INTERNATIONAL JOURNAL OF APPLIED BIOLOGY AND PHARMACEUTICAL TECHNOLOGY

www.ijabpt.com Volume-5, Issue-4, Oct-Dec-2014 Coden : IJABPT Copyrights@2014

ISSN : 0976-4550

Received: 25th July-2014 *Revised: 3rd Aug-2014* *Accepted: 15th Aug-2014*

Research article

THE RELATIVE INCIDENCE OF HYPERTENSION COMORBIDLY OCCURRING WITH DIABETES IN ABO/RHESUS BLOOD GROUPS AND HAEMOGLOBIN GENOTYPES IN SOUTH-WESTERN NIGERIA

Adio J. Akamo[*1], Elizabeth A. Balogun[2], Oladipo Ademuyiwa[1], David A. Ojo[3], Olusola A. Talabi[4], Christopher, A. Erinle[5] and Regina N. Ugbaja[1]

[1]Department of Biochemistry, College of Natural Sciences, Federal University of Agriculture, P. M. B. 2240, Abeokuta, Ogun State, Nigeria;
[2]Department of Biochemistry, University of Ilorin, P.M.B. 1515, Ilorin, Nigeria;
[3]Department of Microbiology, College of Natural Sciences, Federal University of Agriculture, P. M. B. 2240, Abeokuta, Ogun State, Nigeria;
[4]Health Centre, Federal University of Agriculture, P. M. B. 2240, Abeokuta, Ogun State, Nigeria.
[5]Ogun State Hospital, Ijaiye, Abeokuta, Ogun State, Nigeria.
Corresponding Author* Corresponding Author Phone Number: +234806 465 0070
Corresponding Author E-mail address: **ajayngng@yahoo.com**

ABSTRACT: ABO blood groups, Rhesus factors and haemoglobin genotypes are all inherited blood characters. This this study was aimed at investigating the relative incidence of hypertension comorbidly occurring with type 2 diabetes mellitus (T2DM) in ABO/Rhesus blood groups and haemoglobin genotypes in some residents of Abeokuta, South-Western Nigeria. Age and sex matched control subjects (n=150) and patients (n=470) [hypertensive non-diabetics (HND, n=179), normotensive diabetics (ND, n=132), hypertensive diabetics (HD, n=159)] presenting at the Medical Out-Patient Clinic of the State Hospital, Abeokuta, Nigeria were recruited. Standard electrophoretic and haemagglutination techniques were employed in testing the blood samples. Fasting plasma glucose, haemoglobin, plasma creatinine and plasma urea were determined spectrophotometrically. Blood pressure and its component were also determined. Prevalence of hypertension and/or T2DM was observed in subjects with blood O followed by A. The phenotype frequencies of ABO blood group in both hypertensive and diabetic patients and controls (both sexes) are in the order O>A>B>AB. The RhD+ and RhD- distribution were similar in patients and their control counterparts ($p > 0.05$). The spectrum of haemoglobin electrophoresis among the controls and patients can be shown with a general formula HbAA>HbAS>HbAC>HbSS>HbSC>HbCC except in control female and HD female. The results obtained suggest that there is a strong positive relationship between blood group O and hypertension and/or T2DM. Large studies in other ethnic groups are needed to confirm these results.
Keywords: ABO Blood groups, Rhesus factors, haemoglobin genotypes, type 2 diabetes, hypertension

INTRODUCTION

Hypertension (high blood pressure, HBP) is a chronic medical condition in which the systemic arterial blood pressure is elevated. It is classified as either primary (essential) or secondary. About 90–95% of cases are termed primary hypertension, which refers to HBP for which no medical cause can be found. The remaining 5–10% of cases (Secondary hypertension) is caused by other conditions that affect the kidneys, arteries, heart, or endocrine system (Carretero and Oparil, 2000). Persistent hypertension is one of the risk factors for stroke, myocardial infarction, heart failure and arterial aneurysm, and is a leading cause of chronic kidney failure (Pierdomenico *et al*., 2009).

Diabetes mellitus (DM) is a complex disease of carbohydrate, lipid and protein metabolism characterized by hyperglycemia resulting from defects of insulin secretion and/or increased cellular resistance to insulin (International Diabetes Federation. 2013).

DM is generally divided as insulin-dependent diabetes mellitus (IDDM or type I DM), characterized by an absolute deficiency of circulating insulin and non-insulin-dependent diabetes mellitus (NIDDM or type 2 DM/T2DM), characterized by elevated insulin levels that are ineffective in normalizing blood sugar levels or by impaired insulin secretion (International Diabetes Federation. 2013).

It was reported that T2DM is the most common type, accounting for 90 - 95% of all diabetic cases (American Diabetes Association, 2004). In 2013, it was estimated that there were 382 million people with diabetes. Its incidence is increasing rapidly, and it is estimated that by 2030, this number will almost double (International Diabetes Federation. 2013)

Both hypertension and T2DM are cardiovascular diseases (CVD) risk factors (International Diabetes Federation, 2013; Oladapo *et al*., 2010; Pierdomenico *et al*., 2009; American Diabetes Association. 2004; Carretero and Oparil, 2000. CVD is emerging as a significant health problem in sub-Saharan countries such as Nigeria, with a population of over 160 million. These countries are undergoing epidemiological transition from communicable to non-communicable diseases. Epidemiological transition has been closely linked to changes in the demographic, social and economic status of various populations, causing a global rise in chronic diseases, especially CVD. Nigeria has a double burden of communicable and non-communicable diseases (Oladapo *et al*., 2010).

