Original Article

The Role of Hepatic and Portal Vein Dopplers in the Management of Patients with Right Heart Failure

Alka Agrawal[1], Gaurav Bhandari[2], Megha Singh[3], Pragya Verma[3], Shubham Bilgaiyan[3], Ayushi Bansal[3]

[1]Professor and HOD, Department of Radiodiagnosis, MGM medical college, Indore, Madhya Pradesh, India. [2]Assistant Professor, Department of Radiodiagnosis, MGM medical college Indore, Madhya Pradesh, India. [3]PG resident, Department of Radiodiagnosis, MGM medical college Indore, Madhya Pradesh, India

Abstract

Background: Right heart failure (RHF) is characterized by systemic venous congestion, but reliable non-invasive markers to monitor decongestion remain limited. Hepatic vein (HV) Doppler S:D ratio and portal vein pulsatility index (PI) may serve as dynamic surrogates for venous pressures. **Material and Methods:** This prospective observational study enrolled 60 hospitalized RHF patients. Serial HV and portal vein Doppler assessments were performed at admission, Day 2, Day 3 and pre-discharge. HV S:D ratio and portal vein PI were recorded and correlated with clinical response. Statistical analysis was conducted using SPSS 26; significance was set at $p<0.05$. **Results:** At admission 83.3% of patients had reversed HV -S:D ratios only 16.6% were normal. Normalization progressively improved to 75% by discharge. Portal vein PI was elevated in 81.6% of patients at baseline only 18.3% were normal (<0.3). Mean PI declined significantly from 0.42±0.07 at admission to 0.29±0.08 at discharge ($F=19.27$, $p<0.001$) representing a 31% reduction. Despite improvement 25% of patients remained non-responders on HV Doppler. Persistent abnormalities were attributed to hepatic remodeling, severe tricuspid regurgitation, RV-pulmonary uncoupling and reduced venous compliance. Serial Doppler changes correlated closely with therapeutic response. Doppler non-responders may reflect a subgroup with advanced or fixed congestion. **Conclusion:** Serial HV and portal vein Doppler offer reliable, non-invasive monitoring of venous congestion and therapeutic response in RHF. Integration into routine assessment may enhance individualized volume management and identify patients at risk for persistent congestion.

Keywords: Right heart failure, Hepatic vein Doppler, Portal vein, Pulsatility index, Venous congestion, S:D ratio, Ultrasonography.

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INTRODUCTION

Right heart failure (RHF) is a complex clinical syndrome associated with significant morbidity and mortality, posing challenges for both assessment and monitoring.[1] The diverse etiologies, including cardiomyopathies, pulmonary hypertension and volume overload, progressively impair right ventricular function, altering systemic venous hemodynamics.[1,2] Despite the advances RHF assessment remains limited by the complex geometry of the right ventricle and the shortcomings of current imaging modalities.[3]

Conventional evaluation relies on clinical examination, echocardiography and invasive hemodynamic monitoring.[1-3] The physical signs may be absent even with significant hemodynamic compromise.[4] The right ventricle's thin walls, trabeculations and anterior location limit comprehensive echocardiographic evaluation.[3,5] Many echocardiographic indices are preload- and afterload dependent making them unreliable in dynamic hemodynamic states.[3-5] Invasive monitoring though informative carries procedural risks, is costly and unsuitable for serial outpatient monitoring.[2-4]

Venous congestion plays a central role in RHF and the liver serves as a sensitive marker due to its dual blood supply and role as a venous reservoir.[5,6] Normally HV Doppler demonstrates a triphasic waveform reflecting right atrial pressure variations: systolic (S), diastolic (D) and atrial contraction (A) waves.[7-9] Under normal conditions the S wave exceeds D wave (S:D ratio >1) but this reverses with elevated right atrial pressure or tricuspid regurgitation.[7,8]

Also portal vein flow, normally continuous and hepatopetal, becomes pulsatile with increasing central venous pressure.[10] The portal vein pulsatility index (PI) calculated as (Vmax - Vmin)/Vmax, quantifies this pulsatility, with normal values <0.3.[10,11] Elevated PI reflects venous congestion and correlates with poor outcomes in both perioperative and critically ill patients.[9-11] PI changes may precede overt clinical signs, making it a sensitive early marker of systemic venous congestion.[10,11]

Address for correspondence: Dr. Megha Singh, PG resident, Department of Radiodiagnosis, MGM medical college Indore, Madhya Pradesh, India
E-mail: magsingh.megha22@gmail.com

