SURGICAL AND CARDIOLOGIC MANAGEMENT IN A PATIENT WITH A LEFT VENTRICULAR ANEURYSM AND A VENTRICULAR SEPTAL DEFECT AFTER ACUTE MYOCARDIAL INFARCTION

RADU BĂLĂU[1], RADU DEAC[2], MARIUS HARPA[3], HUSSAM AL HUSSEIN[4], CLAUDIU GHIRAGOSIAN[5], HAMIDA AL HUSSEIN[6], HORAȚIU SUCIU[7]

[1,2,3,4,5,6,7]University of Medicine and Pharmacy Tîrgu-Mureş, [1,2,3,4,5,7]Institute for Cardiovascular Diseases and Transplantation Tîrgu-Mureş

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Abstract: *The association of an acute ventricular septal defect (VSD) and left ventricular aneurysm (LVA) is a rare, life-threatening complication of acute myocardial infarction (AMI). We present the case of an apical VSD and LVA after anterior AMI, in a 71-year-old male patient with a history of aortic valve replacement, arterial hypertension and atrial fibrillation. An emergency percutaneous coronary intervention (PCI), with a BMS stent on the left anterior descendent coronary artery was performed. 3rd day echocardiographic reevaluation revealed LVA and apical VSD. After 35 days of cardiologic management, the patient underwent ventriculotomy for VSD repair using a bovine pericardial patch followed by endoventricular patch remodelling of the LVA. He was discharged 16 days after surgery and presented good clinical and echocardiographic evolution during follow-up.*

INTRODUCTION

Left ventricular aneurysms (LVA) and pseudoaneurysms are usually complications of a severe transmural myocardial infarction (MI) leading to chamber dilation and modified geometry of the ventricle, and therefore to hemodynamic degradation. Left ventricular remodelling usually starts within the first two hours after a MI and consists of replacing necrotic with fibrous tissue. This will increase the ventricular volumes as the ejection fraction declines.(1)

Acquired ventricular septal defect (VSD) is a dangerous complication of MI which frequently leads to congestive heart failure. It usually occurs within the first week of an acute myocardial infarction (AMI) (2) and has an incidence of 1%-3%.(3) The acquired VSD is more common in the anterior and apical portions of the septum as a result of an occlusion in the anterior descending artery, and is more accessible for surgical repair than the posterior septal rupture which is usually a consequence of an acute occlusion of the dominant right coronary artery (RCA).(4) The left to right shunt through the VSD will worsen the preexisting pulmonary hypertension determined by the declining systolic function of the left ventricle (LV), which might lead to acute pulmonary edema and rapidly progressive congestive heart failure.(5)

Ventricular aneurysms associated with ruptured interventricular septum represent a rare, but life-threatening mechanical complication(6) of MI, which can appear even if an emergency percutaneous coronary intervention (PCI) has been performed.(7) Surgical treatment of this pathology is technically demanding, especially when performed early after an MI, and presents poor outcome due to the fragility of the infarcted myocardial tissue. Surgical ventricular reconstruction (SVR) using a Pericardium-Dacron double patch, associated with patch closure of the VSD is the optimal surgical option, in order to preserve the ventricular inner contour and restore the geometry of the dilated chamber, therefore reducing the filling pressures and right ventricle (RV) overload.(8) SVR along with the VSD closure could either avoid or decrease the congestive heart failure that is very likely to occur after this complication.

A proper preoperative management of the patient by controlling the heart failure, pulmonary hypertension and renal dysfunction, which are consequences of this pathology(9), is essential for the success of the operation. This management allows delay of surgery for up to 3-4 weeks after the MI, when fibrosis of the VSD-s margins occurs, thus allowing a safer surgical correction.(10)

CASE REPORT

A 71 year-old male patient with a history of arterial hypertension, permanent atrial fibrillation (AF) and a mechanical prosthetic aortic valve (1995), under anticoagulant treatment efficiently administered at home, was admitted at the Emergency Department of Interventional Cardiology, accusing precordial chest pain during the day.

Upon admission, the patient presented NYHA functional class III, blood pressure (BP) of 140/100 mmHg and a heart rate (HR) of 90 bpm. The EKG revealed AF with moderate ventricular response (82/min), left QRS axis deviation, incomplete left bundle branch block, ST elevation in V1-V4 with negative T waves in V5-V6. Echocardiography showed an ejection fraction (EF) of 32%, hypokinetic interventricular septum (IVS), akinetic apex, hypokinetic anterior wall and hyperkinetic postero-lateral wall, as well as a normally-functioning mechanical prosthetic aortic valve, grade II mitral regurgitation (MR), grade II/III tricuspid regurgitation (TR), systolic pulmonary artery pressure (sPAP) of 45 mmHg, and no pathological pericardial space. The final diagnosis was acute anterior MI with ST elevation (STEMI), ischemic and valvular cardiomyopathy, normally-functioning aortic prosthetic mechanical valve, grade II MR, grade II/III TR, moderate pulmonary hypertension and AF with moderate ventricular response.

