



Functional mitral regurgitation in a patient with acute myocardial infarction

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Abstract. This article describes a clinical case of functional mitral regurgitation in a 76-year-old female patient. During emergency hospitalization, echocardiography was performed, which revealed impaired coaptation of the mitral valve leaflets and grade 3-4 mitral valve regurgitation. Therefore, a differential diagnosis was made regarding the cause of mitral regurgitation: between mitral valve chord avulsion and functional regurgitation. After angioplasty and stenting of the infarct-related artery, a decrease in the severity of mitral regurgitation was observed on the 7th day of hospitalization, confirming the effectiveness of the treatment and the functional nature of the condition. This clinical case clearly demonstrates the challenges of differential diagnosis of ischemic mitral regurgitation. Increasing physician awareness and awareness of functional mitral regurgitation can assist in choosing treatment strategies and determining indications for surgical intervention.

Keywords: mitral regurgitation, functional mitral regurgitation, myocardial infarction, conservative treatment

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Introduction. Mitral regurgitation (MR) is a common complication of myocardial infarction (MI). In patients undergoing primary percutaneous coronary intervention (PCI), moderate MR occurs in 9.5-11.5%, while severe MR occurs in 0.7-2.8% [1]. It is worth noting that functional or secondary MR (FMR) occurs in 20-25% of post-MI patients and in 50% of patients with heart failure [2].

Unlike degenerative MR, in which regurgitation is caused by structural changes in the valve apparatus, functional mitral regurgitation is a completely different phenomenon. It can be caused by global or local remodeling of the left ventricle (LV) due to ischemic heart disease, cardiomyopathy, and atrial fibrillation [3]. Risk factors for FMR include older age, female gender, previous myocardial infarction, and multivessel coronary artery disease [4,5].

FMR usually occurs 1 week after MI [6,7] and, from a mechanical point of view, arises from an imbalance between increased leaflet fixation forces and decreased leaflet closure forces [8]. MI of segments located beneath the papillary muscles (usually the lateral or inferior wall of the LV) leads to remodeling of this region and temporary papillary muscle dysfunction. As a result, the papillary muscles are displaced upward, posteriorly, and laterally. The papillary muscle forms inextensible chords on both leaflets; its displacement leads to a more apical position of the leaflets and their closure point, as well as to a characteristic deformity of the anterior leaflet known as the "seagull sign" [9].

In clinical practice, the differential diagnosis of the causes of MR and the choice of treatment tactics remain challenging. This article presents a clinical case of successful conservative treatment of MR in a patient with MI.

Clinical Case.

Patient R., 76, was urgently hospitalized at the regional cardiology clinic (Ryazan) at 9:12 PM complaining of severe shortness of breath and a feeling of shortness of breath.

History revealed a history of hypertension for over 20 years, with a maximum blood pressure (BP) of 180/100 mmHg and a usual BP of 130/80 mmHg. She was not taking regular medication. At 6:00 PM, she developed intense chest pain at rest without radiating pain; as the pain subsided, her shortness of breath worsened. She called an ambulance. An electrocardiogram (ECG) revealed atrial fibrillation (first diagnosed) with ST segment elevation along the anterior septal wall of the left ventricle (Fig. 1). The patient was admitted to the intensive care unit (ICU).

Upon examination, her condition was serious; there was no angina pain. The patient was in a forced position with the head elevated. There was no edema. Objectively, the patient had a hypersthenic build (body mass index 34 kg/m²), pale, clammy, and cold skin, a respiratory rate of 30 breaths per minute, and SaO₂ 84%.

Percussion of the lower lungs revealed dullness. Auscultation revealed weakened vesicular breath sounds, and multiple moist rales throughout the lung fields. The heart rhythm was irregular,

with a weakened first heart sound at the apex, a systolic murmur over the mitral valve radiating to the axilla, and a heart rate of 128 beats per minute. Blood pressure was 170/100 mmHg. Stool was normal the day before, with a normal consistency and color. Urination is free and painless.

Myocardial necrosis biomarkers are elevated: troponin I 27,710 ng/L (normal <29 ng/mL), CPK 3,328 U/L, CPK-MB 453 U/L.

