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Full Length Research Paper

Low birth weight, metabolic syndrome and their associations with the global crisis of 1930 - 1945, rapidly growing economy and coronary heart disease in Central Africa

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The purpose of this study is to determine the prevalence of low birth weight (LBW) and metabolic syndrome (Mets) and their associations with 1930 - 1945 years of global crisis and cardiovascular risk factors in Central Africans. The study was a hospital-based cross-sectional study conducted on Central African patients born between 1930 and 1977. Mets was diagnosed using WHO criteria. Of 407 patients, 262 (64. 6%) and 77 (18.9%) met the criteria of LBW and Mets, respectively. There was association between birth during 1930 global crisis and 1945 second war period; adulthood hypertension, low economic growth, high pulse pressure, type 2 diabetes, decline of renal function, hypercholesterolemia, left ventricular hypertrophy and LBW. There was a U-shaped relationship between current body mass index and Mets in all patients and men, but a linear relationship between current body mass index and Mets in women. Coronary heart disease (OR = 2.3 95%CI 1.1 - 4.8; P = 0.024), LBW (OR = 10 95%CI 3.9 - 25.5; P < 0.0001), elevated fibrinogen (OR = 3.5 95%CI 2 - 6.1; P < 0.0001) were the independent risk factors of Mets in all patients. LBW effect on Mets was lower in men (OR = 7.5 95%IC 2.6 - 22.1; P < 0.0001) than in women (OR = 18 95%CI 2.3 - 37; P = 0.005). In a separate multivariate analysis for only continuous variables, Mets in all patients was independently determined as follows: Y = -1.523 + 0.003 fibrinogen + 0.01 total cholesterol - 0.001 birth weight. LBW, coronary heart disease, malnutrition, elevated fibrinogen, total cholesterol and urea nitrogen may be considered as additional components of Mets in the African patients born between 1930 and 1945 and more in women than in men.

Key words: Low birth weight, fibrinogen, cardiovascular risk factors, metabolic syndrome, Sub-Saharan Africa.

INTRODUCTION

The aim of this study was to determine the prevalence of low birth weight in the community among people born in the period between 1930 and 1977 and to explore the possible association of low birth weight with the metabolic syndrome.

The burden of metabolic syndrome (Mets) is increasing worldwide, most rapidly in African and Asian developing countries (Ford et al., 2002; Ghaffar et al., 2004; Kelliny et al., 2008). This has been attributed to greater availability of food, urbanization and industrialisation and reduced physical activity, resulting in increased adiposity (Misra and Khurana, 2008). Some evidence states that widely prevalent perinatal under nutrition (Low Birth Weight = LBW) may play a significant role in adult-onset of cardio-vascular disease (CVD), type 2 diabetes mellitus (T2DM) and Mets (Barker, 1998).

A thrifty phenotype hypothesis that emphasizes fœtal under-nutrition leading to altered metabolic programming is perhaps a better explanation in resource-poor developing countries because abundant food supply later in life leads to maladaptive increase in weight and in-crease the risk of non-communicable diseases (diabetes, hypertension, CVD)(Prentice and Moore, 2005; Prentice,

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2003; Hales and Barker, 2001; Bavdekar et al., 1999; Mi et al., 2000).

In DRC, black Congolese born with LBW showed higher risk of arterial hypertension (Longo-Mbenza et al., 1999) and people born during 1930 - 1945 periods, suffered so much from famine because of the global crisis in 1930 and the 1940 - 1945 second world war. However, they enjoyed the Belgian colonization: induced improvement in economic situation, westernization after rural-urban migration, aggressive community nutrition intervention programs for undernourished children, control of epidemic infectious disease and welfare between 1950 and 1970. From 1971 till now, Congolese people are facing rapid urbanization, collapse of economy, recession, ethnic conflicts and civil wars. Whether low birth weight (LBW) in adults is an independent risk factor of metabolic syndrome has not been investigated in both developed and developing countries.

This study had four objectives. First, we evaluated the prevalence of Mets according to the World Health Organization (WHO) definition criteria (Alberti and Zimmet, 1998) as waist circumference (WC) was not available at the study period. Secondly, we examined the overlapping of the curves of the distribution of low birth weight (LBW) by birth year and the trend of the growth Product Domestic National of DRC between 1930 and 1955. Thirdly, we determined the univariate association between sex, age, blood pressure, pulse pressure, fasting glucose, urea nitrogen, CVD, creatinin, uric acid, fibrinogen, total cholesterol, left ventricular hypertrophy, arterial hypertension, T2DM, Mets and LBW. Finally, we examined the independent association of LBW and other risk factors with Mets in all patients, men and women.

MATERIALS AND METHODS

This cross-sectional study was conducted by analysing the base line obtained from a clinic-based cohort established in 1997 - 1998 for cardiovascular risk factors at Lomo Medical Clinic, Kinshasa Limete, DRC. There were 419 consecutive black patients, born between 1930 and 1977, eligible for the study. We considered for inclusion only patients who gave their informed consent and had complete data. The present study was conducted in accordance with the Helsinki Declaration II and the local Ethics Committee procedures.

