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Original Article

Evaluation of serum lipid profile of hypertensives in Niger-Delta, Nigeria

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ABSTRACT

Background: Hypertension is a health challenge in both developing and developed nations. It is an important public health challenge worldwide because of its high frequency and concomitant risks of cardiovascular and kidney disease. **Aim**: The aim of the study was to examine lipid profile of hypertensives and compare with normotensive individuals. **Methods**: The volunteers are patients attending the general out-patient clinics in health facilities in Niger-Delta region South-South Nigeria. Venous blood was collected from 1428 volunteers, allowed to clot and separated into serum. Serum total cholesterol, triglycerides, HDL-cholesterol was estimated by standard methods while LDL-C and VLDL-C were calculated. **Results**: Our results show increase levels of total cholesterol, triglyceride, LDL-C and VLDL-C in hypertensives than normotensives while HDL-C is increased in normotensives than in hypertensives. **Conclusion:** We affirm the lipid pattern of hypertensives and normotensives and incriminated dyslipidaemia as a cause of arterial hypertension. We therefore advocate routine lipid profile in the retinue of test panel for hypertensives to nip any complication that may arise in the bud.

Key words: Hypertension, cardiovascular disease, Niger-Delta, lipid profile, hyperlipidaemia

INTRODUCTION

Hypertension is an abnormal state of circulatory function which in the long term can lead to organ damage and severe morbidity. Hypertension which is defined as the blood pressure of equal to or greater than 140/90mmHg[1] has been recognized as the most common cardiovascular disorder and a leading cause of morbidity and mortality in both developed and developing countries.[2] The prevalence of hypertension is higher in the whites than the black population.[3] The prevalence of hypertension has been found to

vary with race, age, geographical pattern, gender and socioeconomic status.[4] Socioeconomic status which is an indicator of lifestyle attributes and is inversely related to the prevalence, morbidity and mortality rates of hypertension.[5] Several factors such as obesity, cigarette smoking, increased salt intake, genetic factors and lack of physical exercise has been attributed to the development of hypertension in human.[5]

An abnormal lipid profile has been strongly associated with atherosclerotic cardiovascular diseases and this has shown a direct effect on the endothelial dysfunction that occurs in hypertension.[6] The role of elevated serum cholesterol in the etiology of atherosclerosis and associated cardiovascular complications is already well established.[7] Hypertension is known to be associated with alteration in lipid metabolism which gives rise to abnormalities in serum lipid and lipoprotein concentrations.[8] It has also been documented that presence of hyperlipidaemia subsequently worsens the prognosis in hypertensive patients.[8] The dyslipidaemia promotes the pathogenesis of coronary heart disease. Different plasma lipids vary significantly in various population group due to difference in geographical, cultural,[9] economic, social conditions,[10] dietary habits and genetic make-up.

The Niger-Delta region of Nigeria is made up of the six states in South-South geopolitical zone and rich with crude oil. There are a lot of industrial activities in the region due to the exploration and exploitation of crude oil. Though various studies[11,12] have related hypertension with hyperlipidaemia in Nigeria but there is paucity of data on hypertensives in this region. Therefore, the objectives of this study is to evaluate the serum lipid profile of hypertensives and normotensives individuals and if by extension relate it to the oil rich environment which they live.

METHODOLOGY

Study area

This study was carried out in the Niger Delta region of Nigeria which comprises of Edo, Delta, Bayelsa, Rivers, Akwa Ibom, and Cross River states. Samples were collected from medical out-patients at Central Hospital Benin (Edo), Central Hospitals in Warri and Agbor (Delta), Federal Medical Centre, Yenagoa (Bayelsa), BMH Port Harcourt (Rivers), General Hospital, Ikot Ekpene (Akwa Ibom), General Hospital, Calabar (Cross River).

Study population

A total of one thousand four hundred and twenty eight (1428) subjects between the ages of 30-80 years were recruited for this study using the unrestricted random sampling. These includes nine hundred and twelve (912) hypertensive patients in medical out-patients and five hundred and sixteen (516) age, sex matched normotensive subjects attending general out-patient clinic for minor ailments in these hospitals. Hypertensives were defined using sphygmomanometer on the basis of blood pressure of ≥140/90mmHg; while the control group was classified on the basis of blood pressure <140/90mmHg. Inclusion criteria include being hypertensive for equal to or greater than one year but not greater than 18months, use of natural antihypertensive agents such as calcium channel blockers, angiotensin converting enzyme inhibitors, and angiotensin II receptor blockers. Exclusion criteria include obesity, pregnancy, treatment with hypolipiduric drugs, cigarette smoking, diabetes mellitus, contraceptives, thiazides, sulfonamides, epinephrine and androgen usage, since they interfere with plasma lipid concentration. Informed consents were obtained from participants. Ethical clearance was also obtained from the various institution ethical committee.

