

Original Research Article

EVALUATION OF RENAL & SPLENIC RESISTIVE INDICES (RI) AND STIFFNESS USING DOPPLER US & SHEARWAVE ELASTOGRAPHY (SWE) IN HEPATORENAL SYNDROME (HRS)

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ABSTRACT**Background:**

Hepatorenal syndrome (HRS) represents a severe, functional renal failure in advanced cirrhosis, driven by profound renal vasoconstriction without intrinsic kidney damage. Traditional diagnostic methods relying on serum creatinine are often delayed and insufficiently specific in cirrhotic populations due to altered creatinine metabolism. Non-invasive imaging modalities, particularly Doppler ultrasonography and shear wave elastography (SWE), offer promising alternatives for early recognition of HRS by detecting functional and hemodynamic changes. **Objectives:** To evaluate the diagnostic efficacy of renal resistive index (RRI), difference in resistive indices between spleen and kidney (DI-RISK), and organ stiffness parameters obtained via SWE in distinguishing HRS from other cirrhotic patients with deranged renal function.

Materials and Methods: This prospective case-control study included 70 cirrhotic patients with elevated serum creatinine, divided into HRS and non-HRS groups based on International Club of Ascites (ICA-AKI) criteria. All participants underwent Doppler ultrasound evaluation of renal and splenic arteries and SWE of the liver and spleen. Key parameters—RRI, splenic RI, DI-RISK, liver stiffness, and spleen stiffness—were compared between groups and analyzed for diagnostic performance.

Results: HRS patients showed significantly elevated RRI (0.79 ± 0.07 vs. 0.67 ± 0.04 ; $p < 0.0001$), splenic RI (0.67 ± 0.07 vs. 0.59 ± 0.05 ; $p < 0.0001$), and DI-RISK (0.13 ± 0.07 vs. 0.09 ± 0.04 ; $p = 0.0175$). Liver and spleen stiffness values were also significantly higher in the HRS group (32.79 ± 9.16 kPa and 38.69 ± 5.60 kPa, respectively). ROC analysis revealed excellent diagnostic performance for RRI > 0.74 (AUC = 0.92), with 89.6% sensitivity and 92.7% specificity.

Conclusion: Doppler and SWE-based ultrasonographic parameters demonstrate strong diagnostic utility in differentiating HRS from other causes of renal dysfunction in cirrhotic patients. This multi-parametric imaging approach can serve as a valuable adjunct to clinical criteria, facilitating earlier diagnosis and targeted management.

Keywords: Hepatorenal syndrome, Doppler ultrasound, Resistive index, Shear wave elastography, Cirrhosis, Renal dysfunction.

INTRODUCTION

Hepatorenal syndrome (HRS) is a functional cause of renal insufficiency in advanced liver disease in the absence of clinical, laboratory, or anatomical evidence of intrinsic renal disease.^[1]

The central pathophysiological mechanism of hepatorenal syndrome (HRS) is profound renal vasoconstriction, which occurs in the absence of structural kidney damage.^[2,3] This process is intricately linked to systemic and splanchnic hemodynamic alterations in advanced liver disease. Cirrhosis induces portal hypertension, leading to significant splanchnic arterial vasodilation mediated by vasodilators such as nitric oxide and endocannabinoids.^[4–6] This vasodilation reduces effective arterial blood volume, which in turn activates compensatory vasoconstrictive and anti-natriuretic systems—including the renin-angiotensin-aldosterone system (RAAS), the sympathetic nervous system (SNS), and antidiuretic hormone (ADH).^[7–10] While these mechanisms initially serve to maintain systemic arterial pressure, they simultaneously reduce renal perfusion, promote sodium and water retention, and ultimately result in decreased glomerular filtration rate (GFR).^[7,11,12]

The ensuing renal hypoperfusion, though histologically bland, forms the physiological basis of HRS,^[3,13] while also distinguishing it from acute tubular necrosis and other intrinsic renal pathologies.^[14]

Additionally, renal autoregulation is compromised, narrowing the kidneys' ability to buffer changes in perfusion pressure. Even modest insults—such as volume depletion, infection, or hypotension—can unmask or worsen renal hypoperfusion.

Conventional diagnostic approaches predominantly depend on the assessment of blood creatinine variations and standard urine analyses to distinguish HRS from other causes of renal impairment. These investigations naturally contribute to the delay in the diagnosis and management of such instances, while also possessing various drawbacks in cirrhotic patients, including incorrect blood creatinine readings influenced by factors such as sarcopenia and altered creatinine metabolism^{15,16}. Novel biomarkers are also constrained by limited availability and clinical integration.

Recent advances in imaging techniques, such as Doppler ultrasound and shearwave elastography (SWE), offer the potential for non-invasive, quantitative assessment of vascular resistance and tissue stiffness in key abdominal organs. Studies suggest that parameters like renal resistive index (RRI) and splenic stiffness may reflect hemodynamic alterations and systemic vascular resistance associated with portal hypertension and HRS.^[17,18]

