Physical Education, Health and Social Sciences

https://journal-of-social-education.org

E-ISSN: 2958-5996
P-ISSN: 2958-5988

Comparative Analysis of Renal Parameters in Diabetic Vs Non-Diabetic Patients

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DOI: https://doi.org/10.63163/jpehss.v3i2.271

Abstract

Objective: To find out comparative analysis of renal parameters in diabetic vs. non-diabetic patients.

Methodology: The present study was conducted by reviewing the medical records of 140 renal patients who affected with and without diabetes at AL Noor Lab & digital X-Ray center from October 2024 to March 2025. Biochemical tests will include renal function tests (serum creatinine and blood urea) and glucose test (fasting, random and HbA1c) conducted to provide a comprehensive biochemical profile for each participant.

Results: This research examined 126 blood samples for blood glucose, urea, and serum creatinine levels by employing standard biochemical techniques. The study population consisted of 51.6% males and 48.4% females in three age groups: 18-33, 34-49 and 50+ years. Statical analysis did not show any significant difference between normal and abnormal values of these parameters ($p > 0.05$). Thus, hypothesis was rejected, which means the values were statistically insignificant.

Conclusion: This research points out that diabetes negatively impacts renal functions, with creatinine levels deviating more than urea. Screening and glycemic control are essential in controlling diabetic nephropathy.

Key words: Blood Glucose, Blood Urea, Serum Creatinine, Hyperglycemia, Renal Function

Introduction

Hyperglycemia caused by failures in insulin production, insulin action. Organs such as the eyes, kidneys, nerves, heart, and blood arteries are particularly vulnerable to the long-term damage, malfunction, and failure caused by diabetes.[1]

One of the leading causes of mortality in individuals with type 1 or type 2 diabetes mellitus (DM) who do not maintain long-term optimal glycaemic control is diabetic kidney disease (DKD), formerly known as diabetic nephropathy (DN). Because of the injury, the kidneys are unable to filter blood properly. Swelling of the ankles, nausea, weakness, and difficulty breathing are some of the symptoms that may develop as a result of the accumulation of waste materials and fluids in the body.[2]

Chronic kidney disease (CKD), sometimes called diabetic nephropathy, is more common in people with diabetes. About one-third of individuals with DM2 may develop diabetic kidney

disease (DKD), making it one of the most common and harmful consequences of the condition. Because of their involvement in gluconeogenesis and tubular reabsorption of glucose, the kidneys join the pancreas, adipocytes, liver, and intestines as key players in glycaemic regulation.[3]

An ever-increasing number of people are living with diabetes mellitus (DM). The group of metabolic diseases is diverse, but they all have the symptom of hyperglycemia, which may be caused by either an inability to secrete insulin or a reduction in the tissues' sensitivity to insulin. The kidney illness that develops in people with diabetes is known as diabetic nephropathy. Diabetes mellitus type 1 and type 2 have many similarities, including kidney pathology, clinical progression, and the likelihood of developing nephropathy.[4]

A severe consequence of diabetes mellitus, diabetic nephropathy (DN) is the primary cause of end-stage renal disease (ESRD) on a global scale. Maintenance dialysis patients with diabetes had a worse chance of survival compared to non-diabetic patients with end-stage renal disease (ESRD) because of the prevalence of other renal disorders. Geographical variations and different selection criteria for renal biopsies contribute to the large range of reported cases of non-diabetic renal disease (NDRD). The histological results of renal biopsies taken from diabetic patients have shown that around a third of these instances show pure diabetic nephropathy, another third show a nondiabetic condition, and a third show diabetic nephropathy with a superimposed illness. Proteinuria without diabetic retinopathy, active urine sediment, quickly declining renal function, and a short term of diabetes are some of the unusual clinical characteristics that have been shown to foretell renal involvement by NDRD in diabetic individuals. It is still difficult to distinguish between DN and NDRD in individual individuals without renal biopsy because of the wide range of clinical manifestations and the prevalence of complicating medical conditions in this group. With the use of clinical and laboratory data, this research aims to investigate the causes of biopsy-proven NDRD in the Thai population and to compare the diagnostic accuracy of NDRD with or without DN to that of isolated DN in patients with type 2 diabetes.[5]

