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Original Research Article

Portal vein Doppler study in evaluation of portal vein pulsatility pattern for assessing severity of cirrhotic patients with portal hypertension

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ABSTRACT

Background: Liver cirrhosis causes progressive hepatic fibrosis leading to increased intrahepatic vascular resistance and portal hypertension. This study aimed to assess portal vein pulsatility index and complete spectral widening on Doppler ultrasonography as non-invasive indicators of disease severity and their correlation with Child-Pugh classification.
Methods: This cross-sectional study was conducted from January 2024 to September 2025 in the Department of Radiology and Imaging at Bangladesh Medical University (BMU), Dhaka. Portal vein Doppler studies were performed on 30 patients with liver cirrhosis and portal hypertension and 30 healthy adults. The parameters evaluated were portal vein pulsatility index (PI) and complete spectral widening (CSW). Data were analyzed using SPSS version 27.0, with a p value <0.05 considered statistically significant.
Results: Among 60 participants (30 cirrhotic, 30 healthy), cirrhotics had mean age 48.2±12.9 years, 66.7% male. Ascites occurred in 76.7%, hematemesis in 30.0%, jaundice in 23.3%; mean albumin 3.04±0.65 g/dl, bilirubin 2.01±0.48 mg/dl, PT 6.56±2.54 s. Child-Pugh classes: A 30.0%, B 36.7%, C 33.3%. PI was lower in cirrhotics (0.15±0.037 vs 0.41±0.044, p<0.001) and decreased with severity (0.19→0.15→0.12, p<0.001). ROC (cut-off 0.35) showed 96.7% sensitivity, 86.7% specificity, 91.7% accuracy (AUC 0.968); CSW present in 80.0%.
Conclusion: Portal vein pulsatility index and complete spectral widening provide a simple, non-invasive, and highly accurate method for diagnosing and grading liver cirrhosis.

Keywords: Liver cirrhosis, Portal hypertension, Pulsatility index, Complete spectral widening

INTRODUCTION

Liver cirrhosis is the end stage of chronic liver disease, characterized by gradual distortion and replacement of normal liver parenchyma with fibrous tissue and regenerative nodules in response to repeated injuries of diverse origins. According to the World Health Organization (WHO), cirrhosis is a progressive disease marked by widespread fibrosis and replacement of normal liver tissue with abnormal nodules, resulting in a distorted liver structure.[1,2] Histologically, cirrhosis involves necrosis of hepatic parenchyma and perivascular fibrosis. As fibrosis progresses, it causes substantial hemodynamic changes, including increased intrahepatic vascular resistance, impaired hepatic venous outflow, altered hepatic arterial resistance, and eventually the development of portal hypertension.[1,2] Portal hypertension is one of the most lethal complications of cirrhosis and is responsible for many clinical manifestations, including esophageal and gastric varices, ascites, hepatic encephalopathy, spontaneous bacterial peritonitis, and hepatorenal syndrome, all of which significantly impact patient survival.[3,4] Early diagnosis

and assessment of portal hypertension hemodynamics are essential for staging liver disease, predicting prognosis, and guiding management.

Clinical assessment of cirrhosis severity is commonly performed using the Child-Pugh scoring system, which incorporates five parameters: ascites, hepatic encephalopathy, serum bilirubin, serum albumin, and prothrombin time or INR.[5] Patients are classified as Child-Pugh Class A (5–6 points: well-compensated), Class B (7–9 points: significant functional impairment), or Class C (10–15 points: decompensated disease).[6] Survival rates correlate closely with Child-Pugh class, with one- and two-year survival of 100% and 85% for Class A, 80% and 60% for Class B, and 45% and 35% for Class C, respectively.[7] However, the Child-Pugh score is primarily a clinical tool and may not fully capture early hemodynamic changes.

Ultrasonography is a well-established, non-invasive, and widely available imaging modality for assessing liver cirrhosis. Conventional B-mode ultrasonography detects architectural changes such as coarse hepatic echotexture, nodular liver surface, splenomegaly, ascites, and alterations in portal vein diameter.[6] Cirrhosis and fibrosis also result in enlargement of the left lateral segment and caudate lobe and reduction of the left medial and right hemi-liver.[8] However, B-mode imaging often fails to detect early cirrhotic changes until substantial structural distortion has occurred.

