THE CASE OF CARDIAC RUPTURE AND THE DEVELOPMENT OF A FALSE LEFT VENTRICULAR ANEURYSM IN A PATIENT WITH MYOCARDIAL INFARCTION

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The article presents the clinical observation of a rare complication of an acute myocardial infarction – a rupture of the left ventricle wall with the development of a false aneurysm in an 84-year-old patient who suffered some diseases that had a myocardial infarction, Bypass surgery for multiple coronary stenosis with severe structural and functional heart disease (calcification, combined aortic defect, relative valve failure, tiny fraction of left ventricular discharge, pulmonary hypertension). The cause of the recurrent heart attack that caused the rupture was the occlusion of one of the left coronary arteries. High diagnostic efficiency of EchoCG, contrast of MSCT and non-specific electrocardiographic pattern of complication was demonstrated.

Keywords: myocardial infarction, cardiac rupture, false aneurysm

Of all the complications of myocardial infarction with the height of the ST segment, rupture of the free ventricular wall ranks second among the deaths after cardiogenic shock [1]. Rupture is more common in patients with posterolateral transmural infarction due to occlusion of the circumflex or anterior descending artery [2]. In 98% of cases, the left ventricle of the heart is affected, and only in 2% – the right, with the front and back walls leading LV in the number of site ruptures (83%). In addition to external breaks, internal breaks (septa, papillary muscles, valves) [3, 4], external breaks are ten times more common [5]. Three-quarters of patients have breakdowns within the first three days, and most have studies within the first two weeks of the onset of acute myocardial infarction, and in almost all cases, the outcome is fatal. The main risk factors and predictors of post-natal myocardial rupture are female, age over 75 years, first heart attack, and use of thrombolytic drugs in treatment – especially in periods exceeding 12 hours from the disease onset [6]. The risk of myocardial rupture is significantly increased in cases of comorbid pathology [7].

Classically, a leaking heart rupture is usually accompanied by some clinical signs immediately preceding it, such as long-term or recurrent pain syndrome, uncontrolled hypertension followed by a significant reduction of blood pressure to the development of shock, tachysystolic, elevated Q-positive, transmural MI, elevation of the ST-segment to 2 mm or more in several adjacent wires, heart failure, leukocytosis, elevated levels of C-reactive protein. The most common form of heart rupture is the type I – a sudden rupture, a sharp breakthrough in the myocardial tissue (hemorrhagic type of dissection) with rapid, extensive hemorrhage into the pericardium, tamponade of the heart and its arrest in the systole. There are slower burst variants – types II and III: with initial myocardial erosion and the so-called «hidden tear» of small size, through which a small amount of blood enters the pericardial cavity, which can last several weeks or days [8]. These variants are likely to activate the adhesive process in the pericardial cavity, creating an intraabdominal cavity larger or smaller at the point of formation of a future false aneurysm, sometimes called the «curable form of myocardial rupture».

Thus, a false aneurysm of the heart (pseudoeurhythmus, covered perforation) is one of the variants for ventricular wall rupture with blood access to the pericardial cavity and its separation. A narrow isthmus connects the cavity of the LV pseudoeurhythmus with the ventricular cavity. The width of the isthmus is not more than 50% of the maximum diameter of the pseudoeurhythmus. That is, the ratio of the size of the isthmus to the diameter of the aneurysm itself, measured with EchoCG, is less than 0.5 (as opposed to 0.9 or more with a true aneurysm), although there are exceptions to the rule [9]. Blood entering the aneurysm cavity through a defect in the LV wall (isthmus), over time, leads to an increase in the size of the pseudoeurhythmus cavity. A decrease in blood flow rate in the aneurysm cavity causes stagnation of blood formation of thrombotic masses, fraught with the development of thromboembolic complications [10].

Pseudoeurhythmus is partially limited by the pericardial, pericardial adhesions, epicardia, remnants of the ruptured wall of the left ventricle, organized thrombotic masses and does not contain a detachment of the myocardial walls [11, 12]. Pseudoeurhythmus of the ventricular heart prevents the development of the tamponade at the end of complete external myocardial wall rupture. Since one of the walls of the pseudoeurhythmus consists only of the pericardium and thrombotic mass, the risk of rupture is observed in more than 30% of patients [13, 14]. The subacute course of the process at a rupture with or without the formation of a pseudoeurhythmus is observed in about 30% of patients. In this case, the rupture develops slowly, within 5–10 days, which often occurs with thrombosis (occlusion) of the anterior coronary, circumflex ramo of the left coronary artery and determines the predominant formation of false aneurysms in the posterior, posterolateral segments of the LV [9, 15].

In general, pseudoeurhythmus of the left ventricle refers to rare complications of a heart attack, being detected, for example, in only 0.23% of almost 3,000 patients undergoing coronary angiography after MI [16].

In addition to myocardial infarction as the most common cause, the false aneurysm can be the result of heart surgery and interventional procedures, thoracic trauma, infectious damage to any of the three heart membranes, Behçet syndrome, coronary artery spasm, or the myocardial bridge that caused MI [9, 17, 18].

