



Research Article

Urinary NGAL and Conventional Renal Markers in Patients with Type 2 Diabetes Mellitus: A Comparative Cross-Sectional Study at a Nigerian Teaching Hospital

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ABSTRACT

Diabetic kidney disease (DKD) is a leading complication of type 2 diabetes mellitus (T2DM); conventional renal markers may not fully capture tubular injury. This study compared demographic, clinical, and conventional renal markers, and urinary neutrophil gelatinase-associated lipocalin (NGAL), between 88 T2DM patients and 44 non-diabetic controls at UITH, Ilorin, Nigeria; examined NGAL's associations with conventional renal markers and clinical characteristics among T2DM participants; and compared the discriminatory performance of NGAL and urinary albumin-to-creatinine ratio (UACR) for reduced eGFR ($<60 \text{ mL/min/1.73m}^2$). T2DM participants were significantly older than controls and had significantly higher blood pressure and fasting glucose (all $p < 0.001$), with similar hypertension prevalence (86.4% vs 72.7%, $p = 0.056$). Compared with controls, T2DM participants had significantly higher serum creatinine, urinary albumin, UACR and NGAL, and significantly lower urinary creatinine and eGFR (all $p \leq 0.001$); median NGAL was approximately 90% higher than in controls (22.85 vs 12.00 ng/mL). Among T2DM participants, NGAL correlated strongly with serum creatinine and UACR, and negatively with eGFR (all $p < 0.001$), and was associated with age and diabetes duration but not sex. Among the 35 of 88 T2DM participants (39.8%) with reduced eGFR, NGAL discriminated this status with AUC 0.852 (95% CI 0.774–0.931), lower than UACR (AUC 0.940, 95% CI 0.895–0.985). NGAL was significantly elevated in T2DM and correlated strongly with conventional renal markers, supporting a potential complementary, rather than an early-detection or predictive, role alongside serum creatinine, eGFR and UACR; the cross-sectional design cannot establish temporal precedence, and larger longitudinal studies are needed before clinical application.

Keywords: Diabetic Kidney Disease; Estimated Glomerular Filtration Rate; Neutrophil Gelatinase-Associated Lipocalin; Type 2 Diabetes Mellitus; Urinary Albumin-to-Creatinine Ratio

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INTRODUCTION

Diabetic kidney disease (DKD) is one of the most consequential microvascular complications of type 2

diabetes mellitus (T2DM), arising from a combination of chronic hyperglycaemia, altered renal haemodynamics, oxidative stress and structural

injury to the glomerulus, tubules and interstitium (Mora-Fernández *et al.*, 2021). Renal impairment in T2DM can, in some patients, precede detectable abnormalities in the conventional markers used to screen for it, so that meaningful tubular or glomerular injury may already be present before serum creatinine, estimated glomerular filtration rate (eGFR) or urinary albumin-to-creatinine ratio (UACR) become clearly abnormal (DeFronzo *et al.*, 2021; Vallon & Thomson, 2020); this study, being cross-sectional, cannot itself establish the temporal sequence of these changes, and instead compares NGAL with these conventional markers at a single time point. Current guidelines recommend the combined use of eGFR and UACR for renal assessment in diabetes (Kidney Disease: Improving Global Outcomes (KDIGO), 2024; American Diabetes Association Professional Practice Committee, 2026), and diabetes, the leading cause of this burden globally, now affects an estimated 589 million adults worldwide (International Diabetes Federation, 2025). However, both eGFR and UACR are indirect measures: eGFR is derived largely from serum creatinine, which is confounded by muscle mass, age and sex (Inker *et al.*, 2021), while UACR reflects glomerular permeability and does not directly capture tubular epithelial injury, a component of DKD that has been reported to occur alongside, and sometimes independently of, glomerular abnormalities in T2DM (Żyłka *et al.*, 2018). Diabetic kidney disease, reduced eGFR, albuminuria, and tubular injury are related but distinct constructs in this manuscript: DKD is used here as the general clinical label for diabetes-associated renal involvement, reduced eGFR and albuminuria are the specific, guideline-defined glomerular criteria used to assess it, and tubular injury refers specifically to epithelial-cell stress or damage in the renal tubules, which UACR does not measure directly and which NGAL is proposed to reflect.

