Original article:

Comparison of lipid profile in hypertensives patients and normotensives subjects living in Sagamu

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ABSTRACT

Objectives: It is uncertain if the lipid levels (total cholesterol and HDL and the ratio which has a significant effect) of individuals can be varied by the cardiovascular event occurring in individuals. Therefore this study was designed to assess the lipid levels in hypertensive and normotensive patients.

Design: This study involved purposive selection of 50 hypertensive patients attending medical outpatient clinic of Olabisi Onabanjo Teaching Hospital, Sagamu. and 50 normotensive patients living in Sagamu aged between 25 and 50 years. The blood pressure was measured with manual sphygmomanometer (mmHg) in order to group them into control and test groups. Fasting blood was taken from each participant. The lipid level (mg/dl) was estimated using standard methods. Data were analyzed using descriptive statistic and student t test.

Results: The mean systolic BP 102.10±1.42 in control and 148.96±2.42 in hypertensive and mean diastolic was 75.64±0.75 in control and 98.58±1.97 in hypertensive. The mean Total cholesterol was 161.48±3.22 in control while to203.82±6.17, HDL 48.52±0.29 in control and 42.64±0.90 in hypertensive while the TC/HDL in control was 3.33±0.07 and 5.01 ±0.27 in hypertensive.

Conclusions: The disturbances in lipid profile between the control and hypertensive subjects will require continuous monitoring of same in hypertensives to prevent cardiovascular events.

Key words: Hypertensive, Normotensive, lipid profile, blood pressure

Introduction

Hypertension is a medical condition in which the blood pressure in the arteries is persistently elevated [(1)] high blood pressure usually does not cause symptoms. Long-term high blood pressure, however, is a major risk factor for coronary artery disease, stroke, heart failure, atrial fibrillation, peripheral vascular disease, vision loss, chronic kidney disease, and dementia [(2)].

The global burden of hypertension and other non-communicable disease (NCDs) is rapidly increasing, and the African continent seems to be the most affected region in the world. The prevalence of hypertension in Nigeria forms a substantial portion of the total burden in Africa because of the large population of the country currently estimated to be over 170 million[(3)].

The increasing prevalence of hypertension in developing countries is of great concern.

According to a report from the World Health Organization [(3)], there was an estimated 972 million people with hypertension in the year 2000. 65% lived in developing world with the number predicted to grow to 1.5 billion by 2025. The increasing prevalence is well reflected in the increase in cardiovascular disease mortalities. This is especially in

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developing countries with high illiteracy rates and a drastic shift in the increase from communicable disease to non-communicable diseases [(4)].

It is estimated that hypertension affects about 1 billion people all over the world and it is the main risk factor for many other cardiovascular diseases [(5)]. The prevalence of hypertension in Nigeria may form a substantial proportion of the total burden in Africa because of the large population of the country currently estimated to be over 170 million [(5)].

With an increasing adult population and changing lifestyle of Nigerians, the burden of hypertension may continue to increase as time unfolds [(6)]. In suggesting an evidence-based context for government and other health policy planners on strategies to reduce this burden in low-resource settings like Nigeria, it is important to have detailed up to date information on the prevalence in order to math available resources.

According to large observational studies, hypertension is thus associated with high incidence of cardiovascular disease, such as stroke, ischemic heart disease, and other vascular diseases [(7)]. An increased incidence of cardiovascular disease has in fact been seen in relation to blood pressure levels across the entire blood pressure distribution [(8)], also within the normal blood pressure range [(9)]. for half a century, treatment and awareness of high blood pressure has been insufficient, as described by "The rule of halves" meaning that only half of those aware were treated, and of those treated only half achieved treatments goals, even though treatment has contributed to a reduction of cardiovascular events, the control of high blood pressure and hypertension can still be improved (10).

Methods

The study involved purposive selection of 50 hypertensive patients attending medical outpatient clinic of Olabisi Onabanjo Teaching Hospital, Sagamu. and 50 normotensive patients living in Sagamu aged between 25 and 50 years. The blood pressure was measured with manual sphygmomanometer (mmHg) in order to group them into control and test groups. Fasting blood was taken from each participant.

Determination of Lipid Profile

Lipid Profile was determined by following the protocol of Trinder, (1969 as described by Ekor, Osonuga, Odewabi and Oritogun (2010).