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While serial monitoring has demonstrated prognostic utility in heart failure, most studies focus on left-sided parameters with limited data for serial hepatic and portal vein Doppler in RHF.[10,12] Serial Doppler assessments may allow non-invasive, real-time monitoring of therapy reducing reliance on invasive monitoring.[10-12] Correlating Doppler changes with clinical improvement may provide additional insights into RHF recovery.[9-11]

Despite these theoretical benefits, there is a paucity of systematic data on hepatic vein S:D ratio and portal vein PI in RHFin their serial changes during hospitalization and correlation with clinical status.[13,14] Existing literature largely focuses on hepatic and portal Doppler findings in liver disease or surgical patients rather than in primary right heart dysfunction. Current treatment response assessment remains largely dependent on subjective clinical parameters and periodic invasive assessments.[13,14]

This prospective study addresses these gaps by systematically evaluating hepatic vein S:D ratio and portal vein PI in RHF patients also assessing their serial changes during hospitalization and correlating these changes with clinical improvement

Materials and Methods

A time-bound, prospective & hospital-based observational study was conducted over one year in the Department of Radiodiagnosis at a Medical College & Super speciality Hospital. Following the approval from Institutional Ethical Committee. Sixty patients clinically diagnosed with right heart failure and referred for hepatic and portal vein Doppler ultrasonography by the Medicine Department were recruited after their consent. All participants received standardized treatment protocols.

Inclusion and Exclusion Criteria

Patients aged 18 years or older clinically diagnosed with right heart failure requiring hospitalization and receiving uniform medical management were included after obtaining informed consent. Patients with known liver pathology, coagulopathy, obesity, veno-occlusive disease, shock or those aged below 18 years were excluded.

After inclusion, detailed history and clinical examination were performed to assess right heart failure signs and symptoms. Patients fasted for 4–6 hours prior to ultrasonography to minimize bowel gas interference. All examinations were performed using a Samsung HS-30 ultrasound machine with a 3–5 MHz low-frequency curvilinear transducer. The patient was positioned supine or in the slight left lateral decubitus position.

Ultrasonographic Evaluation

B-Mode Assessment

Routine evaluation included assessment for hepatomegaly, ascites, pleural effusion and inferior vena cava diameter and collapsibility.

Doppler Assessment

- **Hepatic Vein Doppler:** The right hepatic vein was insonated using a subcostal or intercostal approach. The Doppler cursor was aligned parallel to blood flow, with a 5–10 mm sample volume and Doppler angle ≤60°. Patients were instructed to hold breath at end-expiration. The triphasic waveform was recorded and S:D ratio was calculated based on systolic (S) and diastolic (D) flow velocities.
- **Portal Vein Doppler:** The main portal vein was evaluated using subcostal approach. Doppler sampling was performed along the longitudinal axis with angle correction ≤60°. Flow direction, diameter, velocity (normal 16–40 cm/s) and pulsatility index (PI) were assessed using the formula:

PI = Vmax−Vmin/ Vmax

Doppler evaluations were performed at four time points: admission (Day 1), Day 2, Day 3 and pre-discharge. Serial changes in Doppler parameters were correlated with clinical improvement.

Statistical Analysis: Data were entered into Microsoft Excel and analyzed using SPSS software 26. Quantitative variables were expressed as mean ± standard deviation. Changes in Doppler and clinical parameters over time were assessed using appropriate statistical tests. One-way ANOVA was performed to analyze differences in portal vein PI across serial measurements. A p-value <0.05 was considered statistically significant.

Results

At presentation a large proportion of patients (83.3%) showed abnormal hepatic vein Doppler with reversal of the S:D ratio (D wave > S wave) indicating the significant systemic venous congestion. Only 16.6% had normal S:D pattern at baseline. On portal vein assessment 18.3% of patients demonstrated normal PI (<0.3), while majority had mild congestion (PI 0.3–0.5) seen in 73.3% and 8.3% had severe congestion (PI >0.5). This reflects that most patients admitted with right heart failure were already in a state of advanced volume overload affecting both hepatic and portal systems.

