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EFFECTS OF HYPOGLYCAEMIC AGENT ON STATUS OF MALONDIALDEHYDE, GLYCATED HAEMOGLOBIN AND TOTAL ANTIOXIDANT CAPACITY IN TYPE II DIABETIC PATIENTS ATTENDING SPECIALIST HOSPITAL SOKOTO.

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ABSTRACT

Background: Diabetes Mellitus is a metabolic disorder of multiple aetiology characterized by abnormal Glucose metabolism which results from defects in insulin secretion or insulin action or both. Diabetic Hyperglycaemia triggers glucose autooxidation leading to Reactive Oxygen Species (ROS) release with subsequent molecular and cellular cytotoxicity. Antidiabetic medications inhibits the effects of ROS on cellular and tissues.

Aim: Was to determine the levels of fasting blood glucose (FBG), malondialdehyde and glycated haemoglobin in Type 2 diabetic patients on oral antihyperglycaemic agents.

Materials and Methods: A total of 150 subjects were recruited for the study, the subjects were grouped into 3, Non-DM (apparently healthy, N=50), DM not yet on medication (N=50) and DM on Medication (either Metformin or Daonil, N=50). Patients were asks to fasts, Blood sample were collected for FBG, total antioxidant capacity (TAC), glycated haemoglobin (HBA1c), MDA and antioxidants. Plain container was used for TAC, MDA and ethylene diamine tetra acetic acid was used for glycated haemoglobin, while fluoride Oxalate was used for Fasting Blood Glucose samples. Total antioxidant capacity, malondialdehyde, glucose and HBA1c were analyzed using standard methods. Results generated after laboratory analysis of samples were analyzed using SPSS version 26.0. Results were expressed as Mean ± Standard Error of Mean, P value less than or equal to 0.05 was considered significant while P Value greater than 0.05 was considered not significant.

Results: The results of the present study showed a significantly higher FBG and HBA1c, between Non-DM and DM not on Medication group ($P<0.001$). When compared between Non-DM and DM on Medication Glycated Haemoglobin levels were significantly higher. When compared between DM on Medication and DM not on Medication groups no significant differences were observed in Glycated Haemoglobin ($P>0.05$) with a significantly higher Fasting Blood Glucose levels in DM not on Medication than DM on Medication ($P<0.001$). Equally, when levels of Total Antioxidant Capacity and Malondialdehyde were compared between Non-DM and DM not on Medication groups, a statistically significant difference was observed ($P<0.001$), same was observed, when Non-DM were compared with DM on Medication same was seen ($P<0.001$).

Conclusion: Oral antidiabetic medications reduces hyperglycaemia and abolishes free radical production with MDA reduction while improving TAC in diabetic patients

Keywords: Diabetes Mellitus, Fasting Blood Glucose, Total Antioxidant Capacity, Malondialdehyde, Glycated Haemoglobin

INTRODUCTION

The term diabetes mellitus (DM) describes a metabolic disorder of multiple aetiology characterized by abnormal glucose metabolism resulting from defects in insulin secretion, insulin action, or both [1]. According to WHO (2017), laboratory investigation of fasting blood glucose (FBG) of 7.0mmol/L and random blood glucose (RBG) 11.1mmol/L and HBA1c levels of ≥ 6.5 may be confirmed DM (ADA, 2023). The global prevalence of DM among adults over the years has risen from **4.7%** in 1980 to **10.5%** in 2023 with type II DM making up about 90% of the cases [1,2]. In 2018 DM had a reported prevalence of 9.2% [3]. In Nigeria the prevalence of DM is 5.99% (4), also in North Western Nigeria (our study area inclusive), DM has a reported prevalence of **3.0%** (4). In 2017, diabetes mellitus resulted in approximately 3.2 to 5.0 million deaths [5]. The global economic cost of diabetes related health expenditures in 2018 was estimated at US$727 billion [5].

