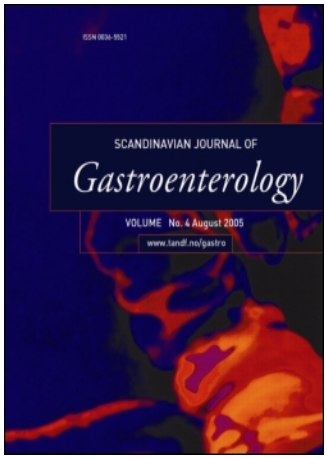

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ORIGINAL ARTICLE

Different hemodynamic patterns of alcoholic and viral endstage cirrhosis: Analysis of explanted liver weight, degree of fibrosis and splanchnic Doppler parameters

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Abstract

Objective. In cirrhosis, portal hemodynamics is usually considered independently of the disease etiology. The objective of this study was to investigate the role of the etiology of liver disease on the relationship between liver blood flow and liver pathology in endstage cirrhosis. ***Material and methods.*** Portal blood velocity and volume, congestion index of the portal vein, and hepatic and splenic pulsatility indices were evaluated with echo-Doppler in cirrhotic patients immediately before liver transplantation. When a patent paraumbilical vein was present, its blood flow was measured and effective portal liver perfusion was calculated as portal blood flow minus paraumbilical blood flow. The hemodynamic parameters were correlated with liver weight and the pattern of the liver fibrosis morphometrically assessed in explanted livers. A total of 131 patients with alcoholic or viral cirrhosis were included in the study. ***Results.*** In alcoholic cirrhosis, liver weight was higher than that in viral disease (1246 ± 295 g versus 1070 ± 254 g, $p = 0.001$), portal liver perfusion per gram of liver tissue was lower (0.49 ± 0.36 ml g^{-1} min^{-1} versus 0.85 ± 0.56 ml g^{-1} min^{-1}, $p = 0.004$) and hepatic pulsatility indices were higher (1.45 ± 0.31 versus 1.26 ± 0.30, $p = 0.018$). The degree of liver fibrosis was similar in alcoholic and viral cirrhosis ($11.7 \pm 5.5\%$ versus $11.0 \pm 4.4\%$, $p =$ NS). An inverse relationship between liver weight and Child-Pugh score was disclosed in viral ($p < 0.001$) but not in alcoholic disease. ***Conclusions.*** A different hemodynamic pattern characterizes the advanced stage of cirrhosis of alcoholic and viral origin. A more severe alteration of intrahepatic portal perfusion, probably coexisting with a more severe hepatocyte dysfunction, and a higher liver weight can be detected in alcoholic cirrhosis.

Key Words: *Cirrhosis, Doppler US, etiology of cirrhosis, hepatic circulation, liver fibrosis*

Introduction

The role of the etiology of liver disease on the relationship between intrahepatic portal flow and liver pathology in cirrhosis has not yet been defined. In fact, after the onset of cirrhosis, the relationships between liver volume and the severity of the disease, liver fibrosis and portal hemodynamics are generally considered as independent of the etiology of the disease [1,2], but no study has specifically been performed to assess this issue in the advanced stage of cirrhosis.

Hepatic function is considered to be dependent on liver volume in patients with chronic liver disease. This idea is supported by the clinical observation of a poor prognosis in cirrhotic patients with liver atrophy and by a series of studies, which reported a relationship between liver volume and hepatic function, in both experimental [3,4] and human studies [5]. Moreover, several papers have demonstrated that hepatic function and volume can be influenced by portal blood flow (PBF) [3,6,7]. In addition, a low or absent PBF is associated with liver atrophy, such as in the experimental model of portacaval shunts [8], and, conversely, PBF is a limiting factor in liver regeneration after liver resection [9,10], after living-related liver transplantation

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[11–13] or after preoperative selective portal vein embolization [14].