Study procedures and data collection

Interviews were conducted by trained nurses through a structured and standardized questionnaire administered during 30 min in one session for recording demographic data (sex, birth year, current age, cigarette smoking habit, alcohol intake and physical activity). Birth weight was obtained from records of maternities and parents or patients for “Matsombe” babies born with small size (n = 17).

The data concerning the Belgian Congo Colony Gross National Product and Growth Product (Domestic National = GP) based on productivity and export between 1800 and 1955, were obtained from the work of Bairoch (1976). Clinical examination included measurements of current body weight, height and BP. Weight in light clothes was measured to the nearest 100 g (0.1 kg) using a Soehnle scale (Soehnle-Waagen GmGh Co, Murrhardt, Germany). And height was measured to the nearest 0.5 cm using portable locally manufactured stadiometers. Body mass index (BMI) was calculated as weight divided by height squared (kg/m^2). Three consecutive diastolic and systolic blood pressures (SBP and DBP) were recorded on the right arm using a standard mercury sphygmomanometer with appropriate cuff sizes at intervals longer than 2 min after 15 min of rest in seated patients. The average of the second and the third readings was used in the present analyses.

A standard 75 g OGTT was performed in all patients between 07.00 AM and 10.00 Am, after an overnight fast of at least 10 - 12 h. In each patient, 12.5 ml of whole blood was drawn before and after 120 min. Plasma glucose determined by the glucose-oxidase method, urea nitrogen, uric acid, creatinin, fibrinogen, total cholesterol, triglycerides and HDL-cholesterol were measured on commercial kits (Biomérieux, Marcy l’Etoile, France) and a Hospitex autoanalyzer (Hospitex Diagnostics, Florence, Italy).

Study definitions

LBW was defined by birth weight < 2500 g. T2DM was defined by either self-report accompanied by use of anti-hyperglycemic medication, or by elevated fasting plasma glucose (FPG) ≥ 126 mg/dl (7.0 mmol/l) or by OGTT ≥ 200 mg/Dl (11.1 mmol/l) (American Diabetes Association, 1997). Mets was defined according to the WHO criteria that require the presence of impaired glucose tolerance, impaired fasting glycemia, diabetes plus two or more of the following: BMI ≥ 30 kg/m^2, fasting triglycerides ≥150 mg/dl (≥1.70 mmol/l, SBP ≥ 140 mmHg and/or DBP ≥ 90 mmHg, and fasting total cholesterol (TC) ≥ 5.2 mmol/l (≥ 200 mg/dl) or use of lowering lipid drugs. BMI ≥ 25 kg/m^2 was considered as total obesity (overweight/obesity) (JAMA, 2003).

Smoking status included current smokers or former smokers versus never smoked. Alcohol intake was defined by regular consumption of ≥ 2 drinks/day. Arterial hypertension was defined by SBP ≥ 140 mmHg and DBP ≥ 90 mmHg, or use of antihypertensive treatment. Increase in pulse pressure (SBP - DBP) meant arterial stiffness/surrogate of subclinical atherosclerosis. Coronary heart disease (CHD) diagnosis in this study was based on history of angina, Minesota codes, electrocardiogram changes, enzymes and documented history of interventional procedures performed abroad (Belgium, France, USA, South Africa). Ischemic stoke occurred for the first time and its diagnosis was confirmed by CTscan of brain performed within 10 h after the onset.

The estimated glomerular filtration rate (e GFR) was calculated using the formula derived from the Modification of Diet in Renal Disease (MDRD) study (Levy et al., 1999) as follows : Cr = creatinin, A = age, f = if female, B = if black; eGFR in mL/min/1.73 m^2 = 186 x $(C2/88.4)^{-1.154}$ x $A^{-0.203}$ x 0.742 (f) x 1.210(b). Chronic kidney disease (CKD) was defined by e GFR< 60 mL/1.73 m^2.

New and additional potential factors of Mets included elevated fibrinogen ≥ 375 mg/dl (median value), hyperuricemia (uric acid ≥ 6 mg/dl, median value), elevated urea nitrogen ≥ 20 mg/dl (median value, smoking, LBW, CHD, CKD, echocardiography-defined left ventricle (LV) hypertrophy components such as increase in end-diastolic LV internal dimension(LViDd), end-diastolic inter-venricular septal thickness (EDIST) and LVmass (de Simone et al., 1992), male sex, age ≥50 years (median value = aging) and age for patients born during global 1930 crisis and period of 1945 second war, increase in pulse pressure and creatinin.

