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Prevalence and Clinical Characteristics of Spontaneous Splenorenal Shunt in Liver Cirrhosis: A Retrospective Observational Study Based on Contrast-Enhanced Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) Scans

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

ABCDEF 1 Xingshun Qi
CD 2 Xiaolong Qi
CD 1 Yongguo Zhang
CD 1 Xiaodong Shao
CD 1 Chunyan Wu
CD 3 Yongji Wang
BC 1 Ran Wang
BC 1 Xintong Zhang
BC 1 Han Deng
BC 1 Feifei Hou
BC 1 Jing Li
ADE 1 Xiaozhong Guo

1 Liver Cirrhosis Group, Department of Gastroenterology, General Hospital of Shenyang Military Area, Shenyang, Liaoning, P.R. China
2 Department of General Surgery, Nanfang Hospital, Southern Medical University, Guangzhou, Guangdong, P.R. China
3 Department of Health Statistics, Fourth Military Medical University, Xi'an, Shaanxi, P.R. China

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Corresponding Authors: Xiaozhong Guo, e-mail: guo_xiao_zhong@126.com and Xingshun Qi, e-mail: xingshunqi@126.com

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Background: This is a retrospective observational study evaluating the prevalence and clinical characteristics of spontaneous splenorenal shunt in liver cirrhosis.

Material/Methods: We included a total of 105 cirrhotic patients who were admitted to our hospital between June 2012 and December 2013 and underwent contrast-enhanced CT and/or MRI scans at admissions. Spontaneous splenorenal shunt was identified. Clinical and laboratory data were compared between cirrhotic patients with and without spontaneous splenorenal shunt.

Results: The prevalence of spontaneous splenorenal shunt was 10.5% (11/105). The prevalence of hepatic encephalopathy was higher in patients with spontaneous splenorenal shunt than in those without spontaneous splenorenal shunt (18.2% *vs.* 4.3%, p=0.062), but the difference between them was not statistically significant. The prevalence of acute upper-gastrointestinal bleeding was lower in patients with spontaneous splenorenal shunt than in those without spontaneous splenorenal shunt (0% *vs.* 18.1%, p=0.205), but the difference between them was not statistically significant. Patients with spontaneous splenorenal shunt had significantly higher Child-Pugh scores (9.50±1.65 *vs.* 7.43±2.02, p=0.002) and MELD scores (11.26±7.29 *vs.* 5.67±6.83, p=0.017) than those without spontaneous splenorenal shunt. In-hospital mortality was similar between them (0% *vs.* 4.3%, p=1.000).

Conclusions: Spontaneous splenorenal shunt might be associated with worse liver function in liver cirrhosis, but not with in-hospital mortality.

MeSH Keywords: Liver Cirrhosis • Prevalence • Risk Factors • Splenorenal Shunt • Survival

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Background

Spontaneous formation of portosystemic shunt is one of classical signs of portal hypertension [1]. The major mechanism is that the blood flow is redirected from the high-pressure portal vessels to the low-pressure systemic vessels [2]. In nature, spontaneous portosystemic shunt is a compensation for portal hypertension [3,4]. However, great damage is caused by the overgrowth of portosystemic shunts. For example, gastroesophageal varices, the most common type of spontaneous portosystemic shunt in cirrhotic portal hypertension, will result in life-threatening upper-gastrointestinal hemorrhage [5]. Large spontaneous portosystemic shunts increase the risk of persistent hepatic encephalopathy [6].

Splenorenal shunt refers to a communication between splenic vein and left renal vein [1]. It is a relatively uncommon type of spontaneous portosystemic shunt and is less recognized in clinical practice [7–9]. An early case-control study suggested that patients with large spontaneous splenorenal or gastrorenal shunt have a higher risk of large esophageal varices, but have a similar risk of variceal bleeding [10]. However, another early study found that only 10% of patients with spontaneous splenorenal or gastrorenal shunt had esophageal varices alone, but 48.8% of patients without spontaneous splenorenal or gastrorenal shunt had esophageal varices alone [11]. Most researchers agree that the risk of chronic hepatic encephalopathy is increased by the presence of spontaneous splenorenal shunt [10]. However, a recent study found that the grade of hepatic encephalopathy was similar between patients with and without spontaneous splenorenal shunt [12]. Due to substantial controversies among studies, more research is needed to explore the clinical significance of spontaneous splenorenal shunt in liver cirrhosis.