Taking advantage of the opportunity to measure the liver weight and the degree of liver fibrosis in patients undergoing orthotopic liver transplants (OLT), in whom a splanchnic Doppler evaluation had been previously performed, we analyzed the relationships between the etiology of the cirrhosis (viral or alcoholic) and splanchnic hemodynamics. In a group of patients, a histomorphologic analysis of tissue wedges of explanted liver was also performed, to measure the amount of liver fibrosis and to study the relationship between etiology, portal flow and fibrosis.

Material and methods

Design of the study

A chart review was performed in all patients aged 18 years or older who underwent OLT and who were referred to our department between January 1993 and January 2003. In all patients, an echo-color-Doppler evaluation of the splanchnic vessels performed on site was a prerequisite for all potential OLT candidates, and it was usually repeated every two to three months until the time of OLT. Only patients with viral or alcoholic cirrhosis were included in the study. Patients with intrahepatic tumor, portal thrombosis, transjugular intrahepatic portosystemic shunt (TIPS) or portacaval shunts were excluded. The following data were obtained from the chart review: patient demographics, etiology of liver disease, presence of intrahepatic tumor, patient's height and weight, body mass index (BMI), the parameters included in the Child-Turcotte-Pugh (CTP) classification [15], the CTP score [15], the weight of the explanted liver, echo-Doppler measurements: portal blood flow mean velocity (PBV), PBF, congestion index (CI) of the portal vein, effective portal liver perfusion (PLP), representing portal flow really perfusing the liver [16] and resistance indices of the liver and the spleen. Doppler measurements analyzed in the study were those of the last evaluation before OLT. The CTP score was used as an estimate of the clinical stage of the disease and of the residual hepatic function.

Each patient included in the study from January 2002 to January 2003 underwent a morphometric assessment of liver fibrosis (expressed as the ratio between the area occupied by fibrotic and epithelial tissue) in eight randomly chosen liver tissue samples (one sample per segment). The study protocol conformed to the ethics guidelines of the 1975 Declaration of Helsinki.

Methods of measurement

Measurement of echo-Doppler parameters. A Toshiba Sonolayer SSA-270A (Toshiba, Tokyo, Japan) with color-Doppler sonography and a 3.75 MHz sector electronic probe was used initially, and a HDI 5000 (ATL, Seattle, Washington) with color-Doppler sonography and a broadband curved array transducer (C5-2 40R) was used after October 1998.

PBV was evaluated as time averaged maximum velocity multiplied by 0.57, assuming the portal velocity profile as parabolic, as previously reported [17,18]. PBF was obtained by multiplying the portal vein cross-sectional area, assuming a circular shape of the portal vein section, by PBV [17,18]. CI was measured in accordance with Moriyasu et al. [19] as the portal vein cross-sectional area divided by PBV. When a patent paraumbilical vein was present, its blood flow was measured and PLP was calculated as PBF minus paraumbilical blood flow [16]. Arterial Doppler resistance indices were measured as the pulsatility index (PI) = (peak systolic–end diastolic velocity)/mean velocity [20]. Hepatic PI was measured in the left and right intrahepatic branches of the hepatic artery [21] and splenic PI was measured near the hilum of the spleen [18].

Measurement of liver weight. After OLT, the native liver was weighed to the nearest gram after the attached ligaments, gallbladder, portal structures, and extraneous tissue had been freed by dissection. No attempt was made to remove the blood from the explant [22]. After being weighed, the liver was formalin fixed, to allow histologic examination. PLP per gram of liver tissue was calculated as PLP/liver weight.

Pathology study

Explanted cirrhotic livers were fixed in 10% formalin for 24–36 h (changing the fixative liquid every 12 h). After fixation, macroscopically to detect any liver "dominant" nodule exceeding 1 cm in its largest diameter, the liver was serially sectioned (i.e. gross sections 1 cm thick). For the histology study, each hepatic segment was randomly sampled (additional samples being taken from any "dominant" nodule). The tissue samples were embedded in paraffin and histological sections (4 micron thick) were obtained and stained with H&E, Perls, periodic acid-Schiff (PAS) after diastase digestion, and van Gieson stain for collagen. In order to evaluate the degree of liver fibrosis, van Gieson-stained histology specimens were used for the morphometric measurements (the principle behind morphometry is based on different colors of hepatocytes and collagen

