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INTERNATIONAL JOURNAL OF ADVANCED RESEARCH (IJAR)

Article DOI: 10.21474/IJAR01/15149
DOI URL: http://dx.doi.org/10.21474/IJAR01/15149

RESEARCH ARTICLE

ANTERIOR ACUTE MYOCARDIAL INFARCTION COMPLICATED WITH VENTRICULAR SEPTAL RUPTURE: CASE REPORTS AND LITERATURE REVIEW

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Manuscript Info

Manuscript History
Received: 05 June 2022
Final Accepted: 09 July 2022
Published: August 2022

Key words:-
Acute Myocardial Infarction, Echocardiography, Ventricular Septal Rupture

Abstract

Post-myocardial infarction (MI)ventricular septal defect (VSD) is a mechanical complication that has become infrequent since the advent of reperfusion strategies; however, it is associated with cardiogenic shock in 12% of cases and represents a significant mortality factor. Surgical treatment is often mandatory in the early period after anterior myocardial infarction(AMI) .Thus, early and appropriate management can reduce 30-40% mortality at 30 days.(1) We present three cases of anterior myocardial infarction complicated by apical VSD, emphasizing the major role of echocardiography in the diagnosis of this fatal complication.

Introduction:-

Among the mechanical complications of post-myocardial infarction is the interventricular communication which is infrequent but has a poor prognosis.Thanks to reperfusion, its incidence has decreased but it remains fatal. It occurs 2-8 days after the infarction and often precipitates cardiogenic shock(2). Surgical repair was considered the gold standard treatment until the last decade when percutaneous closure emerged as an alternative strategy with similar results. Diagnosis is based on echocardiography, which is also useful for guiding percutaneous procedures and follow-up.

We report three cases of patients admitted for anterior myocardial infarction complicated by interventricular communication.

Case 1:
A 69-year-old hypertensive man was hospitalized for acute dyspnea occurring 1 week before admission.

Physical examination revealed a grade 4/6 holosystolic murmur at the left lower sternal border and clinical signs of congestive heart failure and cardiogenic shock with a systolic blood pressure of 90 mmHg, cold extremities and oliguria. The patient was eventually intubated based on hemodynamic and respiratory criteria.

Electrocardiogram (EKG) showed sinus rhythm with ST segment elevation and pathologic Q waves in the anterior leads. Chest x-ray revealed a bilateral interstitial edema.

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Echocardiography revealed inferior and anterior wall akinesia with reduced left ventricular ejection fraction (EF: 36%). The right ventricle was in systolic dysfunction. The ultrasound also revealed an apical VSD, measuring 20mm*10mm, with a left-to-right shunt. (Figure 1)

Blood analysis showed anemia (Hg: 9g/dl) and renal failure (creatinine: 41 mg/l, creatinine clearance: 15 ml/min).

Anti-ischemic medical treatment was initiated, the catheterization and cardiac surgery teams were informed of the patient's condition in order to proceed to immediate coronary angiography with possible correction of the septal defect, but due to refractory shock, the patient died before angiography could be performed.

Case 2:
A 65-year-old patient, with active smoking as a cardiovascular risk factor, who presented 2 days before admission with chest pain complicated by angina recurrences. On clinical examination, the patient was hemodynamically and respiratory stable, with a grade 4/6 holosystolic murmur perceived at the left sternal border and maximum at the xiphoid process, increased by inspiration. The blood pressure was 12/70mmhg with an oxygen saturation of 96%, the rest of the clinical examination was unremarkable.

The EKG showed ST segment elevation with necrosis Q waves in the anterior leads.

Echocardiography revealed akinesia of the apex and adjacent segments with a (EF: 47%). It also revealed a septo-apical, restrictive, muscular VSD with a left-to-right shunt (Figure 2, 3).The CRP was 67mg/l with a troponin >50000 ng/l.

Coronary angiography revealed a severe lesion in the anterior interventricular artery

After stabilization by medical treatment, the patient benefited from surgical closure of the VSD with stenting of the responsible artery with good outcome.

Case 3:
60-year-old patient, chronic smoker, admitted for infarct chest pain for 3 days. On clinical examination the patient had a blood pressure of 90/62 mmhg, a holosystolic murmur of VSD, without signs of heart failure.

