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Original Article

Diameters of left gastric vein and its originating vein on magnetic resonance imaging in liver cirrhosis patients with hepatitis B: Association with endoscopic grades of esophageal varices

Hai-ying Zhou,[1] Tian-wu Chen,[1] Xiao-ming Zhang,[1] Nan-lin Zeng,[1] Li Zhou,[1] Hong-jie Tang,[1] Dan Wang,[1] Su Jian,[1] Juan Liao,[2] Jun-ying Xiang,[2] Jiani Hu[3] and Zishu Zhang[4]

[1]*Sichuan Key Laboratory of Medical Imaging, and Department of Radiology,* [2]*Department of Gastroenterology, Affiliated Hospital of North Sichuan Medical College, Nanchong, China;* [3]*Department of Radiology, Wayne State University, Detroit, and* [4]*Department of Radiology, University of Michigan Health System, Ann Arbor, Michigan, USA*

Aim: To determine whether diameters of the left gastric vein (LGV) and its originating vein are associated with endoscopic grades of esophageal varices.

Methods: Ninety-eight liver cirrhotic patients with hepatitis B undergoing magnetic resonance (MR) portography, and upper gastrointestinal endoscopy for grading esophageal varices were enrolled. Diameters of the LGV and its originating vein – the splenic vein (SV) or portal vein (PV) – were measured on MR imaging. Statistical analyses were performed to identify the association of the diameters with the endoscopic grades.

Results: Univariate analysis showed that the SV was predominantly the originating vein of the LGV, and diameters of the LGV and SV were associated with grades of esophageal varices. Diameters of the LGV ($P = 0.023$, odds ratio [OR] = 1.583) and SV ($P = 0.012$, OR = 2.126) were independent risk factors of presence of the varices. Cut-off LGV diameters of 5.1 mm, 5.9 mm, 6.6 mm, 7.1 mm, 7.8 mm and 5.8 mm; or cut-off SV diameters of 7.3 mm, 7.9 mm, 8.4 mm, 9.5 mm, 10.7 mm and 8.3 mm, could discriminate grades 0 from 1, 0 from 2, 0 from 3, 1 from 3, 2 from 3, and 0–1 from 2–3, respectively.

Conclusion: Diameters of the LGV and SV are associated with endoscopic grades of esophageal varices.

Key words: endoscopy, esophageal varices, left gastric vein, liver cirrhosis, magnetic resonance imaging

INTRODUCTION

MASSIVE HEMORRHAGE OF the upper alimentary tract resulting from esophageal varices, which are mainly supplied by an enlarged left gastric vein (LGV) originating from the splenic vein (SV) or portal vein (PV) and running to the esophagogastric junction along the lesser curvature of stomach, is a major complication of portal hypertension (PHT) secondary to liver cirrhosis.[1,2] At least two-thirds of patients with cirrhosis develop the varices, and approximately 10–60% of patients experience variceal bleeding.[1,3–5] According to the criteria proposed by the Japanese Research Society for Portal Hypertension,[6] grade 2 and 3 varices have a high risk of causing life-threatening upper gastrointestinal hemorrhage. The gold standard method for the identification of the grades of the varices is upper gastrointestinal endoscopy. However, it is invasive and uncomfortable, and this can limit the frequency of examination.[7]

Recent studies have been performed to identify predictive non-invasive factors for esophageal varices such as platelet count of 82 000/uL or less, PV diameter of 11.5 mm or more, and anteroposterior splenic measurement of 103 mm or more, but none of the factors could visualize the varices, and how to grade the varices with these factors were not studied.[8–11] With the development