Hypoglycaemic drugs are used for DM control in type 2 subjects, however, long term use of these agents is associated with a variety of complications [6]. Metformin is an oral hypoglycaemic agent (OHA) of the biguanide class that lowers blood glucose level mainly by decreasing hepatic glucose. Metformin is primarily excreted unchanged by the kidney and renal impairment may cause the accumulation of metformin. Metformin causes elevated concentration of lactic acid, and this has been found to lead to lactic acidosis [7]. A previous study reported that the use of metformin in patients with type 2 DM and advanced CKD was associated with a significantly increased risk of mortality compared with non-users [8].

DM is a chronic metabolic disorder with a rapidly increasing prevalence highlighting the importance of continued research and the need for novel methods to both prevent and treat this pandemic [9]. Although obesity and physical inactivity are known to be major risk factors for type 2 diabetes (T2DM), recent evidence suggests that oxidative stress may contribute to the pathogenesis of T2DM by increasing insulin resistance or impairing insulin secretion. While diabetes mellitus management has largely focused on control of hyperglycemia, the rising burden of this disease is mainly correlated to its vascular complications [9]. This is reflected by a 4-fold increase in the incidence of coronary artery disease, a 10-fold increase in peripheral vascular disease, and a 3- to 4-fold higher mortality rate with as much as 75% of DM patients death occurring from vascular disease [10].

Oxidative stress may play a role in the pathophysiology of diabetes and cardiovascular disease. Consequently, the question of whether antioxidants could have a beneficial effect on reducing the risk of these conditions, especially cardiovascular disease, has been intensively investigated, but the results remain inconclusive [11]. If antioxidants play a protective role in the pathophysiology of diabetes and cardiovascular disease, understanding the physiological status of antioxidant concentrations among people at high risk for developing these conditions, such as people with the metabolic syndrome, is of interest [9].

MATERIALS AND METHODS

STUDY AREA

The study area is Diabetes Mellitus clinic of Specialist Hospital, Sokoto Nigeria. Specialist Hospital, Sokoto is a secondary health institution located in Sokoto State, North-west Nigerian. The metropolitan city of Sokoto State lies between longitude 050.11 to 130.03 east and Latitude 130.00 to 130.06 north and covers an area of 60.33 kms-2.

Sokoto state has twenty-three (23) local governments. There are two main seasons in Sokoto: Dry season and wet or rainy season, the rainy season begins in June and ends in October while the dry season starts from October and last up to May every year, within the dry season, the Hamatan starts from October to February and the warmest months are February to April (temperature may

exceed 45^{0C}). The current prevalence of DM in Sokoto state is 3.0% [4]. The socio-cultural characteristics is homogeneous as majority of its indigenes are Muslims, therefore Islam provides them with a code of conduct of carrying out their way of life. The main occupation of Sokoto state indigenes are farming and animal husbandry while the major languages are Hausa and Fulani.

STUDY POPULATION

The study subjects included diabetic patients attending Diabetic clinic of Specialist Hospital, Sokoto. The study subjects included diabetic patients not on medication, diabetic patients on medication, and non-Diabetic subjects as controls.
Inclusion criteria for diabetic Patients
a. The Diabetic patients with age range between 35-60years.
b. Both males and females were included.
c. Diabetic Patients more than 6months to 1year on medication without any established complication.
d. Patients not on any other medication are also included for the study.
e. Diabetic patients on treatment with Metformin or Daonil.
Exclusion criteria for diabetic Patients
a. Diabetic patients with history of insulin resistance and generalized diseases are excluded.
b. Diabetic patients with an age range outside 35-60yrs are excluded.
d. Diabetic patients on oral anti-diabetic drugs outside metformin, daonil
Inclusion criteria for Controls
a. Apparently healthy subjects without any established diseased condition
b. Both males and females were included
c. Subjects not on any medication for chronic diseases
Exclusion criteria for Controls
a. Age and Sex matched unhealthy subjects were excluded
b. Controls with previous history of insulin resistance were excluded
c. Controls on any medication for chronic illness were excluded
e. Controls with any form of complications were excluded

INFORMED CONSENT

Written consent form was obtained from every study participant.