Principle Total cholesterol level was measured spectrophotometrically using standard laboratory supplied by BIOLABO, France. Cholesterol esters in the presence of cholesterol esterase cholesterol and free fatty acids are separated. The cholesterol formed reacts with oxygen in the presence of cholesterol oxidase to form cholesten-4-one-3 and hydrogen peroxide. The hydrogen peroxide formed reacts with phenol and 4-amino-antipyrine in the presence of peroxidase to give aminoneimine (pinkish in colour) and water. The intensity of the pink/red colour formed is proportional to the cholesterol concentration. The procedure employed was as follows:

The reagent was prepared by adding 5ml of the buffer (1.75mool/L Amino-2-methyl-2-propanol-1) to 5ml of the Chromogen mixture (76umol/L 0-Cresolphtalein Complexon, 3.36mmol/L/L 8 – Hydroxy-Quinoline, 25mmol/L HCI) and allowed to stand for an hour at room temperature.

The reagent solution was prepared by adding equal volumes of the buffer and 5mmol/L Chloro-4-phenol) and the enzyme mixture (100U/L Cholesterol oxidase, 70U/L Cholesterol esterase, 1200U/L peroxidase, 2mmol/L Cholic acid Sodium salt, 0.3mmol Amino antipyrine) and allowed to stand for 5 – 10 minutes while mixing gently at room temperature.

To 10µL of each test sample or standard (5.17mmol/L Cholesterol) was added 1ml of the reagent mixture. This was incubated at 37^{o}C for 5 minutes. The absorbance of the mixture was taken against the blank at a wavelength of 500nm. The blank was made up of 10µL of distilled water and 1ml of the reagent mixture. The cholesterol concentration was determined as follows.

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Total cholesterol concentration (mg/dl) = $\text{Absorbance}_{sample}$ X Standard concentration

Absorbance *standard*

HDL cholesterol level was measured spectrophotometrically using standard lab kits supplied by BIOLABO, France. Low density lipoproteins (LDL) contained in serum are precipitated by addition of phosphotungstic acid and magnesium chloride. High density lipoproteins (LDL) which remains in the supernatant (obtained after centrifugation) react with the cholesterol reagent and proportionally with the cholesterol standard.

The procedure followed was as follows: equal volumes of the serum and reagent mixture (13.9mmol/L phosphotungstic acid and 570mmol/L magnesium chloride) were mixed together and allowed to stand for 10 minutes at room temperature. The reaction mixture was then centrifuged for 10 minutes at 4000rpm to get a clear supernatant. This supernatant was used as sample to get the HDL cholesterol concentration in the serum sample. 1000ul of the Cholesterol reagent was added to test tubes labeled blank, standard and sample containing 50µl water, 50µl of the cholesterol standard and 50µl of the sample respectively. This was well mixed and incubated for 10mins at 37^{o}C. The absorbance of the end sample against the blank was taken at 505nm.

HDL cholesterol concentration (mg/dl) = $\text{Absorbance}_{sample}$ X Standard concentration

Absorbance $_{standard}$

Triglycerides level was measured spectrophotmetically using standard tab kits supplied BIOLABO, France Triglycerides in the presence of lipase form glycerol free fatty acids. Glycerol formed reacts reversibly with adenosine triphosphate (ATP) in the presence of glycerol lipase to form glycerol – 3 – phosphate and ADP. The glycerol 3 phosphate also reacts reversibly with oxygen in the presence of glycerol -3- phosphate oxidase to form dihydroxyacetone phosphate and hydrogen peroxide. The hydrogen peroxide then reacts with chlorophenol and amino antipyrine in the presence of peroxidase to form quinoneimine (pink) and water. The intensity of the pink/red colour formed is proportional to the triglyceride concentration.

The reagent solution was prepared by adding equal volumes of the buffer (3.5mmol/Lchloro-4-phenol, 6mmol/L Magnesium chloride 100mmol/L PIPES) and the enzyme mixture 500U/I Lipase, 1800U/I peroxidase, 400U/I Glycerol 3-phosphate oxidase, 1000U/I Glycerol (lipase. 0.30mmol 4 Amino antipyrine. 1.72mmol/I Adenosine triphosphate Na) and allowed to stand for 5 – 10minutes. To 10µL of each test sample of standard (Glycerol 200mg/dl) was added 1mI of the reagent mixture. This was incubated at 37^{o}C for 5minutes. The absorbance of the mixture was taken against the blank at a wavelength of 500nm. The blank was made up of 10µL of distilled H_20 and 1ml of the reagent mixture. The triglyceride concentration was determined as follows.

Triglyceride concentration (mg/dl) = Absorbance *sample* X Standard concentration

Absorbance *standard*

Ethical Approval and Informed Consent

Ethical clearance for the study was obtained from the Committee on Human Research publication and Ethics of the School of Olabisi Onabanjo University teaching Hospital (OOUTH), Sagamu All participants (100) of this study signed an informed consent form, in accordance to the committee regulations, before answering the questionnaire taking blood pressure and taking blood samples.

