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Giant postinfarction posterolateral left ventricular aneurysm, complicated by mitral insufficiency

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Abstract

Ischemic heart disease holds the leading position in the structure of cardiovascular diseases. Early reperfusion therapy for acute myocardial infarction led to a decrease in mortality and severe complications of coronary artery disease. Despite advances in the treatment of coronary artery disease, dilatation and remodeling of the left ventricle develop in 20% of patients who have had a heart attack, leading to mitral insufficiency and systolic dysfunction of the left ventricle. Aneurysm of the left ventricle is a delayed severe complication of myocardial infarction, which significantly worsens the prognosis. Large aneurysms of the left ventricle cause progressive dilatation of the left ventricle, its volumetric overload with an increase in wall tension in the non-infarction zone, decreased functional

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characteristics of the left ventricle, thrombosis in the aneurysm cavity, life-threatening arrhythmias, and sudden death. Postinfarction left ventricular remodeling can lead to secondary mitral regurgitation, which is an independent predictor of mortality in the long-term period. Surgical treatment of coronary heart disease and its complications is one of the main problems of modern cardiovascular surgery.

Keywords: ischemic heart disease, coronary artery disease, coronary artery disease complications, postinfarction left ventricular aneurysm, ischaemic mitral regurgitation

Conflict of interests. Authors declare no conflict of interest

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AIVB, anterior interventricular branch

BP, blood pressure

CF, circulatory failure

CHF, chronic heart failure

CVC, cardiovascular complication

CVD, cardiovascular disease

ECG, electrocardiogram

EchoCG, echocardiography

EDD, end diastolic dimension

EDV, end diastolic volume

EF, ejection fraction

ESV, end systolic volume

FC, functional class

HF, heart failure

HR, heart rate

IHD, ischemic heart disease

IMA CABG, mammary coronary artery bypass grafting

LCA, left coronary artery

LIMA, left internal mammary artery

LV, left ventricle

MI, myocardial infarction

MV, mitral valve

PASP, pulmonary artery systolic pressure

PCI, percutaneous coronary intervention

RCA, right coronary artery

SPECT, single photon emission computed tomography

TLBAP, transluminal balloon angioplasty

Introduction

Ischemic heart disease (CHD) takes a leading position in the structure of cardiovascular diseases (CVDs). Surgical treatment for coronary artery disease and its complications is one of the main challenges of modern cardiovascular surgery.

Early reperfusion therapy for ST-segment elevation acute myocardial infarction (MI) led to a decrease in mortality and severe complications of coronary artery disease. Despite advances in the treatment of coronary artery disease, left ventricular (LV) dilation and remodeling develops in 20% of patients who have had a heart attack, leading to mitral insufficiency and LV systolic dysfunction [1, 2].

LV aneurysm, being a severe delayed complication of MI, significantly worsens the prognosis. Paradoxical movement of the aneurysmal portion of the myocardium leads to a decrease in cardiac output and the development of congestive heart failure [3]. Large LV aneurysms cause progressive LV dilation, its volume overload with increased wall tension in areas without infarction, decreased LV functional parameters, thrombus formation in the aneurysm cavity, life-threatening arrhythmias, and sudden death [4].

LV aneurysm often develops in extensive transmural MI and, in the vast majority of cases (up to 85%), is localized in the region of the LV apex and anterior wall [5–7]. More rarely, in 3-10% of cases, aneurysm is located on the posterior wall of the left ventricle. In contrast to the aneurysms of the anterior wall and apex, which are predominantly typically true, half of the aneurysms of the posterior localization are false.

It should also be noted that LV remodeling resulted from transmural posterior wall infarctions often leads to a displacement of the papillary muscles, exerting excessive tension on the mitral valve (MV) leaflets and causing loss of their coaptation. As a result, secondary mitral regurgitation develops, which is an independent predictor of mortality in the long term [8–10].

We present a clinical case of a successful surgical treatment of a patient with a giant thrombosed true aneurysm of the inferior LV wall, complicated by mitral insufficiency and severe left ventricular dysfunction.

Clinical Case Report

Patient U., 68 years old, was admitted to the 1st Cardiac Surgery Department of the N.V. Sklifosovsky Research Institute for Emergency

Medicine on December 08, 2020, with the diagnosis reading as follows: “IHD: Stable angina, functional class III [FC III]. Post-infarction cardiosclerosis (primary ST segment elevation MI of the lower LV wall with the formation of a chronic thrombosed aneurysm of the lower LV wall dated February 26, 2020). Atherosclerosis of the aorta and coronary arteries. Condition after percutaneous coronary intervention (PCI): transluminal balloon angioplasty (TLBAP) with stenting of the right coronary artery (RCA) dated February 29, 2020 (1 stent). Hypertension, Grade 3; Grade 3 elevated blood pressure (BP), risk of cardiovascular complications (CVCs) – 4, chronic heart failure (CHF), NYHA Class III-IV; Circulatory failure (CF), 2B stage".

Upon admission, the patient complained of chest pain of a pressing nature, severe shortness of breath on slightest physical exertion, swelling of the legs and feet.

The 68 year old male patient had a long history of severe hypertension.

