e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2026; 18(8); 242-248

Original Research Article

# Microalbuminuria and its Risk Factors in Children with Type 1 Diabetes Mellitus: A Prospective Observational Study from a Tertiary Paediatric Centre in South India

**Priyanka A. R.[1], Karunya Kannan[2], Sathvika Chandrashekara Reddy[3], Alkarani T. Patil[4], Ranjitha M.[5], Jacqueline A. Shah[6]**

[1]Assistant Professor, Department of Paediatrics, Indira Gandhi Institute of Child Health, Bengaluru, Karnataka, India

[2]Senior Resident, Department of Paediatrics, Akash Institute of Medical Sciences and Research Centre, Bengaluru, Karnataka, India

[3]Assistant Professor, Department of Paediatrics, Indira Gandhi Institute of Child Health, Bengaluru, Karnataka, India

[4]Professor & HOD, Department of Paediatrics, Indira Gandhi Institute of Child Health, Bengaluru, Karnataka, India

[5]Assistant Professor, Dept. of Pediatric Medicine, Indira Gandhi Institute of Child Health, Bengaluru, Karnataka, India

[6]Assistant Professor, Dept. of Pediatrics, Indira Gandhi Institute of Child Health, Bangalore, Karnataka, India

**Received: 01-05-2026 / Revised: 15-06-2026 / Accepted: 21-07-2026**

**Corresponding author: Dr. Sathvika Chandrashekara Reddy**

**Conflict of interest: Nil**

**Abstract**

**Background:** The rising incidence of type 1 diabetes mellitus (T1DM) in Indian children has made the prevention of long-term microvascular complications a priority. Persistent microalbuminuria is the earliest clinically detectable marker of diabetic kidney disease and an independent predictor of cardiovascular risk. Data on microalbuminuria in Indian children with T1DM remain limited.

**Objective:** To determine the occurrence of persistent microalbuminuria and to identify the clinical and biochemical factors associated with it in children with T1DM.

**Methods:** A prospective observational study was conducted over 18 months at a tertiary paediatric hospital in Bengaluru, India. Seventy-two children aged 1–18 years with T1DM of more than two years' duration were enrolled after informed consent and assent. Demographic data, disease duration, history of diabetic ketoacidosis (DKA), insulin regimen, anthropometry and blood pressure were recorded. The urinary albumin–creatinine ratio (ACR) was measured on three early-morning midstream specimens collected over 3–6 months; microalbuminuria was defined as an ACR of 30–300 µg/mg and was considered persistent when present in at least two of three specimens. HbA1c, blood urea and serum creatinine were measured, and the estimated glomerular filtration rate (eGFR) was derived using the Schwartz formula. Data were analysed with the chi-square test and ANOVA; $p < 0.05$ was considered significant.

**Results:** Of 72 children (mean age 12 ± 3.7 years), 40 (55.6%) were aged 12–18 years and 43 (59.7%) were female (male: female 1:1.48). Disease duration was 2–5 years in 48 (66.7%). A family history of diabetes was present in 20 (27.8%). Poor glycaemic control (HbA1c > 9%) was documented in 51 (70.8%). Persistent microalbuminuria was detected in 6 children (8.3%), of whom 5 (83.3%) were aged 12–18 years and 5 (83.3%) were female; 3 (50%) had disease duration of 5–10 years and 2 (33.3%) had duration of only 2–5 years. All 6 microalbuminuric children (100%) had poor glycaemic control. Mean HbA1c across three visits was significantly higher in microalbuminuric than normoalbuminuric children (12.8 ± 2.7% vs 9.7 ± 2.0%; $p < 0.001$). Mean eGFR was higher in the microalbuminuric group (131.8 ± 2.8 vs 124.0 ± 2.3 mL/min/1.73 m²; $p = 0.47$), while urea and creatinine were comparable. Blood pressure, BMI, sex, disease duration and family history did not differ significantly between groups. Transient microalbuminuria was observed in a further 3 children (4.2%).

**Conclusion:** Persistent microalbuminuria affected 8.3% of children with T1DM and occurred even in those with less than five years of disease. Poor glycaemic control (HbA1c > 9%) was the only significant risk factor identified, and glomerular hyperfiltration accompanied early albuminuria. Screening for microalbuminuria should therefore begin close to the time of diagnosis rather than being deferred to an arbitrary duration threshold, so that intensification of glycaemic control can be instituted while renal injury is still reversible.

