PREVALENCE OF TARGET ORGAN DAMAGE AMONG HYPERTENSIVE PATIENTS AT MUHIMBILI NATIONAL HOSPITAL

DAR ES SALAAM, TANZANIA

Tuzo Lyuu, MD

MMed Internal Medicine Dissertation

Muhimbili University of Health and Allied Sciences

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By

Tuzo Lyuu

A dissertation Submitted in partial Fulfillment of the Requirements for the Degree of Masters of Medicine (Internal Medicine) of Muhimbili University of Health and Allied Sciences

Muhimbili University of Health and Allied Sciences

October 2012

CERTIFICATION

The undersigned certify that they have read and hereby for recommend for acceptance by the Muhimbili University of Health and Allied Sciences a of dissertation entitled ***Prevalence of Target organ damage among Hypertensive patients at Muhimbili National Hospital***, in fulfillment of the requirements for the degree of Master of Medicine (Internal Medicine) of Muhimbili University of Health and Allied Sciences.

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Date: _____

DECLARATION

AND

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I, Tuzo Lyuu, declare that this dissertation is my own original work and that it has not been presented and will not be presented to any other University for a similar or any other degree award.

Signature..... Date……..

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Thanks.

DEDICATION

This work is dedicated to my parents for their sacrifice and support.

ABSTRACT

Background: Poor controlled hypertension is associated with development of target organ damage and the assessment of target organ damage is important in the evaluation of all hypertensive patients as it provides important information on the severity of the hypertension, the cardiovascular risk and guide management of individual patient.

Objective: This study was done to determine prevalence of left ventricular hypertrophy, hypertensive retinopathy, renal insufficiency and stroke among hypertensive patients at Muhimbili national hospital.

Methodology: This was a cross-sectional hospital based study, carried out for six months, from July, 2011 to December; 2011 at Muhimbili National Hospital. Two hundred and forty hypertensive patients were consecutively recruited into the study. Standardized questionnaires were used to collect socio-demographic characteristics and clinical data of study participants. Target organ damage was assessed by physical examination, ECG, ECHO and estimation of glomerular filtration rate by creatinine clearance.

Results: Among study participants, 206 (85.8%) patients had at least one of the four hypertensive target organ damage studied (left ventricular hypertrophy, hypertensive retinopathy, renal insufficiency and stroke). The organ most affected was the heart presenting as left ventricular hypertrophy observed in 167 (69.6%) followed by hypertensive retinopathy 136 (56.7%), renal insufficiency 72 (30.0%) and Stroke in 22 (9.2%) patients.

Conclusion: This study showed a high prevalence of target organ damage among hypertensive patients admitted in medical wards or attending medical outpatient clinic. These findings illustrate the burden of uncontrolled hypertension in our setting.

Recommendation: Early workup to identify presence of organ damage is recommended in all hypertensive patients.

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ABBREVIATIONS

ACE	Angiotensin Converting Enzyme
ARB	Angiotensin receptor blocker
BP	Blood Pressure
BMI	Body Mass Index
CHD	Coronary Heart Disease
CHF	Congestive Heart Failure
CKD	Chronic Kidney Disease
DBP	Diastolic blood pressure
ECG	Electrocardiogram
ECHO	Echocardiogram
GFR	Glomerular Filtration Rate
HDL	High density lipoprotein
K/DOQI	Kidney Disease Outcomes Quality Initiative
LDL	Low density lipoprotein
LVH	Left ventricular Hypertrophy

LVM	Left ventricular mass
LVMI	Left ventricular mass index
MDRD	Modification of Diet in Renal Disease
MUHAS	Muhimbili University of Health and Allied Sciences
MNH	Muhimbili National Hospital
RBG	Random Blood Glucose
SBP	Systolic blood pressure
TOD	Target Organ Damage
W.H.O	World Health Organization

CHAPTER ONE

1.1.0 BACKGROUND AND LITERATURE REVIEW

Hypertension is a chronic disease that is increasing in prevalence worldwide, affecting all races and gender lines. It is a common risk factor for cardiovascular diseases. [1]

World Health Organization defines Hypertension as a rise in blood pressure (blood pressure, systolic above 140 and diastolic above 90 mm Hg) that increases risk for cerebral, retina, cardiac, and renal events. [2]

*CLASSIFICATION OF HYPERTENSION ACCORDING TO W.H.O

Category of BP	Systolic BP mmHg	Diastolic BP mmHg
Optimal	<120	<80
Normal	120-129	80-84
High normal	130-139	85-89
Grade I –mild	140-159	90-99
Grade II- moderate	160-179	100-109
Grade III- severe	>180	>110
Isolated systolic	>140	<90
Isolated diastolic	<140	>90
Sub group borderline	140-149	<90

*Adopted from Guidelines for the management of hypertension in Tanzania, 2009.

EPIDEMIOLOGY OF HYPERTENSION

Worldwide prevalence of hypertension was estimated to be 26% in 2005, with projected 60% increase in this prevalence by 2025. [3]

Currently, it is estimated that 1 billion individuals worldwide have hypertension, and approximately 7.1 million deaths per year may be attributable to hypertension.

Prevalence of hypertension has been increasing in Africa. Data published in the 1990's shows an overall prevalence of 5%–20%.[4-8]

The effects of a family history of hypertension, alcohol intake, obesity, salt intake, physical inactivity, and advancing age on the prevalence of hypertension are similar to those established for developed nations. [4, 5]

Studies done in Ghana, Nigeria, Egypt and South Africa, suggested that prevalence of hypertension in Africa is increasing and commonly exceeds 20%–25% in rural areas and is over 30% in urban and semi-urban areas.

