

DISORDERS OF LIPIDS AMONG NEWLY DIAGNOSED HYPERTENSIVE PATIENTS IN MAIDUGURI,
NORTHEASTERN NIGERIA.

*MA Talle **O Enyikwola ***A Adelaiye *H Yusuph *MM Baba *B Bakki *BW Goni

Department of *Medicine and **Human Physiology, College of Medical Sciences, University of Maiduguri; ***
Department of Human Physiology, Faculty of Medicine, Ahmadu Bello University Zaria, Nigeria.

Correspondence:

Dr Mohammed A Talle,

Division of Cardiovascular Medicine, Department of Medicine,

College of Medical Sciences,

University of Maiduguri,

PMB 1069, Maiduguri. Nigeria.

E-mail: abdultalle@yahoo.com

ABSTRACT

Background: The prevalence of systemic hypertension is on the increase worldwide. Clustering of hypertension and disorders of lipid accelerates the process of atherosclerosis, leading to increased cardiovascular morbidity and mortality. Assessment of lipids is an essential component of patient evaluation in hypertension.

Objectives: To determine the plasma lipids of untreated hypertensive patients presenting at the cardiology clinic of the University of Maiduguri Teaching Hospital.

Methods: One hundred and fifty untreated hypertensive patients (90 males and 60 females) aged 35 to 55 years were consecutively enrolled. Blood pressure and anthropometric parameters were measured using standard procedures. Fasting blood sample was collected for the determination of serum lipids electrolytes, BUN and blood glucose. Data was analyzed using SPSS version 11 for windows.

Results: The mean (\pm SD) age of the patients was 45.10 ± 6.36 years. Mean Systolic and diastolic blood pressures were 158.64 ± 17.82 mmHg and 103.04 ± 9.40 mmHg respectively. Mean BMI was 27.46 ± 0.39 Kg/m² and 24.59 ± 4.78 Kg/m². About 51% and 17% were overweight and obese respectively. Thirty percent (30%) of patients had hypercholesterolaemia whereas 40% had hypertriglyceridaemia. Combined hypertcholesterolaemia and hypertriglyceridaemia was observed in 24%.

Conclusions: Disorders of lipid is common amongst hypertensive patients in this environment. Effective management of patients with hypertension should include assessment and management of disorders of lipids.

Keywords: *Hypertension; Disorders of lipid; Maiduguri.*

INTRODUCTION

The prevalence of cardiovascular diseases in general and coronary heart disease in particular is on the increase in Nigeria.¹ This is in tandem with the projected escalation in the global burden of cardiovascular diseases, especially in the developing countries where demographic transition is being experienced.² Factors responsible include among others, the demographic shift with altered population age profiles, life style changes (altered diet, decreased physical activity and tobacco use) due to urbanization and industrialization.³

Patients with multiple cardiovascular risk factors are at an increased risk for cardiovascular diseases (CVD) compared with those having single risk factor. The clustering of hypertension and dyslipidaemia, both a substrate for the development and progression of atherosclerotic vascular disease has been reported in a number of studies.^{4,5} Atherosclerosis remains the major underlying predisposition to myocardial infarction and stroke.

The concept of total cardiovascular risk assessment as being advocated in patients with hypertension has determination of serum lipids as a major component.⁶ Studies from northwestern and southern parts of Nigeria have variously documented an increasing prevalence of dyslipidaemia amongst hypertensive patients⁷⁻⁹. There is paucity of information on serum lipids in the northeastern part of Nigeria. Given the heterogeneity of the Nigerian population with different socio-cultural background including dietary habits and the impact it has on lipid metabolism, it is imperative to look at the pattern of disorders of lipids in treatment naïve hypertensive patients in our environment.

MATERIALS AND METHODS

The study was carried out at the University of Maiduguri Teaching Hospital, Nigeria following an approval by the Research and Ethics Committee of the Teaching Hospital.

