Journal of Carcinogenesis
Covering Genesis to Therapeutics

Journal of Carcinogenesis, ISSN: 1477-3163
2025; Vol 24: Issue 8s, https://carcinogenesis.com/

Role Of Portal Vein Color Doppler And Splenic Size To Evaluate Portal Hypertension And Its Correlation With Eosophageal Varices

Surya S[1], Sarvesh R[2], Vasantha kumar M[3]

[1]Postgraduate student, Department of Radiodiagnosis, ACS Medical College and Hospital, Chennai, Tamil Nadu, India

[2]Assistant Professor, Department of Radiodiagnosis, ACS Medical College and Hospital, Chennai, Tamil Nadu, India

[3]Associate professor, Department of radiology, ACS medical College and Hospital, Chennai, Tamilnadu, India

Corresponding Author:

Dr. M Vasantha Kumar

Associate Professor Department of Radiodiagnosis ACS Medical College and Hospital, Chennai-600077, Tamilnadu, India

Email ID mvkvasanth@gmail.com

ABSTRACT

Background:Portal hypertension is a major complication of liver cirrhosis, leading to life-threatening conditions such as gastro-esophageal varices and variceal bleeding. Endoscopy remains the gold standard for variceal detection, but its invasiveness limits routine use. Non-invasive techniques such as Color Doppler ultrasonography offer a safer alternative for assessing portal hemodynamics and splenic size in predicting portal hypertension severity.

Aim:To evaluate the role of portal vein Color Doppler parameters and splenic size in assessing portal hypertension and their correlation with the presence of gastro-esophageal varices in cirrhotic patients.

Materials and Methods:A hospital-based cross-sectional study was conducted among 50 cirrhotic patients at Aarupadai Veedu Medical College and Hospital, Pondicherry, from October 2017 to September 2019. Portal vein diameter, mean flow velocity, splenic size, and related Doppler indices were recorded using Color Doppler ultrasonography. Findings were correlated with upper gastrointestinal endoscopy results for the presence and grade of varices. Statistical analysis was performed using SPSS version 25, with $p < 0.05$ considered significant.

Results:Gastro-esophageal varices were detected in 62% of patients. A significant association ($p < 0.001$) was observed between varices and increased portal vein diameter (>12.25 mm), reduced portal vein velocity, and splenic enlargement (>13.9 cm). Logistic regression confirmed these as significant predictors of portal hypertension.

Conclusion: Portal vein Doppler indices and splenic size serve as reliable, non-invasive parameters for predicting gastro-esophageal varices in cirrhotic patients. These ultrasonographic markers can aid early risk stratification and reduce dependence on invasive procedures..

Keywords: *Portal hypertension, Color Doppler, Splenic size, Gastro-esophageal varices, Cirrhosis, Ultrasonography.*

How to Cite: Surya S, Sarvesh R, (2025) Role Of Portal Vein Color Doppler And Splenic Size To Evaluate Portal Hypertension And Its Correlation With Eosophageal Varices, *Journal of Carcinogenesis*, *Vol.24, No.8s*, 937-944

1. INTRODUCTION

Portal hemodynamics represent the key pathophysiological mechanism in liver cirrhosis and are closely associated with the severity of the disease (1). Cirrhosis, which can develop from a wide range of etiologies, represents the final stage of chronic liver disease (2). It is characterized by diffuse hepatic fibrosis and the formation of regenerative nodules, which are structurally abnormal areas resulting from the distortion and remodeling of normal liver architecture (1). Among the various causes of cirrhosis, the most common and life-threatening complication is portal hypertension, which remains the principal cause of mortality in patients with chronic liver disease. The diagnosis of cirrhosis is generally straightforward during the decompensated stage but remains difficult in the compensated phase. The progression of hepatic fibrosis parallels a gradual rise in portal pressure, reflected by a hepatic venous pressure gradient (HVPG) exceeding 5 mm Hg, a finding often seen in individuals with severe fibrosis even before cirrhosis becomes evident (3). Because fibrosis is heterogeneous, the distinction between severe fibrosis and compensated cirrhosis is often blurred, and chronic liver disease

