Original Article

Increased lipid profile in hypertensive Nigerian males: A possible risk for atherosclerosis

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ABSTRACT

Objectives: This study was aimed at evaluating the risk of atherosclerosis in hypertensive Nigerians attending Ebonyi State University Teaching Hospital, using total cholesterol/HDL ratio.

Methodology: A sex-adjusted assessment of the total cholesterol, high density lipoprotein, and total cholesterol/HDL was performed on 82 hypertensive (45 males; 37 females) and 55 normotensive control (29 males; 26 females) Nigerians, aged between 30-70 years. Analysis of the total cholesterol and high density lipoproteins were by the routinely employed enzymatic and phosphotungstate-phosphomolybdate methods respectively.

Results: Mean total cholesterol (4.9±1.08 mmol/L) and total cholesterol/HDL (5.16±1.89) were significantly higher ($P < 0.05$) in hypertensive males and than in the controls (4.40±0.99 mmol/L and 3.77±1.25 respectively), while the mean HDL concentration was significantly ($P < 0.05$) lower (1.03±0.33 mmol/L) in the hypertensive males than in their control (1.26±0.39 mmol/L) counterparts. In females, the total cholesterol (4.98±1.02 mmHg) HDL (1.12±0.39 mmHg), and total cholesterol/HDL (4.94±1.82) were statistically not different (P > 0.05) from 4.86±1.05 mmHg, 1.29±0.44 mmHg, and 4.09±1.32, respectively obtained from the controls. Also, no sex-adjusted difference was observed in either of the hypertensive or control subjects.

Conclusion: These data underscore the relevance of routine assessment of lipid levels in hypertensive patients, especially in males, to detect increasing risks of atherosclerosis and its sequela.

KEY WORDS: Atherosclerosis, Hypertension, Nigerians, Cardiovascular disease, Cholesterol, Normotensive.

How to cite this article:

Nwosu ZC, Edeogu OC. Increased lipid profile in hypertensive Nigerian males: A possible risk for atherosclerosis. Pak J Med Sci 2011;27(2):269-272

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INTRODUCTION

Atherosclerosis is the hardening of the tunica intima and media of arteries occasioned by the deposition[1-3] of plaques containing cholesterol and its esters, lipoid materials, and lipophages. It is a leading cause of cardiovascular morbidity in developed world,[4-6] but a low incidence has been reported in Nigeria.[7] While the etiology of atherosclerosis is uncertain, hypotheses like the response to injury,[3] and the response to retention hypothesis[8] independently support its pathogenesis, and acknowledge atherosclerosis as an end product of complex inflammatory events.[2] Hypercholesterolemia resulting from imbalanced mobilization of cholesterol account for the earliest stages of atherogenesis, is risk factor for

CVD and is said to interfere with blood pressure regulation.[9] Glass and Witzum[10] concurred to the fact that hypercholesterolemia alone is significant to stimulate lesion development even if other risk factors are absent.

Controlling atherosclerosis and its long-term sequela would entail the control of cholesterol accumulation in blood – feasibly by stepping-up the consumption of dietary fibres, which interferes with the absorption of dietary fat and cholesterol,[11] additives like garlic is recommended in the prevention[12-13] of atherosclerosis, and physical activity. On the other hand, the incidence of hypertension in Nigeria is on the rise,[14] coupled with the fear that the global burden may reach 29% by 2025 (about three-quarter of which is projected to be in developing countries).[15] It is also a concern as the risk of coronary heart disease increases with blood pressure.[4,6,16,17] Hypertension has also remained an important cause of adult morbidity and mortality in sub-Saharan Africa.[18-20]

This study was aimed at estimating the level of the lipid profile hypertensive Nigerians as index for increased risk of atherosclerotic vascular complications.

METHODOLOGY

Study Population: A total of 137 subjects aged between 30-70 were recruited for the purpose of this investigation. They included 82 age and sex matched hypertensive patients in medical out-patient, and 55 normotensive subjects attending general out-patient clinic for minor ailments in Ebonyi State University Teaching Hospital in Abakaliki, the Ebonyi State capital, Nigeria. The subjects were defined as hypertensive using sphygmomanometer, on the basis of systolic to diastolic blood pressure of 140/90 mm Hg or more, while the control group was classified on the basis of a blood pressure less than 140/90 mm Hg.