Doppler ultrasonography offers an advantage by allowing real-time assessment of hepatic and portal hemodynamics, which is valuable for detecting early circulatory changes. In early liver disease, when B-mode ultrasound may be inconclusive, Doppler evaluation of the portal vein can reveal alterations in portal vein flow velocity, portal vein pulsatility, hepatic artery resistance index, and pulsatility index (PI), indicating raised intrahepatic vascular resistance and portal hypertension.[5,9] Normally, the portal vein exhibits mild pulsatility, with PI ranging from 0.2 to 0.5 due to transmitted cardiac pulsations. A PI below 0.2 reflects reduced pulsatility due to hepatic fibrosis, often seen in advanced liver disease.[10]

Multiple studies have consistently reported significantly lower PI values in cirrhotic patients compared to healthy individuals. Subedee et al reported PI values of 0.17±0.03 in cirrhotics versus 0.38±0.10 in controls, while Barakat et al found 0.23±0.08 in cirrhotics versus 0.39±0.10 in controls. Saha et al and Basappa et al observed a progressive reduction in PI with advancing Child-Pugh class highlighting the inverse relationship between PI and disease severity.[1,2,6,7]

In addition to PI, Doppler spectral analysis can detect complete spectral widening (CSW), a marker of disturbed portal venous hemodynamics. CSW reflects turbulent flow and loss of laminar characteristics associated with advanced fibrosis and elevated portal pressure.[1,2] Simultaneous evaluation of PI and CSW may improve the diagnostic sensitivity of Doppler ultrasonography for detecting cirrhosis and assessing disease severity.

The study aimed to determine whether portal vein PI and CSW, measured by Doppler ultrasound, can serve as non-invasive markers for assessing early hemodynamic changes and severity in patients with cirrhosis and portal hypertension, and to correlate these Doppler findings with Child-Pugh classifications.

Objective

To assess the role of Doppler study of the portal vein in evaluating the severity of liver disease in patients with cirrhosis and portal hypertension.

METHODS

This cross-sectional analytical study was conducted at the Department of Radiology and Imaging, Bangladesh Medical University (BMU), a 1,500-bed postgraduate teaching hospital in Dhaka, Bangladesh, between January 2024 and September 2025. A total of 60 participants were included, comprising 30 cirrhotic patients with portal hypertension and 30 non-cirrhotic healthy individuals. Participants were selected based on predefined inclusion and exclusion criteria to evaluate the role of Doppler ultrasonography of the portal vein in assessing portal vein pulsatility patterns and disease severity in liver cirrhosis.

Inclusion criteria

Diagnosis of Hepatitis B, Hepatitis C, or history of alcohol consumption, B-mode ultrasound showing a contracted, nodular liver with heterogeneous parenchyma, raised serum bilirubin, altered serum albumin, prolonged prothrombin time, or INR, presence or absence of ascites and esophageal varices, age 18–70 years, fasting for 6–8 hours prior to imaging

Exclusion criteria

Known hepatic encephalopathy, previous sclerotherapy or band ligation, portal vein thrombosis, portal vein flow reversal or bidirectional flow, uncooperative patients

Variables and measurements

Independent variables included demographic factors (age, sex), clinical features (jaundice, ascites, hematemesis, prothrombin time/INR), and laboratory/investigation data (serum bilirubin, serum albumin, prothrombin time, upper gastrointestinal endoscopy, and abdominal ultrasonography). Outcome variables were portal vein pulsatility index (PI), complete spectral widening (CSW), sensitivity, specificity, predictive values, likelihood ratios, and overall diagnostic accuracy.

Cirrhosis was defined as irreversible liver scarring due to chronic liver disease, and portal hypertension as a portal venous pressure gradient ≥6 mmHg. Disease severity was classified according to Child-Pugh criteria, incorporating bilirubin, albumin, prothrombin time/INR, ascites, and encephalopathy.

Doppler ultrasonography

All participants underwent Doppler ultrasonography (Esaote MyLab X8, 4–15 MHz vector transducer) after overnight fasting. Patients were examined in the supine position with ultrasound gel applied to the abdomen. The sample volume was aligned along the main portal vein at an angle of 60°. The portal vein pulsatility index (PI) was calculated as (Vmax−Vmin)/Vmax. Complete spectral widening (CSW) was recorded when the waveform lacked a clear baseline.

