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RIGHT VENTRICULAR MYOCARDIAL INFARCTION IN A PATIENT WITH CHRONIC THROMBOEMBOLIC PULMONARY HYPERTENSION AGAINST THE BACKGROUND OF SMALL CORONARY ARTERIES

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SUMMARY

Most often acute myocardial infarction of the right ventricle is diagnosed in patients with acute left ventricular lesion and is associated with an atherosclerotic process in the coronary arteries. Isolated acute myocardial infarction of the right ventricle is rarely diagnosed and is often detected only during autopsy. Previously it was believed that in the absence of a significant lesion of the coronary arteries, an acute myocardial infarction of the right ventricle is associated with hypertrophy of the right ventricular myocardium. The results of a study in which acute myocardial infarction of the right ventricle were found in patients who died due to acute massive pulmonary embolism and who did not have right ventricular hypertrophy appeared later. There are many questions regarding the tactics of therapy for this complication, especially in patients with pulmonary hypertension who are on treatment with pulmonary arterial vasodilators.

The article describes a rare clinical case of postmortem detection of myocardial infarction of the right ventricle against the background of small coronary arteries in a 41-year-old patient with a diagnosis of chronic thromboembolic pulmonary hypertension, the difficulties of intravital diagnosis and the restriction of drug therapy.

Keywords: *acute myocardial infarction of right ventricle, chronic thromboembolic pulmonary hypertension, myocardium ischemia in the absence of a coronary atherosclerosis.*

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Right ventricular acute myocardial infarction (MI) is usually associated with the presence of coronary atherosclerosis and almost always accompanies the acute MI of the left ventricle (LV) of the heart. Involvement of the right ventricle (RV) depends on the localization of acute MI: in case of lesion of the lower wall it is recorded in 24-50% of cases; in rare cases, it develops with lesion of the anterior wall of the LV [1]. The relatively small frequency of the RV acute MI can be explained by several factors: lower oxygen consumption due to less muscle mass and workload; high velocity of blood flow during diastole and systole; a more extensive collateral network, including from the left coronary artery; diffusion of oxygen from the intracavitary blood through the thin wall of the RV and through the smallest veins going from the endocardium directly to the coronary arteries of large and medium order [1].

In rare cases, isolated MI of the RV is described in patients with acute pulmonary artery thromboembolism and is diagnosed, as a

rule, posthumously [2]. The clinical case of a 41-year-old female patient who was observed at the National Medical Research Centre of cardiology of Ministry of Health for 11 months against a background of chronic thromboembolic pulmonary hypertension due to recurrent thromboembolism into the system will allow to analyze this rare complication.

For the first time, the patient was admitted to the clinic complaining of dyspnea while doing minimal physical activity (walking for 10 meters) and while speaking, of pain in the left side of the chest, which is unrelated to physical activity and which gets better with body position changes or in 5-10 minutes after taking valocardin, of fast heart beat and recurrent swelling of the lower extremities.

From the anamnesis it is known, that for the first time the patient started to notice dyspnea in the summer of 2012 while walking the stairs (she was forced to stop on the 3rd floor when climbing the stairs). Then the dyspnea was insignificant, and the patient did not apply for the medical help. The patient experienced a sharp deterioration in June 2013, when dyspnea and swelling of legs appeared in 2 days after the intestinal infection while walking at a distance of about 50 meters. For the first time she consulted the doctor on December 16, 2013, when a severe swelling of the lower extremities appeared after the acute respiratory viral infection and also dyspnea began to disturb at rest. On December 25, 2013 the patient was hospitalized at the place of residence. According to medical examination, the level of D-dimer increased to 1.8 μg/ml (<0.5); electrocardiography (ECG) – deviation of the electrical axis to the right, severe hypertrophy and congestion of the right heart, incomplete blockade of the right bundle branch leg of Gies; echocardiography (EchoCG) – the right heart is enlarged, pulmonary artery systolic pressure (PASP) is 120 mm Hg, insufficiency of tricuspid valve of 3-4 degrees; multispiral computed tomography of the lungs (MSCT) – parietal thrombotic masses in the projection of the trunk, left and right pulmonary arteries. A diagnosis was made: recurrent pulmonary artery thromboembolism, severe post thromboembolic pulmonary hypertension. Patient underwent thrombolytic therapy (actinolysis 100 mg), anticoagulant therapy (enoxaparin, warfarin), diuretic therapy, after which her condition stabilized.15 days after the treatment, pulmonary angiography was again performed, where positive dynamics was noted: contrast defects in the pulmonary artery trunk and in left pulmonary artery were not visualized. The volume of thrombotic masses in the right pulmonary artery significantly decreased. After discharge, dyspnea gradually increased and tolerance to physical exercises decreased, even so the patient complied with the regime and was taking prescribed medicine in time.

