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Original Research Article

Assessment of Renal Hemodynamics Using Renal Artery Resistive Index and Its Association with EGFR in Radiologically Diagnosed Chronic Liver Disease

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Abstract

Background: Chronic liver disease (CLD) is frequently associated with renal dysfunction due to progressive portal hypertension and systemic haemodynamic alterations. Renal artery resistive index (RARI), assessed by Doppler ultrasonography, may serve as an early marker of renal haemodynamic impairment, while estimated glomerular filtration rate (eGFR) remains the standard measure of renal function.

Methods: This cross-sectional, observational, and correlational study was conducted in the Department of Radiodiagnosis at Dr. D.Y. Patil Medical College Hospital and Research Institute, Kolhapur, over a period of 18 months. Sixty patients with radiologically diagnosed chronic liver disease were included. Patients with known chronic kidney disease and spontaneous peritoneal infection were excluded. All participants underwent abdominal ultrasonography and renal Doppler evaluation. RARI was calculated from interlobar arterial waveforms obtained from both kidneys, and eGFR was determined using serum creatinine-based estimation. Correlations between RARI, renal function parameters, and portal haemodynamic variables were analysed.

Results: The study population predominantly comprised middle-aged males. Renal function ranged from normal to severely impaired. RARI demonstrated a strong positive correlation with serum creatinine and a strong negative correlation with eGFR, indicating increased renal vascular resistance with declining renal function. RARI showed weak positive correlations with portal vein diameter and spleen size, while a moderate negative correlation was observed with portal vein mean velocity. Patients with retrograde portal venous flow and abnormal hepatic vein waveforms exhibited significantly higher RARI values. Although patients with ascites showed higher RARI and lower eGFR values, these differences were not statistically significant.

Conclusion: Renal artery resistive index is significantly associated with renal functional status and portal haemodynamic alterations in chronic liver disease. Doppler-derived RARI may serve as a useful non-invasive marker for early detection and monitoring of renal impairment and may help identify patients at increased risk of hepatorenal complications.

Keywords: Chronic liver disease, Renal artery resistive index (RARI), eGFR, Doppler ultrasonography, Portal hypertension, Renal dysfunction, Hepatorenal syndrome.

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Introduction

Chronic Liver Disease (CLD) constitutes a significant global health challenge, defined by gradual hepatocellular injury and fibrosis over extended periods.[1] This protracted process frequently arises from diverse etiologies, encompassing viral hepatitis (Hepatitis B and C), sustained alcohol consumption, non-alcoholic fatty liver disease (NAFLD) advancing to non-

alcoholic steatohepatitis (NASH), autoimmune conditions, and inherited metabolic disorders, ultimately culminating in cirrhosis.[1,2] Cirrhosis, the end-stage of chronic hepatic injury, is histologically identified by widespread fibrosis and the formation of regenerative nodules, which deform the liver's normal architecture and compromise its function.[1] The clinical manifestation of CLD exhibits broad variability, ranging from an asymptomatic state to advanced decompensation characterized by complications such as ascites, hepatic encephalopathy, variceal hemorrhage, and hepatocellular carcinoma.[2] The increasing incidence of CLD, particularly NAFLD/NASH, underscores the critical need to understand its systemic complications and to formulate effective strategies for its early diagnosis and management.[1]

Renal dysfunction represents one of the most serious and frequently observed complications in patients with advanced CLD, particularly those with cirrhosis. [2,3] The liver and kidneys are intricately connected through complex circulatory and metabolic pathways, establishing a close anatomical and physiological interdependence. [4] Consequently, hepatic dysfunction often profoundly impacts renal function. Renal involvement in CLD can manifest along a spectrum, from subtle subclinical changes to severe, life-threatening conditions such as acute kidney injury (AKI) and hepatorenal syndrome (HRS).[2,6] The development of renal dysfunction significantly exacerbates the prognosis for patients with CLD, leading to increased morbidity, mortality, and complexities in treatment.[2]

Hepatorenal syndrome (HRS) represents a severe and specific form of acute kidney injury observed in individuals with advanced cirrhosis and ascites.[2,3] Its hallmark is pronounced renal vasoconstriction occurring concurrently with systemic and splanchnic vasodilation.[4] HRS is classified as a functional renal failure, given the absence of intrinsic renal parenchymal damage.[2] The pathogenesis of HRS is intricate and multifactorial, primarily attributed to extensive splanchnic vasodilation, which diminishes the effective arterial blood volume.[2,4] This reduction subsequently triggers compensatory vasoconstrictor mechanisms, such as the renin-angiotensin-aldosterone system (RAAS), the sympathetic nervous system, and the release of antidiuretic hormone.[2] While these responses initially serve as compensatory mechanisms, their sustained activation ultimately induces intense renal vasoconstriction, resulting in a substantial reduction in renal blood flow and glomerular filtration rate (GFR).[4] Distinguishing HRS from other etiologies of AKI in CLD patients holds significant clinical importance due to the divergent management strategies.[3] Moreover, a substantial proportion of CLD patients exhibit subclinical renal hemodynamic alterations prior to the onset of overt HRS, rendering the early identification of renal impairment a critical clinical objective.[6]

The assessment of renal function typically relies on serum creatinine levels, from which the estimated glomerular filtration rate (eGFR) is derived.[8] The eGFR is a recognized and broadly accepted indicator for evaluating renal function and for quantifying the kidney's capacity to eliminate metabolic waste products from the circulation.[8] Standard equations employed for eGFR calculation include the Modification of Diet in Renal Disease (MDRD) equation and the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation, each integrating variables such as serum creatinine, age, sex, and race.[8] While eGFR holds a pivotal position in the diagnosis and staging of chronic kidney disease (CKD), its dependability in patients with advanced liver disease, especially cirrhosis, frequently proves to be constrained.[8] This constraint primarily stems from artifactually low serum creatinine concentrations, which arise from diminished hepatic creatine synthesis, muscle atrophy, and augmented tubular secretion of creatinine in CLD patients.[8] As a result, apparently normal serum creatinine values may obscure underlying renal dysfunction, leading to delayed diagnosis and intervention.[8] These inherent limitations thus underscore the necessity for complementary methodologies to evaluate renal function and hemodynamics in individuals with CLD.

