ASSESSMENT OF RENAL RESISTIVE INDEX IN HYPERTENSIVE PATIENTS: A DOPPLER ULTRASOUND STUDY

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Abstract

Background: Renal resistive index (RI), measured via Doppler ultrasound, is a non-invasive marker used to assess intrarenal hemodynamics and vascular resistance. Elevated RI has been linked to hypertension-related target organ damage (TOD), including microalbuminuria and left ventricular hypertrophy (LVH). This study aims to evaluate RI in hypertensive individuals, those with a family history of hypertension, and normotensive individuals, and to assess its association with early markers of TOD.
Methods: This prospective, single-center study was conducted at Sree Balaji Medical College & Hospital, Chennai, over six months. A total of 60 patients were enrolled, divided into hypertensive (n=20), normotensive (n=20), and individuals with a family history of hypertension (n=20). Doppler ultrasound was performed to measure RI in interlobar arteries. Additional investigations included albumin-to-creatinine ratio (ACR), renal function tests, electrocardiography (ECG), and echocardiography (ECHO) to evaluate TOD. Multiple regression analysis was used to determine independent predictors of RI.
Results: The prevalence of microalbuminuria and LVH was 14% and 5%, respectively. Hypertensive patients exhibited significantly higher RI values compared to normotensive individuals ($P < 0.001$). Elevated RI correlated positively with systolic blood pressure ($r = 0.50$, $P = 0.05$) and ACR ($r = 0.22$, $P = 0.01$), and negatively with renal volume and diastolic blood pressure. Multiple regression analysis identified age, systolic blood pressure, and ACR as independent predictors of RI.
Conclusion: Renal RI serves as a valuable, non-invasive marker for early detection of hypertensive TOD. Hypertensive patients with increased RI are at greater risk for renal impairment and cardiac remodeling. Incorporating

RI measurements into routine hypertension management may aid in early risk stratification and intervention.

Introduction:

Doppler ultrasound (US) of the renal vasculature is a reliable and non-invasive diagnostic tool that has seen increasing clinical applications in recent years. It plays a crucial role in diagnosing conditions such as renal artery stenosis and renovascular disease (1,2) and is also valuable in assessing intrarenal hemodynamics in various pathological conditions, including essential hypertension (3), acute (4) and chronic renal failure (5,6), and graft rejection (7-9). During the cardiac cycle, several hemodynamic parameters can be derived from the shape of Doppler waveforms recorded at different sites of the renal vasculature, both extra- and intraparenchymally. Doppler ultrasound, which relies on detecting frequency shifts caused by moving blood cells, enables the assessment of both arterial and venous blood flow (10,11). The waveform analysis aids in diagnosis, and peripheral arterial resistance within the kidney can be quantified using the Renal Resistive Index (RI), calculated as:

RI = (Peak Systolic Velocity - End Diastolic Velocity) / Peak Systolic Velocity. A normal RI value is generally considered to be 0.60 ± 0.01 (mean ± SD) by most researchers. This technique can be applied to both native and transplanted kidneys. However, an elevated RI serves as a nonspecific marker of disease and indicates increased peripheral vascular resistance (12). This study aims to explore the relationship between US Doppler RI of the intrarenal vasculature and early signs of organ damage, such as left ventricular hypertrophy (LVH), microalbuminuria, and extracardiac vascular changes in patients with essential hypertension. Given the variability in RI among hypertensive patients with chronic kidney disease (CKD) in previous research, we conducted a USG Renal Doppler study at Sree Balaji Medical College & Hospital, Bharath University, Chrompet, Chennai, to assess RI in hypertensive individuals, those with a family history of hypertension, and normotensive patients.

Aims and objectives

- To analyse the renal resistive index (RI) in patients with hypertension.
- To assess the RI using Doppler ultrasound of interlobar arteries and identify early signs of target organ damage (TOD) in both hypertensive and non-hypertensive individuals, with or without a family history of hypertension, aged ≤60 years at SBMCH, Chennai.
- To compare renal resistive index measurements between hypertensive and non-hypertensive patients.

Materials and methods:

Study Design and Methodology: This study was conducted at the outpatient clinic of our department after receiving approval from the Institutional Ethical Committee of Sree Balaji Medical College & Hospital, Bharath University, Chrompet, Chennai. It was designed as

a single-center, non-randomized, prospective study and was conducted over a period of six months, from March 2014 to August 2014. A total of 60 patients were included in the study after being screened according to specific inclusion and exclusion criteria.