Neutrophil gelatinase-associated lipocalin (NGAL) has emerged as a candidate biomarker of tubular injury that could complement these conventional measures (Devarajan, 2021). NGAL is a stress-responsive lipocalin expressed by tubular epithelium and neutrophils that rises with tubular injury, and has shown pooled diagnostic sensitivity and specificity in the region of 80% across observational studies of diabetic kidney disease (He *et al.*, 2020). Individual studies in T2DM populations have reported that NGAL is elevated even among patients with normoalbuminuria, and correlates with UACR and eGFR to varying degrees depending on the population

and disease stage studied (Quang *et al.*, 2021; de Carvalho *et al.*, 2016); because NGAL can also be elevated by urinary tract infection, systemic inflammation, cardiovascular disease and certain medications, these are potential confounders of any between-group NGAL comparison and are considered further in the Discussion and Limitations.

Despite this evidence, comparative data establishing whether NGAL differs meaningfully from serum creatinine, eGFR and UACR between patients with T2DM and non-diabetic controls, evaluated concurrently within a single population, remain limited, and are particularly scarce for Nigerian patients with T2DM. This study therefore compared demographic and clinical characteristics, and conventional renal markers and urinary NGAL, between patients with T2DM and non-diabetic controls at a Nigerian teaching hospital; examined the associations of NGAL with conventional renal markers and selected clinical characteristics among the T2DM participants; and compared the discriminatory performance of NGAL and UACR for identifying reduced eGFR ($<60 \text{ mL/min/1.73 m}^2$) in this population. We use 'associated with' rather than 'predicts' or 'detects early' throughout, since the cross-sectional design cannot establish temporal precedence or predictive validity.

MATERIALS AND METHODS

Study Design and Area

This is a hospital-based, comparative cross-sectional study conducted at the University of Ilorin Teaching Hospital (UIITH), Ilorin, Kwara State, Nigeria. Participants were recruited into two groups: adults with a confirmed diagnosis of T2DM attending the hospital's diabetic outpatient and related clinical services, and apparently healthy, non-diabetic adults recruited from the hospital community (staff, visitors, and other non-patient volunteers), drawn from the same hospital catchment population as the non-diabetic group/Control. Participants with T2DM were not pre-classified into DKD and non-DKD categories; evidence of DKD was instead assessed within the T2DM group using the study's predefined renal criteria (serum creatinine, eGFR and UACR). In this manuscript, 'early' tubular injury is used descriptively, to refer to NGAL elevation observed in T2DM participants irrespective of their conventional renal marker status, rather than to a specific, pre-defined disease stage; this cohort was not stratified into normoalbuminuric, albuminuric, preserved-eGFR and reduced-eGFR subgroups, so the study cannot demonstrate that NGAL elevation precedes

conventional-marker abnormality in any individual participant.

Study Participants

A total of 132 participants (88 with T2DM and 44 non-diabetic controls) were recruited using non-probability purposive sampling and their samples analyzed; this approach allowed recruitment of sufficient confirmed T2DM cases and eligible controls within the study period from the clinics available, though it does not yield a probability sample of the wider clinic population. Sample size was calculated from an assumed 35% DKD prevalence among T2DM patients, using the single-proportion formula $n = Z^2 p(1-p)/d^2$ with 95% confidence ($Z=1.96$) and a 10% margin of error ($d=0.10$), giving a minimum T2DM sample of approximately 88; a 2:1 T2DM-to-control ratio was adopted a priori to preserve adequate control numbers while prioritising precision in the T2DM group, giving 44 controls. This calculation was powered for estimating DKD prevalence within the T2DM group and does not itself guarantee adequate power for the correlation and ROC analyses reported here. Eligible participants were adults aged 18 years and above; individuals with type 1, gestational or secondary diabetes, pre-existing non-diabetic chronic kidney disease, acute kidney injury or acute illness at recruitment, chronic liver disease, active systemic infection, or pregnancy were excluded.

Ethical Considerations

Ethical approval was sought from Kwara State Ministry of Health Ethical Review Committee, Ilorin (UITH/CAT/189/218/791) and the study conducted was guided by the Nigerian National Code of Health Research Ethics and the Helsinki Declaration. Written informed consent was obtained from all participants prior to participation in the study.