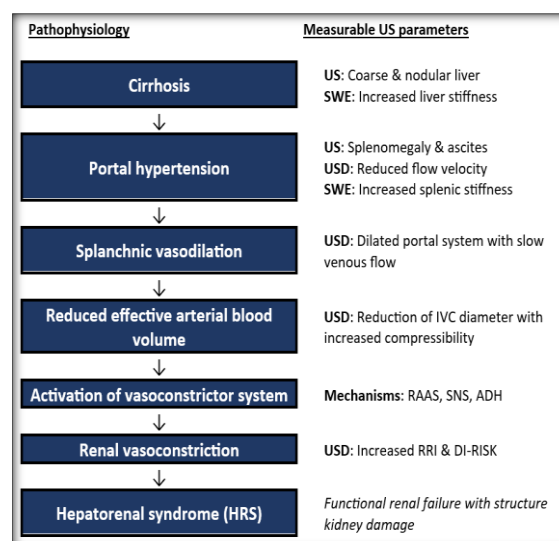


Figure 1: Simplified representation of the sequence in the development of hepatorenal syndrome (HRS) along with stepwise ultrasonographic evaluation, mapping measurable parameters to each stage of disease evolution—from cirrhosis and portal hypertension to renal hypoperfusion and eventual HRS.^[19]

In this context, the study intends to assess the efficacy of Doppler-derived renal and splenic resistive indices, together with SWE-based stiffness assessments, in cirrhotic patients with renal impairment, with the goal of identifying predictive imaging markers for HRS.

MATERIALS AND METHODS

Study Methodology

This **prospective observational study**, conducted at a tertiary center, employs a **case-control design** to assess the efficacy Duplex ultrasound and shear wave elastography (SWE) in the early diagnosis of HRS in the context of cirrhotic diseases presenting with acute renal impairment. Study population comprised of adult cirrhotic patients with renal dysfunction admitted under the **Department of Medicine, Silchar Medical College & Hospital (SMCH)**.

The study used **purposive random sampling** to select cirrhotic patients with deranged serum creatinine admitted under the department of Medicine. Patients were explained about the study, its scope and associated risks and benefits to the patients. Written informed consent was obtained from those who agreed to participate.

A sample size of **seventy (70)** was determined considering epidemiological studies in the closest geographic and patient cohort show approximately **23.8%** of patients with cirrhosis experience HRS-AKI.^[20] Total duration of the study was **twelve months**, from **November 2023 to October 2024**

Inclusion Criteria

- All patients over 18 years of age and willing to participate in the study

- Cirrhotic cases with acute renal dysfunction according to the ICA-AKI criteria for the diagnosis of acute renal injury (AKI).^[21]

Exclusion Criteria

- Patients experiencing emergencies, trauma and post-operative conditions
- Patients with acute infections, malignancies, and pathomorphological abnormalities observed in ultrasound were eliminated.
- Pregnant or lactating women
- Patients aged less than 18 years old
- Patients not willing to participate in the study

Study groups

Participants in the study were divided into two primary groups among cirrhotic patients with deranged serum creatinine:

- HRS case group:** Diagnosed cases of HRS as per ICA-AKI criteria. These cases have a serial rise in serum creatinine with unremarkable urine biochemistry analyses.
- Control group:** Rest of cirrhotic patients with deranged serum creatinine who did not meet the ICA-AKI criteria.

This categorization allowed for the identification of potential predictive markers for HRS and facilitated the evaluation of the effectiveness of the various US techniques as a diagnostic tool for cirrhotic individuals with acute renal impairment.

Study parameters

The study parameters included both **serological and US measurements** to assess the relationship between parenchymal characteristics and the development of HRS. The main parameters were:

- Extrahepatic features of portal hypertension:** This included measurement of portal vein diameter, mean flow velocity and assessment of splenomegaly.
- US Doppler interrogation of kidney and spleen:** This includes averaged measurements of resistive indices of splenic arteries and interlobar renal arteries on both sides.
- Liver and spleen stiffness:** Point-SWE measurements were acquired as per Society for US liver elastography guidelines from liver and spleen.

Examination Procedure

1. **Enrollment:** Eligible participants were enrolled after signing informed consent forms.
2. **Basic US evaluation:** Basic patient history was acquired with relevant laboratory results
3. **Targeted Doppler and B-mode evaluation of the hepatoportal axis** to evaluate for extrahepatic features of portal hypertension.
4. **Targeted Doppler interrogation of the splenic and renal arterial systems**
5. **Elastography (point-SWE) study** of the liver and splenic parenchyma as per **SRU-elastography guidelines**

Instrumentation

All scans were performed on **Samsung RS8AF4W/IN USG Machines** in the USG clinic of

the Department of Radiology, Silchar Medical College & Hospital (SMCH).

Two—dimensional (2D) - shearwave elastography (SWE) and Doppler studies were primarily conducted with the curvilinear 1-7 MHz transducer (**Samsung CA1-7A**)

US evaluation:

Baseline demographic, clinical, and biochemical data (including serial serum creatinine and urine analyses) were recorded. All patients were evaluated according to the ICA-AKI criteria for acute kidney injury.²¹

B-mode: A comprehensive B-mode abdominal scan was performed with the patient in supine, oblique, or lateral decubitus positions using subcostal and intercostal approaches.

Doppler: Targeted Doppler interrogation included the main portal vein, splenic artery, and interlobar renal arteries. Three satisfactory waveforms were obtained from each vessel and averaged. For splenic and renal assessment, measurements were taken from upper, mid, and lower segments during breath-hold to ensure reproducibility.

Elastography: Point shear wave elastography (SWE) of the liver and spleen was performed under breath-hold with the arms elevated to widen the intercostal spaces. Multiple readings were acquired per SRU guidelines²² until 10 valid measurements with acceptable interquartile-to-median (IQR/M) ratios were achieved.

