

Glycated Haemoglobin (HbA1c) and the Assessment of Risk of Nephropathy in Diabetic Patients in Ahmadu Bello University Teaching Hospital Zaria, Nigeria

Mustafa Ibrahim Oladayo^{1*},
Yusuf Tanko¹,
Rasheed Yusuf²,
Sunday Abraham Musa³

Abstract

The study looked into the correlation between glycated haemoglobin A1c (HbA1c) and the risk of developing diabetic nephropathy among diabetic patients attending Ahmadu Bello University Teaching Hospital Zaria, Nigeria, and also determined the level of HbA1c where the risk of nephropathy becomes pronounced.

One hundred and one (101) diabetic patients were used for the study comprising of both male and female patients. About 5mL of blood sample was collected from each of the subjects after about 10 hours of overnight fasting. Then 3mL of the sample was centrifuged and the serum analysed for serum creatinine and also fasting blood glucose (FBG). The Glomerular Filtration Rate (GFR) was then calculated from the serum creatinine value using the Cockcroft-Gault equation. The remaining 2mL from the blood sample was transferred into EDTA bottles and analysed immediately for glycated haemoglobin (HbA_{1c}).

Thirty-seven (37) of the diabetic subjects had mean HbA_{1c} level of 6.96% that correspond to mean FBG level of 91.37mg/dL. Initially no significant correlation was found between HbA1c and the GFR. But there was significant correlation between HbA1c and GFR among patients with HbA1c level $\geq 9\%$ ($R = -0.35$). Patients with this level of HbA1c ($\geq 9\%$) had equivalent FBG ≥ 136 mg/dL.

The risk of developing renal complications among patients was only prominent at the level of 9% HbA1c and above, and monitoring of patients should be done accordingly in order to guard against overtreatment, hypoglycaemia and unnecessary expenses.

Keywords: Glycated haemoglobin (HbA1c); Nephropathy; Diabetes; Serum creatinine; GFR; Blood glucose

- 1 Department of Human Physiology, Ahmadu Bello University, Zaria, Nigeria
- 2 Department of Chemical Pathology, Ahmadu Bello University Teaching Hospital, Zaria, Nigeria
- 3 Department of Human Anatomy, Ahmadu Bello University, Zaria, Nigeria, Nigeria

Corresponding author:
Mustafa Ibrahim Oladayo

✉ oladayomustafa@gmail.com

Tel: 08188773306

Department of Human Physiology, Ahmadu Bello University, Zaria, Nigeria

Citation: Oladayo MI, Tanko V, Yusuf R, et al. (2022) Glycated Haemoglobin (HbA1c) and the Assessment of Risk of Nephropathy in Diabetic Patients in Ahmadu Bello University Teaching Hospital Zaria, Nigeria. J Biomed Sci, Vol. 11 No. 9: 78

Received: 17-Aug-2022, Manuscript No. IPJBS-22-12924; **Editor assigned:** 19-Aug-2022, PreQC No. IPJBS-22-12924 (PQ); **Reviewed:** 02-Sep-2022, QC No. IPJBS-22-12924; **Revised:** 07-Sep-2022, Manuscript No. IPJBS-22-12924 (R); **Published:** 14-Sep-2022 DOI: 10.36648/2254-609X.11.9.78

Introduction

Diabetes is by all odds a big health problem worldwide. The International Diabetes Federation (IDF) Atlas estimated that about 285 million people around the world had diabetes in the year 2010 [1] and close to ten million people now present with the case in Nigeria alone [2].

As Rahbar made the discovery of a diabetic haemoglobin component in people with diabetes in 1968 [3], before long, it was demonstrated that this component had chromatographic

characteristics resembling those of HbA1c (glycated haemoglobin), a minor haemoglobin component [4]. Several clinical studies then showed a close relationship between HbA1c, and the mean plasma glucose few months before doing the HbA1c test [5, 6]. The UK Prospective Diabetes Study (UKPDS) and the Diabetes Control and Complications Trial (DCCT) eventually revealed the link between glycaemic control (as regards HbA1c) and the risk of developing (and aggravating of) chronic diabetic complications [7,8], thereby confirming that HbA1c can be a "gold standard" for assessing medium to long term glycaemic control in diabetic patients. The results from HbA1c testing can

