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Plasma lipid profile including the high density lipoprotein (HDL) subclasses in hypertensive patients in Ouagadougou, Burkina Faso

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The study was undertaken to evaluate the interest of cholesterol subclasses in the management of hypertensive patients recruited at University Hospital Yalgado Ouedraogo of Ouagadougou (Burkina Faso, West Africa). The distribution of hypertensive was reported as 45 (35.4%) without complications, 42 (33%) with cardiovascular complications and 40 (31.4%) with diabetes. Any difference in lipids profile was observed when balanced hypertensive was compared to non-balanced hypertensive. The total cholesterol (TC), high density lipoprotein cholesterol (HDLC) and HDL3 cholesterol (HDL3C) were significantly higher in hypertensive compared to the control group ($p<0.001$). Significant decrease of TC and HDLC levels was observed in women within hypertensive group ($p<0.05$). The increase in triglyceride (TG) and low density lipoprotein cholesterol (LDLC) was significant in obese compared to non-obese. The HDLC level was higher ($p<0.01$) in treated hypertensive compared to untreated, particularly females. The HDLC increased significantly in treated hypertensive without complications ($p<0.01$). The TC and LDLC levels were higher in treated hypertensive with diabetes ($p<0.05$). The HDL2 cholesterol (HDL2C) was significantly lower in treated hypertensive with diabetes ($p<0.05$), and particularly in obese compared to non-obese. A significant decrease of HDL2C was observed in female stage 3 hypertensive ($p<0.05$). The HDL2C might be a better predictor of cardiovascular risks in hypertensive if the relationship between its decrease with severity of hypertension is confirmed by further studies.

Key words: Lipids profile, HDL subclasses, hypertensive.

INTRODUCTION

Hypertension (HT) is one of the most important risk factors of cardiovascular diseases (Durakoğlugil et al., 2014). High blood pressure levels have been associated with elevated atherogenic blood lipid (Saidu et al., 2014). Hence, screening for lipid abnormalities should be an essential part of management of hypertensive patients. It

is recommended to perform an annual screening for blood fasting lipids, including total cholesterol (TC), triglyceride (TG), high density lipoprotein cholesterol (HDLC) and low density lipoprotein cholesterol (LDLC) in hypertensive patients (McPherson et al., 2006). The HDLC has long been considered the main biological marker of protection against the cardiovascular disease. However, many studies have reported cases of patients with low HDLC without additional risk of cardiovascular events, and other increased risks, despite a high value of HDLC (Joy et al., 2008). This atheroprotective role has been attributed to the HDL2C fraction of HDL cholesterol, differentiated on the basis of its density (d = 1,063-1,125 g / ml), whereas the HDL3C fraction (d = 1,125-1,210 g / ml) does not seem involved (Bakogianni et al., 2001; Moriyama et al., 2014). Therefore, the HDL2C subfraction seems to be more atherosclerotic protective than the HDL3C subfraction (McPherson et al., 2006). An inverse correlation has been established between the HDL2C subfraction and atherosclerosis where an increase in the HDL2C subfraction results in decrease in atherosclerosis (Maeda et al., 2012). Several methods of measurement of HDL subclasses have been proposed and many authors have demonstrated the value of knowledge of the HDL subclasses in the monitoring of metabolic diseases (Superko, 2009; Shuhei et al., 2010). This has shown that there could be an association between HDL2C and atherosclerosis in hypertensive patients. This study was undertaken to further evaluate this association using the patients in Bukina Faso in order to build further the body of knowledge in this area. This study also aimed to validate a cost effective method for determining an effective threshold concentration of HDL2C used in assessing cardiovascular risk. The authors propose that this will help Bukina Faso, a resource limited country, in preventing coronary heart diseases.

METHODOLOGY

Ethics statement

The study protocol and consent procedure were approved by the Burkina Faso National Ethics Committee for Research Ouagadougou, Burkina Faso approval number #2012–06–52 on the 7th June 2012. As required by the Helsinki declaration a written informed consent was obtained from all participants prior to conducting any study procedures. After consenting, personal and epidemiological data were collected and recorded. All the data used in this study were anonymous.

