Case report
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Acute Myocardial Injury Detected By ECG-Synchronized Perfusion Single-Photon Emission Computed Tomography in a Polymorbid Female Patient

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ABSTRACT A case of acute focal myocardial injury in a polymorbid female patient with intact coronary arteries according to direct coronary angiography is reported. Acute focal myocardial injury was detected by ECG-synchronized perfusion single-photon emission computed tomography (SPECT) of the myocardium, and confirmed by the results of postmortem examination. The article also discusses the possibility of using perfusion SPECT to diagnose acute focal changes against the background of focal inflammatory myocardial infiltration.

Keywords: acute acute myocardial injury, polymorbid patient, intact coronary arteries, histologic study, focal inflammatory infiltration, ECG-synchronized myocardial perfusion SPECT

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ACS – acute coronary syndrome
CVA – cerebrovascular accident
AST – aspartate aminotransferase
BP – blood pressure
CA – coronary arteries
CAG – coronary angiography
CPK – creatine phosphokinase
CPK-MB – creatine phosphokinase-MB
ECG – electrocardiography
EchoCG – echocardiography
EF – ejection fraction
GGTP – gamma-glutamyl transpeptidase
GSD – gallstone disease
HR – heart rate
i.v. – intravenously
LDH – lactate dehydrogenase
LV – left ventricle
MI – myocardial infarction
MRI – Magnetic Resonance Imaging
SPECT – single photon emission computed tomography

The multiplicity of diseases in one patient (polymorbidity) is one of the most difficult problems in medicine, which is of particular importance in the treatment of elderly and senile patients. The mutual influence of diseases changes the classical clinical pattern, the nature of the course, increases the number of complications and their severity. Often an exacerbation of one chronic disease causes the exacerbation of others [1-3]. The use of many diagnostic methods in such patients in urgent conditions, for example, in acute coronary syndrome (ACS), requires the clinician to know the capabilities of each of these methods.

In the harmonized recommendations of the European and American Cardiological Communities (ESC-ACC/AHA), the myocardial perfusion scintigraphy (single-photon emission computed tomography - SPECT) in patients with ACS obtained class I evidence with level "A" in the diagnosis of ischemia in patients with an unclear diagnosis, and class I of evidence with level "B" in assessing the extent of myocardial infarction (MI) in patients with ST segment elevation, STEMI [4]. The authors believe that myocardial perfusion SPECT should be used in patients who are unable to adequately exercise and with questionable electrocardiography (ECG) data [5].

After changes in the definition of “myocardial infarction” [6], the use of perfusion SPECT has become even more relevant. In these recommendations, ACS “enriched” type 2 MI in patients with intact coronary arteries (CA) according to coronary angiography (CAG), which significantly increases the group of patients with a low or medium probability of cardiac events in accordance with traditional risk markers. Infarction due to coronary thromboembolism in the setting of infective endocarditis or due to coronary thrombosis in the presence of hypercoagulable syndrome has also been classified as type 2 MI [7].

The clinical observation we have described demonstrates the informative value of the radionuclide method in identifying the zone of myocardial necrosis with intact coronary arteries and questionable ECG and echocardiography (EchoCG) data.

Clinical Case Report

A female patient M., 66 years old, turned to the admission department of N.V.Sklifosovsky Research Institute for Emergency Medicine with complaints of pain in the right hypochondrium, nausea, and vomiting. From the medical history it was known that for 20 years the patient had suffered from concomitant mitral heart disease with a predominance of stenosis, cardiac arrhythmias (atrial fibrillation) and type 2 diabetes mellitus (non-insulin-required). In 2015, the patient underwent extirpation of the uterus for cervical cancer. One and a half years before the admission (in April 2017), the patient suffered an acute cerebrovascular accident (ACVA) of the ischemic type. At the same time, atrial fibrillation was first detected at ECG. The patient underwent a repeated stroke 18 days before admission to the Research Institute Clinic. In the Admission (i.e. Emergency) Department, cholelithiasis was diagnosed, acute calculous cholecystitis was suspected. The patient was admitted to the Abdominal Surgery Department, where a detailed examination within 4 days yielded no data that would have confirmed the presence of an acute surgical pathology of the abdominal organs requiring urgent surgical intervention was obtained.