On February 29, 2020, the patient was admitted to Cardiology Department of the Sklifosovsky Emergency Institute with acute chest pain, severe weakness persistent for 3 days that would be relieved only by narcotic analgesics. There were abnormalities on the electrocardiogram (ECG) (ST segment elevation up to 2 mm in leads II, III, aVF). During previous 3 days, the patient had refused hospitalization. Cardioangiography was performed on admission on an immediate basis. A multivessel lesion of the coronary arteries with acute occlusion of the RCA was identified. PCI on the infarct-associated artery was performed, namely, the angioplasty with RCA stenting. Consulted by a cardiovascular surgeon. An elective surgery of myocardial revascularization was recommended. After discharge from

hospital, the patient reported gradually increasing shortness of breath, the frequency and severity of chest pain.

The physical examination at admission revealed a moderately severe status of the patients, complaints on dyspnea, no auscultative findings pathology of the respiratory system, a systolic murmur on the apex, mild lower limbs edema.

Chest X-ray examination: lungs without fresh focal and infiltrative shadows. Pulmonary pattern with signs of pneumosclerosis. The roots of the lungs: the right one is structural, not expanded; the left one is overlapped by a median shadow. The shadow of the mediastinum is not displaced. The shadow of the heart is moderately expanded in diameter transversely due to the left sections. The aorta is indurated. The diaphragm is clear, normally located.

ECG: sinus rhythm, heart rate 75 beats/min, normal axis, q wave in in leads II, III, aVF, negative T-wave in leads II, III, aVF (Fig. 1).

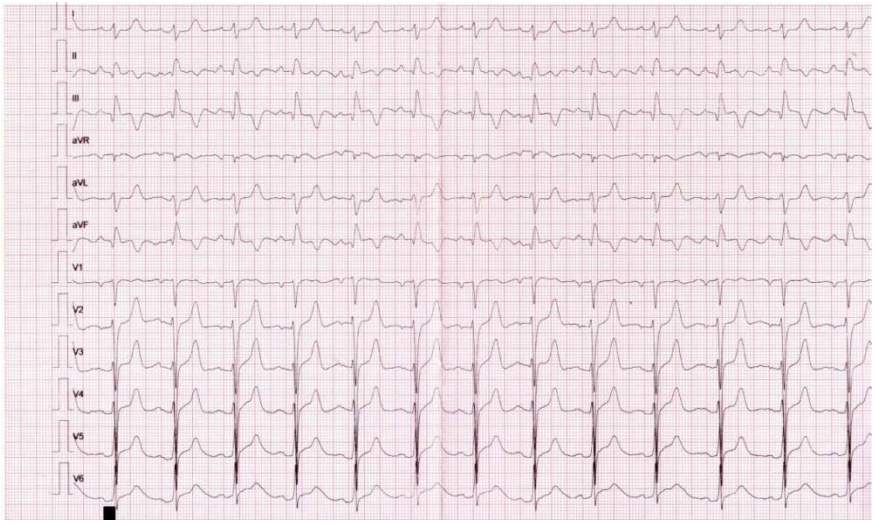


Fig. 1. Electrocardiogram in 12 leads prior surgery. Regular sinus rhythm, heart rate is 78 beats/min. Q wave, and T wave inversion in leads II, III, aVF

Transthoracic echocardiography (EchoCG) demonstrated a significant increase in the size and volume of the left heart chambers: LV end diastolic dimension (EDD) was 89 mm, end diastolic volume (EDV) was 290 mL, end systolic volume (ESV) was 212 mL. The left ventricle contractility function was significantly reduced: the LV ejection fraction (EF) was 26%; while the EF of the LV contracting part was 46%. Along the inferior wall, a giant aneurysm of 66x65 mm in size, 180 ml in volume with the presence of parietal thrombotic masses and with the effect of spontaneous contrast enhancement of blood in the aneurysm cavity was identified. Mitral valve insufficiency of grade 2–3. Pulmonary hypertension of grade 1. (Fig. 2–4).

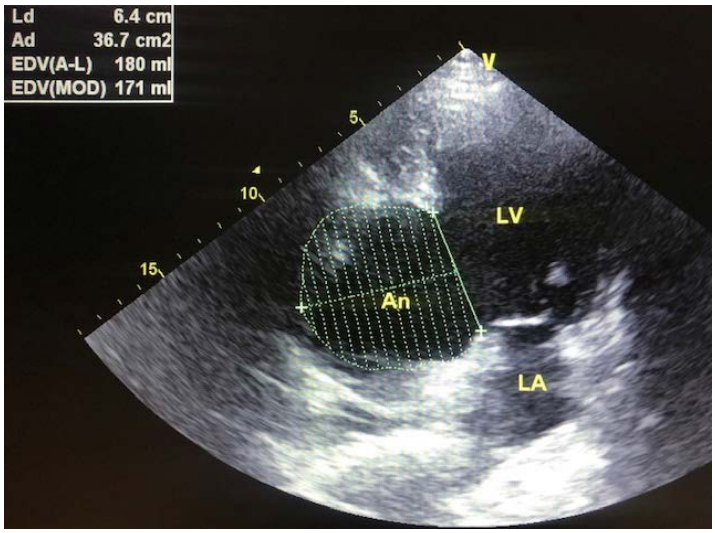


Fig. 2. Echocardiogram. Apical 2-chamber position. LV, left ventricle; LA, left atrium; An, left ventricle aneurysm