Correspondence: Professor Tian-wu Chen, Department of Radiology, Affiliated Hospital of North Sichuan Medical College, 63 Wenhua Road, Shunqing District, Nanchong, Sichuan 637000, China. Email: chentw@aliyun.com
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of imaging technology, magnetic resonance (MR) portography has been described as being comparable to endoscopy for the detection of esophageal varices due to its short acquisition time, high signal-to-noise ratio and no radiation.[12–16] It can not only visualize the anatomical distributions of the varices, but also can analyze the inflowing vein of the varices (LGV) and its originating vein which play important roles in the formation and development of the varices.[2,17–19] Furthermore, cirrhotic patients often receive hepatocellular carcinoma surveillance with MR imaging which could be used as a "one-stop-shop" approach evaluating the varices at the same time without the need for a second study.[20] To our knowledge, there has been no report focusing on the utility of MR imaging to determine the association of the presence and endoscopic grades of the varices with the diameters of the inflowing vessel (LGV) and its originating vein (PV or SV). Therefore, the aim of this study was to determine whether the diameters of LGV and its originating veins are associated with the presence and endoscopic grades of esophageal varices for better understanding and to prevent massive hemorrhage of the upper alimentary tract.

METHODS

Ethics statement

THE STUDY WAS approved by the institutional ethics review board of our university hospital, and written informed consent was obtained from each participant before the study.

Patient population

Patients were enrolled into this study according to the following inclusion criteria: (i) PHT secondary to liver cirrhosis in patients with hepatitis B was confirmed by clinical data, laboratory examinations and imaging study according to the American Association for the Study of Liver Diseases practice guidelines 2007 – Chronic Hepatitis B;[21] and (ii) patients underwent 3-D contrast-enhanced MR portography and upper gastrointestinal endoscopy. The interval between the MR scan and endoscopy was less than 3 days. Patients were excluded from this study if they had a history of upper gastrointestinal bleeding and received any treatment to esophageal varices; or if they had PV or SV emboli, fistula of the hepatic artery–PV, hepatic carcinoma, splenectomy and other diseases which might affect the hemodynamics of the portal venous system.

Between January 2010 and January 2013, 118 consecutive patients (79 men and 39 women; mean age, 50.8 years; age range, 23–71), who met the inclusion criteria and agreed to take part in the study, were recruited. The common clinical manifestations included weakness in health or body ($n = 70$), abdominal distension ($n = 52$) and dull pain in the liver ($n = 40$). According to Child–Pugh classifications, the cohort was composed of 68 patients of Child–Pugh A, 32 of Child–Pugh B and 18 of Child–Pugh C.

Endoscopic technique and interpretation

All patients underwent standard upper gastrointestinal endoscopy performed by two gastroenterologists (9th and 10th authors who had more than 10 years of experience in gastroenterology and upper gastrointestinal endoscopy) in consensus. i.v. sedation was not used in any patient. All endoscopic studies were captured as digital imaging and communications in medicine files, and were reviewed in consensus in a picture archiving and communication system by the previous two experienced gastroenterologists who were unaware of knowledge of the patients' clinical data and MR findings. According to the criteria proposed by the Japanese Research Society for Portal Hypertension (Table 1),[6] patients with the varices were divided into 4 grades based on the severity of the varices as shown on the endoscopic findings. On the basis of their probability for developing an esophageal variceal hemorrhage, the patients were also divided into two groups with low and high risk. Grade 2 and 3 varices were defined as high-risk varices, and grade 0 and 1 varices were defined as low-risk varices.[6]

MR imaging technique

All scans were conducted with a 1.5-T MR scanner (Signa Excite; GE Medical Systems, Milwaukee, WI, USA) with 38-mT/M gradients and a 120-T/M/s slew rate using a phased-array torso coil. The sequences were T_2-weighted axial fast recovery fast spin-echo (FRFSE) fat-suppressed sequence and dynamic 3-D contrast

Table 1 Grading system for esophageal varices proposed by the Japanese Research Society for Portal Hypertension

Grades	Endoscopic criteria†
0	No varices
1	Varices run straight
2	Varices show beaded appearance
3	Varices run in an oblique course and are tortuous with tumor-like appearance

†On the basis of the size and morphology of the largest varix.

enhanced imaging. The scan range was from the level of the left atrium to that of the iliac crests. Each sequence acquisition was performed within a breath-hold. Scanning parameters for the T_2-weighted axial FRFSE fat-suppressed sequence were: repetition time (TR)/echo time (TE), 3000/121.5 msec; bandwidth, 62.5 kHz; section thickness, 5.0 mm; overlap, 2.0 mm; field of view (FOV), 24 cm × 32 cm; and matrix, 256 mm × 192 mm.