On examination: a moderate severity condition, clear consciousness, body temperature 36.8° C; marginal subicteric sclera, cavital edema of the ascites type; no respiratory impairment were revealed: the respiration rate was 18 /min; cardiovascular system: blood pressure (BP) 140/80 mm Hg, muffled heart sounds, systolic murmur, heart rate (HR) was 96 beats/min; the abdomen on palpation was soft, painful in the upper sections and right hypochondrium, the gallbladder was not palpable, there were no symptoms of peritoneal irritation; genitourinary system was without abnormalities. Complete blood count demonstrated hemoglobin 141 g/L, WBC 9.4 x 109 /L, platelets 130 x 109 /L, no other abnormal changes in the formula were found.

The abdominal ultrasound examination (US), in addition to calculus in the neck of the gallbladder, revealed the signs of free fluid in the abdominal cavity, cirrhosis of the liver and splenomegaly. The most pronounced abnormalities requiring urgent medical intervention were recorded with an ECG on the 1st and 2nd days of the patient's stay in hospital. On a series of ECGs: atrial flutter, alternating with the episodes of atrial fibrillation, the ST segment depression of up to 3 mm in leads II, III, aVF, V1 – V6 leads (archived ECGs are not provided for comparison). The patient was examined by a cardiologist: a paroxysmal form of atrial fibrillation, arterial hypertension were diagnosed; and drug therapy was prescribed (intravenous (i.v.) infusion of 600 mg of cordarone) in order to restore sinus rhythm. On the following day, the ECG record demonstrated atrial fibrillation with a heart rate of 72 beats/min. ST segment depression of up to 3 mm in II, III, aVF, V1 – V6 leads. There were no acute focal alterations (Fig. 1).

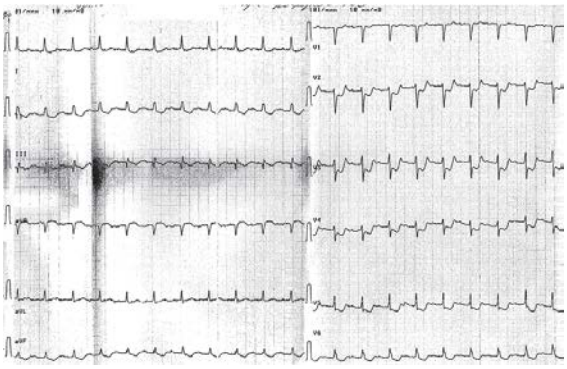

Fig. 1. Electrocardiogram of patient M. with signs of atrial fibrillation

As the treatment turned ineffective, the patient was transferred to the cardiological intensive care unit. During 15 days of intensive treatment in the intensive care unit, ECG and echocardiography, as well as CT and magnetic resonance imaging (MRI) of the brain and computed tomography (CT) of the abdominal organs, were repeatedly performed. According to echocardiography on the first day of the resuscitation period, a combined mitral valve defect was revealed: moderate stenosis of the left atrioventricular orifice, mitral insufficiency of 1–2 degree; the left atrium dilatation (50 mm, 96 ml), the 1st degree tricuspid insufficiency; marginal sclerosis of the aortic valve leaflets; satisfactory systolic function (ejection fraction [EF] 60%); left ventricular [LV]myocardial hypertrophy. Local contractility of the LV myocardium was not impaired. In the region of the middle third of the interatrial septum, a turbulent flow up to 3 mm from left to right was visualized, coinciding with the systole of the ventricles. The suspicion of an open foramen ovale was not confirmed during repeated echocardiography.

Taking into account the increase in the blood level of enzymes (creatine phosphokinase (CPK) up to 787.0 U/L; creatine phosphokinase-MB (CPK-MB) up to 158.0 U/L; troponin I up to 3.5 μg/L), CAG was performed to the patient on the same, and the myocardial perfusion SPECT synchronized with the ECG was performed on the 2nd day of the resuscitation period.

The selective CAG showed the right type of myocardial blood supply, no data for hemodynamically significant CA stenoses were revealed; there was a tortuosity of the distal bed, uneven contours.

