Platelet count to splenic diameter ratio and splenoportal index as non-invasive screening tools in predicting esophageal varices in patients with liver cirrhosis

Rajesh Kumar Wadhwa, Zaigham Abbas, Syed Mujahid Hasan, Nasir Hasan Luck, Mahira Younus[1], Sabiha Anis[2], Muhammed Mubarak[3]

Departments of Hepatogastroentrology, [1]Radiology, [2]Immunology and [3]Pathology, Sindh Institute of Urology and Transplantation (SIUT), Karachi, Pakistan

ABSTRACT

Background: Esophageal varices (EVs) are serious consequences of liver cirrhosis. Several studies have evaluated the possible non-invasive markers for the diagnosis of EVs to reduce the number of endoscopic procedures in patients with cirrhosis but without varices. This study was performed to evaluate the diagnostic performance of two such parameters (platelet count to splenic diameter ratio and splenoportal index) for the detection of EVs. **Materials and Methods:** A total of 111 patients with liver cirrhosis were analyzed after performing upper gastrointestinal endoscopy and non-invasive tests including platelet count and ultrasound abdomen including Doppler study. Appropriate statistical tests were applied to compare the non-invasive tests with the gold standard of endoscopy. **Results:** Of 111 liver cirrhotics, 80 (72.1%) were male and 31 (27.9%) were female. EVs were present in 68 (61.3%) patients and absent in 43 (38.7%) patients. In platelet count to splenic diameter ratio, a cut-off value of 1014 was obtained, which gave a sensitivity of 75.0%, specificity of 65.1%, positive predictive value (PPV) of 77.3%, negative predictive value (NPV) of 62.2% and diagnostic accuracy of 71.2%. In the splenoportal index, a cut-off value of 3.5 cm/s was obtained, which gave a sensitivity of 79.4%, specificity of 72.0%, PPV of 81.8%, NPV of 68.8% and diagnostic accuracy of 76.5% for the diagnosis of EVs. **Conclusions:** The platelet count to spleen diameter ratio and splenoportal index are non-invasive and fairly accurate alternatives in identifying the presence or absence of EVs in patients with compensated cirrhosis.

Key words: Cirrhosis, esophageal varices, portal hypertension, splenomegaly, thrombocytopenia

Address for Correspondence:
Dr. Rajesh Kumar Wadhwa,
Department of Hepatogastroenterology,
Sindh Institute of Urology and Transplantation (SIUT),
Karachi, Pakistan.
E-mail: drwadhva_79@live.com

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INTRODUCTION

Esophageal varices (EVs) are telltale signs of portal hypertension (PHTN) from cirrhosis. Once liver cirrhosis is established, EVs are found in approximately 40% of patients with compensated and 60% of those with decompensated cirrhosis.[1,2] The yearly incidence of gastrointestinal bleeding is 5% in those with small EVs and 15-20% in patients with large EVs.[2] The risk of initial bleeding from varices is 25-35% in 2 years. The rate of variceal bleeding is even higher in those in whom the first bleeding episode occurs within 1 year of detection of varices.[3,4] The mortality rate due to EV bleeding varies from 17% to 57%. The mortality rate from first-time bleeding is 40-60%.[3] The grade of EVs often correlates with the severity of liver disease. While approximately 85% of individuals with Child-Turcotte Pugh (CTP) C cirrhosis have varices, they are present in only 45% in CTP A cirrhosis.[5]

Screening endoscopy in CTP A is recommended only in the presence of thrombocytopenia of <140,000/uL, portal vein diameter of over 13 mm or portosystemic collaterals on ultrasound examination,[1,6] while screening endoscopy is advised in all patients of CTP B and C cirrhosis.

