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HAEMORHEOLOGICAL PARAMETER, GLYCATED HAEMOGLOBIN AND PHYSIOLOGICAL VARIABLES IN TYPE 2 DIABETIC PATIENTS, IN NAUTH, NNEWI, NIGERIA

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ABSTRACT

It has not been established whether there are alterations in haemorheological parameters and their possible correlation with glycated haemoglobin in type 2 diabetics. Aim: To evaluate haemorheological parameters and HbA1c in type 2 diabetics for proper initiation of diagnostic protocol for diabetic subjects. Materials and Methods: Data from 237 subjects that were grouped into: (A) 59 non diabetic/non hypertensive apparently healthy controls, (B) 60 hypertensives, (C)61 Diabetics and (D)57 hypertensive diabetics was collected. Erythrocyte Sedimentation Rate (ESR) by Westergren Method, Glycosylated haemoglobin (HbA1c) by Cation-exchange method; Plasma Fibrinogen Concentration (PFC) by Clauss Method, Relative Plasma Viscosity (RPV) by Reid and Ugwu method (1987) and Fasting Plasma Glucose(FPG) by Glucose Oxidase method. Ethical approval and informed consent was sought and obtained from the hospital and subjects respectively. Anthropometric measurements were taken using standard methods. The results showed that PFC, ESR, RPV, HbA1c and BMI were significantly increased in the diabetic subjects when compared to the control subjects. PFC(mg/dl)A(380.38±21.96); B(444.73±28.21);C:(471.73±30.17); D(425.63±22.11);ESR(mm/hr):A(8.51±2.83),B(24.99±6.40),C(34.80±8.30),D(36.10±10.29);RPV:A(1.81 ±0.07),B(2.04±0.08);C(2.03±0.08),D(2.02±0.09);HbA1c(%):A(4.81±0.51),B(4.70±0.42)C(8.60±2.06),D(8 .90±1.96);BMI(kg/m2):A,B,C,D:(25.13±3.07,32.84±2.63,34.13±2.41,31.71±3.34);respectively ($p<0.001$). All statistical analyses were performed using SPSS software version 20, and data were expressed as mean± standard deviation. Results compared between various groups were evaluated by Student t-test, one-way ANOVA and Bonferroni test while correlation was done using Pearson's correlation coefficient. From this study, diabetics have haemorheological disturbances which may predispose them to cardiovascular risk. These haemorheological alterations do not predict their poor glycaemic control.

KEYWORDS: Haemorheology, Diabetes mellitus, Glycated haemoglobin, Nnewi.

INTRODUCTION

Diabetes mellitus is a heterogenous group of metabolic disorders characterized by chronic hyperglycemia with disturbances of carbohydrate, fat and protein metabolism which results from diminished or absent secretion of insulin or even by reduced tissue sensitivity to insulin (ADA, 2016; IDF, 2015). Similar to other developing countries, Nigeria is experiencing a rise in the incidence of type 2 diabetes as shown by an increase from 2.0% in 1992 to 2.2% in 1997(Cooper *et al.*,1997; Nigerian National Expert Committee on Non Communicable Disease, 1992) to 4.04% in 2011 (IDF, 2011) to approximately 5.8% (about 6 million) of adults (Uloko *et al.*, 2018). Hyperglycemia is a crucial feature in diabetes and report of abnormal glycation, which can adversely affect haemoglobin and membrane proteins in erythrocyte, has been shown to correlate with reduced membrane fluidity while separately, high values of glycosylated haemoglobin have been found to correlate with decreased deformability of erythrocytes (Bauersachs *et al.*, 1989; Donner *et al.*, 1988; Watala *et al.*,1985). Long term complications of chronic hyperglycaemia arise from underlying microvascular and macrovascular diseases (Odusan *et al.*, 2013). The hemorheological disturbances were found to be present even in diabetes patients without clinically detectable microangiopathy (Le Devehat *et al.*,2001). Paisey et al. reported that hyperviscosity in diabetes was strongly related to hyperglycemia and influenced by the quality of diabetic control yet, there is still paucity of data in the

comprehensive approach to effective diagnosis and management of diabetes that will lead to its reduction or possible eradication in our environment or general population. Therefore, there is need to detect the effect of the degree of long term diabetes control using HbA1c on haemorheological parameters in type 2 diabetes patients and investigate whether or not improved glycaemic control leads to haemorheological changes and further improves microcirculation (Odusan *et al*., 2013; Young *et al.*, 2008).

