Case report

MYOCARDIAL INFARCTION SEEN TARDIVELY COMPLICATED WITH VENTRICULAR SEPTAL RUPTURE WITH RIGHT VENTRICULE DYSFUNCTION: A DILEMMATIC SCENARIO.

Abstract

Ventricular septal rupture (VSR) is an uncommon but well-recognized mechanical complication of acute myocardial infarction (MI). Mortality without intervention is 70% in intrahospital or 30 days, and 90% at one year due to cardiogenic shock. Transthoracic echocardiography (TTE) is the choice tool in the diagnosis and evaluation of VSR. We report an original case of VSR secondary to a false aneurysm located at the inferior wall of the left ventricule complicating an acute myocardial infarction seen tardively in an adult man with a known history of hypertension. Inferior wall myocardial infarction complicated with VSR at its basal segment is a rare location compared to forms described in the literatures. Patient died after refractory cardiogenic shock and neurological complications. Treatment of VSR secondary to MI is an emergency based on either an open heart surgery or percutaneous intervention inorder to reduce morbi-mortality rate.

Keywords: Ventricular septal Rupture, Myocardial Infarction, Echocardiography.

Introduction

The incidence of ventricular septal rupture (VSR) represents 0.2% of all ST segment elevation Myocardial Infarction (STEMI), compared to 1-2% before the start of the revascularization era percutaneous [1,2,3]. Ventricular septal rupture (VSR) is an uncommon but well-recognized mechanical complication of acute myocardial infarction (MI). Seen with anterior or inferior MI, it carries a poor prognosis [4]. Mortality without intervention is 70% in intrahospital or 30 days, and 90% at one year. After surgical or percutaneous closure, mortality is 30-40% at 30 days [1,5]. Risk factors for development of a post-infarction VSR are advanced age, sex female, high blood pressure, no history heart attack and late presentation to emergency [6]. Advances in echocardiography, including Doppler and multiplane transesophageal imaging, now enable the reliable diagnosis, localization and quantification of intracardiac shunting in patients with VSR [4,7]. Despite the increasing use of surgery and the reasonable long-term prognosis for most surgical survivors, most patients with VSR secondary to myocardial infarction and cardiogenic shock (CS) do not survive [4,8]. We hereby report a case of an adult man who consulted lately at the emergency department for chest pain and dyspnoea complicated with cerebral vascular accident, diagnosed with VSR secondary to an infero-basal myocardial infarction. Patient died 72 hours after admission due to refractory cardiogenic shock.

Case Report

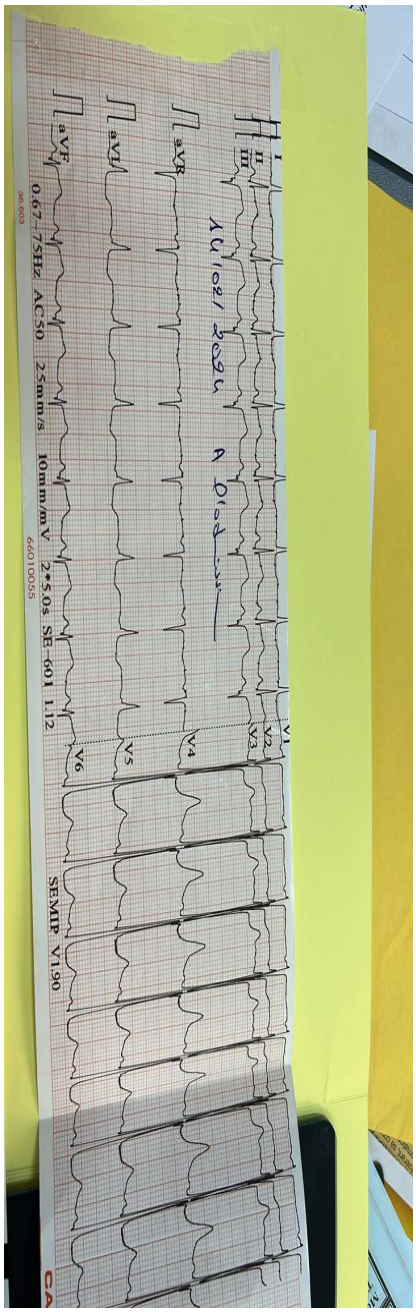
Patient aged 72 years, known active smoker for 40 packet years with a 3 years medical history of poorly monitored hypertension due to non-adherence of his treatment, admitted to the emergency room for atypical chest pain with acute dyspnea.

