solutions as methods for prevention of posttraumatic postoperative complications and controlled them by using a efficacy of phagocytosis and efficiency of elimination of immune complexes.

Therefore, in accordance to liver trauma it is important to determine disorders of immune responsiveness and especially in case of complications.

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ESTIMATION OF SEVERITY OF THE EARLY PERIOD OF TRAUMATIC DISEASE IN VICTIMS WITH LIVER INJURY

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In providing surgical care to victims with abdominal trauma, especially the old and elderly, the most important is to assess the severity of damage and the objectification of the general condition of the patient. Liver injury remains a complicated and unsolved problem due to the peculiarity of its location.

The existing systems to evaluate severity of condition of the victims are not always successfully used in practice, due to the specific emergency care in general surgical wards, and these systems are not adapted to the elderly. Therefore, we conducted a comparative analysis of the previously suggested scales to evaluate patients' condition, determining the most objective ones and their modification considering the requirements occurring during treatment. 20 patients with trauma of the liver were involved in the study. The average age of patients was 48.

In accordance with the criteria determined by the status of the injured on the basis of the numerical system we determine the likelihood of lethal outcome (Px) by the following formula:

Px = eAW /(1 + eAW), where

AW – APACHE II x 0,146 + Wi + W2 + W3; W1–-3,517 (nonspecific coeficient); W2–+0,603 (coeficient for urgent surgery); W3 = diagnostic coeficient for emergency conditions; W3 = +0,503 for diseases of the gastrointestinal tract; W3 – -0,203 for intra abdominal infections.

Considering age and availability of chronic diseases the total score is determined multiplied by the coefficient appropriate for the category of multiple organ injury. According to the score detected among the victims we have differentiated risk groups for development of lethal outcome. The low risk (less than 20 points) was diagnosed in 6 patients in the group of comparison (retrospectively) and in 4 patients of the main group (in the process of treatment). Moderate risk (from 20 to 25 points) was diagnosed in 7 victims of the comparison group and 6 from the main one. High risk (from 30 to 35 points) was diagnosed in 9 patients from the comparison group and 3 of the main group. Extremely high risk (from 35 to 40 points) was diagnosed in 2 victims from the comparison group and 1 – from the main group.

Surgery according to traditional methods including timely operative treatment, adequate therapeutic measures and traditional post-operative management was essential to be performed for the victims with a low risk of lethal outcome.

Maksymyuk V.V.

A CLINICAL COURSE FEATURES OF ACUTE NECROTIZING PANCREATITIS IN PATIENTS WITH POLYMORPHISM OF R122N GENE OF CATIONIC TRYPSINOGEN (PRSS1)

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According to the aim we have learnt distribution of R122H- polymorphism gene PRSS1 and N34S-polymorphism gene SPINK1 among the inhabitants of Chernivtsi region suffering from acute pancreatitis. Associative relations of the carriers of different genotype with its aetiology, complications of clinical courses, morphological forms and its complications were examined. One of the most important genetically determined mechanisms to prevent intraacinous activation of trypsin is neutralized action of cationic trypsinogen. The gene that codes for the synthesis cationic trypsinogen (PRSS1), located on chromosome 7 (7q35). It is known that the long arm of chromosome may meet autosomal dominant mutation - R122N. However, connection between the R122N polymorphism of the PRSS1 gene and the nature of a clinical course of acute pancreatitis remains almost not studied.

Background and Aims: A clinical course feature of acute pancreatitis in patients with R122N polymorphism of PRSS1 gene was examined.

In a study participated 88 persons with various forms of acute destructive pancreatitis. Among them, 53 (60.2%) men and 35 (38.8%) women. The average age of patients was 45 ± 17,1 years. Patients genotype was partitioned into two groups: favorable R122R-genotype (control group), unfavorable R122H- and N122N-genotypes (experimental group). Genetic analysis was performed by setting the polymerase chain reaction.Statistical dependence between the values for normally distributed samplings were checked by way of determining criterion $\chi 2$ according to Pearson and criterion of Fisher. Found that in acute pancreatitis patients with unfavorable R122H and H122H-genotypes polymorphism R122H of PRSS1 gene, develop extensive infected pancreatic necrosis occurs significantly more often than patients with a favorable RR-genotype. This adversely affects the final results of treatment of such

persons. So, the obtained results allow to assess the presence of abnormal H-allele polymorphism R122H of the PRSS1 gene, as a prognostically marker of unfavorable clinical course of acute pancreatitis.

