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Original Research Article

Doppler Ultrasound Assessment of Portal Vein, Hepatic Vein, and Hepatic Artery Hemodynamics in Cirrhosis and Correlation with Child-Pugh Score

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Abstract

Background: Cirrhosis produces progressive hepatic architectural distortion and portal hypertension, altering hepatic vascular hemodynamic. Non-invasive Doppler ultrasound can quantify these changes.

Objective: To evaluate Doppler ultrasound parameters of the portal vein, hepatic artery, and hepatic vein in liver cirrhosis and correlate them with Child-Pugh score.

Methods: A cross-sectional study was conducted on 78 ultrasound-diagnosed cirrhosis patients at a tertiary center. Doppler parameters—including portal vein velocity, hepatic artery resistive/pulsatility indices, hepatic vein waveform, hepatic vascular index (HVI), and arterio-portal ratio (APR)—were recorded and correlated with Child-Pugh class (A/B/C), ascites, and splenomegaly.

Results: Portal vein caliber increased with worsening Child-Pugh class (A: 12.41 mm; C: 14.17 mm; $p<0.001$). Portal vein mean velocity declined significantly (A: 18.25 cm/s; C: 4.25 cm/s; $p<0.001$). Hepatic artery peak systolic velocity increased markedly (A: 62.53 cm/s; C: 125.57 cm/s; $p<0.001$), as did pulsatility index and resistive index. Hepatic vein waveform progressed from triphasic (17.6% in A) to monophasic (100% in C). APR showed a strong positive correlation with Child-Pugh score ($r=0.880$), whereas HVI showed a strong negative correlation ($r=-0.833$). Ascites severity was associated with higher arterial velocities and lower portal flow.

Conclusion: Doppler parameters across all major hepatic vessels correlate strongly with Child-Pugh class and clinical decompensation. Portal vein pulsatility index, hepatic artery pulsatility index, APR, and HVI emerge as robust non-invasive markers of disease severity.

Keywords: Cirrhosis; Doppler Ultrasound; Portal Hypertension; Hepatic Artery; Portal Vein; Hepatic Vein; Child Pugh Score.

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Introduction

Liver cirrhosis represents the end stage of chronic liver disease, characterized by diffuse fibrosis, architectural distortion, and regenerative nodule formation. It is a major cause of morbidity and mortality worldwide, with complications primarily related to portal hypertension and hepatic insufficiency.[1] Accurate assessment of disease severity is essential for prognostication and management.

The Child–Pugh (CP) score remains a widely used clinical tool for stratifying cirrhosis severity into Classes A, B, and C based on biochemical and clinical parameters.[2] Portal hypertension, the key pathophysiological feature of cirrhosis, leads to significant alterations in hepatic hemodynamics, including reduced portal vein flow, compensatory changes in hepatic arterial flow, and loss of normal hepatic vein waveform phasicity.[3–5] Although the hepatic venous pressure gradient is the gold standard for assessing portal hypertension, its invasive nature limits routine use. Colour Doppler ultrasound provides a non-invasive, accessible alternative for evaluating hepatic vascular dynamics, including portal vein velocity, hepatic artery indices, and hepatic vein waveform patterns.[6,7]

This study aims to evaluate Doppler ultrasound parameters of the hepatic vasculature in cirrhotic patients and to correlate these findings with disease severity as assessed by the Child–Pugh score.

Methods

Study Design: This analytical cross-sectional study was conducted in a tertiary care center from 2023 to 2025 after obtaining approval from the Institutional Ethics Committee.

Study Population: Patients with clinically and sonographically diagnosed liver cirrhosis presenting during the study period were included after obtaining written informed consent.

Inclusion Criteria: Patients with ultrasound features suggestive of cirrhosis, including coarse hepatic echotexture, nodular liver surface, and widened interlobar fissures, were included in the study.

Exclusion Criteria: Patients with cardiac or respiratory diseases affecting Doppler waveforms, hepatic masses, prior hepatic surgery, malignancy, or those unwilling to participate were excluded.

Sample Size: The sample size was calculated assuming a prevalence of 50% with a margin of error of 12% and 95% confidence interval, yielding a minimum of 67 patients. After accounting for a 10% attrition rate, a total of 74 patients were required. Finally, 78 patients were included using a convenience sampling technique.

Study Procedure: All eligible patients underwent detailed clinical evaluation, including history, laboratory investigations such as liver function tests, and assessment for features of hepatic decompensation including ascites, jaundice, and splenomegaly. This was followed by ultrasound and colour Doppler examination of the liver and spleen using a standardized protocol. All patients were instructed to fast for at least 6 hours prior to the examination.

