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Research Article

Utility of Urinary Nephrin in Patients With and Without Diabetic Nephropathy- A Cross Sectional Study

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ABSTRACT

Background: Diabetic nephropathy being a major microvascular complication of diabetes mellitus type 2, its early detection helps in preventing irreversible renal injury. Urinary Nephrin, which is a podocyte-derived biomarker,has emerged as a potential bio5;l]marker of early glomerular injury.
Objectives: To assess the urinary Nephrin levels in individuals with type 2 diabetes mellitus, diabetic nephropathy, and healthy controls and to evaluate its diagnostic ability in identifying patients with diabetic nephropathy.
Methods: it was a cross-sectional analytical study, conducted in 88 participants divided into three groups: Type 2 Diabetes mellitus (n = 28), diabetic nephropathy (n = 30), and healthy controls (n = 30). Urine samples were analyzed for urinary creatinine, urinary albumin–creatinine ratio (UACR), urinary Nephrin, and Nephrin–Creatinine ratio using the standard protocols. Correlation of urinary Nephrin levels with other parameters were analyzed using Pearson's correlation coefficient. Diagnostic accuracy was evaluated using receiver operating characteristic (ROC) curve analysis. All the statistical tests were conducted using SPSS software (version 29).
Results: Urinary Nephrin levels were significantly higher in patients with Diabetic Nephropathy (115.80 ± 12.10 ng/mL) compared to those with type 2 diabetes (22.56 ± 6.40 ng/mL) and control groups (5.30 ± 2.10 ng/mL) ($p = 0.01$). Urinary Nephrin showed strong positive correlations with UACR ($r = 0.66$) and a negative correlation with eGFR ($r = –0.54$). ROC analysis demonstrated excellent diagnostic accuracy (AUC = 0.94), with a cut-off > 96.3 ng/mL yielding 93.21% sensitivity and 74.46% specificity.
Conclusion: Urinary nephrin is a highly promising biomarker for early detection of diabetic nephropathy, demonstrating strong diagnostic utility and significant correlation with renal impairment.

Keywords: Urinary nephrin, diabetic nephropathy, UACR, podocyte injury, ROC analysis.

INTRODUCTION

Diabetic nephropathy is among the most important microvascular complications of diabetes mellitus and remains a major contributor to chronic kidney disease and end-stage renal failure worldwide[1]. Though better treatment results has been observed through improved blood sugar management and supportive care, yet many diabetic patients still develop kidney problems which remain hidden for extended periods until their symptoms become apparent[2]. Early detection of subclinical renal injury plays a vital role in preventing kidney damage while enabling fast medical treatment which leads to better long-term health results.

Accumulating evidence suggests that podocyte injury represents one of the earliest structural abnormalities in the development of diabetic nephropathy [3,4,5] . Podocytes maintain the integrity of the glomerular filtration barrier, and subtle alterations in their structure or function may occur well before albuminuria becomes clinically evident [6]. Progressive podocyte injury disrupts slit diaphragm architecture, ultimately contributing to the development of microalbuminuria and, with continued damage, overt proteinuria[7] .Hence lots of research is going on, to identify biomarkers that specifically reflect podocyte dysfunction [8].

Nephrin is a transmembrane protein, which being a basic component of slit diaphragm, controls the selective glomerular permeability [9] . Damage to podocytes results in changes to nephrin production and release of nephrin fragments which appear in urine as nephrinuria [10]. Several studies have demonstrated that elevated urinary nephrin levels can be detected before the appearance of overt proteinuria, suggesting that nephrinuria reflects early glomerular structural damage rather than late functional decline [11,12] . As such, urinary nephrin represents a promising non-invasive marker for detecting subclinical podocyte injury.

In contrast to nephrinuria, traditional markers such as urinary albumin-creatinine ratio (UACR) and serum creatinine typically rise only after significant nephron loss has already occurred [13] . As urinary Nephrin, appears to increase earlier in disease course, it helps in the identification of renal involvement, even while the structural damage may be modifiable. This characteristic feature makes Nephrin a potential marker to identify the risk stratification& clinical monitoring of patients with Diabetic Kidney Disease [14] .

