COMPARATIVE ANALYSIS OF LIPID PROFILES AMONG PATIENTS WITH TYPE 2 DIABETES MELLITUS, HYPERTENSION AND CONCURRENT TYPE 2 DIABETES, AND HYPERTENSION: A VIEW OF METABOLIC SYNDROME

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Type 2 diabetes mellitus and hypertension are independent risk factors for atherosclerotic lesions that are partly linked with dyslipidaemia. This risk is additive when diabetes and hypertension occur concurrently.

In order to determine if concurrent type 2 diabetes and hypertension results in putative increases in dyslipidaemia in a Nigerian population, we compared the plasma lipid levels, atherogenic index and prevalence of dyslipidaemia among age and sex-matched indigenous Nigerians with type 2 diabetes, hypertension and concurrent diabetes and hypertension. Age and sex-matched healthy Nigerians that are free of diabetes and hypertension served as controls.

The patients as a whole were more likely to have dyslipidaemia than controls ($p < 0.05$). High-density lipoprotein cholesterol was similar among patients and controls. Mean total cholesterol, high-density lipoprotein cholesterol; low-density lipoprotein cholesterol and triglyceride levels, atherogenic index and prevalence of dyslipidaemia did not differ significantly among patients with hypertension, diabetes, and concurrent hypertension and diabetes ($p = 0.99$ for each parameter).

It is concluded that concurrent hypertension and type 2 diabetes does not result in a more severe dyslipidaemia than when either of the two conditions occurs in isolation. We attribute this to the common pathogenic link between hypertension, diabetes and dyslipidaemia in metabolic syndrome. Evidence, albeit indirect, of this syndrome among native Africans is, therefore, provided. (*J Natl Med Assoc.* 2003;95:328-334.)

Key words: hypertension ♦ diabetes mellitus ♦ dyslipidaemia ♦ metabolic syndrome

Clusters of multifaceted metabolic disorders including glucose intolerance, essential hypertension, central obesity, dyslipidaemia and insulin resistance coined metabolic syndrome have been described among Caucasians.[1,2] About 10 to 15% and 1 to 2% of Nigerians have hypertension and diabetes mellitus respectively.[3,4] Both conditions coexist frequently in this population, the prevalence of hypertension among diabetics being 20-40%.[5,6]

Several studies established a direct relationship between insulin resistance, enhanced sympathetic nervous activity, hypertension and type 2 diabetes.[1,7] Increased sympathetic nervous activ-

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ity, transmembrane cation transport and renal sodium reabsorption probably contribute to the genesis of hypertension in metabolic syndrome.[8] Insulin resistance causes deficiency of lipoprotein lipase resulting in altered lipid metabolism and dyslipidaemia.[9] Increased influx of free fatty acids in the liver and production of tumor necrosis factor alpha (TNF-α) may play important roles in the mechanism of insulin resistance-related obesity.[10] Recently, a defective gene (cd36), which encodes fatty acid translocase, was identified to underlie insulin resistance, defective fatty acid metabolism and hypertriglyceridaemia in spontaneously hypertensive rats.[11] This may be an important pathogenic mechanism in human metabolic syndrome.

Dyslipidaemia in type 2 diabetes and hypertension are both quantitative and qualitative.[12-14] Quantitative abnormalities include increased levels of total plasma cholesterol, triglyceride and low-density lipoprotein (LDL) cholesterol, and decreased level of high-density lipoprotein (HDL) cholesterol. Qualitative abnormalities include changes in the composition of LDL-cholesterol (small dense LDL-cholesterol, increased triglyceride content and increased electronegativity of LDL-cholesterol). These changes make LDL-cholesterol susceptible to oxidation and glycation, with consequential foam cell formation, endothelial dysfunction and atherosclerosis.[12,14,15]

Information on plasma lipid concentrations and prevalence of dyslipidaemia among patients with type 2 diabetes and/or hypertension is, therefore, important. Several reports have confirmed that diabetes and hypertension are independently associated with dyslipidaemia among Nigerians.[16-19] Data on lipid patterns among diabetic hypertensives is, however, scanty and limited to total plasma cholesterol.[20,21] There also is no documentation of metabolic syndrome in this population, probably because of lack of facilities for insulin measurement.

Given the association between type 2 diabetes and hypertension and dyslipidaemia,[13,14,16-19] the role of lipid abnormalities as risk factors for atherosclerotic complications of diabetes and hypertension[12-15] and the additive nature of these complications when both conditions occur concurrently,[22,23] it may be proposed that putative increases in plasma lipid concentrations would occur in diabetic hypertensives.

