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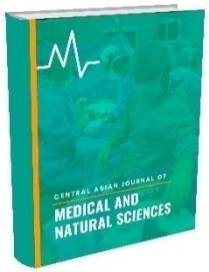

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Characteristics Of Liver Marker Indicators Cholecystokinin-8 And Gastrin-17 In Patients With V Virus Liver Cirrhosis

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Annotation: According to predictions of the World Health Organization (WHO), despite the advances in modern therapy and the development of liver transplantation in recent years, the death rate from liver diseases is expected to double in the next 10-20 years. According to this authoritative organization, approximately 200 million patients suffer from chronic liver diseases.

In our country, more than 50,000 people die from cirrhosis of the liver every year, and this is 3 times higher than the death rate.

The purpose of the study: to study the etiological, epidemiological, clinical and diagnostic characteristics of liver cirrhosis among rural residents of Andijan region, to improve the preventive measures of liver cirrhosis. In order to implement the goals and tasks set before us, the prevalence of chronic liver and gastrointestinal system diseases among the rural population of Markhamat district of Andijan region was studied retrospectively based on the data of 2017-2022.

Keywords: liver cirrhosis, prophylaxis, short-chain peptides, V virus, stomach.

Among the rural residents of Andijan region, a number of works were carried out to study the etiological, epidemiological, clinical and diagnostic features of liver cirrhosis, to improve preventive measures of liver cirrhosis.

In order to implement the goals and tasks set before us, the prevalence of chronic liver and gastrointestinal system diseases among the rural population of Markhamat district of Andijan region was studied retrospectively based on the data of 2017-2022. For this purpose, 6,952 residents aged 18 to 70 belonging to the multidisciplinary hospital and polyclinic of Markhamat district were examined based on a random representative sample, and 694 (10%) were selected. 203 (29.3%) of them were men and 491 (70.7%) were women. Liver cirrhosis was diagnosed in 89 (12.8%) examinees compared to the general population. 80 of them had a viral disease, 7 had an alcoholic disease, and 2 had an unknown etiology.

Results of the study conducted biochemical and immunoferment analysis to study the gastrointestinal system in 41 patients diagnosed with liver cirrhosis with V virus etiology under the classification of child Pyu, 39 with S virus etiology, 2 with unknown and 7 with alcohol etiology.

As a result, the most serological markers of Anti-HBs and Anti-HBs IgG were detected in persons with B virus liver cirrhosis in the compensation stage, and optical density had high signs in them. Small signs of anti-HBe IgG optical density were detected very little (Table 4.1). At the same time, liver tests in these individuals 4.1. it looked like the table.

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1– table

B changes in the taxa of patients with viral liver cirrhosis.

Serum markers	**Healthy individuals**	**Compensation stage**	**Decompensation stage**
Antibodies to the HBV virus			
	%	%	%
HBs- antigen	-	-	58±6,5
HBe – antigen	-	-	79±8,1
Anti-HBs	-	89±9,1	-
Anti-HBe IgG	-	49±5,7	-
Anti-HBc IgG	-	73±6,9	87±9,1
Anti-HBcIgM	-	-	81±9,1
liver tests			
AST(mkmol/c*l) (norm 0,1-0,68)	0,21±0,02	0,41±0,03*	0,74±0,08**
AlT(mkmol/c*l) (norm 0,1-0,68)	0,36±0,04	0,52±0,06	0,93±0,11**
Common bilirubin (mkmol/l) (norm 8,5-20,5)	13,6±1,2	22,9±1,8*	47,9±9,5**

Indirect bilirubin(mkmol/l) (norm 0-5. 0)	2,0±0,1	5,7±0, 5*	26,0±2, 7**
Hydrolases of blood			
Pepsinogen-1(mcg/l) (Meer40–130)	117,4±15,3	75,8±8,6*	24,7±5,3**
Pepsinogen-2 (mcg/l) (Meer4–22)	12,5±1,5	15,6±1,8	17,3±2,1
Peptides			
XCK-8 meer 0.5–1 ng/ml	0,72±0,08	1,23±0,11*	2,37±0,25**
Gastrin-17 norm (morning) < 7 pmol / l	5,6±0,45	9,1±0,82*	21,4±2,3**

* - depending on the circumstances, there is a big difference between cattalicular, **- stable compensation is relatively different from cattalicular

In this, pepsinogen 1 rates fell below the lower limit of the norm in the compensation stage at 75.8±8.6 hepatic cirrhosis, while it was observed to decrease to 24.7±5.3 in the decompensation stage, 3-4 times lower than the norm. Simultaneously, pepsinogen-2 was at the limit of norm in the ikala stage. During the decompensation phase of hepatic cirrhosis, patients experienced an increase in XCK-8 as well as gastrin-17 in the blood due to the apparent impairment of liver function. Patients with viral liver cirrhosis B in the compensation phase were noted to have an unsuspecting increase in their blood from XCK-8 norm 1.23±0.11 while in the decompensation phase it was observed to have a 2.5-fold increase in Meer 2.37±0.25.

