

## ALCOHOLIC CIRRHOSIS OF THE LIVER AND DISARRANGEMENT OF PLASMA ATHEROGENIC FACTORS

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Alcohol is metabolized by alcohol-dehydrogenase into acetaldehyde, and by aldehyde-dehydrogenase and acetyl-coenzyme-A into acetate. Thus produced hydrogen is accepted by nicotinamide-adenine-dinucleotide, which leads to the accumulation of fatty acids in the liver. Cirrhosis of the liver develops due to the intake of alcohol, 80g/day for men and 20g/day for women. The aim of this study was to determine the profile of plasma atherogenic factors in patients with alcoholic cirrhosis of the liver, compared to patients with HCV-cirrhosis, and to determinate their diagnostic significance. The study compared a total cholesterol (TC), triglycerides (Tg), high-density-lipoprotein-cholesterol (HDL-C), low-density-lipoprotein-cholesterol (LDL-C), and apolipoproteins (ApoA1 and ApoB) in plasma of patients with alcoholic cirrhosis compared to patients with HCV-cirrhosis. The values of TC/HDL-C, ApoB/ApoA1 and LDL-C/HDL-C were calculated and compared in both groups. The study included 37 patients with alcoholic cirrhosis, mean age 52.65 years (SD-6.73), who consumed alcoholic beverages during an average of 8.67 years (SD-1.96) and 35 patients with HCV-cirrhosis. There were 21.62% of women and 78.38% of men with alcoholic cirrhosis, and 34.29% of women and 65.71% of men with HCV-cirrhosis. The average values for the following parameters in alcoholic cirrhosis were obtained: TG 11.91mmol/l (SD-2.51), TC 14.63 (SD-2.62), LDL-C - 8.77 mmol/l (SD-2.19), HDL-C 0.41 mmol/l (SD-0.09), apolipoprotein-B 4.01g/l (SD-0.18), apolipoprotein-A1 0.51g/l (SD-0.08); in HCV-cirrhosis, the average values of the studied parameters were as follows: TG 8,62mmol/l (SD-2.31), TC 9.67 (SD-2.39), LDL-C 6.12 mmol/l (SD-1.78), HDL-C 0.76 mmol/l (SD-0.09), apolipoprotein-B 2.38g/l (SD-0.16), apolipoprotein-A1 0.98g/l (SD-0.05). The apolipoprotein-B/apolipoprotein-A1 relation can serve as a diagnostic marker for the presence of alcoholic cirrhosis, and is a better indicator of atherogenic risk. *Acta Medica Medianae 2016;55(3):38-43.*

**Key words:** alcohol, cirrhosis of the liver, atherogenic, plasma

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### Introduction

The most common cause of liver disease, in even 80% of cases, is alcohol. The most difficult form of alcoholic liver disease is cirrhosis of the liver. Cirrhosis is the ninth cause of death, and the sixth cause in the population aged 45-64 years (1-3). The amount of alcohol higher than 80 g of

alcohol per day for men and 20 g for women as well as longer duration of alcohol consumption are the risk factors for alcoholic liver disease, cirrhosis of the liver.

Alcohol in the body, mainly in the liver, is subject of oxidation. In that way, 160-180 g of alcohol per day is metabolized. Alcohol dehydrogenase (ADH), the microsomal oxidation system (MEOS), and catalase are enzymatic systems that metabolize ethanol into the acetate. Ethanol is oxidized, in 80-85% of the cases, with ADH to the stage of a highly toxic acetaldehyde. Acetaldehyde leads to cell necrosis by damaging the cell membrane. In the presence of acetyl-CoA, acetaldehyde is converted into the acetate by an aldehyde-dehydrogenase (ALDH). In that way, by metabolism of ethanol, hydrogen is released and further accepted by nicotinamide-adenine-dinucleotide (NAD). NADH replaces fatty acids as an energy source, which results in the accumulation of fatty acid. The consequence of decreased oxidation of fatty acids is an accumulation of triglycerides in the liver and increased lipoprotein

synthesis. The increased concentration of NADH changes the redox potential of hepatocytes, increases lipid peroxidation, and inhibits protein synthesis. NADH transfers the hydrogen necessary for the conversion of pyruvate into lactate, and in that way increases the levels of lactate and urate levels in serum. MEOS, an enzymatic system, metabolizes 10-15% of ethanol through enzyme cytochrome P450-2E1 (CYP2E1). Catalase system is poorly active in the metabolism of alcohol (4-7).

A food deficient in protein and antioxidant vitamins increases the hepatotoxicity of ethanol. Hepatotoxic effects of ethanol are more noticeable with an increase in intake of unsaturated fatty acids. Ethanol increases absorption of iron from the food and increases its disposal in the liver (8, 9).