People with Diabetes: Upwards of 33% get renal disease. About 14% of patients who do not have diabetes are impacted. Rates of end-stage renal disease (ESRD) from diabetic nephropathy varied between 38.4 and 804.0 per 100,000 person-years, whereas rates of ESRD from all causes ranged between 132.0 and 167.0 per 100,000 person-years in individuals with prevalent diabetes. Compared to the general population, those with diabetes had a greater incidence of end-stage renal disease (ESRD), and the relative risks ranged from 6.2 for whites to 62.0 for Native Americans.[6]

Type 2 diabetes mellitus (DM) has been the leading cause of the dramatic rise in both the incidence and prevalence of DM globally. Between 18% and 20% of persons over 65 have type 2 diabetes worldwide. Nearly 285 million individuals, spanning the ages of 20 to 79, are living with diabetes mellitus (DM). Among them, 70% reside in nations with middle- or low-income levels. The public health care system is facing tremendous problems as a result of the disproportionate impact of this development in type 2 diabetes mellitus (DM2) on emerging nations. Without preventative efforts, this figure is projected to rise by almost 50% in the next 20 years. Diabetes will affect an estimated 438 million adults (or 8% of the total) by the year 2030.[7]

In recent decades, diabetes mellitus (DM) has emerged as a major issue in global health. With an 8.3% prevalence in 2014—equating to 387 million patients—the worldwide incidence of diabetes mellitus has risen dramatically in recent years. Diabetic end-stage renal disease (ESRD) is thought to be a common cause of chronic renal replacement therapy (RRT) in Western nations. According to published research, the percentage of patients with diabetes among those who began RRT varied between 24% (when just diabetic nephropathy was included) and 51% (when all causes were considered). There will be serious personal and societal repercussions for a rise in the incidence of end-stage renal disease (ESRD) due to the rising rates of obesity and diabetes and the better prognosis for diabetic patients. People with

diabetes have a bad outlook since end-stage renal disease (ESRD) is a potentially fatal consequence of the disease. Combining end-stage renal disease (ESRD) with diabetes increases the risk of cardiovascular events, according to epidemiological research. This population has a lower life expectancy due to the high prevalence of cardiovascular disease. Compared to ESRD patients without diabetes, published research show that ESRD patients with diabetes have an increased death risk. And there are a lot of medical bills associated with treating end-stage renal disease. In 1989, the St. Vincent Declaration set a 5-year target of reducing end-stage renal disease (ESRD) caused by diabetes by at least one third. Many epidemiological studies on the prevalence of end-stage renal disease (ESRD) among diabetics have been carried out since then. The presented data show that the incidence of end-stage renal disease (ESRD) varies greatly, particularly when looking at trends across time. It is also difficult to compare the research because of the substantial differences in study population and design. Very few non-systematic reviews have addressed this issue; those that have either looked at the prevalence of end-stage renal disease (ESRD) in the diabetic community relative to the general population or were published before the year 2000. So yet, no comprehensive evaluation has been carried out. Due to the paucity of data, we have undertaken the first comprehensive study to evaluate the rates of end-stage renal disease (ESRD) among people with diabetes versus those without the disease.[8]

Hyperglycemia in uncontrolled diabetes is associated with an increase in blood urea and creatinine, two biomarkers that are known to be associated with the degree of kidney damage in diabetic nephropathy. For this aim, there are readily accessible tests that measure blood urea and creatinine levels. These tests may help diagnose diabetic kidney disease early on and prevent it from progressing to end-stage renal disease (ESRD). Regularly, skeletal muscle releases creatinine phosphate, which is broken down into creatinine. There is a strong relationship between serum creatinine and the percentage of body mass that is skeletal muscle. At low GFRs, the typical reciprocal connection breaks down, and creatinine tends to underestimate how low the GFR has fallen since it is filtered by the glomerulus and secreted into the glomerular filtrate by the proximal tubule. Because of their higher GFR values, patients with early-onset diabetes mellitus are a good group to research to see how renal declines function with time.[9]

