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Successful Surgical Treatment of Postinfarction Rupture of Left Ventricular Myocardium

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ABSTRACT The rupture of the left ventricle free wall is one of the most dangerous complications of myocardial infarction. Due to the widespread availability of echocardiography method, the detection of this fatal complication and the number of lives saved after surgery grew. The survival of patients depends on early diagnosis, stabilization of the patient's condition, promptness and tactics of surgical intervention. We report a case of successful closure of a rupture of the left ventricle free wall on the 15th day after myocardial infarction.

Keywords: myocardial infarction, myocardial rupture, cardiac tamponade, suture myocardial plasty
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ADA — anterior descending artery
BP — blood pressure
CA — circumflex artery
EchoCG — echocardiography
ECG — electrocardiography
EF — ejection fraction
LCA — left coronary artery
LV — left ventricle
MI — myocardial infarction
OMB — hypertensive disease
RCA — right coronary artery

INTRODUCTION

Postinfarction rupture of the left ventricular (LV) myocardium is a rare but most dangerous mechanical complication of myocardial infarction (MI).

Rupture of the LV free wall occurs in 2–4% of cases of MI, while being the cause of 20–25% of all deaths associated with heart attack [1]. It should be noted that the real incidence of this complication is probably higher than these figures, since a significant part of patients die suddenly before the diagnosis is verified [2].

Classic risk factors are old age [3], female gender [4], no history of angina or heart attack [5–9], first transmural anterior or lateral infarction [10–13], and persistent ST segment elevation by more than 2 mm [14] and recurrent chest pain. Although urgent reperfusion is considered necessary to reduce the risk of LV free wall rupture, studies have shown that late or unsuccessful thrombolysis is associated with an increased rupture rate [15–17].

Mortality in LV myocardial rupture is very high and amounts to about 95% [18]. Patient survival depends on early diagnosis, hemodynamic stability, and the promptness of surgical intervention. But even with surgical treatment, the mortality rate ranges from 25 to 50%. Higher mortality was observed in patients operated on shortly after the development of myocardial infarction [19], in patients with cardiogenic shock [20-22], as well as in the short interval between the onset of myocardial infarction and rupture [23].

Clinical case

A 63-year-old male patient B. was admitted on an emergency basis to N.V. Sklifosovsky Research Institute for Emergency Medicine on January 24, 2019 with a diagnosis of coronary heart disease. Primary myocardial infarction of the high lateral parts of the LV with ST elevation from 07.01.19, Atherosclerosis of the aorta, coronary arteries. Condition after percutaneous coronary intervention from 01/07/19: transluminal balloon angioplasty with stenting of the anterior descending artery (ADA) (three stents). Rupture of the LV wall, hemopericardium, condition after puncture of the pericardium on Jan 18, 2019 and Jan 21, 2019

Background: hypertension (HD) grade 3, grade III, risk of cardiovascular complications — 4. Diabetes type 2, target HbA1c level <8%.

Complications: paroxysm of atrial flutter (paroxysm dated Jan 17, 2019). CHA2DS2-VASc risk — 2 points. HAS-BLED — 4 points.

Concomitant diseases: Encephalopathy of mixed origin, obesity of the 1st degree.

Upon admission, the patient complained of weakness, shortness of breath, and dizziness.

It is known from the anamnesis that the patient suffers from hypertension for a long time with a maximum increase in blood pressure (BP) up to 180/100 mm Hg. Adapted to BP figures 120/80 mm Hg. No myocardial infarctions or cerebrovascular accidents in history.

The patient was hospitalized on Jan 07, 2019 in Klin City Hospital with a diagnosis of MI of high lateral LV with ST elevation (Fig. 1).

