

Association between Microalbuminuria and Diabetic Retinopathy in Patients with Type 2 Diabetes Mellitus: A Hospital-Based Cross-Sectional Study

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ABSTRACT

Background: Diabetic retinopathy and diabetic nephropathy share a common microvascular pathogenesis, and microalbuminuria is regarded as an early marker of generalised endothelial injury. This study determined the prevalence of microalbuminuria in type 2 diabetes mellitus and examined its association with the presence and severity of diabetic retinopathy.

Methods: A hospital-based cross-sectional analytical study was conducted on 100 patients with type 2 diabetes mellitus of at least one year duration, aged 30 to 70 years. Patients with macroalbuminuria, non-diabetic renal disease or media opacity were excluded. The urinary albumin-to-creatinine ratio was estimated on early morning samples by immunoturbidimetry and confirmed on repeat testing. Retinopathy was graded on dilated fundus examination and photography using the Early Treatment Diabetic Retinopathy Study classification by an ophthalmologist masked to the urinary results.

Results: Microalbuminuria was present in 38 (38.0%) patients and retinopathy in 34 (34.0%). Retinopathy was significantly more frequent in microalbuminuric than normoalbuminuric patients (57.9% versus 19.4%; $p < 0.001$; odds ratio 5.73, 95% confidence interval 2.33 to 14.11), and sight-threatening disease was largely confined to the microalbuminuric group (18.4% versus 3.2%; $p = 0.010$). The albumin-to-creatinine ratio correlated with retinopathy severity ($\rho = 0.512$, $p < 0.001$). On multivariable analysis, microalbuminuria (adjusted odds ratio 3.86, $p = 0.008$), diabetes duration of ten years or more (3.12, $p = 0.022$) and glycated haemoglobin (1.62 per 1%, $p = 0.010$) were independent predictors.

Conclusion: Microalbuminuria was independently associated with the presence and severity of diabetic retinopathy. Its detection should prompt prioritised retinal screening, although the observed sensitivity was insufficient for it to replace fundus examination.

Keywords: Type 2 diabetes mellitus; Microalbuminuria; Diabetic retinopathy; Albumin-to-creatinine ratio; Microvascular complications

INTRODUCTION

Diabetes mellitus has become one of the defining public health challenges of the present century. The International Diabetes Federation estimated that 536.6 million adults between 20 and 79 years of age were

living with diabetes in 2021, and projected that this number would rise to 783.2 million by 2045, with the greatest proportional increase occurring in low-income and middle-income countries. [1] India carries a disproportionate share of this burden. The

Indian Council of Medical Research India Diabetes study, a nationally representative cross-sectional survey covering all states and union territories, reported a weighted diabetes prevalence of 11.4% and a prediabetes prevalence of 15.3% among adults aged 20 years and above, translating to more than a hundred million individuals with diabetes and a comparable number at imminent risk of conversion. [2] The overwhelming majority of these individuals have type 2 diabetes mellitus, a condition in which chronic hyperglycaemia, insulin resistance and coexisting cardiometabolic risk factors act in concert to produce progressive vascular injury.

Among the microvascular consequences of type 2 diabetes mellitus, diabetic retinopathy is the most feared because of its direct threat to sight. A systematic review and meta-analysis of population-based studies estimated the global prevalence of diabetic retinopathy among people with diabetes at 22.27%, with vision-threatening retinopathy affecting 6.17% and clinically significant macular oedema 4.07%; the number of adults worldwide with diabetic retinopathy was projected to increase from approximately 103 million in 2020 to 160 million by 2045. [3] A pooled analysis of individual participant data from population-based studies across several continents identified longer duration of diabetes, poorer glycaemic control and higher blood pressure as the dominant modifiable determinants of retinopathy, and confirmed the strong graded relationship between these exposures and vision-threatening disease. [4] Retinopathy is traditionally conceptualised as a disorder of the retinal microvasculature, but contemporary evidence indicates that hyperglycaemia-driven oxidative stress, accumulation of advanced glycation end products, activation of the polyol and protein kinase C pathways, low-grade inflammation and vascular endothelial growth factor mediated permeability act together on both the vascular and the neuroglial compartments of the retina, with neurodegeneration

frequently preceding clinically apparent vascular lesions. [5]

Indian epidemiological data indicate that retinopathy is common even where prevalence estimates are lower than in Western populations. The Chennai Urban Rural Epidemiology Study, which examined an urban south Indian population using four-field stereoscopic colour photography, reported a diabetic retinopathy prevalence of 17.6% among individuals with type 2 diabetes, a figure lower than that observed in age-matched Western cohorts but of considerable absolute significance given the size of the diabetic population at risk. [6] The clinical implication is that the absolute number of Indians requiring retinal surveillance greatly exceeds the capacity of available ophthalmic services, and that any clinical or laboratory marker capable of identifying patients at heightened risk would have substantial value in rationalising screening effort.

Diabetic kidney disease follows a parallel natural history. Increased urinary albumin excretion in the range of 30 to 300 mg per gram of creatinine, conventionally termed microalbuminuria, represents the earliest clinically detectable manifestation of glomerular injury and is a well-established predictor of progression to overt proteinuria and declining glomerular filtration. Its significance, however, extends well beyond the kidney. Albumin leakage across the glomerular filtration barrier is thought to reflect a generalised increase in transvascular albumin escape arising from widespread endothelial dysfunction, loss of the endothelial glycocalyx and impaired nitric oxide bioavailability. If this interpretation is correct, then microalbuminuria should function not merely as a renal marker but as an integrative index of systemic microvascular damage, and should therefore track with the presence and severity of microvascular disease in other organs, including the retina. Several lines of evidence support this proposition. The United Kingdom Prospective Diabetes Study demonstrated

that the incidence and progression of retinopathy over six years from diagnosis of type 2 diabetes were strongly determined by baseline and cumulative glycaemic exposure and by blood pressure, establishing that the retinal microcirculation responds to the same systemic exposures that drive glomerular injury. [7] In the Wisconsin Epidemiologic Study of Diabetic Retinopathy, microalbuminuria measured in random urine samples was associated cross-sectionally with the presence of retinopathy, and with proliferative disease in younger-onset participants, leading the investigators to propose that microalbuminuria might serve as a marker of the risk of proliferative retinopathy developing. [8] A large study of patients with non-insulin-dependent diabetes in the United States compared normoalbuminuria, microalbuminuria and overt albuminuria and found that urinary albumin excretion had strong independent associations with retinopathy, neuropathy and cardiovascular disease, prompting the conclusion that albumin excretion in type 2 diabetes may reflect a state of generalised vascular damage rather than isolated renal involvement. [9]

