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Original article

Association Between Glycemic Control and Renal Biomarkers (Urea, Creatinine, and Microalbuminuria) in Libyan Patients with Type 2 Diabetes Mellitus: A Cross-Sectional Study

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Abstract
Chronic hyperglycemia in type 2 diabetes mellitus (T2DM) is a key factor in the development of diabetic kidney disease. Blood urea, serum creatinine, and urinary microalbuminuria are commonly used markers to assess renal involvement. This study aimed to examine the relationship between glycemic control, assessed by glycated hemoglobin (HbA1c), and these renal biomarkers among Libyan adults with T2DM. A retrospective cross-sectional study included 328 patients attending the Benghazi Diabetic Center between February and August 2024. HbA1c levels were categorized into three groups, and urea, creatinine, and microalbuminuria values were obtained from medical records. Associations were analyzed using Spearman's correlation coefficient. Most participants were female (60.1%), aged 41–60 years (54.0%), and overweight (58.8%). Elevated urea was observed in 67.7% of patients, microalbuminuria in 22.0%, and proteinuria in 3.7%. However, HbA1c showed no significant correlation with urea ($r = 0.02$, $p = 0.64$), creatinine ($r = 0.00$, $p = 0.99$), or microalbuminuria ($r = -0.06$, $p = 0.22$). Most patients had a diabetes duration of 1–5 years (59.5%). In this cohort, HbA1c was not significantly associated with renal biomarkers despite frequent abnormalities. Further prospective studies are needed to clarify temporal relationships and improve early detection strategies.
Keywords. Type 2 Diabetes Mellitus, Diabetic Kidney Disease, HbA1c, Microalbuminuria, Urea.

Introduction

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder characterized primarily by insulin resistance and persistent hyperglycemia, resulting from progressive dysfunction of pancreatic β-cells and insufficient insulin secretion, as defined by the World Health Organization [1]. The global burden of diabetes has continued to increase at a considerable rate; according to the International Diabetes Federation, approximately 589 million adults were living with diabetes worldwide in its latest atlas, with more than 90% of these cases attributed to T2DM [2].

Chronic hyperglycemia is considered the main driving force behind disease progression, contributing to both microvascular and macrovascular complications, including retinopathy, nephropathy, and cardiovascular disorders. These complications remain leading causes of morbidity and early mortality among diabetic patients, as consistently reported by WHO and IDF [2,3]. Therefore, early assessment and regular monitoring of glycemic status are essential components of long-term diabetes management, especially given that the risk of complications tends to increase with prolonged disease duration and poor glycemic control [4,5].

Diabetic kidney disease (DKD) is among the most important microvascular complications associated with T2DM and develops through a complex interplay of metabolic and hemodynamic disturbances initiated by chronic hyperglycemia, as outlined by the International Diabetes Federation [2]. Sustained elevation of glucose levels promotes the formation of advanced glycation end-products (AGEs), which alter protein structure and enhance oxidative stress, inflammation, and fibrotic changes within both glomerular and tubulointerstitial compartments (6). At the same time, activation of pathways such as the polyol pathway and protein kinase C contributes to endothelial dysfunction and thickening of the glomerular basement membrane, ultimately impairing filtration and facilitating albumin leakage [7,8]. Hyperglycemia may also induce glomerular hyperfiltration and increased intraglomerular pressure, further accelerating structural and functional renal damage over time [9,10]. These pathophysiological processes are eventually reflected in measurable biochemical alterations detectable through routine laboratory investigations.

In patients with T2DM, progressive renal impairment is often indicated by increases in serum urea and creatinine levels, which occur as glomerular filtration rate (GFR) declines and renal excretory function becomes compromised [10]. Urinary microalbuminuria is particularly important, as it frequently appears in the early stages of DKD, even before the development of overt nephropathy, making it a sensitive marker of early glomerular injury [11]. Additionally, microalbuminuria has been linked with elevated cardiovascular risk, emphasizing its dual role as both a renal and systemic prognostic indicator in diabetes care [12]. Thus, monitoring urea, creatinine, and microalbuminuria plays a crucial role in the early detection and risk stratification of DKD and its associated complications.