fibers). Morphometric measurements of the epithelial/collagen ratio were performed by means of a computerized image analysis system including a photomicroscope (Axioscope model; Zeiss-Jena, Oberkochen, Germany) connected to a 3CCD-JVC color video camera (KY55BE model; JVC, Yokohama, Japan). Images were acquired by means of a frame grabber (Kontron, Eching-Munchen, Germany). Measurements were taken by using green light obtained from an interference (540 ± 10 nm) pass band filter (Schott, Mainz, Germany). Measurement strategy was the following: each of the 8 histological specimens (1 per segment; 8 specimens in total) was scanned at a $100\times$ magnification power, randomly choosing a starting point. Continuously moving along an ideal right line, 16 non-overlapping microscopic fields per specimen were considered (total: 128 fields for every explanted liver). Degree of fibrosis was measured as the ratio (percentage) of the area of fibrosis of the scanned liver tissue and expressed as the mean and standard deviation (SD) in the considered microscopic fields. To minimize the interobserver variation, all the morphometric evaluations were performed by the same investigator (C.M.), who was blinded to any clinical information.

Statistical analysis

Results are reported as mean values $\pm$ SD. Differences between viral and alcoholic cirrhosis were assessed using Student's t-test for unpaired samples and the chi-square test. Correlations were investigated by the least squares method for continuous variables and by non-parametric correlation (Spearman) for discrete variables. Liver fibrosis was expressed as the percentage of fibrosis in relation to the whole liver tissue. The heterogeneity of fibrosis in each liver was measured as the coefficient of variation of the degree of fibrosis calculated in the 128 microscopic fields ($CV = (SD/mean) \times 100$). A p-value of less than 0.05 was considered significant.

Results

Among the 252 OLTs performed during the study period, liver weight was measured in 230 patients. Forty patients were excluded because they had not been transplanted for alcoholic or viral cirrhosis: acute liver failure ($n=8$), primary biliary cirrhosis ($n=5$), sclerosing cholangitis ($n=7$), mixed etiology (biliary and viral) ($n=6$), cryptogenic cirrhosis ($n=7$), others ($n=7$). Twenty-three patients with viral cirrhosis, one with alcoholic and one with mixed (viral and alcoholic cirrhosis) had hepatocellular carcinoma (HCC) demonstrated by the histology of the explanted livers (diameters of the nodules: 1.5 to 6 cm) and were excluded from the analysis. Among the patients with alcoholic and viral cirrhosis, 1 had Budd-Chiari syndrome, 1 a severe arterioportal fistula, 7 had a TIPS, 2 a distal splenorenal shunt, and 1 patient had a latero-lateral portocaval shunt. These patients were excluded as well. In the final analysis only patients with pure viral or alcoholic cirrhosis were included. Therefore, 22 patients with mixed, viral and alcoholic cirrhosis were also excluded. The clinical characteristics of the 96 patients with viral cirrhosis and of the 35 with alcoholic cirrhosis included in the study are reported in Table I. Their liver weight is reported in Table II.