Upper gastrointestinal (GI) endoscopy is the gold standard examination to establish the diagnosis and grade the EVs.[7,8] However, the endoscopy is an invasive procedure and its cost-effectiveness for screening is also questionable.[9,10]

Moreover, not all healthcare centers, especially in rural areas, have such a facility. In addition, the competency of healthcare providers in those places to perform endoscopy is limited. These limitations and the ever-increasing workload on endoscopy units have led many researchers to identify some parameters that can non-invasively predict the presence of EVs.[11-14]

Previously, single non-invasive comparative parameters were studied, e.g., splenomegaly,[15-17] ascites,[18,19] spider naevi,[12] Child grade,[20] platelet count,[11,18,21,22] portal vein diameter, prothrombin time[1], platelet count to splenic diameter ratio (PCSDR),[3,23-28] serum albumin[21] and serum bilirubin[21] as significant predictors for the presence of EVs.

A few studies in Pakistan have also been performed.[19-24] The parameters studied included serum albumin, spleen size, portal vein diameter, PCSDR, portal vein velocity and splenoportal index (SPI).[25]

The PCSDR is a non-invasive parameter that proved to be a simple, reproducible and cost-effective tool while use of SPI for predicting the presence of EVs has two advantages. First, SPI can concomitantly be measured during routine biannual ultrasound (US) screening for hepatocellular carcinoma (HCC) in patients with cirrhosis; hence, it does not add additional cost for the detection of EVs. Secondly, the measurement of splenic index (SI) and mean portal vein velocity (PVV) can be easily performed at the outpatient clinic in patients with varying severity of cirrhosis and etiology of liver disease.[29-36]

The aim of the present study was to assess the sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV) and the diagnostic accuracy of PCSDR and SPI as non-invasive markers in predicting EVs in our setup, taking upper GI endoscopy as the gold standard.

MATERIALS AND METHODS

This was a prospective study conducted at the Department of Hepatogastroenterology, Sindh Institute of Urology and Transplantation (SIUT), Karachi, Pakistan. The study was conducted from November 2010 to April 2011. A total of 111 patients were enrolled in the study. Inclusion criteria included liver cirrhotic patients of 18 years and above of either gender, irrespective of duration and severity of disease, who presented in the GI outpatient department (OPD) for routine clinical visit. The exclusion criteria were: Patients unwilling or unable to undergo invasive endoscopic procedure; pregnancy, acute or severe cardiac or pulmonary disease and marked bleeding dyscrasias in which endoscopy is contraindicated; active GI bleed at admission; those previously undergoing sclerosis or band ligation of EVs; those on primary prophylaxis of variceal bleeding like use of β-blockers and portal vein thrombosis.

All the patients meeting the inclusion criteria were admitted in the ward. A structured proforma was used to collect the data. A written informed consent was obtained from each selected patient. A blood sample for platelet count was obtained. All the patients underwent abdominal US in the morning after an overnight fast of 8 h to see the cirrhotic changes, spleen size and portal vein velocity. It was carried out by a consultant radiologist (with more than 5 years experience) using the US TOSHIBA-apleo 50 Model MCM17545TS. On the same day, all patients underwent upper GI endoscopy at the SIUT endoscopy unit to document the presence or absence of EVs using a video endoscope Olympus GIF-XP180. These endoscopic findings were also documented in the proforma.

PCSDR was defined as the ratio of platelet numbers/mm^3 divided by maximum splenic bipolar diameter as measured in millimeters by the US. The ratio 1014 was considered the cut-off value. PCSDR ≤1014 denoted the presence of varices and >1014 the absence of varices.[3]

SPI was defined as a ratio of SI to mean PVV, calculated by the formula, SPI = SI / PVV mean, whereby SI is the sonographic calculation of splenic size in square centimeters based on the maximum transverse and longitudinal measurements and PVV mean is the velocity of portal blood flow in cm/s calculated automatically by the machine with time-arranged velocity in two to three cardiac cycles. SPI of 3.5 cm/s was considered the cut-off value. SPI <3.5 cm/s denoted the absence of varices and SPI ≥3.5 cm/s the presence of varices.[29]

