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ORIGINAL PAPER

Biochemical parameters study of serum, an ascetic fluid in decompensated cirrhosis

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Abstract.
We have selected a group of 15 patients with decompensate cirrhosis for which we performed the following biochemical investigations in serum and ascetic fluid: ALT, AST, AST/ALT, LDH, Fe, BT, BD, BI, GGT, ALP, protein electrophoresis, CRP, CHE, ALB, CT, TG, Glu, AMY, CPK, urea, Cr, AcU. In the context of necrotic lesions and hepatic fibrosis, the de Ritis ratio has an increasing tendency in serum but also in ascitic fluid. The normal values of total LDH activity from serum and ascitic fluid do not exclude the pathological values of the LDH_4 and LDH_5 isoenzymes which imply the intensification of the anaerobic glycolysis from the cirrhotic liver tissue. The hepatic failure and the mesenchymal inflammation are also expressed by the A/G ratio from the ascitic fluid. The sensitivity to the oxidative stress grows as the hepatoprive and excreto-biliary syndromes (parenchymal decompensation) are more pronounced. The differences without statistical significance for urea, creatinine and uric acid values from serum and ascitic fluid prompt us to consider that the renal failure in decompensate cirrhosis can be established from only the behavior of these analytes in ascitic fluid. The level of AcU is high both in LA and in serum, which makes us consider that xanthinosidase too has higher activity and the superoxide radicals produced by the enzyme are in a higher level.

Abbreviations: A/G-albumin-globulin ratio, AcU-uric acid, ALB-albumin, ALP-alkaline phosphatase, ALT-alanine amino transferase, AMY-amylase, AST/ALT-de Ritis ratio, AST-Aspartate Amino Transferase, AT-aminotransferase, BD-conjugated bilirubin, BI - nonconjugated bilirubin, BT-total bilirubin, CH-cirrhotic hepatitis, CHE-cholinesterase, CPK-creatine phosphokinase, Cr, Creacreatinine, CT-total cholesterol, ELFO-protein electrophoresis, Fe-iron, GGT-gamma glutamiltranspeptidase, Glu-glucose, HTP-portal hypertension, LA-ascitic fluid, LDH-Lactate Dehydrogenase, M-witness, P-patient, TG-triglycerides.

Key words*:* decompensate cirrhosis, HTP, LA, AST/ALT, A/G, CT, Urea, Cr, AcU.

Introduction

Chronic hepatic disorders, which cause cirrhosis, have cellular death as common and compulsory starting point.

Cirrhosis is the final stage of evolution for all the inflammatory or degenerative chronic hepatic diseases. Even when inflammation and necrosis are not present, the pathogenic process of cirrhosis has a progressive character through the expansion trend of the fibrosis and the persistence of regeneration (in the form of nodules), and the coexistence of these processes is mandatory for the cirrhosis diagnosis.

The cellular death causes the regeneration process, without a balance between destruction and regeneration; thus, the normal function of the liver is highly influenced and deteriorated. In the previously works we have studied antioxidative enzymes and other biological markers in the biochemical balance of chronic liver diseases (Başa et al., 2009).

The appearance of ascites marks the start of "decompensate", which is the stage of liver cirrhosis in which the compensatory mechanisms are exceeded and the fluid goes beyond the vascular space as discussed by Henriksen (Henriksen, 1995).

The biological exploration of the cirrhotic patient is a compulsory stage in the clinical, imagistic, and histological examination.

Materials and Methods

The study included 15 patients (10 male patients=66.7%, and 5 female patients = 33.3%), with ages between 55 and 83. (Table 1) In these patients the etiological factors of cirrhosis are the infections with hepatic viruses B and C and alcohol consumption.

For all 15 patients hospitalized in the Internal Diseases department of the Emergency Military Hospital, Constanta there were performed determinations from serum and ascitic fluid for the analytes which characterize: the ***hepatocytolytic syndrome*** (ALT, AST, AST/ALT, LDH, Fe), the ***excreto-biliary syndrome*** (BT, BD, BI, GGT, ALP), the ***mesenchymal inflammation*** **(**protein electrophoresis, CRP**),** the ***hepatoprive syndrome*** (CHE, ALB), ***the lipid profile*** (CT, TG), the ***glucose homeostasis*** (glucose, Glu**),** ***the pancreatic function*** (AMY), ***the muscular activity*** (CPK) ***and the renal function*** **(**urea, creatinine (Cr), uric acid (AcU)**)**

Table 1 Descriptive age/gender statistics regarding the patients with ascitic cirrhosis included in the study

Age interval	Total	Men	Women
50 – 60 years	2 (13.3%)	2 (20%)	-
60 – 70 years	9 (60.0%)	5 (50%)	4 (80%)
70 – 80 years	3 (20.0%)	2 (20%)	1 (20%)
80 – 90 years	1 (6.7%)	1 (10%)	-

The determinations were performed with the Beckman Coulter Synchron CX7 Clinical System automatic analyzer, Concept 2000 analyzer for electrophoresis of serum proteins. The patients were examined when admitted into the Internal Diseases Department of the Emergency Military Hospital, Constanta.

For all the determined analytes we calculated the mean, the standard deviation, minimum and maximum (Table 2); we performed the statistical "Student" test in order to evaluate the difference between the mean of an analyte from the two biological materials (serum, ascitic fluid) (Table 3). The results from the patients with cirrhosis were compared with those of the control group (15 healthy persons).