Sample Collection and Laboratory Analysis

Venous blood and urine were collected from each participant after an overnight fast of at least 8 hours. Fasting blood glucose was measured by the glucose oxidase-peroxidase method, serum creatinine by the modified Jaffe reaction, and urinary albumin by the bromocresol purple dye-binding method, all on an automated chemistry analyser using reagents supplied by Agappe Diagnostics (Kerala, India). UACR was derived from urinary albumin and urinary creatinine, and eGFR was calculated from serum creatinine using the 2021 CKD-EPI creatinine equation without a race coefficient (Inker *et al.*, 2021). Urinary NGAL was measured by commercial sandwich enzyme-linked immunosorbent assay (Eldere ELISA kit, Shanghai Ideal Medical Technology Co., Ltd., Shanghai, China), with absorbance read at

450 nm and concentration derived from the standard curve; NGAL was reported in ng/mL from a single spot urine sample and was not normalized to urinary creatinine, so the values reported here are vulnerable to variation in urine concentration and may not be directly comparable to studies reporting a urinary NGAL-to-creatinine ratio. Duration of diabetes was grouped into three categories (<2 years, 2-5 years, and >5 years) for analysis, based on participant-reported history.

Data Analysis

Data were analysed in IBM SPSS Statistics version 27.0. Categorical variables were compared using the chi-square test. Associations between NGAL, other renal/metabolic parameters, and demographic variables were assessed using Spearman's correlation, the Mann-Whitney U test, or the Kruskal-Wallis H test, as appropriate. The discriminatory performance of NGAL and UACR for reduced eGFR (<60 mL/min/1.73 m²) was assessed by ROC curve analysis, reporting AUC, 95% CI, sensitivity, and specificity at the Youden-optimal cut-off. No multivariable adjustment for confounders was performed. Statistical significance was set at $p<0.05$, two-tailed.

RESULTS

A total of 132 participants were studied, 88 with T2DM and 44 non-diabetic controls (Table 1). T2DM participants were significantly older than controls ($p<0.001$), with a markedly higher proportion aged 65-80 years. Sex distribution did not differ significantly between groups ($p=0.901$). T2DM participants had significantly higher systolic blood pressure (130.0 vs 120.0 mmHg), diastolic blood pressure (83.5 vs 80.0 mmHg) and fasting blood glucose (6.75 vs 3.70 mmol/L) than controls (all $p<0.001$). Among T2DM participants, median diabetes duration was 3.0 years, with 54.5% having had diabetes for 2-5 years and 22.7% for more than 5 years; 76 of 88 T2DM participants (86.4%) were hypertensive, a proportion not significantly different from that among controls (72.7%, $p=0.056$).

Compared with non-diabetic controls, T2DM participants had significantly higher serum creatinine, urinary albumin and UACR, and significantly lower urinary creatinine and eGFR (Table 2, all $p\leq0.001$). Urinary NGAL, the tubular-injury biomarker under study, was also significantly elevated in T2DM: median urinary NGAL was approximately 90% higher than in controls (22.85 vs 12.00 ng/mL, $p<0.001$); NGAL was measured as a single, non-creatinine-normalized urinary concentration.

Table 1: Demographic and Clinical Characteristics of Study Participants

Parameter	Non-DM (n=44)	T2DM (n=88)	Test statistic	p
Age (years)	46.0 (14.0)	56.0 (18.0)	U=1110.00	<0.001
Sex, male, n (%)	19 (43.2)	37 (42.0)	$\chi^2=0.016$	0.901
SBP (mmHg)	120.0 (4.0)	130.0 (20.0)	U=804.50	<0.001
DBP (mmHg)	80.0 (4.0)	83.5 (10.0)	U=1076.00	<0.001
FBG (mmol/L)	3.70 (1.10)	6.75 (3.60)	U=176.50	<0.001
Hypertensive, n (%)	32 (72.7)	76 (86.4)	$\chi^2=3.667$	0.056
Duration of diabetes (years)	—	3.0 (3.0)	—	—

SBP = systolic blood pressure; DBP = diastolic blood pressure; FBG = fasting blood glucose; T2DM = type 2 diabetes mellitus; Non-DM = non-diabetic control; U = Mann-Whitney U statistic; χ^2 = Pearson chi-square statistic. $p<0.05$ was considered statistically significant.

