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Original Research Article

Study Of Type 2 Diabetes Mellitus as a Threat Indicator for the Microalbuminuria

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Abstract:

Background: Type 2 Diabetes Mellitus (T2DM) is a chronic metabolic disorder characterized by persistent hyperglycemia and associated microvascular complications. Microalbuminuria is an early marker of diabetic nephropathy and endothelial dysfunction.

Aim: To evaluate T2DM as a threat indicator for microalbuminuria and to assess its association with glycemic control (HbA1c) and duration of diabetes.

Methodology: A hospital-based cross-sectional observational study was conducted on 50 T2DM patients aged ≥30 years. HbA1c was estimated using the Latex Agglutination Inhibition Method, and urinary microalbumin was measured by the immunoturbidimetric method. Statistical analysis was performed using SPSS version 19. Student's t-test and Pearson's correlation were applied, with $p < 0.05$ considered significant.

Results: Microalbuminuria was present in 40% of patients. Its prevalence increased with rising HbA1c levels, particularly >7.5%. A significant positive correlation was observed between microalbuminuria and HbA1c ($r = 0.61$, $p = 0.002$), and a strong correlation with duration of diabetes ($r = 0.78$, $p < 0.001$). Mean HbA1c was significantly higher in patients with microalbuminuria (8.52 ± 1.18%) than those without (7.46 ± 1.02%) ($p = 0.001$).

Conclusion: Poor glycemic control and longer duration of T2DM significantly predict microalbuminuria, highlighting the importance of early screening and strict metabolic control.

Keywords: Type 2 Diabetes Mellitus, Microalbuminuria, HbA1c, Diabetic Nephropathy, Glycemic Control, Duration of Diabetes.

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Introduction

One of the most problematic health issues of the 21st century is diabetes mellitus. Type 2 diabetes mellitus (T2DM) is also one of its forms and it has almost 85-95 percent of all the diabetes cases in developed nations. The prevalence is observed to be increasing among adolescents through old age with millions of people being affected worldwide. Unless timely intervention is conducted, glycemic control might be worsened and result in severe systemic effects [1]. The alarming rate of incidence, chronicity, and the long-term effects of the disease all predispose T2DM as a significant burden to the population.

Diabetes Mellitus type 2 is a metabolic disorder with polygenic nature, which leads to a state of unremitting hyperglycemia due to either absolute or relative deficiency of insulin [2]. The pathogenesis is complicated by interplay between genetic factors and environmental factors including sedentary lifestyle, overweight, and diet. Contrary to Type 1 diabetes, T2DM patients do not manifest any symptoms in the long term despite persistent hyperglycemia. Due to this silent progress, the disease is often diagnosed in later stages when the complications are already chronic. This non-symptomatic progression highlights the significance of screening and identification of predictive factors that could be used to indicate the imminent damage to the organs.

Diabetes is a chronic disease that in its essence, long-term hyperglycemia has deleterious effects on both microvascular and macrovascular structures. Persistent high sugar in the blood causes small vessels that serve important body parts like kidneys, eyes and peripheral nerves, and large vessels that lead to cardiovascular morbidity [3]. Therefore, diabetic patients are at greater risks of complications with retina (retinopathy), kidney (nephropathy) and

cardiovascular system. One of these, diabetic nephropathy has remained one of the most serious morbidity and mortality causes as it progresses to chronic kidney disease and end-stage renal failure.

Direct toxicity to glucose may occur at the cellular and molecular level. Lorenzi et al [4] found that endothelial cells of humans which were repeatedly subjected to elevated levels of glucose have considerable functional defects which could not be explained by the activity of the polyol pathway alone. These abnormalities are characterized by cell replication and maturation abnormalities, with DNA damage evidence. These results suggest that hyperglycemia triggers intrinsic cellular dysfunction irrespective of reactive metabolism. Moreover, glucose raises induce a greater expression and production of extra cellular matrix of collagen, fibronectin and laminin. This improved matrix production is one reason why the kidneys under diabetic conditions undergo structural changes.

Moreover, high glucose levels in mesangial cells provoke the production and the release of transforming growth factor-beta (TGF- β) a cytokine that is unique in that it increases the synthesis of the matrix and, at the same time, inhibits its destruction. This causes an imbalance thus resulting in an over-accumulation of the extracellular matrix within the glomerulus, which causes glomerulosclerosis and eventual renal damage. There has been a tight association of abnormal endothelial cell activity in the pathogenesis of diabetic nephropathy, therefore. Not only does endothelial dysfunction disturbs the normal filtration processes but also hastens the process of vascular injury in the renal microcirculation.

