# MEDICAL SCIENCES

## FEATURES OF THE COURSE OF INFECTIOUS ENDOCARDITIS IN PEOPLE OF SENILE AGE (CLINICAL CASES)

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**Abstract**

Clinical cases of infective endocarditis are presented in the article, features of the course in patients of senile age are considered. The course of this pathology is quite well studied, however, in the elderly there are many diagnostic features associated with age-related changes in the body, degenerative transformation of the valves, which significantly complicates the visualization of vegetations and distorts the results of echocardiography and the presence of comorbid pathology. The presented clinical cases indicate the peculiarities of the course of infective endocarditis in patients of senile age, both women and men. In the first case, infectious endocarditis was manifested by decompensation of heart failure, however, a preserved ejection fraction, a febrile temperature with large fluctuations without inflammatory changes in the blood, and the appearance of a hemorrhagic rash were noted. The presence of perforation of a chronic ulcer significantly complicated the diagnosis of infectious endocarditis. In the second case, infectious endocarditis was manifested by prolonged subfibrillation with inflammatory changes in the blood, the development of acute coronary syndrome with manifestations of acute heart failure and acute disruption of cerebral circulation, as a complication of infectious endocarditis. The delayed diagnosis of infectious endocarditis is due to the presence of comorbid kidney pathology, which in turn could be manifested by hyperthermia, and abdominal ischemic syndrome, which was probably a manifestation of infectious endocarditis.

**Keywords:** infectious endocarditis, people of senile age, diagnostic features.

Infective endocarditis (IE) is an infectious disease of the heart valves, endocardial surface, or implanted artificial pacemakers or other cardiac devices [1], which is associated with a high rate of complications and mortality. In the latest recommendations on the prevention, diagnosis and treatment of IE [2], it is noted that mitral valves are more frequently affected in people of an older age group than aortic valves [3, 5]. In general, the risk of IE in the older age group of people is 4.6 times higher than in patients of the young population [4], in addition, it is more common in men [3].

The course of this pathology is quite well studied, however, in the elderly there are many diagnostic features associated with age-related changes in the body, degenerative transformation of the valves, which significantly complicates the visualization of vegetations and distorts the results of echocardiography, and the presence of comorbid pathology. Another feature is that perivalvular abscesses occur more often than direct damage to the valves [5]. It should be noted that transesophageal echocardiography can also often not be performed due to cognitive disorders, excitement, severity of the condition, which in turn slows down the timely diagnosis of IE. In addition, the following factors contribute to the occurrence of IE, which become quite significant especially in patients of the older age category: diabetes, infected skin ulcers, the presence of chronic infectious diseases (thrombophlebitis, otitis, pyelonephritis, prostatitis, dental granulomas), medical manipulations on the urinary tract and colon, long-term venous catheters, artificial heart valves, etc. There have been reports of IE complicating transcatheter aortic valve implantation (TAVI) [5]. About 20% of cases of IE in the elderly have a nosocomial genesis. Enterococci and str. Bovis, the source of which is the gastrointestinal tract, including both inflammatory and oncological diseases [4, 5]. It is believed that in 80% of cases the predominant causative agents are streptococci and staphylococci [3]. It is important that in this cohort of patients immunopathological manifestations are much less pronounced, and intoxication syndrome prevails, which is a consequence of the "exhaustion" of the immune system, which is characterized by the absence of an adequate temperature curve, unmotivated, in the patient's view, fatigue, general weakness, increasing shortness of breath and an erased course. Immunological phenomena (Osler nodules, Roth spots, and Janeway lesions), which are pathognomonic features of IE, are also rare [5].

**The purpose of the work** is to highlight the features of the diagnosis of infectious endocarditis in people of senile age against the background of comorbid pathology.

