DOI: 10.23934/2223-9022-2018-7-4-372-377

The Successful Surgical Treatment of a Giant True Left Ventricular Aneurysm: a Case Report

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ABSTRACT We report the successful surgical treatment of a giant true thrombosed aneurysm of the left ventricle.

A 59-year-old male patient Z. was admitted with severe heart failure and chronic thrombosed aneurysm of the left ventricle, formed after acute extensive myocardial infarction, despite successful installation of a stent into the anterior descending artery in the acute period. Echocardiography revealed a significant increase in the volume of the left ventricular cavity, a significant decrease in the contractile function of the left ventricular myocardium (ejection fraction 32–36%), a giant left ventricular aneurysm (9x6 cm) with a parietal lining thrombus in the aneurysm cavity. Coronary angiography showed an aneurysmal dilatation of the circumflex branch of more than 6 mm, hemodynamically significant stenosis of two coronary arteries. According to the scintigraphy, the myocardium beyond the scar tissue was viable. The patient underwent resection of a left ventricular aneurysm, endoventricular plasty (Dor procedure), coronary artery bypass surgery of the circumflex artery and obtuse marginal branch of the left coronary artery.

The patient was discharged in satisfactory condition on day 14 after surgery. At follow-up 6 months after surgery, an increase in the ejection fraction to 39–41% was noted.

Keywords: myocardial infarction, left ventricular aneurysm, surgical treatment

For citation Brand Y.B., Mazanov M.K., Ostroumov E.N. The Successful Surgical Treatment of a Giant True Left Ventrical Aneurysm: a Clinical Case. *Russian Sklifosovsky Journal of Emergency Medical Care*. 2018; 7(4): 372–377. DOI: 10.23934/2223-9022-2018-7-4-372-377 (In Russian)

Conflict of interest Authors declare lack of the conflicts of interests

Acknowledgments The study had no sponsorship

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ABP – arterial blood pressure
ADA – anterior descending artery
CA – circumflex artery
CAD – coronary artery disease
ECG – electrocardiography
EchoCG – echocardiography
EF – ejection fraction
EDS – end-diastolic size
EDV – end-diastolic volume
ESS – end-systolic size
ESV – end-systolic volume
FC – functional class
HR – heart rate
LCA – left coronary artery
LV – left ventricle
MI – myocardial infarction
MSCT – multispiral computed tomography
NYHA – New York Heart Association
OMB – obtuse marginal branch
PIA – posterior interventricular artery
PLB – posterior lateral branch
RCA – right coronary artery
SPPA – systolic pressure in the pulmonary artery
STICH – Surgical Treatment for Ischemic Heart Failure

BACKGROUND

Post-infarction left ventricular (LV) aneurysms, which account for the vast majority of all cardiac aneurysms, are a serious complication of coronary artery disease (CAD).

The LV aneurysm is a local limited bulging of a thinned and non-contractile portion of the LV wall consisting of necrotic or scar tissue. Aneurysms develop after extensive transmural myocardial infarction (MI) and in 85% of cases are located in the region of the apex and anterior wall of the LV [1, 2, 3].

The LV aneurysm is a cause of refractory heart failure, ventricular arrhythmias, and arterial emboli. In some patients, LV aneurysms may be asymptomatic.

The indications for aneurysmectomy remain controversial [4]. One of the main problems of surgical treatment of post-infarction LV aneurysms is the syndrome of small cardiac output, which develops as a result of an excessive reduction in the size of the LV cavity, as well as ventricular arrhythmias and pulmonary insufficiency. During the last decade, hospital mortality in the surgical treatment of LV aneurysms decreased from 2–19% to 3–7%, depending on the method of operation [5, 6].

A clinical case report.

