Quantitative Doppler Evaluation of the Splenoportal Venous System in Various Stages of Cirrhosis: Differences between Right and Left Portal Veins

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ABSTRACT: *Purpose.* The purpose of our study was to evaluate the relationship between the splenoportal hemodynamics in patients with cirrhosis and the stage of the disease.

Methods. Patients with cirrhosis were grouped according to modified Child-Pugh scoring into stages A, B, and C of cirrhosis. A control group of healthy volunteers was included. After gastroenterologic clinical and laboratory examinations, all participants underwent a splenoportal Doppler sonographic evaluation in which the vessels' diameter, area, and blood flow velocity were measured and blood flow rate and the congestion index in the splenoportal venous system were calculated.

Results. Seventy-five patients with cirrhosis (25 women and 50 men) were enrolled; the control group consisted of 30 healthy volunteers (15 women and 15 men) with no liver disease. The mean age (± standard deviation) of the patients was 54.4 ± 14.8 years (range, 13–80 years) and of the control subjects was 47.3 ± 14.5 years (range, 18–72 years). No significant differences in vessel diameter, blood flow velocity, and blood flow rate were found in the main and left portal veins between the study group and the control group. In the right portal vein, we found decreases in the vessel diameter, blood flow velocity, and blood flow rate, and in the splenic vein, we found increases in vessel diameter and blood flow rate. The congestion index was increased in the main portal and splenic veins but was unchanged in the left portal vein.

Conclusions. Although our data indicate that there is no difference in Doppler sonographic parameters of the main portal vein according to Child-Pugh scores, the hemodynamic differences between the left and right branches of the portal vein may be clinically useful in patients with cirrhosis. *J Clin Ultrasound* **30**:537–543, 2002; Published online in Wiley InterScience (www.interscience.wiley.com). DOI: 10.1002/jcu.10114

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The development of Doppler sonography has contributed to a better understanding of splenoportal hemodynamics. Characterizing both the qualitative and quantitative changes in hemodynamics in the splenoportal system is important in patients with cirrhosis. Several variables have been measured and used for diagnosing and assessing the severity of cirrhosis since the introduction of Doppler sonography. The most frequently investigated quantitative Doppler parameters are the mean velocity in the portal vein (PV), the portal blood flow, and the congestion index (CI).[1–5] We undertook this study to investigate the relationship between the quantitative Doppler parameters of the splenoportal system and the clinical, biochemical, and endo-

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scopic findings that are used in the assessment of severity and prognosis of cirrhosis.

PATIENTS AND METHODS

Patient Population

The study group included patients with cirrhosis diagnosed on the basis of clinical, biochemical, endoscopic, and sonographic findings. All of the patients had undergone liver biopsy, and the diagnosis had been verified histopathologically. A control group of healthy volunteers with no liver disease was also included.

To be included, patients could not have experienced gastrointestinal bleeding in the 5 weeks before their enrollment in the study. To eliminate the possible effects of medication on portal hemodynamics, drugs such as beta blockers and diuretics were discontinued for 7 days before the Doppler examination. Patients in whom discontinuation of these drugs was not possible during this washout period were excluded from participation.

Written informed consent was obtained from all patients, and all procedures were conducted in accordance with the Declaration of Helsinki and its revisions.

Sonographic Examinations

All patients were examined after an overnight fast. All sonographic examinations were performed using a 128 XP/10 Doppler ultrasound scanner (Acuson, Mountain View, CA) equipped with a 3.5-MHz curved-array transducer. Liver size (greatest longitudinal dimension) was measured in the subcostal sagittal plane in which the greatest longitudinal dimension of the right kidney was seen. Spleen size was measured using the longest longitudinal dimension from the dome of the diaphragm to the tip of the spleen and using the width of the spleen at the hilum in the coronal plane; however, the length and width measurements of the spleen were multiplied to obtain a calculated area for statistical evaluation.

Each patient was examined by 2 different radiologists; for each parameter, at least 3 measurements were made, and the results of all measurements by both radiologists were averaged.

The main PV (MPV), right and left PVs (RPV and LPV, respectively), and splenic vein (SV) were evaluated by gray-scale sonography for diameter and cross-sectional area measurement. For Doppler examinations, the vessel to be evaluated was visualized along its longitudinal axis, and the sampling gate was placed at the center of the vessel, keeping the angle to less than 60°.