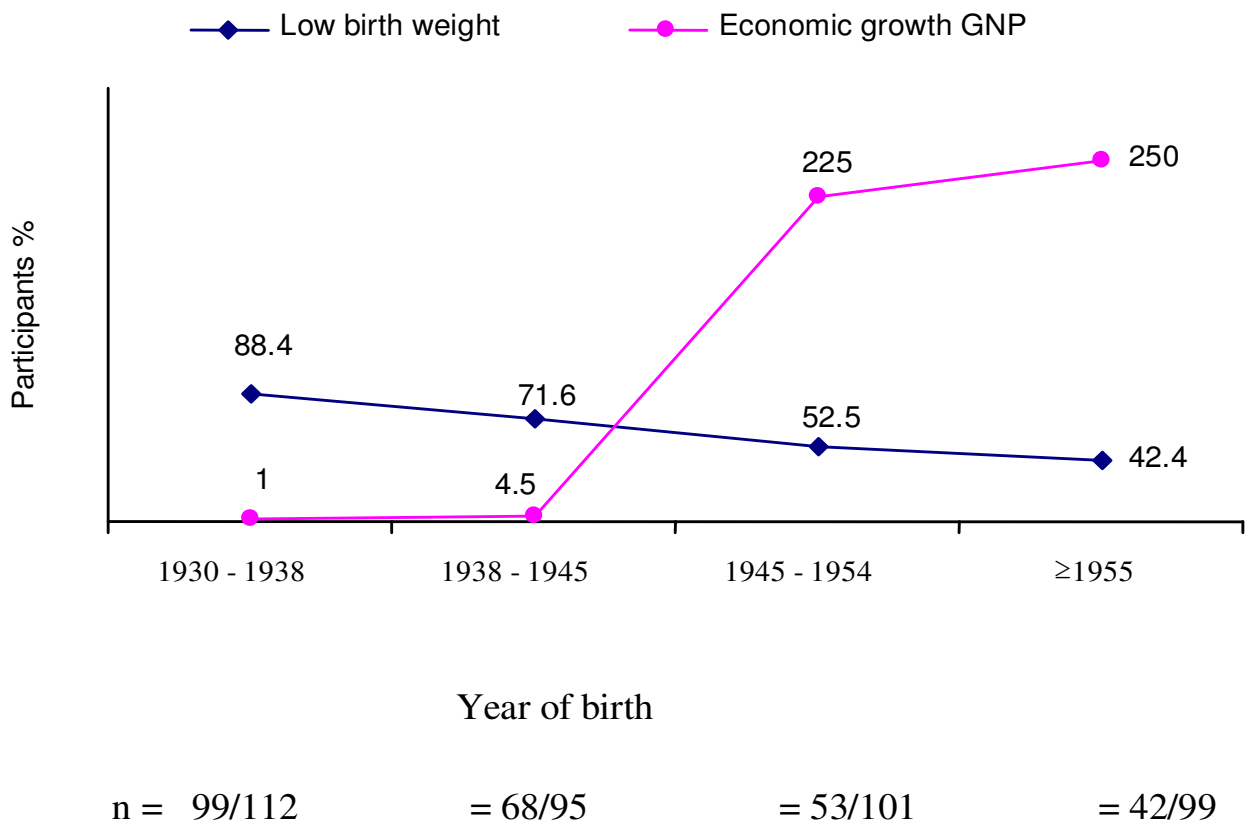

Figure 1. Distribution of participants with Low birth weight by the year of birth (P for trend <0.0001) and economic growth (GNP) in % for Congo between 1930 and 1955.

Statistical analysis

Continuous data were expressed as mean ± SD and categorical data as percentage. Continuous and categorical variables were compared using the student t-test and the Chi-square test, respectively.

Trends of proportion (%) were examined using linear-by-linear Chi-square test and trends of continuous data were examined using simple linear regression models.

Logistic regression models were used to assess the adjusted odds ratios (OR) of Mets prevalence for new and additional potential risk factors of Mets (model 1) and in considering only continuous (Model 2). All statistical tests were two sided and differences with probability values < 0.05 were considered statistically significant. All statistical analysis were performed using SPSS software for Windows (version 13.0; SPSS Inc. Chicago, Ill, USA).

RESULTS

Of 419 eligible patients, 407 (97.1%) accepted to participate. Out of the study population, aged 53.2 ± 26.9 years (range 21 - 88 years) and born with 2664 ± 728.9 g of weight, 58.7% (n = 239) were male and 41.3% (n = 168) were female. The proportions of hypertension, LBW, age ≥ 50 years, smoking, alcohol intake, T2DM, elevated urea nitrogen (UN), elevated fibrinogen, hyperuricemia, hypercholesterolemia (TC) total obesity, CHD, CHF, ischemic stroke and CKD were 54.5% (n = 222), 64.6% (n = 262), 50.9% (n = 207), 17.4% (n = 71), 18.2% (n = 74), 13.5% (n = 55), 39.3% (n = 160), 43.7% (n = 178), 36.1% (n = 147), 35.1% (n = 143), 58.2% (n = 237), 11.1% (n = 45), 21.1% (n = 86), 24.6% (n = 100), and 27.3% (n = 111), respectively.

Figure 1 shows the inverse relationship between LBW and the years of birth, whereas the Economic Growth increased exponentially with years. The probability of LBW decreased with the improvement of economic development is demonstrated by the overlapping of curves in 1945.