The myocardial perfusion SPECT revealed focal alterations in the lateral wall of the LV myocardium (Fig. 2), indirect signs of focal inflammatory infiltration of the LV myocardium; LV diastolic dysfunction; LVEF was normal.

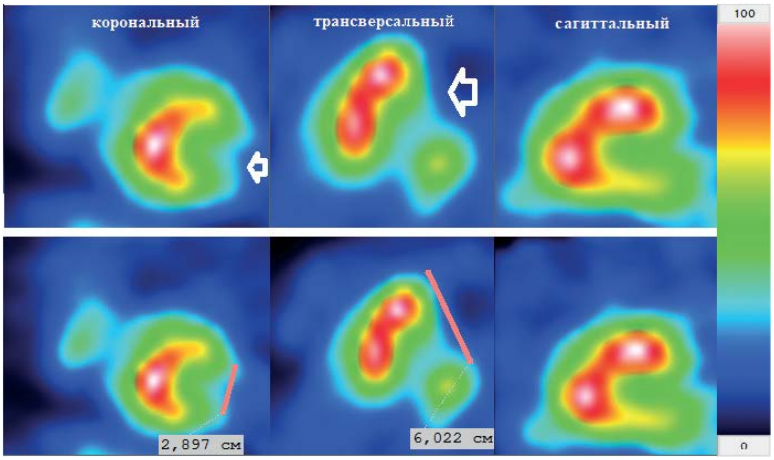

Fig. 2. Perfusion single photon emission computed tomography of the myocardium: middle slices in three standard planes with a defect of radiopharmaceutical accumulation of the infarction area (the white arrow indicates the focus of transmural perfusion disorders in the lateral wall of the left ventricle). The size of the lesion of transmural perfusion disorder is 2.9x6.0 cm (shown in the bottom row of images)

At the CT scan of the brain obtained immediately after scintigraphy, there were no reliable signs of CVA at the time of the investigation; there was internal hydrocephalus (ventricle/brain ratios 2-24%); leukoaraiosis.

Taking into account the ECG and EchoCG data, a patient with a constant form of atrial fibrillation-flutter in the cardiac intensive care unit underwent pulse-reducing therapy. Despite the focal abnormalities revealed at scintigraphy, the diagnosis of MI was not considered.

The patient's condition progressively worsened. Hyperthermia appeared up to 37.5 ° C. At the same time, hemodynamics was stable; there was no tachysystole, moderately pronounced symptoms of heart failure were observed; no acute abdominal symptoms were noted; diuresis was positive, but the signs of renal failure adjoined. There was lethargy, weakness, lack of self-care, and an acute decrease in appetite that attracted doctor's attention. Blood hematology and biochemistry tests demonstrated hyperazotemia (urea up to 26.19 mmol/L, creatinine up to 305.12 mcmol/L), hyperbilirubinemia (total bilirubin up to 39.10 mcmol/L due to direct fraction 28.29 mcmol/L), hyperenzymemia (aspartate aminotransferase [AST] up to 96.0 U/L; lactate dehydrogenase [LDH] 1056.0; gamma-glutamyl transpeptidase [GGTP 365.0 U/L).

At repeated ECGs, the ST segment depression decreased; at repeated EchoCG no dynamics was noted.

The patient was transferred to the intensive care unit for acute endotoxicosis treatment for the complex intensive care that included renal replacement therapy, an infusion-transfusion, antibacterial, hepatoprotective, respiratory and nutritive-metabolic support, as well as monitoring of vital functions and laboratory parameters.

In order to exclude CVA relapse on the 4th day of the resuscitation period,) MRI of the brain and intracranial vessels was performed, which revealed multiple small sub- and supratentorial foci of acute and subacute ischemia, foci of discirculatory genesis in both hemispheres (Fig. 3), internal hydrocephalus, signs of pronounced atherosclerotic alterations in the intracranial arteries.