Collection of samples

Fasting blood samples were collected by standard venepuncture into plain containers. The blood was allowed to clot and then centrifuged at 3000rpm for 10minutes. The serum was separated into a cryovail tubes and kept frozen until required for analysis.

Biochemical analysis

Serum total cholesterol and triglycerides were analyzed using the enzymatic CHOD-PAP method of Trinder[13] as modified by Richmond[14] and HDL-Cholesterol analyzed using the method of Burstein *et al*.[15] LDL-Cholesterol, VLDL-Cholesterol were calculated using Friedewald[16] equation. All test kits used were commercially available and products of Randox Laboratories UK. In all analysis, manufacturer`s instructions were adhered to strictly.

Statistical analysis

The groups mean ± SD was calculated for each analyte and significant difference between means evaluated using the student t-test. Statistical Package for Social Science SPSS version 16.0 software (SPSS Inc., Chicago, IL USA) for windows was used, with $P<0.05$ considered as statistically significant.

RESULTS

A total of one thousand four hundred and twenty eight (1428) individuals participated in the study with nine hundred and twelve, 912 (63.9%) being hypertensives [477 (52.3%)] males and 435 (47.7%) females and five hundred and sixteen [516 (36.1%)] being normotensives [279 (54.1%)] males and 237 (45.9%) females as shown in table 1.

The total cholesterol, Triglycerides, LDL-cholesterol and VLDL-cholesterol were significantly higher except HDL- cholesterol in Hypertensive individual than normal controls. The HDL-cholesterol was observed to be lower in hypertensive than in control subjects as shown in table 2.

Figure 1 shows the lipid parameters of both males and females hypertensives. Total cholesterol, LDL-cholesterol and HDL-cholesterol are higher in male hypertensives than in females' hypertensive while the reverse is the case for triglycerides and VLDL-C.

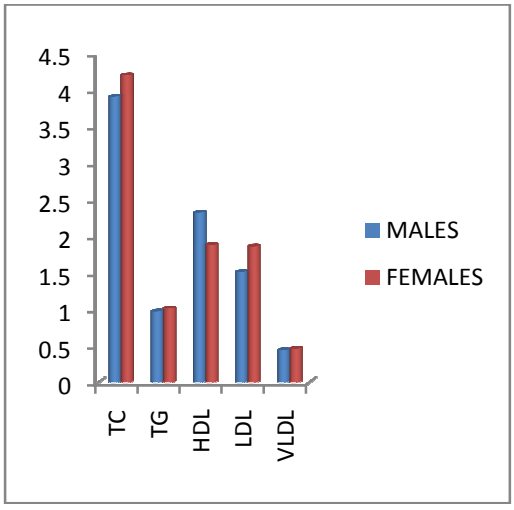

Figure1: Serum Lipid profile of male and female hypertensives

Figure 2 shows the lipid profile of normotensive males and females. The TC and HDL-C are significantly increased in males when compared to their female counterparts. The reverse is the case with TG and LDL-C while there was no significant difference observed in VLDL-C of the two groups.

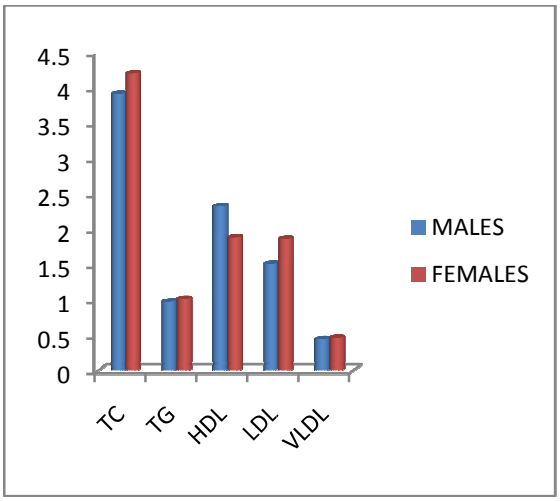

Figure 2: Serum lipid profile of male and female normotensives

Table 3 shows the lipid profile of hypertensive and normotensive males. The TC, TG,LDL-C and VLDL-C of hypertensive males were significantly higher while HDL-C was significantly lower when compared with normotensive males.