Among the traditional continuous parameters defining Mets, only fasting glucose (r = -0.266; P < 0.01), SBP(r = -0.199; P < 0.01) and DBP (r = 0.130; P < 0.01) were significantly and inversely correlated with birth weight, respectively. However, there was a significant and positive association between all new and additional continuous components of Mets (except for uric acid) and LBW (Table 1). There was no association between gender, CHD, ischemic stroke, CHF and LBW (results not shown). But the risk of hypertension (OR = 1.9 95% CI 1.1 - 3.6; P

Table 1. Current characteristics of 407 participants categorized by the level of their birth weight.

		With low birthweight (n = 262)	Without low birthweight (n = 145)
Current variable	**P**	**Mean ± SD**	**Mean ± SD**
Age (yrs)	<0.0001	56.9 ± 32.1	46.3 ± 9.8
SBP (mmHg)	<0.0001	170.9 ± 31.9	156.4 ± 34.3
DBP (mmHg)	0.002	98.3 ± 19.3	91.8 ± 22
Pulse pressure (mmHg)	<0.001	72.6 ± 23.2	64.6 ± 21.9
Fasting plasma glucose (mg/dl)	<0.001	104.3 ±49.8	76.7 ± 16.5
Urea Nitrogen (mg/dl)	0.036	31.8 ± 26.2	26.5 ± 19.3
Creatinin (mg/dl)	0.013	1.3 ± 0.8	1.1 ± 0.6
Uric acid (mg/dl)	0.079	5.9 ± 2.6	5.5 ± 2.4
Fifrinogen (mg/dl)	<0.0001	378.8 ± 143.9	318.9 ± 128.3
Total cholesterol (mg/dl)	<0.001	204.2 ± 58.2	185.7 ± 48.6
LViDd (mmm)	0.007	52 ± 9.3	48.9 ± 9.2

LViDd: End-diastolic LV internal dimension.

Table 2. Means of continuous variables of 407 participants categorized by the presence of metabolic syndrome (Mets) defined according to WHO criteria.

Variable	With Mets (n = 77) Mean ± SD	Without Mets (n = 330) Mean ± SD	P
Age (years)	60.1 ± 56.8	51.5 ± 11.6	0.012
BMI (Kg/m$^{2)}$	33 ± 8.1	27.2 ± 7.4	<0.0001
SBP (mmHg)	174.9 ± 32.3	163.8 ± 33.5	0.016
Pulse pressure (mmHg)	77.3 ± 21.9	68 ± 23	<0.001
Birth weight (g)	2291 ± 413	2751.1 ± 759.1	<0.0001
Fasting plasma glucose (mg/dl)	152.5 ± 66.2	80.9 ± 17.8	<0.0001
Urea Nitrogen (mg/dl)	38.7 ± 30	27.8 ± 22.1	<0.0001
Creatinin (mg/dl)	1.5 ± 0.7	1.2 ± 0.7	0.002
eGFR	72.1 ± 32.6	99.8 ± 50.3	<0.0001
Uric acid (mg/dl)	6.7 ± 2.3	5.5 ± 2.6	<0.0001
Fifrinogen (mg/dl)	434 ± 130.4	339.6 ± 137.9	<0.0001
Total cholesterol (mg/dl)	224.4 ± 69.8	191.4 ± 49.8	<0.0001
LV mass	180 ± 102	150 ± 120	0.042
LViDd (mm)	52.7 ± 9.9	50 ± 9.2	0.022
EDIST (mm)	13.1 ± 5.6	11.6 ± 5.4	0.031

LViDd: end-diastolic left ventricular (LV) internal dimension. EDIST : end-diastolic inter(ventricular septal thickness

< 0.01), T2DM (OR = 2.3 95% CI 1.6 - 3.2; P < 0.001), and CKD (OR = 2.02 95% CI 1.2 - 3.4; P < 0.01) was multiplied two times by LBW, respectively.

Overall, 18.9% (n = 77) met the criteria of Mets defined by WHO Expert Committee and had mean age similar to that of patients without Mets. Tables 2 and 3 show the univariate association between new risk factors and Mets.

There was a significant and positive dose-response relationship between fasting plasma glucose concentrations (Figure 2), LV mass levels (Figure 3) and the prevalence of Mets.

Among patients with Mets, 71.4% (n = 55) and 81.8% (n = 63) were diagnosed with T2DM and total obesity, respectively. However, 28.6% (n = 22) and 18.2% (n = 14) among these patients with Mets were neither diabetic nor obese, respectively.

Table 4 shows that after stepwise adjustments for sex, age, smoking, alcohol intake, elevated UN, hyperruricemia,

Table 3. Characteristics of 407 participants categorized by the presence of metabolic syndrome (Mets) according to WHO criteria.

