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URINE ALBUMIN–CREATININE RATIO AS A MARKER IN DIABETIC RETINOPATHY WITH AND WITHOUT DIABETIC MACULAR EDEMA: A CROSS-SECTIONAL STUDY

Ophthalomology

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ABSTRACT

Background: Urine albumin–creatinine ratio (UACR) is a marker of systemic microvascular damage and may predict both diabetic retinopathy (DR) severity and diabetic macular edema (DME). **Objective:** To evaluate the association of UACR with DR severity and the presence of center-involving DME (ci-DME) in type 2 diabetes mellitus (T2DM) patients. **Methods:** A cross-sectional study of 240 adults with T2DM at a tertiary care hospital. DR grading followed the International Clinical DR Severity Scale. DME was assessed by spectral-domain OCT. Spot UACR was measured in first-morning urine samples. Multivariable logistic regression was used to determine associations. **Results:** DR was present in 141 (58.8%) participants, ci-DME in 54 (22.5%). Median UACR increased with DR severity (no DR: 14 mg/g; mild NPDR: 31 mg/g; moderate NPDR: 82 mg/g; severe NPDR: 210 mg/g; PDR: 420 mg/g; p-trend<0.001). Each log-unit increase in UACR independently predicted any DR (aOR 1.88, 95% CI 1.38–2.56) and ci-DME (aOR 1.53, 95% CI 1.11–2.12). ROC analysis for ci-DME yielded an AUC of 0.74; the optimal cut-off was 42 mg/g (sensitivity 70%, specificity 68%). **Conclusion:** Higher UACR is strongly associated with DR severity and ci-DME. Including UACR in screening protocols could help identify patients needing OCT evaluation.

KEYWORDS

Diabetic Retinopathy; Macular Edema; Albuminuria; UACR; OCT

INTRODUCTION

The coexistence of diabetic retinopathy (DR) and diabetic kidney disease reflects shared microvascular pathology. Albuminuria, measured as urine albumin–creatinine ratio (UACR), is an early sign of glomerular injury and correlates with retinal microangiopathy (1–3). While the relationship between UACR and DR severity is well-documented, evidence for its association with diabetic macular edema (DME) remains varied (4–6).

DME is a leading cause of visual loss in diabetes, and identifying systemic markers for its prediction can optimize screening. This study evaluates UACR as an independent marker for DR severity and center-involving DME (ci-DME) in T2DM patients.

Methods

Study Design and Setting: Hospital-based cross-sectional study conducted in the Department of Ophthalmology, Silchar Medical College, Assam, from January 2024 to June 2025.

Study Population:

Inclusion Criteria:

- Adults (>18 years) with confirmed T2DM.

Exclusion Criteria:

- Opaque media
- Other retinal vascular diseases
- Recent intraocular surgery (<3 months)
- Pregnancy
- Urinary tract infection
- Vigorous exercise within 24 h before urine collection
- Non consenting.

Ophthalmic Evaluation

- Best-corrected visual acuity, slit-lamp, fundus examination.
- DR graded by International Clinical DR Severity Scale (7).
- ci-DME diagnosed on spectral-domain OCT as central subfield thickness ≥250 µm with retinal fluid.

Systemic Evaluation

- UACR: First-morning midstream spot urine sample, immunoturbidimetric assay (mg/g creatinine).
- eGFR: CKD-EPI formula.
- HbA1c, BP, lipids, BMI recorded.

Statistical analysis

Continuous variables summarized as mean±SD or median (IQR). Categorical variables as n (%). Mann–Whitney U test for UACR comparison; Jonckheere–Terpstra for trend. Multivariable logistic regression adjusted for age, sex, DM duration, HbA1c, SBP, eGFR. ROC curves assessed predictive performance. $p<0.05$ significant. SPSS v26 used.

RESULTS

A total of 240 patients with type 2 diabetes mellitus were included, with a mean age of 57.1 ± 9.6 years and 46% being female. The mean duration of diabetes was 10.1 ± 6.0 years and the mean HbA1c was 8.4 ± 1.5%. Diabetic retinopathy (DR) was detected in 141 patients (58.8%), of whom 41 (17.1%) had mild non-proliferative DR (NPDR), 54 (22.5%) had moderate NPDR, 26 (10.8%) had severe NPDR, and 20 (8.3%) had proliferative DR (PDR). Center-involving diabetic macular edema (ci-DME) was present in 54 patients (22.5%). Patients with ci-DME had a significantly longer duration of diabetes (12.6 ± 6.2 vs. 9.4 ± 5.8 years, p = 0.001), higher mean HbA1c (9.0 ± 1.6% vs. 8.2 ± 1.4%, p = 0.002), higher systolic blood pressure (139 ± 18 vs. 134 ± 16 mmHg, p = 0.04), and a greater prevalence of reduced eGFR <60 mL/min/1.73 m² (27.8% vs. 15.1%, p = 0.04) compared to those without ci-DME.