A house-to-house survey in a rural Egyptian population, found a prevalence of hypertension of 27.9%.[6] Cappuccio et al reported a similar prevalence in Ashanti region, Ghana with a higher prevalence in semi-urban areas (32.9%).[7]

In a case finding study done in Nigeria, a prevalence of hypertension was 30.6%. The study revealed that age, occupation, body mass index, waist circumference and hip circumference were significantly associated with high blood pressure.[8]

In a rural adult black population from Limpopo Province in South Africa, Alberts et al found a hypertension prevalence of 25.5% among women and 21.6% among men. More often the relatively high prevalence of hypertension was accompanied by low prevalence of awareness, treatment and control. [9]

Prevalence of hypertension in East Africa is as high as reported in other regions of Africa. A prevalence study which was done in rural Ugandan district, age-standardized prevalence of hypertension was 30.5%. Factors found to be associated with hypertension include past and present alcohol use, being overweight, obesity, female sex and having attained tertiary education.[10]

In two linked cross-sectional population-based surveys of a middle-income urban district and a relatively prosperous rural area in Tanzania, Edwards et al reported an age standardized hypertension prevalence of 37.3% (32.2% to 42.5%) among men and 39.1% (34.2% to 44.0%) among women in the urban district, and 26.3% (22.4% to 30.4%) among men and 27.4% (24.4% to 30.4%) among women in the rural area. [11]

TARGET ORGAN DAMAGE

Uncontrolled hypertension is associated with increased risk of cardiovascular morbidity and mortality. The risk of cardiovascular mortality doubles for each 20 mmHg increase in systolic blood pressure and 10 mmHg increase in diastolic blood pressure above 'normal' blood pressure level. [12]

Poor controlled hypertension is associated with development of target organ damage and the assessment of target organ damage is important in the evaluation of a hypertensive patient. It provides important information on the severity of the hypertension and the cardiovascular risk assessment.[2]

Traditional markers of hypertensive target organ damage are retinal changes, decreased glomerular filtration rate, protenuria and left ventricular hypertrophy (LVH), as assessed either by the electrocardiogram (ECG) or echocardiography.

LEFT VENTRICULAR HYPERTROPHY

Heart disease is the most common cause of death in hypertensive patients. Hypertensive heart disease is the result of structural and functional adaptations leading to left ventricular hypertrophy, diastolic dysfunction, and Congestive heart failure, abnormalities of blood flow due to atherosclerotic coronary artery disease and microvascular disease, and cardiac arrhythmias.

Both genetic and hemodynamic factors contribute to left ventricular hypertrophy. The myocardium undergoes structural changes in response to increased after load. Cardiac myocytes respond by hypertrophy, allowing the heart to pump more strongly against the elevated pressure.

The contractile function of the left ventricle remains normal until later stages. Eventually, LVH lessens the chamber lumen, limiting diastolic filling and stroke volume. The left ventricular diastolic function is markedly compromised in long-standing hypertension.

An aberration in the passive relaxation of the left ventricle during diastole appears to exist. Over time, fibrosis may occur, further contributing to the poor compliance of the ventricle.

Individuals with left ventricular hypertrophy are at increased risk for CHD, stroke, CHF, and sudden death. Aggressive control of hypertension can regress or reverse left ventricular hypertrophy and reduce the risk of cardiovascular disease.[13, 14]

LVH is, to a great extent, a reversible risk factor; its regression in response to antihypertensive treatment significantly improves the cardiovascular outcome and long term prognosis.

Data on prognostic implication of Left ventricular hypertrophy in the Framingham study indicated that LVH has emerged as a powerful indicator of rapidly evolving lethal atherosclerotic disease[15, 16] hence the consistent identification of hypertensive patients with LVH is important in clinical practice.

The electrocardiogram is usually the first method used for assessing the presence of LVH.

QRS complex patterns seen in LVH include increased QRS voltage, prolonged duration of QRS complex, left axis deviation and Left Bundle Branch Block (LBBB).

Many different validated criteria are available for diagnosing LVH using ECG; examples include the Sokolow–Lyon[17], Cornell voltage[18], Cornell voltage QRS duration product criteria.[19]

ECG VOLTAGE CRITERIA

Sokolow + Lyon

S V_1+ R V_5 or V_6 > 35 mm

Cornell criteria

SV_3 + R avl > 28 mm in men

SV_3 + R avl > 20 mm in women

Cornell voltage QRS duration product criteria

[(RaVL+SV3)*QRS duration] ≥ 2400 mm · ms

These ECG criteria generally have high specificities and can therefore identify patients with the greatest LVM and the highest risk. However, they have low sensitivities meaning that a normal ECG does not exclude the presence of LVH.[20]

Since the sensitivity of the different ECG criteria may be as low as 7 to 35 percent with mild LVH and only 10 to 50 percent with moderate to severe disease hence echocardiography is the procedure of choice.

In clinical studies, LVH is frequently identified as Left ventricular mass (LVM) in echocardiogram.

Different measures of body size, such as height, weight, body surface area, are used for normalizing LVM, so as to enable comparison of individuals with different body statures. When indexed to body surface area, Cut-off values from the Framingham study are 132 $g/m^2$for men and 100 g/m^2 for women.

A proposed indexing by (height $^{2.7}$) provides the most accurate estimate of LV mass[21] and it is superior for predicting the impact of LVH on general and cardiovascular mortality in comparison to that using body surface area.

STROKE

Hypertension is an important risk factor for brain infarction and hemorrhage. Approximately 85% of strokes are due to infarction and the remaining percentage is due to hemorrhage (either intracerebral hemorrhage or subarachnoid hemorrhage).[22] The incidence of stroke rises progressively with increasing blood pressure levels, particularly systolic blood pressure in individuals >65 years. Treatment of hypertension convincingly decreases the incidence of both ischemic and hemorrhagic strokes.

RENAL DAMAGE

Despite widespread treatment of hypertension, the incidence of end-stage renal disease continues to rise. The explanation for this rise may be concomitant diabetes mellitus, the progressive nature of hypertensive renal disease despite therapy, or a failure to reduce blood pressure to a protective level. A reduction in renal blood flow in conjunction with elevated afferent glomerular arteriolar resistance increases glomerular hydrostatic pressure secondary to efferent glomerular arteriolar constriction. The result is glomerular hyper filtration, followed by development of glomerulosclerosis and further impairment of renal function.