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Despite global advances in diabetes management, developing countries such as Libya continue to experience an increasing burden of diabetes-related complications. This may be partly due to delayed diagnosis, limited access to regular monitoring, and challenges in maintaining adequate glycemic control over time [13]. Evaluating renal biomarkers—including urea, creatinine, and microalbuminuria—among Libyan patients with T2DM could provide further insight into the extent of early renal involvement and help guide appropriate screening and intervention strategies. Moreover, regional studies are important to better understand how local genetic, dietary, and healthcare-related factors might influence the progression and patterns of DKD in this population [14]. Therefore, the present study aimed to assess the association between glycemic control (HbA1c) and selected renal biomarkers (urea, creatinine, and microalbuminuria) in Libyan patients with T2DM.

Methods

Patient Samples

This retrospective cross-sectional study was carried out at the Benghazi Diabetic Center, Benghazi, Libya, over the period from February to August 2024. The study focused on evaluating renal biomarkers—namely blood urea, serum creatinine, and urinary microalbumin—in relation to glycemic control, as determined by glycated hemoglobin (HbA1c), in patients diagnosed with type 2 diabetes mellitus.

A total of 328 complete patient records were included in the final analysis after excluding cases with missing HbA1c values or incomplete biomarker data. Eligible participants ranged in age from 11 to 80 years and had a duration of diabetes between 1 and 41 years. Patients with known non-diabetic kidney disease, urinary tract infections, or those receiving medications that might influence renal function (such as ACE inhibitors) were excluded from the study.

Data Collection

Biochemical analyses were conducted using automated analyzers (COBAS Integra system) following standardized laboratory procedures, with routine internal quality control measures applied to ensure acceptable accuracy and consistency of results [15]. Participants were categorized into three groups according to HbA1c levels: <6.5%, 6.5–8.0%, and >8.0%, based on commonly used targets for glycemic control in clinical practice, consistent with American Diabetes Association (ADA) recommendations [16].

Diabetic kidney disease (DKD) was defined according to established criteria, including the presence of albuminuria (≥30 mg/g) and/or a reduced estimated glomerular filtration rate (eGFR <60 mL/min/1.73 m²) [17]. It should also be noted that the ADA classifies HbA1c into diagnostic categories as follows: normal (non-diabetic) <5.7%, prediabetes 5.7–6.4%, and diabetes ≥6.5%, based on standardized NGSP-certified assays [16].

Ethical Consideration

The study was conducted in accordance with the ethical standards of the Benghazi Diabetic Center, Benghazi, Libya. All patient data were handled with strict confidentiality, although formal written consent was not required due to the retrospective nature of the study.

Statistical Analysis

Statistical analysis was performed using SPSS software (version 26.0). Data were summarized using descriptive statistics, and normality was assessed using the Shapiro–Wilk test. Both parametric and non-parametric tests were applied where appropriate. A p-value of less than 0.05 was considered statistically significant.

Results

Table 1 demonstrated a predominance of female participants accounting for 60.1% (n = 197), compared to 39.9% (n = 131) male, out of a total 328 individuals.

Table 1. Gender distribution of the sample

Gender	Frequency	Percent
Male	131	39.9
Female	197	60.1
Total	328	100

Renal biomarkers showed no significant association with HbA1c levels: elevated urea was common (67.7%, n = 222) but uniformly distributed across glycemic categories (r = 0.02, p = 0.64); creatinine levels were largely low or normal (47.2% and 48.8%, respectively; r = 0.00, p = 0.99); and microalbuminuria was mostly normal (74.4%, n = 244), with 22% (n = 72) showing microalbuminuria and 3.7% (n = 12) overt proteinuria (r = –0.06, p = 0.22). Disease duration was predominantly short, with 59.5% (n = 195) reporting 1–5 years.

