

Role of Color Doppler Ultrasonography in the Assessment of Portal Hypertension: A Cross-Sectional Study

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DOI: <https://doi.org/10.5281/zenodo.23006921>

Received: 31 August 2026, Revised: 13 September, Accepted: 22 September 2026, Online First: 29 September 2026

ABSTRACT

Background: Portal hypertension is a major consequence of chronic liver disease and is associated with significant morbidity due to ascites, variceal formation, thrombosis, and other vascular complications. Color Doppler ultrasonography provides a non-invasive method for assessing both morphological changes and hemodynamic alterations in the portal venous system. This study evaluated the spectrum of gray-scale and Doppler findings in portal hypertension and assessed the utility of Doppler parameters in differentiating compensated from decompensated cirrhosis. **Methods:** This cross-sectional study was conducted over 18 months from June 2024 to November 2025 in 50 patients with clinically suspected portal hypertension. All patients underwent abdominal ultrasonography with color and spectral Doppler assessment. Portal vein diameter, respiratory variation, splenic size, ascites, portal venous flow pattern, vascular thrombosis, portosystemic collaterals, portal vein peak systolic velocity (PSV), and pulsatility index (PI) were evaluated. Doppler and gray-scale findings were compared between compensated and decompensated cirrhosis. **Results:** The study population comprised 37 males (74%) and 13 females (26%). Cirrhosis was the predominant etiology, accounting for 70% of cases. Portal vein diameter was greater than 13 mm in 60% of patients, while 80% demonstrated less than 20% respiratory variation. Splenomegaly and ascites were present in 78% and 86% of patients, respectively. Hepatopetal portal flow was observed in 74%, hepatofugal flow in 6%, to-and-fro flow in 2%, and absent flow in 18%. Portal vein thrombosis was identified in 28% of patients. Splenorenal collaterals were the most frequent collateral pathway (76%). Mean portal vein PSV and PI were 18.6 ± 5.4 cm/s and 0.42 ± 0.15 , respectively. Compared with compensated cirrhosis, decompensated cirrhosis showed significantly lower PSV (16.6 ± 4.5 vs. 22.4 ± 4.2 cm/s, $p < 0.001$) and PI (0.37 ± 0.14 vs. 0.51 ± 0.13 , $p = 0.006$), along with greater portal vein diameter (14.2 ± 2.1 vs. 12.8 ± 1.4 mm, $p = 0.028$). Splenomegaly, ascites, and portosystemic collaterals were also significantly more frequent in decompensated patients. **Conclusion:** Color Doppler ultrasonography provides comprehensive non-invasive assessment of portal hypertension by demonstrating structural abnormalities, altered portal venous hemodynamics, vascular complications, and collateral pathways. Portal vein PSV and PI, together with gray-scale findings, showed significant differences between compensated and decompensated cirrhosis. Combined gray-scale, color Doppler, and spectral Doppler assessment may therefore aid in evaluating disease severity and identifying advanced portal hypertensive changes.

Keywords: Portal hypertension; Color Doppler ultrasonography; Portal vein; Portal venous flow; Cirrhosis

INTRODUCTION

Portal hypertension is one of the most important causes of morbidity and mortality in cirrhotic patients. [1] Portal hypertension is an important complication of chronic liver disease and is characterized by an increase in portal venous pressure. [2] A hepatic venous pressure gradient (HVPG) greater than 5 mmHg indicates portal hypertension, while clinically significant portal hypertension is defined by an HVPG greater than 10 mmHg. Variceal bleeding may occur when the gradient exceeds 12 mmHg, making early recognition important for prognosis and management [3,4]. The increasing global burden of chronic liver disease, with major contributions from obesity, viral hepatitis, and alcohol-related liver disease, has further increased the clinical importance of portal hypertension [5].

The portal vein is formed by the union of the superior mesenteric and splenic veins and subsequently divides into right and left portal veins within the liver. Normally, portal venous pressure is approximately 1-4 mmHg higher than hepatic venous pressure, maintaining hepatopetal blood flow through the liver. Increased resistance to portal venous flow, most commonly due to cirrhosis, results in elevated portal pressure. Depending on the site of increased resistance, portal hypertension may be classified as presinusoidal, sinusoidal, or postsinusoidal [6,7].

Direct measurement of portal pressure is considered the gold standard for assessment of portal hypertension; however, it is invasive and not routinely suitable for screening or serial assessment. Ultrasonography provides a widely available, non-invasive and cost-effective alternative. Conventional B-mode ultrasonography permits assessment of hepatic morphology and associated findings such as splenomegaly and ascites, while Doppler ultrasonography provides additional information regarding portal venous hemodynamics [8,9].

Color Doppler evaluation can demonstrate alterations in portal venous flow, including changes in flow velocity and direction. Normal portal venous flow is hepatopetal, whereas progressive portal hypertension may be associated with reduced flow or reversal to hepatofugal flow. Doppler imaging can also identify portal vein stenosis, thrombosis and other vascular abnormalities [10]. The combination of color and spectral Doppler therefore permits assessment of both morphological and functional changes in the portal venous system and may provide useful information regarding disease severity [11,12].

Despite the established role of ultrasonography in portal hypertension, assessment of specific Doppler parameters may provide additional information for characterization of disease. In particular, peak systolic velocity (PSV)

and pulsatility index (PI) may demonstrate hemodynamic alterations associated with portal hypertension. Evaluation of these parameters may also assist in differentiating compensated from decompensated cirrhosis.

