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Different Models of Decompensation in Alcoholic versus Nonalcoholic Cirrhotic Patients

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Abstract: ***Background: Worldwide, liver cirrhosis is a significant cause of global health burden and is considered to be the top ten leading cause of death. Ascites is the most common of the three major complications of cirrhosis, followed by esophageal bleeding and encephalopathy. Aim: The aim of the study is to evaluate the different models of decompensation according to the etiology of liver cirrhosis. Patients and Methods: 200 cirrhotic patients hospitalized during 2011 - 2014 in our clinic, divided in two main groups (alcoholic and non-alcoholic etiology), were retrospectively analyzed. Hepatic decompensation was confirmed as presence of ascites, jaundice, hepatic encephalopathy (HE), esophageal varices bleeding, hepatorenal syndrome (HRS), hepatocarcinoma HCC), or spontaneous bacterial peritonitis (SBP). Results: Alcoholic cirrhosis was present in 51% of patients. Male dominated in alcoholic cirrhosis 98% vs 62% in nonalcoholic group (HBV, HCV, cryptogenic, autoimmune), while female dominated in non-alcoholics (38% vs. 2%), p<0.05). Ascites is the main complications in alcoholic cirrhosis (93% vs. 73%, p<0.05), while HCC and death dominated in nonalcoholic group (24% vs. 5.8% and 36.7% vs. 23.5%; p<0.05) respectively. Conclusions: Ascites is the leading initial pattern of decompesation in alcoholic cirrhosis whereas HCC and high mortality dominates in non-alcoholics.***

Keywords: livercirrhosis, cirrhosis decompensation, aetiology, Child-Pough score

1. Introduction

Liver cirrhosis is a significant cause of global health burden, with more than one million deaths in US, with high and/or rapidly increasing mortality [1]. Liver cirrhosis is a major yet largely preventable and underappreciated cause of global health loss. Variations in cirrhosis mortality at country level reflect differences in prevalence of risk factors such as alcohol use and hepatitis B and C infection. Preventive measures to control and reduce liver cirrhosis risk factors should be urgently strengthened [1].

Cirrhosis is defined as the histological development of regenerative nodules surrounded by fibrous bands in response to chronic liver injury, which leads to portal hypertension and end-stage liver disease. Recent advances of the natural history of cirrhosis and in treatment of its complications , have resulted in improved management, quality of life, and life expectancy of patients [3]. The decompensation in liver cirrhosis can be attributed to portal hypertension (ascites, esophageal varices bleeding) or lack of the hepatic function (jaundice, encephalopathy, hepatocarcinoma) [2, 11, 12]. There are several histological models of liver cirrhosis. The histological models of injury in liver cirrhosis varies due to the etiology. Perisinusoidal fibrosis is dominant in alcoholic liver disease versus periportal fibrosis in viral or autoimmune liver disease [3]. It is well known the fact that different models of fibrosis can lead to different models of decompensation [3]. Ascites is the most common of the three major complications of cirrhosis, followed by variceal bleeding and encephalopathy. In our study we assessed different models of hepatic decompensation and their prognostic relevance in alcoholic cirrhotic versus non-cirrhotic patients and analyzed cirrhotic patients at their first admission in our clinic. Ascites is the most common complication of cirrhosis, and 60% of patients with compensated cirrhosis develop ascites within 10 years during the course of disease. The development of ascites is associated with a poor prognosis and impaired quality of life in patients with cirrhosis [3, 13, 16]. Thus it is interesting to evaluated the different patterns of hepatic decompensation and their relevance in alcoholic versus nonalcoholic liver cirrhosis in the Albanian context.

2. Materials and Methods

Two hundred patients with liver cirrhosis admitted to gastrohepatology Service, Durres Hospital between the years 2011 - 2014, were retrospectively analyzed for their first hepatic decompensation Hepatic decompensation was confirmed as presence of ascites, jaundice, encephalopathy, esophageal varices bleeding, hepatorenal syndrome, hepatocarcinoma or spontaneous bacterial peritonitis. Hepatic decompensation has been defined as occurrence of one of several complications of liver cirrhosis according to the Child-Pough score. Several pattern of decompensation were analyzed: ascites (confirmed by ultrasound), jaundice (bilirubin >3 times the upper limit of normal), varices esophageal bleeding (endoscopic report), encephalopathy (clinical evaluation), hepatocellular carcinoma (diagnosed by MRI or CT scan), hepatorenal syndrome (verified as reduction in GFR and renal plasma flow) in the absence of other cause of renal failure.