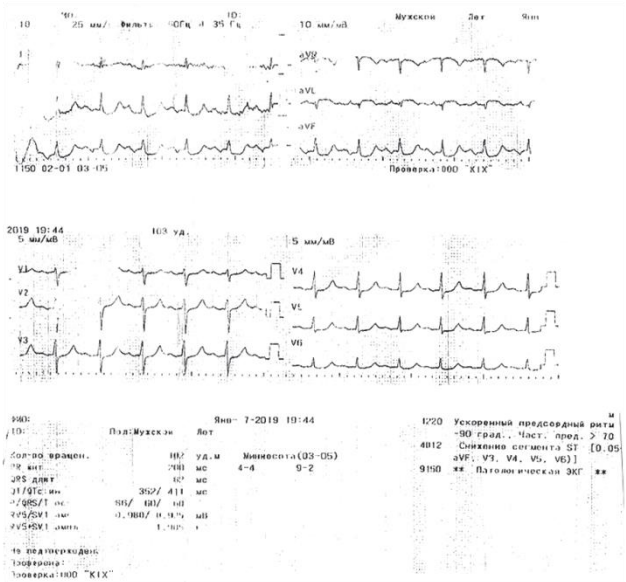

Fig. 1. ECG of 63-year-old male patient B. dated January 7, 2019: myocardial infarction of the high lateral parts of the left ventricle with ST elevation

Coronary angiography: the right type of blood supply. Trunk of the left coronary artery (LCA), circumflex artery (OA), right coronary artery (RCA) — irregular contours, anterior descending artery (ADA) — extended stenosis in the proximal third up to 80% and occlusion in the middle third, obtuse marginal branch (OMB) — 50% stenosis in the middle third (Fig. 2, 3).

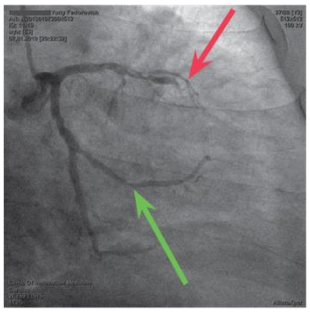

Fig. 2. Left coronary artery. The red arrow indicates occlusion of LADA, the green arrow indicates stenosis of OMB

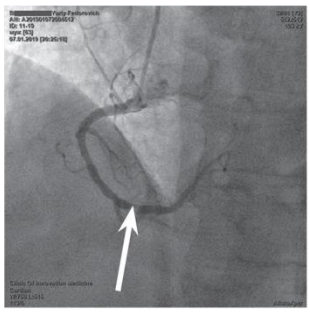

Fig. 3. Right coronary artery (the white arrow)

Balloon angioplasty and stenting of "infarction-associated" ADA were performed (two uncoated stents were installed - Nexgen 2.5x24 mm; Rebel 3.5x24 and 1 drug-eluting stent Promus 3.5x8 mm). Echocardiography (EchoCG) dated January 14, 2019: heart chambers were not dilated. Myocardial contractility was satisfactory. There were no violations of local myocardial contractility. Ejection fraction (EF) 62%. The systolic pressure in the pulmonary artery was 19 mm Hg. No fluid was found in the pericardial cavity. The patient was discharged in satisfactory condition on January 17, 2019. After arriving home, when leaving the car, the patient lost consciousness and fell. An ambulance team was called. Electrocardiography (ECG): paroxysm of atrial flutter. A decrease in blood pressure was noted. The patient was urgently taken again to "Klin City Hospital in the intensive care unit. ECG: atrial flutter, irregular form with atrioventricular conduction (AV conduction) 2: 1.3: 1, tachyarrhythmia. The frequency of ventricular contractions was from 105 to 150 beats/min. Ventricular complex aberration. In the basal parts of the lateral wall, there were signs of focal changes (Fig. 4).

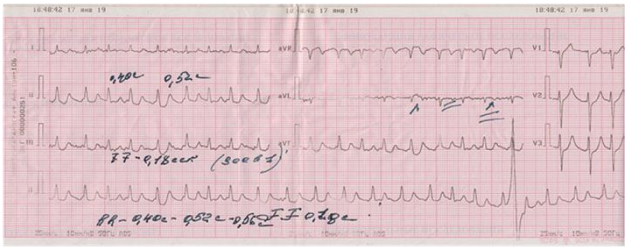

Fig. 4. ECG of a 63-year-old male patient B. dated January 17, 2019: atrial flutter, irregular form with AV conduction 2:1, 3:1, tachyarrhythmia. The frequency of contractions of the ventricles from 105 to 150 beats per minute. Aberration of the ventricular complex. Local abnormalities in the basal sidewall

Sinus rhythm was restored with the help of electro-pulse therapy, hemodynamics was stabilized (Fig. 5).

