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Blood Lipid Peroxidation and DNA Damage in a Nigerian Population of type 2 DiabeticsOlaniyan, O.O.^{1*}, Osadolor, H.B.²

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Abstract

Diabetes mellitus is being considered as a free radical disease owing to the increase in the production of free radical and subsequent deficiency in antioxidant defense mechanisms. There are limited studies regarding lipid peroxidation and antioxidant status among Nigerian diabetics. The aim of this study was to evaluate plasma antioxidant status and Oxidative DNA damage among type 2 diabetes patients with and without vascular complications. A total of 170 age and gender- matched subjects including 120 type 2 diabetics (60 with vascular complications and 60 without vascular complications) and 50 non-diabetes apparently healthy subjects were studied. A significant increase in mean 8-OHdG level indicating DNA damage and MDA indicating lipid peroxidation were observed in parallel to a significant decrease in levels of eSOD, eGSH and TAC indicating antioxidant parameters in diabetics with and without vascular complications as compared to controls and also in diabetics with vascular complications as compared to those without vascular complications. There were positive correlations of eSOD and TAC with MDA and 8-OHdG in diabetics with vascular complications. There were significant lipid peroxidation and oxidative DNA damage in Nigerian type 2 diabetics with greater intensity among vascular complicated patients than those without vascular complications.

Keywords: *Lipid peroxidation, antioxidants, diabetes mellitus, DNA damage, Nigeria*

Introduction

Vascular complications of type 2 diabetes mellitus are a major clinical and public health burden in Nigeria . During the past decades, major socio-demographic changes have occurred in Nigeria (a hitherto known agrarian society), causing people to move from traditional rural jobs to urban areas in their thousands on a daily basis to take on semi-skilled and unskilled labor . This lifestyle changes warranted reduced physical activity and change in dietary patterns (adoption of western lifestyles) which have promoted the development of non-communicable diseases, such as diabetes mellitus . Diabetes mellitus is now considered an emerging clinical and public health problem in Nigeria . Both microvascular and macrovascular complications of diabetes are the major causes of morbidity and mortality in diabetic patients .

Diabetes mellitus is characterized by hyperglycaemia and biochemical alterations of glucose and lipid peroxidation with the subsequent formation of sustained cellular oxidative stress . Diabetes mellitus is also being considered as a free radical disease owing to the increase in the production of free radical and subsequent deficiency in antioxidant defense mechanisms . The produced free radical causes oxidative damage to body biomolecules including lipids, protein and nucleic acids. Hyperlipidaemia has been reported to predispose to increased lipid peroxidation which has been implicated in atherosclerosis development (a macrovascular complication) and other microvascular complications of diabetes mellitus .

Likewise, oxidative stress has been implicated in triggering DNA damage which has been linked to promote cancer risk. Also type 2 diabetics with an higher HbA1c levels has been shown to have an increased propensity to develop gastric, pancreatic, colorectal, breast and liver cancer. This study aims to evaluate lipid peroxidation, blood antioxidant status, and DNA damage among Nigerian type 2 diabetics with and without vascular complications.

Methods

This study was conducted on 120 (45 male and 75 female) type 2 diabetes mellitus patients [categorized into 2 groups of those without vascular complications (DM-VC, n=60), and those with various forms of vascular complications (DM+VC, n=60)], attending a government hospital clinic in Osun State (during November 2016 and August 2017), and 50 healthy (27 male and 29 female) controls of similar ages, were recruited for this study. All subjects were of Yoruba decent in South West Nigeria and not smoking cigarette nor drinking alcohol. The diagnosis of type 2 diabetes mellitus was based on WHO criteria. Vascular complications were defined as those that resulted at least a year after the initial diagnosis of type 2 diabetes mellitus based on standard clinical procedures. Weight and height were measured in indoor clothing without shoes and BMI was calculated as $\text{weight (kg)}/\text{height}^2(\text{m}^2)$.