The onset of symptoms dates 10 days prior to his admission with the appearance of non-typical anginal chest pain with stage III NYHA (New York Heart Association) dyspnea in a context of apyrexia and deterioration in general condition (anorexia, weight loss of 10kg). Due to rapid worsening of symptoms patient finally consults at the emergency department.

The patient was initially treated in the emergency room, the clinical evaluation of which showed a conscious patient with a Glassgow score of 15/15, with neither sensory nor motor deficit. His systolic blood pressure was 110mmHg and diastolic of 80mmHg, a heart rate of 90 beats per minute(bpm), saturation of 93% ambient air, oliguria, without signs of cardiogenic shock. Patient was orthopnea with a persistent localized Basi thoracic chest pain, associated with signs of left heart failure, bilateral and symmetrical crackles at the bases and mid fields of the lungs. Cardiac auscultation reveals a left parasternal holosystolic murmur audible at left bord of the manubrium.

The rest of the clinical examination notes a cachectic state and a diffuse mucocutaneous subicter.

The electrocardiogram shows a regular sinus rhythm, with non-progressive ST segment elevation at the infero-basal leads with lateral mirror image, electrical left ventricular hypertrophy (LVH) with secondary repolarization disorders (Figure 1).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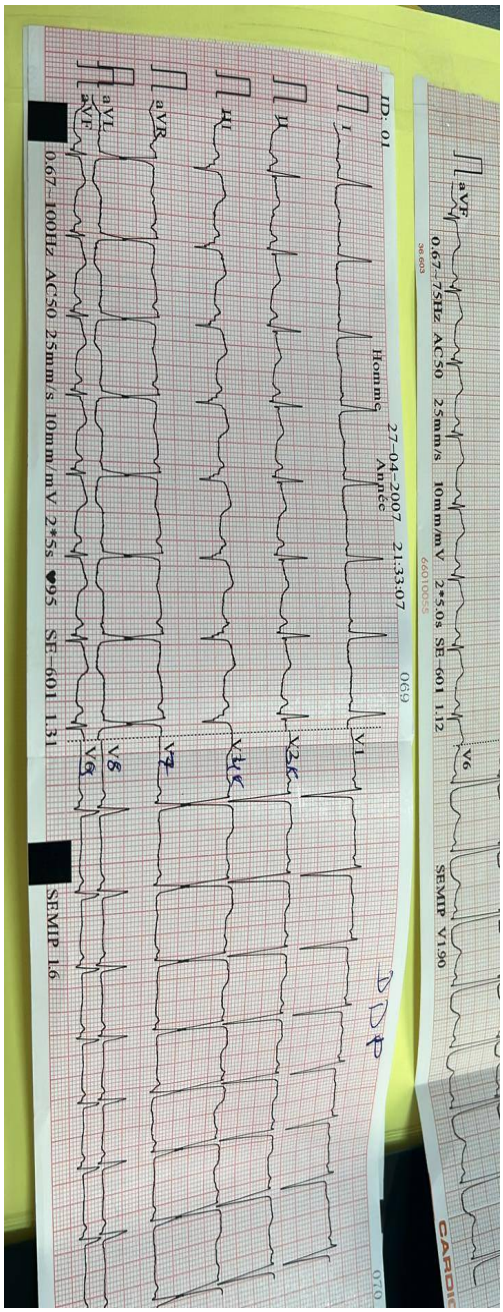

Figure 1A and B: 17 leads Electrocardiogram (EKG): Showing a regular sinus rhythm, with non-progressive ST segment elevation at the infero-basal leads with lateral mirror image, electrical left ventricular hypertrophy (LVH) with secondary repolarization disorders

The transthoracic echocardiography performed showed a hyperkinectic left ventricule with a left ventricular ejection(LVEF) fraction of 64%,Global longidutinal strain of -16.3%, concentric left ventricle hyperthrophy (septal wall mesaured 12mm and Posterior wall 14mm) with a brilliant appearance of the interventricular septum (Figure 2).