represents a continuous pathological spectrum (4). The Baveno VI consensus has therefore proposed the term compensated advanced chronic liver disease (cACLD) to describe the stage encompassing severe fibrosis and early cirrhosis (5). With the growing understanding of disease progression, the concept of cirrhosis has shifted from the histological identification of F4 fibrosis to recognizing patients at risk of complications, particularly those with clinically significant portal hypertension (HVPG ≥ 10 mm Hg). Portal hypertension is the main cause of mortality in chronic liver disease and is defined as a wedge hepatic vein or direct portal vein pressure more than 5 mm Hg above inferior vena cava pressure, a splenic vein pressure exceeding 15 mm Hg, or a surgically measured portal pressure greater than 30 cm H_2O (22 mm Hg) (6). Increased intrahepatic resistance—primarily within the hepatic sinusoids—alters hepatic architecture and leads to portal vein dilatation, splenomegaly, and formation of esophageal and gastric varices, accompanied by complications such as variceal hemorrhage, ascites, encephalopathy, and hypersplenism (7). The development of portosystemic collaterals serves to bypass the increased portal resistance, returning blood to the systemic circulation. Among these complications, gastrointestinal bleeding from ruptured varices is the most severe and carries high mortality, making variceal bleeding one of the most dreaded outcomes of cirrhosis. The prevalence of varices in cirrhotic patients is about 50–60%, with a bleeding risk of 5–15% and a mortality rate of at least 20%. Esophageal varices are key prognostic indicators influencing morbidity and surgical risk. Although endoscopy remains the gold standard for detecting varices, it is invasive, expensive, and carries a potential risk of bleeding. Non-invasive assessment using Color Doppler ultrasonography of the portal venous system and spleen can help predict the presence and severity of gastro-esophageal varices and serve as a tool for long-term monitoring in portal hypertension (8). This study aims to evaluate the correlation between portal vein Color Doppler parameters, splenic size, and the presence of gastro-esophageal varices in patients with cirrhosis.

2. MATERIALS AND METHOD:

This hospital-based cross-sectional study was conducted among inpatients of the medical wards at Aarupadai Veedu Medical College and Hospital (AVMC&H), Pondicherry, over a period of two years from October 2017 to September 2019. A total of 50 patients were included in the study based on predefined inclusion and exclusion criteria. Patients above 18 years of age, of both sexes, diagnosed with liver cirrhosis or portal hypertension with esophageal varices, and those with abnormal liver function tests suggestive of chronic liver disease were included. Patients with recent gastrointestinal bleeding, those who had undergone variceal ligation, sclerotherapy, or Transjugular Intrahepatic Portosystemic Shunt (TIPSS) procedures, terminally ill patients, and those unwilling to participate were excluded. All participants underwent Color Doppler ultrasonography of the portal venous system using a Voluson S6 ultrasound unit equipped with a 3.5–5 MHz convex array probe. Examinations were performed with patients in the supine position, and measurements were obtained from the right hypochondriac region. The following Doppler parameters were recorded: portal vein diameter, mean portal venous flow velocity, direction of flow (hepatopetal or hepatofugal), and splenic vein diameter. Splenic length was assessed in the coronal plane via the left intercostal space, ensuring inclusion of the hilum. Subsequently, upper gastrointestinal endoscopy was performed with the patient in the left lateral position to evaluate the presence and grade of gastro-esophageal varices. Esophageal varices were classified according to the Paquet classification: Grade I (microcapillaries in the distal esophagus), Grade II (one or two small varices), Grade III (medium-sized varices), and Grade IV (large varices in any part of the esophagus). Gastric varices were classified using the Sarin system as gastroesophageal varices type 1 (GOV1), gastroesophageal varices type 2 (GOV2), isolated gastric varices type 1 (IGV1), and isolated gastric varices type 2 (IGV2). The severity of portal hypertensive gastropathy was graded as mild (reddened congested mucosa), moderate (marked redness with fine reticular pattern and raised mucosa), or severe (marked redness, reticular pattern with point bleeding). The presence of gastric-antral vascular ectasia (GAVE) was also noted. Data were analyzed using the Statistical Package for the Social Sciences (SPSS) software version 25.0 (SPSS Inc., Chicago, Illinois). A 95% confidence interval was applied, and a p-value < 0.05 was considered statistically significant. The presence of gastro-esophageal varices was divided into two groups present and absent. Unpaired t-tests were used to compare mean values of portal vein diameter, portal vein velocity, superior mesenteric and splenic vein diameters, splenic size, portal venous flow pattern, and hepatic arterial indices between the two groups. The chi-square test was employed to assess associations between portal vein Doppler parameters and the severity of portal hypertension with respect to varices, while binary logistic regression analysis was used to evaluate the predictive value of Doppler findings and splenic measurements in assessing portal hypertension severity.

