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LIVER CIRRHOSIS ASSOCIATED WITH ALCOHOL CONSUMPTION – LABORATORY PARTICULARITIES

Gorița Adriana, Bugai Rodica
"Nicolae Testemițanu" SUMPh, Republic of Moldova
Discipline of Internal Medicine

INTRODUCTION

Worldwide, excessive alcohol consumption is responsible for approximately 3.3 million deaths annually, accounting for 5.9% of all deaths (7.6% among men and 4.0% among women) and for approximately 139 million cases of disability [1,2]. Alcohol is a major risk factor in over 200 diseases, most of the deaths associated with it being caused by cardiovascular diseases, gastroenterological diseases (especially liver cirrhosis - LC) and various forms of cancer [1,3]. Chronic alcohol consumption affects the entire gastrointestinal system, with the liver being the most vulnerable organ, as it metabolizes over 90% of the alcohol consumed. Most ethanol is absorbed in the intestine (75%), in the stomach (22%) and only about 2% in the oral cavity and esophagus. This means that more than 90% of the absorbed alcohol circulates through the body and is eventually transported to the liver through the portal vein [7,8]. This process leads to the accumulation of toxic metabolites, overloading the hepatic enzyme systems. Alcoholic liver disease (ALD) is the most common pathology associated with alcohol consumption. If alcohol exposure is not stopped in time, liver damage becomes irreversible, with an increased lethal risk.

LC is the most prevalent severe liver disease associated with alcohol consumption, with an estimated 10% to 20% of chronic drinkers developing the condition, generally after at least 10 years of binge drinking. In Europe, 41% of deaths from liver disease are attributed to ALD. In addition, of the 46% of deaths with undetermined hepatic etiology, a significant proportion is probably associated with alcohol consumption [1,2]. In the USA, between 5-10% of the population chronically abuses alcohol and requires medical monitoring [4,5]. After 10-20 years of consumption, about 15% of them develop LC, affecting between 500,000 and 1 million people, with an annual mortality of about 20,000 cases [5,6]. In the Republic of Moldova, 63.2% of the adult population consumes alcohol, and the country remains among those with the highest alcohol consumption per adult (over 15 years old) of 12.9 liters.

AIM OF THE WORK

To evaluate the laboratory particularities of patients with alcohol-associated liver cirrhosis (ALC).

MATERIAL AND METHODS

An analytical-observational-retrospective-prospective cohort survey study was conducted. Patients undergoing treatment in the Gastroenterology department of the MCH "St. Archangel Michael" Municipal Clinical Hospital, Chișinau were selected: study group (SG) with ALC - 58 patients and control group (CG) with LC of various etiology (non-alcoholic) - 7 patients. SG gathered 40 men (69%) and 18 women (31%), aged between 27-86 years, average age of 56±11.66 years, CG – 3 men (43%) and 4 women (57%), aged between 56-75 years, mean age of 65±6.43 years. Alcohol consumption was confirmed by the AUDIT-C test [9]. Specific clinical-paraclinical changes were assessed in accordance with the National Clinical Protocol, in accordance with the recommendations of the European Society of Gastroenterology and Hepatology.

RESULTS

The analysis of the blood count attests anemia with a mean HGB value – 105.41±24.11 g/L, RBC – 3.48±2.41×1012/L, HCT – 0.319±0.065 L/L, an increased MEV – 101.7±10.35 fL, accelerated ESR – 38.44±20.06 mm/hour and a PLT thrombocytopenia – 146.05±102.2×109/L (Table 1). According to p1,2 we observe that there is a statistically significant difference ($p<0.05$) in MEV, platelet and neutrophil leukocyte values between patients with ALC and LC.

