ticks. Itching, swelling and redness of the eyelids after the first course of treatment decreased in 96.5% of patients using Spregal. If at primary eyelash microscopy in the microscope slide were revealed 8-15 ticks in the investigated area, then after the first course of treatment with Spregal their number decreased to 1-2 in the investigated area. After re-treatment, the percentage of negative microscopic eyelash tests approached 100%. Almost similar data were obtained when combining darsonvalization with topical use of gel “Stop demodex”.

Darsonvalization of the eyelids gives a good therapeutic effect. This method involves contacting specific agents with the maximum number of parasites, even deep ones. In our opinion, the spark charge, due to the action on smooth muscle cells of meibomian and sebaceous glands, stimulates the release of their secretion together with the demodex mite, which is exposed to specific drugs previously applied to the skin.

To prevent recurrence of exacerbations of the disease, we recommend daily regular therapeutic eyelid hygiene. For this purpose it is necessary to carry out self-massage of eyelids about 1-2 minutes after a warm compress. The compress is usually performed using cotton swabs, immersed in hot water, squeezed and applied to closed eyelids for 1-2 minutes. Thermal procedures help to improve local metabolic processes and drain the excretory ducts of the meibomian glands.

Self-massage is performed after applying an indifferent eye gel to the eyelash growth area, which helps to clean the surface of the eyelids from toxic agents, scales and crusts.

Our proposed new combined method of treatment of demodicosis blepharitis by sequential application of specific drugs Spregal or Stop Demodex gel on the skin of the eyelids and subsequent darsonvalization of the eyelids is an easy-to-use, affordable and effective way to treat demodicosis.

Daily observance of therapeutic eyelid hygiene (self-massage with a cleansing gel after warm compresses) can significantly reduce the likelihood of exacerbation of demodicosis blepharoconjunctivitis.

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POLYMORPHISM N34S OF THE SPINK1 GENE IN PATIENTS WITH ACUTE PANCREATITIS

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The course of acute pancreatitis is stipulated by one and the same factor. An important role is played by genetically determined defence mechanisms aimed at preventing an intrapancreatic activation of enzymes. One of such basic mechanisms is the neutralizing effect of the secretory pancreatic trypsin inhibitor (the serine protease inhibitor of Kazal’s type I - SPINK1).

The research involved 37 people with different forms of acute pancreatitis. Among them there were 25 men (67.6%) and 12 women (34.2%). An average age of the patients was 48 ± 14,4 years. The patients were divided into 2 groups. The first group included 17 patients with acute edematous pancreatitis. The second group comprised 20 patients with acute necrotizing pancreatitis.

The length of the amplificate of N34S polymorphism of the SPINK 1 gene consisted of 320 pairs of nucleotides (pn). In the presence of the 3rd exon of the nucleotide sequence of the mentioned gene of adenine in the 34th codon, the amplification splits by PstI restrictase into fragments, measuring 320 and 286 pn. In case of transversion A - G the site for PstI restriction was lost.

The presence of the favourable “wild - type” N - allele (“wild - type”, Wt) was detected in the majority of the subjects – in 73,0% cases (27). The pathological "mutant" S – variant was identified in 27,0% of people (10). Also, there were 45.9% of the cases (17) of homozygous carriers of the "wild" NN - genotype (N34), NS - heterozygotes (N34S) - 51,4 % of the cases (19). One patient (2,7%) was a homozygous carrier of the mutant S - allele (SS - genotype, 34S). A distribution of the genotypes according to the polymorphic N34S variant of the SPINK1 gene among the examinees corresponded to expected Hardy – Weinberg’s equilibrium ($p > 0,05$).

On distributing all the patients according to the etiological agent it was found out that the frequency of the NN - and NS - genotypes in patients with biliary pancreatitis involved 52,6% (10)

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,