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THYROTOXICOSIS AND IRRITABLE BOWEL SYNDROME: SERT-GENE POLYMORPHISM

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The mechanisms of bowel dysfunction in thyrotoxicosis poorly investigated. Many studies have explored the role of genetic factors in the onset and progression of irritable bowel syndrome and thyrotoxicosis. In the study of familial inheritance irritable bowel syndrome, in 33% of patients identified genetic predisposition to the disease, whereas in the general population, it was only 2%. In recent years, increasingly studied polymorphisms of candidate genes associated with irritable bowel syndrome. It is known that in the regulation of intestinal motility and secretion are involved various neural and humoral mediators plays a particularly important neurotransmitter serotonin. Gene SERT, encodes a protein-synaptic serotonin transporter with a gap in the presynaptic membrane localized on chromosome 17 in the region of 17q11.2-q1. Depending on the type of gene polymorphism, L (long allele) and S (short allele) form 3 types of genotype: LL (long, long), LS (long-short) and SS (short-short).

The aim of our research was to investigate SERT-gene polymorphism in patients with thyrotoxicosis and irritable bowel syndrome.

We investigated 38 women with diffuse toxic goiter and symptoms of irritable bowel syndrome. All of patients were examined for gene SERT, encoding the serotonin transporter protein. By the nature of violations of the digestive organs of patients divided into 3 groups. The first group included 12 patients with diffuse toxic goiter and with irritable bowel syndrome with diarrhea -type, the second group - 12 patients with constipation. The third group consisted of 14 people with thyrotoxicosis without violations of the digestive system.

In the first group of patients, we found all types of polymorphism: 67% had a homozygous carrier LL alleles SERT, 25% - SS- genotype, and only 1 patient (8%) were heterozygous carriers of LS. Individuals of the second group tended to be short-allele carriers, in particular, 75% of patients were heterozygous of LS, whereas 25% had SS-genotype. In the analysis of a group of individuals without violating the intestinal function number of patients with SS-genotype (79%) was significantly dominated by the number of LS- heterozygotes (21%).

It was found that the type of intestinal dysfunction in diffuse toxic goiter is associated with gene polymorphism SERT, which raises the need for correction of medical tactics in these patients.

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CLINICAL PECULIARITIES AND COMMON MECHANISMS OF DEVELOPMENT, PROGRESSION OF ARTERIAL HYPERTENSION AND OSTEOARTHITIS

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Arterial hypertension (AH) is a disease with marked epigenetic and exogeneous components. Sympathoadrenal system plays a key role in its genesis being activated by the shift of baseline level of blood pressure to higher operative values in a controlling area of ventromedullar brain portion as a result of influence of different factors.

The analysis of modulators of adrenergic processes in the central nervous system was conducted and importance of balance between vasoconstrictive (proinflammatory cytokines, and interleukin-1β, in partial; free radicals and antioxidative defence) and vasodilating(nitric oxide) agents was highlighted.

Osteoarthritis (OA) is one of the most frequently detected variants of concomitant pathology in patients with cardiovascular disorders: they co-exist in 53 % to 78 % of patients, especially those with obesity.

Hypocalcemia and hypercalciuria in AH are suggested to be extremely important factors with are markable input into progression of OA (they influence mineral density of the subchondral bone altering metabolic status of nearly located cartilage.

Progression of atherosclerotic lesions of arteries worsens ischemic state of the synovial-cartilage complex and potentiates further degradation of joint tissue and failure of reparative mechanisms. Hyperlipidemia in AH and OA patients worsens both diseases: oxidized low density lipoproteins are involved into increase of systemic production of reactive species of oxygen by means of stimulation of leptin-like receptors and their accumulation in endotheliocytes and other target tissues.

Progression of OA is linked with generation of a great variety of free radicals. Some of them act locally (modify protein aggregates in extra-cellular matrix of afflicted cartilage), but other ones, those with long half-life period, are transmitted to great distances (contributing to vascular walls rebuilding with rigidity development). Higher concentration of free radicals, peroxinitrites in partial, requires neutralization by antioxidantsin the blood and tissues, that is the background to decrease the concentration of vasodilator nitric oxide as well.

Cartilage and synovial cells express higher levels of cyclooxygenase-2 in case of affliction. Generation of vasoactive prostaglandins is supposed to be pain mediators is a result of its activity.

Chronic pain in OA patients is an extra factor of stable increase of blood pressure. Alertness, limitation of mobility, alteration of social functioning, need to be operated in future, expenses and complicated rehabilitation are forming a wide spectrum of psychological tension in afflicted by OA patients.