The present study therefore aimed to assess the urinary Nephrin levels among individuals with diabetes, diabetic nephropathy, and non-diabetic controls, and to examine its diagnostic ability to detect patients with Diabetic Nephropathy at an earlier stage.

MATERIALS AND METHODS:

Study Design and Setting: This study was designed as a cross-sectional analytical study conducted in the outpatient and inpatient departments of a tertiary care hospital in Tamilnadu. All laboratory investigations, including renal function tests and urinary biomarker estimations, were performed in the hospital's central biochemistry laboratory using standardized protocols.

Study Population: The study population comprised three groups: individuals with type 2 diabetes mellitus without nephropathy, individuals diagnosed with diabetic nephropathy, and healthy controls without diabetes or renal disease. Participants were recruited consecutively from the general medicine units during the study period. Adults aged 35–70 years known cases of type 2 diabetes mellitus with or without nephropathy and healthy controls with normal fasting glucose and no history of renal or systemic illness were included. Pregnant women, individuals with acute or chronic renal conditions unrelated to diabetes, patients with urinary tract infections, hematuria, or systemic inflammatory disorders were excluded. Diabetic nephropathy patients included those with microalbuminuria (UACR : 30–300 mg/24 h, or 30–300 mg/g creatinine) or persistent macroalbuminuria (UACR > 300 mg/24 h, or > 300 mg/g creatinine).

Sample Size and Sampling Technique: A total of 88 participants were included, comprising 28 individuals with diabetes, 30 individuals with diabetic nephropathy, and 30 healthy controls. The sample size was determined based on expected differences in urinary nephrin levels reported in previous literature, with a power of 80% and a 5% level of significance.[1] A consecutive sampling technique was employed to recruit eligible participants until the required sample size was achieved.

After obtaining informed consent, demographic details, anthropometric measurements (height, weight, BMI, waist circumference) and clinical history were documented using a structured proforma.

A fasting venous blood sample was collected for renal function markers (serum urea, serum creatinine, and eGFR) and glycemic parameters (fasting blood glucose, HbA1c). An early morning first midstream spot urine sample was collected for estimation of urine creatinine, urinary albumin–creatinine ratio (UACR), urinary nephrin levels, and nephrin–creatinine ratio.
Urinary nephrin was quantified using a commercially available enzyme-linked immunosorbent assay (ELISA) kit following manufacturer guidelines. All biochemical measurements were performed using calibrated automated analyzers, and quality control protocols were adhered to throughout the testing process. UACR & Nephrin creatinine ratio were calculated.

Statistical Analysis: Data were entered into Microsoft Excel and analyzed using SPSS (version 26). Continuous variables were expressed as mean ± standard deviation. Group comparisons were performed using ANOVA with post-hoc analysis where appropriate. Pearson's correlation coefficients were used to assess the relationship between urinary nephrin and other clinical and biochemical variables. Receiver operating characteristic (ROC) curve analysis was conducted to evaluate the diagnostic performance of urinary nephrin for detecting diabetic nephropathy.

Ethical Consideration: Approval for the study was obtained from the Institutional Ethics Committee prior to commencement. Written informed consent was collected from all participants, and confidentiality of data was ensured throughout the study. All procedures complied with the ethical principles outlined in the Declaration of Helsinki.