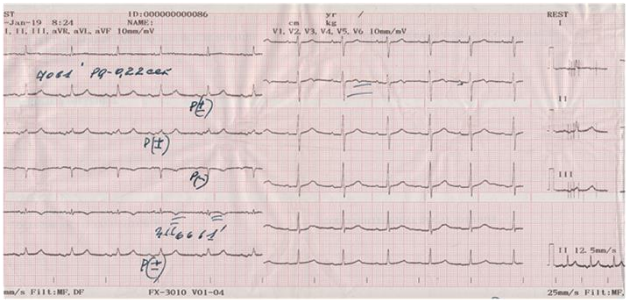

Fig. 5. ECG patient B. dated January 17, 2019. Recovery of sinus rhythm and stabilization of hemodynamics after cardioversion

Echocardiography revealed the presence of fluid in the pericardial cavity without signs of tamponade (1.8–2.3 cm in the apex, 0.6 cm along the anterior and posterior walls, and 0.4 cm in the basal regions) (Fig. 6).

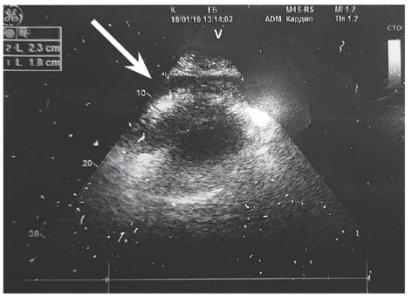

Fig. 6. The ultrasound of the pericardial cavity. The white arrow indicates uncoupling of pericardial leaflets

When performing ultrasound examination and chest X-ray, fluid was revealed in the pleural cavities: pleural layers separation at the level of the sinus on the left — 18 mm, on the right — 30 mm. A puncture of the pericardial cavity was performed on Jan 18, 2019. Evacuation of 90 ml of blood. The drain tube was removed. In the dynamics, an increase in fluid was noted in the pericardial cavity (2.5 cm in the apex, along the anterior and posterior walls — 1.7 cm and 1.4 cm in the basal regions). Repeatedly on 21.01.19, puncture of the pericardial cavity was performed. Evacuation of 330 ml of blood, drainage tube retained. In the biochemical analysis of blood, an increase in the level of alanine aminotransferase, aspartate aminotransferase up to 960 units was noted, so Ticagrelor and Heparin were cancelled. The patient was consulted by a cardiologist. Cardiac magnetic resonance imaging was recommended. It was decided to transfer the patient to a specialized medical institution for further examination and further treatment. The patient was urgently hospitalized at the N.V. Sklifosovsky Research Institute for Emergency Medicine on January 24, 2019

The patient's condition upon admission was stable and severe. Self-breathing through natural airways. SPO_2 without oxygen insufflation 98%. Rough respiration upon auscultation. No wheezing or crackles. The respiratory rate was 19 per minute. Stable hemodynamics. Systolic pressure 110 mm Hg, diastolic pressure 70 mm Hg, pulse rate 72 beats / min. There was no pulse deficit. Sinus heart rhythm, correct. Heart sounds: muffled. The heart rate was 72 per minute.

Transthoracic echocardiography: the chambers of the heart not enlarged. Moderate LV myocardial hypertrophy. Global LV systolic function was preserved, ejection fraction was 55% (according to Simpson). Local LV systolic function was not impaired. The cusps of the aortic and mitral valves were thickened. Mitral regurgitation grade 0-1. Tricuspid regurgitation grade 0-1. Diastolic function was not impaired. There were no signs of pulmonary hypertension. Moderate amount of fluid in the pericardial cavity. Ultrasound examination of the pleural cavities in the sitting position of the patient did not reveal separation of the pleural layers.

According to emergency indications, multispiral computed tomography of the chest organs was performed. Signs of a covered rupture of the anterior wall of the LV were revealed (in the basal parts of the anterior wall of the LV, a transmural defect measuring 10x13 mm was visualized, outside covered with a hyperdense clot. The described defect was located 11 mm lateral from the upper third of the ADA), partially drained hemopericardium (600 cm^3) (Fig. 7). Emphysematous and congestive changes in both lungs, mediastinal lymphadenopathy; bilateral hydrothorax (in the pleural cavities on both sides, a small volume of fluid).