Subsequently, dynamic 3-D contrast enhanced imaging was performed with a bolus injection of gadolinium chelate (Magnevist; Berlex Laboratories, Wayne, NJ, USA) via an automated pump injector (Spectris MR Injection System; Medrad, Indianola, PA, USA) into an antecubital vein according to 0.2 mmol/L per kilogram of bodyweight at the rate of 3.5 mL/s followed by a 20-mL saline solution flush. The scanning delays for triphasic MR imaging were 14 s, 1 min and 3 min after initiation of the contrast injection, representing the arterial, portal and delayed phases, respectively. Because the scanning delay for the hepatic arterial phase was set at 14 s after the bolus injection of contrast medium, the image data obtained at approximately 27 s were used to fill the central k-space lines to obtain entire image contrast of the hepatic arterial phase.

The scanning parameters for arterial and delayed phases with axial slabs were: TR/TE, 3.3–3.8/1.5–1.8 msec; bandwidth, 62.5 kHz; section thickness, 5.0 mm; overlap, 2.5 mm; FOV, 24 cm × 32 cm; and matrix, 256 mm × 192 mm. The portal phase was acquired with axial and coronal slabs, and the scanning parameters for axial slabs were similar to those used for the arterial and delayed phases except for a section thickness of 2.4 mm, and an overlap of 1.2 mm. The parameters with coronal slabs were: TR/TE, 4.3/2.0 msec; bandwidth, 62.5 kHz; section thickness, 3 mm; overlap, 1.5 mm; FOV, 36–40 cm × 36–40 cm; and matrix, 256 mm × 192 mm.

MR images interpretation

All MR image data were transferred to the workstation (AW4.4; GE Medical Systems). The T_2-weighted axial FRFSE fat-suppressed sequence, and arterial and delay enhancement images were used as supplement sequences to review the PV or SV emboli, fistula of the hepatic artery–PV, and hepatic carcinoma for determining whether the patients should be enrolled into or excluded from this study. There was no subject excluded because of suboptimal imaging or coverage. The source images of 3-D dynamic contrast-enhanced sequence were used to review maximum intensity projection (MIP) of the portal venous system. All the MR images were reviewed in consensus by two radiologists including an experienced radiologic professor (the corresponding author, who had 15 years of experience in abdominal radiology) and an experienced radiologist (the first author with 7 years of experience in radiology) with emphasis on the inflowing vessels of the varices and their originating veins. The inflowing vessel of LGV was PV or SV. Subsequently, LGV, PV and SV diameters were measured three times on portal phase imaging with axial slabs using electronic calipers on the above-mentioned workstation by the previous radiologists. The average across the three measurements was the diameter of the corresponding vessel. In the interpretation of MR imaging data of enrolled patients, the difference of the LGV and posterior gastric vein could be clarified when the posterior gastric vein was illustrated in some patients. As for the measuring point of these veins, the LGV was measured at the point which was 1 cm away from its insertion into the SV or PV; the diameter of the PV was measured at the midpoint between the SV–superior mesenteric vein (SMV) confluence and the PV bifurcation which was determined on MIP images; and the diameter of SV was measured at the point which was 1 cm away from the confluence of SMV and SV.[22] To minimize operator-dependent bias, reviewers were blinded to the patients' clinical data and endoscopic grades.

In addition, the diameters were measured repeatedly on the 1st day and the 30th day after the scan by the above-mentioned radiologists working in consensus to test the intraobserver concordance. If the concordance between the two measurements was well, the first measurements were used as the final diameter values of these veins. In instances of poor concordance, the underlying reasons were analyzed.