We performed the present retrospective observational study to evaluate the prevalence and clinical characteristics of spontaneous splenorenal shunt in liver cirrhosis.

Material and Methods

Patient selection

Inclusion criteria were: 1) patients admitted to our hospital between June 2012 and December 2013; 2) patients with a diagnosis of liver cirrhosis; and 3) patients who underwent contrast-enhanced CT and/or MRI scans at admissions. Exclusion criteria were: 1) patients with malignancy, including hepatocellular carcinoma; 2) patients who underwent splenectomy; and 3) patients who underwent surgical splenorenal shunt. Notably, some of them had been included in our previous studies [13–22]. The study protocol of this retrospective observational study was approved by the Medical Ethical Committee of our hospital. The approval number was k (2016)03. The requirement for written informed consent was waived due to the nature of this study.

Data collection

Our study group consistently collected clinical and laboratory data from the electronic medical charts. The accuracy of clinical and laboratory data for the present study was further validated by 2 investigators (XQ and HD). The primary data included age, sex, etiology of liver cirrhosis, history of diabetes, major clinical presentations at their admissions (i.e., ascites, hepatic encephalopathy, and acute upper-gastrointestinal bleeding), presence and severity of esophageal varices at endoscopy, red blood cell, hemoglobin, white blood cell, platelet count, total bilirubin, albumin, alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase, gamma-glutamyl transpeptidase, blood urea nitrogen, creatinine, potassium, sodium, prothrombin time, activated partial thromboplastin time, and international normalized ratio. Child-Pugh score and class and model for end-stage of liver disease (MELD) score were calculated. In-hospital death was also recorded.

The imaging data was evaluated by 1 investigator (XQ), who discussed uncertain cases with other investigators. Presence of spontaneous splenorenal shunt was identified by consecutively tracing the origins of splenic vein and left renal vein and their confluence on the axial contrast-enhanced CT and/or MRI scans. Notably, in the cases with spontaneous splenorenal shunt, multiple planes of CT and/or MRI scans should be studied due to the presence of tortuous collateral circulation. Additionally, the maximum diameters of the spleen, splenic vein, and main portal vein were measured.

Data analysis

Patient characteristics were summarized by the mean with standard deviation and the median with range for continuous variables and the frequency (percentage) for categorical variables. Patients were divided into 2 groups according to the presence of spontaneous splenorenal shunt. The data regarding the clinical symptoms, laboratory tests, and images were compared between patients with and without spontaneous splenorenal shunt. Continuous data were compared by using the independent sample *t* test, and categorical data were compared by using the chi-square or Fisher's exact tests. P value<0.05 was considered statistically significant. All statistical analyses were performed by the SPSS statistical software version 16.0.0.

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Results

Patients

Overall, 113 cirrhotic patients underwent contrast-enhanced CT and/or MRI scans. Among them, 91 patients underwent contrast-enhanced CT scans alone and 22 patients underwent contrast-enhanced MRI scans alone.

Because 8 patients who underwent splenectomy were excluded, 105 patients were finally included. Among them, 85 patients underwent contrast-enhanced CT scans alone, and 20 patients underwent contrast-enhanced MRI scans alone. Patient characteristics are shown in Table 1. A majority of included patients were male, had hepatitis virus- or alcohol-related liver cirrhosis, and had Child-Pugh class A and B. In-hospital mortality was 3.8% (4/105).

The prevalence of spontaneous splenorenal shunt was 10.5% (11/105) (Figures 1, 2). Patient characteristics at admission were compared between patients with and without spontaneous splenorenal shunt (Table 2).