Table 2: Comparison of Conventional and Novel Renal Markers between Non-DM Controls and T2DM Participants

Renal parameter	Non-DM (n=44)	T2DM (n=88)	Test statistic	P
Serum creatinine (mg/dL)	0.80 (0.70–0.90)	0.94 (0.70–1.39)	U=1266.0	0.001
Urinary creatinine (mg/dL)	24.50 (22.00–26.90)	22.75 (19.85–26.25)	U=1278.5	0.001
Urinary albumin (mg/dL)	1.15 $\pm$ 0.49	1.84 $\pm$ 0.67	t=-6.696	<0.001
UACR (mg/g)	42.58 (30.00–60.65)	79.77 (52.69–114.44)	U=821.5	<0.001
NGAL (ng/mL)	12.00 (8.00–17.80)	22.85 (14.00–30.00)	U=700.5	<0.001
eGFR (mL/min/1.73 m ²)	106.21 (94.70–116.25)	75.52 (51.75–111.62)	U=1074.5	<0.001

NGAL = neutrophil gelatinase-associated lipocalin; eGFR = estimated glomerular filtration rate. $p<0.05$ was considered statistically significant.

Among the 88 T2DM participants, NGAL correlated positively with serum creatinine ($p=0.678$), UACR ($p=0.673$) and urinary albumin ($p=0.666$), negatively with eGFR ($p=-0.668$) and urinary creatinine ($p=-0.545$), and moderately with fasting blood glucose (FBG) ($p=0.353$) (Table 3). All of these correlations were significant at $p<0.001$, except the FBG correlation, which was significant at $p=0.001$.

Among the 88 T2DM participants, 35 (39.8%) had eGFR <60 mL/min/1.73 m². Both NGAL and UACR discriminated significantly between participants with and without reduced eGFR (Table 5, Figure 1). UACR showed the higher discriminatory performance (AUC=0.940, 95% CI 0.895–0.985) compared with NGAL (AUC=0.852, 95% CI 0.774–0.931), both $p<0.001$. At the study-derived optimal cut-off NGAL (≥ 26.15 ng/mL) achieved a sensitivity of 74.3% and specificity of 84.9% (Youden index 0.592), while UACR (≥ 98.58 mg/g) achieved the same sensitivity (74.3%)

with higher specificity (96.2%) and the higher Youden index of the two markers (0.705).

Among T2DM participants, age was significantly associated with every renal and metabolic parameter assessed, with the strongest association seen for eGFR ($p=-0.448$, $p<0.001$), and a weaker but significant association for NGAL ($p=0.228$, $p=0.033$) (Table 4). Sex was not significantly associated with NGAL or any conventional renal or metabolic marker (all $p>0.05$), with the single exception of eGFR ($p=0.005$). By contrast, duration of diabetes was significantly associated with every parameter assessed (all $p\leq 0.004$): NGAL ($H=21.864$) differed significantly across all three duration categories on Bonferroni-corrected post-hoc comparison (<2 years vs $2-5$ years, <2 years vs >5 years, and $2-5$ years vs >5 years all significant), whereas serum creatinine and UACR differed most clearly between the >5 -year group and the shorter-duration groups.

Table 3: Correlations of NGAL with Renal and Metabolic Parameters among T2DM Participants (n = 88)

Relationship	P	P
NGAL vs serum creatinine	0.678	<0.001
NGAL vs UACR	0.673	<0.001
NGAL vs urinary albumin	0.666	<0.001
NGAL vs Egfr	-0.668	<0.001
NGAL vs urinary creatinine	-0.545	<0.001
NGAL vs FBG	0.353	0.001

ρ = Spearman's rank correlation coefficient. NGAL = neutrophil gelatinase-associated lipocalin; UACR = urinary albumin-to-creatinine ratio; eGFR = estimated glomerular filtration rate; FBG = fasting blood glucose. $p < 0.05$ was considered statistically significant.