3. RESULTS:

Table 1: Gastro-esophageal varices distribution

Gastro-esophageal varices	Frequency	%

Present	31	62
Absent	19	38

Table 1 represents that out of 50 patients included for this study, 62% had gastro-esophageal varices present whereas in 38% gastro-esophageal varices was absent.

Table 2: Mean distribution of portal hypertension parameters

Varices		**N**	**Mean**	**Std. Deviation**	**Std. Error Mean**
Portal Vein Diameter (PVD)	Present	31	14.426	1.2583	.2260
	Absent	19	9.863	1.7818	.4088
Portal Vein (PV) velocity	Present	31	15.19	3.995	.718
	Absent	19	29.37	5.336	1.224
Superior Mesenteric Vein diameter (SMV)	Present	31	11.458	1.8180	.3265
	Absent	19	7.358	1.4013	.3215
Splenic Vein Diameter (SVD)	Present	31	11.819	1.8591	.3339
	Absent	19	7.405	1.6297	.3739
Splenic size	Present	31	15.958	2.8413	.5103
	Absent	19	10.537	1.4565	.3341
Hepatic Artery Pulsatility index (HA PI)	Present	31	1.3006	.20852	.03745
	Absent	19	.8942	.06440	.01478
Hepatic Artery Resistive index (HA RI)	Present	31	.7048	.05391	.00968
	Absent	19	.5979	.03838	.00881
Hepatic artery Velocity	Present	31	120.10	22.016	3.954
	Absent	19	70.89	14.644	3.359

The table 2 shows the mean values, standard deviation (SD) and standard error mean of portal hypertension parameters in gastro-esophageal varices and nonvarices groups.