The lipid pattern of female hypertensives and normotensives as depicted in table 4 shows a statistically significant increase in TC, TG, LDL-C and VLDL-C but statistically significant decreased HDL-C in hypertertensives when compared with normotensives.

DISCUSSION

Hypertension is recognized globally as a major public health problem.[17] It is a well known risk factor for coronary heart disease, type II diabetes mellitus and renal diseases.[18] Mortality caused by infectious disease is reported to be declining in developing countries whereas the toll of chronic degenerative diseases like stroke and myocardial infarction has been on the increase.[19]

Table 1: Age distribution of hypertensives and normotensives (percentage in parenthesis)

Age (Years)	Hypertensive			Normotensive		
	Male (%)	Female (%)	Total	Male (%)	Female (%)	Total
30-40	147(30.8)	120(27.6)	297(32.6)	84(30.1)	63(26.6)	147(48.5)
41-50	138(28.9)	114(26.2)	252(27.6)	42(15.1)	51(21.5)	93(18.0)
51-60	75(15.7)	99(22.8)	174(19.1)	72(25.8)	48(20.2)	120(23.3)
61-70	51(10.7)	61(14.5)	304(12.5)	60(21.5)	63(26.6)	123(23.8)
71-80	66(13.8)	39(8.9)	75(8.2)	21(7.5)	12(5.1)	33(6.4)
Total	477(52.3)	435(47.7)	912(100)	279(54.1)	237(45.9)	516(100)

Table 2: Serum lipid profile of hypertensive and normotensive patients

	Hypertensive n=912	Normotensive n=516	P Value
Total cholesterol (mmol/l)	5.21±2.17	4.06±0.83	0.86(*P*<0.05)
Triglycerides (mmol/l)	1.80±0.86	0.99±0.51	2.3(*P*<0.05)
HDL-Cholesterol (mmol/l)	1.15±0.25	2.10±0.30	-19.0(*P*<0.05)
LDL-Cholesterol (mmol/l)	3.64±1.14	1.51±0.84	7.1(*P*<0.05)
VLDL-Cholesterol (mmol/l)	0.82±0.39	0.45±0.23	2.3(*P*<0.05)

Table 3: Serum lipid profile of male hypertensives and normotensives

	TC (mmol/l)	TG (mmol/l)	HDL(mmol/l)	LDL(mmol/l)	VLDL(mmol/l)
Hypertensives	4.84± 2.10	1.68±0.67	1.36±0.58	2.72±1.30	0.76±0.30
Normotensives	3.91±1.30	0.97±0.26	2.32±0.72	1.15±0.92	0.44±0.13
P Value	11.6	1.73	6.85	4.13	1.88

Table 4: Serum lipid profile of female hypertensives and normotensives

	TC (mmol/l)	TG (mmol/l)	HDL (mmol/l)	LDL(mmol/l)	VLDL(mmol/l)
Hypertensives	4.40±1.85	1.87±0.88	1.27±0.61	2.28±0.84	0.85±0.40
Normotensives	4.20±1.31	1.01±0.36	1.88±0.21	1.86±0.92	0.46±0.32
P-Value	0.37	1.65	-1.53	-5.25	4.88

Abnormalities in serum lipid and lipoprotein levels (dyslipidaemia) are recognized as major modifiable cardiovascular disease (CVD) risk factors[20] and have been identified as independent risk factors for essential hypertension giving rise to the term dyslipideamic hypertension.[21] About 80% of hypertensive persons have co-morbidities such as obesity, glucose intolerance, hyperisulineamia, low HDL-C, High LDL-C and increased triglycerides.[22] Our study examined the pattern of lipid profile of hypertensive patients compared with normotensive individuals in the Niger-Delta region, South-South, Nigeria. Our study showed significantly increased total cholesterol (TC) in the hypertensives when compared with normotensives. This is in accordance with Asaolu *et al.*[4] who did a similar work in Ado Ekiti. Adu[23] in his work attributed hypercholesterolemia to defective regulatory response of 3-hydroxy-3- methylglutararyl-CoA reductase and hepatic cholesterol 7α-hydroxylase.[24,25,26] These enzymes are rate-limiting enzymes in cholesterol biosynthesis and catabolism to bile acids in human respectively. Hypertensive patients in this study shows hypertriglyceridemia when

