

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

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Abstract:

Background: Diabetes mellitus is a major metabolic disorder characterized by chronic hyperglycemia that may lead to long-term complications affecting various organs, particularly the kidneys. Renal impairment in diabetic patients is commonly assessed using biochemical markers such as blood urea and serum creatinine.

Aim: To compare blood urea and serum creatinine levels between diabetic and non-diabetic individuals and evaluate their association with renal dysfunction.

Methodology: This comparative cross-sectional study included 48 subjects (24 diabetic patients and 24 non-diabetic controls) aged 35–65 years. Blood samples were collected and analyzed for fasting blood sugar, post-prandial blood sugar, HbA1c, blood urea, and serum creatinine using standard biochemical methods. Data were expressed as mean $\pm$ SD and analyzed using Student's *t*-test.

Results: Diabetic subjects showed significantly higher mean values of blood urea (61.25 ± 10.64 mg/dl) and serum creatinine (1.72 ± 0.58 mg/dl) compared to non-diabetics (26.48 ± 4.97 mg/dl and 0.86 ± 0.15 mg/dl, respectively; $p < 0.05$). Elevated urea and creatinine levels were observed only in diabetic patients.

Conclusion: The study demonstrates that diabetic patients have significantly elevated renal function markers, indicating a higher risk of renal impairment. Regular monitoring of blood urea and serum creatinine may help in the early detection of diabetic nephropathy.

Keywords: Diabetes Mellitus, Blood Urea, Serum Creatinine, Renal Function, Diabetic Nephropathy, Comparative Study.

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Introduction

Diabetes mellitus is a metabolic disorder that is usually prevalent all over the world and is primarily brought on by the aberrations in the release of insulin, its action, or a combination of both [1]. It is mainly marked by chronic hyperglycemia which arises out of the defective metabolism of carbohydrates, fats and proteins [2]. Continuous hyperglycemia causes different biochemical and biophysical alterations within the organism that can eventually cause harm to a number of body organs and tissues. The body organs that are most likely to be affected include the eyes, kidneys, heart, nerves, and blood vessels. These complications have a large role in the morbidity and mortality of diabetes mellitus as a whole.

Diabetes has become a significant health issue of concern among the world population based on its swiftly growing prevalence. It is now known as among the most prevalent causes of morbidity and

mortality in the entire world. Diabetes affects approximately 2.2 to 3 percent of the world population and the percentage is likely to rise considerably in the next few years [3]. Diabetes became one of the most problematic health issues in the 21 st century as almost 67 out of 100 people in the world are affected by this disease. Presently, the number of individuals in the world who live with diabetes is approximately 170 million and it is estimated that the figure will increase to nearly 438 million in the year 2030. The ever-growing rate of diabetes prevalence is mainly due to the shift in lifestyle trends, urbanization, and aging of the population [4].

There are a number of risk factors which cause and escalate diabetes mellitus. They are dietary changes, genetic mutations, hypertension, smoking, obesity, high cholesterol levels and inactivity [5]. Unhealthy eating habits and sedentary lifestyle have been a major cause of increased cases of diabetes, especially

among the developing nations. Genetic predisposition is also a significant determinant because the people whose family has a history of diabetes are more susceptible to acquiring the disease. Moreover, there are also metabolic abnormalities that make one prone to diabetes including insulin resistance and impaired glucose tolerance [6].

Other common complications are metabolic and cardiovascular complications that are often related to diabetes mellitus. Dyslipidemia, hypertension, and visceral adiposity are usually associated with diabetes and predispose the patient to the development of chronic diseases and cardiovascular complications significantly [7]. These comorbidities are not only associated with the aggravation of the metabolic imbalance but also with the acceleration of the exertions of diabetes-related complications. Of these complications, renal disorders hold the greatest importance as they can go to an extreme and permanently harm the kidneys unless they are detected and treated early enough.