Results and Discussions

Cirrhosis is a life threatening disease, which appears when the fibrotic tissue replaces the healthy hepatic tissue. The fibrotic tissue can block the blood circulation from the intestine to the liver determining an increase of the pressure portal venous system (HTP), a complication that leads to the accumulation of ascites fluid in the abdominal cavity. The ascites and encephalopathy are due both to, the parenchymal and vascular decompensation. This experiment intends to biologically examine the serum and ascitic fluid in 15 patients with decompensated cirrhosis in order to establish correlations between the analyzed biochemical parameters and to assess the importance of these determinations related to the confirmation of the diagnosis or to the differential diagnosis.

Table 2 Mean value, standard deviation, minimum and maximum of analytes determined in serum and ascitic fluid in cirrhotic patients

	SERUM			**ASCITIC FLUID**		
Analyte	**M ± DS**	Minimum	Maximum	**M ± DS**	Minimum	Maximum
ALT (UI/L)	30.52 ±17.07	11.00	68.00	7.53 ±1.64	5.00	10.00
AST(UI/L)	56.68 ± 29.18	25.00	127.00	16.92 ± 7.12	8.00	31.00
AST/ALT	2.03±0.76	0.96	3.50	2.27 ± 0.86	1.12	4.28
LDH(UI/L)	519.4 ±157.73	320.00	866.00	120.8 ± 23.41	64.00	151.00
BT (mg/dl)	2.8 ± 1.5	0.60	6.30	0.87 ± 0.47	0.20	2.00
BD (mg/dl)	0.98 ± 0.68	0.10	2.40	0.23 ±0.13	0.10	0.60
BI (mg/dl)	1.81 ±0.91	0.50	3.90	0.66 ±0.34	0.10	1.40
GGT(UI/L)	132.52 ±95.49	36.00	303.30	29.82 ± 17.61	10.00	61.00
ALP(UI/L)	104.4 ± 36.68	56.00	180.00	22.74 ± 9.41	13.30	45.00
CT (mg/dl)	140.6 ± 54.50	59.00	265.00	22.2 ± 4.10	14.00	31.00
TG (mg/dl)	86.33 ± 32.71	36.00	140.00	18.73 ± 5.50	10.00	31.00
Urea (mg/dl)	45.0 ± 24.67	17.00	96.00	46.73 ± 28.96	20.00	103.00
Cr (mg/dl)	1.09 ± 0.33	0.60	1.70	0.96 ± 0.28	0.60	1.70
AcU (mg/dl)	6.2 ± 1.67	4.10	10.00	5.55 ± 1.33	3.60	9.00
A% (ELFO)	41.19 ± 6.54	30.9	56.59	44.68 ± 5.91	35.49	57.17
α_1% (ELFO)	4.06 ± 1.38	2.43	7.90	3.68 ±1.49	2.05	6.62
α_2% (ELFO)	8.5 ± 2.73	4.85	14.30	6.5 ± 1.86	4.12	10.55
β% (ELFO)	12.97 ± 3.61	8.14	23.47	15.82 ± 3.32	10.39	23.46
γ% (ELFO)	33.30 ± 7.65	17.83	48.61	29.23 ± 7.08	13.59	43.22
A/G (ELFO)	0.71 ± 0.21	0.44	1.30	0.82 ± 0.20	0.55	1.33
ALB (g/L)	26.6 ± 5.57	17.00	36.00	6.54 ± 2.19	1.20	10.00
CHE (UI/L)	2708.8 ± 767.7	1830.00	4451.00	393.2 ± 157.85	105.00	414.00
AMY (UI/L)	82.46 ± 28.97	16.00	124.00	32.26 ± 17.00	12.00	65.00
CK (UI/L)	62.2 ± 23.05	33.00	115.00	7.0 ± 2.44	3.00	12.00
Fe (μg/dl)	112.0 ± 55.83	41.00	197.00	28.0 ± 7.29	18.00	42.00
GLU (mg/dl)	88.02 ± 15.65	66.00	114.00	124.06 ± 12.38	107.00	145.00

C Reactive Protein (CRP) was analyzed for all 15 patients. The results were positive (≥6 mg/L), both in serum and in ascitic fluid (LA).

Table 3 The result of the Student test performed on the analytes determined in serum and ascitic fluid in cirrhotic patients (statistical significance)

Analyte: serum, LA	Statistical significance	Analyte: serum, LA	Statistical significance
ALT	$p < 0.05$	A%	$p > 0.05$
AST	$p < 0.05$	α_1-globuline %	$p > 0.05$
AST/ALT	$p > 0.05$	α_2-globuline %	$p < 0.05$
LDH	$p < 0.05$	β-globuline %	$p < 0.05$
BT	$p < 0.05$	γ-globuline %	$p > 0.05$
BD	$p < 0.05$	A/G	$p > 0.05$
BI	$p < 0.05$	AMY	$p < 0.05$
GGT	$p < 0.05$	CK	$p < 0.05$
ALP	$p < 0.05$	Fe	$p < 0.05$
CT	$p < 0.05$	Glu	$p < 0.05$
TG	$p < 0.05$	Uree	$p > 0.05$
ALB	$p < 0.05$	crea	$p > 0.05$
CHE	$p < 0.05$	AcU	$p > 0.05$

Hepatocytolisis is one of the ways the liver responds to different brutal assaults (infectious, toxic and anoxic) which lead to cytolysis; the various structures of the liver react differently to aggressions, the hepatocyte sector being the most fragile and the first to respond to aggressions.

