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

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СЛУЧАЙ РАЗРЫВА СЕРДЦА И РАЗВИТИЯ ЛОЖНОЙ АНЕВРИЗМЫ ЛЕВОГО ЖЕЛУДОЧКА У БОЛЬНОГО С ИНФАРКТОМ МИОКАРДА

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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

Приведено клиническое наблюдение редкого осложнения острого инфаркта миокарда – разрыва стенки левого желудочка с развитием ложной аневризмы у больного 84 лет, коморбидного по ряду заболеваний, перенесшего инфаркт миокарда, операцию шунтирования по поводу множественных коронарных стенозов, имеющего тяжёлые структурно-функциональные нарушения сердца (кальциноз, сочетанный аортальный порок, относительную недостаточность клапанов, низкую фракцию выброса левого желудочка, легочную гипертензию). Причиной приведшего к разрыву повторного инфаркта послужила окклюзия одного из шунтов левой коронарной артерии. Продемонстрированы высокая диагностическая эффективность ЭхоКГ, контрастного МСКТ, неспецифичность электрокардиографической картины осложнения.

Ключевые слова: инфаркт миокарда, разрыв сердца, ложная аневризма

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APTT – activated partial thromboplastin time
BMI – body mass index
BP – blood pressure
CABG – coronary artery bypass grafting
CT – computed tomography
DB – diagonal branch
ECG – electrocardiogram
EchoCG – echocardiography
HR – the number of heartbeats
LCA – left coronary artery
LV – left ventricle
LVEF – left ventricle ejection fraction
MCBS – mammacoronary bypass surgery
MI – myocardial infarction
MSCT – multispiral computed tomography
PLBRCA – posterolateral branch of the right coronary artery
RCA – right coronary artery
RIVA – right interventricular artery
SBPPA – systolic blood pressure in the pulmonary artery

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 comorbital 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, tachysistol, extensive 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 (pseudoaneurysm, 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 pseudoaneurysm with the ventricular cavity. The width of the isthmus is not more than 50 % of the maximum diameter of the pseudoaneurysm. That is, the ratio of the size of the isthmus of the aneurysm 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 pseudoaneurysm 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].

Pseudoaneurysm 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 endocardial and myocardial walls [11, 12]. Pseudoaneurysm 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 pseudoaneurysm 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 pseudoaneurysm 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 rami 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, pseudoaneurysm 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, Behcet 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 30 minutes, without irradiation 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 separation within normal limits.

On arrival, the general condition is moderately severe. They were lying position with raised headboards, clear consciousness. BMI=27.04 kg/m^2. 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 postoperative 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, LDL 2.79 mmol/l, HDL 0.80 mmol/l, AI 4.07, AlAT 49.6 U/l, AsAT 97.61 U/l, bilirubin total 19.93 mmol/l, bilirubin conug. 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 (SBPPA 40 mm Hg).

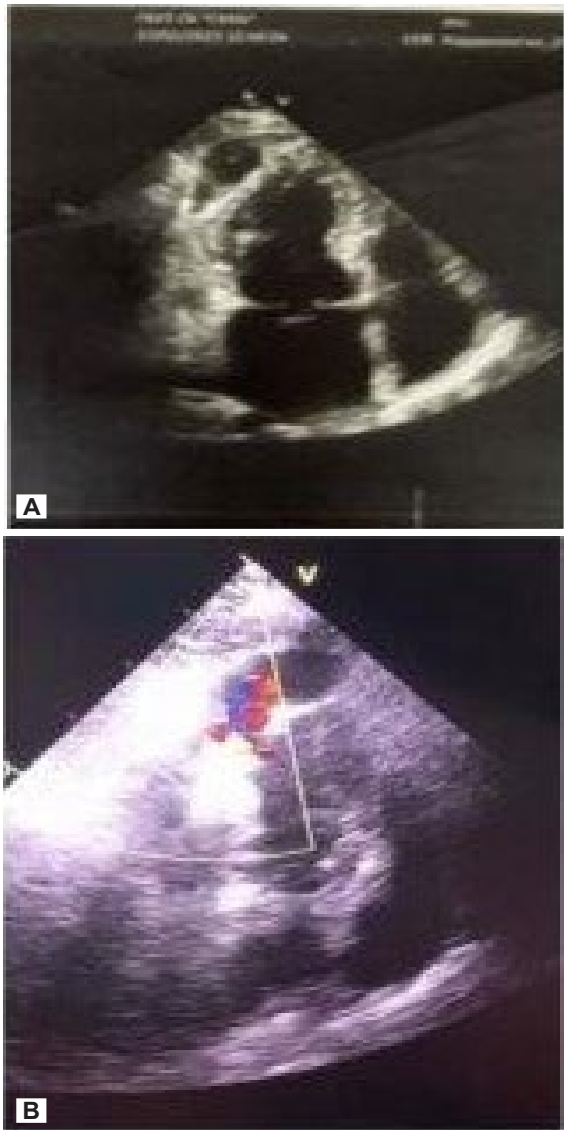

Fig. 2. Echocardiogram: A – false aneurysm of the left ventricle; B – discharge of blood from the ventricle into the pseudoaneurysm cavity through the fistula

