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Dyslipidemia and Associated Cardiovascular Risk Factors Among Non-Diabetic Taxi-Motorbike Drivers Working in Cotonou, Benin

Patrice Hodonou Avogbe* and Ambaliou Sanni

Department of Biochemistry and Cellular Biology, Faculty of Sciences and Techniques, University of Abomey-Calavi, Republic of Benin

***Corresponding author:** Patrice Hodonou AVOGBE, Department of Biochemistry and Cellular Biology, Faculty of Sciences and Techniques, University of Abomey-Calavi, Republic of Benin

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Abbreviations: CVDs: Cardiovascular Diseases; ALP: Alkaline Phosphatase; ALT: Alanine Aminotransferase; AST: Aspartate Aminotransferase; hs-CRP: High Sensitivity C-Reactive Protein; TC: Total Cholesterol; TG: Triglycerides; LDL-C: Low-Density Lipoprotein Cholesterol

ABSTRACT

Background: Dyslipidemia or abnormal lipid level is a major risk factor for Cardiovascular Diseases (CVDs). However, epidemiologic features and influencing factors of dyslipidemia (e.g., liver enzymes) have not been reliably quantified in high-risk populations in Benin. We identified cardiovascular risk factors (CVRFs) associated with dyslipidemia, its prevalence and patterns in taxi-motorbike drivers (TMDs) of Cotonou.

Methods: We conducted a cross-sectional study among 134 TMDs aged 22.0–59.0 years. Fasting glucose, insulin, Alkaline Phosphatase (ALP), Alanine Aminotransferase (ALT), and aspartate aminotransferase (AST), high sensitivity C-reactive protein (hs-CRP), and blood lipids were measured. Dyslipidemia was defined as any or a combination of the following: increased total cholesterol (TC > 5.2 mmol/L), low-density lipoprotein cholesterol (LDL-C > 3.4 mmol/L), triglycerides (TG > 1.7 mmol/L), and reduced high-density lipoprotein cholesterol (HDL-C < 0.9 mmol/L). Predictors of dyslipidemia were analyzed using logistic regression.

Results: The overall prevalence of dyslipidemia was 29.1% (95% CI: 21.4–36.8%). The prevalence of elevated TC, TG, LDL-C, and low HDL-C were 17.2%, 3.7%, 14.2%, 9.0%, respectively. Mixed dyslipidemia was identified in 14.2% of participants. Dyslipidemic forms ranged between 3.2 to 20.6% in hypertensive and between 5.0–21.2% in insulin resistant patients. Logistic regression showed that systolic blood pressure (OR= 1.05, 95% CI: 1.01–1.10, P= 0.015), ALT (OR= 1.10, 95%CI: 1.01-1.20, P= 0.016), and hs-CRP (OR= 1.12, 95% CI: 1.01-1.23, P= 0.027) were independently associated with increased risk of dyslipidemia, whereas alcohol consumption was related to a lower dyslipidemia risk (OR= 0.37, 95% CI: 0.14-0.97, P= 0.047).

Conclusion: The high prevalence of dyslipidemia, which co-occurs with CVRFs such as hypertension, ALT, and hs-CRP in TMDs of Cotonou should raise concern. These markers may help prevent dyslipidemia and associated CVDs within Benin.

Introduction

Cardiovascular diseases (CVDs) remain the greatest cause of mortality worldwide [1]. According to the latest statistics from the World Health Organization, CVDs account for 32.0% of all deaths and 17.9 million deaths in 2019 [2]. A significant proportion of the world's death from CVDs occurred among younger adults in low- and middle-income countries [3]. The pathogenesis of CVDs is multifactorial, involving several Cardiovascular Risk Factors (CVRF) such as dyslipidemia, which is a prominent contributor to CVDs globally and even in Africa [4]. Dyslipidemia is characterized by increased blood levels of Total Cholesterol (TC), Triglycerides (TG), Low-Density Lipoprotein Cholesterol (LDL-C), and by decreased high-density lipoprotein cholesterol (HDL-C) concentrations, occurring singly or in combinations [4]. Dyslipidemia alone or in combination with other known drivers such as hypertension, diabetes, and insulin resistance (IR), contribute to the development of CVDs, leading to increased morbidity and mortality [5,6]. Dyslipidemia can also cause severe diseases in other organ systems, including Non-Alcoholic Fatty Liver Disease (NAFLD) and acute pancreatitis [7]. Moreover, dyslipidemia can induce inflammation, increased production of cytokines, and C-Reactive Protein (CRP) [8]. The liver is the major organ in lipid metabolism, leading the synthesis of new fatty acids, their export and subsequent redistribution to other tissues [9].