Chronic Kidney Disease (CKD) Criteria: Patients were classified as having CKD based on specific criteria. These included the presence of hypertension for at least six months, a family history of hypertension, or a normotensive status. Kidney damage was identified through pathological abnormalities, markers of kidney dysfunction in blood and urine tests, or imaging studies. Doppler ultrasound findings suggestive of CKD included bilateral contracted kidneys, poor corticomedullary differentiation, or abnormal blood flow measurements in interlobar arteries. Additionally, CKD was assessed using velocity-based parameters, calculated from end-diastolic velocity and peak systolic velocity in renal arteries.

Inclusion and Exclusion Criteria: The study included hypertensive patients aged ≤60 years, normotensive individuals with or without a family history of hypertension, and healthy normotensive controls. Patients with diabetes mellitus or those without overt renal disease were excluded from participation.

Study Description: A detailed clinical history was recorded for all participants, with a focus on symptoms related to kidney disease, including decreased urine output, weakness, dry skin, pruritus, constipation, and weight loss. Additionally, a past medical history of hypertension and acute renal failure was documented. All patients underwent a comprehensive clinical examination, which included height, weight, body mass index (BMI), general physical assessment, and systemic evaluation. Vital signs such as pulse, blood pressure, and temperature were recorded, and a fundoscopic examination was performed to assess hypertensive retinopathy. Hypertension was defined according to JNC 8 criteria, where systolic blood pressure (SBP) of ≥150 mmHg and diastolic blood pressure (DBP) of ≥90 mmHg were considered hypertensive for patients aged ≥60 years, while for those younger than 60 years, hypertension was defined as SBP ≥140 mmHg and DBP ≥90 mmHg. The diagnosis was confirmed based on at least three separate BP readings or the presence of ongoing antihypertensive treatment. All participants followed a salt-restricted diet during the study. On the study day, BP was measured using a mercury sphygmomanometer (cuff size: 12.5 × 40 cm) on the right arm, with the patient in a seated position after 5 minutes of rest. The lowest of three consecutive BP readings was recorded, and Korotkoff Phase 5 was used to determine diastolic BP.

Laboratory Investigations: Various laboratory investigations were conducted, including renal function tests (urea, creatinine), spot microalbuminuria, electrocardiography (ECG), and albumin-to-creatinine ratio (ACR). Family history of hypertension was assessed using a standardized questionnaire, where hypertension was defined as DBP >90 mmHg or a BP level requiring antihypertensive therapy in a parent or sibling.

Albuminuria Assessment: Urinary albumin excretion was measured at the end of the washout period using albumin-to-creatinine ratio (ACR), based on three consecutive first-morning urine samples, ensuring a negative urine culture. ACR was calculated as urine

albumin concentration (mg/L) divided by urine creatinine concentration (mmol/L). Creatinine levels were determined using the Jaffe reaction, and albumin levels were analyzed using a radioimmunoassay kit. The criteria used had high sensitivity and specificity for detecting microalbuminuria, defined as an albumin excretion rate between 20–200 mcg/min.

Renal Ultrasound and Doppler Studies: Renal parenchymal echogenicity, renal volume, and mean resistive index (RI) were evaluated in 60 kidney pairs at the end of the study. Renal parenchymal echogenicity was classified based on Hricak's grading system, while renal volume was measured using the ellipsoid formula. Doppler signals were obtained from the interlobar arteries by placing the sample at the edge of the medullary pyramids. Measurements were repeated in different regions of both kidneys (superior, median, and lower poles) to obtain consistent readings. The mean RI was calculated as:
RI = (Peak Systolic Velocity – End Diastolic Velocity) / Peak Systolic Velocity
Each patient's RI was determined using six measurements, three from each kidney.

Echocardiography: The left ventricular mass index (LVMI) was assessed using standard echocardiographic techniques while the patient was at rest in the left lateral position. Echocardiographic images were obtained using parasternal and apical views, following the guidelines of the American Society of Echocardiography. The LV mass was calculated using Devereux's formula, corrected for body surface area (BSA) and expressed in g/m². A single blinded observer analyzed all echocardiograms to eliminate bias. None of the patients showed dysynergic left ventricular segments, ensuring accurate mass calculations.

Statistical analysis: All data are expressed as mean±SEM. Differences between variables are assessed using the appropriate statistical tests based on the underlying distribution of the variables. To study the linear relationship between RI and other variables. Multiple regression analysis was performed to assess the independent contribution of several variables on RI.

