

Demographic and Clinical Correlates of Metabolic Syndrome in Native African Type-2 Diabetic Patients

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Objectives: To describe the metabolic syndrome and its demographic and clinical correlates in native African type-2 diabetic patients.

Methods: Cross-sectional analysis of 254 type-2 diabetic indigenous Nigerians consecutively recruited in a teaching hospital. The main outcome measure was metabolic syndrome. Variables of interest included family history/duration of diabetes mellitus and hypertension, gender, socioeconomic class, occupation and place of domicile (urban or rural). Intergroup comparisons were made with Chi-squared tests or t-tests.

Results: Patients were aged 35–80 years (mean: 52.0 ± 11.7 years) and made of 154 (60.6%) males and 100 (39.4%) females. Full-blown metabolic syndrome was noted in 52 patients (20.5%). Metabolic syndrome, as defined by the WHO, was noted in 150 patients (59.1%). About 72.4% of patients were dyslipidemic, 54.3% were hypertensive, 42.5% were obese, 44.9% were microalbuminuric and 32.3% were hyperuricemic. Ischemic heart disease (myocardial infarction) occurred in only 2.4% of patients. Concurrent hypertension and dyslipidemia; obesity and dyslipidemia; and hypertension and obesity occurred in 44.4%, 42.5% and 33.1% of type-2 diabetics, respectively.

Compared to the diabetics without metabolic syndrome, those with the syndrome had a significantly higher proportion of patients with a family history of hypertension and diabetes (44% versus 25%; $p=0.003$); among the upper/middle socioeconomic class: 52.0% versus 30.8% ($p=0.001$); and among the urban dwelling: 68.0% versus 49.0% ($p=0.004$). Metabolic syndrome was inversely proportional to the physical activity of an individual ($\chi^2=21.69$, $df=5$, $p=0.001$). Blood pressure was significantly higher among patients with metabolic syndrome than those without it ($140.6 \pm 22.9/85.2 \pm 12.9$ mmHg versus 126.9 ± 15.4 mmHg; $P<0.01$).

Conclusions: The development of metabolic syndrome in African type-2 diabetic patients is influenced by demographic and clinical factors. Vigilant dietary habit and physical exercise may reduce the chance of metabolic syndrome in urban Nigerian type-2 diabetics.

Key words: diabetes mellitus ■ metabolic syndrome ■ demographic variables ■ clinical variables

© 2005. From the Department of Medicine, Usmanu Danfodiyo University Teaching Hospital, Sokoto, Nigeria. Send correspondence and reprint requests for *J Natl Med Assoc.* 2005;97:557–563 to: Dr. S.A. Isezuo, Department of Medicine, Usmanu Danfodiyo University Teaching Hospital, Sokoto, Nigeria; e-mail: sisezuo@skannet.com

INTRODUCTION

Clustered metabolic cardiovascular risk factors, including type-2 diabetes mellitus, essential hypertension, obesity, dyslipidemia and ischemic heart disease, known as the metabolic syndrome, have been well-described.¹ Its prevalence rates range from 13–30% and 70–80% among the Caucasian nondiabetic^{2,3} and diabetic^{4,5} populations, respectively. The age-adjusted prevalence of the metabolic syndrome in an Indian urban population was 24.9%.⁶ Genetically determined insulin resistance in a setting of suitable environmental factors is the pivotal pathogenic mechanism underlying the metabolic syndrome.¹ Lipoprotein lipase deficiency largely accounts for the lipid abnormalities,⁷ while systemic hypertension is due to enhanced sympathetic activities, salt sensitivity and increased transmembrane cation transport.^{8,9} The role of tumor necrosis factor in obesity and insulin resistance has also been described.¹⁰

Developing nations are witnessing rapid industrialization, urbanization and increased economic prosperity. The resulting acquisition of the western lifestyle, characterized by calorie excess and physical inactivity, would provide suitable milieu for the development of the metabolic syndrome in genetically predisposed individuals. In Nigeria, hypertension and diabetes mellitus occur in 10–15% and 2–4% of the population, respectively.¹¹ Both conditions coexist frequently, the prevalence of hypertension among diabetics being 20–40%.^{12,13} They are independent risk factors for dyslipidemia.^{12–15} Obesity is also linked with lipid abnormalities among the Africans.¹⁶

As the increased economic prosperity at individual levels in developing nations is uneven, it caused a demographically heterogeneous population. Demographic factors may, therefore, affect the development of the metabolic syndrome. This report describes clustering of cardiovascular risk factors, including the metabolic syndrome in Nigerian type-2 diabetic patients. The demographic and clinical correlates of the syndrome are determined. This information is vital because the metabolic syndrome possesses the potential for altering the epidemiological pattern of

morbidity and mortality in Nigeria. Furthermore, knowledge of the variables influencing the development of the syndrome may be utilized in interventions that could favorably alter its prevalence.

