Plasma Glucose, Creatinine and Urea Levels in Type 2 Diabetic Patients Attending A Nigerian Teaching Hospital

[1]Blessing O. Idonije, [2]Oloruntoba Festus and [3]Olarewaju M. Oluba
[1]Department of Chemical Pathology, [2]Department of Medical Laboratory Science,
College of Medicine, Ambrose Alli University, Ekpoma, Nigeria
[3]Department of Chemical Science, College of Natural Sciences,
Joseph Ayo Babalola University, Ikeji-Arakeji, Osun State, Nigeria

Abstract: Diabetes mellitus is characterized by some specific complications including diabetic nephropathy. Plasma creatinine and urea are useful clinical tools in assessing renal function, despite some limitations. In the present study, plasma glucose, creatinine and urea concentrations were determined in type 2 diabetic patients attending Irrua Specialist Hospital, Ekpoma, Edo state, Nigeria. About 80 (35 males and 45 females) diabetic patients aged between 30-70 years serving as tests and 50 (30 males and 20 females) age apparently healthy individuals of similar age bracket serving as controls were used for the study.

Key words: Plasma glucose, urea, concentrations, Edo state, Ekpoma, Nigeria

INTRODUCTION

Diabetes mellitus is a metabolic disorder characterized by chronic hyperglycemia due to derangement in carbohydrate, fat and protein metabolism that are associated with absolute or relative deficiencies in insulin secretion, insulin action or both (Charles, 1998; Alberti and Zimmet, 1998). Over 170 million people worldwide and about 1-7% of the Nigerian population are affected (Wokoma, 2002; Fabiyi et al., 2002).

According to a Nigerian national non-communicable disease survey, a prevalence rate of about 2.2% for diabetes mellitus with over 90% being type 2 is reported (Akinkugbe, 1997). Epidemiological data show that increasing incidence of diabetes mellitus has made it a global health concern. Diabetic patients are at an increased risk of developing specific complications including: nephropathy, retinopathy, neuropathy and atherosclerosis (Rehman et al., 2005).

Diabetic nephropathy occurs in approximately one third type 2 diabetics (Rehman et al., 2005). In diabetic nephropathy, a number of serum markers are known to be deranged with significant morbidity and mortality (Taniguchi et al., 2003; Puepet et al., 2003). Information on plasma biochemical profiles of diabetic population in Edo state, Nigeria is scarce. The aim of this study was to assess plasma glucose, creatinine and urea concentrations in type 2 diabetic patients resident in Ekpoma, Edo state, Nigeria.

MATERIALS AND METHODS

A total of 80 diabetic subjects of both sexes aged between 30-80 years attending the diabetic clinic of Irrua Specialist Teaching Hospital, Ekpoma, Edo state, Nigeria were selected and served as tests for the study. Diabetes in this study was defined based on laboratory findings of a fasting plasma glucose levels >70.0 mg dL^{-1} on 2 or more occasions (WHO, 1999).

Their medical history and plasma data were obtained via a comprehensive questionnaire after due approval form the ethical committee of the hospital. Patients with diseases such as: dehydration, muscle dystrophy, glomerulonephritis, pyelonephritis, eclampsia and pre-eclampsia, reduced kidney blood flow (shock; congestive cardiac failure), rhabdomyolysis and urinary tract obstruction that have higher than normal plasma creatinine and urea levels were excluded. Also patients on certain drugs that may affect the test were excluded from the study. These drugs include: aminoglycosides, bactrim, cimetidine, heavy metal chemotherapy drug (e.g., cisplatin) and nephrotoxic drugs (e.g., cefoxitin). About 50 age-matched non-diabetic persons attending the family medicine outpatient clinic of the hospital were used as controls in this study.

Informed consent was obtained from all the participants. Fasting venous blood sample was collected from each subject (both tests and controls) into fluoride oxalate containers (for glucose determination) and lithium

Corresponding Author: Olarewaju M. Oluba, Department of Chemical Science, College of Natural Sciences,
Joseph Ayo Babalola University, Ikeji-Arakeji, Osun State, Nigeria

heparin sample containers (for urea and creatinine measurements) using a 5 mL syringe. Each blood samples was mixed gently and spun as quickly as possible at 3000 rpm for 5 min. Plasma was extracted into plain tubes and frozen at -4°C until required for further analysis.