Table 1: Baseline Hepatic Vein Doppler (S:D Ratio) and Portal Vein Pulsatility Index at Admission (1st-Day)

Parameter	Category	Number of Patients	Percentage (%)
Hepatic Vein Doppler (S:D Ratio)	D wave > S wave (Reversed)	50	83.3%
	S wave > D wave (Normal)	10	16.6%
Portal Vein Pulsatility Index (PI)	Normal (<0.3)	11	18.3%
	Mild congestion (0.3–0.5)	44	73.3%
	Severe congestion (>0.5)	5	8.3%

Table 2: Serial Changes in Hepatic Vein Doppler (S:D Ratio) during the stay

Time Point	D wave > S wave (Reversed)	S wave > D wave (Normal)	Demise
Day 1 (Admission)	50 (83.3%)	10 (16.6%)	-
Day 2	47 (78.3%)	13 (21.7%)	-
Day 3	25 (41.7%)	32 (53.3%)	3 (5.0%)
Pre-discharge	12 (20.0%)	45 (75.0%)	-

Serial Doppler assessments demonstrated progressive improvement in hepatic vein flow patterns during hospitalization. The proportion of patients with normal S:D ratio increased from 16.6% at admission to 31.6% by Day 1, 53.3% by Day 3 and 75% at discharge reflecting a gradual resolution of hepatic venous congestion with therapy.

Table 3: Serial Changes in Portal Vein Pulsatility Index (PI) During Hospital Stay

Time Point	Normal (<0.3)	Mild congestion (0.3–0.5)	Severe congestion (>0.5)	Mean ± SD
Day 1 (Admission)	11 (18.3%)	44 (73.3%)	5 (8.3%)	0.42 ± 0.07
Day 2	13 (21.6%)	44 (73.3%)	3 (5.0%)	0.36 ± 0.06
Day 3	19 (31.6%)	37 (61.6%)	1 (1.6%)	0.34 ± 0.07
Pre-discharge	43 (71.6%)	13 (21.6%)	1 (1.6%)	0.29 ± 0.08

In portal vein assessments, normalization of PI (<0.3) improved from 18.3% at admission to 71.6% by discharge. Mean PI values showed significant reduction from 0.42 ± 0.07 on admission to 0.29 ± 0.08 at discharge. Severe congestion (PI >0.5) was seen in 8.3% initially but dropped to 1.6% by discharge. The sequential fall in portal vein PI reflects not only effective volume management but also underlines its utility in tracking response during hospital stay.

Table 4: Doppler-Based Outcome Classification (Hepatic and Portal Vein Doppler)

Outcome Type	Number of Patients	Percentage (%)
Hepatic Vein Doppler Outcome		
Positive Doppler Findings (Responders)	45	75.0%
Non-responders	12	20.0%
Portal Vein Doppler Outcome		
Positive Doppler Findings (Responders)	43	75.4%
Non-responders	14	24.5%

Based on hepatic vein Doppler outcomes 75.0% of patients responded favorably, while 20.0% remained non-responders. Similarly portal vein Doppler classified 75.4% as responders and 24.5% as non-responders. This shows that though majority improved with therapy, a subset continued to exhibit persistent venous congestion even at discharge highlighting the heterogeneity in response.

Table 5: One-Way ANOVA Summary for Portal Vein Pulsatility Index Across Time Points

Variation	SS	df	MS	F	P-value
Between Groups	0.2731	3	0.0910	19.27	<0.001
Within Groups	1.0721	227	0.0047	-	-
Total	1.3452	230			

The ANOVA analysis confirmed that the serial decline in portal vein PI across different time points was statistically significant (F=19.27, p<0.001). This establishes the sensitivity of portal vein Doppler in detecting dynamic changes in venous congestion during decongestive management.

(A) Day 1: Reversed S:D ratio (D > S), indicating elevated right atrial pressure.
(B) Day 2: Persistent reversal of S:D ratio (D > S).
(C) Day 3: S:D ratio normalized (S > D), reflecting reduction in venous congestion.
(D) Pre-discharge: Normal S:D ratio (S > D), confirming sustained hemodynamic improvement.