MATERIALS AND METHODS

Study Area

The study was carried out at the Endocrinology unit of Nnamdi Azikiwe University Teaching Hospital (NAUTH), Nnewi in Anambra State, a tertiary institution in South Eastern Nigeria.

Study Design

Cross sectional study design was used in the study. A total of 237 subjects were recruited in the study which comprised of Sixty one (61) non hypertensive type 2 diabetics, Fifty seven (57) hypertensive type 2 diabetics, Fifty nine (59) non-hypertensive/non-diabetics apparently healthy subjects and Sixty (60) hypertensive non-diabetics apparently healthy individuals serving as control subjects.

Data/Sample Collection

Information (personal lifestyle and medical history) about each subject was collected via questionnaire. Their blood pressure was also measured using sphygmomanometer at the point of blood collection to ascertain absence/presence of hypertension which was defined as history of hypertension, use of BP lowering drugs or systolic blood pressure (SBP)>140 mmHg and diastolic blood pressure DBP)>90 mmHg respectively (Chobanian et al., 2003; WHO 1999). Subjects' weights in kilogram were obtained using a weighing scale and height in meters using stadiometer. The BMI (Body Mass Index) was calculated and expressed as weight (kg)/height(M^2). After obtaining informed consent from each of the subjects, 8ml of venous blood was collected aseptically by venipuncture. 3.5ml was dispensed into sterile container with 0.39ml of 3.8% sodium citrate anticoagulant. 2ml was dispensed into sterile fluoride oxalate bottle for immediate analysis for fasting plasma glucose. 2.5ml was dispensed into sterile K2-EDTA container and analyzed for HbA1c assay. DM was defined as fasting blood glucose >or =7mmol/l or 126mg/dl or on medication for raised blood glucose or with a history of diagnosis of diabetes (WHO 1999).

Sample Size Determination

The sample size was obtained using the formular of Niang *et al.*, (2008), which stated as $N=Z^2pq/d^2$. Hence, the minimum sample size was 59. This indicates that the total sample size should at least be 59, but 237 subjects were used for the study, with the prevalence rate at 4.04% (IDF Atlas, 2011).

Sampling Technique

Purposive sampling technique was employed in selecting the participants based on the inclusion criteria. Patients that gave their consent who also met the selection criteria were recruited as they come to the clinic until the sample size was completed.

ETHICAL CONSIDERATION

Ethical Approval

Ethical approval was sought and obtained from the Ethics Committee of Nnamdi Azikiwe University Teaching Hospital, Nnewi before the commencement of this study (Reference: NAUTH/CS/66/VOL.9/14).

Inclusion Criteria

Participants included in this study were diabetic and non diabetic men and women who were hypertensive or non-hypertensive individual attending Endocrinology Clinic of NAUTH, Nnewi and apparently healthy non diabetic individuals (NAUTH staff, students and others) who gave their consent.

Exclusion Criteria

Those patients with conditions that affect erythrocyte production; or with evidence of chronic medical conditions like renal failure or end stage diabetic nephropathy, liver disease and history of bleeding diasthesis were excluded from the study.

LABORATORY ANALYSIS

Determination of Plasma Fibrinogen Concentration by Clauss Method using Hemostat Fibrinogen Kit (Human Diagnostics Uganda).

Procedure

Determination of HbA1c by Cation Exchange Method using Teco Diagnostic test Kit Spectrophotometrically.