The diameter of the MPV was measured from 1 cm proximal to the hilum in the extrahepatic portion; the diameter of the RPV and LPV branches, from 2 cm distal to the bifurcation of the MPV; and the diameter of the SV, from 1 cm proximal to the splenic hilum in the extrasplenic portion. For all diameter measurements, enlarged B-mode images were used, and diameters were measured from the inner anterior wall to the inner posterior wall.

We then calculated the blood flow rates; CIs; and LPV:MPV, RPV:MPV, and SV:MPV blood flow–volume ratios. A 4-second period was used for measuring the mean blood flow velocity. For the blood flow rate, the following formula was used: blood flow rate (ml/minute) = mean blood flow velocity (cm/second) × cross-sectional area of the vessel (cm^2) × 60. Also, although Moriyasu and colleagues[6] defined the CI for the PV (CI = PV area/maximum PV velocity), we modified it to CI = vein area/maximum vein velocity and calculated CI values for each of the veins examined.

Statistical Analysis

All statistical analyses were performed using SPSS Version 7.5 software (SPSS Inc., Chicago, IL). We used the 2-tailed Mann-Whitney test for comparing the Doppler parameters for the patient and control groups, the Kruskal-Wallis test for a global comparison of the 3 Child-Pugh subgroups of patients for demographics and Doppler data, the chi-square test for categorical variables, and correlation analysis for correlation of the Doppler parameters. A p value of less than 0.05 was considered statistically significant. Results are presented as means ± standard deviations.

RESULTS

In total, 75 patients (25 women and 50 men) with cirrhosis were enrolled, and 30 healthy volunteers (15 women and 15 men) served as the control group. The mean age of the patients was 54.4 ± 14.8 years (range, 13–80 years), and that of the control subjects was 47.3 ± 14.5 years (range, 18–72 years).

Patients were grouped according to their modified Child-Pugh scores.[7] None of the patients had thrombus in the PV or SV, and none had encephalopathy greater than grade 2 (lethargy and moderate confusion). Ascites was classified into three grades: 0, no ascites; 1, ascites that did not require diuretics; and 2, ascites that was controllable with diuretics. We used the system proposed by Beppu et al[8] for the endoscopic grading of esophageal varices (0, no varices; 1, small, smooth

varices; 2, dilated, tortuous varices; and 3, large, coil-shaped varices).

Table 1 summarizes all the clinical and biochemical findings in the patients, grouped by Child-Pugh score, and in the control subjects. In 63 (84%) of the patients, the cause of cirrhosis was viral hepatitis; in 2 (3%), the cause was alcohol; and in 10 (13%), the cirrhosis had another cause. The liver was significantly smaller in patients than in the control subjects ($p < 0.05$), whereas the spleen was significantly larger in patients than in the control subjects ($p < 0.05$). No significant difference in spleen size was observed between the 3 Child groups.

Table 2 shows the various Doppler parameters in the patient groups and control subjects. No significant differences were found in blood flow rate, blood flow velocity, and diameter in the MPV between patients and control subjects, but the CI was statistically significantly higher in patients than in the control subjects ($p < 0.05$). Blood flow rate in the Child A group was significantly larger than it was in the Child B and C groups ($p < 0.05$ for each comparison). In the RPV, blood flow rate, blood flow velocity, and diameter were significantly lower in patients than in the control subjects ($p < 0.05$ for each comparison). CI, blood flow velocity, and blood flow rate in the Child A group were significantly higher than in the Child B and C groups ($p < 0.05$ for each comparison). No statistically significant differences were found in LPV blood flow rate, blood flow velocity, and CI in the patient groups compared with the control group. The diameter of the LPV in the Child A group, however, was significantly higher than in the Child B and C groups ($p < 0.05$). In the SV, blood flow rate, diameter, and CI were significantly higher in the patient groups than in the control subjects ($p < 0.05$), and the diameter and CI in the Child A group were significantly higher than in the Child B and C groups ($p < 0.05$).