A percutaneous coronary intervention (PCI) was performed, with a BMS stent in the 2nd segment of the left anterior descending coronary artery (LAD), and an increase of

[3]Corresponding author: Marius Harpa, Str. Gheorghe Marinescu, Nr. 50, Tîrgu-Mureş, România, E-mail: cardiochirurgiemures@gmail.com, Phone: +40740 751422

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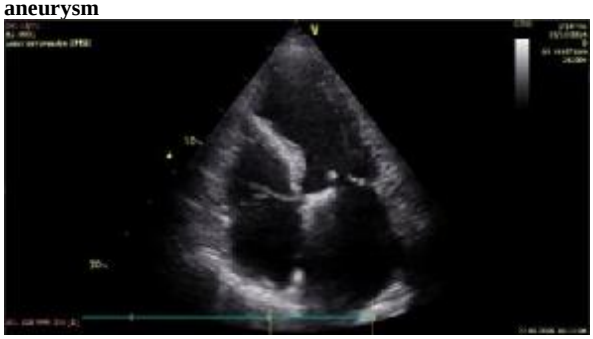
TIMI flow from 0 to II.

Post-PCI echocardiography revealed: mild LV hypertrophy; dyskinetic apex; akinetic IVS in the apical 2/3 and hypokinetic basally; anterior, posterior and inferior walls apically hypokinetic; lateral wall with basal compensatory hyperkinesia; an EF of 32%; RV with preserved contractility; a tricuspid annular plane systolic excursion (TAPSE) of 18mm, and no pathological pericardial space.

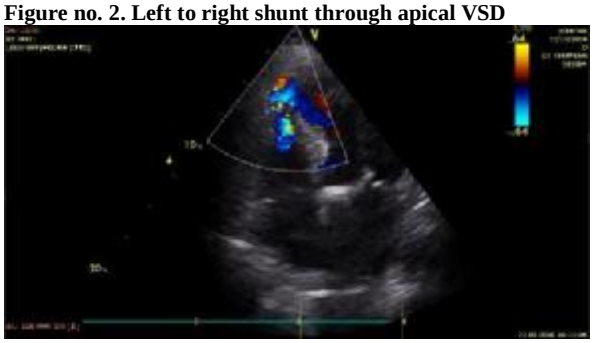
At the 3-day reevaluation, echocardiography showed a "suspect" apical region with a turbulent flow towards the RV in the apical IVS portion, and an RV-RA gradient of 60 mmHg.

Figure no. 1. Diskinetic antero-apical-septal left ventricular aneurysm



The 2 week-reevaluation revealed a VSD with a maximum LV-RV gradient of 20 mmHg, LV apical aneurysm, an EF of 33%, grade I/II TR with an RV-RA gradient of 50 mmHg and an sPAP of 65 mmHg.

Figure no. 2. Left to right shunt through apical VSD



During cardiology admission, the patient received specific treatment for heart failure and ischemic cardiomyopathy, consisting of antiplatelet agents, β-blockers, diuretics, ACE inhibitors, statins, and low molecular weight heparin in doses adjusted to the BP, HR, renal function and oxygen saturation.

After a total stay of 35 days in the Cardiology ICU, a transfer to Cardiovascular Surgery was decided for surgical correction.

The preoperative physical exam showed a healed post-sternotomy scar, normal conformation of the precordial region, apex beat in the 5th intercostal space, mid-clavicular line, arrhythmic heart sounds, mesocardiac IV/6 murmur, aortic III/6 murmur, mitral III/6 murmur, prosthetic click at the aortic site.

Preoperative echocardiography revealed a ruptured IVS in its apical region with left to right shunt, a hypertrophied LV in the mid-ventricular (MV) and apical regions, with an antero-apical-septal aneurysm with a volume of 75 ml, a left ventricular end-diastolic volume (LVEDV) index of 125 ml/m^2 and a left ventricular end-systolic volume (LVESV) index of 81 ml/m^2; dyskinetic IVS in the apical region, akinetic in the MV region and hypokinetic in the basal region; a dyskinetic anterior wall in the apical region, hypokinetic in the MV and basal regions; hypokinetic lateral wall in the apical ½ and normokinetic in the basal ½; normokinetic inferior wall basally and hypokinetic apically and a normokinetic posterior wall, as well as an EF of 30%; grade I/II TR with an RV-RA gradient of 60 mmHg; RV with preserved contractility, TAPSE=18mm, and no pathological pericardial space.