In clinical practice, measurement of concentrations of liver enzymes such as Alanine Aminotransferase (ALT), Aspartate Aminotransferase (AST), Alkaline Phosphatase (ALP) and γ-glutamyltransferase (GGT) are commonly used biomarkers of liver dysfunction [10]. Epidemiologic studies have revealed that increased liver enzymes are major risk factors for hypertension and CVDs [11], CVDs-related mortality [12], and liver disease mortality [13]. Within Benin, an important rise in the prevalence of Non-Communicable Diseases (NCDs) and associated risk factors has been documented over the last decade [14-22]. For example, in Cotonou the prevalence of low HDL-C has increased from 10.0 % in 2007 to 21.1% in 2011 [16,17]. These data suggest that without adequate interventions measures dyslipidemia will continue to rise, thereby aggravating the burden of CVDs. Therefore, identification of CVRFs associated with dyslipidemia are of critical importance for prevention and management of CVDs. However, there are currently few data on epidemiologic features and influencing factors of dyslipidemia within Benin. In particular, the associations of dyslipidemia with liver enzymes have never been explored in high-risk populations such as the Taxi-Motorbike Drivers (TMDs) exposed to high-level of ultrafine particles [22,23]. We sought to determine the prevalence and patterns of dyslipidemia, and its relationships with CVRFs such as CRP, ALP, ALT, and AST in TMDs. We anticipated that dyslipidemia associates with liver dysfunction and tested this hypothesis in a cross-sectional study.

Materials and Methods

Study Design and Study Participants

We conducted several surveys (between 2004–2018) to investigate the health impacts of air pollution on exposed populations, including TMDs. TMDs were offered several health checkups, which included assessment of cardiometabolic markers. This was a retrospective cross-sectional study that analyzed data obtained in our 2009 survey. The study population has been described previously [22,23]. Briefly, demographic and clinical information such as age, alcohol intake, height and weight, systolic (SBP) and diastolic (DBP) blood pressure were obtained from each participant through face-to-face interviews by trained doctors.

Participants Fulfilling the Following Criteria were Included in the Study

male non-smokers without diabetes or CVDs, age ≥ 20 years, and having measurements of fasting glucose (< 7.0 mmol/L), insulin, lipids, ALP, ALT, AST. Patients missing any of these biochemical markers were excluded. A total of 147 TMDs were assessed for eligibility but 134 met predetermined criteria and were included in the analyses reported in this paper. The study was evaluated and approved by the Benin Environmental Agency. Written informed consent was obtained from each participant prior to enrolment in the study.

Blood Collection and Laboratory Testing

Venous blood (5 ml, EDTA-containing tubes) from fasten participants was collected and processed within two hours in our laboratory, in Cotonou. Aliquots of plasma (1 ml) were transported on dry ice to Nancy, where they were stored at – 20ºC until analyzed. Fasting glucose, ALP, ALT, AST, high sensitivity C-reactive protein (hs-CRP), and blood lipids (TC, TG, LDL-C, HDL-C) were measured on a clinical chemistry analyzer (Siemens, Germany). Insulin was determined by radioimmunoassay (Biorad, France). All biological analyses were performed by standardized methods within the research Unit NGERE, Faculté de Médecine, Nancy, France.