**Keywords:** Type 1 Diabetes Mellitus; Microalbuminuria; Albumin–Creatinine Ratio; HbA1c; Diabetic Nephropathy; Hyperfiltration; Children.

**DOI:** 10.25258/ijcpr.18.8.37



## Introduction

Type 1 diabetes mellitus (T1DM) is a chronic autoimmune disorder in which T-cell–mediated destruction of pancreatic beta cells results in absolute insulin deficiency, and it remains the commonest form of diabetes presenting in childhood and adolescence [1,2]. Its incidence is rising globally and in India, so that a growing cohort of children now faces several decades of cumulative exposure to hyperglycaemia [2]. Sustained hyperglycaemia damages the eyes, kidneys, nerves, heart and blood vessels, and the risk of these microvascular and macrovascular complications increases with poorer metabolic control [1,3].

Diabetic kidney disease (DKD) is the most consequential of these complications. It develops in 20–40% of people with diabetes and is the single leading cause of end-stage renal disease worldwide [3]. Clinically it is characterised by persistent proteinuria, a progressive decline in glomerular function, hypertension and eventual renal failure [3,4]. The natural history is a graded one: an initial phase of glomerular hyperfiltration and structural change within the glomerular basement membrane precedes the appearance of small quantities of albumin in the urine, which in turn precedes overt proteinuria and loss of filtration capacity [4,5].

Microalbuminuria — urinary albumin excretion above the normal range but below the threshold detectable by conventional dipstick — marks this incipient stage. It is defined as an albumin excretion rate of 30–300 mg/24 hours, or an albumin–creatinine ratio (ACR) of 2.5–25 mg/mmol (30–300 mg/g) in males and 3.5–25 mg/mmol in females, the sex difference reflecting lower creatinine excretion in females [3,4,6].

Loss of the anionic charge barrier of the glomerular basement membrane, mediated in part by inhibition of N-deacetylase and by accumulation of advanced glycation end-products, together with systemic endothelial dysfunction as proposed in the Steno hypothesis, explains why albumin leak accompanies both renal and cardiovascular injury [6,7,8]. Consistent with this, microalbuminuria predicts not only progression to overt nephropathy but also excess cardiovascular morbidity [9,10].

Reported prevalence in young people with T1DM varies widely, from about 6% to over 60%, reflecting differences in ethnicity, disease duration, pubertal status, metabolic control and — importantly — the rigour of the sampling protocol [11–15]. Between 40% and 50% of children demonstrate transient or intermittent albuminuria that does not progress, which is why a single positive screen cannot be equated with persistent microalbuminuria [5,11]. Current guidance recommends beginning annual screening at 11 years of age, or after two to five years of disease duration [1,16]. Whether this threshold is appropriate for children in resource-limited settings, where glycaemic control is frequently suboptimal, is uncertain: early-onset microalbuminuria within five years of diagnosis has been described in several cohorts [13].

Screening by spot urine ACR is inexpensive, non-invasive, reproducible and feasible in outpatient practice, and identification of microalbuminuria opens a therapeutic window during which intensified glycaemic control, blood-pressure management and renin–angiotensin blockade can retard or reverse progression [1,16,17]. Despite this, paediatric data from India remain sparse compared with the adult literature. The present study was therefore undertaken to assess the occurrence of persistent microalbuminuria and its associated risk factors in children with T1DM attending a tertiary paediatric centre in South India.

**Aim and Objective:** To assess microalbuminuria and the risk factors associated with it in children with type 1 diabetes mellitus.

## Materials and Methods

This was a hospital-based prospective observational study conducted in the Department of Paediatrics, Indira Gandhi Institute of Child Health, Bengaluru, a tertiary-level paediatric referral hospital in Karnataka, India. The study was carried out over a period of 18 months among children attending the paediatric diabetes and nephrology outpatient clinics. The source population comprised approximately 440 children with T1DM registered at the centre.

**Participants:** Children aged 1–18 years with an established diagnosis of T1DM made according to ISPAD criteria, and with a disease duration exceeding two years, were eligible. Children with pre-existing renal disease and those on long-term medications capable of confounding albuminuria or renal function — including chemotherapeutic agents, corticosteroids and angiotensin-converting enzyme inhibitors — were excluded. Consecutive eligible children were enrolled after written

informed consent from a parent or guardian and assent from the child where age-appropriate.

