Original Research Article

A study on evaluating blood urea and serum creatinine in diabetes mellitus patients

Sai Ravi Kiran Biri1, S L V Sankeerthi C H²,.*, Sandhya Rani T³, Rajkumar Gundu⁴, Aravind Vadlakonda⁵

1 Dept. of Biochemistry, Fakir Mohan Medical College, Balasore, Orissa, India
2 Dept. of Biochemistry, Mahavir Institute of Medical Sciences, Hyderabad, Telangana, India
3 Dept. of Microbiology, Sri Lakshmi Naryana Institute of Medical Sciences, Puducherry, India
4 Dept. of Biochemistry, Vinayaka Missions Research Foundation, Salem, Tamil Nadu, India
5 Dept. of General Medicine, Bharath Medical College, Chennai, Tamil Nadu, India

A R T I C L E I N F O

Article history:
Received 08-11-2021
Accepted 11-12-2021
Available online 05-01-2022

Keywords:
Glycosylated hemoglobin
Diabetes mellitus
Renal nephropathy

A B S T R A C T

Background: Diabetes is one of the leading causes for end stage renal disease and nephropathy. Increases of blood urea and serum creatinine are due to abnormal renal function and also reduction in glomerular filtration rate. So, Urea and Creatinine are the ideal biomarkers to correlate the progression of diabetic nephropathy. Aim of the study is to evaluate the blood urea & serum creatinine with HbA1C in Diabetes mellitus patients.

Materials and Methods: A total of 50 cases and 30 controls were selected in our study. Blood samples were collected for blood urea, serum creatinine, HbA1C, Fasting plasma glucose and Post prandial blood sugar with age limit of 35-65 years. Mean ±SD was calculated for all these parameters.

Results: Blood urea and Serum creatinine are statistically significant in Diabetic patients when compared to the controls.

Conclusion: Our study shows that blood urea and serum creatinine can be used as biomarkers in the early detection of diabetic nephropathy. These parameters help in reducing the severity of renal failure.

This is an Open Access (OA) journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

For reprints contact: reprint@ipinnovative.com

1. Introduction

Diabetes mellitus is one of the most common metabolic disorders mainly caused due to defect in the secretion or action of insulin.¹ Mainly characterized by chronic hyperglycemia due to derangement of carbohydrates, fats, and protein metabolism.² This leads to the damage of various organs like eyes, kidneys, heart, nerves, and bloodvessels.

Throughout the world, Diabetes is one of the leading causes of morbidity and mortality and about 2.2 to 3% of world’s population suffers with Diabetes recently and this proportion may even increases in the coming years.³ In the 21st Century, Diabetes is one of the most challenging health problems affecting about 6-7% of world’s population. About 170 million people are affected with Diabetes worldwide and this number may even increase to 438 million people by 2030.Dietary modification, genetic mutations, high blood pressure, smoking, obesity, high cholesterol levels and lack of exercise are the risk factors for increasing the risk of Diabetes.⁴–⁶

Dyslipidemia, hypertension, and visceral adiposity are associated with Diabetes and these are the comorbid risk factors for developing chronic disease and cardiovascular disease.⁷ End stage renal disease and diabetic nephropathy
are mainly associated with renal disorders in diabetic patients. 8 25-45% of diabetic patients clinically develop diabetic nephropathy in their lifetime.8

Glycosylation of tissue proteins causes deterioration of the structure and function of kidney which finally leads to Diabetic nephropathy (DN). In many countries, DN affects 30% of all diabetics which is the leading cause of end stage renal disease (ESRD).9-12 Abnormal renal functions like abnormal blood urea, serum creatinine and macro albuminuria are some of the characteristic features of Diabetic Nephropathy. In uncontrolled diabetes, there may be hyperglycemia associated abnormal increase of blood urea and serum creatinine. So, urea and creatinine are the two important factors to find any abnormality in the kidney.

Serum creatinine when it alters, there will be more reliable reflection in GFR whereas urea formation depends on factors like liver function, protein intake, and rate of degradation of proteins. So, measurement of blood urea and serum creatinine helps in the early detection and prevention of diabetic kidney diseases and prevents the progression of end stage renal disease.13,14 As renal complications are more common in diabetic patients, we aimed to measure the blood urea and serum creatinine levels in diabetic patients and correlate these parameters in non-diabetic patients.

2. Materials and Methods

Present study was conducted in the Department of Biochemistry in Mahaveer medical college, Vikarabad, Telangana state after obtaining institutional ethical clearance. Our study comprises of 50 subjects with age limit between 35-65 years.

Study group consists of 37 males and 13 females with age range between 35-65 years. Our study group compared with 30 normal healthy age matched controls. These are healthy and not having the history of diabetes.

2.1. Subjects

Patients with normal blood glucose and normal renal functions tests are taken as controls.

2.2. Inclusion criteria

Patients with past history of diabetes mellitus for last 3 years were taken as cases.

2.3. Exclusion criteria

Smokers, hypertensives, hyperlipidemic, pregnant women and other chronic disorders are excluded from our study.