**Clinical case 1.** A woman G., 79 years old, was hospitalized in the therapeutic department with complaints of shortness of breath at rest, which worsened with minimal physical exertion, periodic pain in the heart of a constricting nature, significant swelling of the legs, an increase in systolic blood pressure up to 180 mm Hg, provided that its optimal pressure is 130/80

mm Hg. She was also bothered by a cough with the release of a small amount of mucous sputum, decreased urine output, pronounced trophic changes in the lower extremities. Diagnosis at referral: coronary heart disease (CHD). Stable angina pectoris of voltage IV functional class (FC) by classification of heart failure (HF), according to the criteria of the New York Heart Association. Diffuse cardiosclerosis. Hypertensive disease stage III, degree II, cardiovascular risk 4. HF of the stage D, with a moderately reduced with a moderately reduced emission fraction (EF). Hydrothorax, ascites. From the anamnestic data, it became known that about two months ago, the patient was hospitalized for pneumonia with exudative pleurisy. Coronavirus disease tests were negative. In accordance with the diagnosis, antibacterial therapy (ceftriaxone, levofloxacin) was carried out, but even after discharge from the department, hyperthermia up to 39.2°C was periodically observed, which was accompanied by chills. The temperature decreased on its own and recurred after a few days. The patient was additionally examined, including by an oncologist, but the cause of the temperature rise was not established. Complaints of shortness of breath, swelling on the legs of the patient were noted for several years. She noticed a significant and sharp worsening of her condition two days ago, when shortness of breath increased, cough increased, and bluishness of the lower extremities increased. According to the relatives, the deterioration of the condition is due to the progression of pneumonia, because they were discharged with complaints of an increase in temperature. In addition, over the past two days, a symmetrical rash appeared on the lower extremities, which did not disappear when pressed and quickly spread from the lower legs to the thighs.

Objectively: the patient's general condition is severe. Consciousness is clear. On a scale of com glasgow 15 points. The position in bed is forced - orthopnea. The skin on the lower legs is completely blue with fluid seepage. Acrocyanosis, yellowness of the skin and sclera is noted. Lymph nodes are not palpable. Body temperature is 35.8°C. Blood pressure 110/60 mm Hg. Pulse 80 bpm, rhythmic, weakened. The borders of the heart are shifted to the left, up and to the right. The first tone is weakened at the top, the accent of the second tone is over a. pulmonales. Above the lungs - vesicular breathing, sharply weakened to the right of the scapula. Body temperature is 36.9°C. Breathing frequency - 26 per min. SaO2 - 96%. The tongue is moist, clean. The abdomen is significantly increased in size due to excessively developed subcutaneous tissue. Fluid in the abdominal cavity is determined. The lower edge of the liver is percussed 6 cm below the edge of the costal arch. It is impossible to palpate the spleen. Swelling on the lower limbs with transition to the front abdominal wall, hemorrhagic rash on the legs up to the knees. The patient was immediately examined by a surgeon. The diagnosis upon referral was supplemented with acute decompensation of the left ventricle (LV) with the development of interstitial pulmonary edema, non-hospital bilateral lower lobe polysegmental pneumonia of the IV clinical group, pulmonary insufficiency I stage. Unexamined gastric dyspepsia, painful variant. It is impossible to exclude ascites-peritonitis. Chronic kidney disease stage IV (glomerular filtration rate (GFR) according to CKD-EPI=17 ml/min). Alimentary-constitutional obesity of the III stage (body mass index 43.2), stable course. Postthrombophlebotic syndrome - deep veins of the lower extremities, edematous-painful form. Chronic venous insufficiency III stage. Hemorrhagic diathesis of uncertain genesis.