Studies have demonstrated that a reduction in blood pressure may result in improved renal function. Therefore, earlier detection of hypertensive nephrosclerosis using means to detect microalbuminuria and aggressive therapeutic interventions, particularly with ACE inhibitor drugs, may prevent progression to end-stage renal disease.[23]

Nephrosclerosis is one of the possible complications of long-standing hypertension.

The risk of hypertension-induced end-stage renal disease is higher in black patients, even when blood pressure is under good control. Furthermore, patients with diabetic nephropathy who are hypertensive are also at high risk for developing end-stage renal disease.

The renin-angiotensin system activity influences the progression of renal disease. Angiotensin II acts at the afferent and the efferent arterioles, but more so, on the efferent arteriole, which leads to an increase of the intraglomerular pressure. The excess glomerular pressure leads to microalbuminuria. Reducing intraglomerular pressure using an ACE inhibitor has been shown to be beneficial in patients with diabetic nephropathy, even in those who are not hypertensive. The beneficial effect of ACE inhibitors on the progression of renal insufficiency in patients who are non diabetic is less clear. The benefit of ACE inhibitors is greater in patients with more pronounced protenuria.

Hypertension is commonly observed in patients with kidney disease. Volume expansion is the main cause of hypertension in patients with glomerular disease (nephrotic and nephritic syndrome). Hypertension in patients with vascular disease is the result of the activation of the renin-angiotensin system, which is often secondary to ischemia. The combination of volume expansion and the activation of the renin-angiotensin system are believed to be the main factor behind hypertension in patients with chronic renal failure.

The Kidney Disease Outcomes Quality Initiative (K/DOQI) of the National Kidney Foundation (NKF) defines chronic kidney disease as either kidney damage or a decreased kidney glomerular filtration rate (GFR) of less than 60 mL/min/1.73 m^2 for 3 or more months.

K/DOQI classifies the stages of chronic kidney disease, as follows:

Stage 1: Kidney damage with normal or increased GFR (>90 mL/min/1.73 m^2)

Stage 2: Mild reduction in GFR (60-89 mL/min/1.73 m^2)

Stage 3: Moderate reduction in GFR (30-59 mL/min/1.73 m^2)

Stage 4: Severe reduction in GFR (15-29 mL/min/1.73 m^2)

Stage 5: Kidney failure (GFR <15 mL/min/1.73 m^2 or dialysis)

RETINA DAMAGE

Hypertensive retinopathy has been reported more frequently in black patients due to common presentation of severe forms of hypertension among black population.[24]

The retinal circulation undergoes a series of pathophysiological changes in response to elevated blood pressure. [25] In the initial, vasoconstrictive stage, there is vasospasm and an increase in retinal arteriolar tone owing to local auto regulatory mechanisms. This stage is seen clinically as a generalized narrowing of the retinal arterioles. Persistently elevated blood pressure leads to intimal thickening, hyperplasia of the media wall, and hyaline degeneration in the subsequent, sclerotic, stage. This stage corresponds to more severe generalized and focal areas of arteriolar narrowing, changes in the arteriolar and venular junctions (i.e. arteriovenous nicking or nipping), and alterations in the arteriolar light reflex (i.e. widening and accentuation of the central light reflex, or "copper wiring"). This is followed by an exudative stage, in which there is disruption of the blood–retina barrier, necrosis of the smooth muscles and endothelial cells, exudation of blood and lipids, and retinal ischemia. These changes are manifested in the retina as microaneurysms, hemorrhages, hard exudates, and cotton-wool spots. Swelling of the optic disk may occur at this time and usually indicates severely elevated blood pressure (i.e. malignant hypertension). Malignant hypertension is rarely seen because of better methods for the control of blood pressure available in the general population.

However, these stages of hypertensive retinopathy described, may not be sequential. [26]

For example, signs of retinopathy that reflects the exudative stage, such as retinal hemorrhage or microaneurysm, may be seen in eyes that do not have features of the sclerotic stage (e.g. arteriovenous nicking).

The exudative signs are nonspecific, since they are seen in diabetes and other conditions.

EPIDEMIOLOGY OF TARGET ORGAN DAMAGE

Target organ damage has been encountered frequently in hypertensive patients. In a study that was done in Spain, using ECG, LVH was present in 22.9% of the hypertensive patients, renal insufficiency defined by GFR < 60 ml/min/1.73 m^2 45.1%, and abnormal Urine Albumin Excretion in 58.7%.[27]

Another study in the same country but only included patient with resistant hypertension, LVH were seen in 57.1% of all study participants.[28]

In South India, Shantha GP et al found that 18.9% had left ventricular hypertrophy diagnosed by ECG, 39.4% had microalbuminuria and 40% had retinopathy. [29]

In Africa high prevalence of target organ involvement has been reported. Among hypertensive patients in Nigeria, 60.1% of all hypertensive patients participated in one study had at least one target organ involved. [30] In Ghana, prevalence of 47.5% was found among hypertensive civil servants who were perceived to be healthy. [31] In the same study, left ventricular hypertrophy diagnosed by ECG was observed in 33.3% of the participants, history of stroke in 2.8% and 16% had ischemic changes on ECG. CKD was present in 4.1%, hypertensive retinopathy was observed in 70% of participants with 1.0% classified as having Grade 3 retinopathy. None of the participants investigated had hypertensive retinopathy grade 4. [31]

In a hospital based cross-sectional study assessing the state of blood pressure (BP) control and the pattern of target organ complications in Sudanese patients treated in a teaching hospital in

Kassala town in Eastern Sudan , Stroke was present in 6.6%. [32] In another study done in Sudan, in two teaching hospitals, the presence of TOD was 26.7%,53%,57.3%,36.7% and 68.7% for cerebrovascular disease, ischemic heart disease, left ventricular hypertrophy, chronic kidney disease and retinopathy respectively. [33]

Ayodele et al in Nigeria found prevalence of left ventricular hypertrophy by ECG 31.0%, chronic kidney disease 18.2%, heart failure 10.8% and cerebrovascular disease 8.9%. Advanced retinopathy was present in 7.4% of study sample.[30]