Graph 1: Mean distribution of portal hypertension parameters

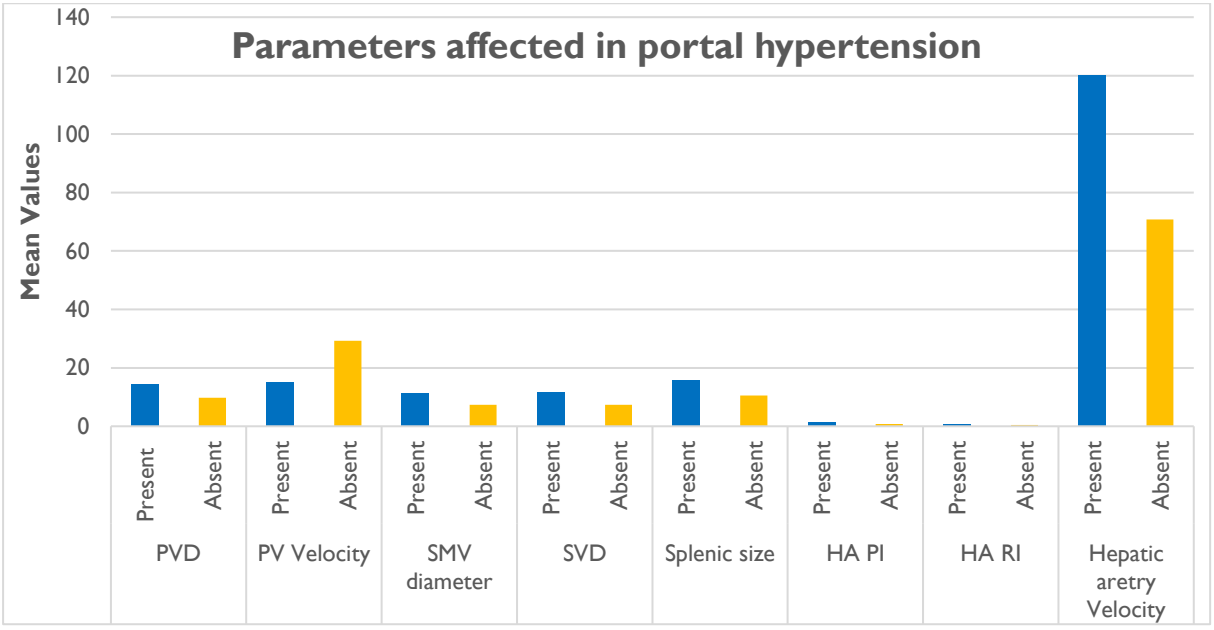

Table 3: Comparison of parameters affected in portal hypertension

Parameters	t-value	df	p-value	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
						Lower	Upper
PVD	10.60	48	.0001*	4.5626	.4302	3.6977	5.427
PV velocity	-10.70	48	.0001*	-14.175	1.324	-16.837	-11.51
	-9.990	30.34	.0001*	-14.175	1.419	-17.071	-11.27
SMV diameter	8.407	48	.0001*	4.1002	.4877	3.1195	5.080
SVD	8.528	48	.0001*	4.4141	.5176	3.3733	5.454
Splenic size	7.699	48	.0001*	5.4212	.7042	4.0054	6.837
HA PI	8.230	48	.0001*	.40643	.04939	.30714	.5057
HA RI	7.542	48	.0001*	.10694	.01418	.07843	.1354
Hepatic artery Velocity	8.625	48	.0001*	49.202	5.705	37.732	60.67

*Significant

Table 3 represents that unpaired t test was applied to compare Portal Vein Diameter (PVD), Portal Vein (PV) velocity, Superior Mesenteric Vein (SMV) diameter, Splenic Vein Diameter (SVD), Splenic Size, Portal Vein (PV) flow, Recanalised Paraumbilical vein, Hepatic Artery Pulsatility Index (HA PI), Hepatic Artery Resistive Index (HA RI), Hepatic artery Velocity between both groups. There was statistically significant (p-value 0.0001) difference between gastro-esophageal and non gastro esophageal groups for all the parameters.

Table 4: Association of gastro-esophageal varices and portal venous flow

		PV Flow		Total	Chi-square	p-value
		Hepatopetal	Hepatofugal			
Varices	Present	28	3	31	1.956	.279 (NS)
	Absent	19	0	19		

NS: Non-significant

Table 4 represents that there was no statistically significant difference (p-value 0.279) found between gastro-esophageal varices and portal venous flow.

Table 5: Association of gastro-esophageal varices and Recanalised Paraumbilical vein

		Recanalised paraumbilical vein		Total	Chi-square	p-value
		Yes	No			
Varices	Present	3	28	31	1.956	.279 (NS)
	Absent	0	19	19		

NS: Non-significant

Table 5 represents that there was no statistically significant difference (p-value 0.279) found between gastro-esophageal varices and recanalised paraumbilical vein.

