THE CLINICAL ASSOCIATIONS OF DYSLIPIDAEMIA AMONG HYPERTENSIVE ADULTS PRESENTING TO THE UNIVERSITY TEACHING HOSPITAL (UTH), LUSAKA ADULT HOSPITAL, ZAMBIA

by

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DECLARATION

I declare that this dissertation is my own work. It has not been submitted before for any degree or examination at this or any other University, and does not represent any interest group(s).

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CERTIFICATE OF APPROVAL

This dissertation of KATONGO HOPE MUTENGO has been approved as partial fulfillment of the requirements for the award of MASTER OF MEDICINE in INTERNAL MEDICINE by the University of Zambia

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ABSTRACT

Co-existence of hypertension and dyslipidaemia, also referred to as dyslipidaemic hypertension (DH), is known to have synergistic effect on the development of cardiovascular disease (CVD). In Zambia, there is scanty information on distinguishing features of dyslipidaemic hypertension. The study aimed at identifying factors associated with dyslipidaemic hypertension in adults at a tertiary hospital in Zambia.

This was a cross-sectional study conducted from January 2017 to July, 2017. One hundred and sixty-one (161) participants were enrolled comprising 88 hypertensives and 73 controls. Relevant demographics, physical examinations, bio-electric impedance analysis and laboratory investigations were performed. Fasting lipid and lipoprotein parameters which included fasting serum total cholesterol (TC), high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDC-C) and triglycerides (TG) were analysed for lipid abnormalities. Data was analyzed using Stata version 15.

The median age was 47 years (IQR; 39, 58) and 38 years (IQR; 38, 48) for the hypertensive and control groups, respectively. 53.3% of hypertensive and 46.7% of controls had at least one lipid abnormality. The factors associated with dyslipidaemic hypertension were; TC [aOR 2.41; 95%CI 1.57, 3.69; p<0.001], TG [aOR 3.28; 95%CI 1.12, 9.63; p=0.031] and HDL-C [aOR 0.10; 95%CI 0.03, 0.39; p=0.001]. On the other hand, the factors noted to be associated with dyslipidaemia only were; TC [aOR 3.61; 95%CI 2.08, 6.28; p<0.001], male gender [aOR 0.22; 95%CI 0.09, 0.53, p=0.001] and HDL-C [aOR 0.03; 95%CI 0.01, 0.11; p<0.001]. Traditional risk factors such as age, body mass index, waist circumference, waist-to-hip ratio did not show strong associations on multi-variate analysis.

The study showed that increase in total cholesterol and triglycerides, and a reduction in high density lipoprotein cholesterol was significantly associated with dyslipidaemic hypertension. It is therefore imperative that management of hypertension should also focus on identifying and correcting associated lipid disorders.

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ABBREVIATIONS

AOC	Adult Outpatient Clinic
BIA	Bioelectric Impedance Analysis
BMI	Body Mass Index
CHD	Coronary Heart Disease
CVD	Cardiovascular Disease
DH	Dyslipidaemic Hypertension
IDF	International Diabetes Federation
IHD	Ischaemic Heart Disease
HDL-C	High Density Lipoprotein Cholesterol
LDL-C	Low Density Lipoprotein Cholesterol
NCD	Non-communicable Diseases
NCEP	National Cholesterol Education Programme
NHANES	National Health and Nutritional Examination Survey
RAAS	Renin Angiotensin Aldosterone System
TC	Total Cholesterol
TG	Triglyceride
TGRLP	Triglyceride Rich Lipoprotein
UTH	University Teaching Hospital
WHO	World Health Organisation

WC	Waist Circumference
WHR	Waist-to-Hip Ratio

CASE DEFINITION

Dyslipidaemia was defined as presence of one or more of the following in accordance with the International Diabetes Federation (IDF); Total Cholesterol of > 5.17 mmol/L (>200mg/dl), Triglyceride > 1.7mmol/l (150mg/dl), decreased HDL-C < 1.03 mmol/L(<40mg/dl) in males and ≤ 1.3 mmol/L (<50mg/dl) in females and elevated LDL-C > 3.36mmol/l (>130mg/dl).

Hypertension was defined according to Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7) as a systolic blood pressure (SBP) ≥140 mmHg and a diastolic blood pressure of (DBP) ≥ 90 mmHg.

Obesity - A BMI of ≥ 30 kg/m^2, or greater according to the third report of the Adult Treatment Panel of the National Cholesterol Education Program (NCEP ATP III).

Central Obesity - Waist circumference ≥94 cm for males and ≥80 cm for females and a waist-hip ratio of ≥ 0.90 for males and ≥ 0.85 for females by anthropometric measurements (IDF).

Abnormal (excess) visceral fat- This was visceral fat rating of 13-59 using segmental fat distribution with Bio-electric Impedance Analysis (BIA) on TANITA body composition analyser.

CHAPTER 1

1.1 Background

Hypertension and dyslipidaemia are both recognised worldwide as major contributors to cardiovascular diseases (CVDs) with a combined multiplicative effect across all ethnicities (1–4). In 2015, CVDs contributed to over 17 million deaths worldwide with about 5 million of these deaths (37%) occurring in low- and middle- income countries (5).

Africa has not been spared by CVD burden. Statistics show that Africa has the highest prevalence of hypertension at 46 % and it's the leading risk for CVDs (6,7). According to the Initiative for Cardiovascular Health Research in the Developing Countries (IC-Health), the CVD burden is expected to double from the year 1990 to 2020 with the poor being at highest risk due to unfavourable socio-economic status and limited access to health care. With the already existing struggle with the current burden of communicable diseases such as Human Immunodeficiency Virus (HIV)-related illnesses, and malnutrition; dealing with CVDs is another added predicament which may have negative effect on the social economic development and may further plummet most countries into poverty (8).

Although hypertension and dyslipidaemia frequently co-exist (2,3,9,10), a common denominator; obesity, particularly central obesity, also accompanies these two conditions. Risk factors for obesity are strongly linked to the development of both dyslipidaemia and hypertension or either [**Figure 1**]. Interestingly, dyslipidaemia and hypertension have a pathophysiological interplay such that occurrence of only one of these conditions may foster the development another (2,11–14). Ultimately, both hypertension and dyslipidaemia, even when independent of each other, can eventually lead to atherosclerosis; the main cause of CAD (11). However, these combined risk factors leading to atherosclerosis can be prevented through lifestyle changes and treatment (6,15,16). The inter-relationship of obesity, hypertension and dyslipidaemia can be summarised as shown below.

