Study Effect Serum Urea and Creatinine in Diabetic Mellitus Patients with Kidney Failure

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Abstract

Background: Diabetes has reached epidemic proportions, significantly impacting global health and the economy. The number of people with diabetes is projected to increase from 415 million (8.8%) in 2015 to 642 million (10.4%) by 2040. Diabetes mellitus (DM) is a group of metabolic disorders of carbohydrate metabolism in which glucose is not adequately utilized, resulting in hyperglycemia. Various statistics have led to diabetes being described as one of the major threats to human health in the 21st century. Diabetes is a serious condition marked by persistently high blood sugar levels, resulting from disruptions in how the body processes carbohydrates, fats, and proteins. This disease arises from either a complete or a partial inability to produce insulin or from the body's ineffective response to it. Alarmingly, diabetes stands as the foremost cause of kidney disease and premature death, with diabetic nephropathy representing a dangerous stage of chronic kidney failure. It is crucial to understand the profound implications of diabetes to prioritize prevention and management effectively. Diabetic nephropathy is a global and public health problem, with 20–40% of diabetic patients developing diabetic kidney disease. Diabetic nephropathy is a progressive kidney disease that develops, peaking after 10-20 years of diabetes. Plasma urea and creatinine are widely considered kidney function tests and are used for two different purposes. The first is to diagnose impaired kidney function, and the second is to detect the presence of progressive loss of kidney function. In diabetic nephropathy, serum urea and creatinine levels rise with uncontrolled hyperglycemia and indicate the severity of kidney damage. These readily available tests are crucial for early detection and prevention of diabetic kidney disease, helping to slow progression to end-stage renal disease. Regular screening is essential for managing kidney health in diabetic patients.

Aim: study the effect of serum urea, serum creatinine, age, sex, and BMI in diabetic mellitus with kidney failure.

Method: The study design was used to describe the variables and the relationships that occur between them in order to achieve the objectives of the current study which studied the effect of urea and creatinine in blood serum of diabetic patients with renal failure in Baquba Teaching Hospital - Haemodialysis Unit. The researcher used the questionnaire shown in the appendix to collect data and information from the study subjects. The study included 80 cases. Laboratory materials from Medical System Biotechnology Co., Ltd. were used to perform biochemical tests.

Results: The study included 50 patients with diabetes and kidney failure, aged between 27 and 85 years, with an average age of 61.82 years. The control group included 30 people, aged between 10 and 70 years, with an average age of 46.3 years. The study cases were divided into groups according to age, sex, family history of the disease, and BMI. The biochemical variables were divided into three groups (hyper value, normal value, and hypo value) according to the Mean ± SD values. The highest percentage (76%) was observed in patients with a family history of the disease. While all biochemical variables increased for most patients, HbA1c levels remained normal, suggesting that familial risk factors may not influence glucose control in the same way.

Conclusion: The present study concluded that most of the patients had a family history of the disease, and most of them were elderly, over 45 years of age. The results showed that the values of biochemical variables were high for the patients and statistically significant at $p < 0.05$ compared to the control group. The blood hemoglobin values for all patients were low, which indicates that patients with diabetic nephropathy suffer from anemia.

Keywords: Hyperglycaemia, Diabetic Mellitus "DM", Kidney Failure, Nephropathy, Serum Urea, Serum Creatinine, Blood Sugar, Hb, BMI, HbA1c.

1. Introduction

Diabetes mellitus is defined by chronic hyperglycemia, marked by elevated blood glucose levels resulting from disruptions in carbohydrate, fat, and protein metabolism. This condition may arise from either an absolute or a relative deficiency in insulin secretion, insulin action, or both [1]. Diabetes has reached epidemic proportions, significantly impacting global health and the economy. The number of people with diabetes is projected to increase from 415 million (8.8%) in 2015 to 642 million (10.4%) by 2040. [2] (see Figure-1).