Written consent was obtained followed by detailed medical personal history and systemic examination. Variables collected were age, gender, fasting & post prandial blood glucose, HbA1C, blood urea and serum creatinine of all subjects.

Blood samples of both cases and controls were collected to study the biochemical parameters like blood urea, serum creatinine, FBS, PPBS, HbA1C. Biochemical parameters were analyzed in clinical biochemistry laboratory using commercial kit adapted to auto analyzer. Serum was separated by centrifugation at 4,000 rpm for 10 min. Plasma glucose level was estimated by glucose oxidase and peroxidase (GOD-POD) end point assay method.15 Blood urea by enzymatic urease method16 while serum creatinine by alkaline Jaffe’s method.17 HbA1C of all subjects in the study was estimated by ion exchange resin method using diagnostic HbA1C kit. Mean ± SD was calculated.

Normal range of fasting plasma glucose is 70-110 mg/dl, post prandial less 140mg/dl. Normal range of urea:15-40mg/dl and creatinine: 0.6-1.4 mg/dl, 0.5-1.2mg/dl for both males and females respectively & HbA1C ≥ 6. WHO criteria is followed to categorize the people with Diabetes Mellitus.

2.4. Statistical analysis

Data collected using excel statistical data was analyzed by students t-test to compare the significance between diabetic and non-diabetic control groups. P value less than 0.05 was considered as statistically significant.

3. Results

In our study, a total number of 50 subjects were taken out of which 37 were males and 13 were females. Their age is between 35-65 years and their mean age is about 56.4 years. Age matched controls are taken in our study.

Out of 50 cases, we had 12 samples with increased Urea, 16 samples with more creatinine and 22 samples increased with both urea and creatinine when compared with controls. In our study group, males having more creatinine value compared to females due to presence of more muscle mass. Increased blood urea and serum creatinine values are observed in diabetic patients when compared with controls. There is no increase in blood urea and serum creatinine in controls.

Table 1: Indicates number of samples showing increased amount of Blood urea and serum creatinine in both diabetics and non-diabetics

| Parameters                  | Cases (n=50) | Controls (n=50) |
|-----------------------------|-------------|----------------|
| Blood Urea increased        | 12          | 0              |
| Serum Creatinine increased  | 16          | 0              |
| Both Urea and Creatinine increased | 22          | 0              |

As we have been taken the diabetic patients, Mean fasting, and post prandial blood sugar was found to be higher in diabetics subjects when compared to non-diabetic.
HbA1C also found to be higher in diabetics. Blood sugar and serum creatinine increases in cases compared with the controls. Both blood urea and serum creatinine shows Statistically high significant value (p < 0.001).

### 4. Discussion

Diabetes mellitus is one of the leading causes of death throughout the world. Impairment of renal function due to diabetes can be assessed by measuring the blood urea and serum creatinine. Measurement of these parameters helps in the early detection of any impairment in the kidney.

As in our study, we have been taken cases with high blood sugar level, it indicates the poor glyemic control, and this is an indicative of renal nephropathy (RN). Glycemic control reflects the risk of nephropathy and other diabetic complications. Increase in urea level indicates the impairment or damage to the kidney whereas creatinine is a marker of GFR. Increase in both creatinine & urea with increased blood sugar clearly indicates the damage of kidney.7

Our study shows significant increase of blood urea and serum creatinine in diabetic patients which may be an indicative of pre-renal damage. This study is similar to the study of Madhusudan Rao et al as they explained the relationship of long-standing plasma glucose level with blood urea level.2 This is also similar to the study of Anjaneyulu and Chopra as they found increased urea and creatinine value in diabetic rats which leads to progressive renal damage.18

As our study shows increased level of blood urea and serum creatinine, it clearly indicates prolonged hyperglycemia which causes irrevocable damage to the nephrons of the kidney. The tiny filtering units of kidneys i.e., nephrons are damaged due to high blood sugar level. As the main function of kidney is to maintain the fluid electrolyte balance, this function got impaired. Increase in serum creatinine & blood urea is due to diminishing of GFR as the creatinine is an indirect measure of glomerular filtration and indicating reduced filtration capacity of the kidney.2

By intensive treatment, elevated levels of HbA1c can be lowered whereas the increased levels of blood urea and serum creatinine can’t be reversed as there is permanent damage of kidneys in DM.9

By this study, we can say blood urea and serum creatinine are the prognostic markers and predictors for renal damage in diabetic patients.19

### 5. Conclusion

In our study, there is a linear relationship of serum creatinine and blood urea with increased levels of HbA1C in Diabetes mellitus patients. Regulation of blood glucose level in proper time will prevent the Progression of Diabetes to Renal Impairment. So, these patients should be monitored regularly with glyemic control and renal failure to avoid the long-term complications of Diabetes Mellitus. By this we can say blood urea and serum creatinine are the simple and useful biomarkers which can serve as predictor tests for assessing the functions of the kidneys.

### 6. Source of Funding

Nil

### 7. Conflict of Interest

The authors declare no conflict of interest.

### References

1. Shankarpasad DS, Gundalli S, Mahantesh B, Kashinakunti SV, Sunitha P. Lipid profile in Diabetes Mellitus. Indian J Pathol Oncol. 2015;2(4):290–4.

