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Original Research Article

ROLE OF COLOR DOPPLER ULTRASONOGRAPHY IN THE EVALUATION OF PORTAL HYPERTENSION AND ITS CORRELATION WITH SEVERITY OF CHRONIC LIVER DISEASE

Charu Vishvakarma[1], Mohit Gambhir[2], Deval Rishi Pandit[3]

[1]*Junior Resident, Department of Radiodiagnosis, Rohilkhand Medical College & Hospital, Bareilly, India.*
[2]*Assistant Professor, Department of Radiodiagnosis, Rohilkhand Medical College & Hospital, Bareilly, India.*
[3]*Junior Resident, Department of Radiodiagnosis, Rohilkhand Medical College & Hospital, Bareilly, India.*

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Corresponding Author:
Dr. Charu Vishvakarma,
Junior Resident, Department of Radiodiagnosis, Rohilkhand Medical College & Hospital, Bareilly, India.
Email: drcharuvishvakarma@gmail.com

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ABSTRACT

Background: Portal hypertension is a major complication of chronic liver disease resulting from increased resistance to portal venous flow. Although hepatic venous pressure gradient measurement is the gold standard, it is invasive and not routinely feasible. Doppler ultrasonography offers a non-invasive alternative for evaluating portal hemodynamics. **Objective:** To assess the role of color Doppler ultrasonography in portal hypertension and its correlation with the severity of chronic liver disease.

Materials and Methods: This cross-sectional study included 76 patients with suspected portal hypertension. All patients underwent grayscale and Doppler ultrasonography. Parameters assessed included portal vein diameter, velocity, flow direction, respiratory phasicity, splenic vein diameter, splenomegaly, collateral circulation, hepatic artery resistive index, and hepatic vein damping index. Disease severity was graded using the Child–Pugh classification.

Results: Portal vein dilatation, reduced velocity, and altered flow patterns were commonly observed. Splenomegaly and portosystemic collaterals were present in a majority of patients. Significant correlations were found between Doppler parameters and Child–Pugh class, with worsening portal hemodynamics in advanced disease. Higher grades of ascites were also associated with reduced portal vein velocity and increased collateral formation.

Conclusion: Color Doppler ultrasonography is a reliable, non-invasive tool for evaluating portal hypertension. Doppler parameters show significant correlation with disease severity and can aid in diagnosis and prognostic assessment.

Keywords: Hypertension, Portal; Liver Cirrhosis; Splenomegaly; Ultrasonography, Doppler, Color.

INTRODUCTION

Portal hypertension is defined as an abnormal increase in portal venous pressure and is most commonly associated with chronic liver disease and cirrhosis.[1-2] It results from increased resistance to portal blood flow and can lead to complications such as ascites, splenomegaly and gastro-oesophageal varices.[3-4]

Although measurement of hepatic venous pressure gradient is considered the gold standard for diagnosis, it is invasive and not routinely performed.[3-4] Doppler ultrasonography has therefore become an important non-invasive imaging modality for evaluating portal venous hemodynamics, including portal vein diameter, flow velocity, flow direction and collateral circulation.[5-6] The present study was conducted to evaluate Doppler ultrasound findings in portal hypertension and to correlate these findings with the severity of chronic liver disease.[7-9]

MATERIALS AND METHODS

After institutional ethical committee approval, the present cross-sectional observational study was conducted over a period of one year to evaluate the role of color Doppler ultrasonography in portal hypertension. Consecutive patients with clinical suspicion or diagnosis of portal hypertension and chronic liver parenchymal disease were included in the study after taking informed consent from all participants.

Inclusion Criteria Included

- Age > 18 years
- Clinical or biochemical evidence suggestive of chronic liver disease

Exclusion Criteria were

- Pediatric patients
- Pregnant women
- Trauma cases.

All patients underwent abdominal ultrasonography followed by Doppler evaluation using a Samsung RS80 ultrasound machine equipped with a 2–5 MHz curvilinear transducer. Grayscale ultrasonography was initially performed to assess liver morphology, echotexture, presence of ascites (Figure 1J) and the splenic size (Figure 1K). Subsequently, Doppler evaluation of the portal venous system was performed to measure portal vein diameter, assess portal vein flow direction, determine portal vein velocity, evaluate respiratory phasicity of the portal vein and identify splenic vein diameter, portosystemic collateral circulation and hepatic artery resistive index (Figure 1A–I).

