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ORIGINAL ARTICLE

Comparison of lipid profiles and 10 year cardiovascular disease risk estimates between indigenous northern diabetic and non-diabetic persons in Adamawa region, Cameroon

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Dyslipidemias are possible debilitating outcomes of diabetes and important predictors of cardiovascular disease risk in diabetic patients. We carried out a cross-sectional, case control study from April to July 2014 at the Ngaoundere Regional Hospital involving 90 patients: 45 diabetics (10 type 1 and 35 type 2) and 45 non-diabetics with the aim of characterizing and comparing the lipid profile between type 1 and type 2 diabetics of northern Cameroon. Blood pressure and anthropometric measurements were obtained, and a fasting blood sample collected per patient on which blood sugar level and lipid profile were determined. Data analysis was performed using R Version 2.13.0 and the French version of Epi Info 7, with the level of significance set at 5%. Following threshold standards according to ANAES, the mean triglyceride level was significantly higher in type 2 diabetics (212.65±49.34 mg/dL) compared to type 1 diabetics (101.60±52.64 mg/dL) and controls (152.24±57.91 mg/dL) ($p<0.0014$). The mean low density lipoprotein (LDLc) was observed to be significantly higher in type 1 (136.78± 33.88 mg/dL) compared to type 2 (113.29 ± 38.00 mg/dL) diabetics and controls (94.62 ± 51.31 mg/dL) ($P = 0.017$), and likewise the median high density lipoprotein (HDLc) of type I, type II, and non-diabetics corresponding to 11.5, 47 and 56 mg/dl respectively were significantly different ($p<0.0001$). Furthermore, mixed hyperlipidemia was absent among the type 1 diabetics but more prevalent in type 2 diabetics (37.1 %) compared to controls (6.7%). According to the D'Agostino (Framingham) model, 37.1% of type II diabetics and 6.8 % of non- diabetics had a high risk of developing cardiovascular disease in 10 years, and the difference here was statistically significant ($p=0.001$). Both type 1 and type 2 diabetics originating from northern Cameroon are very prone to dyslipidemias and thus highly predisposed to cardiovascular diseases.

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INTRODUCTION

Diabetes defined by the American Diabetes Association (ADA) as a fasting blood sugar level >126 mg/dl on two consecutive measurements (Blasco *et al.*, 2014), is a metabolic disease characterized by a chronic hyperglycemia and could be attributed either to an impaired insulin secretion or function or both (Fagot-Campagna *et al.*, 2010). World Health Organization (WHO) estimates a continuous rise in the worldwide prevalence of diabetes, recording a 7-fold increase within a 20 year period and a burden purported to reach 366 million cases on the minimum by 2030 (Wild *et al.*, 2000), with an impoverished country like Cameroon recording a 6.1% prevalence in 2000 (Mbanya *et al.*, 2010). These increasing trends in the prevalence of diabetes are most probably the result of nutritional transitions, urbanization, and technological emergence which favor a sedentary lifestyle (Touze *et al.*, 2011), with approximately 5–10% of the total healthcare budget in most countries allocated for the management

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of diabetics (IDF, 2010).

It is estimated that 6% of the total mortality in sub-Saharan Africa is attributed to diabetes (Wild *et al*., 2000) and over 80% of diabetes related mortality occurs in developing countries (WHO, 2014), with cardiovascular diseases (CVDs) recorded to be responsible for most of these morbidity and mortality cases in several studies (Laing *et al*., 2003; Kemp *et al*., 2005; Orasanu and Plutzky, 2009). Although the mechanism of occurrence of dyslipidemias, progression of atherosclerosis and increased cardiovascular disease risk in diabetes is not well elaborated, it has been suggested that in addition to epigenetically favoured gene-environment interactions, the underlying interaction between hyperglycemia and intracellular metabolic changes may encourage oxidative stress, low-grade inflammation and endothelial dysfunction (Matheus *et al*., 2013). The worldwide prevalence of diabetes has been projected to increase especially in sub-Saharan Africa. This study was aimed at evaluating long-term cardiovascular disease risk among diabetic patients in our setting, thus contributing relevant efforts towards curbing the incidence, morbidity and mortality due to CVDs among diabetic persons.