Morphological changes of the hepatocyte are preceded by a biochemical lesion stage characterized by infrastructural and metabolic changes of the hepatocyte. In this stage the lesions are reversible.

The energy metabolism of the liver cell is disturbed which leads to an increase in the cell membrane permeability and the cytoplasmic components pass into the serum.

The hypoxia persistent inside the liver leads to the substitution of the liver cells with conjunctive tissue and so the sclerosis and cirrhosis are inducted.

The aminotranserases (ALT and AST) from the ascites fluid (LA) have values, which are inside the reference interval even for those patients whose serum values for the same enzymes are higher than LSN. In serum are predominating the normal values for ALT (86,6%) and the pathological values for AST (60%).

The means of serum AST/ALT and ascites fluid AST/ALT values do not differ significantly ($p>0.05$); the AST/ALT mean is higher in LA than in serum (the ascitic fluid ALT mean is approximately 4.2 times smaller than the serum ALT mean), and is higher in women compared to men (both serum and ascitic fluid values) (fig. 1). Therefore we can conclude that the necrotic lesion is larger in women and, from this point of view, the stage of cirrhotic disease is more

advanced in women than in men. The "de Ritis ratio" is important in the assessment of necrotic lesions but also in the evaluation of fibrosis in the context in which its value has also a growth tendency.

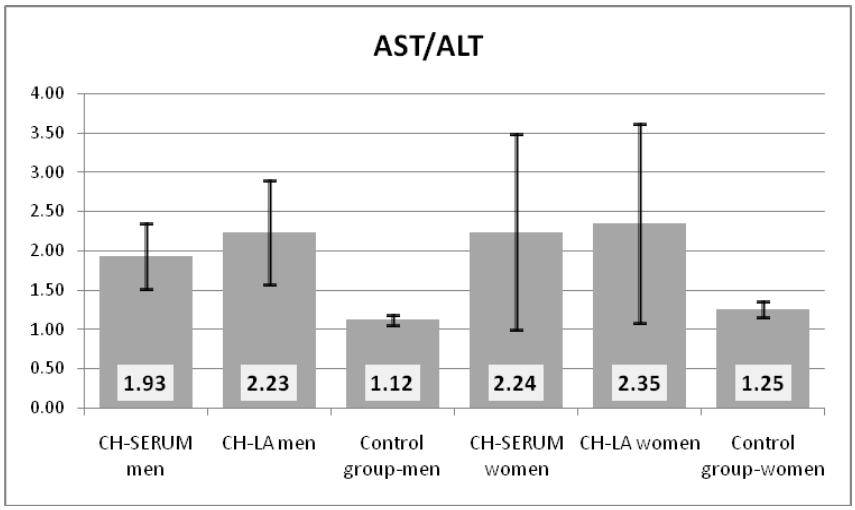

Figure 1 - AST/ALT activity in cirrhosis (serum, LA) and control group (serum)

Normal values of AT do not always exclude a liver disease**.** In severe parenchymal lesions the level of aminotransferases reflect the degree of hepatocyte damage from the determining moment and this is why we consider that dymanic serial determinations are more useful in the more accurate assessment of the evolutive degree of the hepatic disease (Başa et al., 2007), (Başa et al., 2010).
In 46.6% of the cirrhotic patients, serum LDH values exceed the LSN; in LA, the values are between 64 – 151 UI/L (the values are below the inferior normal limit (LIN) of the serum enzyme.
The LDH results obtained in ascitic fluid (which exclude the presence of a spontaneous or secondary bacterial peritonitis with a polymicrobial infection), but also the ratio ascitic LDH/ serum LDH that is not greater than 0.60 (the highest ratio is 0.36) exclude the malignant etiology of ascitic fluid. From the zymograms (table 4) it was observed that, although the total activity of LDH from ascitic fluid has low values, the LDH_4 and LDH_5 isoenzymes have values which exceed the serum reference range and their migration route is similar with that of the serum isoenzymes for both normal and increased total serum LDH activity (Başa et al., 2010). Therefore the level of LDH activity has special importance in determining the benign or malignant etiology of the ascitic fluid, and for the cytolysis evaluation from different developmental stages in cirrhosis is necessary to determine the activity of the LDH isoenzymes with hepatic origin.
Fe in LA has values between 18 and 42 μg/dl, but in serum it exceeds LSN in 26.6% of the patients, the highest value being 197 μg/dl, the respective patient

presenting increased activity for the other cytolysis enzymes. In 20% of patients, the serum iron was determined to be lower than the inferior normal limit; these patients are in an advanced stage of cirrhosis when anemia may also occur and the cytolysis is no longer expressed by the increase of the serum iron and AT values (Başa et al., 2009).
The mean of serum iron values is significantly different from the mean of ascitic fluid iron; the LA values were determined to be lower even than the inferior normal limit of the serum iron in women. So, considering that through cytolysis the iron is released from the hepatocyte, the determination of serum iron could useful in assessing the evolutionary stage of cirrhosis and of chronic hepatic disease in general if we take into account the fact that iron is involved in the processes of peroxidation, inflammation and fibrosis.