Therefore, the present study was undertaken to assess the spectrum of color Doppler sonographic findings in portal hypertension, evaluate changes in PSV and PI, and determine the utility of color Doppler ultrasonography in differentiating compensated and decompensated cirrhosis

MATERIALS AND METHODS

This hospital-based cross-sectional observational study was conducted in the Department of Radiodiagnosis, Sri Aurobindo Medical College and Postgraduate Institute (SAIMS), Indore, Madhya Pradesh, India, over a period of 18 months from June 2024 to November 2025, following approval from the Institutional Ethics Committee.

Patients with clinical suspicion of portal hypertension, irrespective of age and sex, were evaluated using ultrasonography and color Doppler ultrasonography. Written informed consent was obtained from all participants before enrollment. Participation was voluntary, and confidentiality of patient information was maintained throughout the study.

Inclusion and Exclusion Criteria

A total of 50 patients with clinical suspicion of portal hypertension undergoing color Doppler ultrasonography during the study period were planned for inclusion. Patients of any age and sex were eligible for participation. Patients refusing consent, traumatic cases, and pregnant patients were excluded from the study.

Methodology

Following written informed consent, demographic characteristics, presenting complaints, clinical diagnosis, ultrasonographic findings, Doppler findings, and final ultrasonographic diagnosis were recorded using a pre-structured proforma. A detailed clinical history was obtained and correlated with relevant investigations. Patients of both sexes who were clinically suspected of having portal hypertension and referred for color Doppler ultrasonography were enrolled in the study. The collected data were subsequently tabulated and statistically analyzed.

Ultrasonography and Color Doppler Examination

Ultrasonographic examinations were performed by an experienced radiologist using GE Logiq E8/E9 and Philips Affiniti 70G ultrasonography systems with a 3.5-5 MHz curvilinear transducer for imaging and Doppler

evaluation. Both two-dimensional ultrasonography and color Doppler examinations were performed. The relevant findings were recorded in the study proforma. On two-dimensional ultrasonography, relevant findings associated with portal hypertension were assessed and documented. Color Doppler evaluation was performed to assess the portal venous system and its flow characteristics. Portal vein flow direction was assessed as hepatopetal or hepatofugal, with hepatopetal flow representing the normal direction of portal venous flow. The presence of abnormalities such as portal vein stenosis or thrombosis was also assessed where present. The Doppler evaluation additionally included assessment of peak systolic velocity (PSV) and pulsatility index (PI) as specified in the study objectives. These parameters were evaluated in relation to portal hypertension and for differentiation between compensated and decompensated cirrhosis.

Outcome Measures

The primary outcomes were the spectrum of two-dimensional and color Doppler ultrasonographic findings in patients with suspected portal hypertension, assessment of portal venous flow characteristics, evaluation of changes in PSV and PI, and differentiation of compensated and decompensated cirrhosis using color Doppler ultrasonography.

Statistical Analysis

Data were collected and entered into Microsoft Excel and presented using frequency tables. Continuous

variables were planned to be expressed as mean and standard deviation, while categorical variables were presented as frequencies and percentages. Pie charts and bar diagrams were used for graphical presentation of qualitative data. The Chi-square test was used to assess associations between two qualitative variables, while the correlation coefficient test was used to assess the correlation between two quantitative variables. A p-value of <0.05 was considered statistically significant.

RESULTS

A total of 50 patients with clinically suspected portal hypertension were evaluated using gray-scale and color Doppler ultrasonography. The study population was assessed with respect to demographic characteristics, gray-scale findings, portal venous flow patterns, vascular complications, portosystemic collaterals, and underlying etiology.

Demographic Characteristics: Of the 50 patients included in the study, 21 (42%) belonged to the 51-65-year age group, followed by 20 (40%) patients in the 36-50-year age group and 9 (18%) patients in the 20-35-year age group. Thus, the majority of patients (82%) were between 36 and 65 years of age. There was a male predominance, with 37 (74%) males and 13 (26%) females, giving a male-to-female ratio of approximately 2.85:1. [Table 1, Figure 1]

Table 1. Demographic characteristics of the study population

Variable	Category	No. (%)
Age group (years)	20-35	9 (18)
	36-50	20 (40)
	51-65	21 (42)
Sex	Male	37 (74)
	Female	13 (26)
Total		50 (100)