PATIENTS AND METHODS

Patients

Two-hundred-seventy-eight indigenous Nigerians with diabetes mellitus attended the medical out-

patient and inpatient units between January and August 2002 at the Usmanu Danfodiyo University Teaching Hospital (UDUTH), a major referral centre with a catchment population of about eight million. It is located in Sokoto, northwestern Nigeria (sub-Saharan Africa). The northwestern region of Nigeria is located between latitude 11:000 N and 14:000 N and longitude 4:000 E and 7:000 E. Hausa and Fulani are the dominant tribes, while Islam is the dominant religion. Of the 278 diabetics who attended the hospital during the study period, 254 (91.4%) with type-2 diabetes mellitus were studied. The remaining 24 patients had type-1 diabetes mellitus and were excluded from the study. There were 154 (60.6%) males and 100 (39.4%) females. The mean age was 52.0 ± 11.7 years (range: 35–80 years).

Patients with secondary hypertension, nephrotic syndrome, hepato-biliary disease and hypothyroidism were excluded from the study. This was through clinical evaluation, except in nephrotic syndrome, where massive proteinuria was also required as an exclusion criterion. Patients taking lipid-altering drugs, including lipid-lowering agents, were excluded from the study.

Demographic and Clinical Data

Demographic and clinical data, including place of domicile (urban or rural); occupational/educational status; ischemic heart disease (evidenced by history of angina pain, myocardial infarction and coronary artery surgery); family history of diabetes and/or hypertension; diet; alcohol consumption; and cigarette smoking were obtained. Socioeconomic status was stratified into lower, middle and upper classes using the British Registrar General Scale.¹⁷ Consumption of 20 or more units of alcohol weekly and 10 or more cigarettes daily constituted significant alcohol consumption and cigarette smoking, respectively.^{18,19}

Diabetes mellitus was diagnosed using the World Health Organization (WHO) diagnostic criteria.²⁰ A diabetic with no record of ketosis and on oral hypoglycemic drugs and/or a diabetic diet was considered to have type-2 diabetes mellitus. Sitting blood pressure was measured with aneroid mercury sphygmomanometer (Accoson), size 13.5 x 10³ cm, using the patient's nondominant arm and after 10 minutes of rest. Three readings were taken, and the average of the last two was taken as the

Table 1. Baseline Characteristics of Africans with Type-2 Diabetes Mellitus

Characteristics	Values (N=254) N (%)
Gender	
Male	154 (60.6)
Female	100 (39.4)
Place of Domicile	
Urban	153 (60.2)
Rural	101 (39.8)
Literacy	
Literate	82 (32.3)
Illiterate	172 (67.7)
Socioeconomic Status	
Upper	50 (19.7)
Middle	60 (23.6)
Lower	144 (56.7)
Family history of diabetes mellitus (DM)	32 (12.6)
Family history of hypertension (HBP)	32 (12.6)
Family history of HBP and DM	28 (11.0)
Neither family history of HBP nor DM	144 (56.7)
Cigarette smoking	72 (28.4)
Alcohol consumption	34 (13.4)
Concurrent HBP and DM	138 (54.3)
	Mean ± SD
Age (years)	52 ± 11.7
Duration of diagnosis of HBP (years)	6.1 ± 6.7
Duration of diagnosis of DM (years)	6.1 ± 6.6
Systolic blood pressure (mmHg)	135.0 ± 21.1
Diastolic blood pressure (mmHg)	83.1 ± 11.8
Body mass index (kg/m ²)	25.8 ± 6.1
Waist circumference (cm)	95.5 ± 6.2
Fasting blood sugar (mmol/L)	10.5 ± 5.4
Total cholesterol (mg%)	195.5 ± 28.7
High-density lipoprotein cholesterol (mg%)	47.3 ± 12.2
Triglyceride (mg%)	155.7 ± 42.2
Serum uric acid (mg%)	6.9 ± 2.2

