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PLASMA LIPID PROFILES IN NIGERIANS WITH NORMAL BLOOD PRESSURE, HYPERTENSION AND OTHER ACQUIRED CARDIAC CONDITIONS
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PLASMA LIPID PROFILES IN NIGERIANS WITH NORMAL BLOOD PRESSURE, HYPERTENSION AND OTHER ACQUIRED CARDIAC CONDITIONS

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ABSTRACT

Background: **The attention of researchers has recently been drawn to the changing lipid pattern in the Nigerian African because of diet and changing lifestyles. With increasing awareness of systemic hypertension and the role of cholesterol in ischaemic heart disease, indiscriminate emphasis has been placed on the reduction of fat intake and total calorie intake in every patient who has any form of heart disease.**

Objective: **To assess and demonstrate any association between abnormal lipid profile and some acquired cardiovascular diseases, which confer relevance of dietary cholesterol restriction.**

Design: **A cross-sectional study.**

Setting: **Cardiology clinic, University of Benin Teaching Hospital.**

Subjects: **One hundred and twenty two patients (59 males and 63 females) who attended the cardiology clinic in the month of May 2001.**

Results: **The results revealed significant hyperlipidaemia in three groups of Nigerian patients with acquired heart disease: the hypertensive with or without hypertensive heart disease (HHDx), the patient with ischaemic heart disease (IHD), and those with hypertrophic cardiomyopathy (HCM). Rheumatic heart disease (RHD) and dilated cardiomyopathy (DCM) had lower cholesterol levels than the normotensives.**

Concusion: **This study has demonstrated that not all acquired cardiovascular diseases are associated with hyperlipidaemia. Intervention studies may therefore be needed to examine the benefits or adverse effects of dietary restriction in such conditions.**

INTRODUCTION

The attention of researchers has recently been drawn to the changing lipid patterns in Nigerians (1-3). This change has been linked to the changing diet and lifestyle of the average African and to the change in the methods of lipid analysis (from the Lieberman Burchard or ferric chloride reaction to enzymatic methods) (2).

With increasing awareness of systemic hypertension and the role of cholesterol in ischaemic heart disease, there has been indiscriminate emphasis on the reduction of fat intake and total calorie intake in every patient who has any form of heart disease. Earlier works have shown that several factors are to be considered in the interpretation of serum cholesterol level (2-4). These include lipid fractions, disease states, genetics and environmental

factors. Individuals with hypercholesterolaemia, high total cholesterol / high-density lipoprotein -cholesterol ratio and high atherogenic index are all at risk of atheroma induced cardiovascular disease (4). It is also known that the failing myocardium exhibits both nutritional deficiencies and altered nutritional demands which impair myocardial energy metabolism, intracellular calcium homeostasis, and antioxidant defenses (5). This knowledge has led to the fifth paradigm - a nutritional approach to the treatment of heart failure (6). We have decided to examine the lipid profile in different acquired cardiovascular diseases and hence, address the relevance of lipid profile in the management of these conditions.

MATERIALS AND METHODS

One hundred and twenty two patients (59 males and 63 females) of mean age 50.07 ± 12.00 years who attended the cardiology clinic of the University of Benin Teaching Hospital in the month of May 2001 were recruited for this study. This number represented about 12% of the patients that attended the cardiology clinic during the year. They included 19 newly diagnosed hypertensives with no evidence of hypertensive heart disease (ten males, nine females; 53 hypertensives with hypertensive heart disease (22 males, 31 females); 20 patients with rheumatic heart disease (12 males, eight females); 19 patients with dilated cardiomyopathy (nine males, ten females); six patients with ischaemic heart disease (three males, three females) and five patients with hypertrophic cardiomyopathy (three males and two females). Twenty normotensive (NT) controls (nine males, 11 females) of mean age 48.51±6.31 years were also recruited.

The inclusion criteria were as follows:-

(i) Normotensive in apparently good health at the time of recruitment with blood pressure (BP) ≤130/85 mmHg.
(ii) All patients attending the cardiology clinic that has been diagnosed to have any of systemic hypertension (HTN), DCM, IHD. RHD, or HCM.