2. Sivriole MR, Eturi S. A study on blood urea and serum creatinine in diabetes mellitus from Sangareddy District, Telangana, India. Int J Med Health Res. 2017;3(12):132–6.

3. Abdelsalamka M, Elamin AE. Correlation between urea level and HbA1c level in type 2 diabetic patients. Sad Med Lab J. 2011;1(2):1–6.

4. American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care. 2006;29(1):S43–8.

5. Eltaki ME, Raheem AM, Ahmed ES. Evaluation of Lipid Metabolism among Sudanese Patients with Type 2 Diabetes Mellitus. Int J Pure Appl Sci Technol. 2014;23(1):28–33.

6. Adeghate E, Schattner P, Dunn E. An Update on the Etiology and Epidemiology of Diabetes Mellitus. Ann N Y Acad Sci. 2006;1084:1–29.

7. Chutani A, Pande S. Correlation of serum creatinine and urea with glycemic index and duration of diabetes in Type 1 and Type 2 diabetes mellitus: A comparative study. Natl J Physiol Pharm Pharmacol. 2017;7(9):914–9.

8. Whaley-Connell A, Sowers JR, McCullough PA, Roberts T, Mcfarlane SI, Chen SC. Diabetes mellitus and CKD awareness: the kidney Early Evaluation Program (KEEP) and National Health and Nutrition Examination Survey (NHANES). Am J Kidney Dis. 2009;53(4):S11–21.

9. Atkins RC. The epidemiology of chronic kidney disease. Kidney Int. 2005;67(suppl 94):S14–8.

10. Stewart JH, McRerie MR, Williams SM, Jager KJ, Trpeski L, Mcdonald SP. ESRD Incidence Study Group. Trends in incidence of treated end-stage renal disease, overall and by primary renal disease, in persons aged 20–64 years in Europe, Canada and the Asia-Pacific region. Nephrology (Carlton). 1998;12:520–7.

11. Boddana P, Caskey F, Casula A, Ansell D. UK Renal Registry 11th Annual Report (December 2008): Chapter 14 UK Renal Registry

### Table 2: Indicates mean ± SD of blood urea and serum creatinine and correlated with FBS, PPBS & HbA1C in both cases and controls

| Parameters          | Diabetics          | Non-Diabetics      | P value |
|---------------------|--------------------|--------------------|---------|
| FBS                 | 183.24 ± 34.45     | 94.71 ± 9.15       | 0.000*  |
| PPBS                | 273.12 ± 42.12     | 125.28 ± 6.53      | 0.000*  |
| HbA1c               | 6.51 ± 0.02        | 5.15 ± 0.41        | 0.000*  |
| Blood Urea          | 65.81 ± 12.12      | 27.63 ± 5.32       | 0.000*  |
| Serum Creatinine    | 1.89 ± 0.81        | 0.84 ± 0.12        | 0.000*  |

**P value denotes statistically significant differences **
and international comparisons. *Nephron Clin Pract*. 2008;111(suppl 1):269–76.

12. US Renal Data System – USRDS 2010 Annual Data Report. Bethesda, MA: National Institute of Health, National Institute of Diabetes and Digestive and Kidney Diseases.

13. Shlomo M, Polonsky KS, Larsen PR, Kronenberg HM. Diabetes Mellitus. Willams textbook of endocrinology. vol. 18. 12th ed. Philadelphia: Elsevier/Saunders; 2011. p. 1371–1435.

14. Zimmet P, Alberti KG, Shaw J. Global and societal implications of the diabetes. *Nature*. 2001;414(6865):782–7.

15. Trinder P. Determination of glucose in blood using glucose oxidase with an alternative oxygen acceptor. *Ann Clin Biochem*. 1969;6:24–7.

16. Fawcett JK, Scott JE. A rapid and precise method for the determination of urea. *J Clin Pathol*. 1960;13(2):156–9.

17. Owen J, Iggo B, Scandrett FJ, Stewart CP. The determination of creatinine in plasma or serum, and in urine; a critical examination. *Biochem J*. 1954;58(3):426–37.

18. Anjaneyulu M, Chopra K. Quercetin, an antioxidant bioflavonoid, attenuates diabetic nephropathy in rats. *Clin Exp Pharmacol Physiol*. 2004;31(4):244–8.

19. Aldler AI, Stevens RJ, Manley SE, Bilous RW, Cull CA, Holman RR. Development, and progression of nephropathy in type 2 diabetes (the United Kingdom prospective diabetes study). *Kidney Int*. 2003;63(1):225–32.

**Author biography**

**Sai Ravi Kiran Biri**, Associate Professor

**S L V Sankeerthi C H**, Assistant Professor

**Sandhya Rani T**, Assistant Professor

**Rajkumar Gundu**, Assistant Professor

**Aravind Vadakonda**, Assistant Professor

---

Cite this article: Biri SRK, Sankeerthi C H SLV, Rani T S, Gundu R, Vadakonda A. A study on evaluating blood urea and serum creatinine in diabetes mellitus patients. *Int J Clin Biochem Res* 2021;8(4):285-288.