Despite this evidence, the relationship between microalbuminuria and retinopathy is considerably less consistent in type 2 diabetes than in type 1 diabetes. In type 1 diabetes the concordance of renal and retinal microangiopathy is close, and the temporal sequence is relatively predictable. In type 2 diabetes the picture is complicated by the frequent presence of hypertension, dyslipidaemia, obesity, age-related nephrosclerosis and undiagnosed non-diabetic renal disease, all of which may independently raise urinary albumin excretion. Conversely, a substantial proportion of patients with type 2 diabetes develop retinopathy while remaining normoalbuminuric, and others develop declining renal function without albuminuria at all. The clinical consequence is that the predictive value of microalbuminuria for retinal disease cannot simply be extrapolated from type 1 diabetes

and requires direct examination in type 2 diabetic populations, particularly in South Asian patients in whom the phenotype of diabetes, the age of onset and the pattern of complications differ appreciably from those described in Western cohorts.

Current guidance recommends that adults with type 2 diabetes undergo a dilated and comprehensive eye examination at the time of diagnosis and at intervals thereafter determined by the presence and severity of retinopathy and by the adequacy of glycaemic and blood pressure control. [10] In practice, adherence to these recommendations in resource-limited settings is poor. Patients frequently present to general physicians rather than to ophthalmologists, dilated fundus examination requires trained personnel and equipment that are unevenly distributed, and a proportion of patients are first identified only when visual loss has already occurred. Estimation of the urinary albumin-to-creatinine ratio, by contrast, is inexpensive, requires only a spot urine sample, is already recommended annually for renal surveillance and can be performed in most secondary care laboratories. If microalbuminuria reliably identifies patients at elevated risk of retinopathy, it could be used by the treating physician as a trigger for expedited ophthalmological referral, thereby directing scarce specialist capacity towards those most likely to benefit.

The published literature addressing this question in Indian patients remains limited and heterogeneous in design, in the method used to quantify albumin excretion and in the classification system used to grade retinopathy. Studies differ in whether they included patients with macroalbuminuria, whether albuminuria was confirmed on repeat sampling, whether retinopathy was graded clinically or photographically, and whether the analysis adjusted for duration of diabetes and glycaemic control, both of which are powerful confounders of any observed association. These methodological differences plausibly account for much of

the variation in reported effect sizes and justify further carefully conducted work.

The present study was therefore designed to examine, in a well-characterised cohort of patients with type 2 diabetes mellitus attending a tertiary care teaching hospital, the prevalence of microalbuminuria and its relationship with the presence and severity of diabetic retinopathy. Patients with macroalbuminuria were deliberately excluded so that the analysis could isolate the contribution of early, subclinical albumin excretion rather than that of established overt nephropathy, in which an association with advanced retinopathy is already well recognised. Albuminuria was confirmed on repeat sampling to reduce misclassification arising from transient elevations, retinopathy was graded using a standard photographic classification by an observer masked to the urinary findings, and the analysis was adjusted for the principal confounders of duration of diabetes, glycaemic control, blood pressure and age. It was anticipated that the findings would clarify whether a simple and widely available urinary test can be used to stratify retinal risk in routine medical practice.

MATERIALS & METHODS

Study design, setting and duration

A hospital-based cross-sectional analytical study was conducted in the Department of General Medicine, KMCRI Hubli, a tertiary care teaching hospital, in collaboration with the Department of Ophthalmology. The study was carried out over a period of 18 months following approval by the Institutional Ethics Committee. Written informed consent was obtained from every participant before enrolment, and the study was conducted in accordance with the principles of the Declaration of Helsinki.

Sample size

The sample size was calculated using the formula for the estimation of a single proportion, $n = Z^2 \times p \times q / d^2$, where Z was taken as 1.96 corresponding to a 95% confidence level, p was the anticipated

prevalence of microalbuminuria among patients with type 2 diabetes mellitus taken as 35% on the basis of previously published hospital-based Indian data, q was $1 - p$ or 65%, and d was the absolute precision set at 10%. Substitution gave $n = 3.8416 \times 0.35 \times 0.65 / 0.01$, which yielded 87.4 and was rounded to 88. An allowance of approximately 14% was added for non-response, incomplete investigations and ungradable fundus images, giving a final sample size of 100 patients. Consecutive sampling was used, and all eligible patients attending the outpatient and inpatient services during the study period were screened until the required number was reached.

Inclusion criteria

Patients of either sex aged between 30 and 70 years, with an established diagnosis of type 2 diabetes mellitus according to the American Diabetes Association criteria for a minimum duration of one year, who were willing to provide written informed consent and to undergo dilated fundus examination, were included.

Exclusion criteria

Patients with type 1 diabetes mellitus, gestational diabetes mellitus or secondary diabetes were excluded. Patients with macroalbuminuria, defined as a urinary albumin-to-creatinine ratio exceeding 300 mg per gram, or with established overt diabetic nephropathy, chronic kidney disease of any non-diabetic aetiology, or an estimated glomerular filtration rate below 30 mL per minute per 1.73 square metres, were also excluded, so that the analysis addressed early rather than advanced renal involvement. Conditions capable of producing transient or spurious albuminuria were excluded, namely urinary tract infection, febrile illness, haematuria, menstruation, strenuous physical exertion within the preceding 24 hours, uncontrolled heart failure and acute illness or diabetic ketoacidosis. Ocular exclusions comprised dense cataract or other media opacity

precluding adequate fundus evaluation, previous retinal photocoagulation, intravitreal pharmacotherapy or vitreoretinal surgery, glaucoma, uveitis, and retinal vascular disease of non-diabetic origin. Pregnant women and patients with known malignancy or connective tissue disease were also excluded.

Data collection procedure

Each participant was evaluated using a pre-designed and pre-tested proforma. A detailed history was recorded covering age, sex, duration of diabetes, current antidiabetic therapy, history of hypertension and its treatment, smoking and alcohol use, family history of diabetes, and any ocular symptoms. Anthropometric measurements were obtained using standard technique and the body mass index was calculated as weight in kilograms divided by the square of height in metres. Blood pressure was recorded in the sitting position after five minutes of rest using a mercury sphygmomanometer, and the mean of two readings taken ten minutes apart was used for analysis. A complete general physical and systemic examination was performed on every participant.