Within this context, Doppler ultrasonography has become recognized as a valuable non-invasive imaging technique for assessing renal vascular resistance and perfusion. [5,7] The Renal Artery Resistive Index (RARI), derived from Doppler measurements of renal arterial blood flow, offers crucial insights into intrarenal hemodynamics. [7] RARI is computed from peak systolic velocity (PSV) and end-diastolic velocity (EDV) using the following formula:

$$RARI = (PSV - EDV) / PSV.$$

An elevated RARI signifies heightened vascular resistance within the renal circulation, implying compromised renal perfusion and potentially indicating renal dysfunction or vasoconstriction.[7] Prior research has explored the utility of RARI across various nephrological conditions, illustrating its predictive capacity for disease progression and therapeutic outcomes.[7]

Given its non-invasive character and its direct reflection of renal vascular alterations, RARI holds potential as an early indicator of subtle renal hemodynamic irregularities in CLD patients, even before substantial changes in serum creatinine or eGFR become discernible. [7]

Notwithstanding the growing recognition of renal complications in CLD and the established importance of both eGFR and RARI in renal assessment, the precise relationship between these two parameters in patients with radiologically confirmed chronic liver disease remains underexplored.[7,8] Radiological imaging techniques, including ultrasonography, computed tomography (CT), and magnetic resonance imaging (MRI), are instrumental in the diagnosis and staging of CLD.[5] They facilitate the detection of characteristic features such as a nodular liver surface, portal hypertension, splenomegaly, and ascites, often preceding overt clinical decompensation.[5] The inclusion of patients with radiologically diagnosed CLD enables the study of a wider patient cohort, encompassing individuals who may not yet exhibit obvious clinical signs or significant biochemical abnormalities. Consequently, investigating the association between RARI and eGFR within this population carries substantial clinical relevance. [7] Should a significant correlation be identified, RARI could emerge as a valuable adjunct to eGFR, offering a non-invasive, real-time appraisal of renal vascular

changes that might precede a decline in GFR. [7] Such an insight could aid in identifying patients at elevated risk for severe renal complications, including HRS, thereby facilitating earlier therapeutic interventions and more effective management protocols. [2,6]

The principal objective of the current study is to assess the relationship between the renal artery resistive index and the estimated glomerular filtration rate in individuals with chronic liver disease identified through radiological imaging. Through this investigation, the study seeks to ascertain whether RARI can serve as an early, non-invasive indicator of renal dysfunction in these patients, thereby complementing established markers like eGFR. [7,8] Furthermore, this research aims to evaluate how varying stages and etiologies of radiologically diagnosed CLD might influence this observed association. The anticipated findings are expected to enhance our comprehension of renovascular changes in CLD and could inform the development of improved diagnostic methods and prognostic evaluations for renal complications in this patient cohort.[7] Ultimately, the integration of functional parameters such as eGFR with hemodynamic metrics like RARI has the potential to offer a more holistic assessment of renal status in CLD patients, conceivably facilitating earlier intervention, ameliorating clinical outcomes, and diminishing the morbidity and mortality associated with renal dysfunction in chronic liver disease.[2,8]

Methodology

A Cross-sectional, observational and Correlational study was conducted at Dr. D.Y. Patil Medical College Hospital and Research Institute, Kadamwadi, Kolhapur on Patients referred from Department of Medicine for USG Abdomen and pelvis to the Department of Radiodiagnosis, over a period of 18 months.

A written consent/assent was obtained and the detailed history of the patient was taken. The study was started after getting ethical approval from The Institutional Ethics Committee. The participants were selected according to the inclusion and exclusion criteria. The sample size was 60.

Inclusion Criteria: Patient giving consent, radiologically diagnosed cases of chronic liver disease.

Exclusion Criteria: Patients with K/C/O chronic kidney disease, Spontaneous peritoneal infection.

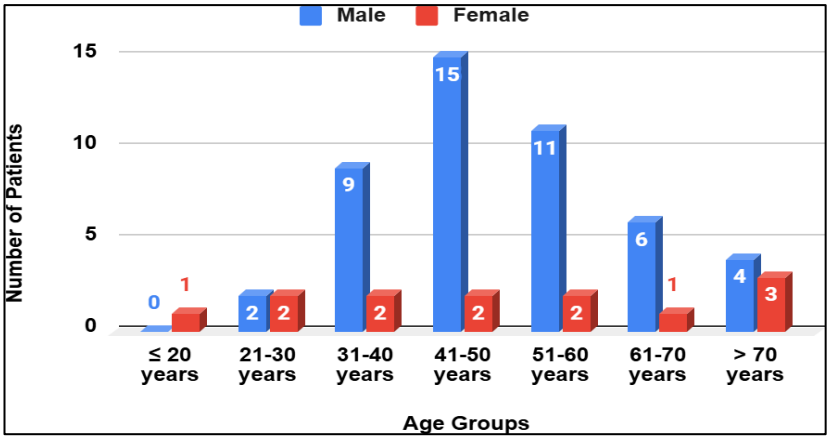
Method

Abdominal ultrasonography and renal Doppler ultrasonography was performed according to the standard protocol using GE Logiq F8, F8 Expert and P10 ultrasound machines with a low-frequency curvilinear transducer (2–5 MHz). For renal Doppler examination, patients were advised to fast for 8 hours before the procedure. The transducer was positioned to obtain visualization of the lateral or posterolateral aspect of the kidney. This approach allowed Doppler assessment at an optimal insonation angle of 0°–60°, facilitating proper evaluation of vascular structures at the peripheral hilum while minimizing interference from bowel gas.