All Doppler studies were performed using high-resolution ultrasound systems equipped with appropriate frequency transducers. To ensure consistency, examinations were conducted by a single experienced radiologist, and equivocal findings were reviewed by a second senior radiologist. Standardized scanning techniques and measurement protocols were followed to minimize interobserver variability.

Ultrasound and Doppler Protocol: Grayscale ultrasound was performed using a curvilinear transducer to assess liver echotexture, surface nodularity, and interlobar fissures. Splenic size was measured in the longitudinal plane, and the presence of ascites was documented.

Colour and spectral Doppler evaluation was performed via intercostal or subcostal approaches. Hepatic artery velocity was measured at the hepatic hilum with an insonation angle ≤60°. Hepatic arterial resistive index (HARI) was calculated as:

$$\mathrm{RI} = \frac{\mathrm{PSV} - \mathrm{EDV}}{\mathrm{PSV}}$$

Portal vein peak velocity was measured in cm/s with an insonation angle ≤60°, and hepatic vein waveform patterns were assessed.

Parameters Studied: The following Doppler parameters were evaluated: vessel caliber, flow direction (hepatopetal/hepatofugal), waveform pattern, average velocity, peak systolic and diastolic velocities, resistive index, pulsatility index, vein pulse index, hepatic vascular index, arterio-portal ratio, spleen size, and presence of ascites.

Statistical Analysis: Data were analyzed using IBM SPSS Statistics. Continuous variables were expressed as mean ± standard deviation, and categorical variables as frequency and percentage. Comparisons among Child–Pugh classes were performed using one-way ANOVA.

Correlations were assessed using Spearman's rank correlation. A p-value < 0.05 was considered statistically significant.

Results

A total of 78 patients with liver cirrhosis were included in the study. The mean age was 50.59 ± 10.69 years, with the majority belonging to the 51–60 years age group (51.3%). Males predominated (69.2%). Alcohol-related cirrhosis was the most common etiology (39.7%), followed by viral hepatitis (19.2%). Ascites was present in 30.8% of patients, and hepatic encephalopathy was observed in 11.6%. Most patients demonstrated biochemical derangement with elevated bilirubin (96.2%), hypoalbuminemia (91.0%), and prolonged INR (84.6%) (Table 1).

Table 1: Baseline Demographic, Clinical and Biochemical Profile of Patients with Liver Cirrhosis (n = 78)

Variable	Category	Frequency (n)	Percentage (%)
Age (years)	30–40 years	17	21.8
	41–50 years	19	24.4
	51–60 years	40	51.3
	>70 years	2	2.6
Sex	Male	54	69.2
	Female	24	30.8
Etiology of Liver Cirrhosis	Alcohol-related	31	39.7
	Viral hepatitis	15	19.2

	Others	32	41.0
Ascites (Clinical)	Present	24	30.8
	Absent	54	69.2
Grade of Ascites	Moderate	24	30.8
	Not applicable	54	69.2
Hepatic Encephalopathy	Grade 1	7	9.0
	Grade 2	2	2.6
	Absent	69	88.5
Serum Bilirubin (>1.2 mg/dL)	Abnormal	75	96.2
	Normal	3	3.8
Serum Albumin	Abnormal	71	91.0
	Normal	7	9.0
INR	Abnormal	66	84.6
	Normal	12	15.4

According to Child–Pugh classification, 21.8% of patients were in Class A, 44.9% in Class B, and 33.3% in Class C, indicating that the majority had moderate to advanced disease (Table 2).

Table 2: Distribution of Child–Pugh Class among Patients with Liver Cirrhosis (n = 78)

Child-Pugh Class	Frequency (n)	Percentage (%)
A	17	21.8
B	35	44.9
C	26	33.3
Total	78	100.0

Ultrasonographic findings showed a significant association with disease severity. Liver surface nodularity, ascites, and hepatic encephalopathy increased significantly with worsening Child–Pugh class ($p < 0.001$). Coarse echotexture was observed in nearly all patients, while splenomegaly was universally present (Table 3).