blood pressure. Systolic blood pressure (Korotkoff phase I) of ≥ 140 mmHg and/or diastolic blood pressure (Korotkoff phase V) of ≥ 90 mmHg or antihypertensive medications were required to make a diagnosis of systemic hypertension.²¹ Anthropometric indices, including weight, height, waist and hip circumferences were measured with patients lightly clothed and without shoes. A body mass index of ≥ 30 kg/m² and a waist circumference of ≥ 88 cm (for females) or ≥ 102 cm (for males) constituted general and central obesity, respectively.²² Weight status was classified using body mass index, into the following categories: lean (< 18.5 kg/m²), normal (18.5 – 24.9 kg/m²), overweight (25 – 29.9 kg/m²) and obesity (≥ 30 kg/m²).²⁰ All patients had resting 12-lead electrocardiography (ECG) to detect ECG evidence of myocardial infarction.

Biochemical Data

The sample analysis was conducted at the Chemical Pathology Department of UDUTH in Sokoto. About 10 ml of fasting serum was withdrawn into heparinized bottles of fluoride oxalate and centrifuged at 100 rpm for five minutes. The supernatant was separated into the appropriate containers for analysis. Samples were analyzed within 24 hours of collection or stored at a temperature of 4°C. Fasting blood glucose was measured using glucose oxidase tests.²³ Total plasma cholesterol was determined using ferric per chlorate methods.²⁴ High-density lipoprotein (HDL) cholesterol was determined after precipitation of low-density lipoprotein (LDL) cholesterol with phosphotungstate and magnesium.²⁵ Triglycerides were measured using the calorimetric enzymatic method.²⁶

LDL cholesterol was calculated from the following formula:

$$\text{LDL cholesterol} = \text{Total cholesterol} - \text{HDL cholesterol} - \text{Triglycerides} / 5$$

A preprepared laboratory standard for lipid analysis was used to ensure quality assurance of the specimens. A plasma uric acid level was determined.

Screening for microalbuminuria was done using early morning urine and the Micral R test (Boehringer-Mannheim, Germany). The screening was done on two occasions over a four-week period. A patient with two positive results was considered to have microalbuminuria. All of our patients, as a routine, had dipstick urinalyses for albumin, nitrite and blood. A urine specimen was also obtained and centrifuged for microscopy. A urinary tract infection, as evidenced by a clinical evaluation or a positive dipstick urinalysis for nitrite, hematuria and white cell count, in the range of 2–5 per high-power field, was excluded before screening for microalbuminuria.

A type-2 diabetic patient with any two of the fol-

lowing was considered to have metabolic syndrome: hypertension; dyslipidemia (triglycerides > 150 mg% or HDL cholesterol < 40 mg%); obesity; and microalbuminuria.²⁰ Microalbuminuria was not utilized as a diagnostic criterion of metabolic syndrome in patients with macroalbuminuria.

Statistical Analysis

Data entry and analysis were done using a statistical software package. Means were presented as follows: value \pm standard deviation. Comparisons of categorical and continuous variables between diabetics with and without the metabolic syndrome were done using Chi-squared tests and the independent sample t-tests (two-tailed), respectively. A p value of < 0.05 was considered statistically significant.

RESULTS

Two-hundred-fifty-four type-2 diabetic patients, aged 52.0 ± 11.7 years (range: 35–80 years) made up of 154 (60.6%) males and 100 (39.4%) females were studied. One-hundred-thirty-eight patients (54.3%) had concurrent hypertension and diabetes mellitus. Of these, hypertension and diabetes were simultaneously diagnosed in 64 patients (46.4%). The diagnosis of hypertension preceded diabetes mellitus by 7.9 ± 5.8 years in 42 patients (30.4%), while that of diabetes mellitus preceded hypertension by 4.8 ± 5.8 years in 32 patients (23.2%).

The demographic, clinical and biochemical characteristics of patients are shown in Table 1. Family history of diabetes mellitus, hypertension and concurrent hypertension and diabetes mellitus occurred in 12.6%, 12.6% and 11.1% of patients, respectively. Though all of the patients adhered to traditional African fiber-rich food choices (rice, millet, corn and beans) as staples, 112 (44.1%) also consumed dairy products and animal fats, including cows' milk in its fresh and fried forms (called "nono" and "manshanu", respectively, in the native language). Though only 32% were engaged in regular formal exercise, all patients had daily activities that were physically demanding.