The patient was examined in the decubitus or semi-decubitus position, ensuring inclusion of the kidney being evaluated along with the abdominal aorta in the imaging field. The lateral edge of the transducer was slightly tilted toward the caudal direction to obtain suitable visualization of the course of the main renal artery and vein. The renal artery resistive index (RARI) was calculated automatically by the ultrasound system. Intrarenal vascular resistance was assessed by measuring interlobar arterial waveforms three times at different regions of each kidney, namely the upper, middle, and lower poles, and the average

value was recorded. Finally, the overall mean resistive index for each subject was determined by calculating the mean RARI of both kidneys.

Results



Graph 1: Age and Sex Distribution of Study Participants

The Graph shows the distribution of participants in different age groups with their gender. A total of 60 patients were included in the study, of which 47 were males and 13 were females, indicating a clear male predominance. Majority of the patients were in the age group of 41-50 years (28.3%) followed by 51-60 years (21.7%) and 31-40 years (18.3%). Only a small proportion of patients were observed in the younger age groups, i.e., 6.7% in the 21–30years group and 1.7% below 20 years. Among the older population, 11.7% of patients each were seen in the 61–70 years and above 70years categories. These findings indicate that most of the study participants were middle-aged adults, with fewer cases observed at the extremes of age.

Table 1: Distribution of Clinical and Laboratory Parameters

Parameters	Mean ± SD	Range
Serum Creatinine level	1.51 ± 0.80	0.5 – 4.0
eGFR	66.92 ± 31.43	16 – 119
RARI	0.72 ± 0.13	0.50 – 0.98

The table shows the summary statistics of important clinical and laboratory parameters assessed in the study population. The mean serum creatinine level was 1.51 ±0.80 mg/dL, with values ranging from 0.5 to 4.0 mg/dL, indicating variation in renal function among the participants. The mean estimated glomerular filtration rate (eGFR) was 66.92 ±31.43 ml/min/1.73m², with a wide range from 16 to 119 ml/min/1.73m², suggesting that patients had varying degrees of kidney function. The mean Renal Artery Resistive Index (RARI) was 0.72 ±0.13, with values ranging between 0.50 and 0.98. These findings demonstrate variability in renal hemodynamic and functional parameters among the study participants. Overall, the results indicate that both biochemical and Doppler parameters showed a broad distribution within the population studied.

Table 2: Distribution of Organ Size Measurements

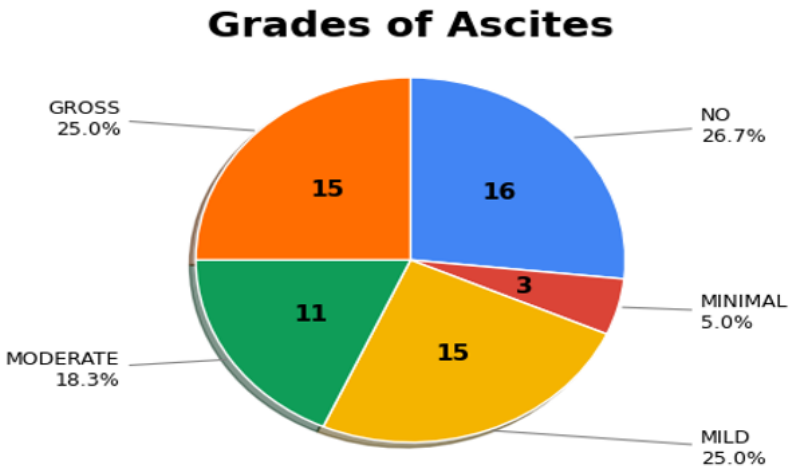
Parameters	Mean ± SD	Range
Liver Size (cm)	13.51 ± 2.36	8.6 – 19.8
Spleen Size (cm)	13.67 ± 2.63	8.2 – 20

The table presents the summary statistics of liver and spleen size measured in the study population. The mean size of the liver was 13.51 ±2.36 cm, with values ranging from 8.6 to 19.8 cm, indicating variability in liver dimensions among participants. Similarly, the mean spleen size was 13.67 ±2.63 cm, with a range between 8.2 and 20 cm. The relatively wide range observed in both liver and spleen measurements suggests differences in organ enlargement among the study subjects. Some patients showed measurements within normal limits, while others demonstrated increased organ size. Overall, these findings reflect the variation in hepatosplenic involvement within the study population.

Graph shows the distribution of severity of ascites in study participants. Among total patients, 16

patients (26.66%) were found to have no ascites on evaluation. The smallest subgroup was represented by 3 patients (5%) with minimal ascites.

Mild ascites was seen in 15 (25%) patients while gross ascites was seen in equal proportion (25%) of patients. Moderate ascites was found in 11 patients (18.33%) of the study population. Results showed that the majority of the patients had ascites of varied severity with mild and gross ascites being the commonest categories in general.

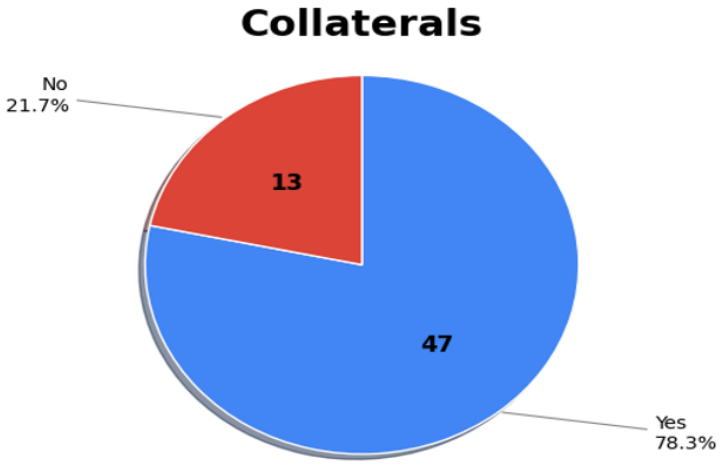


Graph 2: Grades of Ascites

Table 3: Portal and Splenic Venous Doppler Measurements in Study Participants

Parameters	Mean ± SD	Range
Portal Vein Diameter (mm)	11.6 ± 2.29	6 - 17
Splenic Vein Diameter (mm)	8.94 ± 2.74	4.4 - 16
Portal Vein Mean Velocity (cm/s)	24.12 ± 7.03	10 - 36