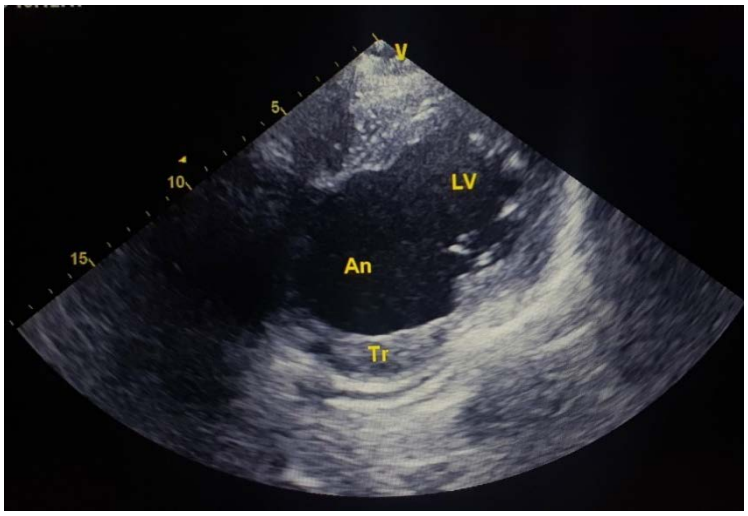


Fig. 3. Echocardiogram. Parasternal position, short axis. LV, left ventricle; An, left ventricle aneurysm; Tr, thrombus

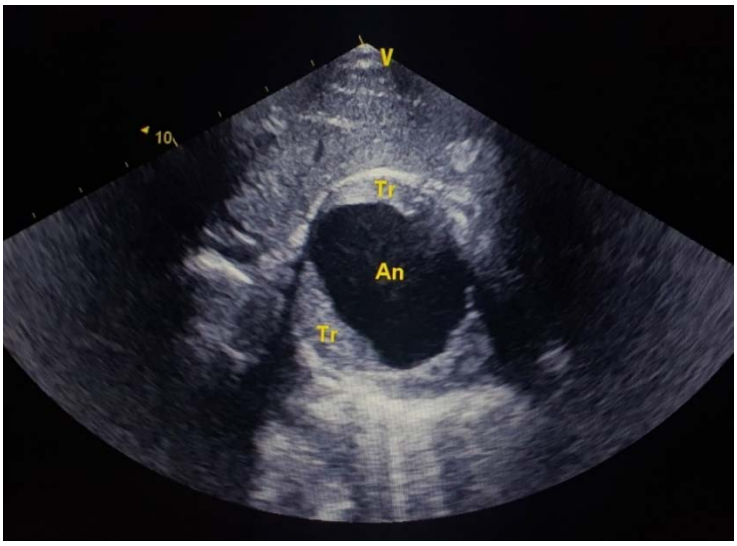


Fig. 4. Echocardiogram. Subcostal position. An, left ventricle aneurysm; Tr, thrombus

Coronary angiography (Fig. 5, 6) revealed a multivessel lesion: the type of coronary blood supply was balanced; the trunk of the left coronary artery (LCA) was normally developed, un-changed; the anterior interventricular branch (AIVB) had uneven contours, stenosis up to 75% in the proximal third, stenosis 70% in the middle third; the diagonal branch was

60% stenosed in the middle third; RCA had uneven contours, restenosis up to 85% in a previously implanted stent in the middle third; the circumflex branch and obtuse marginal branch were without hemodynamically significant stenoses.

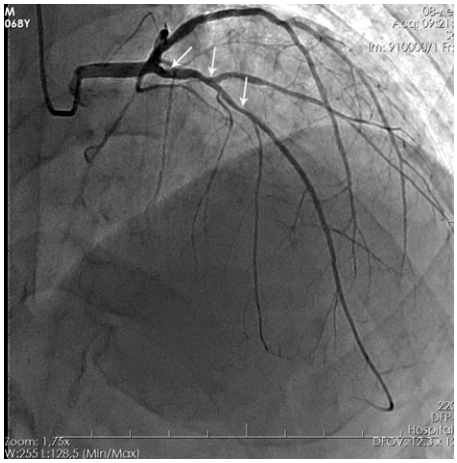


Fig. 5. Selective coronary angiogram of the left coronary artery. Arrows indicate stenoses of the anterior interventricular branch (ostial, bifurcation in the proximal third, postbifurcation - at the border of the proximal third and the middle third)

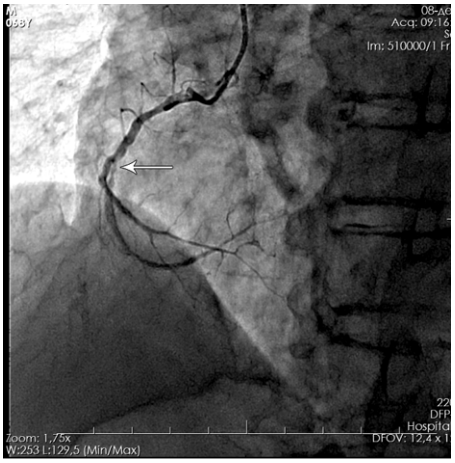


Fig. 6. Selective coronary angiogram of the right coronary artery. The arrow indicates critical stenosis of the right coronary artery

Ventriculography (on 08.12.2020) revealed an aneurysmal expansion

of the inferior wall up to 7.5 cm in size (Fig. 7, 8).

