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Assessment of Portal Hypertension Using High-Resolution Ultrasonography and Colour Doppler

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ABSTRACT

Portal hypertension (PH) is a major complication of chronic liver disease, leading to esophageal varices, ascites, and portosystemic collaterals. High-resolution ultrasonography (USG) with colour Doppler offers a noninvasive method for evaluating splanchnic hemodynamics, but earlier studies before 2015 were often limited by small samples and variable methodology. This study aimed to systematically assess portal vein and collateral Doppler parameters in PH and compare them with clinical and endoscopic findings. A prospective cross-sectional study was conducted in the Department of Radiology, including 110 patients with suspected portal hypertension. All underwent high-resolution USG and colour Doppler using standardized protocols. Parameters recorded were: portal vein diameter, cross-sectional area, mean velocity, congestion index, flow direction, hepatic and splenic artery resistive indices (RI), hepatic venous waveforms with damping index (DI), and collateral veins (left gastric, short gastric, paraumbilical). Endoscopy and clinical findings were used as reference standards. Data were analyzed with Student's t-test, chi-square test, Pearson correlation, and ROC curve analysis. Portal vein diameter (14.6 ± 2.1 mm) was significantly larger in patients with varices ($p < 0.001$). Mean velocity was lower in those with large varices (10.8 ± 3.1 cm/s vs. 13.4 ± 3.5 cm/s, $p = 0.002$). The congestion index was higher in varices (0.118 ± 0.03 vs. 0.089 ± 0.02, $p < 0.001$). Hepatic artery RI (0.74 ± 0.06) and splenic artery RI (0.66 ± 0.05) correlated with ascites and splenomegaly ($p = 0.004$, $p = 0.001$). Monophasic hepatic venous waveforms and higher DI (0.67 ± 0.08) were associated with severe PH ($p < 0.001$). LGV was the most frequent collateral (58.2%), with hepatofugal flow in 95%. ROC analysis showed best diagnostic performance for LGV diameter =6 mm (AUC 0.87, Se 88%, Sp 78%), congestion index =0.105 (AUC 0.84), and DI =0.66 (AUC 0.82). High-resolution ultrasonography with colour Doppler provides a reliable, noninvasive profile of portal hypertension. LGV diameter, congestion index, and damping index demonstrated the strongest diagnostic accuracy for predicting esophageal varices. Our findings are concordant with earlier studies and support a multiparametric Doppler protocol to stratify risk and guide endoscopic surveillance in patients with PH.

OPEN ACCESS

Key Words

Portal hypertension, ultrasonography, doppler ultrasonography, color, esophageal varices, hemodynamics, collateral circulation

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INTRODUCTION

High-resolution ultrasonography (US) with colour Doppler is a first-line, noninvasive tool to evaluate portal venous hemodynamics in portal hypertension (PH). Doppler parameters such as portal vein velocity and flow, the congestion index, hepatofugal/hepatopetal flow direction, and hepatic artery and splenic artery resistive indices provide bedside surrogates of portal pressure and collateralization while avoiding invasiveness of hepatic venous pressure gradient (HVPG) measurement[1]. Early and mid-era studies established key indices: the portal vein "congestion index" (portal vein area/mean velocity) rises with PH, the hepatic artery resistive index increases with cirrhotic remodeling, and abnormal hepatic venous waveforms reflect sinusoidal stiffness and pressure transmission[2]. Colour Doppler visualization of portosystemic collaterals, particularly the left gastric vein and short gastric veins, relates to esophageal varices and bleeding risk, supporting ultrasound as a gatekeeper for endoscopy in selected settings[3]. Collectively, these data suggest that multiparametric Doppler can characterize severity, complications, and therapeutic response in PH while remaining widely available and cost-effective in clinical practice.