Laboratory investigations

Venous blood samples were collected after an overnight fast of at least eight hours. Fasting and two-hour postprandial plasma glucose were estimated by the glucose oxidase-peroxidase method and glycated haemoglobin by high-performance liquid chromatography. Serum creatinine was measured by the modified Jaffe kinetic method and the estimated glomerular filtration rate was derived using the Chronic Kidney Disease Epidemiology Collaboration equation. A fasting lipid profile, complete blood count and urine routine examination were performed for all participants. Urinary albumin was quantified by immunoturbidimetry on an early morning midstream urine sample and urinary creatinine by the modified Jaffe method, and the albumin-to-creatinine ratio

was expressed in milligrams per gram of creatinine. Normoalbuminuria was defined as a ratio below 30 mg per gram and microalbuminuria as a ratio of 30 to 300 mg per gram.

Confirmation of albuminuria and follow-up protocol

Because urinary albumin excretion shows considerable day-to-day variability, an abnormal initial result was not accepted as definitive. Every participant with an initial albumin-to-creatinine ratio in the microalbuminuric range underwent repeat estimation on two further early morning samples collected at approximately six weeks and twelve weeks. Microalbuminuria was confirmed only when at least two of the three samples were in the microalbuminuric range in the absence of any interfering condition, and patients whose repeat values fell consistently below 30 mg per gram were reclassified as normoalbuminuric. Participants were reviewed at three months, at which visit the confirmatory urine result was recorded, blood pressure and glycaemic status were reassessed, and fundus examination was repeated in the small number of patients in whom the initial grading had been equivocal. All patients found to have diabetic retinopathy were referred to the Department of Ophthalmology for definitive management, and all participants received structured counselling on glycaemic control, blood pressure control and the importance of periodic retinal screening.

Ophthalmological evaluation

Each participant underwent a comprehensive ophthalmic assessment comprising best-corrected visual acuity using a Snellen chart, anterior segment examination by slit lamp biomicroscopy, and measurement of intraocular pressure by applanation tonometry. Pupillary dilatation was achieved with tropicamide 1% and phenylephrine 5% eye drops, after which the fundus was examined by indirect ophthalmoscopy and by slit lamp

biomicroscopy using a 90 dioptre lens, supplemented by digital fundus photography. Diabetic retinopathy was graded according to the Early Treatment Diabetic Retinopathy Study severity scale as no apparent retinopathy, mild non-proliferative, moderate non-proliferative, severe non-proliferative or proliferative retinopathy, and the presence of diabetic macular oedema was recorded separately. All grading was performed by a single consultant ophthalmologist who was masked to the urinary albumin results, and the grade of the more severely affected eye was used for analysis.

Statistical Analysis

Data were entered in a Microsoft Excel spreadsheet and analysed using the Statistical Package for the Social Sciences version 25.0. The normality of continuous variables was assessed using the Shapiro-Wilk test. Normally distributed continuous variables were expressed as mean with standard deviation and compared between two groups using the unpaired Student t test, while skewed variables, including the urinary albumin-to-creatinine ratio, were expressed as median with interquartile range and compared using the Mann-Whitney U test. Categorical variables were expressed as frequencies and percentages and compared using the Pearson chi-square test, with the Fisher exact test applied where expected cell counts were below five. The strength of association was expressed as an odds ratio with a 95% confidence interval. The relationship between continuous variables and the ordinal severity grade of retinopathy was examined using the Spearman rank correlation coefficient. Variables associated with the presence of retinopathy at a significance level below 0.10 on univariate analysis, together with variables of established biological importance, were entered into a binary logistic regression model, and adjusted odds ratios with 95% confidence intervals were derived; model calibration was assessed by the Hosmer-Lemeshow goodness-of-fit test. Receiver

operating characteristic analysis was performed to determine the discriminatory ability of the urinary albumin-to-creatinine ratio for the detection of any diabetic retinopathy, and the optimal cut-off was identified using the Youden index. A two-tailed p value below 0.05 was considered statistically significant.

RESULT

A total of 100 patients with type 2 diabetes mellitus who satisfied the eligibility criteria were studied. The mean age of the cohort was 55.2 ± 8.9 years, with the largest proportion of patients falling in the 51 to 60 year age band, which accounted for 39 (39.0%) participants. Fifty-six (56.0%) participants were male and 44 (44.0%) were female, giving a male-to-female ratio of 1.27 to 1. The mean duration of diabetes was 9.3 ± 4.7 years, and 40 (40.0%) participants had been diabetic for more than ten years. The mean body mass index was 26.4 ± 3.5 kg/m² and the mean glycated haemoglobin was $8.2 \pm 1.4\%$. Hypertension was present in 54 (54.0%) participants. Sixty-two (62.0%) participants were receiving oral hypoglycaemic agents alone, 28 (28.0%) were on a combination of oral agents and insulin, and 10 (10.0%) were on insulin alone. The detailed baseline profile of the study population is presented in Table 1.

Microalbuminuria was confirmed on repeat sampling in 38 (38.0%) participants, while the remaining 62 (62.0%) were normoalbuminuric. The median urinary albumin-to-creatinine ratio in the microalbuminuric group was 78.4 mg/g with an interquartile range of 52.6 to 142.8 mg/g, compared with 12.6 mg/g and an interquartile range of 8.4 to 19.2 mg/g in the normoalbuminuric group, and this difference was highly significant on the Mann-Whitney U test ($Z = -8.12$, $p < 0.001$). Comparison of clinical and biochemical parameters between the two groups, summarised in Table 2, showed that microalbuminuric patients had a significantly longer duration of diabetes

(11.6 ± 4.8 versus 7.9 ± 4.1 years; $t = 3.95$, $p < 0.001$), higher glycated haemoglobin ($8.9 \pm 1.4\%$ versus $7.8 \pm 1.2\%$; $t = 4.02$, $p < 0.001$), higher systolic blood pressure (142.6 ± 14.2 versus 132.8 ± 13.1 mmHg; $t = 3.45$, $p = 0.001$), higher diastolic blood pressure (86.4 ± 8.2 versus 82.1 ± 7.9 mmHg; $t = 2.58$, $p = 0.011$), higher fasting plasma glucose (168.4 ± 42.6 versus 146.2 ± 38.4 mg/dL; $t = 2.62$, $p = 0.010$) and higher serum creatinine (1.02 ± 0.21 versus 0.89 ± 0.18 mg/dL; $t = 3.17$, $p = 0.002$). Mean age (56.8 ± 8.4 versus 54.2 ± 9.1 years; $t = 1.41$, $p = 0.162$), body mass index (26.8 ± 3.6 versus 26.1 ± 3.4 kg/m²; $t = 0.97$, $p = 0.335$) and total cholesterol (196.4 ± 38.2 versus 184.6 ± 35.4 mg/dL; $t = 1.55$, $p = 0.124$) did not differ significantly between the groups.