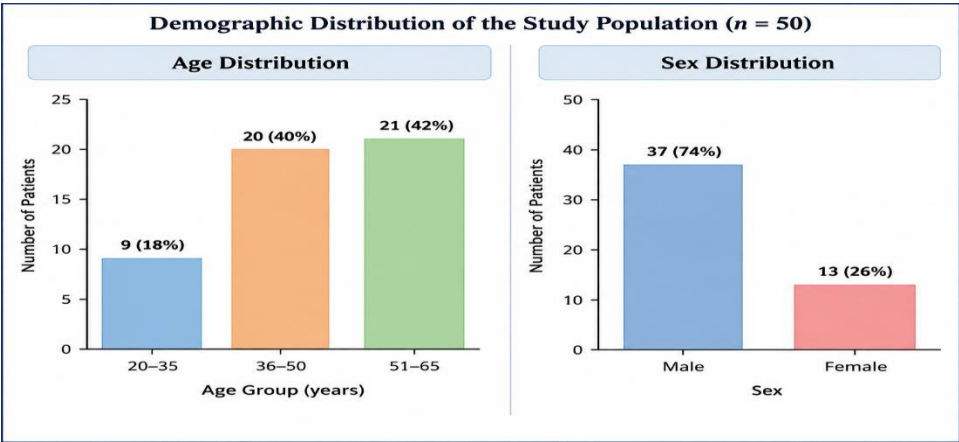


Figure 1: Demographic characteristics of the study population

Gray-Scale Ultrasonographic Findings: An increased portal vein diameter of >13 mm was observed in 30 (60%) patients, whereas 20 (40%) patients had a portal vein diameter of <13 mm. Splenomegaly was present in 39 (78%) patients and absent in 11 (22%). Ascites was the most frequently observed gray-scale finding, being present in 43 (86%) patients. Variation in portal vein diameter of <20% was observed in 40 (80%) patients, while variation of >20% was observed in 10 (20%) patients. [Table 2]

Table 2. Gray-scale ultrasonographic findings

Finding	Category	No. (%)
Portal vein diameter	<13 mm	20 (40)
	>13 mm	30 (60)
Splenomegaly	Present	39 (78)
	Absent	11 (22)
Ascites	Present	43 (86)
	Absent	7 (14)
Variation in portal vein diameter	<20%	40 (80)
	>20%	10 (20)

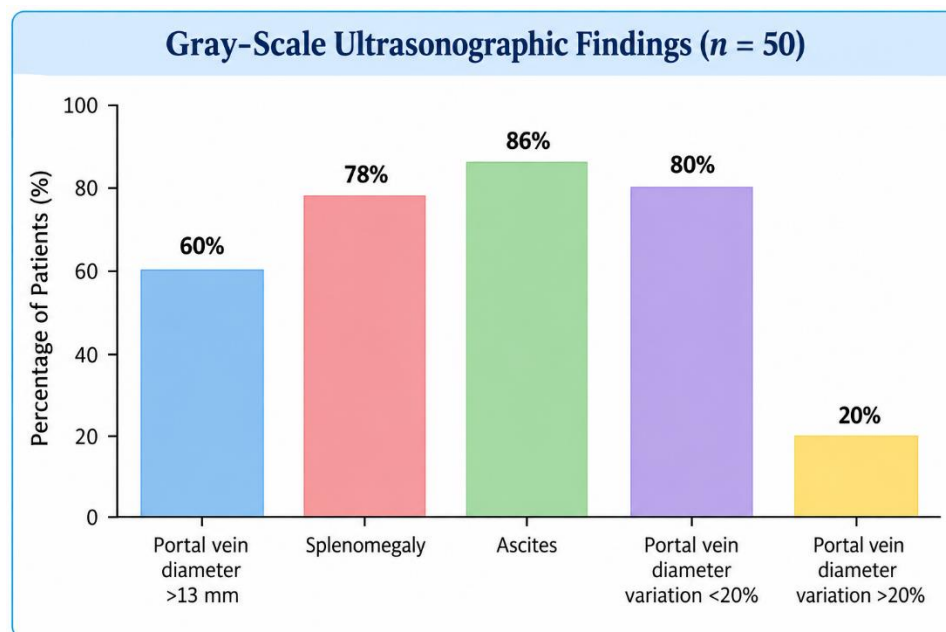


Figure 2: Gray-scale ultrasonographic findings

Color Doppler Assessment of Portal Venous Flow: On color Doppler examination, hepatopetal flow was the predominant portal venous flow pattern, observed in 37 (74%) patients. To-and-fro flow was observed in one (2%) patient, hepatofugal flow in three (6%) patients, and no demonstrable portal venous flow in nine (18%) patients. The splenic vein demonstrated hepatopetal flow in 44 (88%) patients, while one (2%) patient each showed to-and-fro and hepatofugal flow. No flow was demonstrated in four (8%) patients. Similarly, the superior mesenteric vein showed hepatopetal flow in 46 (92%) patients. To-and-fro and hepatofugal flow were each observed in one (2%) patient, while two (4%) patients showed no demonstrable flow. [Table 3, Figure 3]

Table 3. Color Doppler flow patterns in the portal venous system

Vessel	Hepatopetal n (%)	To-and-fro n (%)	Hepatofugal n (%)	No flow n (%)
Portal vein	37 (74)	1 (2)	3 (6)	9 (18)
Splenic vein	44 (88)	1 (2)	1 (2)	4 (8)
Superior mesenteric vein	46 (92)	1 (2)	1 (2)	2 (4)

The portal vein demonstrated a greater frequency of altered or absent flow compared with the splenic and superior mesenteric veins. Abnormal flow patterns, particularly hepatofugal and to-and-fro flow, indicate alteration of the normal

direction of portal venous circulation. The detection of absent flow further emphasizes the importance of color Doppler in identifying significant vascular abnormalities and suspected venous obstruction.

