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causes of such a severe clinical course of acute pancreatitis in a specific case was its hereditary character.

Conclusions. Thus, the frequency of the NN - and NS - genotypes of the SPINK1 gene in the patients examined by us, did not differ significantly from the patients with various forms of acute pancreatitis. The carriage of the unfavorable 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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ACUTE PERITONITIS AS AN URGENT PROBLEM OF MODERN SURGERY

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Introduction. Over the past decades, in connection with the active introduction of new technologies, methods of diagnosis and treatment of acute peritonitis (AP), in particular, the improvement of the technique of surgical interventions, the expansion of the possibilities of drug therapy, as well as the development of complex measures before and during surgical intervention and in the postoperative period, there is a positive trend in the results of the treatment of this pathology. However, the mortality due to AP remains high, varying, depending on the form and prevalence, from 16 to 80% and has no significant downward trend. One of the reasons for the high mortality rate is the lack of in-depth knowledge of the pathogenesis of AP in connection with the incomplete elucidation of the mechanisms of the initiation of the inflammatory process, as well as the factors supporting it, contribute to its progression and spread throughout the peritoneal cavity. This primarily concerns cytokines, especially interleukin 1β (IL-1β), which plays an important role in the regulation of various inflammatory mechanisms. The study of these factors, in our opinion, will make it possible not only to diagnose the presence of peritonitis reliably, but also to predict the nature of its course.

The aim of the study. Study of factors that make it possible to predict and diagnose the course of acute peritonitis

Materials and methods. A comprehensive examination of 115 patients admitted to the hospital with the signs of diffuse peritonitis was conducted. The diagnosis was confirmed surgically. All the patients underwent examination of the variants of IL1β -511C/T gene polymorphism. The material for molecular-genetic examination was DNA isolated from the lymphocytes of the peripheral venous blood of patients by means of the set of reagents «DNA-sorb-B». Polymerase chain reaction (PCR) was conducted using TaqDNA-polymerase and specific primers. The alleles were discriminated by means of specific endonucleases of AVAI and AVAII restriction («Fermentas», Lithuania) in hydrolysis reaction. PCR restriction products were divided by means of electrophoresis in 2% agarous gel with tris borate buffer (TBB) concentrated with ethidium bromide for 30-45 minutes: “mutant” AVAII-resistance Tallele was divided into “wild” C-allele [1]. The fragments were visualized by means of transilluminator with the molecular mass marker available 100-1000 bp. The level of cytokines in the blood serum was evaluated by means of immunoenzyme method on the analyzer STAT-Fax Plus-303 (USA); test systems DIACLON were used (France), DRG (Germany).

Results. It is known that the activity of cytokines is genetically determined. Different versions of the modification of the IL-1β gene (-511C/T), which encodes its synthesis, lead to differences in the nature of the inflammatory process, changes in the parameters of nonspecific resistance and reactivity, which can contribute to the cascade progression of the mechanisms of damage to organs and structures, and disruption of homeostasis. 115 patients undergoing inpatient treatment of various forms of acute peritonitis, which complicated the course of various acute surgical diseases, were studied, and the IL-1β level and the IL-1β -511C/T gene polymorphism were determined.

The conducted studies show that an important mechanism of the development and progression of the inflammatory process in the peritoneal cavity is the excessive activity of IL-1β, the concentration of which in the plasma of patients increases in proportion to the spread of the inflammatory process, it is the highest in CT and TT variants of the genotype, and in the SS variant - the lowest.

Conclusion. Thus, to predict the nature of the course of acute peritonitis, the progression of the inflammatory process in the peritoneal cavity in patients, it is advisable to determine variants of the IL-1β gene (-511C/T): with its CT-, TT- variants, an unfavorable course of peritonitis with the spread of process in the peritoneal cavity and to apply preventive comprehensive prevention of the occurrence of complications.

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BIOMARKERS OF INFLAMMATION IN DIABETIC RETINOPATHY MANAGEMENT

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Introduction. An inflammation is intensively involved in the development of diabetic retinopathy (DR) and its complications. The inflammatory process induces a complex cascade of biological, molecular and cellular signals that alter the physiological responses of the affected eye tissues. Some of inflammatory stimulus (oxygen radicals, diabetes, and infections) may disrupt the natural balance of the eye tissues, thus producing an “inflamed” phenotype. As the result of these processes there is increase of inflammatory cytokines expression which contribute to the onset of different eye diseases. To date, the molecular mechanisms that determine the development of ocular pathologies are not fully clarified and there is no therapy capable of preventing eye damage for people with diabetes. Understanding the cellular and molecular mechanisms that lead to eye damage could be useful for the management of diabetic retinopathy.

The aim. To evaluate the influence of biomarkers of Inflammation on diabetic retinopathy.

Material and methods. Market available biomarkers of Inflammation on diabetic retinopathy management were used.

Results. The evaluation of pathphysiological mechanisms in dabetic retinopathy found that early stages are characterized by histopathological changes which include loss of pericytes, basement membrane thickening, haemodynamic alterations leading to reduced vascular integrity. The later stages of diabetic retinopathy are characterized by complications, which include visual impairment, primarily due to macular edema and proliferative diabetic retinopathy. Also the severity of retinopathy was associated with poorer metabolic control, demonstrated by elevated HbA1c. Diabetic complications accompany the accumulation of advanced glycation end products in diabetic tissues. Increased accumulation of these products has been reported in epiretinal membranes by the use of immunohistochemical technique. Binding of advanced glycation end products to high-affinity receptor in pericytes exerts selective toxicity resulting in their death. Vascular endothelial growth factor exert important role of intraocular neovascularization due to ischemic retinopathy

Conclusions. Early stages of diabetic retinopathy are characterized by histopathological changes which include loss of pericytes, basement membrane thickening, haemodynamic alterations leading to reduced vascular integrity. The later stages of diabetic reinopathy are characterized by complications, which include visual impairment, primarily due to macular edema and proliferative diabetic retinopathy. Binding of advanced glycation end products to high-affinity receptor in pericytes exerts selective toxicity resulting in their death.