Table 3: Ultrasonography Findings in Patients with Liver Cirrhosis (n = 78)

Ultrasound Finding	CPS-A n (%)	CPS-B n (%)	CPS-C n (%)	p-value
Liver Echo Pattern				
Coarse	15 (88.2%)	35 (100%)	26 (100%)	0.025*
Homogeneous	2 (11.8%)	0 (0%)	0 (0%)	
Liver Surface Nodularity				
Present	0 (0%)	11 (31.4%)	24 (92.3%)	<0.001*
Absent	17 (100%)	24 (68.6%)	2 (7.7%)	
Splenomegaly	17 (100%)	35 (100%)	26 (100%)	--
Ascites (Clinical)				
Present (moderate grade)	0 (0%)	2 (5.7%)	22 (84.6%)	<0.001*
Absent	17 (100%)	33 (94.3%)	4 (15.4%)	
Ascites (USG)				
Mild	17 (100%)	31 (88.6%)	3 (11.5%)	<0.001*
Moderate	0 (0%)	4 (11.4%)	20 (76.9%)	
Severe	0 (0%)	0 (0%)	3 (11.5%)	
Hepatic Encephalopathy				
Grade 1	0 (0%)	2 (5.7%)	5 (19.2%)	<0.001*
Grade 2	0 (0%)	0 (0%)	2 (7.7%)	
Absent	17 (100%)	33 (94.3%)	19 (73.1%)	

Pearson Chi-square test applied. P value <0.05 was considered as statistically significant

Portal vein Doppler parameters demonstrated significant changes with disease progression. Portal vein caliber increased, whereas mean velocity, maximum systolic velocity, and pulsatility index decreased significantly with worsening Child–Pugh class ($p < 0.001$). Hepatofugal flow was observed only in advanced disease (Class C) (Table 4).

Table 4: Portal Vein Doppler Findings in Patients with Liver Cirrhosis (n = 78)

Portal Vein Parameter	CPS-A (n=17) Mean ± SD	CPS-B (n=35) Mean ± SD	CPS-C (n=26) Mean ± SD	F	p-value
Caliber (mm)	12.41 ± 0.51	12.64 ± 0.92	14.17 ± 0.98	30.06	<0.001*
Mean Velocity Vm (cm/s)	18.25 ± 2.08	11.91 ± 5.85	4.25 ± 7.30	30.75	<0.001*
Maximum Systolic Velocity Vsmax (cm/s)	24.83 ± 2.53	14.95 ± 7.42	5.80 ± 9.30	34.19	<0.001*
Pulsatility Index (VPI)	0.357 ± 0.018	0.257 ± 0.016	0.205 ± 0.008	593.10	<0.001*
Direction of Flow, n (%)					
Hepatopetal	17 (23.6%)	35 (48.6%)	20 (27.8%)	--	0.002*
Hepatofugal	0 (0%)	0 (0.0%)	6 (100.0%)	--	
Spectral Pattern, n (%)					
Monophasic	16 (94.1%)	35 (100%)	26 (100%)	--	0.162
Pulsatile	1 (5.9%)	0 (0%)	0 (0%)	--	

One-Way ANOVA test applied. P value <0.05 was considered as statistically significant

Hepatic artery Doppler parameters showed a progressive increase with disease severity. Maximum systolic velocity, diastolic velocity, mean velocity, pulsatility index, resistive index, and arterio-portal ratio all increased significantly from Child–Pugh Class A to C ($p < 0.001$) (Table 5).

Table 5: Hepatic Artery Doppler Findings in Patients with Liver Cirrhosis (n = 78)

Hepatic Artery Parameter	CPS-A (n=17) Mean ± SD	CPS-B (n=35) Mean ± SD	CPS-C (n=26) Mean ± SD	F	p-value
Max Systolic Velocity (cm/s)	62.53 ± 6.98	73.26 ± 12.00	125.57 ± 12.83	211.82	<0.001*
Diastolic Velocity (cm/s)	17.31 ± 3.92	20.95 ± 5.92	24.03 ± 1.19	11.92	<0.001*
Mean Velocity (cm/s)	38.63 ± 4.77	39.28 ± 3.09	50.66 ± 1.95	111.47	<0.001*
Pulsatility Index (PI)	1.173 ± 0.034	1.329 ± 0.124	1.998 ± 0.202	222.12	<0.001*
Resistive Index (RI)	0.740 ± 0.032	0.725 ± 0.049	0.802 ± 0.027	29.75	<0.001*
Arterio-Portal Ratio (APR)	2.293 ± 0.290	3.503 ± 0.592	8.679 ± 3.312	72.36	<0.001*

One-Way ANOVA test applied. P value <0.05 was considered as statistically significant

Hepatic vein waveform analysis demonstrated a significant shift from biphasic patterns in early disease to monophasic waveforms in advanced cirrhosis ($p < 0.001$). Hepatic vascular index showed a significant decline with increasing severity (Table 6).