In August 2012, the patient had a syncope for the first time. From 2012 to 2014 there were 6 episodes of syncope in total, so the brain MRI was performed, no pathology was found.

Since February 2014, after repeated hospitalization at the place of residence, the patient received 2.5 mg of warfarin without achieving the target level of the international normalized ratio (INR), bisoprolol 2.5 mg, torasemide 2.5 mg, spironolactone 25 mg per day. Patient was admitted to the National Medical Research Centre of cardiology of Ministry of Health in severe condition. Consciousness is clear. Attention was paid to the pale color of the skin, the presence of blush on the cheeks and acrocyanosis. Mucous membranes of usual color, humidity. Fauces is not hyperemic. The subcutaneous fat layer is developed moderately. The body mass index is 28 kg / m^2. There were no violations of 12-pairs of cranial nerves. Lymph nodes are not enlarged. Bones, joints and muscles, mammary glands, thyroid gland are without any characteristic features. The pastosity of the legs was noted. Respiratory rate – 20 breaths per minute. In the act of breathing, both halves of the thorax were involved evenly. With auscultation of the lungs, breathing was carried out in all departments with a hard hue, there is no wheezing. At auscultation of heart tones are muffled and an accent of the second tone over the pulmonary artery was heard, there is a gentle systolic murmur above the projection of the tricuspid valve. There were no sounds over the main arteries. Heart rhythm is regular, heart rate is 80 per minute, arterial pressure on the right arm is 90/60 mm Hg, on the left – 95/70 mm Hg. Pulsation of peripheral vessels is good, the condition of the veins is satisfactory. The abdomen is soft, painless in all parts, the liver is at the edge of the costal arch, the spleen is not enlarged. The kidneys are not palpable, the symptom of "effleurage" is negative on both sides, there is no dysuria.

At ECG recording, a sinus tachycardia with a heart rate of 105 beats per minute was registered, deviation of the electric axis of the heart to the right, change in the p-pulmonale type atrial component, signs of myocardial changes due to right ventricular hypertrophy, and violation of intraventricular conduction (Figure 1).

