



Table

Dynamic growth and nonspecific resistance of blood and circulating immune complexes in patients with liver injury

Indices	Control group n=15	Postoperative Day				
		1-st n=40	2-nd n=40	5-th n=40	7-th n=40	14-th n=40
Phagocytic index. %	65.14 ±3.48	54.50±4.22 p>0.05	63.65±3.17 p>0.7	64.88±2.86 p>0.9	42.00±2.13 p<0.001	24.08±1.14 p<0.001
Phagocytic number	3.26±0.12	3.00±0.26 p>0.3	2.95±0.23 p>0.2	2.84±0.21 p>0.07	2.42±0.20 p<0.001	1.96±0.10 p<0.001
Index completion of phagocytosis	1.17±0.06	1.59±0.20 p<0.05	1.44±0.20 p>0.1	1.46±0.19 p>0.1	1.29±0.17 p>0.4	0.65±0.17 p<0.01
Circulating immune complexes, units	74.98±2.59	53.60±4.76 p<0.001	93.76±5.57 p<0.01	76.04±6.95 p>0.8	221.60±8.26 p<0.001	189.51±6.81 p<0.001

Thus, as a result of the analysis was performed between the reduction of nonspecific protection and complication after traumatic injury of the liver. Joining postoperative multi organ failure syndrome increases to 35.72% incidence of complicated course of liver trauma. The proposed algorithm allows to improve the prediction of postoperative course and to detect preclinical stage of formation of complicated course.

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POLYMORPHISM N34S OF THE SPINK1 GENE IN UKRAINIAN PATIENTS WITH DIFFERENT FORMS OF ACUTE PANCREATITIS

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The course of acute pancreatitis, whose onset is stipulated by one and the same factor, may be of quite an opposite nature in different patients - from the edematous form to pancreatonecrosis. An important role, hereat, is played by genetically determined defence mechanisms aimed at preventing an intrapancreatic activation of enzymes.

The research involved 37 persons with different forms of acute pancreatitis. Among them: 25 (67.6%) men and 12 (34.2%) women. The mean age of the patients made up 48 ± 14.4 years. The patients were divided into 2 groups. The first group was made up of 17 patients with acute edematous pancreatitis. The second group comprised 20 patients with acute destructive pancreatitis.

The presence of the favourable "wild - type" N - allele ("wild - type", Wt) - 73,0% (27) of the persons was detected in the majority of the subjects. The pathological "mutant" S - variant was identified in 27,0% (10) of the persons. Hereat, there were 45.9% (17) of the cases of homozygous carriers of the "wild" NN - genotype (N34), NS - heterozygotes (N34S) - 51,4% (19) of the cases. One (2,7%) patient was a homozygous carrier of the mutant S - allele (SS - genotype, 34S) (Fig. 1 - 2). 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 made up 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 made up 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). The homozygous mutation SS - genotype was detected in one person of the said group. It should be noted at that the initiation of the disease was associated with the nonbiliary factor in a female patient with the SS - genotype. The course of the disease was characterized by particular "aggressiveness" with the development of acute suppurative subtotal pancreatonecrosis which became complicated by the formation of abscesses of the omental bursa and the right subdiaphragmatic space, retroperitoneal phlegmon, external pancreatic and duodenal fistulae, left - side exudative pleuresy and toxicobacterial shock. The length of the hospital stay of the patient made up 118 bed days 10 step - by - step surgical interferences, having been performed during this period.

Thus, the frequency of the NN - and NS - genotypes of the SPINK1 gene in the patients examined by us, did not differ significantly in 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.