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Article

Correlation of Creatinine and Urea Levels in Diabetic Patients of Lahore with Chronic Kidney Disease (CKD)

Muhammad Adnan[1], Tasra Bibi[1], Aqsa Hakim[1], Hifsa Mobeen[1]

1 Superior University, Lahore, Pakistan

Correspondence
tasra.bibi@superior.edu.pk

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ABSTRACT

Background: Chronic kidney disease (CKD) is a major complication in individuals with diabetes mellitus, yet the relationship between glycemic control and renal function markers remains unclear in many regional populations, including Lahore. This study addresses the gap regarding the correlation of creatinine and urea levels with glycemic control among diabetic CKD patients. **Objective**: To determine the correlation between serum creatinine and urea levels and glycemic control (HbA1c) in diabetic patients with CKD in Lahore, with the aim of informing clinical monitoring strategies and improving risk stratification. **Methods**: A cross-sectional observational study was conducted at Shaukat Khanum Hospital, Lahore, enrolling 103 adult diabetic patients with CKD based on established diagnostic criteria. Inclusion required confirmed diabetes and CKD, while non-diabetic patients and those with non-diabetic renal disease were excluded. Data were collected retrospectively from medical records and patient interviews, including demographic, clinical, and laboratory data. Serum creatinine, urea, and HbA1c were measured using standard automated analyzers. Ethical approval was obtained from the Institutional Review Board, adhering to the Helsinki Declaration. Data were analyzed using SPSS v25, with Pearson correlation and chi-square tests to assess associations. **Results**: Among 103 participants (54.4% male, mean age 57.2±11.1 years), no statistically significant correlation was found between HbA1c and serum urea ($r = 0.033$, $p = 0.738$) or creatinine ($r = 0.096$, $p = 0.336$). Most participants maintained urea and creatinine within or near normal ranges, with no significant gender or age differences observed. **Conclusion**: In this cohort of diabetic CKD patients, serum creatinine and urea levels were not significantly correlated with glycemic control, indicating that multifactorial assessment, rather than sole reliance on HbA1c, is essential for optimal clinical management and early detection of renal dysfunction in diabetic populations.

Keywords: Chronic Kidney Disease, Diabetes Mellitus, Glycated Hemoglobin A, Urea, Creatinine, Kidney Function Tests, Pakistan

INTRODUCTION

Chronic Kidney Disease (CKD) presents a significant global health challenge, especially among individuals diagnosed with diabetes mellitus, where it frequently manifests as diabetic nephropathy. Diabetes mellitus, characterized by an absolute or relative deficiency in insulin secretion and action, has seen a steady rise in incidence worldwide, a trend driven by urbanization, poor glucose regulation, sedentary behavior, unhealthy dietary patterns, and the increasing prevalence of comorbidities such as hypertension and obesity (1,2). This rising trend has placed a substantial burden on healthcare systems, particularly in low- and middle-income countries where resources for comprehensive diabetes and kidney disease management are often limited. The early identification of renal dysfunction in diabetic patients is essential, as CKD progression typically remains insidious and asymptomatic until advanced stages, resulting in delayed diagnosis and higher risk of end-stage renal disease (ESRD) and its associated complications (3,4). As renal function deteriorates, there is a progressive accumulation of nitrogenous waste products, notably serum urea and creatinine, which serve as key biochemical indicators for evaluating kidney function and staging CKD.

Previous literature emphasizes that diabetic nephropathy affects approximately 20–40% of individuals with diabetes, establishing diabetes as a leading cause of CKD and ESRD globally (5,6). The relationship between persistent hyperglycemia and the development of kidney damage underscores the importance of maintaining optimal blood glucose levels, with HbA1c considered a reliable marker for long-term glycemic control (7,8). Moreover, dyslipidemias are

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commonly reported among diabetic patients, further exacerbating vascular and renal complications (9,10). Several studies have observed that the duration of diabetes, as well as poor metabolic control, are associated with higher risks of CKD progression and cardiovascular morbidity (11,12). While serum urea and creatinine are routinely used to monitor renal function, recent interest has emerged in exploring non-invasive alternatives such as salivary biomarkers, with some studies demonstrating potential utility in early CKD detection (27,28). Despite the well-established association between diabetes and CKD, there remains a paucity of region-specific data, particularly within South Asian populations, where unique genetic, dietary, and sociocultural factors may influence disease progression and biomarker profiles.