Figure 2 : Transthoracic echocardiography (TTE) showing a hyperkinectic left ventricule with a left ventricular ejection(LVEF) fraction of 64%,Global longidutinal strain of -16.3%, concentric left ventricle hyperthrophy with a brilliant appearance of the interventricular septum.

We noted a false aneurysm of the basal segment of the inefrieur wall of the left ventricle with a surface area of 21cm^2, ruptured in the right ventricle (RV) creating a restrictive muscular ventricular septal defect (VSD) with a trans defect Gradient of 91mmHg (Figure 3, 4).

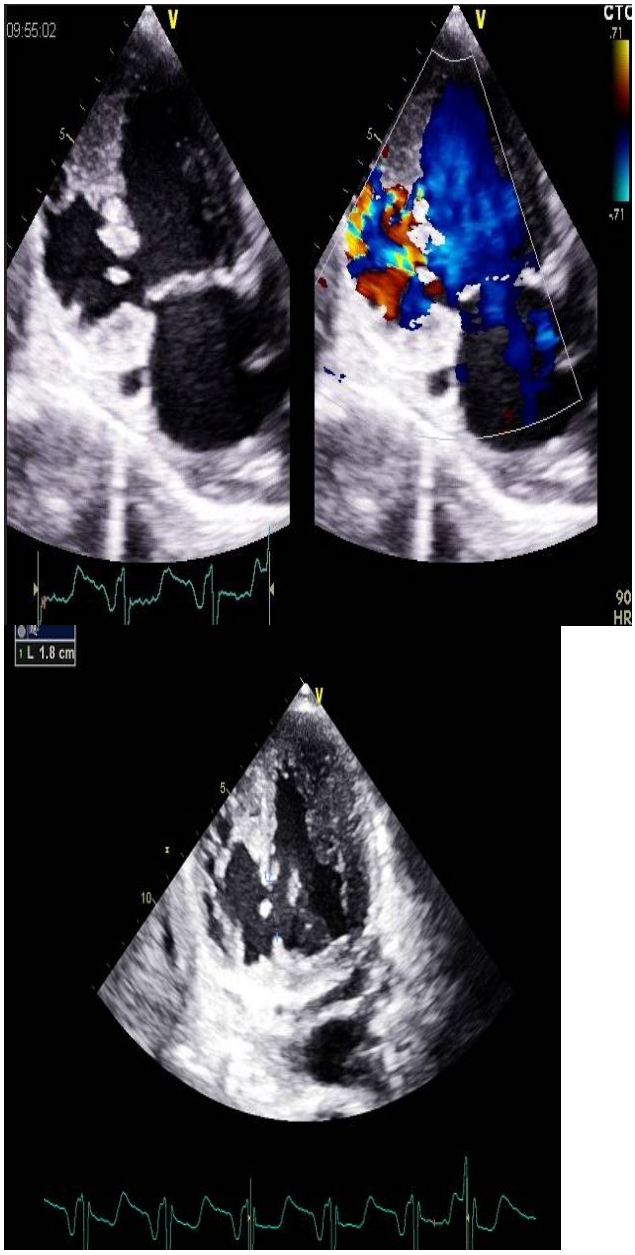

Figure 3 : Transthoracic Echocardiography : Apical section modified 2 chambers view : shows false aneurysm of the basal segment of the inefrieur wall of the left ventricle with a surface area of 21cm^2, ruptured in the right ventricle (RV) creating a restrictive muscular ventricular septal defect mesauring 1,8cm.