Statistical analysis

Statistical analysis was performed by using the Statistical Package for Social Sciences version 13.0 (SPSS, Chicago, IL, USA). A *P*-value less than 0.05 was considered statistically significantly different. All the measured results were given as the mean ± standard deviation. Precision of measurements of the LGV, PV and SV were tested by the concordance correlation coefficient (r_c). r_c of more than 0.85, 0.50–0.85 and less than 0.50 indicated very good, moderate and poor concordance, respectively. The χ^2-test was used to compare the incidence of LGV originating from SV with that from PV in patients with esophageal varices. The univariate associations of the LGV, SV and PV diameters with the presence of the

varices were assessed using χ^2-tests. Based on this analysis, potentially significant parameters were tested for possible interrelationship by multiple logistic regression analysis to identify the diameters of the LGV or its originating vein as a variable for discriminating the presence and endoscopic grades of esophageal varices. Hence, ANOVA was used to compare the diameters among different endoscopic grades of the varices. If significant difference was proved, receiver–operator curve (ROC) analysis was then carried out to determine if the cut-off values of the diameters could discriminate the endoscopic grades of esophageal varices. The diagnostic performance of the cut-off values in classifying endoscopic grades were assessed with the area under the ROC (AUC).

RESULTS

Esophageal varices

OF ALL PATIENTS, as shown on endoscopy, 56 patients had grade 0 esophageal varices, 18 patients grade 1, 30 patients grade 2 and 14 patients grade 3. In patients with esophageal varices of grades 1–3, 20 patients had the varices without other collaterals, 15 cases had the varices with gastric fundic varices, eight with gastrorenal shunt, six with splenorenal shunt, three with venae parumbilicales varices, two with paravertebral varices, and eight with two or more of the above-mentioned shunts on MR imaging. The inflowing vessel of the varices was LGV which originated from the PV in 29.03% patients (18/62) and from the SV (Fig. 1) in 70.97% (44/62).

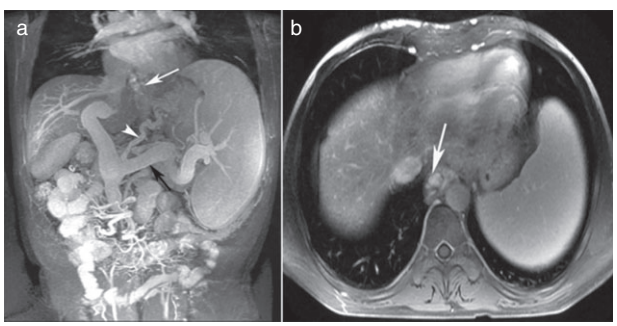

Figure 1 Left gastric vein originating from splenic vein on magnetic resonance maximum intensity projection images. In a 39-year-old male with esophageal varices secondary to post-hepatitic cirrhosis, the images show esophageal varices (a and b, white arrow), and the inflowing vessel of the varices is the left gastric vein (a, white arrowhead) originating from the splenic vein (a, black arrow).

Patients with esophageal varices of grade 0 had no collateral, and PV and SV were displayed well on MR imaging and LGV was visible in 64.29% (36/56) of patients, composed of 30.56% patients (11/36) with the originating vein of PV and 69.44% (25/36) with the originating vein of SV. In the remaining 35.71% of patients (20/56) without esophageal varices, LGV was invisible on MR imaging, and these patients were excluded from this study because the diameter of this vein could not be measured for further performance of this study. Thus, the 36 patients with esophageal varices of grade 0 in which the LGV was visible, and the 62 patients with esophageal varices of grade 1–3, totaling 98 patients, were enrolled for the study. In the enrolled patients, the χ^2-test illustrated that the SV was the predominant originating vein of the LGV ($P < 0.001$).