The exclusion criteria were as follows:

(i) Patients with congenital heart disease.
(ii) Patients with diabetes mellitus.
(iii) Patients with recent history of myocardial infarction.
(iv) Patients in renal failure.
(v) Patients on oral or parenteral contraceptives, (except IUCD) or anabolic steroids.
(vi) Patients who exercised in the last three hours.

Each patient gave his or her informed consent. On the examination day, following an overnight fasting of 12 to 14 hours, routine measurements of weight and height were done using a stadiometer and beam balance scale. Blood pressure was measured after ten minutes of rest using Accosons mercury sphygmomanometer with appropriate standard cuff size. A full clinical examination was then carried out on each subject. The body mass index (BMI) was calculated as weight in kilograms /height in square meter (Kg/m^2). Five millilitres of venous blood was drawn from the arm of each subject into a lithium heparinised plastic bottle, centrifuged immediately at 1500xg for ten minutes and the separated plasma was stored at 4°C before analysing for total cholesterol (TC) and high density lipoprotein cholesterol (HDL-C), and triglycerides (Tg) by standard methods (7,8) respectively. The modified Friedwald formula (8) used to calculate the low-density lipoprotein cholesterol (LDL-C). For the purpose of this study:

(i) Hypertension was based on sitting BP of ≥140/90 mmHg at two or more clinic visits in a subject who is not on antihypertensive medication (9).
(ii) Hypertensive heart disease was based on the presence of a forceful heaving apex, left ventricular hypertrophy by radiology, echocardiography and electrocardiography (10).
(iii) Rheumatic heart disease was based on the presence of thickened valve leaflets or fusion of cusps at echocardiography especially with multiple valve involvement.
(iv) Dilated cardiomyopathy was based on echocardiographic finding of global dilatation and global hypokinesia (10).
(v) Ischaemic heart disease was based on the presence of regional hypokinesia in the absence of cardiac dilatation at echocardiography.

All data obtained were tabulated and analysed by standard statistical methods (11). The student's t-test was used to compare means of two variables. Values of $p<0.05$ were regarded as significant.

RESULTS

Table 1 shows the baseline characteristics of subject studies; all were age sex matched. BMI was significantly higher in the hypertensive groups (with or without HHDx) than in all other groups except IHD.

Table 2 shows the means of serum lipids found in the various groups. Serum cholesterol (TC) was significantly highest in the IHD group (6.35 ± 0.63 mmol/L) followed by the hypertensive groups (5.51±1.33 and 5.52 ± 0.78 mmol/L respectively) and HCM group (5.57 ± 1.50 mmol/L) (P<0.001). There was no significant difference between TC in the two hypertensive groups. TC was significantly higher in the normotensive than in RHD and DCM groups. Triglycerides (Tg) where significantly higher (2.73 ± 1.12 mmol/L) in the HCM compared with any other group (P< 0.001).

This pattern of difference is repeated in serum levels of low-density lipoprotein (LDL-C) and very low-density Lipoproteins (VCDL-C) of the various groups studied.

Table 3 shows the mean of lipid fraction in the various groups. The ratio of TC to HDL-C (TC: HDL-C) was least for IHD group and highest for RHD and DCM groups. The differences were highly significant (P<0.001). The TC: HDL-C ratio was similar in the hypertensive groups, IHD and HCM groups.

The atherogenic index (LDL-C/HDL-C) had a reciprocal relationship in the groups being significantly highest in the IHD group followed by the hypertensive group and HCM groups respectively. Significantly lower values were seen in the normotensive, RHD and DCM groups respectively (P<0.001).