Table 6: Hepatic Vein Doppler Findings in Patients with Liver Cirrhosis (n = 78)

Hepatic Vein Parameter	CPS-A (n=17)	CPS-B (n=35)	CPS-C (n=26)	χ^2	p-value
Waveform Pattern				50.13	<0.001*
Triphasic	3 (17.6%)	0 (0%)	0 (0%)		
Biphasic	14 (82.4%)	20 (57.1%)	0 (0%)		
Monophasic	0 (0%)	15 (42.9%)	26 (100%)		
HVI (mean ± SD)	15.53 ± 1.53	8.92 ± 4.68	2.25 ± 3.69		0.001*

One-Way ANOVA test applied. P value <0.05 was considered as statistically significant

Correlation analysis revealed significant associations between Doppler parameters and Child–Pugh score. Portal vein velocities and pulsatility index showed strong negative correlations, while hepatic artery parameters and arterio-portal ratio showed strong positive correlations. Hepatic vascular index demonstrated a strong negative correlation (all $p < 0.001$) (Table 7).

Table 7: Correlation between Doppler Parameters and Child Pugh Score

Doppler Parameter	Spearman r	p-value	Strength of Correlation
Portal Vein Caliber (mm)	0.598	<0.001*	Strong positive
PV Mean Velocity Vm (cm/s)	-0.773	<0.001*	Strong negative
PV Max Systolic Velocity (cm/s)	-0.770	<0.001*	Strong negative
Portal Vein Pulsatility Index (VPI)	-0.875	<0.001*	Very strong negative
HA Max Systolic Velocity (cm/s)	0.836	<0.001*	Very strong positive
HA Diastolic Velocity (cm/s)	0.458	<0.001*	Moderate positive
Hepatic Artery PI	0.857	<0.001*	Very strong positive
Hepatic Artery RI	0.445	<0.001*	Moderate positive
HA Mean Velocity (cm/s)	0.731	<0.001*	Strong positive
Arterio-Portal Ratio (APR)	0.880	<0.001*	Very strong positive
Hepatic Vascular Index (HVI)	-0.833	<0.001*	Very strong negative

Spearman Rank Correlation test applied. P value <0.05 was considered as statistically significant

Patients with ascites showed significantly altered hemodynamics, including increased portal vein caliber, reduced portal velocities, and increased hepatic arterial parameters ($p < 0.05$) (Table 8).

Table 8: Correlation between Ultrasound Findings of Hepatic Decompensation and Doppler Parameters

Doppler Parameter	Ascites Absent (Mean ± SD)	Ascites Present (Mean ± SD)	Mann–Whitney U	p-value
Portal Vein Caliber (mm)	12.69 ± 0.99	14.02 ± 0.95	234.0	<0.001*
PV Mean Velocity Vm (cm/s)	13.36 ± 6.47	4.85 ± 7.36	146.0	<0.001*
PV Max Systolic Velocity (cm/s)	17.41 ± 8.63	6.49 ± 9.28	161.5	<0.001*
Portal Vein Pulsatility Index (VPI)	0.284 ± 0.054	0.209 ± 0.014	82.5	<0.001*
Hepatic Artery Max Systolic Velocity (cm/s)	72.10 ± 15.39	124.95 ± 16.31	17.0	<0.001*
Hepatic Artery Diastolic Velocity (cm/s)	19.88 ± 5.50	24.11 ± 1.33	406.5	0.009*
Hepatic Artery Pulsatility Index (PI)	1.31 ± 0.18	1.99 ± 0.24	16.0	<0.001*
Hepatic Artery Resistive Index (RI)	0.73 ± 0.04	0.80 ± 0.04	184.5	<0.001*
Hepatic Artery Mean Velocity (cm/s)	39.68 ± 4.34	50.24 ± 3.41	48.0	<0.001*
Arterio-Portal Ratio (APR)	3.32 ± 1.15	8.67 ± 3.60	51.0	<0.001*
Hepatic Vascular Index (HVI)	10.55 ± 5.51	2.71 ± 3.88	103.0	<0.001*

Mann Whitney U test applied. P value <0.05 was considered as statistically significant

Similar trends were observed with increasing spleen size, which correlated positively with hepatic arterial parameters and negatively with portal venous flow indices (Table 9).