Along with the malfunctioning of β and α cells, patients with DM2 also have an altered incretin system. Insulin secretion and other positive effects mediated by the GLP-1 receptor are also impacted by incretin deficiency. This lends credence to a number of recently suggested DM2 pharmaceutical treatments that target incretin efficacy. When it comes to controlling blood sugar levels, the kidneys are just as vital as the pancreas, adipocytes, liver, and intestines. Renal gluconeogenesis plays a major role in glucose regulation via tubular reabsorption of glucose. Research on the kidney's function in regulating blood sugar levels began in 1938 with animal research, and human investigations into this topic didn't begin until the late 1950s. Several hormones have a role in controlling the process of glucose reabsorption by the kidneys; for example, hyperinsulinemia inhibits the release of glucose from the kidneys in the same way it does in the liver. Epinephrine infusion, on the other hand, enhances renal glucose release, while glucagon has no influence on this action. Despite a lack of human data, many investigations have shown that growth hormone and cortisol may increase glucose release from the kidneys.[10]

Changes in renal anatomy and function are the hallmarks of DKD. The glomerular and tubular basement membranes thicken, glomerular sclerosis occurs, and mesangial enlargement occurs as major structural alterations in the kidneys in DKD. In most cases, a clinical condition characterised by chronic albuminuria, hypertension, a gradual decline in glomerular filtration rate (GFR), an increase in cardiovascular events, and death from these events is the clinical manifestation of DKD.[11]

It may not seem complicated, but the kidneys filter blood past the glomerular filtration barrier into Bowman's space to create primary urine that is almost protein-free. This process begins with blood entering the glomerular tuft via the afferent arteriole and perfusing the glomerular

capillaries. The efferent arteriole carries blood out of the tuft, while the renal tubules receive the main urine. The existence of a highly specialised structure, the glomerular filtration barrier (GFB), is crucial for the efficacy of this filtering process. Water, small and medium solutes, and low-molecular-weight proteins up to albumin mass can pass freely through this filtration barrier, allowing for highly selective ultrafiltration of blood plasma. However, proteins with masses greater than 60-70 kDa, particularly those with negative charges, are largely prevented from passing through. Albumin, for instance, often has an estimated sieving coefficient below 0.0005, which means that less than 0.05% of plasma albumin makes it past the GFB and into the urine.[12]

Among the several causes of ESKD, diabetes mellitus (DM) stands out. It is debatable, however, whether diabetic nephropathy (DN) patients have worse renal outcomes than diabetic individuals with non-diabetic renal disease (NDRD). In this prospective observational analysis, we aimed to compare individuals with non-DM chronic kidney disease (CKD) to those with DN, DM, and NDRD.[13]

Several factors contribute to the development of DKD, such as oxidative stress, changes in renal haemodynamics, glomerular hypertension, hypoxia and ischaemia, and an overactive renin-aldosterone system. Unfortunately, treatment targets have not been identified, and the whole pathophysiology of the illness is still unknown. For this reason, treating DKD is identical to treating traditional diabetic nephropathy.[14]

Hyperglycemia stimulates the production of humoral mediators, cytokines, and growth factors by both resident and nonresident renal cells. These alterations result in both structural changes, like an increase in ECM deposition, and functional changes, like shear stress or an increase in glomerular basement membrane permeability. These changes have a role in diabetic nephropathy. Inhabitant renal cells have a surface receptor called GLUT-1 that regulates glucose inflow. The overexpression of GLUT-1 mRNA and overproduction of GLUT-1 protein in mesangial cells were shown by Heilig et al. to occur in vitro at high glucose concentrations ranging from 23 to 30 nM. Furthermore, cellular glucose transport was enhanced. The expression of GLUT-1 is regulated by TGF-β1. The fact that this growth factor regulation depended on both time and dosage was actually shown by Inoki et al. Reductions in GLUT-1 mRNA expression and D-glucose absorption were seen in vitro with addition of an anti-TGF-β1 monoclonal antibody. Ultimately, the activity of endogenous TGF-β1, which is generated by mesangial cells in a high-glucose culture, may boost glucose transport and promote glucose absorption via the activation of GLUT-1 overexpression in both mRNA and protein. As a result, it hastens the onset of metabolic abnormalities in mesangial cells caused by glucose.[15]