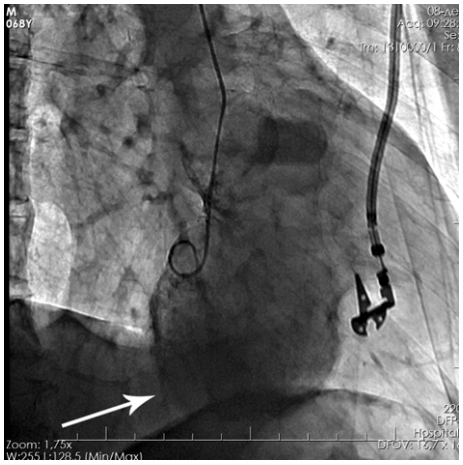


Fig. 7. Aneurysm ventriculogram at systole. The arrow indicates an aneurysm of the left ventricle

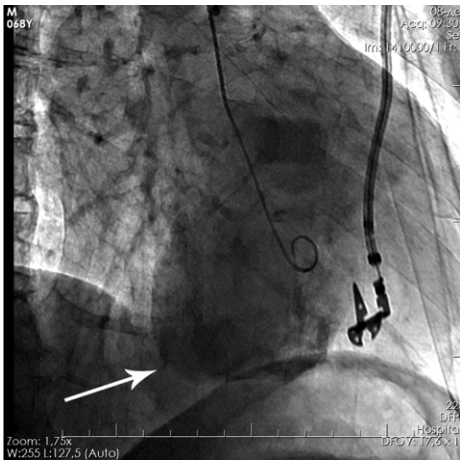


Fig. 8. Aneurysm ventriculogram at diastole. The arrow indicates an aneurysm of the left ventricle

According to multislice spiral computed tomography of the heart with ECG synchronization (Fig. 9), a giant aneurysm of the basal parts of the LV inferior wall with parietal thrombosis was visualized.

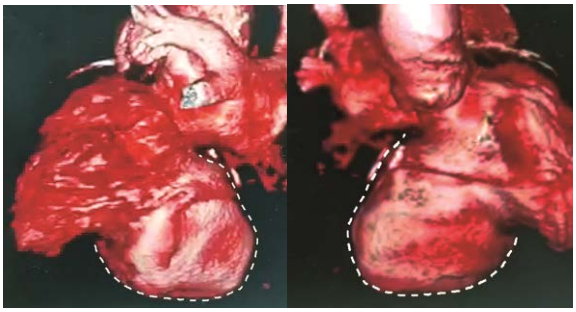
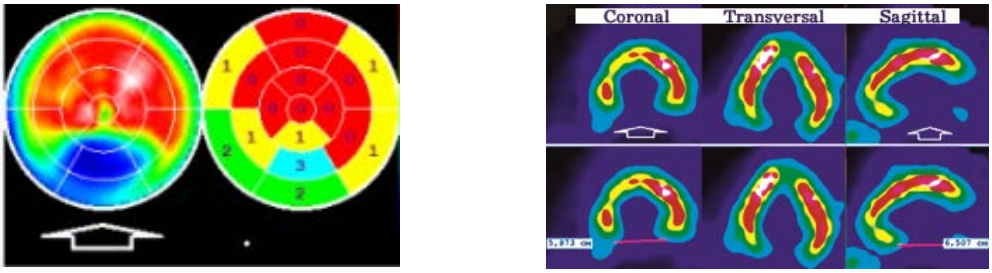


Fig. 9. Multislice spiral computed tomography of the heart before surgery. A giant aneurysm in the basal sections of the left ventricle inferior wall is circled by a dotted line

The single-photon emission computed tomography (SPECT) of the myocardium perfusion visualized the wall of the enlarged LV (LV EDV = 230 ml), a focal transmural absence of perfusion of the LV diaphragmatic wall 6x6 cm in size, diffused hypokinesis and foci of intraventricular pathological asynchrony (Fig. 10 A, B) .



A B

Fig. 10. Single photon emission computed tomogram: a series of tomography scintigrams visualizes significantly decreased perfusion of the anterior and lateral walls of the left ventricle, a focal transmural lack of perfusion in the diaphragmatic wall 6x6 cm in size (indicated with arrows). Color scheme of myocardial perfusion disorders: red means normal perfusion, yellow means moderately decreased perfusion, green means significantly decreased perfusion, blue shows no radiopharmaceutical accumulation: A, single photon emission computed tomography of the "bull's eye" myocardium in diastole before surgery; B, single photon emission computed tomogram before surgery (coronal, transversal, and sagittal sections)

Summing up the results of all the above studies, we denote the following we denote the following: in the presence of hemodynamically significant stenoses in the coronary arteries, a giant thrombosed LV aneurysm, which caused LV dilation with further progressive systolic dysfunction, as well as in the presence of significant mitral insufficiency, a high risk of recurrent myocardial infarction with the development of embolism in the arterial bed, and the futility of drug therapy, we made the decision to perform a surgical treatment.

