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LIVER MARKERS IN PATIENTS WITH CIRRHOSIS OF THE LIVER OF VARIOUS ETIOLOGIES

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Abstract

According to literature data, by the end of 2017, more than 1.32 million people died from cirrhosis of the liver. This accounted for 2.4% of the total mortality rate in the population. Cirrhosis of the liver, occupying one of the main places among the diseases of mankind, is one of the ten chronic diseases that increase the causes of disability and death. The aim of the study was to study the etiological, epidemiological and clinical diagnostic features of cirrhosis of the liver, to improve its prevention measures in rural residents of the Andijan region. To do this, 89 patients with cirrhosis of the liver were studied.

The paper presents the results of a study, biochemical and enzyme immunoassays to study the gastrointestinal tract in 41 patients diagnosed with cirrhosis of the liver in viral etiology, 39 patients with viral etiology, 2 patients of unclear etiology and 7 patients with alcoholic etiology according to the Child-Pugh classification. The effect of gastrointestinal hormones on the course of liver cirrhosis of viral etiology is shown.

Keywords: cirrhosis of the liver, viral hepatitis, alcoholic cirrhosis of the liver.

TURLI ETIOLOGIYALI JIGAR SIRROZI BILAN HASTALANGAN BEMORLARDA JIGAR MARKERLARI

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Annotatsiya

Adabiyotlarda keltirilgan ma'lumotlarga ko'ra, 2017 yil oxiriga kelib, 1,32 milliondan ortiq odam jigar sirrozidan vafot etgan. Bu aholi umumiy o'lim darajasining 2,4 foizini tashkil etdi. Inson kasalliklari orasida asosiy o'rinlardan birini egallagan jigar sirrozi nogironlik va o'lim sabablarini oshiradigan surunkali kasalliklarning o'ntaligiga kiradi. Tadqiqot maqsadi Andijon viloyati aholisida jigar sirrozining etiologik, epidemiologik va klinik diagnostik xususiyatlarini o‘rganish, uning oldini olish chora-tadbirlarini takomillashtirishdan iborat edi. Shu maqsadda 89 nafar jigar sirrozi bilan og‘rigan bemor o‘rganildi.

Maqolada virusli etiologiyali jigar sirrozi tashxisi qo'yilgan virus B etiologiyali 41 bemorda, virus C etiologiyali 39 bemorda, noaniq etiologiyali 2 bemorda va alkogol etiologiyali 7 bemorda oshqozon-ichak traktini o'rganish uchun tadqiqot natijalari, biokimyoviy va immunoenzim tahlillari

keltirilgan. Chayld-Pyu tasnifi. Gastrointestinal gormonlarning virusli etiologiyali jigar sirrozi jarayoniga ta'siri ko'rsatilgan.

Kalit so'zlar: jigar sirrozi, virusli gepatit, alkogolli jigar sirrozi.

ПЕЧЕНОЧНЫЕ МАРКЕРЫ У БОЛЬНЫХ ЦИРРОЗОМ ПЕЧЕНИ РАЗЛИЧНОЙ ЭТИОЛОГИИ

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Аннотация

По литературным данным, по итогам 2017 года, от цирроза печени погибло более 1,32 миллиона человек. Это составило 2,4% от общего показателя смертности в популяции. Цирроз печени, занимая одно из главных мест среди болезней человечества, входит в десятку хронических заболеваний, увеличивающих причины потери трудоспособности и смерти. Целью исследования явилось изучение этиологических, эпидемиологических и клинико-диагностических особенностей цирроза печени, совершенствование мер его профилактики у сельских жителей Андижанской области. Для этого были изучены больные 89 больных циррозом печени.

В работе представлены результаты исследования, биохимические и иммуноферментные анализы для изучения желудочно-кишечного тракта у 41 больного с диагнозом цирроз печени В вирусной этиологии, 39 больных С вирусной этиологии, 2 больных неясной этиологии и 7 больных алкогольной этиологии по классификации Чайлд-Пью. Показаны влияние желудочно-кишечных гормонов на течение цирроза печени вирусной этилогии.

Ключевые слова: цирроз печени, вирусный гепатит, алкогольный цирроз печени.

According to the results of the study, it was found that in individuals with liver cirrhosis of the B virus, high levels of serological markers Anti-HBs and Anti-HbcIgG and their optical density were observed. Anti-NVe IgG was detected in small quantities and the optical density was very low.