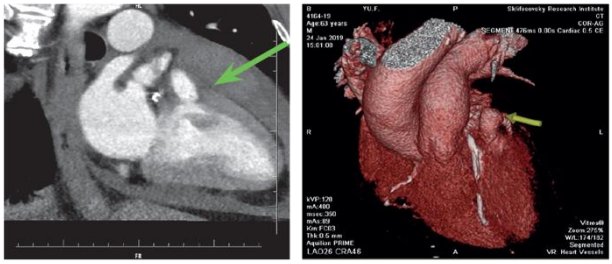

Fig. 7. MSCT of the heart and aorta (the green arrow indicates the area of the aneurysmal protrusion of the LV wall)

In order to assess the state of the coronary arteries and previously installed stents, to exclude mechanical damage to the coronary arteries and to clarify their location in relation to the site of the myocardial defect, the patient underwent control coronary ventriculography. Results: the trunk of the left coronary artery, CA, RCA had uneven contours. ADA had irregular contours, previously stented segments without signs of thrombosis and restenosis (Fig. 8, 9).

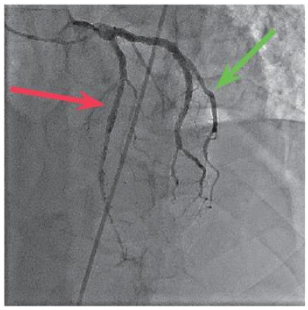

Fig. 8. Left coronary artery: the red arrow indicates the location of stent in the LADA, the green arrow indicates the stenosis of OMB

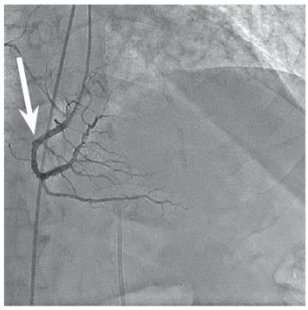

Fig. 9. Right coronary artery (the white arrow)

Left ventriculography: in the basal part of the anterior wall of the LV, an area of paradoxical contractility and an adjacent pathological contrast zone, suspicious of extravasation, are determined (Fig. 10).

Considering that the patient had a myocardial rupture against the background of a subacute course of myocardial infarction of the anterior wall, septum, apex, lateral wall of the LV with ST segment elevation dated Jan 7, 2019, hemopericardium with

signs of cardiac tamponade, the patient was transferred to the cardiac surgery operating room to perform an emergency order according to vital indications.

The patient underwent surgery on Jan 24, 2019: revision, sanitation of the pericardial cavity. Suturing of the LV anterior wall defect.

Intraoperatively: in the pericardial cavity, there was about 600 ml of liquid lysed blood, blood clots at different stages of formation. The cavity is sanitized as much as possible. In the region of the basal part of the anterior wall, closer to the projection of the intermediate artery, a massive hematoma was revealed, covered with clots from the outside under the epicardium (Fig. 11).

Under the conditions of artificial circulation, clots were removed and the epicardium was cut. A through defect of the LV wall with dimensions of 10x25 mm was found (Fig. 12).

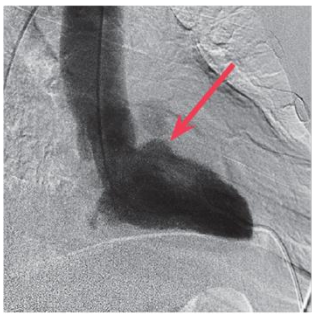

Fig. 10. Ventriculography of the left ventricle (the red arrow indicates the defect of the left ventricle)

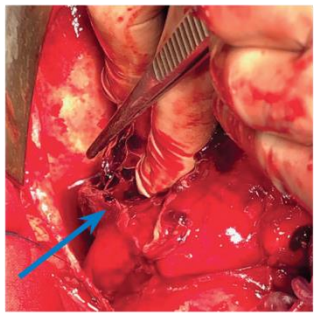

Fig. 11. The blue arrow indicates massive hematoma under the epicardium covered with clots

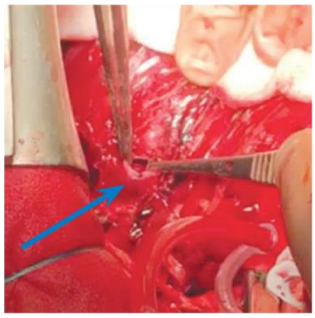

Fig. 12. The blue arrow shows the left myocardial rupture

Sequential suturing of the LV wall with three rows of sutures was performed: on the endocardium, on the myocardium, on the epicardium with Ethicon-Prolene 4 / 0-17 thread. External sealing of the seam line with an autopericardium using a two-component biological adhesive BioGlue.

