and 47,7% (9), respectively and did not differ statistically from that in patients with pancreatitis of nonbiliary genesis – 33,3% (6) and 61,1% (11) respectively ($\chi 2 = 0,003$, $p = 0,95$ and $\chi 2 = 0,68$, $p = 0,4$ respectively).

While analyzing the group of patients with acute edematous biliary pancreatitis, it was established that the homozygous carriers of the favourable "wild" N - allele and heterozygotes occurred with the same frequency - 50% (5) and 50% (5) respectively. However, a tendency towards a domination of the NS – genotype was established in patients with edematous pancreatitis of nonbiliary genesis as compared with the NN – genotype whose frequency of detection involved 85,7% (6) and 14,3% (1) respectively. However, such differences were not statistically significant ($\chi 2 = 2,00$, $p = 0,16$). No homozygous carriers of the mutant S - allele were detected in patients with acute edematous pancreatitis.

In patients with acute destructive pancreatitis of biliary and nonbiliary genesis the frequency of detecting genotypes NN - (N34) and NS - (N34S) did not differ significantly: 55,5% (5) and 44,5% (4) versus 45,5% (5) and 45,5% (5) respectively ($\chi 2 = 0,001$, $p = 0,97$ and $\chi 2 = 0,114$, $p = 0,74$ respectively).

Thus, the frequency of the NN - and NS - genotypes of the SPINK1 gene in the patients examined by us, did not differ significantly from patients with various forms of acute pancreatitis. The carriage of the unfavourable SS - genotype, in our opinion, may be a contributory factor for the onset of the disease and a potentiation of its further progression, as well as a prognostic marker of a severe clinical course of acute pancreatitis with the development of necrotic lesions of the pancreas.

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CURRENT ISSUES OF TREATMENT OF ACUTE PERITONITIS

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Improving the effectiveness of treatment of acute peritonitis is one of the most difficult problems of abdominal surgery. Despite significant advances in the development of treatments for such patients, mortality remains high (from 16% to 63%) and does not tend to decrease. One of the reasons for this is the excessive activity of IL1β, which carries genetic determinism, which serves to progress the inflammatory process in the peritoneal cavity and insufficient effectiveness of existing methods of peritoneal remediation, which leads to prolongation of the inflammatory process, its progression, translocation and generalization of microorganisms.

The use of laparoscopic technologies and the development of methods for predicting the occurrence of the inflammatory process in the peritoneal cavity is one of the most promising ways to improve the results of treatment of patients with acute peritonitis.

However, in diffuse and general peritonitis, laparoscopic techniques do not allow remediation of all pockets and depths of the peritoneum, therefore, preference should be given to laparotomy accesses.

All patients were recognized with a variant of the IL1β 511 C / T gene, and after elimination of the cause of peritonitis, peritoneal remediation was performed by repeated washing with antiseptic solutions, preferring surfactants. We have improved the technology of flushing the peritoneal cavity by supplying the solution to the peritoneal cavity under the pressure created by oxygen. This helped to reduce the number of bacteria, especially anaerobic and provided vibromassage of tissues with oxygenated solution, which improved their microcirculation.

In patients with unfavorable CT and TT gene variants, the clearance of aerobic microflora from peritoneal exudate was 90.7%, aerobic - 64.9%, and from parietal peritoneum and fibrin layers - only 34.9% and 27.5% respectively. Due to this, the need for re-rehabilitation of the peritoneal cavity became obvious. For this purpose, at laparoscopic accesses we left special ports through which we carried out relaparoscopic remediation. At laparotomy accesses we used the programmed laparoperation for its sanitation, control of a course of inflammatory process, viability of fabrics, ability of seams and anastomoses. We have developed technologies for temporary closure of a laparotomy wound for the period between remediations, the current timing of their implementation,

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