One hundred and fifty newly diagnosed hypertensive patients aged 35 to 55 years (comprising 90 males and 60 females) consecutively referred to the cardiology clinic were enrolled. Those with identified secondary causes of hypertension and dyslipidemia (diabetes mellitus, chronic kidney disease, liver disease etc) were excluded. Other exclusion criteria include cigarette smoking and alcohol consumption. Blood pressure was measured with mercury sphygmomanometer in the sitting position using standard protocol and hypertension defined as systolic blood pressure of ≥ 140 mmHg and / or diastolic blood pressure of ≥ 90 mmHg. Blood pressure was classified using the JNC VII guideline.⁶

Anthropometric measurements were carried out using standard procedures and Body Mass Index (BMI) calculated from weight (in Kg) and height (in m²). Classification of BMI was based on international diabetes federation (IDF) guidelines.¹⁰ Fasting blood sample was collected by venepuncture from the non-dominant arm and conveyed to the laboratory within three hours of collection. The enzymatic method was used to analyze for total cholesterol and triglycerides.¹¹ High density lipoprotein -cholesterol (HDL-C) was determined by precipitation method while Friedewald equation was used to calculate LDL-cholesterol (LDL-C).¹² The National Cholesterol Program Adult Treatment Plan III was used in defining dyslipidaemia.¹³

The data was analyzed using SPSS version 11 for windows (SPSS, III, Chicago, USA). Two-way analysis of variance was used in comparing mean values of the male and the female patients and results expressed as Mean \pm standard deviation (SD). P value of ≤ 0.05 was considered significant.

RESULTS

Table 1 is a summary of the demographic characteristics and a comparison of the serum profiles of the patients. Their ages ranged from 35 to 55 years with a mean of 45.10 ± 6.36 years. The mean ages of the male and female patients were 45.83 ± 6.83 and 44 ± 5.46 years respectively ($p > 0.05$)

The mean total cholesterol was 4.64 ± 1.02 mmol/L. Females had a mean total cholesterol of 4.73 ± 1.06 mmol/L compared to 4.58 ± 0.99 mmol/L for the males ($p > 0.05$). The mean HDL-C was 1.06 ± 0.29 mmol/L whereas the mean LDL-C was 3.05 ± 1.02 mmol/L. Females had a mean LDL-C of 3.16 ± 1.19 mmol/L and was significantly higher than the male mean LDL-cholesterol of 2.98 ± 0.89 mmol/L ($p < 0.05$). Mean TG was 1.47 ± 0.53 mmol/L. Male patients had a mean TG of 1.48 ± 0.63 mmol/L compared to 1.44 ± 0.34 mmol/L for the females ($p > 0.05$). The different classes of cholesterol are illustrated in tables 2, 3 and 4.

The mean Atherogenic Index (AI) was 5.27 ± 3.93 . Male patients had a mean AI of 5.49 ± 4.31 while that of the females was 4.93 ± 3.30 ($p > 0.05$). Twenty nine (19.33%) patients made up of 18 (12%) males and 11 (7.33%) females had AI greater than 5.8.

Mean fasting blood sugar (FBS) was 4.63 ± 0.87 mmol/L. The mean FBS for the male patients was 4.74 ± 0.09 mmol/L and slightly higher than the female value of 4.47 ± 0.12 mmol/L ($p > 0.05$). Eight (5.33%) patients had FBS of 6.1 mmol/L but none had FBS in the diabetic range.

Fifty four (36%) patients had hypercholesterolaemia whereas hypertriglyceridaemia was observed in 60 (40%). Combined hypercholesterolaemia and hypertriglyceridaemia was observed in 36 (24%) of patients. Forty four (29.33%) and 49 (32.67%) of the males and females had low HDL-cholesterol, while 2 (1.33%) had a high HDL-C (Table 5).

Mean BMI was 27.46 ± 0.39 kg/m². Thirty eight (26%) patients, made up of 23 (58.97%) males and 16 (41.03%) females had a BMI of less than 25kg/m², while 77 (51.33%) comprising 51 (66.23%) males and 26 (33.77%) females were overweight. The overall prevalence of obesity was 22.63%. Twenty six (17.33%) made up of 13 (50%) males and 13 (50%) females had class 1 obesity whereas six (4%), made up of 2 (33.33%) males and 4 (66.67%) females had class 2 obesity. Only 2 (1.3%) of the patients made up of 1 (50%) male and 1 (50%) female had class 3 obesity. The mean BMI of the females was 27.58 ± 5.38 kg/m² compared to 27.39 ± 4.38 kg/m² for the males ($p > 0.05$). Classification of BMI is illustrated in Table 6.

