



RENAL RESISTIVE INDEX AS AN EARLY NON-INVASIVE MARKER OF HEPATORENAL SYNDROME IN PATIENTS WITH CIRRHOSIS OF LIVER

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ABSTRACT **Background:** Liver cirrhosis is frequently complicated by renal hemodynamic alterations culminating in hepatorenal syndrome (HRS), a condition associated with significant morbidity and mortality. Conventional markers such as serum creatinine often underestimate renal dysfunction in cirrhosis. Renal resistive index (RRI), derived from Doppler ultrasonography, may serve as an early marker of renal compromise. **Objectives:** To evaluate RRI in cirrhotic patients and determine its association with renal status and development of HRS. **Methods:** A prospective observational study enrolled 200 cirrhotic patients at a tertiary care centre over one year. RRI was measured by Doppler ultrasonography; renal status was assessed by serum creatinine. Occurrence of HRS was documented using standard criteria. **Results:** Mean age was 44.3 ± 12.9 years; males comprised 71%. Alcohol was the predominant etiology (65%). Elevated RRI (≥ 0.7) was present in 89.5% of patients with HRS versus 48.1% without HRS ($p < 0.001$). RRI correlated positively with serum creatinine. **Conclusion:** RRI is significantly associated with renal dysfunction and HRS. As a non-invasive Doppler-derived parameter, RRI may serve as a valuable adjunct tool for early risk stratification in cirrhotic patients.

KEYWORDS : Renal resistive index; Liver cirrhosis; Hepatorenal syndrome; Doppler ultrasonography; Renal dysfunction

INTRODUCTION

Liver cirrhosis, the end-stage outcome of chronic liver disease, accounts for approximately 1.3 million deaths annually and ranks fourteenth among causes of adult mortality worldwide.¹ It is characterised by hepatic architectural distortion, regenerating nodules, and pervasive fibrosis, resulting in portal hypertension and impaired hepatic function.

Renal impairment is a major complication of cirrhosis. Hepatorenal syndrome (HRS) is a functional, potentially reversible form of renal failure occurring in approximately 10% of patients with severe cirrhosis or fulminant liver failure.² Its pathophysiology involves nitric oxide-mediated splanchnic vasodilation, activation of the renin-angiotensin-aldosterone system (RAAS), sympathetic nervous system stimulation, and non-osmotic vasopressin release, collectively causing profound renal vasoconstriction despite systemic fluid overload.^{3,4}

Serum creatinine tends to underestimate renal dysfunction in cirrhosis owing to reduced muscle mass, altered creatinine generation, and expanded extracellular volume. This limitation has prompted interest in novel biomarkers (cystatin C, NGAL) and imaging-based hemodynamic markers.⁵

RRI, calculated from Doppler ultrasonography as $(\text{Peak Systolic Velocity} - \text{End Diastolic Velocity}) / \text{Peak Systolic Velocity}$, reflects intrarenal vascular impedance. A value ≥ 0.70 is considered abnormal in adults.⁶ Elevated RRI has been reported in cirrhotic patients with ascites, advanced hepatic dysfunction, or evolving acute kidney injury, and has been associated with HRS and adverse outcomes.⁷ This study evaluated RRI and its association with renal status and HRS in a tertiary care setting in Northeast India.

AIM AND OBJECTIVES

Aim: To evaluate RRI in patients with cirrhosis of liver and determine its association with renal status.

Objectives:

- To determine the correlation between RRI and renal function.
- To determine the association between RRI and hepatorenal syndrome.

MATERIALS AND METHODS

A prospective hospital-based observational study was conducted at the Department of Medicine, Assam Medical College and Hospital (AMCH), Dibrugarh, over one year i.e., from 27th September 2024 to 26th September 2025, following ethical clearance from the IHEC.

Study Population:

Two hundred patients with liver cirrhosis attending Medicine OPD or

admitted to wards were enrolled after written informed consent. Inclusion required age ≥ 13 years and confirmed cirrhosis based on clinical, biochemical, and ultrasonographic (Garcia-Tsao) criteria. Patients with pre-existing intrinsic renal disease, hypertension, diabetes, malignancy, cardiovascular disease, renal artery stenosis or other vascular abnormalities, obstructive uropathy, nephrotoxic drug use, sepsis, hypotension, or pregnancy were excluded.

RRI Measurement: RRI was measured using a SAMSUNG RS80A ultrasound machine with a 2–5 MHz curvilinear transducer within 24 hours of admission. Pulsed wave Doppler was recorded at interlobar or arcuate arteries bilaterally, maintaining insonation angle $< 60^\circ$. Peak systolic velocity (PSV) and end-diastolic velocity (EDV) were averaged over three cardiac cycles. $\text{RRI} = (\text{PSV} - \text{EDV}) / \text{PSV}$; a value ≥ 0.70 was considered elevated. The mean of both kidneys was used for analysis.