Blood is man's complete and unchangeable identity. Although almost 400 blood grouping antigens have been reported, the ABO and Rh are recognised as the major and clinically significant blood group antigens. Rhesus blood group system was the 4th system discovered and yet it is 2nd most important blood group from the point of view of transfusion (Khan *et al*., 2010). The majority of ABO determinants are expressed on the ends of long polylactosamine chains (Daniels, 2002). No disease is known to result from the lack of expression of ABO blood group antigens, but the susceptibility to a number of diseases has been interrelated to a person's ABO phenotype. Such correlations remain conflicting and include the observation that gastric cancer is more common in blood group A individuals, whereas gastric and duodenal ulcers occur more commonly among blood group O individuals (O'Donnell, 2001). Also, there are several common forms of sickle cell diseases such as SS, SC and S-beta thalassemia. The clinical course of sickle cell disease is extremely variable. Some patients have nearly no symptoms while others are severely incapacitated (Adeyemo and Soboyejo, 2006). In Nigeria, data is lacking on frequency distribution of ABO, Rh blood groups and blood genotypes among patients with non-communicable diseases such as hypertension and/or T2DM. Therefore, this study was designed to determine the relative incidence of hypertension comorbidly occurring with T2DM diabetes mellitus in the different ABO/Rhesus blood groups and haemoglobin genotypes in Abeokuta, South-Western Nigeria.

MATERIALS AND METHODS

Study area and subjects

The study was carried out in Abeokuta the capital city of Ogun State, Nigeria, between 2010 and 2012. Abeokuta is an urban township in Southwestern Nigeria with about 800,000 inhabitants based on an annual growth rate of 3.5% from the 1991 census figures (Department of Statistics, Ministry of Finance, Abeokuta, Nigeria). Its topography is undulated i.e. not leveled but rocky. In it is situated the Federal University of Agriculture, Abeokuta with a population of about 15,000 made up of academic and non-academic staff and students from all over the country, with a preponderance of the population from the western coast. They basically consume typical Nigerian low fat, high carbohydrate and protein diets. Apart from this, they live an active life-style in the community (Ademuyiwa *et al*., 2008).

Patients presenting at the Medical Out-patient Clinic, State Hospital, Ijaiye, Abeokuta, Ogun State, Nigeria were used for the study. The protocol for the study was approved by the Research and Ethics Committee of the State Hospital as well as the postgraduate committe of the Department of Biochemistry, Federal University of Agriculture, Abeokuta. Patients (diagnosed by a Consultant Physician in the Department of Internal Medicine of the State Hospital) were made of age and sex-matched indigenous Nigerian normoglycaemic hypertensives; normotensive type 2 diabetes mellitus and patients with comorbidity of hypertension and type 2 diabetes. The diagnosis of diabetes mellitus was based on the World Health Organisation criteria (Idogun *et al*., 2007). Patients on oral hypoglycaemic drugs or whose diagnosis of diabetes was made at the age of 40 years and above with no record of ketosis were considered to have type 2 diabetes mellitus. Hypertensive patients were diagnosed based on World Health Organisation-International Society of Hypertension Guideline cut-off point of 140 mmHg and above for systolic and/or 95 mmHg and above for diastolic blood pressure, and also if it was previously detected and the subject was on treatment (Carretero and Oparil, 2000).

Inclusion criteria included being hypertensive for ≥ one year, use of neutral antihypertensive agents such as calcium channel blockers, angiotensin converting enzyme inhibitors, and angiotensin II receptor blockers. Excluded from the study during routine interviews, clinical investigations and laboratory tests were patients with a history of smoking, drinking alcohol, human immunodeficiency virus (HIV), systemic lupus erythematosus, systemic inflammation or systemic infection, taking oral contraceptives, lipid lowering drugs.

Age and sex-matched volunteers certified clinically and biochemically to be healthy, on no medication; normotensive and normoglycaemic served as controls. They were made of staff and students of Federal University of Agriculture, Abeokuta, Nigeria. They were recruited in the study at the same period with the patients. Participation in the study by individual subject was voluntary. Before enrollment in the study, all subjects were informed about the objectives and requirements of the study, as well as the risks and discomfort that might be involved in participating in the study. Demographic data including age, sex, race, and duration of hypertension and diabetes were collected using questionnaire. Table 1 summarizes the study population.

Table 1: Study population

Subject	Male	Female
Control	74	76
Hypertensive non-diabetics	76	103
Normotensive diabetics	64	68
Hypertensive diabetics	68	91

Measurements of blood pressure

Blood pressure and pulse were measured two times on the left arm of each subject in a supine position using Omron manual inflation blood pressure monitor (model HEM. 412C, Omron Healthcare Inc. Illinois, USA). Each measurement was spaced twenty minutes apart and was performed before collection of blood samples. The average of the two measurements was used for all analyses. To obtain the final measure of blood pressure, the mean of the first two readings was calculated, unless the difference between these readings was greater than 10 mmHg, in which case the mean of the two closest of three measurements was used as the SBP and DBP values. Pulse pressure (PP) was calculated as SBP minus DBP. Mean arterial pressure (MAP) was estimated as (SBP+2DBP)/3 (Napoli and Papa, 2003).