Table 9: Correlation between Ultrasound Findings of Hepatic Decompensation and Doppler Parameters

Doppler Parameter	Spearman r	p-value	Strength of Correlation
Portal Vein Caliber (mm)	0.429	<0.001*	Moderate positive
PV Mean Velocity Vm (cm/s)	-0.721	<0.001*	Strong negative
PV Max Systolic Velocity (cm/s)	-0.697	<0.001*	Strong negative
Portal Vein Pulsatility Index (VPI)	-0.716	<0.001*	Strong negative
Hepatic Artery Max Systolic Velocity (cm/s)	0.663	<0.001*	Strong positive
Hepatic Artery Diastolic Velocity (cm/s)	0.376	0.001*	Moderate positive
Hepatic Artery Pulsatility Index (PI)	0.651	<0.001*	Strong positive
Hepatic Artery Resistive Index (RI)	0.320	0.004*	Moderate positive
Hepatic Artery Mean Velocity (cm/s)	0.607	<0.001*	Strong positive
Arterio-Portal Ratio (APR)	0.755	<0.001*	Very strong positive
Hepatic Vascular Index (HVI)	-0.743	<0.001*	Very strong negative

Spearman Rank Correlation test applied. P value <0.05 was considered as statistically significant

Comparison across ascites severity revealed progressive increase in portal vein caliber and hepatic arterial parameters, along with a decline in portal vein velocities and hepatic vascular index ($p < 0.001$) (Table 10).

Table 10: Comparison of Doppler Parameters According to Severity of Ascites (n = 78)

Doppler Parameter	Mild Ascites (n=51) Mean ± SD	Moderate Ascites (n=24) Mean ± SD	Severe Ascites (n=3) Mean ± SD	F value	p-value
Portal Vein Caliber (mm)	12.73 ± 1.00	13.79 ± 1.15	13.83 ± 0.76	9.12	<0.001*
PV Mean Velocity Vm (cm/s)	13.31 ± 6.66	5.98 ± 8.00	5.07 ± 1.12	9.93	<0.001*
PV Max Systolic Velocity (cm/s)	17.32 ± 8.88	7.98 ± 10.20	7.13 ± 1.15	9.32	<0.001*
Portal Vein Pulsatility Index (VPI)	0.286 ± 0.055	0.215 ± 0.020	0.203 ± 0.006	21.60	<0.001*
Hepatic Artery Max Systolic Velocity (cm/s)	71.88 ± 15.82	117.82 ± 22.32	132.81 ± 16.24	62.15	<0.001*
Hepatic Artery Diastolic Velocity (cm/s)	19.64 ± 5.57	24.14 ± 1.23	23.72 ± 1.63	8.27	0.001*
Hepatic Artery Pulsatility Index (PI)	1.30 ± 0.18	1.90 ± 0.30	2.14 ± 0.28	65.92	<0.001*
Hepatic Artery Resistive Index (RI)	0.74 ± 0.04	0.78 ± 0.05	0.82 ± 0.03	10.36	<0.001*
Hepatic Artery Mean Velocity (cm/s)	39.76 ± 4.45	48.67 ± 5.23	50.92 ± 1.70	34.54	<0.001*
Arterio-Portal Ratio (APR)	3.33 ± 1.18	7.50 ± 3.56	12.54 ± 3.00	45.70	<0.001*
Hepatic Vascular Index (HVI)	10.55 ± 5.67	3.71 ± 4.67	2.41 ± 0.71	15.35	<0.001*

One-Way ANOVA test applied. P value <0.05 was considered as statistically significant

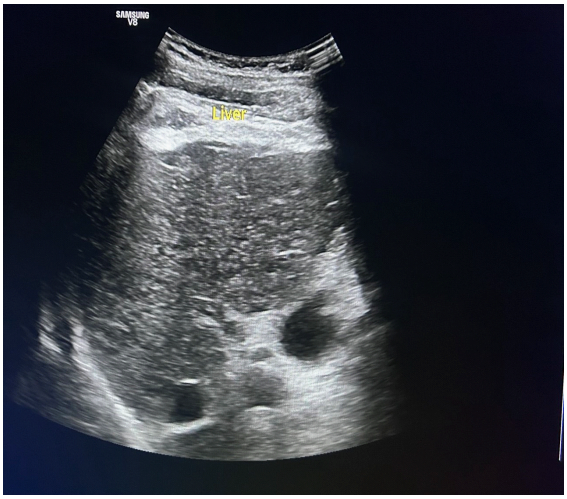


Figure 1(a)

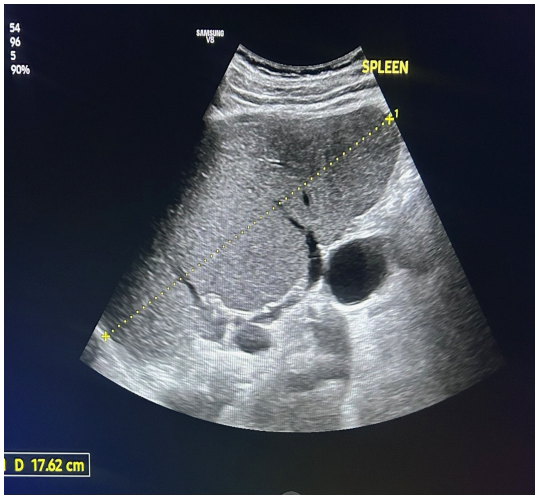


Figure 1(b)

