ORIGINAL RESEARCH PAPER
Volume - 15 | Issue - 06 | June - 2026 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

STUDY OF RENAL RESISTIVE INDEX IN DIABETIC KIDNEY DISEASE

General Medicine

Dr. Afsal Mohammed Hussain — Department of General Medicine, Kanachur Institute of Medical Sciences, Mangalore, Karnataka, India

Dr. Devadas Rai* — HOD & Professor, Department of General Medicine, Kanachur Institute of Medical Sciences and Research Centre, Mangalore, Karnataka, India*Corresponding Author

ABSTRACT

Background: Approximately 1 in 3 adults with diabetes develops kidney disease. Diabetic nephropathy accounts for 15%-40% of patients with end-stage renal disease (ESRD) globally. Early detection of renal hemodynamic changes before measurable decline in estimated glomerular filtration rate (eGFR) remains a major clinical challenge. **Objective:** To assess renal resistive index (RRI) in diabetic patients with and without nephropathy, and to examine its relationship with eGFR and urine albumin-to-creatinine ratio (UACR). **Methods:** This cross-sectional study was conducted at the Department of General Medicine, Kanachur Institute of Medical Sciences, from May 2024 to December 2025. A total of 102 participants were divided into three groups (n=34 each): Group 1 (DM with diabetic kidney disease), Group 2 (DM without complications), and Group 3 (age/sex-matched healthy controls). Renal Doppler ultrasonography was performed under standardized fasting conditions. RRI was derived using the formula: (PSV - EDV) / PSV from interlobar arteries bilaterally. Data were analyzed using SPSS v20; ANOVA for continuous variables andhi-square for categorical data ($p<0.05$ considered significant). **Results:** Mean RRI was 0.90+/-0.15 (Group 1), 0.74+/-0.11 (Group 2), and 0.56+/-0.07 (Group 3), with statistically significant differences ($F=70.346$, $p=0.001$). Mean eGFR was 15.8, 91.4, and 89.46 ml/min/1.73 m2 respectively. UACR averaged 123.32, 10.9, and 9.12 mg/g across groups. Both diabetic groups showed elevated RRI compared to healthy controls, with the highest values in established nephropathy. **Conclusion:** RRI by renal Doppler ultrasonography is a reliable, non-invasive tool for early detection of kidney involvement in diabetic patients, even before microalbuminuria or serum creatinine become abnormal. It correlates significantly with nephropathy stage, eGFR, and UACR.

KEYWORDS

Renal Resistive Index, Diabetic Kidney Disease, Doppler Ultrasonography, Egfr, Diabetic Nephropathy

INTRODUCTION

Type 2 diabetes mellitus (T2DM) has emerged as a global public health crisis. The World Health Organization estimated approximately 35 million people in India were living with diabetes as of 2007, with projections indicating a rise to nearly 70 million by 2025 (Balasubramanyam & Mohan, 2007). T2DM represents roughly 90% of all diagnosed cases globally. Diabetic nephropathy stands as the leading cause of kidney failure worldwide, reported in approximately 15%-40% of patients suffering from ESRD (Rossing, 2006).

Early identification of renal hemodynamic changes preceding measurable eGFR decline remains a clinical challenge. Conventional markers such as serum creatinine and urine albumin often become abnormal only after significant parenchymal damage has occurred. Renal Doppler ultrasonography offers a non-invasive, radiation-free modality that can detect early disturbances in intrarenal blood flow and is safe for patients with pacemakers or metal implants (Viazzi et al., 2014).

The Renal Resistive Index (RRI), calculated as (Peak Systolic Velocity - End Diastolic Velocity) / Peak Systolic Velocity, has been proposed as a sensitive marker of renal vascular resistance and an early predictor of nephropathy progression. Normal RRI values typically fall between 0.50 and 0.70; an elevated RRI (>0.70) often signals poor renal prognosis (Spatola & Andrulli, 2016). This study aimed to examine the relationship between RRI and renal function and to identify potential predictors of elevated RRI among diabetic individuals with and without clinical evidence of nephropathy.

AIMS AND OBJECTIVES

1. To assess renal resistive index in diabetes mellitus patients, including those with and without nephropathy.
2. To examine renal resistive index, eGFR, and urine microalbuminuria in diabetes mellitus patients and their inter-relationships.