Collection of blood samples

Blood samples (10.0 ml) were collected between 08.00 a.m. and 11.00 a.m. on each clinic day from the antecubital vein of the participants after an overnight fast for 12-14 hours. The blood was transferred into a lithium heparin anticoagulated tube, and mixed gently by inverting the stoppered tube several times. The blood samples were stored in a cooler box and transferred to the laboratory for analyses.

Biochemical assays

Plasma was separated from erythrocyes. Fasting plasma glucose (FPG), creatinine and urea concentrations were determined spectrophotometrically also according to the assay methods (Tietz, 1995). Haematocrit and haemoglobin were also determined as discribed by (Tietz, 1995). Blood grouping and heamoglobin genotypes were done according to methods described by (Adeyemo and Soboyejo, 2006). Briefly, for the ABO and Rh test, a drop of blood from each subject was placed on a clean white tile in four places. A drop of each of the antisera, anti A, anti B and anti D was added and mixed with each blood sample, with the aid of glass rods. Blood groups were determined on the basis of agglutination. For the study of blood genotype, cellulose acetate electrophoresis technique was used to determine haemoglobin genotype. A small quantity of venous blood was placed on a tile and mixed with three drops of water to lyse. With the aid of an applicator, the haemolysate was placed on the cellulose acetate paper. Electrophoresis in Tris buffer solution was for 15-20 minutes at an e.m.f of 230V. Haemolysates from blood samples of known genotypes were run as control.

Statistical analysis

Data obtained were entered into SPSS (Statistical Package for Social Sciences) software for Window version 16 (SPSS Inc., Chicago, Illinois, USA). Data were expressed as Mean±S.E.M. Qualitative variables were expressed as count and percentage of status or category. Analysis of Variance (ANOVA) was carried out to test for the level of homogeneity among the groups. Where heterogeneity occurred, the groups were separated using Duncan Multiple Range Test (DMRT). The level of interaction among the parameters was determined using Pearson correlation. p values of < 0.05 were considered to be statistically significant.

RESULTS

The demographic and clinical characteristics of the subjects are shown in Table 2. The controls, hypertensive non-diabetics (HND), normotensive diabetic (ND) and hypertensive diabetics (HD) were similar ($p > 0.05$) in age. The duration of diagnosis of hypertension was 5.04±2.19 years among the HND male and 4.87±1.60 years among the HD male; it was 5.26±1.66 years and 4.94±1.62 years for the HND female and HD female respectively ($p < 0.05$).

The duration of diagnosis of diabetics was also similar ($p > 0.05$) between the ND male, HD male, ND female and HD female; 4.07±1.55 years, 4.05±1.48 years, 4.22±1.19 years and 3.98±1.09 years respectively. In the diabetic patients, fasting plasma glucose (FPG) was similar ($p > 0.05$) among the ND male and ND female (196.42±3.66 mg/dL vs 188.97±4.43 mg/dL ($p > 0.05$) but significantly ($p < 0.05$) higher when compared with HD male and HD female patients (174.39±3.73 mg/dL vs 172.24±3.71 mg/dL). Blood pressure increased significantly ($p < 0.05$) among the HND male, HD male, HND female and HD female (171.26±3.79/109.25±2.12 mmHg, 177.69±3.16/111.13±2.17 mmHg, 168.98±2.80/105.17±1.80 mmHg and 173.54±3.08/106.01±1.97) respectively when compared with their corresponding controls. In both sexes, the pulse pressure (PP), mean arterial pressure (MAP) and heart rate were significantly ($p < 0.05$) increased in HND and HD when compared with their respective control counterparts. While no significant difference ($p > 0.05$) was observed in the mean haematocrit and haemoglobin values of ND male, ND female and HD female when compared with their control counterparts; haematocrit and haemoglobin values of HND male and HND female increased significantly ($p > 0.05$). However, the haematocrit and haemoglobin of HD male decrease significantly ($p < 0.05$). Plasma creatinine increased significantly ($p < 0.05$) as a result of the presence of either or both diseases. The increase was more marked in HD male. While ND male plasma urea has no significant ($p > 0.05$) difference when compared with their control counterparts, plasma urea in other patients increased significantly ($p < 0.05$), the increase was more marked in HND male. Quantitatively plasma urea of the male and female test subjects was between 8% to 18% and 20% to 33% respectively higher than their control counterparts. There was significant ($p < 0.05$) difference in gender creatinine and urea concentrations. In both controls and patients, plasma creatinine and urea were significantly ($p < 0.05$) higher in male when compared with their female counterparts.