The frequencies of individual components of the metabolic syndrome were as follows: dyslipidemia (72.4%), systemic hypertension (54.3%), obesity (42.5%), microalbuminuria (44.9%) and hyperuricemia (32.3%). Ischemic heart disease (myocardial infarction) occurred in six patients (2.4%). Of the 254 diabetics studied, 150 (59.1%) had metabolic syndrome, using the WHO definition (type-2 diabetes mellitus, plus any two of the following: essential hypertension, dyslipidemia, obesity and microalbuminuria). Full-blown metabolic syndrome (type-2 diabetes mellitus, systemic hypertension, obesity, dyslipidemia and microalbuminuria) occurred in 52 patients (20.5%). The various clustered components

of metabolic syndrome are shown in Figure 1. Concurrent systemic hypertension and obesity; systemic hypertension and dyslipidemia; and obesity and dyslipidemia occurred in 114 (44.9%), 108 (42.5%) and 84 (33.1%) diabetics, respectively. Microalbuminuria coexisted with systemic hypertension, dyslipidemia and obesity in 92 (36.2%), 78 (30.7%) and 74 (29.1%) diabetics, respectively.

Type-2 diabetic patients with and without metabolic syndrome are compared in Table 2. Compared to diabetics without metabolic syndrome, those with the syndrome had significantly higher proportions of patients with concurrent diabetes and systemic hypertension (74.7% versus 25.0%; $p < 0.0001$), positive family history of diabetes mellitus and systemic hypertension (44% versus 25%; $p = 0.003$), upper/middle socioeconomic class (52% versus 30.8%; $p = 0.001$) and urban dwelling (68.0% versus 49.0%; $p = 0.004$). Occupation significantly influenced the frequency of metabolic syndrome, with the highest proportion occurring in those associated

with sedentary lifestyles. Of the 62 full-time housewives, 56 civil servants, 76 traders, 16 professionals, 24 manual peasant farmers and 20 belonging to other occupations, metabolic syndrome occurred in 42 (67.8%), 36 (64.3%), 48 (63.2%), 10 (62.5%), four (16.1%) and 10 (50%) patients, respectively ($\chi^2 = 21.69$, $df = 5$; $p = 0.001$).

Blood pressure was significantly higher among patients with metabolic syndrome ($140.6 \pm 22.9/ 85.2 \pm 12.9$ mmHg versus 126.9 ± 15.4 mmHg; $P < 0.01$).

Metabolic syndrome was insignificantly higher in percentage among females than males (64.0% versus 55.8%; $P = 0.57$). Notably, HDL cholesterol was insignificantly higher among diabetics with metabolic syndrome than among those without the syndrome (48.5 ± 12.7 mg% versus 45.5 ± 11.2 mg%; $t = 1.3$; $p = 0.2$) and among obese than nonobese diabetics (48.3 ± 12.4 mg% versus 47.7 ± 10.8 mg%, $p > 0.05$). Age, gender, literacy status, cigarette smoking, alcohol consumption, blood sugar levels, duration of diagnosis of diabetes mellitus, and simultaneous onset of

Table 2. Comparison of Demographic and Clinical Characteristics of Patients with and without Metabolic Syndrome

Characteristics	Patients with Metabolic Syndrome (N=150)	Patients without Metabolic Syndrome (N=104)	P Value
	Mean ± SD	Mean ± SD	
Age (years)	52.9 ± 10.8	50.7 ± 12.8	0.30
Duration of diagnosis of DM (years)	4.4 ± 4.6	3.7 ± 4.0	0.40
Interval between DM and hypertension (HBP) onset (years)	4.6 ± 6.2	5.6 ± 5.3	0.80
Systolic BP (mmHg)	140.6 ± 22.9	126.9 ± 15.4	<0.001
Diastolic BP (mmHg)	85.2 ± 12.9	79.7 ± 9.1	0.01
Body mass index (kg/m ²)	28.4 ± 5.6	22.4 ± 3.6	<0.001
Waist circumference (cm)	100.2 ± 13.3	87.5 ± 8.7	<0.001
Fasting blood sugar (mmol/L)	10.3 ± 5.0	10.8 ± 6.0	0.60
Total cholesterol (mg%)	187.6 ± 28.2	166.0 ± 24.1	<0.001
Low-density lipoprotein cholesterol (mg%)	101.1 ± 21.4	93.9 ± 19.1	0.06
High-density lipoprotein cholesterol HDL-C (mg%)	48.5 ± 12.7	45.5 ± 11.2	0.20
Triglycerides (mg%)	169.2 ± 38.2	135.2 ± 39.5	<0.001
TC:HDL-C ratio	4.0 ± 1.1	3.8 ± 0.9	0.26
Serum uric acid (mg%)	7.6 ± 2.2	5.9 ± 2.7	<0.001
	N (%)	N (%)	
Male:female ratio	1.3	1.9	0.30
Upper and middle class	78 (52)	32 (30.8)	<0.001
Literates	46 (30.7)	36 (34.6)	0.60
Urban-dwelling	102 (68)	51 (49)	<0.001
Positive family history of DM and/or HBP	66 (44)	26 (25%)	<0.001
Cigarette smoking	34 (22.7)	38 (36.5)	0.10
Alcohol consumption	20 (13.3)	14 (13.5)	0.09
Simultaneous diagnosis of HBP and DM	46 (30.7)	18 (17.3)	0.02
Concurrent DM & HBP	112 (74.7)	26 (25.0)	<0.001
Obesity	69 (69.3)	8 (7.7)	<0.01
Dyslipidemia	138 (92)	46 (44.2)	1.01
Hyperuricemia	62 (41.3)	20 (19.2)	<0.001
Albuminuria	98 (65.3)	16 (15.4)	<0.001