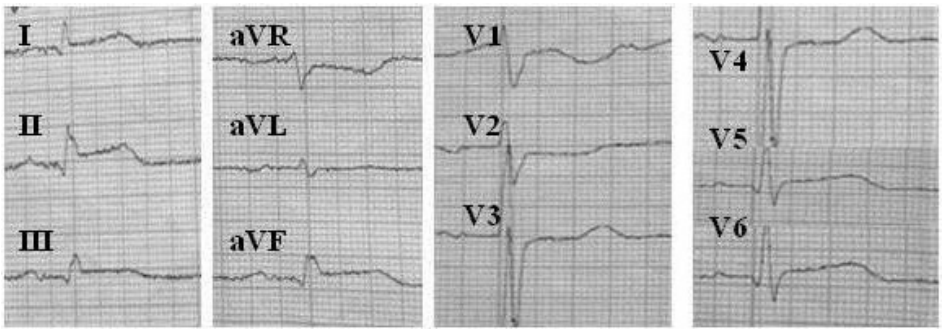

Fig. 1. Patient M. ECG upon admission to the hospital

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 17 mm; in the pleural cavities, 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 associated with a high risk of death. It was decided to continue conservative therapy.

At the hospital, the patient received enoxaparin sodium (enixum), clopidogrel, atorvastatin, omeprazole, acetylsalicylic acid, lisinopril, 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 discomfort 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, coronary stenosis bypass (4 of which were critical)had a myocardial bridge, calcification of mitral rings and mitral valve flaps, calcification and deformation of the semilunar aorta to form a combined defect, a high degree of mitral and tricuspid valve failure, sclerosis and aortic expansion at the Valsalva axillary level, pulmonary hypertension and LVEF reduction (less than 40 %), the presence of a discharge in the pleural cavities and pericardium.

Myocardial infarction was accompanied in the patient by a repeatedly increased (more than 45 times) level of troponin I as one of the indicators of the vastness of the lesion of the heart muscle.

Myocardial rupture and the formation of an aneurysmal sac occurred in the characteristic pseudoaneurysm zone of the myocardium – the posterior and posterolateral segments of the left ventricle. At the same time, in the pericardial cavity along the lateral wall of the LV – at the site of previously detected myocardial hypokinesis, transthoracic echocardiography revealed locally accumulated fluid (blood) 50x20 mm thick, up to 15 mm thick, delimited by walls with dense edges and connecting with the cavity of the left ventricle through a narrow neck (fistula) with a diameter of up to 5 mm (Fig. 2A), which is only 10 % of the maximum diameter of the aneurysm, additionally characterizing it as a false one [12]. Through a myocardial defect in the systole phase, a turbulent flow of blood from the left ventricle into the false aneurysm cavity was observed, recorded during a Doppler study (Fig. 2B). Approximately the same volume of contrast agent in the pericardium and the size of the pathological connection of the pericardium and the left ventricle of the heart were determined by the results of MSCT with contrast.

In the above case, it was possible to document the approximate period of myocardial rupture that occurred in the phase of maximum myomalacia – between 13.02.2022 and 14.02.2022, which corresponded to 4–5 days of exacerbation of the disease (with signs of ST elevation on the ECG, with hypokinesis of the lower, lower lateral LV segments according to the results of EchoCG), and 17.02.2022, when an already formed pseudoaneurysm was diagnosed.

An ECG performed one month after myocardial rupture (Fig. 3) showed «only» the presence of shallow symmetrical T(–) in leads I–III, aVF, V4–V6, and T(+) in aVR. Interestingly, an ECG picture close to the one we obtained was recorded by the authors in another observation of a patient with a similar localization of a

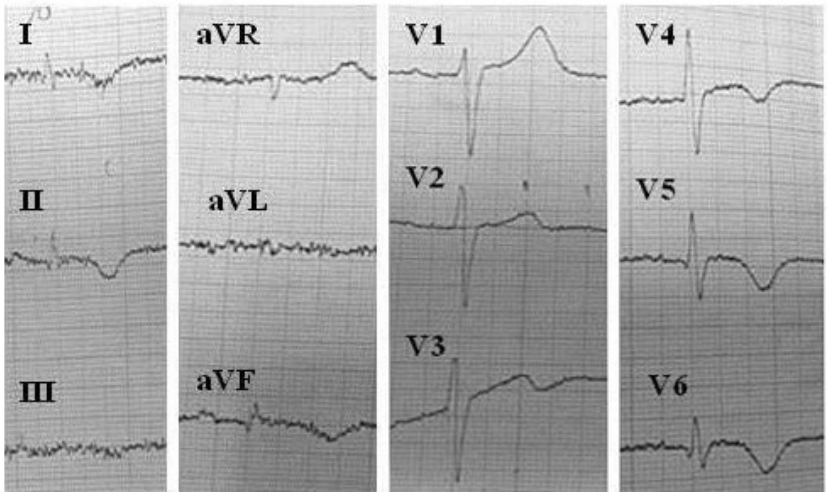

Fig. 3. Patient M. Electrocardiogram 1 month after the formation of a false aneurysm

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 leads on 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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