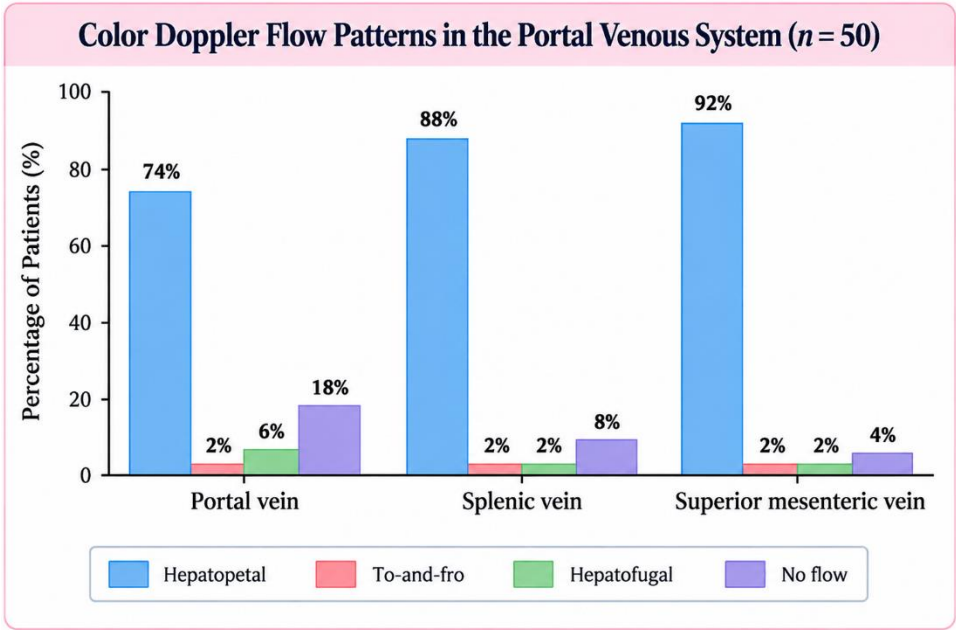


Figure 3: Color Doppler flow patterns in the portal venous system

Vascular Complications and Portosystemic Collaterals: Portal vein thrombosis was identified in 14 (28%) patients, making it the most frequently observed venous thrombotic complication. Splenic vein thrombosis was present in four (8%) patients, while superior mesenteric vein thrombosis was identified in two (4%) patients. Portosystemic collaterals were frequently demonstrated. SR collaterals were the most common, being observed in 38 (76%) patients. GEJ collaterals were present in 22 (44%) patients and PU collaterals in 20 (40%). Gallbladder varices were identified in five (10%) patients, while cavernomatous transformation was observed in three (6%) patients. [Table 4, Figure 4]

Table 4. Vascular complications and portosystemic collaterals

Category	Finding	No. (%)
Venous thrombosis	Portal vein thrombosis	14 (28)
	Splenic vein thrombosis	4 (8)
	Superior mesenteric vein thrombosis	2 (4)
Portosystemic collaterals	SR collaterals	38 (76)
	GEJ collaterals	22 (44)
	PU collaterals	20 (40)
	Gallbladder varices	5 (10)
	Cavernoma	3 (6)

The frequent presence of portosystemic collaterals demonstrates the development of alternative venous pathways in patients with portal hypertension. SR collaterals constituted the most frequent collateral pathway. Portal vein thrombosis was substantially more common than thrombosis involving the splenic or superior mesenteric veins, highlighting the importance of direct evaluation of the portal vein during Doppler examination.

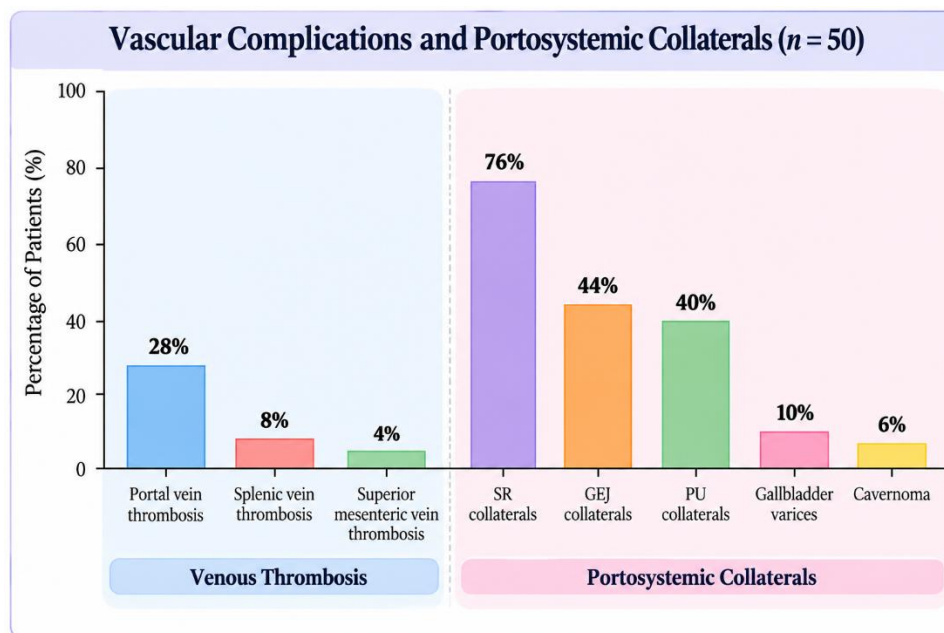


Figure 4: Vascular complications and portosystemic collaterals

Etiological Profile: Cirrhosis was the predominant cause of portal hypertension, accounting for 35 (70%) patients. Portal vein occlusion was identified in five (10%) patients, malignancy in three (6%), and sinistral portal hypertension in two (4%). Other etiologies accounted for five (10%) patients. [Table 5]

Table 5. Etiological profile of portal hypertension

Etiology	No. (%)
Cirrhosis (alcoholic, viral and others)	35 (70)
Portal vein occlusion	5 (10)
Sinistral portal hypertension	2 (4)
Malignancy	3 (6)
Others	5 (10)
Total	50 (100)

Cirrhosis was responsible for the majority of cases, accounting for seven out of every ten patients. The remaining cases demonstrated a heterogeneous etiological profile, including portal venous obstruction, malignancy, sinistral portal hypertension, and other causes.