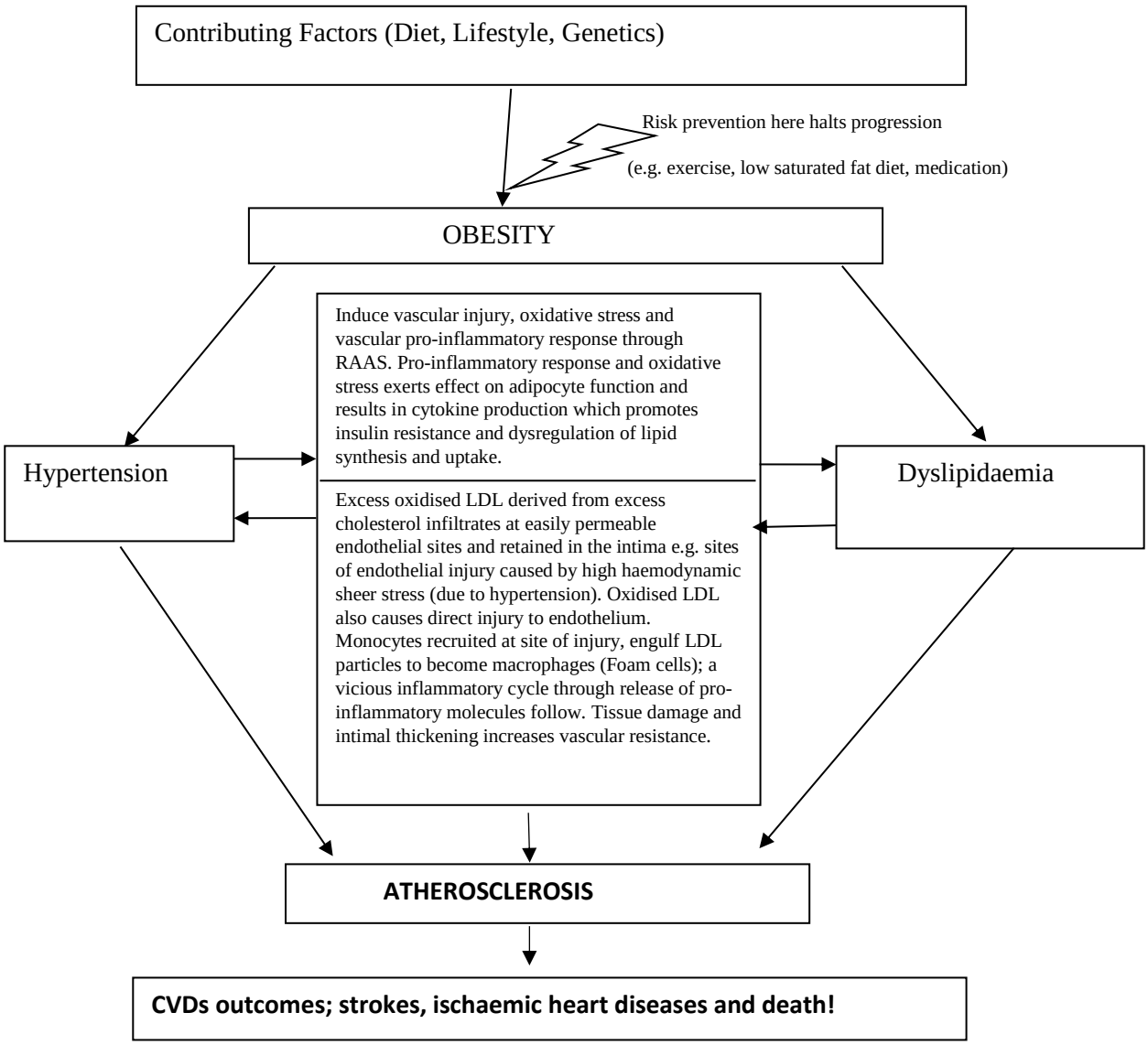

Figure 1. Schema of the link between obesity and hypertension/dyslipidaemia. Arrows: Right- Hypertensive factors leading to dyslipidaemia, Left- some factors in dyslipidaemia leading to hypertension. RAAS- Renin-angiotensin-aldosterone system.

The current burden of hypertension in Zambia, like other low-and middle-income countries cannot be overemphasised. A 2008 survey from Lusaka, the capital city of Zambia, by Goma et al found the prevalence of hypertension and hypercholesterolaemia at 34.8% and 15.8% respectively (17). However, there was no report on other atherogenic lipids and lipoproteins. The co-existence of hypertension and dyslipidaemia in Zambia still remains an area that needs further exploration.

In the wake of hypertension being a major risk factor for stroke at UTH (18), it is fundamental that attention is paid to both lipid and high blood pressure control. This can best be achieved by identifying patients at high risk before they present with established CVD related complications. The aim of this study was to identify clinical factors associated with co-existing hypertension and dyslipidaemia, so as to alert the clinicians about the groups at high multiplicative risk for CVD at presentation.

Bioelectric Impedance Analysis (BIA) by use of the TANITA body composition analyser, was used to assess fat tissue distribution and hence used as a measure of central obesity. The TANITA body composition analyser measures the complete body composition profile that includes weight, body fat percentage, body fat mass, BMI, fat free mass, estimated muscle mass and total body water within 30 seconds. Its accuracy is reportedly within +/- 5 percentage of the standard of body composition analysis using Dual Energy X-ray Absorptiometry DEXA (19). The results are said to be reproducible within +/- 1 percent variation when used under consistent conditions (20). It was therefore used to provide estimates on visceral adiposity (central obesity) which was used as a clinical correlate in the study.

1.2 Statement of the problem

There has been no research to show the factors associated with dyslipidaemia in the adult hypertensive Zambian population. Dyslipidaemia is often asymptomatic and not easily diagnosed and managed despite the fact that it is also a major cause of cardiovascular related morbidity and mortality. It is already recognised that co-existence of hypertension and dyslipidaemia has multiplicative, rather than additive effect on the development of CVDs and the existence of each one of these even independent of the other, pose major risk to poor CVD outcomes.

1.3 Study justification

This study emphasised the importance of identifying dyslipidaemia as a co-existing major cardiovascular risk factor on initial screening of adult hypertensive patient. There is a heavy burden of hypertension and associated CVD outcomes such as strokes among adults in Lusaka, Zambia. Since hypertension and dyslipidaemia both contribute significantly to cardiovascular events, it is imperative that clinicians are pay attention to both lipid and blood pressure control by identifying associated risk factors.

The results from this study will contribute to a pool of knowledge on dyslipidaemic states, not only in Zambia but sub-Sahara region and Africa at large which has diverse racial and regional variations of phenotypes. Prior to this study, there was no available data on clinical associations of dyslipidaemia among hypertensive patients presenting to UTH, Lusaka Adult Hospital, Zambia

1.4 Research question

What clinical factors are associated with adult hypertensive patients who have dyslipidaemia?

1.5 Objectives

General objective

The general objective was to study the clinical associations of dyslipidaemia among adult hypertensive population.

Specific objectives

i. To describe the prevalence and patterns of dyslipidaemia among the hypertensives
ii. To determine the factors associated with dyslipidaemia in the hypertensive population (dyslipidaemic hypertension).
iii. To determine factors associated with dyslipidaemia without hypertension.

CHAPTER 2

Literature review

Dyslipidaemia has been well established as a major risk factor for cardiovascular diseases (1,21). By definition, dyslipidaemia involves abnormalities in atherogenic lipid macromolecules which include high TC, high LDL-C, low HDL-C and high triglyceride levels (22). Other rare triglyceride-rich lipoproteins (TGRLP), elevated very-low density lipoproteins (VLDL) and small density lipoproteins particles are also said to be atherogenic (23). However, because low density lipoprotein (LDL) is the major cholesterol carrying lipoprotein, it has received most attention because of overwhelming evidence that its reduction through lifestyle changes and medical therapy greatly reduces the risk of adverse cardiovascular events (22).

Worldwide, CVD account for about 17 million deaths and most of these deaths occur in low- and middle-income countries with approximately 80% yearly mortality rates (16). Adoption of unhealthy lifestyles in low- and middle-income countries has been attributed to the development of CVD risk factors; hypertension, diabetes, dyslipidaemia and obesity (24). The major identifiable risk factor for the development of cardiovascular diseases, such as strokes and ischaemic heart disease (IHD) is hypertension (21,25,26). Globally, hypertension accounts for 7.5 million deaths which translates to about 12.5% of all causes of death, with Africa having the highest prevalence estimated at 46%, mostly attributed to adoption of western lifestyle and poor access to health care (6,27). Lifestyle changes, such as intake of excessive saturated fatty acids and trans-fatty acids heavy smoking and high alcohol intake have shown a growing trend even in low- and middle-income countries; apart from predisposing to hypertension, these factors also contribute to dyslipidaemia. However, because of other factors such as limited healthcare facilities and treatment, and the focus on high burden of communicable diseases, the burden tend to higher in these countries. (6,24,28). Additionally, physical inactivity; which according to the UK National Institute for Clinical Excellence (NICE) guidelines defined as no reported activity or any physical activity or pair of activities done for less than 20 minutes or less than three times per week, has also been shown to predispose to conditions such as obesity and hypertension (29). The NICE guidelines further recommend moderate

intensity exercises (walking, cycling, gardening) of at least 30 minutes, five times a week to reduce CVD risk.