Table 4 The behavior of LDH isoenzymes in cirrhosis with ascites

Nr. Pacient	LDH Total (UI/L)	LDH_1 (%)	LDH_2 (%)	LDH_3 (%)	LDH_4 (%)	LDH_5 (%)
VN (ser)	266-500	16,10-31,50	29,20-41,60	17,0-26,20	5,90-12,30	3,20-17,30
P02- CH-ser	557	13,04	29,40	21,41	12,96	23,19
P02- CH -LA	118	17,79	27,30	20,68	14,02	20,21
P07- CH- ser	447	17,56	26,03	16,75	15,03	24,63
P07- CH-LA	112	10,53	32,40	23,66	14,29	19,12
P09- CH- ser	320	14,20	26,56	25,17	13,88	20,19
P09- CH-LA	115	18,50	28,44	21,09	12,50	19,47
P11- CH-ser	581	16,65	25,96	18,20	12,94	26,25
P11- CH-LA	149	19,96	24,22	19,20	13,07	23,55
P15- CH-ser	684	9,20	25,15	22,37	14,72	28,56
P15- CH-LA	119	15,47	28,03	20,07	14,53	21,90
M3-ser	402	21,87	35,80	20,81	6,42	15,10
M9-ser	468	24,21	32,64	21,21	8,25	13,69

Excreto-biliary syndrome

The appearance of jaundice in the progress of cirrhosis depends most frequently upon the extension of the hepatocytic necroses and the decrease of the functional remaining hepatocytic mass.
A value above 5 mg/dl of the serum bilirubin is correlated with an unfavorable prognosis (Pascu, 2004). Hepatocellular jaundice associated with cholestasis is present in 66.7% of cases, cholestasis non associated with jaundice is present in 13.3% of cases, hyperbilirubinemia non associated with the increase of cholestasis enzymes is present in 13.3% of cases and just for one of the cases the excreto-biliary syndrome is not biochemical expressed.

Cholestasis is accompanied by alterations in the hepatocyte membrane (the decrease of the membrane's fruidity and of the activity of ATP-ase-Na^+- K^+). The changes result in the inability of the hepatocyte to fix the bile acids failure and, because of this, the biliary flux is reduced (Hăulică, 2000).
The mean of BT values is more than three times over the average value observed in the control group for both sexes, and the means of BD and BI have increased 4.9 times and 1.8 times respectively compared to the normal maximal limit. For the two enzymes with predominantly biliary origin we observed the following: for GGT an increase of 7.34 times (men) and 5.70 times (women) over the average value obtained for the control group (fig. 2), and for ALP an increase of 1.12 times over the normal superior limit (LSN).
In cirrhosis and chronic hepatic diseases in general, the GGT level is correlated with that of ALP, both being sensitive indicators of hepatobiliary tract diseases. GGT increases concomitant with ALP in cholestatic forms and isolated in alcoholics. The increase of alkaline phosphatase level reflects the damage of hepatic excretory function.

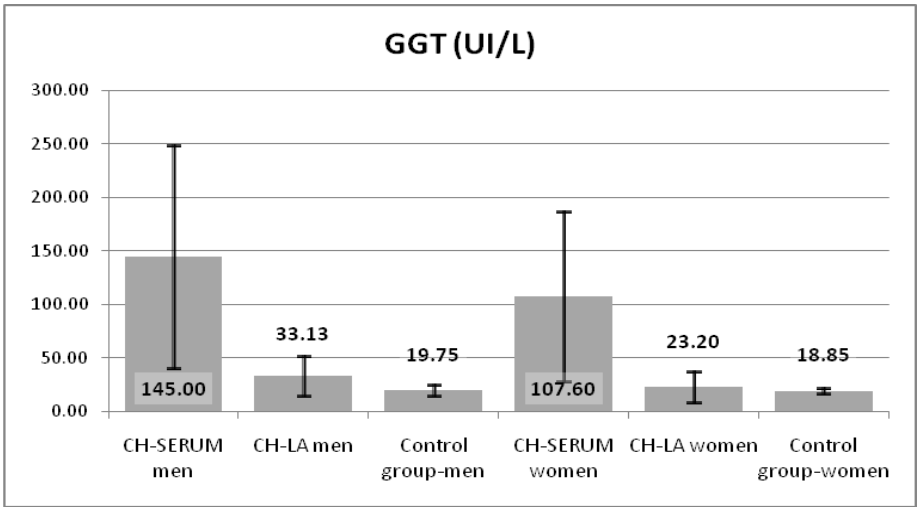

Figure 2 - GGT activity in cirrhosis (serum, LA) and control group (serum)

Lipid profile

Serum cholesterol is decreasing in decompensated hepatic cirrhosis, its low values having poor prognostic significance. The mean of serum CT values in hepatic cirrhosis differs significantly from the mean of CT values determined for the control group ($p<0.05$); this conclusion is valid only for men, for women the difference is not statistically different (fig.3); most probably, in the case of women, the liver failure, expressed also by the cholesterol decrease, occurs later

than in men. In cirrhotics, the difference between the means of serum and ascitic fluid CT is statistically significant for both sexes.