Table 1. Demographic and general characteristics of the patients

	Males (n=90)	Females (n=60)	<i>P</i>
Age (Years)	45.83± 6.83	44.00±5.46	>0.05
SBP (mmHg)	158.60±17.87	158.70±17.90	>0.05
DBP (mmHg)	103.18±9.81	102.83± 8.84	>0.05
TC (mmol/L)	4.58±0.99	4.73±1.06	>0.05
LDL (mmol/L)	2.98±0.89	3.16±1.19	<0.05
HDL (mmol/L)	1.02±0.32	1.12±0.24	=0.05
TG (mmol/L)	1.48±0.63	1.44±0.34	>0.05
AI	5.49±4.31	4.93±3.30	>0.05
FBS (mmol/L)	4.74±0.82	4.47±0.94	>0.05
BMI (Kg/m ²)	27.39±4.36	27.58±5.38	>0.05

n=Number; SBP=systolic blood pressure; DBP=diastolic blood pressure; TC=total cholesterol; LDL=low density lipoprotein; HDL=high density lipoprotein; TG=triglyceride; AI=atherogenic index; FBS=fasting blood sugar; BMI=body mass index

Table 2. Classification of Total cholesterol

Total cholesterol (mmol/L)	Males	Females	Total
<5.17	60	36	96
5.17 – 6.18	27	20	47
>6.18	3	4	7

Table 3. Classification of LDL cholesterol

LDL cholesterol (mmol/L)	Males	Females	Total
<2.59	30	22	52
2.59 – 3.34	32	10	42
3.35 – 4.11	21	13	34
4.12 – 4.89	2	8	10
≥4.90	5	7	12

LDL=low density lipoprotein

Table 4. Classification of Triglyceride in the patients

Triglyceride (mmol/L)	Males	Females	Total
<1.7	50	40	90
1.7 – 2.25	32	20	52
2.26 – 5.63	8	0	8
≥5.64	0	0	0

Table 5. Prevalence of the different dyslipidaemia among patients

Type of dyslipidaemia	Males (%)	Females (%)	Total (%)
Hypertriglyceridaemia	40 (26.7)	20 (13.3)	60 (40)
Hypercholesterolaemia	30 (20)	24 (16)	54 (36)
LDL-Hypercholesterolaemia	28 (18.7)	28 (18.7)	56 (37.4)
HDL-Hypocholesterolaemia	44 (29.3)	49 (32.67)	93 (62)
HDL-Hypercholesterolaemia	0 (0)	2 (1.3)	2 (1.3)
Combined Hypercholesterolaemia And hypertriglyceridaemia	25 (16.7)	11 (7.3)	36 (24)
Abnormal AI	18 (12)	11 (7.3)	29 (19.3)

Legend: LDL=low density lipoprotein; HDL=high density lipoprotein; AI=atherogenic index

Table 6. Classification of Body Mass Index

Class	BMI	CV Risk	Males	Females	Total (%)
Underweight	<18.5	Increased	0(0)	10(7)	10(7)
Normal	18.5 - 24.9	Normal	23(15.3)	15(10)	38(25.3)
Overweight	25 - 29.9	Increased	51(34)	26(17.3)	77(51.3)
Obese					
I	30 – 34.9	High	13(8.7)	13(8.7)	26(17.4)
II	35.0 – 39.9	Very high	2(1.3)	4(2.7)	6(4.0)
III	≥40	Extremely high	1(0.7)	1(0.7)	2(1.4)

Legend: BMI=body mass index; CV= cardiovascular

DISCUSSION

Hyperlipidaemia is acknowledged to be a major risk factor for atherosclerosis and most of the evidences implicate hypercholesterolemia.¹³ The findings in this study are in keeping with reports from other parts of Nigeria where total cholesterol, LDL-C, TG and Atherogenic Index were found to be high among hypertensive patients.⁷⁻⁹

High serum TC level in hypertensive patients as observed in this study confers an additional cardiovascular risk. The finding of higher TC and LDL-C among females is at variance with the reports of workers in other parts of Nigeria, where it is reported to be higher in males.^{8,9,14,15} This reflects the general inconsistencies on the reports of the influence of gender on serum lipids.^{16,17}

High levels of LDL cholesterol have been implicated as being atherogenic by epidemiological investigations of human populations. A direct relationship has been established between levels of LDL cholesterol (or total cholesterol) and the rate of new onset coronary heart disease (CHD) in the Framingham Heart Study, the Multiple Risk Factor Intervention Trial (MRFIT), and the Lipid Research Clinics (LRC) trial.^{18,19,20}

The relatively low levels of HDL-C is worthy of note given its protective role in cardiovascular diseases.^{6,14} Females had a significantly higher HDL-C than males. This is in contrast with previous reports where the difference was not significant.^{9,14} The Helsinki heart study has demonstrated a strong negative association between HDL-C and cardiovascular diseases.²¹