The EKG showed a circumferential ST segment elevation with necrotic Q waves (Figure 4). The echocardiography showed an aspect of ischemic heart disease with diffuse akinesias, an EF of 35%, and a double apical restrictive VSD of 6 and 4 mm (Figure 5). At workup, troponin was >50,000 ng/l.

The coronary angiography was in favor of a very tight stenosis of the marginal artery which benefited from the placement of a stent. The territory of the anterior interventricular artery and the right coronary artery was nonviable. The VSD was then managed surgically with a good outcome.

Discussion:-

The incidence of ventricular septal rupture complicating acute myocardial infarction has decreased since the introduction of reperfusion therapy to approximately 0.2% (compared with 2%) (3,4).

Risk factors for VSD and therefore mortality include high body mass index, arterial hypertension, advanced age and female gender, anterior wall ischemia, and single vessel occlusion (5,6).The right ventricular dysfunction, the presence of CS, and early surgery has been described as significant independent predictors of mortality. (7)Most often, VSD is found in the context of previous AMI by total occlusion of the responsible artery, with minimal collaterals (8)

We describe three types of VSR (the classification made by Becker and van Mantgem): in type I, there is a tear in the wall without thinning. In type II, the infarcted myocardium erodes before rupture and is covered by a thrombus. Finally, type III represents perforation of an already formed aneurysm(2).

Transthoracic echocardiography and transesophageal echocardiography have a role in localizing the VSD. It is observed that anterior MI is related to rupture of the apical septum, whereas in inferior infarction, it often develops at the base of the heart. MI associated with VSR is usually extensive(10).

Echocardiography has also a purpose to measure VSD dimensions, its relation to the surrounding myocardial wall, the degree of shunt, and the ventricular function.

Cardiogenic shock and pulmonary edema are not uncommon in VSD, and require the use of noninvasive ventilation or definitive airway management.A few cases of silent AMI complicated by asymptomatic VSD or manifesting as congestive heart failure have been described. The size of the VSD determines the importance of the left-right shunt and therefore the hemodynamic impact of this shunt as well as the survival.(11)

Concerning the therapeutic component, Vasopressors, such as norepinephrine, are intended to provide coronary blood flow by enhancing perfusion to maintain sufficient cardiac output for organ perfusion (9, 12, 13).

Coronary blood flow can also be provided by an intra-aortic balloon pump (IABP), which tends to decrease afterload as well as the degree of cardiac shunt always with the aim of ensuring a good cardiac onset and thus better target organ perfusion (14,15). ECMO is also used in this perspective.

These therapiesshould not delay definitive treatment with percutaneous closure device or primary surgical treatment (9, 12, 16). Indeed, early and urgent surgical intervention is recommended.However, many studies advocate delaying surgery: Serpytis et al(6) reported a 100% survival rate in patients treated surgically after 3-4 weeks of stabilization compared to 100% mortality in patients operated within 10 days of onset.

Conclusion:-

The prognosis of post-infarction VSD is poor, and surgical management of post-infarction ventricular septal defects is recommended to improve patient survival.

Rapid diagnosis followed by urgent surgical management is crucial to improve prognosis. Optimization of hemodynamic status helps to improve cardiac output, but should not delay surgery.

Figure Legends

Figure 1: 2D transthoracic apical four chambers view showing the VSD.
Figure 2: Apical short axis view showing the VSD.
Figure3:Apical four chamber view showing a L-R shunt through the VSD on color Doppler.
Figure 4: EKG showing a circumferential ST segment elevation with necrotic Q waves.
Figure 5:Color Doppler on a VSD showing a L-R shunt in apical four chamber view.