The patient underwent an LV aneurysm resection thrombectomy, LV endoventriculoplasty to Dor, MV replacement with On-X Valves SZ 25 mm mechanical prosthesis, a direct myocardial revascularization with left internal thoracic artery (LIMA) to left anterior descending artery.

Surgery description. The heart was accessed through the median sternotomy. Connecting a heart-lung machine according to the scheme: aorta − superior, inferior vena cava. Antegrade non-selective cardioplegia with blood cardioplegic solution (St. Thomas' hospital solution). Intraoperatively revealed: a giant linear LV aneurysm, tightly attached to the diaphragmatic part of the pericardium and extending along the inferior wall from the LV apex to the MV annulus. The aneurysm was incised for 5 cm in the middle of the scarred area. A dense thrombus 10x9 cm in size was removed from the LV cavity (Fig. 11). A portion of LV scarred tissue measuring 10x10 cm was excised. Ventriculoplasty according to Dor was completed by placing a collagen-coated polyester patch (Fig. 12). The LV wall was sutured with a two-row twist suture (Fig. 13). Access to the mitral valve was obtained according to Guiraudon through the right atrium and the interatrial septum. At revision, a MV coaptation impairment was noted due to a pathological

modification of the anterior leaflet and its prolapse. The modified anterior leaflet of the mitral valve was excised along with the subvalvular structures. MV was prosthetized with a mechanical prosthesis. The myocardium was revascularized in the extent of IMA CABG: LIMA from the AIVB.

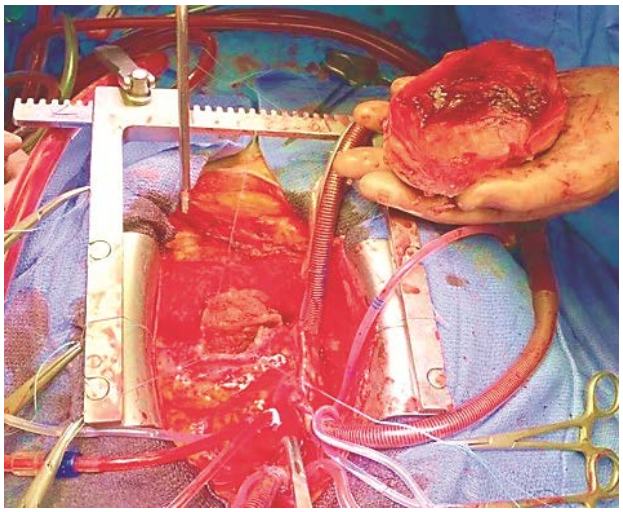


Fig. 11. Intraoperative photo. In the hand of the surgeon, there is an excised portion of the left ventricle aneurysm with thrombus (10x10 cm)