This is one of the most severe long-term effects of diabetes mellitus since it leads to complications with the kidneys. A frequent cause of renal impairment in diabetic patients with diabetes is diabetic nephropathy, which is a leading cause of end-stage renal disease (ESRD) in many countries across the globe. The proportion of diabetic patients that develop diabetic nephropathy throughout their life has been approximated as 25- 45 percent [8]. The occurrence of diabetic nephropathy is tightly connected with the long-term hyperglycemia, hypertension, and other metabolic disorders which are presented in the diabetic patients. The disease takes time to affect the structure and functioning of the kidneys, and eventually renal failure will develop without being treated.

The glycosylation of tissue proteins is one of the greatest pathological mechanisms that cause diabetic nephropathy. Constant hyperglycemia causes non-enzymatic glycosylation of proteins, which causes the formation of advanced glycation end products (AGEs). Such products are stored in different tissues such as kidneys and cause structural and functional impairments. This happens by thickening of glomerular basement membrane, mesangial growth and glomerular sclerosis which are known features of diabetic nephropathy. Diabetic nephropathy occurs almost in 30 percent of patients with diabetes in various countries, and it is regarded as one of the most common causes of end-stage renal disease (ESRD) [9].

Renal dysfunction in diabetic patients is a disease that has to be identified early enough to help avoid additional complications and delay the process of kidney damage. The kidney functioning is evaluated by several biochemical parameters which include blood urea and serum creatinine which are the most

common biochemical parameters used to evaluate kidney functioning. A high concentration of blood urea, serum creatinine, and the occurrence of macroalbuminuria are usually the clues of abnormal renal functioning [10]. These parameters are informative as to the functioning of the kidneys and aid in the diagnosis and testing of renal dysfunction.

In diabetic mellitus, which is uncontrolled, constant hyperglycemia can cause progressive renal damage to cause an abnormal elevation in the levels of blood urea and serum creatinine. The final product of protein metabolism is urea which is synthesized in the liver by the urea cycle. Numerous factors determine its level in the blood including the liver activity, the amount of protein one consumes in the diet and the degradation of the protein [11]. Creatinine, however, is a product of metabolism of creatine and phosphocreatine in muscle tissue. It is synthesized at a fairly stable rate and is mainly released through the kidneys.

Serum creatinine is regarded as being a more accurate measure of the renal functioning due to its close relationship in the bloodstream with the glomerular filtration rate (GFR). As the performance of the kidneys decreases, the capacity of the kidneys to eliminate creatinine also reduces and the creatinine is built up in the bloodstream. Thus, serum creatinine variability is significant in reflecting the change of GFR and renal functioning. Conversely, blood urea can be different under a number of physiological and metabolic conditions although it still gives useful data when measured in combination with serum creatinine levels [12].

Blood urea and serum creatinine measurement is of vital significance in the early diagnosis and treatment of kidney dysfunction in diabetic patients [13]. These parameters should be assessed regularly to detect renal complications at an early stage so that they can be addressed and managed in time. It is also important that diabetic kidney disease be early diagnosed so as to prevent its development and also to lower the chances of end-stage renal disease development [14].

Because of the prevalence of renal complications in patients with diabetes mellitus, assessing blood urea and serum creatinine as the signs of renal functioning is critical in clinical practice. A comparative analysis of the parameters of diabetic and non-diabetic people can be of significance in revealing the effect of diabetes on kidney functioning. Thus, the current research was conducted to determine the quantity and the comparison of the blood urea and serum creatinine levels in diabetic patients and relate them to the levels of non-diabetic patients. This comparative analysis may help in better understanding the relationship between diabetes mellitus and renal function impairment and may also contribute

to the early detection and management of diabetic nephropathy.

Methodology

Study Design: The present research was conducted as a comparative cross-sectional study aimed at evaluating and comparing blood urea and serum creatinine levels between diabetic and non-diabetic individuals. The study design enabled the comparison of renal function markers between two groups at a single point in time in order to assess the possible association between diabetes mellitus and altered kidney function parameters.

Study Area: The study was carried out in the Department of Biochemistry, Bhagwan Mahavir Institute of Medical Sciences, Pawapuri, Nalanda, Bihar, India.

Study Duration: The duration of the study was 8 months from March 2025 to October 2025.