therefore be used to determine the course of future treatment for the patient in order to guard against hyperglycaemic-induced complications [9]. However, the level of 6.5% HbA1c (IFCC (International Federation of Clinical Chemistry) 48 mmol/mol) though specific for the diagnosis of diabetes in most studies, lacks sensitivity and may misdiagnose many diabetic cases. The accuracy of the test is complicated further by many factors which modify levels of HbA1c due to genetic factors like red cell life span, race, haemoglobinopathies; or environmental factors like iron deficiency; or interferences e.g. vitamin C; or biological variability [10]. Microvascular complications such as retinopathy, nephropathy and neuropathy occur in diabetes [11]. And largely due to these complications, globally, diabetes is said to be the fifth leading cause of death [12]. Prevention of diabetes and its complications, early detection of disease stages, and therapeutics that would act in the presence of hyperglycaemia to prevent, delay or reverse the complications are the major concerns. Biomarkers such as glycated haemoglobin, serum creatinine and others are studied for understanding the mechanisms of hyperglycaemia-induced metabolic abnormalities [13]. Diabetic nephropathy, a leading cause of kidney failure and one of the key complications in diabetic patients is defined by either microalbuminuria or by an increase in serum creatinine level, which is in turn used in the calculation of estimated GFR (eGFR) in diabetic patients [14]. While microalbuminuria is a very sensitive test in people with Type 1 diabetes, testing for microalbuminuria alone may miss many cases of diabetic kidney disease in those with Type 2 [15]. Therefore, it is very important to test the kidney function by measuring the serum creatinine level [16]. And using the serum concentration of creatinine in an equation that takes into account the person's weight, age, sex, (and race), one can estimate the GFR to evaluate kidney function. The higher the blood creatinine level, the lower the GFR and the worse shape the kidneys are in [17]. Normal eGFR ranges from 90 to 120 ml/min/1.73m² [17].

Methods

Study subjects

Volunteers comprised of 101 diabetic male and female subjects. The participants had been receiving treatment in the teaching hospital for at least a year. They were recruited over a time period of 12 months from the month of January 2018 through December 2018. The age range of subjects was 35 years and above.

Study site

Zaria, a major city in Kaduna State (North-western region of Nigeria) has a population of about 700,000 people [18]. The denizens of Zaria are of various Nigerian ethnicity and livelihood. The city houses Nigeria's largest University, Ahmadu Bello University. Ahmadu Bello University Teaching Hospital (ABUTH) is a modern hospital and serves patients with myriad forms of ailments including diabetes.

Informed consent and ethics committee approval

The study was approved by the Committee on Ethics for Human Research of Ahmadu Bello University, Zaria with the Approval No: ABUCUHSR/2017/002. Informed consent was gotten from each

of the participants.

Inclusion and exclusion criteria

Diabetic patients that have presented with the ailment for at least a year were selected for the study. Their type of diabetes was Type II and the age range of patients was 35 years and above. Subjects that had any condition that affect erythrocyte turn over; or had evidence of chronic medical conditions like hypertension, renal failure, liver disease and urinary tract infection were all excluded from this study. Also, patients with diabetes for less than a year were excluded. Inclusion and exclusion was done based on the information about subject's personal and health-history data filled in a questionnaire and by scrutinizing the patients' medical record

Sample size

$$n = \frac{Z^2 P(1-P)}{d^2}$$

[19]

Where: n = Sample size, Z = Z statistic for a level of confidence (for the level of confidence of 95%, Z's value is 1.96), P = Expected prevalence or proportion (expressed in proportion of 1 instead of percentage), d = Precision (expressed in proportion of 1 instead of percentage).

Choosing a prevalence of 6.7% [20] at 95% confidence interval, the expected prevalence P = 6.7% (or 0.067) and Precision = 5% (or 0.05), [21].

Substituting the values in the equation:

$$n = \frac{1.96^2 0.067(0.933)}{0.05^2} = 96$$

This indicates that the sample size should at least be 96 but 101 subjects were used for the study, with the attrition rate at 5%.