Table 2: Demograhic and clinical characteristics of the subjects

	Control male	Hypertensive non-diabetics male	Normotensive diabetics male	Hypertensive diabetics male	Control female	Hypertensive non-diabetics female	Normotensive diabetics female	Hypertensive diabetics female
Age (years)	42.58±1.36^{a}	43.29±1.31^{a}	46.45±1.35^{a}	46.33±1.52^{a}	42.70±11.61^{a}	46.75±1.17^{a}	42.46±11.49^{a}	44.80±1.32^{a}
SBP (mmHg)	117.16±1.20^{a}	171.26±3.79^{c}	114.88±1.24^{a}	177.69±3.16^{d}	113.51±1.11^{a}	168.98±2.80^{b}	117.71±2.79^{a}	173.54±3.08^{c}
DBP (mmHg)	77.50±0.98^{a}	109.25±2.12^{b}	78.62±0.76^{a}	111.13±2.17^{b}	76.12±1.15^{a}	105.17±1.80^{b}	78.97±0.79^{a}	106.01±1.97^{b}
PP (mmHg)	39.66±1.30^{a}	62.01±1.96^{b}	36.25±1.08^{a}	66.56±1.47^{bc}	37.39±0.96^{a}	63.81±1.56^{bc}	38.74±2.65^{a}	67.18±1.96^{c}
MAP (mmHg)	90.72±0.86^{a}	129.92±2.63^{b}	90.71±0.81^{a}	133.32±2.45^{b}	88.58±1.04^{a}	126.44±2.05^{b}	91.88±1.20^{a}	128.52±2.21^{b}
Heart rate (beats/mins)	84.60±0.92^{a}	123.76±1.38^{b}	82.86±0.93^{a}	124.96±1.70^{b}	84.14±0.87^{a}	123.40±1.57^{b}	84.06±0.87^{a}	123.04±1.81^{b}
FPG (mg/dL)	71.04±1.32^{a}	77.84±1.40^{a}	196.42±3.66^{c}	174.39±3.73^{b}	75.36±1.41^{a}	75.36±1.73^{a}	188.97±4.43^{c}	172.24±3.71^{b}
Duration of HTN (yrs)	0.00±0.000^{a}	5.04±2.19^{b}	0.00±0.000^{a}	4.87±1.60^{b}	0.00±0.000^{a}	5.26±1.66^{b}	0.00±0.000^{a}	4.94±1.62^{b}
Duration of T2DM (yrs)	0.00±0.000^{a}	0.00±0.000^{a}	4.07±1.55^{b}	4.05±1.48^{b}	0.00±0.000^{a}	0.00±0.000^{a}	4.22±1.19^{b}	3.98±1.09^{b}
Haematocrit (%)	43.34±0.72^{b}	46.60±0.85^{c}	43.44±0.72^{b}	39.26±0.46^{a}	39.16±0.68^{a}	41.84±0.48^{b}	39.24±0.67^{a}	38.58±0.57^{a}
Haemoglobin (g/dL)	14.41±0.23^{b}	15.41±0.28^{c}	14.42±0.23^{b}	13.07±0.15^{a}	13.17±0.25^{a}	14.16±0.19^{b}	13.23±0.24^{a}	12.84±0.19^{a}
Plasma creatinine (mg/dL)	0.97±0.03^{ab}	1.14±0.04^{cd}	1.10±0.04^{cd}	1.20±0.05^{d}	0.88±0.04^{a}	1.05±0.03^{bc}	1.10±0.04^{cd}	1.09±0.04^{cd}
Plasma urea (mg/dL)	29.24±0.81^{b}	34.59±1.08^{d}	31.53±1.30^{bc}	33.88±0.92^{d}	24.25±1.106^{a}	32.45±1.04^{cd}	29.12±1.04^{b}	30.67±0.97^{bc}

Each value represents the mean±S.E.M. Values within the same row with different superscripts are significantly different at $p<0.05$

SBP, systolic blood pressure; DBP, diastolic blood pressure; PP, pulse pressure; MAP, mean aterial pressure; FPG, fasting plasma glucose; HTN, hypertension.

The results as presented in Table 3 showed the blood groups (ABO and Rhesus systems) that were seen among the patients and controls. O and A blood group showed positive or direct association with HND, ND and HD in both male and female patients when compared with their control counterparts ($p < 0.05$). Prevalence of hypertension and/or T2DM was observed in subjects with blood O followed by A. However, no significant ($p > 0.05$) association was found between patients and blood group B and AB. The phenotype frequencies of ABO blood group in both hypertensive and diabetic patients and controls (both sexes) are in the order O>A>B>AB. The frequencies of RhD blood groups are also shown in Table 3. The RhD+ and RhD- distributions were similar in all patients and their control counterparts ($p > 0.05$).

Table 4 shows the frequency of RhD phenotype to the ABO blood groups. RhD negative was more pronounced in blood group A in HND male when compared with other RhD negative. O^+ followed by A^+ blood group showed positive or direct association with HND, ND and HD in both male and female patients when compared with their control counterparts ($p < 0.05$). Prevalence of hypertension and/or T2DM was observed in subjects with blood O^+ followed by A^{+}.

The percentages of the various haemoglobin genotypes obtained in this study are shown in Table 5. There was significant ($p < 0.05$) difference in the distribution of haemoglobin electrophoresis among the patients and the controls. The spectrum of haemoglobin electrophoresis among the controls and patients can be shown with a general formula HbAA>HbAS>HbAC>HbSS>HbSC>HbCC except in control female and HD female.