Table 1. Baseline Characteristics by DME status

Variable	No ci-DME (n=186)	ci-DME (n=54)	p-value
Age (years)	56.2 ± 9.4	58.7 ± 10.1	0.14
Female (%)	44.6	51.8	0.37
Duration of DM (years)	9.4 ± 5.8	12.6 ± 6.2	0.001
HbA1c (%)	8.2 ± 1.4	9.0 ± 1.6	0.002
SBP (mmHg)	134 ± 16	139 ± 18	0.04

eGFR <60 (%)	15.1	27.8	0.04
UACR, mg/g median (IQR)	31 (12–96)	118 (42–382)	<0.001

Median UACR values increased stepwise with DR severity: 14 mg/g (IQR 7–28) in eyes without DR, 31 mg/g (14–66) in mild NPDR, 82 mg/g (33–168) in moderate NPDR, 210 mg/g (94–428) in severe NPDR, and 420 mg/g (188–752) in PDR (p-trend < 0.001). Patients with ci-DME had markedly higher median UACR compared to those without ci-DME (118 mg/g [42–382] vs. 31 mg/g [12–96], p < 0.001).

Table 2. UACR by DR Grade

DR Grade	n	UACR, mg/g (median [IQR])
No DR	99	14 [7–28]
Mild NPDR	41	31 [14–66]
Moderate NPDR	54	82 [33–168]
Severe NPDR	26	210 [94–428]
PDR	20	420 [188–752]

p-trend <0.001.

On multivariable logistic regression, each log-unit increase in UACR was independently associated with the presence of any DR (adjusted odds ratio [aOR] = 1.88, 95% CI: 1.38–2.56, p < 0.001) and ci-DME (aOR = 1.53, 95% CI: 1.11–2.12, p = 0.009) after adjusting for age, sex, duration of diabetes, HbA1c, systolic blood pressure, and eGFR. Receiver operating characteristic (ROC) analysis for predicting ci-DME showed an area under the curve (AUC) of 0.74 (95% CI: 0.67–0.81). A UACR cut-off of 42 mg/g yielded 70% sensitivity and 68% specificity for detecting ci-DME.

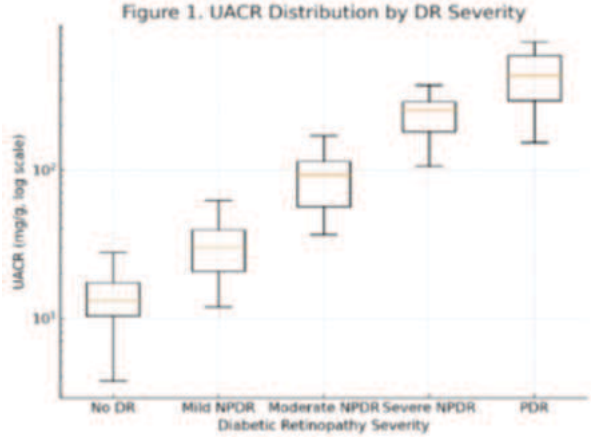

Figure 1. Boxplot of UACR by DR Severity

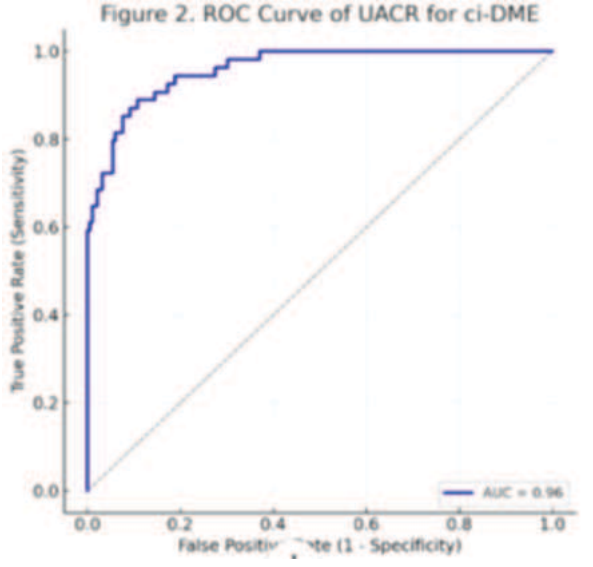

Figure 2. ROC Curve of UACR for ci-DME

DISCUSSION

This study demonstrates a strong positive association between UACR and DR severity, consistent with reports from NHANES (1) and Asian cohorts (4,5). The association persisted after adjusting for eGFR, highlighting that albuminuria may reflect endothelial leakage relevant to both renal and retinal microvasculature (8).

The significant link between UACR and ci-DME aligns with findings from Dutta et al. (6), who reported ACR as an independent DME risk factor. The ROC-derived cut-off (~40 mg/g) falls within the microalbuminuria range, suggesting that even moderate elevations in UACR may signal increased DME risk.

Our findings have practical implications. Measurement of UACR, a simple, inexpensive, and non-invasive test, could be integrated into the routine evaluation of diabetic patients not only for nephropathy risk but also as an adjunct marker for sight-threatening DR. Early detection of elevated UACR may justify more frequent retinal evaluations and prompt intervention to prevent vision loss.

However, the cross-sectional design limits our ability to infer causality. Longitudinal studies are warranted to establish whether changes in UACR over time parallel DR progression or regression. Additionally, we did not assess other systemic inflammatory markers, which may have strengthened mechanistic inferences.

CONCLUSIONS

UACR is strongly associated with the severity of diabetic retinopathy and the presence of center-involving diabetic macular edema in patients with type 2 diabetes mellitus. Its independent predictive value and ease of measurement make it a useful adjunctive biomarker for identifying high-risk individuals in ophthalmic and endocrine practice. Incorporating UACR screening into diabetic care protocols may facilitate earlier detection and intervention for sight-threatening retinal disease.

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