An echocardiogram was performed urgently in the intensive care unit. Left atrial dilation (4.2 cm) and akinesia of the anterior, anteroseptal, and lateral walls were revealed, along with globally impaired LV contractility, with an ejection fraction of 23%. Mitral valve (MV) leaflet coaptation was noted, with grade 3-4 MV regurgitation. A partial rupture of the chordae posterioris leaflet (PPCL) cannot be ruled out. Furthermore, LV cavity dilation toward the apex is observed (suspected aneurysm). A hyperechoic lesion measuring 3 x 4 cm, likely a thrombus, was detected at the LV apex (Fig. 2).

Humidified oxygen at 10 L/min via nasal catheters, intravenous infusion of furosemide 60 mg, heparin 1000 U/h, and nitroglycerin 1 mg/h were prescribed, as well as drug therapy: ticagrelor 180 mg once daily, then 90 mg twice daily, acetylsalicylic acid 100 mg daily, atorvastatin 80 mg daily, lisinopril 10 mg daily, and spironolactone 25 mg daily. Beta-blockers and amiodarone were withheld due to symptoms of acute heart failure (pulmonary edema). Coronary angiography (CAG) performed 12 hours after hospital-



Fig.1. ECG recorded by emergency medical services: tachysystolic atrial fibrillation, heart rate 115 beats/min. ST segment elevation in the leads corresponding to the anterior septal wall of the LV, pathological wave Q V2-6

ization revealed occlusion of the anterior interventricular artery (AIVA) in the superior segment; the post-occlusion regions did not enhance the contrast. The right coronary artery had irregular contours. Conductal recanalization, angioplasty, and stenting of the AIVA were performed, along with kissing dilation of the diagonal branch ostium (Fig. 3).

The ECG after coronary angiography also showed atrial fibrillation with a heart rate of 134 bpm (Fig. 4). Due to patient stabilization, amiodarone infusion was administered, and furosemide and heparin were continued. Blood biochemistry revealed dyslipidemia (LDL cholesterol 3.23 mmol/L), hyperuricemia (uric acid 454.1 mmol/L), and an increase in blood glucose to 14 mmol/L. Creatinine was 88 μ mol/L, and glomerular filtration rate was 55 ml/min/1.73 m² according to the CKD-EPI formula.

Due to the clinical presentation of pulmonary edema and the detection of severe MR, a differential diagnosis was made between papillary muscle avulsion and FMR. Dynamic echocardiography analysis revealed a decrease in the severity of MR to grade 2, an increase in LVEF to 32%, and a decrease in the size of the hyperechoic lesion in the LV apex on day 7 of hospitalization. On hospital days 16 and 35, echocardiogram parameters were unchanged: MRI grade 2, LVEF 36%, and no hyperechoic lesion at the LV apex.

While receiving anticoagulant therapy, the patient was diagnosed with minor

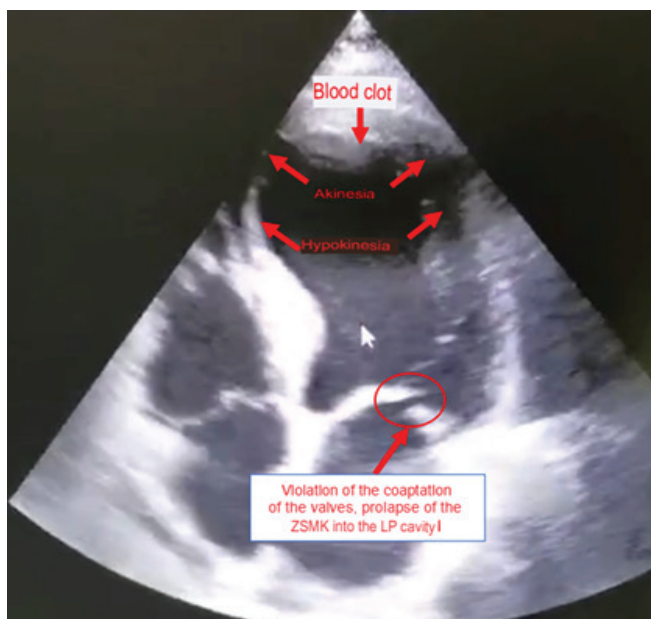


Fig. 2. EchoCG in the intensive care unit, apical four-chamber position: impaired coaptation of the valves of the MC, prolapse of the posterior valve of the MC into the cavity of the left atrium; hyperechoic formation in the area of the apex of the LV (LP – left atrium)

bleeding from an anal fissure on day 25 of hospitalization. On day 30, a nosebleed and mild anemia developed. Due to the nosebleed, anterior packing was performed and oral iron therapy was prescribed (iron III hydroxide polymaltose 100 mg twice daily).