Definitions of Variables

Hypertension was defined as SBP ≥ 140 mmHg or DBP ≥ 90 mmHg [24] Alcohol intake was defined as the average consumption of 1 or more alcoholic drinks per day. The homeostatic model assessment-insulin resistance (HOMA-IR) was calculated using the formula described by Matthews, et al. [25]. IR was defined as the 75th percentile of HOMA-IR value [25]. Elevated liver enzymes were

defined as follows: ALP level >129 UI/L [26], ALT level > 45 UI/L, and AST level >35 UI/L [27]. Dyslipidemia was defined according to the American Heart Association classification, corresponding to any or combinations of the following: TC > 5.2 mmol/L, LDL-C > 3.4 mmol/L, TG > 1.7 mmol/L, and HDL-C < 0.9 mmol/L [28]. The prevalence of dyslipidemia was defined as the proportion of study participants meeting the criteria of dyslipidemia. Mixed dyslipidemia was defined as the presence of ≥2 lipid abnormalities.

Statistical Analysis

Data are expressed as percentage for categorical variables and as median values (interquartile ranges, IQRs 25th–75th) or as mean (± standard deviation) for continuous variables. The student's t test and chi-square test were used to compare differences between subject groups. Factors associated with dyslipidemia were identified by logistic regression analysis. Results were expressed as adjusted Odds Ratios (ORs) with the corresponding 95% confidence intervals (CIs). P–values < 0.05 were considered to indicate a statistical significance. Data analysis was performed using IBM SPSS Statistics 20.0 software.

Results

Clinicodemographic Characteristics, Prevalence and Patterns of Dyslipidemia

Table 1 presents demographic characteristics, CVRFs, and the prevalence of CVRFs. The median age was 39.0 years. Alcohol intake and hypertension were prevalent in 38.1% and 47.0% of participants. The prevalence of elevated ALT and AST were 0.7% and 24.6%, respectively. Overall, the prevalence of dyslipidemia was 29.1% (95% CI: 21.4–36.8%). Among individual lipid abnormalities, the highest prevalence was for elevated TC (17.2%), followed by increased LDL-C (14.2%), low HDL-C (9.0%), and elevated TG (3.7%). Mixed dyslipidemia was present in 19/134 (14.2%), of which 17/134 (12.7%) had TC plus LDL-C (Table 1). As depicted on Figures 1A & 1B, estimates of individual lipid abnormalities were higher in hypertensive (3.2–20.6%) and in IR (5.0–21.2%) than in normotensive (1.4–14.1%) and non-IR and (0.0–15.8%).

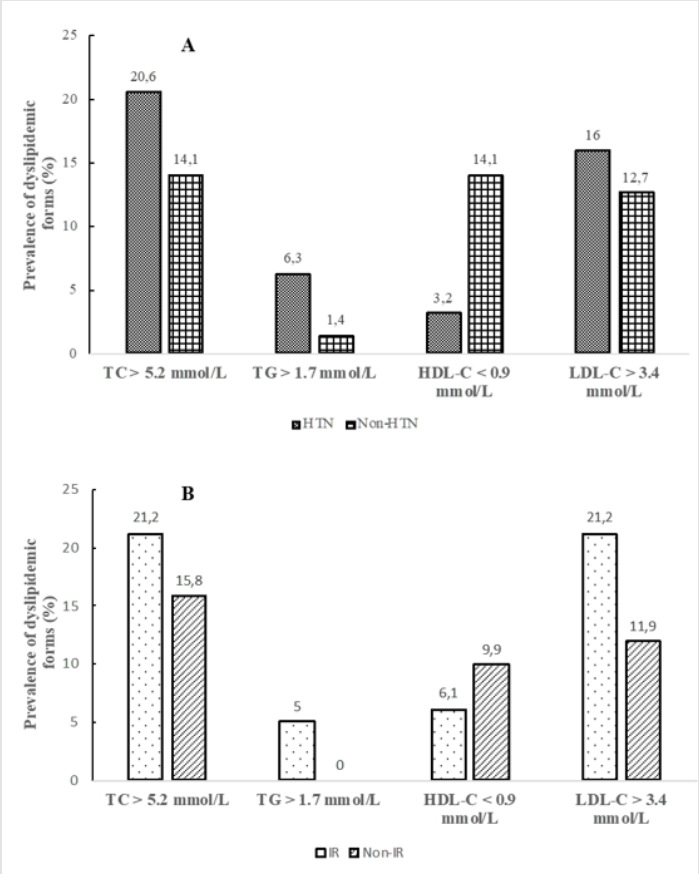

Figure 1: Prevalence of individual lipid abnormalities according to hypertension and insulin resistance statuses.