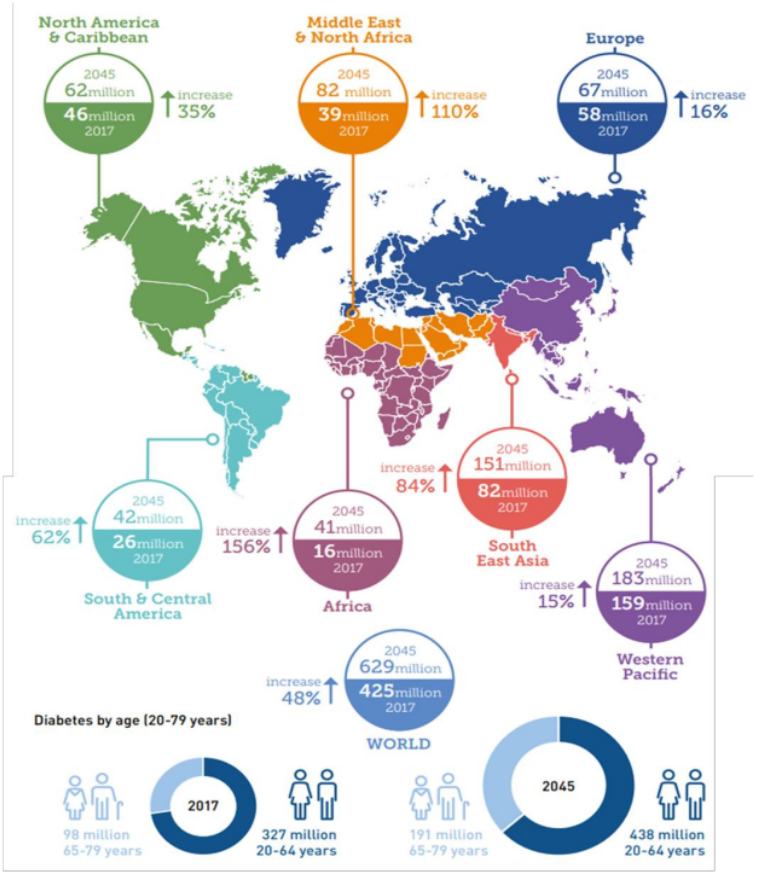


Figure-1: Number of people with diabetes in the world [3].

Diabetes is fundamentally categorized into two types: type I and type II. Type I diabetes primarily stems from idiopathic causes or autoimmune disorders, necessitating individuals to rely on exogenous insulin for survival. This essential therapy not only prevents life-threatening ketoacidosis but also allows those affected to lead normal lives [1].

On the other hand, type II diabetes arises when the beta cells of the pancreas fail to produce sufficient insulin or when the body's cells become resistant to insulin's effects. This resistance occurs in key areas such as muscle, liver, and fat tissue, significantly hampering the management of blood sugar levels. While many people with type II diabetes can initially maintain control through diet and oral hypoglycaemic agents, some may eventually require insulin to effectively regulate their blood glucose levels. By understanding these distinctions and the critical nature of diabetes management, we can empower those living with this condition to take proactive steps in their health journey [4]. Insulin secretion from beta cells in the pancreas plays a crucial role in maintaining normal glucose levels by reducing glucose production from the liver and promoting glucose uptake into skeletal muscle and adipose tissue. However, when beta cells malfunction or insulin resistance occurs in these tissues, hyperglycemia can develop, leading to high levels of glucose in the bloodstream. Many factors can affect insulin secretion and action (see Figure-2), ultimately contributing to the development of conditions such as type II diabetes [5].

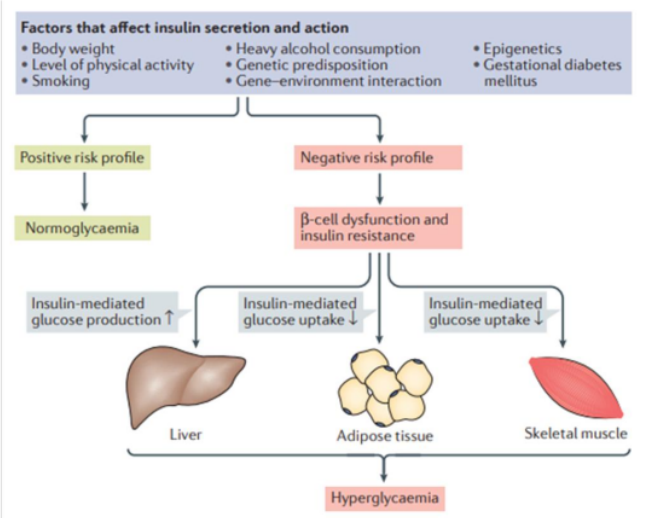


Figure-2: pathophysiology of hyperglycemia in Type II Diabetes mellitus [5].