Data analysis

Data were analyzed using SPSS v23.0. Descriptive statistics were computed for all variables. Comparisons between groups were performed using unpaired t-tests, chi-square tests, and ANOVA where appropriate. Receiver operating characteristic (ROC) curve analysis was used to determine the optimal PI cut-off for differentiating cirrhotic patients from healthy controls.

Ethical considerations

Ethical approval was obtained from the BMU Institutional Review Board, and written informed consent was obtained from all participants. Study findings were used to non-invasively assess cirrhosis and portal hypertension severity, supporting clinical decision-making and patient monitoring.

RESULTS

Table 1 shows the age and sex distribution of the study population, comparing cirrhotic patients with portal hypertension (Group A, n=30) and non-cirrhotic healthy individuals (Group B, n=30). The mean age of Group A was 48.2±12.9 years, slightly higher than Group B (43.4±10.7 years), but the difference was not statistically significant (p=0.124). Male participants predominated in both groups (66.7% in Group A vs 60.0% in Group B), with no significant difference in sex distribution between groups (p=0.592).

Table 2 shows the presenting complaints and key laboratory findings of cirrhotic patients with portal hypertension (n=30). Ascites was the most frequent presenting complaint (23 patients, 76.7%), followed by hematemesis (9 patients, 30.0%) and jaundice (7 patients, 23.3%). Laboratory evaluation revealed hypoalbuminemia with a mean serum albumin of 3.04±0.65 g/dl (range: 2–4), mild hyperbilirubinemia with a mean serum bilirubin of 2.01±0.48 mg/dl (range: 0.90–2.50), and prolonged prothrombin time of 6.56 ± 2.54 seconds above control (range: 3–12), consistent with impaired hepatic synthetic function. Table 3 shows the distribution of cirrhotic patients (n=30) based on the Child-Pugh classification, which assesses the severity of liver disease. Among the participants, 9 patients (30.0%) were classified as Class A, 11 patients (36.7%) as Class B, and 10 patients (33.3%) as Class C.

Figure 1 shows the portal vein PI in cirrhotic patients with portal hypertension (Group A, n=30) and non-cirrhotic healthy individuals (Group B, n=30). The mean PI in Group A was 0.15±0.037 (range: 0.11–0.26), and in Group B it was 0.41±0.044 (range: 0.24–0.50). The difference between the groups was statistically significant (p<0.001).

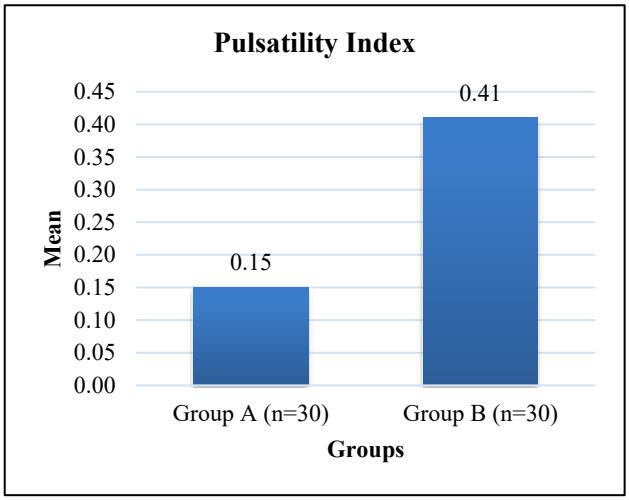

Figure 1: Comparison of portal vein pulsatility index between cirrhotic patients and healthy controls (n=60).

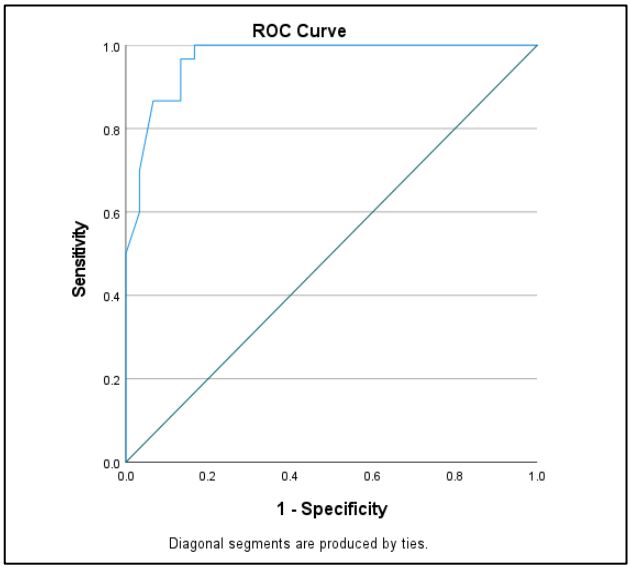

Figure 2: ROC curve for portal vein pulsatility index in differentiating cirrhotic patients from healthy controls.