Several co-morbid conditions such as HIV infection, chronic kidney disease (CKD), malignancies, chronic liver disease (CLD), thyroid disorders and drugs like steroids, Highly Active Anti-retroviral Therapy (HAART), antihypertensive drugs (beta blockers and thiazide diuretics) and oestrogen based contraceptives may affect lipid profiles (30–38). The National Cholesterol Education Programme (NCEP) however, suggests that the occurrence of dyslipidaemia with other major CVD risk factors has a multiplicative, rather than additive effect on the development of cardiovascular morbidity and mortality independent of other co-morbidities. These major risk factors include; advanced age, diabetes mellitus, hypertension, dyslipidaemia and smoking. This is particularly important as dyslipidaemia tends to commonly occur with hypertension or with diabetes mellitus, most importantly as a component of the metabolic syndrome; which has received much attention as a major cause of CVD (2,23,25).

The Framingham Heart Study (FHS) showed that hypertension, a major cardiovascular risk factor, seldom occurs in isolation of other risk factors. At least 80% of the hypertensive population in the FHS had an additional risk factor which included obesity, glucose intolerance, left ventricular hypertrophy and dyslipidaemia. Abdominal obesity, has been shown to be the major determinant of hypertension through its promotion of insulin resistance syndrome (25). This abdominal obesity-hypertension relationship has further been supported by other studies which have indicated a strong correlation of abdominal obesity to insulin resistance or metabolic syndrome (9,13,21,25,39,40). Due to clustering of these conditions, it is important for clinicians to routinely screen for atherogenic risk factors in hypertensive patients as they are modifiable through lifestyle changes and medical therapy.

A large randomised study in the United States of America (USA) showed that a high Body Mass Index (BMI) of greater than 40kg/m^2 was associated with the highest risk for hypertension, dyslipidaemia and diabetes (41). The results of this study were comparable to the National Health and Nutritional Examination Survey (NHANES) done in the USA (42). In contrast, the Asian Indian population in the Chennai Urban

Rural Epidemiology study (CURES) had a lower BMI for similar CVD risks as the USA population (43). This was attributed to a greater degree of central obesity in the Asia Indian population despite having smaller traditional anthropometric measurements in comparison to the USA counterparts. Another study in the Asian India population involving four large states showed that 79% of the general population had at least one lipid abnormality with low HDL-C identified as the common lipid abnormality at 44.9 % (44). Factors attributed to these findings included increased plasma insulin levels, insulin resistance, increased waist circumference, excess visceral fat and low adiponectin levels which were reported to be part of the Asian Indian phenotype (45). These findings reiterate the importance of abdominal adiposity as important parameter in determining CVD risk.

Some African studies also indicate a high prevalence of dyslipidaemia; Oguejiofor O et al in Nigeria found the prevalence of dyslipidaemia at around 60% in apparently healthy adults, with low HDL-C and high LDL-C consistently being high (46). Another study in South-West Nigeria showed a high prevalence of dyslipidaemia (58.7%) in newly presenting hypertensive patient (47). These findings were consistent with a study in North-Central Nigeria in which Adamu C et al found a high prevalence of dyslipidaemia (64%) in newly presenting hypertensive patients(48). Adamu C et al further identified a high BMI as being common in the hypertensive population and linked it with high LDL-C. However, the investigators were silent on the association of abdominal circumference with fat mass distribution as an important clinical parameter in assessing cardiovascular risk (40,49,50).

On the other hand, Lepira FB et al in Congo DR showed that abdominal obesity, was common in the hypertensive population (51). The study also found that dyslipidaemia, particularly hypercholesterolaemia, was common in nearly half of the hypertensive subjects and controls. This is in contrast with the Nigerian studies where high LDL-C and low HDL-C were identified as common abnormal lipid parameters. Furthermore, a study in Soweto, South Africa (SA), by Sliwa K et al on the *de novo* presentations of heart diseases showed that low HDL-C was a common lipid abnormality across ethnicities; African descent, white European, mixed-ancestry and Indian patients (52). Distinctively, raised TC was lowest among Africans in comparison to other ethnicities. Some other studies in the black population of SA have shown similar

trends with TC being identified as the least likely lipid abnormality and low HDL-C as the common lipid abnormality (53,54). However, a report by Swart W showed that TC was higher in South Africans of African descent than in the Indian or mixed race group, while it was lower than in the Caucasian group (55). This study however, involved several states and projected a different picture of dyslipidaemias in SA compared to that in the other aforementioned studies that were confined only one geographical location. All in all, the different findings arising from variable geographical locations further justifies the need to have extensive studies among the African population.

Zambia is a lower middle-income country, and like most similar countries, is also facing the challenges of non-communicable diseases (NCDs). The commonwealth report in 2008 indicated that NCDs accounted for 27% of the deaths across all age groups in Zambia, with cardiovascular diseases being the most prevalent at 12% (56). In 2008, a population based survey in Zambia's capital and largest city, Lusaka, showed a high prevalence of hypertension and cholesterol at 34.8% and 15.8% respectively (17). These results were comparable with those from a South African based survey among black population in Cape Town in which the prevalence of hypertension was at 38.5% and significantly correlated to a high BMI (57). Similarities were also observed in risk factors for hypertension in both the South African and Zambian surveys. Both surveys were however silent on the factors associated with co-existing hypertension and dyslipidaemia in the study population.

CHAPTER 3

Research methodology

3.1 Research design and setting

This was an analytical cross-sectional study where participants were selected on the spot, and without follow-up. Study participants were consecutively enrolled from the Adult Outpatient Clinic (AOC) (Clinic 5) at the University Teaching Hospital, Lusaka Adult Hospital, as they came for their usual medical appointments. This was done from January, 2017 to July, 2017. Patients with a diagnosis of hypertension as per case definition, regardless of duration or control of disease were recruited. Non-hypertensive and non-diabetic hospital staff, patients' relatives including their spouses were used as controls.

3.2 Sampling method

The sampling technique used was simple random sampling. Hypertensive patients were given numbers upon presentation to the hospital on their files as the nurses checked and recorded their vitals. Then two to four files were randomly selected each day from a pile of files for enrolment in the study in no particular order. Those who met the inclusion criteria from the selected files were enrolled. At least 2 to 4 controls were selected from those walking in to the clinic (staff members, relatives and spouses of patients) between 6 am to 9 am in no particular order. Those who met the inclusion criteria and consented were enrolled.

3.3 Sample size

Calculated sample size was 158 based on OpenEpi, version 3, open source calculator for sample size for unmatched case control was used with two-sided confidence level(1-alpha) at 95% and power of 80% to detect differences between cases and controls. Using a ratio of one to one, hypothetical proportions of controls with exposure was taken to be 15 % and hypothetical proportions of cases with exposure at 35.5 %, odds ratio 3.12, a sample size of 158 was calculated with continuity correction.

3.4 Study variables

Dependent variables

Primary outcome variable; Dyslipidaemia with hypertension (dyslipidaemic hypertension).

Secondary outcome variable; Dyslipidaemia without hypertension

Independent variables; demographic characteristics, BMI, abdominal fat composition (percentage), waist-circumference, waist-hip ratio, physical inactivity, educational levels and smoking history.

3.5 Inclusion and Exclusion criteria

Inclusion criteria

Any individual, either gender who were 18 years and above. **A case** was any a person with a systolic blood pressure (SBP) ≥140 mmHg and diastolic blood pressure of (DBP) ≥ 90 mmHg. A **control** was a non-hypertensive without any prior diagnosis of hypertension or previously treated for hypertension. A written consent was sought from each participant prior to recruitment.