The value of pepsinogen 1 in liver cirrhosis in the compensation stage was below the lower limit of the norm. In contrast, pepsinogen-2 was within the normal range, but in liver cirrhosis of viral etiology in the compensation stage, its value was several times higher than the norm. In patients with obvious signs of liver dysfunction in decompensated liver cirrhosis, an increase in CCK-8 and gastrin-17 was observed. It was noted that in patients with liver cirrhosis caused by virus B, in the compensation stage, the increase in CCK-8 in the blood was insignificant. It was also noted that the levels of gastrin-17 were unreliably higher than the norm, and pepsinogen-1 and pepsinogen-2 were within its limits. This indicates the absence of

significant changes in the activity of the digestive glands of the stomach in patients with liver cirrhosis in the compensation period. The obtained data are a sign of decreased functional activity of the digestive glands of the stomach in patients with liver cirrhosis caused by the B virus, and this can be regarded as a latent form of atrophic gastritis. Chronic atrophic gastritis is one of the main causes of damage to the gastric mucosa. The results we obtained give reason to say that CCK-8 is the cause of the development of these changes. Table 6 below shows the optical density values of serum HCV markers in liver cirrhosis caused by the C virus [3, 1].

Table 1

Optical density indices of serum HCV markers in liver cirrhosis caused by C-virus.

Serum markers of HCV	Compensation stage		Decompensation stage	
	%	About optical density	%	About optical density
Anti-HCV total	72.5±6.8	1.762±0.18	68.9±7.1	1.917±0.18
Anti-HCV coreIgG	83.1±7.6	1.945±0.21	79.2±8.3	1.698±0.17
Anti-HCV coreIgM	-	-	85.4±9.1	2.014±0.19
Anti-HCV NS3	-	-	74.1±7.9	0.642±0.07
Anti-HCV NS4	57.3±6.2	0.529±0.06	87.5±9.4	2.323±0.24
Anti-HCV NS5	47.5±5.1	0.683±0.07	81.3±8.5	2.142±0.22

In liver cirrhosis caused by the C virus, serological markers Anti-HCV total and Anti-HCVcoreIgG are detected in most cases. When compared with liver cirrhosis C of viral etiology, signs of increased optical density are observed in the decompensation stage. At the same time, Anti-HCV NS4 and Anti-HCV NS5 were detected relatively little, and the signs of optical density were low. In the compensation phase, serological markers Anti-HCV total, Anti-HCVcoreIgG were lower compared to the decompensation phase. In most cases, Anti-HCVcoreIgM, Anti-HCVNS4 and Anti – HCVNS5 were detected in patients. At the same time, signs of optical density and the stage of compensation of liver cirrhosis were significantly higher. In addition, as shown in the table, the Anti-HCV NS3 marker was rarely found with low optical density values [8].

When studying the characteristics of the indices of gastric digestive hydrolases, as well as cholecystokinin and gastrin-17 in patients with liver cirrhosis caused by the C-virus (table 2), liver tests, in particular, general indices, except for bilirubin, were found to be significantly elevated relative to the norm. The levels of pepsinogen-1 in the blood were significantly lower than the norm. The indices of pepsinogen-2 were noted to be slightly higher than the norm and remained within the normal range. At

the same time, despite the fact that the levels of CCK-8 and gastrin-17 in the blood were slightly higher, they slightly exceeded the upper limit of the norm.

Table 2

Blood indices in patients with liver cirrhosis of C-virus etiology.

Serum markers	Reference indicators	Stage towards compensation	Decompensation stage
Liver function tests			
AST(mmol/h*l) (norm 0.1-0.68)	0.21±0.02	0.91±0.13**	1.09±0.09**
ALT (mmol/h*l) (norm 0.1-0.68)	0.36±0.04	1.24±0.13*	2.26±0.13**
Total bilirubin (μmol/L) (norm 8.5-20.5)	13.6±1.2	29.3±5.3*	41.5±6.7**
Indirect bilirubin (μmol/l) (normal 0-5.0)	2.0±0.1	5.4±0.87*	14.2±4.27**
Blood hydrolase			
Pepsinogen-I (mcg/l) (norm 40–130)	117.4±15.3	41.9±5.3*	14.5±2.3**
Pepsinogen-II(μg/l) (norm 4–22)	12.5±1.5	19.4±2.1	10.2±1.4**
Peptides			
CCK-8 (normal 0.5–1 ng/ml)	0.72±0.08	1.06±0.11*	2.56±0.26**
Gastrin-17 norm (on an empty stomach **)** <7 pmol/l	5.6±0.45	9.1±0.82*	15.3±1.6**

***** - level of reliability in comparison with reference indicators ; ** - level of reliability in relation to the compensation stage.