Table 1

Baseline characteristics of subjects studied (values are given as ±SD)

Group	No.	Age (years) mean (±SD)	Mean BMI kg/m² (±SD)	Significant level
All normotensives	20	49.15 ± 2.30	24.31 ± 0.82	
Males	9	49.70 ± 2.30	22.6 ± 0.11	
Females	11	48.51 ± 6.31	25.7 ± 0.10	NS
Newly diagnosed hypertensives without HHD	19	47.01 ± 14.50	28.84 ± 4.85	
Males	10	48.45 ± 6.45	27.19 ± 4.48	
Females	9	45.25 ± 7.24	30.65 ± 4.82	NS
Hypertensives with HHDx	53	50.10 ± 5.20	29.00 ± 3.20	
Males	22	51.01 ± 4.91	28.40 ± 2.41	
Females	31	49.00 ± 3.52	29.61 ± 4.01	NS
Rheumatic heart diseases (All)	20	51.01 ± 13.41	24.33 ± 1.71	
Males	12	51.10 ± 15.17	22.40 ± 2.32	
Females	8	51.61 ± 9.90	25.51 ± 0.01	NS
Dilated cardiomyopathy DCM (All)	19	50.531 ± 1.42	24.77 ± 1.30	
Males	19	51.371 ± 1.51	24.74 ± 0.65	NS
Females	10	49.751 ± 1.57	25.63 ± 1.80	
Ischaemic heart disease (All)	6	50.50 ± 12.90	28.43 ± 1.96	
Males	3	49.50 ± 2.50	27.6 ± 2.35	
Females	3	51.50 ± 1.50	29.4 ± 1.82	NS
Hypertropic cardiomyopathy (All)	5	51.33 ± 14.31	25.60 ± 1.01	
Males	3	58.50 ± 12.50	24.85 ± 0.05	NS
Females	2	38.12 ± 0.00	27.20 ± 0.00	

P–values between groups:

1 vs 2;	2 vs 3;	1vs 3;	1 vs 4;	4 vs 5;	1 vs 5;	2 vs 6;	6 vs 7
<0.001	NS	<0.001	NS	NS	<0.001	NS	NS

Table 2

Means of serum lipid levels in the different groups of subjects (values are given as ±SD)

Group	No.	TC mmol/L	Tg mmol/L	HDLC mmol/L	LDLC mmmol/L	VLDLC mmol/L	Level of significance
All normotensives	20	4.34 ± 0.11	1.09 ± 0.06	1.45 ± 0.05	2.42 ± 0.08	0.510 ± 0.05	
Males	9	4.49 ± 0.09	1.10 ± 0.06	1.48 ± 0.03	2.47 ± 0.08	0.540 ± 0.04	NS
Females	11	4.34 ± 0.12	1.05 ± 0.05	1.42 ± 0.04	2.40 ± 0.09	0.490 ± 0.05	
Hypertensives without HHDx all	19	5.52 ± 0.78	1.41±0.36	1.47 ± 0.16	3.56 ± 0.69	0.56 ± 0.63	
Males	10	5.46 ± 0.61	1.51±0.42	1.48 ± 0.15	3.70 ± 0.65	0.52 ± 0.01	NS
Females	9	5.45 ± 0.81	1.30±1.32	1.40 ± 0.15	3.44 ± 0.69	0.57 ± 0.1	
Hypertensives with HHD all	53	5.51 ± 1.33	1.59 ± 0.70	1.44 ± 0.46	3.86 ± 0.97	0.53 ± 0.30	
Males	22	5.58 ± 1.34	1.50 ± 0.74	1.34 ± 0.31	3.73 ± 0.13	0.45 ± 0.01	NS
Females	31	5.53 ± 1.27	1.67 ± 0.74	1.70 ± 0.69	4.90 ± 0.73	0.57 ± 0.01	
Rheumatic heart disease all	20	2.99 ± 0.76	1.04 ± 0.34	1.28 ± 0.79	1.55 ± 0.92	0.21 ± 0.07	
Males	9	3.14 ± 0.50	1.05 ± 0.44	1.38 ± 1.66	1.74 ± 0.96	0.21 ± 0.04	NS
Females	10	2.46 ± 0.74	1.00 ± 0.36	1.22 ± 1.02	1.12 ± 0.49	0.22 ± 0.06	
DCM All	19	3.29 ± 0.52	1.11 ± 0.45	1.41 ± 0.67	1.68 ± 0.79	0.23 ± 0.03	
Males	9	3.03 ± 0.34	1.15 ± 0.35	1.35 ± 0.13	1.55 ± 0.53	0.23 ± 0.09	NS
Females	10	3.29 ± 0.65	0.94 ± 0.57	1.26 ± 0.66	1.92 ± 0.66	0.22 ± 0.13	
Ischaemic heart disease all	6	6.35 ± 0.63	1.70 ± 0.52	1.38 ± 0.010	3.58 ± 1.05	0.62 ± 1.10	
Males	3	6.05 ± 0.45	1.45 ± 0.35	1.45 ± 0.05	3.25 ± 0.05	0.75 ± 0.65	NS
Females	3	6.65 ± 0.65	1.95 ± 0.55	1.30 ± 0.10	4.45 ± 0.65	0.55 ± 0.05	
HCM all	5	5.51 ± 1.50	2.73 ± 1.12	1.40 ± 0.08	4.12 ± 6.00	0.73 ± 0.36	
Males	3	4.0 ± 1.00	2.75 ± 1.35	1.30 ± 0.20	3.25 ± 1.45	0.50 ± 0.36	NS
Females	3	7.4 ± 0.69	2.70 ± 0.97	1.35 ± 0.00	4.85 ± 0.00	1.0 ± 0.00	
Significant level		1 vs 2 1 vs 3 1 vs 6 1 vs 7	P<0.001	3 vs 4/5 P<0.001			