Exclusion criteria

Individuals with certain medical condition which may influence outcome of the study were not included in the study. Those excluded were;

i. Those confirmed to be on lipid lowering agents (Atovastatin and Simvastatin) and corticosteroids (Prednisolone) either through file records or review of medications

ii. Hypertensives on thiazide diuretics and beta-blockers

iii. Individuals with diabetes mellitus or co-morbid diabetes mellitus and hypertension according to the case definition

iv. Pregnant women based on Gravindex test or manual palpation or Ultrasound

v. Patients with chronic medical conditions. These included, malignancy, Chronic Kidney disease whether on dialysis or not, HIV-positivity, thyroid disease confirmed CLD through medical records.

vi. Ascites and any palpable abdominal mass on physical examination

vii. Women on oestrogen-containing contraceptives (eg. Microgynon) for at least 3 months preceding the study

The flow chart below shows the enrolment process

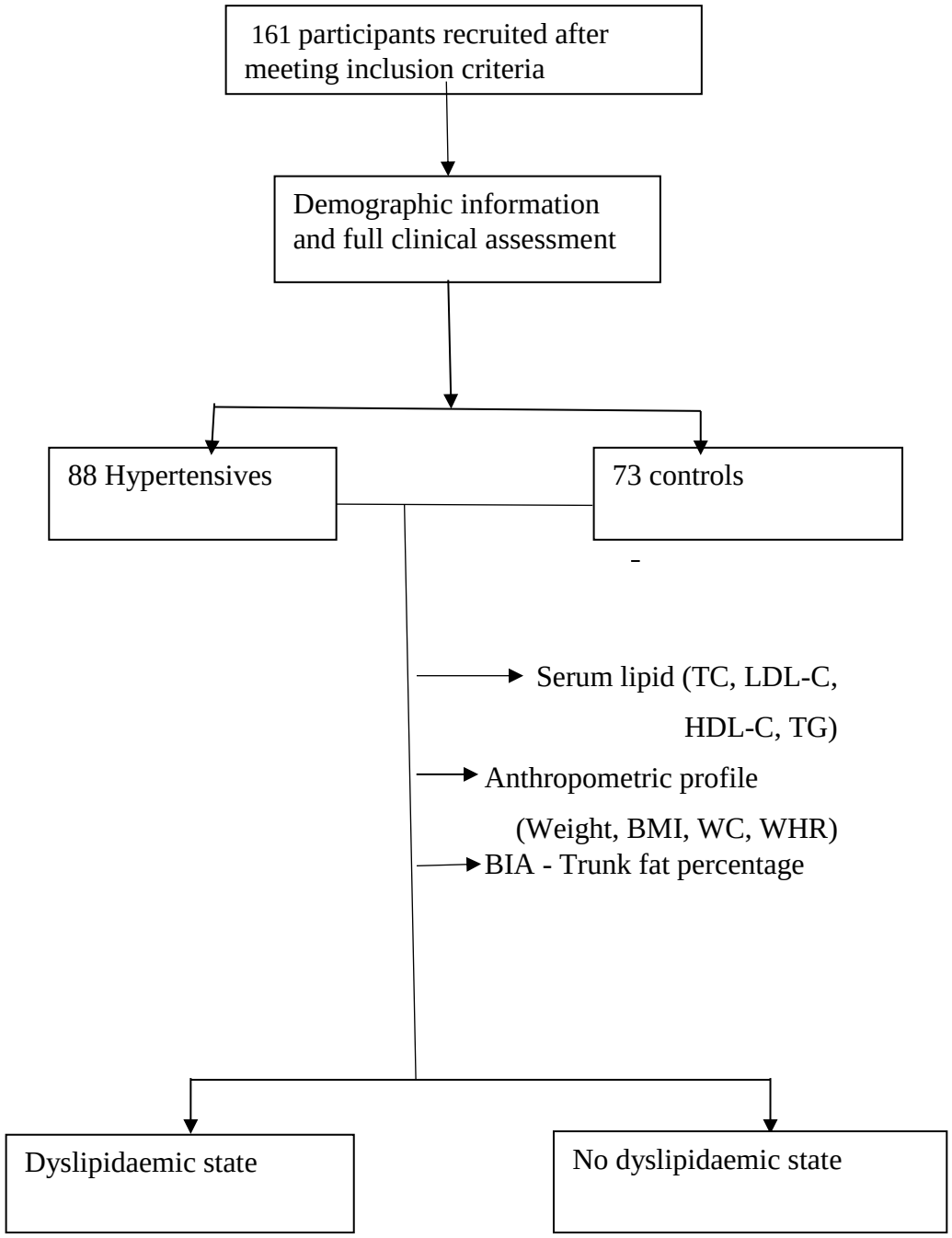

Figure 2: Flow chart of the enrolment process of participants from the AOC

3.6 Clinical parameters

Participants were recruited between 7am and 9am, daily from Monday to Friday. All those in the overnight fasting state of 10-12 hours were identified. Demographic information obtained were age, sex and lifestyle activities which included exercise, diet, alcohol consumption and smoking.

Blood Pressure measurements was taken after 5 minutes of rest using an Omron M10-IT Upper Arm Blood Pressure Monitor with Dual-User Facility and Dual size cuff. The BP was taken in an upright sitting position with the arm fully supported on a flat surface at heart level and feet flat on the floor, observing quietness and avoiding movement during measurements. An average of 3 consecutive readings taken 5 minutes apart were used for analysis.

With a needle prick, Serum fasting blood glucose was measured with ACCUchek glucometer

Physical examination was done to check for xanthomas and xanthelasma.

3.7 Anthropometric measurements

Weight (in kilograms) measured to the nearest 0.1kg and Height (in metres) was measured to the nearest 0.1m in no more than one layer of light clothing and bare feet. Height was measured with a stadiometer. BMI was calculated as weight (kg)/height (m^2) on a TANITA body composition analyser. Waist circumference was taken in an erect posture between the iliac crest and lower border of last rib to the nearest centimetre while hip circumference was taken at the widest part of the buttocks using a dress-makers' tape measure.

3.8 Laboratory parameters

Blood was drawn in heparinised bottles and analysed for serum plasma fasting lipid profile (TC, HDL-C, LDL-C and TG levels). Samples were analysed using standardised Cobus 611 chemistry analyser (TC, LDL-C, HDL-C, TG)..

3.9 Data analysis

Baseline descriptive statistics

General characteristics and demographic information of study participants was presented using simple descriptive statistics. Means, interquartile ranges (IQR), medians and ranges were calculated to understand the variable distribution. Age, height and BMI were not normally distributed while the rest of the continuous variables were normally distributed based on Skewness Kurtosis tests. All normally distributed variables were calculated by use of means and ranges while the skewed ones were calculated by use of medians and IQR. Comparisons were made using Student t-test for normally distributed variables and Mann-Whitney U test for the skewed variables. Chi-square was used to analyse categorical variables. Two sample tests of proportions were used to calculate the differences of two independent samples to ascertain the statistical significance.

Analytical statistics

Logistic regression analysis was used to assess association between the dependent and independent variables. Univariate analysis of the individual independent variables was used to measure their associations with dependent variables under study. The statistically significant associations at p value of < 0.05 in univariate analysis were further assessed in the multivariate model to account for potential confounders.

Significant findings were reported taking into consideration a p- value of < 0.05 for all the results. Data was analysed using STATA version 15

3.10 Study Limitation

Lipid parameters measured in this study only included TC, LDL-C, HDL-C and TG. Other lipids such VLDL, ApoB and Lipoprotein(a) which are useful atherogenic lipids and assessment of familial hypercholesterolaemia were not included. Plasma insulin levels were not measured as an index for insulin resistance but assumptions were made based on other clinical parameters such as acanthosis negricans. Since this study was also carried out in a hospital setting, the study population may not have been a best representative of Lusaka population, though the findings do provide an insight about the bigger picture especially that controls were included. Though care was taken in calculating the sample size, it is possible that the size may not have been adequate

enough resulting in weak correlations between clinical parameters and lipoproteins abnormalities.

