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# The Relationship Between CKD And Hyperglycemia

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## ABSTRACT

**Background:** The relationship between CKD and hyperglycemia is well-established, as hyperglycemia accelerates the development and progression of CKD. Elevated blood glucose levels can lead to glomerular hyper filtration and eventually glomerulosclerosis, which compromises kidney function. Diabetic nephropathy is a common form of CKD among individuals with diabetes and can be characterized by proteinuria, hypertension, and decreased kidney function.

**Methods:** Atotalof100 patients aged 45to 65years were included in this cross-sectional study. Data collection involved assessing blood glucose levels through HbA1c and fasting glucose tests, serum creatinine levels, blood urea nitrogen(BUN),and urine tests for proteinuria.

**Results:** The indicators of kidney disease such as proteinuria, spot urine PC ratio, s. creatine levels, BUN levels and GFR levels were in approximately 20-40% of population.

**Conclusion:** The prevalence of kidney disease is higher among the diabetic patients as compared to

non-diabetics.

**Keywords**: Chronic Kidney Disease, Hyperglycemia, Diabetes Mellitus

## INTRODUCTION

Hyperglycemia is derived from Greek word - Hyper meaning high and glykys meaning sweet. When blood glucose level is more than 125mg/dl in fasting state, it is referred to as hyperglycemia. Decreased insulin secretion and decrease utilization of glucose is responsible for hyperglycemia. In type 1 diabetes, hyperglycemia is due to decrease in the functioning of pancreatic beta cells which results in insulin deficiency and in type 2 diabetes, insulin resistance occurs. Prolonged and uncontrolled hyperglycemia leads to life-threatening complications. Hyperglycemia significantly contributes to chronic kidney disease (CKD) by inducing damage to renal blood vessels. Prolonged exposure to elevated blood sugar levels triggers inflammation, oxidative stress, and the formation of advanced glycation end products in the kidneys. This cascade of events leads to diabetic nephropathy, accelerating CKD progression **(Deedwania 2004)**

Diabetic kidney disease induces structural changes, including thickening of the glomerular basement membrane, loss of podocytes with denuding of the glomerular basement membrane, and mesangial matrix expansion. DKD results from both metabolic and hemodynamic abnormalities. Increased intra glomerular pressure and hyper filtration are often observed in the early phase of diabetes and contribute to the initiation and progression of DKD. In hyperglycemia, a large amount of filtered glucose increases reabsorption of glucose and sodium in the proximal tubule, resulting in decreased delivery of sodium to the distal tubular macula dense. This leads to an inappropriate reduction in afferent arteriolar resistance and a subsequent increase in intra glomerular pressure. It also activates several metabolic pathways, such as polyol pathway, hexosamine pathway, PKC pathway, and advanced gyration end-product (AGE)-related pathway **(Qazi M et al 2022)**

CKD is a global public health issue with significant morbidity and mortality rates. Hyperglycemia, a prevalent condition especially among diabetic populations, is a major risk factor for CKD. Understanding the prevalence of CKD among hyperglycemic individuals helps in estimating the burden of the disease and in planning healthcare resources and interventions**(Kumar et al 2023)**.

A cross-sectional study was conducted in Ethiopia on 214 randomly selected diabetic adults. It stated that diabetes is significant risk factor of CKD. The prevalence of CKD in diabetic patient was found to be 23.8% having e GFR < 60 ml/min/1.73 $m^2$, according to the MDRD and Cockcroft- Gault equations. 22.9% had stage 3 CKD and 0.9% had stage 4 CKD. In this study according to univariate analysis variables such as older age more than 60 years, female gender, type 2 diabetes, longer duration of diabetes (more than 10 years), low monthly income, family history of kidney disease, elevated systolic blood pressure and obesity were significantly associated. In multivariate analysis the factors determined older age, Female gender, obesity, Poor glucose control and family history of CKD that were independently significant **(Fiseha T et al 2014).**

The purpose of this study was to provide a comprehensive understanding of the prevalence of CKD in hyperglycemic individuals, thereby enabling improvement in patient care, guiding public health strategies, and advancing research in kidney disease and diabetes management.