Patients with alcoholic cirrhosis had a higher liver weight than patients with viral cirrhosis: 1246 ± 295 g versus 1070 ± 254 g, $p=0.001$. In patients with viral cirrhosis, but not in those with alcoholic cirrhosis, there was a significant inverse correlation between liver weight and CTP score (Figure 1). When analyzing the single variables of the CTP classification, we found that in viral cirrhosis, liver weight significantly correlated with albumin ($r=0.32$, $p=0.002$) and prothrombin activity ($r=0.45$, $p<0.0001$), but not with bilirubin, presence of ascites or of encephalopathy. Despite the differences in liver weight between the two groups, there was no difference in CTP scores between alcoholic and viral cirrhosis (9.7 ± 1.7 versus 9.8 ± 2.0, $p=$NS), nor did we find any difference in the level of albumin, of bilirubin, prothrombin activity, prevalence of ascites or encephalopathy (Table I). Liver weight was not related to BMI, but instead to patient's height and weight in viral cirrhosis ($r=0.32$, $p=0.0018$ and $r=0.32$, $p=0.0020$, respectively), but not in alcoholic cirrhosis ($r=0.02$, $p=$NS and $r=0.12$, $p=$NS, respectively). The difference in liver weight between alcoholic and viral cirrhosis was also confirmed by analyzing liver weight as the ratio of liver weight to body-weight. In fact, the ratio liver weight/body weight was higher in alcoholic cirrhosis than in viral cirrhosis (0.0183 ± 0.0062 versus 0.0149 ± 0.0035, $p=0.0004$) and was related to CTP score only in patients with viral cirrhosis (Spearman $r=-0.38$, $p=0.0002$ in viral cirrhosis; Spearman $r=+0.07$, $p=$NS in alcoholic cirrhosis). In viral cirrhosis, the ratio liver weight/body weight was related to albumin ($r=0.37$, $p=0.00026$) and to prothrombin activity ($r=0.48$, $p<0.00001$), but not to bilirubin, presence of ascites or encephalopathy.

Splanchnic hemodynamic parameters of the included patients are reported in Table II. Echo-Doppler portal vein measurements were not available or not reliable in 22 patients (10 with alcoholic cirrhosis and 12 with viral cirrhosis).

Table I. Clinical characteristics of the 131 patients included in the study.

	Etiology of cirrhosis		
	Alcoholic	Viral	Comparison between the two groups
No. of patients	35	96	–
Male/female	26/9	82/14	p =NS
Age (years) (mean ± SD)	50 ± 8	51 ± 9	p =NS
Age (years) (range)	22–60	22–63	–
Height (cm) (mean ± SD)	170 ± 9	170 ± 8	p =NS
Weight (kg) (mean ± SD)	72.0 ± 14.0	72.6 ± 11.2	p =NS
Body mass index (mean ± SD)	24.7 ± 3.2	25.0 ± 3.2	p =NS
CTP class (A/B/C)	0/16/19	0/40/56	p =NS
CTP class (score)	9.7 ± 1.7	9.8 ± 2.0	p =NS
S-albumin (mg dl^{-1})	33.2 ± 4.8	31.9 ± 4.9	p =NS
S-bilirubin (μmol l^{-1})	55.3 ± 43.5	70.4 ± 91.1	p =NS
Prothrombin activity (%)	54.8 ± 18.1	53.1 ± 16.1	p =NS
Ascites (yes/no)	28/7	77/19	p =NS
Encephalopathy (yes/no)	12/23	45/51	p =NS
Viral etiology (HBV/HBV–HCV/HBV–HDV/HCV)	–	21/25/19/31	–

Abbreviations: CTP = Child-Turcotte-Pugh; HBV = hepatitis B virus; HCV = hepatitis C virus; NS = not significant.