Etiologies of cirrhosis were classified as alcohol-related and non- alcohol-related. The nonalcoholic cirrhosis included hepatitis B (HBs Ag positive) or hepatitis C (HCV-RNA), autoimmune hepatitis and cryptogenic cirrhosis. Chi square and Mann-Whitney U-test were used to analyze the collected data. A $p < 0.05$ was considered significant.

3. Results

The males consists of 161 (80.5%) and 39 females (19.5 %). The mean age was 58 year with standard deviation of 7.4.

Table 1: Baseline characteristics of the subjects included in the study

Demographic/ lifestyle factors	**n**	**%**
Age at enter point		
25-44	19	9.50%
45-64	126	63%
65+	55	27.50%
Gender		
Male	172	84%
Female	28	16%
Aetiology		
Alcohol	102	51%
Viral hepatitis	80	40%
HbsAg+	66	33%
HCV+	14	7%
AI LD	2	1%
Kriptogen	16	8%
Complications		
Ascitis	165	82.50%
Oezophageal varices	148	74%
Variceal bleeding	46	23%
EH	108	54%
HRS	57	28.50%
HCC	30	15%

In the table 1 it is presented the overall characteristic of the subject including in the study. Related to age at the moment of diagnose, we found 19 patients (9.5%) of age 25-44, 126 patients (63%) at 45-64; 55 patients (27.5%) over 65 years. Related to etiology, we found 102 patients (51%) with alcohol cirrhosis, hepatitis B in 33%, hepatits C in 7% of patients, autoimmune etiology in 1%, and cryptogen cirrhosis was in 8% of patients. According complications ascites was the leading cause of decompensation in 165 patients (82.5%), esophageal varices were in 148 pt (74%), while esophageal varices bleeding occurred in 46 patients (23%), HE in 108 patients (54%), HRS in 57 patients (28.5%) and HCC in 30 patients (15%).

In the table 2 is presented the distribution of subject according the sex and cirrhosis etiology. Alcoholic cirrhosis was present in 102 patients, 98% of them were males, 2% females. Non-alcoholic cirrhosis was seen in 98 patients, 62% males and 38% females. The male predominated in alcoholic cirrhosis 98% *vs.* 62% in nonalcoholic group (HBV, HCV, cryptogenic, autoimmune), while females presented mainly in non-alcoholic cirrhosis (38 % vs 2%), *p <0,05*

Table 2: Distribution of sample according the sex and cirrhosis etiology

	Alcoholic (102)		Nonalcoholic (98)		p value*
	n	%	N	%	
Gender					0.001
Male	100	98	61	62	
Female	2	2	37	38	

**chi square*
p < 0.05 is considered significant

Table 3: Distribution of sample according to the CHILD classification

	n	%	p value
CHILD Classification			0.001
Child A	26	13	
Child B	79	39.5	
Child C	95	47.5	

**chi square*
p < 0.05 is considered significant

In the table 3 is presented the distribution of subject according the Child classification. Patients in Child C dominated *vs* Child B and A: 47.5% *v.s.* 39.5% and 13% respectively and showed poor prognosis and high mortality in non-alcoholic group (36.7% vs. 23.5%).

Table 4: Child Classification related to etiology of cirrhosis

	Aetiology				
	Alcoholic		nonalcoholic		
	n	%	N	%	p value*
CHILD Classification					0.105
Child A	15	14.7	11	11.2	
Child B	46	45	33	33.6	
Child C	41	40.3	54	55.2	

**chi square*
p < 0.05 is considered significant

In the table 4, it is shown distribution of subject according etiology of the cirrhosis and Child classification. The alcoholic cirrhosis was present in 51% of cases, where 14.7% were in Child A; 45% in Child B and 41% in Child C vs. 11.2%; 33.6% and 55.2% respectively in non-alcoholic group. However, there is no significant difference on the distribution of cirrhosis case by etiology (chi square, p>0.05).

