# The Pattern of Plasma Lipid Profile In Untreated And Treated Hypertensives In Ilorin, Nigeria.

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**Abstract**

The pattern of plasma lipids was determined in 50 newly diagnosed hypertensives, 50 established hypertensives on treatment and 50 normotensives as control in order to evaluate the association of hypertension and hyperlipidaemia.

Plasma total cholesterol (TC), triglyceride (TG), high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C), fasting plasma glucose (FPG) and body mass index (BMI) were determined in the subjects.

Hypertensives had significantly higher mean plasma TC (newly diagnosed hypertensives; 4.54mmol/L 1.56, established hypertensives on treatment; 4.80mmol/L 1.51, controls; 3.20mmol/L 1.28), HDL-C (newly diagnosed; 1.28mmol/L 0.45, established hypertensives; 1.16mmol/L 0.37, controls; 0.93mmol/L 0.41), LDL-C (newly diagnosed; 3.21mmol/L 1.34, established hypertensives; 3.62mmol/L 1.30, controls; 2.28mmol/L 1.01) and BMI (newly diagnosed; 26.42kg/$m^2$ 4.75, established hypertensives; 27.20kg/m2 6.57, controls; 23.37kg/$m^2$4.75). FPG was only higher in established hypertensives (established hypertensives; 5.02mmol/L 1.11, controls; 3.72mmol/L 0.64) P 0.05.

Established male hypertensives on treatment had lower HDL-C (1.05mmol/L 0.25) than newly diagnosed hypertensives (1.31mmol/L 0.47) P 0.05. They also had a higher LDL-C (3.96mmol/L 1.42) than in newly diagnosed hypertensives (3.07mmol/L 1.20) P<0.05. Only in new diagnosed female hypertensives, was BMI (29.51kg/$m^2$ 5.01) significantly higher than in same sex control (22.93kg/$m^2$ 3.95) P<0.05.

Increase in plasma lipids observed in hypertension is not due to treatment, but rather a component of metabolic syndrome in which the disease is prominent. The poor lipid profile is worse in male patients on treatment in our environment. Increase in BMI is more relevant to the origin of hypertension in women.

**Key Words:** Lipid profile, newly diagnosed hypertensives, established hypertensives on treatment.

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## Introduction

Hypertension is one of the non-communicable diseases that pose an important threat to the health of the adult African.[1, 2] It is also recognised as the most prevalent cardiovascular disease (CVD)[3,4] and an acknowledged risk factor for the development of coronary heart disease (CHD).[4]

Closely linked to hypertension is hyperlipidaemia, which is either a component of a metabolic syndrome or as result of drugs used in its treatment.[5] The changes in lipoprotein metabolism among other metabolic derangements associated with hypertension, independently increases the risk of CHD.[6] Hence some workers have reasoned that such lipoprotein disorders that have not been considered in primary prevention trials, may account for observed poor results in patients with CHD compared to those with stroke.[6]

In Nigeria, the association between hypertension and hyperlipidaemia has been demonstrated.[7,8] These studies which were conducted in hypertensives on treatment, could not clearly state the origin of the increase in plasma lipids in hypertensives. Whether the observed hyperlipidaemia is due to treatment or as a component of a metabolic syndrome, need to be evaluated in our environment as it has a bearing on drugs prescribed for the treatment of hypertension.

The aim of this study was therefore, to determine whether the reported hyperlipidaemia in hypertensives, is associated with hypertension per se or is due to its drug treatment.

## Materials And Method.

This was a Cross-sectional Study done at the

University of Ilorin Teaching Hospital, Ilorin, Nigeria. The study involved three groups of subjects; newly diagnosed hypertensives, established hypertensives on treatment and normotensives controls.