Table 4 illustrates the portal vein PI across Child-Pugh classes in 30 cirrhotic patients. The mean PI was 0.19±0.03 in Class A (n=9), 0.15±0.02 in Class B (n=11), and 0.12±0.01 in Class C (n=10). The median PI values were 0.19, 0.15, and 0.12, respectively. The ranges were 0.17–0.26 for Class A, 0.12–0.19 for Class B, and 0.11–0.12 for Class C. The difference among the groups was statistically significant (p<0.001). Table 5 shows the distribution of ascites severity among 30 cirrhotic patients according to Child-Pugh classification. Mild ascites was present in 12 patients (40.0%), with 2 in Class A (22.2%), 8 in Class B (72.7%), and 2 in Class C (20.0%). Moderate ascites was observed in 11 patients (36.7%), with 0 in Class A, 3 in Class B (27.3%), and 8 in Class C (80.0%). No ascites was present in 7 patients (23.3%), all in Class A (77.8%). The difference in ascites severity across Child-Pugh classes was statistically significant (p<0.001). Table 6 shows the distribution of complete spectral widening (CSW) among 30 cirrhotic patients according to Child-Pugh classification. CSW was present in 24 patients (80.0%), including 4 in Class A (44.4%), 10 in Class B (90.9%), and 10 in Class C (100.0%). CSW was absent in 6 patients (20.0%), with 5 in Class A (55.6%) and 1 in Class B (9.1%). The difference in CSW presence across Child-Pugh classes was statistically significant (p=0.005).

Figure 2 shows the ROC curve for portal vein PI in differentiating cirrhotic patients with portal hypertension (n=30) from healthy controls (n=30). The area under the curve (AUC) was 0.968 (95% CI: 0.929–1.000), indicating excellent diagnostic performance. At the optimal cut-off value of 0.35, the sensitivity and specificity were 96.7% and 86.7%, respectively. The association between PI and group classification was statistically significant (p<0.001), demonstrating that PI is a highly accurate marker for identifying cirrhotic patients. Table 7 shows the diagnostic performance of portal vein PI with a cutoff value of <0.35 in 30 cirrhotic patients with portal hypertension and 30 healthy controls. PI<0.35 correctly identified 29 cirrhotic patients (true positives) and 26 healthy controls (true negatives), with 1 false negative and 4 false positives. The overall sensitivity was 96.67% (95% CI: 82.78–99.92%) and specificity was 86.67% (95% CI: 69.28–96.24%). Positive predictive value was 87.88% (95% CI: 74.39–94.76%) and negative predictive value was 96.30% (95% CI: 79.02–99.45%). The positive likelihood ratio was 7.25 (95% CI: 2.90–18.10) and the negative likelihood ratio was 0.04 (95% CI: 0.01–0.27). The overall accuracy of the test was 91.67% (95% CI: 81.61–97.24%). The association between PI values and group classification was statistically significant (p<0.001).

Table 1: Demographic characteristics of study participants (n=60).

Variable		Group A (n=30)	Group B (n=30)	P value
Age (in years)	20–29	4 (13.3%)	4 (13.3%)	0.124
	30–39	3 (10.0%)	8 (26.7%)	
	40–49	10 (33.3%)	9 (30.0%)	
	50–59	5 (16.7%)	7 (23.3%)	
	60–69	8 (26.7%)	2 (6.7%)	
	Mean±SD	48.2±12.9	43.4±10.7	
Sex	Male	20 (66.7%)	18 (60.0%)	0.592
	Female	10 (33.3%)	12 (40.0%)	
	Total	30 (100.0%)	30 (100.0%)	

Table 2: Clinical and laboratory profile of cirrhotic patients with portal hypertension (n=30).

Category	Parameter	Value
Presenting complaints	Jaundice	7 (23.3%)
	Ascites	23 (76.7%)
	Hematemesis	9 (30.0%)
Laboratory findings	Serum albumin (g/dl)	3.04±0.65 (2–4)
	Serum bilirubin (mg/dl)	2.01±0.48 (0.90–2.50)
	Prothrombin time (seconds above control)	6.56±2.54 (3–12)

Table 3: Distribution of cirrhotic patients according to child-pugh classification (N=30).