The RPV:MPV blood flow rate ratio in each Child group was significantly lower in patients than in the control group ($p < 0.05$ for each Child group) (Table 3). No significant difference was found in the LPV:MPV blood flow rate ratio in each Child group compared with the control group. The SV:MPV ratio was significantly higher in the Child B and C groups ($p < 0.05$) than in the control group, but this difference was not seen in the Child A group compared with the control group. In the Child B and C groups, the blood flow

TABLE 1
Clinical and Biochemical Features of Cirrhotic Patients (by Child Class) and Control Subjects

	Patient Group				
Characteristic*	Overall (*n* = 75)	Child A (*n* = 18)	Child B (*n* = 26)	Child C (*n* = 31)	Control Group (*n* = 30)
Age, years	54.4 ± 14.8	56.7 ± 14.6	51.0 ± 16.1	55.9 ± 14.1	47.3 ± 14.5
Sex, male/female	50/25	11/7	18/8	19/12	15/15
Etiology					
Hepatitis B	53	13	17	23	0
Hepatitis C	10	2	5	3	0
Alcohol	2	0	2	0	0
Other	10	4	3	3	0
Varix grade					
0	25	7	5	13	0
1	33	7	17	9	0
2	10	3	5	2	0
3	7	2	0	5	0
Variceal hemorrhage	10	2	4	4	0
Ascites grade					
0	8	4	4	0	0
1	42	14	20	8	0
2	25	0	2	23	0
Encephalopathy	17	0	2	15	0
Albumin level, g/dl	2.9 ± 0.8	3.8 ± 0.4	3.2 ± 0.5	2.2 ± 4.0	4.2 ± 2.1
Bilirubin level, mg/dl	3.9 ± 3.7	1.9 ± 2.3	2.2 ± 1.8	6.9 ± 3.7	0.8 ± 0.3
Prothrombin time, seconds	17.8 ± 4.9	14.2 ± 2.4	16.7 ± 3.5	21.0 ± 5.1	13.0 ± 3.2
Liver size, cm†	13.5 ± 2.4	14.3 ± 3.0	13.5 ± 1.9	11.9 ± 1.8	14.9 ± 0.9
Spleen size, cm²‡	96.5 ± 35.9	86.8 ± 36.8	108.0 ± 34.9	91.9 ± 35.5	44.4 ± 7.6

*Data are given as means ± standard deviations or as numbers of patients.

†Liver size (greatest longitudinal dimension) was measured in the subcostal sagittal plane in which the greatest longitudinal dimension of the right kidney was seen.

‡Spleen size is expressed in terms of area (longest longitudinal dimension × longest transverse dimension), calculated for statistical evaluation only.

TABLE 2
Summary of Doppler Parameters in Patients with Cirrhosis and Control Subjects*

Parameter	Patient Group				Control Group (n = 30)
	Overall (n = 75)	Child A (n = 18)	Child B (n = 26)	Child C (n = 31)	
Main portal vein					
Diameter, mm	11.9 ± 3.4	13.8 ± 2.6	11.2 ± 3.4	11.5 ± 3.5	10.8 ± 0.6
Blood flow velocity, cm/second	17.3 ± 9.5	22.7 ± 16.6	14.9 ± 4.8	16.3 ± 5.2	19.6 ± 4.4
Blood flow rate, ml/minute	1361 ± 1337	2186 ± 2200†	1050 ± 801	1134 ± 791	1109 ± 278
Congestion index	0.076 ± 0.044‡	0.085 ± 0.051	0.071 ± 0.03	0.077 ± 0.05	0.05 ± 0.016
Right portal vein					
Diameter, mm	7.4 ± 2.2‡	9.3 ± 2.3	7.1 ± 1.9	6.4 ± 1.6	8.3 ± 0.8
Blood flow velocity, cm/second	15.7 ± 5.9‡	17.7 ± 7.6†	13.9 ± 4.0	16.1 ± 6.0	20.1 ± 3.8
Blood flow rate, ml/minute	478 ± 473‡	850 ± 812†	403 ± 208	317 ± 172	666 ± 168
Congestion index	0.032 ± 0.019	0.044 ± 0.02†	0.031 ± 0.015	0.027 ± 0.021	0.028 ± 0.075
Left portal vein					
Diameter, mm	7.5 ± 2.1	9.1 ± 2.1†	7.1 ± 1.9	6.9 ± 1.8	7.3 ± 1.0
Blood flow velocity, cm/second	15.4 ± 5.0	16.1 ± 4.4	14.3 ± 3.8	15.9 ± 6.4	16.8 ± 4.1
Blood flow rate, ml/minute	453 ± 315	682 ± 416	377 ± 237	381 ± 246	445 ± 174
Congestion index	0.033 ± 0.02	0.045 ± 0.021	0.028 ± 0.012	0.032 ± 0.023	0.026 ± 0.095
Splenic vein					
Diameter, mm	8.5 ± 2.0‡	9.5 ± 2.5†	8.9 ± 1.6	7.4 ± 1.7	6.4 ± 0.7
Blood flow velocity, cm/second	18.2 ± 4.8	17.5 ± 3.5	18.6 ± 6.3	18.3 ± 3.8	17.9 ± 4.0
Blood flow rate, ml/minute	666 ± 397‡	818 ± 550	728 ± 355	517 ± 289	353 ± 136
Congestion index	0.034 ± 0.019‡	0.042 ± 0.019†	0.039 ± 0.021	0.024 ± 0.099	0.019 ± 0.054

