blood, and cardiovascular homeostasis. It was also demonstrated that ACE gene, in addition to the production of angiotensin II, is an activator of bradykinin. Bradykinin is known to be established as a factor of tumor formation due to its ability to stimulate the growth of vascular permeability.

In fact , the genes required for certain functions such as vascular alteration are known to be probably involved in primary tumor progression and metastasis. Cancer cells may disseminate early enough in relation to the period of tumor existence in the body. Angiotensin-converting enzyme is a key enzyme in the RAS and can affect the tissue angiogenesis, cell proliferation, apoptosis, and inflammation. The results of epidemiological and experimental research have shown that the RAS can contribute to paracrine regulation growth of tumor. During the studies it was found out that renin levels increased in patients with hepatic cirrhosis and hepatocellular carcinoma. ACE gene overexpression was noted in extrahepatic cholangiocarcinoma in myeloid leukemic blast cells and in macrophages of the lymph nodes in patients with Hodgkin's disease.

While the work was being performed, we collected and in analyzed the statistical data showing that the ACE gene single nucleotide polymorphism may have a significant impact on the risk of oncological pathology, in particular breast cancer. I / D-polymorphism of ACE gene was associated with a 3-year disease-free survival. Disease free survival for D / D carriers was significantly reduced compared with the I / D and I / I carriers. There is the evidence that RAS inhibitors reduce the tumor growth, progression and metastasis. Angiogenesis, cell growth and invasion of cancer cells have been the targets for new strategies for the treatment of malignant tumors in recent years.

Based on the initial data, we can summarize the following:

1. Genotyping results allow to conclude that I / D ACE gene polymorphism results in an increase of breast cancer and other forms of cancer risk of oncologic pathology.

2. People with D / D genotype of high activity are marked by an increased risk of breast cancer compared to people of II / ID genotype of low activity.

3. RAS inhibitors result in the reduced growth and angiogenesis in tumor cell lines.

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QUANTITATIVE CHARACTERISTICS OF T LYMPHOCYTES IN PATIENTS WITH MULTIPLE SCLEROSIS

Multiple sclerosis (MS) is the central nervous system demyelinating disease characterized by multifocal white matter lesions and chronic paroxysmal-progressive course. The pathogenesis of MS involves an immune attack against the

central nervous system antigens, mediated through the activation of $CD4^+$ myelin-reactive T-cells with the possible contribution of B-cells. The immunopathogenesis of MS associated with the disturbed self-tolerance to other antigens and myelin, the central nervous system results in activated peripheral autoreactive T-cells.

The objective is to study the quantitative characteristics of T lymphocytes in patients with multiple sclerosis.

The tasks are the following:

1. To assess the number of different T lymphocyte subpopulations in patients with MS.

2. To compare the parameters of T-cell immunity in patients with MS and the experimental group.

3. To evaluate and compare the number of subpopulations of T-lymphocytes in patients with different forms of MS course.

The research methods. The number of T-lymphocytes of peripheral blood was determined by flow cytofluorometry.

The results:

1. In patients with multiple sclerosis, the changes in cellular immunity characterized by lymphopenia with a statistically significant reduction in relative value of $CD3^+$T-lymphocytes and γδT lymphocytes in the peripheral blood are revealed. This indicates the possible involvement of γδT lymphocytes in the development and regulation of the specific immune process as cytotoxic, regulatory T cells and antigen-presenting cells.

2. The most pronounced changes in the number of T-lymphocytes are determined in secondary-progredient course of the disease when degenerative processes prevail over the autoimmune reactions. The marked reduction in $CD3^+$ lymphocytes in combination with a statistically significant increase in the subpopulation of T lymphocytes-helpers may indicate an enhancement of humoral immune responses mediated by the production of antibodies by B-lymphocytes to myelin antigens, which enhances the manifestation of destructive processes in the central nervous system.

Conclusions. Considering the leading role of immunological reactions in the pathogenesis of multiple sclerosis, immunological monitoring for the activity and development of the pathological process of this disease is of great importance. On the basis of the data of long-term studies of the immunity in patients with multiple sclerosis it can be said that, firstly, immunological changes are ahead of the clinical ones; secondly, immunopathological process is dynamic, and in the course of the disease the reaction of the immune system changes, the depletion of a number of compensatory reactions occurs, and a number of new protective reactions simultaneously develop.