CHAPTER 4

Results

A total of 175 participants were recruited. Out of these 15 were excluded from the study. Of those excluded, 6 were male and 9 were females. Among the females, 1 was found to have features of Graves' Disease which was later confirmed by laboratory tests, 4 were HIV positive and 4 were found with raised FBS. Among the males, 1 opted out of the study, 2 were HIV positive on treatment, 2 had raised FBS and 1 was on statin therapy. Therefore, a total 161 participants were analysed, of which 88 were hypertensives and 73 controls. BMI had 10 missing values, Visceral (Trunk) fat percentage 11 while Fasting Blood Sugar had 2 missing values.

Demographic, clinical and biochemical parameters are as shown in Table 1. The hypertensives were noted to have a significantly higher median age (47 vs. 38, $p<0.0001$) than the controls. The mean SBP and DBP was also significantly higher in hypertensives than controls; 141mmHg vs 116mmHg ($p< 0.0001$) and 89mmHg vs 74mmHg ($p< 0.0001$) respectively. Other variables noted to be significantly higher in hypertensives was the weight (73 vs. 68, p=0.0491), WC (87kg vs. 78kg, p=0.0058), BMI (27kg/m vs. 25kg/m, p=0.0373), WHR (0.86 vs. 0.81, p=0.0008) and TC (4.4 vs. 4.0, p=0.0152). However, the distribution of height, Visceral fat percentage, HDL-c, LDL-c, TG, FBS and smoking history did not differ in the two group.

Table 2 shows the prevalence and patterns of dyslipidaemia in the hypertensive and controls. Among those who had dyslipidaemia, 53.2% were hypertensive while 46.8% were controls. It was also observed that isolated low HDL-c was the commonest lipid abnormality in both hypertensives and controls (15.9% and 28.8%, respectively, p=0.3789). Combined dyslipidaemia with at least two lipid abnormalities was significantly higher in hypertensives than controls **(14.8 % vs 8.2%, p= 0.0046).** Other lipid abnormalities were not statistically significant in both groups.

Table 1: Demographics, clinical, and biochemical characteristics of the study participants

	Hypertensives	**Controls**	
Variable	**Total =88 (55%)**	**Total=73 (45%)**	**P-value**
Age (years)[mi]	47 (39, 58)	38 (27, 48)	[*]**<0.0001**[w]
Sex			
Female	34 (38.6%)	53 (72.6%)	**<0.001**[c]
Male	54 (61.4%)	20 (27.4%)	
Systolic blood pressure[mr]	141 (85, 209)	116 (91, 133)	***<0.0001**[t]
Diastolic blood pressure[mr]	89 (63, 137)	74 (57, 89)	***<0.0001**[t]
Weight (kgs)[mr]	73 (46, 126)	68 (41, 106)	***0.0491**[t]
Height (meters)[mi]	1.6 (1.6, 1.7)	1.6 (1.6, 1.7)	0.8251[w]
[a]BMI (kg/m)[mi]	27 (18, 41)	25 (16, 39)	***0.0373**[w]
WC (cm)[mr]	87 (75, 95)	78 (70, 91)	***0.0058**[t]
WHR[mr]	0.86 (0.80, 0.90)	0.81 (0.76, 0.86)	***0.0008**[t]
[a]Visceral (trunk) fat percentage[mr]	27 (5, 51)	24 (3, 52)	0.0996[t]
Total cholesterol(mmol/L)[mr]	4.4 (2.4, 8.9)	4.0 (2.2, 6.5)	***0.0152**[t]
HDL-c (mmol/L)[mr]	1.4 (0.5, 2.5)	1.5 (0.05, 3.1)	0.4182[t]
LDL-c (mmol/L)[mr]	2.8 (1.0, 7.3)	2.6 (0.8, 5)	0.2245[t]
TG (mmol/L)[mr]	0.9 (0.3, 3.8)	0.8 (0.2, 2)	0.2216[t]
[a]FBS(mmol/L)[mr]	5.3 (3.0, 7.2)	5.6 (3.4, 7.3)	0.1474[t]
Smoked			
No	76 (86%)	62 (85%)	0.796[c]
Yes	12 (14%)	11 (15%)	

Abbreviations: [mi]median (interquartile range), [mr] mean (range), [w]mann-whitney u-test, [t]student t-test, [c]chi-square, WHR, Waist-to-Hip ratio, WC, Waist Circumference, BMI, Body Mass Index, HDL-c, high-density lipoprotein, LDL-c, low-density lipoprotein cholesterol, TG, triglyceride, FBS, fasting blood sugar *Statistically significant ,

[a] Missing values

Table 2: Prevalence and patterns of dyslipidaemia among study participants

Variable	Hypertensives n (%)	Controls n (%)	P-value
No abnormal lipid parameter	47 (53.4)	37 (50.7)	0.8057
2 lipid abnormalities	13 (14.8)	6 (8.2)	**0.0046**
> 2 lipid abnormalities	4 (4.6)	3 (4.1)	0.895
Hdl	14 (15.9)	21 (28.8)	0.3789
Ldl	3 (3.4)	4 (5.5)	0.5812
Tc	7 (7.9)	0 (0.0)	Na
Tg	0 (0.0)	2 (2.7)	Na

Abbreviation: [na]Not applicable

Table 3 shows the univariate and multivariate analysis of factors associated with dyslipidaemia. During univariate analysis, it was shown that a unit increase in WC (OR 1.03; 95%CI 1.01, 1.06; p=0.007), WHR (OR 218.86; 95%CI 2.04, 23485.35; p=0.024), TC (OR 1.75; 95%CI 1.26, 2.43; p=0.001), LDL-c (OR 2.24; 95%CI 1.47, 3.40; p<0.001), and TG (OR 3.60; 95%CI 1.51, 8.59; p=0.004) were associated with an increased chance of having dyslipidaemia. While a unit increase in HDL-c had a statistically reduced chance of having dyslipidaemia by about 87% (OR 0.13; 95%CI 0.05, 0.35; p<0.001). However, during multivariate analysis, only Sex; male gender was significantly associated with a reduced chance of having dyslipidaemia (OR 0.22; 95%CI 0.09, 0.53; p=0.001**)** as compared to their female counterparts, TC (OR 3.61; 95%CI 2.08, 6.28; p<0.001) and HDL-c (OR 0.03; 95%CI 0.01, 0.1;1; p<0.001) came out as significant predictors of dyslipidaemia.

Table 3: Multivariate and univariate analysis of various clinical and laboratory parameters associated with dyslipidaemia

	Univariate		Adjusted	
Variable	**OR (95%CI)**	**P-value**	**OR (95%CI)**	**P-value**
Age (years)	1.01 (0.99, 1.03)	0.350		
Sex				
Female	R	**R**	R	R
Male	0.58 (0.31, 1.09)	0.089	0.22 (0.09, 0.53)	**0.001**
WC (cm)	1.03 (1.01, 1.06)	**0.007**		
BMI (kg/m)	1.06 (1.00, 1.12)	0.053		
WHR	218.86 (2.04, 23485.35)	**0.024**		
Systolic blood pressure	1.00 (0.99, 1.01)	0.963		
Diastolic blood pressure	0.99 (0.97, 1.01)	0.437		
Total cholesterol	1.75 (1.26, 2.43)	**0.001**	3.61 (2.08, 6.28)	< **0.001**
HDL-c	0.13 (0.05, 0.35)	< **0.001**	0.03 (0.01, 0.11)	< **0.001**
LDL-c	2.24 (1.47, 3.40)	< **0.001**		
TG	3.60 (1.51, 8.59)	**0.004**		
Education status				
None	R	R		
Primary	1.22 (0.41, 3.59)	0.719		
Secondary	0.58 (0.20, 1.65)	0.307		
Tertiary	0.32 (0.08, 1.21)	0.092		
Employment status				
No	R	R		
Yes	1.01 (0.51, 1.98)	0.988		
Smoked				
No	R	R		
Yes	0.66 (0.27, 1.63)	0.369		
Consumed alcohol				
No	R	R		
Yes	0.64 (0.34, 1.19)	0.160		

Table 3,cont.