Prevalence of organ damage has been shown to be high even in a group of patients with new diagnosis of hypertension. In a study which included newly diagnosed hypertensive patients in Nigeria, it showed a high prevalence of TOD in this group of patients. Seventy-one per cent had between grade 1 and 2 hypertensive retinopathy. Almost 89.9% glomerular filtration rate less than 90 ml/min. [34]

Nasheeta Peer et al in a cross-sectional survey among black South Africans diagnosed and on treatment of hypertension found prevalence of renal impairment of 26%. The prevalence of LVH 35% by ECG diagnosed using Sokolow-Lyon criteria. [35]

In a study done at Kenyatta hospital ,LVH using ECG was detected in 33.3% [36], while in Democratic Republic of Congo, study to determine Clinical correlates of left ventricular hypertrophy in black patients with arterial hypertension revealed 48% had LVH using ECG [37].

It has been shown that, the prevalence of TOD is influenced by social demographic factors. Prevalence of TOD increases with age, more prevalent in a group with low education. Also the probability of presenting with of more than one TOD increases with age. TOD prevalence directly related to the presence of cardiovascular risk factors. [38] TOD has also been associated with poor control of blood pressure, with high prevalence among patients with poor control of blood pressure [35, 39, 40].

Essential hypertension reveals an association between metabolic syndrome and organ damage. Approximately 53% of patients with metabolic syndrome had two or three markers of TOD compared with 33% without metabolic syndrome.[41]

It has been shown that, in over weight and Obesity hypertensive patients Metabolic Syndrome maintains its role as risk factor for LVH independently of age, SBP, resulting in a useful predictor of target organ damage in clinical practice. [42]

1.2.0 PROBLEM STATEMENT

Hypertension is considered to be one of major public health problems in the Africa. It is of immense economic importance because of the high prevalence in both urban and semi urban areas [11]. High prevalence of hypertension has also been documented in Tanzania, both in rural and urban settings.[11]

Among hypertensive patients, the rate of treatment and hypertension control has been reported to be low [11, 12]. The reported poor control increases the possibility of developing complications of hypertension with potential damage to the important organs such as the heart, kidney, brain and arterial blood vessels.[12] Knowledge of extent of hypertensive target organ damage is important in raising awareness among health workers as well as in treatment, prevention and resource allocation. However data on the prevalence of organ damage in our setting is not available.

1.3.0 RATIONALE

This study was done to provide information on the extent of target organ damage at Muhimbili National Hospital. The data obtained from this study reflects the health impact of hypertension in our setting. Also it provides the basis for aggressive efforts at prevention, early detection and control of high blood pressure.

1.4.0 OBJECTIVES

1.4.1 Broad Objective

To determine the prevalence of target organ damage among hypertensive patients at Muhimbili National Hospital.

1.4.2 Specific Objectives

1. To determine the prevalence of left ventricular hypertrophy among study subjects.
2. To determine the prevalence of hypertensive retinopathy among study subjects.
3. To determine the prevalence of renal insufficiency among study subjects.
4. To determine the prevalence of Stroke among study subjects.

CHAPTER TWO

2.0 METHODOLOGY

2.1.0 Study Design

The study was hospital based descriptive cross sectional study, conducted from July 2011 to December 2011.

2.2.0 Study Site

The study was conducted at Muhimbili National Hospital, which is located in Dar Es Salaam city. Muhimbili National Hospital is Tanzania's largest tertiary level hospital. It receives referred patients from all over the country.

On average, daily attendance to medical outpatient clinics is about 100 patients with different medical diagnoses. Medical wards receive about 10- 25 new patients every day.

2.3.0 Study Population

Known Hypertensive patients

2.4.0 Inclusion Criteria

Adults aged 18 years and above with documented diagnosis of Hypertension admitted in medical wards or attending medical outpatient clinic.

2.5.0 Sample Size Estimation

Sample size was calculated from the following formula.

$$n = \frac{Z^2 p(1-p)}{d^2}$$

Whereas:

Z = statistic for a level of confidence = 1.96

P = prevalence of Target end organ (Average of prevalence of stroke in Ghana(2.8%) from study Juliet Addo et al and prevalence of stroke of 8.9% in Nigeria, a study done by Ayodele et al)= 6%.

d = precision = 3%

n = minimum sample size=240

2.6.0 Sampling technique:

Patients admitted in medical wards and those attending medical outpatient clinics on Monday, Tuesday and Friday were reviewed. All hypertensive patients were identified. All hypertensive patients who consented to participate in the study were recruited consecutively until the sample size was reached. To avoid double counting, documentation was done in the files of recruited patients.

2.7.0 Procedures

2.7.1 Blood Pressure Measurement

Two BP measurements were taken at 2 minutes intervals using automatic blood pressure machine (Omron) with appropriately sized cuff, after the participant had been seated for 2 minutes. The average of the two measurements was used in the analysis.

A hypertensive patient was considered to have uncontrolled blood pressure if he/she had elevated SBP $\geq$ 140 mmHg and/or DBP $\geq$ 90 mmHg on physical examination.

2.7.2 Weight and height measurements

Weight was measured using the Secca weighing scale, Calibrated every morning using a standard weight. Weighting was done without shoes and in light clothing. Reading recorded to the nearest 0.5 kilograms.

Height measurement was taken using a height measuring rod without shoes and recorded to the nearest 0.5 centimeters.

BMI for each respondent was calculated by dividing weight (kilograms) with height squared (meter). Obesity was defined as BMI of $\geq 30 kg/m^2$.

2.7.3 Echocardiogram

Echocardiographic examinations were done with the patient in partial left lateral decubitus position using PHILIPS 7550 HP ECHO machine. Two cardiologists performed the echocardiography.

Two-dimensional guided M-mode measurements were obtained. Left ventricular mass were calculated using three parameters (End-diastolic left ventricular internal diameter (LVIDd), Intraventricular septum thickness during diastole (IVSDd) and Left ventricular posterior wall thickness during diastole (PWTd).