The clinical picture of alcoholic liver disease varies from asymptomatic disease, fatty liver, alcoholic hepatitis, to heavy cirrhosis of the liver with complications like icterus, ascites, varices oesophagi (10-12). The overlapping of more than one form of alcoholic liver disease in the same patient is often present.

Cirrhosis of the liver is the most heavy form of alcoholic liver disease. It is a chronic disease which is characterized by necrosis of the hepatocytes, nodular regeneration of the liver parenchyma, formation of pseudolobules, ingrowth of collagen into the fibrous tissue, disorders of the lobule architecture, and development of atypical bile ducts.

Alcoholic cirrhosis is often not recognized. The classic clinical picture of the cirrhosis of the liver is presented from asymptomatic state to the manifestation of weakness, fatigue, nausea, anorexia, vomiting and diarrhea, icterus, ascites, hepatic encephalopathy, varices oesophagi, and haematemesis. In laboratory, the cytolysis of hepatocytes and retention of bilirubin and cholestasis are present as well as the reduced synthetic liver function.

Diagnosis is based on properly taken history, including the data on alcoholism, elevated values of aspartate transaminase (AST) and alanine transaminase (ALT) (13). Alkaline phosphatase (ALP) may be elevated up to four times. Gamma-glutamyl-transpeptidase (GGT) is induced by alcohol, and quickly returns to the normal values after stopping the use of alcohol (14-16). Hyperbilirubinemia and prolonged prothrombin time is more common in more severe forms of alcoholic cirrhosis. Hyperuricemia and dyslipidemia follow the chronic alcoholism. More severe forms of cirrhosis are accompanied with leukocytosis, neutrophilia, and anemic syndrome as well as hypoalbuminemia with hypergammaglobulinemia. In chronic consumption of alcohol, the values of IgA are elevated (17-21).

Ultrasonography, computed tomography, and magnetic resonance detect changes in the shape and size of the liver and fibrosis, but also the absence of biliary obstruction and focal changes. Color Doppler detects portal hypertension.

For the diagnosis of alcoholic liver disease, the biopsy of liver has an important role (22-24).

Cirrhosis of the liver caused by hepatitis C virus (HCV) infection C is common. Clinical manifestations and the most of the laboratory analysis results are the same as in alcoholic cirrhosis. The difference is in the serological enzyme immunoassay (EIA) test for C virus, which was positive.

### Aim

The aim of this study was to determine the profile of atherogenic factors in plasma in patients with alcoholic cirrhosis compared to patients with non-alcoholic cirrhosis with hepatitis C virus (HCV) infection.

### Methods

The study followed 37 patients with cirrhosis who consumed alcoholic beverages daily in large quantities above 80 grams, which is the amount of 3-4 units of alcohol for men, and 2-3 units of alcohol for women (one alcoholic unit corresponds to approximately one serving of hard liquor, or 0.5 liters of beer, or 2 dl of wine spritzer or 4 oz) (6). The control group consisted of 35 patients with verified cirrhosis with HCV. The diagnosis of cirrhosis with HCV is set by EIA. All the patients underwent laboratory analyses for aspartate aminotransferase (AST) and alanine-aminotransferase (ALT), total cholesterol (UH), triglycerides, high-density-lipoprotein cholesterol (HDL-C), low-density-lipoprotein cholesterol (LDL-C), and apolipoproteins (Apo-A1 and Apo-B) and blood count. The ratio of UH/HDL and ApoB /ApoA1 and LDL-/HDL-C was calculated.

Diagnosis of cirrhosis is carried out with ultrasound apparatus Siemens X 300 in a supine position, by taking medical history, including the data on alcoholism, laboratory blood test, and EIA.

After performing blood tests, patients with viral hepatitis other than HCV and autoimmune disorders were excluded from the studies, as well as the patients with tumor processes and obstruction of the biliary tree, which was verified by ultrasound examination.

In this paper, we used the standard descriptive methods, mean values, standard deviation and percentage distribution, and the data between groups were analyzed by appropriate statistical tests depending on the type and distribution of features (Man Whitney U test, Student's t test, Hi-square test). The significance level of  $p < 0.05$  was taken as significant. Data are presented in tables.

### Results

The study included 72 patients, of whom 27.78% were women and 72.22% men, mean age  $52.02 \pm 8.56$  years. Patients were divided into two groups, with alcoholic cirrhosis and non-alcoholic cirrhosis with HCV.