Sample collection and analysis

Information about personal data, lifestyle and medical history for each subject was obtained using questionnaire and matching hospital records. Their blood pressure was checked at the point of blood collection to ascertain absence of hypertension. Blood samples were collected from subjects after about 10 hours of fasting. Then 5mL of blood sample (each) was collected via venepuncture. About 3mL of the blood was transferred from syringe into plain bottles, and centrifuged at 4000 rpm for 10 minutes. The serum was kept in the freezer (at -18°C) until analysis. The remaining 2ml was transferred into EDTA bottles to prevent clotting and analysed immediately for glycated haemoglobin (HbA1c).

The glycated haemoglobin (HbA1c) level in the blood samples was measured using the Fineware™ fluorescence immunoassay (FIA) meter and cartridges.

The levels of creatinine and glucose present in the serum samples were measured using the automated Erba® Mannheim XL-200 Full-Auto Chemistry Analyzer by Erba Diagnostics Mannheim, Germany. The creatinine clearance was calculated for each sample from its serum concentration of creatinine using the

Cockcroft-Gault equation [22] where creatinine clearance (C_{CR}) = GFR:

$$C_{cr}(\text{ml/min}) = \frac{(140 - \text{Age}) \times \text{Weight} \times (0.85 \text{ if female})}{72 \times \text{SCr}}$$

Statistical analysis

Results were presented as mean \pm SD and data was analysed using the Descriptive Statistics while relationships between variables was determined using the Pearson's correlation test. A linear regression analysis was also conducted to generate a regression equation. A p -value of < 0.05 was considered statistically significant. All analysis was done using SPSS version 24.

Results

Table 1 simply shows the mean levels of each of the parameters that were measured or determined.

On comparing the interrelationship between the measured parameters, FBG and HbA1c showed a strong positive correlation

Table 1. Mean values of measured parameters of the patients.

Parameters	Mean \pm SD (n= 101)
HbA1c(%)	9.31 \pm 3.24
FBG(mg/dL)	160.62 \pm 87.61
GFR(mL/min)	83.26 \pm 26.93
SCr(mg/dL)	0.96 \pm 0.91

HbA1c = Glycated Haemoglobin, FBG = Fasting Blood Glucose, GFR = Glomerular Filtration Rate and SCr = Serum Creatinine.

with an R value of 0.84 (**Table 2**). The relationship was also significant at a P level of 0.01. Also the GFR and SCr showed a significant correlation ($P < 0.01$) with an inverse correlation coefficient R of -0.49. However, the FBG and HbA1c though showed negative correlations with the GFR, it was not to a significant level.

Although patients with HbA1c $\geq 9\%$ had a strong negative correlation between their HbA1c and GFR, significant at a level of $P < 0.05$ (**Table 3**).

The patients that had HbA1c $\geq 9\%$ were selected and the mean level of each of the measured parameters was calculated (**Table 4**). Their mean HbA1c and FBG levels are expectedly higher than the collective mean HbA1c and FBG levels whereas for the mean GFR and SCr levels, they are about the same as the collective.

Figure 1 is a graph depicting the inverse relationship between the HbA1c and GFR of patients with HbA1c greater than or equal to 9%. In (**Figure 2**), a graph of the relationship between HbA1c and FBG was plot and a line equation generated. Picking a point of

Table 3. Exploring the correlation between HbA1c and GFR of patients with HbA1c $\geq 9\%$.