Variable	With Mets (n = 77; n [%])	Without Mets (n = 330; n [%])	P
Men/Women	46/31 (59.7/40.3)	193/137 (58.5/41.5)	0.840
Hypertension	55 (71.4)	167 (50.6)	<0.001
CHD	17 (22.1)	29 (8.8)	<0.001
CHF	24 (31.2)	62 (18.8)	0.017
Ischemic stroke	30 (39)	70 (21.2)	<0.001
Low birth weight	72 (93.5)	190 (57.6)	<0.001
CKD	30 (39)	69 (20.9)	<0.0001
Hypercholesterolemia	41 (53.2)	102 (30.9)	<0.0001
Hyperuricemia	41 (53.2)	106 (32.1)	<0.001
Elevated fibrinogen	54 (70.1)	124 (37.6)	<0.0001
Elevated Urea nitrogen	45 (58.4)	115 (36.8)	<0.0001

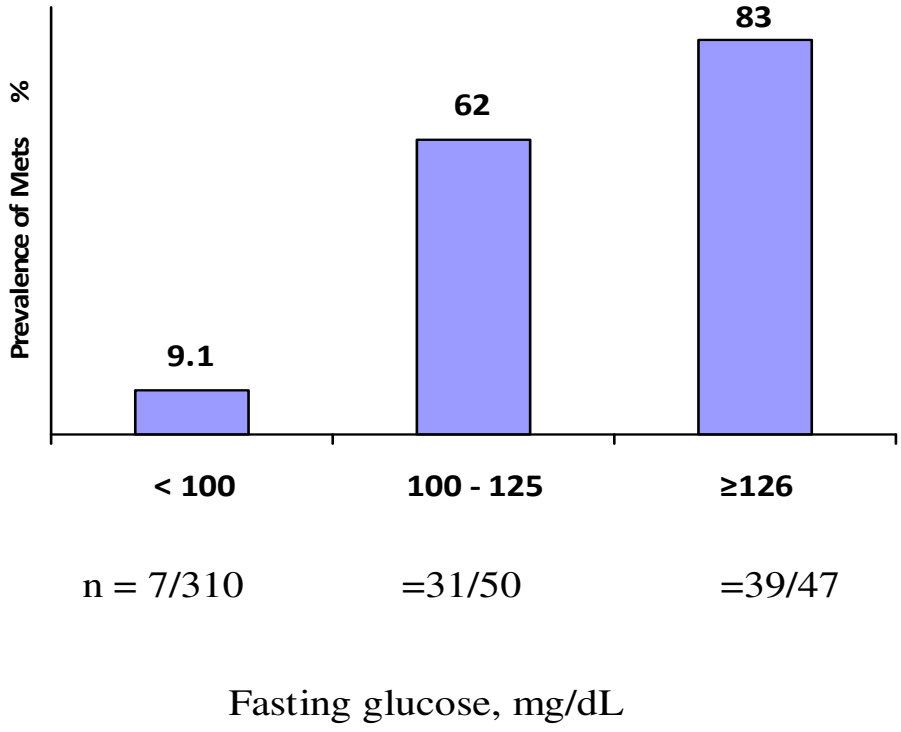

Figure 2. Relationship between the prevalence of the metabolic syndrome defined according to WHO criteria and the levels of fasting plasma glucose in 407 patients. P for trend < 0.0001.

Table 4. Multivariate regression analysis for factors potentially linked to the presence of the metabolic syndrome defined according to WHO criteria among 407 patients.

Independent Individual Variable	Beta coefficient	SE	OR (95% CI)	P
CHD, yes vs. no	0.845	0.373	2.3 (1.1 - 4.8)	0.024
Low birth weight, yes vs. no	2.294	0.482	10 (3.9 - 25.5)	<0.0001
Elevated fibrinogen, yes vs. no	1.247	0.287	3.5 (2 - 6.1)	<0.0001
Constant	- 4.078	0.500		<0.0001

CHD: coronary heart disease. SE: standard error.

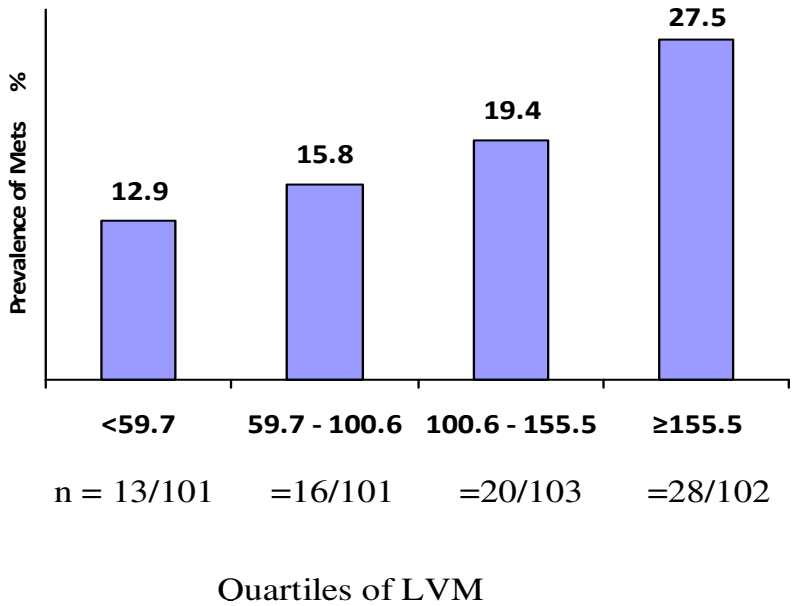

Figure 3. Relationship between the prevalence of the metabolic syndrome defined according to WHO criteria among 407 patients. P for trend = 0.007.

