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Renal Hypoperfusion Associated with Splenorenal Shunts in Liver Cirrhosis[1]

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Purpose: To determine whether spontaneous a splenorenal shunt can be used as an imaging predictor of early renal hemodynamic changes in patients with cirrhosis.

Materials and Methods: The study included 82 cirrhotic patients and 41 control subjects. Three-phase CT was performed and CT attenuation values (Hounsfield units) of the renal cortex in three phases were measured to evaluate renal perfusion. Likelihood ratio tests for trend were conducted for age, presence of ascites, and Child's grade.

Results: The mean CT attenuation values of the renal cortex in cirrhotic patients were significantly lower than the values of control subjects in three phases: 153.3 ± 37.9 versus 173.3 ±25.2 in the arterial phase, 172.6 ± 41.0 versus 197.6 ± 26.5 in the portal phase and136.9 ± 26.0 versus 152.7 ± 20.0 in the delayed phase, respectively. The mean CT attenuation value of cortices in patients with renal hypoperfusion was 119.9 ± 11.8 in the portal phase. Child's class C (aOR: 58.4, 95% CI: 3.6 - 956.2; $p < 0.01$) and the presence of a renal shunt (aOR: 7.5, 95% CI: 1.8 - 30.5; $p < 0.01$) were associated with renal hypoperfusion. The incidence of renal hypoperfusion was associated with Child's grade (trend: $p < 0.01$), and not with the grade of ascites or age.

Conclusion: A dilated spontaneous splenorenal shunt may be a risk factor for renal hypoperfusion in cirrhosis.

Index words : Abdomen, CT
Liver, cirrhosis
Shunts, splenorenal

Renal hemodynamic changes begin early in patients during the course of liver cirrhosis and the hallmark change in such patients is an intense intrarenal vasoconstriction. This vasoconstriction is associated with reduced renal plasma flow and elevated renal arterial vascular resistance that may precede clinically recognized kidney dysfunction (1 - 3). Patients with these abnormalities may be at greater risk for subsequent development of overt hepatorenal syndrome, which is functional renal failure that occurs late in cirrhotic patients with advanced liver disease and ascites (4). A common pathway for this derangement appears to be marked splanchnic vasodilatation that induces an important compensatory response that leads to hyperdynamic circulation and renal functional abnormalities (5). Renal vasoconstriction and hyperdynamic circulation are the compensatory mechanisms that maintain effective arte-

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rial blood volume and arterial pressure when splanchnic arterial vasodilation occurs.

In clinical practice, we have identified decreased perfusion of the renal cortex more frequently in patients with a renal shunt than in patients without a renal shunt. Despite an extensive study of portal hemodynamics in patients with cirrhosis, the interrelationship between the presence of a renal shunt and renal hypoperfusion (6 - 8) has not been studied using CT imaging. As portal inflow is increased due to splanchnic vasodilatation, portal collateral circulation has an important role in modulating advanced portal hypertension; under these circumstances, the collateral circulation may carry as much as 90% of the blood entering the portal system. Extensive shunting of portal inflow is commonly associated with the presence of large shunts, more often through the left renal vein than through other collateral routes. Renal shunts (gastrorenal shunts or splenorenal shunts) usually have a short length with a larger diameter, similar to surgical shunts. The presence of shunts may explain the lower portal venous pressure in patients with large renal shunts.

We assumed that the presence of a renal shunt seen on a CT image indicated the occurrence of splanchnic arterial vasodilatation that was associated with renal vasoconstriction. For the purpose of this study, renal hypoperfusion was defined as the mean attenuation value of CT identified renal cortices less two standard deviations (SDs) as compared to control subjects and nonazotemic patients with liver cirrhosis in the portal phase. Considering a variety of factors that affect renal perfusion, we investigated whether identification of a renal shunt using CT imaging could be used as a predictor of early renal hemodynamic changes in patients with liver cirrhosis.

