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VENTRICULAR SEPTAL DEFECT COMPLICATING ANTERIOR ACUTE MYOCARDIAL INFARCTION: CASE REPORT AND REVIEW OF THE LITERATURE

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ABSTRACT

Early and effective reperfusion of acute myocardial infarction (MI) has resulted in a substantial decline in the incidence of mechanical complications, including ventricular septal rupture which is frequently associated with cardiogenic shock (12% of patients). Hemodynamic stabilization, in most cases using an intra-aortic counter-pulse balloon, is the first step in the care. Then, the decision of a surgical or percutaneous closure and its timing should be evaluated within the heart team, allowing a decrease of mortality to 30-40% at 30 days.

We present a case of anterior AMI complicated by apical VSD and cardiogenic shock, highlighting the role of echocardiography in the diagnosis of this critical complication.

Key words: acute myocardial infarction, echocardiography, ventricular septal rupture.

I. INTRODUCTION

Post-myocardial infarction (MI) ventricular septal defect (VSD) is a rare but potentially catastrophic mechanical complication, it is caused by necrosis of the heart muscle, leading to acute heart failure, cardiogenic shock (CS), and high mortality rate. The incidence has reduced in reperfusion era (0.17%–0.31%), but it remains a life-threatening event. Surgical repair was considered the only definitive treatment. (1) Over the last decade, percutaneous closure (PC) has emerged as an alternative strategy, with results similar to cardiac surgery. Echocardiography plays an essential role for

diagnosis, in guiding percutaneous procedures and follow-up. We present a case of a 69-year-old man with ST-elevated anterior AMI, complicated by apical VSD and cardiogenic shock.

II. CASE REPORT

A 69-year-old man was hospitalized for acute dyspnoea occurred one week before his admission worsening gradually with abdominal pain and vomiting. He had a history of blood hypertension, liver steatosis, without any other cardiovascular risk factors.
Physical examination revealed a grade 4/6 holosystolic murmur at the left lower sternal border with radiation across the precordium and clinical features of congestive heart failure and cardiogenic shock. His systolic blood pressure was 90 mmHg with oliguria, cold, and poorly perfused extremities requiring inotropic agents administration and intravenous (IV) diuretics.
The patient presented a rapid worsening of his respiratory distress with hemodynamic instability which led eventually to intubation and ventilation.
The electrocardiogram (EKG) showed sinus rhythm with ST elevation and pathological Q-waves in the anterior leads (**figure 2**). Chest x-ray provided evidence of a bilateral interstitial edema **figure 3**).
A bedside echocardiography revealed akinesia of the inferior and anterior walls with reduced left ventricle (LV) ejection fraction (EF: 36%). The right ventricle had a systolic dysfunction (tricuspid annular plane systolic excursion:10mm). The echo also revealed an apical VSD, measuring 22mm*10mm, with left-to-right shunting, and pulmonary hypertension
(pulmonary artery systolic pressure: 62mmhg) (**figure 4,5**). Anterior acute myocardial infarction with RV involvement, ventricular septal defect and cardiogenic shock was diagnosed.
The blood tests showed anaemia (hg :9.5) kidney failure (creatinine: 41.3 mg/l, creatinine clearance: 15 ml/mn).
A dual antiplatelet therapy with anticoagulant therapy were administrated, statin was suspended because of history of liver steatosis, angiotensin-converting enzyme inhibitor and betablockers were suspended because of kidney failure and cardiogenic shock. IV diuretics were administrated with inotropic agents.

Both Cath lab and the cardiac surgery teams were informed about the condition of the patient to perform immediate coronarography with eventually correction of the septal defect but because of refractory shock the patient died before angiography could be done.

III. DISCUSSION

The incidence of ventricular septal rupture complicating acute myocardial infarction is approximately 0.2% in the reperfusion era.(2) Before the introduction of reperfusion therapy for MI, the incidence was 0.5% to 2%. (3,4)
Risk factors for the development of VSD and subsequent mortality include hypertension, elevated body mass index, anterior wall AMI, increased age, female gender, first AMI, single vessel occlusion, and absence of smoking history (8-12).