Design: Ethical approval as well as the consent of the subjects was obtained. Following the acquisition of a verbal consent, all the studied subjects were given brief orientation on the role of serum cholesterol in the pathogenesis of vascular diseases. Exclusion criteria were drawn and adhered to with preference for only patients on *fast.* Also, obesity, pregnancy, treatment with lipid lowering drugs, cigarette smoking, diabetics, contraceptives, thiazides, sulfonamides, epinephrine, and androgens were part of the exclusion criteria since they all interfere with serum lipid concentration.[21] Efforts to convince quite a good number of the studied population on the usefulness of the questionnaires proved abortive and so most information as regards the patients status (most of which were geared towards knowing those who qualify to be recruited) were gathered by oral interview.

Sample Collection: Blood sample collection did commence in the morning after 12h fast a night to every sample collection day. During this time, patients were made to sit comfortably, while sample was drawn from the antecubital vein by venipuncture with minimum venous stasis using 2ml syringe. The blood samples were thereafter emptied into well labeled plain borosilicate bottle. The samples were all allowed to clot and serum fraction collected after centrifugation and stored frozen after been put in a clean dry stoppered plastic tube.

Laboratory Analysis of Sera: Before the actual time of analysis, the samples were allowed to thaw by keeping at room temperature. Each serum sample was analyzed for total cholesterol and HDL using enzymatic and phosphotungstate-phosphomolybdate precipitation methods respectively. Quantitation of the analytes (total cholesterol and HDL) was done after complete colour development using spectrophotometer at an absorbance of 500 nm, while total cholesterol/HDL ratio was worked out. All precautions were taken, and standard operational procedures duly followed to ensure accuracy and precision. All values were statistically compared using Biostat statistical software 2008, and expressed as mean±SD at 95% confidence interval *(P<0.05).*

RESULTS

A total of 137 adults aged between 30-70 years, and attending clinics in Ebonyi State University Teaching Hospital, Abakaliki Nigeria were recruited for this investigation. This figure represented 82 hypertensive subjects and 55 normotensives – the former made up of 45 males and 37 females, and the latter 29 males

Table-I: Mean+SD of The Serum Total Cholesterol, HDL-C, and Total Cholesterol/HDL Ratio in Hypertensive and Normotensive Male Subjects.

Parameters	Hypertensive N=45	Control N=29	P
Age (yrs)	49.40±10.85	49.44±12.86	p>0.05
Tchol (mmol/L)	4.90±1.08	4.40±0.99	p>0.05
HDL (mmol/L)	1.03±0.33	1.26±0.39	p<0.05
Tchol/HDL	5.16±1.89	3.77±1.25	p<0.05

Tchol – Total Cholesterol, HDL – High Density Lipoprotein; conversion of mmol/L to mg/dl, multiply by a factor of 38.6

Table-II: Mean+SD of The Serum Total Cholesterol, HDL-C, and Total Cholesterol/HDL Ratio
in Hypertensive and Normotensive Female Subjects.

Parameters	Hypertensive N=37	Control N=26	P. Value
Age (yrs)	52.00±11.36	49.65±12.86	p>0.05
Tchol (mmol/L)	4.90±1.08	4.40±0.99	p>0.05
HDL (mmol/L)	1.12±0.39	1.29±0.44	p>0.05
Tchol/HDL	4.94±1.82	4.09±1.32	p>0.05

Tchol – Total Cholesterol, HDL – High Density Lipoprotein Conversion of mmol/L to mg/dl, multiply by a factor of 38.6

and 26 females. The mean±SD comparison of total cholesterol, HDL, and total cholesterol/HDL for the hypertensive (45) and control (29) males are shown in Table 1. The result showed HDL level to be significantly lower (1.03±0.33 mmol/L and 1.26±0.39 mmol/L), while total cholesterol and total cholesterol/HDL were significantly higher (P<0.05) in the hypertensive males (4.90±1.08 mmol/L and 5.16±1.89 respectively).