Left ventricular Mass was calculated from above parameters using Devereux formula [43] .

$$LV\ mass\ (g) = 0.8\ (1.04\ (IVSd + LVIDd + PWTd)^3 - LVIDd^3) + 0.6$$

The calculated LVM was then divided to height $^{2.7}$ ($m^{2.7}$) to obtain Left ventricular mass index (LVMI). Indexing to height $^{2.7}$ has been shown to provide the most accurate estimate of LV mass [21].

2.7.4 Electrocardiogram

A standard 12- lead resting electrocardiogram (ECG) was performed to each patient at ECG room by investigator. Standard PHILIPS (PAGER) ECG machine was used, with ECG paper running at a speed of 25mm/sec and standard calibration of 10mm per 1mV.

The voltages from the ECG paper were measured by an investigator and then reviewed by a cardiologist for confirmation of presence or absence of LVH by Voltage criteria.

2.7.5 Retinal examination

All recruited patients were reviewed by ophthalmologists at Eye clinic/ medical ward for fundoscopy examination. Ophthalmologists were blinded on patient's blood pressure level. The retinal changes were then classified according to the Keith-Wagener-Baker scale based on the level of severity of the retinal findings.

2.7.6 Biochemical tests

Patients were given an appointment to come for other investigations. After an overnight fasting, blood samples for total serum cholesterol, high density lipoproteins (HDL) low density lipoprotein (LDL), serum triglycerides and blood glucose was taken. 10 mls of venous blood was taken from the antecubital fossa and placed in empty sterile tubes. The samples were processed at Central Pathological Laboratory (CPL) at the Muhimbili National Hospital (MNH) using an automated Abbott AxSymTM Chemistry analyzer system (Abbott USA).

2.7.7 Determination of renal insufficiency and damage

Estimated GFR was computed using creatinine clearance standardized to a body surface area of 1.73 m^2 from the current serum creatinine, according to the Modification of Diet for Renal Disease formula, with the correction factor for subjects of black African ancestry. [44]

$$GFR\ (mL/min/1.73\ m^2) = 175 \times (S_{cr})^{-1.154} \times (Age)^{-0.203} \times (0.742 \text{ if female}) \times (1.212 \text{ if black})$$

Where S_{cr} is patient's serum creatinine in mmol/l.

The calculations were made using online MDRD calculator found on (www.kidney.org/professionals/kdoqi/gfr_calculator.cfm).

The urine protein was assessed using CYBOW 10 urine reagent Strips manufactured by DFI co.ltd, South Korea (2008).

2.8.0 DEFINITION OF TERMS

Left ventricular hypertrophy on Echocardiogram was defined as a LVMI equal to or higher than 51 g/m $^{2.7}$ in men and 47 g/m $^{2.7}$ in women.[21]

On Electrocardiogram, Left ventricular hypertrophy was diagnosed on the basis of fulfillment of at least one of the following voltage criteria. Sokolow - Lyon criteria (S V1+ R V5/ V6 > 35 mm) or Cornell criteria SV3 + R avl > 28 mm in men SV3 + R avl > 20 mm in women.

Retinopathy was graded according to Keith-Wagener-Baker scale.

Grade I when there was 'mild' generalized retinal arteriolar narrowing.

Grade 2 more severe generalized narrowing, focal areas of arteriolar narrowing and arteriovenous nicking.

Grade 3 consisted of grades 1 and 2 signs plus the presence of retinal haemorrhage, microaneurysms, hard exudates and cotton-wool spots.

Grade 4 also referred to as accelerated (malignant) hypertensive retinopathy, with signs of the preceding three grades plus optic disk swelling and macular oedema.

The renal insufficiency was defined as eGFR ≤60 (mL/min/1.73 m^2) according to the Kidney Disease Outcomes Quality Initiative (K/DOQI) of the National Kidney Foundation (NKF).

Results of urine dipstick were interpreted as follows.

Strip reading	mg/dl
None	Negative
Trace	Negative
+1	30
+2	100
+3	300
+4	1000

Significant protenuria was considered when dipstick reading was ≥ +3.

CARDIOVASCULAR RISK FACTORS

Diabetes was defined as a venous fasting plasma glucose of ≥7.0 mmol/l or on diabetic medication and impaired fasting glucose (IFG) as fasting glucose levels equal or > 6.1 <7.0 mmol/l.

Triglycerides > 1.69 mmol/l was considered as high level.

Patient was considered to have hypercholesterolemia when serum cholesterol was > 5.18 mmol/l.

LDL>3.34mmol/l was considered as High level.

HDL >0.99 mmol/l was considered as normal level.

The short version of International physical activity (IPAQ) questionnaire of August 2002 which assesses the activities done during last 7 days was used.

The short version was designed for large population surveillance in which time and space are limited. [45]

In this study, independent self report of activities that the individual done over the past 7 days was identified and recorded according to international physical activity questionnaire. Participant was considered to have Low physical activities if he/she didn't meets criteria of moderate or high physical activity (at least 30 minutes of moderate physical activities in at least 5 days a week or 3-5 days a week of vigorous physical activity).

The CAGE set of four questions was used to assess excessive alcohol intake. Participants were asked four questions (Have you ever felt that you should cut down on your drinking? Have people annoyed you by criticizing your drinking? Have you ever felt bad or guilty about your drinking? Have you ever had a drink first thing in the morning to steady your nerves or get rid of a hangover?). Conclusion for excessive alcohol intake was when $\geq$ 2 questions were answered in the affirmative. [46]

2.9.0 DATA ENTRY AND ANALYSIS

All filled questionnaires were first coded. Data entry was done using Epi Info version 3.5.1 statistical package. Data cleaning was done by consistence checks. Statistical analysis was performed using Epi Info version 3.5.1 statistical package. Quantitative variables were summarized by their mean, standard deviation (SD). Frequency distribution and two way tables were used to summarize the qualitative data. Student's t-test was used to determine association between means, Chi-square (χ^2) and Fisher's exact tests were used to determine the

association between independent and dependent categorical variables. A significance level was set at 5%.