Data analysis

The data were analyzed using Statistical Package for Social Sciences (SPSS) for Windows version 17.0 (SPSS, Chicago, IL, USA). Measures of central tendency (including mean with SD) were calculated for quantitative variables such as age. Numbers and percentages were used for qualitative variables such as gender and frequency of EVs. Sensitivity, specificity, PPV and NPV of each of the indices (SPI and PCSDR) were calculated taking upper GI endoscopy as the gold standard test. Sensitivity, specificity, PPV and NPV were also computed after stratification of age, gender, duration of disease and disease severity.

RESULTS

The main demographic, clinical and laboratory features of all patients are shown in Table 1. A total of 111 liver cirrhotic patients of 18 years and above of either gender were included in the study. The average age of the patients was 43.11 ± 13.58 years (95% CI: 40.55-45.66 years; range: 18-85 years). The average body mass index (BMI) was 21.59 ± 3.85 (95% CI: 20.87-22.32), ranging from 13.5 to 37.0. Of the 111 patients, there were 80 (72.1%) male patients and 31 (27.9%) female patients, with a male to female ratio of 2.6:1. On stratification of age, it was observed that 58 (52.3%) patients were ≥40 years of age and 53 (47.7%) patients were <40 years of age, while 57 (51.4%) patients had a history of duration of cirrhosis <1 year and 54 (48.6%) patients had a history of duration of cirrhosis >1 year.

The severity of liver disease according to the CTP score was observed as follows: CTP-A-46, 41.4%; CTP-B - 51, 45.9%; and CTP-C-14, 12.6%.

Of the 111 patients, EVs were present in 68 (61.3%) patients and absent in 43 (38.7%) patients. Among the 68 positive cases, 25 (22.5%) patients had grade I EV, 27 (24.3%) patients had grade II EV, 13 (11.7%) patients had grade III EV and three (2.7%) patients had grade IV EV.

The diagnostic performance of PCSDR and SPI (cm/s) to predict EV as defined by the upper GI endoscopy (gold standard) is provided in Tables 2 and 3 along with the stratification according to age, gender, duration of disease and severity.

Table 1: Patient characteristics

Total number of patients (%)	111(%)
Males, *n*	80 (72.1)
Females, *n*	31 (27.9)
M:F ratio	2.6:1
Age, mean ± SD (years)	43.11 ± 13.58 (95% CI: 40.55-45.66)
Body mass index, mean ± SD	21.59 ± 3.85 (95% CI: 20.87-22.32)

Table 2: Diagnostic performance of platelet count to splenic diameter ratio, both overall and according to stratification of age, gender, disease severity and duration

Variables	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	Accuracy (%)
Overall	79.40	72.00	81.80	68.80	76.50
Age					
≥40 years	85.20	52.10	72.50	70.50	71.90
<40 years	78.10	86.30	89.20	73.00	81
Gender					
Male	83.30	68.70	80.00	73.30	77.50
Female	77.70	69.20	77.70	69.20	74.10
Duration					
≤1 year	83.30	65.30	73.50	77.20	75
>1 year	80.50	73.60	85.50	66.60	78.10
Disease severity					
CTP A	73.90	91.30	89.40	77.70	82.60
CTP B	87.50	47.30	73.60	69.20	72.50
CTP C	81.80	33.30	81.80	33.30	71.40

Table 3: Diagnostic performance of splenoportal index, both overall and according to stratification of age, gender, disease severity and duration

Variables	Sensitivity (%)	specificity (%)	PPV (%)	NPV (%)	Accuracy (%)
Overall	75.00	65.10	77.30	62.20	71.20
Age					
≥40 years	78.00	52.90	80.00	55.00	70.70
<40 years	70.40	73.10	73.10	70.40	71.70
Gender					
Male	72.00	53.30	72.00	53.30	65.00
Female	83.30	92.30	93.80	80.00	87.10
Duration					
≤1 year	58.80	73.90	76.90	54.80	64.90
>1 years	91.20	55.00	77.50	78.60	77.80
Disease severity					
CTP A	63.20	63.00	54.50	70.80	63.00
CTP B	73.70	76.90	90.30	50.00	74.50
CTP C	100.00	33.30	84.60	100.00	85.70