Table 5: Diagnostic/Discriminatory Performance of NGAL and UACR for Identifying Reduced eGFR (<60 mL/min/1.73 m²) Among T2DM Participants

Marker	AUC (95% CI)	p	Cut-off	Sens. (%)	Spec. (%)	Youden
NGAL (ng/mL)	0.852 (0.774–0.931)	<0.001	≥ 26.15	74.3	84.9	0.592
UACR (mg/g)	0.940 (0.895–0.985)	<0.001	≥ 98.58	74.3	96.2	0.705

AUC = area under the receiver operating characteristic curve; CI = confidence interval; Sens. = sensitivity; Spec. = specificity; Youden = Youden index (sensitivity + specificity – 1) at the study-derived optimal cut-off. NGAL = neutrophil gelatinase-associated lipocalin; UACR = urinary albumin-to-creatinine ratio. These cut-offs are sample-derived and should not be treated as validated diagnostic thresholds.

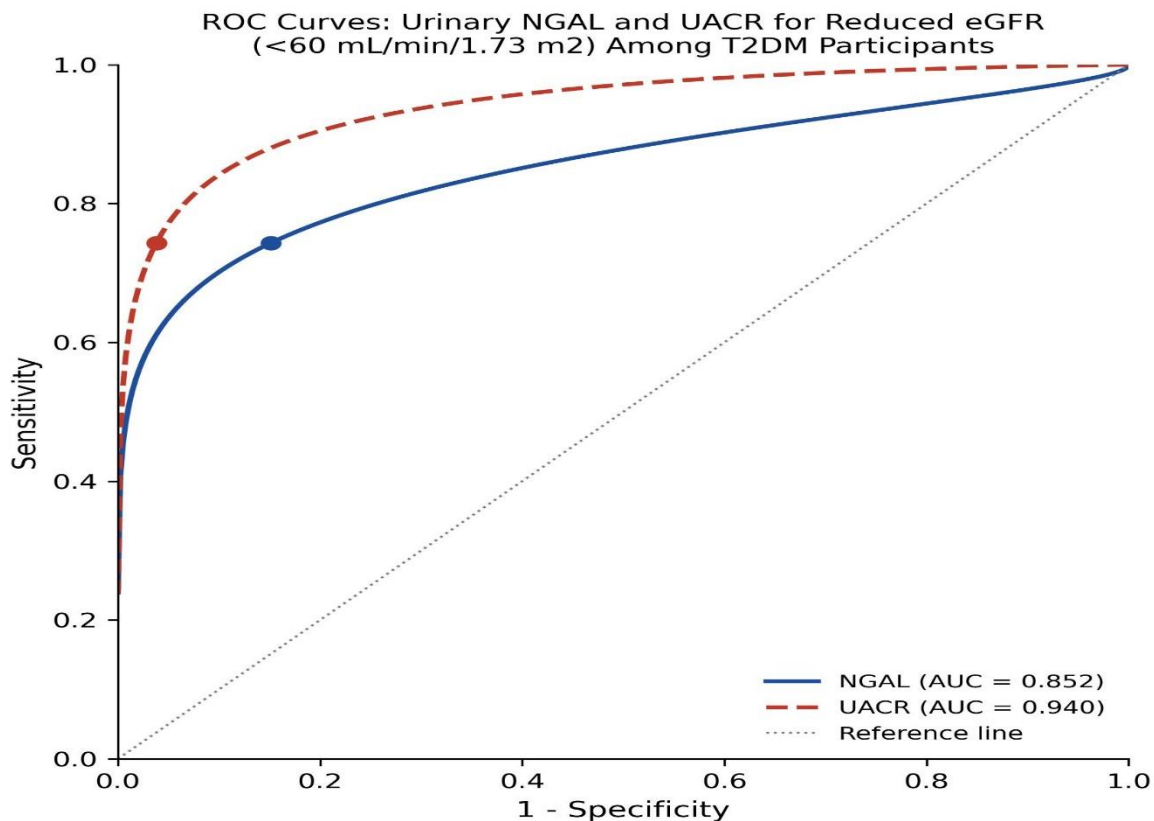


Figure 1: Receiver Operating Characteristic (ROC) Curves for Urinary NGAL and UACR in Identifying Reduced eGFR (<60 mL/min/1.73 m²) Among T2DM Participants

Curves are smooth approximations constructed from the verified AUC, sensitivity, and specificity values in Table 5, rather than plotted from the full set of empirical per-threshold coordinates. NGAL = neutrophil gelatinase-associated lipocalin; UACR = urinary albumin-to-creatinine ratio; eGFR = estimated glomerular filtration rate; T2DM = type 2 diabetes mellitus.