clinical hypertension and diabetes mellitus did not appear to influence the occurrence of metabolic syndrome among type-2 diabetics.

DISCUSSION

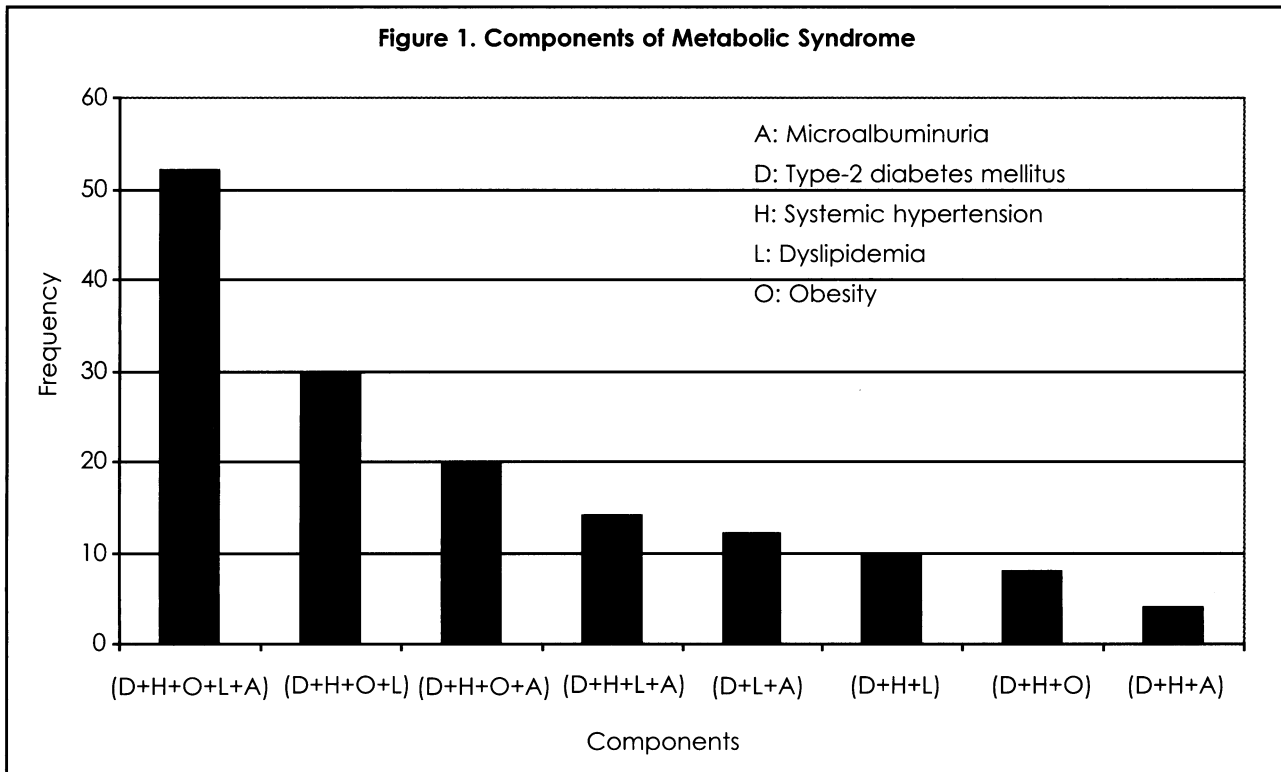
This report describes clustering of cardiovascular risk factors consistent with the metabolic syndrome found among Nigerian type-2 diabetic patients. The prevalence of the syndrome is lower than values ranging from 70–80% among Caucasians with type-2 diabetes mellitus.^{4,5,28} It is, however, higher compared to the recently reported prevalence of 25.2% among type-2 diabetic patients in southern Nigeria²⁹ but similar to the data from Zimbabwean urban type-2 diabetic patients.³⁰ Disparities in the prevalence of metabolic syndrome are largely due to differences in lifestyles, age of the study populations and nonapplication of uniform diagnostic criteria.^{31,32}