DISCUSSION

A number of previous studies have recommended that patients with liver cirrhosis should be screened for EVs. Endoscopy is recommended every 2-3 years in patients without varices and every 1-2 years in patients with small varices.[3] However, this recommendation imposes a major burden on endoscopy units and significant cost on patients. In an attempt to reduce the endoscopy burden, several studies have been performed to identify the non-invasive parameters that can predict the presence of EVs in liver cirrhosis.[3,37-40] Numerous studies have revealed the correlation of PCSDR with the presence of EVs in liver cirrhotic patients.[41] Thrombocytopenia is a prevalent complication of cirrhosis of the liver that has been seen in up to 76% patients, while moderate thrombocytopenia (platelet count, 50,000 uL and 75,000 uL) occurs in approximately 13% of patients with cirrhosis. The occurrence of thrombocytopenia in those patients can be considered as an event with multiple etiologies. Two mechanisms may act alone or synergistically with splenic sequestration. One is central, which involves either myelosuppression because of hepatitis viruses or the toxic effects of alcohol abuse on the bone marrow. The second one involves the presence of antibodies against platelets. It also depends on the stage and etiology of liver disease.[42] Splenomegaly is common particularly in patients with cirrhosis from non-alcoholic etiologies.[43]

It is considered to be caused mainly by congestion of the red pulp as the result of PHTN. However, splenic size does not correlate well with portal pressures, suggesting that other factors may be contributing.[43] The above findings in patients with liver cirrhosis have been analyzed in different combinations to predict the presence of EVs.

Recent studies performed locally and in the Western world show a higher sensitivity and specificity for PCSDR with a cut-off value of 909.[3] In our study, with a pre-determined cut-off value of 1014, the sensitivity, specificity and diagnostic accuracy were 75.0%, 65.1% and 71.2%, respectively. This may be due to the fact that the majority of patients in our study were of CTP class A and B cirrhosis. The trend also shows that these parameters are more helpful in diagnosing the presence and absence of varices in more advanced chronic liver disease (CLD), which is depicted by increasing sensitivity, specificity and NPV with increasing CTP class, sensitivity approaching 100% and NPV also approaching 100% in CTP C patients. Further analysis of the data also testifies the same effect of increasing sensitivity and accuracy with prolonged duration of disease. However, there were no differences in the sensitivity and specificity among the two age groups as well as gender.

In our study, we also prospectively evaluated the clinical value of another non-invasive parameter called SPI by using duplex Doppler US in patients with compensated cirrhosis. The utility of this non-invasive SPI in predicting the presence and absence of EVs could be reasoned by the following pathophysiologic changes. When portal resistance increases in cirrhosis, stagnant portal blood flow causes increased resistance of splenic venous outflow, which leads to congestive splenomegaly. In addition, the increase of splanchnic inflow also causes splenomegaly, which worsens PHTN. The increase in portal pressure not only predisposes to formation of EVs but also aggravates splenomegaly.[43] Previous studies also revealed that the decrease in mean PVV correlated with the severity of PHTN and the risk of EV bleeding.[43] When SPI was set at ≥3.5 cm/s, it showed a high sensitivity of 79.4%, specificity of 72%, PPV of 81.8% and NPV of 68.8% in the study population as well as a high diagnostic accuracy of 76.5%. On further analysis of SPI, it is seen that sensitivity, specificity, PPV, NPV and diagnostic accuracy are greater in compensated cirrhosis like CTP-A and B than in decompensated liver cirrhosis of CTP-C. The possible reason could be that the majority of patients in this study were of compensated cirrhosis. Its sensitivity, specificity and diagnostic accuracy are almost equal when compared with respect to age, gender and duration of disease. By using the cut-off level of ≥3.5 cm/s, majority of our patients showed correct diagnoses with this non-invasive index, comparing favorably with screening endoscopy.

