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Serum lipid profile and lipid pro-atherogenic indices of a cohort of Nigerian adults with varying glycemic and blood pressure phenotypes

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ABSTRACT

Dyslipidemia is common in patients with Type 2 diabetes and those with hypertension. The lipid profile and lipid pro-atherogenic indices of subjects with varying levels of fasting blood glucose and blood pressures may be different and thus was studied. Standard clinical and anthropometric methods were used to determine/measure all necessary parameters. Data from 189 subjects (90 males), aged 22-84 years, who met the inclusion criteria were analyzed. Five glycemic/blood pressure phenotypes were defined, namely: hypoglycemic and normotensive (HN), hypoglycemic and hypertensive (HH), normoglycemic and hypertensive (NH), diabetic and normotensive (DN), and diabetic and hypertensive (DH) and their data compared to the control (normoglycemic and normotensive) group. Hypertensive subjects, irrespective of their fasting blood glucose levels had the worst lipid profile and had higher serum lipid pro-atherogenic indices compared to normotensive subjects. These lipid abnormalities were more prevalent within the older (≥45 years old) and the overweight/obese subjects irrespective of sex. The lipid profile of hypertensive subjects should be monitored regularly and adjusted medically if necessary, irrespective of their fasting blood glucose level. Management of diabetes and hypertension (especially in this environment) should apart from targeting lipid abnormalities, emphasize therapeutic lifestyle changes that encourage weight loss.

Keywords: Diabetes mellitus, dyslipidemia, hypertension, serum lipid profile.

INTRODUCTION

Dyslipidemia is common in patients with Type 2 diabetes. It is believed to be responsible for considerable cardiovascular disease (CVD) – related morbidity and mortality (Krentz, 2003). Diabetic patients exhibit some characteristic changes in plasma lipoproteins. These changes, which influence the proximate determinants of atherosclerosis, include elevated triglycerides and reduced high-density lipoprotein cholesterol levels, with total cholesterol often remaining at or close to the normal range (Derosa et al., 2004). The incidence of Type 2 diabetes might be associated with preexisting hypertension (Izzo et al., 2009). Both dyslipidemia and hypertension are important risk factors for CVD, and studies have found that there is a higher risk of dyslipidemia among individuals with hypertension (Zhang et al., 2009). In fact, the incidence of hypertension in the diabetic population is 1.5–

3 times higher than in non-diabetic age-matched individuals (Arauz-Pacheco et al., 2002). Furthermore, the risk of any cardiovascular event or stroke is almost doubled when the hypertensive patient has diabetes mellitus (Belletti et al., 2010). There is evidence that these chronic diseases are increasing in prevalence in the developing countries as against the more industrialized nations where they are reducing (Gaziani, 2005). This therefore warrants more studies on these conditions, especially in Africa where data is often scanty. It is plausible that those with hypertension or diabetes (independently), or complications of both may have varying serum lipid profiles. The serum lipid profile of diabetic and hypertensive subjects has been studied both in Nigeria and elsewhere (Zwirska-Korczala et al., 2002; Alam et al., 2003; Danbauchi et al., 2005; Martins et al., 2008; Okafor et al., 2008; Dell'Omo et al., 2009; Zhang et al., 2009). However, the available studies did not have a detailed grouping of the subjects based on their glycemic and blood pressure phenotypes, thereby beclouding objective deductions from their data. The present study attempts to fill that vacuum and the results are expected to be useful in the management of diabetes and hypertension complications, especially in Nigeria.

MATERIALS AND METHODS

Subjects

A total of 199 subjects (95 males and 104 females) aged 22-84 years and living in Umuahia, Nigeria participated in the study. Participants were randomly selected and the study explained to them individually. Informed verbal consent was obtained from each participant. Exclusion criteria included pregnancy and hormonal contraceptive use (in women), overt morbidity from any disease including morbid obesity (BMI $\geq$ 40), the taking of anti-hypertensive or anti-diabetic medication, and having fasting blood glucose level that is higher than normal but less than the diabetic level. Data from 189 subjects (52.4% females) were eventually included in the analysis. The study was designed in accordance with the Helsinki Declaration and was approved by the Ethics Committee of the Federal Medical Centre Umuahia and the board of the Department of Biochemistry, Michael Okpara University of Agriculture, Umudike, both in Abia State, Nigeria.