MATERIALS AND METHODS

Study Population and Sample Collection

In this cross sectional case control study spanning an 8 month period from April to October 2014, ninety indigenous north Cameroon participants were enrolled composed of forty-five diabetic patients and forty-five non-diabetic controls, and having resided in the Adamawa region for at least five years. Participants were recruited at the Hypertension and Diabetes Unit of the Ngaoundere Regional Hospital, which represents the main referral center in the region. After having obtained information about the aim, merits, demerits, and consenting to the study, participants were then assisted in filling out a structural questionnaire wherein information on age, gender, profession, ethnicity, medical history, alcohol and tobacco consumption were recorded, and with communication predominantly in the French and Fulfudé (traditional) languages.

The study obtained administrative and ethical clearance from the ethical committee of the Adamawa regional delegation of public health (1229/L/RC/RA/DSP/HR/NGD/CLE). After obtaining an informed consent per participant, blood pressure, weight, height and waist circumference were measured and the body mass index calculated. 5ml of venous blood was collected in sitting position after an 8 to 12 hour overnight fast and blood sugar measured immediately. The remaining part of the blood sample was left to cloth in a dry tube for 10 minutes on the bench top at room temperature, centrifuged at 3000 rpm for 5 minutes and the serum obtained stored in ependorff tubes at -20°C prior to weekly batch analyses. Total cholesterol, triglyceride, and HDL cholesterol were measured and LDL cholesterol calculated using the Friedwald equation. All measurements were performed in accordance with standard operating procedures. Pregnant women, persons on drugs including corticosteroids, β-adrenergic blockers, diuretics estrogen and other hormonal therapy, and HIV/AIDS patients were excluded from the study.

Measurement of Arterial Blood Pressure

Blood pressure measurements were performed following the STEPs procedures elaborated by WHO STEPwise surveillance approach (WHO, 2005). Using a mercury sphygmomanometer (ADC Prosphygmodele 770), in sitting position and following a 10 – 15 minute rest period, measurements were done on both arms (right and left) per patient separated by a 2-3 minute time interval and the averages calculated and recorded. These measurements were controlled for coherence using an electronic blood pressure meter (Omron HEM712C, 6416799LF, China). Values ≥ 140/90 mmHg were considered elevated.

Measurement of Biochemical Markers

Fresh whole blood samples were used for blood glucose level determination using a glucometer prior to clotting. The frozen serum samples were thawed and used to perform total cholesterol, triglyceride and HDL cholesterol measurements, using a spectrophotometer (Mindray BA-88A, BH7AB2710, China) and results were explored fol-

lowing ANAES (Agence Nationale d'Accréditation et d'Evaluation de la Santé) reference values (Chevalier *et al*., 2005). Prior to analysis, calibration was performed using the Calimat calibrator (reference: 62321, lot number: 1003001280 Biomérieux, France) while analytical quality was controlled using the Lyotrol™ N/P quality control serum (reference: 62373, lot number: 1003001280 Biomérieux, France). All biochemical measurements were performed using commercially acquired kits.

Total Cholesterol Measurement

The determination of total cholesterol levels was based on the CHOD-PAP enzyme colorimetric method described by Meiattini (1978), Tietz (1995) and Burtis (1999). Reference values were stated as low risk: < 200 mg/dL, moderate risk: 200-239 mg/dL and high risk: >240 mg/dL.

Triglyceride Measurement

The determination of triglyceride levels was based on the GPO-PAP enzyme colorimetric method which involves lipase catalyzed hydrolysis (Tietz, 1995; Burtis, 1999). The reference values were considered as, males: 40-160 mg/dL and females: 35-135 mg/dL. Moderate and high CVD risk levels were defined at 150 – 200mg/dl and >200.

HDLc Measurement

HDL concentrations were measured using a method comprising 2 phases: first the separation and precipitation of LDL and VLDL lipoprotein fractions mediated by a polysaccharide sulphate in the presence of divalent cations, followed by cholesterol determination in the supernatant containing HDLc fractions. The CVD risk levels were defined as Low: males >40 mg/dl and females > 50 mg/dl; Moderate: males 35 – 49 mg/dl and females 45-49 mg/dl; High: males < 35 mg/dL and females <45 mg/dL.