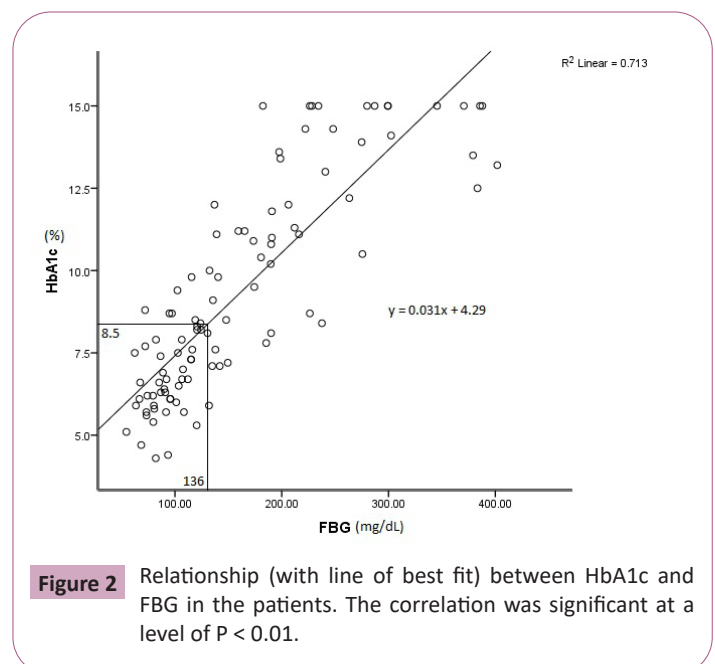
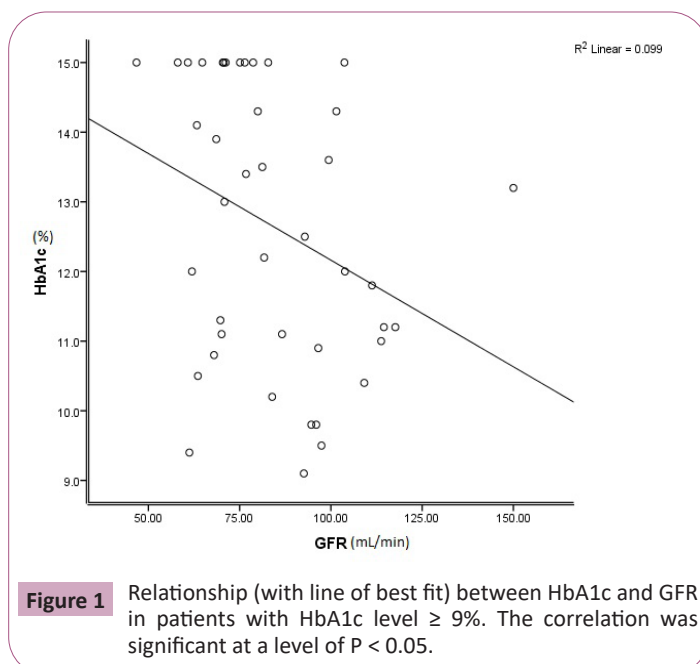
	HbA1c (%)	GFR (mL/min)
HbA1c (%)	1	-0.35*
GFR (mL/min)	-0.35*	1

The asterisk (*) indicates significant difference at the level of $P < 0.05$. HbA1c = Glycated Haemoglobin and GFR = Glomerular Filtration Rate.

Table 2. Exploring the relationship (correlation) between the parameters (HbA1c, FBG, GFR and SCr) for the diabetic subjects.

	HbA1c (%)	FBG (mg/dL)	GFR (mL/min)	SCr(mg/dL)
HbA1c (%)	1	0.84*	-0.02	-0.19
FBG (mg/dL)	0.84*	1	-0.05	-0.11
GFR (mL/min)	-0.02	-0.05	1	-0.49*
SCr(mg/dL)	-0.19	-0.11	-0.49*	1

The asterisk (*) indicates significant difference at the level of $P < 0.01$. HbA1c = Glycated Haemoglobin, FBG = Fasting Blood Glucose, GFR = Glomerular Filtration Rate and SCr = Serum Creatinine.



135 mg/dL FBG on the graph, the equivalent HbA1c is about 8.5%. This is intriguingly about the same HbA1c level where correlation (negative) between HbA1c and GFR is observed. **Table 5** shows varied hypothetical FBG values and their (expected) respective equivalent HbA1c values.

In **Table 6**, subjects that had HbA1_c at 5.7% – the purported point of onset of pre-diabetes – or greater were selected and their FBG compared with their HbA1c. Those whose FBG is correspondingly high (at the prediabetic or diabetic level) are classified as “conforming” while those whose FBG is within the normal range are classified as “deviating”.