Portal vein diameter was measured anterior to the inferior vena cava during quiet respiration and a diameter greater than 13 mm was considered dilated. Doppler spectral analysis was used to determine flow velocity and direction maintaining the Doppler insonation angle below 60° for accurate velocity measurements. Collateral circulation was assessed at the splenic hilum, gastro-oesophageal junction, paraumbilical region and anterior abdominal wall. Hepatic vein damping index (DI) was defined as the ratio of minimum to maximum velocity (Vmin/Vmax) on spectral Doppler waveform; values <0.6 were considered abnormal (Figure 1L). The severity of chronic liver disease was graded using the Child–Pugh scoring system [10] based on serum bilirubin, serum albumin, prothrombin time, ascites and hepatic encephalopathy.

Correlation analysis was performed using Spearman's rank correlation coefficient for non-parametric data and Pearson's correlation coefficient for normally distributed data to evaluate Doppler parameters across Child–Pugh classes and to assess their relationship with the grade of ascites. For group comparisons across Child–Pugh classes, one-way ANOVA (for normally distributed continuous variables) or the Kruskal–Wallis test (for non-parametric data) was used, followed by post-hoc analysis where applicable. Categorical variables were compared using the Chi-square test or Fisher's exact test when expected frequencies were small. To identify Doppler predictors of severe liver disease, multivariate logistic regression analysis was performed, with variables significant in univariate analysis entered into the model; for portal vein velocity, a threshold of <15 cm/s was applied in this model. A p-value < 0.05 was considered statistically significant.

RESULTS

A total of 76 patients were included in this study that underwent grayscale sonography and Doppler evaluation as per the study protocol. Patient demographics and clinical characteristics are summarized in Table 1, and grayscale sonographic findings with disease severity in Table 2.

Table 1: Demographic and clinical profile of the study cohort (n=76)

Parameter	Categories, n (%)
Age group (years)	18–29: 6 (7.9%); 30–39: 12 (15.8%); 40–49: 20 (26.3%); 50–59: 22 (28.9%); ≥60: 16 (21.1%)
Gender	Male: 56 (73.7%); Female: 20 (26.3%)
Presenting complaint (mutually exclusive)	Ascites/distension: 28 (36.8%); Upper GI bleed: 16 (21.1%); Abdominal pain/discomfort: 14 (18.4%); Jaundice: 10 (13.2%); Fatigue/weight loss/others: 8 (10.5%)
Etiology	Alcohol-related liver disease: 24 (31.6%); Chronic hepatitis B: 14 (18.4%); Chronic hepatitis C: 10 (13.2%); NASH/MASLD: 16 (21.0%); Cryptogenic: 4 (5.2%); Others (autoimmune, mixed): 2 (2.6%); Unknown: 6 (7.9%)

Table 2: Grayscale sonographic findings and disease severity (n=76)

Parameter	Categories, n (%)
Liver echotexture	Increased echogenicity: 4 (5.3%); Coarse: 72 (94.7%)
Spleen size	<13 cm: 24 (31.6%); >13 cm: 52 (68.4%)
Child–Pugh class	A: 30 (39.5%); B: 33 (43.4%); C: 13 (17.1%)
Complications	Moderate–massive ascites: 32 (42.1%); Splenomegaly: 52 (68.4%); Portal hypertensive gastropathy: 10 (13.2%); Portal vein thrombosis: 8 (10.5%); Hepatic encephalopathy/HRS: 12 (15.8%)

Evaluation of portal venous hemodynamics using color Doppler ultrasonography demonstrated several characteristic findings in patients with portal hypertension. Portal vein diameter was found to be dilated (≥13 mm) in 76.3%. Assessment of portal vein flow direction showed hepatopetal flow in 60.5%, hepatofugal flow in 23.7%, bidirectional flow in 5.3% and absent flow in 10.5% of cases. Spectral Doppler analysis revealed reduced portal vein velocity (<20 cm/s) in 65.8% of patients.