Results:

The clinical characteristics of the study participants are summarized in Table 1. The overall prevalence of microalbuminuria and left ventricular hypertrophy (LVH) was 14% and 5%, respectively, findings that align with previously published literature on untreated hypertensive patients.

Table 1. Clinical Characteristics of Study Patients

Parameter	Hypertensive (n=20)	Normotensive (n=20)	Family History of Hypertension (n=20)	P-value
SBP (mmHg)	136 ± 3	121 ± 1	127 ± 3	**<0.05**
DBP (mmHg)	96 ± 0.7	75 ± 0.5	83 ± 0.6	NS
Mean Arterial Pressure (mmHg)	100 ± 0.7	90 ± 1.0	95 ± 0.6	NS
Age (years)	62.6 ± 2.1	42.5 ± 1.4	50.5 ± 2.5	NS

Family History of Hypertension (%)	78	43	65	NS
Serum Creatinine (µmol/L)	86.9 ± 0.9	69 ± 1.1	79.8 ± 1.0	**<0.001**
Creatinine Clearance (mL/min)	92 ± 2.1	72 ± 2.2	87 ± 1.6	**<0.001**
Resistive Index (RI)	0.761 ± 0.009	0.576 ± 0.004	0.667 ± 0.002	**<0.001**
LVMI (g/m²)	125.0 ± 2.5	120.3 ± 3.3	123.3 ± 2	NS
LVH (%)	59	36	45	**<0.01**

A significant positive correlation was observed between renal resistive index (RI) and systolic blood pressure (SBP), as well as early markers of end-organ damage, such as albumin-to-creatinine ratio (ACR). Conversely, RI exhibited a negative correlation with renal volume and diastolic blood pressure (DBP). These associations are highlighted in Table 2, which presents the univariate correlation between RI and selected clinical parameters.

Table 2. Univariate Correlation Between RI and Selected Clinical Variables

Variable	Correlation Coefficient (R)	P-value
ACR	0.22	**0.01**
Age	0.25	**0.003**
SBP	0.50	**0.05**
DBP	-0.23	**0.006**

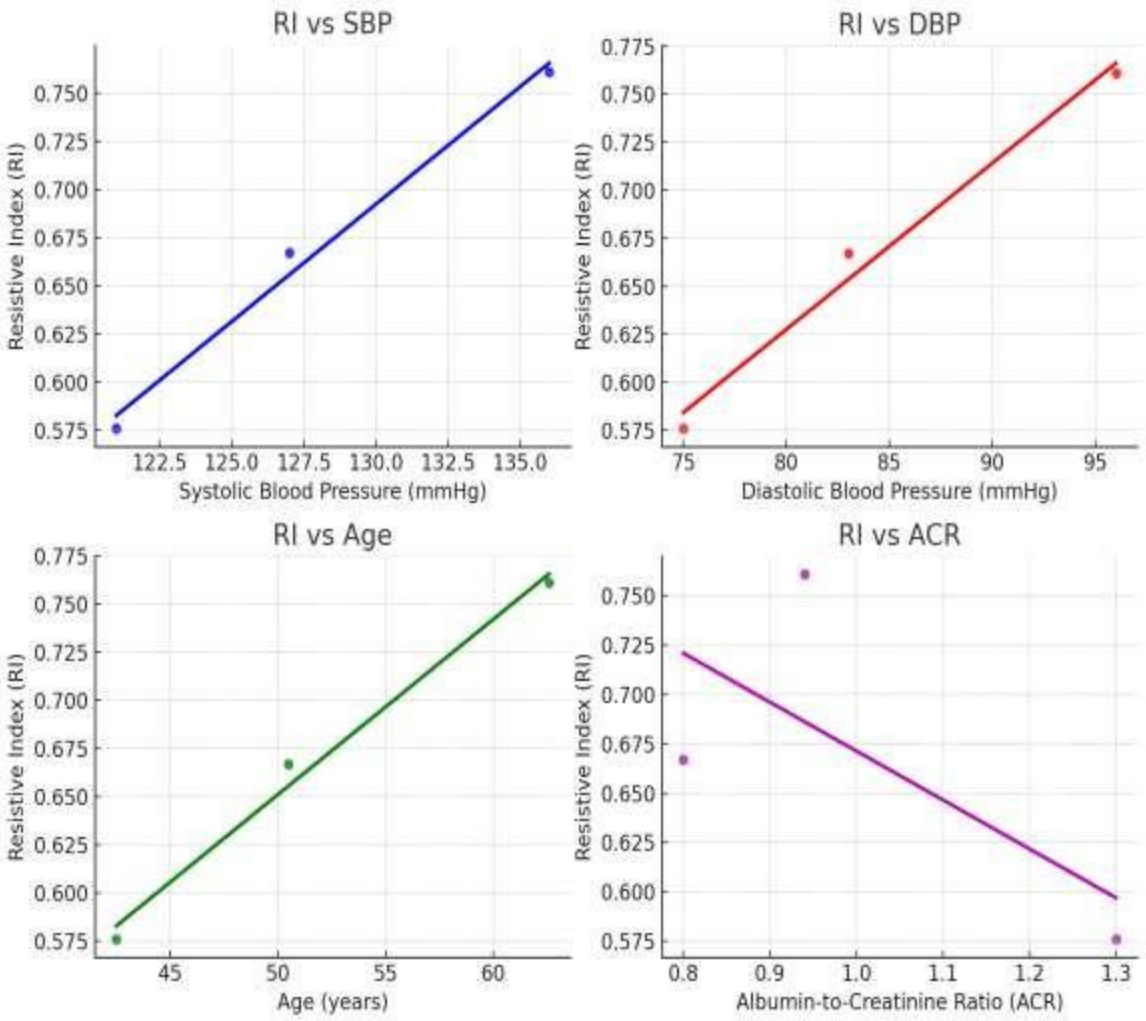
Further analysis using multiple regression models revealed that renal vascular resistance is significantly influenced by age, urinary albumin excretion, and SBP. These three factors collectively accounted for approximately 20% of the variations in renal vascular impedance, as detailed in Table 3.