**Sample Size:** The required sample size was calculated using the formula for a finite population, assuming an expected proportion of microalbuminuria of 12%, a relative precision of 10% and a confidence level of 95% (Z = 1.96), applied to a source population of 440. This yielded 37.14 children; after adding 10% for anticipated non-response, the minimum required sample size was 45. Seventy-two children were ultimately recruited and analysed.

**Data Collection:** A structured proforma was used to record age, sex, age at diagnosis, duration of T1DM, family history of diabetes, past episodes of diabetic ketoacidosis (DKA) and the prevailing insulin regimen. Height and weight were measured using standard techniques and body mass index (BMI) was calculated and categorised, with BMI z-scores derived for age and sex. Blood pressure was recorded with a mercury sphygmomanometer using an age-appropriate paediatric cuff after the child had rested for at least 10 minutes.

**Assessment of Microalbuminuria:** Screening was performed by measurement of the urinary ACR in at least three spot urine specimens collected over a period of three to six months. Early-morning midstream samples were collected at consecutive clinic visits into clean, dry containers and analysed within two hours of voiding. Urine was first screened using Akon Lab urine analysis reagent strips; urinary albumin and creatinine were then quantified by the immunoturbidimetric method on a COBAS 6000 analyser. Microalbuminuria was defined as an ACR of 30–300 µg/mg and was classified as persistent when present in at least two of the three specimens. Children positive on only one specimen were classified as having transient microalbuminuria. Samples obtained during febrile illness, after vigorous exertion, or in the presence of urinary tract infection were deferred.

**Biochemical Assessment:** HbA1c was measured by turbidimetric inhibition immunoassay on haemolysed whole blood at each of the three visits. Glycaemic control was categorised as good (HbA1c < 7.5%), fair (7.5–9%) or poor (> 9%). Blood urea and serum creatinine were measured as markers of renal function, and the estimated glomerular filtration rate (eGFR) was calculated using the Schwartz formula [18].

**Statistical Analysis:** Data were entered into a Microsoft Excel spreadsheet and analysed using SPSS. Categorical variables were expressed as frequencies and proportions and compared using the chi-square test; continuous variables were expressed as mean ± standard deviation and compared using ANOVA. A p value < 0.05 was taken as statistically significant.

**Ethics:** The study protocol was approved by the Institutional Ethics Committee, Indira Gandhi Institute of Child Health, Bengaluru (No. IGICH/ACA/EC/6/2022-23, dated 19 July 2022). Confidentiality of participant data was maintained throughout.

## Results

A total of 72 children with T1DM were enrolled and analysed.

### Baseline Characteristics

**Table 1: Demographic and clinical profile of children with T1DM (n = 72)**

| Variable | Category | n | % |
|---|---|---|---|
| Age (years) | < 6 | 9 | 12.5 |
| | 6–12 | 23 | 31.9 |
| | 12–18 | 40 | 55.6 |
| Sex | Male | 29 | 40.3 |
| | Female | 43 | 59.7 |
| Duration of T1DM (years) | 2–5 | 48 | 66.7 |
| | 5–10 | 16 | 22.2 |
| | > 10 | 8 | 11.1 |
| Family history of diabetes | Present | 20 | 27.8 |
| | Absent | 52 | 72.2 |
| History of DKA | Present | 26 | 36.1 |
| | Absent | 46 | 63.9 |

Mean age of the cohort was 12 ± 3.7 years and mean duration of diabetes was 6 ± 3.1 years. More than half the children (55.6%) were adolescents aged 12–18 years, and only 12.5% were below six years of age. There was a clear female preponderance, with a male-to-female ratio of 1:1.48. Two-thirds of the cohort (66.7%) had been diabetic for between two and five years, and only 11.1% for more than a decade. A family history of diabetes was elicited in just over a quarter of children, and about one-third had experienced at least one prior episode of DKA.