Type and period of the study

This was a case-control study conducted between September to December 2012 in Ouagadougou, the capital city of Burkina Faso in West Africa. The hypertensive subjects were recruited at University Hospital Yalgado Ouedraogo of Ouagadougou and the control group at the Regional Center of Blood Transfusion of Ouagadougou. All laboratory tests were performed at the University Hospital laboratory Yalgado Ouedraogo of Ouagadougou.

Recruitment of study population

Inclusion criteria

The study population comprised of consecutive hypertensive patients attending the cardiology department of University Hospital Yalgado Ouedraogo of Ouagadougou during the period of the study. The hypertensive patients were divided into 4 groups: Group 1: untreated hypertensive; patients; Group 2: treated hypertensive patients without complications; Group 3: treated hypertensive patients with complications (coronary artery diseases; peripheral vascular and cerebrovascular disease; left ventricular systolic dysfunction, nephropathy, retinopathy); Group 4: Treated hypertensive patients with diabetes; Group 5 comprised apparently healthy normotensive controls were recruited at the Regional Center of Blood Transfusion of Ouagadougou.

Exclusion criteria

The hypertensive patients were excluded if taking drugs known to increase blood pressure like steroids and contraceptive pills or to modify the lipids such as statins, nicotinic acid, fibrate and resins. Pregnant women and subjects who are enlisted in any other concomitant study were also excluded.

Biochemical analysis

After an overnight fast, venous blood was collected in a dry tube for biochemical analysis. Serum was separated by centrifugation at 3000 *g* for 10 min at 4°C, stored at -80°C and analyzed within a week. Serum total cholesterol (TC) and triglycerides (TG) were determined using an automated Spintech 240 Biolis 24j analyzer (Spintech, Barcelona, Spain) and the fully enzymatic methods (Spinreact kits Cholesterol-LQ reference TK41021 and Triglycerides-LQ reference TK41031). The dual-step precipitation of HDL subfraction was performed according to the procedure described by Hirano et al. (2008). To isolate total HDLC by precipitation, a combined precipitant consisting of 100 µl (0.02 mmol/L) of dextran sulfate (Mr 500000, SIGMA, France) and 25 µl (200 mmol/L) of $MnCl_2$ ($MgCl_2$-$6H_20$, MERCK, France) was added to 1 ml of serum. After 15 minof standing at room temperature, the mixture was centrifuged at 3,400 *g* for 20 min at 4°C. Aliquots of the resulting supernatant (S1) were taken for the assay of the HDLC and precipitation of the HDL2C. The HDL2C was precipitated by a combined precipitant consisting of 100 µl (0.02 mmol/L) of dextran sulfate (Mr 500000, SIGMA, France) and 50 µl (200 mmol/L) of $MnCl_2$ (MgCl2-6H20, MERCK, France) added to 500 µl of supernatant (S1). After 2 hat room temperature, the mixture was centrifuged at 3,400 *g* for 20 min at 4°C. Aliquots of the resulting supernatant (S2) were taken for the assay of the HDL3C. The measured value for total HDLC was multiplied by 1.125 and that for HDL3 was multiplied by 2.92 to correct for dilution by the

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Table 1. The accuracy and precision of the measurements and day to day coefficient of variations.

Parameter	Accuracy (n = 20)			Precision	
	Reference mean	Mean obtained	Student test calculed t	Intra-serial CV (n = 20)	Inter-serial CV (n=20)
CT	2.87	2.81	0.83	0.56	0.98
HDL-c	0.77	0.65	0.37	1.05	0.94
LDL-c	1.54	1.39	1.67	0.98	1.56
TG	1.23	1.16	0.80	1.39	0.67

TC= total cholesterol; HDL-c= HDL-cholesterol; LDL-c= LDL-cholesterol; TG=triglycerides; CV=coefficient of variations.

reagents. HDL3C was measured by the direct HDLC homogenous assay instead of the original TC assay. The sub-fraction HDL2C was calculated by the following formula: cholesterol HDL2C = HDLC - HDL3C. We calculated the LDLC (in mmol/L) by using the Friedewald formula: LDLC = TC – HDLC – TG/2.2 (Srisawasdi P et al., 2011).