Additionally, when patients were stratified based on RI quartiles, those in the highest quartile had significantly elevated SBP (136 ± 3 mmHg vs. 121 ± 1 mmHg vs. 127 ± 3 mmHg, $P < 0.05$) and increased albuminuria (0.94 ± 0.2 vs. 1.3 ± 0.4 vs. 0.8 ± 0.1 vs. 3.5 ± 1.0, $P = 0.002$). Furthermore, patients with the highest RI values also exhibited a significantly higher prevalence of microalbuminuria and LVH.

Table 3. Multiple Regression Analysis of RI

Independent Variable	β Coefficient	Standard Error (SEβ)	Partial F	P-value
SBP	0.000545	0.000288	1.894	0.06
Age	0.00124	0.000503	2.462	**0.0152**
ACR	0.002955	0.001366	2.164	**0.0323**
Constant	0.4026	0.0508	7.92	**0.00001**

Figure 1: Resistant Index



Discussion

The present study aimed to elucidate the relationship between renal resistive index (RI) and early markers of target organ damage (TOD) in hypertensive patients. Our findings indicate a significant association between elevated RI and increased systolic blood pressure (SBP), microalbuminuria, and left ventricular hypertrophy (LVH). These results underscore the potential of RI as a non-invasive marker for early detection of TOD in individuals with hypertension.

In our cohort, the prevalence of microalbuminuria was 14%, while LVH was observed in 5% of participants. These figures are consistent with previous studies on untreated hypertensive populations. For instance, Pontremoli et al. reported a microalbuminuria prevalence of 13% and LVH prevalence of 40% in a similar cohort [13]. The slight differences in LVH prevalence may be attributed to variations in study populations and diagnostic criteria. Our data demonstrated a positive correlation between RI and SBP ($r = 0.50$, $P = 0.05$), suggesting that increased renal vascular resistance is associated with higher systolic pressures. This finding aligns with previous research indicating that elevated SBP contributes to increased intrarenal arterial stiffness, thereby raising RI values [13]. Conversely, a negative correlation was observed between RI and diastolic blood pressure

(DBP) (r = -0.23, P = 0.006). This inverse relationship may reflect the complex hemodynamic interactions within the renal vasculature, where elevated SBP and reduced DBP collectively contribute to increased pulse pressure, potentially exacerbating renal microvascular damage.

The study found a significant positive correlation between RI and the albumin-to-creatinine ratio (ACR) (r = 0.22, P = 0.01), indicating that higher RI values are associated with increased urinary albumin excretion. Microalbuminuria is a well-established marker of glomerular endothelial dysfunction and has been linked to adverse cardiovascular outcomes [14]. The observed association suggests that elevated renal vascular resistance, as indicated by increased RI, may precede and contribute to glomerular injury, leading to microalbuminuria.