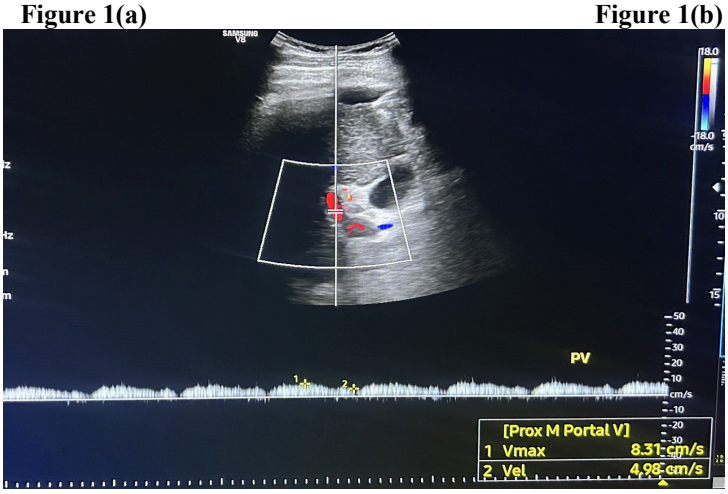


Figure 1(c)

Figure 1: Ultrasound abdomen in a 40-year-old male with alcoholic liver disease (Child Pugh Class B) (a) Grey scale ultrasound shows nodular, coarse echopattern of liver suggestive of cirrhosis; (b) Longitudinal image demonstrates splenomegaly (17.6 cm); (c) Spectral doppler of the portal vein demonstrate hepatopetal flow with monophasic waveform and decreased velocity.

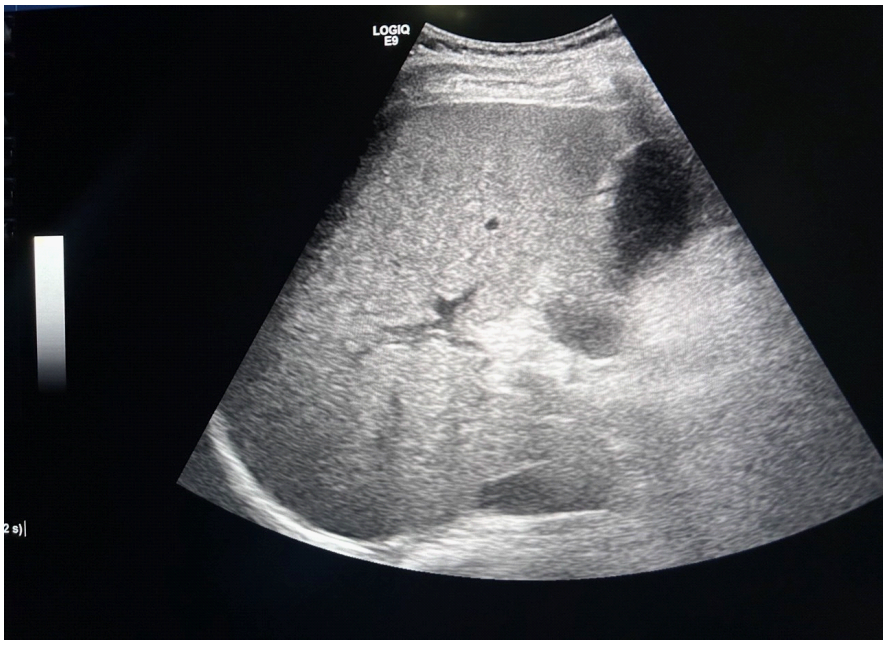


Figure 2(a)