Table-II shows the comparison of the Mean+SD in hypertensive (37) and control (26) females. Here, there is no significant difference (P>0.05) in the mean total cholesterol, (4.98±1.02 mmol/L vs 4.86±1.05 mmol/L), HDL (1.12±0.39 mmHg vs 1.29±0.44 mmol/L), and Tchol/HDL (4.94±1.82 vs 4.09±1.32) between the hypertensive females and their controls.

Tables-III and IV show unique results; a sex-adjusted comparison of the mean total cholesterol, HDL, and Tchol/HDL ratio in either of hypertensive and control subjects showed that there is no significant difference (P>0.05) attributable to sex within either of the categories.

DISCUSSION

The involvement of lipids, especially cholesterol[1-3,8] in atherosclerosis has been exclusively supported by many studies as well as the role of the latter in coronary heart disease.[4-6] However, literatures on these topics in Nigeria is seemingly insufficient. This study was initiated to evaluate total cholesterol, HDL, and total cholesterol/HDL levels, which could serve as assessment tools for risk of atherosclerosis in hypertensive subject. It was observed that hypertension is associated with significantly increased total cholesterol, Tchol/HDL ratio, and a significantly decreased HDL in the male subjects. This is a concern because

many studies have incriminated increased serum cholesterol in pathogenesis of atherosclerosis, although diets and other lifestyle modifications to reduce the serum level of the marker has been proved to produce a significant reduction in the prevalence of the condition.[12,22-24]

Contrarily, HDL is known to play the role of mopping-up cholesterol from the system thus making it a valuable parameter in controlling the risk of atherosclerosis,[25,26] and had been reported to be low in hypertensive patients.[27] But, Lee et al[16] stated that lower HDL may show a lesser but yet increased risk of CHD. Wilson et al[4] asserted that the rate of coronary heart disease is significantly associated with the specified categories of blood pressure, total cholesterol, HDL, and LDL, while Tannasescu et al[28] specifically associated high serum cholesterol with increased risk of CVD in type 2 diabetics. It has also been stated that serum cholesterol may influence the regulation of blood pressure by adrenergic stimulation.[9] A comparison of total cholesterol and HDL in female hypertensive and their control counterpart showed no associated changes in the parameters, as well as was the case when a sex-adjusted analysis was done within the hypertensive and normotensive control groups.

In conclusion, the finding of significantly higher total cholesterol, Tchol/HDL, and lower HDL in hypertensive males points to higher risk of atherosclerosis and other vascular complications. This is because, while total cholesterol is directly associated with increased risk of accumulation, HDL prevents such gradual deposition, and the ratio of the two reflects the overall cholesterol influx and efflux from susceptible vessels. Emphasis should be laid on the importance of routinely monitoring serum lipid profile in

Table-III: Sex-adjusted Comparison of the Mean+SD of Tchol, HDL, and Tchol/HDL in Hypertensive Subjects.

Parameters	Male N=45	Female N=37	P. Value
Age (yrs)	49.40±10.85	52.00±11.36	p>0.05
Tchol (mmol/L)	4.90±1.08	4.98±1.02	p>0.05
HDL (mmol/L)	1.03±0.33	1.12±0.39	p>0.05
Tchol/HDL	5.16±1.89	4.94±1.82	p>0.05

Tchol – Total Cholesterol, HDL – High Density Lipoprotein Conversion of mmol/L to mg/dl, multiply by a factor of 38.6

Table-IV: Sex-adjusted Comparison of the Mean+SD of Tchol, HDL, and Tchol/HDL
in Normotensive Control Subjects.

Parameters	Male N=29	Female N=26	P. Value
Age (yrs)	49.44±12.86	49.65±12.86	p>0.05
Tchol (mmol/L)	4.40±0.99	4.86±1.05	p>0.05
HDL (mmol/L)	1.26±0.39	1.29±0.44	p>0.05
Tchol/HDL	3.77±1.25	4.09±1.32	p>0.05

Tchol – Total Cholesterol, HDL – High Density Lipoprotein Conversion of mmol/L to mg/dl, multiply by a factor of 38.6
NS-Not Significant

all hypertensive Nigerians, to avert consequent vascular complications.

ACKNOWLEDGEMENT

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