The higher TG observed in the males is in keeping with the findings of previous workers.^{9,14,15} Positive associations between triglyceride concentration and the risk of CHD have been observed in many case-control and prospective studies.^{22,23} The literature on epidemiological association between plasma triglyceride and CAD is not consistent²⁴. Biological plausibility and epidemiological data

suggest that triglyceride might promote coronary heart disease and other forms of cardiovascular disease, but the epidemiological and clinical evidence is inconsistent and often flawed.²⁵ A cross-sectional multinational study by WHO have shown that TG levels were significantly higher among subjects with major Q-wave abnormalities compared to subjects without.²⁶ Going by the study of Lindenstrom and colleagues who suggested an association between serum TG and stroke, the co-existence of elevated TG and hypertension (each being a risk factor for stroke) in the subjects worsens the prognosis.²⁷

The TC: HDL (atherogenic index) ratio is an independent, potent, and sensitive predictor of atheroma-formation. The finding in this study is in keeping with the report of previous workers.^{7,8} The higher Atherogenic index in males reflects a comparatively lower level of HDL-C, and partly accounts for their higher risk of atheroma formation and CHD.²⁸

Studies have documented an increased prevalence of age-adjusted myocardial infarction, cerebrovascular disease, peripheral artery disease and coronary artery disease among patients with concomitant dyslipidaemia and hypertension than either in isolation.²⁹ Similarly, targeting dyslipidaemia and hypertension can lead to significant reductions in the prevalence of cardiovascular events.³⁰

The high prevalence of overweight and obesity observed among the hypertensive patients reflects the trend reported in other parts of the country.^{15, 16} A positive correlation has been established between BMI, hypertension, and disorders of lipid except HDL-C where the correlation was negative.³¹ Similarly, clinical trials have shown the effect of weight loss, and hence reduction in BMI, on lowering blood pressure and improving lipid values.³²

Previously thought to be uncommon, disorders of lipid are common among hypertensive patients in this environment and compares with what obtains in other parts of the country. The clustering of hypertension with dyslipidaemia and other cardiovascular risk factors is somewhat a harbinger of an outbreak of cardiovascular diseases, especially in the setting of improved socio-economic circumstances. This calls for an aggressive public enlightenment against the projected escalation in cardiovascular disease including community-based management strategies especially life style modification. The concept of total risk assessment in hypertensive patients as contained in standard guidelines^{6, 33} should also be strictly adhered to.

REFERENCES

1. Odia OJ. Cardiovascular risk factors in Black Africans with ischaemic heart disease. *Niger. Med. J.*, 1995; 28: 119-121
2. Srinath RK. Cardiovascular diseases in the developing countries: Dimensions, determinants, dynamics, and direction for public health action. *Public Health Nutrition*, 2001; 5(1A): 231-237
3. Murray CJL, Lopez AD. Global comparative assessment in the Health sector. Geneva: WHO.1994
5. Rantala AO, Kauma H. Prevalence of Metabolic Syndrome in drug-treated hypertensive patients and control Patients. *J. Int. Med.*, 1999; 245: 163-174
6. Aram VC, George LB, Henry R *et al.*, The seventh report of the Joint National Committee on prevention, detection, evaluation and treatment of high blood pressure. The JNC 7 Report. *JAMA*, 2003; 289(19): 2560-2572
7. Isezuo SA, Badung SLH. Plasma lipids among northwestern hypertensive patients. *Sahel Medical Journal*, 2001; 4(4); 181-186
8. Ajuluchukwu JNA, Jarikre AE, Anorlu IR. Plasma lipid profiles of menopausal Nigerian hypertensive women. *Journal of clinical sciences*, 2001; 1(1): 15-17
9. Agboola-Abu CF, Onabolu A. Plasma lipid levels in patients attending Igbinedion hospital and medical research centre, Okada, Edo state, Nigeria. *Niger Med J*, 2000; 38(1):1-5
10. Zimmet P, Alberty G, Shaw J. A new IDF worldwide definition of metabolic syndrome. *Diabetes Voice*, 2005; 50 (3):31-33
11. National Cholesterol Education Program. Recommendations on lipoproteins measurement. NIH/NHLBI, Bethesda, National Institute of Health. 1995