The contrary may, however, be true, given the established role of hyperinsulinaemia as a central link in the genesis of diabetes, hypertension, and dyslipidaemia in metabolic syndrome.[1,2,7-10]

The objective of this study is to compare the quantitative lipid abnormalities, atherogenic index and prevalence of dyslipidaemia among indigenous age and sex-matched Nigerians with type 2 diabetes mellitus, essential hypertension

Table 1. BASELINE DATA OF PATIENTS AND CONTROLS

Characteristics	Controls N=40	HBP N=40	DM N=40	HBP+DM N=40
Male:Female ratio	1 : 1.7	1 : 1.8	1 : 1.6	1 : 1.9
Age (Years)	47.9±11.4	49.1±12.0	50.8±13.2	52.2±13.5
BMI (Kg/m^2)	25.8±6.0	26.3±3.9	28.0±6.7	27.4±6.8
SBP (mmHg)	110.3±8.4	186.1±26.7	112.5±8.9	178.9±18.8
DBP (mmHg)	79.2±7.4	106.9±22.4	80.8±11.4	99.6±19.8
FBS (mmol/L)	4.1±0.2	4.3±0.3	11.1±3.1	10.3±3.2
Duration of HBP (Years)	----	6.0±3.2	----	5.8±2.2
Duration of DM (Years)	----	----	4.3±1.4	4.2±1.9

SBP: Systolic blood pressure
DBP: Diastolic blood pressure
FBS: Fasting blood sugar
HBP: High blood pressure (Hypertension)
DM: Diabetes mellitus

and concurrent diabetes, and hypertension with a view of providing evidence of metabolic syndrome, if any, in this population.

PATIENTS AND METHODS

Patients were made of age and sex-matched indigenous Nigerian normotensive type 2 diabetes mellitus patients; normoglycaemic hypertensives and patients with concurrent type 2 diabetes and hypertension (40 patients per group). They were recruited at the medical outpatients' clinic of Usmanu Danfodiyo University Teaching Hospital, Sokoto, Northwestern Nigeria. Age and sex-matched volunteers certified clinically and biochemically to be healthy; normotensive and normoglycaemic served as controls. They were made of hospital staff, blood donors and medical students.

The diagnosis of diabetes mellitus was based on the World Health Organisation criteria.[24] 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. Systolic blood pressure ≥ 140mmHg and/or diastolic blood pressure ≥ 90mmHg measured using standard procedures were required to make a diagnosis of hypertension.[25] Height and weight were measured to the nearest centimeters and grams respectively with the subjects lightly clothed and without shoes. Obesity was defined as body mass index > 30Kg/m^2. [26]

The duration of diagnosis of diabetes mellitus was 4.3 ± 1.4 years and 4.2 ± 1.9 years among the normotensive diabetics and diabetic hypertensives respectively. The duration of diagnosis of hypertension was 6.0 ± 3.2 and 5.8 ± 2.2 years among the normoglycaemic hypertensives and type 2 diabetic hypertensives, respectively.

Patients with lipid-altering diseases: nephrotic syndrome, hepato-biliary disease and hypothyroidism were excluded. Other exclusion criteria included frank proteinuria detected by multistix, alcohol consumption, cigarette smoking and use of lipid altering drugs, including lipid lowering drugs, contraceptive medications, diuretics and beta blockers.

Sample Collection and Analysis

After an overnight fasting for 12 to 14 hours, about 6 mls of blood was withdrawn into heparinized bottles of fluoride oxalate and centrifuged at 100 rpm for five minutes. The supernatant was separated into appropriate containers for analysis. Samples were analyzed within 72 hours of collection. Those that could not be analyzed on the same day of collection were stored at a temperature of 4^o C. Fasting blood glucose was measured by glucose oxidase test using aminophenazone as oxygen carrier.[27] Total plasma cholesterol was determined using ferric perchlorate methods.[28] High-density lipoprotein (HDL) cholesterol was determined after precipitation of LDL-cholesterol with phosphtungstate and magnesium.[29] Triglyceride was measured using the colorimetric enzymatic method.[30]

Low-density lipoprotein (LDL) cholesterol was calculated from the formula:

LDL-cholesterol = Total cholesterol – Triglyceride – HDL-cholesterol[31]

Atherogenic index (AI) was defined as the ratio of total plasma cholesterol:

HDL – cholesterol

Plasma cholesterol and triglyceride values were determined in milligram % and converted to millimol/liter by multiplying with a factor of 0.02586 and 0.01129, respectively. A pre-prepared laboratory standard for lipid analysis was used to ensure quality assurance of the specimen.