The classic clinical presentation of DKD is a consistently elevated albuminuria level (>300 mg/g creatinine) and a decreased estimated glomerular filtration rate (eGFR). In individuals with type 1 diabetes who have had the disease for more than ten years, retinopathy, albuminuria (the lack of blood in urine), and a slow but steady decline in glomerular filtration rate (GFR) are the hallmarks of diabetic ketoacidosis (DKD). But the variation in symptoms' manifestations has been increasingly noticeable in recent years.[16]

The screening process allows for the early detection of chronic kidney disease (CKD), counselling, pharmacologic management, and, if necessary, referral to a nephrologist. The American Diabetes Association (ADA) recommends using kidney-protective medicines and maintaining appropriate control of blood sugar and blood pressure. Rationale: renin-angiotensin system inhibitors, sodium-glucose cotransporter-2 inhibitors, glucagon-like peptide 1 receptor agonists, and, more recently, non-steroidal mineralocorticoid receptor antagonists have all been shown to slow down the course of chronic kidney disease. In addition to the presently suggested treatments, there are novel drugs in development with alternative action mechanisms that might further delay or halt the course of the illness.[17]

Different approaches are used to treat DN and NDRD. Apart from the conventional ACEIs and ARBs, immunosuppressants have the potential to treat a wide variety of NDRD lesions. Therefore, differentiating NDRD from DN at an early stage is crucial. Although invasive, a

kidney biopsy is required for diagnosis. A renal biopsy may be risky for patients with DM, leading nephrologists to be hesitant to do the procedure. Possible complications include haematuria, perirenal haematoma, arterial embolisation, and the need for a nephrectomy. The presence of cortical atrophy or a single kidney are two additional reasons why renal biopsy should not be performed. Furthermore, the kidney biopsy is currently not available at many main hospitals. Because of this, nephrologists need to make a diagnosis based on the available clinical and laboratory data before they may do a biopsy. Using logistic regression analysis, Zhou et al. developed a diagnostic model that achieved high levels of sensitivity (90%) and specificity (92%). Results were only statistically significant for the following variables: duration of diabetes, systolic blood pressure (SBP), glycosylated haemoglobin (HbA1c), hemoturia, and diabetic retinopathy (DR). Until renal histology is accessible, other research has only identified characteristics that differentiate NDRD from DN. It is necessary to conduct a systematic evaluation of published findings since the results were inconsistent, most likely as a consequence of variations in the research populations or selection criteria. So, to find out how clinical and laboratory data can help distinguish between NDRD and DN in type 2 diabetic patients, we analysed case-control studies.[18]

Most cases of kidney failure in the elderly are due to diabetic kidney disease (DKD). Nevertheless, DKD is only effectively treated with a combination of renin-angiotensin system inhibitors and interdisciplinary approaches. A new treatment option was added to the list in 2019 when the Canagliflozin and Renal Events in Diabetes with Established Nephropathy Clinical Evaluation (CREDENCE) study demonstrated that sodium-glucose cotransporter 2 (SGLT2) inhibitors were effective against DKD. Nevertheless, the advancement of DKD remains uncontrolled. Even when their blood glucose levels return to normal, people who have brief episodes of hyperglycemia still acquire diabetes complications, such as DKD. Accumulations of advanced glycation end products (AGEs) and epigenetic alterations serve as metabolic memory in response to transient hyperglycemia. Both fundamental and clinical research efforts are focused on developing AGE and histone modification inhibitors as potential medications to enhance metabolic memory. Furthermore, several ongoing clinical studies with renal outcomes as their main goal have shown a reno-protective potential in incretin-related medicines. Because they enhance tubulointerstitial hypoxia, the newly authorised hypoxia-inducible factor prolyl hydroxylase inhibitors for renal anaemia may have reno-protective effects. Moreover, in Bardoxolone Methyl, individuals with DKD saw an improvement in their glomerular filtration rate when given NF-E2-related factor 2 activators.[19]