ETHICAL CONSIDERATION

Ethical clearance was obtained by Ethical Committee of Specialist Hospital Sokoto. The ethical approval number is SHS/SUB/133/VOL.1

DETERMINATION OF SAMPLE SIZE

$$n = \frac{Z^2 P (1-P)}{d2}$$

Where; n = Minimum sample size
z = Value of standard normal deviate which at 95% confidence interval has been found to be 1.96
p = Prevalence of the disease obtained from literature review which is = 3.0% in 2018 (4).
d=standard deviate=0.05

$$n = \frac{(1.96)2x\ 0.030\ (1-0.030)}{0.052}$$

n= 3.8416 x 0.030 x 0.97
0.0025
n =0.11179056
0.0025
n = 44.82
n= 44
in order to enhance Precision, Attrition risk of 14% was added.
14 X 44= 6.16
100
Approximately= 6
New sample size 6+44=50
Therefore 50 samples were adequate for the study.

SAMPLING TECHNIQUES
Subjects Selection
The subjects were selected by convenient random sampling technique.

STUDY DESIGN
This is a cross sectional case control study.
Experimental Design
The data were generated from three groups namely;
1. Non-Diabetic subjects as controls (n=50)
2. Diabetic patients not on Medication (n=50)
3. Diabetic patients on Metformin (n=50)

This design consists diabetic patients not on medication, diabetic patients on medication and non-diabetic subjects as controls.
Initial Diabetes mellitus screening and confirmation was carried out using glucose oxidase-peroxidase method, diabetic and non-diabetic subjects were selected from this criterion, non-diabetic subjects were included as control.
BLOOD SAMPLES COLLECTION AND PROCESSING
Four milliliter (4.0ml) of whole blood was collected using a vacutainer syringe into a plane vacutainer bottle for assay of (TAC) and malondialdehyde (MDA).
Two Milliliter (2.0ml) of whole blood was collected using a monovette vacutainer syringe into fluoride EDTA container for analysis of glucose. Samples in plain containers were centrifuged at 3000rpm for 5minutes, samples were also separated and stored at -20OC until assayed.

ANALYTICAL METHODS
FBS was estimated using glucose oxidase peroxidase method by Trinder P
TAC was estimated using Benzie and Strain method
MDA was estimated by Shah and Walker method
glycated haemoglobin was estimated using fine care machine

STATISTICAL ANALYSIS
The data generated were analyzed using statistical software package (SPSS) version 23. One-way ANOVA and student T-test were used to compare means between the groups for HBA1c, MDA and serum TAC between the two groups.

RESULT
Table 1 shows mean and standard error of mean of Glycated Haemoglobin and FBG. The mean HBA1c levels for control, DM Not on Medication and DM on Medication were 4.00 ± 0.15, 10.25

± 0.48 and 8.20 ± 0.22 respectively. Similarly, FBG for control, DM not on Medication and DM on Medication were 4.47 ± 0.26, 11.58 ± 0.47 and 6.01 ± 0.30 respectively.

Table 2 shows multiple comparison of HBA1c and FBG across all the groups. Other details in the tables below

Table 3 shows mean and standard error of mean of anthropometric measurements across all groups with weight of control, DM not on medication and DM on medication 71.86 ± 2.16, 55.52 ± 1.61 and 63.08 ± 2.01 respectively. The heights were 1.64 ± 0.01, 1.63 ± 0.01 and 1.67 ± 0.02 respectively. Other details refer to table below.

Table 4 shows multiple comparison of the anthropometric measurements between all the groups.

Table 5 shows mean and standard error of mean of TAC and MDA for the study participants, TAC levels were found to be 0.72 ± 0.01, 0.23 ± 0.02 and 0.53 ±0.08 for control, DM not on medication and DM on medication groups while MDA levels were 0.74 ± 0.01, 2.29 ± 0.11 and 1.70 ± 0.07 for control, DM not on medication and DM on medication groups respectively.

