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Case Report

Ventricular Septal Defect Post-Myocardial Infarction: About 5 Cases.

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Abstract:

Post-myocardial infarction (MI) ventricular septal defect (VSD) is a rare but potentially catastrophic mechanical complication that occurs in less than 1% of patients following a myocardial infarction and it is associated with a high morbidity and mortality despite improvements in medical and surgical therapies of revascularization. Our article is based on the study of the occurrence of the VSD after a myocardial infarction in 5 patients. With an average age of 60 years, the prognosis was good if the rupture was small and the patient was hemodynamically stable. VSD tends to occur within the first week after acute myocardial infarction. In most cases, there is an immediate decline in hemodynamics which can lead to cardiogenic shock. VSD is a surgical emergency needing immediate treatment in symptomatic patients. The procedure requires the closure of the VSD and coronary artery bypass grafting.

Keywords: ventricular septal defect (VSD) myocardial infarction (MI), surgery, coronary artery bypass grafting

Introduction:

Ventricular septal defect (VSD) is a rare complication of myocardial infarction (MI), it occurs in only 0.2% of cases. The diagnosis is clinical and echocardiographic [1].

Based on 5 observations of post-MI VSD, we will discuss the predictive prognostic factors and therapeutic management, particularly the timing of surgery in this entity.

Case reports:

Case 1:

67 year old male, diabetic patient admitted on day 6 with an antero-septo-apical MI related to a monotroncular lesion of the anterior interventricular artery (AIV) complicated by an apical thrombus and a left heart failure (LV EF: 26%).

The evolution was marked by the sudden onset of cardiogenic shock on day 18 of the MI. Cardiac auscultation revealed the appearance of a new holosystolic murmur in wheel radius. It led to an echocardiogram which revealed a recent muscular septal defect, non-restrictive, of 1.4cm (v max: 2.6m/s). The patient was initially put on vasoactive drugs and then underwent surgery for closure of VSD and mono-bridging. He died postoperatively.

Case 2:

61 year old male diabetic patient admitted on day 15 with circumferential MI related to AIV artery and right coronary involvement complicated by left heart failure (LV EF: 45%), LV aneurysm and restrictive apical muscular VSD (vmax: 4.1 m/s) of 1.5 cm partially closed by the trabeculations of the RV. After stabilization by the medical treatment the patient benefited from a cure of the aneurysm a mono-bridging of the right coronary and a closure of the VSD. The outcome was favorable.

Case 3:

56 year old male patient with active smoking status, admitted on day 2 with deep septal MI related to bitroncular involvement of AIV artery and the second marginal. He underwent angioplasty with bare stenting of the AIV. On day 3, following a state of cardiogenic shock, the diagnosis of a 6 mm non-restrictive apical VSD was made, leading to surgery on day 4 with good evolution**.**

Case 4:

77 year old male patient with chronic active smoking and type 2 diabetes on diet. Admitted at H4 for a STEMI related to an anterosepto-apical ST-segment elevation which undergone successful thrombolysis at H6. Coronary angiography showed tritruncal lesions (a tight lesion of the AIV, a long tight lesion at the level of the 2nd marginal and a lesion of 70% in the 2nd segment of the right coronary). The TTE showed a serpentine apical ventricular septal defect (double tract) measuring 5 mm in diameter and an apical aneurysm of the left ventricle. The patient remained hemodynamically stable which deferred surgical

treatment.

Case 5:

84 year old female patient, followed for arterial hypertension on treatment, admitted on day 8 of an extensive non-revascularised anterior MI, complicated by flare-up of left ventricular failure, managed with medical treatment. A TTE performed showed a 6mm apical VSD with a left/right shunt associated with an apical aneurysm and moderate LV dysfunction with an LVEF of 42%. The patient remained clinically stable and was then referred for surgery.