RESULTS

Table 1. Baseline Demographic and Anthropometric Characteristics of Study Participants Across the Three Groups

Variable	Type2 Diabetes mellitus (n = 28)	Diabetic Nephropathy (n = 30)	Controls (n = 30)	p-value
Age (years)	53.00 ± 9.69	56.17 ± 12.74	56.75 ± 11.26	0.41
Weight (kg)	62.88 ± 13.45	61.21 ± 11.38	56.90 ± 16.81	0.26
Height (cm)	156.04 ± 8.53	158.58 ± 10.54	155.10 ± 10.03	0.57
BMI (kg/m²)	25.34 ± 5.26	24.45 ± 4.55	23.50 ± 5.96	0.40
Waist Circumference (cm)	38.61 ± 4.79	35.17 ± 4.13	33.85 ± 5.00	0.36

The baseline demographic and anthropometric characteristics of the study participants are presented in Table 1. The mean age of the participants was similar across the three groups, (p= 0.41). Similarly, anthropometric parameters such as weight and height did not differ significantly between groups, with mean weights of 62.88 ± 13.45 kg in the diabetes group, 61.21 ± 11.38 kg in the diabetic nephropathy group, and 56.90 ± 16.81 kg in controls (p = 0.26). The mean height also showed no variation across the groups (p = 0.57). Correspondingly, BMI among the diabetes (25.34 ± 5.26 kg/m²), diabetic nephropathy (24.45 ± 4.55 kg/m²), and control groups (23.50 ± 5.96 kg/m²; p = .40), were similar. Waist circumference, although slightly higher in the diabetes group (38.61 ± 4.79 cm) compared to the diabetic nephropathy (35.17 ± 4.13 cm) and control groups (33.85 ± 5.00 cm), did not reach statistical significance (p =0.36). Overall, there were no significant differences in baseline demographic or anthropometric characteristics across the study groups.

Table 2. Renal Function Markers (Serum Urea, Creatinine, eGFR) in Study Groups

Variable	Type 2 Diabetes mellitus (n = 28)	Diabetic Nephropathy (n = 30)	Controls (n = 30)	p-value
FBS	172.6±82.7	214±84.8	79.7±11.55	**0.001***
HbA1C	8.8±2.3	9±2.8	5.4±1	**0.001***
Serum Urea (mg/dL)	23.61 ± 7.99	44.58 ± 27.78	23.40 ± 4.80	**0.001***
Serum Creatinine (mg/dL)	1.05 ± 0.23	2.15 ± 1.01	0.92 ± 0.15	**0.001***
eGFR (mL/min/1.73m²)	71.46 ± 16.63	38.17 ± 17.58	79.15 ± 20.99	**0.001***

*-statistically significant

Table 2 presents a comparison of the renal function parameters across the three study groups.Serum urea levels were significantly higher among participants with diabetic nephropathy (44.58 ± 27.78 mg/dL) than among those with diabetes without nephropathy (23.61 ± 7.99 mg/dL) and healthy controls (23.40 ± 4.80 mg/dL) (p = 0.001). A similar pattern was observed for serum creatinine, which was markedly elevated in the diabetic nephropathy group (2.15 ± 1.01 mg/dL) relative to the diabetes group (1.05 ± 0.23 mg/dL) and controls (0.92 ± 0.15 mg/dL), p = 0.001. Correspondingly, the estimated glomerular filtration rate (eGFR) was significantly reduced in individuals with diabetic nephropathy (38.17 ± 17.58 mL/min/1.73 m²) compared to both diabetic participants without nephropathy (71.46 ± 16.63 mL/min/1.73 m²) and controls (79.15 ± 20.99 mL/min/1.73 m²), p = 0.001.

Table 3. Urinary biomarkers (Urine creatinine, UACR, urinary Nephrin, Nephrin–Creatinine Ratio) among the three Groups

Variable	Type 2 Diabetes mellitus (n = 28)	Diabetic Nephropathy (n = 30)	Controls (n = 30)	p-value
Urine Creatinine (mg/dL)	58.89 ± 13.37	27.08 ± 8.75	39.90 ± 14.33	0.11
UACR (mg/g)	126.25 ± 15.16	756.42 ± 60.95	8.29 ± 3.74	**0.001***
Urinary Nephrin (ng/mL)	22.56 ± 6.40	115.80 ± 12.10	5.30 ± 2.10	**0.01***
Nephrin–Creatinine Ratio	0.20 ± 0.12	0.90 ± 0.45	0.14 ± 0.06	**0.006***