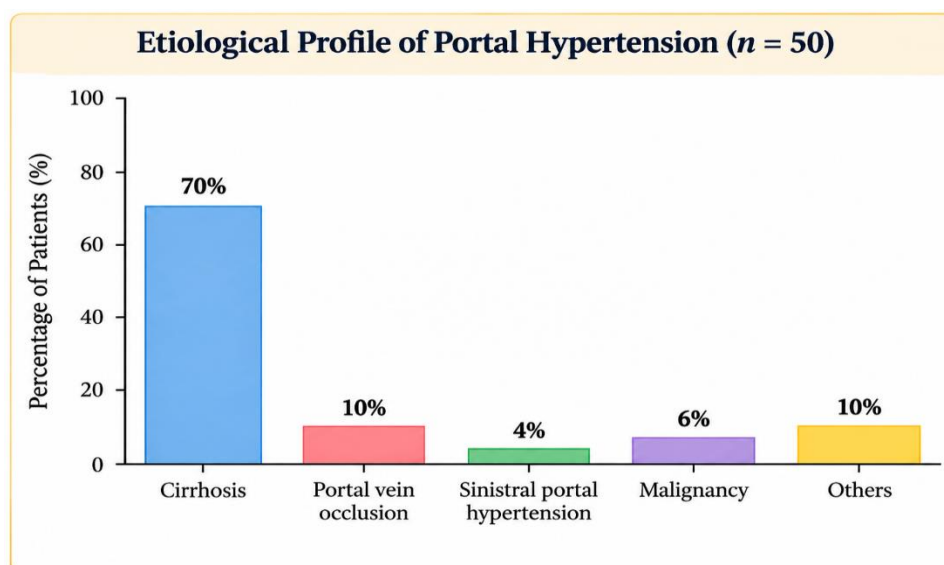


Figure 5: Etiological profile of portal hypertension

Portal Venous Hemodynamic Parameters: Portal venous Doppler examination demonstrated alterations in portal venous hemodynamics. The mean portal vein PSV was 18.6 ± 5.4 cm/s, with a median of 18.0 cm/s and a range of 9.0-31.0 cm/s. The mean portal vein PI was 0.42 ± 0.15 , with a median of 0.40 and a range of 0.16-0.78. These quantitative Doppler parameters complemented the assessment of portal venous flow direction and other sonographic features of portal hypertension. [Table 6]

Table 6. Portal Venous Doppler Parameters

Parameter	Mean ± SD	Median	Range
Portal vein PSV (cm/s)	18.6 ± 5.4	18.0	9.0-31.0
Portal vein PI	0.42 ± 0.15	0.40	0.16-0.78

Differentiation of Compensated and Decompensated Cirrhosis: Among patients with cirrhosis, Doppler and gray-scale findings were compared between compensated and decompensated disease to assess their relationship with the severity of cirrhosis. The decompensated cirrhosis group demonstrated a significantly greater mean portal vein diameter compared with the compensated group (14.2 ± 2.1 mm vs. 12.8 ± 1.4 mm, $p=0.028$). Portal vein PSV was significantly lower in patients with decompensated cirrhosis (16.6 ± 4.5 cm/s) compared with compensated cirrhosis (22.4 ± 4.2 cm/s, $p<0.001$). Similarly, the mean PI was significantly lower in the decompensated group (0.37 ± 0.14 vs. 0.51 ± 0.13 , $p=0.006$). Splenomegaly was more frequently observed among patients with decompensated cirrhosis than those with compensated disease (87.0% vs. 58.3%, $p=0.047$). Ascites was present in 95.7% of patients with decompensated cirrhosis compared with 25.0% of those with compensated cirrhosis ($p<0.001$). Portosystemic collaterals were also significantly more frequent in the decompensated group (91.3% vs. 41.7%, $p=0.002$). [Table 7]

Table 7. Comparison of Doppler and Gray-Scale Parameters Between Compensated and Decompensated Cirrhosis

Parameter	Compensated cirrhosis (n=12)	Decompensated cirrhosis (n=23)	p-value
Portal vein diameter (mm)	12.8 ± 1.4	14.2 ± 2.1	0.028
Portal vein PSV (cm/s)	22.4 ± 4.2	16.6 ± 4.5	<0.001
Portal vein PI	0.51 ± 0.13	0.37 ± 0.14	0.006
Splenomegaly, n (%)	7 (58.3)	20 (87.0)	0.047
Ascites, n (%)	3 (25.0)	22 (95.7)	<0.001
Portosystemic collaterals, n (%)	5 (41.7)	21 (91.3)	0.002

