e-ISSN: 0975-9506, p-ISSN: 2961-6093

Available online on www.ijpqa.com

International Journal of Pharmaceutical Quality Assurance 2026; 17(4); 326-331

Original Research Article

Comparative Evaluation of Blood Urea and Serum Creatinine Levels in Diabetic Patients and Non-Diabetic Individuals

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Received: 12-02-2026 / Revised: 19-03-2026 / Accepted: 21-04-2026

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Conflict of interest: Nil

Abstract:

Background: Diabetes mellitus causes chronic hyperglycemia leading to microvascular complications, particularly diabetic nephropathy. Blood urea and serum creatinine are important biochemical markers for early detection of renal impairment.

Aim: To compare blood urea and serum creatinine levels in diabetic patients and non-diabetic individuals and evaluate their clinical relevance in assessing renal function.

Methodology: A comparative cross-sectional study was conducted on 40 subjects (20 Type-2 diabetics and 20 age-matched controls) aged 35–65 years at a tertiary care hospital. Fasting and post-prandial glucose, HbA1c, blood urea (urease method), and serum creatinine (Jaffe's method) were measured. Data were analyzed using Student's t-test and correlation analysis ($p<0.05$ significant).

Result: Diabetics showed significantly higher blood urea (58.92±11.34 vs 26.80±4.90 mg/dl) and serum creatinine (1.62±0.38 vs 0.86±0.14 mg/dl) ($p<0.001$). Renal abnormalities were present only in diabetics. HbA1c positively correlated with blood urea ($r=0.61$) and creatinine ($r=0.68$). Creatinine was significantly higher in male diabetics.

Conclusion: Diabetes is strongly associated with impaired renal function. Elevated urea and creatinine, particularly with poor glycemic control, indicate early nephropathy; routine monitoring is essential for prevention.

Keywords: Diabetes mellitus, Blood urea, Serum creatinine, Diabetic nephropathy, Renal function, HbA1c.

DOI: 10.25258/ijpqa.17.4.53

Introduction

Diabetes mellitus is a serious health issue and one of the most prevalent metabolic disorders all over the world. It is largely caused by the defects in either insulin secretion or insulin action or both [1]. The condition mainly manifests itself through persistent hyperglycemia caused by imbalance in carbohydrate, fat and protein metabolism [2]. Chronic hyperglycemia in the long run causes structural and functional changes in various tissues such as the eyes, kidneys, heart, nerves and blood vessels, which cause microvascular and macrovascular complications.

Diabetes have been known globally as one of the causes of morbidity and mortality. The percentage has been reported to be approximately 2.2 to 3% and this is likely to rise further in the 21 st century as diabetes has become one of the most difficult challenges in the field of public health with almost 6 -7 percent of the global population affected by the illness [3]. It is estimated that about 170 million people globally are already living with diabetes and this figure might rise to 438 million by the year 2030 [4]. The increase is credited to the fast urbanization, the lack of exercise, and change of diet. A number of risk factors have been found in the pathology of diabetes such as dietary change, gene mutations, hypertension, smoking, obesity, hypercholesterolemia, and physical inactivity [5,6].

Diabetes is not a simple glucose metabolic disorder, it is a complicated metabolic syndrome that is usually accompanied by dyslipidemia, hypertension, and visceral fat. These related disorders are comor-

bid risk factors to chronic and heart-related complications [7]. Renal involvement is one of the long-term complications that are especially highly valued due to their progressive and irreversible nature. The most common renal disorder that is seen in diabetic patients is end-stage renal disease (ESRD) and diabetic nephropathy [8]. The incidence of diabetic nephropathy in diabetic people is about 25-45 percent in their lifetime [9].

Diabetic nephropathy (DN) is a micro-vascular complication of long-term hyperglycemia. The glycosylation of the tissue proteins leads to structural and functional impairment of the renal tissue ultimately culminating to nephropathy. The prevalence of DN in most countries is close to 30% of the total number of diabetic patients and the number one cause of ESRD [10] . Diabetic nephropathy is pathogenies through various mechanisms including non-enzymatic glycation of proteins, oxidative stressful condition, hemodynamic changes as well as inflammatory pathways. Continuous hyperglycemia induces thickening of the glomerular basement membrane, mesangio-expansion, and loss of filtration capacity. These pathological alterations slowly worsen kidney functioning and eventually lead to kidney failure [11].

Early stages of renal impairment in diabetic patients tend to be asymptomatic, so lab research is essential in the early identification of renal impairment. The characteristics indicators of diabetic nephropathy include abnormal renal functions that are manifested through high blood urea, high serum creatinine, and macroalbuminuria. The presence of uncontrolled diabetes is often related to hyperglycemia-induced increase of the levels of blood urea and serum creatinine. Thus, urea and creatinine can be regarded as two significant bio-chemical parameters that could be used to assess kidney functioning [12].