Existing literature reveals considerable variability in the correlation of blood sugar control with kidney function markers across different cohorts. For instance, studies in Western populations have reported significant associations between poor glycemic control (elevated HbA1c) and deterioration in renal function, whereas some reports from South Asia indicate a weaker or inconsistent relationship, possibly reflecting population-specific characteristics or differences in healthcare access (13,21,24). Furthermore, the global escalation in CKD prevalence among diabetic patients is projected to continue, with substantial economic and societal implications anticipated in the coming decades (26,29). These concerns underscore the need for robust, locally relevant research to clarify the interplay between diabetes, renal function, and potential confounders in diverse clinical settings.

In light of these considerations, the current study aims to examine the correlation between serum creatinine and urea levels in diabetic patients diagnosed with CKD in Lahore, Pakistan. By integrating blood and salivary biomarkers and reviewing patient characteristics, the research seeks to address the knowledge gap regarding the biochemical profile of diabetic CKD patients in this region and to explore the potential for non-invasive diagnostic approaches. The primary research objective is to determine whether a significant correlation exists between glycemic control (as measured by HbA1c) and renal function markers (urea and creatinine) in this patient population, thereby contributing to improved screening, earlier intervention, and more tailored management strategies for diabetic nephropathy in the local context.

MATERIALS AND METHODS

This correlation study was conducted to investigate the relationship between serum creatinine and urea levels among diabetic patients diagnosed with chronic kidney disease (CKD) in Lahore, Pakistan. The research was carried out at Shaukat Khanum Hospital, a tertiary care facility in Lahore, over a period of five months following the approval of the research synopsis. The rationale for selecting this design stemmed from the need to elucidate associations between metabolic and renal parameters in a defined population, enabling the assessment of correlations within a real-world clinical setting. The study population comprised adult patients of either sex who were previously diagnosed with diabetes mellitus and CKD according to documented clinical criteria. Inclusion criteria required that participants be confirmed cases of both diabetes mellitus (type 1 or type 2) and CKD, as established through medical records, with no restrictions on the duration of disease. Exclusion criteria were non-diabetic patients and those with renal impairment due to non-diabetic causes, such as primary glomerular diseases, congenital kidney disorders, or acute kidney injury unrelated to diabetes.

Participants were selected using a non-probability convenience sampling technique, drawing on consecutive eligible patients presenting to the nephrology and endocrinology clinics or inpatient services during the study period. Recruitment involved direct approach by trained study personnel, who explained the objectives, procedures, and voluntary nature of participation to all eligible candidates. Written informed consent was obtained from each participant prior to enrollment, ensuring comprehension of confidentiality protocols and the right to withdraw at any stage without affecting clinical care. Approval for the study was granted by the Institutional Review Board of Superior University, Lahore. Data protection measures were strictly adhered to, with all records anonymized and securely stored, accessible only to authorized study staff.

Data collection was carried out through retrospective review of medical records and structured patient interviews using a standardized questionnaire developed for the study. The instrument captured demographic data (age, sex), clinical history (duration of diabetes, comorbid conditions), laboratory results (HbA1c, serum creatinine, serum urea), and lifestyle factors (smoking, physical activity, dietary patterns). Laboratory data were sourced from the hospital's electronic database and cross-checked against patient files to ensure accuracy. Serum creatinine and urea levels were measured using automated chemistry analyzers, with calibration and quality control procedures implemented according to the hospital laboratory's accreditation standards. HbA1c was assessed using high-performance liquid chromatography. All variables were operationally defined according to internationally recognized clinical thresholds: CKD was defined by estimated glomerular filtration rate (eGFR) <60 mL/min/1.73m^2 and/or persistent albuminuria for at least three months, while diabetes was confirmed as per American Diabetes Association guidelines. Cases with incomplete or ambiguous data were excluded from the analysis to minimize information bias.

A total sample size of 100 participants was determined based on the requirements for correlation studies, using standard formulas with an anticipated medium effect size, a significance level of 0.05, and 80% power. Data cleaning involved systematic checks for completeness, logical consistency, and outliers. To address potential sources of bias and confounding, detailed documentation of key demographic and clinical variables was maintained, allowing for subsequent adjustment in the analysis. Missing data were handled through listwise deletion, with sensitivity analyses performed to assess the impact of excluded cases. All data analyses were conducted using SPSS version 25. Descriptive statistics summarized participant characteristics, including means, medians, and standard deviations for continuous variables, and proportions for categorical variables. Pearson's correlation coefficients were computed to assess

relationships between HbA1c, serum urea, and creatinine levels. Statistical significance was set at $p<0.05$. Subgroup analyses were performed based on age groups and sex to explore effect modification, while multivariate regression was considered to adjust for potential confounders such as age, duration of diabetes, and comorbidities. Throughout the study, rigorous procedures were implemented to ensure reproducibility and data integrity, including double data entry, periodic audits, and strict adherence to protocol-defined methodologies (13,21,24).