Sample Size: A total of 48 subjects were included in the study. The participants were divided into two groups consisting of 24 diabetic patients (cases) and 24 non-diabetic healthy individuals (controls). This sample size was selected to allow comparison of biochemical parameters between the two groups.

Study Population: The study population consisted of adult individuals attending the outpatient and inpatient departments of Bhagwan Mahavir Institute of Medical Sciences. Both male and female subjects within the age group of 35–65 years were included in the study. The diabetic group consisted of patients diagnosed with diabetes mellitus, while the control group included apparently healthy individuals without a history of diabetes and with normal blood glucose levels.

Data Collection: Data collection was carried out after obtaining informed consent from the participants. A detailed medical history and clinical examination were conducted for all subjects. Demographic information such as age and gender was recorded along with clinical and biochemical parameters including fasting blood glucose (FBS), post-prandial blood glucose (PPBS), HbA1c, blood urea, and serum creatinine levels. Approximately 5 ml of venous blood was collected from each participant under aseptic conditions. The collected blood samples were processed in the clinical biochemistry laboratory for further biochemical analysis.

Inclusion Criteria

- Patients aged 35–65 years.
- Patients with a known history of diabetes mellitus for at least 3 years (case group).

- Healthy individuals with normal blood glucose and normal renal function tests (control group).
- Individuals willing to provide written informed consent.

Exclusion Criteria

The following individuals were excluded from the study:

- Smokers
- Hypertensive patients
- Hyperlipidemic individuals
- Pregnant women
- Patients with known chronic kidney disease or other chronic systemic disorders

Procedure: Blood samples were collected from both diabetic and non-diabetic subjects under aseptic conditions. The collected samples were centrifuged at 4000 rpm for 10 minutes to separate serum. Plasma glucose levels were estimated using the Glucose Oxidase-Peroxidase (GOD-POD) end-point assay method. Blood urea was measured using the enzymatic urease method, while serum creatinine levels were estimated by the alkaline Jaffe's method. HbA1c levels were determined using the ion exchange resin method with a diagnostic HbA1c kit. All biochemical parameters were analyzed using standard commercial kits adapted to an automated biochemistry analyzer in the clinical laboratory.

Statistical Analysis: The collected data were entered and organized using Microsoft Excel for statistical evaluation. The results were expressed as Mean $\pm$ Standard Deviation (SD). Statistical comparison between diabetic and non-diabetic groups was performed using the Student's t-test to determine the significance of differences in blood urea and serum creatinine levels. A p-value of less than 0.05 was considered statistically significant."

Result

Table 1 shows the distribution of samples with increased blood urea and serum creatinine levels among diabetic cases ($n = 24$) and non-diabetic controls ($n = 24$). In the diabetic group, 6 cases showed elevated blood urea levels, 7 cases had increased serum creatinine, and 11 cases exhibited elevation of both urea and creatinine. In contrast, none of the non-diabetic controls showed increased levels of either parameter. These findings suggest that renal function abnormalities were observed only among diabetic subjects, indicating a possible association between diabetes and impaired kidney function.

Table 1: Distribution of samples showing increased blood urea and serum creatinine levels in diabetic and non-diabetic subjects (N = 48)		
Parameters	Diabetic Cases (n = 24)	Non-Diabetic Controls (n = 24)
Blood Urea increased	6	0
Serum Creatinine increased	7	0
Both Urea and Creatinine increased	11	0

Table 2 compares the mean $\pm$ SD values of biochemical parameters between diabetic (n = 24) and non-diabetic subjects (n = 24). The fasting blood sugar level was significantly higher in diabetics (176.42 ± 31.58 mg/dl) compared to non-diabetics (92.37 ± 8.64 mg/dl). Similarly, post-prandial blood sugar was markedly elevated in diabetics (261.18 ± 39.74 mg/dl) compared with controls (124.51 ± 7.28 mg/dl). The HbA1c level was also higher in diabet-

ics ($6.74 \pm 0.45\%$) than in non-diabetics ($5.09 \pm 0.36\%$). Renal function parameters showed significant differences, with blood urea (61.25 ± 10.64 mg/dl) and serum creatinine (1.72 ± 0.58 mg/dl) being elevated in diabetics compared to non-diabetics (26.48 ± 4.97 mg/dl and 0.86 ± 0.15 mg/dl, respectively). All parameters showed high statistical significance ($p = 0.000$), indicating markedly altered biochemical profiles in diabetic subjects.