The final product of the protein metabolism is blood urea, which is produced in the liver and is released by the kidneys. The level in blood is determined by a variety of physiological factors such as the condition of liver functioning, intake of proteins, level of hydration and the rate of protein catabolism. Serum creatinine, however, is a metabolic derivative of creatine phosphate, produced as a result of muscle metabolism which is mostly eliminated by the glomerular filtration system [13]. Since the production of creatinine is relatively stable and is not substantially influenced by the diet, it has a better indicator of glomerular filtration rate (GFR). Variations in serum creatinine levels are thus regarded as an indicator of kidney malfunction.

Blood urea and serum creatinine measurement is critical in initial detection of kidney involvement in diabetes. Any change in these parameters usually comes before the clinical manifestations and timely action and prevention of the development of the disease can be performed. Early detection of kidney damage allows the adoption of treatment measures that include glucose regulation, blood pressure management and lifestyle changes, which minimize the chances of ESRD [14]. Regarding the fact that renal complications are more common among diabetic patients than non-diabetic ones, comparative assessment of these biochemical indicators becomes a clinical aspect.

Knowledge about the discrepancies in the markers of renal functioning between diabetic and non-diabetic groups could be applied to diagnose the initial nephropathy, the course of the disease, and treatment results. To better screening and preventive healthcare measures, such comparative studies also help. Therefore, the current research will assess the blood urea and serum creatinine concentrations in diabetic patient and compare the parameters with the normal population with no diabetes to establish their clinical significance in the measurement of renal activity and the early signs and symptoms of diabetic kidney disease.

Methodology

Study Design: The present study was conducted as a comparative cross-sectional study aimed at evaluating and comparing blood urea and serum creatinine levels between diabetic patients and non-diabetic individuals. The study intended to assess renal function alterations associated with diabetes mellitus by comparing biochemical parameters between the two groups.

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

Study Duration: The study was conducted over a period of six months from April 2025 to September 2025.

Sample Size: A total of 40 subjects were included in the study. Among them, 20 were diagnosed cases of Type 2 Diabetes Mellitus and were categorized as the case group, while 20 age-matched healthy individuals without diabetes were included as the control group for comparison.

Study Population: The study population comprised adult male and female subjects aged between 35 and 65 years. The case group included patients with a known history of Type 2 Diabetes Mellitus for more than three years attending the outpatient and inpatient departments. The control group consisted of apparently healthy individuals without a history of diabetes and with normal blood glucose levels and renal function tests.

Data Collection: After obtaining written informed consent, a detailed medical history and clinical examination were conducted for all participants. Information regarding age, gender, fasting blood sugar, post-prandial blood sugar, glycated hemoglobin (HbA1c), blood urea, and serum creatinine levels

was recorded. Venous blood samples were collected under aseptic conditions. Serum was separated by centrifugation at 4,000 rpm for 10 minutes. Plasma glucose was estimated using the glucose oxidase-peroxidase (GOD-POD) method. Blood urea was measured by the enzymatic urease method and serum creatinine by the alkaline Jaffe's method. HbA1c estimation was carried out using the ion-exchange resin method with a commercially available diagnostic kit. All biochemical parameters were analyzed using commercial kits adapted to an automated analyzer in the clinical biochemistry laboratory.

Inclusion Criteria

- Individuals aged 35–65 years
- Diagnosed cases of Type 2 Diabetes Mellitus (≥ 3 years duration) for the case group
- Healthy, non-diabetic individuals with normal blood glucose and renal function tests for the control group
- Participants willing to give written informed consent

Exclusion Criteria

- Smokers
- Patients with hypertension
- Hyperlipidemia
- Pregnant women
- Individuals with known renal disease
- Patients with other chronic systemic disorders

Ethical Considerations: The study was conducted after obtaining approval from the Institutional Ethics Committee. Written informed consent was obtained from all participants prior to enrolment. Confidentiality of patient information was maintained throughout the study, and all procedures were carried out in accordance with institutional ethical guidelines and WHO recommendations.

Procedure: Participants were categorized into diabetic and non-diabetic groups based on WHO diagnostic criteria for diabetes mellitus. Fasting blood samples were collected after overnight fasting, and post-prandial samples were collected two hours after meal intake. The collected samples were analyzed for biochemical parameters following standard laboratory protocols to ensure accuracy and reliability of results.

Statistical Analysis: The data collected were entered into Microsoft Excel and analyzed statistically. Results were expressed as mean ± standard deviation. The difference between diabetic and non-diabetic groups was assessed using Student's t-test. A p-value less than 0.05 was considered statistically significant."