2.10.0 ETHICAL ISSUES

Prior to data collection, ethical clearance was sought from the MUHAS ethical committee.

The study involved adults, aged 18 years or above. Patient was asked for consent to participate in the study. All information was kept confidential.

All patients regardless of their willingness to participate in the study received all routine services as any patient in a follow up clinic/admitted in medical ward. Investigation's results were communicated to the patient. When in need of further review and management, appropriate measures were taken.

CHAPTER THREE

3.0.0 RESULTS

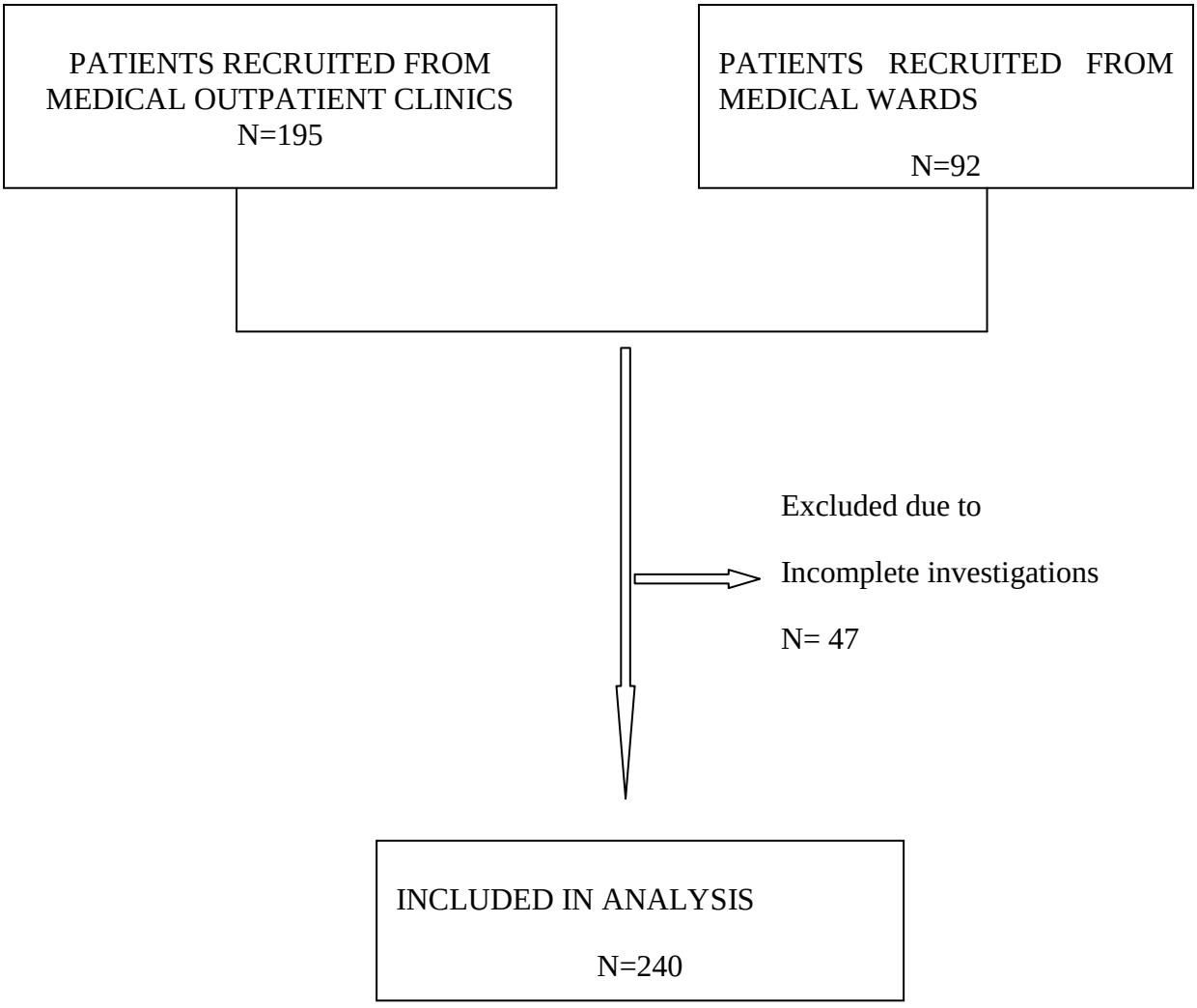


Figure 1: Recruitment procedure

8.1 Demographics and baseline characteristics of study population

The study recruited 240 known hypertensive patients from medical wards and medical outpatient clinics who were consecutively enrolled into the study. Of those recruited 102 (42.5%) were male and 138 (57.5%) were female. The mean age of study population was 53 years standard deviation (±13). Those with primary education were the largest group 87 (36.3%), 40 (16.7%) had no formal education and only 10 (4.2%) had university education. Mean systolic blood pressure was 153 mmHg, standard deviation (±23) and mean diastolic pressure was 91 mmHg, standard deviation (±13). One hundred and forty four patients (60%) were found to have low physical activity, while obesity was noted in 45(18.8%). A higher proportion of females were obese 30 (21.7%) compared to males 15 (14.7%). Two (0.8%) participants were smokers and all were male. None of the participants used excessive alcohol. (Table 1)