Due to difficulty in recruiting untreated hypertensives in our hospital, screening for hypertension was carried out among workers in a Federal Secretariat and a Soft-drink Manufacturing Factory, in Ilorin metropolis. Blood pressure (BP) was recorded in sitting positions, after the patient have rested for ten minutes. Hypertension was defined as BP 140/90mmHg. In subjects with a BP 140/90mmHg, BP measurement was repeated the next morning. If BP remained at 140/90mmHg, such subjects were recruited as Untreated- Hypertensives if they were not receiving treatment for the disease. Among those workers with BP 140/90mmHg, we recruited the normotensive controls. The Treated Hypertensives were recruited from the Cardiology Clinic of our hospital. These were patients who have been receiving treatment for hypertension for at least one year. Any subject with a history suggestive of diabetes mellitus, chronic liver disease, thyroid disease, chronic renal disease, or those on oral contraceptives, was excluded from the study. Hypertensives on Doxazosin and/or Propanolol were also excluded. As most of the subjects were on thiazides, it was difficult to completely exclude those on thiazides.

All the subjects were fasted overnight for at least 10-12 hours, after which 10millitres (mls) of blood was collected from their ante-cubital fossae. About 7mls of the blood was put in EDTA-bottles and the other put in Fluoride oxalate bottles. The blood samples were spun at 2,500rpm for 10 minutes and their plasma separated into clean seperation bottles, which were stored at 20°C. The EDTA-plasma was used for the estimation of plasma total cholesterol (TC),[9] triglyceride (TG),[10] high density lipoprotein-cholesterol (HDL-C) [11] while the fluoride oxalate plasma was used for fasting plasma glucose estimation (FPG).[12] low density lipoprotein-cholesterol (LDL-C) was determined using the Friedwald Formula.[13]

We used standardized scales to measure subjects' weight in kilogram and height in metres. The values got were used to determine the body mass indices (BMI kg/m$^2$). Each subject had on minimal clothing before the body weight was recorded. To determine their heights, subjects stood against a marked wall with occiput, gluteus and calcaneus in contact with the wall and the top of the pinnae in line with the angle of the eye.

In all, 50 subjects were recruited for each of the study group. The results obtained were analysed using Epi-Info Version 6.0 statistical package. The means of the variables in the newly diagnosed hypertensives and established hypertensives on treatment groups were individually compared with the normotensives group. The two hypertensive groups (newly diagnosed hypertensives and established hypertensives on treatment) were also compared. The comparison of means was done using the Student T-test with $P \leq 0.05$ taken as point of significance.

**Results**

The mean age in years of controls (44.06 years 11.93), newly diagnosed hypertensives (45.26 years 7.63) and established hypertensives on treatment (43.38 years, 8.14) did not show any significant difference at $P < 0.05$. Most of the subjects who had hypertension were in the 40-59 years age bracket. More women were found to have hypertension at a younger age ( 40 years). Table 1.

The mean plasma TC in the newly diagnosed hypertensives (4.54mmol/L 1.56) was significantly higher than the value for controls (3.20mmol/L 1.28). P 0.05. The established hypertensives on treatment (4.80mmol/L 1.51) also had a significantly higher TC than the controls (3.20mmol/L 1.28). $P<0.05$. (Table 2) Among the hypertensives, there was no significant difference in plasma TC between males and females. (Table 3, 4)

The mean plasma TG value in the newly diagnosed hypertensives (1.00mmol/L 0.51) and established hypertensives on treatment (1.03mmol/L 0.62) did not differ significantly from the control value (0.87mmol/L 0.43). In newly diagnosed hypertensives, only the males had plasma TG level (1.12mmol/L 0.57) that was significantly higher than controls' (0.87mmol/L 0.28). $P< 0.05$. (Table 3) They also had a mean plasma TG value that was significantly higher than that of female newly diagnosed hypertensives (0.76mmol/L 0.24) $P< 0.05$). This trend was reversed among the established hypertensives on treatment, in which females had a significantly higher plasma TG (1.14mmol/L 0.71) than males (0.83mmol/L 0.29) P 0.05 (Table 4)

Mean plasma HDL-C was significantly higher in newly diagnosed hypertensives (1.28mmol/L 0.45) than in controls (0.93mmol/L 0.41). Similarly, established hypertensives on treatment had a significantly higher HDL-C (1.16mmol/L 0.37) $P< 0.05$. (Table 2) Plasma HDL-C levels were significantly higher in newly diagnosed female hypertensives (1.23mmol/L 0.41) than in