Statistical methodology

Data were analyzed using IBM SPSS version 12. Quantitative variables were expressed as mean $\pm$ SD and qualitative variables as frequencies or percentages. Group comparisons employed the Chi-square, unpaired *t*, or Mann–Whitney *U* tests as appropriate. Correlations between imaging and biochemical parameters were assessed using Spearman's rank test. Diagnostic performance was evaluated via logistic regression and ROC curve analysis, with *p* < 0.05 considered statistically significant.

RESULTS

The study cohort included 52 males (74%) and 18 females (26%) ranging from 21-63 years of age. The mean age of the HRS group was 58.6 ± 10.1 years, while that of the non-HRS group was 56.8 ± 9.4 years. Males predominated in both groups. Alcoholic liver disease was the most frequent etiology in both cohorts, followed by metabolic and infective causes. Autoimmune liver disease was not observed in this population.

Group Comparison Statistics

Comparison of key Doppler, shear wave elastography (SWE), and grayscale ultrasound parameters between HRS cases and cirrhotic controls with renal dysfunction. Mean values, t-test statistics, and *p*-values are reported for each parameter. RRI, splenic artery RI, DI-RISK, liver stiffness, and spleen stiffness were significantly higher in the HRS group,

supporting their diagnostic utility. A p-value < 0.05 was considered statistically significant.

Table 1

Parameter	HRS Mean	Control Mean	t-value	p-value
Extrahepatic features of portal hypertension:				
Portal vein (mm)	12.05	10.82	2.51	0.016
Portal venous flow (cm/s)	15.39	22.81	-4.4	<0.001
Spleen (cm)	11.53	10.33	2.25	0.030
US Doppler parameters:				
RRI (avg)	0.79	0.67	9.07	<0.001
Splenic artery RI	0.67	0.59	5.09	<0.001
DI-RISK	0.13	0.08	3.24	0.002
Shearwave elastography (SWE) parameters (kPa):				
Liver stiffness	32.03	23.62	3.61	<0.001
Spleen stiffness	38.52	29.37	6.76	<0.001

Extrahepatic features of portal hypertension

In our study, three US markers of portal hypertension were evaluated for their diagnostic association with HRS: portal vein diameter, portal venous flow, and spleen size.

Patients were classified based on cut-off values derived from existing literature and ROC analysis—portal vein diameter > 12 mm, portal venous flow < 16 cm/s, and spleen size > 12 cm.

Table 2

	Cases	Controls
Portal vein diameter > 12 mm	16	12
Portal venous flow < 16 cm/s	19	10
Splenomegaly > 12 cm	12	6

Figure 2 Frequency of extrahepatic parameters of portal hypertension in 29 cases of HRS vs 41 cirrhotic cases with renal failure as control group. Chi-square analysis demonstrated that reduced flow and splenomegaly are more consistently associated with HRS than vein caliber alone.

Doppler Ultrasound Parameters

Doppler US provides dynamic insights into renal and splanchnic vascular resistance, which are physiologically altered in HRS. In our study, three Doppler-based parameters were evaluated: **average renal resistive index (RRI)**, **splenic artery resistive index (Splenic RI)**, and the **difference between splenic and renal RIs (DI-RISK)**.

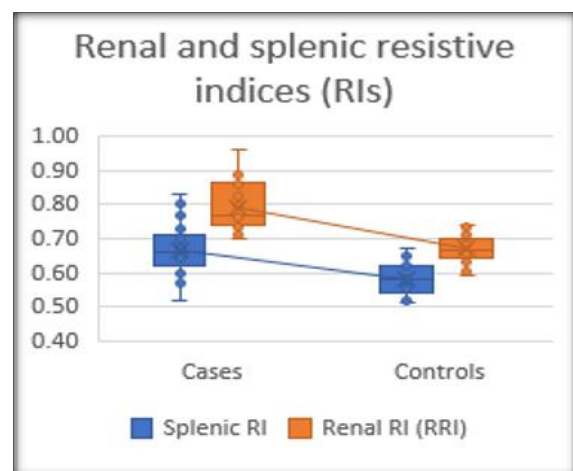


Figure 1

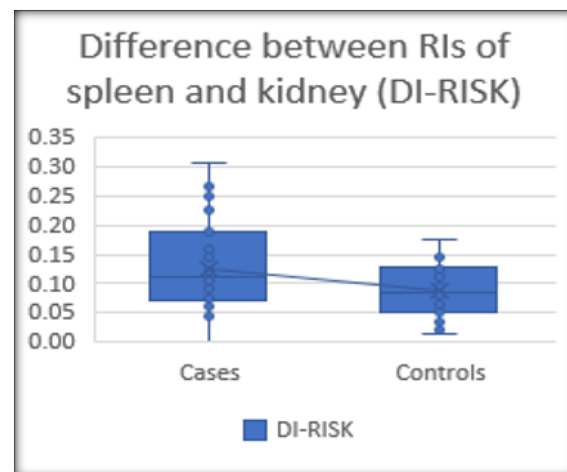


Figure 2

Mean RRI was significantly higher in HRS cases. Given the non-normal distribution in the HRS group, the Mann–Whitney U test was applied in lieu of the t-test, confirming strong statistical significance (U = 1169.5, p < 0.00001). This aligns with the known pathophysiology of intense intrarenal vasoconstriction in HRS. t-test for splenic RI showed a significant difference between groups (t = 5.086, p = 0.00001). Elevated splenic RI in HRS may reflect systemic vascular dysfunction associated with splanchnic vasodilation and compensatory arterial changes. The derived parameter, DI-RISK was also significantly different between HRS and controls (t = 3.243, p = 0.00243), suggesting its utility in highlighting relative vascular resistance shifts between systemic and renal circulations.