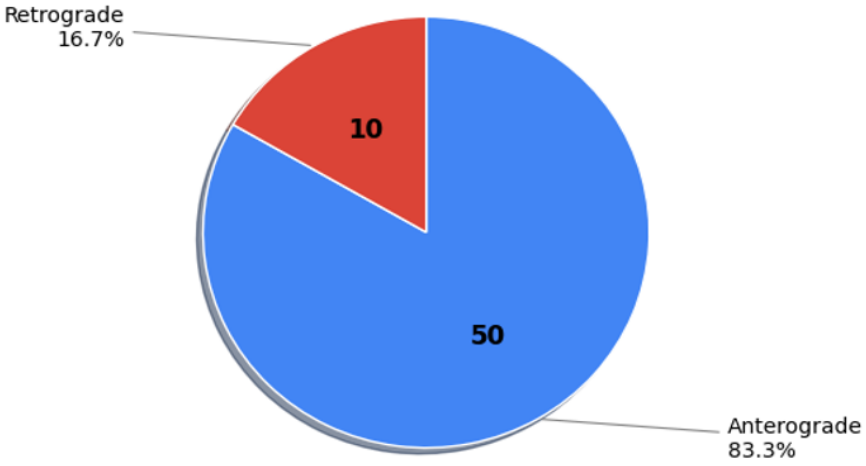
The table presents the summary statistics of portal venous system parameters evaluated in the study population. The mean portal vein diameter was 11.6 ±2.29 mm, with a range from 6 to 17 mm, indicating variation in vessel size among the participants.



Graph 3: Collaterals

The splenic vein diameter had a mean value of 8.94 ±2.74 mm, with measurements ranging between 4.4 and 16 mm. The mean portal vein mean velocity was recorded as 24.12 ±7.03cm/s, with values ranging from 10 to 36 cm/s. The variability observed in these measurements reflects differences in portal venous hemodynamics within the study group. Overall, these findings provide an overview of the Doppler characteristics of the portal venous system in the participants studied.

Distribution of portosystemic collaterals in study participants is shown in above mentioned Graph. On evaluation collaterals were seen in majority of patients 47 (71.7%). Meanwhile, 13 patients (28.3%) did not develop collateral formation. Many patients have collaterals indicating that vascular changes related to portal hypertension occur in the study population. Such findings suggest that collateral circulation was a frequently observed phenomenon among the participants included in the study. In conclusion, the results indicate a high frequency of portosystemic collateral formation in this patient population.



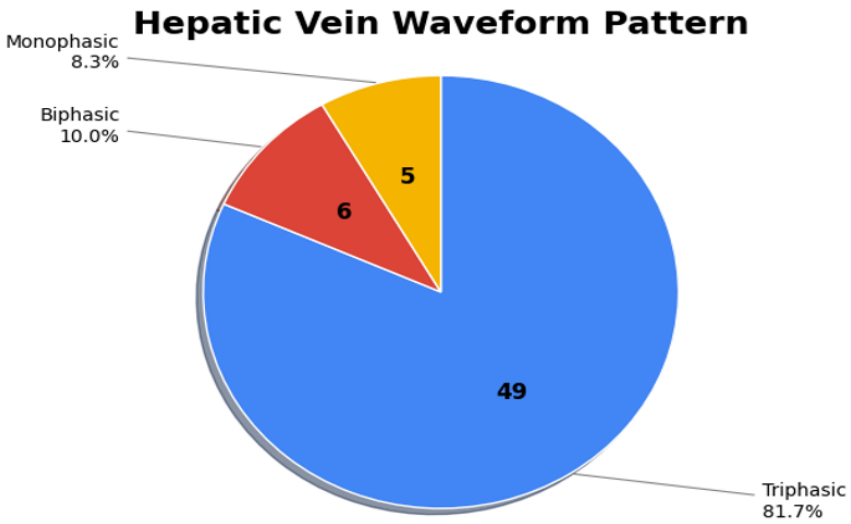
Graph 4: Direction of Portal Vein Blood Flow

The Graph shows the distribution of the direction of portal vein blood flow among the study participants. Anterograde flow was observed in the majority of patients, accounting for 50 individuals (83.30%) in the study. Retrograde flow was seen in 10 patients (16.70%), representing a smaller proportion of the population. The predominance of anterograde flow indicates that most patients maintained the normal direction of portal venous circulation. However, the presence of retrograde flow in some patients suggests altered portal hemodynamics. Overall, these findings demonstrate variation in portal vein blood flow patterns among the study participants.

Table 4: Hepatic Artery Doppler Parameters in Study Participants

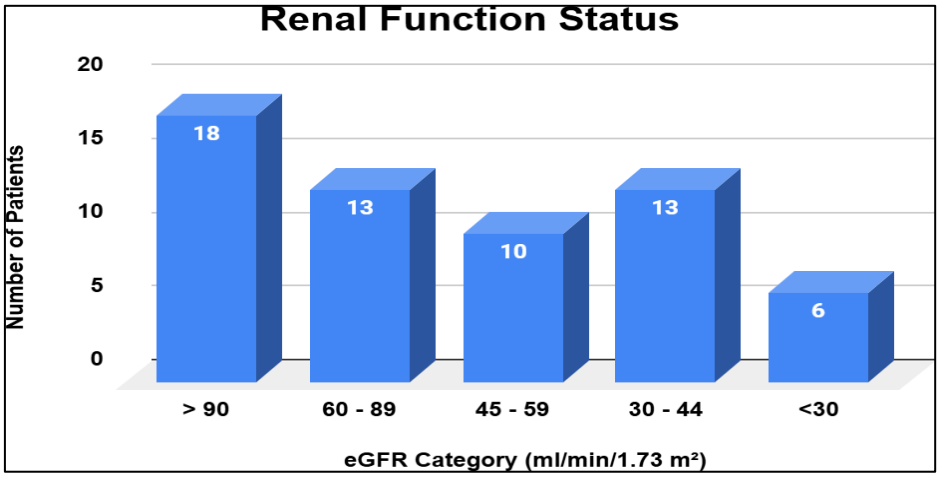
Parameters	Mean ± SD	Range
Hepatic Artery Peak Systolic Velocity (cm/s)	81.57 ± 26.29	10 - 128
Hepatic Artery Resistive Index	0.72 ± 0.09	0.54 - 0.9