Controlling blood sugar levels, lowering blood pressure, controlling lipids, and quitting smoking constitute the standard approach to diabetes treatment. Patients at risk of developing diabetic nephropathy should continue to have glycaemic management as their primary therapeutic goal, as hyperglycemia is the primary cause of the renal level structural abnormalities. To lessen the likelihood of microvascular problems, intensive management of blood glucose levels is recommended. Also, since life expectancy increases, treating blood glucose levels early in juvenile diabetics has a tremendous impact on survival. Diabetes nephropathy, retinopathy, neuropathy, and cardiovascular disease were all shown to be 50% less common in patients whose blood glucose levels were tightly controlled with insulin or sulfonylureas, according to two studies. A multifactorial intervention research by Gaede et al. found a 50% reduction in the risk of microvascular and cardiovascular events.[20]

Material and Methods

A total number of 126 samples from AL Noor Lab & Digital X-Ray were collected within a time period of 6 months from October 2024 to March 2025 in which 63 are confirmed diabetic and 63 are non-diabetic for this retrospective cross-sectional study. All samples were collected to perform Renal Function Test (Creatinine & Urea), Blood Sugar Fasting & Random. Final diagnosis was done by HbA1c test. Data analysis was carried out using the latest version of IBM's SPSS Statistics, version 20.0. Qualitative factors are shown as percentages, whereas

quantitative data are represented as means and standard deviations. A t-test was used for categorical variables in the univariate comparisons across groups, and a one-way analysis of variance test was used for comparing means. Logistic regression method was used to do a multivariate analysis of the dependent variables, which are those that may be indicators of diabetes vs. non-diabetic kidney disease.

Results

One hundred twenty-six blood samples were tested for this study by the biochemistry lab. As a whole, the blood was tested for glucose, urea, and creatinine levels. To estimate blood glucose levels, the GOD-POD method was used. We measured blood urea using Berthelot's technique and serum creatinine using alkaline Jaffe's Picrate method. Table 1 shows that out of the total sample, 51.6% were male and 48.4% were female. People in the age bracket of 18–33 made up 33.33% of the sample, those in the age bracket of 34–49 comprised 26.08%, and those in the age bracket of 50 and over made up 39.59%, as shown in Table 2 and described in Figure 2.

Some descriptive statistics for biochemical parameters are as follows: With a mean of 2.4590 and a standard deviation of ±135.82, the diabetes readings ranged from 85 to 590. With an average of 55.2929 and a standard deviation of ±36.068, the urea levels ranged from 12 to 210. Creatinine levels varied from 0.2 to 65, with an average of 2.9635 and a standard deviation of ±6.1286. The distribution of the data is summarised by these statistics for each parameter in Table 3.

A separate t-test was conducted to determine if the parameters (diabetes, urea, and creatinine) show a statistically significant difference between the normal and abnormal levels. Since Levene's test for equality of variances yielded an F-significance value greater than 0.05, values under "equal variance assumed" were used. According to Table 4, the two-tailed significance values exceeded 0.05. This leads us to conclude that the hypothesis cannot be supported since neither the normal nor the abnormal levels are statistically significant.

Table 1: Gender frequency distribution.