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Table 1. Changes in the blood count of patients with ALC versus LC of non-alcoholic etiology

Note: RBC – erythrocytes, HGB – hemoglobin, HCT – hematocrit, MEV – mean erythrocyte volume, ESR – erythrocyte sedimentation rate

Assessed index (baselines)	Patients with ALC M ± ES	Patients with non-alcoholic LC M ± ES	p1,2	T significance test	df degree of freedom
RBC x10^{12}/L (m. 4,5 – 5,0/ w. 3,7 – 4,7)	3,48±2,41 (max. 21,0 min. 1,64) ↓	3,95±0,79 (max. 5,49 min. 2,59) ↓	p>0,05	-1,02	21
HGB g/L (m. 130 – 160/ w. 120 – 140)	105,41±24,11 (max. 175,0 min. 53,0) ↓	112,0±29,02 (max. 144,0 min. 45,0) ↓	p>0,05	-0,53	7
HCT L/L (0,37 – 0,47)	0,319±0,065 (max. 0,509 min. 0,167)↓	0,339±0,073 (max. 0,436 min. 0,173)	p>0,05	-0,64	7
MEV fL (80 – 100)	101,7±10,35 (max. 121,0 min. 67,0) ↑	85,42±10,06 (max. 100,0 min. 67,0)	p<0,05	3,75	7
Platelets x10^{9}/L (150 - 400)	146,05±102,2 (max. 476,0 min. 22,0) ↓	97,28±37,15 (max. 159,0 min. 57,0) ↓	p<0,05	2,39	18
Leukocytes x10^{9}/L (4,0 – 9,0)	7,55±4,65 (max. 25,5 min. 1,4)	3,57±1,07 (max. 5,2 min. 2,0) ↓	p<0,05	5,27	38
Neutrophils% (47 – 72)	62,25±16,66 (max. 89,4 min. 5,5)	50,9±10,56 (max. 63,1 min. 36,6)	p<0,05	2,34	9
Lymphocytes% (19 – 37)	25,25±15,06 (max. 89,5 min. 4,4)	35,44±11,96 (max. 51,1 min. 17,7)	p>0,05	-1,93	8
Monocytes% (3 – 11)	9,44±3,75 (max. 21,8 min. 2,0)	10,05±3,70 (max. 16,8 min. 6,4)	p>0,05	-0,33	7
Eosinophils% (0,5 – 5)	2,31±2,07 (max. 12,1 min. 0,6)	3,17±1,63 (max. 5,6 min. 1,2)	p>0,05	-1,17	9
Basophils% (0 – 1)	0,56±0,35 (max. 1,8 min. 0,2)	0,42±0,14 (max. 0,6 min. 0,2)	p>0,05	1,7	17
ESR mm/oră (b. 2 – 10/ f. 2 – 15)	38,44±20,06 (max. 78,0 min. 5,0) ↑	27,71±17,62 (max. 57,0 min. 9,0) ↑	p>0,05	1,39	8

According to the biochemical analysis (Table 2), cytolytic syndrome is attested in patients with ALC with a predominance of increased AST, mean value – 106.71±123.93 u/l, ALT 39.31±31.78 u/l. Rittis index in patients with ALC 2.71 (>1.5), in patients with LC 1.44 (<1.5). Cholestatic syndrome present in these patients by significant increase in conjugated bilirubin - 101.05±126.24 mcmol/l and GGTP by approximately 5 times with a mean value of 256.4 ±386.07 u/l. Hypoalbuminemia with a mean value of 25.23±6.22 g/l and a renal impairment due to increased serum urea and creatinine with a mean value of 10.85±12.08 mmol/l, and 135.41±228.14 umol/l, respectively. Hyperglycemia with a mean serum glucose value of 5.87±1.60 mmol/l and hypocholesterolemia - 3.53±1.16 mmol/l [9]. Regarding the values of alkaline phosphatase, GGTP, the 3 fractions of bilirubin, glucose and triglycerides, there is a statistical difference (p<0.05) between the SG and the CG analyzed. Hepatopriv syndrome present by decreasing prothrombin 49.58 ±15.61% mean, fibrinogen – 1.96 ±0.73 g/l and INR – 1.74 ±0.67 [9].