Table 3: Relative incidence of hypertension and/or diabetes in ABO/Rhesus blood

Group	Control male	Hypertensive non-diabetics male	Normotensive diabetics male	Hypertensive diabetics male	Control female	Hypertensive non-diabetics female	Normotensive diabetics female	Hypertensive diabetics female
O	43(58.1)	32(42.1)	26(40.6)	24(35.5)	41(53.9)	46(44.7)	30(44.1)	41(45.1)
A	21(28.4)	27(35.5)	27(42.2)	27(39.7)	24(31.6)	32(31.1)	27(39.7)	32(35.2)
B	7(9.5)	12(15.8)	6(9.4)	7(10.3)	8(10.5)	19(18.4)	6(8.8)	14(15.4)
AB	3(4.1)	5(6.6)	5(7.8)	10(14.7)	3(3.9)	6(5.8)	5(7.4)	4(4.4)
Rh.D+	61(82.4)	59(77.6)	52(81.2)	52(76.5)	62(81.6)	85(82.5)	53(77.9)	73(80.2)
Rh.D-	13(17.6)	17(22.4)	12(18.8)	16(23.5)	14(18.4)	18(17.5)	15(22.1)	18(19.8)
Values are frequencies. Figures in parentheses are in percentage.								

Table 4: Frequency of Rh.D in the various ABO types of subjects

Group	Control male	Hypertensive non-diabetics male	Normotensive diabetics male	Hypertensive diabetics male	Control female	Hypertensive non-diabetics female	Normotensive diabetics female	Hypertensive diabetics female
O+	38(51.4)	25(32.9)	23(35.9)	19(27.9)	33(43.4)	39(37.9)	24(35.3)	35(38.5)
O-	5(6.8)	7(9.2)	3(4.7)	5(7.4)	8(10.5)	7(6.8)	5(7.4)	5(5.5)
A+	16(21.6)	21(27.6)	21(32.8)	21(30.9)	19(25.0)	24(23.3)	20(29.4)	24(26.4)
A-	5(6.8)	6(7.9)	6(9.4)	6(8.8)	5(6.6)	8(7.8)	7(10.3)	9(9.9)
B+	5(6.8)	10(13.2)	4(6.2)	6(8.8)	7(9.2)	17(16.5)	5(7.4)	11(12.1)
B-	2(2.7)	2(2.6)	2(3.1)	1(1.5)	1(1.3)	2(1.9)	2(2.9)	3(3.3)
AB+	2(2.7)	3(3.9)	4(6.2)	6(8.8)	3(3.9)	5(4.9)	4(5.9)	3(3.3)
AB-	1(1.4)	2(2.6)	1(1.6)	4(5.9)	0(0.0)	1(1.0)	1(1.5)	1(1.1)
Total	74(100.0)	76(100.0)	64(100.0)	68(100.0)	76(100.0)	103(100.0)	68(100.0)	91(100.0)
Values are frequencies. Figures in parentheses are in percentage.								

Table 5: Relative incidence of hypertension and/or diabetes in haemoglobin genotypes

Group	Control male	Hypertensive non-diabetics male	Normotensive diabetics male	Hypertensive diabetics male	Control female	Hypertensive non-diabetics female	Normotensive diabetics female	Hypertensive diabetics female
HbAA	34 (68)	27 (54)	30 (60)	24 (48)	35 (70)	30 (60)	31 (62)	20 (40)
HbAS	11 (22)	21 (42)	15 (30)	23 (46)	14 (28)	16 (32)	12 (24)	25 (50)
HbAC	2 (4)	1 (2)	3 (6)	2 (4)	0 (0)	1 (2)	3 (6)	3 (6)
HbSS	1 (2)	1 (2)	1 (2)	1 (2)	1 (2)	2 (4)	3 (6)	0 (0)
HbSC	1 (2)	0 (0)	1 (2)	0 (0)	0 (0)	1 (2)	1 (2)	1 (2)
HBCC	1 (2)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	1 (2)
Total	50 (100)	50 (100)	50 (100)	50 (100)	50 (100)	50 (100)	50 (100)	50 (100)
Values are frequencies; Figures in parentheses are in percentage.								

Table 6 shows that the mean systolic or diastolic blood pressure did not differ significantly ($p > 0.05$) among ABO/RhD blood groups in any patient group except in ND male and HD male where a trend towards lower SBP in blood group AB compared with O, A and B. Also the DBP in HND female O blood group decreased significantly when compared with other blood groups. Meanwhile, a significant ($p < 0.05$) higher value of SBP in RhD negative was seen in control male (120.31±2.71 vs 110.49±1.32 mg/dL), HD male (187.94±5.49 vs 174.54±3.69 mg/dL) and HND female (181.67±6.44 vs 166.29±3.04mg/dL).