Diabetic retinopathy of any grade was detected in 34 (34.0%) of the 100 participants. Mild non-proliferative retinopathy was the commonest pattern, seen in 14 (14.0%) participants, followed by moderate non-proliferative retinopathy in 11 (11.0%), severe non-proliferative retinopathy in 5 (5.0%) and proliferative retinopathy in 4 (4.0%). Sight-threatening disease, comprising severe non-proliferative and proliferative retinopathy, was therefore present in 9 (9.0%) of the cohort. Diabetic macular oedema was documented in 7 (7.0%) participants, of whom 6 belonged to the microalbuminuric group.

The central finding of the study concerned the distribution of retinopathy across albuminuria categories, which is presented in Table 3 and illustrated in Figure 1. Diabetic retinopathy was present in 22 of 38 (57.9%) microalbuminuric patients compared with 12 of 62 (19.4%) normoalbuminuric patients, and this difference was highly significant (chi-square = 15.59, df = 1, $p < 0.001$), corresponding to an unadjusted odds ratio of 5.73 with a 95% confidence interval of 2.33 to 14.11. The severity distribution across the five retinopathy grades also differed significantly between the two groups (chi-square = 17.86, df = 4, $p = 0.001$). Among the microalbuminuric patients, 16 (42.1%)

had no retinopathy, 7 (18.4%) had mild non-proliferative disease, 8 (21.1%) had moderate non-proliferative disease, 4 (10.5%) had severe non-proliferative disease and 3 (7.9%) had proliferative disease. Among the normoalbuminuric patients, the corresponding figures were 50 (80.6%), 7 (11.3%), 3 (4.8%), 1 (1.6%) and 1 (1.6%). Sight-threatening retinopathy was thus present in 7 of 38 (18.4%) microalbuminuric patients compared with 2 of 62 (3.2%) normoalbuminuric patients (Fisher exact test, $p = 0.010$). Taking microalbuminuria as a test for the presence of any diabetic retinopathy, the sensitivity was 64.7%, the specificity 75.8%, the positive predictive value 57.9%, the negative predictive value 80.6% and the overall diagnostic accuracy 72.0%.

Comparison of the participants with and without diabetic retinopathy, shown in Table 4, provided a complementary view of the same relationship. Patients with retinopathy were older (57.9 ± 8.2 versus 53.8 ± 9.0 years; $t = 2.22$, $p = 0.029$), had a longer duration of diabetes (12.4 ± 4.6 versus 7.7 ± 3.9 years; $t = 5.09$, $p < 0.001$), higher glycated haemoglobin ($9.1 \pm 1.3\%$ versus $7.7 \pm 1.2\%$; $t = 5.24$, $p < 0.001$), higher systolic blood pressure (142.8 ± 14.6 versus 133.4 ± 12.8 mmHg; $t = 3.18$, $p = 0.002$) and higher serum creatinine (1.03 ± 0.22 versus 0.90 ± 0.18 mg/dL; $t = 3.10$, $p = 0.003$) than those without retinopathy. The median urinary albumin-to-creatinine ratio was 86.4 mg/g with an interquartile range of 48.2 to 158.6 mg/g in patients with retinopathy compared with 18.2 mg/g and an interquartile range of 9.6 to 34.4 mg/g in those without ($Z = -5.42$, $p < 0.001$). Body mass index did not differ significantly between the two groups (26.9 ± 3.4 versus 26.1 ± 3.5 kg/m²; $t = 1.09$, $p = 0.279$), and there was no significant difference in the distribution of sex ($p = 0.412$).

Spearman rank correlation analysis, presented in Table 5, showed that the urinary albumin-to-creatinine ratio had a moderate positive correlation with the ordinal severity grade of diabetic

retinopathy ($\rho = 0.512$, $p < 0.001$), which was the strongest correlation observed among the variables examined. Glycated haemoglobin ($\rho = 0.436$, $p < 0.001$) and duration of diabetes ($\rho = 0.398$, $p < 0.001$) also correlated significantly with retinopathy severity, as did systolic blood pressure ($\rho = 0.284$, $p = 0.004$) and serum creatinine ($\rho = 0.246$, $p = 0.014$). Body mass index ($\rho = 0.086$, $p = 0.395$) and total cholesterol ($\rho = 0.132$, $p = 0.191$) showed no significant correlation with retinopathy grade.

Binary logistic regression was performed with the presence of any diabetic retinopathy as the dependent variable and microalbuminuria, duration of diabetes of ten years or more, glycated haemoglobin, systolic blood pressure, age and male sex as covariates; the results are shown in Table 6. Microalbuminuria remained independently associated with diabetic retinopathy after adjustment, with an adjusted odds ratio of 3.86 and a 95% confidence interval of 1.42 to 10.52 ($p = 0.008$). Diabetes duration of ten years or more (adjusted odds ratio 3.12, 95% confidence interval 1.18 to 8.24; $p = 0.022$), glycated haemoglobin expressed per 1% increment (adjusted odds ratio 1.62, 95% confidence interval 1.12 to 2.34; $p = 0.010$) and systolic blood pressure expressed per 10 mmHg increment (adjusted odds ratio 1.44, 95% confidence interval 1.02 to

2.03; $p = 0.038$) were also independent predictors. Age (adjusted odds ratio 1.02 per year, 95% confidence interval 0.97 to 1.08; $p = 0.412$) and male sex (adjusted odds ratio 1.18, 95% confidence interval 0.46 to 3.02; $p = 0.731$) were not significant in the adjusted model. The model showed satisfactory calibration on the Hosmer-Lemeshow test (chi-square = 6.42, $df = 8$, $p = 0.601$) and accounted for a substantial proportion of the variance in the outcome (Nagelkerke $R^2 = 0.412$), correctly classifying 78.0% of cases.