A 59-year-old male patient Z. was admitted to the Emergency Coronary Surgery Department of the N.V. Sklifosovsky Research Institute for Emergency Medicine on June 21, 2017 with the diagnosis: "Atherosclerosis of the aorta and coronary arteries. CAD. Exertional angina, 3 functional class (FC). Post-infarction cardiosclerosis (acute extensive MI in February 2017). Balloon angioplasty with a stent in the anterior descending artery in February 2017. Chronic heart failure stage 2A, NYHA III. Chronic post-infarction thrombosed LV aneurysm."

Upon admission, the patient complained of pressing retrosternal pain and dyspnea with minimal exertion.

From the anamnesis it was known that the patient had experienced pressing retrosternal pain with moderate exertion for 2 years. In February 2017, he suffered acute myocardial infarction with the subsequent formation of the LV aneurysm, despite installing a stent in the infarction-dependent ADA. Within the next three months, there were repeated hospitalizations to medical institutions for increasing heart failure and recurrent hypostatic pneumonia. The patient was hospitalized to the Department of Cardiac Surgery of the N.V. Sklifosovsky Research Institute for Emergency Medicine in order to decide on the possibility of surgical treatment.

The condition of the patient was moderately severe upon examination. Harsh breathing, no rales. Respiratory rate – 19 breaths per minute. Muffled heart sounds, regular rhythm, heart rate (HR) 70 per minute. BP 95/60 mm Hg. Pasty lower legs and feet.

X-ray examination of the chest and lungs revealed no changes (Fig. 1).

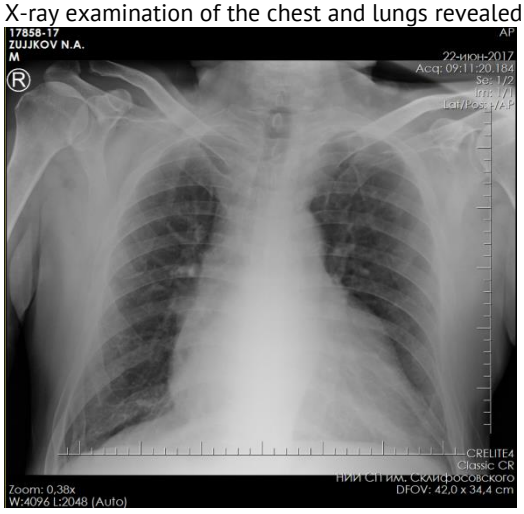

Fig. 1. The survey X-ray of the chest (thickened pulmonary pattern in the upper parts due to moderate venous plethora, a dramatic enlargement of the left heart, partial relaxation of the right dome of the diaphragm).

ECG showed sinus rhythm, signs of scar changes in the anterolateral LV area, aneurysm criteria (persistent ST segment elevation in chest leads from V2 to V4).

Transthoracic echocardiography (EchoCG) revealed a significant increase in the volume of the LV cavity: end-diastolic size (EDS) – 87 mm, end-systolic size (ESS) – 73 mm, end-diastolic volume (EDV) – 267 ml, end-systolic volume (ESV) – 185 ml. The contractile function of the myocardium of the LV was significantly reduced: the ejection fraction (EF) was 32–36%. In the area of the LV apex, interventricular septum, the anterior and lateral walls of the LV (circularly), there

was a giant aneurysm (9x6 cm) with a mural lining thrombus in the cavity. There was pulmonary hypertension of degree I (SPPA – 33 mm Hg) and mitral valve insufficiency of degree I.

According to transesophageal echocardiography, the area of the aneurysmal sac was 37 cm^2, with a diastolic area of LV 34 cm^2 (Fig.2).

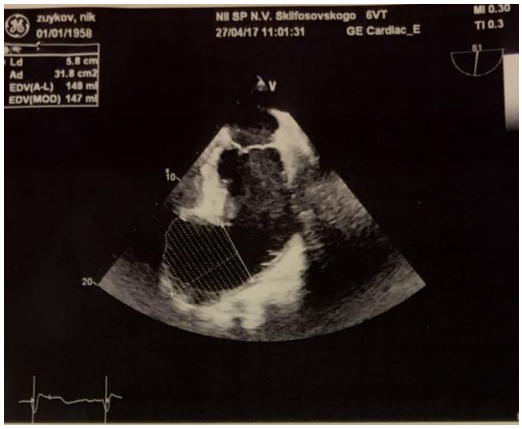

Fig. 2. Trans-esophageal EchoCG, B-mode (the white arrow indicates the LV aneurysm, the red arrow indicates a thrombus in the cavity of the LV aneurysm).