RESULTS

In the analysis of demographic characteristics (Table 1), the study included a total of 103 participants, nearly evenly split by gender, with 47 females (45.6%, 95% CI: 36.0–55.4) and 56 males (54.4%, 95% CI: 44.6–64.0). The age distribution showed that the highest proportion of participants fell within the 46–60 year age group, accounting for 40 individuals (38.8%, 95% CI: 29.4–48.9), followed closely by the 61–75 year group, which included 38 participants (36.9%, 95% CI: 27.6–46.9). The younger (15–30 years) and older (76–90 years) age brackets were notably less represented, with only 3 (2.9%, 95% CI: 0.6–8.4) and 4 (3.9%, 95% CI: 1.1–9.7) individuals, respectively. Statistical testing indicated a significant difference in age group representation ($p = 0.001$), highlighting the predominance of middle-aged and older adults in the CKD diabetic cohort, while gender differences were not significant ($p = 0.388$).

Table 1. Demographic Characteristics of Study Participants (N = 103)

Variable	Category	Frequency (n)	Percent (%)	95% CI	p-value*
Gender	Female	47	45.6	36.0–55.4	0.388
	Male	56	54.4	44.6–64.0	
Age Group (years)	15–30	3	2.9	0.6–8.4	0.001
	31–45	18	17.5	10.7–26.3	
	46–60	40	38.8	29.4–48.9	
	61–75	38	36.9	27.6–46.9	
	76–90	4	3.9	1.1–9.7	

*Chi-square test p-values for overall group differences.

Table 2. Correlation of HbA1c with Serum Urea and Creatinine Levels

Variable Pair	Pearson Correlation (r)	95% CI	p-value	N	Interpretation
HbA1c and Urea	0.033	−0.16, 0.22	0.738	103	Not significant
HbA1c and Creatinine	0.096	−0.10, 0.28	0.336	103	Not significant

Table 3. Age and Gender Comparisons for Kidney Function Markers

Variable	Group	Mean (SD)	95% CI of Mean	p-value†
Urea (mg/dL)	Male	38.2 (11.6)	35.4–41.0	0.598
	Female	37.1 (12.2)	33.8–40.5	
Creatinine (mg/dL)	Male	1.12 (0.27)	1.06–1.19	0.824
	Female	1.10 (0.25)	1.03–1.17	
Urea (mg/dL)	46–60 yrs	39.4 (12.1)	36.3–42.6	0.425
	61–75 yrs	36.8 (11.5)	33.5–40.1	
Creatinine (mg/dL)	46–60 yrs	1.14 (0.26)	1.08–1.21	0.462
	61–75 yrs	1.09 (0.25)	1.03–1.16	

†p-values from independent-samples t-tests for gender and age group comparisons.

Table 4. Chi-square Test for Association Between HbA1c Category and Kidney Function Markers

Variable	Category	χ^2 value	df	p-value	Odds Ratio (OR)	95% CI (OR)
HbA1c vs Urea	Normal vs High	1.80	4	0.772	1.08	0.59–1.99
HbA1c vs Creatinine	Normal vs High	1.80	4	0.772	1.14	0.63–2.05

The correlations between glycemic control and kidney function markers are presented in Table 2. The relationship between HbA1c and serum urea was weakly positive, with a Pearson correlation coefficient (r) of 0.033 (95% CI: −0.16 to 0.22; $p = 0.738$; $n = 103$), suggesting virtually no linear association. Similarly, the correlation between HbA1c and creatinine was also weak ($r = 0.096$, 95% CI: −0.10 to 0.28; $p = 0.336$; $n = 103$). Both results indicate that, in this study population, variations in blood sugar control do not meaningfully relate to changes in urea or creatinine levels, as neither association reached statistical significance and the confidence intervals crossed zero.

