



REAL BROKEN ANVRYSM COMPLICING AN MYOCARDIAL INFARCTION-CASE REPORT AND REVIEW OF LITTERATURE

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ABSTRACT:

INTRODUCTION: Despite the significant reduction in mortality from acute infarcts over the past decades thanks to monitored medical treatment and early reperfusion, a considerable hospital mortality rate persists. This is the consequence of refractory ventricular failure in the majority of cases, or of mechanical complications of the infarction. Mechanical complications include interventricular communication on septal rupture, tamponade on rupture of the free wall of the left ventricle, and acute mitral insufficiency on infarction or rupture of the papillary muscle. While these complications are rare, they have a very poor prognosis. This is why it is very important to know the clinical manifestations so as to clarify the diagnosis by echocardiography and allow urgent medical and surgical management. Interventricular communication (VIC) is caused by a rupture of the interventricular septum. It occurs in less than 0.5% of all STEMI, typically three to five days after an ischemic episode which is most often inaugural and extensive. The main risk factors for postinfarction VIC are advanced age, lack of revascularization and female sex. Over 70% of patients with postinfarction VIC die in hospital, and over 90% within one year. **RESULT:** Our patient presented with an anginal recurrence of infractoid chest pain with stage III dyspnea of NYHA with on the ECG a persistent elevation of the ST segment in the anteroapical and negative T waves in the extended anterior, on the ETT LV dysfunction with disturbed segmental kinetics, estimated LVEF of 20%, enormous aneurysm of the apex and adjacent segments, and restrictive muscle VIC. Due to the hemodynamic stability, the patient underwent postponed surgery for 6 weeks. **CONCLUSION:** The association of VIC and LV infarction is a rare entity and usually occurs within two weeks of an MI. However, and what is the case with our patient, these complications can occur several weeks later. Rapid diagnosis and urgent surgical management significantly improve patient prognosis

Keyword: MI complication IVC

INTRODUCTION:

Myocardial infarction (MI) is a systematized ischemic necrosis of the heart muscle most commonly caused by sudden occlusive thrombosis of a coronary artery. This acute coronary occlusion by a thrombus frequently occurs in atherosclerotic plaque that has become unstable following erosion, ulceration, cracking or rupture [1]. MDI carries a significant fatal risk in the short to medium term, but its morbidity and mortality has nonetheless been reduced significantly over the past three decades thanks to impressive progress made at several levels of intervention. Regardless, it remains a major public health problem in the industrialized world.

MDI is complicated 30% of the time. Apart from the risk of initial ventricular arrhythmias, relatively independent of the size of the necrosis, the frequency and severity of other complications is directly dependent on the necrotic myocardial mass. These complications can be divided into three groups: Early complications: 1st week, with decreasing frequency during this period. Delayed complications: are noted between the 2nd week and the end of the 1st month. Late sequelae.

a) Early complications:

Arrhythmias and conduction disorders,
Heart failure and non-cardiogenic shock.

Mechanical Complications; Apart from the extremely serious free-wall ruptures, which are usually revealed by a picture of cardiac arrest with electromechanical dissociation, these complications must be mentioned before a picture of acute heart failure associated with the appearance of a systolic murmur at the auscultation: two orientations

- Septal perforation: tear of the interventricular septum resulting in acute interventricular communication with massive left-right shunt and pre-capillary pulmonary arterial hypertension
- Acute mitral insufficiency: by rupture or ischemic dysfunction of a mitral pillar with often massive regurgitation, leading to acute left ventricular insufficiency with postcapillary PAH.

* Acute pericarditis; common, in transmural infarction.

*LV thrombosis and thromboembolic accidents

*Threats of extension, which is revealed by the early reappearance of ischemic attacks in situ or in another territory.

b) Delayed complications:

Some of the above complications, in particular heart failure and ventricular arrhythmias may have a delayed onset.

The only really specific complication of this intermediate period is Dressler's syndrome or post-infarction syndrome (of autoimmune etiology).

c) Late sequelae

In extensive infarctions:

- * Risk of heart failure
- * Ventricular arrhythmias, which have a very severe prognosis with a high risk of sudden death.
- * Aneurysm or Ectasia of the Left Ventricle: fairly rare development, linked to a poor healing of the infarcted wall with parietal thinning and formation of a well-defined pocket, animated by a paradoxical movement of systolic expansion.