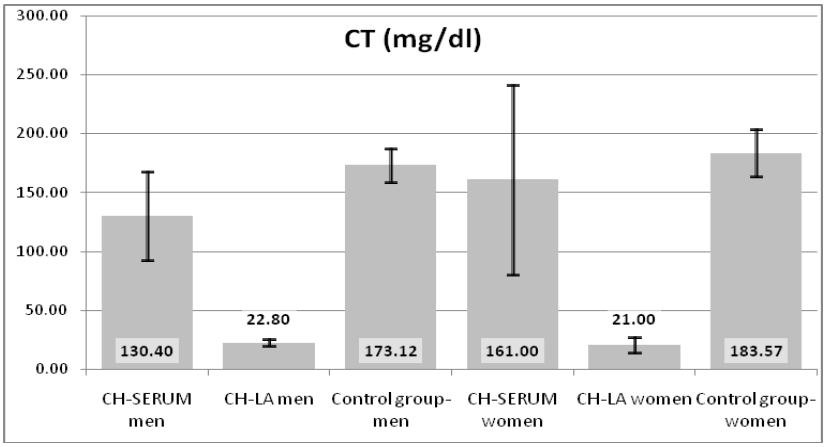

Figure 3 - CT concentration in cirrhosis (serum, LA) and control group (serum)

The cholesterol determination in LA has the greatest diagnostic utility in the differentiation of malignant ascites from the non-malignant ones, especially when the tumor cells are undetectable. Using the threshold of 45 – 48 mg/dl, which differentiates the cirrhotic ascites (with hepatic cause) from the malignant ascites (with peritoneal cause), we observe that all the obtained values for cholesterol from LA correspond to the cirrhotic ascites (fig. 4).

Triglycerides from ascitic fluid have lower values than the serum ones, a fact that excludes the chylous ascites which is caused by lymphatic obstruction (Ciurea et al., 2004); the differences between the serum and ascites fluid TG are statistically significant.

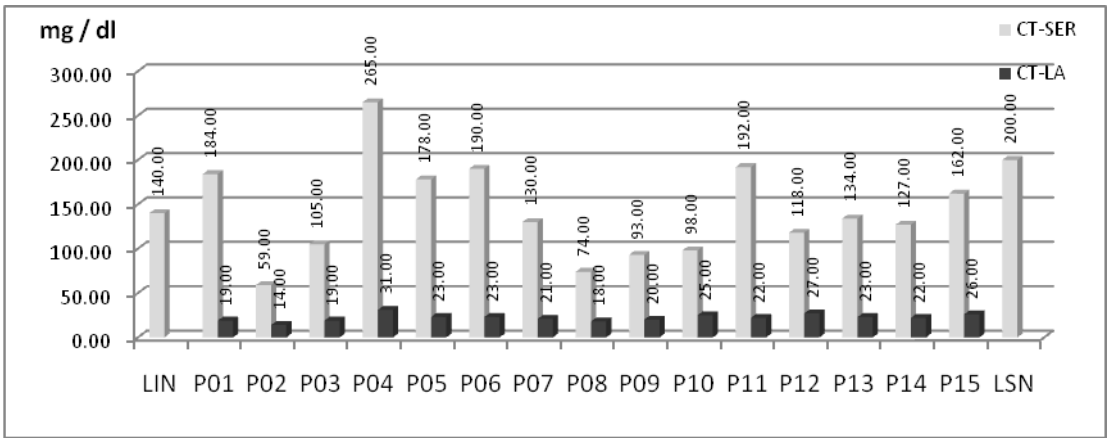

Figure 4 - Total cholesterol variation in serum (CT-SER) and ascitic fluid (CT-LA) in cirrhotics

Hepatoprive and mesenchymal inflammation syndromes

The symptomatology of the hepatic cirrhosis is determined by two major consequences of morphological restructuring: quantitative and functional reduction of the hepatic parenchyma, which leads to hepatic failure and the existence of portal hypertension.

The hepatoprive syndrome appears in the advanced stages of the disease, having predictive value with regard to the survival. The signs of hepatic failure are due to the synthesis deficits and to the lack of liver detoxification. The serum albumin concentration reflects the functional status of the hepatocytes and is useful in tracking the progression of liver disease. In ascitogenic cirrhosis, the liver protein synthesis is low (fig. 5). The albumin decrease is accentuating when the ascites occurs due to the increase of the venous pressure in the portal system over VN 5-10 mmHg or 10-14 cm H_2O manometric measured column; in our study the plasma albumins have an average value of 26,6 ±5,57 g/dl, and the ratio between the ascetic fluid and serum albumins is less than 0.41, which excludes the malignant ascites.

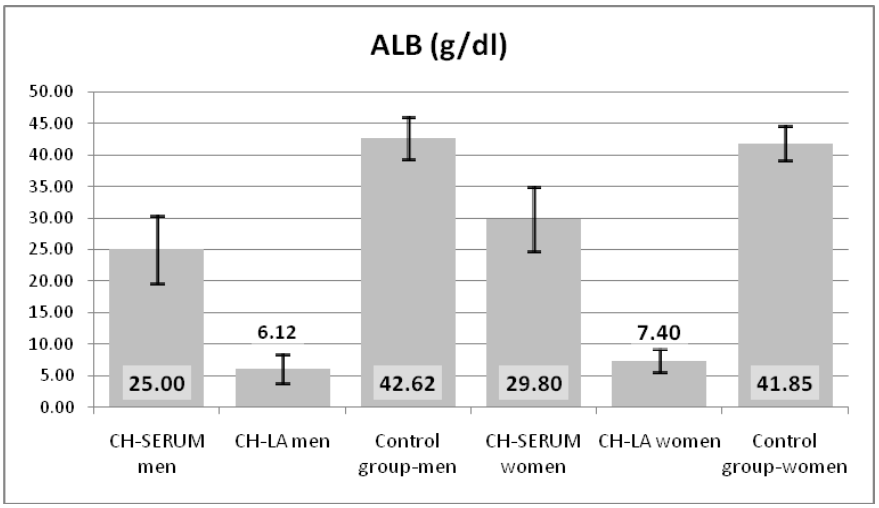

Figure 5 - ALB concentration in cirrhosis (serum, LA) and control group (serum)

The gradient serum albumin/ ascetic fluid albumin has a higher value for the differentiated diagnosis compared to the transudate-exudate concept because the albumin gradient is directly correlated with portal hypertension and, unlike the total proteins determination, is not influenced by diuretic therapy or paracentesis. The patients from our study register values higher than 1.1 g/dl for this gradient, and they have HTP, hypertension that is mentioned also in the result of the abdominal ultrasound. On the other hand, the result of this gradient also excludes: carcinomatosis (most frequently), peritoneal TBC, pancreatic or biliary ascites,

nephrotic syndrome, heart attack or intestinal obstruction, serositis, cases in which the gradient would have been under 1.1 g/dl.