Intraobserver concordance of LGV, PV and SV diameter measurements

In the 98 patients included, the mean LGV, PV and SV diameters were 6.0 ± 3.2 mm (range, 2.0–17.6), 12.9 ± 2.6 mm (range, 6.2–24.2) and 9.3 ± 2.2 mm (range, 4.7–14.9), respectively, for the first measurements. For the repeated measurements, the mean LGV, PV and SV diameters were 5.9 ± 3.1 mm (range, 2.1–17.4), 12.8 ± 2.9 mm (range, 6.4–24.9) and 9.3 ± 2.1 mm (range, 4.5–15.2), respectively. The intraobserver concordance of LGV, PV and SV diameter measurements on MR portography was good because the r_c values were 0.90, 0.92 and 0.98, respectively; and the first measurements were used as the final diameter values.

Association of LGV, PV and SV diameters with presence of esophageal varices

The median value of LGV, SV and PV diameters were 6.0 mm, 9.3 mm and 12.9 mm, respectively. Univariate analysis showed the correlations of the diameters with the presence of esophageal varices (Table 2). Patients with an LGV diameter of 6.0 mm or more and an SV diameter of 9.3 mm or more were more likely to have esophageal varices than with an LGV diameter of less than 6.0 mm ($P = 0.001$) and SV diameter of less than 9.3 mm ($P = 0.002$), respectively; but PV diameter was not associated with the presence of the varices ($P = 0.417$). Before multivariate analysis, the diameters of LGV and SV were chosen as independent risk factors for the presence of the varices, which were identified by multivariate stepwise regression analysis. The diameters of LGV ($P = 0.023$, odds ratio [OR] = 1.583 and 95% confidence interval [CI] for OR of 0.748–3.351] and SV

Table 2 Univariate analysis of left gastric vein, portal vein and splenic vein diameters associated with the presence of esophageal varices ($n = 98$)

Diameters	Patients with esophageal varices ($n = 62$)	Patients without esophageal varices ($n = 36$)	*P*-value
Left gastric vein			0.001
<6.0 mm	22 (35.48)	25 (69.44)	
≥6.0 mm	40 (64.52)	11 (30.56)	
Splenic vein			0.002
<9.3 mm	20 (32.26)	23 (63.89)	
≥9.3 mm	42 (67.74)	13 (36.11)	
Portal vein			0.417
<12.9 mm	24 (38.71)	11 (30.56)	
≥12.9 mm	38 (61.29)	25 (69.44)	

Numbers in the brackets are percentages.

($P = 0.012$, OR = 2.126 and 95% CI for OR of 1.818–5.523) were associated with the varices.

Association of LGV and SV diameters with endoscopic grades of esophageal varices

The relationship of the LGV or SV diameters with endoscopic grades of esophageal varices is summarized in Table 3. LGV or SV diameters could discriminate patients between grades 0 and 1 ($P < 0.001$ or 0.007, respectively), between grades 0 and 2 (both $P < 0.001$), between grades 0 and 3 (both $P < 0.001$), between grades 1 and 3 ($P < 0.001$ or $P = 0.001$, respectively), and between grades 2 and 3 ($P = 0.002$ or 0.022, respectively). However, the diameter of LGV or SV could not differentiate grade 1 from 2 ($P = 0.182$ or 0.139, respectively). Additionally, the differences in LGV or SV diameter between patients with esophageal varices grades 0–1 and 2–3, which were defined as low-risk and high-risk varices, respectively, were of statistical significance (all $P < 0.001$).