His diagnosis can be mentioned:

On clinical examination, on the perception of a second center of beat (double endapex or supra-apical tip shock) on precordial palpation, of a very decomposed gallop (B4> B3) on auscultation.

On the ECG by the persistence beyond the 3rd week of a shift in ST without negativation of the T wave in the infarcted territory [2].

The chest X-ray does not show any abnormalities if the ectasia is posterior, whereas it shows an abnormally "square" aspect of the tip of the heart if the ectasia is anteroapical.

The diagnosis is confirmed by echocardiography and LV angiography.

The aneurysm can be perfectly tolerated, but it can also be the cause of complications: ventricular tachycardias, heart failure and thromboembolic events.

Left ventricular aneurysm (AVG), a rare and late mechanical complication of myocardial infarction, with a historical incidence of approximately 7% [3]. However, given the current management of infarcts with early revascularization, its incidence has decreased considerably. The majority of aneurysms are located on the anterolateral wall (85%), much more rare at the level of the posterior wall.

AVG corresponds to a dyskinetic segment due to the transformation of the necrotic heart wall into fibrous plaque bulging on its surface in systole [4]. It is typically observed 4-6 [5,6] weeks after the onset of extensive anterior myocardial necrosis [7, 8, 9]. Its incidence varies from 3 to 38% [10,11] of cases and is associated with an increased risk of heart failure by several mechanisms (paradoxical ventricular contraction, mitral regurgitation by separation of the papillary muscles) [12, 8], ventricular rhythm disturbances which can be life threatening; Ventricular arrhythmias are present in 15-30% of LV aneurysms, especially when the septum is involved. They are generally resistant to any drug therapy. Thromboembolic events are also described (coronary or systemic) due to a frequent associated mural thrombus [13, 4]. Mortality after AVG is high at one year regardless of the value of the ejection fraction (EF) [14] and other classical prognostic factors. Several authors have studied the determining factors in the constitution of AVG with different results [15,16]. This is probably due to the difference in the definition and interpretation of the aneurysm and to the reduced number of patients included.

CASE REPORT:

Reporting to us the observation of a 74-year-old patient with unbalanced arterial hypertension and type 2 diabetes as cardiovascular risk factors, consulted for an anginal recurrence following infractoid chest pain with vomiting, profuse sweating and dyspnea. stage III of NYHA which she had presented 3 weeks before.

Clinical examination found a conscious patient in fairly good general condition, eupneic at rest, supporting the dorsal decubitus, not in pain. BP is 88/60mmHG in the right arm, 90/60mmHG in the left arm, heart rate is 75 cpm, and SAO2 is 98%.

Cardiovascular examination does not reveal any abnormalities. The chest x-ray found cardiomegaly with a cardiothoracic index at 0.6 mm and a lower left globular arch with a sub-diaphragmatic point.

The ECG shows a regular sinus rhythm with a heart rate of 75 cpm, a persistent anteroseptal and low lateral elevation with negative T waves in the extended anterior (Figure 1).