## AIM OF THE STUDY

To study the prevalence of chronic kidney disease among hyperglycaemic patients in the area of Srinagar, Jammu and Kashmir.

## OBJECTIVES OF THE STUDY

1. To evaluate the prevalence of chronic kidney disease among the patients of hyperglycaemia in Srinagar.
2. To assess the prevalence of deranged kidney function tests among the study population.
3. To relate the incidence of deranged blood sugar levels among the patients with hyperglycaemia.

## MATERIAL AND METHODS

### Materials

A total sample of 100 patients (50 male and 50 female) aged 45 to 65 years were considered for the present study. Sample size was estimated using G-power software. The study was conducted in the Government Sub-District Hospital Sopore India.

**Sampling Technique**

Convenient sampling technique was applied for the present study. This study was a prospective cross-sectional survey of hyperglycaemic patients. The subjects enrolled in this study were followed up for hyperglycaemia and were on treatment. After obtaining informed consent, demographic data was obtained using a questionnaire.

**Methods**

The following methods were applied –

1. The blood glucose level of the patients was recorded by Hb 1 Ac and fasting glucose level.
2. Serum Creatinine test was done from all the subjects. This blood test measures the level of creatinine, a waste product from muscle fibres in blood. Elevated creatinine levels can indicate impaired kidney function.
3. Blood Urea Nitrogen (BUN) test was also be conducted on all the patients. It measures the amount of nitrogen in blood that comes from urea. Elevated BUN levels may suggest kidney dysfunction.
4. Urine Tests were conducted on all the patients. A basic urine test helps to assess various aspects of kidney function. It includes checking for the presence of protein, blood, and other substances in. the urine.
5. Proteinuria Test was conducted on all the patients. A 24-hour urine collection or a spot urine protein-to-creatinine ratio was done to quantify the amount of protein in the urine. Persistent proteinuria can be a sign of kidney damage.
6. Glomerular Filtration Rate (GFR) calculation was done from all the subjects. GFR is a key indicator of kidney function. It can be estimated using formulas that take into account serum creatinine levels, age, sex, and race.
7. Imaging Studies like ultrasound, CT scan or MRI (if needed) were also be considered to visualize the kidneys and identify any structural abnormalities.
8. Some additional tests like renal biopsy or specialized imaging studies (if necessary) were also considered to assess the extent and nature of kidney damage.

# RESULTS

Out of 100 patients, there were 64 males comprising to 64% and 36 females amounting to 36%. On comparing statistically, the p value was 0.42 (>0.05) which was statistically insignificant.

Out of 64 males, maximum patients (34.37%) belonged to the age group of 45-50 years. Least number of patients (15.62%) were in the age group of 56-60 years. In the age group of 51-55 years, there were 21 males (32.81%) and in the age group of 61-65 years, there were 11 males (17.18%).Out of 36 females, maximum patients (30.55%) belonged to the age group of 51-55 years. Least number of patients (19.44%) were in the age group of 61-65 years. In the age group of 45-50 years, there were 09 females (25%) and in the age group of 56-60 years, there were 08 females (17.18%).On comparing statistically, the p value was found to be statistically not significant (0.05) in all the groups among males and females.

Maximum number of patients were in the range of 26-30 kg/m2, with 28 males (43.75%) and 26 females (72.22%). None of the patients in the study population had BMI >40 kg/m2. In the range of 18-25 kg/m2, there were 15 males (23.43%) and 18 females (50%) and in the range of 31-40 kg/m2, there were 21 males (32.81%) and 20 females (55.55%).On comparing statistically, the p value was found to be statistically not significant (>0.05).

Regarding hypertension, 34 males (53.12%) and 30 females (83.33%) had blood pressure less than 140/90 mm Hg with a p value of 0.43 (>0.05, statistically not significant) whereas 10 males (15.62%) and 6 females (16.66%) had blood pressure equal to or more than 140/90 mm Hg with a p value of 0.02 (<0.05, statistically significant.In relation to smoking, none of the females in the study population had history of smoking. There were 20 males (31.25%) who had history of smoking.