A. Prevalence of individual lipid abnormalities according to blood pressure statuses

B. Prevalence of individual lipid abnormalities according to insulin resistance statuses.

Note: IR: Insulin Resistance; HTN: Hypertension, HDL-C: High-Density Lipoprotein Cholesterol; LDL-C: Low-Density Lipoprotein Cholesterol; TC: Total Cholesterol; TG: Triglycerides.

Table 1: Demographic and biological characteristics and prevalence of cardiovascular risk factors in the study population.

Variables	Study population (n = 134)
Age (years)	39.0 (34.0-44.0)
BMI (Kg/m^2)	22.6 (20.7-25.7)
SBP (mmHg)	130.0 (120.0-150.0)
DBP (mmHg)	80.0 (80.0-90.0)
hs-CRP (mg/L)	1.7 (0.6-3.6)
Insulin (µUI/mL)	19.6 (14.2-32.5)
HOMA-IR	3.6 (2.6-6.1)
Glucose (mmol/L)	4.2 (3.8-4-5)
ALP (UI/L)	63.0 (51.0-78.0)
Elevated ALP, n (%)	0 (0.0)
ALT (UI/L)	12.0 (9.0-15.0)
Elevated ALT, n (%)	1 (0.7)
AST (UI/L)	29.0 (26.0-35.3)
Elevated AST, n (%)	33 (24.6)
TC (mM)	4.4 (3.6-4.6)
TG (mM)	0.7 (0.5-1.0)
HDL-C (mM)	1.3 (1.1-1.6)
LDL-C (mM)	2.5 (2.0-3.1)
Individual lipid abnormalities	
TC>5.2 mmol/L, n (%)	23 (17.2)
TG>1.7 mmol/L, n (%)	5 (3.7)
LDL-C> 3.4 mmol/L, n (%)	19 (14.2)
HDL-C>0.9 mmol/L, n (%)	12 (9.0)
Overall prevalence of dyslipidemia, n (%)	39 (29.1)
Prevalence of mixed dyslipidemia*	
TC + LDL-C, n (%)	17 (12.7)
TC + TG, n (%)	2 (1.5)
Alcohol use, n (%)	51 (38.1)
Hypertension, n (%)	63 (47.0)

Note: Data are presented as median (interquartile range, IQR) or n (%).

ALP: Alkaline Phosphatase; ALT: Alanine Aminotransferase; AST: Aspartate Aminotransferase; BMI: Body Index Mass; hs-CRP: high sensitivity C-Reactive Protein; DBP: Diastolic Blood Pressure; HDL-C: High-Density Lipoprotein Cholesterol; HOMA-IR: Homeostatic Model Assessment-Insulin Resistance; LDL-C: Low-Density Lipoprotein Cholesterol; SBP: Systolic Blood Pressure; TC: Total Cholesterol; TG: Triglycerides.

*Mixed dyslipidemia was defined as the presence of ≥2 lipid abnormalities.

Factors Associated with Dyslipidemia

As indicated in Table 2, binary logistic regression showed that hypertension (OR= 1.05, 95% CI: 1.01–1.10, P= 0.015), ALT (OR= 1.10, 95%CI: 1.01-1.20, P= 0.016), and hs-CRP (OR= 1.12, 95% CI: 1.01-1.23, P= 0.027) were independently associated with an increased risk of dyslipidemia, whereas alcohol consumption decreased dyslipidemia risk (OR= 0.37, 95% CI: 0.14-0.97, P= 0.047). Age, BMI, ALP, and AST had no significant influence on dyslipidemia.