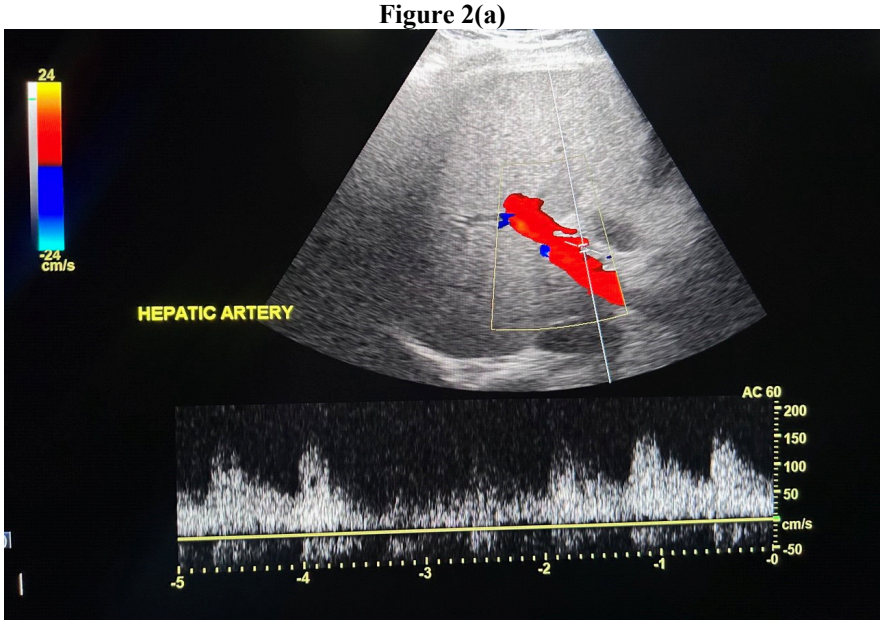


Figure 2(b)

Figure 2: Ultrasound abdomen with hepatic artery doppler in a 50-year-old female patient with viral chronic liver disease (Child Pugh Class C) (a) Grey scale ultrasound image shows coarse echopattern of liver ; (b) spectral doppler of the hepatic artery demonstrates elevated peak systolic velocity.

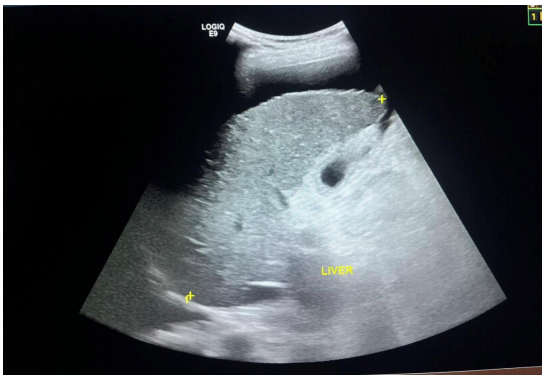


Figure 3(a)

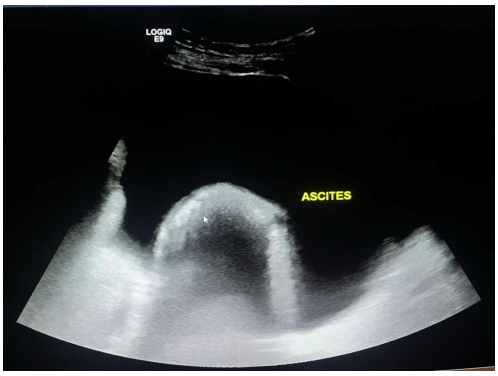
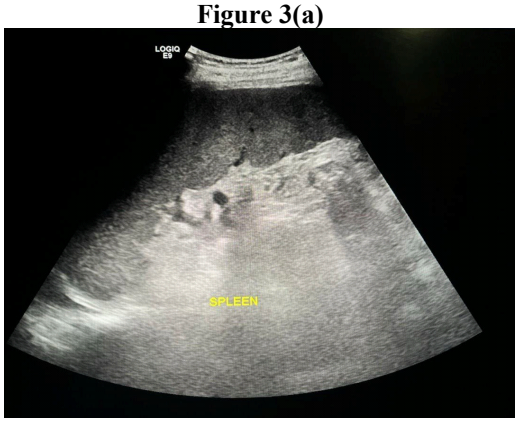


Figure 3(b)



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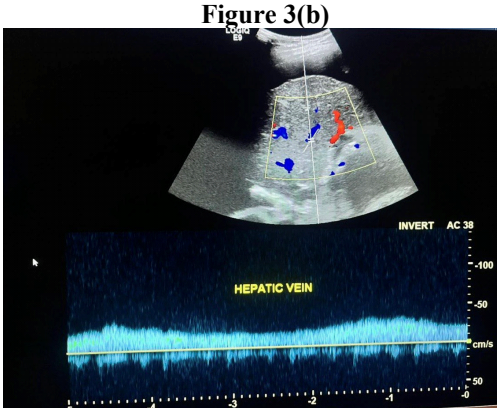

