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CLINICAL CHARACTERISTICS OF ALCOHOLIC LIVER DISEASE

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ABSTRACT

The features of the clinical course of alcoholic liver disease in various ethnic groups were studied in comparison with chronic alcohol-viral and viral hepatitis B.

All patients underwent clinical and laboratory examination.

It was revealed that in indigenous patients chronic alcoholic hepatitis was formed in a shorter time, as evidenced by the high frequency of their detection in the age groups up to 20 and from 20 to 29 years and was characterized by more pronounced clinical manifestations.

The main distinguishing features of alcoholic liver damage, regardless of the presence or absence of CVH infection, are hepatomegaly, the prevalence of pronounced extrahepatic manifestations, increased activity of AIAT in combination with GGT, and immunological changes.

Keywords: alcoholic liver disease, chronic alcoholic hepatitis, alcoholic viral hepatitis, chronic viral hepatitis B.

Introduction. Alcohol abuse has a complex negative effect on the human body, especially on the liver. Alcoholic liver disease (ALD) is manifested by alcoholic steatosis, acute and chronic alcoholic hepatitis, as well as alcoholic liver cirrhosis [1, 2, 4, 5, 6]. Development of ALD is also facilitated by excessive body weight and obesity, hepatopathy virus infection and immune factors [6, 7].

Purpose of the study- to study the features of the clinical course of alcoholic liver disease in different ethnic groups in comparison with chronic alcohol-viral and viral hepatitis B.

Materials and methods of research. Groups of patients with alcoholic hepatitis (84), alcoholic viral hepatitis (72), chronic viral hepatitis B (30) were examined. All patients underwent clinical and laboratory examination.

Results of the study. In 84 patients with chronic active alcoholic hepatitis (ACH), clinical syndromes and symptoms were analyzed in comparison with patients with viral-alcoholic hepatitis (AVH) and viral hepatitis B (CVH-B). Comparative characteristics of clinical manifestations of chronic hepatitis of alcoholic, alcoholic-viral and viral etiology are presented in Table 1.

Asthenic-vegetative syndrome was observed at ACH in 94,6%, at AVH in 90,3% and at CVH-B in 75,3% of patients, and with pain - 98,1%, hepatomegaly - 100%, dyspeptic disorders - 66,6% at patients of two compared groups.

Splenomegaly was observed in patients with ACH - 1.6 (1.9%), AVH - 2 (2.8%), CVH-B - 2 (6.6%). At alcoholic hepatitis, splenomegaly was determined only by ultrasound examination. Asthenic-vegetative syndrome, hepatomegaly occurred in practically all patients of the observed groups. At the same time, pain syndrome, dyspepsia disorders and systemic lesions were much more often detected in patients with ACH and AVH.

Hemorrhagic syndrome was detected in ACH, AVH and CVH-B, respectively, in 25 (30.0%), 26 (36.4%), 10 (32.9%) patients, without significant differences in groups. Jaundice was more often observed in the ACH 18 group (21.4%) than CVH-B 4 (13.3%).

Hepatic signs in the compared groups were encountered in isolated cases. Extrahepatic systemic manifestations were significantly more often detected in alcoholic and alcohol-viral liver lesions compared with viral hepatitis (13.3%) (Table 2).

Systemic lesions were detected at ACH in 78 (92.9%), AVH in 51 (70.8%) and at CVH-B in 4 (13.3%) patients. In indigenous people at ACH systemic manifestations occurred in 53 (67.9%) and AVH - in 35 (68.6%), in non- indigenous patients ACH in 29 (37.2%) and AVH - in 8 (15.6%) respectively. In indigenous patients at ACH and AVH, the frequency of systemic manifestations was much higher than in the non-indigenous.

The gastric lesion in the form of chronic gastritis was observed in 34 (40.5%), pancreatic damage - chronic pancreatitis, more often calcifying in 28 (33.3), lesions of salivary glands in the form of mumps in 21 (25.0%), kidney damage 25 (29.7%) - alcoholic nephritis or pyelonephritis, in 14 (19.4%) heart disease - alcoholic cardiomyopathy with heart failure, quite often with arrhythmia in 16 (19.0%). Lung bronchitis with a protracted clinical course was observed in 7 (8.3%) patients. The Raynaud syndrome was found in isolated cases at ACH and CVH-B. A moderate variant of the disease occurred in 82 (97.6%) patients with ACH, in 43 (59.7%) - AVH and in 17 (56.7%) CVH-B; frequent clinical symptoms were not severe hepatomegaly in patients of viral and alcoholic-viral etiology, a more pronounced increase in the liver was noted at alcoholic chronic hepatitis, together with systemic lesions, as well as pain, dyspeptic and asthenic-vegetative syndromes. A significant clinical variant of the disease was found at ACH in 2 (2.4%), AVH in 8 (11.1%) and at CVH-B in 5 (16.7%) patients (Table 3). At chronic viral hepatitis