The surgical intervention began with a median sternotomy and cardio-pericardial adhesiolysis. Cardio-pulmonary bypass (CPB) was achieved with bicaval venous cannulation and transmitral LV venting. In moderate hypothermia, at 30^0 Celsius, Calafiore cardioplegia was administered. An approximately 5 cm left ventriculotomy parallel to the anterior interventricular groove was performed at the site of the aneurysm. The apical VSD had a diameter of 1.5 cm with irregular edges, which was closed with heterologous pericardial patch. SVR was performed using the endoaneurymorrhaphy technique first described by Cooley in 1989(11), by placing an elliptic pericardium-Dacron double patch at the scarred-to-normal myocardium junction, using a Prolene 3.0 running suture, followed by a double-layered suture of the aneurysmal myocardium, reinforced with Teflon strips over the double patch. The aorta was unclamped, followed by reperfusion and warming. A sinus rhythm was obtained spontaneously, and the CPB was suppressed under a small-dose inotropic protection (Dopamine, Epinephrine and Norepinephrine). The total CPB time was 115 minutes and the ischemia time was 65 minutes.

Norepinephrine was again administered in the first postoperative day, while the Dopamine continued until the 9th day.

RESULTS

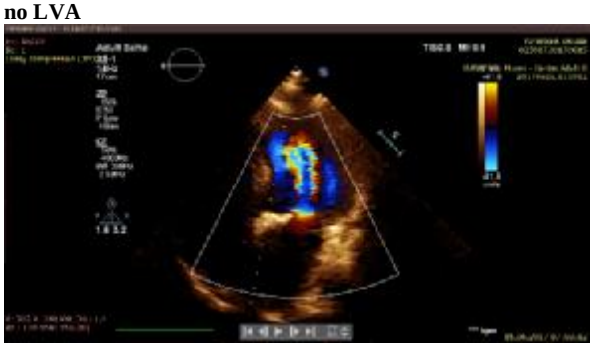
The postoperative evolution was progressively favorable, echocardiography showed an IVS without a residual shunt, grade II MR, grade II TR, an EF of 38%, and no pathological pericardial space. The LVEDV and LVESV indexes were significantly lower than the preoperative values (125 ml/m^2 to 88 ml/m^2 LVEDVI, respectively 81 ml/m^2 to 57 ml/m^2 LVESVI) – a 29% reduction in ventricular index volumes.

Swan-Ganz right heart catheterization revealed a decrease in the pulmonary capillary wedge pressure (PCWP) from 16 mmHg to 8 mmHg, as well as in the pulmonary vascular resistance (PVR), from 211 dyne·sec/cm^5 to 98 dyne·sec/cm^5 in the first 2 postoperative days. Furthermore, a decline in the mean pulmonary artery pressure (mPAP) was observed, form 45 mmHg preoperatively to 15 mmHg in the second postoperative day.

The patient was discharged after a 4 day-stay in the ICU, 16 days stay in the Cardiovascular Surgery Department, with a total of 52 hospitalization days. Upon discharge, the patient was asymptomatic. Clopidogrel, Amiodarone, Carvedilol, Atorvastatin, Perindopril and Acenocoumarol (with periodic monitoring of the INR) were prescribed as long-term treatment.

Clinical and echocardiographic follow-up was performed at 6 months and 1 year after discharge. NYHA functional class was II and there were no complications and no need for rehospitalization. Echocardiographic examinations revealed a constant EF of 40%, mild MR, no residual interventricular shunt, no pulmonary hypertension, and a slight reduction of the LVEDV index (85 ml at 6 months and 83 ml at 1 year) as well as the LVESV index (56 ml at 6 months and 53 ml at 1 year) when compared with the immediate postoperative values.

Figure no. 3. Follow up echocardiography with no VSD and no LVA



DISCUSSIONS

The combination of an acute VSD and anterior LVA is a rare and severe complication of AMI. Surgical treatment is technically demanding, especially when performed early after the MI occurs, with poor results due to the fragility of the infarcted myocardial tissue. Association of previous cardiac surgery (aortic valve replacement in this case) increases the difficulty of the intervention, due to the presence of cardio-pericardial adhesions, therefore increasing the risk of surgical bleeding. The endoventricular patch remodelling procedure used in our case is a useful approach, allowing a direct view for the VSD patch implantation and the aneurysm exclusion, with good postoperative results, no residual interventricular communication, significant reduction of the ventricular volumes, and improvement of the ejection fraction.

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