Table 5. Multivariate Regression Analysis for Factors Potentially Linked to the presence of the metabolic syndrome defined according to WHO criteria among 239 men.

Independent individual variable	Beta coefficient	SE	OR (95% CI)	P
Low birth weight, yes vs. no	2.020	0.549	7.5 (2.6 - 22.1)	<0.0001
Elevated fibrinogen, yes vs. no	1.244	0.378	3.5 (1.7- 7.3)	<0.0001
Constant	- 3.735	0.579		<0.0001

SE: standard error.

Table 6. Multivariate regression analysis for factors potentially linked to the presence of the metabolic syndrome defined according to WHO criteria among 168 women.

Independent individual variable	Beta coefficient	SE	OR (95% CI)	P
Low birth weight yes vs. no	2.888	1.040	18 (2.3 – 37)	0.005
Elevated urea, yes vs. no	0.968	0.468	2.6 (1.1 – 6.6)	0.038
Elevated fibrinogen, yes vs. no	1.117	0.467	3.1 (1.7 – 9.8)	0.017
Constant	- 4.874	1.059		<0.0001

SE: standard error.

ischemic stroke, CHF, CKD and LV mass, only CHD, LBW and elevated fibrinogen were significantly and independently associated with the prevalence of Mets in all patients. LBW had the highest OR for the presence of Mets when compared to ORs for Mets for CHD and increase in fibrinogen. Table 5 shows that stepwise adjustments for age, CHD. Smoking, alcohol intake, elevated UN, hyperuricemia, CKD, CHF, ischemic stroke and LV mass attenuated the OR of Mets for LBW in men. However, stepwise adjustments of age, CHD, smoking, alcohol intake, hyperuricemia, CKD, CHF, ischemic stroke, and LV mass increased two-fold the OR of Mets for LBW, attenuated the statistical strength of OR of Mets for elevated fibrinogen and favoured the entering of elevated UN as independent risk factors of Mets in women (Table 6).

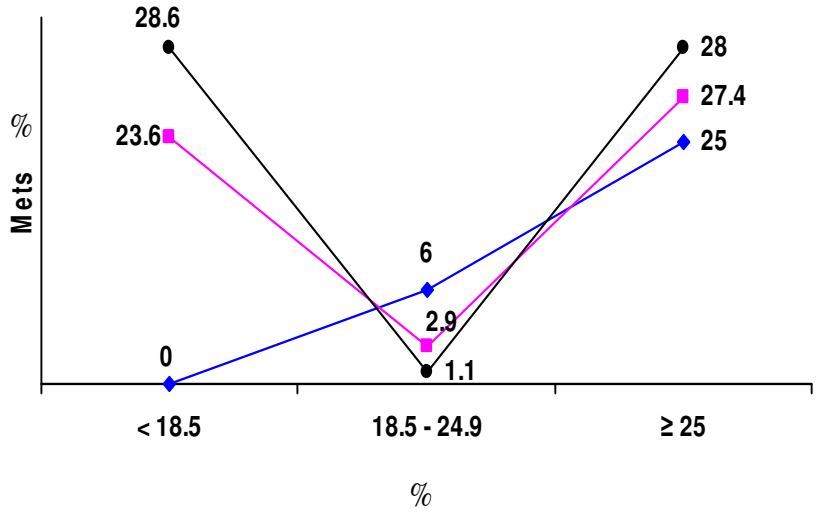

Figure 4. Relationship between the prevalence of metabolic syndrome (Mets) and the nutritional status categories in all patients (▬▬), men (●—●), and women (◆—◆).

In considering continuous variables, only fibrinogen, (OR = 1.003 95% CI 1.001 - 1.01; P < 0.001), total cholesterol (OR = 1.01 95% CI 1.002 - 1.012; P < 0.004), and birth weight (OR = 0.999 95% CI 0.998 - 0.999; P < 0.0001) were independently associated with the presence of the metabolic syndrome as follows:

-Y= - 1.01 + 0.003 fibrinogen + 0.01 TC - 0.001 BW in all patients;
-Y= 1.01 + 0.003 fibrinogen + 0.01 TC - 0.001 BW in men;
-Y= - 1.292 + 0.01 fibrinogen - 0.001 BW in women.