Variable	Univariate OR (95%CI)	P-value	Adjusted OR (95%CI)	P-value
Physical exercise				
No	R	R		
Yes	0.85 (0.44, 1.66)	0.637		
Visceral fat				
Abnormal	R	R		
Normal	0.60 (0.26, 1.38)	0.230		

Abbreviation: R, reference; OR, odds ratio; CI, confidence interval

Table 4 shows the univariate and multivariate analysis of factors associated with dyslipidaemic hypertension. At univariate analysis, it was observed that increase in Age (OR 1.04; 95%CI 1.01, 1.07; p=0.003), WC (OR 1.04; 95%CI 1.01, 1.07; p=0.006), BMI (OR 1.07; 95%CI 1.00, 1.14; p=0.036), SBP (OR 1.03; 95%CI 1.02, 1.05; p<0.001), DBP (OR 1.04; 95%CI 1.01, 1.07; p=0.009), TC (OR 1.92; 95%CI 1.35, 2.73; p<0.001), HDL-c (OR 0.19; 95%CI 0.07, 0.56; p=0.002), LDL-c (OR 1.72; 95%CI 1.18, 2.51; p=0.005) and TG (OR 4.98; 2.00, 12.36; p=0.001) were significantly associated with the occurrence of dyslipidaemic hypertension. While one unit increase in HDL-c significantly reduced the chance of having dyslipidaemic hypertension by 81% (OR 0.19; 95%CI 0.07, 0.56; p=0.002), other factors such as sex, smoking, alcohol consumption, education levels, visceral fat percentage and physical exercises did not show any significant associations at p value of 0.05. However, during multivariate analysis, only lipid parameters; TC (OR 2.41; 95%CI 1.57, 3.69; p<0.001), HDL-C (OR 0.10; 95%CI 0.03, 0.39; p=0.001), and TG (OR 3.28; 95%CI 1.12, 9.63; p=0.031) showed to be significantly associated with occurrence of dyslipidaemic hypertension**.**

Table 4: Univariate and multivariate analysis of various clinical and laboratory parameters associated with dyslipidaemic hypertension.

Variable	Univariate OR (95%CI)	P-value	Adjusted OR (95%CI)	P-value
Age (years)	1.04 (1.01, 1.07)	**0.003**		
Sex				
Female	R	**R**		
Male	1.98 (0.96, 4.06)	0.063		
WC (cm)	1.04 (1.01, 1.07)	**0.006**		
BMI (kg/m)	1.07 (1.00, 1.14)	**0.036**		
WHR	1.63 (0.78, 3.41)	0.197		
Systolic blood pressure	1.03 (1.02, 1.05)	**< 0.001**		
Diastolic blood pressure	1.04 (1.01, 1.07)	**0.009**		
Total cholesterol	1.92 (1.35, 2.73)	**< 0.001**	2.41 (1.57, 3.69)	**< 0.001**
HDL-c	0.19 (0.07, 0.56)	**0.002**	0.10 (0.03, 0.39)	**0.001**
LDL-c	1.72 (1.18, 2.51)	**0.005**		
TG	4.98 (2.00, 12.36)	**0.001**	3.28 (1.12, 9.63)	**0.031**
Education status				
None	R	R		
Primary	0.37 (0.12, 1.13)	0.081		
Secondary	0.38 (0.13, 1.12)	0.078		
Tertiary	0.39 (0.10, 1.53)	0.178		
Employment status				
No	R	R		
Yes	0.82 (0.37, 1.82)	0.629		
Smoked				
No	R	R		
Yes	1.04 (0.38, 2.84)	0.941		
Consumed alcohol				
No	R	R		
Yes	0.84 (0.411.72)	0.627		
Physical exercise				
No	R	R		
Yes	0.86 (0.40, 1.86)	0.701		
Visceral fat				
Abnormal	R	R		
Normal	0.53 (0.19, 1.51)	0.237		

r; reference group; OR, odds ratio; CI, confidence interval

CHAPTER 5

Discussion

The results from this study showed that dyslipidaemia was prevalent in both hypertensive and control groups. Of note is that in both the DH and dyslipidaemia group, TC and HDL-C were the factors strongly associated with the occurrence of dyslipidaemia.

The hypertensive population was significantly older and had higher anthropometric measurements than controls. These were BMI, WC and WHR. These findings were comparable to what Lepira et al. in Congo DR observed (23). Another study in Nigeria by Osuji et al. also reported higher BMI and WHR and mean TC among hypertensives than the controls (24). Some studies have demonstrated that high TC is common among hypertensives. For example, Framingham Heart Study (11) demonstrated a link between cholesterol and hypertension. It is known that high cholesterol levels lead to a reduction in the synthesis and release of Nitric Oxide; a naturally occurring vasodilator, and increases activity in the RAAS and endothelin – both involved in vasoconstriction – which results in a net increase in BP (4). However, other lipids and lipoproteins had similar prevalence in both groups.

Analysis based on the prevalence of dyslipidaemia in both the study group and the controls had comparable results with nearly half of the study participants having dyslipidaemia. This is similar with what was found by Lepira et al. in Congo DR (23) and Akitunde in Nigeria (15) where no differences in the overall prevalence of dyslipidaemia in both hypertensive and controls was observed. Contrary to these findings, Osuji et al. found that hypertensive population had a higher prevalence of dyslipidaemia than controls within the sub-Saharan population (24).

Low HDL-C was the commonest occurring lipid abnormality in both hypertensive and controls. This result was also obtained in previous studies conducted in South West Nigeria (15) and South South Nigeria (25). Different geographical locations in Africa have revealed variations in lipid profiles with some reporting a high TC and low HDL-C as the commonest occurring lipid abnormality (17,23,26). Some contributors to the findings in these studies included rural-urban migration and

adoption of Western lifestyle, but whether this is due to also genetics or environment factors is an area that needs further research.

Even though hypertensives appeared to have high anthropometric indices for obesity, the study showed that only TC, HDL-C and triglycerides showed a strong association with DH. DH is known to constitute the important modifiable component of insulin resistance syndrome (27). Alteration in lipid parameters such is elevated TC, reduced HDL-C and increased triglycerides all form part of insulin resistance syndrome (12) and could have explained this strong association. However, this study did not explore potential causes of lipoprotein abnormalities occurring independent of obesity indices such as normal weight dyslipidaemia (28). Even though prevalence of normal weight dyslipidaemia in the general population is not known, data from Western regions put estimates at 10 to 37% (29). It has been postulated from studies that certain individuals have non-alcoholic fatty liver disease (NAFLD) without obesity (30). And like with obesity, NAFLD may impair metabolic homeostasis manifesting as dyslipidemia, low-grade systemic inflammation, and insulin resistance (IR) all of which are important components of MS (31). This may provide insight for future studies to ascertain the role of normal weight dyslipidaemia among the African population.

Overall, high TC and low HDL-C were strongly associated with the of occurrence of dyslipidaemia regardless of presence or absence of hypertension, after controlling for advanced age, sex, anthropometric measurements and high blood pressure levels. Of note, however, is that male sex had a reducing effect on the occurrence of dyslipidaemia. This finding may again give light to future prospective studies in our population to demystify the role of demographical, genetic and hormonal factors in the occurrence of dyslipidaemia, and possibly the role of sedentarism and type of occupational differences between the sexes. Nonetheless, the screening of all major risk factors is imperative as dyslipidaemia affects substantial proportions of both hypertensive and non-hypertensive population.