The progressive decrease of pseudocholinesterasis, parallel with the decrease of albumin, signifies the formation of advanced and irreversible lesions, the respective analytes being significant for the prognostic. The CHE decreasing degree is directly proportional with the intensity of the hepatic lesions (fig.6).

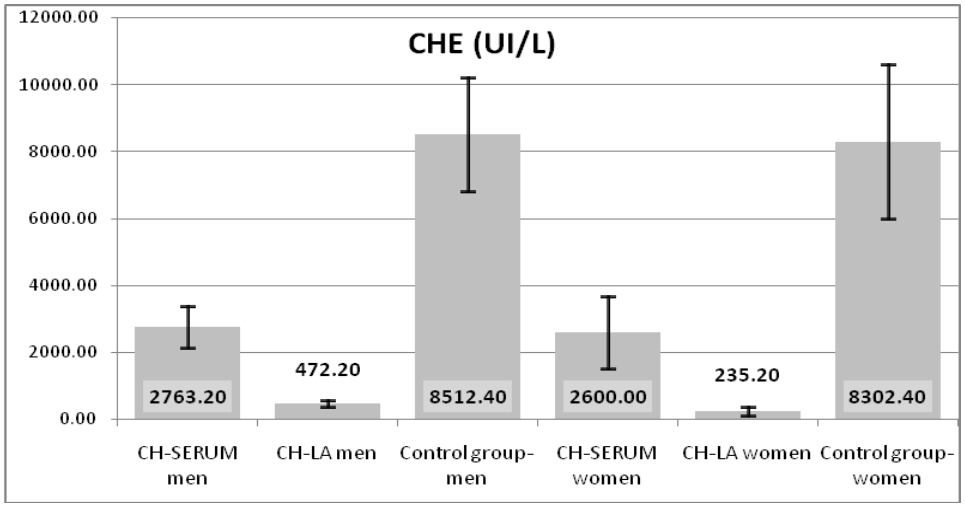

Figure 6 - CHE activity in cirrhosis (serum, LA) and control group (serum)

Cirrhotics cannot maintain an appropriate concentration of the osmotic pressure through albumin synthesis deficiency, fact which boosts the appearance of ascites. Pathological alterations of the plasmatic proteins determine the colloidal plasma instability, optimal ratio of albumin and globulin is not maintained: in cirrhosis, the stabilizing factors (albumin and alfa$_1$-globulins) are decreasing and the factors which favor the precipitation (γ-, β, α_2- globulins) are increasing.

A/G ratio is in the pathologic range both for serum and ascitic fluid determinations. Along with the decline of albumin synthesis, the A/G ratio is decreasing; in the electrophoresis of proteins both plasmic and from ascitic fluid, the percentage values of albumin are decreasing as the γ globulins are increasing. The serum level of globulins indicates the presence of inflammation and is evolving parallel with its severity. The increase of gamma-globulins expresses quite accurately the activation of the hepatic mesenchyme, this increase being directly proportional with the inflammation degree of the mesenchyme.

Hypergammaglobulinemia is an indicator of an inflammatory process which interests the liver and which occurs as a result of antigenic hyperstimulation of the immune system through poor phagocytosis of the antigens from the intestinal tract in SRE from the liver, consecutive to the shunts development (Friedman et al.,

2003), (Zălaru et al.,.Globulin level measurement is often used as an indicator of the reticuloendothelial cell deficit from the hepatic sinusoids to remove the intestinal tract antigens from the portal venous blood.

Carbohydrate homeostasis, pancreatic function, muscle activity

In cirrhotics, à jeune, the energy provided by carbohydrates decreases in a high degree (2% compared to 38% in the control group) while the energy resulted from lipids increases (86% compared to 46% in the control group), and this is possible through the release of hepatic glucose or through the decrease of glycogen reserves in the liver.

After eating, though, cirrhotics, more than the healthy witnesses, immediately utilize the carbohydrates due to the reduced liver storage capacity and after that mobilize the energy from triglycerides. Glucose tolerance tests p.o and i.v. are altered in cirrhosis through the different capacity of the liver to regulate the carbohydrate homeostasis as discussed by Buligescu (Buligescu, 1999). Taking into account the variant that a secondary peritonitis can coexist with a polymicrobial infection, the glucose determination in ascitic fluid has a special importance, knowing that its level decreases less than 50 mg/dl in these types of cases. The determinations of glucose in ascitic fluid made for our study, as well as those of the LDH activities exclude this complication.