Clinical symptoms

The prevalence of hepatic encephalopathy was higher in patients with spontaneous splenorenal shunt than in those without (18.2% *vs.* 4.3%), but the difference between them was not statistically significant (p=0.062).

None of the patients with spontaneous splenorenal shunt presented with acute upper-gastrointestinal bleeding, but 18.1% of patients without spontaneous splenorenal shunt presented with acute upper-gastrointestinal bleeding; however, the difference between them was not statistically significant (p=0.205).

All of the patients with spontaneous splenorenal shunt who underwent upper-gastrointestinal endoscopy had esophageal varices and 84.4% of patients without spontaneous splenorenal shunt who underwent upper-gastrointestinal endoscopy had esophageal varices, but the difference between them was not statistically significant (p=0.066).

Laboratory tests

Patients with spontaneous splenorenal shunt had significantly lower albumin levels than those without spontaneous splenorenal shunt (p<0.001).

Patients with spontaneous splenorenal shunt had significantly higher alkaline phosphatase levels than those without spontaneous splenorenal shunt (p=0.004).

Patients with spontaneous splenorenal shunt had significantly higher Child-Pugh (p=0.002) and MELD (p=0.017) scores than those without spontaneous splenorenal shunt, and the proportion of Child-Pugh class was significantly different between them (p=0.001).

Images

The maximum diameter of the main portal vein was significantly smaller in patients with spontaneous splenorenal shunt than in those without spontaneous splenorenal shunt (p<0.001).

Discussion

In this retrospective study, we explored the prevalence and clinical characteristics of spontaneous splenorenal shunt in liver cirrhosis. Tarantino and Maruyama also performed 2 similar studies on the implications of spontaneous splenorenal shunt in liver cirrhosis [12,23]. In the 2 studies, splenorenal shunt was evaluated by Doppler ultrasound. In Tarantino's study, hepatocellular carcinoma was not excluded. By comparison, the major feature of our study was that we carefully checked the images on the axial contrast-enhanced CT and MRI scans to identify the presence of spontaneous splenorenal shunt; therefore, our findings might be more objective and accurate. Several additional characteristics of the present study are: 1) the clinical and laboratory data were collected by our study group and validated by 2 investigators; 2) the data on diameters of spleen, splenic vein, and main portal vein were also collected; and 3) hepatocellular carcinoma was excluded.

A major finding of our study was that the presence of spontaneous splenorenal shunt was associated with worse liver function (i.e., higher Child-Pugh and MELD scores) in liver cirrhosis. This association was primarily due to a significantly lower albumin level in cirrhotic patients with spontaneous splenorenal shunt. In addition, we should acknowledge that cirrhotic patients with spontaneous splenorenal shunt had higher total bilirubin, creatinine, and international normalized ratio and higher prevalence of hepatic encephalopathy and ascites. Our findings are similar to the findings of Maruyama et al. that hepatic decompensation was more frequently observed in patients with splenorenal shunts, but are in contrast to the findings of Tarantino et al. that Child-Pugh classification did not predict splenorenal shunts (odds ratio: 1.145, 95% confidence intervals: 0.77–1.51, p=0.66). Further research using repeated validation is needed.

We could not determine the order of spontaneous splenorenal shunt and liver dysfunction. If it is true that the occurrence of spontaneous splenorenal shunt results in the deterioration of liver function, the potential mechanism may be as

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Table 1. Patient characteristics.