Table 1

Comparative characteristics of clinical manifestations of CH of alcoholic, alcohol-viral and viral etiology

Clinical signs	Number of patients in groups with the presence of signs					
	ACH n – 84		AVH n – 72		HVG-B n – 30	
	%	n. persons	%	n. persons	%	%
Asthenic-vegetative	79	94.6±3.0	65	90.3±3.5	22	75.3±8.1
Dyspeptic	56	66.6±6.3	56	77.8±4.5	11	38.4±8.8
Painful	83	98.1±1.8	57	79.2±4.9	15	49.3±9.1
Hepatomegaly	84	100	71	98.6±1.4	29	97.3±3.3
Splenomegaly	2	1.9±1.8	2	2.8±1.9	2	6.6±4.6
Hemorrhagic	25	30.0±6.1	26	36.4±5.7	10	32.9±8.6
Jaundice	18	21.4±5.5	13	18.0±4.5	4	13.3±6.2
Hepatic signs:	14	16.6±4.9	7	9.7±3.5	1	3.3±3.3
a) Vascular asterisks	5	5.9±3.4	3	4.2±2.4	1	3.3±3.3
b) palmar erythema	9	10.7±3.8	4	5.5±2.7	-	-
Systemic damage in total:	78	92.9±3.4	51	70.8±5.4	4	13.3±6.2
Clinical variants of CH:						
moderately severe	82	97.6±1.8	68	94.7±2.7	25	82.2±6.8
severe	2.0	2.4±1.8	4	5.3±2.7	5	17.8±6.8

Table 2

Comparative characteristics of systemic lesions in patients with chronic hepatitis of alcoholic, alcoholic-viral and viral etiology

Clinical signs	Number of patients with the presence of signs in groups					
	ACH n-84		AVH n-72		HVG- B n-30	
	n	%	n	%	n	%
Systemic damage in total:	78	92.9±3.4	51	70.8±5.4	4	13.3±6.2
Lymphadenopathy	2	1.8±1.8	1	1.4±1.4	3	10.0±5.5
Fever	12	14.3±4.4	3	4.2±2.4	1	3.3±3.3
Articular syndrome	22	26±5.1	18	25.0±5.1	3	10.0±5.5
Skin syndrome	7	8.3±3.0	7	9.7±3.5	1	3.3±3.3
Renal damage	25	29.7±5.8	9	12.5±3.9	1	3.3±3.3
Heart Attack	16	19.0±4.9	14	19.4±4.7	1	3.3±3.3
Lung infection	7	8.3±3.0	2	2.8±1.9	1	3.3±3.3
Stomach lesion	34	40.5±6.7	25	34.7±5.6	-	
Pancreas lesion	28	33.3±6.5	29	40.3±5.8	-	
Lesion of the salivary glands	21	25.0±5.6	15	20.8±4.8	1	3.3±3.3
Raynaud's syndrome	3	3.6±1.8	-		1	3.3±3.3
Nodular periarteritis	-		-		1	3.3±3.3

B in the clinical picture, systemic lesions with unexpressed hepatomegaly manifested themselves more vividly.

Thus, in the majority of patients with chronic alcoholic hepatitis, systemic lesions were observed at moderately severe variants, and in patients with viral hepatitis - severe variants of the disease.

At studying laboratory indicators (Table 4) at ACH, AVH and viral hepatitis, it turned out that 100% of patients showed hyperaminotransferase, more often than 5 and 10 times higher than normal, but higher than 10 times only in isolated cases. An increase in the level of alkaline phosphatase was noted in 26 (31.0%) in patients with alcoholic chronic hepatitis (ACH), with AVH and CVH-B significantly less. Hypoalbuminemia was observed in 19 (22.6%) patients with ACH, in 9 (12.2%) AVH and in 2 (6.6%) CVH-B.