Table 5: Distribution of complications related to etiology

	Alcoholic (102)		No-alcoholic (98)		p value*
Complication	n	%	N	%	
Ascitis	95	93	72	73	0.001
Esophageal varices	80	78	68	69	0.145
Variceal bleeding	26	25.4	20	20.4	0.393
HCC	6	5.8	24	24.4	0.001
Hepatic coma	56	54.9	52	53	0.795
HRS	26	25.4	31	31.6	0.345
Death	24	23.5	36	36.7	0.041

**chi square*
p < 0.05 is considered significant

As it is shown on the table 5, ascites is the main complication in alcoholic cirrhosis (93% vs. 73%, p<0.05), while HCC and death dominated in nonalcoholic group (24% vs. 5.8% and 36.7% vs. 23.5%, p<0.05) respectively. There is a significant poor prognosis and high mortality in non-alcoholic group (36.7% vs. 23.5% p<0.05).

Table 6: Survival related to etiology of cirrhosis

	etiology				
	Alcoholic		Nonalcoholic		p value*
	median	interquartile range	median	interquartile range	
age (year)	55	15	59.5	18	0.013
time to death	1	8	1.5	3	0.169

**Mann-Whitney U-test*

As it is shown on the table 6, patients with alcoholic cirrhosis are younger than non-alcoholics with male predomination in alcoholic group, $p<0.05$. There is not any significant difference on the time to death by the cirrhosis etiology.

4. Discussion

This study analyzed the differences between the first decompensation in alcoholic vs nonalcoholic liver cirrhosis, because the clinical management and prognosis of cirrhotic patients may differ according to etiology and subsequent complications after hepatic decompensation. Patients with alcoholic liver cirrhosis predominated and were significantly younger than patients with non alcoholic cirrhosis. Alcohol is the leading etiology in several studies investigating the course of liver cirrhosis. Medical reasons for the non alcohol group are the slow progression of natural course of viral liver disease and effective antiviral treatment which prevent development and decompensation of cirrhosis [4]. Patients with alcoholic liver cirrhosis developed more often and more severely ascites than cases with nonalcoholc liver cirrhosis. Two different study (Danish and Norwegian) evidenced ascites as the leading initial hepatic decompensation in 55% and 67% of cases respectively [10, 11,15]. Also a Spanish study observed higher rates of ascites in alcoholic cirrhosis (59 %) [17]. In contrast to the decompensation pattern in alcoholic cirrhosis, hepatocellular carcinoma was the dominating complication in non-alcoholic cirrhosis. Although some authors did not observe a difference in 10 years survival between alcoholic and non-alcoholic liver cirrhosis other investigators described a better or poorer survival for alcoholics compared with other etiologies. These differences may be related to the treatment in non-alcoholic patients [5]. Mortality from cirrhosis of the liver has been examined in few long-term follow-up studies. The 10-year relative survival was worse in patients with alcoholic cirrhosis (34%) or nonspecified cirrhosis (32%) patients with cirrhosis of the liver face reduced life expectancy due to several causes of death [5]. New data on defining HCC risk have emerged for hepatitis B virus, hepatitis C virus, and autoimmune hepatitis. Surveillance is deemed cost-effective if the expected HCC risk exceeds 1.5% per year in patients with hepatitis C and 0.2% per year in patients with hepatitis B [12, 14].

5. Conclusions

Ascites is the leading initial pattern of decompensation in alcoholic cirrhosis whereas hepatocellular carcinoma and high mortality dominates in non-alcoholics. Survival after development of ascites: Ascites limits the prognosis in non-alcoholic cirrhosis. Occurrence of ascites in non-alcoholic cirrhosis should be considered as a negative factor of prognosis and show an early death compared to alcoholic group. Finally, as it can be seen patients with non-alcoholic cirrhosis do not die faster than alcoholic patients after the development of ascites for the first time.

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