	N	Mean or frequency (percentage)	Std. deviation	Median	Minimum	Maximum
Age (years)	105	55.21	13.01	55.19	22.14	85.46
Sex (Male/Female)	105	71 (67.6%)/34 (32.4%)				
Etiology of liver diseases – n.	105					
– Hepatitis B virus alone		32 (30.5%)				
– Hepatitis C virus alone		7 (6.7%)				
– Hepatitis B + C virus		3 (2.9%)				
– Alcohol		30 (28.6%)				
– Hepatitis B virus + Alcohol		7 (6.7%)				
– Hepatitis C virus + Alcohol		1 (1%)				
– Autoimmunity		5 (4.8%)				
– Drug-related		2 (1.9%)				
– Unknown		18 (17.1%)				
Diabetes – n.	105	13 (12.4%)				
Ascites – n.	105	59 (56.2%)				
Hepatic encephalopathy – n.	104	6 (5.8%)				
Acute upper-gastrointestinal bleeding – n.	105	17 (16.2%)				
Esophageal varices at endoscopy – n.	42					
– No		10 (23.8%)				
– Mild		2 (4.8%)				
– Moderate		9 (21.4%)				
– Severe		21 (50%)				
Gastric varices at endoscopy – n.	42	24 (57.1%)				
Hemoglobin (g/L)	102	106.18	30.30	107.00	42.00	170.00
White blood cell count (10^9/L)	102	4.96	3.21	4.15	1.50	20.50
Platelet count (10^9/L)	102	92.97	76.65	73.50	11.00	545.00
Total bilirubin (umol/L)	104	48.44	71.87	23.80	5.10	436.50
Albumin (g/L)	103	32.33	6.72	32.20	11.70	44.30
Alanine aminotransferase (U/L)	104	51.61	63.08	33.00	8.00	429.00
Aspartate aminotransferase (U/L)	104	74.13	98.12	47.00	10.00	889.00
Alkaline phosphatase (U/L)	104	117.67	80.09	92.00	34.00	524.40
Gamma-glutamyl transpeptidase (U/L)	104	159.73	221.69	68.50	12.00	1130.00
Blood urea nitrogen (mmol/L)	102	5.80	2.60	5.26	1.73	17.18
Creatinine (umol/L)	102	59.42	21.61	57.00	29.00	151.00
Potassium (mmol/L)	103	4.04	0.47	4.00	3.01	5.43
Sodium (mmol/L)	103	138.02	6.50	139.20	83.00	144.50
Prothrombin time (seconds)	103	16.34	6.55	14.80	11.40	62.80
Activated partial thromboplastin time (seconds)	103	45.13	16.53	42.10	29.90	180.00
International normalized ratio	103	1.35	0.84	1.16	0.77	7.96
Serum ammonia (umol/L)	53	46.04	32.59	35	9	127
Child-Pugh score	100	7.64	2.07	8.00	5.00	12.00
Child-Pugh class A/B/C	100	37 (37%)/40 (40%)/23 (23%)				
MELD score	100	6.23	7.05	4.86	(5.20)	34.52
Maximum diameter of spleen (mm)	105	140.27	30.77	138.20	83.80	240.90
Maximum diameter of splenic vein (mm)	105	10.68	3.83	10.30	4.30	29.60
Maximum diameter of main portal vein (mm)	105	18.34	5.18	18.00	0.00	31.00
In-hospital mortality	105	4 (3.8%)				

