MAK HILL Publications International Journal of Tropical Medicine

Research Article
doi: 10.59218/makijtm.2020.109.112

# Renal Profile Association with Chronic Diabetes and Chronic Kidney Disease in Diabetic Non Diabetic Patients: A Retrospective Study

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**ABSTRACT**

The chronic hyperglycemia of diabetes is associated with damage and failure of various organs, especially the eyes, kidneys, nerves, heart and vascular system. Diabetes is the major cause of end-stage renal disease and diabetic nephropathy which are also called as diabetic kidney disease. It has been suggested that in lifetime 25-45% of the diabetic patients would be developing clinically evident diabetic nephropathy. The following anthropometric parameters were studied: (i) Age: Was recorded from birthday by calendar to the nearest of year (<6 and >6 months) (ii) height It was measured in cm with the help of height measurement stadiometer (iii) body weight: It was measured in kg, by portable human weighing machine and (iv) body mass index (BMI). It was calculated by the formula. BMI = Weight (in kg)/height ($m^2$). The biochemical parameters were estimated in the clinical biochemistry laboratory using commercial kits adapted to auto analyzer. There was a significant increase in cases as compared to control group. HbA1c levels were significantly higher in cases as compared to control group. There was statistically significant increase in levels of both serum creatinine and urea in both the diabetic groups as compared to control group. Linear relationship of serum creatinine and urea level was found with increased levels of HbA1c in Type 1 diabetic subjects. To monitor the diabetes patients, estimation of blood urea and creatinine level along with HbA1c level is highly recommended. Renal profile, chronic diabetes, chronic kidney disease.

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**Key Words**
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**Received:** 2 November 2020
**Accepted:** 19 November 2020
**Published:** 20 November 2020

**Citation:** Prateek Tripathi, 2020. Renal Profile Association with Chronic Diabetes and Chronic Kidney Disease in Diabetic Non Diabetic Patients: A Retrospective Study. Int. J. Trop. Med., 15: 109-112, doi: 10.59218/makijtm.2020.109.112

## INTRODUCTION

India is the "Diabetic Capital of the World" according to the WHO[1]. Both type 1 and type 2 diabetes are likely to become more common but type 2 diabetes is predicted to become more common in the future due to rising obesity and falling exercise levels. A large portion of the morbidity and death linked to diabetes mellitus are caused by the chronic consequences of the disease which impact multiple organ systems. Measuring long-term glycemic control is a common diagnostic use for glycated haemoglobin (HbA1c). Based on its role as a mean blood glucose indicator, HbA1c predicts the likelihood that diabetic complications would arise in patients with diabetes. Diabetes's chronic hyperglycemia is linked to organ damage and failure, particularly in the kidneys, heart, eyes, nerves and vascular system. Diabetic nephropathy also known as diabetic kidney disease and end-stage renal disease are mostly caused by diabetes. According to certain estimates 25–45% of diabetic patients may develop diabetic nephropathy that is clinically noticeable throughout the course of their lives. In those with Type 1 diabetes the peak onset of nephropathy occurs 10-15 years after the disease first manifests. After 25 years, there is only a 1% annual chance of overt renal disease development for those without proteinuria. Diabetic nephropathy, also known as diabetic kidney disease and end-stage renal disease are mostly caused by diabetes. The measures used to diagnose kidney function include urea and creatinine. Serum creatinine concentration variations more accurately reflect GFR variations than serum urea concentration variations[2]. In type 2 diabetes mellitus, hyperuricemia stands alone as a risk factor for renal impairment[3]. Reports have indicated a positive correlation between the onset of diabetes mellitus and blood uric acid levels[4]. Patients with both type 1 and type 2 diabetes are at risk for developing diabetic nephropathy a deadly late complication of diabetes[5]. This clinical investigation showed a correlation between decreased incidence of microvascular complications, such as chronic kidney disease and improvements in glycemic control, especially early in treatment[6,14]. The Diabetes Control and Complications Trial, in particular, shown a strong correlation between the risk of microvascular complications, such as chronic kidney disease and a decrease in glycosylated haemoglobin (HbA1c) levels[7,6,14] Estimating GG values in healthy individuals, diabetes without chronic kidney disease (CKD) and diabetes with CKD patients were the objectives of this statistical analysis investigation. It was also expanded to determine the relationship between HbA1c and FA levels in CKD patients with diabetes.

## MATERIALS AND METHODS

This present study was conducted in Department of Nephrology during the period from October-March 2019-2020. 60 diabetic subjects as study group and 60 non-diabetic subjects as control group attending out-patient department at the NIMS, Jaipur. Diagnosis of diabetes was done on the basis of WHO criteria. All the subjects between 28-78 years were included in this study. Patients with urinary tract obstruction, congestive cardiac failure, other chronic kidney disease, myopathy or muscular dystrophy were excluded from the study. The following anthropometric parameters were studied: (i) Age: Was recorded from birthday by calendar to the nearest of year (<6 and >6 months), (ii) height: It was measured in cm with the help of height measurement stadiometer, (iii) body weight: It was measured in kg, by portable human weighing machine and (iv) body mass index (BMI): It was calculated by the formula: BMI = Weight (in kg)/height ($m^2$). The biochemical parameters were estimated in the clinical biochemistry laboratory using commercial kits adapted to auto analyzer. Blood samples from subjects and controls were collected in ethylenediaminetetraacetic acid bulb in all the diabetic patients for investigation of fasting and post-meal plasma glucose. Estimation of serum glucose was carried out by glucose oxidase and peroxidase method[15]. Serum was separated from blood by centrifugation at 3000 rpm for 10 min. Serum urea was estimated by Berthelot's method[16] while creatinine was estimated by alkaline Jaffe's Picrate method[17]All parameters were analyzed by commercially available reagents and kits on semi auto analyzers and auto analyzer in Clinical Biochemistry laboratory, SMC, Shahjaha-npur, U.P. eGFR was Calculated by using Cockroft Gault equation[18].

