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Pulmonary embolism with comorbid acute myocardial infarction: a clinical case

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ABSTRACT

The article presents a case of pulmonary embolism with comorbid acute inferior myocardial infarction in the 54-year-old patient who was admitted to the Regional Center for Percutaneous Coronary Interventions. Coronary angiography revealed a multivessel lesion with angiographic signs of instability in the proximal third of the right coronary artery. Pulmonary angiography revealed signs of pulmonary embolism with moderate impairment of pulmonary perfusion.

The described combination is challenging in terms of both diagnosis and subsequent treatment strategy. A feature of this case is the use of a double surgery, consisting of revascularization of the infarct-related artery and fragmentation of thrombotic masses in the pulmonary artery, in combination with thrombolytic therapy.

Key words: acute myocardial infarction, pulmonary embolism, thrombolysis, coronary angiography, pulmonary angiography.

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Тромбоэмболия легочной артерии в сочетании с острым инфарктом миокарда: клинический случай

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РЕЗЮМЕ

Представлен случай сочетания тромбоэмболии легочной артерии и острого нижнего инфаркта миокарда у 54-летнего пациента, который поступил в региональный центр по проведению чрескожных коронарных вмешательств. При выполнении коронароангиографии обнаружено многососудистое поражение с ангиографическими признаками нестабильности в проксимальной трети правой коронарной артерии, при ангиопульмонографии – картина тромбоэмболии легочной артерии с нарушением перфузии легких средней степени тяжести.

Описанное сочетание представляет собой трудную задачу как с точки зрения диагностики, так и последующей лечебной тактики. Особенностью данного случая является применение двойного интервенционного вмешательства, состоящего из реваскуляризации инфаркт-зависимой артерии и фрагментации тромботических масс в легочной артерии в сочетании с тромболитической терапией.

Ключевые слова: острый инфаркт миокарда, тромбоэмболия легочной артерии, тромболизис, ангиография коронарных сосудов, ангиопульмонография.

Конфликт интересов. Авторы декларируют отсутствие явных и потенциальных конфликтов интересов, связанных с публикацией настоящей статьи.

Источник финансирования. Авторы заявляют об отсутствии источника финансирования при проведении исследования.

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INTRODUCTION

Pulmonary embolism (PE), having no clinical specificity, is one of the diseases that require differential diagnosis, including differential diagnosis with acute myocardial infarction (AMI). Treatment of patients with a combination of these diseases is particularly difficult [1]. An example of successful application of the interventional strategy in managing a patient with pulmonary embolism with comorbid AMI is described below.

CLINICAL CASE

A 54-year-old patient was delivered by an ambulance team to the Regional Center for Percutaneous Coronary Interventions with the diagnosis of acute coronary syndrome with ST-segment elevation. He had considered himself sick for 1.5 hours, when severe weakness, shortness of breath, and tightness in the chest with loss of consciousness appeared for the first time in his life.

He had a history of smoking up to one pack of cigarettes a day and did not measure blood pressure. There were no other risk factors for cardiovascular diseases. Consciousness was clear. The skin was of a normal color. Body mass index (BMI) was 26 kg / m^2. There was no edema on the lower extremities. Breathing was adequate, with a rate of 16 breaths per minute. Upon auscultation, vesicular breathing was noted, both lungs were clear, SpO_2 90%. The heart tones were rhythmic and muffled; no heart murmurs were detected. Blood pressure in both arms was 120 / 80 mm Hg, the heart rate was 100 beats per minute (against the background of a dopamine infusion at a dose of 3 mcg / kg / min). The abdomen was soft and painless. The lower edge of the liver was at the border of the costal arch. When examining other organs and systems, no pathological abnormalities were detected.

Laboratory findings: troponin I and creatine phosphokinase-MB (CPK-MB) were negative upon admission to the hospital (upon repeated examination after 12 hours, the upper limit of normal values was exceeded by 10 times), D-dimer upon admission was > 8,000 ng / ml.