It should be remembered that true postinfarction aneurysms of the heart, occurring in about 5% of cases of MI, unlike false ones, more often have an anterior and/or apical localization, less often lower and especially lateral, and are relatively rarely complicated by rupture. Subepicardial and subendocardial aneurysms associated with myocardial infarction have been described [19, 20].

Clinical case. Patient M., 84 years old, was admitted to the cardiology department of the regional hospital (transferred from the city hospital) on 14.02.2022 with complaints of increased shortness of breath and the periodic occurrence of previously absent chest pains of a pressing nature lasting up to 50 minutes, awakening from sleep, completion with little physical exertion (walking up to 100 meters, climbing to the 1st floor), relieved by nitroglycerin, for episodes of increased blood pressure – up to a maximum of 160/90 mm Hg (adapted to blood pressure 120–130/80 mm Hg), for palpitations, weakness, fatigue. In 2014, the patient suffered an acute myocardial infarction, bypass surgery; coronary angiography revealed a myocardial bridge of the middle third of the RIVA with a maximum systolic narrowing of 30%, stenosis of the trunk of the LCA (70%), RIVA (90%), CA (70%), RCA (99%), DB (70%), PLBRCA (50%). In 2020, he underwent popliteal-popliteal autogenous bypass surgery on the left. He suffers from hypertension, dyslipidemia, aortic atherosclerosis with calcification and lunar half-moon deformation, combined aortic valve defect (moderate insufficiency and stenosis), a relative deficiency of mitral (2–3), and tricuspid (2).

Deterioration on 09.02.2022 – the above complaints appeared. He was hospitalized in the city hospital with an acute Q-positive myocardial infarction of the lower lateral area of the left ventricle, a persistent form of atrial fibrillation (ventricular normosystole) with a sinus rhythm recovery of 12.02.2022. At EchoCG performed the day before, the aortic walls, fibrous rings, and valve flaps are compacted and calcified. LV myocardium of heterogeneous structure. Hypertrophy of the interventricular septum. Reduction of global LV contractility. Hypokinesis of the lower, lower lateral segments of the LV. Enlargement of the cavities of both atria. Dilation of the aortic lumen at the level of the Valsalva sinuses. Significant mitral valve failure, moderate trivial, and pulmonary valve failure is minor hemodynamic. Combined aortic valve defect: mild stenosis and insufficiency. Moderate pulmonary hypertension. Pericardial effusion is not compromised by rupture. On arrival, the general condition is moderately severe. They were lying position with raised headboards, clear consciousness. BMI=27.04 kg/m². The skin and visible mucous membranes are of standard color; no periphe-
ral edemas exist. There is a percussive pulmonary sound above the lungs, vesicular breathing. There is a post-operative scar in the sternum area. The left border of cardiac dullness is shifted to the left by 1–1.5 cm; the tones are muted, rhythmic. Heart rate is 78 beats in 1 minute; BP-right – 140/80 mm Hg, BP-left – 142/82 mm Hg. The abdomen is regular, soft, and painless; the liver is not palpable and is not enlarged on percussion. Physiological functions are not significantly disrupted.

Total blood count: Er. 3.8 × 10^{12}/l, Hb 111 g/l, Ht 31.5 %, Thromb. 254 × 10^9/l, L. 9.1 × 10^9/l, St. 3 %, Segm. 77 %, Lymph. 16 %, M. 3 %, E. 1 %.

General urine analysis: relative density 1025, pH 5.5, protein 1.12 g/l, leukocytes 1–2–2 in the field of vision, erythrocytes altered more than 50 in the field of vision, squamose and transitional epithelium 0–1–2 in the field of vision, mucus (+), uric acid crystals (3+).

Prothrombin index was 100 %, prothrombin according to Quick 99.3 %, prothrombin time 11.4 sec, APTT 24.8 sec, fibrinogen 5.9 g/l. Troponin I 20.4–22.78–3.85 ng/ml (norm 0–0.5 ng/ml).

Total cholesterol 4.06 mmol/l, triglycerides 1.21 mmol/l, HDL 2.79 mmol/l, LDL 0.80 mmol/l, AI 4.07, AlAT 49.6 U/l, AsAT 97.61 U/l, bilirubin total 19.93 mmol/l, bilirubin conj. 7.46 mmol/l, glucose 7.51 mmol/l, potassium 5.50 mmol/l, sodium 143.17 mmol/l, creatinine 121.10 mmol/l, urea 15.45 mmol/l, LDH 582.28 U/l.

Radiography of the thoracic cavity. In lungs without signs of infiltration, the pattern is enhanced by perivascular and peribronchial consolidation. The roots of the lungs are behind the mediastinum shadow; the pleural sinuses are differentiated. The shadow of the heart expands across the aorta fragmentary sclerotized. There is a sternal suture in the projection of the sternum. Signs of pneumosclerosis, widening of the heart boundaries.

Ultrasound of pleural cavities: The right pleural cavity is defined by a liquid with a layer thickness of 50 mm, and the left pleural cavity is characterized by a liquid with a layer thickness of 55 mm.