Although it was previously thought that the incidence of septal rupture increased with thrombolytic therapy, placebo-controlled trials failed to confirm an increased risk of rupture with thrombolytic therapy(5,6). Early occurrence of ventricular septal rupture has been observed, however, after thrombolytic therapy. (7)

VSD generally develops about 3–5 days after AMI onset. This interval has decreased in reperfusion era .VSD has been more often observed in patients with total occlusion of the infarct-related artery and minimal collaterals, generally in a context of single-vessel disease, after anterior AMI. (13)

RV dysfunction, development of CS, and early surgery have been reported as significant independent predictors of mortality. (1)

Transthoracic echocardiography and transoesophageal echocardiography provide informations about: * VSD site, dimensions, and relation with surrounding myocardial wall, ** shunt amount, and *** ventricular function.

The clinical features of the patients presenting with VSD commonly involve cardiogenic shock and fulminant pulmonary oedema, which necessitate the use of non-invasive ventilation (NIV) or definitive airway management.

Vasopressors, such as norepinephrine, are needed in hypotensive patients to improve peripheral perfusion and ensure adequate coronary blood flow to maintain adequate cardiac output for end-organ perfusion (14, 15, 16). Non-pharmacologic measures for afterload reduction include the use of an intra-aortic balloon pump (IABP) which also augments coronary blood flow, impella placement, and veno-arterial extracorporeal membrane oxygenation (ECMO), which also augments cardiac output (18-21) IABP decreases afterload, thereby reducing the degree of left to right shunt, and increasing cardiac output, systemic pressures, and end-organ perfusion. Hemodynamic optimization should not delay definitive treatment via percutaneous closure device or primary surgical treatment (14, 15,23).

Both STEMI ESC guidelines (24) and ACC/AHA guidelines (25) recommend an emergency or early surgical intervention. However, several studies suggest to delay surgery: Serpytis *et al*.(12) reported 100% survival rate in patients surgically treated after 3–4 weeks of stabilization versus 100% mortality in patients having surgery within 10 days from symptoms onset.

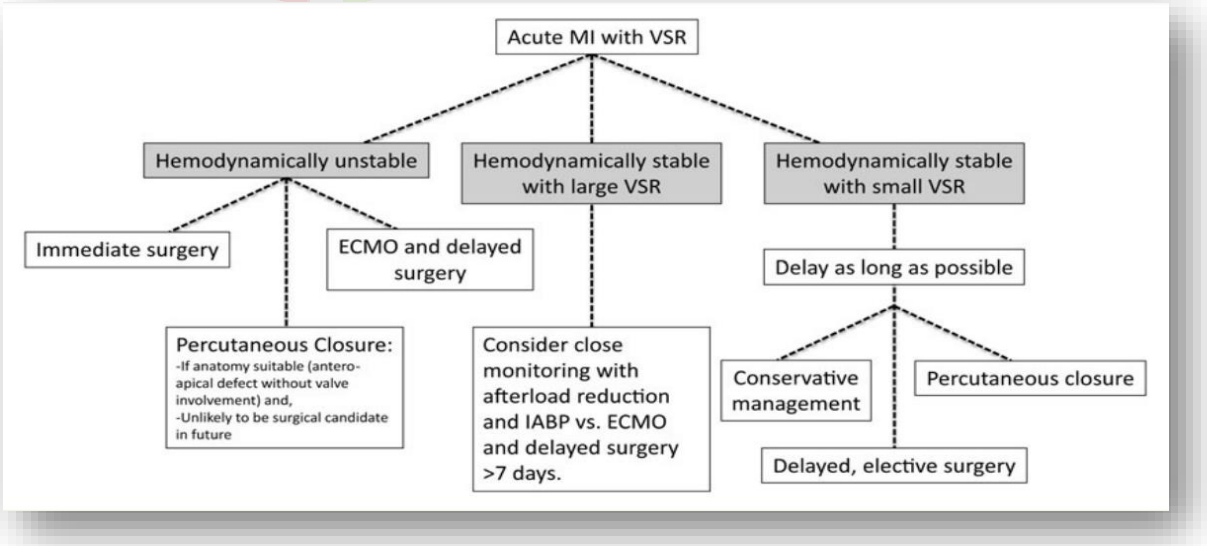

Figure 1. A multidisciplinary approach for managing acute ventricular septal rupture(27)

Thirty day mortality for patients with VSR approaches 87% in those with VSR and concomitant cardiogenic shock, and VSR is always fatal in patients who do not receive operative repair (21, 26).

In our case, and in the absence of non-pharmacological measures, vasopressors alone could not improve circulatory collapse and the patient died rapidly after his admission.