The table shows the summary statistics of hepatic artery Doppler measurements observed in the study population. The mean hepatic artery peak systolic velocity was 81.57 ±26.29 cm/s, with values ranging from 10 to 128 cm/s, indicating variability in arterial flow velocity among the participants. The hepatic artery resistive index had a mean value of 0.72 ±0.09, with a range between 0.54 and 0.9. The wide range observed in peak systolic velocity suggests differences in hepatic arterial hemodynamics within the study group. Similarly, variations in the resistive index reflect changes in vascular resistance in the hepatic artery. Overall, these findings provide an overview of hepatic arterial Doppler characteristics in the participants included in the study.



Graph 5: Hepatic Vein Waveform Patterns

The Graph shows the distribution of hepatic vein waveform patterns observed on Doppler evaluation among the study participants. The majority of patients, 49 (81.7%), demonstrated a triphasic waveform pattern, which is considered the normal hepatic venous flow pattern. A biphasic waveform was observed in 6 patients (10.0%), indicating mild alteration in hepatic venous flow dynamics. Monophasic waveform was seen in 5 patients (8.3%), representing the least common pattern in the study. The presence of biphasic and monophasic waveforms in some patients suggests changes in hepatic venous hemodynamics. Overall, the findings indicate that most participants maintained a normal triphasic hepatic vein waveform pattern.

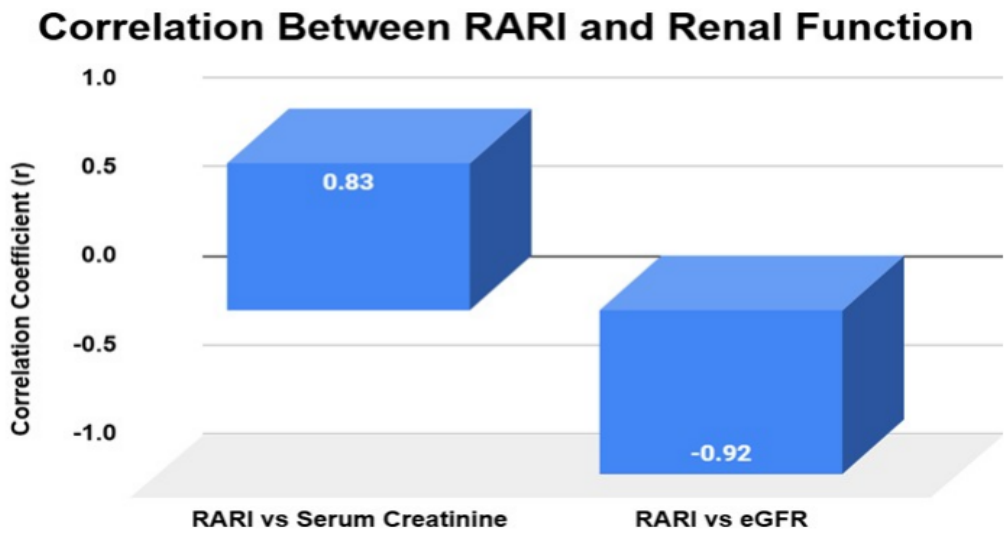


Graph 6: Renal Function Status

The table shows the distribution of study participants based on eGFR categories and corresponding renal function status. A total of 18 patients (30%) had normal renal function with eGFR values greater than 90 ml/min/1.73 m². Mild decrease in renal function was observed in 13 patients (21.70%) with eGFR between 60 and 89 ml/min/1.73 m². Mild to moderate decrease was noted in 10 patients (16.60%) in the 45–59 ml/min/1.73 m² category; 13 patients (21.70%) had moderate to severe decrease in renal function with eGFR between 30 and 44 ml/min/1.73 m². Severe reduction in renal function with eGFR less than 30 ml/min/1.73 m² was seen in 6 patients (10%). Finally, the results showed that a considerable number of patients had different degrees of renal dysfunction.

Table 5: Correlation of RARI with Renal Function Parameters

Variables	Correlation Coefficient (r)	p-value
RARI vs Serum Creatinine	0.83	<0.001
RARI vs eGFR	-0.92	<0.001



Graph 7: Correlation of RARI with Renal Function

Table shows correlation analysis of Renal Artery Resistive Index (RARI) with renal function parameters in the study population. A significant positive correlation was observed between RARI and serum creatinine (r = 0.83) which was statistically highly significant with a p value of <0.001. This indicates a correlation of higher renal arterial resistance with higher serum creatinine. Conversely, a significant inverse correlation was observed between RARI and eGFR (r = -0.92), which was also highly significant statistically with p <0.001.

This means that the RARI value increases with decreasing eGFR. The present results show a strong relationship between Doppler-based renal vascular parameters and biochemical markers of renal function. In conclusion, the results suggest the clinical significance of RARI for the evaluation of changes in renal function.