The following diagnosis was made:

Combined underlying disease: coronary artery disease: extensive anterior myocardial infarction with Q waves, acute

stage. Coronary artery atherosclerosis with acute occlusion of the left anterior atrial artery (LAA), irregular contours of the right coronary artery (RCA). Conductor recanalization, percutaneous transluminal coronary angioplasty and stenting of the LAA, kissing dilation of the diagonal branch. Newly reported persistent atrial fibrillation. EHRA IIA.

Background: Stage III hypertension, target blood pressure achieved. Risk 4.

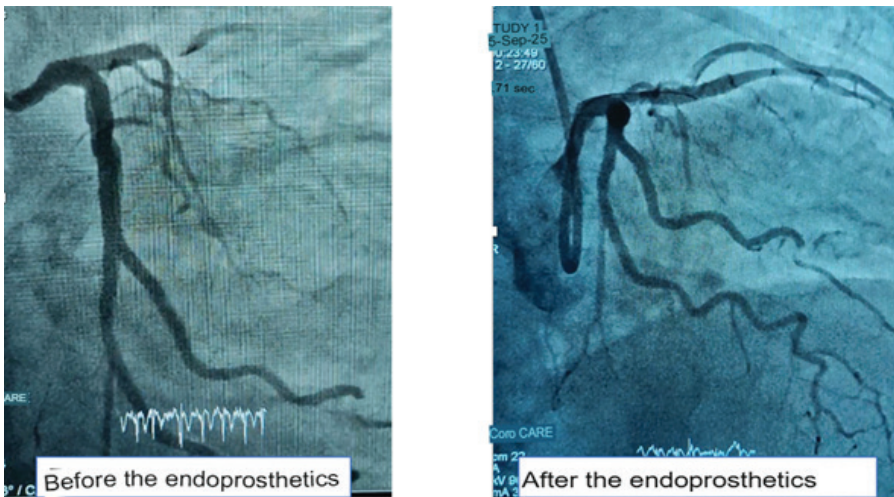


Fig. 3. Coronarangiograms, right oblique caudal projection: permanent residence in the upper segment is occluded, postocclusive sections do not contrast

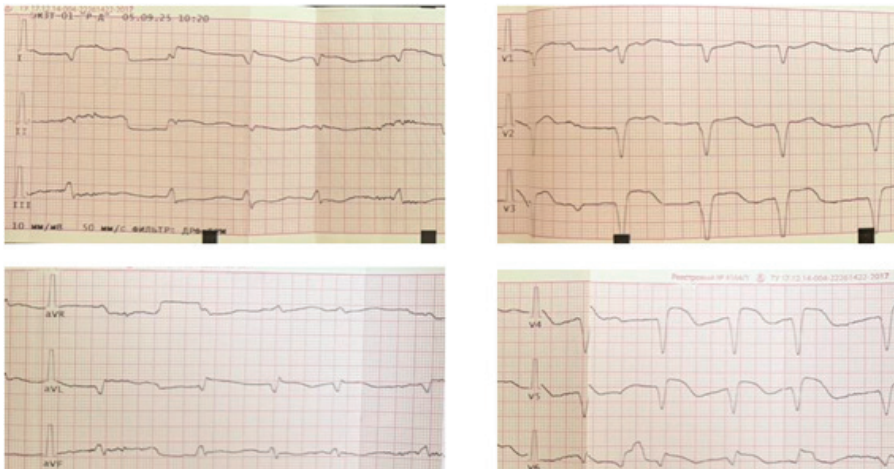


Fig. 4. ECG after CAG: atrial fibrillation with a heart rate of 134 beats/min; dynamics of antero-septal myocardial infarction

Type 2 diabetes mellitus, target HbA1C <7.5%. Stage 1 obesity.

Complications: Acute heart failure. Killip II. Pulmonary edema. SCAI B. Right hydrothorax. Left ventricular septal-apical aneurysm. Left ventricular apical thrombus. Mitral valve insufficiency (MR stage 3-4 → MR stage 2). Significant pulmonary hypertension. CHF stage 1 (2A) with reduced LVEF (36%). NYHA FC 2.