Gender	**Frequency**	**Percent**
Female	61	48.4%
Male	65	51.6%
Total	**126**	**100.0%**

Figure 1: Gender Distribution Graph

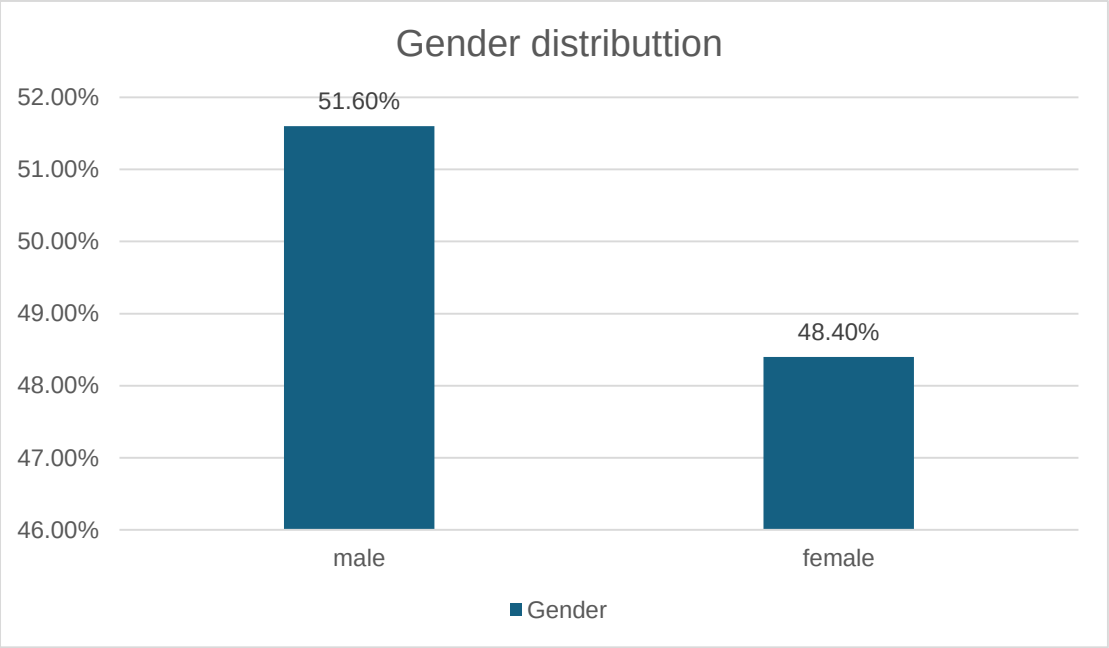
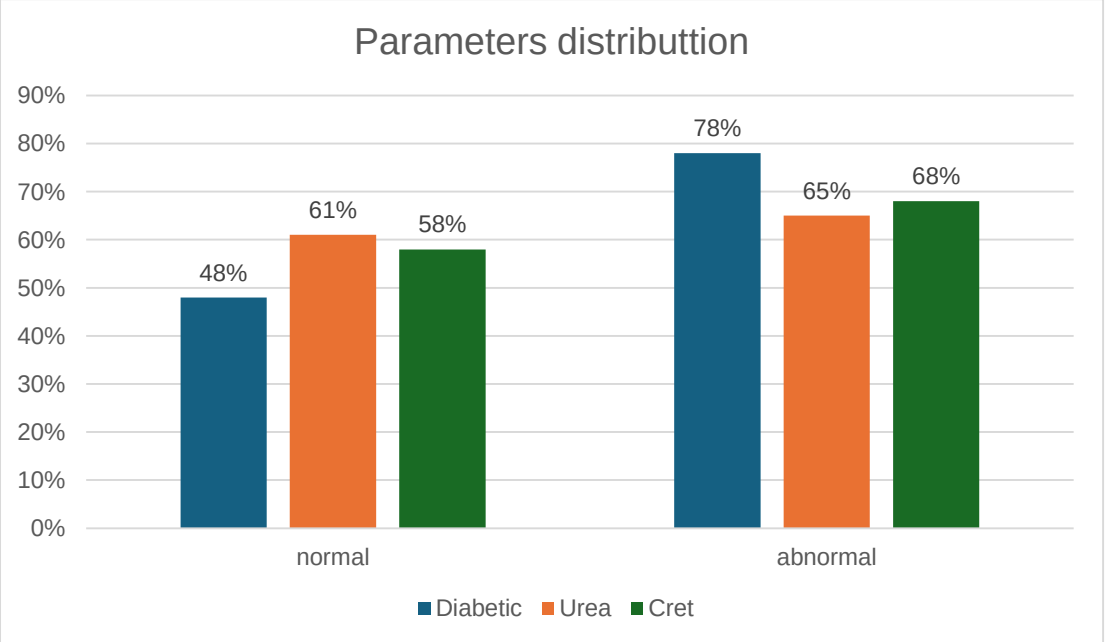