CHAPTER 6

Conclusion and Recommendations

Our study showed that the prevalence of dyslipidaemia was high in both the hypertensive population and controls although combined dyslipidaemia was significantly higher in the hypertensive population. An increase in total cholesterol and triglycerides, and a reduction in high density lipoprotein cholesterol was significantly associated with dyslipidaemic hypertension. Traditional risk factors such as age, physical inactivity, smoking status, alcohol consumption and obesity indices (BMI, WC, WHR, Visceral fat rating) did not show strong associations.

Based on the findings above. The following are the recommendations;

i. The management of hypertension should also focus on identifying and correcting associated lipid disorders as a high percentage of hypertensives had dyslipidaemia.
ii. Protocols should be put in place to treat dyslipidaemia in the hypertensive patients.
iii. There is need to do geographical and genetic profiling of lipid and lipoprotein abnormalities in our population to ascertain the extent of dyslipidaemia in the population without obvious traditional risk factors.

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APPENDICES

Appendix I: Information sheet

Information sheet for the study on **"The Clinical Associations of Dyslipidaemia Among Hypertensive Adults Presenting to The University Teaching Hospital (UTH), Lusaka Adult Hospital, Zambia."**

You are invited to take part in this study, which is looking at dyslipidaemia and its clinical profile, as well as its relationship to abdominal fat content, in hypertensive patients presenting to UTH. This study is being done as part of requirement for a master of internal medicine degree. Information about this study is supplied in this document. One of the study team investigators will be on hand to explain the contents of this document and answer all your questions. Please make sure that you understand everything in this document. If you decide to participate you will be asked to give consent before you take part.

Participation in this study is completely voluntary. You are under no obligation to take part in the study and are free to withdraw from this study at any time. Withdrawal or refusal to participate will have no consequences on your further medical care. No financial reward will be given to any persons taking part in this study.

Title of study

The Clinical Associations of Dyslipidaemia Among Hypertensive Adults Presenting to The University Teaching Hospital (UTH), Lusaka Adult Hospital, Zambia

- **Who is doing the study?**

Dr. Katongo Hope Mutengo is the main researcher under the supervision of Dr. Brown Kamanga and Dr Soka Nyirenda. The main researcher is responsible for the day to day running of the study. We can be contacted via Department of Medicine, University Teaching Hospital, Lusaka, Zambia**.**

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The study has been approved by the Biomedical Research Ethics Committees of the University of Zambia (UNZA BREC) and the School of Medicine post graduate forum. They can be contacted on the following number: +260 1 256067 or at this address: Biomedical Research Ethics Committee, University of Zambia, Ridgeway Campus, PO Box 50110, Lusaka, Zambia.

- **What is the purpose of this study?**

  The purpose of this study is to identify people who may be at increased risk for complications of heart diseases. These complications include changes in the structure of heart muscles also known as remodelling, which may impair the function of the heart resulting heart failure, a slow or fast heartbeat, or an irregular heart beat collectively known as arrhythmias. Both heart failure and arrhythmias can impair blood flow to the important organs of the body, such as the brain and kidneys. They can also increase one's chance of developing heart attack. Consequences of heart failure may include breathlessness, getting tiredness easily even on minimal activity, such as walking, climbing stairs or doing household chores. Sometimes one may experience intense chest pain or sudden death. There may also be clots of blood forming in the heart and blood vessels due to slowing of blood flow as a consequence of heart failure and arrhythmias. The blood clots may sometimes dislodge and flow with the blood to the brain where they can lodge in smaller blood vessels, blocking further flow of blood to a part of the brain resulting in stroke. Very high blood pressure can also rupture a blood vessel in the brain leading to stroke. Consequences of strokes include loss of certain functions that the affected person was able to do, such as walking and talking. People with hypertension have an increased risk for complications of heart disease. The risk is multiplied by the having certain abnormal fats in the blood, which can arise from the consumption of diets that are high in bad fats (e.g., animal fat), or sugar and/or unhealthy habits such as smoking and not exercising.

- **What is dyslipidaemia?**

  Dyslipidaemia, is having excess "bad" fats in the blood beyond the acceptable levels or reduction in the "good" fats which are protective. It can be due to

excess intake of fat and sugar, or the condition may run in the family. Dyslipidaemia puts one at an increased chance of heart disease as these 'bad" fats may block blood flow in the blood vessels preventing oxygen in blood to reach the important tissue. This may lead to one having complications such as heart attack or stroke.

Procedure of the study

- If you agree to take part in this study you will be asked to sign or print a consent form. You will be given a copy of this information sheet and the consent form to keep
- Preliminary questions about your age, residence, occupation and some illnesses (in both you and your immediate family members) will be asked. You will also be asked about your social activities such as alcohol consumption and smoking. This will take about 20 minutes.
- Your blood sample will then be collected between 7am to 9pm. It is alright to eat your usual meals at supper time but a blood sample will be collected only if you ate at least 10-12 hours prior to collection time. You are not required to eat any meals thereafter including breakfast until all measurements have been taken. Ensure you carry your breakfast with you which you will eat after the completion of the process.
- Ensure you are wearing light clothing inside such as boxers, bras, petticoats and cycling shorts. Your weight measurements and abdominal diameters will be measured in your light clothing by qualified personnel in a private comfortable room.
- Your blood pressure will be measured 3 times every 5 minutes after you have had 5 minutes' rest. The whole process of clinical examination will take about 30 minutes.
- You will then stand on a machine which appears a like a scale, with your bare feet. This machine will measure the components of fats in your body around the abdomen, arms and legs. It will also measure your body mass index (BMI) after entering your height, which is a ratio of your weight to your height calculated as weight (in kilograms)/ height (metre^2). It will provide information to you on the expected weight for your height.

- An HIV test is a requirement to participate in the study, trained counsellors will be available to provide the services to you. You have the right to refuse the test or knowledge of the test results.
- If you wish to know all your results, we will avail them to you using the contacts details you will provide. **No treatment will be provided to you during the procedure**, in the event that you require it, you will be referred to the appropriate persons to see you.
- The information you will provide will be analysed together with the other information from other participants.

Are there any risks for people taking part in this study?

- People often get alarmed at the knowledge that they (may) have a potentially life-threatening heart disease or at high risk of such. Since this study may bring out such, proper psychological care will be provided in such an event and appropriate measures for referral will be provided in good time. Some questions asked may also be personal, for instance, this study may bring out weight problems which may be communicated to you. Therefore, if you feel uncomfortable, you have the right to stop the interview.

Benefits

- The benefit of this study is the understanding of clinical factors associated with dyslipidaemia in adults presenting with hypertension at UTH. We hope the information obtained from this study would help clinicians to risk stratify patients upon presentation to the hospital.

Confidentiality

All information that you give in the interview is confidential. Only number codes will be used for your identity during data collection. Your identity will not be disclosed in any publication resulting from this study. All data obtained will be kept securely and only medical personnel participating in the research will have access your information. The University of Zambia and department of internal medicine at UTH may however review the data for verification.

*If you have any questions about this study, please ask them now. If you have any later questions or concerns please contact**, DR KATONGO H. MUTENGO AND THE CHAIRPERSON, UNZA BREC on numbers and addresses provided above**.*

Kindly ensure this information sheet is kept safe.