Coronary angiography: left type of blood supply. Critical multivessel lesion of coronary arteries (Fig. 3, 4). The trunk of the left coronary artery (LCA) with irregular contours without significant stenosis. ADA – uneven contours in the proximal third; a stent previously installed in the middle third of ADA, without signs of repeated stenosis, there was a residual stenosis of up to 20%, then uneven contours without significant narrowing. The circumflex artery (CA) – aneurysmal expansion of more than 6 mm in the middle third. The obtuse marginal branch (OMB) – stenosis in the mouth up to 70%, then – without hemodynamically significant contractions. The posterior lateral branch (PLB) – no hemodynamically significant contractions. The posterior interventricular artery (PIA) – stenosis in the middle third up to 70%, then without hemodynamically significant contractions. The right coronary artery (RCA) was poorly developed.

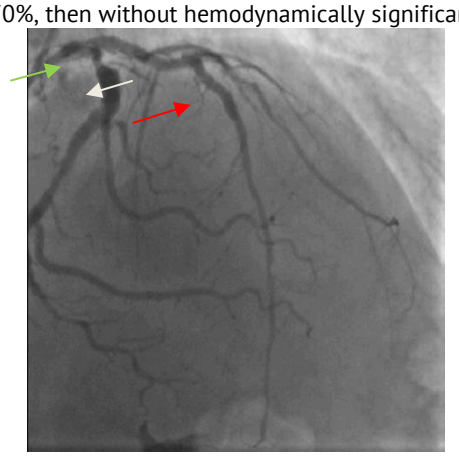

Fig. 3. The left coronary artery: the red arrow indicates the place where the stent was placed in the ADA, the green arrow indicates the place of the stenosis of the CA, the white arrow indicates the aneurysmal expansion of the CA.

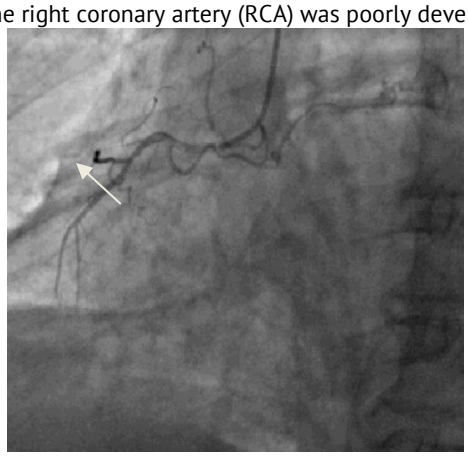

Fig. 4. The right coronary artery (the white arrow). The system is not developed.

The LV ventriculography revealed increased volume, aneurysmal expansion in the anterolateral and apical segment, hypo- and akinesis of the anterobasal segment and normokinesis of the posterior basal and diaphragmal segment. It was not possible to reliably measure EF due to a large LV aneurysm (about 32-38%) (Fig.5).

Fig. 5. LV ventriculography (the red arrow indicates the aneurysmal part of the LV).

Myocardial scintigraphy: significantly enlarged LV (EDV 270 ml) with a focus of absent perfusion in the apex of at least 5.5x5.5 cm. There was a focus of pathological asynchrony at the edge of non-perfusion area, the remaining walls were not altered, hypokinetic, the posterior lateral wall of the left ventricle was most actively functioning; EF 32%. The right ventricle was not enlarged, the right ventricular EF was 37%. The interventricular asynchrony was 120 ms, the LV was the first to contract. The myocardium of the other walls was preserved and potentially viable (Fig. 6).