Procedure

A haemolysate constituting 500ul of lysis reagent and 100ul of well mixed whole blood was prepared and allowed to stand for 5minutes. Three milliliter(3ml) of well mixed glycohaemoglobin cation-exchange resin was added to 100ml of haemosylate and mixed for 5minutes in a rotator after which a filter separator was used to separate non-glycosylated haemoglobin bound resin from glycohaemoglobin(GHb) supernatant and the absorbance of the supernatant was taken. 5ml of deionised water was added to 20ul of haemosylate and absorbance of total haemoglobin(THb) taken as well. The ratio of absorbance of glycosylated haemoglobin and total haemoglobin was used to calculate the percentage of glycosylated haemoglobin(GHb%) is calculated as follows:

(GHb%) = R(unknown)/R(standard) X Standard concentration.

Where: R(unknown)= Absorbance of GlycoHb unknown/Abs. of Total Hb unknown

R(standard)= Absorbance of GlycoHb standard /Abs. of Total Hb standard

Determination of Relative Plasma Viscosity Using Reid and Ugwu Method (1987)

Procedure

One milliliter syringe with a hypodermic needle was used to draw plasma into the syringe up to 1ml mark avoiding air bubbles. The plunger was carefully removed and the time taken for the entire plasma to drain was noted. This was done twice for each sample and the average was taken. The whole process was repeated using distilled water. The ratio of the flow rate of plasma to that of water was obtained as the plasma viscosity.

Determination of Fasting Plasma Glucose (FPG) Spectophotometrically using Oxidase Method

Procedure

Ten microlitre of serum was added to one thousand microlitre of the glucose reagent in a test tube and allowed to stand at room temperature for 10mins after which the absorbance was measured using cuvette in spectrophotometer. Level of glucose in mmol/l was calculated using the formular:

Fasting Plasma Glucose (mmol/l) = Absorbance of Test/ Absorbance of Standard X concentration of standard

Quality Control

All the reagents were quality controlled and stored at 2-8^{0}C before use according to the manufacturers guide. In-House control was used for the Full Blood Count test. Daily maintainence were performed on the equipment before use and the standard operating procedures for the tests were strictly adhered to.

Statistical Analysis

The data obtained was analyzed using Statistical Package for Social Sciences (SPSS) version 20. Data were expressed as mean ±SD. Comparism of mean ±SD of all the groups were done using one-way ANOVA and post hoc analysis was done using Bonferroni test. The test for correlation was done using Pearson's correlation coefficient. The level of statistical significance was p <0.05 and at 95% confidence interval.

RESULTS

Table 1 shows that the mean ±SD of RPV was significantly higher in groups B(2.04±0.08); C(2.03±0.08), D(2.02±0.09) when compared with the control group A(1.81±0.07) (p<0.001 in each case). There was significant decrease in mean ±SD in group C(2.03±0.08) when compared to group B(2.04±0.08) (p<0.001). The levels of ESR(mm/hr) was significantly higher in groups B(24.99±6.40); C(34.80±8.30); D(36.10±10.29) when compared to group A(8.51±2.83) (p<0.001). There was significant increase in mean ±SD between group (B and C) and (B and D)(B:24.99±6.40 and C:34.80±8.30) and (B:24.99±6.40 and D:36.10±10.29) (p<0.001) respectively. Similarly, the levels of PFC(mg/dl) was significantly increased in B(444.73±28.21);C(471.73±30.17);D(425.63±22.11)(p< 0.001) when compared to control group A(380.38±21.96). There was significant increase in mean ±SD of group B (444.73±28.21) when compared to C(471.73±30.17) but a significant decrease in mean ±SD of B(B: 444.73±28.21) when compared to (D: 425.63±22.11); and C: 471.73±30.17) when compared to group D: 425.63±22.11) respectively (p<0.001 in each case). HbA1c(%) was significantly increased in groups C(8.60±2.06) and D(8.90±1.96) when compared to control group A(4.81±0.51) (p<0.001 in each case). FPG(mmol/l) significantly increased in the study groups C(7.68±1.55) D(8.19±1.75)(p<0.001) except B(5.32±0.95) when compared to control group A(4.66±0.56).