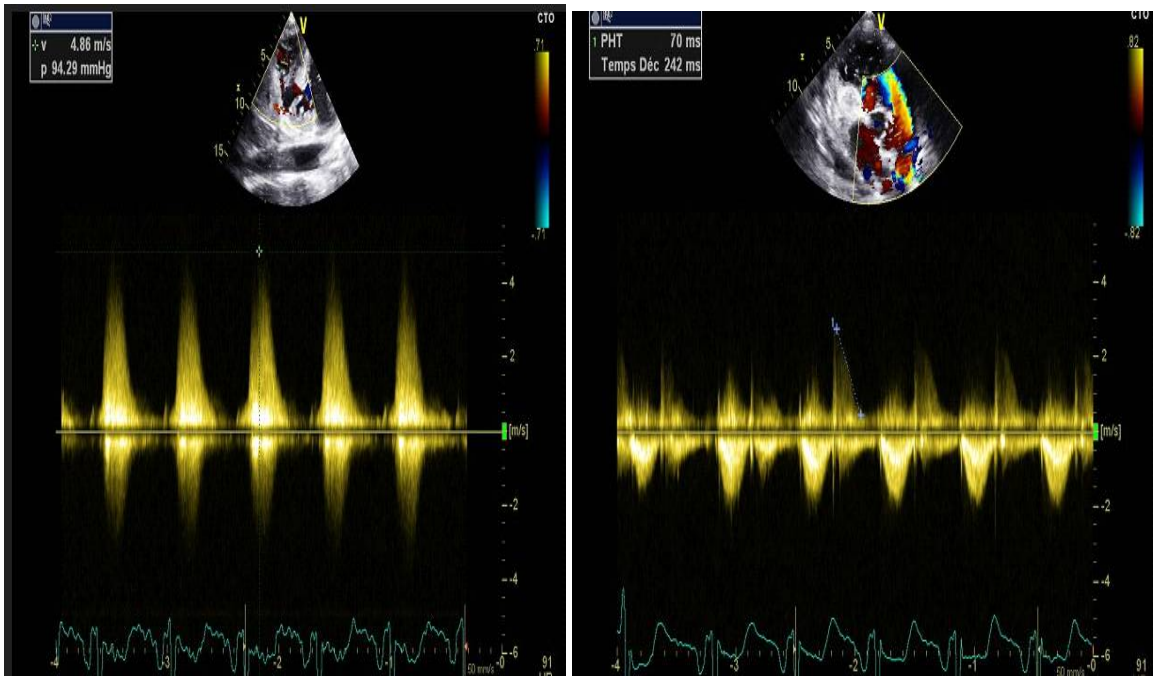

Figure 4 A and B : Color doppler and Continous wave doppler : Gradient a trans Ventricular septal defect of 91mmHg and a PHT value of 70ms.

This defect was partially closed by the pericardial wall of the RV associated with akinesia of the free wall of the RV. Altered systolic longitudinal function of the right ventricule with a systolique pressure of 39mmHg +10mmHg of right atrial pressure (RAP).We also noted an elevated filling pressure of the right ventricle. Absence of mitral and aortic valve diseases, also absence of pericardial effusion with a dilated inferior vena cava (Diameter 22mm) Figure 5.