**Table 1: shows the distribution of serum creatinine levels in the study population**

| Creatinine (mg/dl) | Male | Female | p-value |
|---|---|---|---|
| 0.5-1.0 | 23 (35.93%) | 12 (33.33%) | 0.34 |
| 1.1-1.4 | 13 (20.31%) | 09 (25%) | 0.02 |
| >1.4 | 28 (43.75%) | 15 (41.66%) | 0.01 |

28 males (43.75%) and 15 females (41.66%) had their serum creatinine levels more than 1.4 mg/dl with a p value of 0.01 (<0.05, statistically significant). 13 males (20.31%) and 9 females (25%) had their creatinine levels within the range of 1.1-1.4 mg/dl with a p value of 0.02 (<0.05, statistically significant). Normal serum creatinine levels were in 23 males (35.93%) and 12 females (33.33%) with a p value of 0.34 (>0.05, statistically not significant).

**Table 2: shows the distribution of BUN levels in the study population**

| BUN (mg/dl) | Male | Female | p-value |
|---|---|---|---|
| 6-24 | 30 (46.87%) | 20 (55.55%) | 0.54 |
| 25-40 | 20 (31.25%) | 09 (25%) | 0.02 |
| >40 | 14 (21.87%) | 7 (19.44%) | 0.01 |

14 males (21.87%) and 7 females (19.44%) had their BUN levels more than 40 mg/dl, with a p value of 0.01 (<0.05, statistically significant). In the range of BUN levels between 25-40 mg/dl, there were 20 males (31.25%) and 9 females (25%), with a p value of 0.02 (<0.02, statistically significant). Normal serum BUN levels were in 30 males (46.87%) and 20 females (55.55%), with a p value of 0.54 (>0.05, statistically not significant).

**Table 3: shows the distribution of urine protein findings in the study population**

| Urine protein | Male | Female | p-value |
|---|---|---|---|
| Negative | 32 (50%) | 20 (55.56%) | 0.54 |
| Trace | 18 (28.12%) | 10 (27.78%) | 0.42 |
| +1 | 9 (14.06%) | 4 (11.12%) | 0.36 |
| +2 | 5 (7.81% | 2 (5.56%) | 0.26 |

A large number of the population had negative results comprising of 32 males (50%) and 20 females (55.56%) and on comparing statistically, the p value was 0.54 (>0.05), statistically not significant. 18 males (28.12%) and 10 females (27.78%) had traces of urine protein with p value of 0.42 (>0.05), statistically not significant. Findings of +1 protein were in 9 males (14.06%) and 4 females (11.12%)

with a p value of 0.36 (>0.05), statistically not significant. Higher levels of urine protein (+2) were found in 5 males (7.81%) and 2 females (5.56%) with a p value of 0.26 (>0.05), statistically not significant.

**Table 4: shows the distribution of spot urine protein-to-creatinine ratio in the study population**

| Spot Urine PC ratio | Male | Female | p-value |
|---|---|---|---|
| <0.15 | 46 (71.87%) | 28 (77.67%) | 0.46 |
| 0.15-0.28 | 17 (26.56%) | 8 (22.22%) | 0.02 |
| >0.28 | 1 (1.56%) | 0 | |

Maximum number of patients, 46 males (71.87%) and 28 females (77.67%), had their spot urine PC ratio within normal range, with a p value of 0.46 (>0.05, statistically not significant). 17 males (26.56%) and 8 females (22.22%) had their spot urine PC ratio up to 0.28. there was only one male (1.56%) who had spot urine PC ratio more than 0.28 whereas none of the females had spot urine PC ratio more than 0.28.