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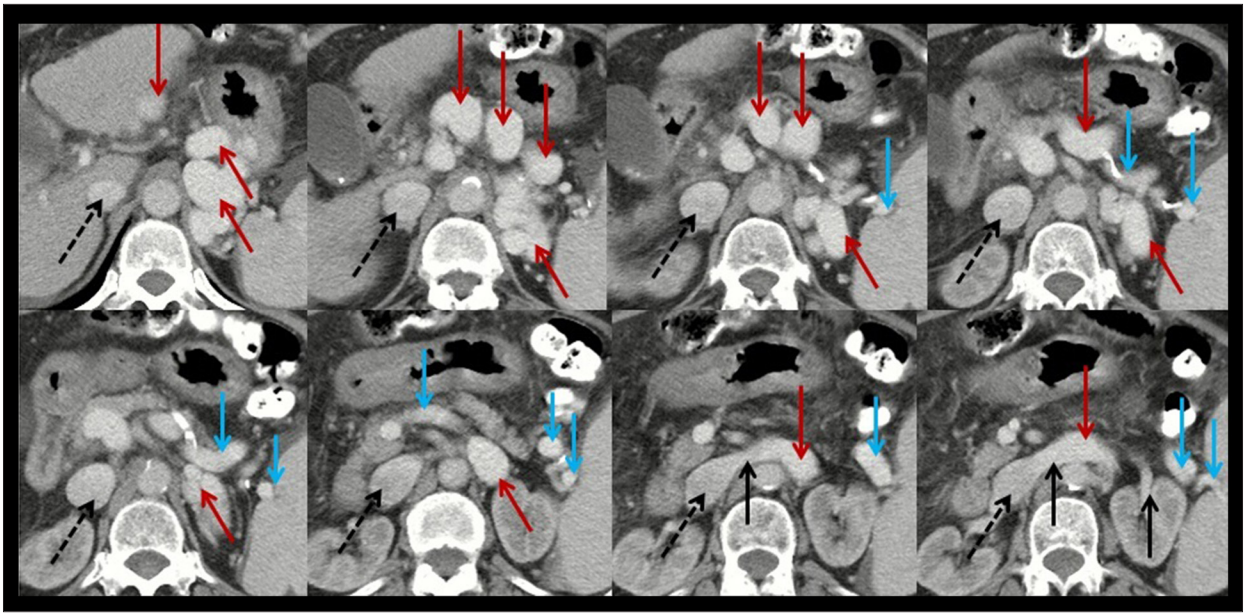

Figure 1. Axial contrast-enhanced computed tomography at the portal vein phase in a 63-year-old female patient with spontaneous splenorenal shunt (HXY). Black dashed arrows represent the inferior vena cava; black solid arrows represent the left renal vein; blue solid arrows represent the splenic vein; red solid arrows represent the communication between splenic vein and left renal vein.

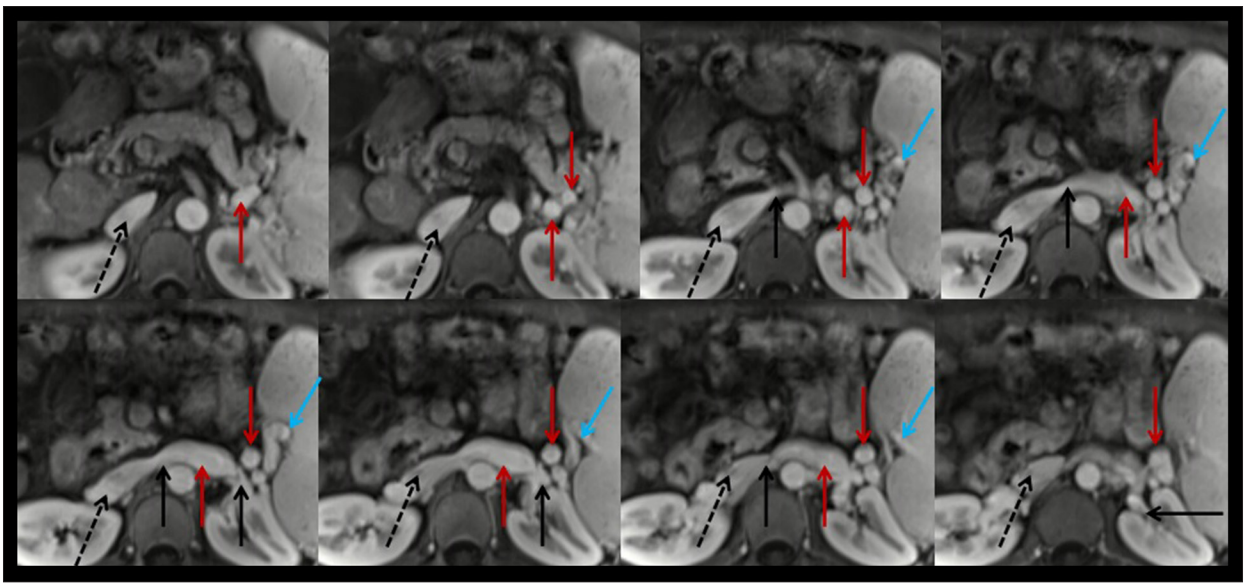

Figure 2. Axial contrast-enhanced MRI scans at the portal vein phase in a 44-year-old male patient with spontaneous splenorenal shunt (MQS). Black dashed arrows represent the inferior vena cava; black solid arrows represent the left renal vein; blue solid arrows represent the splenic vein; red solid arrows represent the communication between splenic vein and left renal vein.

