Western European Journal of Modern Experiments and Scientific Methods
Volume 3, Issue 2, February, 2025
https://westerneuropeanstudies.com/index.php/1
ISSN (E): 2942-1896 Open Access| Peer Reviewed

A CLINICAL CASE OF INFECTIOUS ENDOCARDITIS IN A PATIENT WITH MARFAN SYNDROME

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"Infectious endocarditis (IE) is a serious disease characterized by inflammation of the endocardium, most often caused by a bacterial infection. Damage to the heart valves can lead to their dysfunction, embolism, and other serious complications. Timely diagnosis and adequate treatment, including antibiotic therapy and, in some cases, surgery, are crucial for improving outcomes. "In this article, we will look at a clinical case illustrating various aspects of the diagnosis and treatment of IE."
Keywords: infectious endocarditis, diagnosis, treatment, bacteremia, echocardiography, antibiotic therapy, valve damage, complications.

Introduction:

Infectious endocarditis (IE) is a malfunction of the heart valves caused by their destruction, associated with infectious (more often bacterial) inflammation of the endocardium, accompanied by systemic inflammatory and autoimmune reactions.(3) The clinical picture of modern IE consists of manifestations of infectious septic intoxication, hemodynamic disorders due to damage to valves and myocardium, and specific complications [1, 2] IE is a very difficult disease to diagnose. Despite significant medical advances, IE remains a disease with high morbidity and mortality. Its prevention, diagnosis and treatment is still a serious problem in clinical practice. [2]

Timely diagnosis of infectious endocarditis is a determining factor in the prognosis of a serious disease.

The purpose of the work: to highlight the difficulties of diagnosing secondary infectious endocarditis using the example of a clinical case.

Research material: Patient N., 35 years old (born in 1979), was hospitalized in TMA clinics with complaints of shortness of breath, a feeling of lack of air, a rise in body temperature to 38-39°C, dry painful cough, chills, profuse sweating, discoloration of urine, headaches, dizziness, general weakness, fatigue, decreased appetite, pain in the phalanges of the fingers, brittle nails.

From anamnesis: considers himself ill during the last year. Initially, there were rises in body temperature to 37.8-38°C, pain in the temporal region. He was diagnosed with neuralgia and received therapy, the pain stopped bothering him, but his body temperature continued to rise periodically. He was examined and radiographically diagnosed with right-sided focal pneumonia. He received antibacterial therapy, and his condition improved. But after 20 days, body temperature rises of a longer nature several times a day. He was treated inpatient, and received therapy: antibiotics, nonsteroidal anti-inflammatory drugs. The improvement was short-term, unstable, and he took analgesics and NSAIDs on his own. After 11 months from the onset of the disease, he began to notice a significant deterioration in his condition, and the complaints described above joined in. After consultation at the Republican Surgery Center named after Academician V.V. Vakhidov was hospitalized.

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Anamnesis vitae: at the age of 7 months, Marfan syndrome and congenital heart disease were diagnosed: an open ductus arteriosus. He grew and developed according to his age, and they paid attention to his asthenic physique, long limbs, and poor nutrition. He was a member of the basketball team. According to the patient, his maternal grandmother had a congenital heart defect (he doesn't know which one).

Objectively: the condition is severe, the position is orthopedic, asthenic build, low nutrition, the limbs are disproportionately long, the fingers are thin, long, the terminal phalanges are in the form of "drumsticks". The skin is pale yellowish in color, acrocyanosis, mucous membranes are subicteric, dryish. There is increased pulsation of the carotid arteries and swollen cervical veins. On the skin of the abdomen, elbow, and above the joints, there is a small-point petechial rash that does not protrude above the skin surface and is painless when pressed. The chest is deformed – kyphoscoliosis of the chest and thoracic spine, expansion of intercostal spaces. Above the lungs, a percussive-pulmonary sound with a boxy tinge, on the right with 4-5 intercostal space - shortening. Auscultation - weakened vesicular breathing, fine-bubbled wet wheezing on the right from the 3rd intercostal space and below. The apical shock is visible to the eye, amplified, diffuse, shifted to the left and down. The left boundary of the relative dullness of the heart is defined in the 6th intercostal space 1.5 cm outwards from the left midclavicular line. Auscultation of the heart - at the tip of the I tone is weakened, systolic noise, the emphasis of the II tone is on the pulmonary artery, to the left of the sternum in the 1-2 intercostal space – systolic–diastolic ("machine-like") murmur. The pulse is rhythmic, 98 in 1 min. BP 110/60 mmHg. Abdomen is soft, slightly sensitive in the right hypochondrium, liver according to Kurlov 11*10* 9 sm. The spleen protrudes 1.5 cm from the hypochondrium. There is no swelling in extremities.