Quality control

Laboratory analysis was conducted by trained and competent personnel following standard laboratory procedures as stated above. The laboratory was enrolled in a National External Quality Assurance (EQA) program for cholesterol and HDL fractions determination with a frequency of 2 surveys per year. During the period of the experiment, the laboratory performance was within the stated acceptable limits of National EQA program. To ensure the accuracy and precision of the test results, internal quality controls were performed daily before analysis and Standard Deviation (SD) and coefficient of variations (CV) calculated. In Table 1 the accuracy and precision of the measurements during the study were within the acceptable criteria stated in literature (Thalameh et al.,1986; Vassault et al., 1999).

Statistical analysis

Quantitative variables were expressed as means ± standard deviation and qualitative variables in percentages. The Analysis of Variance (ANOVA) was used to determine quantitative variables with normal distribution, followed by the Bonferonni multiple comparisons test to compare the means between groups. The statistical analysis was performed using the statistical software PASW, version 18 for Windows (SPSS CPSC., Chicago, USA). Probability levels of 0.05 or less were considered significant.

RESULTS

A total of 201 study subjects of which 158 (78.6%) were hypertensive with an average age of 55 ± 11 years were studied (Table 2). The other 43 (21.4%), making the control group, were normotensive with an average age of 51 ± 8 years. The 158 hypertensive patients, 53 (33.5%) and 105 (66.5%) were males and females respectively. Of the 43 in the control group, 24 (55.8%) were males and 19 (44.8%) females. There were significantly more females hypertensive than males hypertensive in the study group ($p<0.05$). The hypertensive patients were significantly older than the controls (Control group mean = 51 ± 8; hypertensive group mean = 55 ± 11; $p<0.05$). The male hypertensive patients were older (mean = 58 ± 12 years) than female (mean = 53 ± 10 years). The body mass index (BMI) for hypertensive patients (Mean = 27 ± 6 kg/m²) was significantly higher ($p<0.001$) compared to normotensive controls (Mean =21.7 ± 3 kg/m^2). The prevalence of obesity in hypertensive patients was 57.6% (87.5% in females versus 12.5% in males). Among the 158 hypertensive patients, 127 (80%) were on antihypertensive therapy. The 127 treated hypertensive patients comprised 45 (35.4 %) without complications, 42 (33%) with cardiovascular complications and 40 (31.4 %) with diabetes. Both systolic and diastolic blood pressures (BP) for untreated hypertensive patients were significantly higher than those on treatment. According to the World Health Organization (WHO) classification of hypertensive stages of severity, the patients were divided into 3 groups: 42 (26.6%) in stage I; 33 (20.9%) in stage II; 16 (10.1%) in stage III and 67 (42.4%) were balanced.

The TC, HDLC and HDL3C (Table 3) were significantly higher in hypertensive patients compared to control group ($p<0.001$). Particularly, the increase in TC and HDLC was significant in women within hypertensive group ($p<0.05$).

Significant increase of the triglyceride and LDLC was observed in obese compared to non-obese and the decrease of HDLC and HDL3C was significant in obese ($p<0.05$), while an decrease in HDL2C was observed in obese males compared to non-obese males (Table 4).

The blood lipids profile in Table 5 shows that the HDLC level increased significantly ($p<0.01$) in treated hypertensive compared to untreated hypertensive, especially in females. The highest HDLC level was recorded in treated hypertensive without complications ($p<0.01$). The TC and LDLC levels were significantly higher in treated hypertensive with diabetes ($p<0.05$). In males, the decrease of HDL2C was significant in treated hypertensive with diabetes.

The study of lipid levels according to the stage of hypertension recorded in Table 6 shows only a significant decrease of HDL2C in stage 3 females hypertensive ($p<0.05$). No difference in lipids profile was observed between balanced hypertensive and non-balanced hypertensive.

Table 2. Demographic and clinical characteristics of the population.