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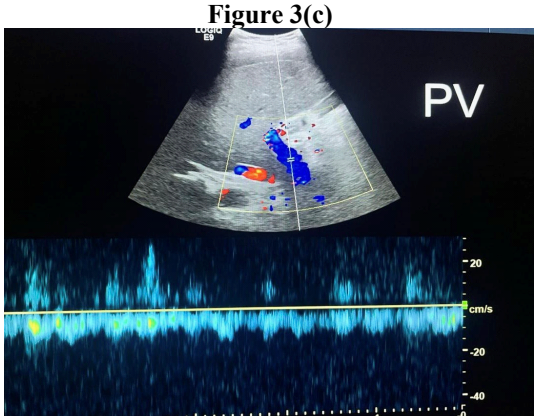

Figure 3(e)

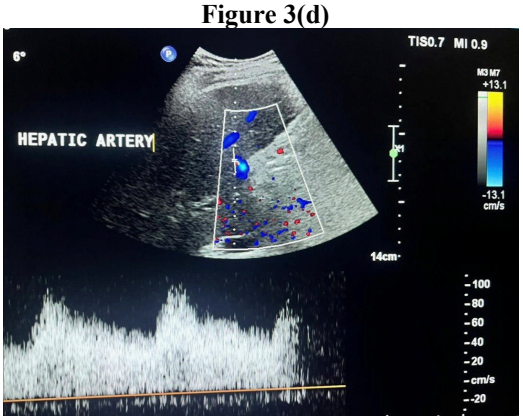

Figure 3(f)

Figure 3: Ultrasound abdomen with liver doppler in a 40 year-old male patient with alcoholic liver disease (Child Pugh Class C)) The grayscale ultrasonography image of the abdomen demonstrates (a) liver with coarse echopattern and surface nodularity suggestive of liver cirrhosis. (b) Anechoic free fluid is visualized in the peritoneal cavity, consistent with ascites. (c)The spleen is enlarged, extending beyond its normal dimensions. (d)Spectral Doppler evaluation of the hepatic vein shows loss of the normal triphasic waveform pattern, with a monophasic waveform noted. (e)Doppler assessment of the portal vein demonstrates reversed (hepatofugal) flow. (f)Spectral analysis of the hepatic artery reveals elevated peak systolic velocity.

Discussion

The present study demonstrates significant alterations in hepatic hemodynamics across the portal vein, hepatic artery, and hepatic vein with increasing severity of liver cirrhosis. These Doppler changes showed strong correlations with Child–Pugh class and features of portal hypertension, highlighting the utility of colour Doppler ultrasound as a non-invasive tool for disease assessment. Portal vein parameters showed progressive dilatation with a significant reduction in flow velocities and pulsatility index as disease severity increased.

These findings are consistent with previous studies, which have reported decreased portal venous flow due to increased intrahepatic resistance and development of portosystemic collaterals. The presence of hepatofugal flow exclusively in advanced disease further reflects severe portal hypertension.

Hepatic artery Doppler parameters demonstrated a compensatory increase in velocities and indices with worsening cirrhosis, consistent with the hepatic arterial buffer response. The arterio-portal ratio showed a strong positive correlation with disease severity, indicating a shift from portal to arterial dominance in hepatic perfusion. The observed variations in resistive index across stages reflect the complex interplay between vascular resistance and compensatory mechanisms.

Hepatic vein waveform analysis revealed a progressive loss of normal phasicity from triphasic to monophasic patterns with advancing disease, reflecting reduced hepatic compliance due to fibrosis. The hepatic vascular index showed a strong negative correlation with Child–Pugh score, emphasizing its value as a composite indicator of hepatic hemodynamic changes.

Additionally, Doppler parameters showed significant associations with markers of hepatic decompensation such as ascites and splenomegaly. Patients with ascites demonstrated reduced portal venous flow and increased hepatic arterial parameters, indicating advanced portal hypertension and hemodynamic compromise.

Overall, the findings are consistent with existing literature and support the role of Doppler ultrasound in evaluating the severity of cirrhosis. The integration of portal, arterial, and venous parameters provides a comprehensive assessment of hepatic hemodynamics.

Limitations

This study is limited by its cross-sectional design, modest sample size, and lack of correlation with invasive measurements such as hepatic venous pressure gradient. Additionally, operator dependency of Doppler ultrasound may influence measurements.

Conclusion

Colour Doppler ultrasound demonstrates significant and progressive alterations in hepatic hemodynamics that correlate strongly with the severity of liver cirrhosis as assessed by the Child–Pugh score. Portal vein parameters show reduced flow and increased caliber, hepatic artery parameters demonstrate compensatory increases, and hepatic vein waveforms show loss of phasicity with advancing disease.

Composite indices such as arterio-portal ratio and hepatic vascular index serve as reliable markers of disease severity. Thus, Doppler ultrasound provides a valuable, non-invasive modality for assessing and monitoring cirrhosis and its complications.

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