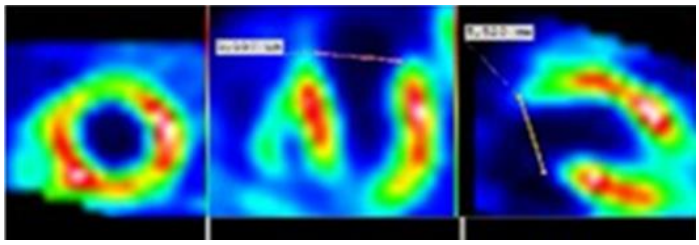

Fig. 6. The tomoscintigraphy image myocardium before surgery.

Additionally, multispiral computed tomography of the heart (MSCT) was performed and revealed cardiomegaly, LV aneurysm from the level of the lower third of the LV to its apex on the posterior wall with a dramatic thinning of the myocardium and the presence of a parietal thrombus (the thickness was 5-9 mm), a small amount of fluid in the pericardial cavity with maximum separation up to 19 mm, atherosclerotic lesion of the aorta and coronary arteries (Fig. 7).

Fig. 7. MSCT of the heart and aorta (the red arrow indicates the aneurysmal part of the LV).

The presence of a critical lesion of the coronary arteries, the giant LV aneurysm with a thrombus, a high risk of recurrent fatal myocardial infarction with the progression of chronic heart failure and hopeless medical therapy were indications for surgical treatment.

On July 7, 2017 the patient underwent surgery: resection of the LV aneurysm, Dor endoventriculoplasty, coronary artery bypass grafting of the circumflex artery and obtuse marginal branch of the left coronary artery.

Intraoperative: the pericardial cavity was filled with loose adhesions. The heart was separated from adhesions by blunt and sharp dissections. The cardiac operative exploration revealed a giant (10x15 cm in diameter) saccular aneurysm in the anterior, lateral, and posterior wall and the apex of the LV. Under conditions of cardiopulmonary bypass and blood cardioplegia, the cavity of the aneurysm was opened in the middle of the scarred area over 10 cm. In the aneurysmal cavity, there were organized thrombotic masses fixed to the wall, which were removed. The LV cavity was repeatedly washed with saline to prevent material embolism. The LV aneurysm of 3.5-5x22 cm was excised. After plication of the interventricular septum, the LV cavity was reconstructed using a xeno-pericard patch of 7x3 cm. (Fig. 8a). The coronary arteries (CA and OMB) were bypassed using an autovenous graft (the large saphenous vein).

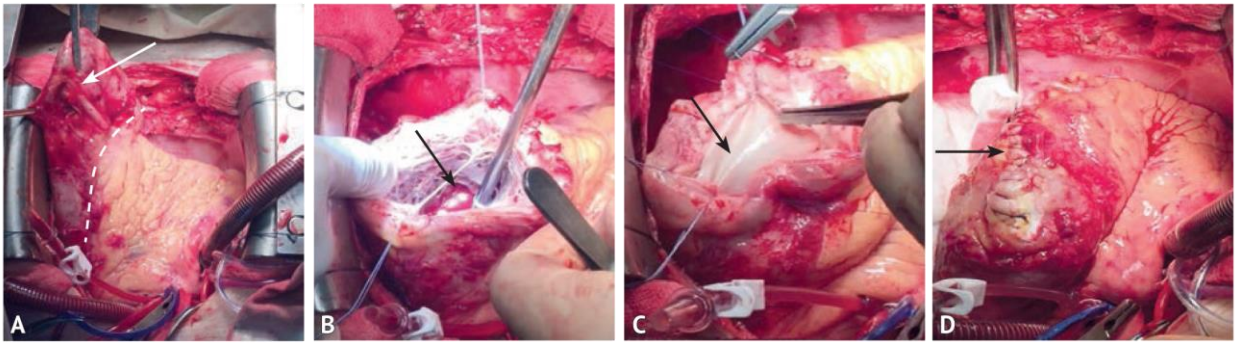

Fig. 8. The intraoperative photo. A - the white arrow indicates the aneurysmal part of the LV. The dashed line indicates the border between the viable myocardium and scar tissue; B -the excised aneurysm. The black arrow indicates the LV septum. C - endoventriculoplasty stage. The black arrow indicates a patch of xeno-pericardium. D - the incision in the LV is sutered with a double row circular stitch (the black arrow).