Table 6: ABO-Rh blood types and blood pressure of the subjects

SBP (mmHg)	Control male	Hypertensive non-diabetics male	Normotensive diabetics male	Hypertensive diabetics male	Control female	Hypertensive non-diabetics female	Normotensive diabetics female	Hypertensive diabetics female
O	118.88±1.76^{a}	161.34±6.33^{a}	117.65±1.79^{b}	165.75±4.97^{a}	114.15±1.63^{a}	160.46±3.58^{a}	120.57±5.92^{a}	166.22±4.87^{a}
A	115.90±1.62^{a}	182.30±5.64^{a}	113.70±1.88^{b}	183.48±4.41^{ab}	113.08±1.82^{a}	180.56±4.63^{a}	114.81±2.19^{a}	182.47±4.36^{b}
B	111.71±3.06^{a}	167.67±8.49^{a}	117.17±4.18^{b}	173.29±12.29^{ab}	111.50±3.12^{a}	166.26±7.49^{a}	116.67±3.78^{a}	164.43±8.42^{a}
AB	114.00±6.11^{a}	183.80±12.20^{a}	104.00±3.76^{a}	193.80±7.25^{b}	113.67±6.69^{a}	181.17±14.75^{a}	117.40±5.47^{a}	200.25±8.62^{b}
Rh.D+	110.49±1.32*	170.51±4.39	114.94±1.45	174.54±3.69*	113.79±1.30	166.29±3.04*	119.17±3.48	171.29±3.67
Rh.D-	120.31±2.71**	173.88±7.60	114.58±2.27	187.94±5.49**	112.29±1.91	181.67±6.44**	112.53±2.56	180.72±5.16
DBP (mmHg)								
O	77.53±1.13^{ab}	104.12±3.52^{a}	79.23±1.33^{a}	103.21±3.91^{a}	75.66±1.71^{a}	98.50±2.43^{a}	79.50±1.27^{a}	100.61±2.80^{a}
A	78.86±1.78^{ab}	116.00±3.03^{a}	78.37±1.16^{a}	114.30±2.77^{a}	76.08±2.04^{a}	113.59±2.94^{b}	78.59±1.19^{a}	114.75±2.77^{a}
B	71.43±5.21^{a}	105.33±5.13^{a}	78.33±2.08^{a}	106.43±7.97^{a}	77.12±2.25^{a}	104.63±4.21^{b}	78.67±2.63^{a}	99.71±5.61^{a}
AB	81.67±3.67^{b}	115.00±6.77^{a}	77.20±2.33^{a}	124.90±3.66^{a}	80.00±4.73^{a}	113.17±8.77^{b}	78.20±3.17^{a}	113.50±9.61^{a}
Rh.D+	77.56±0.94	108.64±2.53	78.31±0.85	109.08±2.61	76.05±1.34	103.39±1.96	79.00±0.93	105.30±2.33
Rh.D-	77.23±3.57	111.35±3.59	80.00±1.80	117.81±3.20	76.43±2.08	113.61±4.04	85.50±1.42	108.89±3.14

Values are mean±S.E.M. Values within the same column with different superscripts are significantly different at $p<0.05$

Table 7: ABO-Rh blood types and blood pressure component of the subjects

PP (mmHg)	Control male	Hypertensive non-diabetics male	Normotensive diabetics male	Hypertensive diabetics male	Control female	Hypertensive non-diabetics female	Normotensive diabetics female	Hypertensive diabetics female
O	41.35±1.77^{a}	57.22±3.32^{a}	38.42±1.707^{b}	62.54±2.36^{a}	38.49±1.44^{a}	61.96±2.09^{a}	41.07±5.82^{a}	65.61±3.51^{a}
A	37.05±1.68^{a}	66.30±2.99^{a}	35.33±1.41^{b}	69.19±2.18^{b}	37.00±1.54^{a}	66.97±2.53^{a}	36.22±1.51^{a}	67.72±2.80^{a}
B	40.29±6.59^{a}	62.33±4.43^{a}	38.83±3.10^{b}	66.86±5.02^{a}	34.38±2.42^{a}	61.63±4.94^{a}	38.00±2.86^{a}	64.71±3.48^{a}
AB	32.33±2.60^{a}	68.80±5.62^{a}	26.80±4.68^{a}	68.90±4.42^{a}	33.67±2.33^{a}	68.00±6.39^{a}	39.20±3.93^{a}	86.75±7.44^{a}
Rh.D+	38.93±1.30	61.86±2.22	36.63±1.27	65.46±1.74*	37.74±1.10	62.91±1.80	40.17±3.34	65.99±2.42
Rh.D-	43.08±4.21	62.53±4.27	34.58±1.62	70.12±2.58**	35.86±1.91	68.06±2.58	33.67±1.95	71.83±3.51
MAP (mmHg)								
O	91.32±1.09^{a}	123.20±4.38^{a}	92.04±1.26^{a}	124.06±4.14^{a}	88.49±1.54^{a}	119.15±2.69^{a}	93.19±2.28^{a}	122.48±3.23^{a}
A	91.21±1.54^{a}	138.10±3.84^{a}	90.15±1.28^{a}	137.36±3.25^{ab}	88.42±1.83^{a}	135.92±3.39^{a}	90.67±1.43^{b}	137.32±3.12^{a}
B	84.86±3.40^{a}	126.11±6.10^{a}	91.28±2.56^{a}	128.71±9.33^{a}	88.58±2.31^{a}	125.18±5.01^{a}	91.33±2.75^{ab}	121.29±8.19^{a}
AB	92.44±4.46^{a}	137.93±8.55^{a}	86.13±1.86^{a}	147.87±4.71^{b}	91.22±5.35^{a}	135.83±10.71^{a}	91.27±3.64^{b}	142.42±8.60^{a}
Rh.D+	90.54±0.89	129.27±3.10	90.52±0.90	130.90±2.90	88.63±1.22*	124.36±2.22*	92.39±1.47	127.30±2.61
Rh.D-	91.59±2.65	132.20±4.88	91.53±1.81	141.19±3.92	96.67±1.82*	136.30±4.82**	90.09±1.64	132.83±3.56
Heart Rate (beats/minutes)								
O	83.33±1.45^{a}	125.47±1.84^{a}	84.53±1.59^{a}	124.58±3.80^{a}	85.12±1.29^{a}	119.00±2.54^{a}	84.11±1.43^{a}	122.06±3.23^{a}
A	87.57±1.24^{a}	123.83±2.39^{a}	82.54±1.40^{a}	122.48±2.29^{a}	83.44±1.67^{a}	123.48±2.49^{a}	83.64±1.35^{a}	122.92±2.51^{a}
B	84.00±1.32^{a}	118.75±2.96^{a}	80.50±2.06^{a}	125.75±5.89^{a}	82.43±2.13^{a}	128.00±3.62^{a}	86.50±2.84^{a}	127.00±6.75^{a}
AB	83.33±3.28^{a}	127.00±5.23^{a}	79.33±0.88^{a}	132.00±3.69^{a}	84.00±2.00^{a}	130.25±1.32^{a}	84.00±2.52^{a}	124.67±8.35^{a}
Rh.D+	84.56±1.02	124.30±1.64	82.11±1.05	124.77±2.08	84.80±1.05	122.83±1.80	84.29±1.02	125.50±2.16
Rh.D-	84.78±2.18	122.23±2.66	85.25±1.92	125.40±3.00	81.50±0.82	124.73±3.20	83.53±1.74	116.71±2.72