When comparing the two non-invasive parameters, i.e., PCSDR and SPI, for diagnosis of the presence or absence of EVs, both had significant sensitivity, specificity and diagnostic accuracy in our population. But, when compared with the western world, the sensitivity and specificity of SPI and PCSDR are higher than in our population.[3,29] The best explanation of this could be the etiology of liver cirrhosis, as in our study the majority of patients had virus-related cirrhosis while the major cause of cirrhosis in the western world is non-viral related, e.g., alcoholic cirrhosis, metabolic cirrhosis, primary biliary cirrhosis and primary sclerosing cholangitis. We have found that PCSDR is a non-invasive parameter that has proved to be a simple, reproducible and cost-effective tool while the SPI can be measured in the outpatient clinic in patients with varying severity of cirrhosis and etiology of liver disease during routine biannual US screening for HCC.

Therefore, we believe that both these parameters can be used as simple non-invasive screening tools for the diagnosis of the presence or absence of EVs. The tests would be helpful for those centers especially in the rural areas of our country, where endoscopy facilities are not available or there is limited competency of healthcare providers. By using the above non-invasive parameters, they can refer their patients to endoscopy facility centers.

CONCLUSION

In this study, we have demonstrated that both the PCSDR and the SPI have a fairly high sensitivity, specificity and accuracy for diagnosing EVs in liver cirrhosis. Both parameters are simple, reproducible, cost-effective, non-invasive and fairly accurate in identifying the presence or absence of EVs in patients with compensated cirrhosis. Applying these indices can reduce the need for screening endoscopy.

REFERENCES

1. Schepis F, Camma C, Niceforo D, Magnano A, Pallio S, Cinquegrani M, *et al*. Which patients with cirrhosis should undergo endoscopic screening for esophageal varices detection? Hepatology 2001;33:333-8.
2. D'Amico G, Luca A. Natural history. Clinical-hemodynamic correlations. Prediction of the risk of bleeding. Bailliere's Clin Gastroenterol 1997;11:243-56.
3. Giannini E, Botta F, Borro P, Risso D, Romagnoli P, Fasoli A, *et al*. Platelet count/spleen diameter ratio: Proposal and validation of non-invasive parameter to predict the presence of oesophageal varices in patients with liver cirrhosis. Gut 2003;52:1200-5.
4. The Northern Italian Endoscopic Club for the Study and Treatment of Esophageal Varices. Prediction of the first variceal hemorrhage in patients with cirrhosis of the liver and esophageal varices: A prospective multicenter study. N Engl J Med 1988;319:983-9.
5. Chalasani N, Imperiale TF, Ismail A, Sood G, Carey M, Wilcox CM, *et al*. Predictors of large esophageal varices in patients with cirrhosis. Am J Gastroenterol 1999;94:3285-91.
6. Grace ND, Groszman RJ, Tsao GG, Burroughs AK, Pagliaro L, Makuch RW, *et al*. Portal hypertension and variceal bleeding: An AASLD single topic symposium. Hepatology 1998;28:868-80.