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Statistical analysis was carried out by using student test. The data obtained was analyzed using computer statistical programme package SPSS version 15.0 Probability value of **P** less than 0.05 was considered statistically significant.

Results:

Table 1 showed the mean systolic BP 102.10±1.42 in control and 148.96±2.42 in hypertensive and mean diastolic was 75.64±0.75 in control and 98.58±1.97 in hypertensive. The mean Total cholesterol was 161.48±3.22 in control while 203.82±6.17mg/dl in hypertensives, HDL 48.52±0.29 in control and 42.64±0.90 in hypertensive while the TC/HDL in control was 3.33±0.07 and 5.01 ±0.27 in hypertensive. All values with $P<0.05$±SEM are significant.

Figure 1 showed the comparison between the hypertensive patients and normotensives subjects where there were higher levels in both total cholesterol and HDL levels.

Figure 2 showed the mean systolic blood pressure and mean diastolic blood pressure between the hypertensive patients and normotensives subjects where there were higher levels in hypertensives.

Figure 3 showed the mean TC/HDL ratio between the hypertensive patients and normotensives subjects where there were higher levels in hypertensives.

Table 1: Mean lipid profile and blood pressure of hypertensives and non-hypertensive controls($p<0.05$±SEM).

Variables	Hypertensives (n=50)	Controls (n=50)	t-value	p-value
Total Cholesterol (mg/dl)	203.82±6.17	161.48±3.22	6.081	0.000*
HDL (mg/dl)	42.64±0.90	48.52±0.29	-6.222	0.000*
SBP (mmHg)	148.96±2.42	102.10±1.42	16.734	0.000*
DBP (mmHg)	98.58±1.97	75.64±0.75	10.866	0.000*
TC/HDL ratio	5.01±0.27	3.33±0.07	6.035	0.000*

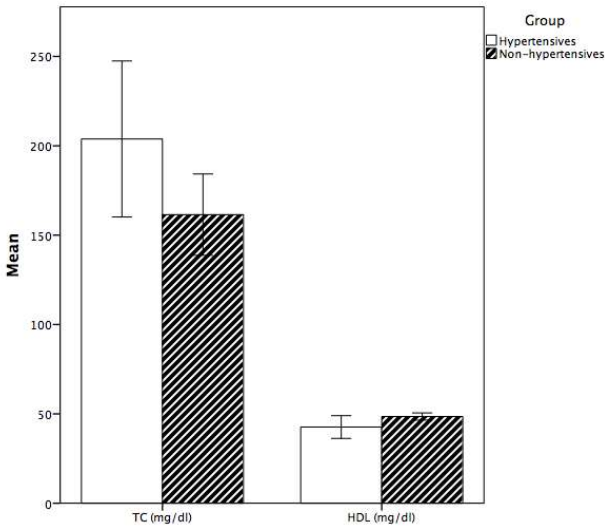

Figure 1 showed the comparison between the hypertensive patients and normotensives subjects where there were higher levels in both total cholesterol and HDL levels. ($p<0.05$±SEM).

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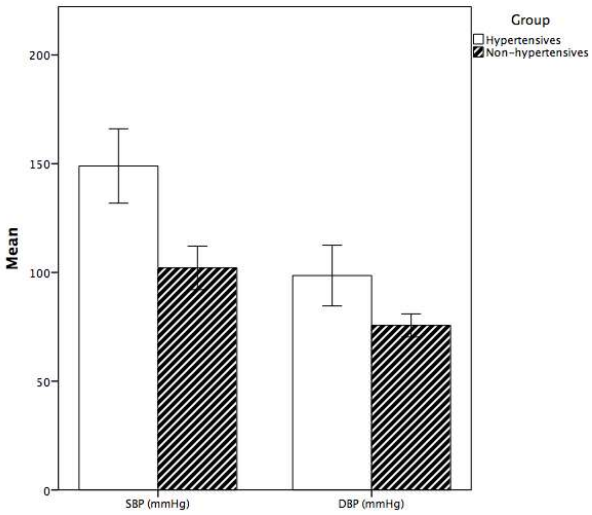

Figure 2 showed the mean systolic blood pressure and mean diastolic blood pressure between the hypertensive patients and normotensives subjects where there were higher levels in hypertensives. ($p<0.05\pm SEM$).