Hypergammaglobulinemia was noted in a small number of patients in all 3 groups. A significant increase in gamma-glutamyltranspeptidase (GGTP) was observed in patients with ACH, statistically significantly differed from those of the CVH-B group, and an increase in GGTP was noted in patients with AVH. Hypocholesterolemia was observed only in patients with chronic hepatitis B 16 (54%). Hypercholesterolemia was detected mainly in patients with alcoholic hepatitis. Anemia was more frequent in patients with alcoholic and alcohol-viral hepatitis. Leukopenia, leukocytosis and an increase in ESR occurred in isolated cases. From the laboratory indicators more characteristic for alcoholic hepatitis were hypoalbuminemia, increased activity of gamma-glutamyltranspeptidase and

Table 3

Clinical variants of chronic hepatitis in different ethnic groups

Clinical option	Number of patients in ethnic groups																	
	ACH						AVCH						CVH-B					
	indigenous n=53		non-indigenous n=31		total n=84		indigenous n=45		non-indigenous n=27		total n=72		indigenous n=21		non-indigenous n=9		total n=30	
	абс.	%	абс.	%	абс.	%	абс.	%	абс.	%	абс.	%	абс.	%	абс.	%	абс.	%
Latent							3	6.7±3.7	18	66.7±9.1	21	29.2±5.4	3	14.3±7.6	5	55.6±16.6	8	26.7±8.1
Moderately severe with systemic manifestations	53	100.0	29	93.5±5.1	82	97.6±1.8	35	77.8±6.2	8	29.6±8.8	43	59.7±5.8	14	66.7±10.3	3	33.3±15.7	17	56.7±9.0
severe			2	6.4±5.1	2	2.4±1.8	7	15.5±5.4	1	3.7±3.6	8	11.1±3.7	4	19.0±8.6	1	11.1±10.5	5	16.7±6.8

Table 4

Comparative analysis of laboratory parameters in patients with alcoholic, alcoholic-viral and viral etiology

Indicator	Number of patients in groups					
	ACH n-84		AVCH n-72		CVH- B n-30	
	n	%	n	%	n	%
Hyperbilirubinemia (more than 20. 5 µmol / l)	21	25±5.5	5	6.8±3.0	4	13.7±6.2
Increase in the level of aminotransferases in total:	84	100	72	100	30	100
including less than 5 times	46	54.7±6.6	32	44.5±5.9	14	46.7±9.1
5 to 10 times	38	45.2±6.6	39	54.1±5.9	15	50.0±9.1
over 10 times	-		1	1.4±1.4	1	3.3±3.3
Increase in the level of alkaline phosphatase	26	31.0±6.1$^{x2.3}$	4	5.4±2.7^{x1}	2	6.6±4.6^{x1}
Hypoalbuminemia (less than 32g.l)	19	22.6±5.6^{x3}	9	12.2±3.9	2	6.6±4.6^{x1}
Hypergammaglobulinemia	3	3.6±5.0	3	4.2±2.4	1	3.3±3.3
Gammaglutamyltranspeptidase	80	95.2±3.0^{x3}	61	84.7±4.2^{x1}	1	3.3±3.3$^{x1.2}$
Rheumatoid factor	-		-		1	3.3±3.3
Positive LE-cell test	-		-		1	3.3±3.3
Hypocholesterolemia	3	3.6±5.0^{x3}	1	1.4±1.4^{x3}	16	54.0±9.1$^{x1.2}$
Hypercholesterolemia	57	67.8±6.2^{x3}	40	55.5±5.9^{x3}	1	3.3±3.3$^{x1.2}$
Thrombocytopenia	1	1.2±1.8	1	1.4±1.4	1	3.3±3.3
Anemia	19	22.6±5.6^{x3}	23	31.9±5.5^{x3}	1	3.3±3.3$^{x1.2}$
Leukopenia	4	4.7±3.0	2	3.1±1.9	2	6.6±4.6
Leukocytosis	1	1.2±1.8	-		-	
Increased ESR	4	4.7±3.0	-		1	3.3±3.3

hypercholesterolemia.

Conclusion. Thus, at chronic alcoholic hepatitis, the presence of extrahepatic systemic lesions is due to the toxic effect of alcohol on the endocrine glands, vessels, nervous system, and organs of the gastrointestinal tract, which was observed by other researchers. However, in indigenous patients they developed early and their number and severity were significantly higher [1, 5, 7].

Pain syndrome in patients with chronic alcoholic hepatitis in most cases is associated with the development of concomitant secondary chronic pancreatitis, gastritis and duodenitis. A number of authors have noted the presence of these diseases as the cause of the onset of pain. Chronic hepatitis combined with clinical symptoms is closer to alcoholic than to viral hepatitis. Very important was the fact that CVH replication markers were absent in all patients [2, 3, 6].