Child-Pugh classification	Number of patients (n=30)	%
Class A	9	30.0
Class B	11	36.7
Class C	10	33.3
Total	30	100.0

Table 4: Comparison of portal vein pulsatility index among Child-Pugh classes in cirrhotic patients (n=30).

Pulsatility index	Child Pugh class			P value
	Class A (n=9)	Class B (n=11)	Class C (n=10)	
Mean±SD	0.19±0.03	0.15±0.02	0.12±0.01	<0.001
Median	0.19	0.15	0.12	
Range (min–max)	0.17–0.26	0.12–0.19	0.11–0.12	

Table 5: Distribution of ascites severity according to Child-Pugh class in cirrhotic patients (n=30).

Ascites grade	Total	Child Pugh class			P value
		Class A (n=9)	Class B (n=11)	Class C (n=10)	
Mild	12 (40.0%)	2 (22.2%)	8 (72.7%)	2 (20.0%)	<0.001
Moderate	11 (36.7%)	0 (0.0%)	3 (27.3%)	8 (80.0%)	
Absent	7 (23.3%)	7 (77.8%)	0 (0.0%)	0 (0.0%)	
Total	30 (100.0%)	9 (100.0%)	11 (100.0%)	10 (100.0%)	

Table 6: Presence of complete spectral widening according to Child-Pugh class in cirrhotic patients (n=30).

CSW grade	Total	Child Pugh class			P value
		Class A (n=9)	Class B (n=11)	Class C (n=10)	
Present	24 (80.0%)	4 (44.4%)	10 (90.9%)	10 (100.0%)	0.005
Absent	6 (20.0%)	5 (55.6%)	1 (9.1%)	0 (0.0%)	
Total	30 (100.0%)	9 (100.0%)	11 (100.0%)	10 (100.0%)	

Table 7: Diagnostic performance of pulsatility index (<0.35) in differentiating cirrhotic patients with portal hypertension from healthy controls (n=60).

Pulsatility index	Groups		Total (n=60)	P value
	Cirrhotic patients with portal hypertension	Healthy control		
<0.35	29 (TP)	4 (FP)	33	<0.001
>0.35	1(FN)	26 (TN)	27	
Total	30	30	60	
Diagnostic validity test		**Values**	**95% CI**	
Sensitivity		96.67%	82.78 to 99.92	
Specificity		86.67%	69.28 to 96.24	
Positive likelihood ratio		7.25	2.90 to 18.10	
Negative likelihood ratio		0.04	0.01 to 0.27	
Positive predictive value		87.88%	74.39 to 94.76	
Negative predictive value		96.30%	79.02 to 99.45	
Accuracy		91.67%	81.61 to 97.24	

DISCUSSION

This cross-sectional analytical study was conducted at BMU between January 2024 and September 2025 to evaluate the utility of Doppler study of the portal vein in assessing portal vein pulsatility patterns for staging the severity of liver cirrhosis with portal hypertension. A total of 60 participants were enrolled, including 30 clinically diagnosed cirrhotic patients with portal hypertension and 30 non-cirrhotic healthy individuals. In this study, the mean age of cirrhotic patients was 48.2±12.9 years, while that of the non-cirrhotic group was 43.4±10.7 years, with no statistically significant difference between the groups (p=0.124). Similar age distributions have been reported in previous studies. Subedee et al reported a mean age of 49.6±14.7 years among cirrhotic patients1, and Saha et al. documented a mean age of 47.9±11.5 years.[6] These findings suggest that cirrhosis primarily affects middle-aged to elderly individuals, consistent with the chronic progressive nature of liver disease. In the present study, males constituted 66.7% of the cirrhotic group and 60.0% of the non-cirrhotic group, with no significant difference between the groups (p=0.592). Male predominance among cirrhotic patients has been consistently reported in previous studies. Syed et al. reported 62% male patients, Subedee et al reported 63.46% males1, Afif et al reported 67.9% and Saha et al reported 63.6% male