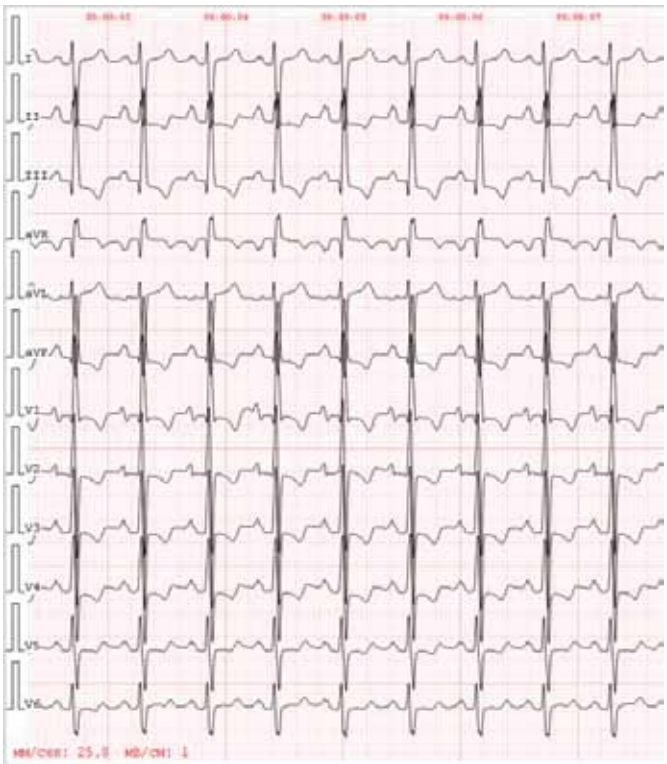

Figure 1. Electrocardiography of the patient during the first hospitalization

Daily monitoring of the ECG according to Holter: the main rhythm is sinus, the average heart rate is 80 beats per minute, the maximum is 154 beats per minute, and the minimum is 54 beats per minute. Ischemic dynamics of the ST segment was not detected, 3 ventricular ecstasyoles were registered, supraventricular extrasystoles and pauses were not detected.

The results of echocardiography are presented in Table 1. Attention is drawn to the dilatation of the right heart with signs of right ventricular overload pressure, myocardial hypertrophy of the right ventricle, dilatation of the pulmonary arteries and its branches, high pulmonary hypertension, relative deficiency of tricuspid valve of II degree, pulmonary artery valve of I-II degree, widening of inferior hollow vein, signs of compression of left ventricle due to compression of the enlarged right ventricle.

Table 1. Results of EchoCG of patient G. in dynamics

	Norm	First hospitalization	Recurrent hospitalization
Aorta, cm	2,9-4,5	3,4	3,0
LA, cm	2,0-4,0	3,1	3,6
LV EDD, cm	4,0-5,5	3,8	2,6
IVST, cm	0,7-1,1	1,0	1,0
LV PWT, cm	0,7-1,1	1,0	1,0
S RA, cm^2	<18	32	32
AP RVD, cm	≤2,9	4,3	4,8
RVAWT, cm	≤0,5	0,9	1,1
PASP, mm Hg	<30	105	105
TR, degree	0-I	II	III
PR, dergee	0-I	I-II	I-II
PAT, cm	≤2,6	4,1	4,8
RPA / LPA, см	≤1,5	3,5 / 3,0	4,3 / 3,0
IVC, cm	≤2,2	2,4 / 1,0 on inspiration	2,6 / 1,6 on inspiration

According to chest radiography, focal and infiltrative changes were not detected, the pulmonary pattern is diffusely poor due to the vascular component; extended segmental and subsegmental arteries of high intensity are visualized. The heart is widened in diameter, the pulmonary artery trunk protrudes along the left contour. The quantitative signs of pulmonary arterial hypertension (PAH) are visualized (Table 2). Pleural sinuses are free.

Table 2. Quantitative signs of pulmonary hypertension based on chest radiography in patient Z. in dynamics

Indicators	Norm	May 2014	February 2015
Right root, mm	≤15	18	18
left root, mm	-	40	44
Coefficient of Moore, %	≤30	38	40
Coefficient of Luthi, %	≤33	39	39
CTI, %	≤50	55	58

When the combined single-photon emission computed tomography and computed tomography of the lungs with intravenous injection of the radiopharmaceutical 99mTc-MAA were performed, the area of heterogeneous decrease in accumulation of radiopharmaceutical drug in the region S4 of the middle lobe of the right lung with a sufficiently sharp contour was recorded. For the rest parts of the lungs, the distribution of radiopharmaceutical drug is even. When evaluating computed tomography (SPECT/CT-tomography) lung intravenous radiopharmaceutical 99mTc-MAA a site of a non-uniform decrease in accumulation of RFP in the region S4 of the middle lobe of the right lung with a sufficiently sharp contour is recorded. When evaluating CT research, focal and infiltrative changes in lung tissue are not determined. Cardiothoracic index >50%. Fluids in the pleural cavities are not detected. The scintigraphic picture can show embolism in the basin of the right branch of the pulmonary artery.