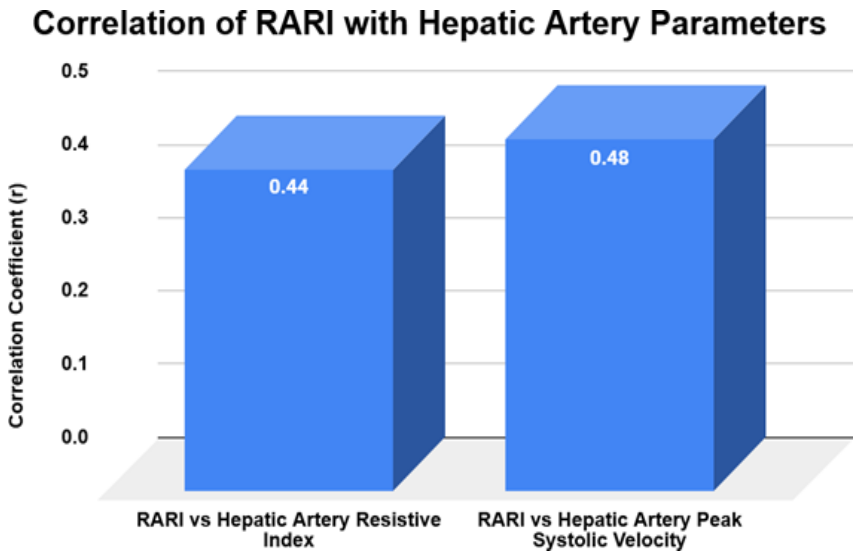
Table 6: Correlation of RARI with Portal Hemodynamic and Splenic Parameters

Variables	Correlation Coefficient (r)	p-value
RARI vs Portal Vein Diameter	0.04	0.766
RARI vs Portal Vein Mean Velocity	-0.49	<0.001
RARI vs Spleen Size	0.19	0.156

Table presents correlation analysis between renal artery resistive index (RARI) and selected portal haemodynamic and splenic parameters in study participants. Very weak positive correlation between RARI and portal vein diameter (r = 0.04) was seen, which was not statistically significant (p = 0.766). There was a moderate negative correlation between RARI and mean portal vein velocity (r = -0.49) which was statistically highly significant (p < 0.001). This suggests an association between lower portal vein velocity and higher RARI values. A weak positive correlation was observed between RARI and spleen size (r = 0.19), which was also not statistically significant (p = 0.156). Overall, these findings suggest that among the studied parameters, portal vein mean velocity showed a significant association with RARI, while portal vein diameter and spleen size did not show significant correlation.

Table 7: Correlation of RARI with Hepatic Artery Doppler Parameters

Variables	Correlation Coefficient (r)	p-value
RARI vs Hepatic Artery Resistive Index	0.44	0.001
RARI vs Hepatic Artery Peak Systolic Velocity	0.48	<0.001



Graph 8: Correlation of RARI with Hepatic Artery Doppler Parameters

Table shows the correlation analysis between Renal Artery Resistive Index (RARI) and hepatic artery Doppler parameters in study subjects. We found a moderate positive correlation between RARI and hepatic artery resistive index (r=0.44) which was statistically significant (p=0.001). Similar positive correlation was observed between RARI and hepatic artery peak systolic velocity (r = 0.48) which is statistically highly significant (p < 0.001). These results indicate that increased hepatic arterial resistance and increased peak systolic velocity are correlated with increased renal arterial resistance. The results suggest a relationship between renal and hepatic vascular hemodynamics in the study population. Overall, the correlations demonstrate a significant association between RARI and hepatic artery Doppler parameters.

Table 8: Comparison of Mean RARI According to Presence of Ascites

Parameter	Ascites Present	Ascites Absent	p-value
Mean RARI	0.74	0.68	0.107
SD	0.12	0.14	

Table shows the comparison of Renal Artery Resistive Index (RARI) in patients with and without ascites. The mean RARI was slightly higher in patients with ascites (0.74) than in those without ascites (0.68). In both groups (with and without ascites) there was some variation with a standard deviation of 0.12 (patients with ascites) and 0.14 (patients without ascites). However, the difference between the two groups was not statistically significant (p-value of 0.107). This indicates that RARI is generally higher in patients with ascites, but not statistically significant in the present study. Overall, the results suggest that the presence of ascites did not significantly alter RARI values.

Table 9: Comparison of Mean eGFR According to Presence of Ascites

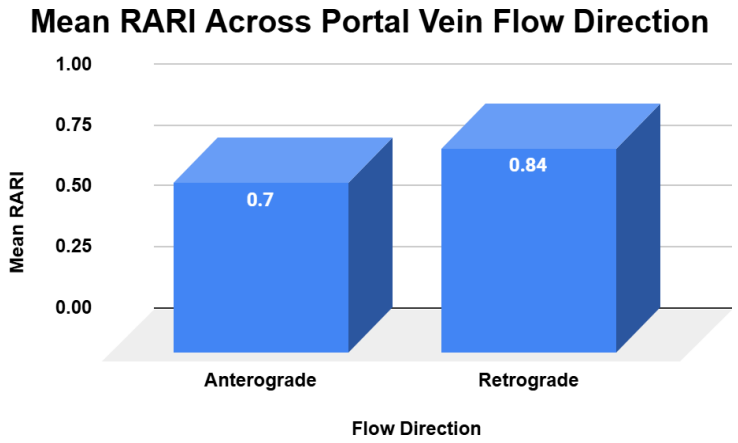
Parameter	Ascites Present	Ascites Absent	p-value
Mean eGFR	62.73	78.44	0.087
SD	30.96	30.79	—

The estimated glomerular filtration rate (eGFR) in patients with and without ascites is presented in Table 9. Mean eGFR was lower in patients with ascites (62.73 ml/min/1.73 m2) as compared to those without ascites (78.44 ml/min/1.73 m2). The standard deviation was 30.96 for patients with ascites and 30.79 for those without ascites, indicating similar variation in both groups. Renal function was relatively lower in patients with ascites, but the difference between the two groups was not statistically significant (p = 0.087). However, the absence of strong statistical

evidence in this study suggests a trend toward lower eGFR in patients with ascites. Overall, the findings suggested a possible association between ascites and impaired renal function that was not statistically significant.