*-statistically significant

There were significant differences in urinary biomarker levels across the three study groups. (Table 3). Urinary albumin-creatinine ratio (UACR) was markedly elevated in participants with diabetic nephropathy (756.42 ± 60.95 mg/g) compared to those with diabetes mellitus type 2, without nephropathy (126.25 ± 15.16 mg/g) and healthy controls (8.29 ± 3.74 mg/g), demonstrating a statistically significant difference ($p < .001$). Urinary Nephrin levels also showed a clear gradient, with highest concentrations observed in the diabetic nephropathy group (115.80 ± 12.10 ng/mL), followed by the diabetes group (22.56 ± 6.40 ng/mL) and controls (5.30 ± 2.10 ng/mL), $p = 0.01$. Similarly, the nephrin–creatinine ratio was substantially elevated in the diabetic nephropathy group (0.90 ± 0.45) compared to the diabetes (0.20 ± 0.12) and control groups (0.14 ± 0.06), $p = .006$.

Table 4. Correlation Between Urinary Nephrin Levels and Clinical, Biochemical, and Renal Parameters

	Variable	Pearson's r	p-value	Interpretation
Urinary NEPHRIN	Nephrin–Creatinine Ratio	0.92	0.01*	Very strong positive correlation
	UACR	0.66	0.001*	Strong positive correlation
	HbA1c	0.52	0.001*	Moderate positive correlation
	eGFR	–0.54	0.001*	Moderate negative correlation

*-statistically significant ($p<0.05$)

The correlation analysis examining the association between urinary nephrin levels and various clinical, biochemical, and renal parameters demonstrated several statistically meaningful relationships. Urinary nephrin showed a very strong positive correlation with the nephrin–creatinine ratio ($r = 0.92$, $p = .01$), indicating that higher nephrin excretion closely paralleled elevations in the ratio. A strong positive correlation was also observed with UACR ($r = 0.66$, $p = .001$), suggesting increasing nephrin levels with worsening albuminuria. Additionally, urinary nephrin demonstrated a moderate positive correlation with HbA1c ($r = 0.52$, $p = .001$), reflecting higher nephrin concentrations among individuals with poorer glycemic control. Conversely, a moderate negative correlation was identified between urinary nephrin and eGFR ($r = –0.54$, $p = .001$), consistent with declining kidney function in individuals with elevated nephrin levels. No significant correlations were noted with age, anthropometric parameters, blood pressure, fasting blood glucose, serum urea, serum creatinine, or other variables assessed. (Table 4)

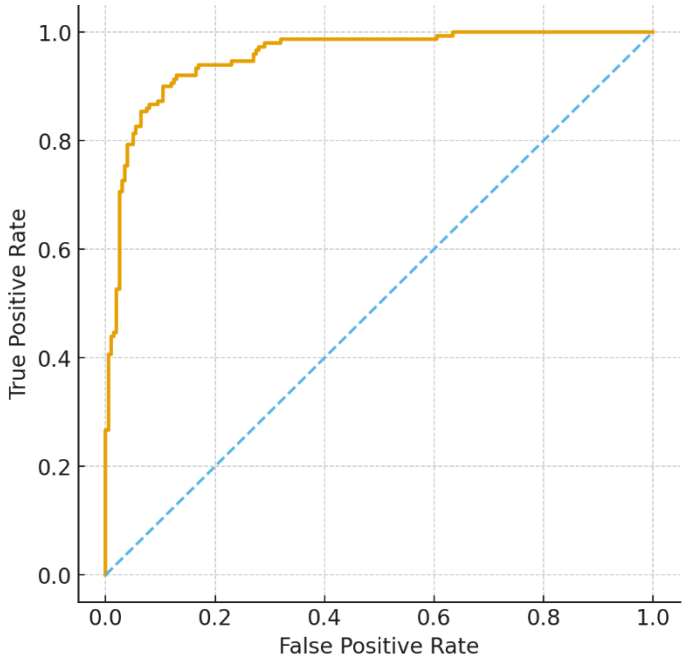
A strong positive correlation is observed with UACR ($r = 0.66$, $p = .001$), suggesting increasing nephrin levels with increasing albuminuria. Urinary nephrin levels also correlated with HbA1c values, correlation coefficient $r = 0.52$, $p = .001$. Nephrin and eGFR values were negatively correlated , $r = –0.54$ with $p = .001$. No significant correlations were noted with age, anthropometric parameters, blood pressure, fasting blood glucose, serum urea, serum creatinine, or other variables assessed.