Pain and inflammatory edema can mask manifestations of congestion in the large circulation circuit in case of chronic heart failure. Hypodynamia that is the result of pain facilitates development of obesity, which, in its turn, switches on a lot of additional molecular interactions worsening the course of underlying pathology.

Hence, AH and OA are comorbid pathology: they are related pathogenetically. Clinical manifestation of any of the couple worsens the second one.The search of criteria of early diagnostics and prediction of complications remains a topical task of medicine nowadays.

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METABOLIC SYNDROME IN PATIENTS WITH RHEUMATOID ARTHRITIS

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Rheumatoid arthritis and metabolic syndrome are considered to be diseases with common traits that can increase the risk of cardiovascular disease incidence. The prevalence of metabolic syndrome (MS) among rheumatoid arthritis (RA) patients is 37%, which almost corresponds to the prevalence of metabolic syndrome among patients with coronary heart disease - 41% and occurs with greater frequency than in the population (10-30%). Patients with rheumatoid arthritis have an increased risk and a higher mortality from cardiovascular diseases, the rheumatologist should be aware of those MS risk factors and attempt to modify them.

The aim of our study was to investigate some criteria of MS (based on criteria recommended by the International Federation of Diabetes, 2005) in patients with RA.

The study involved 30 patients with RA who were hospitalized in the rheumatology department of Chernivtsy regional clinical hospital. The control group consisted of 20 healthy individuals. Clinical examination of each patient included general clinical and special studies. For the study of carbohydrate metabolism conducted laboratory studies of blood to the definition of indicators of blood glucose and insulin levels. The level of insulin resistance (IR) was calculated using the formula HOMA-IR. Waist circumference measured by tape at the navel.

Increased waist circumference (central obesity type) in women> 80 cm in men> 94 cm was observed in 40% of women and 36.7% of men in patients with RA. In the control group - 25 and 20%, respectively ($p < 0,05$). IP is observed in 20% of patients with RA, diabetes type 2 - 3.3% increase in fasting blood glucose> 5.6 mmol/l - in 23.3% of patients with RA in the control group IR 5% and improving fasting blood glucose by 10% ($p < 0,05$). Increased blood pressure (> 130/85 mm Hg) and / or the use of antihypertensive therapy was found in 46.7% of patients with RA and 10% in the control group ($p < 0,05$).

So, signs of metabolic syndrome in patients with rheumatoid arthritis are significantly more likely than in the control group. Combined course of disease requires attention from clinicians to develop a differentiated approach to the prevention of metabolic syndrome among patients with rheumatoid arthritis.

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QUALITY OF LIFE IN PATIENTS WITH CHRONIC HEART FAILURE AND DIABETES MELLITUS TYPE 2 AND POSSIBILITY OF ITS CORRECTION

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Diabetes mellitus (DM) is one of the leading medical-social problem of the modern society due to its high incidence, frequent comorbidity with concomitant pathology, increased mortality, high risk of chronic vessel complications. In Ukraine, same as in the world, the number of diabetic patients is continuously increasing mainly due to people with diabetes mellitus type 2, the number of which totally in the population of patients with this disease is around 90% (Pankiv V.I., 2010).

The aim of the research was to determine the impact of chronic heart failure and diabetes mellitus type 2 on the quality of life of elderly and senile patients.

A comprehensive survey of 108 patients with chronic heart failure (HF) of ischemic origin and DM type 2 was conducted. The average age of the patients was 76,04±1,84 years. All examined patients according to their comorbidities were randomized into the following subgroups: I – patients with HF without DM type 2 (n=32), II – patients with HF, complicated by concomitant DM type 2 (n=76). The control group for comparative studies comprised 24 people without HF and DM type 2, whose age was not significantly different from the average age of the patients of the experimental groups. All patients received basic therapy of the main and concomitant diseases. Moreover to achieve the objective of the investigation telmisartan was prescribed additionally. Therefore, patients with heart failure and diabetes mellitus type 2 were randomized into subgroups according to the prescribed treatment: IIA subgroup – patients who received only basic therapy (26 people); IIB subgroup (30 patients) – those for whom in the scheme of the standard treatment substitution of ACE inhibitor by angiotensin II receptor blocker telmisartan (MIKARDIS®, Boehringer Ingelheim) was conducted. Telmisartan was prescribed in the daily dose of 40 mg after meals. Duration of hospital treatment was 21-24 days, in addition, it was recommended to continue treatment with telmisartan up to 3 months. Quality of life was determined by Mezzich J. E., Cohen M., Ruiperez N. et al. questionnaire.