MATERIALS AND METHODS

Study Design and Setting

This cross-sectional study was conducted at the Department of General Medicine, Kanachur Institute of Medical Sciences, Mangalore , India, from May 2024 to December 2025. The study began only after receiving ethics committee approval, and every participant provided signed consent before inclusion.

Study Groups

A total of 102 participants were enrolled and divided into three equal groups (n=34 each): Group 1 - Diabetes mellitus with diabetic kidney disease (DKD); Group 2 - Diabetes mellitus without complications; Group 3 - Age- and sex-matched healthy individuals.

Inclusion Criteria

Participants were selected based on the American Diabetes Association diagnostic framework for diabetic nephropathy: (a) persistently elevated urinary albumin levels exceeding 300 mg/day or 200 microg/min, verified at least twice over a 3-6 month period; (b) a gradual drop in glomerular filtration rate; and (c) elevated arterial blood pressure.

Exclusion Criteria

Non-diabetic kidney disease, renal artery stenosis, active urinary tract infection, urinary tract abnormalities, hypotension, bradycardia, and hemoglobinopathy.

Sample Size

Sample size was estimated using observed differences in proportion of elevated RRI from Jinadu et al. (2022): 30% (DM without nephropathy), 43.4% (DM with nephropathy), and 12.5% (healthy controls). Using the formula $N = 3(Za/2 + Zb)2\ P(1-P)/(p1-p2)2$ at alpha=0.05 and 80% power, a minimum of 34 per group was calculated.

Measurements and Procedure

All patients underwent standardized assessment including history, physical examination, renal function tests (semi-automated chemistry), HbA1c, fasting lipid profile, urine albumin-to-creatinine ratio, and renal arterial Doppler ultrasonography. Patients fasted overnight (minimum 8 hours) prior to Doppler examination to minimize bowel gas artifact. PSV and EDV were obtained from interlobar arteries bilaterally using M-mode analysis. Three to five reproducible waveforms were captured per kidney. RRI was derived using the standard formula: RRI = (PSV - EDV) / PSV. The average of three readings per kidney was used for final analysis. eGFR was calculated using the CKD-EPI equation.

Statistical Analysis

Data were entered in Microsoft Excel and analyzed using SPSS version 20. Qualitative variables were summarized as proportions and

illustrated using bar charts. Quantitative variables were described using means and standard deviations. ANOVA was used for numerical variables; chi-square for categorical data. A p-value under 0.05 indicated statistical significance.

RESULTS

Table 1. Comparison of Key Clinical and Biochemical Parameters Across Study Groups

Parameter	Group 1 (DM+DKD)	Group 2 (DM Only)	Group 3 (Controls)
Predominant Age Group	51-60 yrs (41.2%)	41-50 yrs (55.9%)	31-40 yrs (47.1%)
Sex Female : Male	21 : 13	19 : 15	19 : 15
DM Duration (Mean+/-SD, yrs)	9.7 +/- 6.7	7.8 +/- 5.4	N/A
HbA1c % (Mean+/-SD)	7.58 +/- 1.2	7.87 +/- 1.31	5.6 +/- 0.4
Serum Creatinine (mg/dL)	2.3 +/- 0.66	1.25 +/- 0.43	0.72 +/- 0.6
BUN (mg/dL)	46.2 +/- 28.1	33.3 +/- 20.4	31.0 +/- 7.8
eGFR (ml/min/1.73 m2)	15.8 +/- 9.4	91.4 +/- 4.9	89.46 +/- 4.36
UACR (mg/g)	123.32 +/- 87.9	10.9 +/- 7.54	9.12 +/- 4.13
RRI (Mean +/- SD)*	0.90 +/- 0.15	0.74 +/- 0.11	0.56 +/- 0.07

*F=70.346, p=0.001 (Significant). DKD=Diabetic Kidney Disease; DM=Diabetes Mellitus; N/A=Not Applicable; UACR=Urine Albumin-to-Creatinine Ratio; RRI=Renal Resistive Index.

Table 2. Distribution of Kidney Disease Stage Across Study Groups

CKD Stage	Group 1 n (%)	Group 2 n (%)	Group 3 n (%)
Nil / Normal	0 (0)	0 (0)	34 (100.0)
Stage 1	0 (0)	20 (58.8)	0 (0)
Stage 2	12 (35.3)	14 (41.2)	0 (0)
Stage 3	16 (47.1)	0 (0)	0 (0)
Stage 4	6 (17.6)	0 (0)	0 (0)

Chi-square = 165.231, df = 8, p = 0.000 (Significant).