Receiver operating characteristic analysis, illustrated in Figure 2, was performed to assess the ability of the quantitative urinary albumin-to-creatinine ratio to discriminate between participants with and without diabetic retinopathy. The area under the curve was 0.786 with a 95% confidence interval of 0.692 to 0.880 ($p < 0.001$). The optimal cut-off identified by the Youden index was an albumin-to-creatinine ratio of 41.5 mg/g, at which the sensitivity was 73.5% and the specificity 75.8%, giving a Youden index of 0.493. This threshold lay above the conventional diagnostic boundary of 30 mg/g, indicating that within the microalbuminuric range the risk of retinopathy increased progressively with the degree of albumin excretion rather than in a simple dichotomous fashion.

Table 1. Baseline demographic, clinical and biochemical characteristics of the study population (N = 100)

Variable	Frequency (n)	Percentage / Mean $\pm$ SD
Age group (years)		
30–40	8	8.0%
41–50	24	24.0%
51–60	39	39.0%
61–70	29	29.0%
Mean age	—	55.2 $\pm$ 8.9 years
Sex		
Male	56	56.0%
Female	44	44.0%
Duration of diabetes		
< 5 years	22	22.0%
5–10 years	38	38.0%
> 10 years	40	40.0%
Mean duration	—	9.3 $\pm$ 4.7 years
Body mass index	—	26.4 $\pm$ 3.5 kg/m ²
Systolic blood pressure	—	136.5 $\pm$ 14.2 mmHg
Diastolic blood pressure	—	83.7 $\pm$ 8.1 mmHg

Coexisting hypertension	54	54.0%
Current or former smoking	27	27.0%
Family history of diabetes	41	41.0%
Fasting plasma glucose	—	154.6 ± 41.2 mg/dL
Glycated haemoglobin	—	8.2 ± 1.4%
Serum creatinine	—	0.94 ± 0.20 mg/dL
Total cholesterol	—	189.1 ± 36.7 mg/dL
Treatment		
Oral hypoglycaemic agents alone	62	62.0%
Oral agents with insulin	28	28.0%
Insulin alone	10	10.0%
Microalbuminuria present	38	38.0%
Diabetic retinopathy present	34	34.0%

SD = standard deviation. Percentages are of the total study population of 100.

Table 2. Comparison of clinical and biochemical parameters between microalbuminuric and normoalbuminuric patients

Parameter	Microalbuminuria (n = 38)	Normoalbuminuria (n = 62)	Test value	p value
Age (years)	56.8 ± 8.4	54.2 ± 9.1	t = 1.41	0.162
Male sex, n (%)	23 (60.5)	33 (53.2)	$\chi^2 = 0.51$	0.475
Duration of diabetes (years)	11.6 ± 4.8	7.9 ± 4.1	t = 3.95	< 0.001
Body mass index (kg/m ²)	26.8 ± 3.6	26.1 ± 3.4	t = 0.97	0.335
Systolic blood pressure (mmHg)	142.6 ± 14.2	132.8 ± 13.1	t = 3.45	0.001
Diastolic blood pressure (mmHg)	86.4 ± 8.2	82.1 ± 7.9	t = 2.58	0.011
Fasting plasma glucose (mg/dL)	168.4 ± 42.6	146.2 ± 38.4	t = 2.62	0.010
Glycated haemoglobin (%)	8.9 ± 1.4	7.8 ± 1.2	t = 4.02	< 0.001
Serum creatinine (mg/dL)	1.02 ± 0.21	0.89 ± 0.18	t = 3.17	0.002
Total cholesterol (mg/dL)	196.4 ± 38.2	184.6 ± 35.4	t = 1.55	0.124
Urinary ACR (mg/g), median (IQR)	78.4 (52.6–142.8)	12.6 (8.4–19.2)	Z = -8.12	< 0.001
Diabetic retinopathy, n (%)	22 (57.9)	12 (19.4)	$\chi^2 = 15.59$	< 0.001

Values are mean ± standard deviation unless stated otherwise. ACR = albumin-to-creatinine ratio; IQR = interquartile range. Continuous variables compared using the unpaired Student t test, skewed variables using the Mann-Whitney U test and proportions using the Pearson chi-square test. A p value below 0.05 was considered significant.

Table 3. Distribution and grade of diabetic retinopathy according to albuminuria status

Grade of diabetic retinopathy	Microalbuminuria (n = 38), n (%)	Normoalbuminuria (n = 62), n (%)	Total (N = 100)	p value
No apparent retinopathy	16 (42.1)	50 (80.6)	66	
Mild non-proliferative	7 (18.4)	7 (11.3)	14	
Moderate non-proliferative	8 (21.1)	3 (4.8)	11	0.001
Severe non-proliferative	4 (10.5)	1 (1.6)	5	
Proliferative retinopathy	3 (7.9)	1 (1.6)	4	
Any retinopathy	22 (57.9)	12 (19.4)	34	< 0.001
Sight-threatening retinopathy	7 (18.4)	2 (3.2)	9	0.010
Diabetic macular oedema	6 (15.8)	1 (1.6)	7	0.013

Sight-threatening retinopathy comprises severe non-proliferative and proliferative disease. The p value of 0.001 refers to the comparison of the five-grade severity distribution between groups ($\chi^2 = 17.86$, df = 4). The comparison for any retinopathy gave $\chi^2 = 15.59$, df = 1, with an odds ratio of 5.73 (95% confidence interval 2.33–14.11). Comparisons for sight-threatening retinopathy and macular oedema used the Fisher exact test.