The patient underwent multispiral computed tomography with contrast enhancement (MSCT-angiopulmonography): the expansion of the pulmonary artery was determined to 4.2 cm, the right pulmonary artery (RPA) – 3.6 cm, the left pulmonary artery (LPA) – 3.4 cm. On the lower contour of the right pulmonary artery, a thrombus with a thickness of up to 1.2 cm was visualized, stenosing the artery lumen to 35%, distal to the level of subsegmental branches of filling defects was not revealed. The trunk of the pulmonary artery and the LPA to the level of the subsegmental branches were contrasted without stenotic changes. The right parts of the heart are greatly expanded. Lungs are without fresh focal and infiltrative changes. The pulmonary pattern is diffusely strengthened. Fibrotic changes in the middle lobe of the right lung. Lumens of trachea and large bronchia are free. The roots of the lungs are structural. Axillary, intrathoracic lymph nodes are not enlarged. Bone destruction at this examination was not detected. Conclusion: a thrombus along the lower wall of the right pulmonary artery. Signs of pulmonary hypertension.

When conducting a catheterization of the right heart, the SPAP was 121 mm Hg, the average – 69 mm Hg, the average pressure in the right atrium – 3 mm Hg, pulmonary artery wedge pressure – 6 mm Hg, saturation of capillary blood – 94%, blood saturation of the pulmonary artery 50%, cardiac index 1.3 l / min x m^2, pulmonary vascular resistance – 2260 dyn x s / cm^5; acute pharmacological test – positive (a decrease in the average pressure of the left artery from 69 to 32 mm Hg, cardiac output increased from 2.3 to 3.4 l / min). When performing selective pulmonary angiography, the trunk and main branches of the pulmonary artery were enlarged; significant violations of perfusion were not detected.

According to ultrasound, the size of the liver within normal values, its contours are clear, even, an increased echogenic, structure - diffusely homogeneous, fine-grained, vascular pattern without features. Additional structures in the parenchyma have not been identified. The portal vein is not enlarged, the inferior vena cava is at the upper border of the norm (23 mm, norm – up to 22 mm), the hepatic veins are not dilated. Extrahepatic bile ducts, holedoch are not dilated. Additional structures in the lumen are not determined. Gallbladder with an inflection in the neck, of normal size, the walls are not thickened, moderately compacted, the contents of the cavity are homogeneous. Ultrasound dopplerography of veins of lower extremities – thrombotic masses were not detected.

According computer spirometry lung capacity and performance airway within the age norm. (VC >0%, FEV1 >70%).

In blood tests, attention was attracted to a slight increase in the level of total bilirubin up to 41.3 μM/L (norm – 3-29 μMol / L), uric acid up to 619 μM / L (norm – 142.8-339.2 μMol/L), C-reactive protein up to 1.06 mg / dl (norm – up to 0.5 mg / dL), hemoglobin up to 16.2 g / dl (12-16 g/dL), D-dimer up to 2.58 μg/ml (norm up to 0.5 μg/ml). The level of international normalized ratio (INR) at admission – 1.49, which indicates the lack of anticoagulation therapy. When carrying out a genetic blood test to identify hereditary thrombophilia, heterozygous polymorphism in the gene of the inhibitor / activator of plasminogen of type 1 was detected.

Based on medical examination, the diagnosis was confirmed: chronic thromboembolic pulmonary hypertension (CTEPH). Functional class III-IV (WHO). Recurrent thromboembolism in the pulmonary artery system. Pulmonary heart: insufficiency of tricuspid valve of II degree, pulmonary valve of I-II degree. Chronic heart failure – II A stage. Chronic hemorrhoids. Chronic adnexitis.

Therapy was conducted with enoxaparin 160 mg, torasemide 5-7.5 mg, spironolactone 50 mg per day. Bisoprolol was replaced by diltiazem with a dose up to 180 mg per day, pathogenetic therapy with sildenafil 60 mg per day was started with good tolerability.