Table 6: Binary logistic regression for portal hypertension parameters

Portal hypertension variables		Beta	S.E.	p-value	Odd's ratio	95% C.I.		R square
						Lower	Upper	
	PVD	-.236	.301	**0.04***	.79	.438	1.42	0.626
	PV velocity	-.048	.087	**0.03***	.95	.80	1.13	
	SMV diameter	-1.236	.565	**0.02***	.29	0.96	.88	
	SVD	1.482	.617	**0.04***	4.40	1.31	14.75	
	Splenic size	-.317	.293	**0.001***	.728	.410	1.29	
	PV Flow	-18.75	23090.7	**0.001***	0.74	.00	-	
	Recanalised Paraumbilical vein	-4.55	2.597	.10	.011	.00	1.71	
	HA PI	-7.61	4.63	.825	.00	.00	4.29	
	HA RI	2.24	10.06	.464	9.38	.00	3.482	
	Hepatic artery Velocity	-.018	.025	.99	.98	.935	1.03	

*Significant

Note: variables entered in the model: Portal Vein Diameter (PVD), Portal Vein (PV) velocity, Superior Mesenteric Vein

(SMV) diameter, Splenic Vein Diameter (SVD), Splenic Size, PV Flow, Recanalised Paraumbilical vein, Hepatic Artery Pulsatility Index (HA PI), Hepatic Artery Resistive Index (HA RI), Hepatic artery Velocity.

Table 6 shows that a logistic regression was performed for all the parameters of portal hypertension to ascertain the effects on gastro-esophageal varices on the likelihood of severity that participants are diagnosed with portal hypertension. The logistic regression model was statistically significant, $p < 0.05$ for portal vein diameter (PVD), portal vein (PV) velocity, superior mesenteric vein (SMV) diameter, splenic vein diameter (SVD), splenic size and PV Flow. The model explained 62.6.0% (Nagelkerke R^2) of the variance in portal hypertension and correctly classified 82.0% of cases. Increased portal vein diameter (PVD), portal vein (PV) velocity, superior mesenteric vein (SMV) diameter, splenic vein diameter (SVD), splenic size and PV Flow was associated with an increased likelihood of exhibiting portal hypertension.

4. DISCUSSION:

Liver cirrhosis is a progressive chronic liver disease in which clinical manifestations depend on the underlying etiology and disease duration. In this study involving 50 patients, 62% presented with gastroesophageal varices, while 38% had none. Of these, 48 were male and 2 female, aged 35–55 years. Doppler ultrasonography was utilized to assess hepatic and portal hemodynamics noninvasively (9). Barakat et al emphasized its value in studying blood flow characteristics in cirrhotic patients (10). Accordingly, duplex Doppler ultrasound was used to evaluate hemodynamic parameters such as portal vein diameter (PVD), portal vein velocity (PVV), superior mesenteric vein (SMV) diameter, splenic vein diameter (SVD), splenic size, hepatic artery resistive index (HARI), and hepatic artery velocity. Previous studies have demonstrated relationships between these parameters and the presence of esophageal varices.

Doppler indices such as portal vascular resistance, hemodynamic liver index, and splenoportal index have been suggested as noninvasive predictors of varices, with sensitivities and specificities around 63–76% and 92%, respectively (11). However, accuracy in detecting large varices remains limited due to operator and equipment dependency. In this study, the portal vein diameter was significantly increased in patients compared to controls (14.43 mm vs. 9.86 mm, $p < 0.001$). Similar findings were reported by Sarwar et al., Nicolau et al., and Hagen-Ansert, who associated larger PVDs (>11–13 mm) with high-grade varices and portal hypertension (12,13,14).

Reduced portal vein flow velocity (<16 cm/s) and increased diameter are characteristic of portal hypertension (15). Our findings showed significantly decreased PV velocity in patients (15.19 cm/s) compared to controls (29.37 cm/s; $p < 0.001$), consistent with Liu et al., who correlated decreased PV velocity with variceal bleeding risk (16). Similarly, S. Plestina et al. identified portal vein size as a predictor of varices, though Feng Hua Li et al. found no such association (17,18).