### Glycaemic control and insulin regimen

**Table 2: Glycaemic control and insulin regimen in children with T1DM (n = 72)**

| Variable | Category | n | % |
|---|---|---|---|
| Glycaemic control | Good (HbA1c < 7.5%) | 8 | 11.1 |
| | Fair (HbA1c 7.5–9%) | 13 | 18.1 |
| | Poor (HbA1c > 9%) | 51 | 70.8 |
| Insulin regimen (basal–bolus) | Regular insulin + glargine | 45 | 62.5 |
| | Regular insulin + NPH | 27 | 37.5 |

All children were on a basal–bolus regimen. Glycaemic control was poor in the great majority: only 11.1% achieved an HbA1c below 7.5%, while 70.8% had values above 9%. A long-acting analogue (glargine) formed the basal component in 62.5% of children, the remainder using NPH insulin.

**Occurrence of Microalbuminuria**

**Table 3: Persistent microalbuminuria in children with T1DM (n = 72)**

| Persistent microalbuminuria | n | % |
|---|---|---|
| Present | 6 | 8.3 |
| Absent | 66 | 91.7 |

Persistent microalbuminuria, defined by a raised ACR in at least two of three specimens, was documented in 6 of 72 children (8.3%). In addition, 2 children (2.8%) had an isolated elevated ACR on the first specimen that normalised on the second and third, and 1 child (1.4%) who was normoalbuminuric on the first two visits had an isolated elevation on the third — giving a transient microalbuminuria rate of 4.2%, which underlines the necessity of serial rather than single-sample testing.

**Comparison of normoalbuminuric and microalbuminuric children**

**Table 4: Socio-demographic and clinical characteristics by albuminuria status**

| Variable | Normoalbuminuria (n = 66) | Microalbuminuria (n = 6) | p value |
|---|---|---|---|
| **Age group** | | | 0.28 |
| < 6 years, n (%) | 8 (12.1) | 1 (16.7) | |
| 6–12 years, n (%) | 23 (34.8) | 0 (0) | |
| 12–18 years, n (%) | 35 (53.0) | 5 (83.3) | |
| Mean age ± SD (years) | 11.8 ± 3.6 | 13.5 ± 3.9 | |
| **Sex** | | | 0.21 |
| Female, n (%) | 38 (57.6) | 5 (83.3) | |
| Male, n (%) | 28 (42.4) | 1 (16.7) | |
| **Family history of diabetes** | | | 0.11 |
| Present, n (%) | 20 (30.3) | 0 (0) | |
| **Age at diagnosis**, mean ± SD (years) | 6.6 ± 3.9 | 6.7 ± 2.4 | 0.97 |
| **Duration of T1DM** | | | 0.21 |
| 2–5 years, n (%) | 46 (69.7) | 2 (33.3) | |
| 5–10 years, n (%) | 13 (19.7) | 3 (50.0) | |
| > 10 years, n (%) | 7 (10.6) | 1 (16.7) | |
| Mean ± SD (years) | 5.2 ± 3.0 | 6.8 ± 3.2 | 0.48 |
| **History of DKA**, n (%) | 24 (36.4) | 2 (33.3) | 0.88 |
| **BMI category** | | | 0.39 |
| Underweight, n (%) | 42 (63.6) | 3 (50.0) | |
| Normal, n (%) | 23 (34.8) | 3 (50.0) | |
| Overweight, n (%) | 1 (1.6) | 0 (0) | |
| Mean BMI ± SD (kg/m²) | 17.8 ± 2.9 | 18.8 ± 2.5 | 0.32 |
| **Insulin regimen** | | | 0.12 |
| Regular + glargine, n (%) | 43 (65.2) | 2 (33.3) | |
| Regular + NPH, n (%) | 23 (34.8) | 4 (66.7) | |

Children with microalbuminuria were on average older (13.5 ± 3.9 vs 11.8 ± 3.6 years) and had a longer duration of diabetes (6.8 ± 3.2 vs 5.2 ± 3.0 years), and five of the six were adolescents; none of these differences reached statistical significance in this small subgroup. Notably, two of the six microalbuminuric children (33.3%) had a disease

duration of only two to five years, indicating that early-onset albuminuria is not confined to long-standing disease. Female sex, family history of diabetes, age at diagnosis, previous DKA, BMI and the type of basal insulin were not significantly associated with albuminuria status.