The patient was admitted to the hospital again in 11 months after the first hospitalization. When questioned, it was found that soon after discharge she lowered the dose of sildenafil to 12.5 mg per day due to the complications of drug provision and the appearance of persistent hypotension (systolic blood pressure 80-90 mm Hg). Since discharge, the patient had three episodes of deterioration, which were accompanied by increased dyspnoea and edema, but she did not seek medical help.

At admission, she complained about dyspnoea at rest and with minimal physical activity (walking for a distance of 10 meters), intense pains of a stabbing character in the left half of the chest with irradiation into the left arm, arising without connection with physical activity, passing with a change in the position of the body or in 5-10 minutes after taking Valokardin, episodes of cardiopalmus, self-stopping, periodic swelling of the lower extremities.

Patient's condition is heavy, the skin is pale, pronounced blush on the cheeks, the pastosity of the shins. At auscultation of the lungs, breathing was carried out in all departments, with a hard tinge, without wheezing, respiratory rate – 20 breaths per minute. Percutally the relative border of the heart is widened to the right. At auscultation heart tones are rhythmic, the accent of the second tone over the projection of the pulmonary artery is heard, and also systolic sound above the projection of the tricuspid valve and diastolic noise along the left edge of the sternum are heard. Heart rate – 80 per minute, blood pressure 80/60 mm Hg. At palpation the abdomen was soft, painless. The liver protruded by 3 cm from under the edge of the costal arch, slightly painful at palpation. From the side of the urinary and nervous systems without particular features.

According to the electrocardiography, at admission, sinus rhythm with a heart rate of 65 per min., deviation of the electric axis of the heart to the right, atrial component change as p-pulmonale type, signs of myocardial hypertrophy of the right ventricle. When compared with ECG data in the dynamics there were no significant changes (Fig. 2). During hospitalization, the ECG was performed again against a background of pains in the chest, but the configuration of the complex remained unchanged.

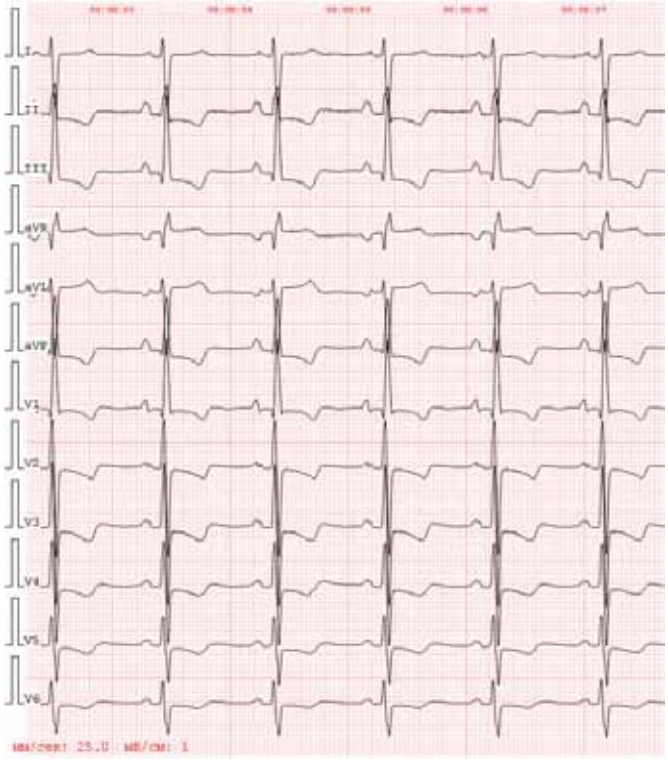

Figure 2. Electrocardiography of patient G. during second hospitalization

According to chest radiography, focal and infiltrative changes are not detected, cells of focal and infiltrative changes are not detected, the pulmonary pattern is diffusively poor on the left and on the periphery to the right due to the vascular component, in the projection of the left root, extended and "chopped" segmental arteries are defined. In dynamics there is an increase in the size of the left root, the Moor coefficient and cardio-thoracic index (Table 2).