Table 3 Diameters of left gastric vein and splenic vein stratified by endoscopic grades of esophageal varices

Grades	Diameter of left gastric vein (mm)	Diameter of splenic vein (mm)
0 ($n = 36$)	4.6 ± 1.7 (4.2–4.9)	7.3 ± 1.6 (6.9–7.6)
1 ($n = 18$)	6.3 ± 1.6 (5.5–7.1)	8.6 ± 1.7 (7.8–9.4)
2 ($n = 30$)	7.0 ± 1.4 (6.5–7.5)	9.4 ± 2.4 (8.5–10.3)
3 ($n = 14$)	8.8 ± 3.3 (6.9–10.7)	10.7 ± 1.9 (9.6–11.8)
0–1 ($n = 54$)	5.0 ± 1.6 (4.6–5.3)	7.6 ± 1.5 (7.3–7.9)
2–3 ($n = 44$)	7.6 ± 2.3 (6.9–8.2)	9.8 ± 2.3 (9.1–10.5)

Diameters of left gastric vein and splenic vein are expressed as mean ± standard deviation; and numbers in the brackets are 95% confidence intervals of the diameter.

ROC analysis for utility of diameters of LGV and SV to classify endoscopic grades of esophageal varices

By ROC analysis in all of the 98 patients enrolled, we found that the cut-off diameters of LGV of 5.1 mm, 5.9 mm, 6.6 mm, 7.1 mm, 7.8 mm and 5.8 mm, or the cut-off diameters of SV of 7.3 mm, 7.9 mm, 8.4 mm, 9.5 mm, 10.7 mm and 8.3 mm, could discriminate endoscopic grades 0 from 1, grades 0 from 2, grades 0 from 3, grades 1 from 3, grades 2 from 3, and grades 0–1 from 2–3 (Fig. 2), respectively. The sensitivity, specificity and AUC of the diameters for discrimination of the endoscopic grades in all of the 98 patients are summarized in Table 4.

DISCUSSION

APPROXIMATELY HALF OF cirrhotic patients have esophageal varices at the time of diagnosis, and incidence of varices may increase to 90% in the long-term follow up.[23] Among endoscopic grades of esophageal varices, grades 2 and 3 are of particular importance because they can cause life-threatening upper gastrointestinal hemorrhage. Therefore, it is crucial to grade the varices for prevention and treatment of the hemorrhage.[24] The LGV, which is the inflowing vein of the varices and originates from the SV or PV as shown on ultrasonography, plays an important role in the formation and development of the varices.[17,25] Recent studies showed a correlation between the variceal bleeding and hepatofugal flow in the LGV on ultrasonography, and the LGV velocity and diameter were found to correlate with the occurrence of variceal bleeding.[17,26] However, others found that dilatation of the LGV could not be present at the time of the occurrence of variceal hemorrhage.[27] These published articles suggest that there is an inconsistency regarding the association of this variceal hemorrhage with LGV velocity or diameter. In this study, we initially used MR portography to visualize the LGV and its originating vein, and to determine whether their diameters could be associated with the presence and endoscopic grades of the varices.

Our study initially suggested that the diameters of LGV and its main originating vein – the SV – measured on MR imaging could be used to identify the presence and endoscopic grades of the varices. Compared to other researches which have been performed to identify