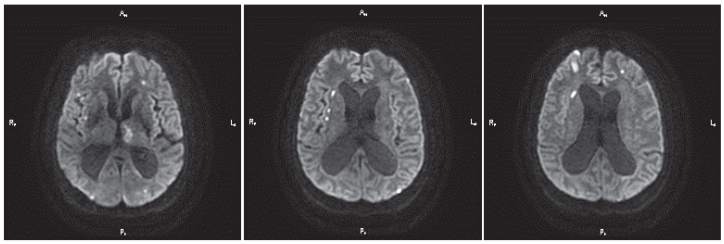

Fig. 3. Slices of the brain (magnetic resonance imaging) with multiple foci of acute and subacute ischemia

Doppler ultrasonography of the neck vessels revealed atherosclerotic stenosis of the right internal carotid artery (50%).

On the same day, the patient was examined by a neurosurgeon, gynecologist, and cardiac surgeon. The neurosurgeon's conclusion: multiple foci of ischemia do not produce a mass-effect; no data of acute neurosurgical pathology were obtained. The gynecologist's conclusion: there is no data for acute gynecological pathology. The cardiac surgeon's conclusion: there are no indications to emergency cardiac surgery.

The echocardiography in the projection of the posterior cusp of the mitral valve on day 10 of patient's stay in the intensive care unit localized a movable structure of medium echogenicity of 0.6 cm in size (vegetation?); no signs of pulmonary hypertension were found; type 1 LV diastolic dysfunction; no impairments of segmental contractility were identified; EF was 61%. The patient refused in writing from transesophageal echocardiography.

On day 13, the CT scanning of the abdominal organs was performed, which (the same as the abdominal ultrasonography on the 1st day) revealed diffuse focal changes in the liver (more likely, cirrhotic), the enlarged spleen, focal changes in the spleen (more likely, of post-ischemic nature). To exclude the abscess formation, a dynamic control was recommended. The patient was diagnosed with ascites, chronic calculous cholecystitis, the duodenal wall edema, the mass formation in the left breast, the condition after the extirpation of the uterus with appendages.

The patient's condition continued to deteriorate due to hyperazotemia, progressive renal failure and hepatic encephalopathy.

According to ECG data: the accelerated junctional rhythm (heart rate 100 bpm) turns into atrial fibrillation; deviation of the heart electrical axis to the right; complete blockade of the right bundle branch block; the right ventricle overload; insufficient blood supply to the myocardium of the anterior wall and the antero-septal region. Suspicion of focal alterations in the posterior wall? (Fg. 4.).

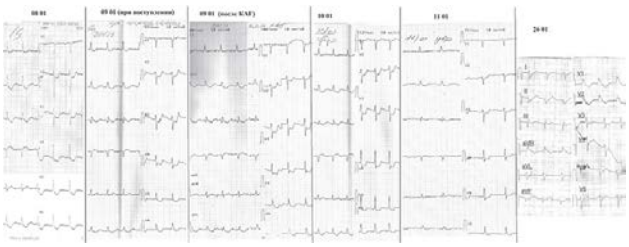

Fig. 4. Electrocardiogram of patient M. over time. The switch from the accelerated nodal rhythm to atrial fibrillation, right bundle branch block, insufficient blood supply to the myocardium of the anterior wall and anteroposterior region

On day 15, at therapist's examination, a suspicion of patient's having infective endocarditis was suggested.

On day 19 from admission, the patient's condition acutely deteriorated: bradycardia appeared with a transition to asystole. Resuscitation measures (chest compressions, artificial ventilation, intravenous 4% sodium bicarbonate 400 ml, 0.1% epinephrine 1.0 i.v. 6 times) had no effect for 30 minutes.

Biological death was stated.

Final clinical diagnosis

Underlying diseases: 1. Liver cirrhosis. Ascites. Hepatosplenomegaly. 2. Cerebrovascular disease: cerebral infarction of a certain age. Syndrome of vertebrobasilar insufficiency of the arterial system. 3. Degenerative combined mitral valve disease with the forming of the left atrioventricular foramen stenosis of moderate severity, 1–2 degree mitral insufficiency. Permanent atrial fibrillation-flutter (risk on the CHA2DS2 – VASc score 5, the HAS – BLED Risk Assessment score 5).

Background diseases: stage III arterial hypertension, grade 3 risk of cardiovascular complications 4. Atherosclerosis of the aorta, carotid arteries, cerebral arteries, CA. Atherosclerotic stenosis of the right ICA (50%).