LDLc Estimation

LDLc was calculated using the Friedwald's formula for all corresponding triglyceride levels <400 mg/dL (Friedwald *et al*., 1972) which states: LDLc (mg/dL) = TC - TG/5 – HDL, where TC=total cholesterol and TG=triglycerides. CVD risk levels were defined as Low: <130 mg/dL, Moderate: 130 – 160 mg/dL, and High: >160 mg/dL.

Mixed dyslipidemia

Participants with abnormal lipid levels in at least two of the following lipid categories were considered as having a mixed dyslipidemia. Triglycerides > 150 mg/dl, LDLc >130 mg/dl and HDLc < 35 mg/dl.

Estimation of 10 year Cardiovascular Disease (CVD) Risk

The corresponding CVD risk levels within a 10 year period were determined using the D'Agostino (D'Agostino *et al*., 2008) and SCORE (Brotons *et al*., 2014) models for all participants older than 20 years old. These calculations were performed on the www.cardiorisk.fr website and risk levels were explored accordingly: D'Agostino: low risk < 10%, intermediate risk 10-20%, high risk >20% and SCORE reduced risk < 5%, high risk >5%.

Ethical Considerations

The decision to voluntarily participate and quit this study at any time was a right fully reserved for and exercised by participants without any repercussions. Every potential candidate prior to recruitment was furnished with adequate information on this study and duly consented in writing to participate in the study. Minors were obligatorily represented by at least one of their parents or guardian. All information obtained from participants was handled confidentially and participants noted to have results indicative of a possible dyslipidemia were referred for cardiological assessment.

Statistical Analysis

Data were analyzed using both the French version of Epi info 7 and R version 2.13.0., and the results were presented as mean ± standard deviation or as Median. Differences in prevalence were explored using the Fischer's exact test and group means were compared using ANOVA or Kruskal Wallis tests, with Post Hoc Tukey for multiple comparisons. Significant differences were considered at $P<0.05$.

RESULTS

A total of 90 participants were enrolled into this study ranging in age from 10 to 78 years old and

composed of 52 females and 38 males (sex ratio 1.37:1). Study participants were recruited into two study groups and one control group being type I diabetics (10), type II (35), and non-diabetics (45) respectively, with female predominating both among type II diabetics and control group contrary to the type I diabetics. The mean age of the study population was 47.16 ± 16.92 years, with the mean ages of the type I, type II and control groups being 18.10 ± 4.55, 54.65 ± 11.23, and 47.78 ± 15.52 years respectively, and demonstrating a significant difference (P=0.001).

The prevalence of hypercholesterolemia among type I, type II and non-diabetics recorded 30%, 28.6%, 22.2% respectively and recorded a significant difference (P=0.041). While hypertriglyceridemia was prevalent at 20%, 15.6%, and mixed dys- lipidemias 37.1% and 6.7% among type II diabetics and non-diabetics respectively, no cases were recorded among type I diabetics in these categories. Furthermore, significant differences were observed between the diabetic and non-diabetic groups following the comparison of means of fasting blood sugar, triglycerides, HDL, LDL, systolic and diastolic blood pressures (Table 1).

A significant proportion of type II diabetics (37.1%) were noted to be at high risk of developing a cardiovascular disease within 10 years compared to the controls (6.8%) with respect to the D'Agostino model (Table 2). Moreover, the mean SCORE (P=0.004) and mean log of D'Agostino (P<0.0001) estimates between type II diabetics and controls were significantly different when compared.

Table 2: Distribution of Type II diabetics and controls by CVD risk levels

Risk Level	Type II (%)	Control (%)	P-value
Low[a]	34.3	70.5	0.001
Intermediate[a]	28.6	22.7	
High[a]	37.1	6.8	
< 5%[b]	74.3	81.8	0.580
>5%[b]	25.7	18.2	

[a]D'Agostino model; [b]SCORE model

DISCUSSION

The rising prevalence in diabetes is attributed to a simultaneous increase in the incidence of both type I and type II with a greater increment observed in the type II class (Matheus *et al*., 2013). CVDs make up a larger proportion of the disease burden among diabetic persons, with type II diabetics (without history of myocardial infarction) purported to be equally predisposed to coronary artery disease (CAD) as non-diabetic persons with a history of myocardial infarction (Laing *et al*., 2003; Kemp *et al*., 2005; Orasanu *et al*., 2009).

In this study, it was observed that there was a significantly high prevalence of hypercholesterolemia among type I diabetics (30%), and type II (28.6%), compared to non-diabetics (22.2%) (P=0.041).