Result

Table 1 demonstrates that renal impairment was observed only among diabetic cases and not in controls. Increased blood urea was found in 6 patients, elevated serum creatinine in 7 patients, and both parameters were raised in 5 patients among diabetics, whereas none of the controls showed any abnormality. Only 2 diabetic subjects had values within normal limits compared to all 20 controls, indicating a clear association between diabetes and deterioration of renal function.

Table 1: Number of samples showing increased Blood Urea and Serum Creatinine in diabetics and controls (N=40)

Parameters	Cases (n = 20)	Controls (n = 20)
Blood Urea increased	6	0
Serum Creatinine increased	7	0
Both Urea & Creatinine increased	5	0
Within normal limits	2	20

Table 2 shows that all biochemical parameters were significantly higher in diabetic subjects compared to non-diabetics. Fasting blood sugar (178.40 ± 28.52 mg/dl vs 92.65 ± 8.41 mg/dl), post-prandial blood sugar (264.75 ± 36.10 mg/dl vs 121.20 ± 7.30 mg/dl), and HbA1c (7.82 ± 0.91% vs 5.23 ± 0.40%) were markedly elevated in diabetics ($p < 0.001$). Renal function markers were also significantly raised, with blood urea (58.92 ± 11.34 mg/dl vs 26.80 ± 4.90 mg/dl) and serum creatinine (1.62 ± 0.38 mg/dl vs 0.86 ± 0.14 mg/dl) higher among diabetics ($p < 0.001$), indicating impaired kidney function associated with diabetes.

Table 2: Mean ± SD of biochemical parameters in diabetic and non-diabetic subjects

Parameters	Diabetics (n = 20) Mean ± SD	Non-Diabetics (n = 20) Mean ± SD	P value
Fasting Blood Sugar (mg/dl)	178.40 ± 28.52	92.65 ± 8.41	0.000*
Post Prandial Blood Sugar (mg/dl)	264.75 ± 36.10	121.20 ± 7.30	0.000*
HbA1c (%)	7.82 ± 0.91	5.23 ± 0.40	0.000*
Blood Urea (mg/dl)	58.92 ± 11.34	26.80 ± 4.90	0.000*
Serum Creatinine (mg/dl)	1.62 ± 0.38	0.86 ± 0.14	0.000*

Table 3 compares renal parameters between male and female diabetics. Mean blood urea levels were slightly higher in males (60.12 ± 10.80 mg/dl) than females (57.15 ± 11.92 mg/dl), but the difference was not statistically significant (p = 0.31). However, serum creatinine levels were significantly higher in males (1.71 ± 0.35 mg/dl) compared to females (1.48 ± 0.32 mg/dl) (p = 0.04), indicating comparatively poorer renal function among male diabetic patients.

Table 3: Comparison of Blood Urea and Serum Creatinine according to gender in diabetics

Parameter	Male (n=12) Mean ± SD	Female (n=8) Mean ± SD	P value
Blood Urea (mg/dl)	60.12 ± 10.80	57.15 ± 11.92	0.31
Serum Creatinine (mg/dl)	1.71 ± 0.35	1.48 ± 0.32	0.04*

Table 4 shows a significant positive correlation between glycemic control (HbA1c) and renal function parameters among diabetics (n=20). HbA1c demonstrated a moderately strong positive correlation with blood urea (r = 0.61, p = 0.003) and an even stronger positive correlation with serum creatinine (r = 0.68, p = 0.001). These findings indicate that higher HbA1c levels are associated with increased blood urea and creatinine levels, suggesting worsening renal function with poor glycemic control.

Table 4: Correlation of HbA1c with renal parameters in diabetics (n=20)

Parameter	Correlation Coefficient (r)	P value
HbA1c vs Blood Urea	0.61	0.003*
HbA1c vs Serum Creatinine	0.68	0.001*

Discussion

The current research revealed significant modification in the renal biochemical parameter of diabetic patients against non-diabetic controls. Blood urea (58.92 11.34 mg/dl) and serum creatinine (1.62 0.38 mg/dl) were significantly higher in diabetics compared to controls (26.80 4.90 and 0.86 0.14mg/dl respectively) and only two diabetics fell within the normal range and all other non-diabetics had a normal renal index. The findings of these studies are a good indication that chronic hyperglycemia causes early renal failure. The same results have been indicated by Sirivole and Eturi (2017) [2], who also reported an increase in urea and creatinine in diabetics, which is a characteristic of failing renal function that is linked to long-term hyperglycemia. As Chutani and Pande (2017) [7] also showed, urea and creatinine continued to rise as the glycemic index worsened, and the length of time the participants had diabetes, proving that renal damage is associated with metabolic control."