Instruments and measures

Blood pressure was measured on a single visit, using a standard mercury sphygmomanometer and appropriate cuff sizes, with the subject in a seated position, and having rested for at least 10 minutes. Three separate readings were taken per subject, after two minutes intervals and the average of the second and third readings recorded. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were taken at the 1st and 5th Korotkoff sounds respectively. The same trained personnel took all blood pressure measurements.

Weights and heights of participants were taken, with participants dressed in light clothing, and BMI calculated with the formula: BMI = [weight (kg)/height^2 (m^2)].

Self-reported age at last birthday was recorded per participant. Based on their ages, and taking into consideration that age ($\geq$45 years for men, and $\geq$55 years for women) is one of the risk factors for coronary artery disease (CAD) listed by NCEP (2001), we grouped subjects into age ranges as follows: 22-34 years, 35-44 years, 45-54 years, 55-64 years and $\geq$65 years.

Fasting blood samples (4ml) was drawn from each participant, allowed to stand at ambient temperature until clotting took place, and the serum separated by centrifugation for 5 minutes at 1000 g. From a drop of whole blood (before separation), blood glucose concentration was measured by the glucose oxidase method (Washako and Rice, 1961). From the serum, total cholesterol, HDL-Cholesterol and triglycerides were measured by enzymatic colorimetric methods (Allain et al., 1974; Lopes-Virella et al., 1977; Tietz, 1990). LDL-Cholesterol was determined by difference (Friedwald et al., 1972). VLDL-

Cholesterol was calculated using the formula: VLDL = [Triglycerides (mg/dl)]/5. Non HDL-Cholesterol was calculated as the difference between total cholesterol and HDL-cholesterol (Contreras et al., 2010). Serum lipid pro-atherogenic indices were calculated thus: Total cholesterol (TChol) to HDL-Cholesterol ratio = TChol/HDL; Non HDL-Cholesterol to HDL-Cholesterol ratio = Non HDL/HDL; and LDL-Cholesterol to HDL-Cholesterol ratio = LDL/HDL.

Definitions

We defined hypertension as a systolic blood pressure that is 140 mmHg or more and/or a diastolic blood pressure that is 90 mmHg or more (WHO, 1999), and normotension as SBP/DBP < 140/90. Based on subjects' fasting blood glucose level, they were classified as hypoglycemic (FBG < 75 mg/dl), normoglycemic (75-115 mg/dl) and diabetic (> 125 mg/dl) (Kratz et al., 2004). We then defined 5 glycemic and blood pressure phenotypes. Subjects who were normotensive and normoglycemic served as control subjects. Those who were hypoglycemic and normotensive were grouped as HN; those who were hypoglycemic and hypertensive were grouped as HH; those who were normoglycemic and hypertensive were grouped as NH; those who were diabetic and normotensive were grouped as DN; while those who were diabetic and hypertensive were grouped as DH. Lipid and lipoprotein level were described as optimal/normal, borderline, high or low as the case may be, using the Kratz et al. (2004) reference values. TChol/HDL, Non HDL/HDL, LDL/HDL ratios were taken as critical if the values were ≥ 5, 4, and 4 respectively.

Statistical analyses

Descriptive statistics on all the data generated was done (sex-wise and glycemic/blood pressure phenotype-wise) and reported as means ± standard deviations. Differences between means of the different groups compared to the control were separated by ANOVA post hoc tests with the least significant difference fixed at 0.05. We calculated the prevalence of the different disorders as the number of such cases divided by the number of subjects in that category, and the answer multiplied by 100. Descriptive statistics and group comparisons were done using SPSS for windows version 11.0 (SPSS Inc Chicago IL).