*Data are given as means ± standard deviations.
†$p < 0.05$ versus both other Child groups.
‡$p < 0.05$ versus control group.

TABLE 3
Blood Flow Velocities and Blood Flow Volume Ratios in Patient and Control Groups*

Group	Blood Flow Velocity, cm/second				Blood Flow Rate Ratio		
	MPV	RPV	LPV	SV	RPV:MPV	LPV:MPV	SV:MPV
Child A	22.7 ± 16.6	17.7 ± 7.6	16.1 ± 4.4	17.5 ± 3.5	0.41†	0.37	0.43
Child B	14.9 ± 4.8†	13.9 ± 4.0†	14.3 ± 3.8	18.6 ± 6.3	0.39†	0.42	1.62†
Child C	16.3 ± 5.2†	16.1 ± 6.0†	15.9 ± 6.4	18.3 ± 3.8	0.38†	0.43	0.65†
Control	19.6 ± 4.4	20.1 ± 3.8	16.8 ± 4.1	17.9 ± 4.0	0.61	0.40	0.34

Abbreviations: MPV, main portal vein; RPV, right portal vein; LPV, left portal vein; SV, splenic vein.
*Data are given as means ± standard deviations.
†$p < 0.05$ versus control group.

velocities in the MPV and RPV were significantly lower than they were in the control subjects, but there was no significant difference in blood flow velocities in the LPV between the patient and control groups.

DISCUSSION

The presence of extensive fibrosis and numerous regenerative nodules replacing the normal liver parenchyma is characteristic of cirrhosis. Two principal mechanisms—elevated vascular resistance, which is an initiating factor, and increased portal blood inflow, which plays an important role in maintaining a chronic portal hypertensive state—have been reported to be involved in the development of portal hypertension secondary to cirrhosis.[9] Portal blood flow changes are generally the result rather than the cause of hepatic dysfunction and structural changes. Determination of the disease's prognosis is important from the perspective of treatment. Child-Pugh scoring can accurately assess disease severity.[10] Various quantitative Doppler parameters (eg, mean portal flow velocity, blood flow rate, and CI) have been evaluated in the literature for diagnosing and grading the severity of cirrhosis.[1,11] However, no consensus on the role of these parameters has been reached.

Diffuse progressive fibrosis in the liver causes an increase in microvascular resistance and a decrease in blood velocity in the PV.[12,13] As the resistance increases, portosystemic collaterals open, and the PV diameter returns to normal and

then decreases.[13] It has been reported that as the degree of cirrhosis increases, portal blood flow and velocity decrease.[14,15] Our study found no significant changes in diameter, blood flow velocity, or blood flow rate in the MPV between the study groups and the control group. Although our unexpected results seem to differ from what has been reported in most previous articles, blood flow velocity was significantly lower in the Child B and C groups than in the control group. As the Child-Pugh score increased from A to B and C, blood flow rate was also found to be significantly lower. Our results are similar to those in the vast majority of published studies in Child B and C groups but not in the Child A group. This result could indicate that the Doppler parameters studied were unaltered in the Child A group and that changes should be expected in later stages of the disease.

In the literature, it has been reported that portal blood flow volume in the MPV in cirrhotic patients with moderate and severe liver failure, many of whom had ascites, was significantly lower than it was in those who were considered to be in good condition according to Child-Pugh criteria.[15] These data indicate that in many patients with cirrhosis, portal venous blood flow actually appears to be increased in the early phases of the disease. Although our study lacked the statistical power to determine significance in this regard, our results in patients with Child A stage cirrhosis clearly illustrate this fact.