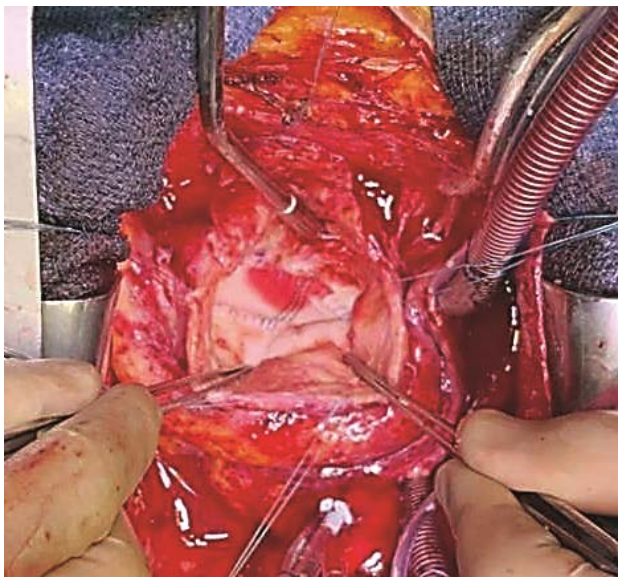


Fig. 12. Intraoperative photo. Polyester patch of the left ventricle

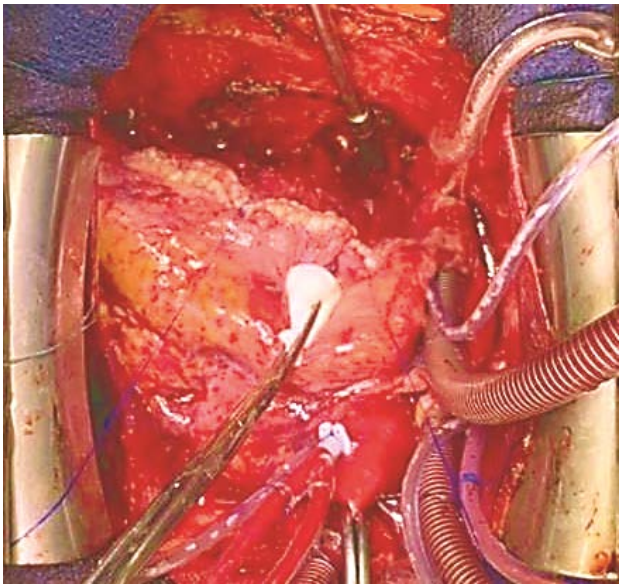


Fig. 13. Intraoperative photo. Double row suture of the left ventricle

The early postoperative period proceeded without any signs of heart or respiratory failure. EchoCG showed a reduction in the LV volume (EDV 143 ml, ESV 86 ml), an increase in LV EF up to 39-41%. The function of the prosthesis in the mitral position was satisfactory: the mean gradient was 3.4 mm Hg, transprosthetic regurgitation of grade 1.

The patient was discharged to the outpatient treatment in a satisfactory condition on the 15th day.

At dynamic examination after 2 months of the follow-up: there are no complaints; heart failure of NYHA functional class I. The EchoCG data: LV EDV is 145 ml, LV ESV is 75 ml, EF is 48%, the prosthesis function is satisfactory (Fig. 14-16).

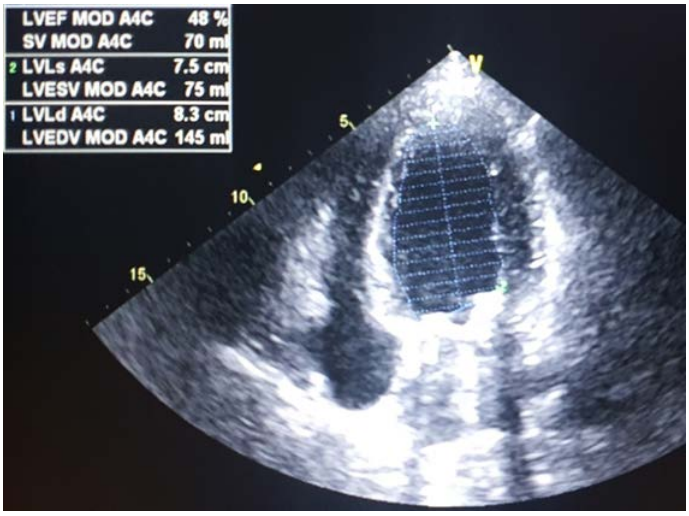


Fig. 14. Echocardiogram. Apical 4-chamber position. The end diastolic volume of the left ventricle is 145 mL, the left ventricle ejection fraction is 48%

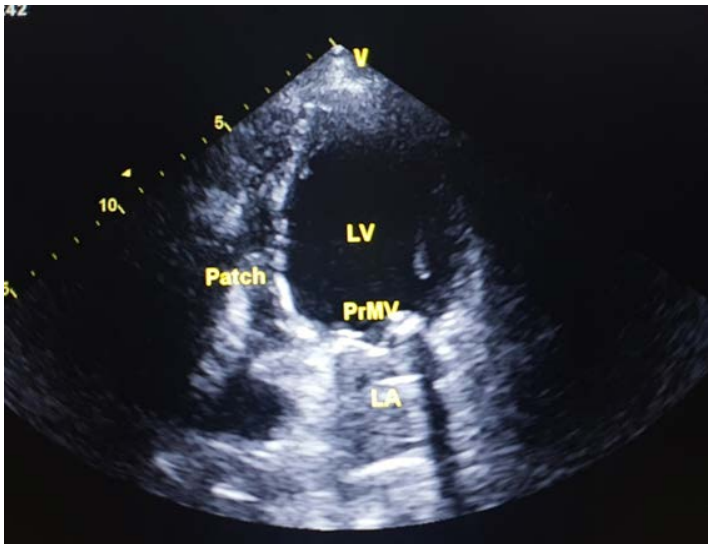


Fig. 15. Echocardiogram. Apical 2-chamber position. LV, left ventricle; LA, left atrium; PrMV, mitral valve prosthesis; Patch, left ventricle patch

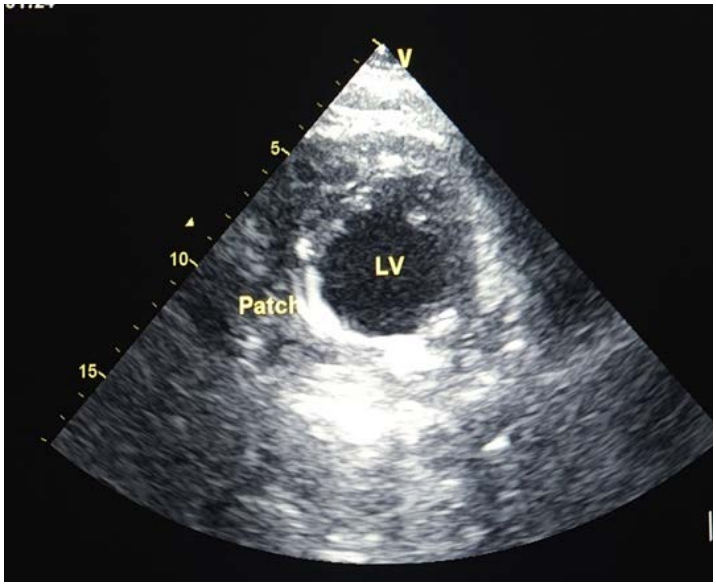


Fig. 16. Echocardiogram. Parasternal position, short axis. LV, left ventricle; Patch, left ventricle patch

The results of the control SPECT (Fig. 17 A, B, 18) demonstrate a significant reduction in the LV volume, functional recovery myocardium segments, mainly the LV anterior and lateral walls, and an increase in LV EF (from 25 to 48%).