patients6.[1,5,6,11] These findings suggest that males are more frequently affected by liver cirrhosis, which may relate to higher rates of alcohol consumption, viral hepatitis exposure, and other risk factors commonly observed among males. Ascites was the most frequent presenting complaint in this study (76.7%), followed by hematemesis (30.0%) and jaundice (23.3%). These findings are consistent with those of Basappa et al who reported ascites in 75% of cirrhotic patients.[7] A similar clinical spectrum was described by Chaurasiya et al who observed that ascites, jaundice, and gastrointestinal bleeding were among the most common presenting features in cirrhotic patients with portal hypertension reflecting decompensated disease.[12] Mital et al also observed ascites and encephalopathy as frequent complications, particularly in patients with advanced liver disease.[3] Likewise, Nouh et al emphasized that gastrointestinal bleeding secondary to varices and the presence of ascites remain hallmark manifestations of portal hypertension in cirrhosis.[4]

Cirrhotic patients in this study also exhibited characteristic laboratory abnormalities. The mean serum albumin level was 3.04±0.65 g/dl, indicating hypoalbuminemia, while the mean serum bilirubin was 2.01±0.48 mg/dl, reflecting mild hyperbilirubinemia; prothrombin time was prolonged by 6.56±2.54 seconds above control. These findings are in agreement with Joshi et al who reported that the majority of cirrhotic patients with portal hypertension had serum albumin levels below 3.5 g/dl, along with elevated bilirubin and prolonged coagulation parameters, reflecting impaired hepatic synthetic function.[13]

Similar laboratory trends were documented by Mital et al who noted progressive hypoalbuminemia, hyperbilirubinemia, and worsening coagulopathy with advancing Child–Pugh stages.[3] Basappa et al reported comparable derangements in liver function parameters, and Nouh et al also observed significant abnormalities in biochemical liver tests among cirrhotic patients with portal hypertension.[4,7] Collectively, these findings indicate progressive deterioration of hepatic synthetic and excretory capacity with advancing liver disease, corroborating the laboratory profile observed in the present study.

The distribution of cirrhotic patients according to Child-Pugh classification in this study revealed 36.7% in Class B, 33.3% in Class C, and 30.0% in Class A, indicating a broad spectrum of disease severity with a slight predominance of moderate to advanced stages. Similar distributions have been reported previously. Mital et al observed 42% in Class C3, Basappa et al reported 46.7% in Class C7, and Nouh et al documented representation across all Child-Pugh classes.[4] These findings highlight the reproducibility of Child-Pugh classification as a reliable tool for stratifying cirrhosis severity. The portal vein PI was significantly lower in cirrhotic patients (0.15±0.037) compared to healthy controls (0.41±0.044, $p<0.001$). The decline in PI reflects progressive hepatic fibrosis, increased intrahepatic vascular resistance, and reduced hepatic parenchymal compliance. Subedee et al reported a mean PI of 0.17±0.03 in cirrhotics and 0.38±0.10 in controls, while Saha et al reported 0.18±0.08 in cirrhotics and 0.32±0.09 in controls. Barakat et al observed 0.23±0.08 in cirrhotics and 0.39±0.10 in healthy controls.[1,2,6]

Despite minor variations, all studies consistently report a lower PI in cirrhotic patients, supporting its role as a sensitive, non-invasive marker of altered portal hemodynamics. PI progressively decreased with advancing Child-Pugh class: 0.19±0.03 in Class A, 0.15±0.02 in Class B, and 0.12±0.01 in Class C. Subedee et al reported 0.215±0.025 in Class A, 0.181±0.023 in Class B, and 0.149±0.030 in Class C1. Barakat et al found 0.25±0.08, 0.23±0.08, and 0.21±0.07, respectively.[2] Basappa et al reported 0.36±0.06, 0.29±0.07, and 0.20±0.047, and Saha et al observed 0.28±0.11, 0.20±0.03, and 0.14±0.056. These consistent patterns confirm an inverse correlation between PI and disease severity.