**TABLE 1: Age and sex distribution in the hypertensive groups and control.**

| AGE GRP (YEARS) | NORMOTENSIVE CONTROL | | NEWLY DIAGNOSED HYPERTENSIVE | | ESTABLISHED HYPERTENSIVES ON TREATMENT | |
|---|---|---|---|---|---|---|
| | Male (n) | Female (n) | Male(n) | Female(n) | Male(n) | Female(n) |
| 20-29 | 2 | 3 | 1 | 1 | - | - |
| 30-39 | 7 | 4 | 1 | 6 | 2 | 4 |
| 40-49 | 10 | 9 | 10 | 6 | 5 | 6 |
| 50-59 | 4 | 3 | 14 | 9 | 10 | 11 |
| 60-69 | 5 | 3 | 1 | 1 | 6 | 6 |
| **TOTAL** | **28** | **22** | **27** | **23** | **23** | **27** |

**TABLE 2:** Comparison of mean plasma lipids, FPG, and BMI of newly diagnosed hypertensives and established hypertensives on treatment with control.

| Parameters | Control Mean (SD) | New diagnosed hypertensives Mean (SD) | Control Mean (SD | Estab. Hypertensives on treatment Mean(SD) |
|---|---|---|---|---|
| TC(mmol/L) | 3.20 (1.28) | 4.54 (1.56)* | 3.20 (1.28) | 4.80 (1.51)* |
| TG(mmol/L) | 0.87 (0.43) | 1.00 (0.51) | 0.87 (0.43) | 1.03 (0.62) |
| **HDL-C(mmol/L)** | 0.93(0.41) | 1.28 (0.45)* | 0.93 (0.41) | 1.16 (0.37)* |
| **LDL-C(mmol/L)** | 2.28 (1.01) | 3.21 (1.34) | 2.28 (1.01) | 3.62 (1.30)* |
| FPG(mmol/L) | 3.72 (0.64) | 3.57 (0.76) | 3.72 (0.64) | 5.02 (1.11)* |
| BMI(kg/m$^2$) | 23.37 (4.75) | 26.42 (4.75)* | 23.37 (4.75) | 27.20 (6.57)* |
| **SYSTOLIC BP** | 119.50 (7.97) | 152.96 (15.63)* | 119.50 (7.97) | 139.70 (16.46)* |
| **DIASTOLIC BP** | 77.70 (4.54) | 96.00 (7.07)* | 77.70 (4.54) | 88.40 (9.34)* |

* = significant P 0.05
Estab = Established

**TABLE 3:** Mean plasma lipids, FPG and BMI compared by sex in newly diagnosed hypertensives and established hypertensives with their controls.

| Parameters | Newly diagn. male Hypertensive Mean(SD) | **Male Control Mean(SD** | Estab. male hypertensive on treatment Mean(SD) | Newly diagn. female Hypertensive Mean(SD) | **Female Control Mean(SD)** | Estab. Female Hypertensive on treatment Mean(SD) |
|---|---|---|---|---|---|---|
| TC(mmol/L) | 4.48 (1.43)* | 3.53 (1.39) | 4.84 (1.83)* | 4.65 (1.81)* | 2.77 (0.84) | 4.79 (1.34)* |
| TG(mmol/L) | 1.12 (0.57)* | 0.87 (0.28) | 0.83 (0.29) | 0.76 (0.24) | 0.88 (0.57) | 1.14 (0.71) |
| **HDL-C(mmol/L)** | 1.31(0.47)* | 1.05 (0.45) | 1.05 (0.25) | 1.23 (0.41)* | 0.77 (0.29) | 1.22 (0.42) |
| LDL-C(mmol/L) | 3.07 (1.20)* | 2.49 (1.230 | 3.96 (1.42)* | 3.48 (1.57)* | 2.01 (0.56) | 3.45 (1.22)* |
| FPG(mmol/L) | 3.65 (0.80) | 3.59 (0.68) | 5.34 (1.36)* | 3.72 (0.66) | 3.81 (0.55) | 4.86 (0.94)* |
| BMI(kg/m$^2$) | 24.82 (3.77) | 23.71 (5.35) | 26.28 (7.10)* | 29.5 (5.01)* | 22.93 (3.95 | 27.67 (6.34)* |
| SYSTOLIC BP | 154.64(17.70)* | 121.43(7.56) | 139.71(16.53)* | 149.71(10.23)* | 117.05(7.97) | 139.70(16.67)* |
| DIASTOLIC BP | 95.15 (7.55)* | 78.75 (3.76) | 89.71 (10.68)* | 97.65 (5.89)* | 76.36 (5.16) | 87.73 (8.67)* |