12. Friedewald WT, Levy RI, Friedrickson DS. Estimation of the concentration of low density lipoprotein cholesterol without the use of the preparative ultracentrifuge. *Clin. Chem.* 18; 499. 1972
13. James IC. Executive Summary of the 3rd report of the National Cholesterol Education Program (NCEP). Expert panel on detection evaluation and treatment of high blood cholesterol in Adults (Adult Treatment Panel III). *JAMA*, 285(19): 2486-2497. 2001
14. Jarikre AE, Dim DC, Ajuluchukwu JNA. Plasma lipids in Nigerian hypertensive patients: The gender factor. *Nigerian Quarterly Journal of Hospital Medicine*, 1996; 6(4): 293-298
15. Ukoh VA, Oforofuo LAO. Fluvastatin and hyperlipidaemia: Effects on hypertensive patients with myocardial ischaemia. *Nigerian Medical Practitioner*, 2003; 43(1): 12-16
16. Aduba O, Onwamaeze I, Oli J, Udeozo K. Serum cholesterols and high density lipoprotein cholesterol in Nigerian diabetics. *E. Afr. Med. J.*, 1984; 61: 35-39
17. Jarikre AE, Ofogba CJ, Emuvegan EE. Reference values for the nutritional indices in urbanized adult Nigerians living in the Lagos area. *Journal of Clinical Practice*, 1998; 1(1): 22-25
18. Wilson PWF, D'Agostino RB, Levy D, Belanger AM, Silbershatz H, Kannel WB. Prediction of coronary heart disease using risk factor categories. *Circulation*, 1998;97:1837-47
19. Stamler J, Wentworth D, Neaton JD, for the MRFIT Research Group. Is relationship between serum cholesterol and risk of premature death from coronary heart disease continuous and graded? Findings in 356,222 primary screenees of the Multiple Risk Factor Intervention Trial (MRFIT). *JAMA*, 1986; 256:2823-8

20. Lipid Research Clinics Program. The Lipid Research Clinics Coronary Primary Prevention Trial results. I: Reduction in the incidence of coronary heart disease. JAMA, 1984;251:351-64
21. Manttari M, Elo O, Haapa K *et al.*, The Helsinki Heart Study: Basic design and randomization procedure. Eur. Heart J.,1987; 8(suppl 1): 1-29
22. Gotto AM, Gory GA, Thompson JR *et al.*, Relationship between plasma lipid concentrations and Coronary Artery Disease in 496 patients. Circulation, 1977;56: 875-883
23. Barbir M, Wile D, Trayner I, Aber VR, Thompson GR. High prevalence of hypertriglyceridaemia and lipoprotein abnormalities in Coronary Artery Disease. Br Heart J, 1988; 60: 397-403
24. Mellisa A A. Plasma triglyceride and Coronary Heart Disease. Arteriosclerosis and thrombosis, 1991; 11:2-14.
25. Carber AM, Avins AL. Triglyceride concentration and Coronary Heart Disease. BMJ, 1993; 309:2-3.
26. West KM, Ahuja MMS, Bennett PH, Cryzyk A, Deacosta DM, Faller JH et al. The role of circulating glucose and triglyceride concentration and their interaction with other risk factors as determinants of arterial disease in nine diabetic population samples from the World Health Organization multinational study. Diabetes Care, 1983; 6: 361-369
27. Lindenstrom E, Boysen G, Nyboe J. Risk factors for stroke in Copenhagen, Denmark I. Basic demographic and social factors. Neuroepidemiology, 1993; 12: 37-42
28. Chan MK. Lipid metabolism in renal failure. Clin. Biochem.,1990; 23:61-65
29. Michael LJ, Kenneth P, David SB, Rebecca JB. Prevalence of comorbid hypertension and dyslipidemia and associated cardiovascular disease. Am J Manag Care, 2004; 10: 926 – 932

30. Wald NJ, Law MR. A strategy to reduce cardiovascular disease by more than 80%. *BMJ*, 2003; 138: 1419 – 1423
31. Clarice DB, Millicent H, Kaven AD, Frederick CR, Robert G, Eva O et al. Body mass index and the prevalence of hypertension and dyslipidaemia. *Obesity Research*, 2000; 8(9): 605-619
32. Wood PD, Stefanick ML, Williams PT, Haskell WL. The effects on plasma lipoproteins of a prudent weight-reducing diet with or without exercise, in overweight men and women. *N Eng J Med*, 1991; 325:461-466
33. New Consensus Guidelines from European Society for Hypertension / European Society for Cardiology for the Management of arterial Hypertension (2007). *Journal of Hypertension*, 2007; 25(6): 1113 – 1124