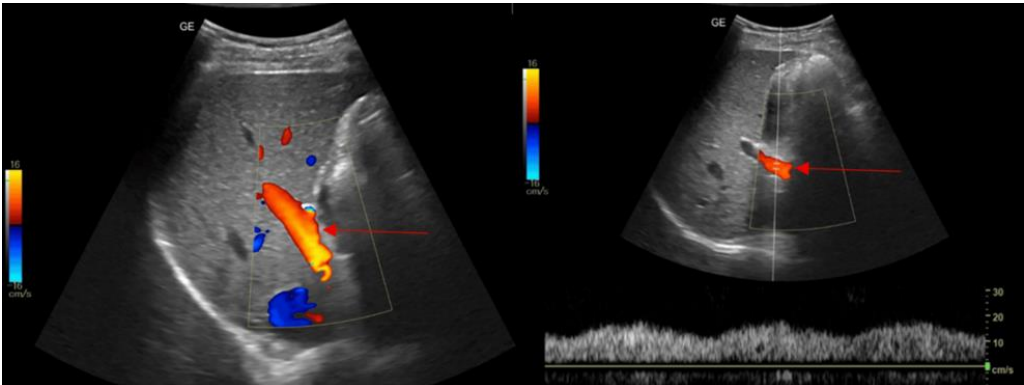


Figure 6: Colour and pulsed Doppler images of the portal vein show normal hepatopetal low (red arrows), normal velocity and waveform pattern

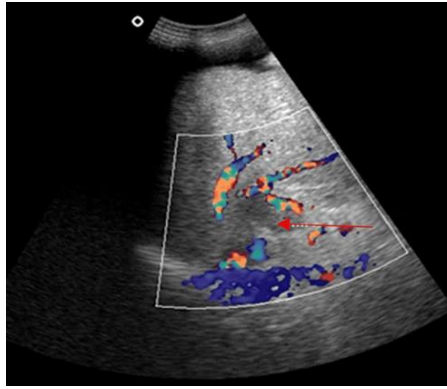


Figure 7: Colour Doppler image of the portal vein shows no colour flow in the portal vein suggesting portal vein thrombosis (red arrow).

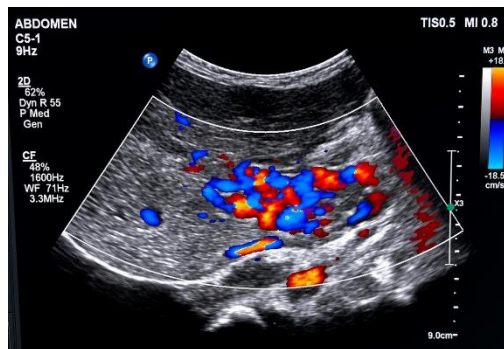


Figure 8: Cavernoma formation- shows multiple tortuous vessels near the hilum



Figure 9: Dilated Portal Vein

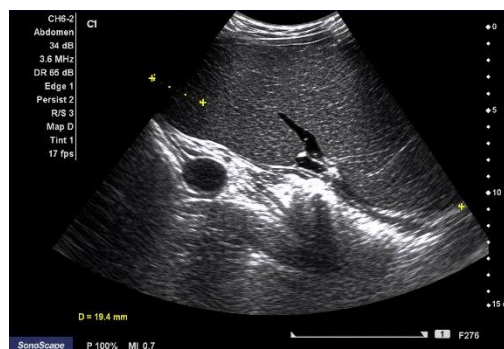


Figure 10: Splenomegaly

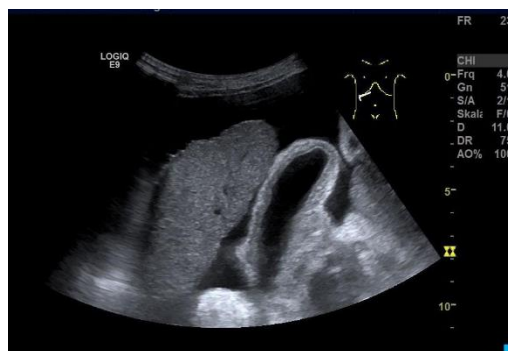


Figure 11: Ascites

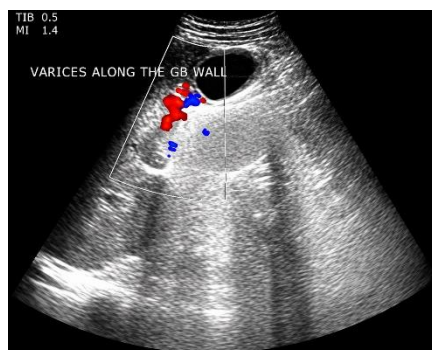


Figure 12: GB Wall Varices

DISCUSSION

Color Doppler ultrasonography provides both morphological and hemodynamic information in patients with portal hypertension. In the present study, 50 patients with clinically suspected portal hypertension were evaluated for gray-scale findings, portal venous flow characteristics, vascular complications, collateral pathways, and quantitative Doppler parameters. The examination demonstrated multiple coexisting abnormalities, highlighting the importance of combining conventional ultrasonography with color and spectral Doppler rather than relying on a single sonographic marker [1,6,8,10].

The study population showed a predominance of middle-aged and older patients, with 42% in the 51-65-year group and 40% in the 36-50-year group. Males accounted for 74% of the study population. This demographic pattern was comparable with that reported by Ruthira et al., who observed a greater representation of patients aged 51-65 years along with male predominance [7]. Nimmakayala et al. also reported a mean age of 57.22 ± 9.26 years and a marked male predominance [5]. Differences between studies may reflect variation in referral patterns, underlying liver disease, and study population characteristics.