follows: hepatic perfusion is largely reduced in patients with spontaneous splenorenal shunt, thereby leading to the liver function abnormality. If the perspective that the development of spontaneous splenorenal shunt is secondary to the deterioration of liver function is supported, the potential mechanism may be as follows: portal pressure is largely elevated in cirrhotic patients with poor liver function, thereby inducing the development of spontaneous splenorenal shunt. Certainly, we should never neglect a synergistic effect between spontaneous splenorenal shunt and liver dysfunction.

Our study did not find any significant association between spontaneous splenorenal shunt and in-hospital death. This is primarily explained by the small number of patients included

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Table 2. Comparison of characteristics between patients with and without splenorenal shunt.

Variables	With splenorenal shunt (n=11)			Without splenorenal shunt (n=94)			P value
	N	Mean or frequency (percentage)	Std. deviation	N	Mean or frequency (percentage)	Std. deviation	
Age (years)	11	56.89	12.98	94	55.02	13.07	0.653
Sex (Male/Female)	11	6 (54.5%)/ 5 (45.5%)		94	65 (69.1%)/ 29 (30.9%)		0.327
Etiology of liver diseases – n.	11			94			0.059
– Hepatitis B virus alone		4 (36.4%)			28 (29.8%)		
– Hepatitis C virus alone		0 (0%)			7 (7.4%)		
– Hepatitis B + C virus		0 (0%)			3 (3.2%)		
– Alcohol		0 (0%)			27 (28.7%)		
– Hepatitis B virus + Alcohol		3 (27.3%)			7 (7.4%)		
– Hepatitis C virus + Alcohol		0 (0%)			1 (1.1%)		
– Autoimmunity		3 (27.3%)			2 (2.2%)		
– Drug-related		0 (0%)			2 (2.2%)		
– Unknown		1 (9.1%)			17 (18.1%)		
Diabetes – n.	11	2 (18.2%)		94	11 (11.7%)		0.537
Ascites – n.	11	7 (63.6%)		94	52 (55.3%)		0.599
Hepatic encephalopathy – n.	11	2 (18.2%)		94	4 (4.3%)		0.062
Acute upper-gastrointestinal bleeding – n.	11	0 (0%)		94	17 (18.1%)		0.205
Esophageal varices at endoscopy – n.	3			39			0.066
– No		0 (0%)			10 (25.6%)		
– Mild		1 (33.3%)			1 (2.6%)		
– Moderate		0 (0%)			9 (23.1%)		
– Severe		2 (66.7%)			19 (48.7%)		
Gastric varices at endoscopy – n.	3	3 (100%)		39	21 (53.8%)		0.247
Hemoglobin (g/L)	10	89.90	24.53	92	107.95	30.45	0.073
White blood cell count (10^9/L)	10	5.19	4.25	92	4.94	3.10	0.814
Platelet count (10^9/L)	10	90.50	60.55	92	93.24	78.47	0.915
Total bilirubin (umol/L)	11	52.56	31.01	93	47.95	75.34	0.842
Albumin (g/L)	11	25.70	5.92	92	33.12	6.39	<0.001
Alanine aminotransferase (U/L)	11	56.09	70.28	93	51.08	62.57	0.804
Aspartate aminotransferase (U/L)	11	91.09	59.43	93	72.12	101.77	0.547
Alkaline phosphatase (U/L)	11	182.85	134.31	93	109.96	68.21	0.004
Gamma-glutamyl transpeptidase (U/L)	11	218.27	254.72	93	152.81	217.97	0.357
Blood urea nitrogen (mmol/L)	10	6.98	3.73	92	5.68	2.45	0.134

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Table 2 continued. Comparison of characteristics between patients with and without splenorenal shunt.