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Table 2. Comparative biochemical changes in patients with ALC versus LC of non-alcoholic etiology

Assessed index (baselines)	Patients with ALC M ± ES	Patients with non-alcoholic LC M ± ES	p1,2	T significance test	df degree of freedom
Total protein g/l (62 – 80)	63,53±9,27 (max. 86,0 min. 49,0)	63,14±7,60 (max. 72,0 min. 52,0)	p>0,05	0,11	8
Albumin g/l (38 – 54)	25,23±6,22 (max. 40,6 min. 13,2) ↓	30,81±8,13 (max. 46,0 min. 21,0) ↓	p>0,05	-1,49	6
Urea mmol/l (2,5 – 8,3)	10,85±12,08 (max. 54,0 min. 1,78) ↑	8,04±3,72 (max. 16,3 min. 4,5)	p>0,05	1,26	24
Creatinine mcm/l (53 – 115)	135,41±228,14 (max. 1555,0 min. 37,0) ↑	100,12±36,90 (max. 146,6 min. 58,8)	p>0,05	1,03	56
Total bilirubin mcmol/l <17	157,84±186,46 (max. 726,2 min. 6,8) ↑	35,92±15,68 (max. 52,0 min. 13,7) ↑	p<0,05	4,73	61
Conjugated bilirubin mcmol/l <5,1	101,05±126,24 (max. 529,1 min. 2,6) ↑	12,58±4,76 (max. 21,5 min. 6,9) ↑	p<0,05	5,20	57
Free bilirubin mcmol/l <15	58,07±66,49 (max. 268,0 min. 2,1) ↑	23,34±12,08 (max. 39,4 min. 6,5) ↑	p<0,05	3,34	50
Glucose mmol/l (3,89 – 5,84)	5,87±1,60 (max. 12,3 min. 3,6) ↑	5,19±0,64 (max. 6,31 min. 4,4)	p<0,05	1,99	16
ALAT u/l <49	39,31±31,78 (max. 179,0 min. 7,0)	44,42±37,2 (max. 131,0 min. 13,0)	p>0,05	-0,32	7
ASAT u/l <46	106,71±123,93 (max. 832,0 min. 14,0) ↑	64,14±49,24 (max. 178,0 min. 23,0) ↑	p>0,05	1,63	16
Alpha-amylase u/l (0 – 86)	67,16±79,41 (max. 579,0 min. 13,9)	66,85±29,59 (max. 107,0 min. 30,0)	p>0,05	0,01	18
Alkaline phosphatase u/l (100 - 290)	160,98±108,35 (max. 780,0 min. 38,0)	108,85±19,01 (max. 149,0 min. 82,0)	p<0,05	3,10	52
GGTP u/l (5 - 55)	256,4±386,07 (max. 2509,0 min. 16,0) ↑	42,0±10,14 (max. 62,0 min. 29,0)	p<0,05	4,03	54
Total cholesterol mmol/l (3,87 – 5,2)	3,53±1,16 (max. 6,2 min. 1,3) ↓	3,54±1,0 (max. 4,9 min. 1,74) ↓	p>0,05	-0,01	6
Triglycerides mmol/l (0,68 – 1,88)	1,34±0,90 (max. 5,6 min. 0,39)	0,74±0,18 (max. 0,98 min. 0,43)	p<0,05	3,92	36
Serum iron mcmol/l (9,0 – 31,3)	14,22±8,27 (max. 34,8 min. 1,7)	18,08±14,51 (max. 47,8 min. 1,2)	p>0,05	-0,63	7

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CONCLUSIONS

Alcohol is a primary risk factor for the onset and progression of liver disease to cirrhosis, which aggravates the clinical course of the disease, survival rate and mortality. In these patients, there is an increase in MEV, conjugated bilirubin, a predominance of an increase in AST with an increased Rittis index and GGTP of approximately 5 times; Hepatopriv syndrome manifested by decreased serum albumin, prothrombin, fibrinogen and INR.

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