Table 3

Means of lipoprotein fractions in the various groups studied (values are given as SD) Groups (n)

Parameter		No.	Normotensive without HHDx	No.	Hypertensives with HHDx	No.	Hypertensive VHD	No.	Rheumatic	No.	DCM	No.	IHD	No.	HCM
LDLC:HDLC	Males	9	1.31 ± 0.15	10	3.04 ± 0.26	22	3.08+0.28	12	1.36 ± 0.11	9	1.15 ± 0.18	3	2.24 ± 1.70	3	2.51 ± 0.70
	Females	11	1.22 ± 0.27	9	2.75 ± 0.83	31	2.88 ± 0.55	8	0.92 ± 0.01	10	1.54 ± 0.16	3	3.42 ± 0.41	2	3.59 ± 0.05
	Total	20	1.21 ± 0.26	19	3.06 ± 0.81	53	3.58 ± 0.43	19	1.21 ± 0.13	19	1.20 ± 0.17	6	2.59 ± 0.50	5	2.94 ± 0.83
TC:HDL-C	Males	19	3.03 ± 0.28	10	3.89 ± 0.48		3.82 ± 0.82	8	2.27 ± 0.32	9	2.24 ± 0.45	3	4.17 ± 0.05	3	3.08 ± 1.0
	Females	11	3.03 ± 0.30	9	3.57 ± 0.44		4.16 ± 0.88	12	2.55 ± 0.32	10	2.61 ± 0.48	3	5.12 ± 0.04	2	5.48 ± 0.30
	Total	20	3.03 ± 0.29	19	3.77 ± 0.45		4.04 ± 0.90	20	2.34 ± 0.57	19	2.33 ± 0.04	6	4.60 ± 0.03	5	3.94 ± 0.32

Table 4

Comparing baseline characteristics lipids and lipoprotein fractions of cardiac patients: P-values between groups

	Group								
Mean age	1 vs 2 NS	1 vs 4 NS	1 vs 6 NS	2 vs 3 NS	2 vs 4 NS	2 vs 6 NS	4 vs 6 NS	4 vs 5 NS	6 vs 7 NS
Mean body mass index (BMI)	<0.001	NS	<0.001	NS	<0.001	NS	<0.001	NS	NS
Total cholesterol	<0.001	NS	<0.001	NS	<0.001	NS	<0.001	NS	NS
Triglycerides	<0.001	NS	<0.001	NS	<0.001	NS	<0.001	NS	NS
LDL-C	<0.001	NS	<0.001	NS	<0.001	NS	<0.001	NS	NS
HDL-C	NS	NS	NS	NS	NS	NS	NS	NS	NS
VLDL-C	<0.001	NS	<0.001	NS	<0.001	NS	<0.001	NS	NS
TC: HDLC	<0.001	NS	<0.001	NS	<0.001	NS	<0.001	NS	NS
LDL-C: HDLC	<0.001	NS	<0.001	NS	<0.001	NS	<0.001	NS	NS