The control EchoCG in the postoperative period revealed a decrease in LV volumes (EDS – 60 mm, ESS – 49 mm, EDV – 112 ml, ESV – 178 ml) and an increase in EF up to 38%.

The control myocardial scintigraphy before discharge (Fig. 9) showed the decrease in the size of the LV cavity and the growth of EF up to 38%, the diminished perfusion defect at the LV apex (indicated by arrows) and the decreased systolic thickening mainly along the LV anterior wall.

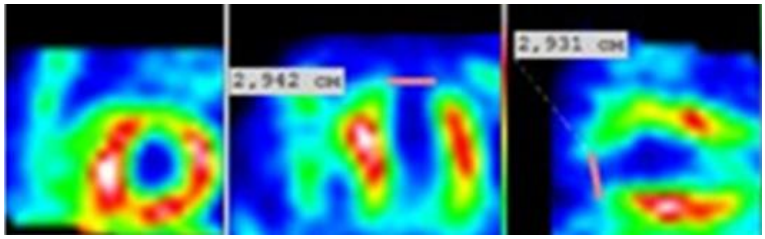

Fig. 9. The tomoscintigraphy image of the myocardium after surgery.

The patient was discharged in satisfactory condition on the day 14 after surgery. The follow-up 6 months after surgery: no complaints, NYHA I heart failure.

According to EchoCG: EDV – 173 ml, ESV – 102 ml, the growth of the LV EF up to 39-41%.

DISCUSSION

The LV aneurysm is a frequent complication of acute extensive myocardial infarction and is mainly located in the apex of the LV due to ADA occlusion and lack of collateral blood flow [1, 2, 3]. Only in 10–15% of observations an aneurysm is detected on the inferior wall of the LV with occlusion of the right coronary artery and even less often develops in the LV lateral wall during occlusion of the circumflex artery [7, 8]. Earlier, it was reported that LV aneurysms developed in 30–35% of patients of the total number of patients with Q myocardial infarction. The proper use of thrombolytic drugs, percutaneous angioplasty and stents as well as drugs that reduce afterload, has significantly reduced the percentage of this serious complication. The frequency of LV aneurysms formation is currently 8–15% [9, 10].

When the LV aneurysm develops, the timely diagnosis is vital because of the threat of serious complications that may accompany (heart failure, arterial thromboembolism, ventricular tachyarrhythmias).

Today, indications for aneurysmectomy still remain controversial. Some authors insist that the presence of resistant ventricular tachyarrhythmias, systemic emboli and refractory heart failure in a patient are good reasons for surgical treatment [5, 6, 10, 11, 12, 13, 14].

Aneurysmectomy combined with coronary artery bypass surgery is indicated in patients with severe heart failure and ineffective conservative therapy (class IIa in accordance with the recommendations of the American College of Cardiology/American Heart Association). At the same time, according to a STICH study (Surgical Treatment for Ischemic Heart Failure), the surgical treatment of LV aneurysms does not lead to an improvement in the functional class in these patients, a decrease in mortality or hospitalizations associated with cardiovascular diseases [4 , 15].

Thus, the indications for aneurysmectomy are set individually by surgeons after assessing the severity of heart failure, measuring LV size, severity of associated mitral insufficiency, condition and area of myocardial scar and myocardial viability in other parts of the LV, the presence of a blood clot in the aneurysm cavity and the risk of systemic embolic complications.

An important point is that such operations should be performed strictly in cardiac surgery centers with high surgical experience.

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