In the present report, metabolic syndrome is not associated with HDL hypocholesterolemia, and ischemic heart disease is a relative rarity. These findings are strikingly at variance with the Caucasians^{2,3} but consistent with previous reports on African diabetics and hypertensives.^{13,14,33} Normal or elevated HDL cholesterol in diabetes mellitus^{13,34} and a direct relationship between obesity and HDL cholesterol¹⁶ are well-documented among Africans. Obesity is diet-related among Africans. Obese Africans, therefore, tend to consume correspondingly high amounts of fiber. Epidemiological studies have linked high fiber-rich food consumption with

reduced risk of coronary artery disease.^{35,36} In diabetes mellitus, HDL hypocholesterolemia is due to a reduction of HDL₂ subfraction of HDL cholesterol arising from increased HDL cholesterol catabolism. It is not known if fiber reduces catabolism of HDL cholesterol in type-2 diabetics.

Two types of type-2 diabetic populations have been described in blacks: one with insulin resistance and increased risk of cardiovascular mortality and morbidity, and the other that is insulin sensitive with no increased risk of cardiovascular disease.^{37,38} Insulin resistance is genetically determined and underlies the pathogenic mechanism of the metabolic syndrome.¹ Family history of hypertension, type-2 diabetes mellitus and obesity are recognized markers of genetic predisposition to the syndrome.³⁹ The present study demonstrated that genetic predisposition to the metabolic syndrome is not increased in type-2 diabetics with a family history of concomitant hypertension and diabetes mellitus, compared to those with a family history of either of the two conditions occurring in isolation.

In addition to insulin resistance, suitable environmental factors are an important prerequisite for the clinical expression of the metabolic syndrome. Variations in these factors partly explain the population differences in the frequency of the syndrome. We recorded a significantly higher percentage of metabolic syndrome among type-2 diabetics in the upper/middle socioeconomic class compared to the lower class. Differences in dietary habits largely



account for socioeconomic class-related variations in the prevalence of the metabolic syndrome. In the Chennai Indian population, for example, individuals in the middle class had significantly higher monthly income, calorie and fat intake and increased prevalence of metabolic syndrome, compared to those in the lower class.⁴⁰

A rural-urban difference in the prevalence of metabolic syndrome has also been documented in the Palestinian West Bank community.⁴ Affluent and sedentary lifestyles, characterized by high calorie and fat intake, are more likely among the urban population than the rural population. In the developing nations, the prevalence of hypertension and diabetes is higher in the urban areas than in rural areas.¹¹ It is, therefore, not surprising that the percentage of occurrence of metabolic syndrome is significantly higher among urban- rather than rural-dwelling type-2 diabetes mellitus patients. Given the increasing urbanization in developing nations, an association of urbanization with the increased clustering of cardiovascular risk factors among type-2 diabetics should be a major public health concern.

The effect of gender on the prevalence of metabolic syndrome is uncertain. While some reports showed a higher value among females than males,⁴¹ others showed no such relationship.² The higher percentage of the syndrome among female type-2 diabetes mellitus patients in the present report with an Islam-dominated population may be explained by the sedentary lifestyle arising from the practice of purdah, a religious obligation that restricts women to their homes.

Data on the specific effects of occupation on the metabolic syndrome are lacking. It is, however, well-documented that type-2 diabetics that are engaged in physical exercise have decreased clustering of cardiovascular risk factors.^{42,43} Peasant farming is the predominant occupation in the developing nations. It is manual and physically demanding. This probably accounts for the inverse relationship between the metabolic syndrome and physical activities of individuals in the current report.

We were constrained by the lack of facilities for the determination of insulin sensitivity. The high drop-out rate of patients from the clinic in the setting where this work was carried out compelled us to screen for microalbuminuria over a four-week period instead of the recommended three-to-six month period.

In conclusion, the occurrence of metabolic syndrome in type-2 diabetes mellitus patients is affected by clinical and demographic variables that could be potentially modified to favorably alter the prevalence of the syndrome in this population.