EchoCG in dynamics revealed an increase in the cavity and thickness of the anterior wall of the right ventricle, the pulmonary artery trunk and its right branch, and a decrease in the left ventricular end-diastolic dimension (Table 1).

According to ultrasound, signs of right ventricular heart failure as enlargement of the liver and stagnation in the liver, dilation of lower hollow veins and hepatic veins. MSCT of the chest with contrast enhancement was performed: the expansion of the pulmonary artery up to 5.3 cm, right pulmonary artery 4.4 cm, left pulmonary artery 3.9 cm. On the lower contour of the right pulmonary artery, a thrombus with a thickness of up to 1.2 cm is determined, stenosing artery lumen up to 35%, the thrombus extends to the mouth of the mid – lobe branch, obturating the lumen all over. The trunk of the pulmonary artery and the left pulmonary artery up to the level of the subsegmental branches are contrasted without stenotic changes. The right parts of the heart are markedly dilated. In the lungs, the pattern is diffusely enhanced, a "mosaic" perfusion of the lungs is determined. Fibrotic changes in the middle lobe of the right lung. The lumen of the trachea and large bronchi are free. The roots of the lungs are structural. The enlarged axillary and intrathoracic lymph nodes are not determined.

Thus, the patient was admitted to the hospital in an extremely severe condition due to the presence of high pulmonary hypertension because of recurrent pulmonary thromboembolism, as well as decompensation of chronic heart failure. Given persistent hypotension, the correction of previously selected therapy was made: diltiazem replaced with ivabradine 15 mg /day, warfarin with enoxapyrine 140 mg/day; given the persistent hypotension sildenafil was prescribed at a dose of 15 mg per day, with a further increase in the dose under the control of blood pressure. The presence of heart failure, the need for parenteral loop diuretics required the installation of a central catheter to infuse pressor amines, against which there was a temporary stabilization of blood pressure at a level of 90-110/60-80 mm Hg. The diuresis was positive (+ 300-500 ml). Inhalations of iloprost are added to the therapy – 20 μg / day.

Given the severity of the condition, persistent hypotension, negative diuresis, the inability to continue therapy with sildenafil and iloprost in prescribed doses, the patient was transferred to the Intensive Care Team, a central catheter was installed, infusion of dobutamine was started at a rate of 10 μg/kg/min, against which there was a relative increase in blood pressure to 95-105/62-70 mm Hg. On the bedside monitor, a sinus rhythm was recorded with signs of an overload of the right heart (Figure 3). Later there was a tendency to hypotension 80-85/64 mm Hg, in connection with which the speed of dobutamine was increased to 15 mcg/kg/min. Anuria remained throughout the observation period in the Intensive Care Team. Due to the appearance of vomiting, the increase in cough, dyspnea inhalation of iloprost was terminated. Against the background of ongoing therapy, including the infusion of dobutamine the patient suddenly lost consciousness and wheezed