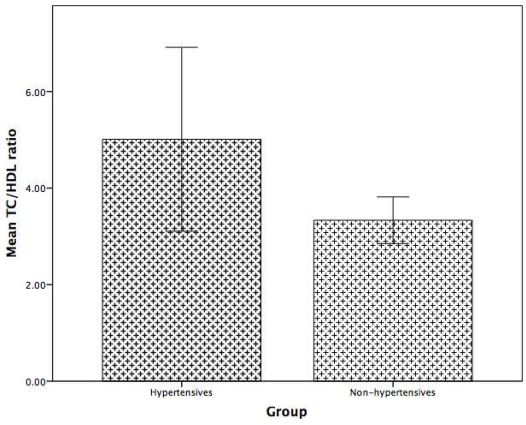

Figure 3 showed the mean TC/HDL ratio between the hypertensive patients and normotensives subjects where there were higher levels in hypertensives. ($p<0.05\pm SEM$).

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Discussion

The main risk associated with high cholesterol is coronary heart disease. If cholesterol is too high, it builds up on the walls of the arteries which is known as atherosclerosis. This condition causes arteries to become narrowed, and the narrowed blood vessels reduced blood flow to the heart. This can result in angina pectoris resulting from reduced blood flow to the heart, or a heart attack in cases when a blood vessels is blocked completely [(11).]

In this study, serum TC concentrations are significantly higher in hypertensive patient. This is consistent with earlier observations elsewhere and in other parts of Nigeria [(12)]. High levels of serum cholesterol are known to increase the risk of developing macrovascular complications such as coronary heart disease (CHD) and stroke. Many epidemiological studies indicate a progressive increase in CHD risk as the serum TC exceeds 5.0 mmol/L [(13)]. It was therefore suggested by Lewis [(14)] that levels of serum TC in the range 5.0-6.5 mmol/L be considered undesirable.

Moreover, noteworthy is the positive and significant correlation between serum TC and both systolic and diastolic BP in hypertensive patients; suggesting that as blood pressure increase so also does TC.

This observation is expected, and may be due to common risk factors for hypertension, obesity and dyslipidaemia. Obesity is known to play a central role in the causation and sustenance of insulin resistance [(15)]. Insulin resistance is considered the underlying factor in the pathogenesis of hypertension, dyslipidaemia and the metabolic syndrome in some populations [(15)].

In African patients however, there are doubts as to the role of insulin resistance in the aetiology and sustenance of hypertension (Bakari, 2004, [(16)] suggesting that other mechanism or chance may be responsible for the observed relationship between these variables.

The occurrence of high serum TC levels in hypertensive patients, as found in the present study may be due to variety of causes such as genetic factors, increased consumption of dietary animal fats, lack of physical exercise, stress.

The risk of becoming hypertensive in later life is considerable, as studies from almost all high-income countries have shown that blood pressure rises with increasing age [(17)]. The incidence of hypertension is likely to vary depending on the initial blood pressure and the intra-individual variation of blood pressure measurements [(17)].

Although there are subjects in whom the hypertension can be traced to an underlying disease, i.e secondary hypertension, those cases are rare and lost hypertensive cases have primary hypertension. Primary hypertension stems from interaction between multiple genetic and environmental factors, involving complex pathogenesis mechanism [(18)]. Hypertension has long been recognized to cluster within families in cross-sectional studies, and a positive family history of hypertension doubles the prevalence of hypertension. More recent studies in twins (Kupper et al.[(19)] have concluded that approximately 60% of the family association of blood pressure is explained by shared genes and approximately 40% by shared environment. Thus, even though genetic might account for the largest impact, there is still a major influence of lifestyle and environmental factors which are potentially preventable[(20)].

Hypertension may cause structural changes in blood vessels and in the heart, and include macrovascular complications, such as ischemic heart disease and heart failure[(20)].

These conditions develop due to the interplay between high bloods pressure and metabolic disorders, such as in the metabolic syndrome and are also influenced by genetic and lifestyle [(21)]. Insulin resistance with the activation of the renin-angiotensin-aldosterone system (RAAS) and inflammation represent common mechanisms in these conditions. RAAS is important for the regulation of salt-water balance in the body and contributes to blood pressure regulation in several ways [(22)]. The endothelium in peripheral vessels is another key factor, as endothelial dysfunction with reduced production of nitric oxide (NO) will affect vasodilatation, insulin, sensitivity, platelet adhesion, and thrombus formation. The sympathetic nervous system is also believed to play a role in initiating primary [(22)].

CONCLUSION

In conclusion, the disturbances in lipid profile between the control and hypertensive subjects will require continuous monitoring of same in hypertensives to prevent cardiovascular events. Hence, hypertensive patients need to be watchful of what they eat in order not to result in disturbances of their lipid levels.

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CONFLICT OF INTEREST

No conflict of interest.

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