Assays: Blood glucose was estimated by glucose-oxidase (GOP POD) method. Determination of plasma creatinine was carried out using Jaffe's method described by Bowers and Wong (1980). Urea was estimated using urease-Berthelot's method described by Richterich and Kuffer (1973).

Statistical analysis: Data are means±SD. Data was analyzed using SPSS 11.0. The comparism of difference in the means was calculated by student's t-test and difference in proportions was compared by χ^2-test of proportions. About p-value of 0.05 was considered significant.

RESULTS AND DISCUSSION

Impairment of renal function due to type 2 diabetic mellitus was assessed by measurement of plasma concentrations of creatinine and urea in both tests (type 2 diabetic patients) and controls (non-diabetic subjects). Fasting blood glucose concentration, plasma creatinine and urea concentrations were observed to be significantly higher in males (Table 1) and females (Table 2) type 2 diabetic patients (test subjects) compared to non-diabetic control subjects. Results obtained showed no significant difference in the test parameters between male and female test subjects.

Plasma creatinine and urea are established markers of Glomerular Filtration Rate (GFR). Though plasma creatinine is a more sensitive index of kidney function compared to plasma urea level. This is because creatinine fulfills most of the requirements for a perfect filtration marker (Perrone *et al.*, 1992). According to Mitch and Walser (1986) if a graph of reciprocal of plasma creatinine is plotted over time a straight line will be obtained. Thus,

if a patient is losing kidney function at a constant rate, one could be able to extrapolate the graph out of time and get a rough idea of when kidney will fail completely and when initiation of dialysis may be required and to determine efficacy of treatment to halt progression of renal failure. Plasma creatinine is also helpful in recognizing when there is an acute drop in kidney function in addition to chronic loss. Thus, plasma creatinine is used for monitoring disease progression (Mitch, 1986; Mitch and Walser, 1986).

CONCLUSION

Results obtained from the present study showed that in addition to elevated blood sugar level in type 2 diabetes mellitus, plasma creatinine and urea concentration are also significantly increased in male and female diabetics compared with their levels in apparently healthy non-diabetic male and female controls.

This observation is in accord with the reports of Aldler *et al.* (2003), Judykay (2007) and Wagle (2010). Aldler *et al.* (2003) in their report submitted that raised plasma creatinine and urea levels in diabetic patient may indicate a pre-renal problem such as volume depletion. Judykay (2007) in his submission suggested that high creatinine levels observed in diabetic patients may be due to impaired function of the nephrons. Researcher also posited that high urea levels in diabetes mellitus patients could be attributed to a fall in the filtering capacity of the kidney thus leading to accumulation of waste products within the system.

In addition, a report on the comparative study of serum sugar and creatinine levels in male and female type 2 diabetic patients showed that blood glucose and serum creatinine concentrations are elevated in type 2 diabetic patients compared with non-diabetic male and female controls (Wagle, 2010). Wagle (2010) reports showed a progressive decrease in renal function in male and female diabetic patient as from age 40 years and beyond as a result of increased serum creatinine levels. Male diabetic patients were found to present significantly higher serum creatinine level than females.

Table 1: Plasma glucose, creatinine and urea concentrations in male type 2 diabetes and male non-diabetic controls

Parameters	Control	Test	t-value	p-value
Glucose (mg dL^{-1})	178.5±67.2	83.4±10.7	7.7	p<0.05
Creatinine (mg dL^{-1})	1.2±1.20	0.8±0.20	2.0	p<0.05
Urea (mg dL^{-1})	28.1±13.9	20.0±7.00	2.9	p<0.05

Table 2: Plasma glucose, creatinine and urea concentrations in female type 2 diabetes and female non-diabetic controls

Parameters	Control	Test	t-value	p-value
Glucose (mg dL^{-1})	179.3±54.9	85.5±12.3	7.5	p<0.05
Creatinine (mg dL^{-1})	1.1±0.50	0.8±0.20	2.6	p<0.05
Urea (mg dL^{-1})	28.7±15.6	19.0±5.20	2.7	p<0.05

REFERENCES

Akinkugbe, O.O., 1997. National expert committee on Non-Communicable Diseases (NCD) in Nigeria. Final Report of a National Survey, Lagos, pp: 64-90.

