, the American guide of myocardial infarction with ST-elevation in its section on complications recommended surgical emergency treatment regardless of the patient's hemodynamic status. On the other hand taking into account the benefit of the delay in the surgical time European 2017 guide promotes the surgical time delay elective treatment in patients who respond to conservative treatment initially [11, 12].

The theoretical basis of this appearance is clear of the pathophysiology of myocardial scarring; MMP-9 activity has its peak at day 7, deposition of collagen starts between days 2 and 4 and necrotic myocardium is entirely replaced by collagen at 28 days approximately [13].

The option to surgical treatment is Percutaneous treatment, originally described in 1988 by Lock et to the., most likely to close defects are minors to 15mm, simple, medium to apical septum and not involving the subvalvular device, there are now devices specially designed for the closure of interventricular septal defect examples (Amplatzer PI) myocardial infarction. The mortality reported in a meta-analysis of case series oscillates around 32%, with a success rate of closure of 89 %. There is an association between the clinical status of the patient with mortality, reaching more than 50% in patients that are operated in cardiogenic shock [14, 15].

In regards to the medical treatment, this is addressed primarily to the post-load decrease in order to limit the consumption of myocardial oxygen and reduce the severity of the short, the drugs most used are sodium nitroprusside and Levosimendan [3]. A line in the decisions for the patient with RSIV algorithm is the implantation of ventricular assist devices. Ventricular support that has been most frequently used in these patients is the aortic balloon counter pulsation with up to 65% of patients requiring ventricular support [10].

The indication for the use of the same is IIA according to European guide on its section of mechanical complications post infarction, the grade of recommendation for the use of mechanical circulatory support in the short term as a bridge to recovery is IIA [12, 16].

However, we advise against the use of Intra-Aortic Balloon since after 48hrs there is arterial vascular complications and what we seek is to endure it for at least seven days. So we suggest the use of extracorporeal oxygenation membrane of preference (extracorporeal membrane oxygenation [ECMO]), which aims to stabilize the patient. The use of the Impella 5 device - 0/L has been reported as a bridge to surgery with a mortality rate of 40% after 30 days. The Tandem Heart device is a management option; the date forecast reports are infrequent. The treatment approach that we suggest is described in figure 5.

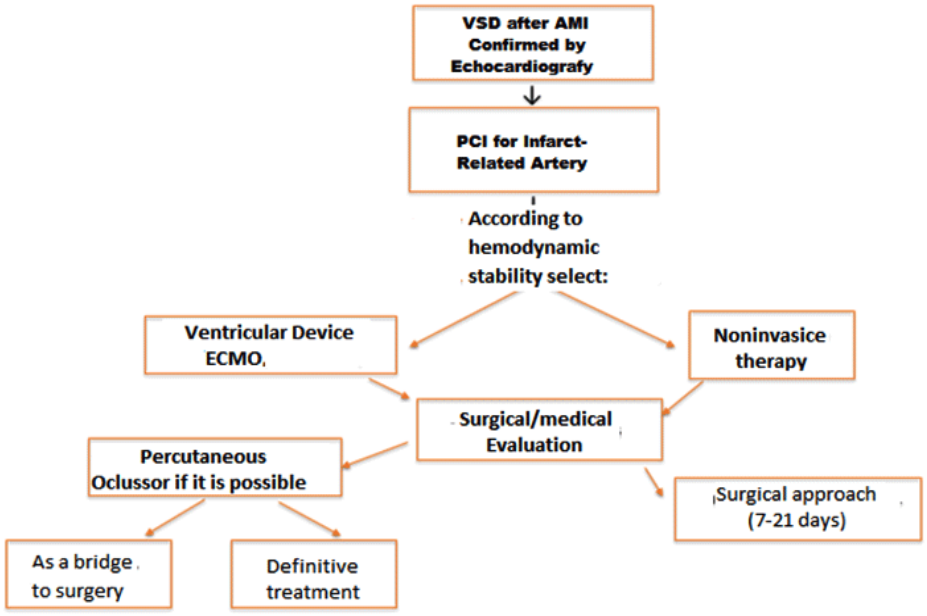

Figure 5: Algorithm showing the recommended clinical route in case of ventricular septal rupture post AMI

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