IV. CONCLUSION

Despite being a rare complication of acute myocardial infarction. VSR is a serious surgical emergency that requires urgent diagnosis and rapid intervention for optimal outcomes. Rapid diagnosis followed by emergent surgical treatment is crucial for prognosis improvement. Optimization of hemodynamic condition with IABP and vasodilators may help to reduce the afterload on the compromised ventricle following acute myocardial infarction and improve cardiac output in the short term, but should not delay surgical intervention.

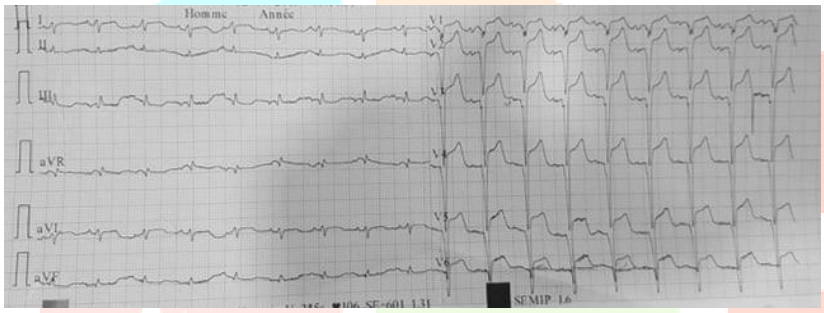

Figure 2: Electrocardiogram showing ST-elevation with pathological Q-waves in the anterior leads.

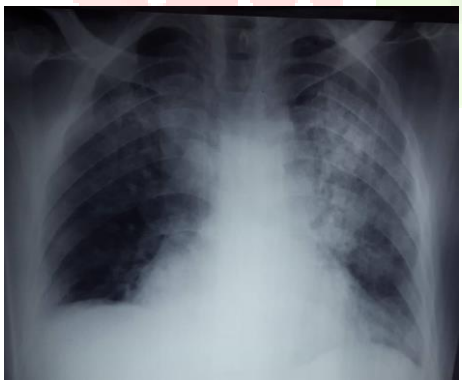

Figure 3: Chest X-ray demonstrating a bilateral interstitial edema

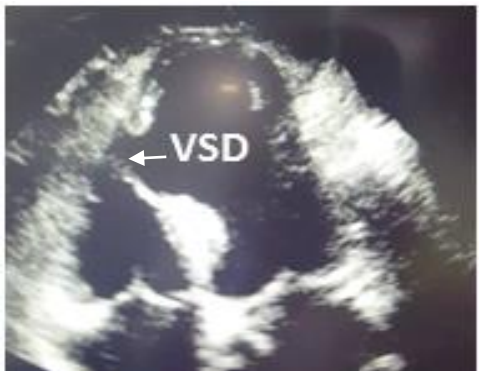

Figure 4: two-dimensional transthoracic apical four-chamber view showing the ventricular septal defect (arrow)

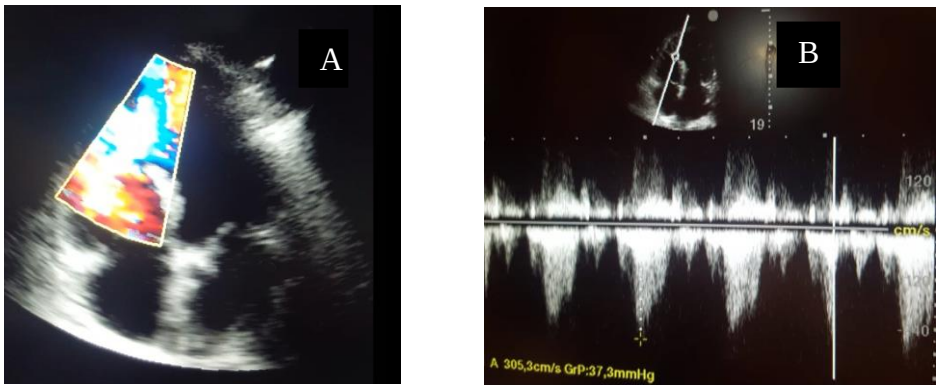

Figure 5: A : two-dimensional transthoracic apical four-chamber view on color doppler shows the left-to-right shunt.
B : two-dimensional transthoracic apical four-chamber view on continuous-wave doppler shows a left to right shunting across the VSD with a gradient about 37,3 mmHg .

Competing interests

The authors declare no competing interest.

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