Values are mean±S.E.M. Values within the same column with different superscripts are significantly different at $p<0.05$

Table 7 depicts the ABO-Rh blood types and blood pressure component [pulse pressure (PP), mean arterial pressure (MAP) and heart rate] in the controls and patients. Resting heart rate did not differ ($p > 0.05$) among ABO-RhD blood groups for patients and controls in both sexes. While PP in ND male AB blood group decreased significantly, the PP in A blood group increased significantly when compared with other blood groups. Meanwhile, a significant ($p < 0.05$) higher value of PP in RhD negative was observed in HD male (70.12±2.58 vs 65.46±1.74 mg/dL), also a significant ($p < 0.05$) higher value of MAP in RhD negative was observed in control female (96.67±1.82 vs. 88.63±1.22) and HND female (136.30±4.82 vs. 124.36±2.22 mg/dL).

DISCUSSION

Associations of blood groups with several diseases including immunological disorders are considered to be markers of impending health hazards. Data on association between distribution of ABO blood types and diseases are conflicting, some studies reported no association and others showed positive association. A well established claim for a relationship between blood groups and disease was that between group A and carcinoma of stomach. It had been reported that the risks for developing carcinoma of stomach was in the ratio 1.2:1 for the blood group A subjects compared with subject having blood group O or B. The most prevalent view is that A-phenotype has greater susceptibility for cardiovascular diseases, atherosclerotic peripheral vascular disease and several other types of cardiovascular diseases than non A-phenotypes, particularly the O-phenotypes (Khan *et al*., 2010). According to Jorgenson 2009, group O is more susceptible to carcinoma of stomach while pernicious anaemia occurs more frequently in group A subjects than group O. Group A is more susceptible to duodenal ulceration as well. Similarly, blood groups A, B, and O are equally more susceptible to malaria infection, while blood group AB individuals are less susceptible (Singh *et al*., 2007). Comparing Plasmodium falciparum and Plasmodium vivax, it was found that significantly lower frequency of Plasmodium falciparum was observed among individuals with blood groups A and O, while, in groups B and AB, no difference in Plasmodium vivax and falciparum was observed (Singh *et al*., 2007; Ojo and Mafiana, 2007). A study from Australia showed a very high incidence of coronary heart disease in blood group A subjects, while in Britain and Pakistan relationship between ischemic heart disease and blood group A has also been established (Khan *et al*., 2010). A study carried out in India suggested that there was no association between the ABO blood groups and DM type 2 (Khurshid *et al*., 1992). There are many studies which showed a significant excess of blood group A among male diabetics, such as a combined series from Lancashire, Cheshire, and Oxford (Muhammad *et al*., 2010). In addition, there were reports from Italy and Trinidad (Jorgenson, 2009) showing an increased frequency of blood group B among diabetics, but in Germany (Singh *et al*., 2007) and in Glasgow (Apostolopoulos *et al*., 2002) it was concluded that there was no significant ($p > 0.05$) association between ABO blood groups and diabetes.

As obvious from the above discussion, certain populations do show a positive association with ABO blood groups. The results in this present research O and A blood groups showed positive or direct association with HND, ND and HD in both male and female patients when compared with their control counterparts. Prevalence of hypertension and/or T2DM was observed in subjects with blood O followed by A. This implied that O followed by A blood group patients have more chances of HND, ND and HD. However, no significant association was found between patients and blood group B and AB. The phenotype frequencies of ABO blood group in both hypertensive and diabetic patients and controls (both sexes) are in the order O>A>B>AB. The frequencies of RhD blood groups were similar in patients and their control counterparts.