Importantly, a cutoff value of RRI > 0.73 yielded an odds ratio of 121.88 (95% CI: 20.76–715.60), confirming it as the most powerful independent predictor of HRS in this cohort. Splenic RI > 0.59 also demonstrated strong diagnostic power (OR = 15.02), while DI-RISK showed statistical separation but lacked definitive predictive utility when thresholded, likely due to sample distribution skew. These findings support the integration of Doppler indices into the diagnostic framework for HRS, particularly in settings where early recognition is essential for optimizing outcomes.

Elastography parameters

Shear wave elastography (SWE) was employed to quantify liver and spleen stiffness in cirrhotic patients with renal dysfunction. The aim was to assess whether these parameters could reliably differentiate patients with HRS from those without.

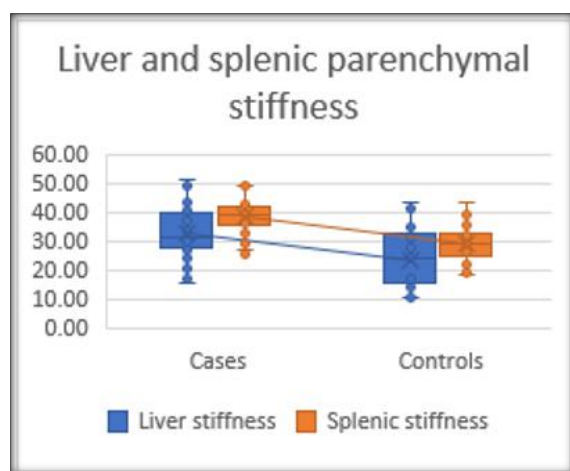


Figure 3

Liver stiffness was significantly higher in HRS cases. Spleen stiffness demonstrated even greater discriminatory power. With normal distributions in both groups, a t-test confirmed a highly significant difference ($t = 6.756$, $p < 0.00001$). A threshold of > 34.8 kPa provided an odds ratio of 27.60 (95% CI: 7.54–100.97), making it the strongest SWE-based predictor of HRS in this cohort.

Diagnostic Accuracy of Combined Imaging Parameters

Multivariate Logistic Regression and ROC Curve Analysis

To evaluate the diagnostic accuracy and predictive power of Doppler ultrasound and shear wave elastography (SWE) parameters in distinguishing HRS from other causes of renal dysfunction in cirrhotic patients, a multivariate logistic regression model was constructed.

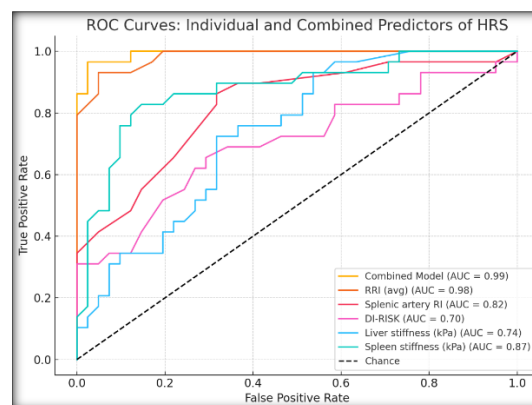


Figure 4

Figure 3 ROC curves comparing the diagnostic performance of individual Doppler and shearwave elastography (SWE) parameters with the combined multivariable logistic regression model for predicting hepatorenal syndrome (HRS). The combined model demonstrated the highest AUC, indicating superior discriminative ability. Among individual parameters, RRI and splenic RI showed strong predictive performance, followed by spleen stiffness. The diagonal dashed line represents the line of no discrimination (AUC = 0.5).

The logistic regression analysis revealed that **RRI**, **Splenic RI**, and **DI-RISK** were extremely strong predictors of HRS, with odds ratios exceeding 10^{14} , though their individual p -values were undefined due to collinearity. **Liver stiffness** did not reach statistical significance (OR = 1.02, $p = 0.83$), suggesting its limited additive value in the presence of stronger Doppler-based predictors.

Given the high degree of correlation between several predictors (as supported by Spearman analysis), a **ROC curve analysis** was performed to assess both individual and combined model performance.

The **combined multivariable model** achieved the highest diagnostic accuracy, with an **AUC of 0.99**, indicating near-perfect discrimination between HRS and non-HRS cases. Among individual predictors:

- **RRI (avg)** and **Splenic RI** demonstrated strong discriminatory ability (AUC > 0.90)
- **Spleen stiffness** also showed good performance (AUC > 0.85)
- **DI-RISK** contributed meaningfully in combination but was less robust as a standalone marker
- **Liver stiffness** had the lowest individual AUC, supporting its reduced weight in multivariable models

These findings affirm that a **multi-parametric approach**, combining Doppler ultrasound and SWE parameters, provides superior diagnostic precision compared to individual measures alone. Such an integrated model may facilitate earlier identification of HRS and guide clinical decision-making in cirrhotic patients with renal dysfunction.

DISCUSSION

The early diagnosis of hepatorenal syndrome (HRS) in cirrhotic patients with renal dysfunction remains a persistent challenge. Traditional reliance on serum creatinine, a delayed and non-specific marker in liver disease, often leads to late recognition and missed opportunities for early intervention.^[23] In this context, the utility of non-invasive ultrasound-based parameters has emerged as a promising solution. Our study reinforces the clinical relevance of integrating **Doppler US and shear wave elastography (SWE)** to accurately detect HRS in patients with decompensated cirrhosis.