Examinations: Complete Blood Count (CBC): Hemoglobin (Hb) – 47 g/L, Red Blood Cells (RBC) – 1.7 × 10^{12}/L, Color Index (CI) – 0.7, Platelets – 289 × 10^{9}/L, White Blood Cells (WBC) – 8 × 10^{9}/L, Erythrocyte Sedimentation Rate (ESR) – 18 mm/h. Urinalysis (UA): Protein – 0.099 g/L, Epithelial cells – 5-6 per field, Leukocytes – numerous, Erythrocytes – 4-5 per field. Biochemistry: Total protein – 53.1 g/L. Coagulogram: Hematocrit (Ht) – 24%, Fibrinogen – 288 mg%, Prothrombin Index (PTI) – 60%, Thrombotest – Grade V, INR – 1.15, APTT – 29.84. ECG: Sinus rhythm, Heart rate (HR) – 98 bpm, Electrical axis of the heart (EAS) – shifted to the left.LVH with impaired myocardial blood supply.

Echocardiography: left ventricular dimensions are normal, EDD - 5.0 cm, EF - 53.3%. Mitral regurgitation grade 2, aortic regurgitation grade 3. Patent ductus arteriosus = 0.66 cm. Pulmonary hypertension grade 2-3. Pericardial effusion of approximately 200 ml.Conclusion: Bacterial endocarditis with vegetations on the right coronary cusp of the aortic valve, the anterior leaflet of the mitral valve, and the atrial septum. Perforation of the right coronary cusp of the aortic valve (0.5 cm).

MSCT of the chest revealed signs of right-sided middle and lower lobe pneumonia, moderate right-sided hydrothorax (59-100 ml), cardiomegaly (possible congenital anomaly?), and moderate hydropericardium. Bacteriological urine culture identified Staphylococcus aureus with determined antibiotic susceptibility.

Diagnosis: Secondary infectious endocarditis, subacute course, localized on the mitral and aortic valves and the atrial septum, immunoinflammatory phase, grade 2 activity. Hepatosplenomegaly. Right-sided middle and lower lobe pneumonia complicated by mild exudative pleuritis. Arthralgia. Severe hypochromic anemia.

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Background conditions: Marfan syndrome, congenital heart disease (patent ductus arteriosus). **Complications:** Perforation of the right coronary cusp of the aortic valve, aortic and mitral valve insufficiency, heart failure stage IIB (NYHA class III), and hydropericardium. Conducted Therapy: Antibacterial therapy, Egilok 12.5 mg twice daily, Ramipril 1.25 mg twice daily, Heparin 5,000 IU subcutaneously four times a day for 5 days, followed by Fraxiparine 300 mg subcutaneously for 7 days, Diclofenac 3 ml intramuscularly for 5 days, Verospiron 25 mg once in the morning, Reosorbilact 200 ml intravenously (drip) ×4, Ferrofer 200 mg (5 ml) in 100 ml saline solution intravenously (drip), Ascorbic acid 5% – 5 ml in 100 ml saline solution intravenously (drip).Patient's Condition and Outcome: The patient demonstrated positive dynamics, with normalization of body temperature, stabilization of hemodynamics, and resolution of intoxication symptoms, arthralgia, skin rash, lung rales, and pleural effusion. Discharged with a recommendation for a follow-up consultation at the Republican Center of Surgery named after Acad. V.V. Vakhidov.

Conclusion: Despite a history of prolonged fever, chills, profuse sweating, intoxication symptoms, and dynamic changes in objective status characteristic of subacute bacterial endocarditis, the diagnosis was established nearly a year after disease onset. This delay was likely due to the condition developing against the backdrop of a complex congenital anomaly, and only the addition of echocardiographic examination (ECHO) allowed for diagnosis verification, appropriate treatment, and prevention of further severe complications while enabling the development of a strategy for further patient management.

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