Hepatic PI was not available or nor reliable in 42 patients (14 with alcoholic cirrhosis and 28 with viral cirrhosis) and splenic PI in 33 patients (12 with alcoholic cirrhosis and 21 with viral cirrhosis). Four patients with viral cirrhosis had a reversed PBF. No correlation was apparent between liver weight or CTP score and splanchnic hemodynamic parameters. PLP and PLP per gram of liver tissue significantly correlated with serum albumin in viral cirrhosis ($r = 0.34$, $p = 0.017$ and $r = 0.24$, $p = 0.030$, respectively), but not in alcoholic cirrhosis. Forty-one patients had a patent paraumbilical vein. These patients had similar liver weights (1107 ± 298 g versus 1115 ± 268 g, p =NS), a similar CTP score (9.8 ± 2.0 versus 9.7 ± 1.9, p =NS), a similar PBF (1045 ± 656 ml^{-1} min^{-1} versus 944 ± 530 ml min^{-1}, p =NS), similar hepatic and splenic PI values (1.28 ± 0.30 versus 1.34 ± 0.36, p =NS, and 1.03 ± 0.22 versus 1.13 ± 0.26, p =NS, respectively), but a lower PLP (538 ± 426 ml min^{-1} versus 944 ± 530 ml min^{-1}, $p = 0.0001$) and a lower PLP per gram of liver tissue (0.54 ± 0.48 ml g^{-1} min^{-1} versus 0.90 ± 0.57 ml g^{-1} min^{-1}, $p = 0.0017$) in respect of the other patients. Splanchnic hemodynamics was not related to liver weight, but differences were found according to the etiology of cirrhosis. In alcoholic cirrhosis, PLP was lower (585 ± 399 ml min^{-1} versus 865 ± 525 ml min^{-1}, $p = 0.020$), PLP per gram of liver tissue was lower (0.49 ± 0.36 ml g^{-1} min^{-1} versus 0.85 ± 0.56 ml g^{-1} min^{-1}, $p = 0.004$) and hepatic PI was higher (1.45 ± 0.31 versus 1.26 ± 0.30, $p = 0.018$) in respect of viral cirrhosis. Prevalence of a patent paraumbilical vein was higher in alcoholic cirrhosis (56% versus 29%, $p = 0.011$).

Morphometric assessment of liver fibrosis was performed in 30 patients (M/F: 23/7, age: 49 ± 12 years). Ten patients were affected by alcoholic cirrhosis and 20 by viral cirrhosis (4 HBV, 5 HBV-HDV, 3 HBV-HCV, 8 HCV). In patients with

Table II. Liver weight and splanchnic hemodynamic parameters in patients with alcoholic or viral cirrhosis.

	Alcoholic cirrhosis	Viral cirrhosis	Comparison between the two groups
Liver weight (g)	1246 ± 295	1070 ± 254	$p = 0.0010$
Liver weight/body-weight	0.0183 ± 0.0062	0.0149 ± 0.0035	$p = 0.0004$
PV diameter (mm)	14.9 ± 3.5	14.4 ± 2.8	p =NS
PV velocity (cm s^{-1})	9.4 ± 2.6	10.4 ± 3.3	p =NS
Congestion index (cm s^{-1})	0.198 ± 0.125	0.180 ± 0.078	p =NS
Portal blood flow (ml min^{-1})	921 ± 654	997 ± 527	p =NS
Portal liver perfusion (ml min^{-1})	585 ± 399	865 ± 525	$p = 0.020$
Portal liver perfusion per gram of liver tissue (ml g^{-1} min^{-1})	0.49 ± 0.36	0.85 ± 0.56	$p = 0.004$
Hepatic PI	1.45 ± 0.31	1.26 ± 0.30	$p = 0.018$
Splenic PI	1.18 ± 0.27	1.08 ± 0.25	p =NS
Spleen size (cm)	16.5 ± 3.0	17.8 ± 3.2	p =NS

Abbreviations: PV = portal vein; PI = pulsatility index.