Materials and Methods

Patients and Clinical Features

Eighty-two patients with liver cirrhosis (66 men and 16 women; mean age 57.5 ± 10.9 years; age range, 41 - 78 years) and 41 patients without chronic liver disease (control group, 23 men and 18 women; mean age 52.0 ± 11.7 years; age range, 45 - 81 years) were referred for three-phase spiral CT imaging of the liver to assess liver lesions. The study was reviewed and approved by the institutional review board. Patients with heart failure or sepsis affecting renal perfusion were not included in the study group. The control group consisted of patients with a variety of focal liver lesions who had normal liver function tests (16 hemangiomas, 10 metastases, 8 cysts and 7 others lesions). The diagnosis of cirrhosis was based on a liver biopsy for 12 patients and on clinical, laboratory, and imaging data for the remainder of patients. Cirrhosis was due to a viral origin in 46 patients, an alcoholic origin in 22 patients, a biliary origin in 3 patients and a cryptogenic origin in 11 patients (Table 1). The clinical status of the patients with cirrhosis, including 34 cases with hepatocellular carcinoma with no portal vein invasion, was Child's A in 27 cases, Child's B in 40 cases, and Child's C in 15 cases. All participants, including the patients with liver cirrhosis as well as the control group of subjects, had a serum creatinine concentration of less than 1.5 mg/dL and no renal abnormalities at the time of abdominal CT imaging.

CT Exams

For dedicated renal parenchymal imaging of subjects with a suspected perfusion abnormality of the kidney, we studied each patient prospectively with the use of three-phase spiral CT imaging. The CT protocol was as follows. All images were obtained with a spiral CT scanner (Somatom plus 4 scanner, Siemens, Erlangen, Germany) following the oral administration of 800 - 1000 ml of 3% diatrizoate meglumine (Gastrografin, Bracco Diagnostics, Princeton, NJ U.S.A.). After a nonenhanced study was performed including the entire structures of the liver and kidneys, 120 ml of iopamidol (Ultravist, 300 mg I/ml, Schering, Germany) was injected at a rate of 3 ml/sec into an antecubital vein using a mechanical power injector. The delay between administration of the contrast material and the onset of scanning was 30 seconds, 60 seconds and 5 minutes, which corresponded to the arterial, portal and delayed phases. Scanning times ranged from 12 - 18 sec (mean, 15 sec). The section thickness was 8 mm, with a pitch of 1.5:1. We assessed the attenuation value of the renal cortex using Hounsfield units (HU) in a fixed section at the hilar level by assigning three circular regions of interest (ROI).

Table 1. Patient Characteristics

Control Group (n = 41)	Cirrhotic Group (n = 82)
Hemangioma (n = 16)	Cyst (n = 8)
Metastasis (n = 10)	Others (n = 7)
Viral cirrhosis (n = 46)	Alcoholic cirrhosis (n = 22)
Biliary cirrhosis (n = 3)	Cryptogenic cirrhosis(n = 11)

Note: Control group of subjects has normal hepatic function

Data Analysis

In each case, the ROIs were chosen to include as much of the renal cortex as possible, with exclusion of the renal medulla or cystic areas (Fig. 1). The three measured values for each phase (i.e., for each image) were then averaged. These mean values were compared using a t-test and scattered plots for patients with cirrhosis and control subjects in three phases. Before determining whether associated factors correlated with renal hypoperfusion, we set out to identify cases with renal hypoperfusion in patients with liver cirrhosis. Renal hypoperfusion associated with cirrhosis was defined as the mean attenuation value of CT identified renal cortices less than two SDs as compared to the mean attenuation values less than two SDs for control subjects in the portal phase. The following factors were included in the simple logistic regression analysis for assessing the risk of renal hypoperfusion: age, sex, presence of ascites, renal shunt and Child's grade. The diameter of the renal shunts was measured at a location immediately before entering the left renal vein. The degree of ascites was classified into four grades as follows: grade 1: no ascites; grade 2: easily detected but of relatively small volume; grade 3: obvious ascites but less than tense; grade 4: tense ascites. To exclude the influence of multiple variables on renal enhancement characteristics, a multiple logistic regression analysis was used to adjust for factors. Likelihood ratio testing for trends was conducted for age, presence of ascites, and the Child's grade. Statistical analysis was performed using the SAS statistical package (SAS Institute, Cary, NC U.S.A.). A P-value less than 0.05 was considered as statistically significant.