Respiratory phasicity of the portal vein was reduced or absent in 63.2% of patients. Splenic vein dilatation was observed in 55.3% of patients, while splenomegaly (spleen size ≥13 cm) was noted in 68.4% of patients. Portosystemic collateral circulation was identified in 67.1% of patients, most commonly involving perigastric collaterals, splenorenal shunts, paraumbilical veins and anterior abdominal wall varices. In addition, evaluation of hepatic venous waveforms demonstrated a decreased hepatic vein damping index in a significant proportion of patients indicating reduced hepatic venous pulsatility associated with advanced liver disease. Doppler findings in the study cohort are summarised in Table 3.

Table 3: Doppler sonographic findings (n=76)

Parameter	Categories, n (%)
Portal vein diameter	<13 mm: 18 (23.7%); ≥13 mm: 58 (76.3%)
Portal vein flow velocity	<20 cm/s: 50 (65.8%); ≥20 cm/s: 14 (18.4%); Could not be evaluated: 12 (15.8%)
Portal vein flow direction	Hepatopetal: 46 (60.5%); Hepatofugal: 18 (23.7%); Bidirectional: 4 (5.3%); No flow: 8 (10.5%)
Respiratory phasicity	Preserved: 16 (21.1%); Reduced/absent: 48 (63.2%); Not assessable: 12 (15.8%)
Portal vein lumen	Clear: 64 (84.2%); Thrombosis: 8 (10.5%); Portal cavernoma: 4 (5.2%)
Splenic vein diameter	<10 mm: 34 (44.7%); ≥10 mm: 42 (55.3%)
Collaterals/varices	Paraumbilical vein: 8 (10.5%); Splenorenal: 10 (13.1%); Anterior abdominal wall: 12 (15.8%); Gall bladder varices: 6 (7.8%); Perigastric: 12 (15.8%); No collaterals: 25 (32.9%)
Damping index of hepatic vein	<0.6: 65 (85.5%); >0.6: 11 (14.5%)

Comparison of Doppler parameters across Child–Pugh classes summarized in Table 4 demonstrated worsening portal hemodynamics with increasing disease severity. Mean Doppler parameters were compared across Child–Pugh classes using one-way ANOVA or Kruskal–Wallis test. A statistically significant difference was observed for portal vein diameter, portal vein velocity, splenic vein diameter, hepatic artery resistive index, hepatofugal flow, and presence of collateral vessels ($p < 0.05$). Patients in Child–Pugh class C showed greater portal vein dilatation (p=0.001), lower portal vein velocity (p<0.001), higher frequency of hepatofugal flow (p=0.004) and collateral circulation (p=0.041) compared with those in classes A and B. Box plots comparing the Doppler parameters across various Child–Pugh classes are shown in Figure 2A. Multivariate logistic regression summarized in Table 5 revealed that portal vein diameter ≥13 mm (AOR 2.80, p=0.015), portal vein velocity <15 cm/s (AOR 3.60, p=0.007) and hepatofugal flow (AOR 4.90, p=0.002) were independent Doppler predictors of severe liver disease indicating that progressive portal hypertension and reversal of portal flow are strongly associated with advanced hepatic dysfunction.

Table 4: Comparison of Doppler parameters across Child–Pugh classes

Parameters				P-value
	Class A (n=30)	**Class B (n=33)**	**Class C (n=13)**	
Portal vein diameter (mm), Mean ± SD	12.9 ± 1.6	14.3 ± 1.8	15.3 ± 2.1	**0.001**
Portal vein velocity (cm/s), Mean ± SD	16.4 ± 3.0	13.7 ± 3.1	11.8 ± 2.9	**<0.001**
Splenic vein diameter (mm), Mean ± SD	10.3 ± 1.7	11.2 ± 1.8	12.6 ± 2.2	**<0.001**
Hepatic artery RI, Mean ± SD	0.75 ± 0.05	0.78 ± 0.05	0.81 ± 0.06	**0.002**
Hepatofugal flow, n (%)	1 (3.3)	8 (24.2)	9 (69.2)	**0.004**
Predominant collaterals present, n (%)	13 (43.3)	27 (81.8)	11 (84.6)	0.041

p-values calculated using one-way ANOVA for normally distributed data and Kruskal–Wallis test for non-parametric data.