The poor glycemic control has been pointed as one of the crucial determinants of diabetic nephropathy among other risk factors of the disease. The risk of developing microvascular complications is high because of persistent hyperglycemia, which is evidenced by high levels of glycated hemoglobin (HbA1c). Poor glycemic control is known to be one of the greatest risk factors in the occurrence of microalbuminuria, which accelerates the process of renal disease [5]. Hence, it is both important to monitor the glycemic status to gain control over metabolism and predict the long-term renal outcomes.

Microalbuminuria is described as the excretion of albumin in the urine of between 20-300 mg daily [6]. It is a sensitive and early clinical indicator of dysfunction of renal endothelial and can be regarded as one of the first manifestations of diabetic nephropathy [7]. Micro albumin in the urine indicates glomerular hyper-permeability and premature glomerular damage. Notably, microalbuminuria is the earliest sign of proteinuria and may be detected long before the onset of renal impairment that is evident in clinical terms.

The most frequent cause of chronic renal damage is diabetic nephropathy which is responsible in about thirty percent in the Indian population. It has a complex etiopathogenesis which is a polygenic interaction with a contribution of genetic susceptibility and environmental factors [8]. Since the burden of diabetes and its complications is very high, there is an urgent need to identify the people at risk as early as possible. Microalbuminuria is not only a predictor of progressive nephropathy, but also an indicator of overall endothelial malfunction, which can be associated with a higher cardiovascular risk.

Early therapeutic intervention is also among the great benefits associated with the detection of microalbuminuria. Early diagnosis of diabetic nephropathy through proper management measures, such as stringent glycemic control, changes in lifestyle and pharmacological treatment can prevent or delay the pathophysiology of diabetic nephropathy to irreversible renal failure. Microalbuminuria is a threat indicator, which presents far earlier than overt nephropathy and so is a useful warning sign, as it indicates that the management needs to be more vigorous prior to structural damage.

Microvascular complications are also influenced by the length of the period of diabetes mellitus. The cumulative vascular damage with prolonged exposure to hyperglycemia raises the risk of renal involvement. The time of diabetes and the levels of HbA1c are usually measured in clinical practice in order to determine the probability of complications. Nevertheless, they could be more effective as predictors in regular screening procedures by creating a clear relationship between these parameters and microalbuminuria.

In this regard, the current research paper was conducted to assess Type 2 diabetes mellitus as a risk factor of microalbuminuria. In particular, the focus of the study is to evaluate the relationship between microalbuminuria and the length of diabetes mellitus and glycemic control using HbA1c on random patients with Type 2 diabetes. The study will also help in enhancing preventive methods to combat diabetic nephropathy by investigating this correlation.

Methodology

Study Design: The present study was designed as a hospital-based cross-sectional observational study conducted to evaluate Type 2 Diabetes Mellitus as a potential threat indicator for the development of microalbuminuria. The study aimed to assess the association between glycemic control, as indicated by HbA1c levels, and urinary microalbumin levels among diagnosed diabetic patients.

Study Area: The study was conducted in the Department of Biochemistry, Anugrah Narayan Magadh Medical College, Gaya, Bihar, India.

Study Duration: The study was carried out over a period of six months from March 2025 to August 2025.

Sample Size: A total of 50 patients diagnosed with Type 2 Diabetes Mellitus were included in the study. The sample size was determined based on feasibility within the study duration and availability of eligible patients attending the Medicine OPD.

Study Population: The study population comprised patients diagnosed with Type 2 Diabetes Mellitus attending the Medicine OPD during the study period. Diagnosis was based on clinical history, physical examination, and relevant biochemical investigations. Patients were selected using a random sampling method to minimize selection bias.

Data Collection: Detailed clinical history including duration of diabetes, treatment history, and associated complications was recorded using a structured proforma. After obtaining informed consent, fasting venous blood samples were collected under aseptic precautions for estimation of HbA1c. HbA1c was measured by the Latex Agglutination Inhibition Method using RANDOX Kit – HA 3830 in a Randox Daytona autoanalyzer. For assessment of microalbuminuria, the first morning midstream urine sample was collected in a clean, dry container after discarding the initial and terminal portions of urine. Urinary microalbumin was estimated by the immunoturbidimetric method using RANDOX Kit – MA 2426 in a Randox autoanalyzer. All laboratory procedures were performed according to manufacturer guidelines.

Inclusion Criteria

- Patients diagnosed with Type 2 Diabetes Mellitus
- Age ≥ 30 years
- Patients willing to participate and provide informed consent
- Patients attending Medicine OPD during the study period

Exclusion Criteria

- Patients with Type 1 Diabetes Mellitus
- Patients with known chronic kidney disease unrelated to diabetes
- Patients with urinary tract infection
- Pregnant women
- Patients with severe systemic illness

Procedure: Eligible patients were identified in the Medicine OPD and informed about the purpose and objectives of the study. After obtaining consent, demographic and clinical details were recorded. Fasting blood samples and first morning urine samples were collected and analyzed for HbA1c and urinary microalbumin levels respectively. The obtained results were documented systematically for further statistical evaluation to determine the association between glycemic status and microalbuminuria.