Besides parenchymal changes, morphologic characteristics of the liver are altered by atrophy of the right lobe and the medial segment of the left lobe and by hypertrophy of the lateral segment of the left lobe and of the caudate lobe.[2,16–20] The left lobe of the liver is less affected than the right lobe is in cirrhosis.[2,19] Nishihara et al[19] reported results supporting regional differences in portal blood flow. They reported a significant decrease of blood flow in the anterior branch of the RPV in patients compared with normal subjects; however, they did not find a similar decrease in the posterior branch of the RPV or in the MPV. We found that the RPV's diameter, blood velocity, and blood flow rate were significantly lower in patients than in control subjects. However, there was no significant difference in the values obtained for the LPV between patient and control groups. In addition, the lower RPV:MPV blood flow rate ratio in each Child group and the absence of such a decrease in the LPV:MPV ratio compared with those in the control group in our study indicate a regional heterogeneity in the portal circulation. There is no consistent explanation for focal atrophy and hypertrophy of the different lobes. The streamline theory, which refers to the preferential distribution of blood flowing from the superior mesenteric vein and the SV into the RPV and LPV, respectively, has recently been confirmed with magnetic resonance angiography.[21] According to this theory, we can suggest that the increased portal inflow due to increased SV blood flow rate, as shown by our results, maintains an almost normal hemodynamic status in the LPV and could cause hypertrophy of the left lobe. Thus, the changes in LPV hemodynamics are less significant than are those in the RPV.

Evaluating the splenic hemodynamics is important in patients with cirrhosis. Chronic passive congestion due to a long-standing pressure increase in the splenoportal venous system is a common cause of splenomegaly. Splenic blood flow rate increases due to splenomegaly in turn cause an increase in the diameter of the SV.[4,22] Our results also support these data. Yin et al[22] reported that the SV:MPV ratio is increased in cirrhotic patients and found a significant correlation with esophageal variceal bleeding. In our study, the SV:MPV ratio was increased only in patients in the Child B and C groups, not in those in the Child A group. Since the number of patients with a history of variceal hemorrhaging was too low to draw a definite conclusion, we did not evaluate this relationship.

Some factors, such as variations in the measurement technique, affect the diagnostic accuracy of Doppler sonography.[18] In our study, we paid special attention to the appropriate placement of the sampling gate and kept the Doppler angle below 60°. In addition to technical factors, some physiologic factors, such as patient position, exercise status, phase of respiration, and fasting state could lead to variations in results.[18,23] To prevent these potential sources of error, we followed a rigorous evaluation protocol and used the same equipment for all participants. Since the diameter-measurement techniques used in the calculation of blood flow could lead to errors, we used a method similar to that of Walsh et al.[13] Most authors[14,18,19,24–27] have measured the diameter of the PV assuming the vein to be circular and have used the formula $\pi d^2/4$. However, one should keep in mind that some vessels are oval in cross section, including the PV.[28] An error of 1 mm in measuring the PV diameter would cause a 12–36% difference in blood flow. For that reason, we used the "trace" function of the scanner to outline the cross section of the vessel and obtain the most accurate measurement of the vessel's cross-sectional area, regardless of its shape. The use of

direct measurement of cross-sectional area of the vessels is more accurate than estimating it from the diameter at 1 site, provided, however, that the transverse scan plane is truly perpendicular to the vessel's axis. To minimize measurement errors, we used the mean of 3 different measurements performed by 2 different radiologists.

In conclusion, the blood flow rate in the MPV was not significantly different between the cirrhotic patients and the healthy controls in our study. We did find increases in the diameter, blood flow, and CI in the SV in cirrhotic patients compared with control subjects. For that reason, we believe that quantitative Doppler evaluation of the SV should be included in the Doppler examination of cirrhotic patients. Since the diameter, blood flow rate, and blood flow velocity values in the RPV showed a decrease and the corresponding values in the LPV did not differ significantly in cirrhotic patients compared with control subjects, there could be regional heterogeneity in the portal blood circulation owing to streamlined flow in the PV. It also seems that parenchymal damage and vascular fibrosis affect the liver parenchyma supplied by the RPV more than that supplied by the LPV. Thus, we suggest that one should pay special attention to and perform a detailed hemodynamic examination of both the RPV and LPV besides the MPV during a portal Doppler evaluation of cirrhotic patients.

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