Table 1: Clinical and biochemical characteristics of study population

Variables	Type I	Type II	Control	P-value
Age (years)	18.10 ± 4.55	54.65 ± 11.23	47.78 ± 15.52	0.001
SBP	111 ± 14.49	138.57 ± 21.71	125.78 ± 23.79	0.001
DBP	67 ± 11.60	76.86 ± 20.40	73.47 ± 23.87	0.039
FBS	127.5	162.0	88.0	0.001
TC	166.30 ± 35.60	188.85 ± 37.92	183.11±47.78	0.350
TG	81.60 ±52.64	158.37 ± 49.34	152.24 ± 57.91	0.001
HDL	11.5	47.0	56.0	0.000
LDL	136.78 ± 33.88	113.29 ± 38.00	94.62 ± 51.31	0. 017

SBP=systolic blood pressure, mmHg; DBP=diastolic blood pressure, mmHg; FBS=fasting blood sugar, mg/dL; TC= total cholesterol, mg/dL; TG=triglyceride, mg/dL; HDL=high density lipoprotein, mg/dL; LDL=low density lipoprotein, mg/dL

Conversely, the difference between the mean cholesterol levels of these three groups was not significant. This contradicts the results of Sapna and Alok (2008) where they recorded significantly higher mean cholesterol levels among diabetics compared to non-diabetics controls. While no case was recorded among type I diabetics, 20% of type II diabetics compared to 15.6% of non-diabetic controls were hypertriglyceridemic, this being lower than the 64.1% obtained by Yadav and colleagues (2014). Moreover the mean triglyceride level was significantly highest among type II diabetics (P=0.0014). Comparing the median HDLc levels, the type I diabetes group portrayed significantly lowest value (P<0001). This differs from the results of Fendi and coworkers (2010), who observed lowest mean HDLc levels among type II diabetics.

Interestingly, we obtained significantly high mean LDLc levels among type I diabetics compared to the type II and non-diabetic controls (P=0.017). This is in line with several studies which recorded similar results and suggesting that LDL cholesterol represents the principal cardiovascular risk factor in the management of dyslipidemias among type I diabetics (Aqeella and Javaid, 2008; Dionadji *et al*., 2011). Also we recorded the prevalence of mixed dyslipidemias at 37.1% and 6.7% among type II diabetics and non-diabetics respectively. Higher rates were reported by Oguejiofor and colleagues (2012) and likewise by Dionadji and colleagues (2011). Diabetes, especially type II potentiates CVD risk following increase in very low density lipoprotein (VLDL), triglycerides and LDL levels, while reducing HDL levels. These lipoproteins, when subjected to possible chemical modifications such as oxidation and glycosylation, result in a decrease in vascular compliance thus predisposing patients to an acute type atherosclerosis. This mechanism may alone be insufficient in type I diabetic patients as atherosclerotic progression could be slowed under conditions of intense insulin-based diabetic control (Matheus *et al*., 2013; Renard *et al*., 2004).

Comparing the 10 year CVD risk between type II diabetics and non-diabetics at high risk, a significant difference was observed with the D'Agostino model while the SCORE model failed to establish a significant difference. This may be due to the fact that the SCORE model does not consider diabetic status in its calculations while the D'Agostino model multiplies the risk by an estimated factor of 2 for diabetics.

CONCLUSION

Our study coheres with previous studies in demonstrating and increased prevalence of hyperlipidemias, dyslipidemias, and high 10 year CVD risk among diabetics compared to non-diabetic controls, thus the need for adequate patient-centred management and follow-up using both hospital-based and public health methods.

LIMITATIONS

This study was a cross sectional thus limiting the ability to make strong inferences on serum lipoprotein variations in diabetes, moreover, measurements were performed only ones giving room for a possible white coat effect. Furthermore, the non-verification of the initial diabetes diagnosis and classification diabetic patients may have led us to wrongly classify some patients on the basis of their medical history. Since age ≥ 20 years is a prerequisite in estimating 10year CVD risk scores using the d'Agostino and SCORE models, the unavailability of older type I diabetics during our study period rendered it impossible to estimate their CVD risk scores. Lastly, our sample size was a limitation to the strength of data analyses.

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COMPETING INTERESTS

The authors declare that they have no competing interests.

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