RESULTS

The age, BMI and lipid profile of the population are displayed in Table 1. BMI did not vary so much between the control group on the one hand, and the other groups on the other hand (Table 1). Only hypoglycemic and hypertensive (HH) females had critical mean serum lipid pro-atherogenic indices. In fact, they had critical mean values in the 3 indices studied (Table 2). Many of the normoglycemic and hypertensive (NH), diabetic and normotensive (DN) and diabetic and hypertensive (DH) subjects had an unfavorable lipid profile. Majority of them had higher total cholesterol levels and lower HDL-cholesterol level than is desirable (Table 3). The majority of subjects who had diabetes and hypertension complications were in the older age ranges as against the control group were the majority were in the younger age ranges (Tables 4 and 5).

DISCUSSION

Impaired insulin action [which usually results from derangements in carbohydrate (glucose) metabolism] leads to increased rates of intracellular hydrolysis of triglycerides with the release of non-esterified fatty acids (NEFA). The rise in NEFA provides substrate for the liver and, in the presence of impaired insulin action and relative insulin deficiency, is associated with complex alterations in plasma lipids, described as dyslipidemia (Zwirska-Korczala et al., 2002). In a typical dyslipidemic state, plasma VLDL-cholesterol, triglycerides and LDL-cholesterol levels are raised while HDL-cholesterol levels drop (Krentz, 2003).

Table 1: Age, BMI and lipid profile of the population, stratified according to glycemic and blood pressure phenotype.