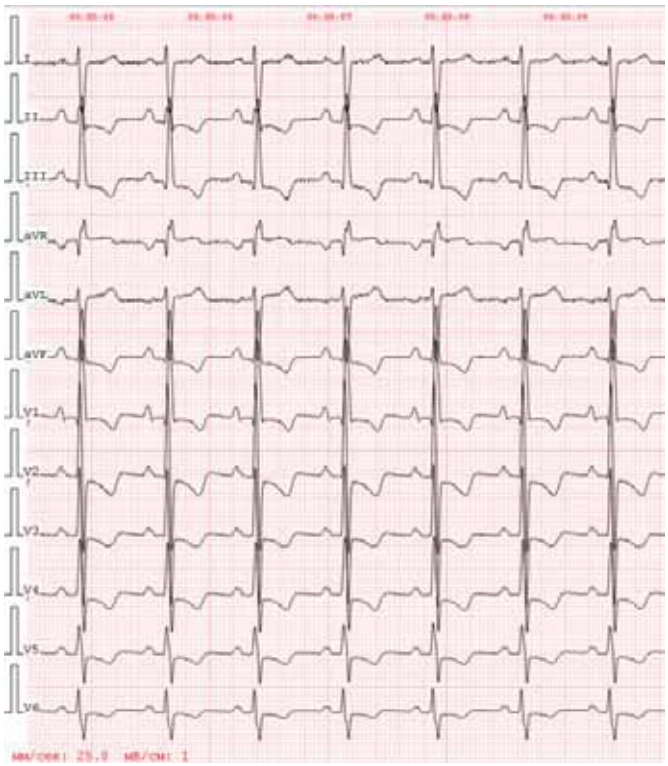

Figure 3. Electrocardiography of patient G. during second hospitalization at pain syndrome

in 7 hours. On the monitor – the decrease of the hemodynamically inefficient sinus rhythm to 30 beats/min and below, reanimation measures started: indirect heart massage, artificial ventilation by an Ambo bag, intravenous administration of atropine, epinephrine, intubation of the trachea performed, hardware-assisted ventilation started and intravenous infusion of adrenaline with increasing speed. When carrying out emergency echocardiography, you can notice the absence of a mechanical systole of the ventricles – electromechanical dissociation. Despite the resuscitation measures carried out within 50 minutes, it was not possible to restore cardiac and respiratory activity, biological death was ascertained.

An autopsy revealed a chronic thromboembolic syndrome, manifested itself with thromboembolism of the segmental branch of the pulmonary artery of the right lung, hemorrhagic infarction of the middle lobe of the right lung, atelectasis of the lower lobe of the right lung, chronic pulmonary heart. In addition, in the area of the tip of the right ventricle, a pale brown anemic section of irregular shape was found, measuring 4.0x2.5x2.0 cm. At histological research – the center of a necrosis in the field of an apex of the right ventricle, which appeared about a day ago. Coronary arteries of the heart are convoluted, yawn, the walls are thickened due to dense white and yellow plaques. Hemodynamically significant atherosclerotic lesion of the coronary arteries was not revealed. The immediate cause of death was acute pulmonary-cardiac failure.

Thus, in a patient with recurrent thromboembolism, high pulmonary hypertension, autopsy revealed an acute myocardial infarction against a background of almost unchanged coronary arteries, the clinical manifestations of which were poorly characterized for this disease. The patient complained of pains in the region of the heart, but they were atypical, and occurred with no connection with physical activity and stopped after taking sedative medication. The ECG initially had changes in the form of severe hypertrophy and right ventricular overload and in dynamics did not show changes, that are typical of AMI.

Isolated myocardial infarction of the right ventricle is extremely rare, in 1,9-4,2% of cases as a finding in autopsy [2]. The most common cause is atherosclerotic lesion (30-50% of all cases) of the right coronary artery, rarely – lesion of the envelope artery [3].

Previously it was believed that in the absence of significant coronary lesion, the right ventricular AMI is associated with RV myocardial hypertrophy [4]. Later, results of a study appeared in which AMI of the RV was found in 6 patients who died due to acute massive pulmonary embolism and without ventricular hypertrophy, while 5 patients had significant stenosing coronary lesions [5]. At the same time, there are several cases of detection of the right ventricular acute MI in patients with acute pulmonary embolism, no right ventricular myocardial hypertrophy, and hemodynamically insignificant coronary lesions (there were coronary artery stenoses <50%) [2]. Also, cases of development of AMI of the RV are described in newborns and infants in the presence of perinatal asphyxia, myocarditis, pulmonary dysplasia and pulmonary arterial hypertension of newborns [6].

Thus, in patients with pulmonary hypertension, especially with CTEPH, in the case of a sharp deterioration in the presence of pain syndrome and unstable hemodynamics, the right ventricular acute MI should be excluded. Differential diagnosis should also be performed with recurrence of thromboembolism in the pulmonary artery system, pulmonary artery dissection, compression of the left coronary artery trunk.