References

1. Abdurakhmanov D.T. Alkogol'nyj gepatit [Alcoholic hepatitis] Klinicheskaya gepatologiya [Clinical hepatology]. Moscow, 2008, No. 4 (2), P. 3-10.
2. Bueverov A.O. Pavlov A.I., Ivashkin V.T. Alkogol'naya bolezn' pecheni: vozmozhno li uluchshenie prognoza? [Alcoholic liver disease: is it possible to improve the prognosis?] Klinicheskie perspektivy gastroehnterologii i gepatologii [Clinical perspectives of gastroenterology and hepatology]. Moscow, 2011, No. 2, P. 3-10.
3. Mayer K.P. Gepatity i posledstviya gepatita [Hepatitis and the consequences of hepatitis]. Moscow, Geotar, 2004, P.720.
4. Abittan C. Alcoholic liver disease /Abittan C., Lieber C.//Clin. Perspect. in Gastroenterol. -1999. -V. 2. -P.57 -63.
5. Achord J.L. Review and treatment of alcoholic hepatitis: a meta-analysis adjusting for confounding variables // Gut. -1995. -V. 37. -P. 1138.
6. Buckwold V.E., Xu Z., Chen M., Yen T.S., Ou J.H. Effects of a naturally occurring mutation in the hepatitis B virus basal core promoter on precore gene expression and viral replication. J Virol -1996;70:5845-51.
7. O'Shea R.S. Alcoholic liver disease /O'Shea R.S., Dasarathy S., McCullough A.J.//Hepatology. -2010. -V.51. -P. 307-328.

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MORPHOLOGY OF MUCOSA-ASSOCIATED LYMPHOID TISSUE (MALT) OF LARYNX IN GENERAL HYPOTHERMIA

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ABSTRACT

We have studied the cytoarchitectonics of mucous-associated diffuse lymphoid tissue (MALT - mucosa-associated lymphoid tissue) of the larynx in persons who died from hypothermia. The material was collected in the summer (June, July, and August) and winter (December, January, and February) seasons of the year with the support from the State Budget Institution of the Bureau of Forensic Medical Examination of the Sakha (Yakutia) Republic. Death caused by low natural temperature occurred most often at ambient air temperatures ranging from -34°C to -40°C, less often at -31°C to -33°C. As one of the most frequent factors contributing to the onset of death from hypothermia is alcohol intoxication, for comparative characteristics of the morphology of lymphoid tissue, we also investigated the group of persons who died from general hypothermia with underlying alcohol intoxication.

Significant changes in the cellular composition of the mucous-associated diffuse lymphoid tissue of the larynx were revealed: a decrease in T- and B- lymphocytes and plasma cells and an increase in the number of destructively altered cells and macrophages.

Keywords: mucosa-associated diffuse lymphoid tissue (MALT), larynx, cytoarchitecture, hypothermia.

The study of the effects of low temperatures on the human body is one of the topical areas of basic and clinical medical sciences. The Sakha (Yakutia) Republic is a region with extreme climatic and geographical conditions, where the cold is one of the main environmental factors of adverse effects on the human body [1, 2, 5]. When activating protective reactions of the organism, an important role belongs not only to the central organs of the immune system, but also to its peripheral structures, in particular the immune (lymphoid) tissue of the walls of hollow organs. Located on the border between the external internal bodily environments, the peripheral parts of the immune system provide sanitation to the tissue complex. Destroying foreign substances and detoxification of the body [6, 13] depends on the functional activity of these structures.

When a person adapts to the conditions of the North, the mucosa-associated lymphoid structures of the respiratory organs are primarily affected by low temperatures, as they are the first "target" in the path of cold air penetration. In this regard, one of the promising areas of research on the pathological influence of the cold factor on the human body is the study of the immune structures of the larynx, which are located at the surface of the organ and are the first specific barrier to the penetration of antigens, in particular cold air.

In both domestic and foreign scientific literature, there is zero data on the morphology of the mucosa-associated lymphoid tissue of the laryngeal walls related to death from general hypothermia, as well as when this type of death is combined with alcohol intoxication under conditions of low air temperatures in the Sakha (Yakutia) Republic. The purpose of our study is to optimize the postmortem diagnosis of death from general hypothermia by examining the cellular com-