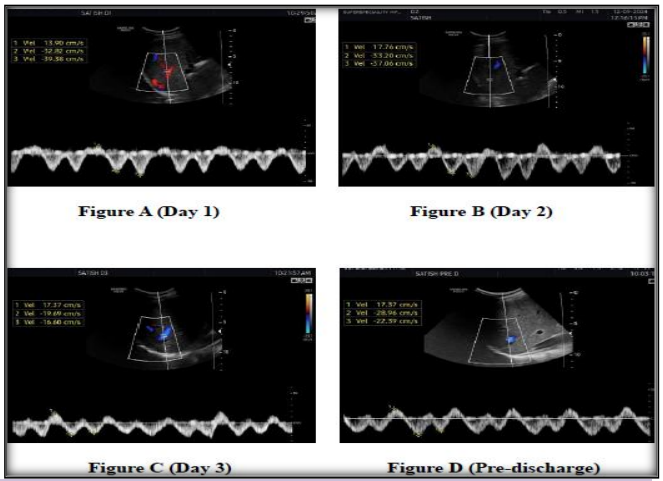

Figure 1a: Serial Hepatic Vein Doppler Changes Showing S:D Ratio Normalization During Decongestion

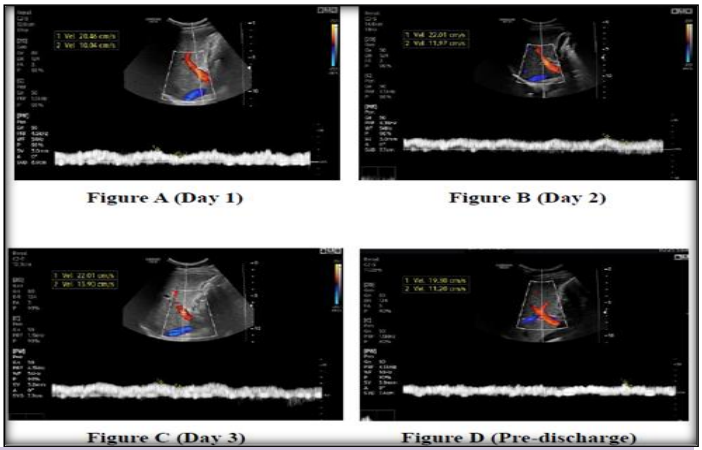

Figure 1B: Serial Portal Vein Doppler Changes Showing Progressive Reduction in Pulsatility Index

(A) Day 1: PI elevated at 0.50.
(B) Day 2: PI remains elevated at 0.49.
(C) Day 3: PI reduces to 0.41.
(D) Pre-discharge: PI normalizes to 0.30, indicating resolution of portal venous congestion.

Discussion

This prospective study demonstrates the value of hepatic vein (HV) Doppler S:D ratio and portal vein pulsatility index (PI) as dynamic, non-invasive indicators of systemic congestion in patients with right heart failure (RHF). Both parameters closely tracked clinical improvement during decongestive therapy.

At admission, abnormal HV waveforms with reversed S:D ratios were seen in 83.3% of patients [Table 1], consistent with elevated right atrial pressure (RAP) and impaired right ventricular compliance. Only 10 of 60 patients (16.6%) demonstrated normal S:D ratios on admission, which improved to 45 of 60 (75%) by discharge [Table 2]. These waveform alterations align with prior findings by Jefkins and Chan who had emphasized that S:D reversal reflects elevated RAP and the hemodynamic burden of tricuspid regurgitation.[15]

Portal vein PI was also elevated at baseline, with only 18.3% of patients exhibiting normal values (<0.3) [Table 3]. With treatment portal vein PI decreased significantly from 0.42 ± 0.07 to 0.29 ± 0.08 (ANOVA F=19.27; p<0.001) [Table 4], representing an approximate 31% relative reduction during hospitalization. Kuwahara et al. similarly reported PVPR reduction from 0.29 ± 0.20 to 0.18 ± 0.15 following therapy in acute HF patients.[16] Notably, their control group exhibited a mean PVPR of 0.08 ± 0.07, highlighting the substantial elevation seen in RHF populations. Improvement in PVPR in their cohort was primarily driven by increased minimum portal vein velocity (12.6 ± 4.5 cm/s to 14.6 ± 4.6 cm/s; p=0.03), indicating restoration of splanchnic venous outflow.[15,16] Ohara et al. introduced the hepatic venous stasis index (HVSI), which showed strong associations with RAP, BNP levels and cardiac outcomes, validating hepatic vein Doppler as a congestion surrogate.[17]