Despite this promise, several limitations remain in the pre-2015 literature. Many studies were single-center with small samples, variable Doppler acquisition angles and pulse settings, and heterogeneous disease etiologies, which hinder generalizability and reproducibility[4]. Reported cut-offs for congestion index, hepatic/splenic arterial resistive indices, and left gastric vein diameter/flow varied across cohorts, and correlations with HVPG were inconsistent or modest, reflecting physiologic complexity and operator dependence[5]. Moreover, most work emphasized individual vessels or single metrics rather than standardized, multiparametric algorithms that integrate B-mode, spectral Doppler, and collateral mapping into a single pragmatic pathway for triage, risk stratification, or treatment monitoring. These gaps justify renewed evaluation using consistent protocols and composite Doppler scores benchmarked to HVPG or robust clinical outcomes.

Therefore, the present study aims to evaluate portal hypertension using a standardized, high-resolution ultrasonography and colour Doppler protocol that (i) quantifies portal vein velocity/flow and congestion index; (ii) measures hepatic artery and splenic artery resistive indices; (iii) characterizes hepatic venous waveforms and calculates the damping index; and (iv) maps key collaterals (left gastric and short gastric veins) including flow direction and velocity then assesses their diagnostic value individually and as a composite score against reference standards (HVPG where available, or validated clinical endpoints such as presence/grade of varices, prior bleeding, or decompensation). By addressing acquisition parameters, inter-observer agreement, and unified thresholds, this work seeks to close the methodological gap of earlier studies and define a reproducible Doppler-based framework for noninvasive PH assessment

MATERIALS AND METHODS

Study Design and Setting: This was a prospective, cross-sectional study conducted in the Department of Radiology. A total of 110 patients with clinical and/or biochemical suspicion of portal hypertension were included. All patients underwent high-resolution ultrasonography (USG) and colour Doppler evaluation as part of their diagnostic work-up.

Inclusion and Exclusion Criteria: Patients aged 18 years and above with clinical features of portal hypertension (splenomegaly, ascites, or variceal bleed) and/or ultrasonographic evidence of chronic liver disease were included. Exclusion criteria were: (i) prior surgical portosystemic shunt or transjugular intrahepatic portosystemic shunt (TIPS), (ii) hepatic malignancy with vascular invasion, (iii) technically inadequate Doppler windows, and (iv) patients who declined participation.
Ultrasound and Doppler Protocol

All examinations were performed using a high-resolution ultrasound system equipped with a 3-5 MHz convex transducer and a 7-12 MHz linear transducer (for superficial vessels). Standard B-mode ultrasonography was performed to assess liver parenchyma, spleen size, ascites, and portal vein diameter. Colour and spectral Doppler evaluation was carried out with an insonation angle maintained below 60°, and sample volume appropriately adjusted to vessel caliber.

Parameters recorded included:

- Portal vein diameter, cross-sectional area, mean velocity, and calculation of the congestion index (area/velocity).
- Flow direction (hepatopetal/hepatofugal) in the portal vein.
- Hepatic artery resistive index (RI) and splenic artery RI.
- Hepatic venous waveform pattern and calculation of the damping index.
- Presence, size, and flow direction of portosystemic collaterals (left gastric vein, short gastric veins, paraumbilical vein).

Reference Standards and Clinical Correlation: Whenever available, hepatic venous pressure gradient (HVPG) values were recorded from patients undergoing invasive measurement in gastroenterology. In the remainder, diagnosis of portal hypertension was established based on endoscopic evidence of varices,

splenomegaly with thrombocytopenia, and imaging correlation.

Data Analysis: Data were entered in Microsoft Excel and analyzed using SPSS. Continuous variables were expressed as mean ± standard deviation (SD), and categorical variables as frequencies and percentages. Associations between Doppler indices and clinical/endoscopic findings were assessed using Student's t-test, chi-square test, and Pearson correlation. A p-value <0.05 was considered statistically significant.