**Glycaemic control by albuminuria status**

**Table 5: HbA1c across three visits by albuminuria status**

| HbA1c | Normoalbuminuria (n = 66) | Microalbuminuria (n = 6) | p value |
|---|---|---|---|
| **Visit 1** | | | 0.33 |
| Good (< 7.5%), n (%) | 9 (13.6) | 0 (0) | |
| Fair (7.5–9%), n (%) | 13 (19.7) | 0 (0) | |
| Poor (> 9%), n (%) | 44 (66.7) | 6 (100) | |
| Mean ± SD (%) | 9.7 ± 2.1 | 13.5 ± 3.5 | |
| **Visit 2** | | | < 0.001 |
| Good (< 7.5%), n (%) | 11 (16.7) | 0 (0) | |
| Fair (7.5–9%), n (%) | 15 (22.7) | 1 (16.7) | |
| Poor (> 9%), n (%) | 40 (60.6) | 5 (83.3) | |
| Mean ± SD (%) | 9.7 ± 2.1 | 13.5 ± 3.5 | |
| **Visit 3** | | | 0.02 |
| Good (< 7.5%), n (%) | 6 (9.1) | 0 (0) | |
| Fair (7.5–9%), n (%) | 16 (24.2) | 0 (0) | |
| Poor (> 9%), n (%) | 44 (66.7) | 6 (100) | |
| Mean ± SD (%) | 9.8 ± 1.9 | 11.6 ± 1.1 | |
| **Mean of all three visits (%)** | 9.7 ± 2.0 | 12.8 ± 2.7 | < 0.001 |

Every child with persistent microalbuminuria (6/6, 100%) had poor glycaemic control at the first and third assessments. Averaged across all three visits, mean HbA1c was 3.1 percentage points higher in the microalbuminuric group, a difference that was highly significant ($p < 0.001$). Poor glycaemic control was thus the only variable in this study that showed a statistically significant association with persistent microalbuminuria.

**Blood pressure and renal parameters**

**Table 6: Blood pressure and renal parameters by albuminuria status (mean ± SD)**

| Parameter | Normoalbuminuria (n = 66) | Microalbuminuria (n = 6) | p value |
|---|---|---|---|
| Systolic BP (mmHg) | 105 ± 5.66 | 102 ± 5.66 | 0.09 |
| Diastolic BP (mmHg) | 68 ± 7.13 | 65.3 ± 7.0 | 0.08 |
| Blood urea (mg/dL) | 21.8 ± 6.5 | 21.8 ± 3.0 | 0.53 |
| Serum creatinine (mg/dL) | 0.5 ± 0.1 | 0.5 ± 0.2 | 0.21 |
| eGFR (mL/min/1.73 m²) | 124.0 ± 2.3 | 131.8 ± 2.8 | 0.47 |

No child in either group was hypertensive, and mean systolic and diastolic pressures were in fact marginally lower in the microalbuminuric group, differences that were not statistically significant. Blood urea and serum creatinine were identical between groups, confirming that excretory function was preserved. However, mean eGFR was approximately 8 mL/min/1.73 m² higher in microalbuminuric children, in keeping with the glomerular hyperfiltration that characterises the earliest stage of diabetic kidney disease.

**Discussion**

In this prospective cohort of 72 Indian children with T1DM of more than two years' duration, persistent microalbuminuria was present in 8.3%. This figure sits comfortably within the range reported by comparable paediatric series: 6.2% among 65 Kenyan children [11], 9.3% among 471 North American youth [15] and 12.1% in a Kuwaiti cohort of 302 children [13]. It is markedly lower than the 60% reported from Port Harcourt, Nigeria [12], and the 77.5% reported from Alexandria, Egypt [14]. Much of this heterogeneity is methodological. Both of the high-prevalence studies relied on a smaller number of urine specimens, and neither required confirmation across serial samples. In our cohort, an additional 4.2% of children had an isolated raised ACR that normalised on repeat testing; had we accepted a single positive specimen, our reported prevalence would have more than doubled.

Transient and intermittent albuminuria is well recognised in young people with T1DM, occurring in up to 40–50% of some series, and does not necessarily herald progressive nephropathy [5,15]. Serial confirmation is therefore not a refinement but a requirement.