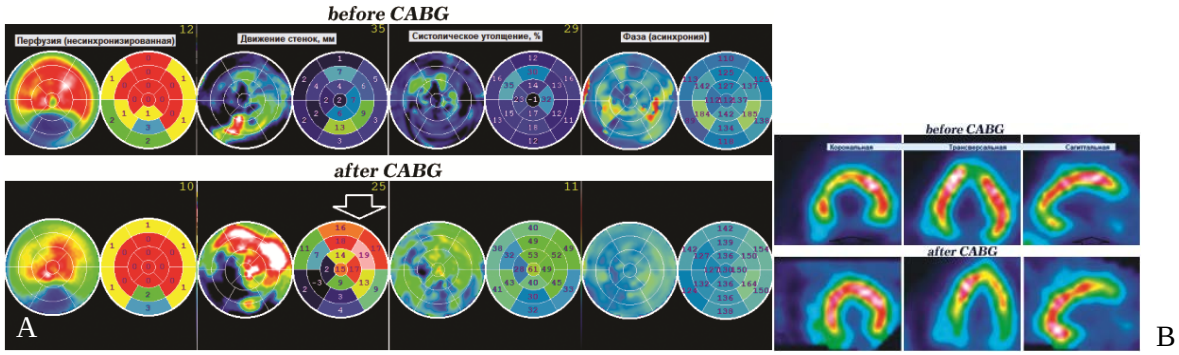


Fig. 17. A, single photon emission computed tomogram of the "bull's eye" myocardium in diastole before and after surgery; B, single photon emission computed tomogram of the myocardium (transversal, coronal, and sagittal sections) before and after surgery

On a series of control tomoscintigrams (Fig. 17 A, B) recorded at rest, there is an improvement in myocardial perfusion, mainly in the left ventricle anterior and lateral walls.

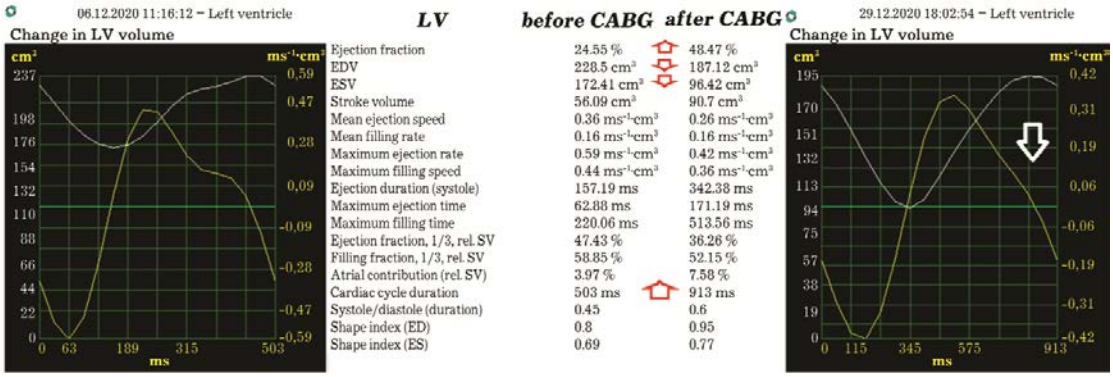


Fig. 18. When comparing the parameters of the left ventricle before and after the operation, a significant reduction in the left ventricle volume and the increase in the left ventricle ejection fraction from 25 to 48% were noted (indicated by red arrows)

Discussion

Postinfarction LV aneurysms are formed in 10–35 % of cases of acute myocardial infarction [11, 12]. Currently, there is a trend towards a decrease in the incidence of this complication as a result of the widespread use of PCI followed by coronary artery stenting, and also the drugs that reduce afterload [13–15].

Nevertheless, a postinfarction aneurysm remains a serious complication leading to the development of heart failure, arrhythmias, and arterial thrombosis [16, 17].

LV aneurysm is predominantly localized in the apex and occurs due to AIVB occlusion and the lack of the developed network of intersystem and

intrasystem collaterals [18]. The incidence of posterior aneurysms varies within 10%, meanwhile the aneurysms not affecting the mitral valve complex structure are extremely rare [14, 19, 20]. The development of progressive mitral valve insufficiency after posterior inferior MI complicated by an aneurysm is usually associated with the reposition of ischemic papillary muscles due to LV dilation and remodeling [21, 22].

A number of studies have revealed the prognostic significance of ischemic mitral insufficiency. M.S. Feinberg et al. demonstrated that the annual mortality among patients with AMI was 4.8% in absent mitral insufficiency, 12.4% in case of mild or moderate one, and 24% in severe mitral insufficiency [23]. Thus, both moderate and severe ischemic mitral regurgitation determine a poor prognosis and are associated with a significant increase in the risk of developing heart failure (HF) and death.