REFERENCES

1. Reaven GM. Role of insulin resistance in human disease. *Diabetes*. 1989; 37:1595-1607.
2. Ford ES, Buft G, Dietz W. Prevalence of the metabolic syndrome among U.S. adults: finding from the 3rd National Health Nutrition Examination Survey. *JAMA*. 2002;287:356-359.
3. Movakovic B, Popovic M. Occurrence of the metabolic syndrome in the population of the town of Novi Sed. *Med Pregl*. 2001;54:17-20.
4. Abdu-Rahim HF, Hussein A, Bjertness F, et al. The metabolic syndrome in the West Bank population: an urban-rural comparison. *Diabetes Care*. 2001;24:275-279.
5. Balkau B, Charles MA, Drivsholm T, et al. Frequency of WHO-defined metabolic syndrome in European cohort and an alternative definition of an insulin resistance syndrome. *Diabetes Metab*. 2002;28:364-376.
6. Gupta R, Deedwania PC, Gupta A, et al. Prevalence of metabolic syndrome in an Indian urban population. *Int J Cardiol*. 2004;97:257-261.
7. Verges BL. Dyslipidemia in diabetes mellitus. Review of the main lipoprotein abnormalities and their consequences on the development of atherosclerosis. *Diabetes Metab*. 1999;25:32-40.
8. Rosolova H. The sympathetic nervous system and insulin resistance. *Vntr Kek*. 2003;49:61-65.
9. Ginner V, Coca A, Sierra A. Increased insulin resistance in salt-sensitive hypertension. *J Hum Hypertens*. 2001;15:481-485.
10. Hotamisligil GS. The role of TNF-alpha and TNF receptors in obesity and insulin resistance. *J Intern Med*. 1999;245:621-625.
11. Akinkugbe OO. Noncommunicable diseases in Nigeria: the next epidemics. Abayomi Memorial Lecture. *Nigerian Journal of Medical Practice*. 2000;3:904-907.
12. Ikem RT, Akinola NO, Balogun MO, et al. What does the presence of hypertension portend in the Nigerian with noninsulin diabetes mellitus? *WAJM*. 2001;20:127-130.
13. Isezuo AS, Badung SLH, Omotoso ABO. Comparative analysis of lipid profiles among patients with type-2 diabetes, hypertension and concurrent hypertension and diabetes: a view of metabolic syndrome. *J Natl Med Assoc*. 2003;95:328-334.
14. Isezuo AS, Badung SL. Plasma lipid profiles among northwestern Nigerian hypertensives. *Sahel Medical Journal*. 2000;4:181-186.
15. Agboola-Abu CF, Onabolu A. Plasma lipid levels in patients attending Igbinedion Hospital and Medical Research Centre, Okada, Edo State, Nigeria. *Nigerian Medical Journal*. 2000;38:1-5.
16. Njelekela AH, Nedishi Y, Nara T, et al. Obesity and lipid profiles in middle aged men and women in Tanzania. *East Afr Med J*. 2002;79:58-64.
17. Stevenson THC. British Registrar General Scale: classification of occupation according to their socioeconomic significance. *Royal Stat Soc*. 1928;91:207.
18. Doll R, Peto R, Hall H. Mortality in relation to consumption of alcohol: 13 years observation of male British Doctors. *Br Med J*. 1994;209:911-918.
19. Doll R, Peto R, Wheatler K. Mortality in relation to smoking: 40 years observation of male British Doctors. *Br Med J*. 1994;209:901-911.
20. World Health Organization. Definition, diagnosis and classification of diabetes mellitus and its complications: report of a WHO consultation, part I: diagnosis and classification of diabetes mellitus. Geneva, Switzerland; 1999.
21. 7th report of the Joint National committee on prevention, detection, evaluation and treatment of high blood pressure. *JAMA*. 2003;289: 2560-2572.
22. World Health Organization. Consultation on obesity. Classification according to BMI. Geneva, June 3-5, 1997.
23. Trinder P. Determination of blood glucose using 4-aminophenazone as oxygen carrier acceptor. *J Clin Pathol*. 1969;22:246.
24. Levine JB, Zak B. Ferric Chloride method of determination of total cholesterol. *Clin Chim Acta*. 1964;10:381-384.
25. Busterin M, Scholnick HR, Morfin R. Rapid method for the isolation of lipoproteins from human serum by precipitation with polyanions. *J Lip Res*. 1970;11:583-595.

