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Case Report

Cardiology

A Rare Combination of Mechanical Complications Following Myocardial Infarction: Ventricular Septal Rupture and an Extensive Ventricular Aneurysm

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Abstract

Mechanical complications following acute myocardial infarction (MI) are rare and account for <1% of the total cases, with the advent of the newest revascularization methods. Although this low incidence, the mortality rate is high, especially among older patients. A 51 years old female, with a history of diabetes and asthma, was admitted to our cardiac intensive care unit for heart failure symptoms. She was diagnosed about 3 weeks ago, with acute anterior wall MI. She did not undergo any revascularization because of the lack of means and was on optimal medical therapy. Transthoracic echocardiography (TTE) revealed akinesia of the inferior, septal, and lateral wall, and all apical segments with a giant aneurysm of 10mm, and a posterior interventricular rupture with a left-right shunt, with a reduced left ventricular (LV) ejection fraction of 19%. An enhanced computed tomography (CT) scan revealed a ventricular septal (VS) defect with a giant left ventricular aneurysm (LVA), which was connected to the LV posterior wall. Urgent cardiac surgery after a percutaneous cardiac intervention was not realized because of lack of means. The association of VS

surgery after a perculateous cardiac intervention was not realized occause of fack of means. The association of VS rupture and LVA, found in our patient, is an extremely rare and highly lethal complication requiring urgent surgical management. Its incidence is between 1% and 2% of MIs. TTE has very high sensitivity and specificity, to provide diagnosis and evaluation of VS rupture and LVA. Color-flow Doppler identifies abnormal flow within the aneurysm and helps to highlight ventricular ruptures as an additional image in the endocardium. CT is fast, provides a clear resolution of the LV, and may be used to demonstrate the complications of infarction. The management of VSD is surgical, with the difficulty of repair from fragile infarcted tissue. Medical therapy should stabilize the hemodynamics and act as a bridge to surgery. The simultaneous presence of a VS rupture and LVA is rare and usually occurs within the MI. However, as shown in our patient, these complications may appear up to several weeks later, and it is unusual in the revascularization era.

Keywords: Left ventricular aneurysm, ventricular septal rupture, post-myocardial infarction complications, case report. Copyright © 2023 The Author(s): This is an open-access article distributed under the terms of the Creative Commons Attribution 4.0 International License (CC BY-NC 4.0) which permits unrestricted use, distribution, and reproduction in any medium for non-commercial use provided the original author and source are credited.

INTRODUCTION

Coronary disease is the most common cause of death in the world and its frequency increases worldwide. Mechanical complications following acute myocardial infarction (AMI) are rare and account for <1% of the total cases, with the advent of the newest revascularization methods. Although this low incidence, the mortality rate is high, especially among older patients [1].

The association of ventricular septal rupture (VSR) post-MI and left ventricular aneurysm (LVA) is extremely rare and usually occurs during the first week following MI.

CASE REPORT

A 51 years old female, with a history of diabetes and asthma, was admitted to our cardiac intensive care unit for heart failure symptoms (NYHA class IV symptoms) without chest pain. She was diagnosed about 3 weeks ago, with an acute anterior wall MI complicated by mid-range Heart Failure ejection fraction. She did not undergo any revascularization because of the lack of means and was on optimal medical therapy.

Electrocardiogram (EKG) shows sinus tachycardia, left axis deviation, ST elevation in lead V2 to V6, and T inversion in lead II, III, and aVF.

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Chest X-ray showed bilateral vascular congestion and minimal pleural bilateral effusion. Transthoracic echocardiography (TTE) revealed akinesia of the inferior, septal, and lateral wall, and all apical segments with a giant aneurysm of 10mm (Figure 1), and a posterior interventricular rupture with a left-right shunt (Figure 2), with a reduced left ventricular ejection fraction of 19%. The pulmonary systolic pressure estimated from the tricuspid regurgitation jet

was at 60 mmHg. An enhanced computed tomography (CT) showed a giant LVA, which was connected to the LV posterior wall (Figure 3).

The patient was managed medically in the intensive care unit. Urgent cardiac surgery was decided, after a percutaneous cardiac intervention, but not realized because of lack of means.

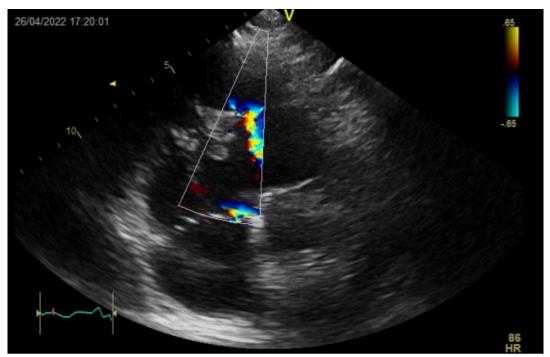


Figure 1: TTE view showing the ventricular septal rupture

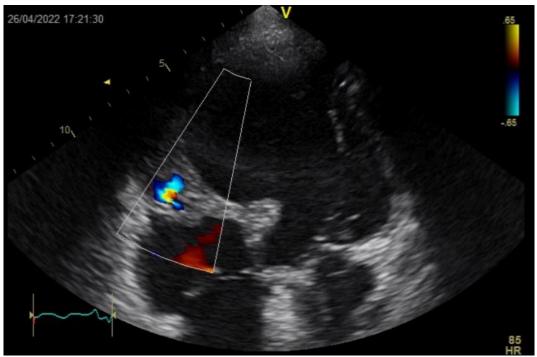


Figure 2: TTE four cardiac chamber view showing the LVA and the posterior VSR



Figure 3: Axial CT-Scan view showing the LVA

DISCUSSION

Complications of MI are numerous and constitute the whole severity of the coronary disease. There are many risk factors for post-MI cardiac rupture, among them: the first non-managed MI event, advanced age, female gender, and high admission blood pressure. More than half of patients with VSR present a cardiogenic shock. They may present also angina pain, dyspnea, nausea, sudden or progressive hypotension, decreased urine output, altered mentation, and a new mitral regurgitation murmur.