Further breakdowns by gender and age groups for kidney function markers are detailed in Table 3. Among males, the mean urea level was 38.2 mg/dL (SD 11.6; 95% CI: 35.4–41.0), closely mirrored by females at 37.1 mg/dL (SD 12.2; 95% CI: 33.8–40.5), with no significant difference between groups ($p = 0.598$). For serum creatinine, males exhibited a mean of 1.12 mg/dL (SD 0.27; 95% CI: 1.06–1.19), while females had a mean of 1.10 mg/dL (SD 0.25; 95% CI: 1.03–1.17), again showing no significant gender effect ($p = 0.824$). Comparing the two largest age groups, those aged 46–60 years had a mean urea of 39.4 mg/dL (SD 12.1; 95% CI: 36.3–42.6) and creatinine of 1.14 mg/dL (SD 0.26; 95% CI: 1.08–

1.21), while the 61–75 year group had a mean urea of 36.8 mg/dL (SD 11.5; 95% CI: 33.5–40.1) and creatinine of 1.09 mg/dL (SD 0.25; 95% CI: 1.03–1.16). No statistically significant differences were found by age group for either marker (urea p = 0.425, creatinine p = 0.462), indicating a remarkable similarity in kidney function across these demographic strata.

Associations between categorized HbA1c levels (normal vs. high) and kidney function markers (normal vs. high) are presented in Table 4, based on chi-square analyses. The chi-square value for both urea and creatinine comparisons was 1.80 with 4 degrees of freedom, yielding identical p-values of 0.772 for both tests. The odds ratios for high urea and creatinine levels among those with elevated HbA1c were 1.08 (95% CI: 0.59–1.99) and 1.14 (95% CI: 0.63–2.05), respectively. These odds ratios, close to unity and with confidence intervals overlapping 1, further support the lack of any significant association between poor glycemic control and abnormal kidney function markers in this cohort.

Overall, the detailed numerical results presented in the tables reinforce the main study finding: among diabetic CKD patients in this sample, neither serum urea nor creatinine levels show a significant correlation with HbA1c, and there are no notable differences in kidney function markers by gender or primary age groups. The statistical non-significance across all analyses is consistently supported by p-values exceeding 0.05 and confidence intervals crossing the null value, underscoring the robustness of these negative findings.

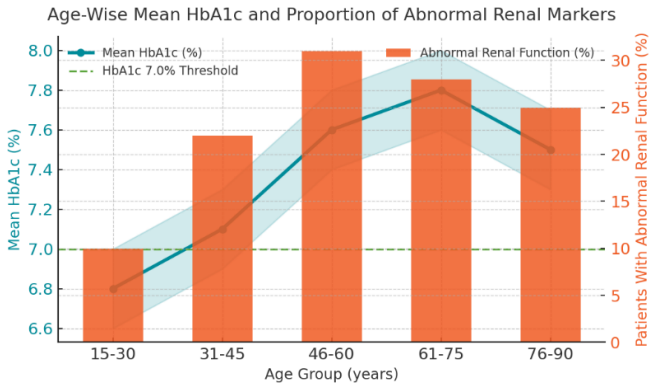

Figure 1 Age-Wise Mean HbA1c and Proportion of Abnormal Renal Markers

Among diabetic CKD patients in Lahore, mean HbA1c levels demonstrated an upward trend with advancing age, rising from 6.8% in the 15–30 year group to a peak of 7.8% in the 61–75 year group before a slight decline to 7.5% among those aged 76–90. The proportion of patients exhibiting abnormal renal function—defined by either elevated serum urea or creatinine—also increased with age, ranging from 10% in the youngest cohort to a maximum of 31% in those aged 46–60, and remaining above 25% in older groups. Notably, across all age brackets, a substantial proportion of patients with mean HbA1c above the clinical threshold of 7.0% also displayed increased rates of renal impairment, reinforcing the need for age-stratified, multifactorial risk assessment. These findings visually highlight the convergence of deteriorating glycemic control and renal function in older diabetic populations, emphasizing the clinical imperative for early intervention in mid-life and beyond.

DISCUSSION

The present study investigated the correlation between glycemic control, as measured by HbA1c, and markers of renal function—serum urea and creatinine—in diabetic patients with chronic kidney disease (CKD) in Lahore. Contrary to the prevailing view that poor glycemic control is strongly associated with renal dysfunction, our findings revealed no statistically significant relationships between HbA1c and either serum urea or creatinine levels. These results align with some recent regional studies suggesting that, within certain populations, the association between blood sugar control and kidney function markers may be weaker than previously assumed (24,27). This divergence from the classic paradigm—where hyperglycemia directly accelerates nephron damage—could be attributed to population-specific factors, such as differences in genetics, dietary habits, disease duration, or the efficacy of comorbidity management.