Table 2: Cardiovascular risk factors associated with dyslipidemia.

Variables	Univariate Analysis			Multivariate Analysis*		
	Dyslipidemia Absent (n=95)	Dyslipidemia Present (n=39)	P-value	OR	95% CI	P-value
Age (years), mean (SD)	39.1 (7.9)	39.9 (7.4)	0.584	–	–	–
BMI (Kg/m^2), mean (SD)	23.1 (3.6)	24.7 (4.3)	0.02	1.1	0.95–1.20	0.287
SBP (mmHg), mean (SD)	132.3 (17.6)	138.7 (20.9)	0.074	1.05	1.01–1.10	0.015
DBP (mmHg), mean (SD)	84.4 (12.9)	85.5 (14.0)	0.669	0.95	0.90–1.01	0.078
hs-CRP (mg/L), mean (SD)	2.4 (3.5)	5.4 (7.6)	0.028	1.12	1.01–1.23	0.027
HOMA-IR, mean (SD)	5.2 (4.9)	5.0 (4.0)	0.832	–	–	–
Glucose (mmol/L), mean (SD)	4.2 (0.7)	4.3 (0.6)	0.296	–	–	–
ALP (UI/L), mean (SD)	66.4 (20.3)	66.9 (21.4)	0.894	–	–	–
ALT (UI/L), mean (SD)	11.8 (4.5)	15.1 (8.5)	0.028	1.1	1.01–1.20	0.016
AST (UI/L), mean (SD)	31.9 (10.0)	37.0 (21.5)	0.161	–	–	–
Alcohol intake, n (%)	39 (76.5)	12 (23.5)	0.265	0.37	0.14–0.97	0.047
Insulin resistance, n (%)	24 (72.7)	9 (27.3)	0.79	–	–	–

Note: ALP: Alkaline Phosphatase; ALT: Alanine Aminotransferase; AST: Aspartate Aminotransferase; BMI: Body Index Mass; hs-CRP: high sensitivity C-Reactive Protein; DBP: Diastolic Blood Pressure; HDL-C: High-Density Lipoprotein Cholesterol; HOMA-IR: Homeostatic Model Assessment-Insulin Resistance; SBP: Systolic Blood Pressure; CI: Confidence Interval; OR: Odd Ratio

*Dyslipidemia was considered as dependent variable and other clinicodemographic parameters were set as independent variables.

Discussion

This is the first study to examine the prevalence and associated CVRFs of dyslipidemia in TMDs. Our results showed that the overall prevalence of dyslipidemia was 29.1% and that individual lipid abnormalities ranged between 3.7–17.2%, similarly, to reported estimates in other countries [29,30]. For example, Xi et al. reported an overall prevalence of 31.2% in China [31]. Estimates of lipid abnormalities ranged between 7.6%–29.5% in Nigerian [30]. Moreover, in Africa, the pooled prevalence in the general population from population-based studies was 23.6% [4]. However, the prevalence of dyslipidemia in TMDs was lower than estimates reported in Togo, 60.3% [32], India, 50.7% [33], and Iran, 51.8% [34]. This discrepancy could be ascribed to differences in studied populations, sample size, socio-economic status, lifestyles [34], and the cutoff used for dyslipidemia [4]. Within Benin, the majority of previous studies assessed the prevalence of individual lipid abnormalities, but not the overall prevalence of dyslipidemia. In an earlier study by Sodjinou, et al. [16], estimates of elevated TG and low HDL-C were 3.0% and 10.0%, respectively. However, a study evaluating the evolution of CVRFs over four years in apparently healthy patients from Cotonou found elevated TG and low HDL-C at 2.2% and 26.2%, respectively [35]. Further, in Parakou, estimates of individual lipid abnormalities ranged between 11.2%–39.4% and 25.3%–53.0% in hospital-based and population-based studies, respectively [36,37]. Collectively, these data indicate a significant increase in the prevalence of dyslipidemia, which varied widely across populations and regions.