Complications of the underlying disease: right-sided polysegmental pneumonia of moderate severity. Lower limb vein thrombosis. Multiple organ (hepatic-renal, cardiovascular, respiratory) failure.

Concomitant diseases: gallstones, chronic calculous cholecystitis. Type 2 diabetes mellitus. Chronic kidney disease, grade IV (glomerular filtration rate 21.18 ml/min/1.73 m2). Condition after the extirpation of the uterus with appendages for malignant cervical canal neoplasm. The left breast cyst. Grade 1 obesity. Organic disease of the central nervous system with amnestic-intellectual personality decline and subdepressive syndrome.

From the anatomical pathologist's protocol of the autopsy

Heart weight is 550 g. Atrioventricular orifices are patent: the left one is narrowed, the mitral valve perimeter is 8.5 cm; the thickness of the LV myocardium is 1.7 cm, and that of the right ventricle myocardium is 0.4 cm, that of the interventricular septum is 1.6 cm; ventricular index is 0.47. The endocardium is smooth, shiny, slightly dense in the left atrium, whitish, gray-pink layered deposits up to 0.7 cm thick are tightly fixed on the leaflets of the mitral valve, occupying most of the atrial surface of the leaflets. The myocardium in the section is pale brown, with multiple dense grayish foci up to 0.3 cm in diameter.

In the lateral wall of the LV along its entire length, a clearly demarcated focus of a variegated appearance is visible due to dull gray-yellow and sunken gray-red areas of 10.0x4.0 cm (Fig. 5).

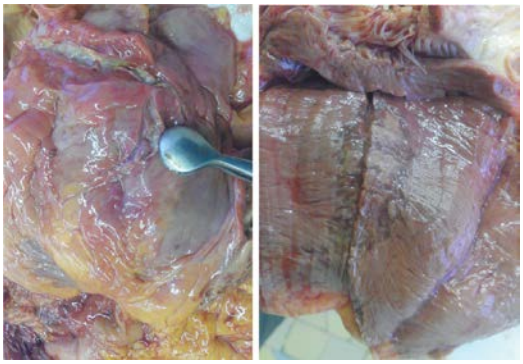

Fig. 5. Specimen of the heart. A delimited focus of a variegated appearance due to dull gray-yellow and sunken gray-red areas 10.0x4.0, corresponding to the area of myocardial necrosis

Cardiac valves: leaflets, with the exception of the mitral valve, are thin, grayish. The leaflets of the mitral valve are thickened, compressed, somewhat deformed, partially fused along the commissures. There is also sclerosis, shortening, thickening of the chords. The coronary arteries are patent for a long extent, there are few flat fibrous plaques in their intima. In the lumen of the middle third of the obtuse edge branch, a parietal gray-pink thrombus of 0.3x0.2x0.2 cm in size is loosely fixed. The blood supply to the heart is from the right. Aorta has a moderate amount of fibrous plaques in the abdominal region. The renal and mesenteric arterial outlets are freely patent. The pulmonary arteries contain liquid blood and loose, dark-red clots. Their intima is yellowish, smooth. The extracranial arteries are wide, elastic, with few flat fibrous plaques in their intima. Large veins are freely patent, contain liquid blood and loose dark-red clots.

At histological examination:

Brain: pericellular edema, hyperemia, there are scattered siderophages in the walls of small cysts, mild glial reaction.

Myocardium: uneven hypertrophy of cardiomyocytes, small-focal and perivascular sclerosis, the focus of cardiomyocyte and stromal necrosis, with a pronounced fibroblastic reaction and a zone of granulation tissue along the periphery, mixed lymphocytic-leukocyte infiltration with an admixture of siderophages; in scarce areas, the infiltration is purely leukocytic with nuclear detritus, scattered colonies of bacteria spread deep into the infarction focus; Mitral valve: severe sclerosis, densely fixed massive fibrin deposits on the surface with the signs of organization, with an admixture of polynuclear cells, macrophages, lymphocytes, as well as bacterial colonies (Fig. 6).