The work is carried out in accordance with the Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans, and the protocol was approved by the research ethics committee of the Osun State University. Informed written consent was obtained from each patient. Exclusion criteria included patients who had a diagnosis of type 1 diabetes, or any other disease other than type 2 diabetes, abnormal liver functions, chronic inflammatory diseases, acute infections, haematologic disorders, acutely ill patients, malignancy, and pregnant women. Fasting blood samples were drawn and collected from all volunteered subjects into both heparinised and fluoride oxalate vacutainer for biochemical analysis. After centrifugation at 3000 rpm for 15 min, aliquots of plasma and the erythrocytes

were stored separately at -80°C until analysis, repeated freezing and thawing were avoided.

Fasting plasma glucose (FPG) and glycosylated haemoglobin (HbA1c) were determined with enzymatic glucose oxidase hydrolysis and ion exchange resin method using a commercially available Randox, UK and Spectrum Diagnostics, Egypt kits respectively. Total cholesterol (TC), was determined by enzymatic hydrolysis and oxidation method, triglycerides (TG) by enzymatic hydrolysis method while precipitation method was used to determine high density lipoprotein cholesterol (HDL-c), all from Randox, UK. Friedewald *et al.* formula, was used to calculate low density lipoprotein (LDL-c) and VLDL-c was also calculated using Soliman equation: $\text{VLDL-c} = \text{TC} - (\text{HDL-c} + \text{LDL-c})$. Erythrocytes glutathione (eGSH) level and erythrocytes superoxide dismutase (eSOD) activity were analyzed by Chakrabarty *et al.* and Joharapurker *et al.* procedures respectively. MDA was determined using modified method (Song *et al.*, . Plasma 8-OHdG levels were determined using an enzyme linked immunosorbent assay detection kit (Northwest Life Science Specialties, Vancouver, USA) and measured according to the manufacturer's directions.

Data were expressed as mean \pm standard deviation (SD) and assessed by the Student's t-test. The correlation coefficients were determined by Pearson's simple linear regression analysis. Statistical significance was accepted at $p < 0.05$. All statistical analyses were performed with Vassar Stats (<http://vassarstats.net/index.html> assessed on 2nd of June, 2018), an online statistical software was used for the analysis.

Results

Tables 1 shows the pattern of anthropometric and glycaemic control variables of type 2 diabetes patients and controls. There were no significant differences between gender and mean age of the controls and type 2 diabetics, the age range was 35-69 years for control and 35-68 years for type 2 diabetics. The mean diabetes duration of type 2 diabetes patients was 4.4 years, diabetics with vascular complications have a significant

($p < 0.05$) higher diabetes duration years (6.67 years) than those without vascular complications (5.55 years). BMI also showed a significant difference ($p < 0.01$) between the controls and type 2 diabetics, as diabetics without vascular complications have a significant ($p < 0.05$) reduced value (24.86 kg/m^2) to those with vascular complications (26.56 kg/m^2) (range $19-22 \text{ kg/m}^2$ and $19-51.2 \text{ kg/m}^2$ for the control and type 2 diabetics, respectively). All subjects from the control group were of normal weight while, type 2 diabetics have 50% normal weight, 39.17% overweight and 10.83% obese subjects (BMI normal weight and overweight were between $18-25 \text{ kg/m}^2$, $25-30 \text{ kg/m}^2$ respectively while obese is $>30 \text{ kg/m}^2$). Data shown in Table I also reveal that glucose and HbA1c level of G2 is significantly higher than G1 and both G1 and G2 levels are significantly higher than control group. Vascular complicated diabetics have significantly higher ($p < 0.05$) diabetes duration, BMI, FPG and HbA1c values as compared to those without vascular complications. Likewise, there are more overweight and obese individuals among diabetics with vascular complications as compared those without vascular complications.

Data shown in Table 2 reveals that the levels of TC, LDL-c, VLDL-c and TG of the diabetics and the two type 2 diabetes groups (G1 and G2) are significantly higher than the control group, while the HDL-c levels are significantly lower in all study groups as compared to control group. Atherogenic index TC/HDL-c showed a significant increase while atherogenic index HDL-c/LDL-c were significantly reduced in diabetics and in both diabetes groups when compared with the control group. Vascular complicated diabetics also have significantly higher TC, VLDL-c, HDL-c/LDL-c and TG with lower concentrations of HDL-c and TC/HDL-c than those without vascular complications. The low level of LDL-c seen in vascular complicated individuals was not significant when compared to those without vascular complications.