Concomitant symptoms: Past nose-bleed. Left anterior tamponade. Acute anal fissure. Chronic cholecystitis, remission. Mild iron deficiency anemia. Chronic pyelonephritis, remission.

At discharge, the following recommendations were given for taking drug therapy: clopidogrel 75 mg for 12 months, apixaban 5 mg 2 times a day, atorvastatin 80 mg, lisinopril 2.5 mg, metoprolol 12.5 mg / day with a recommendation for further titration of the dose under supervision at the place of residence, spironolactone 50 mg in the morning and at lunch, torasemide 15 mg / day, pantoprazole 20 mg, iron III hydroxide polymaltose 200 mg / day.

Discussion. In the acute phase of myocardial infarction, if FMR is detected during Doppler echocardiography, it is recommended to determine the quantitative parameters of regurgitation and its severity [10]. Given that FMR is largely due to changes in the shape of the LV, correction of the underlying condition that led to remodeling is primarily necessary.

If FMR develops in the setting of acute myocardial ischemia, treatment should be aimed at its correction. The most effective method for eliminating myocardial ischemia is coronary revascularization [11]. In the presented clinical case, timely percutaneous coronary intervention led to significant clinical improvement and a decrease in the severity of MR on the 7th day of hospitalization. It is worth noting that optimal use of angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, mineralocorticoid receptor antagonists, and sodium-glucose cotransporter-2 inhibitors, as well as cardiac resynchronization therapy, can halt LV remodeling and alleviate MR in a significant proportion of patients [12,13].

In contrast, mitral valve surgery for FMR has conflicting results. In two randomized clinical trials involving patients with moderate to severe MR due to coronary artery disease but with preserved LV function (ejection fraction >40%), mitral valve reconstruction using ring annuloplasty or chord-sparing replacement did not have a long-term beneficial effect on clinical outcomes and resulted in a high rate of MR recurrence [14,15].

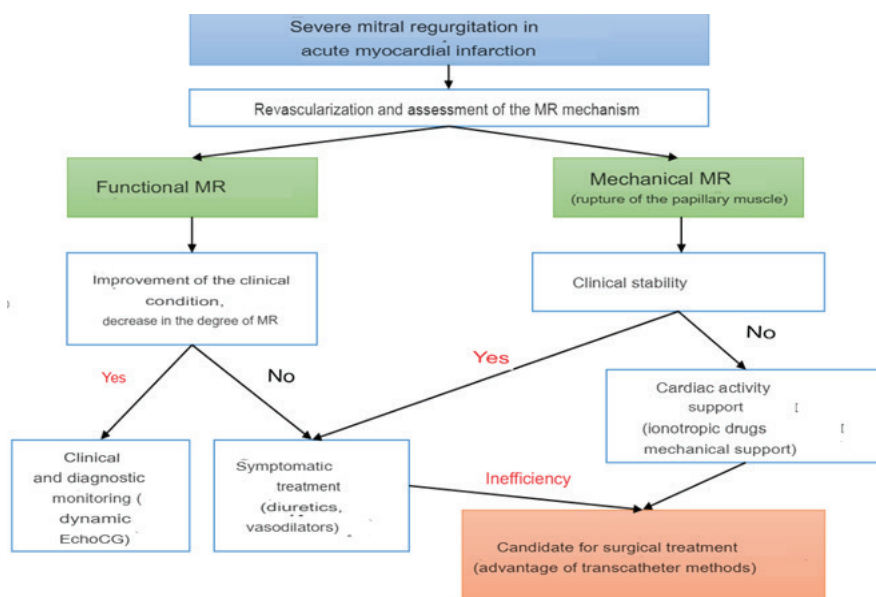


Fig.5. Management tactics for patients with severe ischemic mitral regurgitation (with Estévez-Loureiro R modification [16])

Figure 5 presents the management strategy for patients with severe ischemic MR. It is noteworthy that surgical treatment of FMR is used only when conservative treatment is ineffective.

Conclusions. Acute MR is a common complication of myocardial infarction: approximately 25% of cases are functional. Treatment of FMR should primarily be aimed at restoring the structure and function of the left ventricle by correcting ischemia (coronary revascularization), while also incorporating drug therapy aimed at managing the symptoms of acute heart failure.

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