Table 6 shows multiple comparison of TAC and MDA between all the groups. For more details refer to tables 1- 6 below:

Table 1: Glycated Hemoglobin and Fasting Blood Glucose among Study Participants

GROUP	N	HBA1c (%)	FBG (mmol/L)
A	50	4.00 ± 0.15	4.47 ± 0.26
B	50	10.25 ± 0.48	11.58 ± 0.47
C	50	8.20 ± 0.22	6.01 ± 0.30
p-value	-	p<0.001	p<0.001

Values are expressed as Mean ± Standard Error of Mean (SEM), N: Total Number of Subjects, HBA1c: Glycated Hemoglobin, FBS: Fasting Blood Sugar, A: Non-DM, B: DM not yet on Medication, C: DM on Medication

Table 2: Post hoc analysis of HBA1c and FBG between groups

GROUP		HBA1c (%)	FBG (mmol/L)
A vs B	-	p<0.001	p<0.01
A vs C	-	p<0.001	p>0.05
B vs C	-	p>0.05	p<0.001

HBA1c: Glycated Hemoglobin, FBS: Fasting Blood Glucose, A: Non-DM, B: DM not yet on Medication, C: DM on Medication

Table 3: Levels of Anthropometric Measurement in Diabetic and Control group

GROUP	N	Weight (kg)	Height (m)	BMI (kg/m^2)
A	50	71.86 ± 2.16	1.64 ± 0.01	27.10 ± 0.96
B	50	55.52 ± 1.61	1.63 ± 0.01	21.17 ± 0.71
C	50	63.08 ± 2.01	1.67 ± 0.02	23.81 ± 0.91
p-value	-	p<0.001	p>0.05	p<0.001

Values are expressed as Mean ± Standard Error of Mean (SEM), N: Total Number of Subjects, BMI (Body Mass Index) A: Non-DM, B: DM not yet on Medication, C: DM on Medication

Table 4: Post hoc analysis of anthropometric measurements between groups

Group		Weight (kg)	Height (m)	BMI (kg/m^2)
A vs B	-	p<0.001	p>0.05	p<0.001
A vs C	-	p<0.001	p>0.05	p<0.001
B vs C	-	p<0.001	p>0.05	p>0.05

A: Non-DM, B: DM not yet on Medication, C: DM on Medication

Table 5: Total Antioxidant Capacity and Malondialdehyde among Study Subjects

GROUP	N	TAC (nmol/L)	MDA (nmol/L)
A	50	0.72 ± 0.01	0.74 ± 0.01
B	50	0.23 ± 0.02	2.29 ± 0.11
C	50	0.53 ± 0.08	1.70 ± 0.07
p-value	-	p<0.001	p<0.001

Values are expressed as Mean ± Standard Error of Mean (SEM), N: Total Number of Subjects, TAC: Total Antioxidant Capacity, MDA: Malondialdehyde, A: Non-DM, B: DM not yet on Medication, C: DM on Medication

Table 6: Post hoc analysis of TAC and MDA between groups

Group		TAC (nmol/L)	MDA (nmol/L)
A vs B	-	p<0.001	p<0.001
A vs C	-	p<0.001	p<0.001
B vs C	-	p<0.001	p<0.001

TAC: Total Antioxidant Capacity, MDA: Malondialdehyde, A: Non-DM, B: DM not yet on Medication, C: DM on Medication