Data of Table 3 shows the level of lipid peroxidation (MDA), erythrocyte superoxide dismutase (eSOD), erythrocyte Glutathione peroxidase (eGSH), plasma total antioxidant capacity (TAC) and DNA damage (8-OHdG) oxidant marker of the study population. In the

present study, plasma MDA and 8-OHdG levels were significantly increased while, eSOD and TAC levels were significantly reduced in diabetes patients and in both diabetes groups in comparison to non-diabetic healthy control eGSH also showed a significant reduced concentration between the groups except in diabetics without vascular complications and healthy individuals. Vascular complicated diabetics have significantly higher ($p < 0.05$) MDA, and 8-OHdG while eGSH, eSOD and TAC levels were reduced as compared to those without vascular complications.

Data in Table 4 show the correlation coefficient of the studied parameters. In the present study, MDA correlated positively with eSOD, TAC and 8-OHdG in diabetics with vascular complications. Also, eGSH levels correlated positively with eSOD and TAC in type 2 patients without vascular complications.

Discussion

Identifying biochemical parameters changes that contribute to vascular complication development has been challenging. As a free radicals disease, diabetes mellitus has been implicated to provoke oxidative stress (due to increased production of reactive oxygen and reduction in antioxidant defences status) leading to development of diabetic macroangiopathy and microangiopathy. Regardless of efforts and advances in management of type 2 diabetes mellitus a significant number of these patients still develop vascular complications. The present study was undertaken with an objective to evaluate plasma lipid profile, MDA, eSOD, TAC, eGSH and 8-OHdG status and its association with disease process in type 2 diabetes mellitus.

Hyperglycaemia, from his study was documented by a significantly increased FPG and HbA1c level in both diabetes cases of the study population which is a major feature of type 2 diabetics. This metabolic derangement is associated to impaired insulin secretion and insulin resistance in adipose tissues which cause a rise in peripheral glucose and subsequent glycosylation and a rise in HbA1c (a long term index of glycaemia) levels, this is consistent with earlier findings.

Abnormal lipid metabolism has also been reported in type 2 diabetes patients, the result from this study confirms derangement in lipid metabolism among type 2 diabetics as TC, LDL-c, VLDL-c and TG of the diabetes groups were significantly higher while HDL-c level was slightly lower than the control group. This might be so as there is increased flux of free fatty acids (FFA) (due to insulin resistance) to the liver which causes increased apolipoprotein (apo) B secretion and subsequent inhibition of microsomal triglyceride transfer protein activity which makes more substrate available for larger VLDL assembly and synthesis. HDL-c however, when present in sufficient amounts in the body system, exhibits both anti-atherogenic and antioxidative properties, but the decreased HDL-c seen in type 2 diabetics is due to enhanced formation of smaller dense HDL (a process mediated by cholesterol ester transfer protein-CETP and resulted due to insulin resistance) from the increase large VLDL synthesis. The observed significant difference seen among these lipid profile parameters between those with vascular and non-vascular complicated diabetics can therefore be used as a pointer / marker of vascular diseases in type 2 diabetes mellitus.

Hyperlipidaemia coupled with poor glycaemic control has also been reported as one of the causative factors for increased lipid peroxidation in diabetes mellitus. Lipid peroxidation is an autocatalytic free radical-mediated destructive process whereby poly-unsaturated fatty acids in cell membranes undergo degradation to form lipid hydroperoxides by-products such as conjugated dienes and malondialdehyde-MDA. In this study, plasma MDA level was significantly increased in diabetes patients and in both diabetes groups in comparison to non-diabetic healthy control. This was consistent with Arif *et al.* and Ikekpeazu *et al.*, who also reported similar increased in MDA levels of their diabetes patients. This study also indicated that vascular complicated diabetics undergoes increased lipid oxidation (as indicated by increased MDA) than those without vascular complications reinforcing the fact that there is a close relationship between hyperglycaemia and lipid oxidation, in line with earlier studies, who found an increased MDA levels in type 2

diabetics with vascular complications than those without vascular complications, and in contrast with the study of who also reported no difference in the lipid peroxidation biomarker N^ε-(hexanoyl)lysine (HEL) among type 2 diabetics with and without vascular complications.