Among all evaluated parameters, the **renal resistive index (RRI)** stood out as the **most robust individual predictor** of HRS, showing statistically significant elevation in cases compared to controls (mean RRI: 0.79 vs 0.67, $p < 0.00001$). The optimal cutoff value of **> 0.73** demonstrated **93.1% sensitivity** and **95.1% specificity**, with an AUC of 0.92. These findings are consistent with earlier works such as **Bardi et al. (2002)**,^[24] who reported $RRI > 0.70$ as predictive of HRS, and **Mogawer et al. (2021)**,^[25] who suggested an RI cutoff > 0.77 with comparable diagnostic performance. Importantly, our findings reaffirm the pathophysiological link between **intrarenal vasoconstriction** and elevated RRI, offering clinicians a reliable and immediate tool for HRS suspicion even before creatinine rise (**Wong, 2015**).^[15]

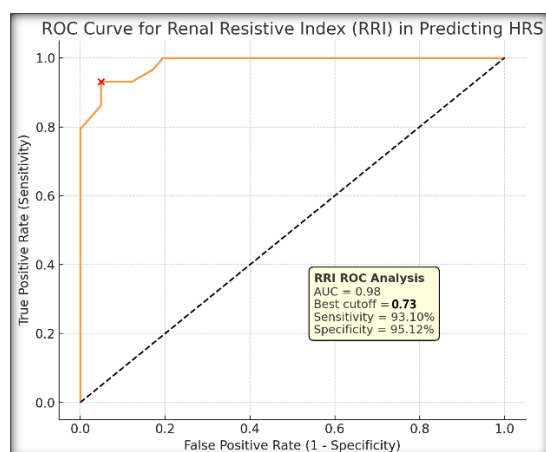


Figure 5

Figure 4 “ROC curve for Renal Resistive Index (RRI) in predicting hepatorenal syndrome (HRS). The curve plots sensitivity against 100-specificity, with the optimal cutoff value for RRI identified at **> 0.73**. At this threshold, RRI achieved a sensitivity of 93.1% and a specificity of 95.1%, demonstrating excellent diagnostic accuracy for distinguishing HRS from non-HRS cases. The diagonal line represents the line of no discrimination (AUC = 0.5).”

This finding of intense renal vasoconstriction is consistent with our current understanding of HRS. The renal resistive index (RRI), derived from

Doppler US, directly reflects intrarenal arterial resistance. In HRS, elevated RRI values mirror the increased vascular resistance caused by this neurohumoral activation and impaired renal perfusion, often preceding biochemical changes such as serum creatinine elevation.^[15,26,27] Since HRS is primarily a functional disorder without structural renal changes, Doppler-based indices like RRI are ideally suited to detect early hemodynamic shifts that would not be captured through traditional imaging or lab parameters.^[23,28]

Moreover, as demonstrated in earlier studies, even cirrhotic patients without overt azotemia may exhibit elevated RRI values, suggesting that renal Doppler US can uncover subclinical alterations before frank renal failure develops.^[24,29] This makes RRI not just a diagnostic tool, but also a potential early warning biomarker for clinicians monitoring cirrhotic patients at risk of decompensation.

A novel contribution of our study is the incorporation of **DI-RISK** (difference between splenic and renal RI), which demonstrated statistically significant elevation in HRS patients ($p = 0.0175$), and moderate diagnostic power. This marker, previously described in chronic kidney disease populations (Grün et al., 2012),^[30] shows emerging relevance in hepatic disease contexts, as it potentially isolates **renal-specific hemodynamic changes** from generalized systemic vascular influences. While DI-RISK along may not rival the diagnostic strength of RRI, it enhances specificity when interpreted in conjunction with splenic Doppler parameters.

Shear wave elastography parameters further contributed diagnostic strength, especially **spleen stiffness**, which outperformed liver stiffness in differentiating HRS cases. A threshold of >34.8 kPa yielded an odds ratio of 27.60 and an AUC of 0.85, with strong sensitivity and specificity values. These results are supported by Colecchia et al. (2015) and Talwalkar et al. (2013),^[18,31] who highlighted spleen stiffness as a reliable marker of **portal hypertension severity**, which is intimately linked to the hemodynamic cascade of HRS. While **liver stiffness** was significantly elevated in HRS (mean: 32.8 kPa vs 23.7 kPa, $p = 0.0002$), its standalone predictive value was lower—aligning with literature suggesting liver stiffness reflects fibrosis more than acute functional shifts (SRU Guidelines, 2015; Fang et al., 2021).^[22,32] While spleen and renal stiffness values measured using shear wave elastography (SWE) showed significant differences between groups, these findings must be interpreted with caution. Given that HRS typically occurs in the advanced stages of liver disease, where severe portal hypertension is almost universally present, higher stiffness values are more likely a reflection of this shared pathophysiological endpoint rather than a direct indicator of HRS. Thus, while SWE adds valuable diagnostic context, it should not be interpreted in isolation. Its role is best understood as part of a broader hemodynamic picture, complementing but not replacing functional vascular indices like the RRI, which more directly

capture the intrarenal circulatory compromise that defines HRS.^[18,33]