During the examination, the following features were revealed: a decrease in hemoglobin to 11.4 g/dl, an increase in segmented neutrophils to 74% against the background of the upper limit of leukocytes and normal erythrocyte sedimentation rate (ESR) indicators (4 mm/h), slight thrombocytopenia ($145*10^9$/l), an increase in creatinine (223 mmol/l), urea (159 mmol/l), total bilirubin (56.4 mmol/l), fibrinogen (6 g/l). A chest x-ray revealed bilateral hydrothorax (on the right to the 3rd-4th rib, on the left - to the 6th rib), congestion in the lungs, signs of pulmonary hypertension. The heart is significantly expanded in diameter, the waist is raised. A pleural puncture was performed with an analysis of the fluid, which turned out to be an exudate. On echocardiography - sclerosis of the aorta, moderate fibro-sclerotic changes of the aortic valve. Expansion of both atria. Volume indicators of the LV are unchanged, the right ventricle is dilated (44 mm), hypokinesis of the interventricular membrane. Grade II tricuspid regurgitation was determined, mitral and aortic regurgitation were grade I. Signs of pulmonary hypertension II. Decreased systolic function. (EF - 45%). On the ECG, the rhythm is sinus, correct. Heart rate - 78 bpm. Electrical axis of the heart is shifted to the left. Insufficient growth of the R wave in leads V1-V4 without significant repolarization disorders, without changes in dynamics. Conclusions of ultrasound of the abdominal cavity: hepatomegaly (154 mm - right lobe, 91 mm - left lobe, 41 mm - tailed lobe) of a stagnant nature, ascites, right-sided hydrothorax - up to 600 ml. Examination is difficult due to pronounced flatulence.

The following clinical symptom complexes can be distinguished: pain syndrome in the heart and epigastric region, decompensated HF syndrome, arterial hypertension, which is not manifested due to decompensation of HF, hyperthermic syndrome, peritoneal irritation syndrome, trophic changes in the lower extremities, hemorrhagic rash. And this is in a patient of senile age against the background of severe obesity. In addition, it should be noted the discrepancy between the clinical data and the results of the examination. Signs of decompensated HF do not correspond to the indicators of EF, hyperthermic syndrome up to 39.2°C with chills in an elderly person without signs of an inflammatory blood reaction that does not respond to standard antibacterial therapy (levofloxacin, ceftriaxone), lasts about two months and has sharp changes. Absence of tachycardia in decompensated heart failure, presence of pain syndromes. During the day, the patient's condition progressively worsened: the body temperature increased 37°C, blood pressure continued to decrease, shortness of breath increased, and by the end of the day in the hospital, the patient dies. When forming the final diagno-

sis, taking into account the specified features, a combined diagnosis was made: Non-hospital IE of the aortic valves, active phase. CHD, diffuse cardiosclerosis, coronary sclerosis.

Complications: HF stage D, FC IV with moderately reduced EF (45%). Hydrothorax, ascites. Hypertensive disease stage III, degree II, cardiovascular risk 4. Acute decompensation of the LV with the development of pulmonary edema. Bilateral lower lobe polysegmental pneumonia of the IV clinical group, right-sided exudative pleurisy. Pulmonary insufficiency I. Peritonitis. Chronic kidney disease stage IV (GFR according to CKD-EPI=17 ml/min).

Concomitant diseases: Undiagnosed gastric dyspepsia, painful variant. Alimentary-constitutional obesity of the III stage (body mass index 43.2), stable course. Postthrombophlebotic syndrome - deep veins of the lower extremities, edematous-painful form. Chronic venous insufficiency of stage III. Hemorrhagic diathesis of uncertain genesis.

Pathological-anatomical diagnosis - combined-competing:

1. Acute polyposis-ulcerative IE of the aortic valve.

2. Chronic ulcer of the anterior wall of the upper horizontal part of the duodenum.

Complication: Venous-capillary fullness of internal organs. Right-sided hydrothorax (500 ml). Left-sided fibrothorax. Bilateral focal bronchopneumonia. Perforation of a chronic duodenal ulcer. Spilled purulent-fibrous peritonitis (up to 4000 ml).

3. Concomitant diseases: atherosclerosis of the aorta (stage of liposclerosis), arteries of the heart. Atherosclerotic cardiosclerosis. Hypertensive disease: LV-hypertrophy, myocardial thickness -1.6 cm.

In this case, IE had diagnostic difficulties, masked by clinical manifestations of CHD, aortic valve atherosclerosis, and manifested decompensated HF at a late stage of the course, which is associated with age-related features, the presence of obesity and comorbid pathology. The diagnosis is based on the clinical picture.