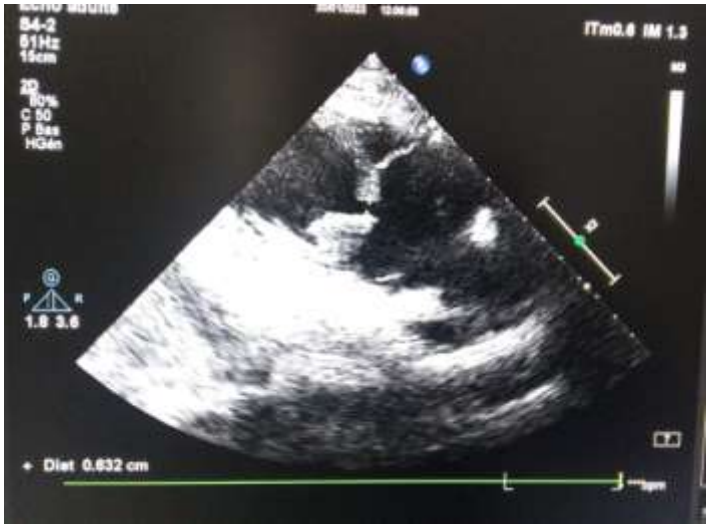
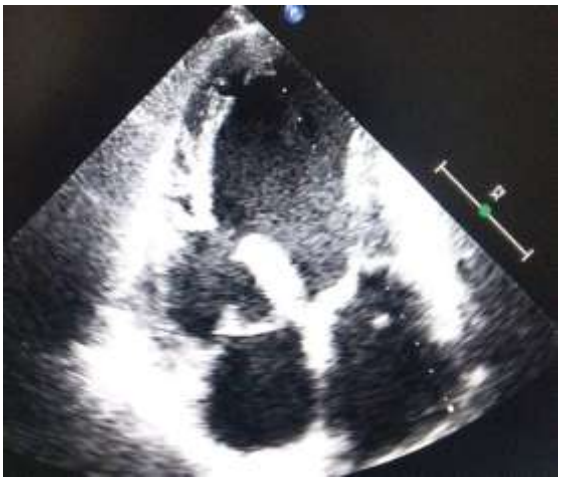


Figure 1:-



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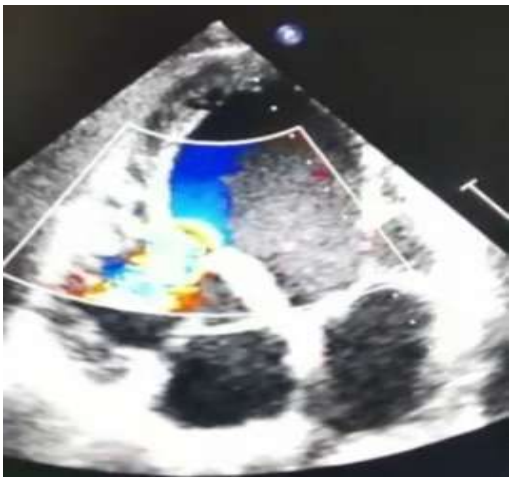

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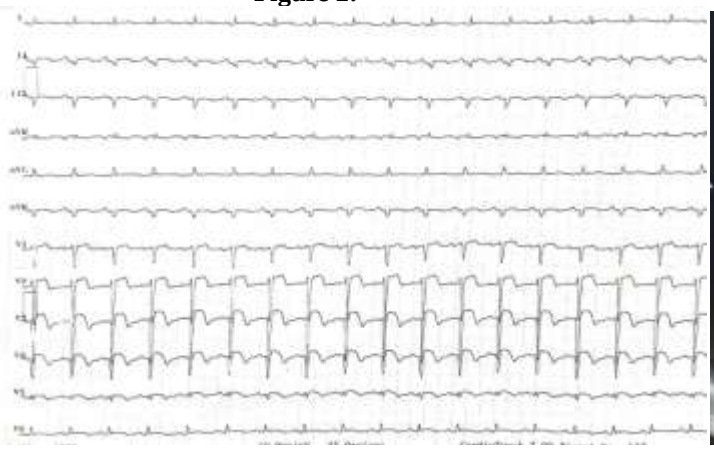

Figure 4:-

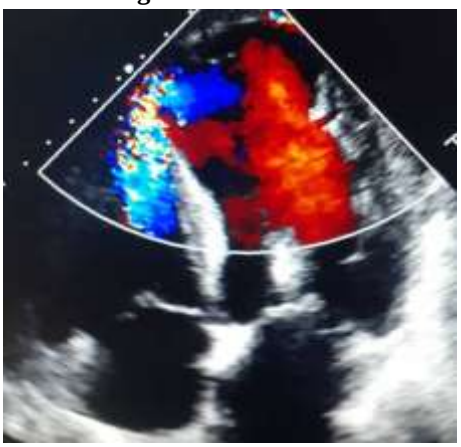

Figure 5:-

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