Variables	With splenorenal shunt (n=11)			Without splenorenal shunt (n=94)			P value
	N	Mean or frequency (percentage)	Std. deviation	N	Mean or frequency (percentage)	Std. deviation	
Creatinine (umol/L)	10	71.75	38.85	92	58.08	18.73	0.057
Potassium (mmol/L)	10	3.89	0.50	93	4.05	0.46	0.298
Sodium (mmol/L)	10	136.91	4.09	93	138.14	6.71	0.573
Prothrombin time (seconds)	10	18.88	4.94	93	16.06	6.66	0.198
Activated partial thromboplastin time (seconds)	10	49.23	13.81	93	44.68	16.80	0.411
International normalized ratio	10	1.61	0.55	93	1.32	0.86	0.309
Serum ammonia (umol/L)	6	60.50	27.19	47	44.19	33.01	0.698
Child-Pugh score	10	9.50	1.65	90	7.43	2.02	0.002
Child-Pugh class A/B/C	10	0 (0%)/ 3 (30%)/ 7 (70%)		90	37 (41.1%)/ 37 (41.1%)/ 16 (17.8%)		0.001
MELD score	10	11.26	7.29	90	5.67	6.83	0.017
Maximum diameter of spleen (mm)	11	147.71	25.84	94	139.40	31.30	0.399
Maximum diameter of splenic vein (mm)	11	10.43	1.79	94	10.71	4.01	0.816
Maximum diameter of main portal vein (mm)	11	12.30	5.35	94	19.05	4.70	<0.001
In-hospital mortality	11	0 (0%)		94	4 (4.3%)		1.000

and the low in-hospital mortality. Additionally, information regarding long-term outcome was not available in our study. Therefore, additional work is needed to establish whether spontaneous splenorenal shunt influences the long-term survival of cirrhotic patients. Recently, Maruyama et al. examined the clinical effect of splenorenal shunt and short gastric vein on long-term outcomes in patients with cirrhosis. They found that the 1-, 3-, and 5-year cumulative survival rates were 80%, 66.6%, and 58.3% in patients with splenorenal shunt; 94.1%, 87.4%, and 72.8% in those with short gastric vein; and 88.3% 73.1%, and 58%, respectively, in those without splenorenal shunt or short gastric vein. The difference among the 3 groups was not statistically significant (p=0.2). It appears that patients with short gastric veins have the best survival, followed by patients without splenorenal shunt or short gastric vein and patients with splenorenal shunt.

Another finding of our study was a very low risk of acute upper-gastrointestinal bleeding in cirrhotic patients with spontaneous splenorenal shunt. This finding is readily explained by the fact that spontaneous splenorenal shunt reduces the portal pressure, thereby preventing development of variceal bleeding. It is seemingly contradictory that the prevalence of esophageal varices at endoscopy was higher in cirrhotic patients with spontaneous splenorenal shunt than in those without spontaneous splenorenal shunt. However, it should be noted that only a low proportion of cirrhotic patients underwent endoscopic examinations. Thus, the potential selection bias should be emphasized.

Several additional limitations of our study should be acknowledged. First, angiography was not regularly performed due to its invasiveness. Second, due to the retrospective nature of our study, not all included patients had relevant clinical and laboratory data. Third, the data at previous admissions were not collected.

Conclusions

In conclusion, the presence of spontaneous splenorenal shunt indicates more severe liver damage. Additionally, it might be associated with a higher prevalence of hepatic encephalopathy, but a lower prevalence of acute upper-gastrointestinal bleeding. Considering its clinical significance, physicians should pay more attention to the presence of spontaneous splenorenal

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shunt. Further well-designed prospective studies are warranted to explore the clinical implications of spontaneous splenorenal shunt in liver cirrhosis.

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Conflict of interest

None.

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