Moreover, these reports suggest that a large proportion of individuals within Benin may be eligible for lipid-lowering therapy, which has shown favorable impact on CVDs mortality [38]. Pathophysiologically, elevated TC is known to play important roles in both initiation and progression of CVDs [39]. High LDL-C level is a critical risk factor for CVDs [6] and lowering LDL-C concentrations is the primary target for treatment and prevention of CVDs [40]. Dyslipidemia and hypertension commonly coexist as components of the metabolic syndrome in CVDs [41]. Here, hypertension was positively associated with risk of dyslipidemia, similarly to what was reported in findings of previous studies [30,42,43]. We also found that dyslipidemia was more prevalent in IR patients than in non-IR patients. Dyslipidemia mediated IR was classically associated with elevated TG and low HDL-C, which can be detected years before the clinical diagnosis of diabetes in IR patients [44]. This study also established a positive association between elevated ALT and risk of dyslipidemia, suggesting that patients with dyslipidemia may have a higher chance of developing liver disease than non-dyslipidemia. This finding was in complete agreement with reports by Park et al. who demonstrated significant increases in the levels of several lipid profiles (e.g., TG, TC/HDL-C) with increasing ALT levels [45]. Similarly, positive associations of elevated TG, TC, and LDL-C with ALT as well as AST and GGT were reported in Bangladesh [46]. Further, persistent elevations of ALT and GGT increased cardiovascular risk in white and black adults followed over 12 years [47]. Consistent with this, Zhang et al. revealed that increased ALT was associated, in a dose-response manner, with multimorbidity

(e.g., hypertension, diabetes, dyslipidemia, and stroke) [48].

Interestingly, a prospective study from China associated elevations of ALT and AST with increased incident type 2 diabetes risk [49]. Overall, these data indicate that monitoring of ALT may have significant impacts on risk of developing NCDs in patients with dyslipidemia. Alcohol decreased lipolysis of circulating chylomicrons and VLDL by a reduced activity of lipoprotein lipase. Excessive alcohol intake was associated with increased TG and CVDs [50]. However, our results indicated that alcohol reduced risk of dyslipidemia, similarly to findings of study conducted in China [31]. Evidence suggested that the favorable impact of alcohol on blood lipids could be related to the type of alcoholic beverage as well as genetic polymorphisms [50-52]. In this study, CRP, which is a highly sensitive systemic marker of inflammation and tissue damage, showed a linear relationship with risk of dyslipidemia. This was consistent with reports in homozygous familial hypercholesterolemia patients [53]. Further, Koenig suggested that hs-CRP could better predict future cardiovascular outcomes than traditional CVRFs [54]. Consistently, elevated hs-CRP and risk of CVDs was demonstrated in several reports [55,56]. We anticipated that continuous monitoring of hs-CRP in dyslipidemic patients could have favorable impacts on CVDs. We acknowledge that there are important limitations to this study. The sample size is relatively small. The study included only TMDs and did not evaluate influence of sex or lifestyle factors, limiting generalizability of our findings. A further limitation of this study was its cross-sectional nature, which limits our understanding of causal links between dyslipidemia and associated factors. Thus, obtained result represent associations, not causation. In spite of these limitations, this study has several strengths. This is the first study that reports an association of dyslipidemia with risk factors such as hs-CRP and ALT in TMDs. Therefore, this study adds additional flows to existing medical literature on dyslipidemia within Benin. As such, our study provides evidence-based foundation for future broader studies that may help policy makers when planning and implementing interventions to control risk factors of CVDs.

Conclusion

Our findings revealed that three in 10 TMDs had dyslipidemia, which co-occurs with hypertension, increased ALT, and hs-CRP. These are worrying findings given that TMDs without diabetes or CVDs already have coexistence of dyslipidemia with multiple CVRFs. Therefore, dyslipidemia should be considered as a serious public health problem, and strategies for prevention, early detection, and treatment using lipid-lowering therapy are required to reduce the burden of CVDs in high-risk populations. This study provides useful information for future broader studies with different designs to better quantify the relationship of dyslipidemia with CVRFs.

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Competing Interests

The authors declare no competing interests.

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