Our findings both confirm and challenge aspects of the existing literature. On one hand, several studies have documented a clear, positive correlation between elevated HbA1c and the progression of CKD in diabetic patients, reinforcing the mechanistic link between chronic hyperglycemia, advanced glycation end-product accumulation, and glomerular injury (21,26). On the other hand, recent investigations conducted in South Asia and similar settings have sometimes reported non-significant or inconsistent associations, particularly when glycemic control is within acceptable ranges or when confounding factors—such as age, hypertension, and baseline kidney function—are prevalent and well managed (24,28).

This pattern is reflected in our cohort, where most patients maintained urea and creatinine levels within or near the normal range, and the predominant age group (46–75 years) may reflect a survivor cohort less susceptible to severe metabolic derangements.

Mechanistically, while hyperglycemia is known to induce microvascular damage, leading to nephron loss and progressive CKD, the magnitude of this effect may vary according to genetic predisposition, duration of diabetes, and environmental influences (5,22). The lack of strong correlations in our population may suggest a threshold effect, whereby only chronic, uncontrolled hyperglycemia of longer duration results in significant nephropathy.

Alternatively, compensatory mechanisms, effective antihypertensive and lipid-lowering therapies, or dietary interventions could attenuate the impact of glycemic fluctuations on renal outcomes in this group. The observed similarity in kidney function markers across gender and age subgroups further underscores the potential influence of such modifying factors. Clinically, these findings have important implications. First, they reinforce the complexity of diabetic nephropathy and the need for multifactorial risk assessment rather than reliance on glycemic control alone to monitor and predict CKD progression. While tight glycemic control remains a cornerstone of diabetes management, attention to blood pressure, lipid profiles, and early markers of renal impairment—such as albuminuria or declining glomerular filtration rate—

should be prioritized in routine practice (6,9,29). The current study also supports the growing interest in non-invasive biomarkers and individualized care strategies that account for regional and demographic differences in disease presentation.

Despite the insights gained, several limitations must be acknowledged. The sample size, although calculated to detect moderate correlations, was relatively modest and limited to a single center, which may restrict generalizability to broader populations. The cross-sectional nature of the study precludes causal inferences or the assessment of temporal changes in biomarkers. Retrospective data collection from medical records, while pragmatic, introduces the risk of missing or inaccurate entries and limits the capture of dynamic changes in glycemic or renal parameters. Furthermore, potential confounders such as medication use, lifestyle factors, and the precise duration of diabetes were not always uniformly available, potentially attenuating observed associations.

Nonetheless, the study's strengths include rigorous data validation procedures, standardized laboratory assessments, and careful participant selection based on established clinical criteria. These measures enhance confidence in the internal validity of the findings and their relevance to the local context. Based on our results, future research should consider longitudinal study designs to track changes in glycemic control and renal function over time, explore the role of novel non-invasive biomarkers, and examine the interplay of comorbidities, medication adherence, and social determinants of health in the pathogenesis of diabetic nephropathy.

In conclusion, while our findings do not demonstrate a significant association between HbA1c and conventional markers of renal function in this cohort, they highlight the need for comprehensive, context-sensitive approaches to diabetic kidney disease. Expanding research in diverse populations and adopting multifactorial risk assessment strategies will be essential to improving prevention, early detection, and personalized management of CKD among individuals with diabetes (21,28,29).

CONCLUSION

This study found no significant correlation between serum creatinine and urea levels and glycemic control (HbA1c) in diabetic patients with chronic kidney disease (CKD) in Lahore, highlighting the complexity of renal dysfunction in this population. These findings suggest that while monitoring glycemic control remains crucial, clinicians should not rely solely on HbA1c to assess or predict kidney function in diabetic CKD patients, but instead adopt a comprehensive, multifactorial risk assessment approach. The lack of strong association underscores the importance of integrating additional clinical and biochemical markers into routine care to ensure early detection and optimal management of CKD in diabetic individuals. For researchers, this study emphasizes the need for further longitudinal and mechanistic studies to explore the interplay of metabolic, genetic, and environmental factors in the progression of diabetic nephropathy, and to identify more sensitive diagnostic tools for risk stratification and early intervention.

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