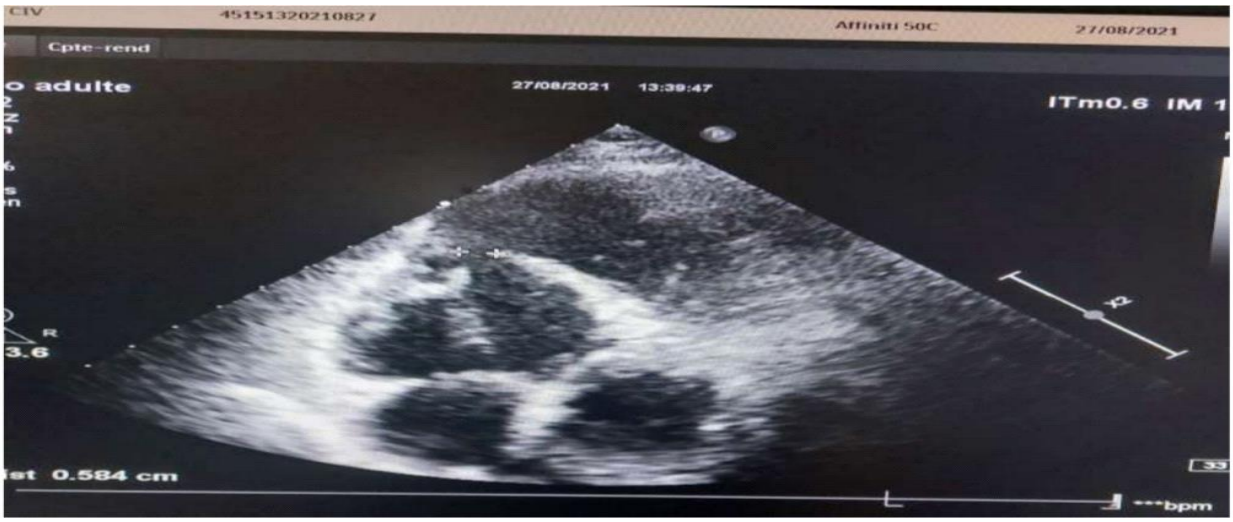

Image 1: 4-cavity section showing an apical VSD with a serpentine path

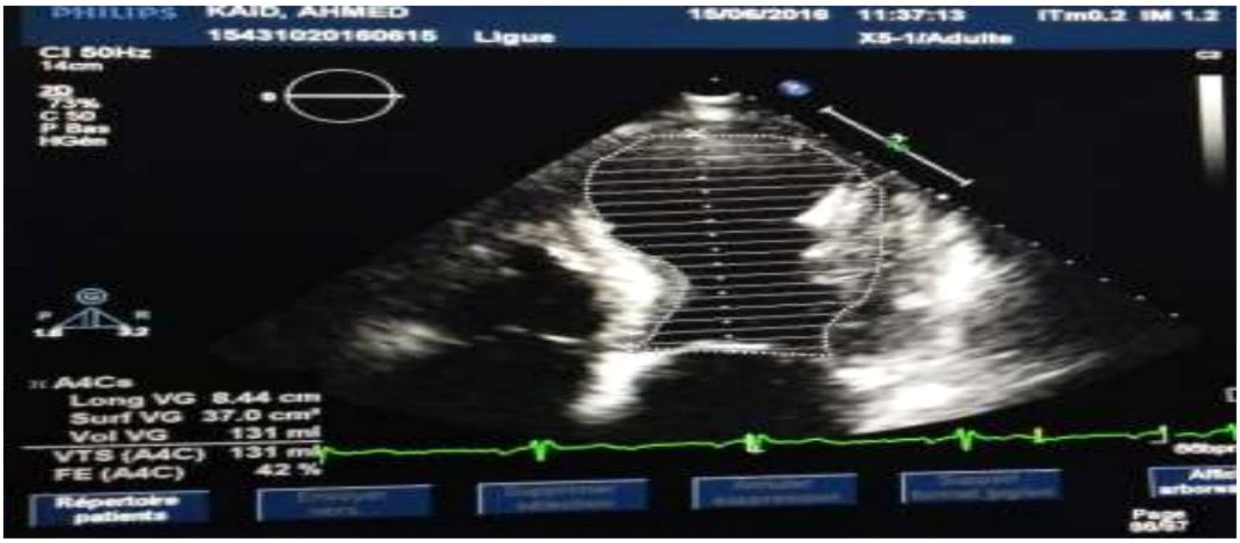

image 2: 4-cavity slice showing an apical left ventricular aneurysm.