Alberti, K.G. and P.Z. Zimmet, 1998. Definition, diagnosis and classification of diabetes mellitus and its complications: Part 1. Diagnosis and classification of diabetes mellitus, provisional report of a WHO consultation. Diabetic Med., 15: 539-553.

Aldler, A.I., R.J. Stevens, S.E. Manley, R.W. Bilous, C.A. Cull and R.R. Holman, 2003. Development and progression of nephropathy in type 2 diabetes: The United Kingdom Prospective Diabetes Study (UKPDS 64). Kidney Int., 63: 225-232.

Bowers, L.D. and E.T. Wong, 1980. Kinetic serum creatinine assays II. A critical evaluation and review. Clin. Chem., 26: 555-561.

Charles, F.A., 1998. Endocrinology. In: Mayo Internal Medicine Board Review, Udaya, B.S.P., O.E. Millhouse and M.S. Leann (Eds.). 3rd Edn., Lippincott, UK., pp: 187-278.

Fabiyi, A.K., B.A. Kolawole, O. Adeshinto and R.T. Ikem, 2002. The impact of knowledge, attitude, practice and belief on type 2 Nigerian diabetes patients on drug compliance. Diabetes Int., 291: 15-17.

Judykay, T., 2007. Nutrition for reducing urea and cratinine in the blood. Diabetes Care, 27: 2191-2192.

Mitch, W.E. and M. Walser, 1986. Nutrition Therapy of Uremic Patients. In: The kidney, Brenner, B.M. and F.C. Rector (Eds.). Saunders, Philadelphia, pp: 1759-1790.

Mitch, W.E., 1986. Measuring the Rate of Progression of Renal Insufficiency. In: Contemporary Issues of Nephrology: Progressive Nature of Renal Disease, Mitch, W.E., B.M. Brenner and J.H. Stein (Eds.). Vol. 14, Churchill Livingstone, New York, pp: 167-187.

Perrone, R.D., N.E. Madias and A.S. Levey, 1992. Serum creatinine as index of renal function. Clin. Chem., 38: 1933-1953.

Puepet, F.H., E. Agaba, and C. Chuhwak, 2003. Some metabolic abnormalities in type 2 diabetes in Jos, north central Nigeria. Nig. J. Med., 12: 193-197.

Rehman, G., S.A. Khan and M. Hamayun, 2005. Studies on diabetic nephropathy and secondary diseases in type 2 diabetes. Int. J. Diab. Dev. Ctries., 25: 25-29.

Richterich, R. and H. Kuffer, 1973. The determination of urea in plasma and serum by a urease/Berthelot method, adapted to the Greiner Electronic Selective Analyzer GSA II (authors transl). Z. Klin. Chem. Klin. Biochem., 11: 553-564.

Taniguchi, A., M. Fukushima, Y. Seino, M. Sakai and S. Yoshii *aaat al.*, 2003. Platelet count is independently associated with insulin resistance in non-obese Japanese type 2 diabetes patients. Metabolism, 52: 1246-1249.

WHO, 1999. Definition Diagnosis and Classification of Diabetes Mellitus and its Complications. Part 1: Diagnosis and Classification of Diabetes. WHO/NCD/NCS 99, 2, Geneva, pp: 1-58.

Wagle, T.J., 2010. Genderwise comparison of serum creatinine and blood sugar levels in type 2 diabetic patients. Bombay Hospital J., 52: 64-68.

Wokoma, F.S., 2002. Diabetes and hypertension in Africa: An overview. Diabetes Int., 12: 36-40.