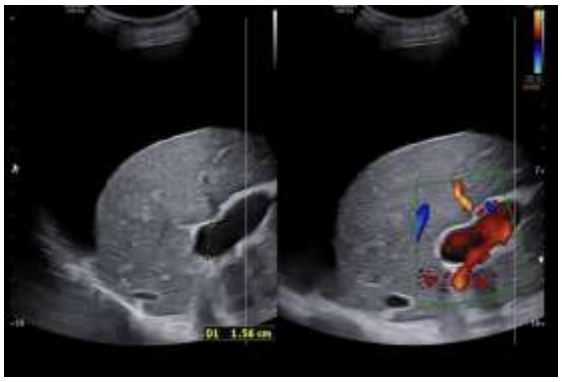
Table 5: Multivariate analysis for predictors of severity of liver disease based on Child–Pugh class

Predictor (coded)	β Coefficient	Adjusted OR (AOR)	95% CI	p-value
Portal vein diameter ≥13 mm	1.03	2.80	1.20–6.53	**0.015**
Portal vein velocity <15 cm/s	1.28	3.60	1.42–9.12	**0.007**
Hepatofugal flow (yes)	1.59	4.90	1.82–13.16	**0.002**
Splenic vein diameter ≥10 mm	0.64	1.90	0.82–4.39	0.132
Recanalized para-/umbilical vein (yes)	0.83	2.29	0.92–5.71	0.075

Correlation analysis also revealed that patients with higher grades of ascites had significantly reduced portal vein velocity (p=0.003) and more frequent collateral formation (p=0.001). Correlation analysis comparing Doppler parameters with ascites grade and the corresponding box plots are summarised in Table 6 and Figure 2B.

Table 6: Correlation analysis between Doppler parameters and ascites severity

Variable	Mild/No ascites (n=44)	Moderate–massive (n=32)	p-value
Portal vein diameter (mm), Mean ± SD	13.4 ± 1.8	14.8 ± 2.1	**0.003**
Portal vein velocity (cm/s), Mean ± SD	15.1 ± 3.2	12.7 ± 3.4	**0.001**
Splenic vein diameter (mm), Mean ± SD	10.7 ± 1.9	12.0 ± 2.3	**0.002**
Recanalized para-/umbilical vein, n (%)	3 (8.8)	9 (21.4)	0.122



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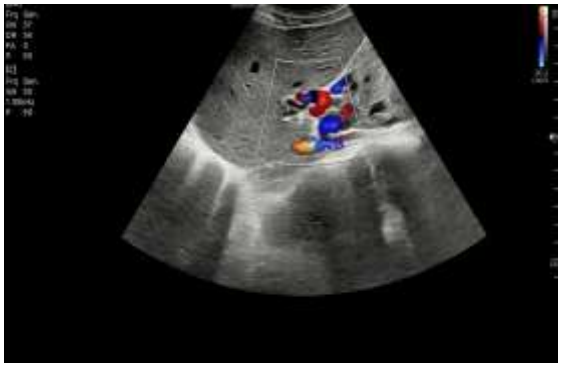

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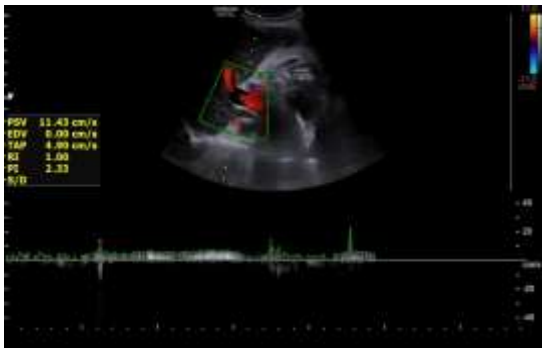