Diabetic nephropathy is an important cause of morbidity and mortality in both type I and type II diabetes. It is now the most common cause of end-stage renal failure in developed countries and accounts for between (20-50)% of patients starting renal replacement therapy [6], Diabetic nephropathy impacts a staggering 30 percent of individuals living with diabetes and stands as the foremost cause of end-stage kidney disease. This alarming statistic underscores the critical need for awareness and proactive management of diabetes to protect kidney health and improve quality of life [7].

The growing prevalence of diabetes is largely driven by rising obesity rates (known as body mass index "BMI"), as seen in the United States and China. Conversely, Cuba experienced a notable decline in diabetes rates in the early 1990s when nationwide body weight decreased due to an economic crisis, demonstrating the strong link between obesity management and diabetes prevention [5]. Evaluating kidney function serves two key purposes: diagnosing impaired function and monitoring progressive loss. In diabetic nephropathy, serum urea and creatinine levels rise with hyperglycemia in uncontrolled diabetes, indicating the severity of kidney damage. Measuring these biomarkers is a readily available test that helps detect and prevent diabetic kidney disease early, reducing the risk of progression to end-stage renal disease (ESRD) [8,9]. Creatinine, a waste product from creatinine phosphate in skeletal muscles, is released at a constant rate. Its serum levels reflect muscle mass and are filtered by the glomerulus, with a small amount secreted by the proximal tubule. However, when GFR is low, this correlation can break down, causing serum creatinine levels to underestimate the reduction in GFR [5,10,11]. Anaemia is common in diabetic patients with CKD [12]. One in five diabetic patients with chronic kidney disease (CKD) has anemia, with severity increasing in advanced stages. Key causes include iron deficiency, erythropoietin deficiency, and a poor response to erythropoietin [13].

Aim of the Study

Study the effect of serum urea, serum creatinine, age, sex, and body mass index “BMI” in diabetic mellitus with kidney failure.

2. Methods

2.1. Design of the study: the study design was used to describe the variables and the relationships that occur between them in order to achieve the objectives of the current study which studied the effect of urea and creatinine in the blood serum of diabetic patients with renal failure in Baquba Teaching Hospital - Hemodialysis Unit during the period from 15 October 2024 to 25 December 2024.

2.2. Administrative and ethical permission: the college has obtained permission from the college to conduct the study. Research objectives and benefits were explained to samples personally.

2.3. Data collection: the researcher used the questionnaire shown in the appendix to collect data and information from the study subjects. The study included 80 cases (50 patients and 30 controls) with ages 10–85 years. Out of 30 control subjects, 15 were female and 15 were male, whereas among patients, 24 were females and 26 were males.

2.4. Statistical Data Analysis: several statistical measures were used by using Statistical Package of Social Sciences (SPSS) version 26, and Microsoft Excel (2013) in order to analyze and evaluate the results of the study.

2.5. Materials: laboratory materials from Medical System Biotechnology Co., Ltd. were used to perform biochemical tests (blood sugar, serum creatinine, serum urea, and HbA1c). A blood picture was taken to determine blood hemoglobin (Hb). The scale was used to measure weight and the measuring tape to measure height to calculate the BMI according to equation-1 and divide the study subjects into four groups according to Table-1 shown below [14].

$$\mathrm{BMI} = \frac{\mathrm{mass_{kg}}}{\mathrm{height_m}^2} \quad \text{---------- equation-1}$$

Table-1: Categories of BMI

Category	BMI (kg/m^2)
Underweight	≤ 18.4
Normal range	18.5–24.9
Overweight (Pre-obese)	25.0–29.9
Obese	≥ 30.0

2.6. Reagents and procedure: the reagents and method were mentioned in the test kit. The method mentioned in the test kit was applied.