Statistical Analysis: The collected data were entered and analyzed using SPSS software version 19. Results were expressed as mean ± standard deviation. Student's t-test was applied to compare mean values between relevant groups, and Pearson's correlation coefficient was used to assess the relationship between HbA1c levels and urinary microalbumin levels. A p-value of less than 0.05 was considered statistically significant."

Result

Table 1 shows that the prevalence of microalbuminuria increased progressively with higher HbA1c levels among the 50 patients. In the <6.5% group (n = 8), only 1 patient had microalbuminuria, whereas in the 6.5–7.0% group (n = 10), 2 patients were affected. The proportion further increased in the 7.1–7.5% group (n = 12), with 4 cases of microalbuminuria. Notably, in patients with HbA1c >7.5% (n = 20), 13 had microalbuminuria, representing the highest burden. Overall, 20 patients (40%) had microalbuminuria, while 30 (60%) did not, indicating a clear association between poor glycemic control and the presence of microalbuminuria.

Table 1: Distribution of Microalbuminuria according to HbA1c levels (N = 50)

HbA1c (%)	Total Patients (n)	Microalbuminuria Present (n)	Microalbuminuria Absent (n)
< 6.5	8	1	7
6.5 – 7.0	10	2	8
7.1 – 7.5	12	4	8
> 7.5	20	13	7
Total	**50**	**20 (40%)**	**30 (60%)**

Table 2 shows that the prevalence and severity of microalbuminuria increased with longer duration of diabetes among the 50 participants. In patients with duration <5 years (n = 18), only 1 case of mild microalbuminuria was observed, with no moderate or severe cases. Among those with 5–10 years of diabetes (n = 14), 3 had mild, 2 had moderate, and 1 had severe microalbuminuria. In the 10–15 years group (n = 10), the distribution shifted toward greater severity, with 2 mild, 3 moderate, and 2 severe cases. Patients with duration >15 years (n = 8) showed the highest severity, including 1 mild, 2 moderate, and 3 severe cases. Overall, of the 50 patients, 7 had mild, 7 had moderate, and 6 had severe microalbu-

minuria, indicating a clear trend of increasing severity with longer duration of diabetes.

Table 2: Distribution of Microalbuminuria according to Duration of Diabetes (N = 50)

Duration of Diabetes (Years)	Total Patients (n)	Mild (20–50 mg/dl)	Moderate (50–100 mg/dl)	Severe (100–300 mg/dl)
< 5 years	18	1	0	0
5 – 10 years	14	3	2	1
10 – 15 years	10	2	3	2
> 15 years	8	1	2	3
Total (Positive cases)	**50**	**7**	**7**	**6**

Table 3 demonstrates a significant association between microalbuminuria and both HbA1c levels and duration of diabetes among the study participants (N = 50). The mean HbA1c was 8.04 ± 1.26%, showing a statistically significant correlation with microalbuminuria ($p = 0.002$) and a moderate positive correlation ($r = 0.61$). Additionally, the mean duration of diabetes was 7.12 ± 4.18 years, which showed a highly significant association ($p < 0.001$) and a strong positive correlation with microalbuminuria ($r = 0.78$). These findings suggest that poor glycemic control and longer disease duration are strongly linked to the presence of microalbuminuria.

Table 3: Assessment of Different Parameters and Their Significance with Microalbuminuria (N = 50)

Parameters	Mean ± SD	Significance by Student t-test	r value
Microalbuminuria and HbA1c (%)	8.04 ± 1.26	$p = 0.002$	0.61
Microalbuminuria and Duration (years)	7.12 ± 4.18	$p < 0.001$*	0.78

Table 4 shows that the mean HbA1c level was significantly higher in patients with microalbuminuria (8.52 ± 1.18%) compared to those without microalbuminuria (7.46 ± 1.02%), and this difference was statistically significant ($p = 0.001$). This indicates that poorer glycemic control is strongly associated with the presence of microalbuminuria.

Table 4: Comparison of Mean HbA1c in Patients with and Without Microalbuminuria

Parameter	Microalbuminuria Present (n=20)	Microalbuminuria Absent (n=30)	p-value
HbA1c (%) (Mean ± SD)	8.52 ± 1.18	7.46 ± 1.02	0.001*

Discussion

In the current study, 40 percent of patients with type 2 diabetes mellitus had microalbuminuria. This is a bit higher than the prevalence rates in previous studies that were quoted between 25-35 percent (Ghai et al., 1994; Patel et al., 1999) [9,10]. The findings that we have are however similar to the findings that were recorded by Varghese et al. (2001) [11] who reported prevalence rate of about 36% among patients visiting a diabetes center in southern India. Likewise, Mather et al. (1998) [12] also found similar frequencies of various ethnic groups indicating that microalbuminuria is a frequent complication in a broad dissemination of the population. The marginally increased frequency in our study could be explained by the fact that our subjects had poorer glycemic control or longer period of disease."