Despite the need for cautious interpretation, portal hypertension parameters such as spleen stiffness remain clinically significant and should not be disregarded. As the natural history of cirrhosis progresses, the emergence of extrahepatic manifestations—splenomegaly, varices, and renal vasoconstriction—marks a shift toward systemic decompensation. In this context, the diagnostic value of SWE increases, particularly when interpreted alongside Doppler ultrasonography. Several studies have highlighted that the positive predictive value of noninvasive markers such as spleen stiffness improves markedly when signs of systemic or extrahepatic portal hypertension are present.^[17,18,33] In our study, both splenomegaly and slow portal vein flow were significantly more prevalent among patients with HRS, supporting the notion that these features reflect advanced circulatory dysregulation and splanchnic vasodilation typical of late-stage portal hypertension.^[17,34]

Given their non-invasive detectability, these parameters can serve as early indicators prompting further Doppler assessment—particularly **RRI**, which in our study showed the highest diagnostic accuracy for HRS. This layered approach not only refines diagnostic specificity but also provides a practical framework for clinicians performing standard ultrasound exams in cirrhotic patients. Incorporating these extrahepatic signs as triage criteria for more advanced Doppler workup may enhance early recognition of HRS, facilitating timely management and potentially improving outcomes.^[15,18]

Our findings strongly support a **combined diagnostic approach**. The **multivariate logistic regression model**, integrating Doppler and SWE parameters, yielded a near-perfect AUC of ~0.99. This validates earlier hypotheses proposing that combining functional (RRI, splenic RI), hemodynamic (DI-RISK), and structural (SWE) markers captures the multifactorial nature of HRS more effectively than any single test (**Busk et al., 2016; Salerno et al., 2008**).^[14,28] The synergy of Doppler and elastographic indices provides a holistic and early insight into the renal impact of systemic circulatory dysregulation in cirrhosis.

While a combined diagnostic approach incorporating SWE and Doppler parameters offers the highest diagnostic accuracy, it is important to acknowledge the practical advantages of using RRI alone in certain clinical contexts. In resource-limited settings or acute care scenarios where full elastography may not be readily available, an **RRI above the cutoff value identified in this study (≥ 0.73)** can serve as a **strong, rapid, and non-invasive screening tool**. Given its robust sensitivity and specificity demonstrated in our cohort, RRI provides clinicians with a functional vascular marker that reflects real-time hemodynamic compromise—a hallmark of HRS pathophysiology. This makes RRI not only an

adjunct in comprehensive evaluation but also a **viable frontline tool** in the early identification of patients at risk for HRS, enabling timely intervention even before SWE data is available.^[15,26]

In sum, this study affirms and extends previous findings by demonstrating the **clinical utility of ultrasound**—particularly RRI and spleen stiffness—as immediate, reproducible, and bedside-accessible markers for the diagnosis of HRS. The potential role of **DI-RISK as a novel adjunct** deserves further prospective exploration.

Limitations

This study has several limitations that should be considered while interpreting the results:

- **Limited patient mobility:** Many patients with hepatorenal syndrome (HRS) presented in advanced stages and were directly admitted to intensive care units. Due to their critical condition, they could not be transported to the ultrasound suite where the primary equipment used for Doppler and elastography assessments was located.
- **Unmeasured ascites severity:** The degree of ascites was not quantitatively assessed. Since increased intra-abdominal pressure can influence Doppler waveforms and shear wave propagation, this might have introduced variability in renal and splenic measurements.^[35,36]
- **Indirect assessment of portal hypertension:** Instead of using the standard hepatic venous pressure gradient (HVPG) technique, portal hypertension was inferred from indirect ultrasound findings, which are less sensitive and specific.^[37]
- **Temporal variability:** Patients were evaluated at different points during their hospital stay. This temporal heterogeneity might have contributed to variation in resistive indices and stiffness values.^[38]
- **Single-center design:** A single tertiary care center design was another limiting factor in the study.

CONCLUSION

Summary

This prospective observational study was conducted to assess the diagnostic utility of **Doppler ultrasound and shear wave elastography (SWE)** in distinguishing **hepatorenal syndrome (HRS)** from other forms of renal dysfunction in patients with liver cirrhosis. Seventy patients with cirrhosis and deranged serum creatinine were enrolled and categorized into HRS and non-HRS groups based on ICA-AKI criteria.

The key findings were as follows:

- **Renal resistive index (RRI)** was the strongest single predictor of HRS, with high diagnostic accuracy (AUC = 0.92, OR = 121.88).

- **Splenic RI** and **DI-RISK** added additional discriminatory value, particularly in combination.
- **Spleen stiffness** was the most powerful elastographic marker (OR = 27.60, AUC = 0.85), outperforming liver stiffness.
- **Portal hypertension features** such as low portal vein flow and increased spleen size also contributed to diagnosis.
- A **combined model** incorporating Doppler and SWE parameters showed excellent classification accuracy (AUC ≈ 0.99), confirming the utility of a multi-parametric imaging approach.

These findings suggest that a **composite US approach** can aid in the **early identification and differentiation of HRS**, potentially improving timely therapeutic intervention and prognosis. Alternatively, **averaged RRI** emerged as the single biggest predictor with fairly high sensitivity and specificity and can be a viable marker for the practical assessment of HRS in the context of chronic liver disease with acute renal dysfunction.

Conclusion

The diagnosis of HRS is often delayed due to non-specific biochemical markers and overlapping clinical features. This study demonstrates that **Doppler US and shear wave elastography (SWE)** provide accurate, non-invasive alternatives for identifying HRS in its early stages.

