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patients with CHF and DM). As severity of anemia increased, progressive depletion of glucocorticoid function of adrenal glands was observed due to cortisol content decreasing by 26% ($p < 0,05$ compared to patients with CHF, DM and mild anemia).

Perspectives of future investigations are connected with finding out of possible ways of the pharmacological correction of the revealed changes.

FREQUENCY AND CHARACTERISTICS OF ANEMIC SYNDROME IN PATIENTS WITH CHRONIC HEART FAILURE

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It is known that decreasing of hemoglobin (Hb) level below 12 g/dL is accompanied by progression of chronic heart failure (CHF). Aim of our study was to find out frequency and origin of anemic syndrome (AS) in patients with CHF.

We analyzed 2056 case records of hospitalized patients with CHF. AS was diagnosed in case of Hb below 130 g/dL in males and below 120 g/dL in females (WHO, 2003). Among all examined patients AS was found in 69,21% (1423 cases), which corresponds to the literature data. Among MALE patients AS was diagnosed in 1147 cases (76,22%), in females – in 276 cases (49,19%). We found out that in patients after 45 years anemia is more frequent in males than in females. Only in rare cases anemia was documented as a separate diagnosis (2,81% in case of mild anemia, 50% in case of moderate anemia, 65,8% in case of severe AS). Hyperchromic anemia (MCH > 33 pg) was diagnosed in 23 patients (1,62%), hypochromic anemia (MCH < 27 pg) – in 128 patients (8,99%); in most cases AS was of normochromic character (1272 patients, 89,39%). Mild macrocytosis (MCV 95-108) was found occasionally (19 cases, 1,34%), microcytosis (MCV < 80) – in 163 cases (11,45%), normocytosis – in 1241 patients (87,21%).

Therefore, AS is comorbid to CHF in 69,21% of patients, predominantly in males. In most patients with CHF concomitant anemia is normochromic and normocytic, which requires further investigation of its etiology for an adequate correction of hemoglobin level.

DEVELOPMENT FEATURES OF CARDIOVASCULAR SYSTEM DISORDERS UNDER THE INFLUENCE OF CONCOMITANT STREPTOCOCCAL TONSILLITIS

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Disorders of the cardiovascular system, which are etiologically associated with acute and chronic tonsillitis and nasopharyngitis, which by the way occur quite often

in adult age are rarely considered by family doctors as a direct consequence of some diseases. This knowledge would significantly speed up diagnosis and improve determining an adequate treatment algorithm focusing on the etiological link of pathogenesis.

Objective: to study the literature to determine the features of the course of myocarditis, which are etiologically associated with acute and chronic tonsillitis and nasopharyngitis, while identifying the main links in the pathogenesis that can be directly influenced during the diagnosis and treatment of cardiovascular diseases and the prevention of complications.

Materials and methods: literature review and confirmed clinical cases

In today's world, there are more than 100 different pathological conditions, syndromes and diseases that are associated in one way or another with the development of chronic streptococcal tonsillitis and nasopharyngitis in childhood (and not so rarely in adulthood). Despite the fact that the etiologic factor is more often a viral infection, group A streptococcus causes diseases in 15-30% of cases in children and 10-20% in adults. And a striking etiological factor in the development of cardiovascular disorders is chronic tonsillitis in its decompensated form

Results and discussion: The pathogenesis of disorders arising from prolonged persistence of the pathogen in the upper respiratory tract, including neuro-reflex, bacterial, toxin and allergic factors. The main principle is a shift in the immune status of the human body, which is manifested (at the first stages) by an imbalance of T- and B-lymphocytes and their subpopulations, followed by circulating immune complexes, sensitization of basophils (as a result, inflammatory mediators such as histamine are released, serotonin, bradykinin), neutrophils, eosinophils with bacterial antigens and the subsequent rapid development of cascade reactions. CICs appear as a result of the binding of pathogen antigens, among which the most aggressive are M-protein, streptolysins O and S, streptokinase, hyaluronidase, and deoxyribonuclease B, with antibodies produced by the body.

The antigen-antibody pair has a chemotoxic property, capable of increasing the proteolytic functionality of macrophages, which will unexpectedly lead to a reduction in tonsil lymphoid tissue, denaturation of proteins that acquire antigenic properties and circulate in the blood, inevitably causing the formation of autoantibodies. As a result, the tonsils become the site of constant delayed-type sensitization to antigens streptococcus. The immune process will lead to further disorganization of connective tissue with the formation of specific rheumatic granulomas (in the perivascular connective tissue or in the myocardial interstitium) and the occurrence of acute rheumatic fever.

The neuro-reflex mechanism of chronic tonsillitis and the formation of an associated pathological condition was also described. It is known that there are afferent connections of the palatine tonsils with the structures of the central nervous system, in particular, areas of the subthalamic zone (hypothalamus). These areas of the brain that are responsible for the regulation of active immune status of the body, and when pathological afferent impulses are received a chain of reactions is triggered,

which will lead to decompensated immunological reactivity of the body: activation of the nuclei of this zone, excitation of adrenergic receptors, disruption of the balance between the sympathetic and parasympathetic systems, which will lead to disorders in other organ systems as a result of the cascade of reactions.