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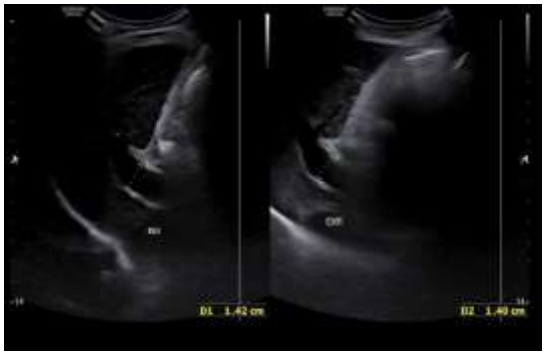

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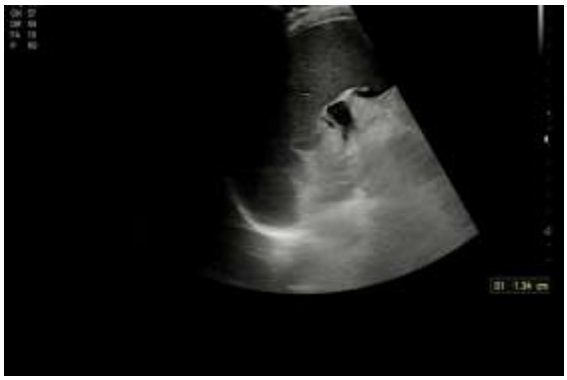

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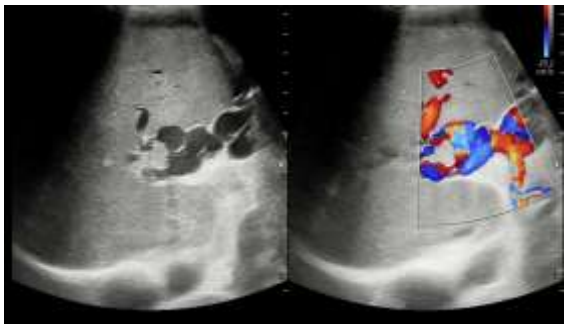

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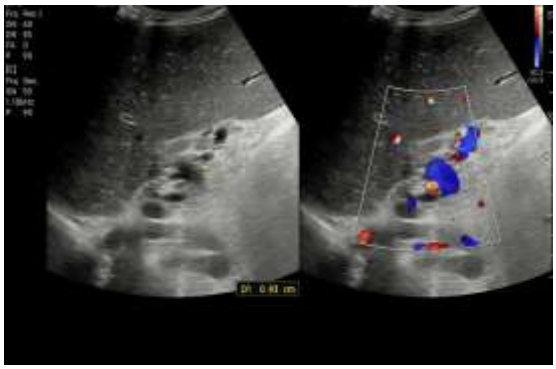

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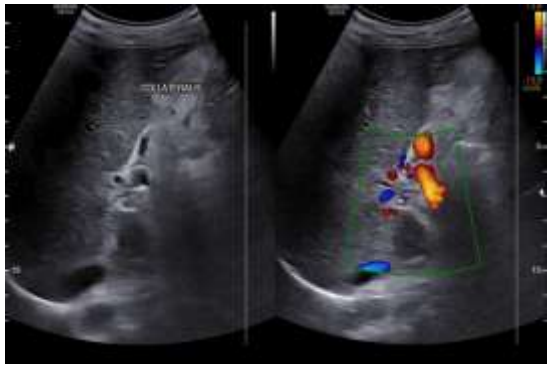

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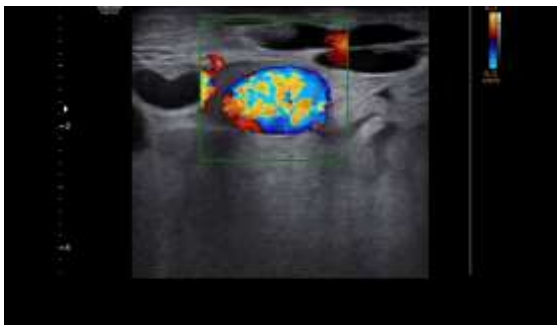
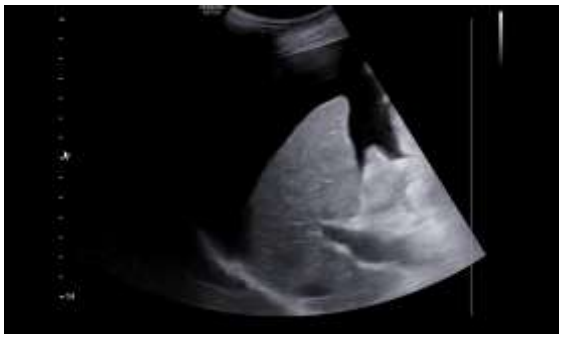