3. Results and Discussion

3.1. Distribution of Study Cases: The study cases were divided into groups according to age, sex, family history of the disease, and BMI, as shown in Table-2.

Table-2: Frequency and percentage for study subjects.

Parameters / Groups		Patient Group n= 50		Control Group n= 30	
		F*	%*	F*	%*
Age (years)	10 – 28	1	2	6	20
	29 – 47	3	6	7	23
	48 – 66	27	54	13	43
	67 – 85	19	38	4	14
Sex	Female	24	48	15	50
	Male	26	52	15	50
Family History	Yes	38	76	13	43
	No	12	24	17	57
BMI	≤ 18.4	0	0	0	0
	18.5 – 24.9	7	14	13	43
	25.0 – 29.0	25	50	13	43
	≥ 30	18	36	4	14

*F: Frequency, %: percentage.

3.1.1. Demographic Characteristics:

3.1.1.1. Age distribution:

Age plays a major role in increasing the risk of developing diabetes nephropathy [15]. The study included 50 patients with diabetes and kidney failure, aged between 27 and 85 years, with an average age of 61.82 years. The control group included 30 people, aged between 10 and 70 years, with an average age of 46.3 years. The two groups were divided into four groups according to age, as shown in Table-2. The highest percentage in the patient group was in the age group (48-66) years, with a percentage of 54%, followed by the age group (67-85) years, with a percentage of 38%, as shown in Figure-3. This contradicts previous studies, as the disease was prevalent in the age group (20-30) years [4,16,17]. In the control group, the highest percentage was in the age group (48-66) years, as it reached 43%, while the percentages of the remaining age groups were almost equal.

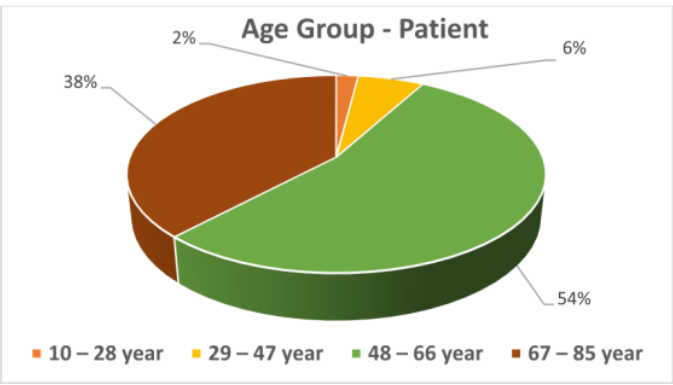


Figure-3: Age distribution for the patient group

3.1.1.2. Sex distribution:

The patient and control groups were divided according to gender into females and males, as shown in Table-2. The proportion of females and males in the control group was equal, while in the patient group, the ratio of males was higher than the proportion of females, as shown in Figure-4.

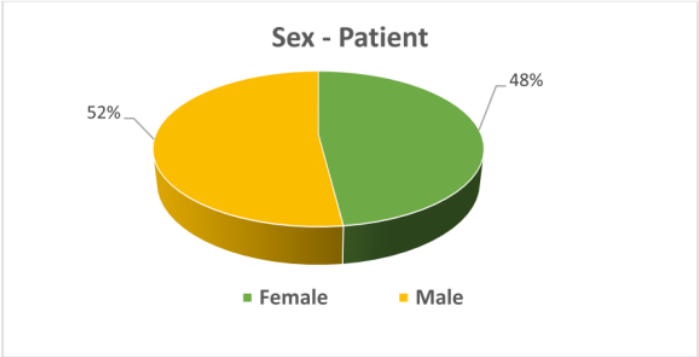


Figure-4: Sex distribution for the patient group.

3.1.2. Genetic Characteristics – Family History of the Disease Distribution:

The percentage of patients with a family history of the disease (76%) was higher than the percentage of patients without a family history of the disease (24%), as shown in Table-2. This is consistent with previous studies [4,18], as the genetic factor or family history had a major impact on the incidence of the disease.