ECG at admission (14.02.2022). Sinus rhythm. Low voltage of the waves in standard leads. Small q wave (qR) and elevation of ST I, II, III, aVF, V5–V6, reciprocal depression of ST aVR, V1–V3: a picture of the damage (acute phase of infarction) to the myocardium of the lower lateral wall of the left ventricle (Fig. 1).

EchoCG (17.02.2022). Aortic walls, fibrous rings, and valve cusps are consolidated and calcified. LV myocardium has a heterogeneous structure with areas of increased echogenicity. Along the lateral wall of the left ventricle, an accumulation of fluid 50×20 mm with the presence of blood flow is located, communicating with the cavity of the left ventricle through a fistula up to 5 mm, a hemopericardium with a layer thickness of up to 15 mm: a false aneurysm of the left ventricle – hematoma (Fig. 2). Hypokinesis of the myocardium of the lower apical and lateral segments of the left ventricle. Decrease in global contractile and pumping function of the left ventricular myocardium (LVEF according to Teicholtz 39 %). Dilation of all cavities of the heart. Insufficiency of the mitral, tricuspid, and aortic valves, fibrosis, and calcification of the valves of the mitral and aortic valves. Dilation of the aortic lumen at the level of the Valsalva sinuses. Moderate degree of pulmonary hypertension (tSBPPA 40 mm Hg).

CT with a contrast of the aorta and its branches was performed. From the ascending part of the aorta, CABG is determined to the RCA system with a diameter of up to 5 mm at the entrance; it is traced throughout, and the filling is satisfactory. Also, from the ascending aorta, the place of departure of the shunt (presumably to the LCA system) with a diameter of up to 4 mm is determined.
and the shunt is visualized for 7 mm; in subsequent sections, the shunt is not visualized. A mammacoronary shunt to the LCA system is defined and visualized throughout; its filling is satisfactory. In the pericardial cavity, the liquid layer thickness is up to 18 mm.

A pathological connection is determined between the left ventricular cavity and the pericardial cavity up to 4-5 mm in size with extravasation of the contrast agent into the pericardial cavity, with a limited accumulation of up to 43-20-49 mm. Conclusion: Condition after sternotomy: CABG, MCBS (2017), signs of occlusion of one of the CABG shunts. Signs of a pathological fistula between the left ventricle and the pericardial cavity with signs of extravasation of the contrast agent into the pericardial cavity, signs of hydropericardium (hemopericardium?), bilateral hydrothorax.

Cardiovascular consultation: surgical correction of complications of acute MI in a patient with coronary bypass surgery, plastic surgery on the breast is conducted. 

At the hospital, the patient received enoxaparin sodium (enixum), clopidogrel, atorvastatin, cimetidine, acetylsalicylic acid, bisoprolol, dinitrate isosorbide, spironolactone, and torasemide.

Due to the appearance of respiratory syndrome on the third day of hospitalization and the detection of SARS CoV2 patient was transferred to an infectious diseases hospital. Subsequently, he continued treatment on an outpatient basis.

During observation and treatment in the hospital and at the subsequent outpatient stage, complaints of general weakness were noted periodically (mainly during the daytime). Short-term discomort was behind the sternum, a feeling of lack of air. On an ECG performed one month after discharge from the hospital (17.03), a low QRS voltage is determined in standard leads, T neg. I, II, III, aVF, V4–V6, small q I, II, aVF, V4–V6: signs of focal changes in the lower lateral LV wall and the apex area (Fig. 3). The fate of the patient was traced to 27.03.2022 when sudden death occurred, a sectional study was not conducted.

Discussion. This clinical observation characterizes a rare complication of myocardial infarction – rupture of the left ventricle wall, limited locally brazed pericardium, which contributed to «sealing the tear» with the formation of a cavity – a false aneurysm connecting to the left ventricular cavity, which enabled, at least in the initial period of this fatal event, to save the patient’s life.

The clinical picture of the development of pseudoaneurysm in the case of M. did not differ in specificity: there was an increase in heart failure in the form of increased shortness of breath, the resumption of angina attacks, probably due to the occlusion of one of the shunts (to LCA), destabilization of blood pressure, the appearance of arrhythmia, general weakness, increased fatigue. At the same time, it should be remembered that in more than 10 % of cases, patients with a false aneurysm may be asymptomatic [21].

Patients over 80 years of age suffered some non-communicable diseases: hypertension, atherosclerosis destruction of lower limb vessels, MI,

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false aneurysm at the same time (1 month) after the rupture [22]. In both cases, attention was drawn to the absence of a distinct Q-positivity in the ECG, characterizing the lateral wall of the left ventricle with the undoubted transmurality of the suffered infarcts.

**Conclusion.** Considering the absence of specific clinical, electrocardiographic, and laboratory signs of pseudoaneurysm, the EchoCG method for posterior and posterolateral myocardial infarction probably should be used not only for complex diagnosis at the initial stage of the disease but also for monitoring the condition of the myocardium after the active phase of treatment (late stage of inpatient or early outpatient treatment) where there is a high risk of rupture.

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**References**