The first group included 37 patients with alcoholic cirrhosis, aged 32 to 67 years, mean age  $52.65 \pm 6.73$  years. They consumed alcoholic drinks in quantities greater than 80 g for 5 to 11 years, on average  $8.67 \pm 1.96$  years. Among the respondents, there was 21.62% of women and 78.38% of men. In the second group of patients, there were 35 subjects with non-alcoholic cirrhosis with HCV, aged 31 to 65 years, mean age  $47.31 \pm 5.63$  years, who did not consume alcoholic beverages. Among them, there was 34.29% of women and 65.71% of men. There was no significant differences in gender distribution and average age between the examined groups (Table 1).

**Table 1.** General characteristics of the studied group of patients

	Women / men % / % (n / n)	Age (years)	Length of alcohol consumption (years)
Alcoholic cirrhosis	21.62/78.38 (8/29)	$52.65 \pm 6.73$	$8.67 \pm 1.96$
Non-alcoholic cirrhosis with HCV	34.29/65.71 (12/23)	$47.31 \pm 5.63$	-
Total	20/52 (27.78/72.22)	$52.02 \pm 8.56$	-

Data are presented as n/n or (%/%) and mean  $\pm$  SD; NS for all parameters between groups

**Table 2.** Markers of hepatocellular damage and blood count

	Alcoholic cirrhosis	Non-alcoholic cirrhosis with HCV
The markers of hepatocellular damage		
AST (IU/l)	$91.36 \pm 6.45$	$75.37 \pm 6.21^{**}$
ALT (IU/l)	$83.89 \pm 11.49$	$69.76 \pm 9.31^{**}$
AST/ALT	1.09	$0.76^{**}$
GGT (IU/l)	$196.7 \pm 14.42$	$100.2 \pm 9.56^{**}$
albumini (g/L)	$30.04 \pm 5.54$	$31.03 \pm 6.43$
INR	$1.4 \pm 0.09$	$1.4 \pm 0.08$
Hematologic parameters		
The number of leukocytes (G/L)	$9.78 \pm 0.68$	$7.03 \pm 0.71^*$
Hb (g/L)	$114.3 \pm 12.49$	$112.4 \pm 11.31$
Hct	$31.29 \pm 3.19$	$32.08 \pm 3.51$
PLT (G/L)	$110.12 \pm 16.11$	$168.09 \pm 26.34^*$

Hb-haemoglobin, Hct-haematocrit, PLT-platelets, INR-international normalized ratio, GGT-gamma glutamic transferase, AST-aspartate aminotransferase, ALT-alanine aminotransferase  
Data are presented as mean  $\pm$  SD; \* $p < 0.05$ , \*\* $p < 0.01$  vs. alcoholic cirrhosis

The average values of AST, ALT, GGT and AST/ALT ratio in the alcoholic cirrhosis group were

significantly higher than in non-alcoholic cirrhosis with HCV group ( $p < 0.01$ ), prospectively. There were no differences in albumin concentration, INR and parameters of anemia between the groups. The number of leukocytes was significantly higher, and the number of platelets lower in alcoholic cirrhosis group ( $p < 0.05$ ) (Table 2).

**Table 3.** Atherogenic indicators

	Alcoholic cirrhosis	Non-alcoholic cirrhosis with HCV
TC (mmol/l)	$14.63 \pm 2.62$	$9.67 \pm 2.39^{**}$
LDL-C (mmol/l)	$8.77 \pm 2.19$	$6.12 \pm 1.78^{**}$
HDL-C (mmol/l)	$0.41 \pm 0.09$	$0.76 \pm 0.05^{**}$
TG (mmol/l)	$11.91 \pm 2.51$	$8.63 \pm 2.31^{**}$
TC/HDL-C	$32.91 \pm 8.21$	$12.05 \pm 7.11^{**}$
LDL-C/HDL-C	$17.93 \pm 5.21$	$8.66 \pm 3.22^{**}$
apoB (g/L)	$4.01 \pm 0.18$	$2.28 \pm 0.17^{**}$
apoA1 (g/L)	$0.51 \pm 0.08$	$0.98 \pm 0.03^{**}$
apoB/apoA1	$8.21 \pm 1.40$	$2.41 \pm 1.22^{**}$

TC-total cholesterol, TG-triglycerides, ApoA1-apolipoprotein A1, ApoB-apolipoprotein B

Data are presented as mean  $\pm$  SD; \*\* $p < 0.01$  vs. Alcoholic fatty liver

All examined patients had highly reduced HDL-C (less than 1 mmol/l) as well as highly elevated TC (more than 6.2 mmol/l) and TG (more than 5.65 mmol/l) in both alcoholic cirrhosis group and non-alcoholic cirrhosis with HCV group. Patients with alcoholic cirrhosis had values of LDL-C higher than 4.9 mmol/l, while this was not the case in non-alcoholic cirrhosis with HCV group.