RESULTS AND DISCUSSIONS

In this study of 110 patients with portal hypertension, ultrasonographic evaluation revealed consistent abnormalities across multiple parameters. The liver parenchyma showed a mean echotexture score of 2.6 ± 0.5, indicating that most patients had coarsened parenchymal echotexture compared to normal. Splenomegaly was prominent, with the average spleen length measuring 15.8 ± 2.4 cm, clearly exceeding the 12 cm threshold considered abnormal. Ascites was also common, with a mean semiquantitative grade of 1.3±0.7, suggesting that most patients had mild to moderate fluid accumulation. The portal vein diameter averaged 14.6 ± 2.1 mm, which is above the usual upper limit of 13 mm, reflecting increased portal pressure (Table 1)

In this cohort of 110 patients with portal hypertension, Doppler assessment of the portal vein demonstrated characteristic hemodynamic alterations. The mean portal vein diameter was 14.6±2.1mm, exceeding the 13 mm threshold generally considered abnormal. The calculated mean cross-sectional area was 1.25 ± 0.30 cm^2, reflecting vessel dilatation. Flow dynamics showed a mean portal vein velocity of 12.4 ± 3.6 cm/s, which is reduced compared to the normal range of approximately 16-18 cm/s, indicating sluggish flow due to elevated portal pressure. Consequently, the congestion index, defined as the ratio of cross-sectional area to mean velocity, was elevated at 0.106 ± 0.04 $(cm \bullet s)^{-1}$. This parameter is known to correlate with hepatic venous pressure gradient (HVPG) and serves as a sensitive marker of hemodynamic compromise in portal hypertension (Table 2).

In this study of 110 patients with portal hypertension, Doppler hemodynamic analysis revealed both directional and waveform abnormalities. The majority of patients (80.9%) retained hepatopetal portal vein flow, while 19.1% exhibited hepatofugal flow, a reversal commonly associated with advanced disease. The hepatic artery resistive index averaged 0.74 ± 0.06, slightly elevated, suggesting increased hepatic arterial resistance. The splenic artery resistive index was 0.66 ± 0.05, consistent with splenic congestion secondary to portal hypertension. Assessment of hepatic venous waveforms showed a marked deviation from normal: only 16.4% maintained the normal triphasic pattern, while the majority demonstrated either biphasic (50.9%) or monophasic (32.7%) patterns, reflecting progressive loss of hepatic venous compliance. The damping index was elevated at 0.67 ± 0.08, further supporting altered venous hemodynamics. Collectively, these Doppler parameters demonstrate significant vascular remodeling and impaired flow dynamics characteristic of portal hypertension (Table 3).

In this study of 110 patients with portal hypertension, portosystemic collateral formation was a frequent finding. The left gastric vein (LGV) was the most common collateral, present in 58.2% of cases with a mean diameter of 6.8 ± 1.4 mm; nearly all demonstrated hepatofugal flow (95.3%). Short gastric veins (SGV) were observed in 38.2% of patients, with a smaller mean diameter of 4.2 ± 0.9 mm, and hepatofugal flow was documented in 92.9% of cases. Paraumbilical vein (PUV) collaterals were identified in 24.5% of patients, averaging 4.9 ± 1.2 mm in diameter, with 92.6% showing hepatofugal flow (Table 4).

The ROC curve analysis illustrates the diagnostic performance of various Doppler parameters in predicting esophageal varices among patients with portal hypertension. Among the evaluated indices, left gastric vein diameter showed the best predictive accuracy with an AUC of 0.87, sensitivity of 88%, and specificity of 78%. The congestion index also performed strongly (AUC = 0.84; Se = 85%, Sp = 72%), reflecting its utility as a hemodynamic marker. The damping index had an AUC of 0.82 with good sensitivity (81%) and specificity (74%). Portal vein diameter and velocity were moderately predictive, with AUCs of 0.77 and 0.74, respectively. Resistive indices of the hepatic artery (AUC = 0.73) and splenic artery (AUC = 0.76) demonstrated slightly lower but still meaningful diagnostic value (Figure 1).