3.1.3. Lifestyle Characteristics – BMI Distribution:

The study participants were divided into four groups according to their BMI as shown in Table-1, where the highest percentage of patients with a BMI of 25-29 was 50%, and the percentage of those with obesity (BMI ≥30) was 36% as shown in Figure-5.

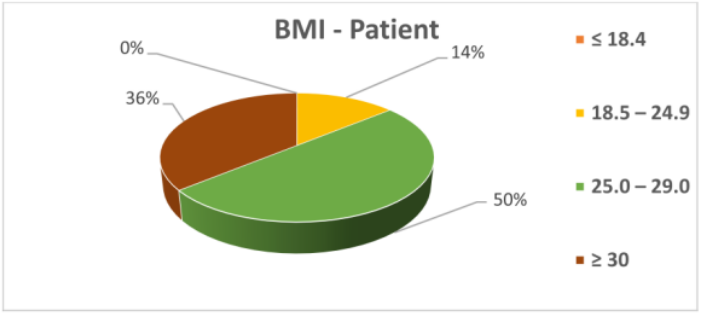


Figure-5: BMI distribution for the patient group

3.2. Mean ± SD for Biochemical Variables:

The biochemical variables were divided into three groups (hyper value, normal value, and hypo value) according to the mean ± SD values as shown in Table-3. We note from the table that all patients had a low hemoglobin level (9.67±0.83), as anemia is common in diabetic patients with chronic kidney disease [12]. As for blood sugar, the percentage was high in most patients (164.75±41.62), and the HbA1c level was within the normal range for 31 patients (6.00±0.41), and for 19 patients the level was high (7.49±0.39). We conclude from this that the high HbA1c level with diabetic nephropathy requires many future studies. This is consistent with another study [19], which found that the high HbA1c level in non-diabetic patients is associated with chronic kidney disease and that the HbA1c level in diabetic nephropathy patients requires future studies. Another study [20] showed a close relationship between HbA1c and renal failure, as HbA1c level was high in diabetic nephropathy patients with statistical significance at ($p < 0.001$), which is consistent with the present study, as significant differences were recorded at ($p < 0.05$ and $p < 0.001$) for the patient group compared to the control group. We note that serum creatinine (7.57±1.48) and serum urea (140.06±32.22) were high for all patients, which is consistent with the study conducted by SA Bamanikar et al.[7], as serum creatinine and serum urea levels were high and statistically significant at ($p < 0.05$) compared to the control group.

Also, other study [1] gave the same results, which is consistent with the results obtained in our study, as all values of variables were statistically significant at ($p < 0.05$) compared to the control group.

Table-3: Mean ± SD for Biochemical Variables.

Variables	Statue	n	Patient Group Mean ± SD	n	Control Group Mean ± SD	P value
Hb	Normal (12 – 16)	0	-	18	13.11 ± 1.00	< 0.05
	Hyper (>16)	0	-	4	16.9 ± 0.64	< 0.001
	Hypo (<12)	50	9.67 ± 0.83	8	10.44 ± 0.97	
Blood sugar (mg/dL)	**Normal (70 - 120)**	**10**	**98.58 ± 13.51**	**21**	**98.81 ± 9.53**	**< 0.05**
	Hyper (> 120)	**40**	**164.75 ± 41.62**	**9**	**157.33 ± 31.16**	**< 0.001**
	Hypo (< 70)	**0**	**-**	**0**	**-**	
HbA1c %	Normal (4.2 – 6.5)	31	6.00 ± 0.41	25	5.19 ± 0.56	< 0.05
	Hyper (> 6.5)	19	7.49 ± 0.39	2	7.00 ± 0.42	< 0.001
	Hypo (< 4.2)	0	-	3	4.03 ± 0.057	
Serum Urea (mg/dL)	**Normal (17 – 43)**	**0**	**-**	**28**	**29.13 ± 3.78**	**< 0.05**
	Hyper (> 43)	**50**	**140.06 ± 32.22**	**2**	**45.5 ± 0.7**	**< 0.001**
	Hypo (< 17)	**0**	**-**	**0**	**-**	
Serum Creatinine (mg/dL)	Normal (0.4 – 1.2)	0	-	28	0.79 ± 0.157	< 0.05
	Hyper (> 1.2)	50	7.57 ± 1.48	2	1.4 ± 0.141	< 0.001
	Hypo (< 0.4)	0	-	0	-	

3.2.1. Mean ± SD for Biochemical Variables According To Family History:

From Table-4, we notice that patients with a family history of the disease have lower values of vital variables than patients without a family history of the disease. This is different from the other study [2], where the values of the variables for patients with a family history of the disease were higher than the values of the variables for patients without a family history of the disease. The values of variables for all patients were high compared to the control group and the normal values of variables.