(I)



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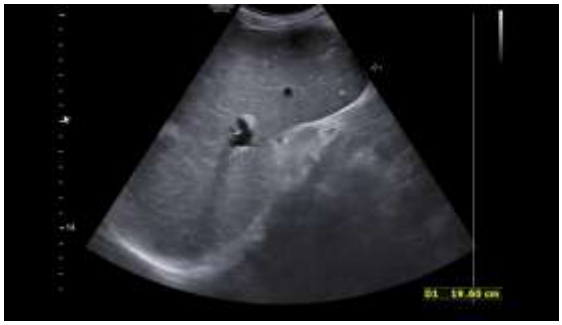
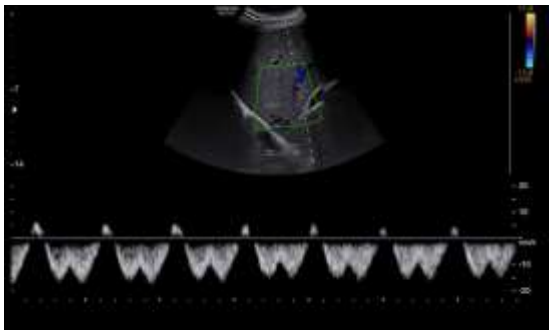


(K)



(L)

Figure 1: Grayscale and color Doppler ultrasonography findings in portal hypertension. (A–I) portal venous system evaluation including vein diameter, flow direction, velocity, respiratory phasicity, splenic vein and portosystemic collaterals; (J) liver echotexture and ascites; (K) splenomegaly; (L) hepatic vein waveform for damping index

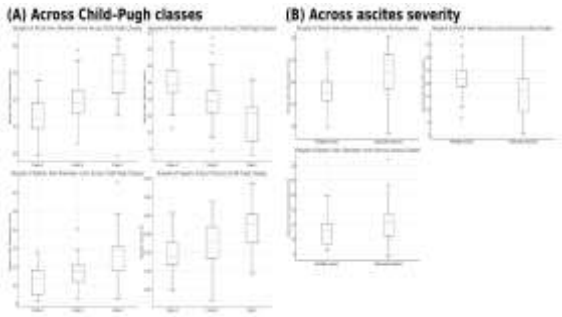


Figure 2: Box-plot comparison of Doppler vascular parameters. (A) portal vein diameter, portal vein velocity, splenic vein diameter and hepatic artery resistive index across Child–Pugh classes; (B) portal vein diameter, portal vein velocity and splenic vein diameter across ascites severity grades

DISCUSSION

In the present study, we evaluated 76 patients with clinically suspected portal hypertension using color Doppler ultrasonography to assess portal venous hemodynamics and its correlation with the severity of chronic liver disease. Our study population included predominantly middle-aged individuals with a male predominance, and alcohol-related liver disease being the most common etiology, followed by non-alcoholic steatohepatitis and viral hepatitis similar to earlier studies which have reported chronic liver disease and portal hypertension to be more prevalent among middle-aged males due to higher rates of alcohol consumption and viral hepatitis infection.[3-4,11]