Table 4: Association of Renal and Metabolic Parameters with Age, Sex, and Duration of Diabetes among T2DM Participants (n = 88)

Parameter	Age, ρ (p)	Sex, U (p)	Duration, H (p)
Serum creatinine (mg/dL)	0.220 (0.039)	896.0 (0.688)	20.373 (<0.001)
Urinary creatinine (mg/dL)	-0.254 (0.017)	813.5 (0.272)	17.397 (<0.001)
Urinary albumin (mg/dL)	0.329 (0.002)	757.0 (0.115)	22.175 (<0.001)
UACR (mg/g)	0.351 (<0.001)	734.0 (0.077)	22.926 (<0.001)
NGAL (ng/mL)	0.228 (0.033)	836.0 (0.363)	21.864 (<0.001)
eGFR (mL/min/1.73 m ²)	-0.448 (<0.001)	609.0 (0.005)	18.990 (<0.001)
FBG (mmol/L)	0.298 (0.005)	836.0 (0.363)	11.161 (0.004)

ρ = Spearman's rank correlation coefficient, UACR = urinary albumin-to-creatinine ratio; NGAL = neutrophil gelatinase-associated lipocalin; eGFR = estimated glomerular filtration rate; FBG = fasting blood glucose. $p < 0.05$ was considered statistically significant.

DISCUSSION

This study found that patients with T2DM had significantly poorer conventional renal indices than non-diabetic controls, and, concurrently, significantly higher urinary NGAL. NGAL is not itself a direct histological measure of tubular injury; it is interpreted here as a marker of tubular stress, whereas UACR and eGFR reflect glomerular filtration and permeability. This pattern of concurrently abnormal glomerular/filtration and tubular-stress markers is consistent with the current understanding that diabetic renal injury involves interacting glomerular, tubular and interstitial processes rather than a single mechanism confined to the glomerulus (Mora-Fernández *et al.*, 2021; Vallon & Thomson, 2020); it does not, on its own, establish that NGAL detects renal injury before conventional markers, predicts future renal decline, or improves diagnostic classification, since the T2DM participants already had significantly different serum creatinine, UACR and eGFR values compared with controls at the same cross-sectional time point.

The strong correlations of NGAL with serum creatinine, UACR and eGFR among the T2DM participants reinforce the biological plausibility of this marker as an indicator of renal involvement, and are consistent with earlier reports of positive correlation between urinary NGAL and UACR in T2DM populations (Quang *et al.*, 2021; de Carvalho *et al.*, 2016). NGAL also correlated with fasting blood glucose, although this correlation was more modest than its correlations with the conventional renal indices, indicating that glycaemia alone does not account for the observed elevation. However, because NGAL, UACR, serum creatinine and eGFR are all correlated with one another and with overall renal function, the strong pairwise correlations observed here may partly reflect shared, overall renal dysfunction rather than an independent tubular-

specific signal; this study cannot distinguish these two possibilities, and both are compatible with the reported correlations.

The elevation of NGAL observed in this cohort, together with the concurrent decline in conventional renal indices, mirrors previous reports describing simultaneous tubular and glomerular abnormality in early diabetic kidney disease. Żyłka *et al.* (2018) similarly found tubular injury markers, including NGAL, to be elevated alongside indices of glomerular function in patients with type 2 diabetes, supporting the view that tubular injury in T2DM is an early, parallel process rather than one that only follows established albuminuria. A systematic review and meta-analysis of urinary NGAL in diabetic kidney disease has likewise reported consistent elevation of this biomarker across diverse study populations, in keeping with the substantially higher NGAL concentrations observed here in T2DM participants relative to non-diabetic controls (He *et al.*, 2020). The direction and strength of NGAL's correlation with UACR and eGFR observed in this study are also broadly consistent with earlier work in T2DM populations, although reported correlation coefficients have varied across studies, likely reflecting differences in assay platform, sample size, urine normalization practice and disease stage (Quang *et al.*, 2021; de Carvalho *et al.*, 2016). On ROC analysis for identifying reduced eGFR, UACR outperformed NGAL (AUC 0.940 versus 0.852; Figure 1). This is unsurprising given that UACR and eGFR are both established, guideline-recommended components of renal assessment in diabetes that are, to some extent, physiologically linked (KDIGO, 2024; American Diabetes Association Professional Practice Committee, 2026); a marker of albuminuria would be expected to track a filtration-based reference standard closely. The AUC obtained for NGAL here (0.852) falls within the range reported by prior