Age and Sex: The highest proportion of Group 1 participants were aged 51-60 years (41.2%), Group 2 patients were predominantly in the 41-50 year range (55.9%), and Group 3 controls were mostly aged 31-40 years (47.1%). This age-group difference was statistically significant (chi-square=42.801, p<0.001). Female participants predominated across all three groups (61.8%, 55.9%, and 55.9% respectively); gender distribution was not statistically significant (p=0.851).

Diabetes Duration and Medication: Average diabetes duration was 9.7 years in Group 1 and 7.8 years in Group 2 (p=0.216, NS). Approximately 50.0% of Group 1 and 47.1% of Group 2 were on oral hypoglycemic agents; the remainder were on insulin or combination therapy. This difference was statistically significant (chi-square=104.175, p<0.001).

Biochemical Parameters: Mean HbA1c levels were 7.58%, 7.87%, and 5.6% in Groups 1, 2, and 3 (F=46.577, p<0.001). Mean serum creatinine was 2.3, 1.25, and 0.72 mg/dL respectively (F=65.195, p<0.001). BUN levels averaged 46.2, 33.3, and 31.0 mg/dL (F=5.395, p=0.001). All differences were statistically significant.

Renal Resistive Index: Mean RRI was 0.90+/-0.15 in Group 1 (DM+DKD), 0.74+/-0.11 in Group 2 (DM without complications), and 0.56+/-0.07 in Group 3 (healthy controls). This difference was statistically significant (F=70.346, p=0.001). Both diabetic groups showed markedly elevated RRI compared to healthy controls, with the highest values observed in those with established DKD.

Renal Function Indices: Mean eGFR was 15.8+/-9.4 ml/min/1.73 m2 in Group 1, 91.4+/-4.9 in Group 2, and 89.46+/-4.36 in Group 3 (F=1437.157, p<0.001). Mean UACR levels were 123.32+/-87.9, 10.9+/-7.54, and 9.12+/-4.13 mg/g respectively (F=55.95, p<0.001). All differences were statistically significant.

DISCUSSION

This study demonstrated that renal resistive index is significantly elevated in diabetic patients even without overt nephropathy compared to healthy controls. This is consistent with findings by Jinadu et al. (2022), who reported significantly greater mean intra-renal artery resistance indices in diabetics (0.60+/-0.04) versus controls (0.56+/-0.04, p=0.02). In that study, high RRI in patients without nephropathy was associated with elevated HbA1c (OR 2.81; CI: 1.73-9.03), highlighting glycemic control as a key driver of early vascular changes.

In a study by Tahir et al. (2020) involving 150 type 2 diabetic patients with mean HbA1c of 7.58, bilateral RRI averaged 0.72 - consistent with our Group 2 findings. Sistani et al. (2019) investigated 100 patients with diabetic nephropathy and reported strong associations between RRI and systolic blood pressure (R=0.75), UACR (R=0.67), and eGFR (R=0.76), paralleling our findings. Li et al. (2022) demonstrated that an RRI threshold of 0.66 predicted diabetic kidney disease with 69.2% sensitivity and 80.9% specificity - supporting the utility of RRI as a discriminatory marker even before conventional parameters become abnormal.

The significantly elevated serum creatinine (2.3 mg/dL), reduced eGFR (15.8 ml/min/1.73 m2), and markedly elevated UACR (123.32 mg/g) in Group 1 confirm established kidney disease; while the elevated RRI in Group 2 (0.74) - where eGFR was preserved and UACR near-normal - underscores the potential of RRI as a pre-clinical marker. This aligns with the observation by Shirin et al. (2015) that 73.6% of diabetic patients had RRI less than 0.7, with a mean of 0.71, and positive associations with serum creatinine and albuminuria. Their study concluded that RRI from Duplex Doppler ultrasonography is an effective diagnostic tool for kidney failure in diabetic nephropathy, with high correlation to serum creatinine and albuminuria.