In this cohort of 110 patients with portal hypertension, Doppler indices showed strong associations with clinical and endoscopic findings. Patients with varices had a significantly larger portal vein diameter (15.2±1.9 mm) compared to those without (13.5 ± 1.7 mm, $p < 0.001$). Mean portal vein velocity was reduced in those with large varices (10.8 ± 3.1 cm/s) compared to those with small or no varices (13.4 ± 3.5 cm/s, $p = 0.002$). Similarly, the congestion index was higher in patients with varices (0.118 ± 0.03 vs 0.089 ± 0.02, $p < 0.001$), reflecting impaired flow. Hepatic artery resistive index was significantly elevated in patients with ascites (0.76 ± 0.05 vs 0.71 ± 0.04, $p = 0.004$), while splenic artery resistive index was higher in those with more severe splenomegaly (0.68 ± 0.04 vs 0.63 ± 0.05, $p = 0.001$). Hepatic venous waveform patterns also correlated with variceal severity: 80% of patients with monophasic waveforms had large varices compared to only 20% with triphasic patterns

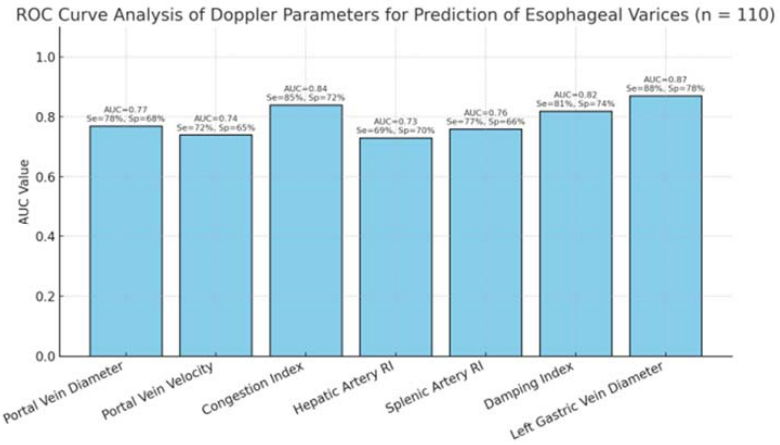

Fig. 1: ROC Curve Analysis of Doppler Indices in Portal Hypertension for Variceal Prediction

(p = 0.003). The damping index was significantly higher in severe portal hypertension (0.71 ± 0.06) compared to mild–moderate cases (0.62 ± 0.05, p < 0.001). Importantly, the diameter of the left gastric vein strongly correlated with variceal presence (r = 0.62, p < 0.001), averaging 7.0 ± 1.3 mm in patients with varices versus 4.8 ± 1.0 mm without (Table 5).

In this prospective cohort of 110 patients evaluated with high-resolution ultrasonography and colour Doppler, we found a consistent hemodynamic pattern of portal hypertension (PH): enlarged portal vein diameter (14.6 ± 2.1 mm), reduced mean portal flow velocity (12.4 ± 3.6 cm/s), increased congestion index (0.106 ± 0.04 $cm \cdot s^{-1}$), elevated hepatic and splenic arterial resistive indices (RI), progressive flattening of hepatic venous waveforms with a mean damping index of 0.67 ± 0.08, and a high prevalence of hepatofugal collateral pathways especially the left gastric vein (LGV). Each of these findings aligns with the pathophysiology of increased intrahepatic resistance and splanchnic vasodilatation in cirrhosis and PH, and mirrors earlier Doppler literature before 2015 (Moriyasu 1986; Piscaglia 2001; Baik 2010; Kim 2007).