Portal vein dilatation was observed in the majority of patients in this study which is a well-recognized ultrasonographic finding in portal hypertension patients indicating increased portal venous pressure. Similar observations have been reported by Bolondi et al. and Zoli et al., who demonstrated that portal vein dilatation is frequently associated with increased portal venous pressure in patients with cirrhosis.[7-8] However, Lafortune et al. emphasized that portal vein diameter alone may not always reliably diagnose portal hypertension due to overlap between normal individuals and patients with chronic liver disease.[12] Reduced portal vein velocity was another important finding in this study reflecting increased intrahepatic vascular resistance and reduced portal blood flow. Similar findings have been reported by Patriquin et al. and Mittal et al. who demonstrated significant reductions in portal vein velocity in patients with portal hypertension and advanced liver disease.[13-14] Loss of respiratory phasicity is another important finding in this study suggesting loss of normal venous compliance associated with portal hypertension. Assessment of portal venous flow direction revealed hepatopetal flow in the majority of patients, while hepatofugal flow was seen in a smaller but clinically significant proportion. Reversal of portal venous flow is considered a hallmark of advanced portal

hypertension and is frequently associated with extensive collateral formation. Herbay et al. also reported that hepatofugal flow represents an important Doppler indicator of severe portal hypertension and advanced liver disease.[15] Splenomegaly was observed in a large proportion of patients in the present study. Splenic enlargement occurs due to congestion of the splenic circulation secondary to increased portal venous pressure. Similar observations have been described in earlier studies evaluating ultrasonographic findings in portal hypertension.[16] Splenic vein dilatation was also observed in several patients, which further supports the presence of portal venous congestion. Portosystemic collateral circulation was identified in majority of the patients in this study representing an adaptive mechanism that helps decompress the portal venous system in response to sustained portal hypertension. Subramanyam et al. demonstrated that collateral veins such as paraumbilical veins, splenorenal shunts, and perigastric collaterals can be effectively detected using Doppler ultrasonography.[17] The presence of these collateral channels is often associated with more advanced disease and increased risk of complications such as variceal bleeding. Evaluation of hepatic venous waveforms also revealed decreased hepatic vein damping index in patients with more severe liver disease. Decreased damping index reflects reduced hepatic venous pulsatility due to increased hepatic parenchymal stiffness and altered venous compliance, which has been described in previous Doppler studies of cirrhosis.[18]

Further analysis of Doppler parameters across Child–Pugh classes in the present study demonstrated progressive deterioration of portal venous hemodynamics with increasing severity of liver disease. These findings are consistent with previous studies that have demonstrated progressive deterioration of portal hemodynamics with increasing Child–Pugh class. Bolondi et al,[7] Zoli M et al,[8] and Herbay AV et al,[15] reported that patients with advanced liver disease show significantly reduced portal vein velocity and increased portal vein diameter due to rising intrahepatic resistance. Similarly, hepatofugal flow and the presence of portosystemic collaterals are more frequently observed in patients with advanced cirrhosis and are considered markers of severe portal hypertension.

In the present study, Doppler parameters also showed significant variation with the grade of ascites, indicating worsening portal venous hemodynamics with increasing ascites severity. These observations are in concordance with previous studies by Mittal P et al,[13] and Patriquin H et al,[14] which have demonstrated a significant association between the severity of ascites and worsening portal hemodynamics. Reduced portal vein velocity in patients with higher grades of ascites has been attributed to increased intrahepatic resistance and splanchnic vasodilatation. Additionally, Subramanyam BR et al,[17] reported that the increased frequency of collateral formation in patients with severe ascites reflects the development of alternative pathways to decompress the portal venous system.

Thus, the findings of the present study demonstrate that color Doppler ultrasonography provides valuable information regarding portal venous hemodynamics and the severity of chronic liver disease. Doppler parameters such as portal vein diameter, portal vein velocity, direction of flow, collateral circulation and hepatic vein damping index can serve as useful non-invasive markers for assessing the severity of portal hypertension and predicting advanced liver disease. Our present study highlights the important role of Doppler ultrasonography as a readily available, non-invasive imaging modality for the evaluation and prognostic assessment of patients with portal hypertension.

CONCLUSION

Color Doppler ultrasonography is a reliable and non-invasive imaging modality for the evaluation of portal hypertension. Doppler parameters such as portal vein diameter, portal vein velocity, direction of flow, splenomegaly and presence of portosystemic collaterals demonstrated characteristic hemodynamic changes associated with portal hypertension. These Doppler findings also showed correlation with the severity of chronic liver disease and Child–Pugh classification. Thus, Doppler ultrasonography serves as an effective diagnostic and prognostic tool for assessing portal hypertension and monitoring disease severity in patients with chronic liver disease.

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