**Table 5: shows the distribution of GFR in the study population**

| GFR (ml/min./1.73 $m^2$) | Male | Female | p-value |
|---|---|---|---|
| >90 | 42 (65.62%) | 20 (55.45%) | 0.56 |
| 60-89 | 15 (23.43%) | 12 (33.33%) | 0.42 |
| 15-59 | 6 (9.37%) | 4 (11.11%) | 0.32 |
| <15 | 1 (1.56%) | 0 | |

Most of the patients, 42 males (65.62%) and 20 females (55.45%), had glomerular filtration rate (GFR) more than 90 ml/min./1.73$m^2$. 27 patients comprising of 15 males (2343%) and 12 females (33.33%) had their GFR between 60-89 ml/min./1.73$m^2$. There were 10 patients, 6 males (9.37%) and 4 females (11.11%), had GFR between 15-59 ml/min./1.73$m^2$. There was only one male (1.56%) whose GFR fell below 15 ml/min./1.73$m^2$. None of the females had their GFR below 15 ml/min./1.73$m^2$.

# DISCUSSION

The relationship between CKD and hyperglycaemia is well-established, as hyperglycaemia accelerates the development and progression of CKD. CKD is a global public health issue with significant morbidity and mortality rates.

34 males (53.12%) and 30 females (83.33%) had blood pressure less than 140/90 mm Hg with a p value of 0.43 (>0.05) whereas 10 males (15.62%) and 6 females (16.66%) had blood pressure equal to or more than 140/90 mm Hg with a p value of 0.02 (<0.05). In relation to smoking, none of the females in the study population had history of smoking. There were 20 males (31.25%) who had history of smoking.**Akpor et al (2022)** in their investigation found that the majority of patients were normotensives or had controlled blood pressure. **Fenta ET et al (2023)** also found hypertension and smoking as associated risk factors for developing CKD.

28 males (43.75%) and 15 females (41.66%) had their serum creatinine levels more than 1.4 mg/dl with a p value of 0.01 (<0.05). 13 males (20.31%) and 9 females (25%) had their creatinine levels within the range of 1.1-1.4 mg/dl with a p value of 0.02 (<0.05). Normal serum creatinine levels were in 23 males (35.93%) and 12 females (33.33%) with a p value of 0.34 (>0.05).**Kheirandish M et al (2024)** also found that the approximately 30-40% of the population with higher blood sugar levels had borderline or raised BUN and S. creatinine levels. Theyalso found that the approximately 430-40% of the population with higher blood sugar levels had borderline or raised BUN and S. creatinine levels, which were similar to our findings.

A large number of the population had negative results comprising of 32 males (50%) and 20 females (55.56%) and on comparing statistically, the p value was 0.54 (>0.05), statistically not significant. 18 males (28.12%) and 10 females (27.78%) had traces of urine protein with p value of 0.42 (>0.05). Findings of +1 protein were in 9 males (14.06%) and 4 females (11.12%) with a p value of 0.36 (>0.05). Higher levels of urine protein (+2) were found in 5 males (7.81%) and 2 females (5.56%) with a p value of 0.26 (>0.05).**Kim KS et al (2018)** in their study concluded that the patients with high blood sugar levels had proteinuria with levels ranging from traces to +1. They also concluded that patients with higher blood sugar levels had associated higher urine spot PC ratio, if associated with proteinuria along with, similar to our findings.

The distribution of GFR in the study population showed that, most of the patients, 42 males (65.62%) and 20 females (55.45%), had glomerular filtration rate (GFR) more than 90 ml/min./1.73m$^2$. 27 patients comprising of 15 males (2343%) and 12 females (33.33%) had their GFR between 60-89 ml/min./1.73m$^2$. There were 10 patients, 6 males (9.37%) and 4 females (11.11%), had GFR between 15-59 ml/min./1.73m$^2$. There was only one male (1.56%) whose GFR fell below 15 ml/min./1.73m$^2$. None of the females had their GFR below 15 ml/min./1.73m$^2$.**Stojceva-Taneva O et al (2016)** found in their study that the number of patients having low GFR were very less and they belonged to older age group.

## CONCLUSION

Out of the total study population 20-40% patients had the features indicating diabetic kidney disease such as borderline BUN and s. creatinine levels, marginally raised proteinuria, spot urine PC ratio and decreased GFR.

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