The mean SMV and SVD diameters were greater in patients (11.46 mm and 11.82 mm, respectively) than in controls (7.36 mm and 7.41 mm). Spleen size was also significantly increased in patients with varices (15.96 cm) compared to those without (10.54 cm; $p < 0.001$), aligning with Shankar et al., and Serag et al., who reported spleen size cutoffs >13.5–13.9 cm as strong predictors of varices (sensitivity 90–94.5%, specificity 75–80%) (8,19).

Increased hepatic artery pulsatility index (HA PI) was observed in patients (1.30 vs. 0.89 in controls), similar to Elbarbary et al. (20). The hepatic artery resistive index (HA RI) was also elevated (0.70 vs. 0.60; $p < 0.001$), consistent with Liu et al., who linked higher HA RI and reduced PV velocity to increased variceal bleeding risk (21). Hepatic artery velocity was significantly higher in patients (120.10 cm/s) than controls (70.89 cm/s; $p < 0.001$), indicating compensatory hepatic arterial hyperperfusion due to reduced portal flow (22).

A comparison of Doppler parameters between patients with and without gastroesophageal varices revealed statistically significant differences ($p < 0.001$), except for recanalized paraumbilical vein ($p = 0.279$). These findings align with Mandal et al., Shankar et al., and Bhattarai et al., who all demonstrated strong correlations between portal vein diameter, spleen size, and variceal grades (7,8,23). Logistic regression analysis confirmed that increased PVD, PV velocity, SMV and SVD diameters, splenic size, and PV flow were significantly associated with portal hypertension severity ($p < 0.05$), explaining 62.6% of the variance and correctly classifying 82% of cases. Recanalized umbilical veins, commonly seen in cirrhotic patients, were also linked with increased portal velocity (24,25).

In summary, duplex Doppler ultrasound serves as a reliable, noninvasive tool for assessing portal hemodynamics in liver cirrhosis, providing valuable correlations between vascular parameters and the presence or severity of gastroesophageal varices.

5. CONCLUSION:

In patients with cirrhosis and portal hypertension, this study demonstrates that a portal vein diameter greater than 12.25 mm is significantly associated with an increased likelihood of developing gastroesophageal varices. A reduction in mean portal vein velocity correlates with greater severity of portal hypertension and a higher risk of variceal bleeding, while the presence of a recanalized umbilical vein may indicate elevated portal venous velocity. Similarly, a spleen size exceeding 13.9 cm is linked to an increased probability of variceal formation. Therefore, assessment of portal vein Doppler parameters and spleen size through ultrasonography can serve as reliable, non-invasive indicators for predicting gastroesophageal

varices in patients with portal hypertension. Further large-scale studies are warranted to enhance the precision and diagnostic value of these non-invasive modalities in evaluating portal hypertension and its complications.

REFERENCES

[1] Kim MY, Jeong WK, Baik SK. Invasive and non-invasive diagnosis of cirrhosis and portal hypertension. World J Gastroenterol 2014;20:4300-4315.

[2] British Medical Journal. Cirrhosis definition. Updated Jul 07, 2017. Available from:http://bestpractice.bmj.com/best-practice/monograph/278/basics/definition.html

[3] Kumar M, Kumar A, Hissar S et al. Hepatic venous pressure gradient as a predictor of brosis in chronic liver disease because of hepatitis B virus. LiverInt2008;28:690–8.

[4] Regev A, Berho M, Jeffers LJ. Sampling error and intraobserver variation in liver biopsy in patients with chronic HCV infection. AmJGastroenterol2002;97:2614–18.

[5] de Franchis R. Expanding consensus in portal hypertension: report of the Baveno VI Consensus Workshop: Stratifying risk and individualizing care for portal hypertension.2015;63:543–5.

[6] Wilson SR, Withers CE. The liver. In: Rumack CM, Wilson editors. Diagnostic ultrasound. 3rd ed. St. Louis, Missouri: Elsevier Mosby; 2005. pp. 97-101.

[7] Mandal L, Mandal, Bandyopadhyay D, Datta S. Correlation of portal vein diameter and splenic size with gastro-oesophageal varices in cirrhosis of liver. JIACM 2011;12(4): 266-70.