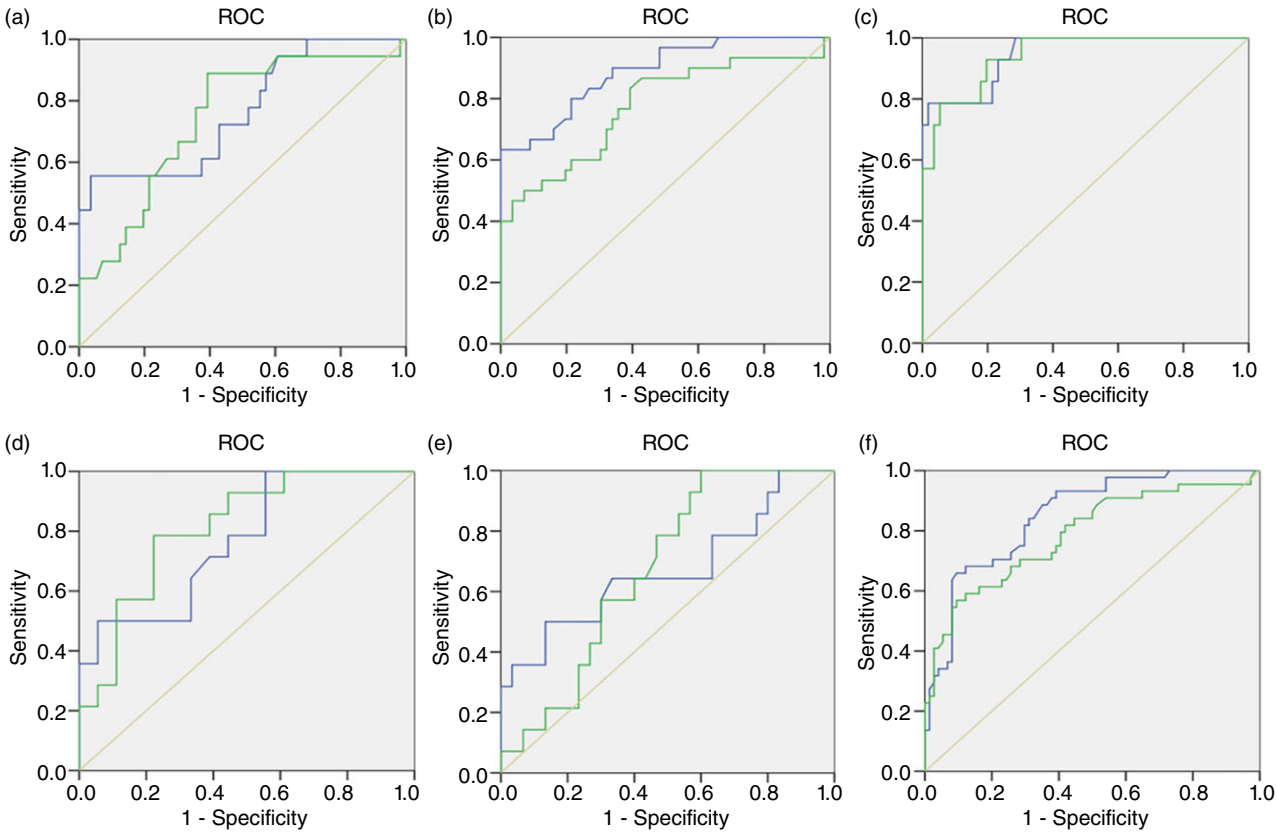

Figure 2 Receiver–operator curves (ROC) show the cut-off left gastric vein diameters of 5.1 mm, 5.9 mm, 6.6 mm, 7.1 mm, 7.8 mm and 5.8 mm, or the cut-off splenic vein diameters of 7.3 mm, 7.9 mm, 8.4 mm, 9.5 mm, 10.7 mm and 8.3 mm for discriminating endoscopic grades 0 from 1 (a), grades 0 from 2 (b), grades 0 from 3 (c), grades 1 from 3 (d), grades 2 from 3 (e), and grades 0–1 from 2–3 (f), respectively. Source of the curve: —, left gastric vein; —, splenic vein; —, reference line.

Table 4 Receiver–operator curve analysis for utility of diameters of left gastric vein or splenic vein to classify endoscopic grades of esophageal varices

Grades	Cut-off diameter (mm)	Sensitivity (%)	Specificity (%)	AUC
Left gastric vein				
Grade 0 vs 1	5.1	72.24	58.65	0.76
Grade 0 vs 2	5.9	80.02	78.59	0.89
Grade 0 vs 3	6.6	78.62	98.24	0.95
Grade 1 vs 3	7.1	78.58	55.63	0.77
Grade 2 vs 3	7.8	64.33	66.74	0.68
Grades 0–1 vs 2–3	5.8	84.09	69.81	0.85
Splenic vein				
Grade 0 vs 1	7.3	66.72	69.56	0.75
Grade 0 vs 2	7.9	83.29	60.67	0.78
Grade 0 vs 3	8.4	92.94	80.43	0.94
Grade 1 vs 3	9.5	78.61	77.85	0.81
Grade 2 vs 3	10.7	57.17	70.08	0.67
Grades 0–1 vs 2–3	8.3	70.49	71.66	0.79

AUC, area under the receiver–operator curve.

predictive non-invasive factors for the varices such as platelet count of 82 000/uL or less, PV diameter of 11.5 mm or more, and anteroposterior splenic measurement of 103 mm or more,[8–11] we used MR portography to display the varices, the inflowing vein of the varices and its originating vein, which was visualized and effective to investigate the previous associations.