Despite overall improvements, 25% of patients did not normalize their hepatic vein S:D ratios by discharge, identifying a non-responder subgroup [Table 2]. Several plausible mechanisms may account for the persistent non-responders observed in our study. Chronic hepatic congestion may induce sinusoidal fibrosis and structural remodeling of hepatic vasculature, leading to reduced venous compliance even after decongestion. While liver stiffness and FIB-4 indices were not evaluated in this study, prior work by Ohara et al. has linked such structural hepatic changes with persistent hepatic venous Doppler abnormalities.[17] Severe tricuspid regurgitation (TR) may further contribute to these persistent abnormalities, as ongoing regurgitant flow maintains elevated RAP and directly blunts the S-wave component of hepatic vein flow despite systemic volume reduction.[15] In addition, right ventricular-pulmonary arterial (RV-PA) uncoupling may limit effective reduction of right atrial pressures even after systemic decongestion, thereby preventing Doppler normalization in certain patients.[18] Finally, reduced venous compliance from comorbidities such as cirrhosis, chronic kidney disease, diabetes and longstanding hypertension may further impair Doppler waveform reversibility despite appropriate therapy.[17]

Physiologically both the PI and S:D ratios reflect the direct transmission of right atrial pressure into the splanchnic venous system. Shih et al. reported portal vein PI levels of 87.8% ± 32.3% in patients with RAP ≥10 mmHg.[19] A.Arab et al. demonstrated that even preload-responsive healthy individuals exhibited acute PI elevation following volume expansion, underscoring its sensitivity to RAP fluctuations.[10]

Beyond diagnosis portal vein Doppler parameters hold prognostic relevance. Kuwahara et al. demonstrated that elevated PVPR at discharge correlated with increased cardiovascular events over one year.[16] Eljaiek et al. reported that portal vein pulsatility ≥50% independently predicted major postoperative complications, with 18.3% of cardiac surgery patients exceeding this threshold (OR 5.83, CI 2.04–16.68; p=0.001).[18]

Several important limitations exist in the interpretation and application of these Doppler parameters. Firstly technical challenges limit hepatic vein Doppler feasibility particularly in obese or critically ill patients, where acquisition failure rates have been reported as high as 23%.[16] Secondly threshold variability must be recognized across different clinical settings. While a portal vein PI greater than 0.5 is widely accepted as indicative of RHF-related congestion lower thresholds of approximately 0.3 to 0.5 may reflect clinically significant congestion in postoperative cardiac surgery patients.[18,19] Further Abou-Arab et al. demonstrated that portal vein pulsatility can vary physiologically even among healthy individuals after volume expansion, highlighting the need for careful context-specific interpretation of these values.[10]

The observed PI reduction (F=19.27, p<0.001) exceeds thresholds considered clinically meaningful. Prior studies suggest that PVPR reductions ≥0.10–0.12 associate with improved clinical outcomes.[16] The ~0.13 absolute reduction observed here supports its relevance as a marker of meaningful decongestion. While this study was not powered for outcome correlations previously reported odds ratios highlight the prognostic weight of unresolved congestion.[18]

From a clinical standpoint, these findings support incorporating hepatic and portal vein Doppler into bedside volume assessment algorithms. Based on observed trends [Table 4], reassessment by Day 3 may serve as a practical therapeutic checkpoint to evaluate decongestive efficacy. Integration with validated scoring systems such as VExUS, alongside biomarkers (BNP, creatinine), can refine congestion profiling and guide individualized volume management.[20,21]

A key strength of this study is its focus on isolated RHF, a population underrepresented in prior Doppler studies that often evaluated mixed left-right HF or postoperative patients.[18,19] Because venous congestion is the principal driver of morbidity in isolated RHF, non-invasive splanchnic Doppler may offer uniquely actionable insights into dynamic preload assessment in this cohort. In support of this, Murayama et al. demonstrated that hepatic vein systolic filling fraction (HV-SFF), a Doppler-derived parameter, showed an inverse correlation with right atrial

pressure ($r = -0.35$; $p < 0.001$), further validating hepatic vein Doppler as a reliable marker of systemic venous congestion.[22]

Our findings affirm that hepatic and portal vein Doppler provide reproducible, sensitive and real-time assessment of right-sided pressures and response to decongestive therapy. Despite certain technical and population-specific limitations, they represent valuable adjuncts to conventional echocardiographic and biomarker-based assessment strategies in RHF management.

Conclusion

Serial hepatic and portal vein Doppler effectively monitor venous congestion in RHF. Reversal of S:D ratio and fall in portal PI strongly reflect therapeutic response. Persistent abnormalities suggest severe or a irreversible hemodynamic burden and warrant aggressive volume or valve-directed interventions.

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Conflicts of interest

There are no conflicts of interest.

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