231. PULMONARY COMPLICATIONS OF INFECTIVE ENDOCARDITIS: A CASE REPORT

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Background. Infective endocarditis (IE) is a severe septic disease, with the most frequent localization of the microbial graft on native or prosthetic valves, which causes serious complications and high mortality. The annual incidence of IE is 3-10 cases per 100,000 persons, with an increasing tendency in elderly patients, whereas the overall mortality, according to the Global Burden of Disease (GBD) estimates, is 1 per 100,000 persons, representing 65,000 deaths in 2013 [1,2]. The high mortality of patients with IE is mostly caused by cardiovascular, pulmonary and renal complications. Pulmonary complications occur in 10-65% of cases, more commonly in patients with right sided IE: septic pulmonary embolism, pulmonary infarction, pneumonia, pulmonary abscesses, pleural effusion, empyema, pneumothorax and fungal aneurysms of the pulmonary arteries [3&#93].
Case report. We report a case of a 29-year-old male patient, who was admitted to the Internal Medicine Department of „The Holy Trinity" Municipal Clinical Hospital with suspected pneumonia, investigated clinically and paraclinically, and diagnosed with Infective Endocarditis. We studied the pulmonary complications in an intravenous drug user with IE and their impact on the evolution and prognosis of the disease. At the admission the patient presented the following complaints: fever, chills, night sweats, dyspnea at minimal physical exertion, dry cough, fatigue. According to the patient's history, the disease started 2 months ago after an infection of the right lower limb. He consulted the surgeon, but did not follow the indicated treatment. After being in cold, the fever came back 39˚C and he addressed the family physician. Even after administering short-term antimicrobial treatment, the fever persisted and the patient was admitted to the Internal Medicine Department with suspected pneumonia. Antibiotic therapy was started but considering that the patient has been an intravenous drug user for 8 years, EchoCG was performed to exclude the Infective Endocarditis. Objective findings: pale, moist skin, petechiae on the upper and lower limbs, Janeway lesions. Fever 39˚C. Free breathing with a respiratory rate of 24 r/min. Lung auscultation revealed a reduced inferior vesicular murmur bilaterally, wet rales. Rhythmic heart sound, S1 heart sound diminished at the apex, systolic murmur in p. IV of auscultation of the heart. Heart rate - 120 beats per minute, blood pressure - 100/70 mm Hg. Palpation revealed moderate hepato-splenomegaly. Laboratory findings. Positive blood culture, the collected Staphylococcus aureus sensitive to cephtriaxon, vancomycin, gentamicin, resistant to penicillins, erythromycin. EKG: Sinusal rhythm with HR - 94 b/min, vertical electrical axis of the heart, incomplete right bundle branch block. Echocardiographic conclusion: Considerable enlargement of RA (54mm), moderate enlargement of LA (46mm), RV (32mm). Large, mobile vegetation with a diameter of 20 mm on the tricuspid valve. EchoCG Doppler: EF-65%, IIIrd degree tricuspid valve insufficiency, IInd degree mitral valve insufficiency, Ist degree aortic valve insufficiency. Chest X-ray. Right lower lobe destructive pneumonia. Laboratory findings. Hemogram: microcytic anemia: Hgb - 70g/l, Red blood cells - $2.8x10^{12}$, White blood cells - $11.2x10^{9}$, leukocytosis with polynuclear neutrophils, ESR - 64 mm/hour. Urine test: leukocyturia, RBC

in urine. The treatment with intravenous infusions of Vancomycin 2g/day (6 weeks) and i/v administration of Gentamicin 240 mg/day, antifungal medications, vitamins and aspirin improved the patient's condition by eradicating the infection and resolving the pneumonia. After being treated conservatively, the patient was consulted by the cardiac surgeon. At the moment he does not require surgical correction of the tricuspid valve.

Conclusions. Patient Y, 29 years old, an intravenous drug user, develops an Infective Endocarditis of the right side in intact valves, the source of bacteremia being the intravenous administration of the drug and also the skin infection. The febrile syndrome and the recurrent pulmonary complications, together with the auscultative changes of the tricuspid valve, anemia, leukocytosis, increased ESR led to an early diagnosis. Following the combined antibacterial treatment, according to the standard schemes, the infectious process has been definitively resolved, but the patient requires long-term monitoring and schooling regarding intravenous drug cessation and lifestyle change.

Key words: Infective endocarditis, pulmonary complications, septic pneumonia.

232. CHRONIC INFLAMMATION AS A NEW CARDIOVASCULAR DISEASE FACTOR

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Introduction. Cardiovascular disease (CVD) is a major public health problem, in most areas of the world. While traditional risk factors for the development of CVD have been researched, the science community has recently identified chronic Inflammation as an additional risk factor. Inflammation is the result of the body's immune system activity recognizing and removing harmful stimuli to start the healing process. Chronic inflammation is referred to as a long-term disorder. Chronic inflammatory disorders include diseases such as rheumatoid arthritis (RA), systemic sclerosis (SSc), systemic lupus erythematosus (SLE), ankylosing spondylitis (AS) and psoriatic arthritis (PsA) etc., which play a crucial role in the process of atherogenesis.

Aim of the study. This research was on studying cardiovascular patients, that previously have been diagnosed with a form of chronic inflammation, to show that patients with chronic inflammatory diseases are likely at high risk of developing CVD.

Materials and methods. The aim of the research consisted in studying cardiovascular patients, that have been previously diagnosed with a form of chronic inflammation, to show that patients with chronic inflammatory diseases are likely to be at a high risk of developing CVD.

Results. By studying the significant inflammatory indicators like C-reactive protein, fibrinogen, Cytokines interleukin, the helper T cells, LDL cholesterol, triglycerides, etc. and their effects on atherosclerosis we can underline the pathophysiology of atherogenesis. When the pro-inflammatory activity starts, it also commences the alteration of lipoprotein concentrations, oxidative stress, and macrophage accumulation, the injury of the endothelial and the activation of the immune system. All these factors and many others are increasing the risk of the atherosclerosis/arteriosclerosis and supported by the traditional factors they create the best conditions for the development of CVD. Patients with rheumatoid arthritis are in the