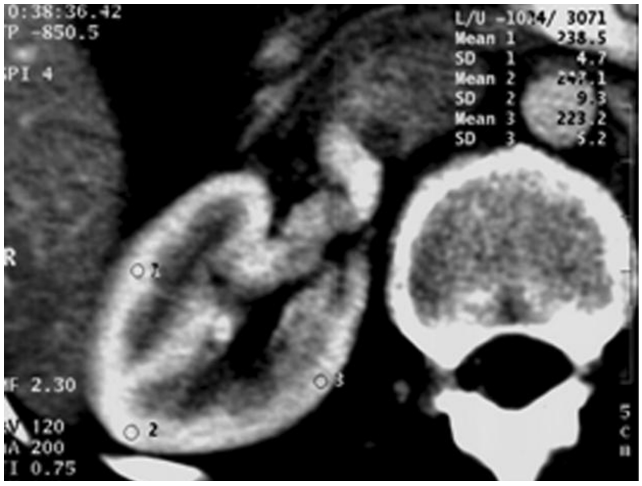

Fig. 1. Measurement of HU at the three regions of interest (ROI) in the renal cortex. Small circles represent the three ROIs. ROIs were chosen to include as much of the renal cortex as possible, with exclusion of the renal medulla.

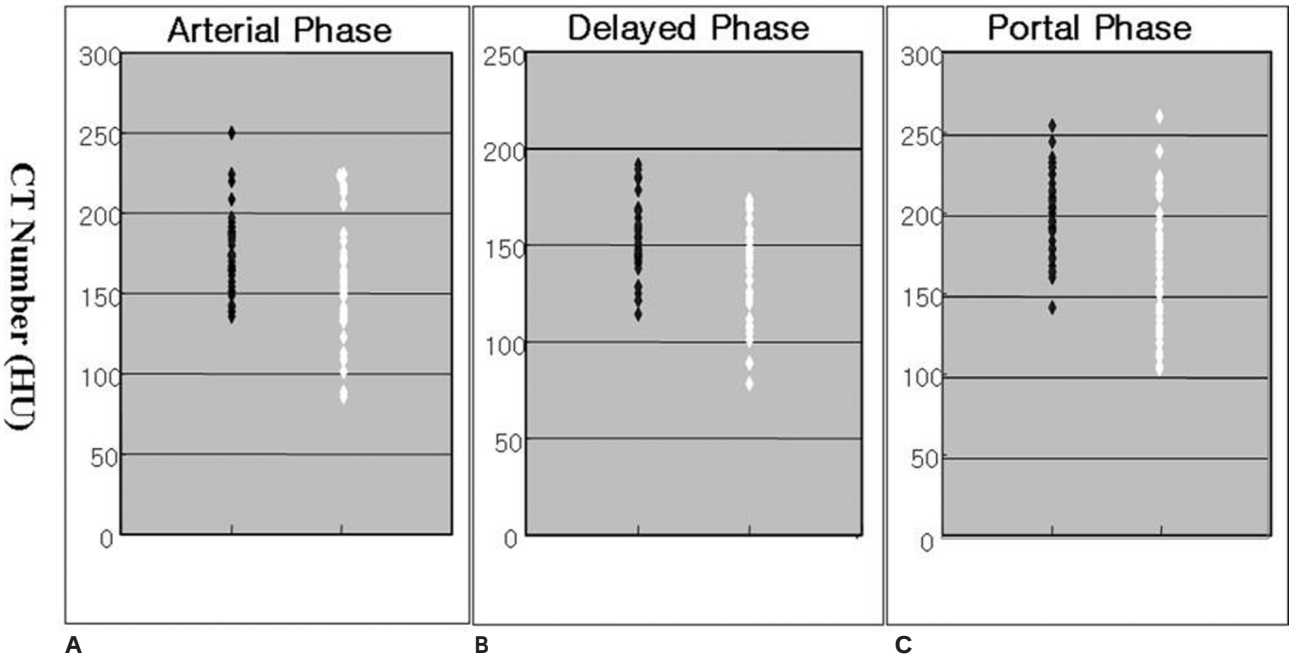

Fig. 2. Scatter plots for renal cortical CT attenuation value (HU) in the control group of subjects (black dots) and cirrhotic group of patients (white dots). For the renal cortices, the mean CT attenuation values were measured in the arterial (**A**), portal (**B**), and delayed (**C**) phases. Although the two groups exhibited different values for the mean CT attenuation value, there is considerable overlap in the individual values in comparisons between the two groups.