* = significant P 0.05
Estab. = Established
Diagn. = Diagnosed

**TABLE 4:** Comparison of mean plasma lipids, FPG and BMI in newly diagnosed hypertensives and established hypertensives on treatment and their sex differences

| Parameters | Newly diagn. hypertensive Mean(SD) | Estab. Hypert. on treatment Mean(SD) | Newly diagn. male hypert. Mean (SD) | Newly diagn. female Hypert. Mean(SD) | Estab male hypertensives on treatment Mean(SD) | Estab. female Hypertensives on treatment Mean(SD) |
|---|---|---|---|---|---|---|
| TC (mmol/L) | 4.54 (1.56) | 4.80 (1.51) | 4.48 (1.43) | 4.65 (1.81) | 4.84 (1.83) | 4.79 (1.34) |
| TG (mmol/L) | 1.00 (0.51) | 1.03 (0.62) | 1.12 (0.57) | 0.76 (0.24)* | 0.83 (0.29) | 1.14 (0.71)* |
| HDL-C (mmol/L) | 1.28 (0.45) | 1.16 (0.37) | 1.31(0.47) | 1.23 (0.41) | 1.05 (0.25) | 1.22 (0.42)* |
| LDL-C (mmol/L) | 3.21(1.34) | 3.62 (1.30) | 3.07 (1.20) | 3.48 (1.57) | 3.9 (1.42) | 3.45 (1.22) |
| FPG (mmol/L) | 3.57 (0.76) | 5.02 (1.11)* | 3.65 (0.80) | 3.72 (0.66) | 5.34 (1.36) | 4.86 (0.94) |
| BMI ($kg/m^2$) | 26.42 (4.75) | 27.20 (6.57) | 24.82 (3.77) | 29.51 (5.01)* | 26.38 (7.10) | 27.6 (6.34) |
| SYSTOLIC BP | 152.96(15.63) | 139.70(16.46)* | 154.64(17.70) | 149.71(10.23) | 139.71(16.53) | 139.70(16.67) |
| DIASTOLIC BP | 96.00 (7.07) | 88.40 (9.34)* | 95.15 (7.55) | 97.65 (5.89) | 89.71 (10.68) | 87.73 (8.67) |

* = significant P 0.05
Hypert. = Hypertension
Diagn. = diagnosis
Estab. = established

Table 5: Comparison of mean plasma lipids, FPG and BMI by sex in control group, newly diagnosed hypertensives and established hypertensives on treatment groups.

| Parameters | Male control Mean(SD) | Female control Mean(SD) | Newly diagn. male hypert. Mean(SD) | Estab. Male hypert. on treatment Mean(SD) | Newly diagn. female hypertensive on treatment Mean(SD) | Estab. Female hypert. on treatment Mean(SD) |
|---|---|---|---|---|---|---|
| TC (mmol/L) | 3.53 (1.39) | 2.77 (0.84)* | 4.48 (1.43) | 4.84 (1.83) | 4.65 (1.81) | 4.79 (1.34) |
| TG (mmol/L) | 0.87 (0.28) | 0.88 (0.57) | 1.12 (0.57) | 0.83 (0.29)* | 0.76 (0.24) | 1.14 (0.71)* |
| HDL-C (mmol/L) | 1.05 (0.45) | 0.77 (0.29)* | 1.31 (0.47) | 1.05 (0.25)* | 1.23 (0.41) | 1.22 (0.42) |
| LDL-C (mmol/L) | 2.49 (1.23) | 2.0 (0.56)* | 3.07 (1.20) | 3.96 (11.42)* | 3.48 (1.57) | 3.45 (1.22) |
| FPG (mmol/L) | 3.59 (0.68) | 3.81 (0.55) | 3.65 (0.80) | 5.34 (1.36)* | 3.72 (0.6) | 4.86 (0.94)* |
| BMI ($kg/m^2$) | 23.71(5.35) | 22.93 (3.95) | 24.82 (3.77) | 26.28 (7.10) | 29.51 (5.01) | 27.67 (6.34) |
| SYSTOLIC BP | 121.43(7.56) | 117.05(7.97*) | 154.64(17.70) | 139.71(16.53)* | 149.71(10.23) | 139.70(16.67)* |
| DIASTOLIC BP | 78.75 (3.76) | 76.36 (5.16) | 95.15 (7.55) | 89.71 (10.68)* | 97.64 (5.89) | 87.73 (8.67)* |