Parameter		Non hypertesive n= 43 M 24 F 19	Hypertensive					
			Total n=158 M 53 F 105	Untreated hypertensive n=31 M=13 F=18	Treated hypertensive n=127 M=40 F=87	Treated hypertensive without complications n=45 M=9 F=36	Treated hypertensive with complications n=42 M=21 F 21	Treated hypertensive with diabetes n=40 M=10 F=30
Age(years)	Participants	51 ± 8	55±11	49±13	56±11	53±11	60±11	56±10
	Male	54 ± 9	58±12	53±14	60±12	54±14	62±13	60±7
	Female	48 ± 7	53±10	46±12	54±10	52±10	58±9	54±11
Sex	Male	24(55.8%)	53(34.6)	13(24,5%)	40(31,5%)	9(17%)	21(39%)	10(18,9%)
	Female	19(44.2)	105(66.4)	18(17,1%)	87(68,5%)	36(34,3%)	21(20%)	30(28,6%)
	Sex ratio	1.3	0.5	0.7	0.4	0.2	1	3
BMI (Kg/m^2)		21.7±3	27±6	27±5	28±6	28±6	25±5	30±7
Waist circumference (cm)	Male	85±9	91±11	87±11	93±13	90±9	93±14	96±12
	Female	87±10	94±11	89±12	95±11	94±10	92±11	99±11
Blood pressure (mmHg)	Systolic	120±10	144±21	155±21	141±22	143±23	141±23	140±18
	Diastolic	85±10	84±10	93±13	83±10	85±10	82±10	80±10
Hypertension Stage	Stage 1	-	42(26.6%)	10(24%)	32(25%)	15(36%)	7(17%)	10(24%)
	Stage 2	-	33(20.9%)	8(24%)	25(20%)	9(27%)	8(24%)	8(24%)
	Stage 3	-	16(10.1)	8(50%)	8(6%)	3(19%)	4(25%)	1(6%)
	Balanced		67(42.4)	5(8%)	62(92%)	18(27%)	23(34%)	21(31%)

DISCUSSION

The present study assessed plasma lipid profile among hypertensive subjects. The total cholesterol (TC) and HDLC increased significantly in hypertensive compared to control group. The increase of TC is common in hypertensive but generally a low HDLC is reported in hypertensive (Kanaya et al., 2003; Cai et al., 2012; Sun et al., 2014). The findings of higher mean HDLC among hypertensive patients when compared with normal controls was also documented in Nigeria (Okeahialam et al., 2003; Karaye et al., 2008; Adamu et al., 2013; Saidu et al., 2014). Particularly, significant elevation of TC and HDLC levels was noted in women within hypertensive group. This can be explained by the number of postmenopausal women in the group (53 ± 10 years). This observation is consistent with other studies (Freedman et al., 2004; Ai et al., 2010; Skoczyńska et al., 2013) as well as with National Cholesterol Education Program report, ATPIII: prior to the age of menopause, females have lower total cholesterol levels than males of the same age. After menopause, however, cholesterol levels tend to rise in women (Stone et al., 2005).

In this study, the relationship between dyslipidemia and the traditional cardiovascular risk factors including body mass index (BMI), waist circumference (WC) and blood pressure (BP) was studied. The triglyceride and LDLC increased in obese compared to non-obese and the decrease of HDLC was significant in obese. These findings are in agreement with those of previous studies (Kanaya et al., 2003; Cai et al., 2012; Wang et al., 2012;Sun et al., 2014).

Table 3. Lipids values of hypertensive group compared to non-hypertensive.

Lipids (mmol/L)		Hypertensive n=158 (M 53 F 105)	Non hypertensive n=43 (M 24, F 19)	*p*
Total Cholesterol	Population	5.39±1.20	4.69 ± 0.84	0.0004
	Male	5.11±1.18	4.71 ± 0.94	0.147
	Female	5.52±1.17	4.67 ± 0.72	0.04
	p	0.039	0.883	
Triglycerides	Population	1.24±0.65	1.10 ± 0.43	0.18
	Male	1.24±0.70	1.09 ± 0.40	0.27
	Female	1.25±0.58	1.10 ± 0.47	0.28
	p	0.90	0.965	
LDL cholesterol	Population	3.42±1.04	3.23 ± 0.8	0.26
	Male	3.26±1.03	3.26 ± 0.86	0.24
	Female	3.49±1.04	3.20 ± 0.74	
	p	0.189	0.821	
HDL cholesterol	Population	1.53±0.39	1.23 ± 0.3	0.00005
	Male	1.42±0.30	1.19 ± 0.27	0.002
	Female	1.57±0.40	1.28 ± 0.34	0.003
	p	0.017	0.331	
HDL_3 cholesterol	Population	1.02±0.23	0.76 ± 0.18	0.000001
	Male	0.99±0.22	0.76 ± 0.20	0.000003
	Female	1.03±0.22	0.76 ± 0.15	0.000001
	p	0.23	0.941	
HDL2 cholesterol	Population	0.51±0.36	0.50 ± 0.28	0.86
	Male	0.44±0.30	0.45 ± 0.29	0.89
	Female	0.54±0.37	0.56 ± 0.28	0.81
	p	0.08	0.216	

Table 4. Comparison of Lipid levels between obese and non-obese.