Appendix II: Informed consent form

Informed consent form for the study on "The Clinical Associations of Dyslipidaemia Among Hypertensive Adults Presenting to The University Teaching Hospital (UTH), Lusaka Adult Hospital, Zambia

I have been invited to take part in a research project being conducted at the University Teaching Hospital by

Dr. Katongo Hope Mutengo
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Email: katmutengo@yahoo.com

1. The study is being supervised by Dr. Brown Kamanga and Dr. Soka Nyirenda, Department of Internal Medicine; Tel +260 972 758 137 and +260977775662.
2. I have been told the purposes of this research and understand the processes involved. I understand the potential distress that may occur.
3. I have been given a list of names and addresses of people and institutions I may contact in relation to this research.
4. I have read the information in the **Information sheet for the study on "The Clinical Associations of Dyslipidaemia Among Hypertensive Adults Presenting to The University Teaching Hospital (UTH), Lusaka Adult Hospital, Zambia** or have had it read or explained to me.
5. I have had the opportunity to ask questions and have had these answered satisfactorily.
6. I understand that I have the right torefuse to participate in this study or withdraw from the study at any time. I understand that refusing to take part or withdrawing from the study will in no way compromise my clinical care.
7. I agree to take part in the study

Participant's information:

Signature (or fingerprint): _____

Surname:

Name: _____ (please print)

Date: _____

The person who conducts the informed consent discussion must also sign and date this form.

Signature: _____

Date: _____

Surname: _____

Name: _____ (please print)

Signature of witness, if applicable.

Witnessed by: (print name): _____

Signature of Witness: _____

Appendix III: Data collection sheet

1	**Subject ID: _____** **Contact details: phone _____ other_____**
2	**Sex: □ Male □ Female □ DOB:____ /___/ ____**
3	**Education level (Tick as appropriate):** **No education □ Primary□ Secondary □ Tertiary□**
4.	**Employment status: □ Yes □No** **Salary/month:** **□Less than K1,100 (less than 100USD)** **□ K1,100-5,000 (1100-5,500USD)** **□K 5,000-10,000 (500-1000USD)** **□ K10,000-15,000 (1100-1, 650USD)** **□ Greater than K15,000 (1, 650 USD)**
5	**Cigarette/Tobacco Smoking (tick as appriopriate)** **Have you ever smoked?** **a) Yes_____2. No_____ (If no skip to question 6)** **b) Are you a current smoker 1. Yes_____ 2. No (If no, skip to part e.)** **c) Age started smoking_____** **d) No. of cigarettes per day i. (1-5)___ ii. (6-9) iii. (10-20)___** **e) Former smoker for more than one year i._____ ii. Less than one year_____ (tick as appropriate)**
6	**Alcohol** **(Tick as appropriate)** **a) Have you ever taken alcohol i. Yes_____ ii. No_____(if no skip to question 7)** **b) Are you still taking alcohol 1. Yes_____ii. No_____ (if no skip to part f.)**

	c) What type of alcohol do you take i. Opaque____ ii. Beer_____iii. Wine_____ iv. Distilled Spirits_____ **d) How long have you been taking alcohol i. less than 5 years_____ ii. 5-10 years_____ iii. More than 10 years_____** **e) How much alcohol do you take/week i. 1-5 bottles ii. 5-10 bottles_____ iii. More than 10 bottles iv. Other_____** **f) When did you last take alcohol? i. < 1 year____ ii. 1-5 years iii. > 5 years____**
7	**Diet** **1. Dietary Fiber Intake:** **A. Servings of fruits (and vegetables)** **a) Daily i) Yes_____ ii) No_____(Proceed to part 7.2B if Yes)** **b) Weekly i) Yes_____ ii) No_____** **c) Number of times per week if Yes i. once_____ b. twice_____ ii. 3x or more_____** **2. High fat and salty foods:** **B. Frequency of eating fast foods (e.g. instant noodles, hamburgers, French fries, fried chicken skin):** **a. Daily i) Yes_____ ii) No_____ (proceed to part d. if Yes)** **b. Weekly i) Yes_____ ii) No_____ (Proceed to part d. if Yes)** **c. Monthly i) Yes_____ ii)No_____ (Proceed to part 8, if No)** **d. Number of times i) once_____ ii) twice_____ iii) 3x or more_____**

Number of time spent on the following activities in last week

8		**None**	**Less than one hour**	**1 to 3 hours**	**More than 3 hours**
1	**Physical exercise such as swimming, jogging, aerobics, football, tennis, gym workout etc.**				

2	**Cycling, including cycling to work and during leisure time**				
3	**Walking, including walking to work, shopping, for pleasure etc.**				
4	**Housework/Childcare Gardening/DIY**				

***Adapted from General Practice Physical Activity Questionnaire, NHS with some modifications**

CLINICAL INFORMATION

9	**Family history:** **i. Hypertension: a. Yes_____; b. No_____** **ii. Informed by blood tests they have too much fat? a. Yes _____; b. No____** **iii. Others_____**
10	**A. Past Medical history** **i. Hypertension a. Yes_____ b. No_____ (If No skip to part 10B.v)** **ii. Year of diagnosis with Hypertension _____** **iii. Other Chronic Diseases □ Cancer □ CKD □ Dialysis therapy □ CLD □ HIV □ Thyroid disease** **iv. Others_____** **v. Bleeding disorder a. Yes_____ b. No_____** **B. Drug History:** **i. Anti-hypertensive Date/month/year started Date/month/year stopped Duration**

	1) Nifedipine_____ ____ /_____ /___ ____ /_____ /____ _____ **2) Enalapril _____ ____ /_____ /____ ____ /_____ /____ _____** **3) Losartan_____ ____ /_____ /____ ____ /_____ /____ _____** **4) Atenolol _____ ____ /_____ /____ ____ /_____ /____ _____** **5) Modiuretic_____ ____ /_____ /____ ____ /_____ /____ _____** **6) Amlodipine_____ ____ /_____ /____ ____ /_____ /____ _____** **7) Other_____ _____ _____ _____** **ii. Any herbal medications taken to control high Blood Pressure? Yes_____ No_____(if no, skip to iv.)** **iii. If yes, state which one 1. _____ 2. _____ 3._____** **iv. Use of Asprin/Clopidogrel a. Yes_____ b. No_____ c. Duration i. _____months** **v. Hormonal contraceptive use:** **a) Current use: i. Yes_____ (if yes, skip to part d.) ii. No_____** **b) Previous use: i. Yes_____ ii. No_____(if no, skip to part 11)** **c) Last used if Yes in b) a. less than 3 Months _____b. More than 3 months _____ c. More than 6 months_____** **d) Type of contraception i. Oral_____ ii. Injectable_____ iii. Depot_____ iv. Implant_____v. Others_____** **e) Duration of use i. 1-2 years ii. >2-5 years iii. > 5 years**
11	**Previous use of** **a. Statins use** **b. Corticosteroids (Predinosolone/Hydrocortisone Dexamethasone)** **c. Niacin** **i. Yes-_____** **Yes-_____** **Yes_____** **ii. No- _____** **No-_____** **No_____**

PHYSICAL EXAMINATION

12	**i. Weight (kg): _____** **ii. Height(metres): _____** **iii. Waist circumference (cm): _____** **iv. Hip circumference (cm)_____** **v. Total body fat content: Absolute___ kg _____%** **vi. Trunk fat content: _____ kg _____%** **vii. Calculated BMI: _____**
13	**Presence of stigmata for dyslipidaemia:** **i. Xanthelasma a. yes_____ b. No_____** **ii. Corneal arcus a. yes_____b. No_____** **iii. Cutaneous Eruptive xanthomas a. yes_____ b. No_____**
14	**Blood Pressure after 5 minutes of rest with uncrossed legs** **Blood Pressure at 0 mins;_____ Blood Pressure at 5 mins;_____** **Blood Pressure at 10 mins_____** **Average Blood Pressure;_____**

LABORATORY INVESTIGATIONS

15	**i. Fasting blood glucose(mmol/l): _____** **ii. Total cholesterol: _____** **iii. LDL-C: _____** **iv. HDL-C: _____** **v. Triglyceride levels: _____** **vi. Liver enzymes: ALT: _____ AST_____** **vii. Creatinine_____ Urea_____**