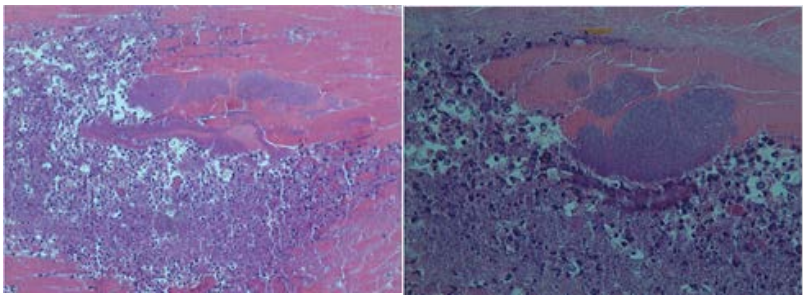

Fig. 6. Histological section of the myocardium. Staining with hematoxylin and eosin x400. Focus of cardiomyocytes necrosis with marked stromal fibroblastic reaction and the area of granulation, mixed lymphocytic-leukocytic infiltrate

Lungs: hyperemia of interalveolar septum capillaries.

Liver: the girder structure is impaired, the tissue is represented by various-sized false lobules divided by thin and wide fibrous layers with diffuse lymphocytic infiltration, which does not extend into the lobules, with pronounced proliferation of small ductules, and unevenly expressed small- and medium-drop fatty degeneration of hepatocytes.

Kidneys: edema, hyperemia, mild stromal sclerosis, extensive foci of dystrophy, necrobiosis and necrosis of tubular epithelium without reactive inflammatory abnormalities.

Spleen: pronounced hyperemia and paucicellular red pulp, the stroma is exposed somewhere, the follicles are few, small, without light centers of reproduction, there is a focus of necrosis with hemorrhagic suffusion, with leukocyte infiltration along the periphery.

Small and large intestine: edema, uneven blood supply, areas of hemorrhagic suffusion of the mucous membrane with necrosis of its superficial sections.

Stomach: edema, uneven blood supply, the edge of an acute ulcer deep within the mucous membrane, with hemorrhages in the edge, without sclerosis or significant reactive inflammation.

Mammary gland: lipomatosis, fibrosis, glandular structures are atrophic, ducts are mostly preserved, part of the ducts is cystically dilated; in one of the dilated ducts, the epithelium with proliferation without signs of atypia forms papillary structures.

The final anatomic pathologist's diagnosis (after histological examination)

The underlying disease: subacute bacterial polyposis-ulcerative thromboendocarditis of the mitral valve: massive thrombotic deposits on the valves with the initial signs of their organization, with microbial contamination.

Comparing the final clinical diagnosis and the anatomical pathologist's diagnosis

The diagnoses did not coincide. Thromboendocarditis, septic myocardial infarction, small and large intestine infarctions were missed at diagnosis. The etiology of heart disease was incorrectly determined. The cause was the underestimation of the specialist's (therapist) consultation and the instrumental examination results (a suspicion of focal abnormalities in the posterior wall myocardium at ECG; a suspicion of the vegetations on the mitral valve posterior leaflet at EchoCG; scintigraphy revealed focal alterations in the LV lateral wall, not being in doubt, and indirect signs of inflammation).

DISCUSSION

The timing of the myocardium focal changes revealed at autopsy by pathologist, taking into account the results of the histological examination, is within 7-14 days. Meanwhile, along with the changes observed in the classical course of infarction in the 2nd week of the disease, later microscopic alterations were also revealed in the form of mixed lymphocytic-leukocyte infiltration areas with an admixture of hemosiderophages. SPECT revealed focal transmural alterations in the LV myocardium of the same lateral localization on hospitalization day 2 (the following day after CAG), that is, 18 days before death.

A detailed assessment of CAG already after postmortem autopsy examination showed the presence of distal occlusion of the tiny D2 (the branch of the blunt edge-2), which could be regarded as embolic (Fig. 7; the occlusion site is indicated by an arrow). But, in any case, the occlusion of such a small branch by itself could not lead to focal changes in the myocardium of the lateral wall measuring 6.0x10.0 cm.