The index TC/HDL-C was elevated and at high-risk (more than 4.5) with an average value of  $32.91 \pm 8.21$  in patients with alcoholic cirrhosis and  $12.05 \pm 7.11$  in non-alcoholic cirrhosis with HCV. The apoB/apoA1 index was calculated, and all values in both groups were over 1.1, which also classifies them as high risk patients, with an average value of  $8.21 \pm 1.40$  in patients with alcoholic cirrhosis and  $2.41 \pm 1.22$  in patients with non-alcoholic cirrhosis with HCV. Index LDL-C/HDL-C was higher than 3.5, which represents a very high atherosclerotic risk in all patients, with an average value of  $17.93 \pm 5.21$  in patients with alcoholic cirrhosis and  $8.66 \pm 3.22$  in non-alcoholic cirrhosis with HCV.

There were significant differences in lipid parameters and apolipoproteins between the groups. Higher values of TG, TC, LDL-C, apoB, apoB/apoA1 ratio, and lower values of HDL-C and apoA1 were registered in alcoholic cirrhosis compared to non-alcoholic cirrhosis with HCV group (Table 3).

## Discussion

The examined group of patients with alcoholic cirrhosis and non-alcoholic cirrhosis with HCV were predominantly male, with similar mean age. During the period of alcohol consumption in alcoholic cirrhosis, there was a manifestation of

the most heavy form of alcoholic liver damage, cirrhosis of the liver. All patients had elevated transaminases, with AST predominance, so the AST/ALT ratio was 1.09. The values of GGT were also elevated. In non-alcoholic cirrhosis with HCV group, which were not consumers of alcoholic beverages, the transaminases and GGT- were increased, with predominance of ALT. The AST/ALT ratio was lower (0.76) compared with patients with alcoholic cirrhosis (Table 1 and 2).

Laboratory analysis verified the elevation of the values of TC, TG and LDL-C, as atherogenic factors, and decreased values of anti-atherogenic HDL-C. The lipid disorders were more prominent in alcoholic cirrhosis than non-alcoholic cirrhosis with HCV (Table 3).

In the body, alcohol is subject to oxidation, mainly in the liver. Ethanol is metabolized in the acetate by using the three-enzyme system: alcohol dehydrogenase (ADH), the microsomal oxidation system (MEOS) and catalase. Ethanol is oxidized, in 80-85% of cases, to highly toxic acetaldehyde which damages the cell membrane, leading to cell necrosis. Ethanol oxidation produces the accumulation of fatty acid and triglycerides in the liver and increased lipoprotein synthesis. The increased concentration of NADH changes the redox potential of hepatocytes, leading to the inhibition of protein synthesis and increase in lactate and urate levels in serum. MEOS is responsible for the metabolism of 10-15% of the ethanol. Thus, the consumption of oxygen and production of acetaldehyde are increased, as well as the lipid peroxidation. Catalase system is poorly active in the metabolism of alcohol (4-7).

The values of apoA1 and apoB were determined. Among patients with alcoholic cirrhosis, apoA1 and HDL-C were lower compared to non-alcoholic cirrhosis with HCV group. ApoA1 is a component of an anti-atherogenic lipoprotein and was decreased in alcoholic cirrhosis (Table 3). The low values of apoA1 created conditions for the development of atherogenic effect. ApoB, as atherogenic component, was extremely increased in alcoholic cirrhosis. Due to the reduced apoA1 and increased apoB, in patients with chronic consumption of ethanol and developed alcoholic cirrhosis and non-alcoholic cirrhosis with HCV, atherogenic affect in plasma was created (4, 20, 21).

Apolipoprotein B is the primary apolipoprotein of chylomicrons and LDL, and is responsible for transporting of the cholesterol to tissues (5, 8). ApoB in particles of LDL is a ligand for the LDL receptors of the cells, and it "unlocks" cells for

the transport of cholesterol. By an unknown mechanism, high values of apoB lead to the formation of plaques in blood vessels and the development of atherosclerosis. Thus, the determination of apoB is a better and more significant indicator of atherosclerosis risk than analysis of LDL and total cholesterol. As in patients with alcoholic cirrhosis, there is a significant elevation of apoB, as well as increased risk of plaques formation in blood vessels, and consequently, there is a risk of the development of atherosclerosis.

Apolipoprotein A1 is a major component of plasma HDL-C. It leads to the so-called fat-efflux from tissues to the liver (14). Thus mobilized fat is than excreted from the liver. ApoA1 is a cofactor of lecithin cholesterol transferase (LCAT) important for the synthesis of cholesterol esters of the plasma. ApoA1 is an ingredient of prostacyclin (PGI<sub>2</sub>), responsible for the realization of antiaggregational effects.