7. de Franchis R. Evolving consensus in portal hypertension report of the Baveno IV consensus workshop on methodology of diagnosis and therapy in portal hypertension. J Hepatol 2005;43:167-76.
8. Spiegel BM, Targownik L, Dulai GS, Karsan HA, Gralnek IM. Endoscopic screening for esophageal varices in cirrhosis. Is it ever cost effective? Hepatology 2003;37:366-77.
9. Arguedas MR, Heudebert GR, Eloubeidi MA, Abrams GA, Fallon MB. Cost-effectiveness of screening, surveillance and primary prophylaxis strategies for esophageal varices. Am J Gastroenterol 2002;97:2441-52.
10. Amico GD, Morabito A. Noninvasive markers of esophageal varices: Another round, not the last. Hepatology 2004;39:30-4.
11. Pilette C, Oberti F, Aube C, Rousselet MC, Bedossa P, Gallois Y, *et al*. Non-invasive diagnosis of esophageal varices in chronic liver diseases. J Hepatol 1999;31:867-73.
12. Madhotra R, Mulcahy HE, Willner I, Reuben A. Prediction of esophageal varices in patients with cirrhosis. J Clin Gastroenterol 2002;34:81-5.
13. Thomopoulos KC, Labropoulou-Karatza C, Mimidis KP, Katsakoulis EC, Iconomou G, Nikolopoulou VN. Non-invasive predictors of the presence of large esophageal varices in patients with cirrhosis. Dig Liver Dis 2003;35:473-8.
14. Amarapurkar DN, Parikh SS, Shankaran K, Chopra K, Dhawan P, Kalro RH, *et al*. Correlation between splenomegaly and oesophageal varices in patients with liver cirrhosis. Endoscopy 1994;26:563.
15. Chalasani N, Imperiale TF, Ismail A, Sood G, Carey M, Wilcox CM, *et al*. Predictors of large esophageal varices in patients with cirrhosis. Am J Gastroenterol 1999;94:3285-91.
16. Zaman A, Becker T, Lapidus J, Benner K. Risk factors for the presence of varices in cirrhotic patients without a history of variceal hemorrhage. Arch Intern Med 2001;161:2564-7.
17. Sharma SK, Aggarwal R. Prediction of large esophageal varices in patients with cirrhosis of the liver using clinical, laboratory and imaging parameters. J Gastroenterol Hepatol 2007;22:1909-15.
18. Giannini EG, Zaman A, Kreil A, Floreani A, Dulbecco P, Testa E, *et al*. Platelet count/spleen diameter ratio for the noninvasive diagnosis of esophageal varices: Results of a multicenter, prospective, validation study. Am J Gastroenterol 2006;101:2511-9.
19. Khan H, Iman NU. Hypoalbuminemia: A marker of oesophageal varices in chronic liver disease due to hepatitis B and C. Rawal Med J 2009;34:98-101.
20. Javed IF, Hameed A, Qazi I, Farooq A, Masoodur R. Predictors of esophageal varices in patients of liver cirrhosis. J Postgrad Med Inst 2007;21:60-4.
21. Gill ML, Atiq M, Sattar S, Khokhar N. Non-endoscopic parameters for the identification of Esophageal Varices in patients with chronic hepatitis. J Pak Med Assoc 2004;54:575-7.
22. Muhammad K, Naveed YK, Muhammad A, Mian MA, Hamamatul BK, Zubai H, *et al*. Association of platelet count to splenic index ratio with presence of esophageal varices in patients with hepatitis C virus related compensated cirrhosis. Pak J Gastroenterol 2006;20:37-42.
23. Shahid S, Altaf A, Anwaar AK, Arshad KB, Farzana S, Waqar Hassan S, *et al*. Platelet count / spleen diameter ratio: Can it predict the presence of varices in patients with cirrhosis of liver? Proc Shaikh Zayed Postgrad Med Inst 2004;18:21-6.
24. Sethar GH1, Ahmed R, Rathi SK, Shaikh NA. Platelet count/splenic size ratio. A parameter to predict the presence of esophageal varices in cirrhotics. J Coll Physicians Surg Pak 2006;16:183-6.