TABLE 1: DEMOGRAPHICS AND BASELINE CHARACTERISTICS OF STUDY POPULATION

CHARACTERISTIC	MALE	FEMALE	TOTAL	*P-value*
	N=102 (%)	N=138 (%)	N=240 (%)	
Age (years)				
<40	17 (16.7)	20 (14.5)	37 (15.4)	0.861
40-65	67 (65.7)	95 (68.8)	162 (67.5)	
>65	18 (17.6)	23 (16.7)	41 (17.1)	
Education				
No Formal	17 (16.7)	23 (16.7)	40 (16.7)	0.250
Primary	29 (28.4)	58 (42.0)	87 (36.3)	
Secondary	24 (23.5)	26 (18.8)	50 (20.8)	
Vocational	27 (26.5)	26 (18.8)	53 (22.1)	
University	5 (4.9)	5 (3.6)	10 (4.2)	
Cardiovascular risk factors				
Smoking	2 (2.0)	0	2 (0.8)	
Diabetes	11 (10.8)	19 (13.8)	30 (12.5)	0.490
Obesity	15 (14.7)	30 (21.7)	45 (18.8)	0.168
Low physical activity	66 (64.7)	78 (56.5)	144 (60.0)	0.201
Lipid profile	**N=80(%)**	**N=106 (%)**	**N=186 (%)**	
High Triglycerides	26 (32.5)	36 (34.0)	62 (33.3)	0.834
High LDL	38 (47.5)	61 (57.5)	99 (53.2)	0.174
Low HDL	39 (48.8)	54 (50.9)	93 (50.0)	0.767
High total Cholesterol	21 (26.3)	42 (39.6)	63 (33.9)	0.060

8.2 Prevalence of target organ damage

Of the 240 hypertensive patients recruited in this study, 206 (85.8%) patients had at least one of the four hypertensive target organ damage studied (left ventricular hypertrophy by echocardiogram, hypertensive retinopathy, renal insufficiency and stroke). Eighty three patients (34.6%) had two organ damage while 77 (32.1%) had one target organ damage and 4 (1.3%) had all four target organ damage. The organ most affected was the heart presenting as left ventricular hypertrophy diagnosed by Echocardiogram that was observed in 167 (69.6%) followed by hypertensive retinopathy 136 (56.7%). Stroke was observed in 22 (9.2%) patients. Male had higher prevalence of TOD than female.

Patients with uncontrolled blood pressure had had higher prevalence of all four target organ damage compared to those with controlled blood pressure. The *P* value was < 0.05 for LVH, retinopathy and renal insufficiency. (Table 2, Figure 2 and 3)

TABLE 2: PRESENCE OF TARGET ORGAN DAMAGE AMONG STUDY PARTICIPANTS

Number of Target Organ damage	Frequency N=240	%
0	34	14.2
1	77	32.1
2	83	34.6
3	43	17.9
4	3	1.3