	Control	**HN**	**p**	**HH**	**p**	**NH**	**p**	**DN**	**p**	**DH**	**P**
Age (years)											
Total	45.4 ± 12.0	*39.3 ± 3.6	0.012	*57.6 ± 15.2	0.012	*59.1 ± 13.0	<0.001	*52.3 ± 10.9	0.005	*58.3 ± 8.3	<0.001
Male	44.0 ± 10.7	45.4 ± 13.1	0.706	57.0 ± 18.1	0.073	*61.3 ± 11.3	0.001	*54.5 ± 11.7	0.003	*58.5 ± 8.4	<0.001
Female	46.5 ± 12.9	*35.5 ± 12.8	0.001	58.0 ± 15.6	0.066	*57.1 ± 14.8	0.023	50.2 ± 9.8	0.262	*57.9 ± 8.6	0.015
BMI (kg/m^2)											
Total	23.3 ± 3.6	23.7 ± 4.5	0.616	25.4 ± 2.1	0.194	*27.3 ± 5.4	0.001	24.5 ± 3.6	0.130	*27.1 ± 4.0	<0.001
Male	23.8 ± 3.2	22.8 ± 3.9	0.450	25.3 ± 0.7	0.560	27.2 ± 5.9	0.053	24.7 ± 3.9	0.471	*27.8 ± 3.5	0.002
Female	22.9 ± 4.0	24.3 ± 4.8	0.210	25.5 ± 2.9	0.229	*27.3 ± 5.3	0.006	24.4 ± 3.4	0.190	25.5 ± 4.9	0.101
Total Cholesterol (mg/dl)											
Total	222.7 ± 88.5	*170.3 ± 61.9	0.001	206.5 ± 59.8	0.613	246.6 ± 104.2	0.302	223.2 ± 59.8	0.976	217.0 ± 83.4	0.088
Male	224.2 ± 90.1	187.4 ± 69.1	0.155	185.0 ± 45.0	0.424	215.2 ± 85.2	0.794	228.5 ± 59.6	0.856	245.0 ± 103.7	0.397
Female	221.5 ± 88.6	*159.7 ± 55.9	0.004	222.6 ± 70.7	0.980	274.2 ± 116.9	0.098	217.8 ± 60.8	0.869	277.4 ± 87.3	0.079
LDL-Cholesterol (mg/dl)											
Total	115.5 ± 77.1	*83.0 ± 60.7	0.040	94.7 ± 67.9	0.501	116.0 ± 90.6	0.984	95.4 ± 66.8	0.202	132.0 ± 104.1	0.363
Male	111.9 ± 80.8	91.6 ± 65.6	0.422	66.3 ± 55.0	0.341	112.0 ± 67.4	0.998	91.5 ± 67.1	0.383	127.1 ± 112.6	0.526
Female	118.3 ± 75.3	77.7 ± 58.2	0.500	116.1 ± 76.0	0.957	119.5 ± 111.8	0.969	99.3 ± 67.9	0.388	86.0 ± 30.4	0.411
Triglycerides (mg/dl)											
Total	167.6 ± 66.6	150.1 ± 82.0	0.244	195.3 ± 57.5	0.348	190.1 ± 80.9	0.293	154.1 ± 74.8	0.369	151.7 ± 69.8	0.355
Male	173.6 ± 58.8	163.8 ± 71.9	0.673	177.6 ± 56.9	0.928	150.9 ± 88.3	0.464	172.1 ± 71.1	0.947	*129.5 ± 61.6	0.047
Female	163.2 ± 72.5	141.7 ± 87.9	0.259	208.6 ± 62.4	0.235	*224.4 ± 59.2	0.032	136.2 ± 75.6	0.182	204.3 ± 61.9	0.149
HDL-Cholesterol (mg/dl)											
Total	74.5 ± 25.0	*58.3 ± 25.1	0.005	72.3 ± 44.0	0.841	81.6 ± 31.4	0.383	*96.1 ± 30.8	<0.001	*94.1 ± 30.8	0.003
Male	78.6 ± 23.4	63.0 ± 21.1	0.086	83.0 ± 41.7	0.797	72.0 ± 32.0	0.587	*102.6 ± 24.2	0.005	94.4 ± 35.5	0.066
Female	71.5 ± 26.1	*55.4 ± 27.2	0.031	64.2 ± 50.2	0.626	*90.0 ± 30.4	0.045	*91.3 ± 31.1	0.013	*93.4 ± 16.7	0.049
VLDL-Cholesterol (mg/dl)											
Total	33.5 ± 13.3	30.0 ± 16.4	0.244	39.1 ± 14.5	0.348	38.0 ± 16.2	0.293	30.8 ± 15.0	0.369	30.3 ± 14.0	0.355
Male	34.7 ± 11.8	32.8 ± 14.9	0.673	35.5 ± 11.4	0.928	30.2 ± 17.7	0.464	34.4 ± 14.2	0.947	*25.9 ± 12.3	0.047
Female	32.6 ± 14.5	28.3 ± 17.6	0.259	41.7 ± 12.5	0.235	*44.9 ± 11.8	0.032	27.2 ± 15.1	0.182	40.9 ± 12.4	0.149
Non HDL-Cholesterol											
Total	147.84±80.58	*111.93±63.56	0.035	134.22±77.65	0.681	165.02±99.11	0.475	126.25±73.49	0.202	160.49±112.88	0.514
Male	145.58±85.18	124.33±68.01	0.430	101.98±63.82	0.394	143.15±70.83	0.946	125.93±75.78	0.431	150.57±121.21	0.846
Female	149.53±78.30	*104.30±60.76	0.041	158.40±86.74	0.841	184.17±120.19	0.294	126.57±13.00	0.237	184.04±92.97	0.296

Control subjects are those who are normoglycemic and normotensive. HN, HH, NH, DN and DH represent Hypoglycemic Normotensive, Hypoglycemic Hypertensive, Normoglycemic Hypertensive, Diabetic Normotensive and Diabetic Hypertensive respectively. p values < 0.05 are significant. * indicates values that differ significantly from the control.

Table 2: Serum lipid pro-atherogenic indices, stratified according to glycemic and blood pressure phenotype.