The electrocardiogram showed ST segment elevation in aVL of 1 mm, ST segment depression in II, III, and V_{3-6} of up to 2 mm, and right bundle branch block (Fig. 1). Coronary angiography revealed arteriosclerosis, calcification of the coronary arteries; stenosis

of the left main coronary artery before bifurcation of more than 65%; stenosis of the proximal third of the anterior interventricular branch of more than 70%; stenosis of the middle third of the anterior interventricular branch of about 60%; stenosis of the orifice of the diagonal branch of more than 40%, stenosis of the orifice and the proximal third of the intermediate artery of about 50%; stenosis of the proximal third of the right coronary artery (RCA) of more than 85% with angiographic signs of instability; and angiographic signs of mural thrombosis of the middle third of the RCA (Fig. 2).

Stenting of critical stenosis of the proximal third and thrombosis-affected area of the middle third of the RCA was performed with a good angiographic result (Fig. 3).

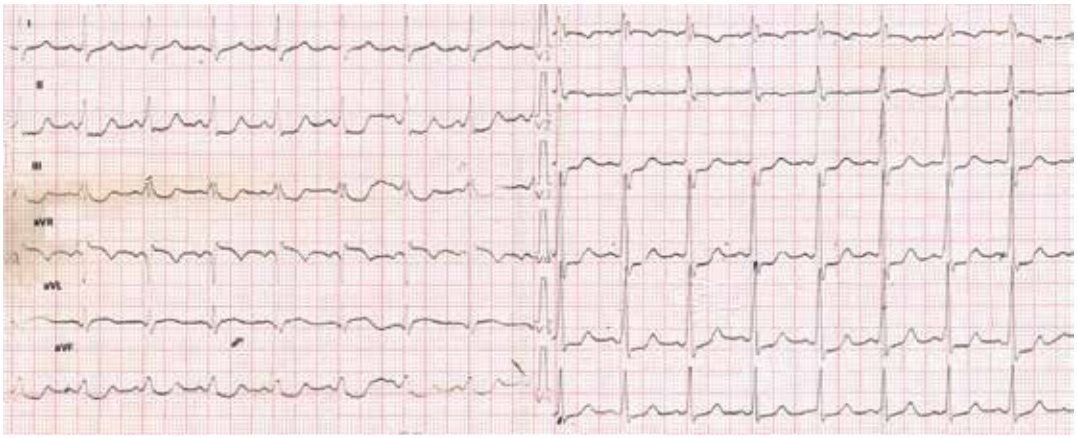

Fig. 1. Electrocardiogram of the patient upon admission: speed – 25 mm / sec, voltage – 10 mm