Out of total 620 subjects, haemoglobin genotypes were determined from the 400 subjects [50 subject each from hypertensive non-diabetics male (HNDM), hypertensive non-diabetics female (HNDF), normotensive diabetic male (NDM), normotensive diabetic female (NDF), hypertensive diabetics male (HDM) and hypertensive diabetics female (HDF)] which is the first aspect of the research work. The percentages of the various haemoglobin genotypes obtained in this study showed that there was significant ($p < 0.05$) difference in the distribution of haemoglobin electrophoresis among the patients and the controls. The spectrum of haemoglobin electrophoresis among the controls and patients can be shown with a general formula HbAA>HbAS>HbAC>HbSS>HbSC>HbCC except in control female and HD female. The importance of the knowledge of the blood groups and genotypes in regards to the health of an individual is enormous. The different types of information are useful for medical diagnosis, genetic information, genetic counseling and also for the general wellbeing of individuals (Adeyemo and Soboyejo, 2006). In conclusion, there was a strong relationship between blood group O and hypertension and/or T2DM, and the association was positive as this group was more common in hypertension and/or T2DM patients (both sexes). Large studies in other ethnic groups are needed to confirm these results.

ACKNOWLEDGEMENTS

We appreciate the efforts of technical staff of Biochemistry department FUNAAB, Mrs. J. O. Adebawa, Mr. O. J. Olurinde, Mr. O. A. Awoyemi and Mrs. T. O. Adeleye. We also acknowledge the roles of Mr. Oloyede – a medical laboratory scientist at the State Hospital, Abeokuta and his team, who assisted in the blood collections during the period of this project. Finally our thanks goes to all participating Medical Doctors and the patients as well as the control subjects who gave us all necessary support in the course of this study.

REFERENCES

Ademuyiwa, O., Regina N. Ugbaja and Solomon O. Rotimi. (2008). Plasma lipid profile, atherogenic and coronary risk indices in some residents of Abeokuta in south-western Nigeria. Biokemistri 20(2):85-91.

Adeyemo, O. A. and Soboyejo, O. B. (2006). Frequency distribution of ABO, RH blood groups and blood genotypes among the cell biology and genetics students of University of Lagos, Nig. Afr. J. Biotechnol. 5 (22):2062-2065.

American Diabetes Association. (2004). The diagnosis and classification of diabetes mellitus. Diabetes Care 27: s5 - 10.

Apostolopoulos, K., Labropoulou, E. Konstantinos, B., Rhageed, S. and Ferekidis, E. (2002). Blood group in otitis media with effusion. J. Otorhinolar. Relat. Spec. 64:433–435.

Carretero, O. A. and Oparil, S. (2000). Essential hypertension. Part I: definition and etiology. Circ. 101 (3):329–335.

Daniels G. (2002). Human blood groups. 2nd ed. Oxford, UK: Blackwell Scientific.

Idogun, E. S., Unuigbe, E. P., Ogunro, P. S., Akinola, O. I. and Famodu, A. A. (2007). Assessment of serum lipids in Nigerians with type 2 diabetes mellitus complications. Pak. J. Med. Sci. 23:708-712.

International Diabetes Federation. (2013). IDF Diabetes Atlas, 6th edn. Brussels, Belgium. Pp 1 – 160.

Jorgenson, G. (2009). The ABO blood group polymorphism in the multifactorial genetic system. In: Human Genetics. 3rd Edition. New York: McGraw Hill. 967.p. 264.

Khan, M. S., Ahmed, Z., Hanif, R., Zaman, S., Ali, I., Rahman, J. U. and Zaman, F. (2010). Relationship between blood groups and male infertility. J. Ayub. Med. Coll. Abbo. 22 (1):154-156.

Khurshid, B., Naz, M., Hassan, M. and Mabood, S. F. (1992). Frequency of ABO and Rh (D) blood groups in district Swabi N.W.F.P (Pakistan). J. Sc. Tech. Uni. Pesha. 16:5–6.

Muhammad, K.., Hamid, A. N. and Narazah M. Y. (2010). Association of ABO blood groups with diabetes mellitus. Libyan J Med. 5:1-4.

Napoli, M. and Papa, F. (2003). Association between blood pressure and C-reactive protein levels in acute ischemic stroke. Hypertension. 42:1117–1123.

O'Donnell, J. and Laffan, M. (2001). The relationship between ABO histoblood group, factor VIII and von Willebrand factor. Transfus Med. 11: 343 - 351.

Ojo, D. A. and Mafiana, C. F. (2007). Prevalence of malaria and typhoid infections in endermic community of Ogun State, Nigeria. Bio. and Environ. Sc. J. Trop. 4(2): 185-192.

Oladapo, O. O., Salako, L., Sodiq, O., Shoyinka, K., Adedapo, K. and Falase A. O. (2010). A prevalence of cardiometabolic risk factors among a rural Yoruba south-western Nigerian population: a population-based survey. Cardiovasc. J. Afr. 21:26-31.

Pierdomenico, S. D., Di, N. M. and Esposito, A. L. (2009). Prognostic Value of Different Indices of Blood Pressure Variability in Hypertensive Patients. Ameri. J. Hypert. 22 (8):842–847.

Singh, N, Shukla, M. M., Uniyal, V. P., Sharma, V. P. (2007). ABO blood groups among malaria cases from district Mandla, Madya Pradesh. Indian. J. Malariol. 32 (2):59–63.

Tietz, N.W. (1995). Clinical Guide to Laboratory Test 3rd edition, Philadelphia, Pa: WB Saunders Company, 518-522.