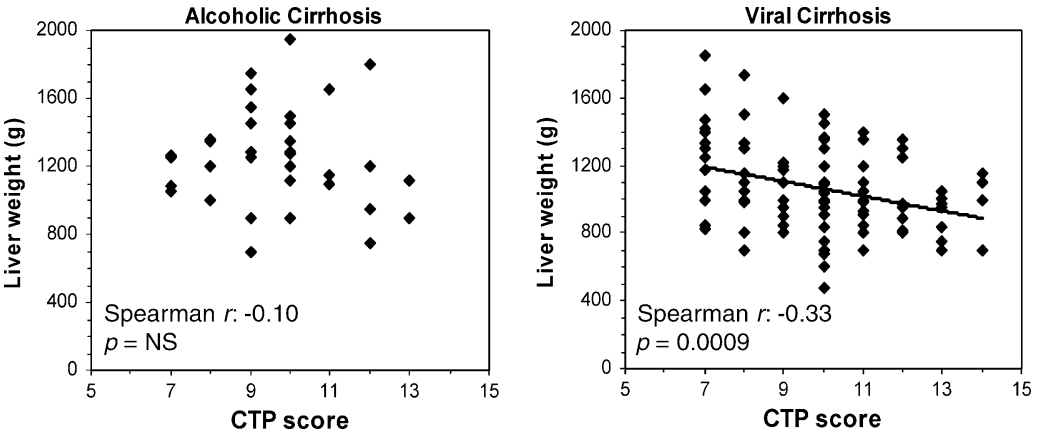

Figure 1. Relationship between Child-Turcotte-Pugh (CTP) score and liver weight in patients with alcoholic and viral cirrhosis.

alcoholic cirrhosis, liver weight was heavier than in patients with viral etiology, without significant difference in the degree of fibrosis (Table III). There was no relationship between the degree of fibrosis and liver weight. Fibrosis varied considerably among the various patients, independently of the etiology of liver disease (3.8% to 21.3% in viral cirrhosis and 7.3% to 26.4% in alcoholic cirrhosis). The degree of fibrosis was also heterogeneous among the various microscopic fields of the same patient, in both alcoholic and viral cirrhosis, but the CV of fibrosis measurement in the 128 microscopic fields was significantly higher in viral cirrhosis than in alcoholic cirrhosis (Table III). In viral, but not in alcoholic cirrhosis, CV was inversely related to the percentage of fibrosis ($r=0.78$, $p<0.0001$ and $r=0.45$, $p=$NS, respectively).

Discussion

This study demonstrates that PLP was lower and hepatic arterial resistance indices were higher in alcoholic cirrhosis in relation to viral cirrhosis. This result suggests a more serious sinusoidal distortion, with higher resistance, in alcoholic patients. In order to understand the cause of the different hemodynamic patterns detected in the two types of cirrhosis, we analyzed the relationships with liver pathology (liver weight and degree of fibrosis) and with the severity of the disease estimated by CTP score. In cirrhosis of viral origin, liver weight was inversely related to CTP score and directly related to albumin and prothrombin activity. Conversely, there was no such relationship in alcoholic cirrhosis, in which, despite a similar CTP score, liver weight was higher. As the degree of fibrosis was similar in alcoholic and in viral cirrhosis, it seems that in respect of viral cirrhosis, patients with alcoholic cirrhosis have impaired hepatocyte function with lower portal flow (the portal flow effectively perfusing the liver was estimated as PLP per gram of liver tissue). The residual hepatic function seems to be related to liver volume only in viral cirrhosis.

The systematic difference in liver weight between alcoholic and viral cirrhosis was an unexpected finding. The difference in weight is not trivial, averaging at 16%, and it was detected despite a similar degree of severity of the disease. To explain the difference in weight, a different histology between viral and alcoholic cirrhosis cannot be taken into account, as alcoholic and non-alcoholic patients are often indistinguishable based on histology alone [23]. Even though a different pattern of fibrosis and fatty change has sometimes been reported in alcoholic cirrhosis [24,25], no quantitative histologic difference has been found between the two different conditions [23,26,27]. Similarly, in our patients the

Table III. Histomorphometry analysis of the livers of the 30 patients in whom the pathology evaluation was performed.

	All patients ($n=30$)	Alcoholic cirrhosis ($n=10$)	Viral cirrhosis ($n=20$)	Comparison of alcoholic and viral cirrhosis
Liver weight (g)	1152±216	1297±198	1081±192	$p=0.007$
Fibrosis (%)	11±4.7	11.7±5.5	11.0±4.4	$p=$NS
Fibrosis SD (%)	12.4±3.4	12.7±3.2	11.7±3.9	$p=$NS
Fibrosis CV (%)	116±24	103±17	122±25	$p=0.028$
Fibrotic mass (g)	131±62	152±72	120±55	$p=$NS

Abbreviations: SD = standard deviation; CV = coefficient of variation; NS = not significant.

degree of fibrosis was similar in alcoholic and viral cirrhosis.