Table 2 shows that there was significant difference in mean age(yrs) of the study groups B(48.72±8.51); C(55.12±8.25);D(53.66±8.07) (p<0.001 in each case) when compared to the control group A(47.98±11.10), however, group C(55.12±8.25) shows a significantly higher mean(p<0.001).

Table 1: Levels of some haemorheological and glycaemic control variables in control, hypertensives, hypertensives and diabetics, and diabetic group.

	RPV	ESR(mm/hr)	PFC(mg/dl)	HbA1c(%)	FPG(mmol/l)
Control (A)	1.81±0.07	8.51±2.83	380.38±21.96	4.81±0.51	4.66±0.56
Hypertensives only (B)	2.04±0.08	24.99±6.40	444.73±28.21	4.70±0.42	5.32±0.95
Hypertensives + Diabetics (C)	2.03±0.08	34.80±8.30	471.73±30.17	8.60±2.06	7.68±1.55
Diabetics only (D)	2.02±0.09	36.10±10.29	425.63±22.11	8.90±1.96	8.19±1.75
f-value	114.842	170.591	128.101	150.327	106.674
p-value	<0.001	<0.001	<0.001	1.000	<0.001
A vs B	<0.001	<0.001	<0.001	<0.001	0.036
A vs C	<0.001	<0.001	<0.001	<0.001	<0.001
A vs D	<0.001	<0.001	<0.001	<0.001	<0.001
B vs C	<0.001	<0.001	<0.001	<0.001	<0.001
B vs D	0.868	<0.001	<0.001	<0.001	<0.001
C vs D	1.000	1.000	<0.001	1.000	0.194

KEY: RPV-Relative Plasma Viscosity; ESR- Erythrocyte Sedimentation Rate; HbA1c-Glycosylated haemoglobin; FPG- Fasting Plasma Glucose-; PFC- Plasma Fibrinogen Concentration; P value <0.05 is considered significant.

There was significant increase in mean ±SD observed in DBP (mmHg) among groups B(98.75±6.64) and C(100.04±7.61) ($p<0.001$) when compared to control group A (81.32±4.20). There was also, significant increase in levels of SPB(mmHg) observed in groups B(157.53±10.29) and C(161.75±6.63) ($p<0.001$) when compared to controls group A (121.41±6.49).There was also significant decrease in mean ±SD of group D(123.80±8.32) when compared to group B(157.53±10.29) and group C(161.75±6.63)($p<0.001$). BMI(kg/m^2) shows a significant increase in mean of groups B,C,D(32.84±2.63; 34.13±2.41; 31.71±3.34) respectively ($p<0.001$ when compared to the control group A(25.13±3.07). There was no significant difference between groups (B and C) (32.84±2.63& 34.13±2.41) and (B and D)(32.84±2.63 and31.71±3.34) however, there was significant decrease in the levels BMI in group C(34.13±2.41) and group D(31.71±3.34) ($p<0.001$).

Table 2: Levels of demographic and anthropometric data in control, hypertensives, hypertensives and diabetics, and diabetic group.

GROUPS	Age(years)	DBP(mm/Hg)	SBP(mm/Hg)	BMI(kg/m^2)
Control (A)	47.98±11.10	81.32±4.20	121.41±6.49	25.13±3.07
Hypertensives only (B)	48.72±8.51	98.75±6.64	157.53±10.29	32.84±2.63
Hypertensives + Diabetics (C)	55.12±8.25	100.04±7.61	161.75±6.63	34.13±2.41
Diabetics only (D)	53.66±8.07	77.56±5.86	123.80±8.32	31.71±3.34
f-value	9.010	209.566	414.112	112.430
p-value	<0.001	<0.001	<0.001	<0.001
A vs B	1.000	<0.001	<0.001	<0.001
A vs C	<0.001	<0.001	<0.001	<0.001
A vs D	0.004	0.006	0.641	<0.001
B vs C	0.002	1.000	0.032	0.099
B vs D	0.018	<0.001	<0.001	0.196
C vs D	1.000	<0.001	<0.001	<0.001

KEY: - DBP-Diastolic Blood Pressure; SBP- Systolic Blood Pressure; BMI- Body Mass Index

Table 3: Correlation of blood pressure, BMI, FBS, and HbA1c with haemorheological variables in control subjects.