Table 4. Comparison of study parameters between patients with and without diabetic retinopathy

Parameter	Retinopathy present (n = 34)	Retinopathy absent (n = 66)	Test value	p value
Age (years)	57.9 ± 8.2	53.8 ± 9.0	t = 2.22	0.029
Male sex, n (%)	21 (61.8)	35 (53.0)	$\chi^2 = 0.67$	0.412
Duration of diabetes (years)	12.4 ± 4.6	7.7 ± 3.9	t = 5.09	< 0.001
Body mass index (kg/m ²)	26.9 ± 3.4	26.1 ± 3.5	t = 1.09	0.279
Systolic blood pressure (mmHg)	142.8 ± 14.6	133.4 ± 12.8	t = 3.18	0.002
Glycated haemoglobin (%)	9.1 ± 1.3	7.7 ± 1.2	t = 5.24	< 0.001
Serum creatinine (mg/dL)	1.03 ± 0.22	0.90 ± 0.18	t = 3.10	0.003
Urinary ACR (mg/g), median (IQR)	86.4 (48.2–158.6)	18.2 (9.6–34.4)	Z = -5.42	< 0.001
Microalbuminuria, n (%)	22 (64.7)	16 (24.2)	$\chi^2 = 15.59$	< 0.001

ACR = albumin-to-creatinine ratio; IQR = interquartile range. Values are mean ± standard deviation unless stated otherwise.

Table 5. Spearman rank correlation of study variables with severity grade of diabetic retinopathy

Variable	Correlation coefficient (rho)	p value
Urinary albumin-to-creatinine ratio	0.512	< 0.001
Glycated haemoglobin	0.436	< 0.001
Duration of diabetes	0.398	< 0.001
Systolic blood pressure	0.284	0.004
Serum creatinine	0.246	0.014
Age	0.198	0.048
Total cholesterol	0.132	0.191
Body mass index	0.086	0.395

Retinopathy severity was coded as an ordinal variable from 0 (no apparent retinopathy) to 4 (proliferative retinopathy).

Table 6. Binary logistic regression analysis of predictors of diabetic retinopathy

Predictor variable	Adjusted odds ratio	95% confidence interval	p value
Microalbuminuria (present versus absent)	3.86	1.42–10.52	0.008
Duration of diabetes ≥ 10 years	3.12	1.18–8.24	0.022
Glycated haemoglobin (per 1% increase)	1.62	1.12–2.34	0.010
Systolic blood pressure (per 10 mmHg)	1.44	1.02–2.03	0.038
Age (per 1 year increase)	1.02	0.97–1.08	0.412
Male sex	1.18	0.46–3.02	0.731

Dependent variable was the presence of any diabetic retinopathy. Hosmer-Lemeshow goodness-of-fit $\chi^2 = 6.42$, df = 8, p = 0.601; Nagelkerke R² = 0.412; overall correct classification 78.0%.

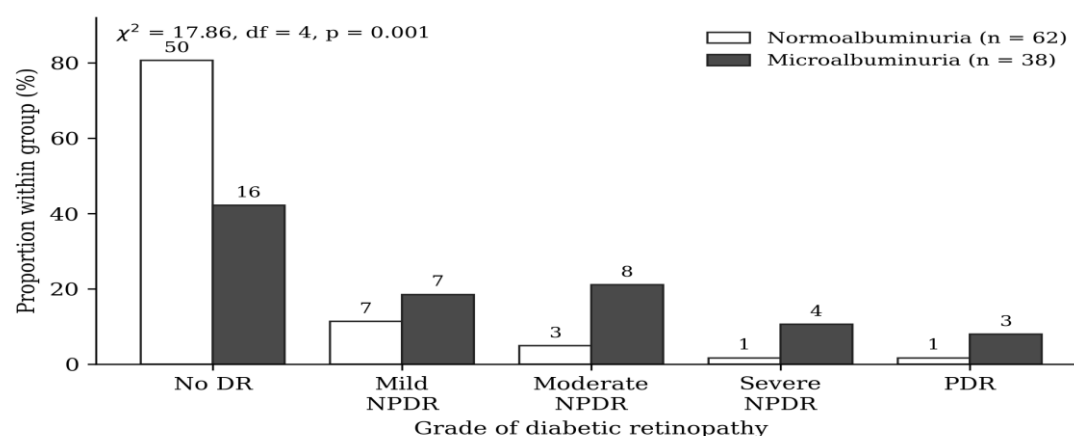


Figure 1. Distribution of diabetic retinopathy grades among microalbuminuric and normoalbuminuric patients. Bars represent the proportion within each albuminuria group and the numbers above the bars are absolute patient counts. The severity distribution differed significantly between the two groups.

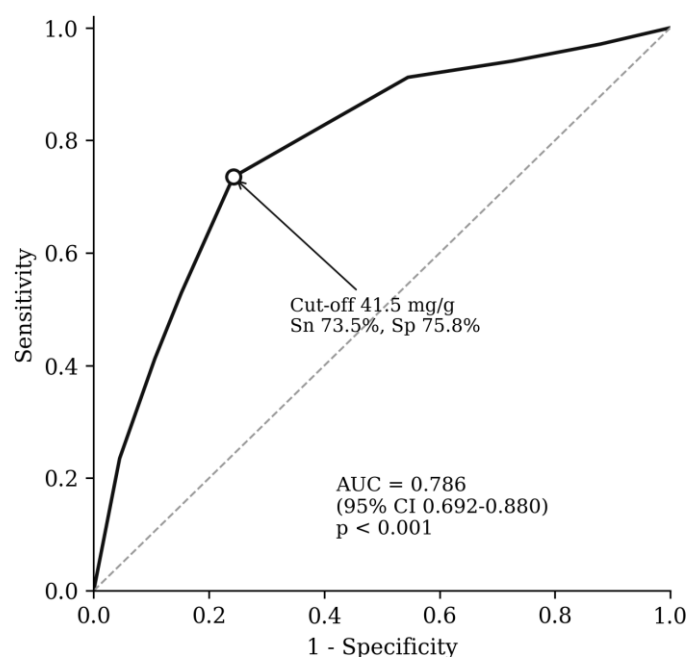


Figure 2. Receiver operating characteristic curve for the urinary albumin-to-creatinine ratio in the detection of any diabetic retinopathy. The optimal cut-off identified by the Youden index was 41.5 mg/g.

DISCUSSION

The present cross-sectional study of 100 patients with type 2 diabetes mellitus demonstrated that microalbuminuria was confirmed in 38.0% of participants and that diabetic retinopathy was almost three times as frequent among microalbuminuric patients as among normoalbuminuric patients. The association persisted after adjustment for duration of diabetes, glycaemic control, blood pressure, age and sex, and the quantitative albumin-to-creatinine ratio correlated with the ordinal severity of retinopathy. Taken together, these findings support the view that albumin excretion in type 2 diabetes reflects an underlying state of generalised microvascular injury rather than an isolated renal abnormality, and that the retina and the glomerulus respond in parallel to the same systemic exposures.