Table-4: Mean ± SD for Biochemical Variables according to family history.

Family History / Variables		Mean ± SD				
		Serum Urea (mg/dL)	Serum Creatinine (mg/dL)	HbA1c (%)	Hb	Blood Sugar (mg/dL)
Patient n=50	(-) Family History n=12	151.28 ± 37.32	8.25 ± 1.42	6.58 ± 0.78	9.75 ± 0.94	160.69 ± 48.96
	(+) Family History n=38	136.52 ± 30.13	7.36 ± 1.45	6.57 ± 0.91	9.64 ± 0.81	148.62 ± 45.48
Control n=30	(-) Family History n= 17	33.64 ± 5.29	0.84 ± 0.15	4.78 ± 0.54	14.05 ± 1.95	106.94 ± 19.11
	(+) Family History n= 13	25.77 ± 7.91	0.76 ± 0.32	5.75 ± 0.70	11.40 ± 1.45	128.69 ± 42.66

3.2.2. Mean ± SD for Biochemical Variables According To BMI:

We note from Table-5 that patients with BMI ≥ 18.5 had high and statistically significant values for the variables at ($p < 0.05$) except for HbA1c, which was high for patients with BMI 18.5 - 24.9. As for the rest of the groups, the values for the variables were normal compared to the control group. This is contrary to other study [2], where the development of diabetes with kidney disease was related to BMI ≥ 25.

Table-5: Mean ± SD for Biochemical Variables according to BMI.

BMI / Variables		Mean ± SD				
		Serum Urea (mg/dL)	S.Creatinine (mg/dL)	HbA1c (%)	Hb	Blood Sugar (mg/dL)
Patient n=50	≤ 18.4 n= 0	-	-	-	-	-
	18.5 – 24.9 n= 7	139.96 ± 30.21	6.94 ± 0.70	7.21 ± 1.09	9.64 ± 0.75	172.42 ± 56.62
	25 – 29 n= 25	141.89 ± 29.80	7.51 ± 1.37	6.50 ± 0.8	9.62 ± 0.86	146.17 ± 36.3
	≥ 30 n= 18	137.45 ± 37.49	7.91 ± 1.79	6.28 ± 0.77	9.75 ± 0.86	158.17 ± 54.01
Control n=30	≤ 18.4 n= 0	-	-	-	-	-
	18.5 – 24.9 n= 13	27.68 ± 6.39	0.75 ± 0.18	5.04 ± 0.53	13.43 ± 2.66	114.38 ± 18.84
	25 – 29 n= 13	32.23 ± 7.28	0.81 ± 0.16	5.12 ± 0.89	12.73 ± 1.3	112.46 ± 32.48
	≥ 30 n= 4	32.00 ± 11.05	0.95 ± 0.53	5.95 ± 0.62	11.75 ± 1.26	135.50 ± 34.07

3.2.3. Mean ± SD for Biochemical Variables According To Age:

Diabetes and chronic kidney disease pose a major risk and challenge to elderly patients[21], as the disease is closely related to age, as shown in our current study. We note from Table-6 that the highest incidence rate of the disease was among the elderly over 45 years of age, and all patients recorded high rates for all variables with statistical significance at ($p < 0.05$) except for HbA1c, where all values for all ages were within the normal range ($HbA1c \leq 6.5$).

Table-6: Mean ± SD for Biochemical Variables according to Age.