All the considered indices in liver cirrhosis of viral C etiology in the decompensation stage were significantly higher than in the compensation stage. Thus, the level of pepsinogen-1 in this group of patients was below the lower limit of the norm and significantly lower compared to the compensation stage. It was noted that the indices of pepsinogen-2 were within the normal range, but several times lower than the compensation stage, while the indices of CCK-8 and gastrin-17 were significantly high compared to the compensation stage and exceeded the upper limit of the norm by 2.5 times (table 2).

Thus, in liver cirrhosis of C-virus etiology: the concentration of pepsinogen-1 in the blood serum up to 40 μg / l and at the same time a significant decrease in the secretion of hydrochloric acid indicate the development of atrophic gastritis; an

increase in gastrin-17 in the blood and a decrease in the secretion of hydrochloric acid are additional causes of the development of atrophic gastritis; an increase in CCK-8 in the blood is an indicator of liver dysfunction, which leads to a decrease in the neutralization of this peptide in the liver. In our opinion, an increase in CCK-8 in the blood is the main factor leading to the development of gastric disorders; when examining patients with liver cirrhosis of C-virus etiology, it is advisable to examine the functional state of the stomach, as an organ involved in the pathological process, namely, the determination of digestive hydrolases in the blood, as well as peptides that control these organs. This informative screening test is considered as a risk trigger for gastric bleeding [7, 5].

When studying changes in the cholecystokinin-8 and gastrin-17 indices in patients with alcoholic liver cirrhosis, it was found that seven patients with alcoholic liver cirrhosis had significantly and reliably elevated liver function indices. In addition, in this group of patients, the levels of gamma-glutamyl transferase and immunoglobulin A were significantly higher than normal. At the same time, it was observed that the pepsinogen-1 indices were significantly lower than the lower limit of normal. It was noted that the Pepsinogan-2 indices were within the normal range. The CCK-8 and gastrin-17 indices were significantly high compared to the norm and exceeded its upper limit by 2.5 times.

A higher than normal level of CCK-8 in the blood of patients with alcoholic liver cirrhosis confirms a decrease in its breakdown by the liver. A decrease in pepsinogen-1 and a significant increase in gastrin-17, which are mainly produced by glandular cells of the body and fundus of the stomach, indicate a decrease in the enzyme-secreting property of the stomach. At the same time, the concentration of serum pepsinogen-1 from 40 μg/l and the secretion of hydrochloric acid are significantly reduced, and atrophic gastritis is observed. Thus, with alcoholic liver cirrhosis, as with viral liver cirrhosis, the metabolism of short-chain peptides is disrupted. High degree of increase in CCK-8. As a result, gastric secretion decreases and atrophic gastritis develops [2].

When studying changes in blood cholecystokinin-8 and gastrin-17 levels in liver cirrhosis of unknown etiology, it is of great interest to study changes in short-chain peptide CCK-8 and long-chain peptide gastrin-17 levels in patients with liver cirrhosis not associated with alcohol or infection. Below is an analysis of two studies conducted in patients with liver cirrhosis without identifying an etiologic factor, in which the reference values were negative and liver tests were within normal limits. Pepsinogen-1 and pepsinogen-2, as well as CCK-8 and gastrin-17, were also within normal limits. In all patients diagnosed with liver cirrhosis of unknown etiology, the pepsinogen-1 level was slightly below the lower limit of normal and significantly below normal. However, pepsinogen-2 levels were within normal limits. CCK-8 and

gastrin-17 levels exceeded the upper limit of normal.

In patients with liver cirrhosis of unknown etiology, an increase in the amount of CCK-8 above the norm indicates a decrease in its neutralization in the liver. A decrease in pepsinogen-1 and a significant increase in gastrin-17, which are mainly produced by glandular cells of the body and fundus of the stomach, indicate a decrease in the enzyme-secreting activity of the stomach. A decrease in the concentration of pepsinogen-1 in the blood serum to less than 40 μg/l is observed with a significant decrease in the secretion of hydrochloric acid and the development of atrophic gastritis. But regardless of the etiological factor, in liver cirrhosis, its neutralization by the liver is impaired, and the concentration of CCK-8 in the blood increases. As a result, there is a decrease in gastric secretion and the development of atrophic gastritis [4,1].

The obtained data show that the effect of CCK-8 on the functional activity of the stomach, atrophic gastritis and thinning of the gastric mucosa can be taken as a trigger for an increased risk of gastric bleeding. As a result, it became possible to more accurately diagnose atrophic gastritis, as well as promptly assess the risk of bleeding from the upper gastrointestinal tract in liver cirrhosis of unknown etiology and prescribe pathogenetic treatment.

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