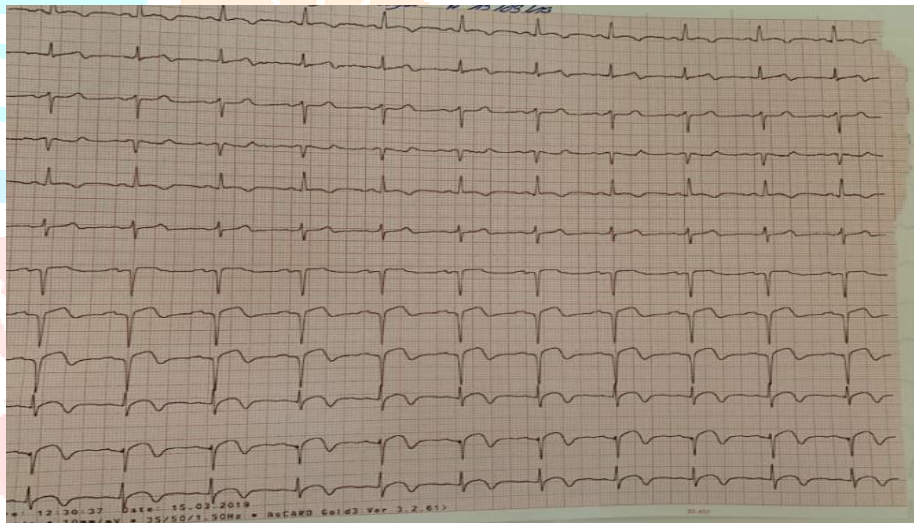


Figure 1: ECG on D 15 of our patient's hospitalization showing the persistent ST supershift in the anteroseptal and negative T waves in the extended anterior.

The biological assessment shows troponin in declining kinetics, serum potassium at 4.5 mEq / l, fasting blood sugar at 1.77 g / l, HBA1C at 7%, correct renal function and lipid balance and hepatic without abnormality. transthoracic cardiac ultrasound (TTE) shows a dysfunctional LV with segmental kinetic disturbances such as apical akinesia and adjacent segments, akinesia of the lateral and lower wall with an estimated LVEF of 20% as well as communication 16mm restrictive muscular inter-ventricular at the level of the apical segment of the infero-septal wall (flow estimated at 4.18m / s), and a large apical aneurysm of the LV and adjacent segments with a wide neck (Figure 2) with high left ventricular filling pressures. Note also the presence of a pericardial effusion of low circumferential abundance with deposits organized in a mattress suggesting a hemopericardium probably secondary to the ruptured aneurysm (Figure 3)

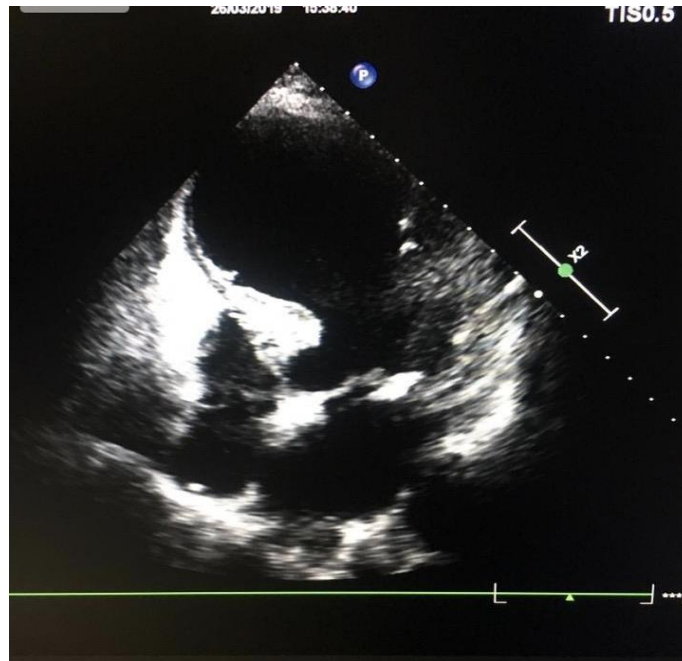


Figure 2: 4-cavity ETT section showing the apical LV aneurysm and adjacent segments with a non-obstructive septal pad.