Variables	Age	Mean ± SD				
		Serum Urea (mg/dL)	**S. Creatinine (mg/dL)**	**HbA1c (%)**	**Hb**	**Blood Sugar (mg/dL)**
Patient n=50	**10 - 28 n= 1**	-	-	-	-	-
	29 - 47 n= 3	146.47 ± 26.17	9.50 ± 0.97	6.17 ± 0.63	9.23 ± 1.13	189.43 ± 51.47
	48 - 66 n= 27	136.31 ± 33.14	7.78 ± 1.58	6.54 ± 0.87	9.76 ± 0.71	142.36 ± 37.17
	67 - 85 n= 19	146.28 ± 32.39	6.96 ± 1.03	6.5 ± 0.91	9.67 ± 0.71	162.83 ± 47.11
Control n=30	**10 - 28 n= 6**	27.17 ± 4.75	0.74 ± 0.15	4.73 ± 0.77	13.17 ± 3.41	94.00 ± 11.01
	29 - 47 n= 7	27.14 ± 8.9	0.63 ± 0.21	5.17 ± 0.54	11.89 ± 1.49	123.43 ± 43.77
	48 - 66 n= 13	33.60 ± 7.20	0.85 ± 0.15	5.28 ± 0.75	13.65 ± 1.85	119.62 ± 29.63
	67 - 85 n= 4	29.25 ± 7.54	1.08 ± 0.40	5.68 ± 1.11	11.85 ± 1.40	127.00 ± 39.11

3.2.4. Mean ± SD for Biochemical Variables According To Sex:

The study showed that male patients had higher levels of variables than female patients, and all patients had higher levels of all variables than the control group as shown in Table-7. For serum creatinine, the results obtained in this study are consistent with other studies [7,22], this may be due to the storage of creatinine in muscle mass in males [23,24]. For blood sugar, one study showed no relationship between blood sugar and sex [7]. These results are similar to the results shown by other studies [22,25,26,27]. For blood hemoglobin, the levels obtained in this study were low, indicating that patients with diabetes and kidney disease are anemic with hemoglobin levels below 12, which is consistent with the results obtained in another study [28] where hemoglobin levels were below 8.

Table-7: Mean ± SD for Biochemical Variables according to sex.

Sex / Variables		Mean ± SD				
		Serum Urea (mg/dL)	**S.Creatinine (mg/dL)**	**HbA1c (%)**	**Hb**	**Blood Sugar (mg/dL)**
Patient n=50	Female n= 24	134.28±28.06	7.30 ± 1.21	6.58 ± 0.81	9.43 ± 0.92	137.64 ± 40.46
	Male n=26	145.41±35.35	7.82 ± 1.68	6.56 ± 0.94	9.89 ± 0.69	164.32 ± 48.04
Control n=30	Female n= 15	28.60 ± 8.1	0.84 ± 0.3	5.28 ± 0.77	11.97 ±1.29	112.47 ± 26.82
	Male n= 15	31.85 ± 6.85	0.78 ± 0.17	5.11 ± 0.81	13.83 ±2.52	120.27 ± 38.39

4. Conclusion

The present study highlighted that a significant portion of patients had a family history of the disease, and many were elderly, specifically over 45 years of age. The findings revealed that biochemical variable values were elevated for these patients, with statistical significance established at $p < 0.05$ when compared to the control group. Notably, the blood hemoglobin levels among all patients were low, indicating that individuals with diabetic nephropathy are indeed affected by anemia. This aligns with earlier findings that emphasize the prevalence of anemia in patients with chronic conditions, particularly those with chronic kidney disease and diabetes.

5. Recommendations

I. Conducting future studies on the relationship between HbA1c and diabetic nephropathy.

II. Conducting the same study for a specific age group with a difference of five or ten years to make the study more accurate.

III. Studying the association between insulin resistance in diabetic patients and its effect on chronic kidney disease or nephropathy.

6. Appendix – The Questionnaire

No.	**Age (y.)**	**Sex**	**Family History**	**Weight (kg)**	**Height (cm)**	**BMI**	**S. Urea**	**S. Creatinine**	**HbA1c %**	**Hb**	**Blood Sugar**
1											
2											
3											
4											
5											
6											
7											
8											

Study Effect Serum Urea and Creatinine in Diabetic Mellitus Patients with Kidney Failure

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