As shown in our study, esophageal varices could be found in most of the cirrhotic patients, the LGV could be the inflowing vein of the varices, and the diameter of the LGV and of the predominant originating veins (SV) of this inflowing vein would increase with the progress of the varices from grade 0 to 3. The possible mechanism of these findings may be explained as follows. Because of portal outflow obstruction (elevated intrahepatic portal vascular resistance) in cirrhotic patients, increased blood flow in the PV and SV cannot enter the liver via the PV, and a considerable percentage of the PV and SV flow is forced to bypass the liver.[1,28,29] One of the most important shunting routes is LGV originating from PV or SV, and our findings suggested that SV was the predominant originating vein. When the blood flow in the SV or PV prominently increases, the diversion of a large quantity of portal flow via the LGV would result in LGV dilation and the occurrence of esophageal varices together with the dilation of the predominant originating vein (the SV).[17,25] In this study, we found that the LGV diameter increased with the increasing endoscopic grades of the varices, which suggested that the diameter of LGV or SV could have a potential association with the endoscopic grades of the varices. We confirmed that the diameters of the LGV or SV could be independent risk factors for the presence of esophageal varices, and be used to discriminate the grades of the varices.

Based on the present data with the ROC analysis, the LGV and SV diameter measurements could be used as referential criteria to classify the endoscopic grades of esophageal varices except for discriminating grade 1 from 2. This indiscrimination between grade 1 and 2 may be because the endoscopic grading system for the varices used in our study is on the basis of the size and morphology of the largest varix, and the difference in the endoscopic grades between grade 1 and 2 is not so obvious. Patients between grades 0–1 and 2–3, which were defined as low-risk and high-risk varices, respectively, could be discriminated by the LGV and SV diameter measurements. According to the AUC which was used to assess the diagnostic performance of the cut-off diameters in classifying the endoscopic grades of esophageal varices, the cut-off diameter of the LGV was found to be better than that of the SV in classifying grades 0 from 1, grades 0 from 2, and grades 0–1 from 2–3. The potential explanation may be because the SV is not only the predominant originating vein of the LGV but also the originating vein of other shunts such as splenorenal shunt and gastric fundic varices, which may have an affect on the hemodynamics and diameter of the SV.[1,23] On the other hand, the cut-off diameter of the SV was found to have similar diagnostic performance to that of the LGV in classifying grades 0 from 3, and grades 2 from 3; and the cut-off diameter of the SV was better in classifying grades 1 from 3. Therefore, recognition of the dilated LGV and SV may be an additional secondary sign of esophageal varices, and the diameter measurements are crucial to classify endoscopic grades of the varices for guiding the therapy to prevent the potential hemorrhage.[24]

However, there was a limitation in this study. The enrolled patients in this study had post-hepatitic cirrhosis secondary to chronic hepatitis B, but our findings are specific to liver cirrhosis in patients with hepatitis B.

In conclusion, we used a portography with MR imaging to visualize the inflowing vessel and its originating vein of esophageal varices secondary to liver cirrhosis in patients with hepatitis B. On MR portography, the diameter of the LGV or SV could be associated with the presence and endoscopic grades of the varices, and could be used to discriminate the high-risk varices from the low-risk ones. We hope that these findings could be helpful in better understanding the occurrence and severity of esophageal varices for appropriate treatment to prevent variceal bleeding.

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