The early postoperative period was uneventful.

In the control echocardiography in the postoperative period, the cardiac cavities were not dilated. LV contractile function was satisfactory. End-diastolic size 47 mm, end-systolic size 33 mm, end-diastolic volume 97 ml, end-systolic volume 44 ml. EF up to 56%. The patient was discharged on the 12th day in a satisfactory condition.

DISCUSSION

In 1647, the outstanding anatomist W. Harvey discovered and described in detail a rupture of the free wall of the LV with concomitant hemotamponade of the heart and severe calcification of the coronary arteries for the first time [24]. Later T. Morgagni, after analyzing 10 such cases, reported a possible connection between myocardial rupture and damage to the coronary arteries [25].

Indeed, this often fatal complication is based on transmural infarction resulting from complete occlusion of the coronary artery.

Ruptures of the LV free wall usually occur within the first week after the development of MI, and 94% of them occur within the first 16 hours [26]. This associated with the fact that in the first hours of a heart attack, reactive myocardial edema increases and, as a consequence, the strength of the affected LV wall is critically reduced [27]. The increasing intracardiac pressure on the necrotic myocardium is the main hemodynamic rupture factor, with hyperkinesis of the intact myocardium at the border with the infarcted area playing a special role [28–30].

Morphologically, two main types of rupture of the LV free wall are distinguished: an acute form, which is fatal in most cases, and a subacute form, when a blood clot closes the defect and leads to the formation of a pseudoaneurysm [31, 32]. The subacute course is characterized by repeated intermittent bleeding, which occurs in one third of cases [33, 34].

Despite the high mortality rate, surgical intervention remains the only effective method of treating the rupture of the LV free wall; however, both the optimal time and the method of choosing a correction are still being discussed [35, 36].

Surgical treatment of myocardial rupture is associated with great technical difficulties, especially during the period of maximum myomalacia (2-10 days after the development of a heart attack). Since the zone of rupture is surrounded by the infarcted myocardium, the main problem when suturing the myocardium on pads is the gradual eruption of sutures and recurrent bleeding. Therefore, today, in addition to simple suturing of the defect, various methods of treating patients with external rupture of the heart wall are offered: excision of infarcted area with and without synthetic patch repair, the use of a wide range of synthetic and biological adhesives [37, 38], conservative treatment (in patients with the formation of pseudoaneurysms and rapid stabilization of hemodynamics), including the use of extracorporeal membrane oxygenation [39] followed by surgical treatment.

Analyzing the presented clinical case, it should be noted that the patient initially had an increased risk of myocardial rupture: age, episode of MI in history, no history of angina pectoris, isolated coronary artery disease, localitionof infarction (transmural infarction of high lateral parts of the LV). However, the lack of clear diagnostic criteria for this complication did not allow timely suspicion and detection of a LV wall defect. Only upon re-hospitalization due to loss of consciousness was a rupture of the LV free

wall diagnosed, and the patient was transferred to the cardiac surgery department. The patient had a subacute course of rupture of the LV free wall with the formation of a pseudoaneurysm that limited the volume of pericardial bleeding, which led to the relative stability of hemodynamics in the acute period. During this time, the restored blood flow through the PNA promoted reparative processes in the infarction zone, and the volume of scar tissue necessary for successful surgical correction was formed in the area of the rupture, which made it possible to successfully perform suture plasty of the defect.

CONCLUSION

1. For the timely diagnosis of myocardial rupture as a result of transmural infarction, a high level of alertness and careful monitoring of clinical manifestations and instrumental data in patients are required.

2. Urgent transthoracic echocardiography is the gold standard for diagnosing left ventricular free wall rupture.

3. The presence of a rupture of the free wall of the left ventricle with the development of hemotamponade is an indication for emergency surgical intervention.

4. Simple suturing of the defect in the subacute course of rupture of the left ventricle can be successfully performed in case of a stable course of the disease.

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