Table 5. ROC Curve Characteristics of urinary Nephrin& Nephrin/Creatinine ratio for detecting Diabetic Nephropathy

Condition	AUC	Youden Index	Cut-off Value	Sensitivity (%)	Specificity (%)	Positive LR	Negative LR
Nephrin	**0.94**	**0.87**	**> 96.3**	**93.21**	**74.46**	**2.92**	**0.60**
Nephrin/Creatinine ratio	**0.452**	**0.33**	**> 6.8**	**33%**	**98%**	**25.6**	**0.67**

Receiver operating characteristic (ROC) analysis was performed to evaluate the diagnostic accuracy of urinary Nephrin for identifying patients with diabetic nephropathy. Urinary nephrin demonstrated excellent discriminative ability, with an area under the curve (AUC) of 0.94, indicating strong predictive performance. Based on the Youden Index (0.87), at a cut-off value of > 96.3 ng/mL, urinary nephrin achieved a sensitivity of 93.21% and a specificity of 74.46%, corresponding to a positive likelihood ratio of 2.92 and a negative likelihood ratio of 0.60. These findings suggest that urinary Nephrin could be a very effective biomarker for distinguishing patients with diabetic nephropathy from those without the condition. (Table 5) (Chart 1)

Chart 1. ROC curve of Urinary Nephrin for Detecting Diabetic Nephropathy



DISCUSSION

In the present study, urinary Nephrin levels were assessed in 88 indviduals with Diabetes mellitus type 2, Diabetic Nephropathy and healthy controls. In our study we found the Nephrin levels to be significantly elevated in patients with Diabetic Nephropathy compared to those with Diabetes Mellitus type 2 & healthy controls.

Nephrin is a 180 KD transmembrane protein expressed in the glomerular podocytes. Podocytes, endothelial cells & basement membrane together formthe glomerular filtration barrier[15,16]. These podocytes tend to get damaged in glomerular diseases like diabetic nephropathy, minimal change disease (MCD),membranous glomerulopathy, focal segmental glomerulosclerosis(FSGS) etc[17,18]. There is also histological evidence for down regulation of Nephrin expression in kidney biopsies & excess excretion in urine, correlating with albuminuria In Diabetic Nephropathy, occurrenceof Nephrinuria even in normo albuminuric individuals (before micro albuminuria), could be due to dysregulation of Nephrin residues in the glomerular podocytes. Many type 2 Diabetes mellitus patients with normoalbuminuria, also had coexistent nephrinuria, indicating that nephrinuria could be biomarker for early glomerular damage caused by podocyte injury[19]

Surya et al. demonstrated comparable demographic characteristics among normoalbuminuric, microalbuminuric, and macroalbuminuric diabetic individuals, indicating that nephrin elevation occurs independently of age or anthropometric status[1]. Nascimento et al. likewise reported minimal anthropometric variation across groups stratified by glucose tolerance, despite marked differences in podocyte-related markers [2] . Although Hliel et al. observed age and BMI differences between certain diabetic subgroups, nephrin levels in their study showed a stronger association with renal parameters[5] . Taken together, these findings support the view that podocyte injury markers such as nephrin primarily reflect renal pathology rather than baseline demographic variation.