The prevalence of retinopathy of 34.0% observed in the present study is higher than the 17.6% reported from the population-based Chennai Urban Rural Epidemiology Study. [6] This difference is anticipated and is largely explained by case mix. Population-based surveys enumerate all individuals with diabetes in a defined

community, including a substantial proportion with short duration and newly detected disease, whereas a tertiary care cohort such as the present one is enriched for patients with longer duration, poorer glycaemic control and established complications. The mean duration of diabetes in the present cohort was 9.3 years and the mean glycated haemoglobin was 8.2%, both of which are consistent with a referral population. Hospital-based Indian series have reported comparable figures; an evaluation of 306 patients with type 2 diabetes attending an ophthalmic service in Bengaluru documented retinopathy of any grade in 43% and proliferative disease in 8.5%, [15] while a cross-sectional study of 590 type 2 diabetic patients from Iran reported an overall retinopathy prevalence of 39.3% with proliferative disease in 5.4%. [12] The prevalence of 34.0% in the present study therefore sits comfortably within the range reported from comparable clinic-based cohorts.

The observed prevalence of microalbuminuria of 38.0% is higher than the 15.9% reported in the population-based Sankara Nethralaya Diabetic Retinopathy Epidemiology and Molecular Genetic

Study, which examined 1414 subjects with type 2 diabetes from the Chennai metropolis,¹¹ and higher than the 25.9% reported by Manaviat and colleagues. [12] The same explanation of referral bias applies, compounded by the fact that the present study deliberately excluded patients with macroalbuminuria; removing the most advanced renal cases concentrates the remaining cohort towards the microalbuminuric band and inflates its apparent share. Differences in the method of ascertainment are also relevant, since the present study used quantitative immunoturbidimetric estimation of the albumin-to-creatinine ratio confirmed on repeat sampling, whereas some earlier series relied on semi-quantitative dipstick methods that are less sensitive at the lower end of the microalbuminuric range.

The strength of the association observed here is consistent with, and in the same direction as, the major published literature. The Sankara Nethralaya investigators reported that the prevalence of diabetic retinopathy rose progressively across albuminuria categories, being 14.1% in normoalbuminuric subjects, 31.0% in microalbuminuric subjects and 60.5% in those with macroalbuminuria, with a corresponding gradient in disease severity; they concluded that subjects with microalbuminuria were approximately twice as likely to have retinopathy as those without, and that this risk rose to nearly six-fold in the presence of macroalbuminuria. [11] The unadjusted odds ratio of 5.73 and adjusted odds ratio of 3.86 obtained in the present study are numerically larger than the approximate doubling of risk reported in that population-based cohort, a difference that most plausibly reflects the higher baseline severity of the present hospital cohort and the smaller sample, which widens the confidence interval and permits a larger point estimate. In the Wisconsin Epidemiologic Study of Diabetic Retinopathy, microalbuminuria measured in random urine samples was likewise associated cross-sectionally with the

presence of retinopathy and, in younger-onset participants, with proliferative disease. [8] A study of 947 patients with non-insulin-dependent diabetes in the United States found that urinary albumin excretion had strong independent associations with retinopathy, neuropathy and cardiovascular disease on multiple logistic regression, and the investigators explicitly interpreted albumin excretion as a marker of generalised vascular damage. [9] The present findings replicate that pattern in an Indian tertiary care setting.

The gradient of retinopathy severity across albuminuria categories deserves particular emphasis, because it is this gradient rather than the simple dichotomous association that carries clinical weight. In the present study, sight-threatening retinopathy was present in 18.4% of microalbuminuric patients but in only 3.2% of normoalbuminuric patients, and diabetic macular oedema showed a similar skew. A cross-sectional study of 1214 patients with type 2 diabetes from Brazil examined this question directly and found that, after patients with macroalbuminuria and those on dialysis were excluded, microalbuminuria remained independently associated with proliferative diabetic retinopathy with an odds ratio of 3.3 and a 95% confidence interval of 1.56 to 6.98. [13] That study is methodologically the closest analogue to the present work, since it too isolated early albuminuria by excluding overt nephropathy, and its effect estimate is closely comparable to the adjusted odds ratio of 3.86 obtained here. An Indian study of 250 patients with type 2 diabetes from Bhubaneswar similarly reported that microalbuminuria was associated with all grades of retinopathy with a skew towards the more severe grades. [16] The convergence of these estimates across different populations and analytical approaches strengthens confidence that the association is genuine rather than an artefact of confounding by duration or glycaemic control.

A further observation of the present study was that the risk of retinopathy appeared to increase continuously with the degree of albumin excretion rather than at a fixed diagnostic threshold. The receiver operating characteristic analysis identified an optimal cut-off of 41.5 mg/g, above the conventional boundary of 30 mg/g, and the albumin-to-creatinine ratio correlated with retinopathy grade. This is consistent with a large study from China that examined 4739 subjects with type 2 diabetes cross-sectionally and 297 subjects prospectively, and demonstrated that the odds of retinopathy rose progressively across deciles of albumin excretion, with a threshold well below the conventional definition of microalbuminuria discriminating future retinopathy risk; in the prospective arm, subjects with higher albumin excretion within the so-called normal range developed retinopathy at more than twice the rate of those with lower excretion over a mean follow-up of 4.5 years. [14] The implication of both that work and the present analysis is that the dichotomy between normoalbuminuria and microalbuminuria is a clinical convenience rather than a biological boundary, and that the quantitative value carries information that is lost when it is reduced to a binary category. A study of 306 Indian patients likewise found that both microalbuminuria and lower haemoglobin concentration predicted the occurrence and increasing severity of retinopathy, of clinically significant macular oedema and of hard exudate formation, even after correction for duration of diabetes and other systemic risk factors. [15]