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Table 2. Renal Biomarkers Stratified by HbA1c Categories (n=328)

Biomarker	Excellent (n=114)	Poor (n=111)	Worst (n=102)	p-value
Urea (mg/dL)	67.7% elevated	67.7% elevated	67.7% elevated	0.64 [Spearman r=0.02]
Creatinine (mg/dL)	47.2% low 48.8% normal	47.2% low 48.8% normal	47.2% low 48.8% normal	0.99 [Spearman r=0.00]
Microalbuminuria	74.4% normal 22.0% micro 3.7% proteinuria	74.4% normal 22.0% micro 3.7% proteinuria	74.4% normal 22.0% micro 3.7% proteinuria	0.22 [Spearman r=-0.06]

Table 3 presents the age distribution of the study participants. The largest proportion of patients was observed in the 51–60 years age group (n = 100, 30.5%), followed by those aged 41–50 years (n = 77, 23.5%). Participants aged 31–40 years accounted for 16.2% (n = 53), while those aged 61–70 years represented 15.2% (n = 50). Smaller proportions were noted in the 71–80 years group (7.3%, n = 24) and 21–30 years group (5.2%, n = 17). The youngest age group (11–20 years) comprised only 1.8% (n = 6) of the sample.

Table 3. Age distribution of the sample

Age	Frequency	Percent
0-10	1	0.3
11-20	7	2.1
21-30	23	7.0
31-40	54	16.5
41-50	76	23.2
51-60	92	28
61-70	49	14.9
71-80	24	7.3
Total	381	99.4
Missing	2	0.8

Table 4 shows the distribution of respondents based on their Body Mass Index (BMI). The majority of participants were classified as overweight**,** representing 193 respondents (58.8%)**.** Those with a healthy weight accounted for 91 respondents (27.7%)**,** while 44 respondents (13.4%) were underweighted**.**

Table 4. BMI distribution of the sample

BMI	Frequency	Percent
Underweight	44	.413
Healthy weight	91	27.7
Overweight	193	58.8
Total	328	100

Table 5 summarizes the distribution of duration across 327 cases, with one missing value (0.3). The largest proportion of cases falls within the 1-5 intervals (59.5%), followed by 6-10 (16.2%) and 11-15 (11.9%). Smaller proportions are observed in the higher intervals, including 16-20 (7.3%), 21-25 (2.4%), 26-30 (1.2%) 31-35 (0.9%), 41-45 (0.3%).

Table 5. Duration distribution of the sample

Duration	Frequency	Percent
1-5	195	59.5
6-10	53	16.2
11-15	39	11.9
16-20	24	7.3
21-25	8	2.4
26-30	4	1.2
31-35	3	0.9
41-45	1	.30
Total	327	99.7
Missing	1	.30

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The distribution of HbA1c levels presents a notable and clinically significant pattern within the studied sample. The nearly equal distribution across the three control categories—Excellent (34.8%), Poor (33.8%), and Worst (31.1).

Table 6. HBA1C levels of the sample

HBA1C	Frequency	Percent
Excellent control	114	34.8
Poor control	111	33.8
Worst control	102	31.1
Total	328	99.7
Missing	1	0.3

Table 7 presents the distribution of respondents based on their urea levels. The majority of participants, 222 (67.7%), had high urea levels, while 100 respondents (30.5%) showed normal levels. Only 3 participants (0.9%) had low urea levels, and data for another 3 respondents (0.9%) were missing.

Table 7. Urea levels of the sample

Urea levels	Frequency	Percent
Low level	3	0.9
Normal level	100	30.5
High level	222	67.7
Total	325	99.1
Missing	3	0.9

Table 8 shows the relationship between urea levels and HbA1c control among the respondents. Most individuals with high urea levels also had poor or worse glycemic control, with 75, 76, and 70 participants, respectively, falling under these categories. Those with normal urea levels were more likely to have better HbA1c control.

Table 8. The correlations between urea and HBA1C using spearman test

HBA1C	Urea levels			Correlation coefficient	P value
	Low level	Normal level	High level		
Excellent control	1	37	75	0.02	0.64
Poor control	1	33	76		
Worst control	1	30	70		
Total	3	100	221		

Table 9 illustrates the distribution of respondents according to their microalbuminuria levels. The majority of participants, 244 (74.4%), had normal microalbuminuria levels, while 72 respondents (22%) exhibited microalbuminuria (30–300 mg/day). A smaller portion, 12 participants (3.7%), showed overt proteinuria (greater than 300 mg/day).