A correlation between liver weight and the severity of the disease, estimated with the CTP score, was found only in patients with viral cirrhosis, while in alcoholic cirrhosis liver weight was higher, without any relationship with CTP score. The Spearman *r* of the regressions between liver weight and CTP score was noticeably different between viral and alcoholic cirrhosis (-0.33 versus -0.10 when analyzing liver weight and -0.38 versus $+0.07$ when analyzing the ratio liver weight/body-weight). Therefore, the different behavior patterns of the two groups cannot be simply accounted for by the different number of the included patients. As a consequence, also considering the equal amount of fibrosis, impaired hepatocyte function in alcoholic cirrhosis in relation to viral cirrhosis seems a plausible hypothesis. A possible difference from viral cirrhosis may be the presence of hepatocyte swelling, a characteristic of alcoholic liver disease [28]. Hepatocyte swelling might explain why, with the same function, alcoholic cirrhosis has a higher liver weight, and why there is a loss of relationship between weight and function. But hepatocyte swelling has been clearly demonstrated only after acute administration of alcohol [29–31], and all our patients had been abstinent for at least six months (prerequisite for entry on the transplant waiting list).

The lower PLP per gram of liver tissue in alcoholic cirrhosis may be in keeping with the finding of Vidins et al. [32], who reported that patients with alcoholic liver disease showed a marked reduction in relative sinusoidal area, with an increase in hepatocyte size, when compared with patients with non-alcoholic liver disease. However, in that paper alcoholic patients were not abstinent and therefore a direct toxic effect of alcohol on hepatocytes cannot be ruled out. At any rate, the lower PLP per gram of liver tissue suggests a more marked sinusoidal distortion, with higher sinusoidal resistance in alcoholic cirrhosis in relation to viral cirrhosis. This hypothesis is supported on the one hand by the contemporary significant increase in hepatic PI values detected in alcoholic cirrhosis, as hepatic PI is a parameter related to portal resistance [21,33], and on the other hand by the higher prevalence of a patent paraumbilical vein detected in alcoholic cirrhosis, which is a finding also reported by other investigators [34]. This interpretation is also supported by a few studies which reported a higher portal pressure in alcoholic cirrhosis in relation to viral cirrhosis [35–37]. Even though the relationship between sinusoidal resistance and portal pressure is not strictly linear, because portal pressure is influenced also by portal blood flow and by the extent of portal-collateral circulation, the higher portal pressure reported in alcoholic cirrhosis [35–37] sustains the hypothesis of higher sinusoidal resistance in this group of patients.

PLP was not related to liver weight, or to the single variables of the Child classification, i.e., bilirubin, albumin, prothrombin activity, ascites and encephalopathy. A significant relationship with albumin was detected in viral cirrhosis only. Therefore, it seems that PLP does not play a major role in the regulation and maintenance of liver function in endstage cirrhosis. In alcoholic cirrhosis, PLP values were lower, but these lower values were not related to findings of decompensated cirrhosis. For this reason, the increase in liver volume in alcoholic cirrhosis does not seem to be linked to a specific alteration in portal perfusion, but probably to the etiology *per se*.

Alcoholic cirrhosis and viral cirrhosis are traditionally defined as micronodular and macronodular, respectively, according to the different aspects of their pathology [38,39]. Our study suggests also a different, specific hemodynamic pattern. As a result, in endstage liver cirrhosis the intrahepatic hemodynamic pattern and liver weight are not independent of the etiology of the disease. The difference between the two types of cirrhosis (alcoholic and virus-related) was also confirmed by the analysis of the variability of the degree of fibrosis in the liver parenchyma. In fact, the heterogeneity of fibrosis among microscopic fields in the same liver, estimated as CV, was higher in virus-related cirrhosis (Table III), particularly in patients with the lowest degree of fibrosis.

In conclusion, a different hemodynamic pattern characterizes the advanced stage of cirrhosis of alcoholic and viral origin. A more severe alteration of intrahepatic portal perfusion and a higher liver weight can be detected in alcoholic cirrhosis.

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