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Study Of Portal Vein Doppler Indices and Other Noninvasive Markers as Predictors of Esophageal Varices in Cirrhotic Patients

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KEYWORDS

Cirrhosis, Portal hypertension, Esophageal varices, Noninvasive parameters, hepatic vessel Doppler indices, Platelet – spleen diameter ratio.

ABSTRACT:

Background: Portal hypertension commonly accompanies liver cirrhosis. The development of esophageal varices (EV) is one of the major complications of portal hypertension. Guidelines recommend that all cirrhotic patients should be screened for the presence of EV when liver cirrhosis is diagnosed.

Objectives: To evaluate portal hypertension parameters in liver cirrhosis by using Doppler ultrasound and other non-invasive parameters in predicting esophageal varices and to correlate portal hypertension parameters in predicting esophageal varices and upper GI bleed from esophageal varices.

Materials and Methods: 99 Cirrhotic patients were enrolled in our study based on their clinical presentation, Child-Pugh status, co-morbid conditions, baseline blood parameters, liver function test, etiology and endoscopic grading of varices were noted. All patients will be subjected to Doppler evaluation of portal system and other indices like Liver vascular index, congestion index, portal hypertensive index and presence of porto-systemic collaterals also noted. Platelet/spleen ratio, AST/platelet ratio were calculated.

Results: The most common aetiology of cirrhosis in this part of country is Alcohol related liver disease (29%), followed by cryptogenic and Hepatitis B related liver disease (28% & 21% respectively). Non-invasive parameters like Platelet count (114578 in patients without varices vs. 78113 in patients with large varices, P=0.049), Prothrombin time (16.53±4.3 vs. 18.18±4.5, P=0.030), Platelet count/spleen diameter ratio (957 vs. 627, P=0.011) predicted the presence of large esophageal varices. Among the Colour Doppler Ultrasound study parameters, the Portal vein mean velocity (15.44±4.63 vs. 11.91±3.97, P=0.019), Liver vascular index (14.38±5.56 vs. 9.38±4.01, P=0.001), Spleen size>16.2 cm (13.34±4.20 vs.16.29±3.42, P=0.05) predicted the presence of large esophageal varices, increasing the risk for upper gastrointestinal bleeding.

Conclusion:

Results of our study indicate that non-invasive tools like platelet count, prothrombin time, platelet/spleen diameter ratio, spleen size >16.2 cm, and Doppler parameters like portal vein velocity, liver vascular index are predictors of presence of large esophageal varices.

Journal of Chemical Health Risks

www.jchr.org

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INTRODUCTION:

Portal Hypertension is the most common and lethal complication of chronic liver diseases. It is responsible for the development of gastroesophageal varices, variceal hemorrhage, ascites, renal dysfunction, portosystemic encephalopathy, hypersplenism and hepatopulmonary syndrome. Portal hypertension commonly accompanies liver cirrhosis. The development of esophageal varices (EV) is one of the major complications of portal hypertension.[1] The prevalence of EV in patients with liver cirrhosis ranges from 60% to 80%. [2,3] Numerous evidences suggest that varices develop and enlarge with time. Christensen and colleagues showed that the cumulative incidence of varices in patients with cirrhosis increased from 12% to 90% over 12 years.[4] In a study, Cales and Pascal et al. showed 20% of patients who did not have varices developed new varices and 42% of patients with small varices showed definite enlargement.[5]

The risk of bleeding from these varices is associated with the severity of the liver disease and the size of varices, which are the most important predictors of bleeding.[6,7] It is estimated that approximately 60%–80% of patients with cirrhosis develop esophageal varices during their life at a rate of 8% per year, and the progression from small to large varices occurs in 5%–10% of patients after the first year.[8-11] Portal hypertension related upper GI bleeding accounts for 15-20% of all upper GI bleeding cases in Western population[12] and around 45% cases in Indian population.[13] Large EVs (LEVs) are more likely to bleed than small EVs (SEVs)[14] due to high variceal wall tension.[15] Among 60% of cirrhotic patients who develop gastroesophageal varices, 50% will experience an episode of variceal hemorrhage within 2 years of the diagnosis of the varices.[16,17] Majority of initial bleeds occurring with 1 year from the time of detection of varices.[6, 18] Up to one-third to half of the patients with advanced liver disease and large varices die after the first attack of variceal bleeding.[19]