Discussion

Diabetic nephropathy is said to affect up to 20 or 30 percent of patients with diabetes and it is a common cause of kidney failure [23]. In Nigeria, it is reported to be the third most common cause of chronic renal failure. Though diabetic nephropathy is said to present at its earliest stage with microalbuminuria, it is important to test for the estimated GFR especially in patients with type 2 diabetes. For instance, patients with type 2 diabetes in NHANES III (Third National Health and Nutrition Examination Survey), low GFR was present in 30% of patients in the absence of micro- or macro albuminuria and retinopathy [24]. Gross et al, 2005, [25] therefore recommended that GFR should be routinely estimated for a proper screening of diabetic nephropathy, as it is regarded as the best parameter for overall kidney function [26,27]. In this study, the GFR was first observed not to drop significantly with increasing HbA1c. Whereas the GFR, an index for determining kidney function is known to drop with increasing level of HbA1c, a glycaemic marker which increases progressively with increase in FBG and used to monitor glycaemic control among diabetic patients [28-30]. But as from 9% glycation of haemoglobin (9% HbA1c) onward, the GFR dropped proportionally and significantly as the HbA1c increased. This occurred because below the level of 9% haemoglobin glycation, there isn't the expected correspondingly high FBG level among the patients which would actually compromise the renal function and cause the GFR to

Table 4. Mean values of measured parameters of the patients with HbA1c ≥ 9%.

Parameters	Mean ± SD (n= 43)
HbA1c (%)	12.58 ± 2.02
FBG (mg/dL)	234.06 ± 82.44
GFR (mL/min)	85.05± 21.06
SCr(mg/dL)	0.91 ± 0.12

HbA1c = Glycated Haemoglobin, FBG = Fasting Blood Glucose, GFR = Glomerular Filtration Rate and SCr = Serum Creatinine.

drop – HbA1c being just a marker of glycaemic level. Thus, it is only in the patients with HbA1c above 9% that depreciating kidney function with increasing HbA1c is seen. The mean FBG at this level is also seen to be correspondingly very high.

The cut-point (point of onset of diabetes and risk of complications) of HbA1c from the diagnostic point of view is still controversial [31,32] and there is need to investigate local equivalent levels for a given blood glucose range [9]. As seen from figure 2, the equivalent level of HbA1c for FBG of about 136 mg/dL is 8.5% suggesting the cut-point for the diagnosis of diabetes [33]. It is worthy of note that it is around the same level (~9%) that significant correlation between the rising HbA1c and the dropping GFR was observed among the patients. This conjointly points at that HbA1c level (of ~9%) as an important point where diagnosis of diabetes and prognosis of complications – especially nephropathy – can be made.

Though a significant downward trend of GFR against rising HbA1c was observed among patients with HbA1c ≥ 9%, their mean GFR seemed not to be lower than the collective mean GFR level. This is likely due to the effectiveness of treatment among this group of patients moving more of them towards the relatively higher GFR (as observed in the mean value) and thus, an improving kidney function. There is however the necessity of further studies to investigate the relationship and interdependence between local HbA1c levels and accompanying renal function status among diabetic patients using microalbuminuria and/or albumin-creatinine ratio as an index or indices for determining kidney function.

Table 5. Glucose values and equivalent glycated haemoglobin levels based on the regression equation: $y = 0.031x + 4.29$ gotten from the study.

FBG (mg/dL)	HbA1c (%)
68	6.40
97	7.30
126	8.20
152	9.00
183	9.96
212	10.86
240	11.73
269	12.63
298	13.53
326	14.40
355	15.30

HbA1c = Glycated Haemoglobin, FBG = Fasting Blood Glucose.

Table 6. Proportion of Diabetic Subjects with Deviating HbA1c Levels (5.7% and above) Not Conforming to their Corresponding FBG Levels.

	n = 64 Conforming HbA1c	n = 37 Deviating HbA1c	t	p-value
% of total N	63.37	36.63		
HbA1c (%)	10.67 ± 3.31	6.96* ± 1.08	8.25	< 0.001
FBG (mg/dL)	200.65 ± 87.15	91.37* ± 15.89	9.75	< 0.001
SCr (mg/dL)	0.95 ± 0.18	0.99* ± 0.20	-0.92	< 0.001
GFR (mL/min)	82.99 ± 22.19	83.72* ± 33.95	-0.12	< 0.001

The asterisk (*) indicates significant proportion at the level of $p < 0.001$. **Conforming HbA1c** = HbA1c level that matches its supposed glucose range. **Deviating HbA1c** = HbA1c level that does not match (higher than) its supposed glucose range. HbA1c = Glycated Haemoglobin, FBG = Fasting Blood Glucose, GFR = Glomerular Filtration Rate and SCr = Serum Creatinine.