Lipids (mmol/L)	Non obese (BMI <25 Kg/m^2)			Obese(BMI ≥25 Kg/m^2)		
	Total	Male	Female	Total	Male	Female
Triglyceride	1.10±0.5	1.06±0.5	1.14±0.5	1.50±0.92[a]	1.72±1.2	1.29±0.64
Total Cholesterol	5.23±1.12	4.95±1.11	5.52±1.13[c]	5.48±1.33	5.39±1.44	5.57±1.23
LDLcholesterol	3.24±1.15	3.1±1.00	3.39±1.03	3.60±1.09[a]	3.66±1.09[b]	3.55±1.09
HDL cholesterol	1.55±0.30	1.49±0.33	1.62±0.32	1.42±0.38[a]	1.28±0.31[b]	1.57±0.46
HDL2cholesterol	0.50±0.30	0.46±0.34	0.55±0.28	0.47±0.34	0.39±0.27[b]	0.55±0.42
HDL3cholesterol	1.06±0.19	1.05±0.22	1.08±0.21	0.95±0.20[a]	0.88±0.22[b]	1.02±0.24

Significant difference (P<0.05) between [a]Non obese hypertensive and obese hypertensive; [B]male Non obese hypertensive and Male obese hypertensive; [c]male non-obese hypertensive and female non-obese hypertensive.

The blood lipids profile showed a significant elevation of the TC and HDLC in treated hypertensive compared to untreated and particularly in females. Moreover, the TC and LDLC levels were higher in treated hypertensive with diabetes. The dyslipidaemia seen in the Type 2 diabetes mellitus (T2DM) patients with hypertension could be due to the effects of antihypertensive treatment. Indeed, in other studies, treatment of hypertension with b-blockers, as well as high doses of thiazide diuretics have been shown to exacerbate the dyslipidaemia in patients with hypertension and diabetes mellitus (Andrew and Clifford, 1994; Iaccarino et al., 2005).

Table 5. Blood Lipids profile of untreated hypertensive and treated hypertensive.

Parameters (mmol/L)		Untreated hypertensive n=31 M=13 F=18	Treated hypertensive n=127 M=40 F=37	Treated hypertensive without complications n=45 M=9 F=36	Treated hypertensive with complications N=42 M=21 F 21	Treated hypertensive with diabetes N=40 M=10 F=30
Triglyceride	Population	1.22±0.91	1.25±0.6	1.16±0.52	1.17±0.48	1.44±0.76
	Male	1.40±1.29	1.18±0.56	1.10±0.48	1.08±0.49	1.46±0.73
	Female	1.09±0.52	1.29±0.62	1.18±0.53	1.25±0.46	1.44±0.79
Total cholesterol	Population	5.19±1.20	5.44±1.22	5.47±1.31	5.04±1.14	5.83±1.11[h]
	Male	5.32±1.00	4.98±1.27	4.83±1.35	4.71±1.22	5.7±1.14
	Female	5.10±1.35	5.65±1.16[f]	5.62±1.28	5.38±0.97	5.88±1.11
LDL Cholesterol	Population	3.24±0.98	3.46±1.08	3.31±1.00	3.26±1.07	3.85±1.11[h]
	Male	3.34±0.97	3.21±1.08	2.9±1.15	3.08±1.06	3.78±0.91
	Female	3.16±1.00	3.58±1.08	3.41±0.95	3.43±1.08	3.88±1.18
HDL Cholesterol	Population	1.38±0.37	1.57±0.40[d]	1.61±0.47[h]	1.54±0.39	1.56±0.34
	Male	1.43±0.40	1.44±0.32	1.47±0.26	1.48±0.38	1.32±0.15[g]
	Female	1.34±0.35	1.63±0.42[e,f]	1.64±0.50	1.6±0.39	1.65±0.35
HDL3 cholesterol	Population	0.98±0.23	1.04±0.23	1.01±0.22	1.04±0.23	1.07±0.25[h]
	Male	1.05±0.29	0.99±0.21	0.93±0.14	1.03±0.23	0.95±0.23[g]
	Female	0.92±0.17	1.06±0.24	1.02±0.23	1.05±0.22	1.1±0.25
HDL2 Cholesterol	Population	0.41±0.33	0.54±0.38	0.6±0.45	0.51±0.34	0.5±0.31
	Male	0.39±0.37	0.46±0.31	0.54±0.3	0.48±0.35	0.36±0.17[h]
	Female	0.43±0.31	0.57±0.40	0.61±0.48	0.55±0.33	0.55±0.34