The mortality rate from first episode of bleeding is 40%.[20] Mortality from each rebleeding episode is 20-30%. The reported overall mortality from variceal bleeding ranges from 17% to 57%. [2, 3] Introduction of prophylactic antibiotics and pharmacotherapy have shown to reduce the mortality.[2, 3, 21] The American Association for the Study of Liver Disease and the Baveno V Consensus Conference on portal hypertension recommended that all cirrhotic patients should be screened for the presence of EV when liver cirrhosis is diagnosed.[22, 23] Other authors have suggested repeating endoscopy at 2–3 year intervals in patients without varices and at 1–2 year intervals in patients with small varices so as to evaluate the development or progression of varices.[9, 24] Upper gastrointestinal endoscopy, which is the most common and accurate procedure for evaluation of varices, is at times inconvenient for patients.[25,26] It also bears a small risk of complications like esophageal perforation, aspiration of gastric contents and bacteremia.[27,28] Moreover, sedation with benzodiazepines usually used for this procedure can significantly exaggerate hepatic encephalopathy.[29] Investigators have attempted to identify characteristics that 'noninvasively' predict the presence of varices. These studies have shown that biochemical, clinical, and ultrasonographic parameters alone or together have good predictive power for noninvasively assessing the presence of EV.[30-43] Overall, the most common result of these studies was that parameters such as splenomegaly, thrombocytopenia, Childs score, ascites, portal flow patterns, and platelet count—spleen diameter ratio were predictors of the presence of EV.

Doppler ultrasonography can be regarded as an attractive and non-invasive alternative method and may provide useful functional information. Many investigations reported correlations between different hepatic vasculature Doppler indices and the severity of portal hypertension and the resultant esophageal varices.[20, 35, 44-50] Our study aimed to determine what Doppler indices of hepatic vessels can be used to predict the presence of esophageal varices and to evaluate the severity of esophageal varices.

AIM & OBJECTIVES

1.To evaluate portal hypertension parameters in liver cirrhosis by using Doppler ultrasound.

2.To evaluate other non-invasive parameters in predicting esophageal varices.

Journal of Chemical Health Risks

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JCHR (2025) 15(3), 2727-2738 | ISSN:2251-6727

3.To correlate portal hypertension parameters in predicting Esophageal varices and upper GI bleed from esophageal varices.

MATERIALS AND METHODS

The study was conducted after getting approval by the Ethics Committee of our institution. Ninety-nine Cirrhotic patients registered in liver clinic and admitted in ward (both old and new patients) in Dept. of medical gastroenterology; Tertiary care teaching medical college hospital were included in this prospective study. The study period from January 2024 to October 2024. All subjects included in the study provided informed consent to participate. All patients underwent a detailed clinical evaluation at entry, with the following data:

Age, Gender, Duration of illness, Details of treatment prior to registration. Etiology of cirrhosis was arrived based on history of alcohol intake including quantity & total duration of consumption; blood for viral serology (HBsAg &HBV DNA assay for hepatitis B and HCV RNA & Anti-HCV for hepatitis C); serum ceruloplasmin (<20mg%), presence of Kayser Fleischer ring and 24 hours urine copper estimation (>100mg%) for Wilsons disease; and antinuclear antibody, hypergammaglobulinemia (>3.5 gm%) for autoimmune related cirrhosis. Apart from details of past blood transfusion, surgery, family members with liver disease, details of associated co-morbid illness were also recorded.

Relevant history and physical characteristics including symptoms and signs of liver failure (spider angioma, palmar erythema etc.), hepatomegaly, splenomegaly, and abdominal vein collaterals were recorded. Ascites was graded as none, mild (detectable only on ultrasound), moderate (visible moderate symmetrical abdominal distension) or severe (marked abdominal distension). Hepatic encephalopathy was graded from grade 0 to IV, as per the Conn's grading. Diagnosis of cirrhosis was based on clinical, biochemical, and ultrasonographic findings.

Patients with evidence of hepatocellular carcinoma on ultrasonography, Portal vein thrombosis and Previous H/O surgical intervention for portal hypertension were excluded. Hematological and biochemical workup included measurement of haemoglobin, total leukocyte count, platelet count, prothrombin time, and serum concentrations of bilirubin (total and conjugated), protein, albumin, alanine aminotransferase and aspartate aminotransferase. All patients were tested for HBsAg and antibodies to hepatitis C virus to determine the cause of liver cirrhosis. Tests for other causes of cirrhosis (serum ceruloplasmin and slit lamp examination for Wilson's disease, tests for autoantibodies for autoimmune liver disease, iron studies for hemochromatosis) were carried out only if there was a suggestive clinical clue. For each patient, a modified Child-Pugh score was calculated. Child Turcotte Pugh (CTP) score was applied to grade the severity of cirrhosis. CTP score is based on serum bilirubin, serum protein, ascites, prothrombin time and hepatic encephalopathy. Minimum score of CTP is 5 and maximum score is 15. Based on scoring system, cirrhosis was classified as Childs A when the total score was 5 & 6, Childs B when the total score was 7 to 9, and Childs C when the total score is exceeded 9.