Results

The mean CT attenuation values of the renal cortices in cirrhotic patients were significantly lower than the values in control subjects for all three phases: 153.3 ± 37.9 versus 173.3 ± 25.2 in the arterial phase, 172.6 ± 41.0 versus 197.6 ± 26.5 in the portal phase and 136.9 ± 26.0 versus 152.7 ± 20.0 in the delayed phase, respectively; $p < 0.01$ (Table 2). Individual CT attenuation values of the renal cortices for the two groups are represented in a scatter plot (Fig. 2). Although the two groups had different mean CT attenuation values, there was considerable overlap of the individual values between the two groups. In patients with cirrhosis, 23 cases of renal hypoperfusion were identified using a cutoff value of 144.6 HU (-2SD) in the portal phase (Fig. 3). The mean attenuation value of the cases identified on CT with renal hypoperfusion was 111.7 ± 20.1 HU for arterial phase, 119.9 ± 11.8 HU for portal phase and 104.3

Table 2. Mean CT Attenuation Values (HU) of Renal Cortices in the Studied Groups

Group	Control	Cirrhosis	*p*-Value	Hypoperfusion
Arterial Phase	173.3 ± 25.2	153.3 ± 37.9	< 0.01	111.7 ± 20.1
Portal Phase	197.6 ± 26.5	172.6 ± 41.0	< 0.01	119.9 ± 11.8
Delayed Phase	152.7 ± 20.0	136.9 ± 26.0	< 0.01	104.3 ± 12.5