Group 1 = Normotensive
2 = Hypertensive without hypertensive heart disease
3 = Hypertensives with hypertensive heart disease
4 = Rheumatic valvular heart disease
5 = Dilated cardiomyopathy
6 = Ischaemic heart disease
7 = Hypertrophic cardiomyopathy

DISCUSSION

It has been shown in this study that hypertensive Nigerians have higher BMI than their normotensive counterparts; a finding which is in agreement with earlier studies (12,13). The patients with IHD also had high BMI. The association of high BMI and IHD is well known. Men with BMI of between 25 and 29 have a 70% greater risk of coronary heart disease (10). Normotensives and patients with RHD, DCM, and HCM were found to have similar BMI and were not obese.

The study has demonstrated dyslipidaemia in three groups of Nigerian cardiac patients: the hypertensive, the one with IHD, and the patient with HCM. The values obtained are similar to those of Jarikre *et al* (2). Several studies have demonstrated that while the Nigerian Africans than their Caucasian counterparts, a significant difference in lipid levels still exist between the hypertensives and normotensives (12,13). The findings of this study emphasise the additive effect of hyperlipidaemia/ dyslipidaemia on systemic hypertension as risk factors of coronary artery disease. Systemic hypertension, obesity and hyperlipidaemia are independently known major risk factors for IHD (14,15). Ajayi and Rotimi found that HHDx was the most common cause of sudden cardiac death among autopsy cases at the Ile-Ife Teaching Hospital (16).

An interesting finding in this study is the relatively but significantly low TC in DCM and RHD. These are conditions that have been found to be associated with infections, malnutrition and genetic traits (17,18). The presence of low serum cholesterol has been blamed on either the disease itself or the disease process (18).

In this study RHD and DCM had similar HDL-C levels with all other groups but had lower LDL-C levels even lower than the healthy normotensives. They also had significantly low atherogenic indices similar to the normotensive group. According to the Adult Treatment Panel (ATP III) of the National Cholesterol Education Programme released in May 2001 these conditions do not by any means belong to any risk category for coronary artery disease (19). Furthermore, recent works have found that lowering LDL-C below moderate to normal levels elicits diminishing return in terms of coronary event rates (20).

Recent works on the pathogenesis of congestive cardiac failure (CCF) suggest that the nutritional demand on the pathological process of myocardial failure may result in conditioned nutrient requirement deficiencies for the heart (7). Coenzyme Q_{10}, a very important endogenous antioxidant which protects the cell membrane and circulating LDL-C from oxidation is lacking in the failing myocardium (21), and this may result in weakening of endothelium. Coenzyme Q_{10} is found in every food group. Thiamine, a co-enzyme in a variety of enzyme systems particularly those related to carbohydrate metabolism is greatly depleted following use of diuretics. The latter are almost constantly employed in large doses in the management of RHD and DCM. Thus the underlying heart disease relentlessly progressive in almost all patients who develop symptoms of CCF and so mortality remains very high (7,21). Supplementation of such food items can only come from adequate intake of such food items as yeast, liver, milk, eggs and cereals, which are usually denied these patients. Thus, lowering LDL-C in those cardiac patients who already have very low LDL-C is likely to produce diminishing returns in terms of cardiac performance.

In conclusion, this study has shown the association of dyslipidaemia/hyperlipidaemia with hypertensive heart disease, IHD and HCM. It shows the total absence of dyslipidaemia/ hyperlipidaemia in RHD and DCM. Intervention studies may therefore be needed to examine the benefits or adverse effects of dietary restriction in such conditions.

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