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GENETIC PROGNOSTIC MARKERS OF SEVERE ACUTE PANCREATITIS AND DEVELOPMENT OF ITS COMLICATIONS

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The character of development of acute pancreatitis (AP) and its complications depends on the ability of the mechanisms of antienzymatic protection, which realization is considerably determined by genetic factors. However, among many factors promoting the development and progressing of the disease genetically determined predictors are the least investigated. According to results of modern genetic researches a number of mutations are found which are considered as susceptibility factors to pancreatitis. A special attention among the latter ones is paid to gene transversion of cationic trypsinogen (PRSS1) and pancreatitis inhibitor of trypsin (SPINK1). They are accompanied by disorders of genetically determined mechanisms of intracellular trypsin inactivation. Although the influence of these hereditary factors on the clinical development of acute pancreatitis and its complications still remains practically uninvestigated.

According to the aim we have learnt distribution of R122H- polymorphism gene PRSS1 and N34S-polymorphism gene SPINK1 among the inhabitants of Chernivtsi region suffering from acute pancreatitis. Associative relations of the carriers of different genotype with its etiology, complications of clinical courses, morphological forms and its complications were examined.

88 persons were involved into the study including 53 (60,23%) men and 35 (39,77%) women with different forms of acute pancreatitis. The distributions of R122H-polymorphism gene PRSS1 and N34S-polymorphism gene SPINK1 were determined.

Studying distributions of genotypes R122H-polymorphism gene it was found that acute pancreatitis patients are often carriers of the R-allele (RR- and RH-genotypes 27,27% and 64,77%, respectively) in case of less quantity of pathological HH-homozygote (7,96% persons). As the result the quantity of heterozygote carriers of mutational RH-genotype was 64,77% (57 persons). The number of RR- and HH-homozygote prevailed reliably – 27,27% (24) and 7,96% (7) persons respectively ($p<0,05$).

Studying the distribution of genotype N345-polymorphism gene SPINK1 a lot of patients with acute pancreatitis demonstrated availability of favourable "wild" N-allele («wild-type» (D. Whitcomb, 2013), Wt) – 69,32% (61) persons, while the pathological "mutant" S-variant was identified in 30,68% (27) persons. Homozygote carriers of «wild» NN-genotype (N34) were 42,05% (37) persons, NS-heterozygote (N34S) – 54,55% (48) persons, but homozygote carriers of «mutant» S-allele (SS-genotype, 34S) - 3,40% (3) persons.

Genotype distribution by polymorphous variants of R122H gene PRSS1 and N24S gene SPINK1 among the examined patients with acute pancreatitis corresponded to Hardy-Weinberg equilibrium.

Performed genetic investigations formed the basis to study peculiarities of clinical development of acute pancreatitis in persons with different RH- and NS-genotypes. The examined patients with acute pancreatitis were found to have favourable R-allele of R122H-polymorphism gene of cationic trypsinogen – PRSS1 (RR- and RH-genotype – 27,27% and 64,77% persons respectively) with less quantity of pathological HH- homozygote (7,96% persons). In case patients have unfavourable H-allele of R122H-polymorphism gene PRSS1 the probability of severe clinical development of acute pancreatitis with diffuse pancreonecrosis is reliably higher than in patients with favourable R122R-genotype (55,0% persons against 17,9% persons, $\chi^2 = 9,274$, $p<0,01$). Such patients have more often pancreatogenic abscess of the peritoneal cavity – (48,3% against 21,4% persons, $\chi^2 = 4,250$, $p<0,05$) and diffuse (general) peritonitis (38,3% against 7,15% persons, $\chi^2 = 7,663$, $p<0,01$). These findings enable us to determine mutation R122H- and H122H-genotype as candidate hereditary factors of severe acute pancreatitis and its complications.

Patients with acute pancreatitis were found to be carriers of favourable N-allele of N34S-polymorphism gene of secretory pancreatitis inhibitor of trypsin more often – SPINK1 (NN-genotype – 42,05% and NS-genotype – 54,55%), with less quantity of pathological SS-homozygote (3,40%). In case patients have S34S-polymorphism of gene SPINK1 the probability of severe acute pancreatitis with diffuse pancreonecrosis development is reliably higher than in patients with favourable N34N- and N34S-genotype (100% against 21,7% persons, $\chi^2 = 5,741$, $p<0,05$). It enables to determine mutation S34S-genotype as a genetic predictor of unfavourable development of acute pancreatitis.

Identified peculiarities of the clinical course of acute pancreatitis in persons with genetically determined disorders of intracellular trypsin inactivation are the backgrounds for elaboration of two new ways to prognosticate its course and development of complications.

The main point of the first suggested method is the following: R122H-polymorphism of gene PRSS1 should be determined in patients with acute pancreatitis. In case mutational R122H- or H122H-genotypes are found severe clinical course of the disease with susceptibility to the development of diffuse necrotizing damage of the pancreas and fast formation of purulent necrotic complications can be prognosticated (patent on useful model № 68121 UA).

Another method is to investigate N34S-polymorphism of gene SPINK1. In case two mutation S-alleles are determined severe clinical course of acute pancreatitis with a high risk of development of spread infectious pancreonecrosis and early occurrence of its purulent necrosis complications can be predicted (patent on useful model № 66811 UA).