Parameters	Pearson's correlation	RPV	ESR (mm/hr)	PFC (mg/dl)
DBP (mm/Hg)	r	-0.032	-0.057	-.017
	p-value	0.810	0.668	0.897
SBP (mm/Hg)	r	-0.096	-0.055	0.036
	p-value	0.470	0.680	0.786
BMI m^2/kg	r	0.282	0.081	0.015
	p-value	0.031*****	0.544	0.913
FPG (mmol/l)	r	0.122	0.226	-.087
	p-value	0.358	0.085	0.517
HbA1c(%)	r	0.143	-0.048	0.170
	p-value	0.280	0.717	0.202

KEY: - *: significant correlation; RPV-Relative Pslasma Viscosity; ESR- Erythrocyte Sedimentation Rate; HBA1C-Glycated haemoglobin; FPG- Fasting Plasma Glucose-; PFC- Plasma Fibrinogen Concentration; DBP-Diastolic Blood Pressure; SBP- Systolic Blood Pressure; BMI- Body Mass Index

In Table 3, BMI correlated positively with RPV (r: 0.282;p: 0.031) Other parameters show a casual relationship.

Table 4: Correlation of blood pressure, BMI, FPG, and HbA1c with haemorheological variables in Hypertensive subjects.

Parameters	Pearson's correlation	RPV	ESR (mm/hr)	PFC (mg/dl)
DBP	r	0.055	0.065	-0.080
	p-value	0.679	0.622	0.543
SBP	r	-0.036	0.228	-0.042
	p-value	0.787	0.079	0.748
BMI	r	-.0259*	-0.243	0.071
	p-value	0.045	0.062	0.592
FPG	r	0.112	-0.168	0.116

	p-value	0.392	0.201	0.378
HbA1c	r	0.004	0.090	0.335*
	p-value	0.974	0.493	0.009

KEY: - * significant correlation; RPV-Relative Pslasma Viscosity; ESR- Erythrocyte Sedimentation Rate; HBA1C-Glycated haemoglobin; PFC- Plasma Fibrinogen Concentration; DBP-Diastolic Blood Pressure; SBP- Systolic Blood Pressure; BMI- Body Mass Index

In Table 4, there was a negative correlation between BMI and RPV (r: -.0259; p: 0.045.). HbA1c weakly correlated positively with PFC (0.335; p: 0.009). There was no correlation between DBP,SBP, HbA1c with the haemorheological variables.

Table 5: Correlation of blood pressure, BMI, FPG, and HbA1c with haemorheological variables in hypertensive and diabetic subjects

Parameters	Pearson's correlation	RPV	ESR (mm/hr)	PFC (mg/dl)
DBP	r	-0.044	-0.011	0.144
	p-value	0.749	0.936	0.287
SBP	r	-0.213	0.042	0.185
	p-value	0.114	0.755	0.169
BMI	r	-0.125	-0.119	-0.215
	p-value	0.360	0.380	0.108
FPG	r	-0.014	0.134	-0.060
	p-value	0.917	0.321	0.656
HbA1c	r	0.029	0.096	0.016
	p-value	0.832	0.479	0.907

KEY: - * significant correlation; RPV-Relative Pslasma Viscosity; ESR- Erythrocyte Sedimentation Rate; HBA1C-Glycated haemoglobin; FPG- Fasting Plasma Glucose-; PFC- Plasma Fibrinogen Concentration; DBP-Diastolic Blood Pressure; SBP- Systolic Blood Pressure; BMI- Body Mass Index

TABLE 6: Correlation of blood pressure, BMI, FPG, and HbA1c with haemorheological variables in diabetic subjects.