The single significant risk factor identified was poor glycaemic control. All six microalbuminuric children had an HbA1c above 9%, and the mean

value across three visits was 12.8 ± 2.7% compared with 9.7 ± 2.0% in normoalbuminuric children ($p < 0.001$). This mirrors the findings of Al-Eisa et al. (13.8 ± 1.5% vs 9.3 ± 2.0%) [13], Omar et al., who found 88.8% of microalbuminuric children to be poorly controlled [14], and Poovazhagi et al. from Chennai (10.9 ± 2.3% vs 9.2 ± 1.6%; $p = 0.004$) [19]. The mechanistic basis is well established: sustained hyperglycaemia drives mesangial expansion, thickening of the glomerular basement membrane and accumulation of advanced glycation end-products, while inhibition of N-deacetylase reduces heparan sulphate synthesis and dissipates the anionic charge barrier, allowing albumin to escape into the filtrate [7,8,20]. The corollary — demonstrated by the DCCT/EDIC cohort — is that intensification of insulin therapy substantially reduces the incidence of albuminuria [21].

Two findings deserve particular emphasis. First, one-third of our microalbuminuric children had been diabetic for only two to five years. Al-Eisa et al. similarly found that 66% of their microalbuminuric children had disease duration under five years [13].

This argues against deferring screening until an arbitrary duration threshold has been crossed, and supports commencing ACR surveillance close to diagnosis, particularly where metabolic control is known to be poor. Second, eGFR was higher in the microalbuminuric group (131.8 vs 124.0 mL/min/1.73 m²), replicating the pattern seen by Al-Eisa et al. (204 vs 182.5 mL/min/1.73 m²) [13]. Glomerular hyperfiltration, driven by afferent arteriolar vasodilatation and raised intraglomerular pressure, precedes and predicts structural injury [4,22]. Its presence alongside normal urea and creatinine is a reminder that conventional excretory markers are insensitive to early DKD.

Unlike several other series, we found no association between microalbuminuria and blood pressure; indeed mean pressures were marginally lower in the microalbuminuric group. Al-Eisa et al. [13], Jawad et al. [23] and Ramaphane et al. [24] all reported higher systolic pressures in microalbuminuric children.

The discrepancy is plausibly explained by our small microalbuminuric subgroup, the low BMI of our cohort (63.6% of normoalbuminuric children were underweight) and the young age at which hypertension has not yet supervened. Similarly, sex, family history, prior DKA and BMI showed no significant association, though the female preponderance among microalbuminuric children (83.3%) is consistent with the female excess reported by Omar et al. [14] and Ramaphane et al. [24].

**Limitations.** The single-centre design, modest sample size and — critically — the small number of microalbuminuric children (n = 6) limit statistical power to detect associations of moderate effect size, and preclude multivariable adjustment. Pubertal staging was not formally assessed despite its known influence on insulin resistance and albumin excretion [16]. Longitudinal follow-up beyond the study period was not undertaken, so progression to overt nephropathy could not be assessed. Larger multicentre cohorts with longer follow-up are needed to confirm these observations in the Indian paediatric population.

## Conclusion

Persistent microalbuminuria was present in 8.3% of children with type 1 diabetes mellitus, and glomerular hyperfiltration accompanied it while urea and creatinine remained normal. Importantly, microalbuminuria was not restricted to children with more than five years of disease: a third of affected children had been diabetic for only two to five years.

Poor glycaemic control (HbA1c > 9%) was the only statistically significant risk factor identified, whereas age, sex, family history, previous ketoacidosis, BMI and blood pressure were not. Because a single positive specimen substantially overestimates the burden of disease, screening should rest on serial early-morning albumin–creatinine ratios. Taken together, these findings support initiating microalbuminuria screening at or soon after the diagnosis of T1DM rather than deferring it, and prioritising aggressive optimisation of glycaemic control, so that progression to diabetic nephropathy and end-stage renal disease may be prevented while renal injury remains reversible.

## References

1. American Diabetes Association. Standards of medical care in diabetes—2022. Diabetes Care. 2022;45(Suppl 1):S1–264.
2. Maahs DM, West NA, Lawrence JM, Mayer-Davis EJ. Epidemiology of type 1 diabetes. Endocrinol Metab Clin North Am. 2010;39(3):481–97.
3. Alicic RZ, Rooney MT, Tuttle KR. Diabetic kidney disease: challenges, progress, and possibilities. Clin J Am Soc Nephrol. 2017;12(12):2032–45.
4. Drummond K, Mauer M; International Diabetic Nephropathy Study Group. The early natural history of nephropathy in type 1 diabetes: II. Early renal structural changes in type 1 diabetes. Diabetes. 2002;51(5):1580–7.
5. Singh A, Satchell SC. Microalbuminuria: causes and implications. Pediatr Nephrol. 2011;26(11):1957–65.