It is equally important to acknowledge the substantial body of evidence that qualifies or contradicts a simple one-to-one correspondence between renal and retinal microangiopathy in type 2 diabetes. In the present cohort, 12 of the 34 patients with retinopathy, amounting to 35.3% of those affected, were normoalbuminuric, and conversely 16 of the 38 microalbuminuric patients, amounting to 42.1%, had no detectable retinopathy. The negative

predictive value of 80.6% was considerably better than the positive predictive value of 57.9%, and the sensitivity of 64.7% would be inadequate for microalbuminuria to function as a stand-alone screening test. This dissociation is a recognised feature of type 2 diabetes and contrasts with the tighter concordance described in type 1 diabetes. [11] Analysis of 64,784 patients from the German and Austrian prospective diabetes follow-up database found that although microalbuminuria was significantly associated with any retinopathy, glycated haemoglobin above 8%, hypertension, body mass index above 35 kg/m² and male sex were also independently associated, and that it was glycated haemoglobin together with micro- and macroalbuminuria that constituted the strongest predictors of severe retinopathy, indicating that albuminuria operates as one determinant within a multifactorial model rather than as a sufficient marker in its own right. [19] The nationwide Taiwanese cohort study of patients with newly diagnosed diabetic nephropathy reported adjusted hazard ratios for the development of non-proliferative and proliferative retinopathy of 5.01 and 9.7 respectively when compared with matched patients without nephropathy, confirming a strong longitudinal link but also demonstrating that the relationship is best characterised in terms of relative hazard over time rather than as a fixed cross-sectional correspondence. [18] The explanation for the discordant cases is likely to be multifactorial and includes non-diabetic causes of albuminuria such as hypertensive nephrosclerosis and age-related glomerular change, genetic variation in susceptibility to retinal as opposed to renal injury, differential effects of renin-angiotensin system blockade on albumin excretion, and the phenomenon of normoalbuminuric diabetic kidney disease. The independent contributions of duration of diabetes, glycated haemoglobin and systolic blood pressure observed in the present regression model are entirely concordant with established evidence. The

United Kingdom Prospective Diabetes Study, which followed 1919 patients with retinal photographs at diagnosis and again six years later, found that the incidence of retinopathy was strongly associated with baseline glycaemia, with cumulative glycaemic exposure and with higher blood pressure, and that progression among those with existing retinopathy was associated with older age and hyperglycaemia. [7] The pooled analysis of population-based studies from multiple continents reached the same conclusion regarding duration, glycaemic control and blood pressure as the dominant determinants of both any retinopathy and vision-threatening retinopathy. [4] The persistence of an independent effect of microalbuminuria in the present model after these variables were included is therefore the informative finding, since it indicates that albumin excretion contributes information beyond that supplied by the conventional risk factors.

The reverse inference is also supported by the literature and is of practical importance to the physician. A cross-sectional study of 648 patients with diabetes reported that the presence of retinopathy significantly predicted neuropathy with an odds ratio of 2.23 and nephropathy with an odds ratio of 5.68, establishing that the relationship between retinal and renal microangiopathy is bidirectional and that either may serve as a sentinel for the other. [17] In routine practice this means that the finding of microalbuminuria in a patient with type 2 diabetes should prompt fundus examination, and equally that the detection of retinopathy should prompt assessment of renal function and albumin excretion. Current standards of care recommend a dilated and comprehensive eye examination at diagnosis and at intervals thereafter. [10] Where ophthalmic services are stretched, as they are across much of India, the albumin-to-creatinine ratio offers a means of triage: it is inexpensive, requires only a spot urine sample, is already recommended annually for renal surveillance and can identify a subgroup in whom the pre-test probability

of sight-threatening disease is several times higher than average. Given the projected rise in the number of adults with diabetic retinopathy worldwide from approximately 103 million in 2020 to 160 million by 2045, [2] and the magnitude of the Indian diabetic population, [2] such risk-stratified approaches to screening are likely to become increasingly necessary.

Limitations

Several limitations should be borne in mind when interpreting these findings. The cross-sectional design permits the demonstration of association but not of causation or temporal sequence, and it cannot establish whether microalbuminuria precedes, follows or merely accompanies the development of retinopathy. The sample size of 100 was adequate for the primary comparison but limited the precision of the effect estimates, as reflected in the wide confidence intervals in the regression model, and precluded meaningful subgroup analysis by severity grade. The study was conducted at a single tertiary care centre and the cohort was consequently enriched for patients with longer duration and poorer control, so the prevalence figures reported here should not be extrapolated to the community. Patients with macroalbuminuria were excluded by design, which sharpened the focus on early albuminuria but prevented examination of the full albuminuria spectrum. Although albuminuria was confirmed on repeat sampling, the influence of antihypertensive therapy, particularly angiotensin-converting enzyme inhibitors and angiotensin receptor blockers, on measured albumin excretion could not be fully controlled. Retinopathy was graded by a single masked observer without a second independent grader, so inter-observer reproducibility could not be quantified. Finally, several variables of potential relevance, including haemoglobin concentration, serum lipid subfractions, duration and adequacy of blood pressure control and markers of systemic inflammation, were not incorporated into

the analysis, and residual confounding by these factors cannot be excluded.

CONCLUSION

In this hospital-based cross-sectional study of 100 patients with type 2 diabetes mellitus, microalbuminuria was present in 38.0% and diabetic retinopathy in 34.0%. Retinopathy was significantly more frequent among microalbuminuric than among normoalbuminuric patients, and the association remained significant after adjustment for duration of diabetes, glycaemic control, blood pressure, age and sex. The severity of retinopathy increased across albuminuria categories, sight-threatening disease was largely confined to microalbuminuric patients, and the quantitative albumin-to-creatinine ratio correlated with the ordinal grade of retinopathy, with discrimination that was moderate rather than excellent.

The findings indicate that microalbuminuria in type 2 diabetes mellitus should be interpreted as a marker of generalised microvascular injury rather than as a purely renal abnormality, and that its detection identifies a subgroup at appreciably elevated risk of retinal disease. The clinical implication is that estimation of the urinary albumin-to-creatinine ratio, which is inexpensive and already part of routine annual diabetes care, can be used by the treating physician to prioritise referral for dilated fundus examination in settings where ophthalmological capacity is limited. It must be emphasised, however, that the sensitivity observed was insufficient for microalbuminuria to replace retinal screening, since a substantial proportion of patients with retinopathy were normoalbuminuric; the test should therefore be used to escalate rather than to withhold ophthalmic assessment. Prospective studies with larger cohorts, serial quantification of albumin excretion and standardised photographic grading are needed to define the temporal relationship between the two complications and to determine whether interventions that reduce albumin excretion

also modify the course of diabetic retinopathy.

Declaration

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