	Control	**HN**	**p**	**HH**	**p**	**NH**	**p**	**DN**	**p**	**DH**	**P**
Total Cholesterol to HDL-Cholesterol ratio											
Total	3.16 ± 1.19	3.54 ± 2.31	0.344	*5.10 ± 5.13	0.015	3.35 ± 1.47	0.736	2.61 ± 1.32	0.176	3.28 ± 2.18	0.797
Male	3.00 ± 1.17	3.21 ± 1.56	0.743	2.61 ± 1.16	0.742	3.36 ± 1.31	0.670	2.43 ± 1.04	0.327	3.36 ± 2.53	0.550
Female	3.27 ± 1.21	3.74 ± 2.67	0.363	*6.97 ± 7.35	<0.001	3.34 ± 1.68	0.928	2.79 ± 1.55	0.379	3.07 ± 1.07	0.794
Non-HDL-Cholesterol to HDL-Cholesterol ratio											
Total	2.16 ± 1.19	*2.54 ± 2.31	0.035	4.10 ± 5.73	0.681	2.35 ± 1.47	0.475	1.61 ± 1.32	0.202	2.28 ± 2.18	0.514
Male	2.00 ± 1.17	2.21 ± 1.56	0.743	1.61 ± 1.16	0.742	2.36 ± 1.32	0.670	1.43 ± 1.04	0.327	2.36 ± 2.53	0.550
Female	2.27 ± 1.21	2.74 ± 2.67	0.363	*5.97 ± 7.35	<0.001	2.34 ± 1.68	0.928	1.79 ± 1.55	0.379	2.07 ± 1.07	0.794
LDL-Cholesterol to HDL-Cholesterol ratio											
Total	1.68 ± 1.06	1.88 ± 1.86	0.056	*3.06 ± 4.42	0.042	1.71 ± 1.42	0.938	1.26 ± 1.21	0.221	1.90 ± 2.01	0.567
Male	1.56 ± 1.08	1.65 ± 1.46	0.868	1.10 ± 0.92	0.651	1.88 ± 1.37	0.657	1.06 ± 0.89	0.314	2.02 ± 2.33	0.368
Female	1.76 ± 1.04	2.02 ± 2.09	0.563	*4.52 ± 5.64	0.002	1.57 ± 1.54	0.771	1.46 ± 1.46	0.516	1.61 ± 0.96	0.818

Control subjects are those who are normoglycemic and normotensive. HN, HH, NH, DN and DH represent Hypoglycemic Normotensive, Hypoglycemic Hypertensive, Normoglycemic Hypertensive, Diabetic Normotensive and Diabetic Hypertensive respectively. p values < 0.05 are significant. * indicates values that differ significantly from the control.

Table 3: Prevalence of glycemic and blood pressure phenotypes, stratified by Kratz et al. (2004) reference values.

	Control (%) (n = 56)	HN (%) (n = 42)	HH (%) (n = 7)	NH (%) (n = 15)	DN (%) (n = 42)	DH (%) (n = 27)
Total Cholesterol						
Desirable <200 mg/dl	44.6	71.4	57.1	53.3	35.7	37.0
Borderline 200-239 mg/dl	17.9	16.7	28.6	0.0	26.2	14.8
High ≥ 240	37.5	11.9	14.3	46.7	38.1	48.1
LDL-Cholesterol						
Optimum/Near optimum <100 – 129 mg/dl	67.9	83.3	71.4	60.0	66.7	63.0
Borderline 130-159 mg/dl	10.7	7.1	14.3	26.7	7.1	7.4
High/Very High ≥160 mg/dl	21.4	9.5	14.3	13.3	26.2	29.6
HDL-Cholesterol						
Low <40 mg/dl	7.1	23.8	28.6	13.3	4.8	7.4
High ≥60 mg/dl	71.4	38.1	57.7	80.0	92.9	85.2
Triglycerides						
Desirable <160 mg/dl	48.2	50.0	42.9	26.7	54.8	55.6
High ≥ 160 mg/dl	51.8	50.0	57.1	73.3	45.2	44.4
VLDL-Cholesterol						
Desirable <32 mg/dl	48.2	50.0	42.9	26.7	54.8	55.6
High ≥ mg/dl	51.8	50.0	57.1	73.3	45.2	44.4

Control subjects are those who are normoglycemic and normotensive. HN, HH, NH, DN and DH represent Hypoglycemic Normotensive, Hypoglycemic Hypertensive, Normoglycemic Hypertensive, Diabetic Normotensive and Diabetic Hypertensive respectively.