systematic review of urinary NGAL for diabetic kidney disease (He et al., 2020), lending some external support to the discriminatory performance observed, although the present AUCs should be interpreted as specific to this study population and cross-sectional reference standard (eGFR <60 mL/min/1.73 m²) rather than as universally applicable estimates; the cut-off derived here by maximum Youden index (NGAL ≥26.15 ng/mL) is sample-derived and should not be treated as a validated diagnostic threshold.

Age and duration of diabetes, but not sex, were significantly associated with NGAL as well as with the conventional renal markers. The association with age is consistent with the known decline in renal functional reserve with advancing age, compounded in diabetes by cumulative metabolic and haemodynamic stress (Mora-Fernández et al., 2021). However, because T2DM participants were also significantly older than controls, age is a plausible residual confounder of the between-group NGAL comparison itself, and no adjustment for age was performed in this analysis. The association with duration of diabetes, in which NGAL differed significantly across all three duration categories rather than only between the longest- and shortest-duration groups (as observed for serum creatinine and UACR), may indicate that this tubular-stress marker tracks a more continuous, cumulative injury process across the course of the disease, though this cross-sectional design cannot establish this as a causal or temporal relationship. The single significant sex association, for eGFR only, without a corresponding difference in serum creatinine, UACR or NGAL, is difficult to interpret biologically from these data and may reflect the demographic composition of this sample rather than a genuine sex-specific renal effect.

Taken together, these findings support an association, and potential complementary value, for NGAL alongside conventional renal markers in T2DM: this biomarker was significantly elevated relative to non-diabetic controls, correlated strongly with established indices of glomerular and tubular-stress-adjacent function, showed moderate discriminatory performance for reduced eGFR, and tracked age and duration of diabetes independently of sex. These findings should not be read as demonstrating that NGAL detects renal injury earlier than conventional markers, predicts subsequent renal decline, or improves diagnostic classification beyond UACR and eGFR, since UACR outperformed NGAL on ROC analysis and no longitudinal outcome data were collected. Rather than replacing serum creatinine,

eGFR and UACR, urinary NGAL may serve as a potential complementary indicator alongside conventional markers, providing additional information on renal involvement in T2DM that these markers do not fully capture alone, pending confounder-adjusted, longitudinal confirmation.

CONCLUSION

In this comparative cross-sectional study at the University of Ilorin Teaching Hospital (UIH), patients with T2DM had significantly poorer conventional renal indices and significantly higher urinary NGAL than non-diabetic controls. Among T2DM participants, NGAL was significantly associated with conventional renal markers, age and duration of diabetes, but not sex, and showed moderate discriminatory performance for reduced eGFR (AUC 0.852), lower than UACR (AUC 0.940). Urinary NGAL was elevated and associated with conventional renal indices in T2DM; these findings should not be read as establishing early detection, predictive validity, or clinical utility for NGAL, given the cross-sectional, unadjusted design and the absence of longitudinal outcome data. These findings support a potential complementary role for NGAL alongside UACR and eGFR in the renal assessment of patients with T2DM, pending larger, confounder-adjusted, longitudinal studies to determine whether this biomarker adds predictive information for subsequent renal decline.

Author Contributions: Ibrahim Olayinka Musa conceived the study and designed the methodology. Mathew Folaranmi Olaniyan served as the main supervisor of the study, with additional supervision provided by Wasiu Olanrewaju Garuba. Suleiman Ibrahim Eleha and Imran Oluwatobi Sarumi performed the statistical analysis. Ogunwale Kolawole Ayobami Tajudeen and Oluwatoni Olabimpe Olawumi carried out the biochemical analyses. Seidat Omolabake Hassan did manuscript writing and review. All authors read and approved the final manuscript.

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Data Availability: The dataset analysed during the current study is available from the corresponding author on reasonable request.

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