The study Incremental Decrease and Events through Aggressive Lipid Lowering (IDEAL), and INTERHEART study (25,26) emphasize that the determination of the relation apoB/apoA1 is a significant prognostic factor for atherogenic effects. Individual monitoring of TC, LDL-C or apoB, as atherogenic factors, and HDL-C, and ApoA1, as antiatherogenic factors, as well as determining the relation TC/HDL-C or LDL-C/HDL-C is less significant when compared to relation apoB/apoA1.

Determining the relationship between apoB/ApoA1 proved to be the most important and most coherent marker for the existence of atherogenic plasma profile.

### Conclusion

The determination of TC, TG, LDL-C, HDL-C, apoA1, apoB1 and the relation apoB/apoA1, LDL-C/HDL-C, TC/HDL-C is important for the follow-up of atherogenic factors in plasma. Alcoholic cirrhosis and non-alcoholic cirrhosis with HCV lead to increase in Apo B. It is an indicator of atherogenic status of plasma, and increased risk for the formation of plaques in blood vessels, including atherosclerosis. The values of TC, TG, LDL-C, HDL-C, LDL-C/HDL-C and TC/HDL-C levels showed great variation, reaching very elevated values. Determining the value of the ratio apoB/apoA1 is a less variable and more coherent parameter for tracking the atherogenic changes in the plasma. The lipids and apoB/apoA1 relation, besides markers of hepatocellular damage, can serve as diagnostic criteria for the presence of alcoholic cirrhosis, thus being better indicators of atherogenic risk.

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## ALKOHOLNA CIROZA JETRE I POREMEĆAJ ATEROGENIH FAKTORA U PLAZMI

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Alkohol se metaboliše alkoholnom dehidrogenazom do acetaldehida, pa aldehid dehidrogenazom uz acetil koenzima-A do acetata. Tako nastali vodonik prihvaća nikotinamid adenin dinukleotid, te se akumuliraju masne kiseline u jetri. Ciroza jetre nastaje unosom alkohola od 80 g/dan za muškarce, a 20 g/dan za žene. Cilj studije bio je da se utvrdi profil aterogenih faktora plazme bolesnika sa alkoholnom cirozom jetre u odnosu na bolesnike sa cirozom jetre uzrokovanom HCV infekcijom i odredi njihov dijagnostički značaj. Studija upoređuje ukupni holesterol (TC), trigliceride (Tg), visokogustine lipoprotein holesterol (HDL-C), niske-gustine lipoprotein holesterol (LDL-C) i apolipoproteine (ApoA1 i ApoB) u plazmi bolesnika sa alkoholnom cirozom jetre i bolesnika sa cirozom jetre i HCV infekcijom. Izračunavan je i upoređivan odnos TC/HDL-C i ApoB/ApoA1 i LDL-C/HDL-C u obe grupe bolesnika. Studija uključuje 37 bolesnika sa alkoholnom cirozom jetre, prosečne starosti 52,65 godina (SD-6,73), koji su konzumirali alkoholne napitke prosečno 8,67 godine (SD-1,96) i 35 bolesnika sa cirozom jetre i HCV infekcijom. Žena je bilo 21,62%, a 78,38% muškaraca sa alkoholnom cirozom jetre; 34,29% žena i 65,71% muškaraca sa cirozom jetre izazvanom HCV-om. Prosečne vrednosti Tg su 11,91 mmol/l (SD-2,51), TC 14,63 (SD-2,62), LDL-C 8,77 mmol/l (SD-2,19), HDL-C 0,41 mmol/l (SD-0,09), apolipoprotein-B 4,01 g/l (SD-0,18), apolipoprotein-A1 0,51 g/l (SD-0,08) kod alkoholne ciroze jetre, a u HCV cirozi Tg su prosečne vrednosti 8,62 mmol/l (SD-2,31), TC 9,67 (SD-2,39), LDL-C 6,12 mmol/l (SD-1,78), HDL-C 0,76 mmol/l (SD-0,09), apolipoprotein-B 2,38 g/l (SD-0,16), i apolipoprotein-A1 0,98 g/l (SD-0,05). Odnos apolipoprotein-B/apolipoprotein-A1 može poslužiti kao dijagnostički marker prisustva alkoholne ciroze i bolji je pokazatelj aterogenog rizika. *Acta Medica Medianae 2016;55(3):38-43.*

**Ključne reči:** alkohol, ciroza jetre, aterogeni, plazma

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