There was a significant (P<0.0001) and U-shaped relationship between the Mets prevalence and the adulthood nutritional status in all patients (P < 0.001) and in men (P < 0.001) but a positive linear and exponential relationship between Mets and nutritional status in women (for trend = 0.002) (Figure 4). There were 8 cases of Mets (8 men and 0 women) in under-nutrition, 4 cases of Mets (1 man and 3 women) in normal nutrition and 65 cases of Mets (37 men and 28 women) in over-nutrition groups.

DISCUSSION

The main findings of this study are as follows: i) The prevalence of LBW was very high and decreased with time as GNP increased; ii) Birth weight was negatively associated with higher cardio-metabolic and renal risk, ii) The unexpected high prevalence of Mets; iii) The independent association between CHD, LBW, high fibrinogen, high urea nitrogen and the presence of Mets; iii) The higher adjusted risk of LBW for Mets in women; iv) The highest rates of Mets in both under-nutrition and over-nutrition (total obesity) in comparison with that observed in normal nutrition status.

In this hospital-based study, the prevalence of LBW (64.5%) was similar with 63% of LBW in our previous population-based study (Longo-Mbenza et al., 1998). These rates reflecting public health and welfare pattern of a developing country were higher than the rate of 3.6% of LBW recently reported from a Brazilian population-based study (Pellanda et al., 2008). The significant influence of 1930 global crisis - 1945 Second World War (famine, low economic level) and that of economic development between 1945 and 1955 on the rates of LBW were well demonstrated in this study. Furthermore, we demonstrated that low birth weight (LBW) was significantly associated with traditional CVD risk factors (hypertension, diabetes, left ventricular hypertrophy and hypercholesterolemia) and new CVD risk factors such as CKD, Mets increase in pulse pressure, urea nitrogen, uric acid and fibrinogen. These findings are consistent with our previous study and many studies in the literature and based on the underlying link between intrauterine growth retardation and increased cardiovascular (Barker, 1998; Bavdekar et al., 1999; Mi et al., 2000; Pellanda et al., 2008; Barker et al., 1989; Barker, 1993; Kaijser et al., 2008; Balci et al., 2009; Bertrand and Levy-Marchal, 2008; Rotteveel et al., 2008).

The present study shows that these Central Africans witness a dramatic increase in Mets prevalence (18.9%) which is higher than that reported in 2007 in an adult sample of a general population in Central Africa using the same WHO definition for Mets (Fezeu et al., 2007). Individually, there was a significant positive and univariate

association between SBP, pulse pressure, urea nitrogen, creatinin, uric acid, fibrinogen, total cholesterol, LV mass, LViDd, EDIST, CHD, CHF, ischemic stroke, CKD, low weight and the estimated Mets prevalence among these patients at high CVD risk (54.5% hypertensives, 50.9% aged ≥ 50 years, 17.4% smokers). Adjusted for confounding risk factors, only low birth weight, CHD and increase in fibrinogen concentrations were identified as independent contributing risk factors of Mets in all patients. When sex was considered, low birth weight and elevated fibrinogen were the independent risk factors of Mets in men, whereas low birth weight, elevated urea nitrogen and elevated fibrinogen were the independent risk factors of Mets in women.

The sanitary transition has created double jeopardy of infectious (HIV, malaria, tuberculosis, emergent and re-emergent infections) and non communicable disease in other developing countries (Ford et al., 2002; Ghaffar et al., 2004; Kelliny et al., 2008; Misra and Khurana, 2008; Barker, 1998; Misra, 2002; Reddy, 2002). And because of nutrition transition characterized by the patterns from evolutionary biology, the relationship between nutrition transition, urbanization/industrialization, low birth weight and the emergence of Mets is complex in these Central African patients. Phase 1 nutrition transition was defined by hunter gatherer Africans before Belgian colonization in 1908 with a diet rich in fiber, carbohydrates, low in fat, lean body phenotype and high activity profile. In Phase 2 between 1930 and 1945, many Africans existed in famine-like situation by the 1930 global crisis and 1940 - 1945 War (low protein and fat diets) and having epidemic rates of LBW. In Phase 3 between 1945 and 1954, famine vanished because of economic growth and nutrition improved with increase in the consumption of vegetables, fruits and animal protein after rural-urban migration. Phase 4 was observed in 1997 - 1998 and was conducive to the emergence of Mets, CHD, hypertension, T2DM, CHF and CKD. A rapid shift from phase 3 to phase 4 (life expectancy, smoking, stress) in central Africa may be responsible for steep increase in the Mets (Misra and Khurana, 2008). It is suggested that thrifty genotype is propagated by survival natural selection during famines and leads heightened tendency to develop obesity and T2DM when food becomes abundant and available. However, thrifty genes are not yet identified. A thrifty phenotype hypothesis emphasizes low birth weight implication in the adulthood high risk of hypertension, diabetes, renal decline inflammation but not high BMI levels in this study.

Furthermore, there was a significant U-shaped relationship between BMI and Mets prevalence in all patients and in men in this study. In women from the present study, however, the Mets prevalence was not present in the undernutrition category, but increased proportionally with the increase in BMI. This is consistent with the presence of insulin resistance in undernutrition, normal nutrition and over nutrition/total obesity categories of observed in the general Congolese population (Kasim et al., 2007).