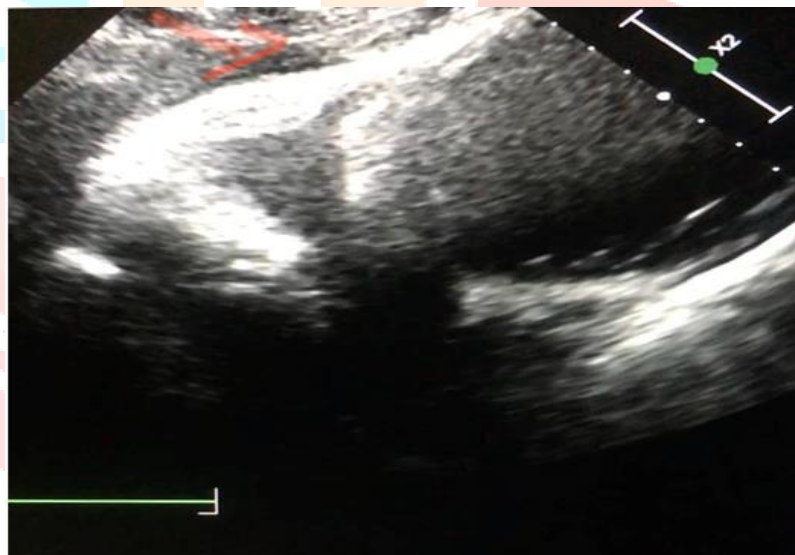


Figure 3: Subcostal ETT section showing pericardial effusion with deposits organized in a mattress suggesting a hemopericardium.

The surgical indication was asked but deferred due to the patient's hemodynamic stability and the absence of other hemodynamic or thromboembolic complications. Surgical correction consisted of a ventriculotomy in the middle of the aneurysm area parallel to the interventricular artery, with resection of the necrotic tissue. We find an apical VIC with friable and necrotic edges, which was closed by a dacron patch sewn by a crown of patched U-stitches. Due to the oblong character of the LV aneurysm, the fairly generous size of the residual cavity and the location of the mitral pillars, we opted for the linear resection technique with direct closure of the aneurysm edges by separate U-shaped points.

DISCUSSION:

The complications of MI are numerous and constitute the whole severity of coronary artery disease. The association of VIC and a left ventricular aneurysm, found in our patient, constitutes an extremely rare and highly lethal complication requiring urgent surgical management [17]. The incidence of this complication has been evaluated between 1% and 2% of MI, but it is responsible for 5% of deaths in the acute phase of infarction [17]. Its current incidence is lower (0.2%) in the GUSTO-I study studying the benefit of early thrombolysis in acute MI [18]. VICs can appear within a few hours to a week of necrosis, with a majority between the second and fourth days [18, 19]. This delay is, however, five weeks in our patient. Transthoracic echocardiography is the examination of choice in the diagnosis and evaluation of post-infarction VIC with very high sensitivity and specificity [20]. Its management is surgical, with the difficulty of repairing from fragile infarcted tissues. Two attitudes are generally adopted: a deferred repair 6 to 8 weeks after the IDM in order to carry out a repair on stronger scar tissue in the event of hemodynamic stability and small VIC; or even an emergency repair due to the patient's hemodynamic instability. [21] According to the recommendations of the American College of Cardiology/American Heart Association (ACC-AHA, recommendation class I) [21], surgical repair should not be postponed, regardless of the clinical condition of the patient.

Despite adequate management, the prognosis for this pathology is poor, with an estimated mortality of 30%. Prognostic factors are mainly represented by systemic arterial pressure, right atrium pressure, and cardiopulmonary bypass surgery (CPB). [22] The first interventions for a ventricular aneurysm were described in 1944 by Beck [23], who carried out an external reinforcement of the aneurysmal wall by the aponeurosis of the fascia lata. The first repair of an aneurysm by direct linear suture under CEC is described by Cooley et al. [24] in 1958. These are the work of Dor et al. [25] and Jatene [26] on the importance of maintaining an elliptical ventricular kinetics and geometry which allowed the development of LV repair. The incidence of LV aneurysms is estimated at 7.6% in the CASS study [27]. It is certainly lower at the present time thanks to the early reperfusion of infarcts (thrombolysis, angioplasty) and the use of converting enzyme inhibitors, which act respectively on the initial phase of aneurysmal formation and late post-infarction remodeling. from VG.

Regarding myocardial revascularization associated with surgical treatment of the complication of MI, opinions are divided. Bypasses associated with emergency surgery for VIC and LV aneurysm do not increase inpatient morbidity and mortality [28–29]. On the other hand, some authors do not recommend this combined surgery because of the lack of medium-term benefit [30]. Our patient did not have bypass surgery due to the lack of viability of the heart muscle on magnetic resonance imaging.

CONCLUSION

The association of VIC and LV infarction is a rare entity and usually occurs within two weeks of an MI. However, and what is the case with our patient, these complications can occur several weeks later. Rapid diagnosis and urgent surgical management significantly improve patient prognosis

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