Parameters	Pearson's correlation	RPV	ESR (mm/hr)	PFC (mg/dl)
DBP	r	-0.008	-0.099	0.193
	p-value	0.950	0.446	0.137
SBP	r	0.064	-0.060	0.107
	p-value	0.623	0.645	0.410
BMI	r	0.097	0.057	0.281*
	p-value	0.457	0.661	0.028
FPG	r	-0.208	0.122	0.154
	p-value	0.107	0.349	0.235
HbA1c	r	0.065	0.059	0.014
	p-value	0.616	0.652	0.918

KEY: - * significant correlation; RPV- Relative Plasma Viscosity; ESR- Erythrocyte Sedimentation Rate; HBA1C-Glycated haemoglobin; FPG- Fasting Plasma Glucose-; PFC- Plasma Fibrinogen Concentration; DBP-Diastolic Blood Pressure; SBP- Systolic Blood Pressure; BMI- Body Mass Index

DISCUSSION

In this study, mean levels of the haemorheological parameters (PFC, RPV, ESR) were significantly increased in the diabetic group: (hypertensive and normotensive) and hypertensive non- diabetic when compared with the control subjects. The levels of Plasma fibrinogen concentration (PFC) was significantly increased in the diabetic group (hypertensive and non hypertensive) in this study compared with the non diabetic group (hypertensive non diabetic and apparently healthy control) which is similar to the findings of Odusan *et al*., (2013); Reid *et al.*, (1984). Also, PFC was significantly higher in hypertensive diabetics when compared to normotensive diabetics and non diabetic hypertensives. This increase can be as a result of Relative insulin defficiency in diabetic patients resulting in different protein synthesis with 50% increase in fibrinogen synthesis (Odusan *et al*.,2013). Also, glycosylated fibrinogen is less susceptible to plasma degradation (Reid and Oli, 1986) thereby increasing the plasma levels. However, Reid *et al*., (1984) contrasts the significant increase between hypertensive diabetics and hypertensive non diabetics.

Relative plasma viscosity was significantly raised in the study group (hypertension, normotensive diabetics and hypertensive diabetics) than the control with significantly higher value noted in the hypertensive diabetics when compared to the normotensive diabetics. This result is similar to the findings of Memeh and Reid, (1998) and Odusan *et al., (*2013). This suggests that diabetics may be prone to thrombotic complication since abnormal plasma viscosity contributes to disturbance in normal blood flow and metabolism thereby playing essential role in development of both micro-circulatory disorders and hypertension (Memeh and Reid, 1998).

Estimation of glycosylated haemoglobin provides useful information about long term glycemic control. In this study, glycosylated haemoglobin was significantly increased in the diabetic group (normotensive and hypertensive) compared to the non diabetic group (hypertensive and control). This corresponds to findings of Analike *et al*.,(2019); Chinenye *et al.*,(2012); Sakpa and Idemudia, (2014) and Adebisi *et al*.,(2009) in their different studies. This implies that there was poor glycemic control in the diabetic subjects with mean of HbA1c >8.5% and this did not meet the ADA and IDF glycemic target of 7% and 6.5% (Leszek, 2009) respectively. Reason for this is multifactorial with financial constraint as key factor (Sakpa and Idemudia, 2014). The mean HbA1c of the non diabetic hypertensive and control subjects were within the normal range (less or equal to 6.5%) however, there was a significant higher difference in the control compared to the hypertensive subjects which can be as a result of undetected impaired glucose tolerance among the control, since their screening was done via a one spot sample collection, hence, authenticating the use of HbA1c as a more effective diagnostic test for diabetes. Another reason may be the type of antihypertensive drug used which may have a role in causing relative hypoglycaemia (Ronald *et al.,* 1997), hence the significant difference in HbA1c. Fasting plasma glucose was significantly higher in the diabetic group (normotensive and hypertensive when compared to non diabetic group (hypertensive and control subjects). This is in keeping with reports of disturbances in carbohydrate metabolism which occurs in diabetics. FPG did not correlate with PFC, RPV, ESR. A significantly increased mean ±SD for HbA1c and PFC in the diabetic group was noted, though there was only a casual relationship between them in this study and this contrasts Adebisi *et al*., (2009). Drugs and environment may be reason for this.