The study shows that about 37% of diabetic subjects used had HbA1c levels that do not match their glucose level – they had higher HbA1c (at the supposed diabetic or prediabetic level) for their (normal) blood glucose range. This indicates that 37 of the patients were capable of being over-treated and indulge in unnecessary or inimical expenses.

Finally, this study has provided a gateway for the underlying physiologic factors responsible for such a relatively wide range in normal HbA1c level to be investigated in the nearest future.

Conclusion

Glycated haemoglobin level of 8.5% should be taken as the threshold for the diagnosis of diabetes and the point of likelihood for the development diabetic complications particularly

nephropathy. Also, the treatment of diabetic patients should be targeted at points well below this level with effort to provide adequate care for them. In addition, taking 8.5% as the “point of worry” could help guard against overtreatment, treatment-induced hypoglycaemia and unnecessary expenses.

Other Information

Data are available on request.

Funding

The study was privately funded.

Conflict of Interest

No conflict of interest declared.

References

- 1 Shaw JE, Sicree RA, Zimmet PZ (2010) Global estimates of the prevalence of diabetes for 2010 and 2030. *Diab Res Clin Prac* 87: 4-14.
- 2 Oghagbon EK (2014) Commentary: Improving persistently elevated HbA1c in diabetes mellitus patients in Nigeria. *Eth Dis* 24: 502-507.
- 3 Rahbar S (1968) An abnormal haemoglobin in red cells of diabetes. *Clin Chim Acta* 22 296-298.
- 4 Schnek A, Schroder W (1961) The relation between the minor components of whole normal human adult haemoglobin as isolated by chromatography and starch block electrophoresis. *J Am Chem Soc* 83: 1472-1478.
- 5 Gabbay K, Hasty K, Breslow JL, Ellison RC, Bunn HF, et al. (1977) Glycosylated haemoglobin and long-term blood glucose control in diabetes mellitus. *J Clin Endo Metab* 44: 859-864.
- 6 Boronat M, Saavedra P, Varillas VF, No`voa FJ (2009) Use of confirmatory factor analysis for the identification of new components of the metabolic syndrome: the role of plasminogen activator inhibitor-1 and haemoglobin A1c. *Nutr Metab Cardiovasc Dis* 19: 271-276.
- 7 DCCT Research Group (1993) The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. *N Engl J of Med* 329 977-986.
- 8 UK Prospective Diabetes Study (UKPDS) Group (1998) Intensive blood glucose control with sulphonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS 33). *Lancet* 352: 837-853.
- 9 Robert MC, Christopher JL (2012) When the blood glucose and the HbA1c don't match: turning uncertainty into opportunity. *Diab Care* 35: 2421-2423.
- 10 Leslie RD, Cohen RM (2009) Biologic variability in plasma glucose, haemoglobin A1c, and advanced glycation end products associated with diabetes complications. *J Diab Sci Tech* 3: 635-643.
- 11 Beisswenger PJ (2010) Glycation and biomarkers of vascular complications of diabetes. *Amino Acids* 42: 1171-1183
- 12 Roglic G, Unwin N, Bennett PH, Mathers C, Tuomilehto J, et al. (2005) The burden of mortality attributable to diabetes: realistic estimates for the year 2000. *Diab Care* 28: 2130-2135.
- 13 Marshall S, Flyvbjerg A (2006) Prevention and early detection of vascular complications of diabetes. *Brit Med J* 333: 475-480.
- 14 Amos AF, McCarty DJ, Zimmet P (1997) The rising global burden of diabetes and its complications: estimates and projections to the year 2010. *Diab Med* 14: 1-85.
- 15 Koroshi A (2007) Microalbuminuria, is it so important? *Hippokratia* 11: 105-107.
- 16 Lewis G, Maxwell AP (2014) Risk factor control is key in diabetic nephropathy. *Practitioner* 258: 13-17.
- 17 Mula-Abed WAS, Rasadi KA, Al-Riyami D (2012) Estimated Glomerular Filtration Rate (eGFR): A Serum Creatinine-Based Test for the Detection of Chronic Kidney Disease and its Impact on Clinical Practice. *Oman Med J* 27: 108-113.
- 18 National Population Commission (2006).
- 19 Daniel WW (1999) *Biostatistics: A Foundation for Analysis in the Health Sciences* seventh ed. John Wiley & Sons.
- 20 Edo AE, Akhmemokhan K (2012) Relationships between haemoglobin A1c and spot glucose measurements in Nigerians with type 2 diabetes mellitus. *Nig J Clin Pract* 15: 23-26.
- 21 Naing L, Winn T, Rusli BN (2006) Practical Issues in Calculating the Sample Size for Prevalence Studies. *Arch Orof Sci* 1: 9-14.
- 22 Cockcroft DW, Gault MH (1976) Prediction of creatinine clearance from serum creatinine. *Nephron* 16: 31-41.
- 23 Navarro JF, Mora C, Muros M, Maca M, Garca J (2003) Effects of penroxifylline administration on urinary N- acetyl- bet a-D glucosaminidase (NAG) excretion in type 2 diabetic patients: a short term prospective randomized study. *Am J Kidney Dis* 42: 264-270.
- 24 Kramer HJ, Nguyen QD, Curhan G, Hsu CY (2003) Renal insufficiency in the absence of albuminuria and retinopathy among adults with type 2 diabetes mellitus. *J Am Med Assoc* 289: 3273-3277.
- 25 Gross JL, De Azevedo MJ, Silveiro SP, Canani LH, Caramori ML, et al. (2005) *Diabetic Nephropathy: Diagnosis, Prevention, and Treatment*. *Diab Care* 28: 176-188.
- 26 Levey AS, Coresh J, Balk E, Kausz AT, Levin A, et al. (2003) National Kidney Foundation practice guidelines for chronic kidney disease: evaluation, classification, and stratification. *Ann Int Med* 139: 137-147.