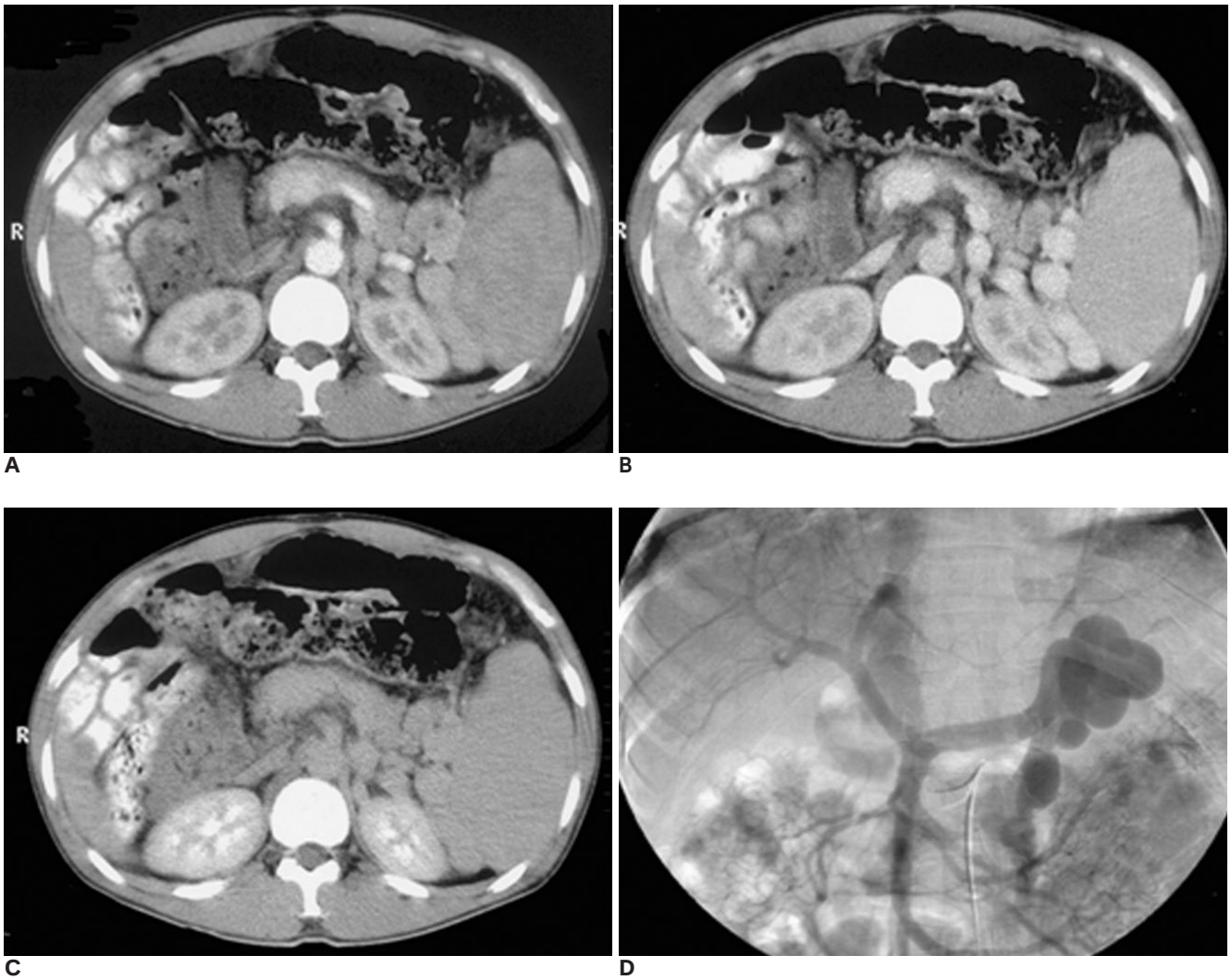

Fig. 3. A three-phase CT scan (**A**-arterial phase, **B**-portal phase, **C**-delayed phase) and arterial-portography (**D**) performed in a patient with a renal shunt. Much of the portal venous inflow is diverted through the renal shunt, showing early opacification of the inferior vena cava. Prominent hypoperfusion of the renal cortex (108 HU in **A**, 112 HU in **B**, 107 HU in **C**) is noted in the images of the three phases studied. This patient has no ascites.

± 12.5 for delayed phase (Table 2). The mean diameter for the renal shunt was 10.6 ± 3.7 mm (range, 5 - 18 mm). Univariate logistic analysis demonstrated that three factors (Child's class B, Child's class C and the presence of a renal shunt) were significantly associated with renal hypoperfusion. However, multiple logistic regression analysis demonstrated only two factors were significantly associated with renal hypoperfusion: Child's class C [adjusted Odds ratio (aOR): 58.4, 95% confidence interval (CI): 3.6 - 956.2] and the presence of a renal shunt (aOR: 7.5, 95% CI: 1.8 - 30.5). Although the renal hypoperfusion in the Child's B group was statistically significant (OR: 11.1, 95% CI: 1.4 - 91.8; $p < 0.01$), this factor was not significant when adjusted for other factors (aOR: 7.8, 95% CI: 0.8 - 76.6; $p < 0.01$). While renal perfusion tended to decline as the Child's grade increased (trend: $p < 0.01$), the same trend was not demonstrated for grade of ascites or age. Interestingly, renal hypoperfusion was not related to either the presence of ascites or the degree of ascites (Table 3).

Discussion

In contrast to the vasodilatation seen in the splanchnic and systemic circulatory systems, the central hemodynamic abnormality occurring in the renal vascular bed in cirrhosis is vasoconstriction. It has been estimated that up to 80% of cirrhotic patients hospitalized with ascites exhibit renal hypoperfusion caused by renal vasoconstriction (6). The mean CT attenuation value of renal cortices in cirrhotic patients was significantly lower than the mean value of control subjects for the three CT phases studied. Moderate degrees of renal vasoconstriction are commonly overlooked in clinical practice, as the sensitivity of the tests used to estimate the glomerular filtration rate (GFR) in the clinical setting is low in patients with cirrhosis. The existence of moderate renal vasoconstriction is important clinically for several reasons. First, patients with moderate renal vasoconstriction have more marked sodium retention and they require higher doses of diuretics than are required by patients with normal renal perfusion for the treatment of, or prevention of, ascites (4). Second, patients with moderate renal vasoconstriction are predisposed to the development of hepatorenal syndrome (7, 8). Third, cirrhotic patients may develop renal dysfunction when treated with a variety of medications, especially nonsteroidal anti-inflammatory drugs (NSAIDs) and aminoglycosides. For the early detection of renal hyperperfusion, there are significant limitations using serum creatinine as a marker for renal function in patients with cirrhosis. Ideally, the GFR should be measured in cirrhotic patients; however, this is not feasible in most settings.