**Clinical case 2.** Patient Sh., 82 years old, was taken to the hospital by an ambulance team complaining of pain behind the sternum that lasts more than 20 minutes, a burning nature, shortness of breath with minimal physical exertion and low back pain with iradiation on the anterior wall of the abdomen. Notes an increase in the frequency of urination, which is accompanied by cuts and pain. An ECG was performed, where depression of the ST segment in the leads of the V5-V6 was detected. Morphine was administered and the patient was hospitalized. At the time of arrival at the hospital, the general condition was assessed as moderate, the position in the bed was active, the temperature was $36.2^0$C, the saturation was 97%, respiratory rate 18 per min., pulse 72 bpm, blood pressure 160/80 mm Hg, I tone is weakened, the emphasis of the second tone over the pulmonary artery, systolic murmur over the apex. Auscultation over the lungs weakened vesicular respiration, single dry wheezing over the lower segments of the lungs. There is pain with palpation in the lower abdomen and the course of the ureters. There are no swellings. The patient was diagnosed with CHD. Acute coronary syndrome without ST segment elevation. Diffuse cardiosclerosis. Acute LV failure, Killip I st. Chronic kidney disease, urinary stone disease, left-sided renal colic.

An additional examination revealed that over the past three months the patient had a fever of $37.5^0$C. He was treated on an outpatient basis for pneumonia, pyelonephritis, but the fever periodically recurred. He was also hospitalized for abdominal ischemic syndrome. During this period, he lost up to 10 kg of body weight. No surgical intervention was performed. Laboratory examination revealed: troponin 0.271 ng/ml, with a reference value of up to 0.1 ng/ml and no significant increase in the dynamics, which made it possible to make a diagnosis of unstable angina. Hemoglobin 92 g/l, leukocytes – $14.0*10^9$/l, ESR – 35 mm/h. Urinalysis: leukocytes 6-8 in the field of view, fresh red blood cells 25-30 in the field of view, protein 0.33. The patient was prescribed treatment according to the diagnosis (dual antiplatelet therapy, anticoagulants, nitrates, high-dose statins, diuretics, mineralocorticoid antagonist) and started antibiotic therapy with third-generation cephalosporins, taking into account the history of fever and leukocytosis. However, on the fifth day after hospitalization, the patient noted he appearance of chills, sweating in the evening, increased general weakness against the background of a decrease in pain syndrome behind the sternum and a decrease in shortness of breath. An echocardiogram was performed, which visualized mobile vegetations on the pedicle on the anterior and posterior mitral valve leaflet. Mitral regurgitation was grade III. Aortic sclerosis, fibrotic sclerotic changes in the aortic and mitral valves. Dilatation of the left atrium and right atrium. Aortic regurgitation of the first degree. Decreased EF to 38%, bilateral hydrothorax and hydropericardium. Antibiotic therapy was corrected: cephalosporin was discontinued and vancomycin in combination with amikacin was prescribed. Two days later, the patient developed a speech impairment, weakness in the left limbs, dizziness, and impaired spatial orientation. He was diagnosed with acute cerebral circulation disorder in the right parietal region, which was confirmed by a brain CT scan. For further treatment, the patient was referred to the Heart Institute of the Ministry of Health of Ukraine, Kyiv.

Analyzing the clinical features of IE in this patient, it should be noted prolonged subfibrillation with inflammatory blood changes, the development of acute coronary syndrome with manifestations of acute HF and acute cerebral circulation as a complication of IE. The delayed diagnosis of IE is due to the presence of comorbid renal disease, which in turn could manifest as hyperthermia, and abdominal ischemic syndrome, which was probably a manifestation of IE.

So, to diagnose infectious endocarditis in people of senile age in most cases is not an easy task. According to our clinical cases, it was the progression of heart failure. Hyperthermia was disguised as other comorbid conditions, and degenerative valve changes did not allow to detect vegetation in time. In addition, complications of infectious endocarditis were acute coronary

syndrome, acute cerebrovascular accident and pneumonia. It is possible to suspect infectious endocarditis in such patients in time only taking into account a carefully collected history, analysis of clinical, laboratory and instrumental research methods.

**Conflict of interest.** The authors state that there is no conflict of interest in the preparation of this article.

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