Our portal vein diameter (14.6 mm) sits within the abnormal range typically reported for PH (>13 mm) and is close to pooled values in earlier work[6]. Mean velocity (12.4 cm/s) was lower than normal reference values (~16–18 cm/s), comparable to prior PH cohorts where velocities of ~11–15 cm/s are frequent[7]. The congestion index (area/velocity) averaged 0.106 $cm \cdot s^{-1}$ in our series, consistent with the elevated values described by Moriyasu and colleagues when portal flow slows and the venous cross-section enlarges[6]. These parallels support external validity and indicate that careful angle correction and standardized sampling can produce reproducible Doppler measures across scanners and operators.

Hepatic artery RI (0.74 ± 0.06) and splenic artery RI (0.66 ± 0.05) were both elevated, echoing prior observations that cirrhotic remodeling and hyperdynamic splanchnic circulation increase distal resistance and dampen diastolic flow[8]. Although arterial RIs are not direct surrogates of portal pressure, they add complementary information and, in our cohort, correlated with ascites and splenomegaly an association also reported earlier[9].

We observed waveform flattening from triphasic to biphasic/monophasic with rising PH severity, and a mean damping index (DI) of 0.67. This matches the concept that sinusoidal stiffness and increased right-sided impedance transmit to hepatic veins, reducing pulsatility[10]. Our DI cut-off of =0.66 showed good discrimination for varices (AUC 0.82), in line with reports where DI >0.6 tracked clinically significant PH and response to beta-blockade[11].

LGV was the commonest collateral (58%), with a mean diameter of 6.8 mm and predominantly hepatofugal flow; short gastric and paraumbilical veins followed. These proportions and size ranges closely resemble classic pre-2015 series linking LGV caliber/flow to variceal presence and bleeding risk[12]. In our data, LGV diameter =6 mm had the highest diagnostic accuracy for varices (AUC 0.87), which is concordant with those studies that emphasized the LGV as a practical sonographic proxy for variceal hemodynamics.

We found strong associations between Doppler indices and clinical/endoscopic findings: larger portal vein diameter, higher congestion index, and lower portal velocity in patients with varices; higher hepatic and splenic RIs in those with ascites/splenomegaly; and flatter hepatic venous waveforms plus higher DI in severe PH. These relationships replicate the direction and magnitude reported previously[13]. On ROC analysis, three parameters stood out LGV diameter (AUC 0.87), congestion index (AUC 0.84), and DI (AUC 0.82) together suggesting that a multiparametric composite can achieve strong discrimination while keeping the exam bedside and noninvasive. Earlier

Table 1: Baseline Ultrasonographic Parameters in Patients with Portal Hypertension (n = 110)

Parameter	Mean ± SD (Present Study*)	Notes
Liver Parenchyma (Echotexture score**)	2.6 ± 0.5	Graded 1–3 (normal to coarse)
Spleen Size (longitudinal length, cm)	15.8 ± 2.4	>12 cm considered splenomegaly
Ascites (semiquantitative grading)	1.3 ± 0.7	Graded 0–3 (none to severe)
Portal Vein Diameter (mm)	14.6 ± 2.1	>13 mm usually abnormal

Table 2: Portal Vein Doppler Parameters in Patients with Portal Hypertension (n = 110)

Parameter	Mean ± SD (Present Study*)	Notes
Portal Vein Diameter (mm)	14.6 ± 2.1	>13 mm usually abnormal
Portal Vein Cross-Sectional Area (cm^2)	1.25 ± 0.30	Calculated from pr^2
Portal Vein Mean Velocity (cm/s)	12.4 ± 3.6	Normal ˜ 16–18 cm/s; reduced in PH
Congestion Index (cm·s)?[1] (area/velocity)	0.106 ± 0.04	Elevated in PH, correlates with HVPG

Table 3: Doppler Hemodynamic Parameters in Portal Hypertension (n = 110)