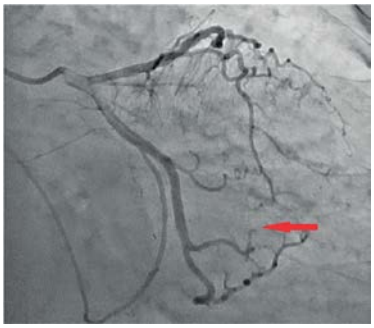

Fig. 7. Selective coronary angiography. The distal part is tortuous, the contours are irregular (indicated by the arrow). No hemodynamically significant stenosis of the coronary arteries was detected

This means that our patient had both an unusually slow repair and an unusually large area of focal damage with a small diameter of the occluded distal vessel. The presence of large colonies of bacteria and their toxins [8] could disproportionately lengthen and suppress the inflammatory phase. This, in turn, could lead to long-term tissue damage and "improper" healing, the formation of defective scars and increased death of myocytes, contributing to the expansion of the infarction zone [9, 10].

On the other hand, according to the scintigraphy results, it is clear that the focal alterations in the myocardium were already present at the time of hospitalization, and they can be classified as type 2 MI.

These findings, unfortunately, did not become a part of the clinical diagnosis. But the SPECT (scintigraphy) conclusion completely coincided with the results of postmortem autopsy examination, with regard both to the presence of focal alterations, and to the diagnosis of focal inflammatory infiltration in the myocardium (Fig. 8; "bull's eye").

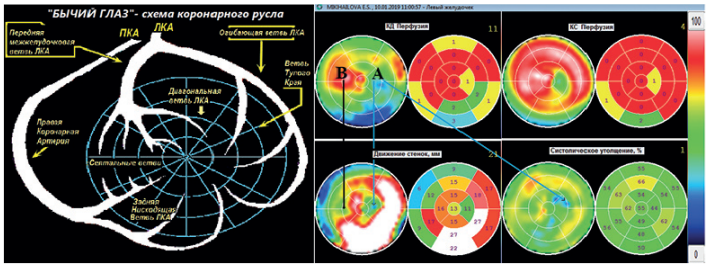

Fig. 8. On the left — a diagram of the coronary vessels in 17 segments of the bull's eye. Top row: on the left – images of the distribution of perfusion in the left ventricular myocardium during the diastole, on the right — during the systole. Bottom row: on the left — images of the movement of the walls of the left ventricle, on the right — images of the distribution of systolic thickening. The arrows indicate: A — focal decrease in myocardial perfusion, topographically coinciding with the areas of decreased movement and systolic thickening (focal myocardial alteration); B — focus of maximum perfusion, topographically coinciding with the area of hypokinesis in the image of wall movement (focal myocardial inflammation)

It is important to note that, despite this coincidence with the anatomical pathologist's diagnosis, the myocardial perfusion scintigraphy (SPECT) is not reflected in the Russian Clinical Guidelines for the

diagnosis and treatment of myocarditis [3]. Nevertheless, we have successfully used this method many times [11]. It did not let us down in this case either.

Secondary infective endocarditis in our patient proceeded without its common clinical manifestations, and typical instrumental test findings, namely:

- without severe hyperthermia, the main sign indicating infective endocarditis;
- without distinct vegetations at ultrasonography of the heart;
- without segmental contractility impairments, according to EchoCG.

Only myocardial perfusion SPECT, synchronized with ECG, as early as on hospitalization day 2 revealed deep, transmural focal alterations in the LV lateral wall and the signs of inflammation in the LV myocardium. The underestimation of these instrumental examination findings noted by pathologists when comparing the clinical and pathologist diagnoses, unfortunately, did not include the results of scintigraphy. Infrequent use of the radionuclide method in our country and the resulting lack of knowledge and confidence of clinicians in the results of scintigraphy leads to diagnostic errors as early as at the stage of emergency medical care [12].

CONCLUSION

This clinical case has demonstrated frequent mistrust in the results of myocardial scintigraphy (single-photon emission computed tomography), which is due to the small experience of using the radionuclide method in homeland cardiological practice. It should be noted that the myocardial perfusion single-photon emission computed tomography synchronized with electrocardiography makes it possible to detect myocardial ischemia in patients with an unclear diagnosis, to estimate the size and depth of the myocardial infarction focus, and should serve as a permanent diagnostic tool for cardiologists and cardiac surgeons in solving various clinical problems in polymorbid patients, especially in the diagnosis of type 2 acute myocardial infarction.

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