Acute pancreatitis is excluded for the patients included in our study (the ratio between ascites fluid AMY and serum AMY is lower than 2). Serum amylase decreases in severe hepatic failures. The mean of serum AMY values differs significantly from the mean of ascites fluid AMY values ($p<0.05$.). The level of AMY and ALP cam is used to detect lesions of the pancreas or small intestine. For the entire studied group of patients we did not detect in ascites fluid any AMY or ALP values that exceed the serum levels, thus the ascites can be attributed to liver cirrhosis. The amylase concentration in ascites fluid is useful for detecting pancreatic ascites and perforations with secondary bacterial peritonitis

In accordance with the obtained results we note that in liver cirrhoss the CK serum activity decreases towards the inferior normal limit, especially in terminal stages, and the ascites fluid CPK values may not be relevant in the diagnosis of liver cirrhosis.

Renal function

Disorders of albumin synthesis and catabolism in cirrhotic liver (alcoholic and viral) constitutes a cause for hypoalbuminemia which is responsible for the decrease of oncotic intracapillary pressure followed by the water transfer in the interstices leading to the appearance of ascites, edemas or both. The water transfer is accompanied by the transfer of some compounds with low molar mass (urea, sodium, phosphate ion) or high molar mass (albumin, cytokines, insuline etc.).

Serum creatinine is a more specific and sensitive indicator of the renal function compared to urea; a disturbance of the renal function reduces the excretion of creatinine, determining the increase of serum creatinine, whose blood value varies only in relation to its renal elimination, thus representing an important parameter in the determination of the functional capacity of the kidney. The increase of the creatinine level from blood is usually the first sign of acute renal failure (Henriksen, 1995).

Persistently elevated levels of serum urea denote a significant alteration of the glomerular filtration rate, which represents a useful instrument in the diagnosis of renal failure (Battaler et al., 1998).

The values from ascitic fluid of urea, creatinine and uric acid are about at the same concentrations with those from serum (figure 7, 8, 9,10,11).

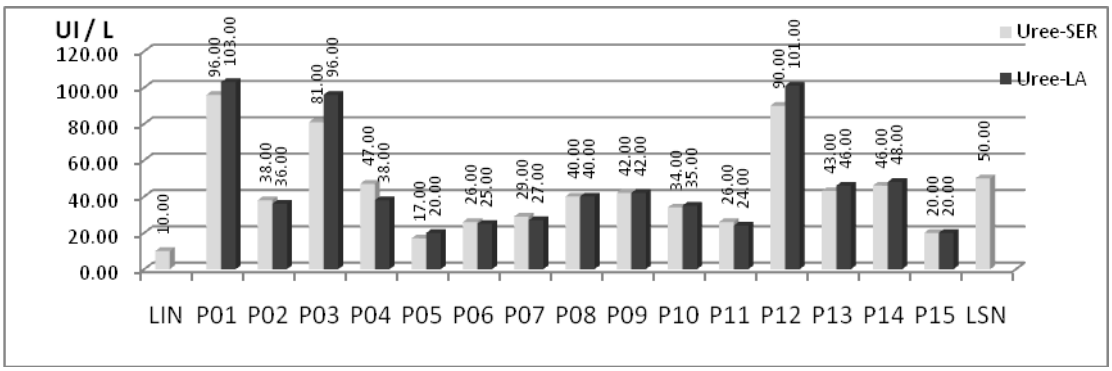

Figure 7 - Urea variation in serum (Uree–SER) and in ascitic fluid (Uree–LA) in cirrhotics

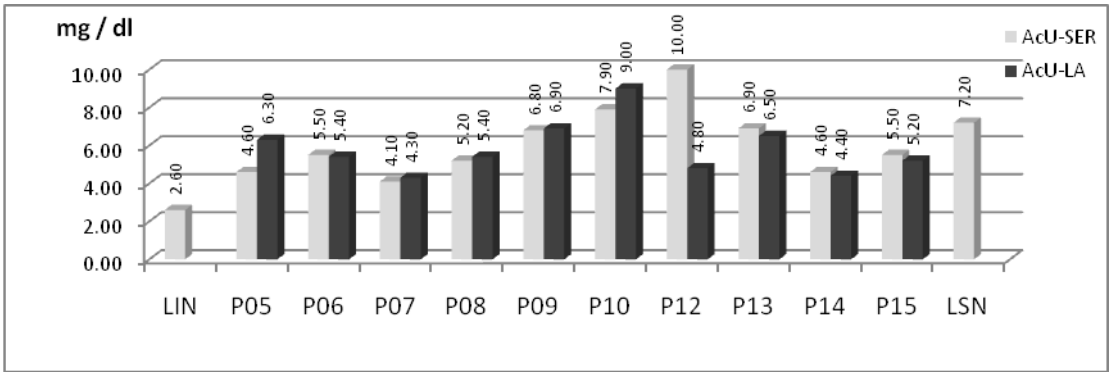

Figure 8 - Uric acid varioation in serum (AcU–SER) and in ascitic fluid (AcU–LA) in cirrhotics-men

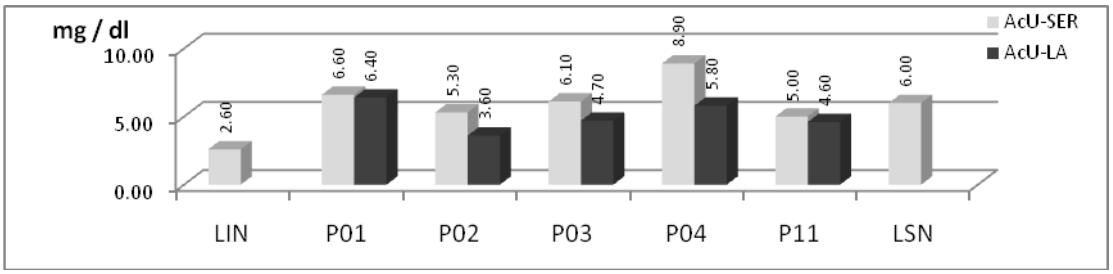

Figure 9 - Uric acid varioation in serum (AcU–SER) and in ascitic fluid (AcU–LA) in cirrhotics-women