All patients underwent ultrasonography and the following details were recorded: maximum vertical span of the liver; nodularity of liver surface; spleen size (length of its longest axis); diameter of the portal and splenic veins; presence of portal-systemic collaterals; and presence of ascites. All Doppler assessments were performed by a single radiologist using a 3.5 MHz curvilinear transducer of EASOATE MyLab40 (Germany) machine. Patients were examined while fasting and in supine position and quiet respiration. Subcostal or intercostals ultrasonic windows were used to obtain longitudinal view of middle hepatic vein, portal vein and hepatic artery (in front of portal vein) with an ultrasound beam incidence angle of less than 60º. Outer-to-outer main portal vein diameter (mm) was measured in midway between the spleno-portal junction and its intrahepatic bifurcation. Several Doppler ultrasonographic parameters were measured such as Liver and spleen sizes, portal and splenic vein diameters, portal vein mean velocity (PVV), hepatic artery resistive index (HARI), hepatic artery pulsatility index (HAPI), portal vein cross sectional area, Splenic artery resistive index (SARI), presence of portal-systemic collaterals and following indices were calculated: liver vascular index

Journal of Chemical Health Risks

www.jchr.org

JCHR (2025) 15(3), 2727-2738 | ISSN:2251-6727

(LVI), congestion index (CI), portal hypertension index (PHI).

All patients underwent upper gastrointestinal endoscopy for assessment of esophageal and gastric varices after Doppler ultrasound examination. If EVs were present, their size was graded as I-IV, using the Paquet grading system. Grade 0: No varices, grade I: Varices, disappearing with insufflation, grade II: Larger, clearly visible, usually straight varices, not disappearing with insufflation, grade III: More prominent varices, locally coil-shaped and partly occupying the lumen, grade IV: Tortuous, sometimes grape-like varices occupying the esophageal lumen. Further, patients were classified dichotomously either as having large EVs (grade III-IV) or as not having these (no varices) or small EV (grade I-II). Presence of gastric varices, portal hypertensive gastropathy, and duodenopathy were recorded wherever appropriate.

Statistical analysis: Data were analyzed with Analysis of Variance (ANOVA) Technique. Descriptive statistics including means, standard deviations, and frequencies were computed. For determining associations, univariate analysis was performed by ANOVA. A p value less than 0.05 was considered statistically significant. A multivariate ordinal logistic regression (OLR) model was used for determining the adjusted associations between size of esophageal varices and hepatic hemodynamic determinants.

RESULTS:

Ninety-nine cirrhotic patients (70 men, 29 women) were enrolled in the study. Mean age of the study population was 44 ± 11.5 years. Cirrhosis was predominantly observed in men (Male: Female - 2.4:1). Table 1 shows the patients' baseline characteristics **(Table 1)**.

Characteristics	No (%)
Total No of cases	99
Mean age (years)	44±11.5
Gender distribution	
Male	70 (71%)
Female	29 (29%)
Literacy status	
Yes	78 (79%)
No	21 (21%)
Alcohol Ingestion	
Yes	43 (43%
No	56(57%)
Smoking	
Yes	33(33%)
No	66(67%)

Table 1: Demographic characteristics of the study population

Alcohol related liver disease constitutes the most common etiology of cirrhosis in our study followed by cryptogenic and Hepatitis B related liver disease (**Table 2**).

Etiology	n	%
Alcohol	32	29.63%
Alcohol+Hepatits B	8	7.41%
Alcohol+Hepatits C	2	1.85%
Autoimmune	2	1.85%
Cryptogenic	30	27.78%
Hepatitis B	23	21.30%
Hepatitis C	7	6.48%
Wilsons disease	3	2.78%

Table 2: Etiology of cirrhosis

Journal of Chemical Health Risks

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JCHR (2025) 15(3), 2727-2738 | ISSN:2251-6727

The severity of liver disease as assessed by Child Pugh scoring was: Child A in 31%, Child's B and C in 45% and 23% respectively (**Table 3**).