Table 3: Descriptive Statistics: SD in Glucose and Renal parameters.

Parameters	N	Minimum	Maximum	Mean	SD
Glucose	126	85.00	590.00	2.459042	135.82085
Urea	126	12	210.00	55.2929	36.06381
Creatinine	126	0.2	65.00	2.9635	6.12864

Figure 3: Normal and Abnormal Values of Parameters.



Discussion

The findings of the study highlight critical observations regarding the variation in urea and creatinine levels in diabetic patients across different hyperglycemic categories. The data clearly indicate that as blood glucose levels increase, there is a corresponding rise in percentage variation of creatinine levels, which significantly exceeds the variation observed in urea levels. This suggest that creatinine is a more sensitive marker for renal function impairment in hyperglycemic states compared to urea.[21]

In the first hyperglycemic category (130-190 mg/dl), a notable percentage of samples showed variations in both urea (2.22-17.77%) and creatinine (7.14-71.42%) levels, with creatinine exhibiting a greater range of deviation. This trend persisted in the second category (190-260 mg/dl), where creatinine variation (14.28-78.57%) again exceeded that of urea (4.44-22.22%). The trend peaked in the highest hyperglycemic category (260-390mg/dl), where creatinine exhibited a striking percentage variation (85.71-100%), while urea maintained relatively minor fluctuations (6.66-26.66%).

These findings reinforce the hypothesis that hyperglycemia contributes significantly to kidney dysfunction, with creatinine levels being disproportionately affected compared to urea.[22]

Moreover, the rejection of the hypothesis in the t-test analysis further suggests that normal and abnormal levels of diabetes, urea, and creatinine do not differ significantly with in the studied population. This statical in significance could point to the need for a larger sample size or the inclusion of additional variables to capture subtle differences.

The present study aligns with prior research emphasizing the importance of renal function tests (RFTs) in diabetic care and underscores the need for focused monitoring of creatinine levels in hyperglycemic patients to detect potential renal complications.[23] However, the study also highlights gaps in the availability of comparative data on diabetic or non-diabetic renal disease in the Pakistani population.

Bauza and Mosquera (2003) identified hyperglycemia as a key driver of progressive renal damage.[24] Anjaneyulu et al. (2004) demonstrated that diabetic rats exhibiting significantly higher urea and creatinine levels, indicating progressive renal damage.[25] Gungor et al. (2006) supported these findings, reporting substantially elevated creatinine levels in diabetic patients compared to non-diabetic controls.[26] Furthermore, Kamal A et al. (2014) emphasizing the role of hyperglycemia in diabetic nephropathy through mechanisms like hyperfiltration and increased glomerular filtration rate (GFR). These processes result in microvascular damage, worsening renal impairment.[27]

Conclusion

This study demonstrates that diabetes significantly influences renal parameters, serum creatinine being more distant from normal range compared to blood urea. Raised levels of abnormal urea and creatinine along with blood glucose reflects abnormal kidney function in diabetic subjects. Diabetes mellitus incidence among human population has been reaching epidemic rates across the globe, keeping stringent control on blood glucose may aid in its delaying the disease progression. It is necessary to develop effective strategies for the prevention of diabetes and its complications. Blood urea and serum creatinine routine screening can help with early detection of pre-renal complications, rendering valuable prognostic benefits in the management of diabetic nephropathy globally. Further studies are required to evolve the understanding of this association.

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