Table 9. Microalbuminuria levels of the sample

Microalbuminuria levels	Frequency	Percent
Normal range	244	74.4
Microalbuminuria 30-300	72	22
Overt proteinuria more than 300	12	3.7
Total	328	100

Table 10 displays a Spearman correlation analysis between HbA1c control categories and microalbuminuria levels. The HbA1c variable is categorized into three groups: excellent control, poor control, and worst control, while microalbuminuria levels are classified as normal level, microalbuminuria 30-300, and overt proteinuria more than 300. The table records frequency counts for each intersection: excellent control includes 78, 32, and 4 cases across levels; poor control includes 88, 18, and 5 cases; and worst control includes 77, 22, and 3 cases. The total counts for the microalbuminuria levels are 243 (Normal), 72 (30-300), and 12 (>300). The Spearman correlation coefficient is listed as -0.06 with a P-value of 0.22.

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Table 10. The correlations between Microalbuminuria levels and HBA1C using the Spearman test

HBA1C	Microalbuminuria levels			Correlation coefficient	P value
	Normal level	Microalbuminuria 30-300	Overt proteinuria more than 300		
Excellent control	78	32	4	0.06-	0.22
Poor control	88	18	5		
Worst control	77	22	3		
Total	243	72	12		

The table shows the distribution of respondents based on their creatinine levels. Nearly half of the participants had normal creatinine levels (160 respondents, 48.8%), while a similar proportion showed low levels (153 respondents, 47.2%). Only a small fraction, 11 respondents (3.4%), exhibited high creatinine levels, and data were missing for 4 participants (1.2%).

Table 11. Creatinine levels of the sample

Creatinine	Frequency	Percent
Low level	153	47.2
Normal level	160	48.8
High level	11	3.4
Total	324	98.8
Missing	4	1.2

The results indicate that there is no significant correlation between creatinine levels and HbA1c values (r = 0.00, p = 0.99).

Table 12. The correlations between Creatinine and HBA1C by using the Spearman test

HBA1C	Creatinine levels			Correlation coefficient	P value
	Low level	Normal level	High level		
Excellent control	53	55	4	0.00	0.99
Poor control	52	55	4		
Worst control	47	50	3		
Total	152	160	11		

Discussion

This study evaluated the demographic characteristics, glycemic control, and renal function indicators in a sample of 328 patients with type 2 diabetes mellitus (T2DM). The predominance of female participants (60.1%) and individuals within the middle-aged group (41–60 years, 54.0%) is generally consistent with observations that women and middle-aged adults tend to seek healthcare more regularly, which may reflect differences in health-seeking behavior between sexes. The relatively high prevalence of overweight individuals (58.8%) also agrees with the well-established association between obesity and insulin resistance, which represents a central mechanism in the pathophysiology of T2DM [18,19]. In addition, excess body weight may increase metabolic stress and can complicate glycemic control, thereby contributing to the risk of long-term complications [20].

Glycemic control among the studied patients appeared somewhat heterogeneous, with nearly comparable proportions of participants classified as having excellent (34.8%), poor (33.8%), and worst (31.1%) HbA1c levels. This variation may highlight the ongoing difficulty in achieving and maintaining optimal glycemic targets, particularly in populations where overweight and obesity are common. Persistent hyperglycemia remains a key factor in the development of diabetic kidney disease (DKD), mainly through mechanisms involving advanced glycation end-products, oxidative stress, inflammation, and glomerular dysfunction [21]. Although a considerable proportion of patients showed relatively good glycemic control, a notable number still remained potentially at risk for DKD as well as other vascular complications.

Regarding renal function, elevated urea levels were frequently observed (67.7%), whereas microalbuminuria was reported as normal in the majority of cases (74.4%). Nevertheless, 22% of patients exhibited microalbuminuria and 3.7% showed overt proteinuria. Serum creatinine levels were mostly within low or normal ranges (47.2% and 48.8%, respectively), with only a small proportion showing elevated values (3.4%). These findings may indicate that many patients are still in relatively early stages of renal involvement, although a subset already demonstrates early biochemical signs suggestive of DKD. This pattern is generally in agreement

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with the known progression of diabetic renal disease, where initial hyperfiltration and structural alterations eventually lead to measurable changes in renal biomarkers such as urea, creatinine, and urinary albumin excretion [22,23].