CTP Grade	n	%
Grade A	31	31%
Grade B	45	45%
Grade C	23	23%
Total	**99**	**100%**

Table 3: Severity of liver disease as assessed by CTP score

The mean value of laboratory investigations and mean values of Doppler study parameters are shown in **Table 4 and Table 5** respectively.

Investigation	Mean	Std dev
Hb (gms%)	9.64	2.61
Platelet Count(no.)	95818.18	42936.68
PT (sec.)	18.49	4.64
INR	1.42	0.40
Creatinine (mg/dl)	2.23	13.48
TB mg/dl)	2.60	2.74
AST (U/L)	77.63	60.24
Albumin (g/dl)	2.84	0.73

Table 4: Mean values of Laboratory investigation

Doppler study	Mean	Std dev
Liver size (cms)	11.98	1.45
PV DM (cms)	1.20	0.26
PV velocity (cm/sec)	12.97	4.60
HARI	0.71	0.12
HAPI	1.32	0.44
PV cs area(cm2)	1.05	0.58
Spleen Size (cms)	14.85	3.36
SARI	0.67	0.08
Liver Vascular Index	10.73	5.01
Congestive Index	0.11	0.14
PHT Index	0.05	0.11

Table 5: Mean values of Doppler study parameters

Of the 99 patients, 19 (19%) did not have esophageal varices at endoscopy, 36 (36%) had small esophageal varices (SEV) and 44 (45%) have large esophageal varices (LEV).

Table 6 shows the relationship of absence of EV or presence of SEV/LEV with various clinical, laboratory and ultrasonographic characteristics on univariate analysis.

Parameter	No Varices (n=19)	SEV (n=36)	LEV (n=44)	P-Value
MELD	11.00± 4.22	14.39±5.59	12.39±4.94	0.500
Platelet Count	114578±53732	95333±44346	78113±34290	0.049*
PT(sec)	16.53±4.33	19.90±4.59	18.18±4.55	0.030*
INR	1.34±0.37	1.48±0.37	1.41±0.43	0.490
TB (mg/dl)	1.94±1.86	3.25±3.09	2.35±2.69	0.176
AST (U/L)	83.74±69.87	78.94±47.42	73.91±66.06	0.830

Journal of Chemical Health Risks

www.jchr.org

JCHR (2025) 15(3), 2727-2738 | ISSN:2251-6727

PV Velocity (cm/sec)	15.44±4.63	12.96±4.90	11.91±3.97	0.019*
HARI	0.67±0.08	0.74±0.15	0.71±0.09	0.108
HAPI	1.13±0.34	1.38±0.53	1.35±0.38	0.117
PV cs area (cm^2)	1.08±0.78	0.98±0.51	1.10±0.55	0.638
Spleen Size (cm)	13.34±4.20	15.10±2.56	16.29±3.42	0.05*
SARI	0.71±0.09	0.66±0.07	0.67±0.08	0.092
Liver Vascular Index	14.38±5.56	10.46±4.99	9.38±4.01	0.001*
Congestive Index	0.10±0.13	0.11±0.14	0.12±0.15	0.816
PHT Index	0.04±0.06	0.03±0.03	0.06±0.16	0.444
P/S Ratio	957.00±607.54	668.86±343.94	627.68±325.51	0.011*
APRI	0.07±0.04	0.10±0.07	0.11±0.12	0.288

Table 6: Comparison of different parameters according to Varices:

Six factors were found to be significantly different between the three groups. These were Platelet count (P=0.049), Prothrombin time (P=0.030), Portal vein velocity (P=0.019), Liver vascular index (P=0.001), Spleen size (P=0.05) and Platelet count/spleen ratio (P=0.011). No significant difference is observed for the other parameters.

The results of a logistic regression analysis in 99 patients. In this analysis no factors found to have independent predictive value for the presence of LEV (**Table 7**).