DISCUSSION

Prior to oral antidiabetic treatments, FBG levels were significantly higher in DM not on medication group compared with non-DM group. These results indicated the confirmation of diabetic mellitus in the study participants due to insulin resistance as a result of relative insulin functional deficiency (12). However, FBG was significantly lower in non-DM group compared with DM on medication and DM not on medication ($p<0.001$). FBG was significantly higher in DM not on medication group compared to DM on medication group suggesting that oral antidiabetic medication (Metformin) had antihyperglycaemic effect in the diabetic subjects ($p<0.001$). Furthermore, no significant difference was observed for FBG level between DM on medication and non-DM groups which may suggest that this antihyperglycaemic effect of OHAS (Oral hypoglycaemic agents) was able reduce blood glucose level (13). When compared for FBG between DM on medication and DM not on medication groups, no statistically significant difference ($p>0.05$) was found implying that OHAS was able to reduce ROS production secondary to hyperglycaemia induced glucose autooxidation. The results were in agreements with previous work carried out by Muriki et al. 2020 (14), which reported a reduction of FBG levels in Type 2 DM patients, similar results have been reported in Benin city (Southern part of Nigeria), where Velentino et al. (15) reported a reduction in FBG with the use of OHAS (metformin). The results were in line with previous work on diabetic animal models. Al Hariri et al. reported a reduction in FBG levels in STZ induced diabetic animal model supplemented with bee bread and metformin.

Equally, HBA1c levels were significantly higher in the DM groups (DM not on medication and DM on medication) compared to non-DM group ($p<0.001$). These results indicate that the hyperglycaemic effects due to diabetes mellitus were chronic in nature (progressive and long standing). Furthermore, no statistically significant difference was observed for HBA1c levels between DM on medication and DM not yet on medication groups ($p>0.05$). This result indicates accumulation of sugar in the red cells which in DM not on medication group signifies chronic hyperglycaemia.

In the present study, weight in DM group was significantly lower than non-DM group when compared to non-DM and DM on medication groups ($p<0.001$), the findings was similar with other studies on Indonesian bee bread (17). This was suggested to be due reduced circulating plasma insulin secondary to relative insulin deficiency as a result of type 2 diabetes mellitus (12). Insulin is necessary for the intracellular transport of glucose for further glucose catabolism. However, in DM, body cells are deprived of glucose due to reduced circulating insulin and, in order to compensate for reduced glucose metabolism, there is enhanced proteolysis in skeletal muscle and lipolysis in adipose which could lead to weight loss (12). On the other hand, the body weight in non-DM and DM on medication groups can be attributed to the anabolic effects of insulin (18). A similar study reveals that chronic hyperglycaemia causes abnormal protein and

lipid metabolism, cellular damage and eventually induction of apoptosis in diabetic animal model (16). Therefore, the lower body weight in DM group could be compensated by the consequences of type 2 DM induced hyperglycaemia (19).

In contrast, the DM group on medication showed significantly higher body weight when compared with DM not on medication group . This suggests an antihyperglycaemic effect of diabetic medication in prevailing weight loss or improving body weight in the treated group. This finding was in accordance with previous study on bee bread in Indonesia (17), in which there is significantly higher body weight gain in diabetic animal model supplemented with bee bread. The diabetic group treated with standard antidiabetic drug showed improved body weight was similar to bee bread treated and non-DM groups (17). Body weight was significantly reduced in type 2 DM, possibly due to severe proteolysis and mobilization of proteins for energy generation. With the current study, body weight was significantly improved with the use of OHAS which shifts utilization of proteins and lipid for energy generation.

No statistically significant differences were observed for body height when compared across all the study groups ($p>0.05$) as physiologically all study participants have attained their peak heights and in addition, diabetes has no known proven effects on body height after puberty. When compared between DM on medication and DM not yet on medication groups for BMI, although higher but no statistically significant difference was observed ($p>0.05$). Similarly, a statistically significant difference was observed when DM not yet on medication group was compared to non-DM group ($p<0.001$), the results were in agreement with previous work carried out by Valentine et al. (15) in Benin city (Southern part of Nigeria).