Among all evaluated parameters, **renal resistive index (RRI)** emerged as the **most powerful individual marker**, aligning with prior research on intrarenal vasoconstriction in HRS. The addition of **DI-RISK**, a novel index comparing splenic and renal resistance, offers an additional layer of specificity in assessing systemic versus renal vascular influences. Likewise, **spleen stiffness** better reflected the severity of portal hypertension than liver the and was highly predictive of HRS.

The integration of these imaging markers into a **combined logistic model** dramatically enhanced diagnostic precision, reinforcing the value of a **multi-parametric ultrasound approach**. Given their accessibility and reproducibility, these tools can be seamlessly incorporated into routine bedside evaluation, facilitating **earlier diagnosis, more accurate stratification, and targeted management** of HRS in cirrhotic patients.

Future studies should focus on validating **DI-RISK and spleen stiffness cutoffs** in larger populations and evaluating the prognostic utility of these markers in treatment response and survival.

REFERENCES

1. Epstein M. The Hepatorenal Syndrome — Newer Perspectives. *N Engl J Med.* 1992;327(25):1810-1811. doi:10.1056/NEJM199212173272509
2. Coffman TM, Schrier RW, eds. *Schrier's Diseases of the Kidney*. 9th ed. Wolters Kluwer Health/Lippincott Williams & Wilkins; 2013.
3. Fagundes C, Ginès P. Hepatorenal Syndrome: A Severe, but Treatable, Cause of Kidney Failure in Cirrhosis. *American Journal of Kidney Diseases.* 2012;59(6):874-885. doi:10.1053/j.ajkd.2011.12.032
4. Martin PY, Ginès P, Schrier RW. Nitric Oxide as a Mediator of Hemodynamic Abnormalities and Sodium and Water Retention in Cirrhosis. Epstein FH, ed. *N Engl J Med.* 1998;339(8):533-541. doi:10.1056/NEJM199808203390807
5. Iwakiri Y, Groszmann RJ. The Hyperdynamic Circulation of Chronic Liver Diseases: From the Patient to the Molecule. *Hepatology.* 2006;43(Supplement 1):S121-S131. doi:10.1002/hep.20993
6. Ros J, Clària J, To-Figueras J, et al. Endogenous cannabinoids: A new system involved in the homeostasis of arterial pressure in experimental cirrhosis in the rat. *Gastroenterology.* 2002;122(1):85-93. doi:10.1053/gast.2002.30305
7. Cárdenas A, Ginès P. Portal hypertension: Current Opinion in *Gastroenterology.* 2009;25(3):195-201. doi:10.1097/MOG.0b013e328329e154
8. Schrier RW, Arroyo V, Bernardi M, Epstein M, Henriksen JH, Rodés J. Peripheral arterial vasodilation hypothesis: A proposal for the initiation of renal sodium and water retention in cirrhosis. *Hepatology.* 1988;8(5):1151-1157. doi:10.1002/hep.1840080532
9. Bernardi M, Trevisani F, Gasbarrini A, Gasbarrini G. Hepatorenal Disorders: Role of the Renin-Angiotensin-Aldosterone System. *Semin Liver Dis.* 1994;14(01):23-34. doi:10.1055/s-2007-1007295
10. Henriksen JH, Møller S, Ring-Larsen H, Christensen NJ. The sympathetic nervous system in liver disease. *Journal of Hepatology.* 1998;29(2):328-341. doi:10.1016/S0168-8278(98)80022-6
11. Facciorusso A, Cosimo Nacchiero M, Rosania R, et al. The Use of Human Albumin for the Treatment of Ascites in Patients with Liver Cirrhosis: Item of Safety, Facts, Controversies and Perspectives. *CDS.* 2011;6(4):267-274. doi:10.2174/157488611798280906
12. Facciorusso A, Del Prete V, Crucinio N, et al. Angiotensin receptor blockers improve survival outcomes after radiofrequency ablation in hepatocarcinoma patients. *J of Gastro and Hepatol.* 2015;30(11):1643-1650. doi:10.1111/jgh.12988
13. Ginès P, Schrier RW. Renal Failure in Cirrhosis. *N Engl J Med.* 2009;361(13):1279-1290. doi:10.1056/NEJMra0809139
14. Salerno F, Gerbes A, Ginès P, Wong F, Arroyo V. Diagnosis, prevention and treatment of hepatorenal syndrome in cirrhosis. *Postgraduate Medical Journal.* 2008;84(998):662-670. doi:10.1136/gut.2006.107789
15. Wong F. The evolving concept of acute kidney injury in patients with cirrhosis. *Nat Rev Gastroenterol Hepatol.* 2015;12(12):711-719. doi:10.1038/nrgastro.2015.174
16. Sherman DS, Fish DN, Teitelbaum I. Assessing renal function in cirrhotic patients: Problems and pitfalls. *American Journal of Kidney Diseases.* 2003;41(2):269-278. doi:10.1053/ajkd.2003.50035
17. Ma X, Wang L, Wu H, et al. Spleen Stiffness Is Superior to Liver Stiffness for Predicting Esophageal Varices in Chronic Liver Disease: A Meta-Analysis. *Herrero JI, ed. PLoS ONE.* 2016;11(11):e0165786. doi:10.1371/journal.pone.0165786
18. Colecchia A, Montrone L, Scafoli E, et al. Measurement of Spleen Stiffness to Evaluate Portal Hypertension and the Presence of Esophageal Varices in Patients With HCV-Related Cirrhosis. *Gastroenterology.* 2012;143(3):646-654. doi:10.1053/j.gastro.2012.05.035
19. Macnaughtan J, Thomas H. Liver failure at the front door. *Clinical Medicine.* 2010;10(1):73-78. doi:10.7861/clinmedicine.10-1-73
20. Arora MS, Kaushik R, Ahmad S, Kaushik RM. Profile of Acute Kidney Injury in Patients with Decompensated Cirrhosis at a Tertiary-Care Center in Uttarakhand, India. *Dig Dis.* 2020;38(4):335-343. doi:10.1159/000504836
21. Angeli P, Gines P, Wong F, et al. Diagnosis and management of acute kidney injury in patients with cirrhosis: revised consensus recommendations of the International Club of Ascites. *Gut.* 2015;64(4):531-537. doi:10.1136/gutjnl-2014-308874