Statistical Analysis: Data were analyzed using SPSS v21.0 and Microsoft Excel 2010. Continuous variables are expressed as mean $\pm$ SD; categorical data as number and percentage. Chi-square or Fisher's exact test was used as appropriate; $p < 0.05$ was considered significant.

RESULTS

A total of 200 patients were enrolled and analyzed. The mean age was 44.3 ± 12.9 years; 71% were male. Majority of the study population belonged to 40–49 years age group (29%). Alcohol was the leading etiology (65%), followed by others (16%), HBV (10%), and HCV (9%).

Table 1: Demographic and Etiological Profile

Parameter	Category	n (%)
Age (years)	40–49 (most common)	58 (29%)
Gender	Male / Female	142 (71%) / 58 (29%)
Etiology	Alcohol / HBV / HCV / Others	130 (65%) / 20 (10%) / 18 (9%) / 32 (16%)

Among the study population, 56% had elevated RRI (≥ 0.7); 52% had normal renal function, 29% had AKI, and 19% had HRS. Serum creatinine exceeded 2 mg/dL in 25% of patients.

Table 2: Distribution by Serum Creatinine, RRI and Renal Status

Parameter / Category	N	%
Serum creatinine < 1.0 mg/dL	86	43.0
Serum creatinine 1.0–2.0 mg/dL	64	32.0
Serum creatinine 2.1–4.0 mg/dL	36	18.0
Serum creatinine > 4.0 mg/dL	14	7.0
RRI < 0.7	88	44.0
RRI ≥ 0.7	112	56.0
Normal renal function	104	52.0

Acute Kidney Injury	58	29.0
Hepatorenal Syndrome	38	19.0

A highly significant association was observed between elevated RRI and HRS: 89.5% of HRS patients had RRI ≥ 0.7 versus 48.1% of non-HRS patients ($p < 0.001$, Fisher's exact test).

Table 3: Distribution of RRI According to HRS Status

RRI	HRS Present n (%)	HRS Absent n (%)	p-value
<0.7	4 (10.5%)	84 (51.9%)	<0.001*
≥ 0.7	34 (89.5%)	78 (48.1%)	
Total	38	162	

RRI also correlated significantly with serum creatinine levels ($p < 0.001$): the proportion of patients with RRI ≥ 0.7 rose from 30.4% in those with creatinine < 1 mg/dL to 85.7% in those with creatinine > 4 mg/dL.

Table 4: Correlation of RRI with Serum Creatinine

Serum Creatinine	RRI <0.7 N	RRI <0.7 %	RRI ≥ 0.7 N	RRI ≥ 0.7 %	Total
<1.0 mg/dL	52	59.1	34	30.4	86
1.0–2.0 mg/dL	24	27.3	40	35.7	64
2.1–4.0 mg/dL	10	11.4	26	23.2	36
>4.0 mg/dL	2	2.3	12	10.7	14
Total	88	100.0	112	100.0	200

*Fisher's Exact Test; p-value significant at 5% level

DISCUSSION

This prospective study of 200 cirrhotic patients comprehensively evaluated RRI and its association with renal dysfunction. The demographic profile — mean age 44.3 years, male predominance, and alcohol as the leading etiology — is consistent with published literature from India and South Asia, including reports by Regmi et al.,² Vinodh et al.,⁹ and Anil Kumar et al.¹⁰

Elevated RRI (≥ 0.7) was observed in 56% of patients, reflecting high prevalence of intrarenal vascular resistance in this hospital-based cohort, in agreement with Mogawer et al.¹¹ and Senthil et al.¹² The strong association between elevated RRI and HRS — 89.5% of HRS patients versus 48.1% of non-HRS patients having RRI ≥ 0.7 ($p < 0.001$) — is the key finding of this study and aligns with prior evidence that renal vascular resistance rises markedly in functional renal failure complicating cirrhosis. The significant positive correlation between RRI and serum creatinine further supports worsening renal hemodynamics with advancing dysfunction.

Limitations include the single-centre design, which may affect generalizability, and the absence of longitudinal RRI tracking with therapy. Prospective multicentre studies with serial RRI measurements and clinical outcomes data are needed.

CONCLUSION

Renal resistive index is significantly associated with the degree of renal dysfunction and the occurrence of hepatorenal syndrome in cirrhotic patients. As a non-invasive, reproducible Doppler-derived parameter, RRI can serve as a valuable adjunct for early detection of renal hemodynamic alterations and risk stratification. Routine renal Doppler assessment in cirrhotic patients may improve identification of high-risk individuals and guide clinical decision-making. Larger prospective studies with longitudinal follow-up are needed to define standardised cut-offs and evaluate the impact of RRI-guided management on patient outcomes.

DISCLOSURES

Conflict of Interest: None declared.

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Ethical Approval: Obtained from the IHEC, Assam Medical College and Hospital, Dibrugarh.

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