* = significant P 0.05
Hypert. = Hypertension
Estab. = established
Diagn. = diagnosis

controls (2.28mmol/L 1.01). Similarly, it was significantly higher in established hypertensives on treatment (3.62mmol/L 1.30) than in controls (2.28mmol/L 1.01). P 0.05. In males, the established hypertensives on treatment's mean plasma LDL-C level (3.96mmol/L 1.42) was significantly higher than

that of newly diagnosed hypertensives (3.07mmol/L 1.20). P 0.05. This was not so in the female hypertensives (newly diagnosed hypertensives; 3.48mmol/L 1.57, established hypertensives on treatment; 3.45mmol/L 1.22)

Fasting plasma glucose levels in newly diagnosed hypertensives (3.57mmol/L 0.76) and established hypertensives on treatment (5.02mmol/L 1.11) showed a significant difference. This was higher in the treated group. P 0.05. Fasting plasma glucose in established male hypertensives on treatment (5.34mmol/L 1.36) and established female hypertensives (4.86mmol/L 0.94) were significantly higher when individually compared with values in newly diagnosed males hypertensives (3.65mmol/L 0.80) and newly diagnosed female hypertensives (3.72mmol/L 0.66) respectively. P 0.05.

The BMI in newly diagnosed hypertensives (26.42kg/$m^2$ 4.75) was significantly higher than controls value (23.37kg/m2 4.75). So was it in established hypertensives (27.20kg/$m^2$ 6.75) compared to controls (23.37kg/m2 4.75). P 0.05. Table 2. Established male hypertensives on treatment had significantly higher BMI (26.28kg/$m^2$ 7.10) than male controls. P 0.05. Female hypertensives (newly diagnosed; 29.51kg/$m^2$ 5.01, established on treatment; 27.67kg/$m^2$ 6.34) had significantly higher BMI than female controls (22.93kg/$m^2$ 3.95) P 0.05. Table 3.

**Discussion**

The peaked prevalence of untreated hypertensives in the 40-59 years bracket in both sexes, suggests that as age increases the prevalence of hypertension rises. Despite the few subjects of this study, this trend have been demonstrated in other studies [14,15]. In subjects below 40 years of age, women had more cases of newly diagnosed hypertension. It is our opinion that this had to do with their higher BMI in compared to that of newly diagnosed male hypertensives. A study in Ibadan found that obesity and age less than 40 years were associated with hypertension[16]. Another study in India found that obesity and age less than 45 years were more associated with hypertension in women[17]. It is possible that in addition to increased BMI, urbanisation has some role to play in women being more prone to developing hypertension at earlier age.

As in some Nigeria[7,8], Tanzania[18] and Cameroun[19] studies, we demonstrated increase in plasma TC in subjects with hypertension. This increase was not worsened by treatment as untreated hypertensives and treated ones, had similar plasma TC. If anything, treatment of hypertension arrested the increase of plasma TC due to hypertension. This was earlier noted in the ALLHAT Study where control of high BP was associated with a reduction in plasma TC[20].