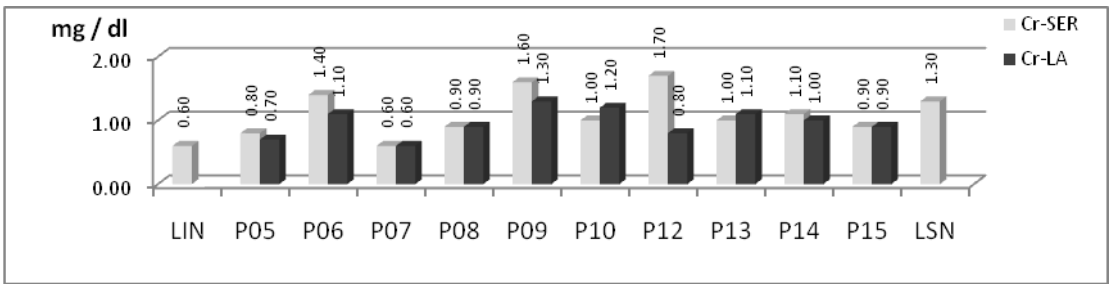

Figure 10 - Creatinine variation in serum (Cr–SER) and in ascitic fluid (Cr–LA) in cirrhotics-men

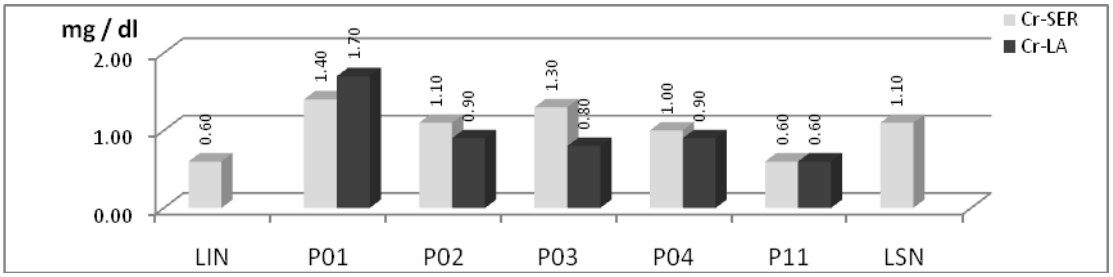

Figure11 - Creatinine variation in serum (Cr–SER) and in ascitic fluid (Cr–LA) in cirrhotics-women

Conclusions

1. Normal values of ALT, compared with AST, are found in a higher number of cirrhotic patients with ascites, the AST enzyme being biloclar and in cirrhosis we observe a predominance of structural alterations of mitochondria along with a functional alteration of the same mitochondria and the decrease of ATP quantity.
2. De Ritis ratio in serum is not statistically very different from that in ascites fluid, although the means of AST and ALT values in serum versus

ascites fluid are significantly different from a statistical point of view; as a result, the value of this ratio, both in serum and ascites fluid, is important when evaluating the degree of cytolysis, necrosis and even hepatic fibrosis.

3. The values of LDH in LA don't increase LIN for seric enzyme, so determination of total activity of LDH and of zimograme LDH, in ser and in ascites fluid has an importance in confirmation of diagnostic but also in evaluation of hepatocytolisis level.

4. In excreto-biliary syndrome, all the analytes we determined in ascites fluid (BT, BD, BI, GGT, and ALP) do not present values higher than those determined in serum; this determination is important for the differential diagnosis.

5. Serum cholesterol decreases in decompensate cirrhoses, its low values (60%) being significant in unfavorable prognosis; CT values in LA correspond to cirrhotic ascites, not exceeding the limit, which separates the malign ascites from the cirrhotic ones.

6. The values of gamma globulins in serum and ascitic fluid do not differ significantly, because the fractions have a similar migration path and their level indicates an increased immunological reactivity and a high degree of inflammation of the hepatic mesenchyme; the determinations from ascites fluid comes useful in the confirmation of the cirrhotic etiology of the ascites fluid.

7. The A/G ratio is higher for the values obtained from the protein electrophoresis in ascitic fluid, compared to the serum values (the albumins that reached the ascitic fluid are in a bigger proportion than globulins), but the mean is not statistically different, thus the hepatic failure and the mesenchymal inflammation in decompensate cirrhosis is expressed also by the A/G ratio from the ascitic fluid.

8. The result of the ratio ascitic fluid albumins/serum albumins from our study confirms the cirrhotic ascites and excludes the malignant etiology.

9. Taking into account the fact that the differences between the means of urea, creatinine and uric acid from serum and ascitic fluid are not statistically significant, we consider that, by determining these parameters only in ascitic fluid, it is possible to establish the presence of renal failure in the decompensate cirrhosis.

10. Portal hypertension is evidenced by the value of albumin gradient between serum and ascitic fluid.

11. Exploratory paracentesis accompanied by an appropriate serological of the ascites fluid is a rapid method for etiologic diagnosis of ascites.

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