Cirrhosis was the leading etiology in the present study, accounting for 70% of cases. Portal vein occlusion was identified in 10%, malignancy in 6%, sinistral portal hypertension in 4%, and other causes in 10%. This predominance of cirrhosis is consistent with the established association between chronic liver disease and

increased intrahepatic vascular resistance [3,4,6]. Ruthira et al. reported cirrhosis in 62.5% of patients, while Nimmakayala et al. identified alcoholic liver disease as the predominant etiology in 62% [5,7]. The presence of non-cirrhotic causes in the present series also demonstrates the importance of evaluating the portal venous system for obstruction, thrombosis, and other vascular abnormalities.

Portal vein diameter exceeded 13 mm in 60% of patients, while 80% demonstrated less than 20% respiratory variation. Ruthira et al. reported a similar frequency of reduced respiratory variation, with less than 20% variation in 80% of patients, whereas Nimmakayala et al. reported decreased respiratory variation in 83% [5,7]. Bolondi et al. demonstrated the diagnostic value of diminished respiratory response of portal vessels in portal hypertension [13]. These findings support assessment of portal vein caliber during respiration as a useful adjunctive marker. Nevertheless, portal vein diameter is influenced by several physiological and technical factors and should therefore be assessed alongside other sonographic findings [1,8].

Splenomegaly was observed in 78% of patients, making it one of the commonest gray-scale abnormalities. Ruthira et al. and Nimmakayala et al. reported comparable frequencies of 85% and 77%, respectively [5,7]. Splenic enlargement results from increased portal venous pressure and chronic splenic congestion. However, its diagnostic value is enhanced when it occurs together with portal vein dilatation, altered flow,

ascites, or collateral vessels, as splenomegaly alone is not specific for portal hypertension [8,10].

Ascites was identified in 86% of the study population. Nimmakayala et al. reported ascites in 78% of patients [5]. The high frequency observed in the present study may reflect the inclusion of patients with advanced portal hypertensive disease. A particularly notable finding was the difference between compensated and decompensated cirrhosis, with ascites present in 25% of compensated patients compared with 95.7% of decompensated patients ($p < 0.001$). This marked difference supports the role of ascites as an important sonographic manifestation of advanced hepatic dysfunction [3,6].

Hepatopetal flow remained the predominant portal venous flow pattern, occurring in 74% of patients. To-and-fro flow was seen in 2%, hepatofugal flow in 6%, and no demonstrable flow in 18%. Color Doppler is particularly useful for this assessment because it directly demonstrates the direction of portal venous flow, thereby providing information that cannot be obtained reliably from gray-scale imaging alone [1,8]. Cannella et al. also emphasized detailed Doppler evaluation of venous hemodynamics, including assessment of the left gastric vein, for understanding collateral circulation in portal hypertension [2].

The mean portal vein PSV was 18.6 ± 5.4 cm/s, with a median of 18.0 cm/s and a range of 9.0-31.0 cm/s. Portal venous velocity reflects the functional consequences of increased vascular resistance and redistribution of portal blood flow. Its measurement is affected by respiration, the site of sampling, angle of insonation, technical factors, and collateral circulation, and should therefore be interpreted in the context of the overall examination [1,8]. Nouh et al. demonstrated the value of portal color Doppler parameters as non-invasive predictors of esophageal varices in cirrhotic patients, supporting the clinical utility of quantitative Doppler assessment [3].

Hepatofugal flow was observed in 6% of patients. Ruthira et al. reported hepatofugal flow in 7.5% [7], while Ditchfield et al. demonstrated reversed portal venous flow as an important Doppler sign of portal hypertension [15]. Reversal of portal flow represents a substantial alteration from the normal hepatopetal circulation and may occur with advanced portal hypertension and development of collateral pathways. Its detection is therefore particularly relevant when accompanied by thrombosis, extensive collateralization, or other signs of advanced disease.

The mean portal vein PI in the present study was 0.42 ± 0.15 , with a median of 0.40 and a range of 0.16-0.78. Assessment of PI alongside PSV provides additional information regarding portal venous hemodynamics. Kumhari et al. emphasized that gray-scale ultrasonography demonstrates structural manifestations,

whereas color and spectral Doppler contribute information regarding flow direction, velocity, vascular patency, and hemodynamic changes [1]. Consequently, simultaneous evaluation of portal vein diameter, respiratory variation, PSV, PI, and flow direction provides a broader assessment of the portal circulation than any isolated parameter.

An important observation was the significant difference in portal venous parameters between compensated and decompensated cirrhosis. Portal vein PSV was 22.4 ± 4.2 cm/s in compensated patients and 16.6 ± 4.5 cm/s in decompensated patients ($p < 0.001$). PI was also significantly lower in decompensated disease, measuring 0.37 ± 0.14 compared with 0.51 ± 0.13 in compensated cirrhosis ($p = 0.006$). Portal vein diameter was significantly greater in the decompensated group, measuring 14.2 ± 2.1 mm compared with 12.8 ± 1.4 mm in compensated disease ($p = 0.028$). These differences indicate progressive modification of portal venous hemodynamics with worsening cirrhosis and are consistent with the role of ultrasonography in non-invasive assessment of portal hypertension [8].

Hepatic venous Doppler parameters can provide complementary information about the effects of chronic liver disease on intrahepatic circulation. Ruthira et al. described alteration of the normal triphasic hepatic venous waveform and assessed the hepatic vein damping index in portal hypertension [7]. Nimmakayala et al. further demonstrated increasing damping index values across Child-Pugh classes, with a significant difference between disease severity groups [5]. These observations suggest that hepatic venous waveform analysis may complement portal venous Doppler measurements when assessing the severity of chronic liver disease.