Table 3. Logistic Regression Analysis in the Cirrhotic Group

	Variable	Renal Perfusion		OR	aOR
		Normal	Decrease	95% CI	95% CI
Sex	Female	11	5	1	1
	Male	48	18	0.8 (0.3 - 2.7)	1.1 (0.2 - 6.3)
Age	< 55 years	15	9	1	1
	55 - 64 years	25	8	0.6 (0.2 - 1.9)	1.1 (0.2 - 5.8)
	> 65 years	19	6	0.7 (0.2 - 2.3)	1.3 (0.2 - 7.4)
				Trend: $p = 0.49$	Trend: $p = 0.76$
Child's	A	26	1	1	1
	B	28	12	11.1 (1.4 - 91.8)	7.8 (0.8 - 76.6)
	C	5	10	52.0 (5.4 - 502.1)	58.4 (3.6 - 956.2)
				Trend: $p < 0.01$	Trend: $p < 0.01$
Ascites	1	34	8	1	1
	2	13	7	2.3 (0.7 - 7.6)	0.7 (0.1 - 4.2)
	3	7	6	3.6 (1.0 - 13.8)	1.1 (0.2 - 7.5)
	4	5	2	1.7 (0.3 - 10.4)	0.3 (0.1 - 3.9)
				Trend: p = 0.13	Trend: p = 0.54
SRS	NO	47	7	1	1
	YES	12	16	9.0 (3.0 - 26.7)	7.5 (1.8 - 30.5)

Note: SRS = spontaneous splenorenal shunt; OR = Odds ratio; aOR = adjusted Odds ratio; CI = confidence interval
Renal hypoperfusion is defined as the mean CT attenuation value of the identified renal cortices less two standard deviations (SD) as compared to the value for control subjects determined in the portal phase.

An alternative diagnostic approach is the use of Doppler sonography to assess the resistive index (RI) of the renal vasculature. Previous studies have shown (7, 9) that an abnormal renal RI predicts an increased chance for development of hepatorenal syndrome and kidney dysfunction. Although abnormal values may identify patients at high risk, an elevated RI (greater than 0.70) usually suggests the presence of a number of other intrinsic renal diseases (10); however, more information is required before the use of the RI can be recommended as a standard procedure. With increasing use of cross-sectional imaging, dynamic CT, which can acquire the corticomedullary phase of renal enhancement, has assumed a greater role in the evaluation of renal perfusion. The attenuation value of renal cortices, as identified by CT, may provide data for calculating important physiological parameters, such as renal perfusion and the GFR (11). Changes in the CT attenuation value, in a region after an intravenous injection of contrast medium, may be used to calculate blood flow per unit volume of tissue. As the CT attenuation value of renal cortices in the corticomedullary phase documents renal perfusion, images obtained during this phase might be of particular value for detecting abnormalities in renal perfusion (11). A hyperattenuating cortical nephrogram with corticomedullary differentiation is obtained during the corticomedullary phase, which occurs approximately 25 - 80 seconds after the start of intravenous contrast material administration (12). With our method, the time interval between the initiation of injection of contrast agent and the beginning of CT scanning is predetermined, and is the same in all patients. Although the CT attenuation values of renal cortices in cirrhotic patients was significantly lower than the values of control subjects for the three phases, the arterial phase (30 sec) image may not ensure maximum enhancement of the renal cortex in all cases; as it does not account for differences among individuals for circulation time or blood volume. Therefore, for the three phase images studied, only the portal phase (60 sec) image was used for the selection of patients with renal hypoperfusion, as the density of the renal cortex was greatest at approximately one minute after the bolus injection of contrast material. As differential CT values for renal hypoperfusion do not exist for normal renal perfusion and decreased renal perfusion, we therefore defined renal hypoperfusion in cirrhotic patients to have less than two standard deviations in the number of renal cortices on CT imaging as compared to control patients during the portal phase imaging. Based on our results, although the cutoff value (144.6 HU) was relatively high, the mean CT attenuation value of the renal cortices for patients with hypoperfusion was very low (119.9 HU). Contrast material starts to appear in the collecting system in the delayed phase, which occurs approximately three to five minutes after the start of intravenous contrast material administration. Delayed phase imaging was useful to rule out a urinary obstruction that could have affected the cortical nephrogram.