Table 10: Comparison of Mean RARI According to Portal Vein Flow Direction

Flow Direction	Mean RARI	SD	p-value
Anterograde	0.70	0.13	0.001
Retrograde	0.84	0.08	

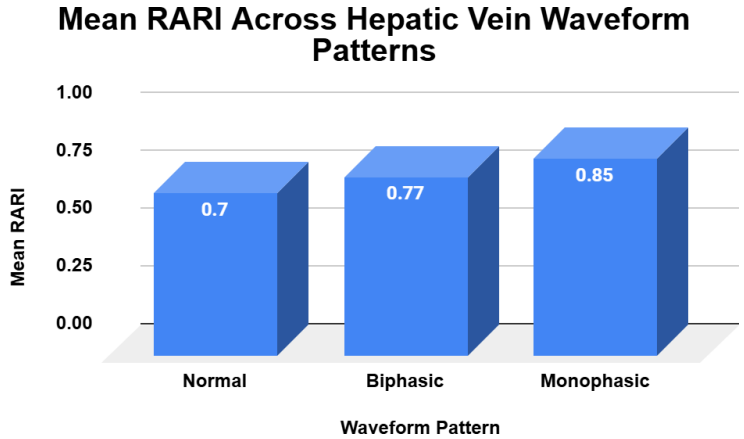


Graph 9: Mean RARI According to Portal Vein Flow Direction

Table shows comparison of renal artery resistive index (RARI) according to direction of portal vein blood flow among the study participants. Mean RARI was lower in patients with anterograde portal vein flow (0.70) than in those with retrograde flow (0.84). There was some variability within the groups as indicated by the standard deviation of 0.13 in the anterograde group and 0.08 in the retrograde group. The difference between the two groups was significant (p = 0.001). This finding indicates that those patients with retrograde portal vein flow are more likely to have an increased renal arterial resistance. The results, in general, show a significant association of portal vein flow direction with RARI values.

Table 11: Comparison of Mean RARI According to Hepatic Vein Waveform Pattern

Waveform Pattern	Mean RARI	SD	p-value
Normal	0.70	0.13	0.031
Biphasic	0.77	0.11	
Monophasic	0.85	0.07	



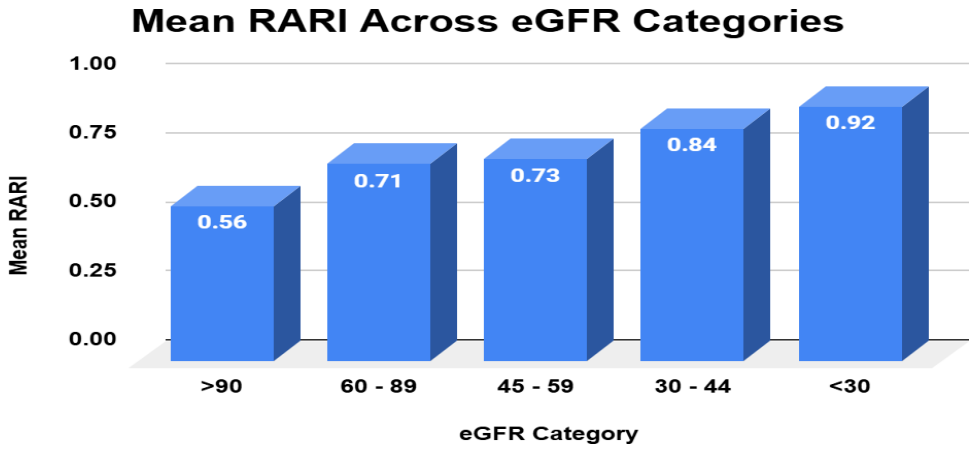
Graph 10: Mean RARI According to Hepatic Vein Waveform Pattern

The table shows the comparison of Renal Artery Resistive Index (RARI) among different hepatic vein waveform patterns observed in the study participants. Patients with a normal (triphasic)

waveform pattern had a mean RARI of 0.70 with a standard deviation of 0.13. Higher mean RARI values were observed in patients with biphasic waveform (0.77 ±0.11) and monophasic waveform (0.85 ±0.07). This indicates a gradual increase in renal arterial resistance as the hepatic vein waveform pattern becomes more abnormal. The overall difference among the groups was statistically significant, with a p-value of 0.031. These findings suggest an association between altered hepatic venous flow patterns and increased RARI values. Overall, the results indicate that abnormal hepatic vein waveforms are linked with higher renal vascular resistance in the study population.

Table 12: Comparison of Mean RARI Across Different eGFR Categories.

eGFR Category	Mean RARI	SD	p-value
>90	0.56	0.06	<0.001
60–89	0.71	0.05	
45–59	0.73	0.05	
30–44	0.84	0.04	
<30	0.92	0.04	



Graph 11: Mean RARI Across Different eGFR Categories

The table shows the comparison of mean Renal Artery Resistive Index (RARI) across different categories of eGFR. Patients with eGFR greater than 90 ml/min/1.73 m² had the lowest mean RARI value of 0.56 ±0.06. As renal function declined, the mean RARI progressively increased, with values of 0.71 ±0.05 in the 60–89 category and 0.73 ±0.05 in the 45–59 category. A further rise in mean RARI was observed in patients with eGFR between 30–44 (0.84 ±0.04) and less than 30 (0.92 ±0.04). The difference between these groups was statistically highly significant ($p<0.001$). Overall, the findings demonstrate a clear inverse relationship between renal function and RARI values in the study population.