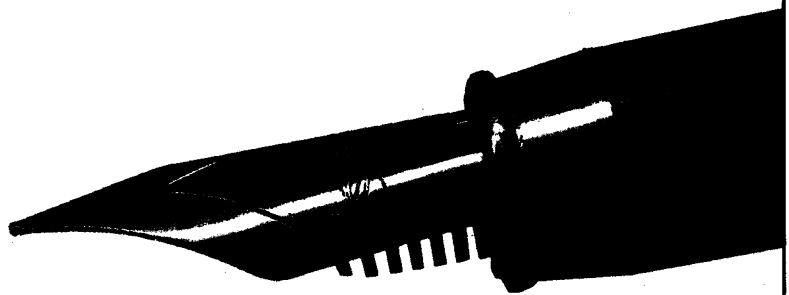
26. McGowan MW, Artiss JD, Strandergh DR, et al. Peroxidase-coupled method for the calorimetric determination of serum triglycerides. *Clin Chem*. 1983;29:583-542.
27. Friedwald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma without use of preparative ultracentrifugation. *Clin Chem*. 1972;18:499-502.
28. Bruno G, Merletti F, Biggeri A, et al. Metabolic syndrome as a predictor of all cause and cardiovascular mortality in type-2 diabetes: the Casale Monferrato Study. *Diabetes Care*. 2004;27:2689-2694.
29. Alebiosu CO, Odusan BO. Metabolic syndrome in subjects with type-2 diabetes mellitus. *J Natl Med Assoc*. 2004;96:817-821.
30. Makuyana D, Gomo Z, Munyombwe T, et al. Metabolic syndrome disorders in urban black Zimbabweans with type-2 diabetes mellitus. *Cent Afr J Med*. 2004;50:24-29.
31. Park HS, Oh SW, Cho S I, et al. The metabolic syndrome and associated lifestyle factor among South Korean adults. *Int J Epidemiol*. 2004;33:328-336.
32. Marchesini G, Forlani G, Cerrelli F, et al. WHO and ATP III proposals for the definition of the metabolic syndrome in patients with type-2 diabetes mellitus. *Diabet Med*. 2004;21:383-387.
33. Sharma MD, Pavuk VN. Dyslipidemia in African Americans, Hispanics and whites with type-2 diabetes and hypertension. *Diabetes Obes Metab*. 2001;3:41-45.
34. Aduba O, Onowamaeze I, Oli J, et al. Serum cholesterol and high density lipoprotein cholesterol in Nigerian diabetics. *East Afr Med J*. 1984;61:35-39.
35. Jerikins DJ, Axelsen M, Kendall CW, et al. Dietary fiber, carbohydrates and the insulin resistant diseases. *Br J Nutr*. 2000;83:157-163.
36. Wirfalt E, Hedblad B, Gullberg B, et al. Food patterns and components of the metabolic syndrome in men and women: a cross-sectional study with the Malmo Diet (a cancer cohort). *Am J Epidemiol*. 2001;154:1150-1159.
37. Chaiken RL, Benjeri MA, Pasmantier R. Patterns of glucose and lipid abnormalities in black NIDDM subjects. *Diabetes Care*. 1991;14:1036-1042.
38. Banerji MA. Diabetes in African Americans: unique pathophysiological features. *Curr Diab Rep*. 2004;4:219-223.
39. Hunt KJ, Haiss G, Sholinsky PD, et al. Family history of metabolic disorders and the multiple metabolic syndrome: the NALBI family heart study. *Epidemiology*. 2000;19:395-409.
40. Mohan U, Shantherenis, Deepa R, et al. Intraurban differences in the prevalence of the metabolic syndrome in Southern India—the Chennai Urban Population Study. *Diabet Med*. 2001;18:28-287.
41. Dizare T, Kobayashi M, Sato Y, et al. Possible link between a low prevalence of cardiovascular disease and dyslipidemia: a study in Japanese patients with type-2 diabetes. *Diabet Med*. 1993;10:431-437.
42. Wannamethee SG, Shaper AG, Alberti KG. Physical activity, metabolic factors and the incidence of coronary artery disease and type-2 diabetes. *Arch Intern Med*. 2000;160:2108-2116.
43. Brage S, Wedderkopp N, Ekelund U, et al. Features of the metabolic syndrome are associated with objectively measured physical activity and fitness in Danish children: the European Youth Heart Study. *Diabetes Care*. 2004;27:2141-2148. ■

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