This study did not find any significant difference in plasma TG in both newly diagnosed and established hypertensives compared to the controls. In the Nigeria studies earlier mentioned[7,8], hypertensives on treatment had higher plasma TG levels. The increase in plasma TG, was noted to be higher in males than females, in one of the studies[8]. The present study found this increase to be present in newly diagnosed male hypertensives. Unlike in the males, treatment appears not to reduce plasma TG in established female hypertensives, as its value was higher than controls'. In Cameroun[19] treated female hypertensives had a higher plasma TG than controls, just like what we found. Also, the Camerounians did not find any significant difference in plasma TG in treated male hypertensives and controls. This gender differences are possibly due to hypertension and or increased BMI, since these were not present in the controls. The association of hypertriglyceridaemia as an independent risk factor for CHD is controversial.[21,22]. However, it is more predictive of heart disease in women than in men[23]. The import of this is that female hypertensives in this area should have their plasma TG closely watched. They could benefit from discretionary drug treatment and body weight control[24].

Mean plasma HDL-C was higher in hypertensives in this study as in Lagos[8] and Cameroun[19] studies. While the other Lagos study[7] did not show a significant difference in plasma HDL-C in hypertensives and controls, another Nigerian study[25] found a decreased HDL-C in hypertensives. Reports of low HDL-C in hypertensives is a pattern commonly noted in some Caucasian study[26] and not in others[27]. In patients with CHD, adult blacks have been shown to have higher plasma HDL-C than whites[28] and this partly explains their reduced incidence of CHD[29]. Only in the males, was the established hypertensive subjects found to have lower plasma HDL-C than in newly diagnosed group. This suggests that the established male hypertensives in this study have a poorer plasma lipid profile.

The dyslipidaemia found in the hypertensives in this study also demonstrated a higher plasma LDL-C. This increase was noted in both groups of hypertensives. Some other studies have earlier shown this increase in plasma LDL-C in hypertensives[8,18,19,25]. Among the males, the established hypertensives had higher plasma LDL-C than newly diagnosed hypertensives. This was not the case in female

hypertensives. The Cameroun study[19] found increased plasma LDL-C in male hypertensives. These workers were of the opinion that male hypertensives on treatment have a more marked deterioration of lipid profile, which worsens with increasing duration of treatment. In the present study, the combination of increased level of LDL-C and reduced level of HDL-C in male hypertensives could provide a ground for increased susceptibility to CHD. Low Density Lipoprotein Cholesterol which is a major atherogenic lipoprotein has been positively correlated with the risk of CVD[30].

In this study, glucose intolerance, which is associated with hypertension, did not show as abnormal FPG in newly diagnosed hypertensives. Similar pattern was found in studies carried out in Norway[27] and United Kingdom [31]. But in established hypertensives on treatment, there was a higher FPG than in controls. This increase is thought to be due to the type of treatment received by the subjects. There are data suggesting significant differences in the effect of antihypertensives on insulin resistance and glycaemic control, both between antihypertensive classes and, even within the same classes[32].

Cardiovascular risks, including hyperlipidaemia is said to decrease with treatment of hypertension, especially when accompanied by weight loss in blacks[24]. There seem to be inadequate attention paid to weight management in the study subjects. This seems the case in males in which BMI was significantly higher in the established hypertensives on treatment than in controls. But in females, both newly diagnosed and established hypertensives had higher mean BMI than controls. This suggests that increased BMI is more contributory to the origin of hypertension in females in our study population.

Since weight loss is the most effective non-pharmacological means of lowering blood pressure[33] and the most natural target of primary prevention of cardiovascular disease[34], it should be emphasised in management of hypertension in this part of the country. In addition, the observed clustering of dyslipidaemia and the potential benefits of treatment among hypertensives, demonstrates the need for screening and treating other cardiovascular risk factors, beyond simply controlling blood pressure[35].

These factors, and the observation of regional[36] and ethnic[37] differences in plasma lipid patterns, make it necessary for similar studies to be carried out in other parts of the Country. The findings will help us to delineate peculiar plasma lipid maps in hypertensives in different parts of the country, if any.

The noted short comings of this work are, the small number of subjects recruited for the study and the inclusion of subjects on thiazide diurectics. It should be noted that effort was made to include only those who had been on thiazide for a short time, especially less than one year.

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