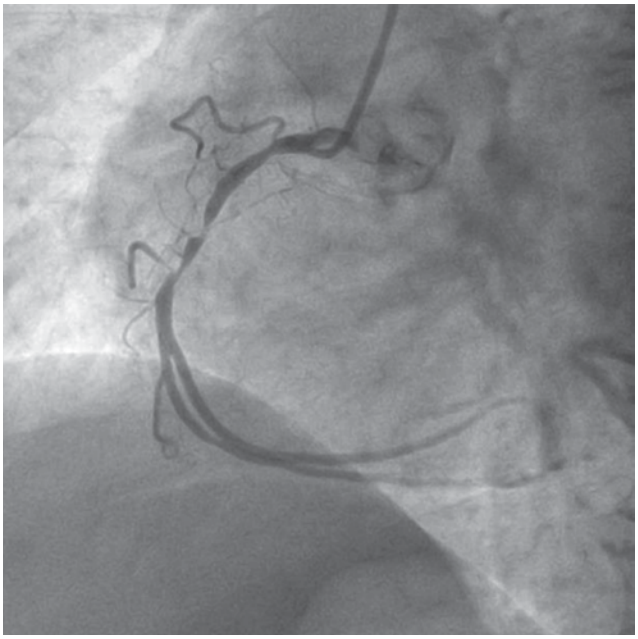

Fig. 2. Image of the RCA obtained during coronary angiography

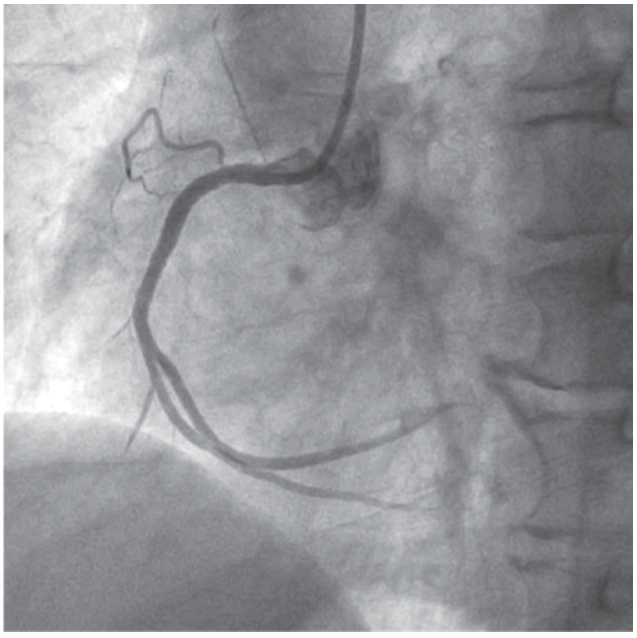

Fig. 3. Image of the RCA after stenting

After stenting, taking into account the features of the clinical presentation and the results of D-dimer determination, it was decided to perform pulmonary angiography, which revealed signs of pulmonary embolism (mainly on the right) of moderate severity (Miller index 19) (Fig. 4). Mechanical fragmentation of the thrombus with selective thrombolysis (alteplase – 15 mg in 15 minutes) and subsequent systemic administration of a thrombolytic (alteplase – 85 mg in 1.5 hours) were performed (Fig. 5).