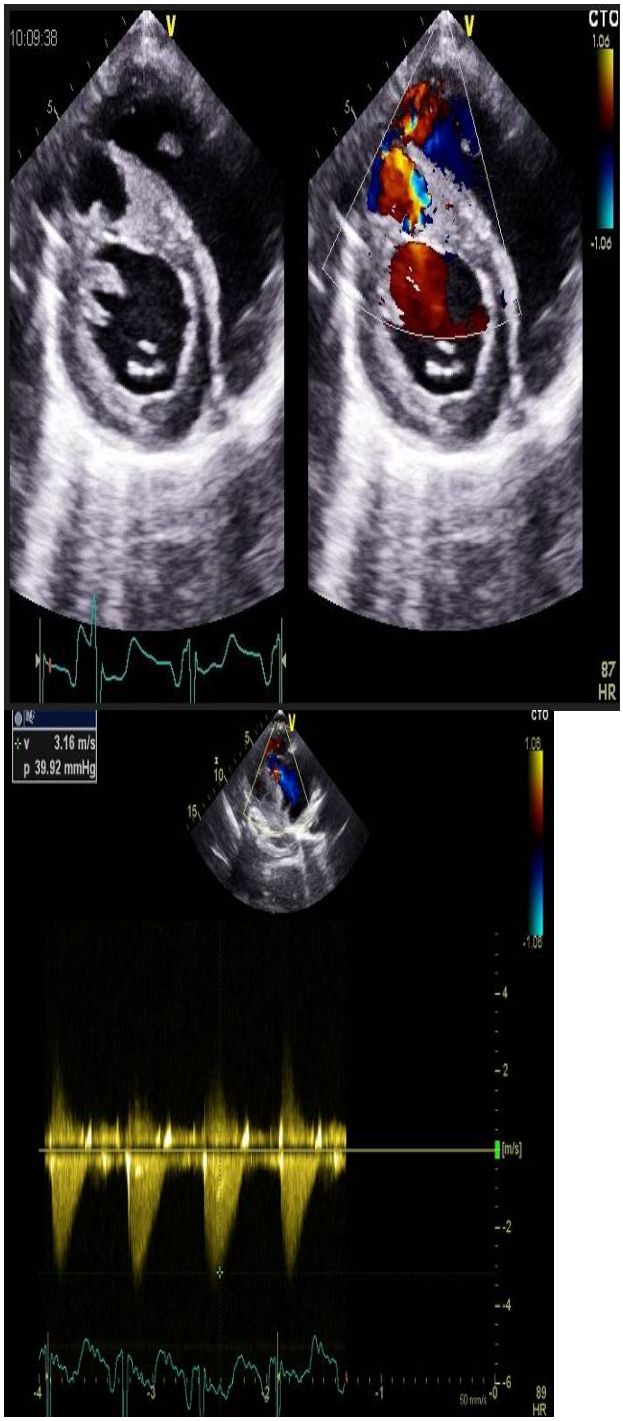

Figure 5A : Transthoracic echocardiography : subcostal view : showing VSD defect partially closed by the pericardial wall of the RV associated with akinesia of the free wall of the RV. B-Altered systolic longitudinal function of the right ventricule with a systolique pressure of 39mmHg +10mmHg of right atrial pressure (RAP).

Biologically assessment showed a blood level of troponin IC of 13 (normal range <0.001), impaired renal function (GFR: 23ml/min/m^2), hepatic cytolysis with elevated hepatic

enzymes (ASAT, ALAT) of values 30 times the normal and c-reactive protein (CRP) at 76ng/l. The rest of the assessment showed a hemoglobin (HB) value of 12.2g/dl with hyperleukocytosis of 18020/ul predominately neutrophiles at 15990/ul with Platelets levels at 187000/ul. Spontaneous low prothrombin level of 40% with normal fibrinogen value and a high value of prostrate specific antigen of 12 (Normal less than 4ng/l).

At this stage, the diagnosis retained was myocardial Infarction of the inferior wall of the left ventricle complicated by aneurysmal rupture of the basal segment, extended to the RV.

The patient was placed on double anti-platelet aggregation(DAPT) treatment based on acetyl-salycylic acid 100 mg/day and Clopidogrel 75 mg/day, anticoagulation based on hemiparin sodium 7500 IU/D, intravenous loop diuretic 40 mg three times a day and hypolipimiant treatment based on artovastatine at 80 mg a day.

Fours hours upon admission to the intensive care unit (ICU), the patient presented suddenly a right hemiparesis with contralateral facial paralysis of which he recovered totaly. A cerebral MRI imaging was conducted showing a multiple-age ischemia vascular cerebral accident (IVCA), one acute, localized at the level of the left PICA and the right AICA, associated with significant vascular leukopathy (Figure 6).

A renal ultrason was conducted which showed kidneys with normal anatomical size, well differenciated without other structural abnormalities.