Variables	P-Value	Odds Ratio
Group A	0.440	1§
Group B	0.761	1.778
Group C	0.360	5.511
MELD	0.559	0.880
Hb	0.904	0.975
Platelet	0.794	1.000
PT	0.047	0.692
INR	0.055	185.554
TB	0.764	0.915
AST	0.182	1.041
PV_DM	0.609	0.344
PV_Velocity	0.214	1.161
HARI	0.596	62.255

Journal of Chemical Health Risks
www.jchr.org
JCHR (2025) 15(3), 2727-2738 | ISSN:2251-6727

HAPI	0.613	0.393
PV_CS	0.764	1.333
Spleen size	0.114	0.692
SARI	0.053	168
Liver_Vascular index	0.315	1.212
Congestive_Index	0.271	19.845
PHT_Index	0.429	10.125
P/S_Ratio	0.568	0.999
APRI	0.175	0.000

Table 7: Logistic Regression Results

DISCUSSION:

Variceal gastrointestinal bleeding is a major complication of portal hypertension with significant morbidity and mortality. However, this complication occurs primarily in patients with LEV and is uncommon in those with small varices. Because the occurrence of variceal bleeding can be prevented using pharmacological agents like beta-adrenergic receptor antagonists, it is important to recognize patients who have LEV and are thus at a higher risk of developing variceal bleeding and likely to benefit from such interventions. It has therefore been recommended that patients with liver cirrhosis should be screened for the presence of LEV at the time of initial diagnosis and at periodic intervals thereafter throughout life. Efforts have been made to identify clinical, laboratory and imaging characteristics that may non-invasively predict the presence or absence of LEV with a high degree of accuracy, either reducing or eliminating the need for screening endoscopy.

Our study, based on information achieved from 99 liver cirrhosis patients from tertiary care hospital in south India, including 44 with LEV, showed that 6 factors had predictive ability for the presence of LEV on univariate analysis. These were Platelet count (P=0.049), Prothrombin time (P=0.030), Portal vein mean velocity (P=0.019), Liver vascular index (P=0.001), Spleen size (P=0.05) and Platelet count/spleen ratio (P=0.011). However, on multivariate analysis, no factors were found to have independent predictive value.

Our study population was composed mainly of patients with liver cirrhosis due to alcohol abuse or chronic hepatitis B infection, which represent more than 50% of the causes of liver cirrhosis, followed by cryptogenic in 27%. In our study, there was no correlation between the presence of EV and CTP classification. These findings were also reported by other researchers,[51] but in a study by zaman et al. showed CTP class B or C were nearly 3 times more likely to have varices on endoscopy than CTP class A.[34] MELD score also was not a good predictor of the presence of EV or LEV in our study. Burton et al.[52] and Levy et al.[53] also demonstrated no predictive value for MELD for the presence of EV. But in a recent study by Tafarel et al.[54] showed that the presence of EV could be predicted by MELD score higher than 8 points (sensitivity 80.1% and specificity 51.2%). The importance of platelet count has been alluded to in many studies. [55-58] The values of thrombocytopenia related to the presence of EV were different among various published studies.

In our study platelet count less than 95000/mm3 was associated with the presence of SEV and platelet count less than 78000/mm3 associated with the presence LEV. This is in line with existing studies that have documented LEV with platelet count less than 100000/mm3. Thrombocytopenia and EV are associated

Journal of Chemical Health Risks

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JCHR (2025) 15(3), 2727-2738 | ISSN:2251-6727

because both resulted from deterioration of liver functional reserve, leading to hemodynamic changes. In our study, patients with LEV group had large spleen size (16.29±3.42 cm) in comparison to those in SEV (15.10±2.56cm) & no varices (13.34±4.20 cm) group with p =0.05. In the only available Indian study, Amarapurkar et al.[36] found that presence of splenomegaly was associated with presence of esophageal varices but not with LEV. In our study, LEV were more often associated with a splenomegaly (size>162mm, p=0.05) as has been observed in other parts of the world.

The presence of splenomegaly in cirrhotic patients is likely the result of vascular disturbances that are mainly related to portal hypertension. Even though low platelet count and splenomegaly were used as important predictors of presence of EV, the use of platelet count alone as a non-invasive predictor of EV can be misleading and cannot be solely attributed to portal hypertension. In patients with chronic liver disease the presence of decreased platelet count may depend on several factors other than portal hypertension, such as shortened platelet mean lifetime, decreased thrombopoietin production, or myelotoxic effects of alcohol or hepatitis viruses. In this situation Giannini et al. [57] introduced a new parameter, platelet count/spleen diameter (p/s) ratio. He showed that platelet count/spleen diameter ratio with cut off value of 909 had 100% negative predictive value for non-invasively predicting the presence of EV in patients with either compensated or decompensated liver cirrhosis. 100% of patients with a p/s ratio >909 were free from EV. The platelet count/spleen diameter ratios represent an acceptable surrogate marker for clinically relevant portal hypertension. In concordance with the study by Giannini et al. [57] our study also showed similar results.