Table 2: Mean $\pm$ SD values of biochemical parameters in diabetic and non-diabetic subjects			
Parameters	Diabetics (n = 24) Mean $\pm$ SD	Non-Diabetics (n = 24) Mean $\pm$ SD	P value
Fasting Blood Sugar (mg/dl)	176.42 ± 31.58	92.37 ± 8.64	0.000*
Post-Prandial Blood Sugar (mg/dl)	261.18 ± 39.74	124.51 ± 7.28	0.000*
HbA1c (%)	6.74 ± 0.45	5.09 ± 0.36	0.000*
Blood Urea (mg/dl)	61.25 ± 10.64	26.48 ± 4.97	0.000*
Serum Creatinine (mg/dl)	1.72 ± 0.58	0.86 ± 0.15	0.000*

Table 3 shows the demographic distribution of the study participants (N = 48), consisting of 24 diabetic cases and 24 non-diabetic controls. Among the diabetic group, 15 were males and 9 were females, while the control group included 14 males and 10 females, resulting in a total of 29 males and 19 females in the study. With respect to age distribution,

13 participants were in the 35–45 years age group, 17 in the 46–55 years group, and 18 in the 56–65 years group, indicating that the majority of participants belonged to the middle-aged and older adult population. Overall, the demographic characteristics were relatively comparable between the diabetic and non-diabetic groups.

Table 3: Demographic distribution of study participants (N = 48)			
Gender	Diabetic Cases (n = 24)	Non-Diabetic Controls (n = 24)	Total
Male	15	14	29
Female	9	10	19
Age Group (years)			
35–45	6	7	13
46–55	9	8	17
56–65	9	9	18

Discussion

The study involved 48 participants with 24 diabetic patients and 24 non-diabetic controls. The results of the current study indicated significantly high levels of blood urea (61.25 ± 10.64 mg/dl) and serum creatinine (1.72 ± 0.58 mg/dl) in diabetes patients compared to non-diabetic controls with the respective mean values of 26.48, 0.86. Sirivole and Eturi, (2017) experienced similar results and found that the blood urea and creatinine concentrations were much higher in diabetic subjects than in non-diabetic participants in a research design that was conducted in Telangana, India. According to them, the mean level of blood urea of diabetic subjects was reported to be about 55-60 mg/dl and the serum creatinine was about 1.6 mg/dl which is quite similar to the present

study and thus confirms the relationship between diabetes and compromised renal functioning [2].”

The observed elevated fasting blood sugar (176.42 ± 31.58 mg/dl) and post-prandial blood sugar (261.18 ± 39.74 mg/dl) of the diabetic subjects in the current study show that glycemic control is not good which is a critical factor in the development of diabetic nephropathy. Glycemic control is poor, which results in longer periods of hyperglycemia, resulting in structural and functional changes of renal tissues. A comparable situation was also observed in a study made by Chutani and Pande (2017) [7], who reported that the elevation of blood urea and serum creatinine levels were strongly associated with poor glycemic status and extended diabetes. The research was able to show a mean serum creatinine of more

than 1.5 mg/dl in uncontrolled diabetic patients, which can be compared to the average creatinine of 1.72 mg/dl in the current study. This resemblance indicates that chronic hyperglycemia plays an important role in progressive kidney injury of diabetic patients.

Besides the blood glucose values, glycated hemoglobin (HbA1c) is also a significant measure of long-term glycemic control. The average HbA1c value of diabetic patients and non-diabetic controls was 6.74 and 5.09 respectively with a standard deviation of 0.45 and 0.36 respectively in the current study. High HbA1c indicates the continued presence of hyperglycemia and the formation of risk factors, such as nephropathy. Similar results have been noted by Abdelsalamka and Elamin, (2011) [3] who showed that there is a positive correlation between the levels of HbA1c and blood urea in type 2 diabetes patients. Their study revealed that patients who had a higher level of HbA1c of more than 6.5% exhibited a substantial rise in the level of urea, indicating that renal functionality declined as the level of glycemic regulation deteriorated. These results are similar to the results of the current study in which there was a correlation between the higher levels of HbA1c and the higher levels of renal markers.