- 27 American Diabetes Association (2004) Nephropathy in diabetes (Position Statement). *Diab Care* 27: 79-83.
- 28 Writing Team for the Diabetes Control and Complications Trial/ Epidemiology of Diabetes Interventions and Complications Research Group (2003) Sustained effect of intensive treatment of type 1 diabetes mellitus on development and progression of diabetic nephropathy: the Epidemiology of Diabetes Interventions and Complications (EDIC) study. *J Am Med Assoc* 290: 2159-2167.
- 29 World Health Organization (WHO) (2011) Use of Glycated Haemoglobin (HbA1c) in the Diagnosis of Diabetes Mellitus. Abbreviated Report of a WHO Consultation Geneva: WHO.
- 30 Koenig RJ, Peterson CM, Jones RL, Saudek C, Lehrman M, et al. (1976) Correlation of glucose regulation and hemoglobinA1c in diabetes mellitus. *N Engl J Med* 295: 417-420.
- 31 Sherwani SI, Khan HA, Ekhzaimy A, Masood A, Sakharkar MK (2016) Significance of HbA1c Test in Diagnosis and Prognosis of Diabetic Patients. *Biomark Insights* 11: 95-104.
- 32 Ziemer DC, Kolm P, Weintraub WS, Vaccarino V, Rhee MK, et al. (2010) Glucose-independent, black-white differences in hemoglobin A1c levels: a cross-sectional analysis of 2 studies. *Ann Int Med* 152: 770-777.
- 33 Mustafa IO, Tanko Y, Yusuf R, Musa SA (2019) The use of glycated haemoglobin (HbA1c) in determining glycemic control (and relevance of BMI) in diabetic patients in Ahmadu Bello University Teaching Hospital Zaria, Nigeria. *Diab Metab Syndr: Clin Res Rev* 13: 2967-2972.