Patient was stable 48 hrs after a transient vascular cerebral accident under monitoring. Surgery was to be performed at day 3 of hospitalization before which the patient presented a state of shock made up of neurological distress with hemodynamic and respiratory instability requiring the use of vasoactive drugs and mechanical respiration. Biological assessment showed worsening of renal function, GFR 11ml/min/m^2 consistent with clinical oliguria despite the increase of loop diuretics doses ; an elevation of CRP at 290ng/l, hyperleucytosis of 220000/ul predominately neutrophilies and fall in the level of plaletets at 80000u/l at fibrinogen marker. Blood cultures, chest X-ray and urinary analyse were conducted and a prophylaxie antibiothrapy based on 3rd generation ceftriaxon IV was started. Few hours later patient presented a cardio-respiratory arrest refractory despite resuscitation measures.

Discussion

Ventricular septal rupture secondary to Myocardial infarction diagnose tardively corresponds to the rupture of the ventricular septum between the healthy and infarcted tissus. It is a rare complication still associated with a high mortality rate [1]. The impact of this complication has been estimated between 1% and 2% of MIs, but it is responsible for 5% of deaths in the acute phase of Myocardial infarction [9]. The incidence of VSR secondary to MI is lower (0.2%) in the GUSTO-I studys of the benefit of early thrombolysis in acute MI [10]. VSRs can appear within a few hours to one week after

necrosis, with a majority between the second and fourth day [9, 11]. This period is, however, ten days in our patient.

MI can be complicated by right ventricular heart failure in 45% of cases, left ventricular heart failure in 35% of cases and global heart failure in 20% of cases. Cardiogenic shock is described in 60% of cases [11], which is the cause of dead in our patient three days prior to hospitalization.

Anamnesis typically reveals retrosternal pain often in 2 stages (the first being the infarction and the second, rupture of the interventricular septum) and dyspnea. Physical exams is marked by the presence of a holosystolic murmur of high rasping intensity predominantly in the parasternal left-side of the manubrium, radiating like a chariot wheel associated with signs of right heart failure (mainly turgor jugular) [12]. Our patient presented thesame symptoms and signes except clinical left side heart failure.

Transthoracic echocardiography (TTE) is the choice in the diagnosis and evaluation of VSR complicating a myocardial infarction with sensitivity and very high specificity [9, 13]. The TTE will make it possible to objectify the VSR, to locate it, to measure its size, to observe the direction of the shunt, to calculate the shunt, to estimate the systolic pressures functions of the left ventricle at that of systolic function of the right ventricle. As in our patient, TTE helps in confirming the diagonsis ; what's peculier in our findings was a false aneurysm complicated into VSR located at basal segment of inferior wall of left ventricule compared to literature with apical localization[4]. Angiography has better sensitivity, but remains a technique invasive and irradiating. The scanner allows to obtain also a good spatial definition as well as cardiac magnetic resonace imaging [14].

The recommendations of the European Society of cardiology regarding coronary revascularization of 2014 report that urgent cardiac surgery must be carried out in the event of a mechanical complication of myocardial infarction and cardiogenic shock (1C) [15]. Mortality after surgery is 30-50% when surgery to close VSR is carried out within the first 3 weeks, however, many studies show that mortality is lower when surgical closure is carried out beyond these 3 weeks, with the bias that unstable patients dies before intervention [1]. As in our case, patient died after refractory cardiogenic shock 3 days prior to hospitalization. This shows the unpredictable mortality in stable patients, hence the necessity of rapid treatment. Percutaneous VSR closure is another less aggressive alternative treatment with good results with the use of Amplatzer type devices in the absences of poor prognostic factors for percutaneous closure of VSR inclusing[2, 3, 12] ;

-VSR more than 15 mm in diameter,

-an apical localization without banks,

-a winding pathway between the VSR and infarcted tissus.

Despite adequate management, the prognosis of this pathology is grim, as medical management resulted in almost 100% mortality, and surgical outcome was poor once

cardiogenic shock developed [4].

Conclusion

The association of VSR and LV myocardial infarction is a rare entity and usually occurs within two weeks after a MI as shown in this case, however complications may occur several weeks later. A rapid diagnosis and urgent surgical or percutaneous closure of the VSR treatment significantly improves the patient's prognosis. Mortality rate is almost 100% in patients under medical therapy only.

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