## RESULTS

The Mean±SD of the fasting and post-meal blood sugar levels as well as the HbA1c levels between the case and control groups are displayed in Table 1. Cases were significantly higher than in the control group. The case's HbA1c values were noticeably greater than those of the control group. The serum creatinine and urea levels in non-diabetic individuals (control), Type 1 diabetics and Type 2 diabetics are displayed in Table 2 as Mean±SD. Serum creatinine and urea levels were higher in the diabetes groups than in the control group, both statistically significantly. Other terms used in this context are ANOVA (analysis of variance) IQR (interquartile range) IDDM (insulin dependent diabetes) and NIDDM (non-insulin dependent diabetes).Table 3 displays the Pearson's coefficient correlation between the HbA1c diabetes group and blood urea, creatinine and uric acid. Renal profiles and HbA1c were revealed to significantly positively correlate.

Table 1: Comparison of parameters between cases and controls.

| Parameters | Cases (Mean±SD) | Controls (Mean±SD) | p-value |
|---|---|---|---|
| FBS (mg dL) | 188.62±38.59 | 86.69±8.51 | <0.001 |
| PPBS (mg dL) | 239.27±43.8 | 132.4±9.21 | <0.001 |
| HbA1c (%) | 8.55±0.07 | 6.33±0.53 | <0.001 |
| Urea (mg dL) | 38.9±7.3 | 25.45±7.5 | <0.001 |
| Creatinine (mg dL) | 1.67±0.32 | 1.09±0.48 | <0.002 |
| Uric acid (mg dL) | 7.31±0.8 | 5.29±0.88 | <0.001 |
| EGFR (mL min. 1.73m$^2$) | 63.04±15.2 | 83.05±17.5 | <0.002 |

(Statistically significant p<0.05)

Table 2: Comparison of baseline variable between prehypertensives and normal.

| Parameters | Groups | Mean±SD | Median | IQR | One-way ANOVA test | |
|---|---|---|---|---|---|---|
| | | | | | f-value | p-value |
| Serum urea (m dL) | Control | 28.24±3.98 | 28.01 | 3.00 | 34.510 | 0.000* |
| | NIDDM | 32.17±4.11 | 32.00 | 6.00 | | |
| | IDDM | 32.01±4.68 | 33.00 | 7.00 | | |
| Serum creatinine (mg dL) | Control | 1.09±0.28 | 1.19 | 0.29 | 51.969 | 0.000 |
| | NIDDM | 1.31±0.22 | 1.34 | 0.21 | | |
| | IDDM | 1.39±0.27 | 1.39 | 0.13 | | |

*p<0.005 indicates a statistically significant difference.

## DISCUSSION

A significant contributor to morbidity and death is diabetes mellitus. Diabetes-related kidney damage is known as diabetic nephropathy. An worldwide study found that as the length of the disease increased, the management of diabetes deteriorated, with neuropathy emerging as the most prevalent complication and being followed by retinopathy, cardiovascular, renal and foot ulcers. In this study, we found that patients with diabetes had renal function test values that were closer to higher reference limits. The levels of uric acid, creatinine and urea in patients were statistically significantly higher than in controls. In a population-based investigation, Blessing *et al*. Meera *et al*. and Rohitash *et al*.[19,20] discovered the similar outcomes. Our research demonstrates a statistically significant positive association between renal characteristics and HbA1c in certain circumstances. Diabetes patient's lower kidney filtering capacity would cause an accumulation of waste products, which would raise uric acid, creatinine and serum urea levels[21]. High blood creatinine levels in diabetes patients are caused by impaired nephron function[22]. Serum urea and creatinine elevations indicate glomerular impairment, which cannot be corrected with a rigorous treatment regimen. Patients with type 2 diabetes, whether or whether they have chronic kidney disease (CKD) may also have

Table 3: Correlation of study parameters with HbA1c.

| Renal Profile | HbA1c |
|---|---|
| Urea | 0.018 |
| Creatinine | 0.011 |
| Uric acid | 0.0147 |

elevated HbA1c levels as a result of persistent hyperglycemia brought on by impaired glucose metabolism. The large elevation of the observed HbA1c value, which was derived from the regression equation using serum FA, may be connected to the significant increase in GG in the CKD group. In comparison to patients with diabetes who do not have any chronic difficulties the study also showed that projected HbA1c increases with the development of chronic complications. The rise in intracellular and nextracellular (FA) HbA1c glycation could be the cause of this. The study came to the conclusion that diabetes and the chronic difficulties that go along with it may be caused by a substantial intracellular glycosylation process. The precise role of GG in diabetes and its consequences has to be further studied. Serum urea and creatinine elevations indicate glomerular impairment which cannot be corrected with a rigorous treatment regimen. Early detection and intervention would be the sole approach to control this growing glomerular injury and therefore elevated levels of serum and creatinine web.

## CONCLUSION

Linear relationship of serum creatinine and urea level was found with increased levels of HbA1c in Type 1 diabetic subjects. To monitor the diabetes patients, estimation of blood urea and creatinine level along with HbA1c level is highly recommended. Serum urea and creatinine are simple and useful biomarkers which can serve as predictor tests for assessing kidney functions (nephropathy) India betic patients. FA together with GG may be considered a correct interpretation of the glycosylation processes and GG and will be useful as a research probe quantitating variation between intracellular and extracellular glycemic control to identify sources of population variation in diabetic complications beyond glycemic control.

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