25. Liu CH, Hsu SJ, Liang CC, Tsai FC, Lin JW, Liu CJ, *et al*. Esophageal varices: noninvasive diagnosis with duplex US in patients with compensated cirrhosis. Radiology 2008;248:132-9.
26. Zimbwa TA, Blanshard C, Subramaniam A. Platelet count/spleen diameter ratio as a predictor of oesophageal varices in alcoholic cirrhosis. Gut 2004;53:1055.
27. Baig WW, Nagaraja MV, Varma M, Prabhu R. Platelet count to spleen diameter ratio for the diagnosis of esophageal varices: Is it feasible? Can J Gastroenterol 2008;22:825-8.
28. Agha A, Anwar A, Bashir K, Savarino E, Giannini EG. External validation of the platelet count/spleen diameter ratio for the diagnosis of esophageal varices in hepatitis C virus-related cirrhosis. Dig Dis Sci 2009;54:654-60.
29. Sarangapani A, Shanmugam C, Kayanasundaram M, Rangachari B, Thangavelu P, Subbarayan JK. Noninvasive prediction of large esophageal varices in chronic liver disease patients. Saudi J Gastroenterol 2010;16:38-42.
30. Zaman A, Hapke R, Flora K, Rosen HR, Benner K. Factors predicting the presence of esophageal or gastric varices in patients with advanced liver disease. Am J Gastroenterol 1999;94:3292-6.
31. Finn JP, Kane RA, Edelman RR, Jenkins RL, Lewis WD, Muller M, *et al*. Imaging of the portal venous system in patients with cirrhosis: MR angiography vs duplex Doppler sonography. AJR Am J Roentgenol 1993;161:989-94.
32. Gines P, Quintero E, Arroyo V, Terés J, Bruguera M, Rimola A, *et al*. Compensated cirrhosis: Natural history and prognostic factors. Hepatology 1987;7:122-8.
33. D'Amico G, Morabito A, Pagliaro L, Marubini E. Survival and prognostic indicators in compensated and decompensated cirrhosis. Dig Dis Sci 1986;31:468-75.
34. Grace ND. Prevention of initial variceal hemorrhage. Gastroenterol Clin North Am 1992;21:149-61.
35. Smith JL, Graham DY. Variceal hemorrhage: A critical evaluation of survival analysis. Gastroenterology 1982;82:968-73.
36. Graham DY, Smith JL. The course of patients after variceal hemorrhage. Gastroenterology 1981;80:800-9.
37. Ripoll C, Groszmann R, Garcia-Tsao G, Grace N, Burroughs A, Planas R, *et al*. Hepatic venous pressure gradient predicts clinical decompensation in patients with compensated cirrhosis. Gastroenterology 2007;133:481-8.
38. Garcia-Tsao G, Bosch J, Groszmann RJ. Portal hypertension and variceal bleeding — unresolved issues. Summary of an American Association for the Study of Liver Diseases and European Association for the Study of the Liver single-topic conference. Hepatology 2008;47:1764-72.
39. Sarin SK, Selhi KK, Nanda R. Measurement and condition of wedged hepatic, intrahepatic, intrasplenic and intravariceal pressures in patients with cirrhosis of the liver and non-cirrhotic portal fibrosis. Gut 1987;28:260-6.
40. de Franchis R. Evolving consensus in portal hypertension. Report of the Baveno IV consensus workshop on methodology of diagnosis and therapy in portal hypertension. J Hepatol 2005;43:167-76.
41. Madhotra R, Mulcahy HE, Willner I, Reuben A. Prediction of esophageal varices in patients with cirrhosis. J Clin Gastroenterol 2002;34:81-5.
42. Watanabe S, Hosomi N, Kitade Y, Kurokohchi K, Arima K, Kawabata H, *et al*. Assessment of the presence and severity of esophagogastric varices by splenic index in patients with liver cirrhosis. J Comput Assist Tomogr 2000;24:788-94.
43. Iwao T, Toyonaga A, Oho K, Tayama C, Masumoto H, Sakai T, *et al*. Value of Doppler ultrasound parameters of portal vein and hepatic artery in the diagnosis of cirrhosis and portal hypertension. Am J Gastroenterol.1997;92:1012-17.

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