Erythrocyte sedimentation rate (ESR) was significantly increased in the study groups (hypertensive, normotensive diabetics and hypertensive diabetics) respectively when compared to the control subjects. This corresponds to the findings of William *et al.,* (2018) and Odusan *et al.,* (2013). Hypertensives and diabetics are known to be prone to recurrent infections which are seen in the increased levels of ESR as a result of chronic inflammatory processes which occur in these subjects.

The mean age in the diabetic group (normotensive and hypertensive) in this study was 53.66±8.07 and 55.12±8.25. This is similar to the result of Sakpa and Idemudia, (2014) and observations from McMichael and Beaglehole, (2000) and Wild *et al*., (2004) in other Sub Saharan African countries with an age range of 45-64 years but, it's low when compared to reports from developed countries where most of the diabetic patients were over 64 years (Wild *et al.,* 2004).The hypertensive diabetic subjects had a significant higher (55.12±8.25) ($p<0.001$) mean age when compared with the control (47.98±11.10). This implies that increased age may be a risk factor for cardiovascular disease in diabetics.

The mean ±SD levels of BMI was significantly higher in the study groups (hypertension, normotensive diabetics and hypertensive diabetics) compared to the control subjects. This is similar to the findings of Sakpa and Idemudia,(2014) suggesting that diabetes is associated with increased BMI. Diabetics with hypertension had significantly raised BMI than those without. Urbanization which increase the risk of diabetes 2-5 fold is associated with decreased physical activity energy expenditure(PAEE)(Uloko *et al.,*2018; Chalterjee *et al*.,2010; Saquib *et al.,* 2012), an independent risk factor for metabolic syndrome (Assah *et al.,*2011) may be the cause of increased BMI in this group.

BMI correlated with PFC ($r=0.281$, $p<0.028$) in the normotensive diabetics in this study and this is similar to the findings of Harsoor *et al*., (2014).

DBP and SBP were significantly raised in the hypertensive group (diabetics and non diabetics) compared to non hypertensive group (normotensive diabetics and control subjects) with the levels significantly higher in hypertensive diabetics than their normotensive diabetic counterparts. This is similar to the findings of Memeh, (1990). There was no significant difference between their levels in the non diabetic hypertensive and hypertensive diabetic subjects.

This study reveals that fibrinogen levels and relative plasma viscosity are increased in diabetics especially when they coexist with hypertension.

Glycaemic target according to ADA and IDF have not been achieved in our locality hence proper management protocol to reverse poor glycaemic control should be initiated.

There is increase in haemorheological parameters which may predispose to impaired blood flow (which is a CVD risk factor) in diabetics. There is no correlation between haemorheological parameters in this study with glycaemic control variable (HbA1c).

CONCLUSION

This study reveals that hyperglycemia and poor glycaemic control is substantial in diabetic patients in

our environment with increased risk of cardiovascular complications when it coexist with hypertension as seen in their altered haemorheological parameters. Hence, the control of blood glucose level is important to reduce the occurrence of these complications. Thus, evaluation of haemorheological parameters and estimation of glycosylated haemoglobin levels in diabetics is needful as it provides useful information about cardiovascular risk factors and long term glycaemic control, thereby optimizing patient care.

Since alteration in haemorheological parameters is substantial in type 2 DM, with significant poor glycemic control in this study, it is therefore recommended that haemorheological parameters should be an affordable and integral part of diagnostic assessment in diabetic patients for early detection and arrest of cardiovascular complications thereby reducing the burden of morbidity and mortality in these patients. This study advocates for a packaged protocol termed "Diabetes Algorithm Test" (HbA1c, PFC, RPV and FBC), that will be a compulsory, free or very affordable preliminary screening for all diabetics especially hypertensive diabetics. It is recommended that government, non-governmental organizations and hospital management should create policies that can make this realizable.

Limitation of the Study

For further studies, the lifestyle of the subjects, that is, whether they are smokers or non smokers and the duration of diabetes, noted as limitation in this study may be considered.

Declaration of Conflict of Interest

There is no conflict of interest in this study.

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