compared to normotensives. This is consistent with the report of previous authors.[24,25,26] This high level of triglycerides can be attributed to down- regulation of lipoprotein lipase which is found in skeletal muscle, myocardium and adipose tissue, which are the principal site of storage.[23] Hypertriglyceridemia is a risk factor for stroke as there is increase in chylomicrons and very low- density lipoprotein which can lead to blockage of blood vessels hence resulting in stroke as found in hypertensives.[27] Low density lipoprotein (LDL-C) of hypertensives was significantly increased when compared with controls. This is in agreement with work of Festus *et al.*[27] This increased LDL-C can be explained by severe reduction of hepatic LDL receptor protein abundance despite normal LDL receptor mRNA abundance and gene translation rate.[23] This point to inefficient translationand/or increased LDL receptor protein turnover as a cause of LDL receptor deficiency. Based on the critical role of LDL receptor, acquired LDL receptor deficiency will contribute to hypercholesterolemia, elevation of plasma LDL-C, and impaired LDL clearance.[28] Hypertensives show significantly increased level of Very low density lipoprotein (VLDL-C) when compared with normotensive individuals. This is in accordance with Osuji *et al.*[25] High density lipoprotein cholesterol (HDL-C) in hypertensive patients was significantly decreased than in normotensive individuals. This is consistent with earlier studies in parts of the world and in other parts of Nigeria.[4,22,24,25,26,] Low serum HDL-C levels contribute to structural and functional alterations which led to arterial rigidity.[29] Studies carried out in animal models and humans[30] showed that low HDL-C levels are associated with significant endothelial dysfunction and compromised peripheral vasodilatation. The cause of increased lipids except HDL-C in hypertensive can be attributed to the influence exerted on lipids especially cholesterol by the renin angiotensin system. This greatly affects lipid homoestasis in hypertensives.[31]

The results of our investigation on normotensive individuals confirm previous reports that healthy Nigerians have significantly lower lipid profile than Caucasians.[19,32] The lipid pattern of male subjects was observed to be lowered statistically except HDL-C that is increased when compared with female of both hypertensive and normotensive individuals. This sex difference is attributed to the presence of testosterone in males which reduces cholesterol level and estrogen in females which increases cholesterol level.[33]

High levels of serum cholesterol are known to increase the risk of coronary heart disease (CHD) and stroke.[34] Epidemiological studies indicate a progressive increase in CHD risk as the serum total cholesterol exceeds 5.0mmol/l.[35] It is therefore recommended that the treatment of hypertension should, in addition to lowering arterial blood pressure, focus on modifying dyslipidaemia.

Based on our results obtained from this study, we therefore affirm that the lipid profile of the inhabitants of the Niger-Delta region, South–South, Nigeria does not differs significantly from the lipid profile of other Nigerians as earlier proposed by various authors. This low lipid profile among the Niger-Delta inhabitants can be attributed to palm oil consumption which contain olein, a hypocholesteroleamic compound.[36] Palm oil is found in large quantity in the Niger Delta region due to the fact that it is one of their agricultural produce.[37] It exerts hypocholesterolemic effect on total cholesterol, triglycerides and LDL-cholesterol and exerts hypercholesterolemic effect on HDL-cholesterol.[37] Also, the ingredients used in the preparation of food have direct blood cholesterol lowering effect by affecting pathways that regulate whole body cholesterol metabolism.[36] Dietary fibre, soy protein, plants sterol, rice oil, oryzanol and other ingredients in traditional diets have a positive association for regulating whole-body metabolism.[36] The constituents of these ingredients have been shown to inhibit 3-hydroxy-3-methylglutaryl-CoA reductase (HMG-CoA) which is the rate–limiting enzyme in cholesterol biosynthesis and enhances higher activities of LDL receptors which in turn lower the LDL-cholesterol concentration in the body.[38] Bandana[39] in a report advocated the use of mustard oil for cooking due to cholesterol lowering effects. Also the serum lipid profile especially total cholesterol, triglycerides and LDL-cholesterol levels have positive association or correlation with hypertension. Higher levels of these lipids may be due to physical inactivity, consumption of dietary fat, increase stress and consumption

of alcohol. This study has been able to reveal that dyslipidaemia is majorly the cause of hypertension because low HDL-C contributes to structural and functional alterations which led to arterial rigidity. The hardened arteries, in turn, lead to an increase in the speed of return of arterial pressure waves reflected from the peripheral circulation, so that there is an increase in peak systolic blood pressure, which ultimately leads to hypertension. Also most of the local food and ingredients been consumed in the Niger Delta region are natural hypolipiduric which has tremendous effects on the lipid pattern of this region.

We therefore advocate routine lipid profile testing as well as consumption of local palm oil and ingredients for all hypertensive patients in order to nip lipid derangement in the bud to prevent complications such as coronary heart disease.

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