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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.