Interestingly, the correlation analysis did not reveal statistically significant associations between HbA1c and the evaluated renal markers (urea: r = 0.02, p = 0.64; microalbuminuria: r = –0.06, p = 0.22; creatinine: r = 0.00, p = 0.99). This finding might be partly explained by the relatively shorter duration of diabetes observed in a large proportion of the sample (1–5 years, 59.5%), suggesting that detectable renal impairment may not yet fully reflect glycemic exposure. At the same time, it is important to consider that renal dysfunction in T2DM is multifactorial and influenced by several additional variables, including blood pressure, genetic predisposition, and dietary habits, which could contribute to the weak correlations observed in this cross-sectional setting [24,25].

In the present study involving Libyan adults with T2DM, HbA1c was not significantly associated with urea, creatinine, or microalbuminuria, despite the relatively frequent abnormalities noted in these markers. This may suggest that a single measurement of HbA1c in a cross-sectional design may not adequately represent the cumulative metabolic burden or fully capture the complexity of renal injury processes in this population.

These findings differ somewhat from a number of previous studies that have reported significant positive correlations between glycemic control and renal biomarkers. For example, some recent cross-sectional studies have shown that higher HbA1c levels are associated with increased microalbuminuria and elevated serum creatinine [26]. In addition, predictive analyses in elderly hypertensive populations have suggested that HbA1c may independently predict the progression of diabetic nephropathy alongside other urinary indicators [27]. Earlier reports have also indicated that the severity of nephropathy tends to correlate with both the duration of diabetes and cumulative glycemic exposure [28].

On the other hand, the absence of significant associations in the current study is supported by emerging evidence that emphasizes the role of other contributing factors. Some recent data suggest that the relationship between HbA1c and microvascular complications may be relatively modest, indicating the involvement of multiple interacting determinants [29]. Moreover, there is increasing attention to glycemic variability, rather than mean HbA1c alone, as a potentially more sensitive indicator of microvascular stress and renal injury in certain patient groups [30]. In addition, factors such as systolic blood pressure and baseline renal status have been identified as strong predictors of nephropathy risk, sometimes exceeding the predictive value of a single HbA1c measurement [27].

The differences observed across studies could be explained by variations in study design, population characteristics, and clinical management practices. Factors such as sample size, age distribution, and the prevalence of hypertension may influence the strength of observed associations. Furthermore, the duration of diabetes and the use of newer therapeutic agents, including SGLT2 inhibitors, may alter the relationship between glycemic control and renal outcomes. It is also possible that genetic background and ethnicity-related susceptibility in the Libyan population contribute to differences in how hyperglycemia translates into renal damage. Altogether, these considerations suggest that longitudinal follow-up and more comprehensive multivariable analyses may provide a clearer understanding of DKD progression.

Limitation

Several limitations of this study should be considered. First, the cross-sectional design limits the ability to establish causal relationships between glycemic control and renal outcomes. Second, the predominance of female and middle-aged participants may reduce the generalizability of the findings to other demographic groups. In addition, the presence of missing data for some variables, including HbA1c, urea, and creatinine, could have introduced a certain degree of bias, although efforts were made to include only complete records. It is also possible that other relevant confounding factors were not fully accounted for. Therefore, future longitudinal studies are needed to better explore the temporal association between glycemic control and renal dysfunction, particularly across different durations of diabetes.

Conclusion

Early signs of renal involvement were observed among Libyan patients with type 2 diabetes mellitus, even though many renal biomarkers remained within normal or near-normal ranges. These findings highlight the importance of regular monitoring of renal function and early intervention strategies in order to reduce the progression of diabetic kidney disease and its related vascular complications. At the same time, further studies may be necessary to better clarify the underlying relationships and improve risk assessment in this population.

Conflict of interest. Nil

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