Table 4: Prevalence of glycemic and blood pressure phenotypes, stratified by age-range.

	Control (%) (n = 56)	HN (%) (n = 42)	HH (%) (n = 7)	NH (%) (n = 15)	DN (%) (n = 42)	DH (%) (n = 27)
22-34 years	21.4	45.2	0.0	0.0	2.3	0.0
35-44 years	26.8	26.2	28.6	13.3	21.4	3.7
45-54 years	25.0	9.5	0.0	26.7	38.1	33.3
55-64 years	21.4	14.3	28.6	20.0	21.4	40.7
≥ 65 years	5.4	4.8	42.9	40.0	16.7	22.2
Total	29.6	22.2	3.7	7.9	22.2	14.3

Control subjects are those who are normoglycemic and normotensive. HN, HH, NH, DN and DH represent Hypoglycemic Normotensive, Hypoglycemic Hypertensive, Normoglycemic Hypertensive, Diabetic Normotensive and Diabetic Hypertensive respectively.

Table 5: Prevalence of glycemic and blood pressure phenotypes within the age-ranges.

	22-34 years (%) (n = 32)	35-44 years (%) (n = 40)	45-54 years (%) (n = 47)	55-64 years (%) (n = 43)	≥ 65 years (%) (n = 27)
Control	37.5	37.5	29.8	27.9	11.1
HN	59.4	27.5	8.5	14.0	7.4
HH	0.0	5.0	0.0	4.7	11.1
NH	0.0	5.0	8.5	7.0	22.2
DN	3.1	22.5	34.0	20.9	25.9
DH	0.0	2.5	19.1	25.6	22.2
Total	16.9	21.2	24.9	22.8	14.3

Control subjects are those who are normoglycemic and normotensive. HN, HH, NH, DN and DH represent Hypoglycemic Normotensive, Hypoglycemic Hypertensive, Normoglycemic Hypertensive, Diabetic Normotensive and Diabetic Hypertensive respectively.

The results of these are post-prandial hyperlipidemias due to elevated VLDL-cholesterol, thrombogenic alterations in the coagulation system due to hypertriglyceridemia, and a reduction of cholesterol efflux from the tissues and anti-atherogenic activities due to reduced HDL-cholesterol (Zhang et al., 2009). LDL-cholesterol particles also become small and dense, thereby becoming more atherogenic (since they are subject to oxidation and easily adhere to and invade arterial walls) (Krentz, 2003). It is these lipid disturbances that are postulated to be involved in the increased incidence of arterial hypertension and Type 2 diabetes mellitus (Zwirska-Korczala et al., 2002).

We found the highest prevalence of those with high total cholesterol among subjects with both diabetes and hypertension (DH) (48.1%) followed by those who were normoglycemic but hypertensive (NH) (46.7%) and then diabetic and normotensive (DN) subjects (38.1%). The prevalence of subjects with high/very high LDL-cholesterol was highest in the DH (29.6%) group, while the prevalence of subjects with low HDL-cholesterol was highest in the HH group (57.7%). High triglyceride and VLDL-cholesterol levels were most prevalent in the NH group (57.1%). These findings suggest that these lipid abnormalities were more associated with hypertension as DN (but not NH) subjects had prevalence values that were comparable to those of the control group. This is understandable as hypertension, even in the absence of diabetes mellitus causes insulin resistance which in turn results in elevated plasma lipids (Hsueh et al., 2010). Our finding that irrespective of sex, NH subjects had the worst mean lipid profile (having both hypercholesterolemia and hypertriglyceridemia) followed by the HH group (that had only hypertriglyceridemia), while DN subjects, and hypoglycemic and normotensive (HN) subjects had mean lipid profiles that were between desirable and borderline, also support the exacerbation of serum lipid abnormalities by hypertension. These data agree with earlier reports from Nigeria (Okafor et al., 2008; Idogun et al., 2007) and elsewhere (Akbar, 2001; Alam et al., 2003).