The most widely accepted theory suggests that renal vasoconstriction is the consequence of underfilling of the systemic arterial circulation secondary to marked vasodilatation of the splanchnic circulation (1 - 4). This vasodilatation is a characteristic feature of cirrhosis and contributes to the hyperdynamic circulatory state, resulting in an increase of portal venous inflow. Portal venous flow does not reflect portal venous inflow (which is equivalent to the sum of portal and the collateral blood flow) as much of the latter is diverted into portal-systemic collaterals, and because portal venous flow is normal or reduced in patients with cirrhosis (13). Even though the collateral vessels serve to decompress the hepatic vascular bed, they are actually high-resistance vessels. Thus, there is inadequate decompression of the high-pressure portal vascular bed, and portal pressure remains high. The increase in portal venous inflow resulting from splanchnic vasodilatation represents not only an important factor that contributes to maintain or worsen not only portal pressure elevation but also hyperdynamic circulation. However, for evaluation of hyperdynamic circulation, measurement of portal inflow is not feasible in clinical practice as diverse collateral routes may be present in patients with cirrhosis. Therefore, a representative collateral route indicating hyperdynamic circulation is needed among various collateral routes. Ohm's law defines the pressure in any vessel: P = Q X R, where P is the pressure, Q is the flow, and R is the resistance to flow through a vessel. The resistance can be further measured using Poiseulle's law: R = 8nL/r4, where L is the length of the vessel, r is the radius of the vessel, and n is the coefficient of viscosity. The radius of the vessel appears to be the most important factor because small changes in the radius are associated with large changes in resistance. As most collateral vessels, including esophageal or umbilical collateral vessels, have a long length and small radius, the flow volume through these collateral routes may be relatively small due to high vascular resistance,

and therefore cannot be used as a predictor of hyperdynamic circulation. However, renal shunts that are identified by imaging may be useful factors to suggest an increase of diverting portal inflow as they usually have a short length and larger diameter, similar to surgical shunts. Therefore, renal shunts may be a representative collateral route that can be used to characterize hyperdynamic circulation. This concept is supported by the fact that the portal venous pressure has been shown to be significantly lower in patients with large gastrorenal shunts (14). In our study, the clinical factors significantly associated with renal hypoperfusion, as determined by logistic regression analysis, were Child's class C and the presence of a renal shunt. Thus, our findings suggest that a renal shunt (hyperdynamic circulation) and renal vasoconstriction are intimately related to each other, and serve as compensatory mechanisms to maintain effective arterial blood volume and arterial pressure in patients with cirrhosis. With the advent of modern imaging techniques, such as sonography, CT, and MR imaging, renal shunts can now be easily detected.

In conclusion, the presence of a renal shunt may be a useful predictor for renal hypoperfusion in addition to the Child's grade in patients with cirrhosis. Identification of moderate renal hypoperfusion can aid in the management of patients with cirrhosis.

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간경변 환자에서 콩팥 관류감소와 연관된 지라콩팥 단락[1]

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목적: 자발 지라-콩팥 단락이 간경변 환자의 초기 콩팥 혈역동학 변화의 예견 인자로 사용될 수 있는지 알아보고자 하였다.

대상과 방법: 간경변 환자 82명과 대조군 41명을 대상으로 하였다. 3기 전산화단층촬영이 시행되었고 콩팥 관류를 알아보기 위해 콩팥 피질에서 CT 값을 측정하였다. 콩팥 관류감소 발생 성향을 나이, 복수, Child 등급에 따라 알아보았다.

결과: 간경변 환자에서 콩팥 피질 평균 CT 값은 대조군에 비해 3기 모두에서 낮았다(동맥기: 153.3 ± 37.9 대 173.3 ± 25.2, 문맥기: 172.6 ± 41.0 대 197.6 ± 26.5, 지연기: 136.9 ± 26.0 대 152.7 ± 20.0). 콩팥 관류 감소 환자에서 피질 CT 값은 문맥기에 119.9 ± 11.8였다. 두 가지 요인이 콩팥 관류 감소와 연관이 있었다: Child 등급 C (교정 확률 비: 58.4, 95% 신뢰구간: 3.6 - 956.2; $p < 0.01$) 와 지라콩팥 단락(교정 확률 비: 7.5, 95% 신뢰구간: 1.8 - 30.5; $p < 0.01$). 또한, 콩팥 관류 감소는 복수량 또는 나이와 상관이 없었고 Child등급에 따라 (경향: $p < 0.01$) 진행하였다.

결론: 심한 간질환과 연관해서 늘어난 자발 지라콩팥 단락은 간경변에서 콩팥 관류 감소의 위험 요인이 될 수 있다.