Drug treatment in patients with severe chronic HF is low effective; a five-year survival rate is no more than 40% [17, 24]. The only method to improve the prognosis and reduce mortality in patients with postinfarction aneurysms, mitral insufficiency, and progressive heart failure is a surgical intervention aimed at an adequate reconstruction of the left ventricle, improvement of its contractility, and restoring MV function [7].

However, the operation in such patients is associated with a high risk of in-hospital mortality. Factors influencing the possibility of an unfavorable outcome include: low LV EF (less than 30%) [15, 25–35], cardiac index lower than 2.1 mL/min/m^2, the pulmonary artery systolic pressure (PASP) over 33 mm Hg, creatinine exceeding 180 μmol/L [21], severe mitral regurgitation [27, 28], NYHA Class III-IV heart failure, and age ≥ 75 years [29].

In the described case, the patient had a high risk of surgical

intervention: a giant aneurysm of the posterior wall, exceeding the size of the left ventricle, severe systolic and diastolic dysfunction, mitral insufficiency, grade 2 pulmonary hypertension, CHF of III-IV FC. The only efficient way to improve the patient's condition would be surgical treatment.

It should be noted that one of the key points of successful surgical treatment for LV aneurysm is preoperative planning of the scope of the intervention using multimodal imaging [36, 37]. Two-dimensional echocardiography is the gold standard, since it allows determining the location and size of the aneurysm, gives an idea of the LV systolic and diastolic function, the presence of thrombus, and the MV functional impairment. Meanwhile, when determining the expediency of surgical treatment, the EF of the LV contracting part is rather more important than the total LV EF is [30]. Computed tomography provides detailed information about the location of the aneurysm, the extent of its impact on the LV and MV geometry, as well as about potential LV remodeling after surgical reconstruction. MRI or perfusion scintigraphy gives an idea of the viability of the ischemic myocardium not involved in the aneurysm. Polypositional contrast ventriculography allows visualization of the aneurysm, evaluation of LV volume parameters and the extent of mitral valve insufficiency.

Despite the use of various imaging modalities with great diagnostic accuracy, the differential diagnosis between true and false aneurysms can be difficult. In our case, this diagnostic dilemma also arose. The inferior posterior location was suggestive of a false aneurysm, as only a small percentage of posterior aneurysms are thought to be true ones [31]. At the same time, a number of authors, based on the results of 65 autopsy sections, showed an equal distribution of aneurysms by location [7]. The absence of

contrast enhancement of the coronary arteries in the aneurysm wall during coronary angiography also testified in favor of a false aneurysm [32, 33]. On the other hand, the wide neck of the aneurysm (the ratio of less than 0.5 between the width of the neck and the maximum internal diameter of the aneurysm) and the absence of bidirectional blood flow through it [34, 35] suggested a true aneurysm. It was also impossible to trace the myocardium continuity and determine if there were thrombotic masses inside or outside the LV cavity. The intraoperative assessment did not clarify either; only a histological examination finally established that the aneurysm was true.

At present, debatable remains the question of the expediency of performing resection of an LV aneurysm with subsequent surgical reconstruction of its cavity [38]. The decision is made on an individual basis and should consider such factors as: the LV and aneurysmal cavity volume, the severity of mitral insufficiency, the location and extent of scar tissue, LV myocardial reserve, the presence of thrombotic masses, the severity and progression of heart failure, and the efficacy of conservative therapy [20].

In our case, the indisputable indications for surgical treatment were: the patient's having a giant posterior aneurysm with large thrombotic masses in its cavity, a high risk of developing arterial embolism, heart failure refractory to conservative therapy, the mitral valve dysfunction, hemodynamically significant stenosis of AIVB, and also satisfactory values of EF of the LV contracting part [39].

Conclusions

1. Despite the widespread use of percutaneous coronary intervention for early reperfusion of the coronary arteries in acute myocardial infarction, postinfarction aneurysms remain a serious problem leading to the

development of congestive heart failure, ventricular arrhythmias, and embolic complications.

2. Aneurysms of the myocardium posterior wall often cause a change in the left ventricle geometry with deformation of the mitral valve structures and the development of mitral valve incompetence. In this case, the goal of the surgical intervention is the aneurysm resection, the correction of the left ventricle geometry and volumes, restoration of mitral valve function, and revascularization of the ischemic myocardium in presence of hemodynamically significant stenoses of the coronary arteries.

3. The key to a successful surgical treatment is the preoperative planning of the intervention using multimodal imaging.

4. Differential diagnosis between a true and false aneurysm can be difficult. Verification requires a thorough study of the obtained images using various instrumental methods, visual intraoperative evaluation, and a mandatory histological examination.

5. The case we have described demonstrates that a thorough preoperative assessment and planning of the intervention, as well as the choice of optimal surgical tactics, made it possible to achieve a successful result in a patient with a high risk of surgical treatment, restore the function of the left ventricle, mitral valve and, by reducing the progression of heart failure, significantly improve the patient's quality of life.

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