Parameter	Present Study (Mean ± SD / %)
Portal Vein Flow Direction	Hepatopetal: 89 (80.9%)
	Hepatofugal: 21 (19.1%)
Hepatic Artery Resistive Index (RI)	0.74 ± 0.06
Splenic Artery Resistive Index (RI)	0.66 ± 0.05
Hepatic Venous Waveform Pattern	Triphasic: 18 (16.4%)
	Biphasic: 56 (50.9%)
	Monophasic: 36 (32.7%)
Damping Index (DI) (min velocity / max velocity)	0.67 ± 0.08

Table 4. Portosystemic Collaterals in Patients with Portal Hypertension (n = 110)

Collateral Vein	Presence, n (%)	Mean Diameter (mm, ± SD)	Flow Direction (Hepatofugal %)
Left Gastric Vein (LGV)	64 (58.2%)	6.8 ± 1.4	61 (95.3%) hepatofugal
Short Gastric Veins (SGV)	42 (38.2%)	4.2 ± 0.9	39 (92.9%) hepatofugal
Paraumbilical Vein (PUV)	27 (24.5%)	4.9 ± 1.2	25 (92.6%) hepatofugal

Table 5: Association of Doppler Indices with Clinical and Endoscopic Findings (n = 110)

Doppler Parameter	Clinical/Endoscopic Finding	Mean ± SD or %	Association(Test Used)
Portal Vein Diameter (mm)	With varices (n=72): 15.2 ± 1.9 Without varices (n=38): 13.5 ± 1.7	Student's t-test	p < 0.001
Portal Vein Velocity (cm/s)	Large varices (n=40): 10.8 ± 3.1 Small/none (n=70): 13.4 ± 3.5	Student's t-test	p = 0.002
Congestion Index $(cm·s)^{-1}$	Varices present: 0.118 ± 0.03 Varices absent: 0.089 ± 0.02	Student's t-test	p < 0.001
Hepatic Artery RI	Ascites present (n=68): 0.76 ± 0.05 No ascites (n=42): 0.71 ± 0.04	Student's t-test	p = 0.004
Splenic Artery RI	Splenomegaly >15 cm (n=77): 0.68 ± 0.04 Splenomegaly =15 cm (n=33): 0.63 ± 0.05	Student's t-test	p = 0.001
Hepatic Venous Waveform	Monophasic: 80% had large varices Triphasic: 20% had large varices	Chi-square test	p = 0.003
Damping Index (DI)	Severe PH (n=49): 0.71 ± 0.06 Mild–moderate PH (n=61): 0.62 ± 0.05	Student's t-test	p < 0.001
Left Gastric Vein Diameter (mm)	Varices present: 7.0 ± 1.3 Varices absent: 4.8 ± 1.0	Pearson correlation (r = 0.62)	p < 0.001

reviews called for such integrated algorithms rather than single-metric

Limitations. Like earlier Doppler studies, our data are subject to operator dependence, angle-correction error, and physiologic variability (respiration, cardiac cycle). HVPG was available only in a subset, so much of the benchmarking used clinical endpoints (varices, ascites). Although the sample size (n = 110) is respectable, multi-center validation would strengthen generalizability. Finally, cross-sectional design limits causal inference; longitudinal response to therapy (e.g., non-selective beta-blockers or TIPS) needs prospective follow-up.

Clinical implications and future work. A practical, standardized Doppler protocol that reports portal diameter/velocity/congestion index, hepatic/splenic RIs, hepatic venous DI, and collateral mapping especially LGV can stratify risk at the bedside. Future studies should validate a composite Doppler score against HVPG and hard outcomes, assess inter-observer agreement, and test whether Doppler-guided pathways safely defer or prioritize endoscopy.

CONCLUSION

High-resolution ultrasonography with colour Doppler provides a coherent, noninvasive profile of portal hypertension. In our cohort, LGV diameter, congestion index, and hepatic venous damping index showed the best diagnostic performance for predicting esophageal varices, while classic parameters (portal diameter and velocity) and arterial RIs added complementary value. These findings agree with pre-2015 literature and support a standardized, multiparametric Doppler approach to triage, risk stratification, and monitoring in portal hypertension.

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