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ANALYSIS OF DIAGNOSTIC VALUE OF POPULATION AND SUBPOPULATION STRUCTURE OF LYMPHOCYTES IN NORMAL AND HIV-INFECTION CONDITIONS

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So, despite many opportunities of typing of various markers of lymphocytes and other cells, results of their research sometimes have insufficient informational content. In particular, at considerable deviations in the state of health of the person results of a research of population structure of lymphocytes show the level of physiological values. Taking into account this phenomenon of clinical laboratory dissociation, development and studying of approaches to interpretation of results of immunophenotyping of peripheral blood lymphocytes represents an important and current problem. Now it is solved with involvement of specialists in the field of mathematics, cybernetics and informatics whose efforts are able to afford to achieve new success in understanding of norm and aberrations in the field of population structure of lymphocytes.

Keywords: receiving antibodies, immunophenotyping of peripheral blood lymphocytes of group of clinically healthy donors and a group of persons with HIV infection, functional qualities of the lymphocytes of patients, possibilities of the organization of lymphocytes in the field of immunity.

Taking into account the relevance of a subject the purpose of the real research consists of the analysis of diagnostic value of population and subpopulation structure of lymphocytes in the normal condition and with a lymphotropic infection (HIV infection). For realization of the purpose the following tasks have been set: how to analyse the database on immunophenotyping of peripheral blood lymphocytes of group of clinically healthy donors and a group of persons with a lymphotropic infection (HIV infection); to estimate functional qualities of lymphocytes of the examined persons from a position of full value of their function and a possibility of the organization by them of work in the field of immunity were set.

The contingent of surveyed included a group of 24 HIV-positive persons, including12 men and women at the age of 18-35 and a group of 21 clinically healthy donors, as a part of 12 men and 9 women. For immunophenotyping of lymphocytes peripheral blood was used. The straight line the IFA for immunophenotyping of lymphocytes on the provided protocol was put (with registration of results by method of a flowing tsitofluorimetriya).

Thus, the conducted research with use of monoclones to markers of a population and subpopulation order of lymphocytes and a method of a flowing tsitometriya allows to draw the following conclusions:

1) HIV infection is followed by significant changes of population structure of lymphocytes:

– oppression of a B-lymphopoiesis is observed;

– the quantity of NK lymphocytes decreases;

– the accelerated death T-helperov;

– there is an increase T-cytotoxic.

Loss of CD4 cells is considered a key indicator of progressing of HIV infection.

2) the changes of population structure which are available at HIV infection reflect development of physiological and functional changes of lymphocytes, resulting an organism in the status of an immunodeficiency.

Having calculated CD4/CD8 ratio - lymphocytes of two groups, we see that at group of HIV-positive patients very low level of the immunoregulatory index is observed. It demonstrates that patients have some of the following diseases: congenital immunodeficiency disorders, infections (virus and bacterial, including HIV infection), chronic diseases, multiple myeloma, stress, oncological diseases.

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PATHOLOGICAL ROLE OF AUTOPHAGY IN OSTEOARTHRITIS

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The incidence of osteoarthritis is influenced by many factors. Among them are mechanical, genetic, senescence-associated, ecological and other. Autophagy is considered a key factor in the pathogenesis of OA. Arising in healthy cartilage cells as a protective mechanism, it becomes defective and leads to development of the osteoarthritis.

Keywords: autophagy, osteoarthritis, senescence, inflammation, aging, chondrocytes, cartilage.

Osteoarthritis is a type of chronic joint disease that is characterized by the degeneration and loss of articular cartilage and hyperplasia of the synovium and subchondral bone. Articular cartilage is very susceptible to the senescence-related changes, because of the low turnover of chondrocytes and extracellular matrix. There are very small part of proliferating cells in mature cartilage. According to this, cells are prone to accumulate changes related to trauma, mechanical or oxidative stress over time. These changes include reduction of oxidative defense system, aberrant gene expression, which is responsible for abnormal protein synthesis, and altered responses to growth factors and cytokines. These mechanisms are critical to maintenance of chondrocyte survival and normal function.

Autophagy has role in pathogenesis of several diseases. It also regulates the aging process. It is a highly regulated cellular mechanism with both beneficial and pathogenic effects. Cellular homeostasis require a well-regulated balance between protein synthesis and degradation. There are two basic mechanisms for degradation in eukaryotic cells by the proteasome and autophagy. So autophagy involved in the degradation of long-lived proteins, whereas the ubiquitin proteasome system degrades specific short-lived proteins. Autophagy protects against neurodegeneration, heart diseases, infections and even cancer. The autophagy mechanisms are loosen with aging and related to the failure of the lysosomal hydrolases. That leads to accumulating of protein catalysis products and slow clearance of autophagosomes in the aging tissues. In addition, there are some hormonal changes.

In articular cartilage, the role of autophagy in the maintenance of cellular homeostasis and function is particularly important, due to the low rate of chondrocyte proliferation. Autophagy is considered a key factor in the pathogenesis of OA. Homeostasis in chondrocytes is maintained by intercellular interaction, organelle functioning and normal biosynthesis functioning. A common feature of degenerative diseases (including OA) is the accumulation of destructive macromolecules, which leads to the loss of the extracellular matrix, cell dysfunction, and death. Chondroptosis is the term, which describes the death of chondrocytes in articular cartilage. This process includes apoptosis and autophagy. In OA patients, autophagy activation function is missing, thus leading to chondrocyte death and tissue destruction.

The functional deficiency of autophagy can lead to mitochondrion dysfunction and abnormal accumulation, further increasing the risk of OA. For instance, lack of effective mitochondrial coupling in OA causes deficiency of the reparation ability of articular cartilage. Additionally, pathological chondrocytes contain large amount of reactive oxidants. Moreover, the increase in oxidative stress, reduction of chondrocyte proliferation, inflammation, and death of chondrocytes are all related to mitochondrial dysfunction. The mitochondrial dysfunction plays an important role in OA pathogenesis. Autophagy can be activated to combat the dysfunction of the mitochondria in human chondrocytes. It is one of the indispensable regulatory mechanisms for intracellular homeostasis.

Autophagy does not only regulate nutrient provision but can also play an important role in the removal of dysfunctional organelles and macromolecules, an activity that can be confirmed in OA. Animal studies have indicated that the activation of autophagy can prevent cartilage from mechanical damage in OA. With aging chondrocytes lose some mechanisms of autophagy, which helps to defend them and maintain homeostasis. Therefore, autophagy became defective and leads to OA development.