The reasons for the development of acute right ventricular ischemia in the absence of a significant atherosclerotic lesion of the coronary bed are not sufficiently studied at present time. Earlier, we showed the presence of signs of myocardial ischemia in patients with pulmonary arterial hypertension from synchronized single-photon emission computer tomography of the myocardium with 99Tc-MIBI at rest and in combination with stress tests [7]. Probably, in conditions when the right ventricle works against the high pulmonary vascular resistance, sometimes it's enough to have a slight decrease in the total lumen of the pulmonary bed (for example, the embolism of one segment of the lung), an additional increase in pulmonary vascular resistance for the development of acute myocardial ischemia even without significant atherosclerotic lesion in oxygen [8]. Hypertrophy of the right ventricle should be considered as a predisposing factor in this situation. With respect to a similar mechanism of development of left ventricular ischemia, in 2013 the European Society for the Treatment of Stable Angina proposed the term "microvascular angina" [9].

Diagnosis of acute myocardial infarction of the right ventricle in patients with pulmonary hypertension is very difficult. Clinical signs are pain in the chest, arterial hypotension, swelling of neck veins, a symptom of Kussmaul (increased pressure in the neck veins and their expansion on the inspiration), with the absence of physical and radiographic signs of stagnation in the small circulatory system [3]. It is worth noting that such clinical symptoms are present in most patients with pulmonary hypertension in the clinically developed stage of the disease.

All patients with suspected acute myocardial infarction of the right ventricle need to perform ECG recording in V1 lead, and especially V3R and V4R (ST elevation ≥1 mm) [10], although under the conditions of the initially changed ECG with signs of hypertrophy and overload of the right heart, this can be of little informative. Additional Echocardiography may also be not informative: characteristic features of myocardial infarction of the right ventricle, such as an expansion of the right ventricular

cavity, increased right atrial pressure (up to 10 mm Hg or more) and tricuspid regurgitation are detected in all patients with pulmonary hypertension. Lesion of the right ventricle myocardium can be confirmed by a radionuclide study (accumulation of Tc pyrophosphate in the lesion or absence of microcirculation in the appropriate area) or by MRI with contrast.

Many questions remain regarding the tactics of therapy. In acute myocardial infarction of the right ventricle, an increase in blood flow to the right heart is recommended due to the intravenous administration of a volume substitution solutions (plasmoexpanders) [10]. However, in conditions of high pulmonary vascular resistance, an increase in right ventricular preload is not advisable, since it can lead to the development of electromechanical dissociation. In the conditions of hypotension and oliguria, the appointment of sympathomimetics is justified. In this situation, the use of dobutamine is more preferable. It has been shown that the use of dobutamine leads to an increase in the cardiac index, right ventricular stroke volume [11]. Dobutamine also can reduce pulmonary vascular resistance, thereby decreasing the post-loading of the right ventricle, which is important for patients with pulmonary hypertension. However, dobutamine should be used with caution in patients with acute myocardial infarction, since at high doses it can lead to a heart rate increase and myocardial contractility, which leads to an increased need for oxygen and worsening of ischemia [12].

In acute myocardial infarction of the right ventricle, diuretics and, especially, peripheral vasodilators should be avoided. However, in patients with pulmonary hypertension, there are often manifestations of heart failure requiring the use of loop diuretics, and modern PAH-specific drugs are vasodilators and their cancellation is associated with the risk of pulmonary vascular resistance increase.

Thus, this clinical case and the analysis of literature data demonstrate that in patients with CTEPH, a sharp deterioration in the condition may be due to the development of an acute myocardial infarction of the right ventricle. The mechanisms of this complication have not been sufficiently studied until recently, the disease is diagnosed mostly posthumously, so treatment requires considerable caution. Nevertheless, timely diagnosis significantly increases the chances of a favorable outcome. Given the difficulties described above in managing patients with acute myocardial infarction of the right ventricle, it is advisable to pay more attention to early diagnosis of the disease, prevention of complications of pulmonary hypertension, careful monitoring of the state in the dynamics, timely correction of PAH-specific therapy.

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