The process and pathogenesis of the consequences, in particular in the cardiovascular system with the occurrence of carditis, chronic rheumatic fever, changes in the valvular apparatus, heart defects and the formation of heart failure, are described in detail. In the course of rheumatic fever is characterized by three phases. The first phase is characterized by the formation of a primary streptococcal focus in the upper respiratory tract, followed by a rather a long incubation period of 1.5-2 months, ending with the actual onset of rheumatism, which lasts unchanged for years.

Rheumatic carditis is more often valvular than associated with myocardial dysfunction and disorganization. It should be noted that it is very common to use anti-inflammatory drugs to mask many cases of rheumatic carditis, and there is still no convincing evidence that it does not change the natural course and pathogenesis of the disease.

The treatment of rheumatic carditis involves several basic but important steps that must include primary prevention - eradication of streptococci from the primary focus and prevention of new infections, followed by anti-inflammatory treatment, supportive treatment and treatment of complications, and secondary prevention - preventing of recurrent attacks.

However, as far back as 1947 Gore and Safir described 12 cases of myocarditis on the background of streptococcal tonsillitis that did not meet the Jones criteria for acute rheumatic fever, and this form of the disease was called "non-rheumatic myocarditis". It turned out that these were not isolated cases. The clinical forms and symptomatic manifestations of this disease are diverse - from the onset of angina pectoris to conduction changes, arrhythmias, blockages, with the appearance of ECG ST-segment elevation and positive cardiobiomarkers, and a clean picture on coronary angiography.

Such a manifestation of myocarditis may be the result of coronary microvascular dysfunction with increased sensitivity to vasoconstrictor stimuli and limited microvascular vasodilator capacity (in particular, given the above-described imbalance of sympathetic and parasympathetic NS direct exposure to an infectious pathogen that is tropic to the tissue of the heart muscle, and a cascade of immunologic changes in the large organism). In 30% of cases, this condition leads to further inflammatory and dilated cardiomyopathies, but most patients recover spontaneously without any consequences and relapses.

Conclusion: Acute and chronic upper respiratory tract infection can lead to various consequences, either by affecting the connective tissue of the body with the development of acute rheumatic fever, valve dysfunction and development of carditis or with the occurrence of "non-rheumatic myocarditis", the main pathogenetic link of which is ischemia caused by microcirculatory disorders as a reaction to an

inflammatory stimulus, therefore a detailed study of the life history with regard to ENT features is an important component of high-quality diagnosis and a guarantee of rational treatment of cardiovascular diseases.

THE EFFECTIVENESS OF DIHYDROPYRIDINE CALCIUM CHANNELS BLOCKER AMLODIPINE FOR THE COMBINATION TREATMENT OF PATIENTS WITH ARTERIAL HYPERTENSION AND METABOLIC SYNDROME

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Relevance of the work. Metabolic syndrome (MS) is a cluster of risk factors in the cardiovascular continuum, with the main role of the insulin resistance (IR) in the sequence of its development, which is, according to WHO experts, is a pandemic of the 21st century.

The drugs of choice in the complex treatment of arterial hypertension (AH) and MS should have a high antihypertensive potential, the ability to improve glucose metabolism, not worsen tissue IR, normalize the blood lipid spectrum, and limit the effect of neurohumoral factors on the cardiovascular system.

The unique properties of amlodipine are the ability to inhibit the proliferation of vascular smooth muscle cells, which underlies the antisclerotic effect of the drug, and a powerful antioxidant activity, which contributes to the inhibition of myocyte apoptosis in HF. There is evidence that the drug has antiaggregatory and antithrombotic effects.

Purpose of the work. To study the efficacy and safety of amlodipine in the complex treatment of arterial hypertension in patients with MS.

Research materials and methods. We examined 30 patients with AH with MS (4 - men, 26 - women), mean age - 62.3 ± 5.8 years. All patients was suffering of AH grade 1-2 against the background of compensated type 2 Diabetes Mellitus (DM) (15 patients) or insulin resistance (15 patients) in combination with dyslipidemia and overweight (10 patients) or obesity grade 1-2 (20 patients). The duration of DM ranged from 1 to 10 years (average 2.6 ± 1.4 years). AH was diagnosed simultaneously with DM or preceded it (mean disease duration 3.5 ± 1.2 years). We prescribed amlodipine together with ACE inhibitor at an initial daily dose of 5 mg. The effectiveness of the combination treatment was evaluated 1, 2 and 3 weeks after the start of therapy. In the inadequate response to therapy the dose of amlodipine was increased to 7.5-10 mg/day. The safety of the treatment was assessed by the level of fasting and stimulated glycemia and lipid profile indicators.

Results. A decrease in SBP and DBP was noted in all patients, the effectiveness of a starting dose of 5 mg was confirmed in 12 patients (grade 1 AH), 7.5-10 mg in 14 patients. In 4 patients, the target level of BP was not achieved, antihypertensive therapy was combined with thiazide diuretic (patients with stage II