indications for suturing the surgical wound. The number of programmed laparation operations depended on the nature of the inflammatory process and averaged 3.2 ± 1.4. According to the results of microbiological studies, the number of microorganisms before suturing the surgical wound was significantly lower than the etiologically significant concentration.

For the period between the openings of the peritoneal cavity, we used the designed method of peritoneosorption, placing in all its departments containers with sorbents, which were given antimicrobial properties, which were replaced during the next laparation. This allowed up to 80% of peritoneal exudate to be adsorbed together with microorganisms, reducing their peritoneal damage and preventing translocation.

Thus, the evaluation of variants of the IL1β 511 C / T genotype makes it possible to predict the nature of the inflammatory process, and the use of treatment tactics through the use of improved techniques of peritoneal rehabilitation can significantly increase the effectiveness of treatment of patients with acute peritonitis.

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LABORATORY TESTING OF RETINAL PIGMENT EPITHELIUM DYSFUCTION IN DIABETIC RETINOPATHY

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A major complication of diabetes and a leading cause of blindness in working-age population of the developed countries is a Diabetic retinopathy (DR). It is traditionally regarded as a disorder of blood-retina barriers. There is a leakage of blood content as a major pathological characteristic of the disease. The vascular leakage through the retinal pigment epithelium (RPE) barrier in the disease has not been widely acknowledged, while the breakdown of the endothelial barrier in DR has been investigated extensively. The leakage of blood content through the RPE barrier causes excessive water influx to the retina. The resultant breakdown of the RPE barrier is likely to play a causative role in the development of some forms of diabetic macular edema. The latter is a major cause of vision loss in DR.

A causative role in DR is a breakdown of RPE barrier, particularly for some forms of DME. Currently the extent and significance of the diabetes-induced RPE barrier breakdown in humans are not clear. However, treatment of the RPE barrier breakdown should be considered as an intervention in DR for the following reason. So as the endothelial and RPE barrier are interconnected to the fluidal retina, the leakage through both barriers are additive to the overall insults.

The diabetes-induced endothelial barrier breakdown was reduced dramatically in Muller cell-specific VEGF knockout mice. This reduction in retinal VEGF is overall to approximately 50% of that in wild-type controls. Reducing the overall insults under a “pathological threshold” is essential for keeping the disease under the control.

Genetic disruption of VEGF signaling in the mouse RPE caused a measurable reduction of overall diabetes-induced vascular leakage and inflammation. Anti-VEGF therapies on the treatment of DME certainly support the beneficial effect of this idea.

A lack of progress in developing the methodology for clinical diagnosis and for research in the biology of the RPE barrier certainly makes it difficult to advance the field in a more significant way, although many achievements have been made in the biology of the RPE barrier. That is why, not as many experiments related to the RPE barrier were carried out in in vivo settings.

New technology of fluorescent microscopic assay is needed for imaging the RPE barrier-specific leakage in experimental animals, and perhaps in humans, as future goals. That is why active work with bioengineers is on.

The potential use of recent developments in tissue-specific gene expression tools for the RPE and animal models of RPE-specific gene knockout could be manipulated to the RPE barrier specifically. The significance of the RPE barrier breakdown in DR, as well as that in other retinal

diseases, in conjunction with a combination of approaches in vivo and in vitro, will be recognized appropriately in the near future.

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DIFFERENTIAL DIAGNOSIS OF NODULAR GOITER ON THE BACKGROUND AUTOIMMUNE THYROIDITIS AND DIFFERENTIATED THYROID CANCERS

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In modern literature there are many publications dealing with a study of nodular goiter combined with autoimmune thyroiditis (NGAIT) morphology. However, some issues remain unresolved including the question of the role of autoimmune thyroiditis (AIT) in the development of tumor processes. According to the literature, AIT leads to metaplasia processes in the thyroid epithelium, hyperplasia of lymphoid tissue, which undoubtedly can be considered as an optional precancerous condition.

The information that papillary cancer and lymphomas occur three times more frequently in patients with NGAIT confirms this idea.

The total accuracy of clinical, instrumental and laboratory diagnostic methods for the establishment of morphological origin of nodular new growths in the TG even in the most daring conclusions does not exceed 80%. This result cannot be satisfactory either for surgeons (unjustified over diagnosis of thyroid cancer) or endocrinologists (inadequate and ill-timed selection of patients for surgical treatment). Unfortunately, the chemical reagents used in the preparation of drugs for morphological studies by a standard method, block most of the antigenic determinants. That is why immunocytochemical and morphological studies of the biopsy material are performed on individual drugs, which lead to additional needle biopsies and prevent from the morphological identification of the cells reacting with antibodies. Instead, the best for PCE is the option when cytomorphological and immunocytochemical study is carried out consistently on the same smear of a puncture material. One of the mechanisms of malignant transformation and progression is a cell cycle dysregulation with apoptosis inhibition and proliferation activation.

It is quite necessary to solve these problems, because the correct choice of treatment strategy, timely surgical treatment and therefore the patient’s survival largely depend on the accuracy of PCE. That is why our aim was to study the processes of proliferation and apoptosis in thyroid puncture material under NGAIT using immunohistochemical method of investigation as well as determining the proliferative activity index.

We examined 75 women with nodular NGAIT and 12 patients with differentiated thyroid cancer during 2016-2019. While preparing the smears we used a method of restoration of antigen determinants activity designed and patented in V. I. Komisarenko Endocrinology Institute laboratory. It enabled us to combine cytomorphological and immunocytochemical researches in one cytological preparation and provided a possibility to compare morphological and immunocytochemical characteristics of certain cellular elements.

The results of immunohistochemical reaction were evaluated by means of semiquantitative analysis, proposed by A.K. Khmelnytskyi, according to the intensity of color "+ -" - small "+" - poor, "++" - moderate, "+++" - pronounced. Assessment of immunoreactive cells was calculated by the formula (Fas, FasL, Bcl-2, P53, Ki-67) = N1 / N2 × 100%, where N1 was the number of immuno-positive cells to Fas, FasL, Bcl-2, P53, Ki-67 receptors, N2 - the total number of the cellular nuclei per 1 square millimeter. Morphometric analysis was performed by means of the microscope Bresser BioScience Bino (Germany) with a digital camera Nikon DS-Fil, personal computer with installed software NIS-Elements F 3.2.

The results showed the degree of proliferative activity in the thyroid tissue NGAIT. A high proliferative activity of lymphoid tissue, moderate proliferative activity in the area of thyrocytes lymphoid infiltration and low - outside. Marked expression of Fas and FasL in t thyrocytes in areas of lymphoid infiltration indirectly indicates that when there NGAIT immunologically caused apoptosis thyrocytes. This has been an increase in the expression of FasL in patients punctate DTC,