Though it is known that treatment of hypertension with β-blockers, as well as high thiazide diuretics exacerbates dyslipidemia in subjects with hypertension and diabetes (Idogun et al., 2007), we doubt if this could be the cause of the noticed dyslipidemia in this cohort since our subjects were not taking any prescription medicines at the time of sampling. The increase in lipid abnormalities may be due to the significantly higher BMI of the hypertensive group (excluding the HH group), relative to the control group. Normotensive subjects (males and females alike) had a normal BMI while their hypertensive counterparts were overweight. Visceral fat accumulation which usually accompanies overweight/obesity is known to be associated with hyperinsulinaemia and insulin resistance (Carr and Brunzell, 2004; Meigs et al., 2006; Ejike and Ezeanyika, 2008). Impaired insulin action leads to a cascade of events that culminates in dyslipidemia (Krentz, 2003). Since dyslipidemia involves (among others) a drop in the level of the cardio-protective/anti-atherogenic HDL-cholesterol and an increase in the level of small dense LDL-cholesterol that are known to be highly atherogenic, hypertension often ensues (Krentz, 2003; Wajchenberg, 2000). This may explain the deranged lipid profile within the hypertensive group.

Our data further shows that subjects with diabetes, hypertension or both were more in the ≥ 45 years age group. Only the HN group was significantly younger than the control group. The other groups were significantly older than the control group. Like BMI, age was significantly higher in hypertensive subjects (irrespective of glycemic phenotype) compared to the others. Age is a very important and well established risk factor for hypertension, diabetes and other chronic diseases (NCEP, 2001; Pockok et al., 2001; Mokdad et al., 2001). As men age,

physiological imbalances favor dyslipidemia and the attendant health consequences (Ejike and Ezeanyika, 2008). For women, menopause erodes the cardio-protective roles of estrogen due to their reduced circulating concentrations (Sakao et al., 2010). These may therefore explain the higher prevalence of the studied abnormalities in the older population.

It is important to note that a derangement in the serum lipid profile has serious implications in the predisposition of an individual to cardiovascular events. Epidemiological studies show that an increase in total cholesterol level by 1% increases the risk of a cardiovascular event by 2% (Law et al., 1994), whereas the greater the HDL-Cholesterol achieved in an individual, the greater the cardioprotective benefit the individual has (Devendra et al., 2010). Additional greater cardiovascular risk is known to be related to the increase of plasma lipid pro-atherogenic indices. For example, a value ≥ 5 for TC/HDL ratio is related to four times greater risk of a cardiovascular event in patients with a triglyceride level below 200 mg/dl, and increased seven times when the triglyceride level is above 200 mg/dl (Assmann and Schulte, 1992). This clearly implies that the risk of cardiovascular events in our sample could be high, especially among the hypoglycemic and hypertensive female subjects.

The strengths of our study lie in its cross sectional nature, suggesting that subjects studied were not hospitalized or visiting a healthcare facility. This has the advantage of making the findings easy to extrapolate for the general population. Inherent in this however, is a possible under-estimation of the lipid profile of subjects with diabetes or hypertension or both. Obviously, if the disorders progressed to clinical stages, these factors would be expected to be altered more markedly. Our sample size is also not very large, but should suffice in an under-studied population that (for cultural reasons) resists cooperation with researchers who require their blood. Measurement of blood pressure and fasting blood glucose level on a single visit is clearly not a sure way of determining if a person is hypertensive or diabetic. This is a limitation of this study (and cross-sectional studies in general) and has, implicit in it, the potential for over-estimating disease presence in the community. These therefore warrant a cautious interpretation of the data presented in this report.

Conclusion

We found that subjects with hypertension (diabetic or non-diabetic, but not hypoglycemic) had the worst lipid profile compared to the control group. They were also older and heavier (BMI-wise) than the others. Female subjects who were hypoglycemic and hypertensive had the highest serum lipid pro-atherogenic indices. Hypertensive subjects, irrespective of fasting blood glucose level should have their lipid profile monitored regularly and adjusted medically if necessary. Management of diabetes and hypertension (especially in this environment) should apart from targeting lipid abnormalities, emphasize therapeutic lifestyle changes that encourage weight loss.

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