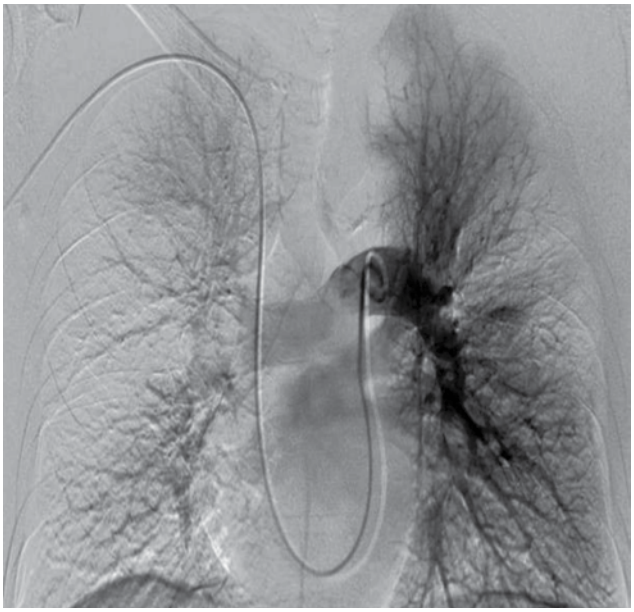

Fig.4. Pulmonary angiography image

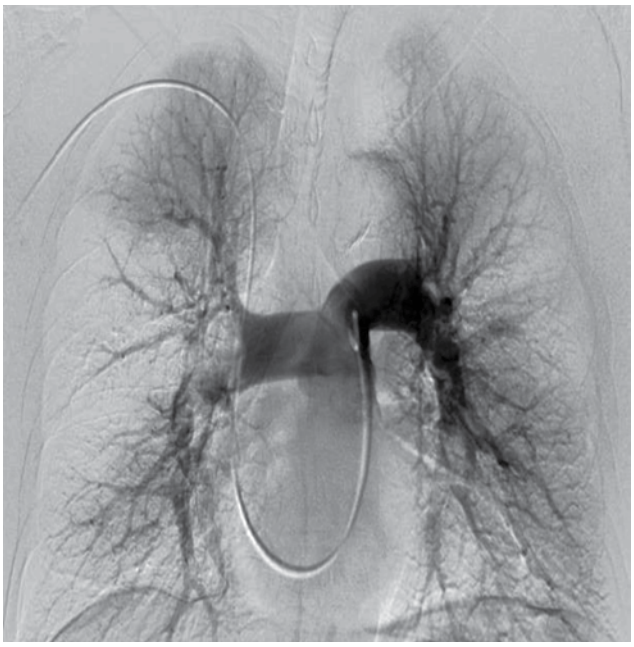

Fig. 5. Pulmonary arteries after fragmentation of thrombotic masses and selective thrombolysis

In echocardiography (after percutaneous coronary intervention (PCI)), dilation of the right ventricle (RV) and left ventricle (LV) cavities, aortic dilation at the level of the sinus of Valsalva, asymmetric hypertrophy of the LV myocardium (posterior wall – 0.9–1.3 cm, interventricular septum – 1.2–1.5 cm), hypokinesis of the posterior LV wall, and LV ejection fraction of 47% were detected.

Ultrasound examination of the lower extremities (after PCI) revealed thrombotic masses with signs of recanalization of up to 20% in one of the posterior tibial veins on the right side; mural thrombotic masses with signs of recanalization of up to 50% in the deep femoral vein; and thrombotic masses with signs of recanalization of up to 50% in the sural veins on the left side.

The postoperative period was uneventful. On the 14th day, the patient was discharged for further treatment in an outpatient setting with a recommendation to continue double antiplatelet therapy in combination with dabigatran under the supervision of a cardiologist and vascular surgeon.

DISCUSSION

Three types of a combination of AMI and PE are described in the literature: PE as a complication of AMI; AMI resulting from paradoxical embolism with a functioning foramen ovale, combined with PE; and AMI as a complication of PE [2–4]. We believe that in the case under discussion, the third variant is most likely to develop against the background of critical stenosis of RCA, which is consistent with the 4th Universal definition of myocardial infarction [5].

The following reasons for ST segment elevation in PE are suggested: 1) true myocardial ischemia (occlusion, embolism, atherosclerotic plaque); 2) insufficiency of coronary artery blood flow due to an acute increase in the right ventricular afterload [6]; 3) transmural ischemia of the right ventricle due to hypotension, hypoxemia, pulmonary arterial hypertension, and hypercatecholaminemia [7]; 4) compression of the coronary arteries by a dilated pulmonary artery due to developmental abnormalities [8, 9].

In addition to the difficulty in diagnosing such a combination of diseases, there are also difficulties in determining the most optimal treatment strategy. In most of the described cases, systemic thrombolysis was used, which was performed at various time intervals, due to delayed diagnosis of PE by contrast enhanced computed tomography (CT). There is a description of a clinical case using a double intervention [10].

According to the latest European recommendations for the diagnosis and management of patients with acute pulmonary embolism, pulmonary angiography is indicated for elective invasive percutaneous treatment of PE [11]. In our case, taking into account the patient's being in the catheter laboratory, in order to minimize time loss associated with patient's

transportation, it was decided to perform an emergency pulmonary angiography with subsequent intervention.

CONCLUSION

The given clinical example demonstrates the complexity of diagnosing and treating a patient with a combination of life-threatening conditions. In the presence of initial hypotension (shock) with an impossibility to perform immediate CT angiography, bedside echocardiography is a method for verifying the diagnosis of high-risk PE, the results of which will allow to differentiate PE and AMI in the shortest possible time [11].

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