22. Barr RG, Wilson SR, Rubens D, Garcia-Tsao G, Ferraioli G. Update to the Society of Radiologists in Ultrasound Liver Elastography Consensus Statement. *Radiology*. 2020;296(2):263-274. doi:10.1148/radiol.2020192437
23. Low G, Alexander GJM, Lomas DJ. Hepatorenal Syndrome: Aetiology, Diagnosis, and Treatment. *Gastroenterology Research and Practice*. 2015;2015:1-11. doi:10.1155/2015/207012
24. Bardi A, Sapunar J, Oksenberg D, et al. Intrarenal arterial doppler ultrasonography in cirrhotic patients with ascites, with and without hepatorenal syndrome. *Rev Med Chil*. 2002;130(2):173-180.
25. Mogawer MS, Nassef SAR, Elhamid SMA, et al. Role of renal Duplex ultrasonography in evaluation of hepatorenal syndrome. *Egypt Liver Journal*. 2021;11(1):34. doi:10.1186/s43066-021-00104-9
26. Platt JF, Ellis JH, Rubin JM, Merion RM, Lucey MR. Renal duplex Doppler ultrasonography: a noninvasive predictor of kidney dysfunction and hepatorenal failure in liver disease. *Hepatology*. 1994;20(2):362-369.
27. Sacerdoti D, Bolognesi M, Merkel C, Angeli P, Gatta A. Renal vasoconstriction in cirrhosis evaluated by duplex doppler ultrasonography. *Hepatology*. 1993;17(2):219-224. doi:10.1002/hep.1840170210
28. Busk TM, Bendtsen F, Møller S. Hepatorenal syndrome in cirrhosis: diagnostic, pathophysiological, and therapeutic aspects. *Expert Review of Gastroenterology & Hepatology*. 2016;10(10):1153-1161. doi:10.1080/17474124.2016.1196132
29. Kastelan S, Ljubic N, Kastelan Z, Ostojic R, Uravic M. The role of duplex-doppler ultrasonography in the diagnosis of renal dysfunction and hepatorenal syndrome in patients with liver cirrhosis. *Hepatogastroenterology*. 2004;51(59):1408-1412.
30. Grün OS, Herath E, Weihrauch A, et al. Does the Measurement of the Difference of Resistive Indexes in Spleen and Kidney Allow a Selective Assessment of Chronic Kidney Injury? *Radiology*. 2012;264(3):894-902. doi:10.1148/radiol.12111533
31. Talwalkar JA, Yin M, Venkatesh S, et al. Feasibility of In Vivo MR Elastographic Splenic Stiffness Measurements in the Assessment of Portal Hypertension. *American Journal of Roentgenology*. 2009;193(1):122-127. doi:10.2214/AJR.07.3504
32. Fang Y, Zhu H, Gao C, et al. Value of shear wave elastography in predicting hepatorenal syndrome in patients with liver cirrhosis and ascites. *Int J Clin Pract*. 2021;75(11). doi:10.1111/ijcp.14811
33. Kazemi F, Kettaneh A, N'kontchou G, et al. Liver stiffness measurement selects patients with cirrhosis at risk of bearing large oesophageal varices. *Journal of Hepatology*. 2006;45(2):230-235. doi:10.1016/j.jhep.2006.04.006
34. Berzigotti A, Tsochatzis E, Boursier J, et al. EASL Clinical Practice Guidelines on non-invasive tests for evaluation of liver disease severity and prognosis – 2021 update. *Journal of Hepatology*. 2021;75(3):659-689. doi:10.1016/j.jhep.2021.05.025
35. Kim BH, Lee JM, Lee YJ, et al. MR elastography for noninvasive assessment of hepatic fibrosis: Experience from a tertiary center in asia. *Magnetic Resonance Imaging*. 2011;34(5):1110-1116. doi:10.1002/jmri.22723
36. Berzigotti A, Seijo S, Reverter E, Bosch J. Assessing portal hypertension in liver diseases. *Expert Review of Gastroenterology & Hepatology*. 2013;7(2):141-155. doi:10.1586/egh.12.83
37. De Franchis R, Bosch J, Garcia-Tsao G, et al. Baveno VII – Renewing consensus in portal hypertension. *Journal of Hepatology*. 2022;76(4):959-974. doi:10.1016/j.jhep.2021.12.022
38. Garcia-Tsao G, Bosch J, Groszmann RJ. Portal hypertension and variceal bleeding—Unresolved issues. Summary of an American Association for the study of liver diseases and European Association for the study of the liver single-topic conference. *Hepatology*. 2008;47(5):1764-1772. doi:10.1002/hep.22273.