LVA is a scarred area that loses its contractile function and leads to a localized dilation of the left ventricular (LV). It is the most common mechanical complication of AMI and its incidence is approximately 15% [2, 3].

VSR is an uncommon complication of myocardial infarction (MI) and can appear within a few hours to a week after necrosis, with a majority between the second and fourth days. It represents a defect in the interventricular septum caused by ischemic necrosis [4].

The association of VSR and LVA, found in our patient, is an extremely rare and highly lethal complication requiring urgent surgical management. Its incidence is between 1% and 2% of MIs but is responsible for 5% of deaths in the acute phase. Its current incidence is lower with the large use of revascularization methods.

LVA is frequently located in the anterior wall about 22% within 3 months of infarction, and unusually

in the inferoposterior wall (9%) when they are rarely extensive. The diagnosis of small aneurysms is done by routine investigations and is frequently asymptomatic. The large ones are presented with thromboembolism symptoms, wall motion abnormalities, arrhythmias, contractility alteration, and sudden cardiac death.

The ventricular rupture is the most dramatic complication of LVA. The rupture of an immature aneurysm can cause cardiac shock and tamponade. Mature ones rupture less due to dense fibrous tissue.

Some signs at the EKG are predictive of this complication: the persistent ST-segment elevation with T wave inversion, sudden onset of electromechanical dissociation, and Goldberger's sign [2, 5].

High cardiac mortality is reported in the presence of an LVA, and is estimated at 67% and 80%, for 3 months and 1 year, respectively [2]. Post-MI death incidence is at 5%, and 1% to 3% of all MIs complicated by VSR. They are frequently located in the anterior septum than the posterior (60% vs 40%).

Becker proposed a classification in free wall rupture [6]: Type I: Acute presentation, slit-like tear, in the 24 hours Type II: Sub acute presentation with erosion of infarcted myocardium Type III: Late presentation, with the aneurysm formation and rupture, associated with older infarcts.

Identifying a narrow neck compared to the size of the cavity in TTE can make the difference between a false and true aneurysm. It is often associated with thrombus. TTE has very high sensitivity and specificity to provide the diagnosis and evaluation of VSR and LVA. Color-flow Doppler identifies abnormal flow within the aneurysm and helps to highlight ventricular ruptures as an additional image in the endocardium [4]. Transesophageal echocardiography remains the gold standard that can accurately locate and measure the VSD to guide surgical strategy [2, 3].

CT helps to demonstrate regional wall thickening, as well as the complications of infarction, such as LVA and mural thrombus. For patients with ventricular aneurysms, CT is fast and provides a clear resolution of the LV. It also provides the site and the extent of anterior and posterior LVA.

CT requires identifying the small ostium that connects the aneurysm with the LV cavity to differentiate a true aneurysm from a pseudo aneurysm. False aneurysms are usually substantially larger than true aneurysms; they frequently arise from the posterior or inferior wall of the LV.

Magnetic Resonance Imaging (MRI) can be useful in identifying small LVA's and assessing resectability. Dark-blood imaging defines anatomy and helps in delineating the bulging of the aneurysm.

The management of VSD is surgical, with the difficulty of repair from fragile infarcted tissue. Medical therapy should stabilize the hemodynamics and act as a bridge to surgery, however, despite optimal treatment, mortality remains 45% in surgically treated patients and 90% in those managed medically [5].

Two attitudes can be adopted: to defer the repair for 6 to 8 weeks after MI to carry it out on more solid scar tissue, in the case of hemodynamic stability and small VSD, or an emergency repair when the patient is hemodynamically unstable. However, 90% of mortality is observed in patients managed medically and about 45% for those treated surgically, despite optimal medical therapy.

Factors that predict survival after surgery are systolic blood pressure, mean right atrial pressure and cardiopulmonary bypass time [4, 7].

Surgical repair of LVA results in improved survival in this subset of high-risk patients and is indicated in refractory CHF, resistant ventricular tachyarrhythmias, and recurrent thromboembolism. 9.9% is the mean operative mortality rate [6, 7].

Regarding myocardial revascularization associated with the surgical cure of the MI complication, opinions are divided: Bypass surgery associated with emergency surgery for VSD and LVA does not increase hospital morbidity and mortality. On the other hand, some authors do not recommend this associated surgery because of the lack of medium-term benefits [4].

Our patient is unique in that, she presented a double mechanical complication: LVA and VSD, three weeks after an AMI with no signs of re-infarction. The combined presence of these complications is unusual in the revascularization era.

CONCLUSION

Simultaneous presence of VSD and LVA is rare and usually occurs within the AMI. However, as shown in our patient, these complications may appear several weeks later.

High clinical suspicion and thorough physical examination with immediate surgery can lead to a better prognosis and complete recovery.

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