Analysis of urinary biomarkers revealed marked elevations in UACR, urinary nephrin, and the nephrin–creatinine ratio among individuals with diabetic nephropathy, allowing clear discrimination from both diabetics without nephropathy and healthy controls. We have found significant increase in Nephrin excretion in urine among the Diabetic Nephropathy patients, compared to other groups.

Our results match with the results which other researchers have recorded in their independent studies. The research by Surya et al. revealed that urinary nephrin levels show a progressive increase from normoalbuminuria through to macroalbuminuria which indicates podocyte damage occurs before proteinuria becomes visible[1] . The research

conducted by Nascimento et al. demonstrated that nephrin mRNA levels rose with albuminuria progression and provided useful predictions of pathological albumin excretion[2] .Kondapi et al. similarly observed that rising urinary Nephrin levels were accompanied by increasing serum creatinine, suggesting a close relationship between podocyte injury and declining renal function[7] .

In our study, we have got significant negative correlation of urinary Nephrin levels with e-GFR.Urinary nephrin showed strong positive correlations with UACR and HbA1c, alongside a significant inverse association with eGFR. These relationships suggest that nephrin excretion reflects both renal structural injury and metabolic control. Similar results have been observed by Hliel et al & Kostovska et al , who also demonstrated an inverse relationship of urinary nephrin, with eGFR [5,8]. This further reinforces the concept that nephrin excretion increases as glomerular filtration capacity declines [8]. Other studies have also shown the correlation of urinary Nephrin with albuminuria (UACR), glycemic indices & eGFR [1,2] Rangaswamaiah et al. have reported elevated urinary nephrin levels even in diabetic individuals with normal UACR, supporting the utility of nephrin as an early indicator of renal injury [4] . From these findings we can infer that urinary Nephrin rises earlier and more compared to other conventional markers of renal damage.

Collectively, these observations indicate that podocyte injury, as reflected by nephrinuria, progresses along with renal impairment in diabetic nephropathy& it could have an important role as an indicator of diabetic kidney disease progression.

Receiver operating characteristic analysis in the present study demonstrated excellent diagnostic performance of urinary nephrin, with an AUC of 0.94 for identifying diabetic nephropathy. This level of discrimination is comparable to, and in some cases exceeds, that reported in earlier investigations. Kondapi et al. reported high sensitivity and specificity for urinary nephrin at a cut-off value closely matching that identified in the present study [7] . Kostovska et al. similarly observed a high predictive probability for nephrin in identifying diabetic nephropathy [8] . In addition, Hliel et al. reported strong diagnostic accuracy for nephrin in ROC analyses, further supporting its value as a diagnostic biomarker [5] . All these findings point to towards the robustness of urinary nephrin as a sensitive tool for detecting diabetic nephropathy.

LIMITATIONS

The sample size, particularly within the diabetic nephropathy group, was relatively modest, which may limit the generalizability of the results to broader populations. In addition, the cross-sectional design restricts interpretation of temporal or causal relationships and does not allow evaluation of the prognostic or predictive utility of urinary nephrin over time. Longitudinal follow-up would be necessary to determine whether nephrinuria precedes conventional markers and predicts progression of renal dysfunction.

CONCLUSION

Urinary nephrin levels were markedly higher in patients with diabetic nephropathy compared with diabetic individuals without nephropathy and healthy controls. It also displayed a high diagnostic accuracy for discriminating DN patients. From a clinical perspective, measurement of urinary nephrin may serve as a useful adjunct in the evaluation of diabetic patients, presenting with normoalbuminuria who may have underlying podocyte injury, which may otherwise go undetected. If along with UACR, urine Nephrin levels are also analyzed, it could be a very useful biomarker for predicting the future development of Diabetic Nephropathy. However, before routine clinical implementation can be recommended, larger and well-designed longitudinal studies are required to confirm its predictive value, determine optimal cut-off thresholds, and assess its performance across different populations. Such studies would help clarify whether incorporating urinary nephrin into routine assessment can meaningfully influence clinical decision-making and long-term renal outcomes.

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