Definition of Dyslipidaemia

Dyslipidaemia was defined as below using the European Atherosclerosis Society[32] except hypertriglyceridaemia that was defined on the basis of the local value for Nigerians[33] because it differs significantly from the Europeans.

- Total cholesterol (TC) > 5.2mmol/L
- Low-density lipoprotein (LDL) cholesterol > 3.5mmol/L
- Triglyceride (TG) > 1.75mmol/L
- High-density lipoprotein (HDL) cholesterol < 0.9mmol/L
- Atherogenic index (AI) > 5.8

Statistical Analysis

Data entry and analysis were done using EPI-Info software. Means are presented as values ± standard deviation. Student's *t*-test and chi-square test were used to compare means and proportions between two groups respectively. Analysis of variance (ANOVA) and chi-square tests were utilized in comparing the means and proportions respectively between normoglycaemic hypertensives, normotensive type 2 diabetics, and type 2 diabetic hypertensives.

RESULTS

The baseline data of patients and controls are shown in Table 1. The controls, normoglycaemic hypertensives, normotensive diabetics and diabetic hypertensives were similar in age, body mass index, and male to female ratio. The duration of diagnosis of hypertension was 6.0 ± 3.2 years among the normoglycaemic hypertensives and 5.8 ± 2.2 years among type 2 diabetic hypertensives (t=0.3; p>0.05). The duration of diagnosis of diabetes was similar among the normotensive diabetics and diabetic hypertensives (4.3 ± 1.4 years versus 4.2 ± 1.9 years) (t=0.3; p>0.05). In the diabetic patients, fasting blood sugar was similar among the normotensive (11.1 ± 3.1mmol/L) and hypertensive groups (10.3 ± 3.2mmol/L) (t=1.12; p>0.05). Blood pressure also was similar among the normoglycaemic hypertensives and diabetic hypertensives (186.1 ± 26.7/106.9 ± 22.4mmHg) versus (178.9 ± 18.8/99.6 ± 19.8mmHg) (t=1.2; p>0.05).

Comparing the patients and controls (Table 2), atherogenic index, and the levels of total plasma cholesterol, triglyceride, and LDL-cholesterol were significantly higher among patients than controls ($p < 0.05$ for each parameter). HDL-cholesterol was similar among patients and controls ($p > 0.05$). Comparing the three groups of patients (Table 2), atherogenic index, total plasma cholesterol, triglyceride, LDL-cholesterol, and HDL-cholesterol concentrations did not differ significantly among normoglycaemic hypertensives, normotensive diabetics, and diabetic hypertensives (F=0.0; p=0.99 for each parameter).

Hypertriglyceridaemia and hypercholesterolaemia were the most frequent dyslipidaemia. Of the 120 patients studied, 37 (31.1%) and 25 (20.8%) had hypertriglyceridaemia and hypercholesterolaemia, respectively. The frequency of dyslipidaemia among the normoglycaemic hypertensives, normotensive diabetics and diabetic hypertensives were 22 (55%), 23 (57.5%) and 22 (55%), respectively. The differences were not statistically significant (x^2=0.70; p=0.97).

DISCUSSION

Our results agree with the previous reports

Table 2. COMPARISON OF PLASMA LIPID LEVELS AMONG CONTROLS, HBP, DM AND HBP+DM PATIENTS

Parameters	Controls N=40	HBP N=40	DM N=40	HBP+DM N=40
*TC (mmol/L)	3.14±0.78 (2.59-4.65)	4.16±1.25 (0.55-6.23)	4.36±1.32 (1.08-7.02)	4.31±1.77 (2.20-6.21)
HDL-C (mmol/L)	1.21±0.31 (0.83-2.63)	1.21±0.48 (0.50-2.55)	1.20±0.55 (0.30-2.48)	1.29±0.61 (0.66-3.00)
*LDL-C (mmol/L)	1.92±0.63 (1.42-3.13)	2.26±1.00 (0.67-4.51)	2.37±1.22 (0.55-5.32)	2.34±0.81 (0.69-4.71)
*TGA (mmol/L)	1.22±0.33 (0.87-2.25)	1.74±0.60 (0.71-2.90)	1.79±0.56 (0.45-2.88)	1.82±0.29 (0.75-3.08)
*AI	3.10±1.08 (3.50-5.95)	4.26±1.79 (1.08-8.11)	4.16±1.10 (1.99-11.40)	4.21±1.04 (1.17-9.73)