6. Khosla N, Sarafidis PA, Bakris GL. Microal buminuria. Clin Lab Med. 2006;26(3):635–53.
7. Waghmare P, Goswami K. Microalbuminuria: a mere marker or an ominous sign? J Assoc Physicians India. 2016;64(3):61–5.
8. Satchell SC, Tooke JE. What is the mechanism of microalbuminuria in diabetes: a role for the glomerular endothelium? Diabetologia. 2008;51(5):714–25.
9. Mogensen CE. Microalbuminuria predicts clinical proteinuria and early mortality in maturity-onset diabetes. N Engl J Med. 1984;310(6):356–60.
10. Lydakis C, Lip GY. Microalbuminuria and cardiovascular risk. QJM. 1998;91(6):381–91.
11. Amolo P, Mbori-Ngacha D, Were F, Mungai L. Prevalence of microalbuminuria in children and adolescents with type 1 diabetes mellitus. Int J Endocrinol Diabetes. 2022;5(2):1–7.
12. Yarhere IE, Jaja T, Anolue M. Microalbuminuria in type 1 diabetes mellitus children in University of Port Harcourt Teaching Hospital, Nigeria. Pan Afr Med J. 2020;36:161.
13. Al-Eisa A, Al-Hajri A, Al-Shuaib S, Al-Abdul Razzak D, Al-Basiri I. Early-onset microalbu uminuria in children with type 1 diabetes in Kuwait. Curr Pediatr Res. 2017;21(2):254–9.
14. Omar MA, Rezk MM, El-Kafoury AA, Kandil MS. Microalbuminuria and glycated hemoglobin in children with type 1 diabetes mellitus. Alexandria J Med. 2015;51(1):83–8.
15. Alleyn CR, Volkening LK, Wolfson J, Rodriguez-Ventura A, Wood JR, Laffel LM. Occurrence of microalbuminuria in young people with type 1 diabetes: importance of age and diabetes duration. Diabet Med. 2010;27(5):532–7.
16. Donaghue KC, Marcovecchio ML, Wadwa RP, Chew EY, Wong TY, Calliari LE, et al. Microvascular and macrovascular complications in children and adolescents. Pediatr Diabetes. 2018;19(Suppl 27):262–74.
17. Silverstein J, Klingensmith G, Copeland K, Plotnick L, Kaufman F, Laffel L, et al. Care of children and adolescents with type 1 diabetes: a statement of the American Diabetes Assoc iation. Diabetes Care. 2005;28(1):186–212.
18. Schwartz GJ, Work DF. Measurement and estimation of GFR in children and adolescents. Clin J Am Soc Nephrol. 2009;4(11):1832–43.
19. Poovazhagi V, Senguttuvan P, Padmaraj R. Prevalence of microalbuminuria in children with type 1 diabetes mellitus. Pediatr Oncall. 2012;9(7).
20. Sheth JJ. Diabetes, microalbuminuria and hypertension. Clin Exp Hypertens. 1999;21(1–2):61–8.
21. Nathan DM, Cleary PA, Backlund JY, Genuth SM, Lachin JM, Orchard TJ, et al. Intensive diabetes treatment and cardiovascular disease in patients with type 1 diabetes. N Engl J Med. 2005;353(25):2643–53.
22. Jefferies C, Nakhla M, Derraik JGB, Gunn AJ, Daneman D, Cutfield WS. Preventing microvascular complications in children with type 1 diabetes. Pediatr Diabetes. 2015;16(4): 271–85.
23. Jawad K, Meaad K, Lamia M. Microalbuminuria in children and adolescents with type 1 diabetes mellitus. Med J Basrah Univ. 2017;25(2):5–11.
24. Ramaphane T, Gezmu AM, Tefera E, Gabaitiri L, Nchingane S, Matsheng-Samuel M, et al. Prevalence and factors associated with microalbuminuria in pediatric patients with type 1 diabetes mellitus at a large tertiary-level hospital in Botswana. Diabetes Metab Syndr Obes. 2021;14:4415–22.
25. Margeirsdottir HD, Larsen JR, Brunborg C, Overby NC, Dahl-Jørgensen K. High prevalence of cardiovascular risk factors in children and adolescents with type 1 diabetes: a population-based study. Diabetologia. 2008; 51(4):554–61.