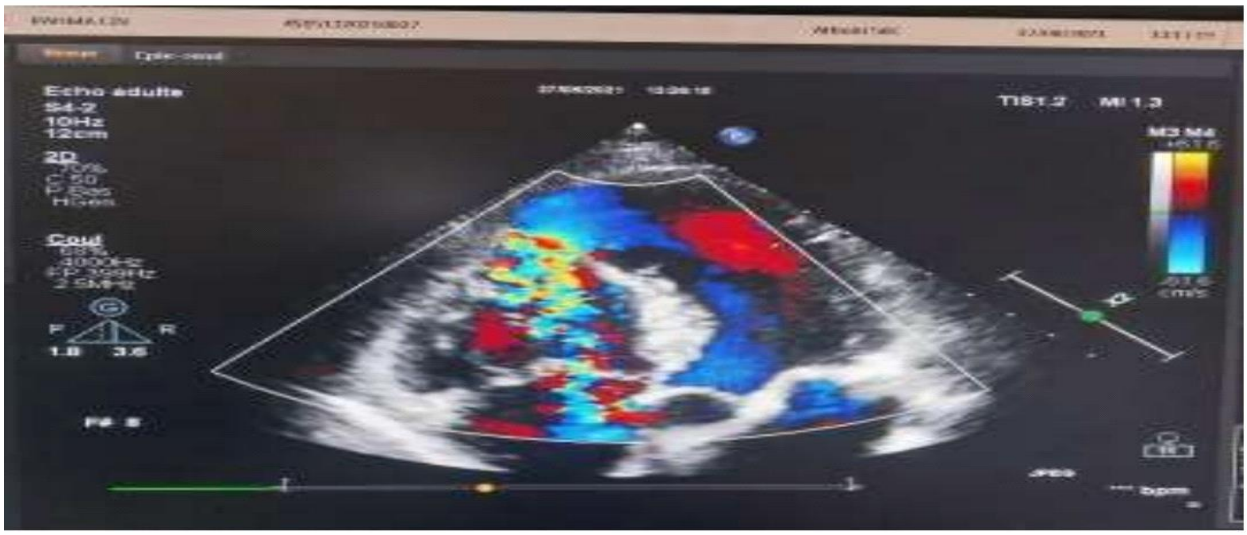

image 3: 4-cavity section in Doppler mode showing a serpentine apical VSD .

Discussion

Mechanical complications of myocardial infarction (MI) are fearful, and frequently occur following transmural MI. The combination of VSD with left ventricular aneurysm found in our patients, is a rare and highly lethal complication requiring urgent surgical management in case of hemodynamic instability [1].The incidence of this complication has been estimated at between 1% and 2% of MI, but it is responsible for 5% of deaths in the acute phase of infarction [1]. Its current incidence is lower (0.2%) in the GUSTO-I study investigating the benefit of early thrombolysis in acute MI [2]. VSD can occur within hours to a week after necrosis, with the majority occurring between the second and fourth day [2, 3]. The average delay was ten days for our patients. Transthoracic echocardiography is the test of choice for the diagnosis and evaluation of post-infarction VSD with a very high sensitivity and specificity [4]. Its management is surgical, with the difficulty of repairing fragile infarcted tissue. Two approaches are generally adopted: a delayed repair of 6 to 8 weeks after the MI in order to achieve a repair on stronger scar tissues, this is done in cases of hemodynamic stability and small VSD, as in our patient's case. Alternatively, an emergency repair can be performed because of the patient's hemodynamic instability. [5]

According to the recommendations of the American College of Cardiology-American Heart Association (ACC-AHA, Class I recommendation), surgical repair should not be delayed, regardless of the patient's clinical condition [5]. Despite adequate management, the prognosis for this condition is poor, with an estimated mortality of 30%. The prognostic factors are mainly represented by the systemic arterial pressure, the right atrial pressure and the duration of extracorporeal circulation (ECC).

Conclusion:

VSD is a rare and fearful mechanical complication of MI. Treatment is primarily surgical.

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