Reduced insulin sensitivity but not obesity may be the hall mark of these conditions, and might be present in early life (Bertrand and Levy-Marchal, 2008). Stimuli or insulin during critical or sensitive periods of foetal life (reduced size, reduced muscular mass, reduced number of nephrons) and early post natal conditions would permanently affect the development and function of beta-cell mass, or lead to peripheral insulin resistance with subsequent adverse consequences in adult life “programming” (Fox et al., 2004). Physiological alterations in these neuroendocrine systems may have potent effects on high risk of Mets and CVD through their influence on risk factors such as plasma glucose, fibrinogen, urea nitrogen, total cholesterol, uric acid and blood pressure.

LBW was associated with inflammatory state indicated by the elevated concentrations of fibrinogen in adulthood. This maker of acute phase reactants (elevated cortisol and fibrinogen) and endothelial activation in adult life of four US communities had been predicted by low birth weight (Pellanda et al., 2008). Thus, Mets and CVD appear as a result of innate immunity activated by environmental factors (adipokines, cytokines, monocyte/ macrophage), genetics, nutrition, physical inactivity, stress, age and the foetal ongoing programming (Low birth weight) (Balci et al., 2009).

This study showed that Mets, CKD and CVD could develop in parallel. In non-diabetic Black South Africans, Okpechi et al. (2007) and Fox et al. (2004) showed a significant association between decline-in eGFR and increasing number of Mets components. Elevated urea nitrogen was also identified as an independent determinant and an additional risk factor of Mets in this study. This is because urea nitrogen is also a metabolic maker for protein metabolism and may be linked to an excess of amino acids or animal proteins from the western diet adopted by these patients after their rural-urban migration.

As reported by Dallas Heart Study in US populations (Wassink et al., 2008), manifest atherosclerosis (CHD) was associated with Mets prevalence in these Central Africans.

The present study showed a non significant influence of gender upon the Mets prevalence in these Central Africans, contrasting with higher prevalence of Mets among women than men in the third NHANES sample from USA (Park et al., 2003). This study reveals a gender-specific correlation between LBW and Mets: higher vulnerability for Mets in women with LBW than in men.

Our observations raise major clinical and public health concerns, especially for our developing countries where there is simultaneous presence of high prevalence of low birth weight, inflammation and emerging Mets, CKD, CHD and obesity. Seasonal disparities in food availability, low birth weight, the year/period of birth and the

economic development need more in depth studies in sub-Saharan Africa. The economic cost of low birth weight and Mets-related non communicable diseases (CHD, CKD and CHF) will impact adversely on meagre health budgets in developing Central African settings. To prevent increasing morbidity due to low birth weight and Mets in these Central Africans, there is an urgent need to initiate large-scale community prevention and intervention programmes focusing on increased physical activity, quality of life (no wars, no famines) and healthier food options, particularly for pregnant women and children. The interest in the Mets should, however, not displace the use of other similar risk scoring tools from their primary place in the identification of individuals at high risk of CVD.

LIMITATIONS

Potential limitations of this study merit discussion. Being cross-sectional and observational in nature, the obtained hospital-based findings may affect the generalisability of the general populations. Residual confounding cannot be ruled out as an explanation for the results. The nutritional status of mothers in terms of malnutrition/anaemia during pregnancy and genes expression were not available. The weakness of the study may be bias and accuracy of the data of the babies who were underweight, were not selected by random sampling. The strength of the study resides in the documented birth weight to avoid individuals not knowing their birth weight and their potential misclassification. Residual confounding due to subclinical disease appears unlikely to have occurred, since the association between birth weight and chronic inflammatory state was significant. Moreover, the association between birth and Mets increased after adjustment for CHD in women. It is also important to consider that the programming of genes responsible for inflammatory and Mets conditions or endothelial activation in response to intrauterine environmental stress did not vary because of ethnically homogeneous black Bantu Central Africans examined. The same urban environment and globalization of diet were shared by the study population patients. Indeed, differences between whites and blacks in terms of inflammation and related chronic disease are well known (Schmidt et al., 1996). The difference between correlations is also suggested by the simultaneous identification of low birth weight and elevated fibrinogen levels as independent risk factors of the present Mets prevalence.

A new concept of salt sensitivity in the metabolic syndrome which was not done in this study should be mentioned (Chen et al., 2009; Yang, 2009). This may explain the concept that salt sensitivity plays an important role in the aetiology of hypertension in the blacks in Sub-Saharan Africa.

Conclusion

High prevalence of low birth weight, metabolic syndrome and cardiorenal diseases were observed in these African patients with economic development improvement and sanitary transitions. Low birth weight associated with all adulthood CVD risk factors except BMI, CHD, increase in total cholesterol and elevated fibrinogen levels were independently associated with metabolic syndrome in all patients. The U-shaped association between nutritional status and metabolic syndrome may be due to the nutrition transition.

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