**P<0.05 (Patients versus controls)*

that plasma lipid concentrations are higher among Nigerian hypertensives and diabetics than controls,[16-19] and that plasma lipid concentrations are lower in this population than in Caucasians.[34,35] Racial variations in plasma lipid concentrations are largely attributable to differences in the fiber component of diet. Traditional African diet is high in plant fiber and low in fats. In Rhodesia, for example, fats made up 17.8% and 42.7% of diet among Africans and whites, respectively.[36] High fiber diet reduces plasma lipids through reduction of total fat intake, reduction of fat absorption, and increased bile secretion.[37] The non-intake of cigarettes and alcohol among our patients are additional factors contributing to the comparatively lower plasma lipid concentrations obtained in the current study.[38]

Blood pressure and glycaemic control among our patients were poor in spite of having been commenced on treatment. These observations have also been made in the recent nationwide survey of noncommunicable diseases in Nigeria.[3] Poor blood pressure control among Nigerians has been attributed to poor compliance.[39]

It is further demonstrated in this study that plasma lipid concentrations, atherogenic index and prevalence of dyslipidaemia do not differ significantly among normoglycaemic hypertensives, normotensive diabetics and type 2 diabetic hypertensives. In a recent comparative profile of patients with diabetes mellitus and those with concurrent hypertension and diabetes, total plasma cholesterol did not differ significantly between the two groups: 4.6 ± 1.2 versus 4.6 ±0.8mmol/L.[20] Another report, however, showed that the prevalence of hypercholesterolaemia was significantly higher among a population of Nigerian hypertensives with or without diabetes than those with diabetes alone: 49.3% versus 19.4%.[21] This was not primarily a comparative lipid study, and confounding factors of dyslipidaemia were not excluded.

The absence of putative increases in plasma lipid concentrations in patients with concurrent type 2 diabetes and hypertension demonstrated in this study supports previous reports[40,41] that neither hyperglycaemia nor elevated blood pressure is responsible for hyperlipidaemia among patients with diabetes or hypertension. Specifically, dyslipidaemia does not occur in patients with secondary hypertension despite elevated blood pressure[40] or those with insulin-like-growth factor-1 deficiency (Laron dwarf) despite persistent hyperglycemia.[41]

Furthermore, if hypertension or diabetes influences lipid values, such values would have been magnified in the current study because of poor blood pressure and glycaemic control among our patients.

A link between diabetes, hypertension, and dyslipidaemia has been clearly described among Caucasians.[1,7] In a hypertensive animal model, a defective gene was recently identified as underlying insulin resistance and defective fatty acid metabolism.[11] The recognized role of genetically determined insulin resistance as a common pathogenic mechanism underlying the genesis of dyslipidaemia, diabetes, and hypertension in

Table 3. PREVALENCE OF DYSLIPIDAEMIA AMONG HBP, DM AND HBP+DM PATIENTS

Parameters	HBP N=40	DM N=40	HBP+DM N=40	Total
TC > 5.2mmol/L N (%)	8(20.0)	7(17.5)	10(25.0)	25
HDL-C < 0.9mmol/L N (%)	7(17.5)	9(22.5)	6(15.0)	22
LDL-C > 3.5mmol/L N (%)	6(15.0)	5(12.5)	4(10.0)	15
TGA > 1.75mmol/L N (%)	11(27.5)	10(25.0)	16(40.0)	37
AI > 5.8 N (%)	7(17.5)	6(15.0)	7(17.5)	20
Total	39	37	43	119

P>0.05 unless otherwise stated

human metabolic syndrome[1] may explain the lack of additive increases in plasma lipid concentrations when type 2 diabetes and hypertension occur concurrently.

Our results, therefore, appear to suggest the existence of metabolic syndrome among Nigerians. In spite of the relatively low plasma concentrations, metabolic syndrome would have important implications with respect to the potential rise in the incidence of ischaemic heart disease that has thus far been relative very low among Nigerians.[42,43]

In conclusions, concurrent diabetes and hypertension does not result in excess hyperlipidaemia than when either of the two conditions occurs in isolation. Evidence, albeit indirect, of metabolic syndrome among native Africans is provided.

We were constrained by lack of facilities for measurement of insulin and parameters of adrenergic tone such as cathecolamines in a developing country like Nigeria with scarce resources.

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