Discussion

Chronic liver disease (CLD) is associated with significant haemodynamic alterations affecting both the portal and systemic circulation, frequently leading to renal dysfunction. Progressive portal hypertension and circulatory disturbances contribute to renal vasoconstriction and impaired renal perfusion, reflected by an increased Renal Artery Resistive Index (RARI). Simultaneously, renal function may deteriorate, commonly assessed by estimated glomerular filtration rate (eGFR). Evaluating the relationship between RARI and eGFR is clinically important because Doppler-derived indices may identify renal dysfunction before marked biochemical abnormalities develop. [22,35,39,49,50]

In the present study, most patients were middle-aged, particularly between 41–50 years, and males constituted the majority of the study population. This demographic pattern is comparable to the findings of Sharma et al., who reported a higher prevalence of chronic liver disease among middle-aged men, largely due to greater exposure to alcohol, metabolic syndrome, and chronic viral hepatitis. [51]

The clinical and laboratory profile demonstrated considerable variability in renal function and renal vascular resistance among CLD patients. Such findings are expected because progressive portal hypertension and systemic circulatory dysfunction are known to influence renal perfusion and vascular resistance. [25,40] Similar observations were reported by Platt et al., who noted increasing renal artery resistive indices with worsening renal function. [37]

Assessment of organ size revealed variations in liver and spleen dimensions. Many patients exhibited splenomegaly, a recognized consequence of portal hypertension. These findings are consistent with the observations of Bolondi et al., who reported increased spleen size and variable liver dimensions in patients with chronic liver disease and portal hypertension. [52] The presence of hepatosplenic structural alterations in the present study further supports the progressive nature of portal hypertensive changes in CLD.

Ascites was present in the majority of patients, with mild and gross ascites being more common than moderate ascites. Ascites is a well-established manifestation of advanced portal hypertension and hepatic decompensation. Similar findings were documented by Runyon et al., who described ascites as one of the most frequent complications of chronic liver disease, with severity varying according to disease progression. [53]

Doppler evaluation demonstrated alterations in portal haemodynamics. Increased portal vein and splenic vein diameters, along with variable portal vein velocities, reflected the haemodynamic consequences of portal hypertension. These findings correspond with those of Bolondi et al., who showed that portal vein diameter and blood flow velocity are significantly altered in patients with chronic liver disease and portal hypertension. [52] Doppler ultrasonography therefore remains a valuable non-invasive tool for assessing portal circulatory changes.

A large proportion of patients exhibited portosystemic collaterals, indicating significant portal hypertension and the development of alternative venous pathways to decompress the portal circulation. Similar observations were reported by Bolondi et al., who found collateral vessel formation to be a common feature of advanced portal hypertension. [52] Likewise, most patients demonstrated normal hepatopetal portal flow, whereas a smaller subset showed retrograde flow, suggesting advanced portal haemodynamic derangement. Reversal of portal venous flow has been recognized as an indicator of severe portal hypertension and advanced disease. [52]

The hepatic artery Doppler parameters also showed substantial variability. Alterations in hepatic artery peak systolic velocity and resistive index likely reflect compensatory changes associated with the hepatic arterial buffer response. Similar findings were reported by Piscaglia et al., who demonstrated significant changes in hepatic arterial haemodynamics in chronic liver disease patients. [54]

Evaluation of hepatic vein waveforms showed that most patients retained the normal triphasic pattern, while smaller proportions exhibited biphasic or monophasic waveforms. These abnormal waveforms are generally associated with increased hepatic stiffness and reduced vascular compliance in advanced liver disease. Comparable findings were reported by Bolondi et al., who observed progressive waveform abnormalities with worsening hepatic pathology. [55]

Renal function assessment revealed a broad spectrum of eGFR values ranging from normal renal function to severe impairment. Although many patients maintained relatively preserved renal function, varying degrees of renal dysfunction were observed, consistent with previous reports demonstrating that renal impairment is a frequent complication of chronic liver disease. [56]

A key finding of the present study was the significant relationship between RARI and renal function. RARI demonstrated a strong positive correlation with serum creatinine and a strong negative correlation with eGFR. These findings indicate that increasing renal vascular resistance is associated with worsening renal function. Similar observations were reported by Platt et al., who showed that elevated renal resistive indices correlate with progressive renal dysfunction in patients with liver disease. [37] This supports the potential utility of RARI as a non-invasive marker for early renal haemodynamic impairment.

The correlation between RARI and portal haemodynamic parameters showed variable results. Weak positive correlations were observed with portal vein diameter and spleen size, whereas a moderate negative correlation was found with portal vein mean velocity. This suggests that reduced portal venous flow may be associated with increased renal vascular resistance. Similar findings have been described by Bolondi et al., who proposed that portal circulatory alterations influence renal haemodynamics in cirrhotic patients. [52] Among the evaluated variables, portal vein velocity demonstrated the strongest association with RARI.

When patients were stratified according to the presence of ascites, mean RARI values were higher and mean eGFR values were lower among those with ascites. Although these differences were not statistically significant, the observed trends suggest that renal vascular resistance increases and renal function declines with

advancing hepatic decompensation. Similar findings have been reported by Platt et al. and Fede et al., who demonstrated greater renal dysfunction among patients with advanced liver disease and ascites. [37,56]

Patients with retrograde portal vein blood flow had significantly higher RARI values than those with normal hepatopetal flow. This finding indicates a strong relationship between severe portal haemodynamic disturbances and renal vascular compromise. Similar observations have been reported by Bolondi et al., who noted that abnormal portal flow patterns are frequently associated with more advanced systemic haemodynamic abnormalities. [52]

Finally, RARI showed a significant association with hepatic vein waveform patterns. Patients with triphasic waveforms had the lowest RARI values, whereas progressively higher values were observed in those with biphasic and monophasic waveforms. These findings suggest that worsening hepatic venous haemodynamics are accompanied by increasing renal vascular resistance. Comparable results have been described by Bolondi et al., supporting the concept that hepatic and renal haemodynamic abnormalities progress simultaneously in advanced chronic liver disease. [55]

Overall, the present study demonstrates that renal vascular resistance assessed by Doppler ultrasonography is closely related to renal functional status and portal haemodynamic alterations in chronic liver disease. RARI may therefore serve as a useful non-invasive marker for identifying patients at increased risk of renal dysfunction and monitoring disease progression. [37,52,55,56]

Conclusion

The results indicate that doppler derived RARI could represent a useful non-invasive marker for early detection and monitoring of renal impairment in patients with chronic liver disease and could help to identify patients at risk of developing complications such as hepatorenal syndrome.

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