Blood urea (r = 0.61) and serum creatinine (r = 0.68) were found to have a moderate positive and strong positive correlation with the high fasting blood sugar (178.40 ± 28.52 mg/dl), post-prandial glucose (264.75 ± 36.10 mg/dl), and HbA1c (7.82 ± 0.91%). The same case was reported by Abdelsalamka and Elamin (2011) [3] who established that there was a significant correlation between HbA1c and urea concentration indicating that as glycemic control worsens, nephropathy progresses at a faster rate. Similar findings support the assumption that chronic hyperglycemia harms the glomerular basement-membrane thus resulting in diminished filtration ability and nitrogenous waste products build-up.

The pathophysiology of diabetic nephropathy is also advocated by our observation that a number of diabetic patients were simultaneously found to have increased both renal markers. Chronic hyperglycemia causes glomerular hyperfiltration, then subsequent thickening of the basement membrane and mesangial expansion which in the end leads to the reduction of glomerular filtration rate (GFR). Since the creatinine is an indirect measure of GFR and urea buildup is an indicator of impairment of excretion, the increase in both parameters is indicative of progressive impairment of renal excretion. The same progressive worsening of the renal functioning with poor glycemic control was also mentioned in the United Kingdom Prospective Diabetes Study where the authors underlined that these markers could predict diabetic nephropathy (Adler et al., 2003) [15]. Similarly, Whaley-Connell et al. (2009) [8] have established that diabetes is among the major causes of chronic kidney disease in the world and biochemical surveillance can be used to identify renal damage at its early stages.

In our study, there were slight differences in gender in blood urea of males (60.12 ± 10.80 mg/dl) and females (57.15 ± 11.92 mg/dl) which were not significant, whereas there were significant differences between serum creatinine in males (1.71 ± 0.35 mg/dl) and females (1.48 ± 0.32 mg/dl). The difference can be explained by a greater muscle mass and metabolic turnover in males resulting in an increased level of creatinine. Even though they did not specifically highlight similar gender-related differences in Sirivole and Eturi (2017) [2], they observed that there was a difference in the levels of creatinine that depended on physiological factors such as muscle mass. We can therefore find that our results do not

conflict with previous literature but are in line with physiological expectations.

A similar study in Anjaneyulu and Chopra (2004) [16] by use of animal experimental research revealed that diabetic rats experienced progressive kidney damage with an increase in urea level, and also, creatinine level which supports our clinical observation that hyperglycemia directly harms kidney tissue. The causal relationship between diabetes and nephron injury is enhanced by the fact that both experimental and clinical results are similar. In addition, epidemiological investigations provide evidence that diabetes is a significant cause of end-stage renal disease in the global context, and therefore it has a clinical significance of early biochemical testing (Atkins, 2005; Stewart et al., 1998) [9,10].

Also, our findings indicated that renal parameters of only two diabetic subjects remained normal even with the increased level of glucose in the body, which pointed to an early diagnosis of the disease or a milder stage. This is contrary to Chutani and Pande (2017) [7], who have found progressive rise in renal markers with the increased duration of diabetes, suggesting that the duration and extent of nephropathy are dictated by duration and severity of diabetes. Thus, this difference within the sample can be explained by reduced length of disease or personal metabolic variations.

The high positive relationship between HbA1c and serum creatinine that exists in our study suggests that long-term glycemic status is more predictive, compared to single glucose measurements. This observation is in line with the hypothesis put forward by Shlomo et al. (2011) [13] that microvascular complications are caused by chronic sustained hyperglycemia and not by a transient elevation of glucose. Chronic glycation of proteins causes end products of glycation that destroy renal capillaries and diminish their filtration ability.

In general, our results agree with majority of the past clinical and experimental studies that demonstrate considerable increase of blood urea and serum creatinine in diabetics as compared to non-diabetics. Small variations in the magnitude between the studies could be explained by the period of diabetes, demographics, and treatment status. The fact that no abnormal renal markers were present in all the control subjects further supports the association of hyperglycemia with renal dysfunction as opposed to the variation with age. Therefore, in line with previous studies, blood urea and serum creatinine are good prognostic factors to identify and follow up diabetic nephropathy and should be regularly checked on diabetic patients to help in averting the onset of chronic kidney disease.

Conclusion

The current research shows that diabetic patients have significantly different renal biochemical parameters relative to non-diabetic controls, which shows an initial renal involvement in diabetes mellitus. The proportion of diabetic subjects that experienced increase in their blood urea and serum creatinine was quite high and all subjects who were not diabetic were kept within the normal range. Mean values of glycemic marker and renal function marker were significantly different between the diabetics as it supports the fact that chronic hyperglycemia and damaged kidney functioning are related to each other. Gender analysis indicated that there was a great difference in serum creatinine with values being relatively higher in men but there was no significant difference in the level of blood urea. Moreover, the positive association between HbA1c and the two renal parameters also means that the deteriorating glycemic control is associated with deteriorating renal dysfunction. In general, the results highlight the significance of regular tests of renal biomarkers in patients with diabetes to prevent and focus on the early stages of diabetic nephropathy.

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