Portosystemic collateralization was frequent in the present study. Splenorenal collaterals were identified in 76%, followed by gastroesophageal collaterals in 44%, paraumbilical collaterals in 40%, gallbladder varices in 10%, and cavernomatous transformation in 6%. Ruthira et al. reported splenorenal/gastrorenal collaterals in 95%, gastroesophageal collaterals in 55%, gallbladder varices in 12.5%, and cavernoma in 7.5% [7]. Nimmakayala et al. reported splenorenal collaterals in 93% [5], while Subramanyam et al. demonstrated the usefulness of sonography for identifying portosystemic collateral pathways [16]. Variation in collateral prevalence between studies may be related to differences in patient selection and the severity of portal hypertension.

Gallbladder varices were present in 10% of patients, broadly comparable with the frequency reported by Ruthira et al. [7]. Chawla et al. specifically investigated gallbladder varices in patients with portal vein thrombosis, emphasizing their relevance as an additional collateral pathway [17]. Cavernomatous transformation was present in 6% of the current study population and

represents an important imaging manifestation of chronic portal venous obstruction. Color Doppler facilitates recognition of these vascular channels and helps establish their relationship with portal venous occlusion.

Portal vein thrombosis was detected in 28% of patients, while splenic vein and superior mesenteric vein thrombosis were identified in 8% and 4%, respectively. The relatively high prevalence of portal vein thrombosis emphasizes the importance of systematically assessing portal venous patency. Ditchfield et al. highlighted the diagnostic contribution of duplex Doppler assessment of the portal vein and spleen in portal hypertension [15]. In the present study, absent or altered flow patterns on Doppler provided an important clue to vascular obstruction, while associated cavernomatous transformation supported chronicity [1,8]. Differences from earlier studies may reflect variations in disease severity and case selection.

The frequency of several sonographic abnormalities increased significantly with decompensation. Portosystemic collaterals were present in 91.3% of decompensated patients compared with 41.7% of compensated patients ($p=0.002$), while splenomegaly was present in 87.0% and 58.3%, respectively ($p=0.047$). When considered together with the lower PSV and PI and larger portal vein diameter observed in decompensated disease, these findings indicate that progression of cirrhosis is associated with increasingly evident structural and hemodynamic abnormalities. The particularly large difference in ascites and collateral formation further supports their value in recognizing advanced disease.

The findings are consistent with the pathophysiological progression of portal hypertension, in which increasing intrahepatic vascular resistance is accompanied by development of collateral pathways and progressive changes in portal venous flow [4,12]. The Baveno consensus emphasized the clinical importance of clinically significant portal hypertension in the development of cirrhotic complications [4]. Since direct measurement of portal pressure is invasive, ultrasonography offers an accessible and repeatable method for evaluating indirect morphological and hemodynamic manifestations [6,8].

Despite the comprehensive evaluation, the study had certain limitations. The relatively small sample size of 50 patients and single-centre design may limit generalizability. The cross-sectional design precluded longitudinal assessment of disease progression. Doppler measurements may also be influenced by technical factors, respiration, and operator dependence. Direct portal pressure measurements were not performed for comparison.

Overall, the present study demonstrates that no single Doppler or gray-scale feature adequately represents the entire spectrum of portal hypertension. Portal vein diameter, respiratory variation, flow direction, PSV, PI, splenic size, ascites, vascular patency, and collateral pathways provide complementary information. The significant differences between compensated and decompensated cirrhosis particularly indicate the potential value of an integrated Doppler approach for assessing disease severity. Therefore, combined gray-scale, color Doppler, and spectral Doppler ultrasonography represents a practical non-invasive method for evaluating portal hypertension, identifying its vascular complications, and providing useful information for differentiation of compensated and decompensated cirrhosis.

CONCLUSION

Color Doppler ultrasonography is a valuable, non-invasive modality for evaluating portal hypertension. The study demonstrated frequent structural and hemodynamic abnormalities, including portal vein dilatation, splenomegaly, ascites, altered portal venous flow, thrombosis, and portosystemic collaterals. Portal vein peak systolic velocity and pulsatility index were significantly lower in decompensated compared with compensated cirrhosis. Decompensated patients also showed greater portal vein diameter, splenomegaly, ascites, and portosystemic collaterals. Thus, combined gray-scale and Doppler assessment provides useful information regarding portal hypertension severity, vascular complications, and differentiation between compensated and decompensated cirrhosis.

DECLARATIONS

Ethics Approval and Consent to Participate

The study was conducted in accordance with the ethical standards of the institution and the principles of the Declaration of Helsinki. Ethical approval was obtained from the Institutional Ethics Committee. Written informed consent was obtained from all participants before inclusion in the study.

Consent for Publication

Not applicable.

Availability of Data and Materials

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Competing Interests

The authors declare that they have no competing interests.

Funding

The authors received no specific funding for this study.

Authors' Contributions

All authors contributed to the conception and design of the study, data collection, analysis and interpretation of the findings, drafting or critical revision of the manuscript, and approved the final version for publication.

Acknowledgements

The authors would like to thank all participants and the clinical and radiology staff who contributed to the completion of this study.

AI Use Disclosure

The authors declare that no generative artificial intelligence tools were used to generate or substantially modify the scientific content of this manuscript.

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