Gastrin-17 was observed to have an unreliable increase of 9.1±0.82 in the compensation phase. This indicates that patients with cirrhosis of the liver during the period of compensation do not have significant changes in the functioning of the gastric glands.

At the stage of decompensation of viral liver cirrhosis V, it was noted that in the blood of patients XCK-8, a clear increase in the norm. This testifies to the reduced ability of the liver to harm XCK-8 and the functional activity of the stomach, as well as the presence of atrophic gastritis. The fact that pepsinogen-1, which is mainly produced by the gland cells in the body and bottom of the stomach, decreases from the norm and gastrin-17 is significantly increased from the norm indicates a decrease in the enzyme-secreting foliation of the stomach. In this case, the reduction of pepsinogen-1 concentration in whey to signs less than 40 mcg/l is observed in a significant decrease in hydrochloric acid excretion and the development of atrophic gastritis [2, P.1204-1208]. In gland cells in all sections of the stomach.

These results show that atrophic gastritis in patients during the decompensation phase of viral hepatic cirrhosis shows extra-hepatic manifestations, but the mechanisms of these changes are not covered in the literature.

The detected changes B confirm a decrease in the functional activity of the digestive glands of the stomach in viral liver cirrhosis. This may be a sign of atrophic gastritis.

In our opinion, this is due to the metabolism or physiological absorption by the liver of low-molecular or short-chain peptides, including XCK-8. This was shown by us in experiments previously carried out in dogs in Adti's Research Laboratory [9; - S. 252-253; 10th S. 24-26] and has also been confirmed by other researchers [2; 1204-1208-b, 3; 1-3-b.].

In our opinion, an increase in short-chain peptides, which are excreted in the body after the ingestion of food into the stomach, is observed when liver function is disrupted. This can be confirmed by the fact that XCK-8 is metabolized in significantly healthier individuals and to a lesser extent in hepatic cirrhosis at the compensation stage. As a result, XCK-8 is not considered to be its main form in the plasma of regulatory subjects, but it increases significantly in patients with liver cirrhosis [3 - P. 1-3.; 6; - R. 19-26; 8;– P. 880-884]. XCK-8 Type A may be important in inhibiting the stimulation of gastric acid release due to activation of XCK receptors. It also exerts control over the release of gastric acid, blood plasma gastrin, and somatostatin [1; – P. 3924-3933;. 4; - P. 1038-1040.; 10; - S. 24-26].

Thus, it can be assumed that in moderation, XCK-8, which is highly absorbed by the liver, during the decompensation stage of hepatic cirrhosis, this process is disrupted and its amount in the blood increases.This is due to the fact that at the same time a decrease in gastric secretion and the development of atrophic gastritis is noted.

The data obtained indicate that chronic atrophic gastritis, the development of Portal hypertonic gastropathy, at the stage of decompensation of hepatic cirrhosis occupies a key place in increasing the factor of gastric bleeding.Under the influence of XCK -8, gastric emptying is left behind, the remnants of food acquire a tendency to damage the changed mucous membrane even without it, and this increases the risk of gastric bleeding. In our opinion, XCK - 8 is the main factor that is responsible for the development of the indicated disorders.

Our observation and analysis noted that atrophic gastritis in patients with viral liver cirrhosis V is manifested in addition to the liver, but the mechanisms of these changes are not covered in detail in the literature.

Thus, a sign of a decrease in the functional activity of the digestive glands of the stomach at the stage of decompensation of hepatic cirrhosis, its manifestation in the form of atrophic gastritis, and it can be considered one of the main causes of complications in the digestive system. The results we are able to obtain are related to the metabolism or physiological absorption by the liver of low-molecule or short-chain peptides, including XCK-8.As we have already mentioned, this is in experiments previously carried out in dogs in Adti's Research Laboratory [9; - S. 252-253] and co-authored by other researchers [2;- P.1204-1208; 3; - P. 1-3.]. XCK - 8 is of leading importance in inhibiting the stimulation of gastric acid excretion due to the activation of Type A XCK receptors, and it is also necessary to co-monitor the control of gastric acid in the release of gastrin and somatostatin in the blood plasma [1– - P. 3924-3933, 5; - P. 47-50.].

Thus, it can be assumed that in moderation, XCK-8 is highly absorbed by the liver. In viral liver cirrhosis, especially at the stage of decompensation, its absorption by the liver is impaired, and the concentration of XCK-8 in the blood increases. As a result of this, as described above, the cessation of gastric secretion and the development of atrophic gastritis are noted.

Data obtained in patients with viral liver cirrhosis B decreased functional activity of the digestive glands of the stomach/

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