Significant difference (P<0.05) between [D]untreated hypertensive and Treated hypertensive, [E]female untreated hypertensive and female treated hypertensive, [F]male treated hypertensive and female treated hypertensive, [G]male treated hypertensive with diabetes and female treated hypertensive with diabetes, [h] treated hypertensive without complications and treated hypertensive with diabetes

Table 6. Lipids levels according to the grade of hypertension.

Parameters (mmol/L)		Balanced hypertensive	Non balanced hypertensive	Hypertensive		
				Stage 1	Stage 2	Stage 3
Triglyceride	Population	1.23±0.7	1.26±0.66	1.13±0.71	1.28±0.66	1.28±0.53
Total Cholesterol	Population	5.39±1.2	5.39±1.25	5.29±1.2	5.33±1.19	5.77±1.48
LDL Cholesterol	Population	3.46±0.98	3.39±1.13	3.28±1.09	3.38±1.13	3.69±1,24
HDLCholesterol	population	1.57±0.37	1.51±0.43	1.60±0.51	1.42±0.33	1.46±0.35
	Male	1.46±0.41	1.42±0.28	1.43±0.34	1.41±0.25	1.43±0.25
	Female	1.61±0.34	1.56±0.48	1.68±0.56	1.43±0.38	1.48±0.39
HDL2cholesterol	population	0.52±0.33	0.51±0.40	0.58±0.47	0.45±0.30	0.46±0.38
	Male	0.44±0.34	0.45±0.31	0.44±0.35	0.44±0.29	0.48±0.34
	Female	0.55±0.32	0.54±0.44	0.64±0.51	0.46±0.32	0.45±0.41[i]
HDL3cholesterol	Population	1.05±0.21	1.01±0.24	1.02±0.25	0.98±0.24	1.05±0.25
	Male	1.01±0.2	0.99±0.24	0.99±0.21	0.97±0.23	1.12±0.41
	Female	1.06±0.22	1.01±0.24	1.04±0.27	0.98±0.25	1.03±0.15

Significant difference (P<0.05) between [i]Stage 1; Stage 2 and stage 3 hypertensive.

The measurement of cholesterol sub-fractions reported a significant increase of HDL3C in hypertensive compared to control group and HDL3C level was in accordance to TC and HDLC values. This observation is consistent to over studies reporting that HDLC of hypertensive patients had a markedly increased relative content of HDL3C

while their HDL2C fraction was reduced by other 50% (Dobiasova et al., 1992). The HDL3C level was higher in treated hypertensive with diabetes and particular in males. This HDL3C elevation can be due to the hypertension effect on lipids profile and also to the treatment reported to increase HDL3C in patients on antihypertensive drugs as beta-blokers (Szollár et al., 1990).

The high density lipoprotein 2 (HDL2C) decreased in obese compared to non-obese. In males, the decrease in HDL2C was significant in treated hypertensive with diabetes. This decrease in HDL2C can be explained by the fact that the apoAI, major protein component of HDL2C, is reduced in type 2 diabetes (Van Linthout et al., 2010). According to the stage of hypertension, a significant decrease of HDL2C was observed only in stage 3 female hypertensive while the other lipids levels were not related to the severity of hypertension.

Conclusion

The HDL2C might be a better predictor of hypertensive complications if the relationship between its decreases with hypertensive stage of severity is confirmed by further studies.

Conflict of interests

The authors did not declare any conflict of interest.

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