It was also identified in the present research that 6 diabetic participants had high levels of blood urea, 7 high levels of serum creatinine and 11 subjects had high levels of both parameters, but none of the non-diabetic subjects had abnormal levels. This observation is a strong pointer that the abnormalities of renal functions were mainly linked to diabetes among the population under study. The same findings were noted in experimental research by Anjaneyulu and Chopra (2004) who showed elevated levels of urea and creatinine in diabetic rats as a result of progressive renal impairment. Their results justified the fact that hyperglycemia causes oxidative stress and structural injury in nephrons, which results in a decreased glomerular filtration rate and the process of retrieving nitrogenous waste products in blood [15].

The pathophysiology of the increased urea and creatinine in diabetes is mostly associated with the destruction of the glomerular filtration system. Chronic hyperglycemia causes thickening of the glomerular basement membrane and expansion of mesangium which in the end lines up the filtration capacity of the kidneys. Creatinine is believed to be an indirect measure of glomerular filtration rate (GFR), and a rising serum creatinine level is a sign of renal failing. These mechanisms have been reported to be similar in giant epidemiological studies on chronic kidney disease in diabetic patients. Indicatively, Atkins (2005) [9] indicated that diabetes is among the major causes of chronic renal kidney disease in the whole world and has been linked to gradual deterioration of the renal filtration capacity.

A different population-based study by Whaley-Connell et al., (2009) [8] in the Kidney Early Evaluation Program helped to point out that diabetic patients often present with high serum creatinine and low renal function as compared to non-diabetic persons. Their results indicated the significance of the biochemical screening every now and then to identify early kidney damage among diabetic patients. The current research is in line with these reports, as the renal functional markers were significantly higher in diabetic patients than in the healthy controls even in a relatively small sample size.

The current study population was characterized by a slightly a greater number of males than females, diabetic and non-diabetic. The presence of similar gender ratios has been observed in most of the epidemiological studies conducted on diabetes with males usually constituting a larger number of cases diagnosed. The epidemiological research regarding the occurrence of diabetes by Adeghate et al., (2006) [6] also revealed that the prevalence of diabetes predisposes to metabolic and kidney complications among the middle aged and the geriatric age groups. These observations are in line with the age distribution of the current study, the majority of whom are within the 46-65 years old age group.

Generally speaking, the results of the current research aligning with the existing literature demonstrate that diabetic patients have a much higher urea level in their blood and serum creatinine level than non-diabetic patients do. These high concentrations indicate compromised renal filtration because of hyperglycemia over a long period and gradual nephron loss. Despite the fact that intensive glycemic control is able to lower the levels of HbA1c and decrease the rate of complications, structural kidney damage might not always be reversible once it has taken place. As such, early detection and treatment of diabetic nephropathy require regular follow-up of renal functioning parameters, including blood urea and serum creatinine. The findings of this paper also confirm the significance of these biochemical parameters as significant prognostic factors of the presence of renal impairment in diabetic patients (Chutani & Pande, 2017; Aldler et al., 2003) [7,16].

Conclusion

The current research concludes that diabetic patients have an evident inclination towards an increase in renal biochemical indicators as opposed to non-diabetic people. Higher percentages of diabetic subjects had higher levels of blood urea and serum creatinine, and the same was not true of the non-diabetic controls. The mean values of glycemic indicators and renal function parameters were also significantly higher in the diabetic group, indicating a strong association between diabetes and impaired kidney function. The demographic distribution showed that both genders and various middle-to-older age

groups were represented in the study population. Overall, the findings suggest that diabetes has a notable impact on renal function, highlighting the importance of regular monitoring of blood urea and serum creatinine levels in diabetic patients for early detection and prevention of potential kidney complications.

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