Our analysis revealed that patients in the highest RI quartile exhibited a significantly higher prevalence of LVH (59%) compared to those in lower quartiles. This association underscores the interplay between renal and cardiac target organ damage in hypertension. Elevated RI may reflect systemic arterial stiffness and increased afterload, contributing to left ventricular remodeling and hypertrophy. This finding is consistent with studies that have demonstrated a link between increased arterial stiffness, as measured by pulse wave velocity, and the development of LVH in hypertensive patients [15].

Multiple regression analysis identified age, SBP, and ACR as independent predictors of RI, collectively accounting for approximately 20% of its variability. This finding suggests that while these factors significantly influence renal vascular resistance, other unmeasured variables may also contribute to RI variations. Potential contributors include genetic predispositions, inflammatory markers, and the presence of comorbid conditions such as diabetes mellitus or dyslipidemia (16). Further research is warranted to explore these additional factors and their impact on RI. The significant associations between elevated RI and markers of TOD highlight the utility of RI as a non-invasive tool for early detection of organ damage in hypertensive patients (17). Incorporating renal Doppler ultrasonography into routine clinical practice may facilitate the identification of individuals at heightened risk for cardiovascular and renal complications, enabling timely intervention. Moreover, monitoring RI over time could serve as a valuable marker for assessing the efficacy of antihypertensive therapies and guiding treatment adjustments (18).
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Several limitations of this study should be acknowledged. The cross-sectional design precludes the establishment of causal relationships between elevated RI and target organ damage. Longitudinal studies are necessary to determine whether increased RI predicts the progression of organ damage over time. Additionally, the sample size was relatively small, which may limit the generalizability of our findings. Future studies with larger, more diverse populations are needed to confirm these associations (19). Finally, while RI is a useful marker of renal vascular resistance, it does not provide direct information about the underlying histopathological changes. Combining RI assessment with other imaging modalities or biomarkers may offer a more comprehensive evaluation of renal and systemic vascular health (20).
In summary, our study demonstrates that elevated renal resistive index is significantly

associated with increased systolic blood pressure, microalbuminuria, and left ventricular hypertrophy in hypertensive patients. These findings support the use of RI as a non-invasive marker for early detection of target organ damage, which may facilitate timely therapeutic interventions aimed at reducing the burden of hypertension-related complications (21).

Conclusion

This study highlights the significant association between an elevated renal resistive index (RI) and early markers of target organ damage (TOD) in hypertensive patients. Our findings demonstrate that increased RI correlates positively with systolic blood pressure (SBP), microalbuminuria, and left ventricular hypertrophy (LVH), underscoring its potential as a non-invasive marker for early detection of hypertensive complications.

Among the study population, 78.4% of hypertensive individuals exhibited increased RI, suggesting a higher susceptibility to renal impairment. Furthermore, 23.6% of patients with a family history of hypertension also had elevated RI, indicating a potential predisposition to vascular dysfunction. In contrast, normotensive individuals showed no significant changes in RI, reinforcing the role of hypertension in driving renal vascular resistance. The positive correlation between RI and albumin-to-creatinine ratio (ACR) suggests that renal microvascular damage may contribute to glomerular dysfunction, leading to microalbuminuria. Additionally, the strong association between RI and LVH highlights the interplay between systemic arterial stiffness and cardiac remodeling in hypertension. These findings are consistent with previous research, further validating the utility of RI in identifying early hypertensive end-organ damage. From a clinical perspective, our results support the integration of Doppler ultrasound-based RI measurement in routine hypertension management. Early detection of increased RI may facilitate timely interventions, including blood pressure control and renal function monitoring, to prevent the progression of organ damage. Moreover, serial RI assessments may serve as a valuable tool for evaluating the efficacy of antihypertensive therapies and adjusting treatment strategies accordingly. Despite these significant findings, this study has certain limitations. The cross-sectional design restricts causal inferences, necessitating longitudinal studies to establish whether elevated RI predicts the progression of organ damage over time. Additionally, the relatively small sample size may limit the generalizability of our findings, emphasizing the need for larger, multicenter studies to validate these results. Further research exploring genetic, inflammatory, and metabolic factors influencing RI will provide a more comprehensive understanding of its role in hypertension-related vascular changes. In conclusion, the renal resistive index emerges as a promising, non-invasive marker for detecting early hypertensive organ damage. By incorporating RI measurements into clinical practice, healthcare providers may enhance the early identification of at-risk individuals, allowing for prompt therapeutic interventions to mitigate the long-term complications of hypertension.

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