[8] Shankar R, Banerjee S, Anshul, Ganguly S, Bansal S, Uppal A. A Study of Association of Portal Vein Diameter And Splenic Size With Gastro-Oesophageal Varices in Liver Cirrhosis Patients. Journal of Dental and Medical Sciences 2016;15(9):125-129.

[9] Baik SK, Jee MG, Jeong PH. Relationship of hemo-dynamic indices and prognosis in patients with liver cirrhosis. Korean J Intern Med 2004; 19:165 –170.

[10] Barakat M. Non-pulsatile hepatic and portal vein waveforms in patients with liver cirrhosis: concordant and discordant relationships. Br J Radiol 2004; 77:547 –550.

[11] Bintintan A, Chira RI, Bintintan VV, et al. Value of hepatic elastography and Doppler indexes for predictions of esophageal varices in liver cirrhosis. Med Ultrason. 2015;17:5–11.

[12] Sarwar S, Khan AA, Alam A. Non endoscopic prediction of presence of esophageal varices in cirrhosis. J Coll Physicians Surg Park 2005; 15:528 –531.

[13] Nicolau C, Bianchi L, Vilana R. Gray-scale ultrasound in hepatic cirrhosis and chronic hepatitis: diagnosis, screening, and intervention. Seminars Ultrasound CT MR 2002; 23: 3–18.

[14] Hagen-Ansert SL. Textbook of diagnostic ultrasonography. 6th ed. St. Louis, MO: Elsevier Mosby; 2006. 2 :1–6.

[15] Berzigotti A, Seijo S, Reverter E, Bosch J. Assessing portal hypertension in liver diseases. Expert Rev Gastroenterol Hepatol. 2013;7: 141–155.

[16] Liu C, Hsu S, Lang C, Tsai F, Lin J, et al. Esophageal varices: non-invasive diagnosis with duplex Doppler US in patients with compensated cirrhosis. Radiology 2008; 248:132 –139.

[17] Pleština Color Doppler ultrasonography is reliable in assessing the risk of esophageal variceal bleeding in patients with liver cirrhosis. Wiener klinische Wochenschrift October 2005, Volume 117, Issue 19-20, pp 711-717.

[18] Feng Hua Li, Jian-Guo Xia, Hong-Li Li, et al. Hemodynamic analysis of esophageal varices in patients with liver cirrhosis using color Doppler ultrasound; World J Gastroenterology 2005 Aug 7; 11 (29): 4560-5.

[19] Serag Esmat and Dalia Omran. Study of noninvasive predictors of portal hypertension in liver cirrhotic Egyptian patients. Journal of American Science 2011; 7(1):962-968.

[20] Aly A. Elbarbarya , Mohamed M. Elbedewyb , Amr M. Elbadry. Hemodynamic analysis of portal hypertension in patients with liver cirrhosis. Tanta Medical Journal 2014, 42(4):130–137.

[21] Liu C, Hsu S, Lang C, Tsai F, Lin J, et al. Esophageal varices: Non invasive diagnosis with duplex Doppler US in patients with compensated cirrhosis. Radiology 2008; 248:132 –139.

[22] McNaughton DA, Abu-Yousef MM. Doppler US of the liver made simple. Radiographics 2011;31: 161-188.

[23] Bhattarai S, Gyawali M2, Dewan KR, et al. Study of Portal Vein Diameter and Spleen Size by Ultrasonography. NJR 2014; 4(7) 6-14.

[24] Domland M, Gebel M, Caselitz M, et al. Comparison of portal venous flow in cirrhotic patients with and without paraumbilical vein patency using duplex-sonography. Ultraschall Med. 2000;21(4):165-9.

[25] Gupta D, Chawla YK, Dhiman RK, et al. Clinical significance of patent paraumbilical vein in patients with liver cirrhosis. Dig Dis Sci. 2000;45(9):1861-4.