In our study, prothrombin time predicts the presence of LEV in univariate analysis. But no other study demonstrated prothrombin time as a predictor of EV.Among the hemodynamic characteristics of portal vein and hepatic artery, our study showed the Mean portal vein velocity (15.44±4.63 vs. 12.96±4.90 vs 11.91±3.97, p=0.019), and Liver vascular index (14.38±5.56 vs. 10.46±4.99 vs. 9.38±4.01, p=0.001) were the predictors of EV in univariate analysis. There was a significant association between the mean portal vein velocity and the size of varices (p = 0.019). According to Korner et al. the overall sensitivity for prediction of variceal bleeding in case of decreased portal vein mean velocity was 88%. Although none of other portal vein measurements had statistically significant associations with the size of varices, some articles have shown that portal vein diameter is also increased in large varices,[20,47] such a finding was not observed in our study. Iwao, et al, showed that not only portal venous velocity was significantly lower, but the hepatic arterial pulsatility index was also significantly higher in patients with esophageal varices.[50] Our study results did not disclose any significant associations between hepatic artery resistive index, pulsatility index and esophageal varices status.

In a recent study by Tarzamni et al.[59] Portal vein diameter, congestion index (CI) (0.11 ± 0.03 vs. 0.06 ± 0.03, P < 0.0005), portal hypertensive index (2.62 ± 0.79 vs. 1.33 ± 0.53, P < 0.0005), and hepatic (0.73 ± 0.07 vs. 0.66 ± 0.07, P < 0.001) and splenic artery resistance index (SARI) (0.73 ± 0.06 vs. 0.62 ± 0.08, P < 0.0005) were found to be significantly higher in patients with LEV and portal vein flow velocity (13.25 ± 3.66 vs. 20.25 ± 5.05, P < 0.0005), liver vascular index (8.31 ± 2.72 vs. 17.8 ± 6.28, P < 0.0005) were significantly lower in patients with LEV. A logistic regression model confirmed spleen size >15.05cm, (P = 0.002) and portal hypertensive index (P = 0.040) as independent predictors for the occurrence of large esophageal varices (LEV). Our study correlates only with liver vascular index and portal vein velocity which was significantly lower in patient with LEV.

Piscaglia et al[60] proposed a PHI cutoff of 1.2 s/m as the parameter with the highest accuracy (≈75%) for PHT and useful tool for detecting esophageal varices. Our study did not show significant association between PHI and esophageal varices. Shabestari et al.[61] showed significant correlation between the size of esophageal varices and portal vein mean velocity (p=0.04) and logistic regression analysis did not show any significant associations between Doppler parameters and the size of esophageal varices. He also concluded that none of hepatic vasculature Doppler measurements had a significant role in predicting the size of esophageal

Journal of Chemical Health Risks

www.jchr.org

JCHR (2025) 15(3), 2727-2738 | ISSN:2251-6727

varices. De Bem, et al.[48] also revealed that there is no good correlation between Doppler ultrasound parameters of the portal system and the presence of gastroesophageal varices in cirrhotic patients. According to Liu et al.[62] mean PVV (P =0 .001), SARI (P =0.04), were predictive of the presence of esophageal varices at univariate analysis, but in multivariate logistic regression analysis, only mean PVV was independently associated with the presence of esophageal varices. Our study is also in line with the above studies, in which portal vein diameter, HARI, HAPI, SARI, congestive index, portal hypertensive index did not predict the presence of esophageal varices. Multivariate logistic regression analysis did not show any significant association between Doppler parameters and esophageal varices.

CONCLUSION:

In conclusion, results of our study indicate that non-invasive tools like platelet count, prothrombin time, platelet/spleen diameter ratio, spleen size >16.2 cm, and Doppler parameters like portal vein velocity, liver vascular index are predictors of presence of large esophageal varices. But there were no independent noninvasive predictors of large esophageal varices by multivariate analysis in our study. Values for the noninvasive indicators from this study and comparables need to be validated by randomised prospective studies. Applying the non-invasive techniques including hepatic vessel hemodynamics by Doppler study for the detection of esophageal varices and assess the risk for bleeding may be cost effective and safer than the “scope all strategy”. But further randomised studies are needed to evaluate the accurate predictors of esophageal varices in our population. Till then Upper Gastrointestinal endoscopy remains the gold standard procedure for screening esophageal varices and assessing risk for bleeding.

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Conflicts of Interest:

There are no conflicts of interest

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