Diabetes mellitus (DM) patients are prone to oxidative stress as a consequence from glucose auto-oxidation following diabetic induced hyperglycaemia leading to free radical release with subsequent end organ damage (20). MDA level is widely used in assessing lipid peroxidation or lipid damage in the cells, tissues and body fluids (21). In the present study, significantly higher level of MDA was found in DM groups compared with the non-DM group ($p<0.001$) suggesting the presence of oxidative stress in diabetic subjects. The finding was in line with other study carried out by Muriki et al. (14), which reported an increase in MDA in type 2 diabetic patients signifying a gross increase in lipid peroxidation as a result of increase in ROS in DM patients. This finding corroborates to other studies in alloxan induced DM rats after 4 weeks (22). Therefore, it is plausible to suggest that diabetic hyperglycaemia may cause higher oxidant activity, ROS release, increased oxidative stress status and eventually may cause cell and tissue damage (21). These findings are similar with other works where by Iranian BB at 200mg and 300mg /kg per day for 4 weeks significantly reduces MDA levels (23). Indonesian BB at 200, 300 and 400 mg/kg per day for 4 weeks significantly reduces MDA (17), Iranian BB at 100 and 200 mg/kg per day reduces MDA and improves TAC after 6 weeks of treatment (23) and Saudi Arabia BB at 300mg/kg per day significantly reduces MDA after 21 days of treatments (16), in diabetic animal models. These findings suggest that anti-oxidative effect of bee bread was active to curtail against hyperglycaemia-induced glucose autoxidation and cellular damage in this diabetic model (24). In the Present study, the antihyperglycaemic effects of antidiabetic medication (metformin) was active to curtail against hyperglycaemia induced glucose autoxidation that produces free radicals and or ROS which leads to lipid peroxidation that raises serum MDA levels in the diabetic subjects. Similarly, MDA was significantly lower in DM on medication when compared with DM not yet on medication group ($p<0.001$) suggesting an ongoing process of lipid peroxidation in DM not on medication group. Equally, MDA levels were significantly higher and statistically significant different was found in the DM on medication group compared with the non-DM group ($p<0.001$), the results suggest that diabetic medication abolishes hyperglycaemia with a reduction but ongoing lipid peroxidation.

TAC is the primary measure and marker to evaluate the status of antioxidants in the body, which reveals the synergistic interaction of all components. The present study showed that, TAC was measured in non-DM, DM not on medication and DM on medication groups. TAC was significantly lower in DM not on medication group compared with non-DM group ($p<0.001$). This finding was in agreement with a previous study by Muriki et al. (14), that reported a reduction in plasma level of TAC due to utilization of its components to curtail against the increased ROS production following DM induced hyperglycaemia in diabetic patients. Similar results have been reported in alloxan induced diabetic animal model (25). This significant reduction of TAC level in DM group might represent low level antioxidant capacity (26). However, TAC was significantly higher in DM on medication group compared with DM group ($p<0.001$). This finding was in line with previous report on animal models where by TAC level was significantly higher in STZ-induced diabetic male rats supplemented with Iranian Bee Bread at doses 100 and 200 mg/kg per day for 4 weeks (23). These findings might suggest that reduction in hyperglycaemia has an ability to reduce oxidative stress in diabetic subjects. However, when compared a statistically significant difference was found for TAC level in DM not yet on medication and DM on Medication groups ($p<0.001$), suggesting that reduction of oxidative stress by diabetic medication occurs due to abolishment of hyperglycaemia in diabetes mellitus. (24). When compared for TAC levels between DM on medication and non-DM groups, a statistically significant differences were observed implying that diabetic medication reduces hyperglycaemia with an ongoing free radical production, hence damage to biomolecules (Proteins, Lipids and Carbohydrates). This may probably be due to poor dietary habit of these diabetic subjects that triggers hyperglycaemia with subsequent production of free radicals and end organ damage.

CONCLUSION:

FBG was significantly higher in DM groups which entails DM was established in the study participants. HBA1c levels were not significant between DM not on Medication and DM on medication groups. This suggests a chronic longstanding hyperglycaemia in the in DM not on Medication and failure of good dietary control in the DM on Medication group.

Body weight was significantly improved with the use of oral antihyperglycaemic agent (metformin), no significant differences were observed for body height across all groups. BMI was also improved with the use of oral antihyperglycaemic agents.

Level of TAC was significantly lower in the Diabetic group, which suggests increase in ROS activities, MDA was also significantly elevated in the DM groups which suggests the presence of lipid peroxidation in vivo.

Conflict of interest:

The authors declared no conflict of interest.

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