One of the main results of our research was that the level of microalbuminuria was gradually increasing in direct proportion to the level of HbA1c. Microalbuminuria was found in 1 of the 8 patients with hbA1c less than 6.5 percent only and 13 of the 20 patients with hbA1c above 7.5 percent. The average HbA1c in patients with and without microalbuminuria was significantly greater (8.52 ± 1.18 and 7.46 ± 1.02 respectively) and the relationship between the two groups was statistically significant ($p = 0.001$) yet exhibited moderate level of positive correlation ($r = 0.61$, $p = 0.002$). These are aligned with the results of Patil and Paunipagar (2019) [13] that showed that high levels of microalbuminuria and high levels of HbA1c levels were significantly related to patients with type 2 diabetes. Similarly, Idogun and Kasia (2011) [14] found that the mean levels of HbA1c were significantly higher in the patients who had microalbuminuria than in those who did not, which also supported the importance of poor glycemic control in causing renal involvement.

The fact that we have observed a threshold effect, where microalbuminuria rose significantly above a critical level when HbA1c rose above 7.5% is consistent with the hypothesis that persistent hyperglycemia above a critical range promotes glomerular damage. The same tendencies were characterized by Varghese et al. (2001) [11], according to which the microalbuminuria was independently linked to worsening of glycemic control. Nonetheless, there

are also reports on some studies that have shown opposite results. Huraib et al. (1995) [15] did not observe significant relationship between HbA1c with microalbuminuria in Saudi patients with non-insulin-dependent diabetes mellitus. On the same note, Afkhami-Ardekani et al. (2008) [16] noted that there was no statistically significant correlation between glycemic control and microalbuminuria in their cohort. Such discrepancies can be attributed to sample size variations, genetic predisposition, or lifestyles, or some other confounding variables like hypertension and lipid abnormalities.

The other significant detail of our research was that the duration of diabetes was strongly positively related to the presence and intensity of microalbuminuria. Duration of diabetes and microalbuminuria had a high correlation coefficient ($r = 0.78$, $p < 0.001$), and therefore, there was a strong relationship. Patients with 15 years and above duration of diabetes had higher proportion of moderate and severe microalbuminuria than patients with shorter duration of diabetes. This observation confirms the fact that cumulative exposure to hyperglycemia is central in the development of renal damage. Varghese et al. (2001) [11] and Mather et al. (1998) [12] also reported a similar observation and observed that longer period of diabetes enhanced the risk of microalbuminuria significantly. Huraib et al. (1995) [15] too proved the increase in the prevalence of diabetic nephropathy with the increase in disease duration although their analysis did not reveal any definite correlation with the level of HbA1c.

The progression pattern in the severity of diabetic nephropathy noted in our study also highlights the progressive nature of diabetic nephropathy. Mild microalbuminuria was observed among patients with shorter disease duration (less than 5 years) but moderate and severe microalbuminuria were observed among patients with longer disease duration. This gradation is similar to the results of Patel et al. (1999) [10] and Ghai et al. (1994) [9], which indicated that microalbuminuria is likely to develop to open nephropathy with continued metabolic insult. It is a biological possibility that this process occurs because of the pathophysiological processes of the involvement of advanced glycation end products, oxidative stress, and endothelial dysfunction (Bucala et al., 1991) [17].

In general, our research supports already available evidence that the poor glycemic control and the long-term presence of type 2 diabetes mellitus is an important indicator of threat of microalbuminuria development and progression. Although some studies have exhibited opposing findings on the relationship between HbA1c and micro albuminuria, most of the available evidence, including ours indicates a significant positive and clinically significant relationship. Timely microalbuminuria and tight glycemic control can thus be significant in prevention or postponement of the overt diabetic nephropathy and complications.

Conclusion

The current research concludes that Type 2 Diabetes Mellitus is a considerable threat marker to the phenomenon of microalbuminuria development. The results show that there is a distinct relationship between bad glycemic control and the presence of microalbuminuria whereby increasing the levels of HbA1c are strongly positively correlated with urinary albumin excretion. Also, the microalbuminuria severity was considerably associated with the diabetes period, which also points to the fact that the long-term development of this disease predisposes the risk of renal involvement. The mean level of HbA1c in patients with versus without microalbuminuria was relatively higher, which further indicated the contribution of persistent hyperglycemia to early diabetic nephropathy. On the whole, the analysis emphasizes the fact that poor glycemic level and the time of Type 2 Diabetes Mellitus are significant predictors of microalbuminuria, which explains the necessity to screen early and curb metabolism to avoid kidney problems.

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