Physical activity was significantly lower in diabetic groups compared to controls (chi-square=12.812, p=0.002), and the proportion of sedentary participants was highest in Group 1 (64.7%). This is consistent with the well-established relationship between physical inactivity and accelerated nephropathy progression in T2DM. The predominantly female distribution in our cohort (61.8%, 55.9%, 55.9%) contrasts with male predominance reported by Sistani et al. and Li et al., which may reflect regional referral patterns. The higher proportion of Stage 3 CKD in Group 1 (47.1%) confirms the expected spectrum of advanced diabetic kidney disease.

SUMMARY

This study evaluated the renal resistive index (RRI) across three groups: diabetic patients with nephropathy, those without nephropathy, and healthy controls. Findings revealed that both groups of diabetic individuals exhibited higher RRI values compared to the control group, with the highest indices observed in those with established diabetic kidney disease. Diabetic nephropathy was associated with elevated clinical markers such as reduced eGFR, increased UACR, and more advanced stages of renal impairment. The study shows patients with and without diabetic nephropathy exhibited high RRI compared to healthy controls. RRI helps in early recognition of kidney failure even before microalbuminuria and serum creatinine become abnormal. Hence RRI is a better non-invasive tool for early recognition of renal failure.

LIMITATIONS

This study was conducted at a single centre only. There can be individual variability in RI, and it cannot differentiate early and late kidney changes. Longitudinal follow-up data were not available to assess prognostic outcomes.

CONCLUSION

This study evaluated the renal resistive index across three groups and demonstrated that both groups of diabetic individuals exhibited higher RRI values compared to the control group, with the highest indices in those with established diabetic kidney disease. RRI shows potential as a first indicator of renal involvement in diabetes, correlating significantly with eGFR, UACR, serum creatinine, and disease stage. While the present study offers valuable contributions to understanding the role of RRI in diabetic kidney disease, further large-scale and longitudinal research is warranted to confirm its prognostic utility and broader clinical application.

REFERENCES:

[1] Balasubramanyam, M., & Mohan, V. (2007). Diabetes in 2007: What are the problems

and challenges? Indian Journal of Medical Research, 125, 195-199.
[2] Jinadu, Y. O., Raji, Y. R., Ajayi, S. O., Salako, B. L., Arije, A., & Kadiri, S. (2022). Resistivity index in the diagnosis and assessment of loss of renal function in diabetic nephropathy. Cardiovascular Journal of Africa, 33(1), 26-32.
[3] Li, H., Shen, Y., Yu, Z., Huang, Y., He, T., Xiao, T., Li, Y., Xiong, J., & Zhao, J. (2022). Potential role of the renal arterial resistance index in the differential diagnosis of diabetic kidney disease. Frontiers in Endocrinology, 12, 731187.
[4] Maksoud, A. A. A., Sharara, S. M., Nanda, A., & Khouzam, R. N. (2019). The renal resistive index as a new complementary tool to predict microvascular diabetic complications in children and adolescents. Annals of Translational Medicine, 7(17), 422.
[5] Rossing, P. (2006). Diabetic nephropathy: Worldwide epidemic and effects of current treatment on natural history. Current Diabetes Reports, 6(6), 479-483.
[6] Shirin, M., Sharif, M. M., Gurung, A., & Datta, A. (2015). Resistive index of intrarenal artery in evaluation of diabetic nephropathy. Bangladesh Medical Research Council Bulletin, 41, 125-130.
[7] Sistani, S. S., Alidadi, A., Moghadam, A. A., Mohamadnezhad, F., & Ghahderijani, B. H. (2019). Comparison of renal arterial resistive index in type 2 diabetic nephropathy stage 0-4. European Journal of Translational Myology, 29(4), 8364.
[8] Spatola, L., & Andrulli, S. (2016). Doppler ultrasound in kidney diseases: A key parameter in clinical long-term follow-up. Journal of Ultrasound, 19(4), 243-250.
[9] Tahir, S., Shafique, M., Ali, D., Khan, M. B., Sadiq, T., & Rahman, H. (2020). Determination of renal resistivity index in patients having type II diabetes mellitus. Pakistan Armed Forces Medical Journal, 70(3), 649-653.
[10] Viazzi, F., Leoncini, G., Derchi, L. E., & Pontremoli, R. (2014). Ultrasound Doppler renal resistive index: A useful tool for the management of the hypertensive patient. Journal of Hypertension, 32(1), 149-153.