In this study, 40.0% of patients presented with mild ascites, 36.7% with moderate ascites, and 23.3% had no ascites detectable at the time of assessment. The relatively high prevalence of ascites may reflect that many patients were in advanced stages of cirrhosis at enrollment. Hussain et al reported a significant association between the severity of liver disease (Child Pugh class) and clinical manifestations of portal hypertension, noting that decompensation features such as ascites tend to increase with worsening Child Pugh class, with the highest frequency observed in Class C patients, demonstrating similar trends to our cohort.[14]

Similarly, Tsoris et al highlighted that ascites severity is an integral component of the Child Pugh scoring system, increasing progressively as liver function deteriorates, which explains why more advanced classes exhibited higher grades of ascites while early-stage patients often had absent fluid accumulation.[15] Subedee et al reported a progressive increase in ascites frequency with advancing Child–Pugh class, occurring in 11.1% of Child A, 66.7% of Child B, and 100% of Child C patients1. Mital et al. documented ascites in 74.4% of cirrhotic patients, predominantly in decompensated stage, and Basappa et al reported ascites in 75% of cases.[3,7] Despite minor variations in prevalence across studies, the consistently high occurrence of ascites in advanced cirrhosis underscores its clinical importance as a key indicator of portal hypertension and hepatic decompensation. CSW was observed in 80.0% of cirrhotic patients, including 44.4% of Class A, 90.9% of Class B, and 100% of Class C patients, and was absent in healthy controls. Spectral widening reflects disrupted laminar flow due to architectural distortion, increased portal pressure, and altered vascular resistance. Subedee et al documented CSW in 76.92% of cirrhotics and none in controls, with a

stepwise increase across Child Pugh classes (28.5% in A, 66.6% in B, 100% in C). Saha et al reported CSW in 60.6% of cirrhotic patients.[1,6] Basappa et al demonstrated increasing spectral width index with advancing Child Pugh class (0.82±0.09 in A, 0.83±0.14 in B, 0.92±0.07 in C), confirming the correlation between spectral widening and disease severity.[7]

These observations are further supported by Rakheeb et al who reported progressive loss of the normal triphasic hepatic vein waveform and spectral broadening in cirrhotic patients, with higher Child Pugh classes and the presence of varices strongly associated with abnormal Doppler patterns.[16] Similarly, Khan et al demonstrated that abnormal hepatic vein waveforms, including spectral widening, were significantly more frequent in Child Pugh B and C patients compared to Class A, reinforcing that CSW is a sensitive non-invasive marker of hepatic dysfunction and portal hypertension severity.[17]

ROC curve analysis demonstrated excellent diagnostic performance of PI for differentiating cirrhotics from controls, with an AUC of 0.968. At a cutoff of 0.35, sensitivity was 96.7%, specificity 86.7%, PPV 87.88%, NPV 96.3%, positive likelihood ratio 7.25, negative likelihood ratio 0.04, and accuracy 91.67%. Previous studies did not always report ROC analysis for PI specifically. Iwao et al. reported a hepatic artery PI cutoff of 1.1 with 85% sensitivity and 81% specificity.[18] Other Doppler indices have shown AUC values between 0.88 and 0.99. This study provides one of the few ROC-based evaluations specifically for portal vein PI, supporting its diagnostic utility in cirrhosis and portal hypertension.

Limitations

The study had several limitations.

Single-center study

The study was conducted at a single tertiary care center, which may limit the generalizability of the findings to other populations or healthcare settings.

Small sample size

The relatively small sample size (30 patients and 30 controls) may reduce the statistical power to detect subtle differences, especially in subgroup analyses.

Operator dependency of doppler ultrasound

Doppler ultrasonography is highly operator-dependent, and minor variations in technique could affect PI and spectral widening measurements.

Lack of invasive hemodynamic correlation

The study did not include invasive measurements (such as hepatic venous pressure gradient, HVPG) to directly validate Doppler findings against gold-standard portal pressure assessments.

Exclusion of other etiologies and comorbidities

Patients with complicating factors such as portal vein thrombosis, previous interventions, or severe encephalopathy were excluded, which may limit applicability to more complex cirrhosis cases.

Limited pediatric and early-stage representation

The study population mostly included adult cirrhotic patients, with limited representation of pediatric patients or very early-stage compensated cirrhosis.

CONCLUSION

In conclusion, this study shows that portal vein PI, measured by Doppler ultrasonography, is significantly lower in cirrhotic patients compared to healthy individuals and progressively decreases with advancing Child-Pugh class. The presence of CSW was also closely associated with cirrhosis and disease severity. ROC curve analysis confirmed the excellent diagnostic performance of PI, with high sensitivity and specificity. These findings support the use of PI and CSW as reliable, non-invasive Doppler markers for the detection, staging, and monitoring of cirrhosis and portal hypertension. Incorporating these Doppler parameters into routine clinical evaluation may enhance non-invasive assessment of liver disease and reduce reliance on invasive procedures.

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