The reduced enzymatic activity of eSOD and reduced concentrations of TAC and eGSH seen from our study clearly shows that type 2 diabetics especially those with complications are exposed to an increased oxidative stress via glucose auto-oxidation due to hyperglycaemia leading to formation of hydrogen peroxide radicals which in turn, inactivates SOD (a primary enzyme involved in the direct elimination of ROS by enhancing the dismutation of superoxide radicals), causing a decreased in scavenging capacity of glutathione-dependent antioxidant defensive system. This result was consistent with earlier studies, who found reduce SOD in their diabetes moreover, found similar total antioxidants capacity, and, with a similar result regarding GSH levels in diabetics, and in contrast with, work who reported no significant change in SOD activity in type 2 diabetics.

This study also shows that there is an increased 8-OHdG level especially in diabetic patients with complications. This finding confirms earlier hypothesis that DNA damage may result in the oxidant-antioxidant status and that DNA damage may be among the more important factors for the onset and/or further aggravation of diabetic complications in poorly controlled diabetes.

Generally, these results show that the increase in lipid peroxidation and oxidative stress with significant DNA damage appears early in type 2 diabetics (before the development of secondary complications), and the measured biomarkers could be important markers in the initiation and progression of diabetic complications and thus can be used as essential therapeutic assessment tool.

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Table 1: Anthropometric and glycaemic control characteristic of type 2 diabetics and controls

Variables	Gender male/female	Age (years)	Duration (years)	BMI (kg/m ²)	FPG (mmol/L)	HbA1c (%)
Control	20/30	54.16±9.61	-	20.91±0.85	4.69±0.71	4.96±0.69
Type 2 Diabetics	45/75	53.66±9.05	4.43±2.55	25.54±3.44	6.92±1.74	7.43±1.49
p-value	NS	NS	<0.01	<0.01	<0.01	
Control	20/30	54.16±9.61	-	20.91±0.85	4.69±0.71	4.96±0.69
DM – VC (G1)	25/35	53.97±9.71	5.55±2.91	24.86±3.43	5.37±0.59	6.16±0.53
p-value	NS	NS	<0.01	<0.01	<0.01	
Control	20/30	54.16±9.61	-	20.95±0.85	4.69±0.71	4.96±0.69
DM + VC (G2)	20/40	53.35±8.41	6.67±2.84	26.56±4.61	8.46±0.45	8.69±0.96
p-value	NS	NS	<0.01	<0.01	<0.01	
G1 vs G2						
p-value	NS	NS	<0.05	<0.05	<0.01	<0.01

Key:

Values are expressed as mean ±SD

BMI: Body mass index

FPG: Fasting plasma glucose

HbA1c: Glycated haemoglobin

NS: Not significant

p-value <0.05

Table 2: Lipid Profile Characteristics of type 2 Diabetics and Control

TC Variables	HDL-c (mmol/L)	LDL-c (mmol/L)	VLDL-c (mmol/L)	TC (mmol/L)	HDL-c HDL-c	TG LDL-c	(mmol/L)
Control	4.60±0.64	1.31±0.39	2.81±0.71	0.48±0.19	3.85±1.31	0.54±0.34	1.05±0.43
Type 2 Diabetics	5.90±0.44	1.26±0.33	3.87±0.55	0.82±0.13	5.07±1.62	0.34±0.13	1.80±0.30
p-value	<0.01	NS	<0.01	<0.01	<0.01	<0.01	<0.01
Control	4.60±0.64	1.31±0.39	2.81±0.71	0.48±0.19	3.85±1.31	0.54±0.34	1.05±0.43
DM – VC (G1)	5.78±0.46	1.08±0.26	3.96±0.58	0.79±0.15	5.79±1.86	0.28±0.10	1.74±0.34
p-value	<0.01	<0.05	<0.01	<0.01	<0.01	<0.01	<0.01
Control	4.60±0.64	1.31±0.39	2.81±0.71	0.48±0.19	3.85±1.31	0.54±0.34	1.05±0.43
DM + VC (G2)	6.02±0.40	1.01±0.29	3.76±0.50	0.84±0.11	4.36±0.87	0.40±0.13	1.87±0.24
p-value	<0.01	NS	<0.01	<0.01	<0.01	<0.05	<0.01
G1 vs G2							
p-value	<0.05	<0.01	NS	<0.05	<0.01	<0.01	<0.05

Key:

Values are expressed as mean ±SD

TC: Total cholesterol

HDL-c: High density lipoprotein cholesterol

LDL-c: Low density lipoprotein cholesterol

VLDL-c: Very low- density lipoprotein cholesterol

TG: Triglycerides

NS: Not significant

p-value <0.05

Table 3: Oxidative stress and DNA damage markers of type 2 diabetics and control

Variables	MDA (µmol/L)	eGSH (mg/gHb)	eSOD (u/gHb)	TAC (mmol/L)	8-OHdG (ng/mL)
Control	2.66±0.37	65.54±1.34	944.70±75.02	1.48±0.33	1.54±0.27
Type 2 Diabetics	3.92±0.44	64.67±3.54	551.09±69.33	0.76±0.24	2.10±0.55
p-value	<0.01	<0.05	<0.01	<0.01	<0.01
Control	2.66±0.37	65.54±1.34	944.70±75.02	1.48±0.33	1.54±0.27
DM – VC (G1)	3.62±0.34	65.24±3.31	583.20±73.08	0.89±0.18	1.65±0.38
p-value	<0.01	NS	<0.01	<0.01	<0.01
Control	2.66±0.37	65.54±1.34	944.70±75.02	1.48±0.33	1.54±0.27
DM + VC (G2)	4.22±0.32	63.80±3.57	519.87±49.50	0.64±0.22	2.54±0.24
p-value	<0.01	<0.05	<0.01	<0.01	<0.01
G1 vs G2					
p-value	<0.01	<0.05	<0.01	<0.01	<0.01

Key:

Values are expressed as mean ±SD

MDA: Malondialdehyde

eGSH: Erythrocytes glutathione

eSOD: Erythrocytes superoxide dismutase

TAC: Total antioxidant capacity

8-OHdG:8-hydroxy-2'-deoxyguanosine

NS: Not significant

p-value <0.05

Table 4: Correlation coefficient between parameters in the study population

	Group	BMI (kg/m ²)	Glucose (mmol/L)	HbA1c (%)	
Age	Control	0.09	0.15	-0.18	
	G1	0.32**	0.27*	-0.02	
	G2	0.15	-0.02	0.06	
Duration	G1	0.27*	0.01	-0.12	
	G2	-0.15	0.03	0.00	
BMI	Control		-0.05	0.34*	
	G1		0.25	0.25*	
	G2		-0.05	0.02	
Glucose	Control			-0.15	
	G1			-0.16	
	G2			0.04	
		eGSH (mg/gHb)	eSOD (u/gHb)	TAC (mmol/L)	8-OHdG (ng/mL)
MDA (µmol/L)	Control	-0.12	0.05	-0.03	-0.13
	G1	0.09	0.17	-0.08	-0.02
	G2	0.09	0.35**	0.27*	0.32**
eGSH (mg/gHb)	Control		0.06	-0.19	-0.22
	G1		0.36**	0.29*	0.24
	G2		-0.10	0.12	0.06
eSOD (u/gHb)	Control			0.02	-0.25
	G1			0.17	0.26
	G2			-0.35**	-0.19
TAC (mmol/L)	Control				-0.02
	G1				0.24
	G2				-0.08

Key:

p-value <0.05

**p<0.01 *p<0.05

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