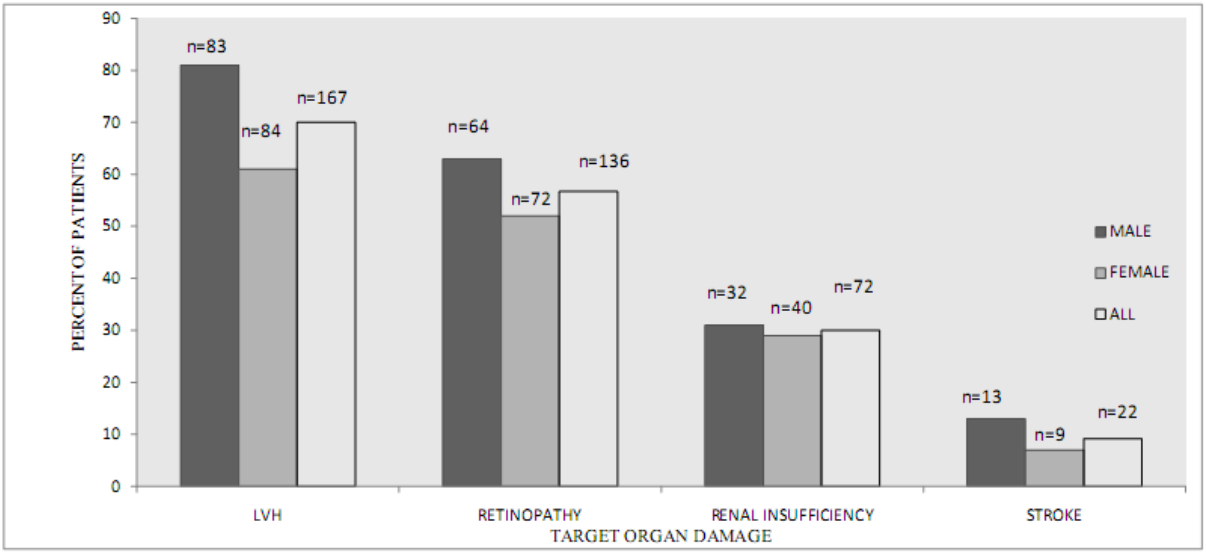


FIGURE 2: COMPARISON OF PRESENCE OF TARGET ORGAN DAMAGE BY SEX

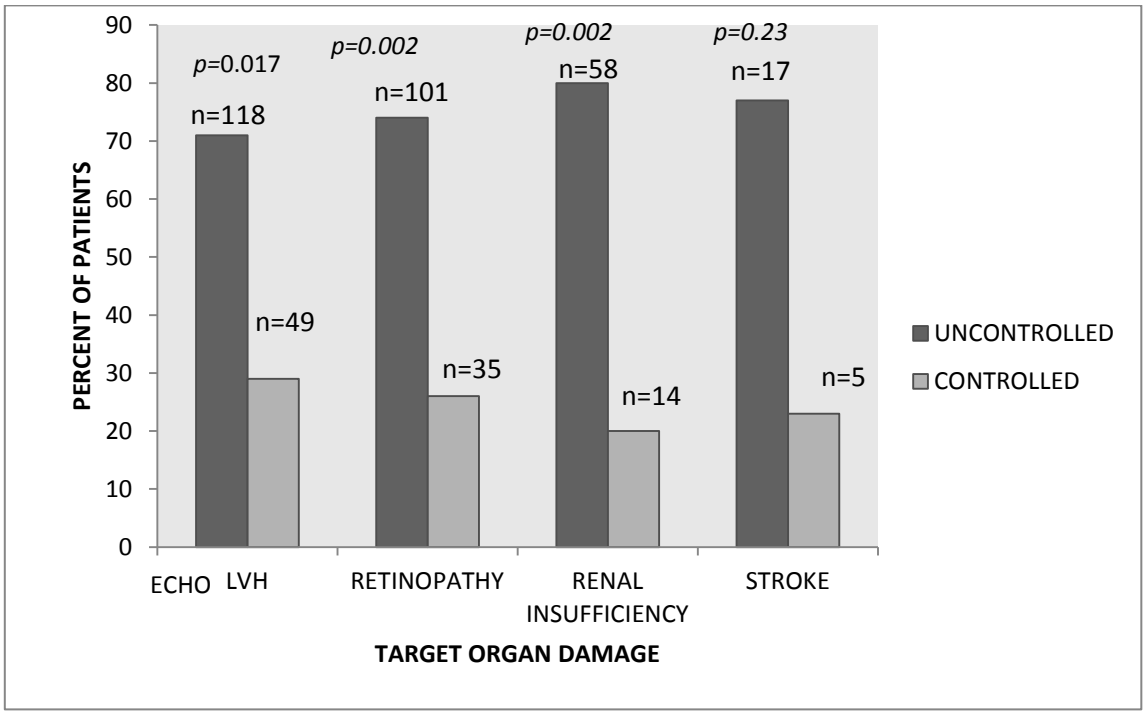


FIGURE 3: COMPARISON OF PREVALENCE OF TARGET ORGAN DAMAGE WITH BLOOD PRESSURE STATUS

8.3 Prevalence of left ventricular hypertrophy

Among 240 patients studied, prevalence of left ventricular hypertrophy that was diagnosed by echocardiogram based on left ventricular mass index was 167 (69.6%). Males had a higher prevalence of left ventricular hypertrophy compared to females 83 (81.4%) vs. 84 (60.9%). p=0.0006. On electrocardiogram, 114 (47.5%) of all subjects had left ventricular hypertrophy. On ECG, a higher proportion of male 61 (59.8%) presented with LVH compared with female. 53(38.4%) p=0.001 (Table 3)

TABLE 3: ECHOCARDIOGRAPHIC AND ELECTROCARDIOGRAPHIC PREVALENCE OF LEFT VENTRICULAR HYPERTROPHY BY SEX

LEFT VENTRICULAR HYPERTROPHY	MALE n=102(%)	FEMALE n=138(%)	TOTAL N=240(%)	*P-value*
By Echocardiogram				
LVH	83(81.4)	84(60.9)	167(69.6)	0.0006
NO LVH	19(18.6)	54(39.1)	73(30.4)	
By electrocardiogram				
LVH	61(59.8)	53(38.4)	114(47.5)	0.001
NO LVH	41(40.2)	85(61.6)	126(52.5)	

8.4 Prevalence of hypertensive retinopathy

Hypertensive retinopathy was present in 136 (56.7%) of the study population. According to Keith-Wagener-Baker classification, which is based on the level of severity of the retinal findings, the majority presented with grade 1 and 2 retinopathy, 38 (15.8%) and 71 (29.6%) respectively. Advanced retinopathy (grade 3 and 4) was present in 27 (11.3%) participants. (Table 4)

TABLE 4: PREVALENCE OF HYPERTENSIVE RETINOPATHY ACCORDING TO KEITH-WAGENER AND BAKER CLASSIFICATION

Hypertensive retinopathy Grade	Frequency n=240	%
Absent	104	43.3
1	38	15.8
2	71	29.6
3	22	9.2
4	5	2.1

8.4 Prevalence of renal insufficiency

Of the 240 study participants, 168 (70%) patients had eGFR of more than 60 mL/min/1.73 m^2. Renal insufficiency defined as estimated glomerular filtration rate less than 60 mL/min/1.73 m^2 was present in 72 (30%) patients. Based on K/DOQI classification, End stage renal disease defined as GFR less than 15 mL/min/1.73 m^2 was noted in 15 (6.2%) of the study participants. (Table 5)

Thirty percent of all participants had protenuria. The presence of protenuria was observed more in a group with renal insufficiency with statistical significant difference (P value < 0.001). Table 6

TABLE 5: PREVALENCE OF RENAL INSUFFICIENCY STAGES ACCORDING TO K/DOQI

eGFR(mL/min/1.73 m^2)	Frequency n=240	%
≥60	168	70.0
30-59	36	15.0
15-29	21	8.8
<15	15	6.2

TABLE 6: RELATIONSHIP BETWEEN PROTEINURIA AND GFR

Urine albumin	GFR<60 (mL/min/1.73 m^2) n=72	GFR≥60 (mL/min/1.73 m^2) n=168	Total N=240	*P value*
Absent	31 (43.1)	137 (81.5)	168 (70.0)	< 0.001
Present	41(56.9)	31(18.5)	72 (30.0)	

CHAPTER FOUR

4.0.0 DISCUSSION

This study has revealed a high prevalence of target organ damage among hypertensive patients attending Muhimbili National Hospital.

Eighty six percent of all participants had at least one target organ damaged. This means majority of hypertensive patients attending medical clinics or admitted in medical wards presents with hypertensive complications which may results into high morbidity and mortality among hypertensive patients. These complications arise with poor control of blood pressure.

This finding of high prevalence can be contrasted to prevalence of target organ damage in hypertensive patients attending medical outpatient department of a state hospital in Nigeria [30] and among hypertensive civil workers in Ghana, [31] where 60.1% and 47.5% respectively were reported to have at least one organ involved. All three studies showed a high prevalence of the target organ damage among patients with hypertension. Use of ECG which could under diagnose LVH in other two studies as well as studying only out patients may contribute to the difference in prevalence with our study. Also we speculate that, late presentation to health care facilities could contribute to the high prevalence of TOD as many patients present when they already have hypertensive complications.

Left ventricular hypertrophy detected by ECHO was the commonest target organ damage encountered. The percentage of Left Ventricular Hypertrophy by ECHO in this study was 69.6%. Data on prognostic implication of Left ventricular hypertrophy in the Framingham study indicated that LVH has emerged as a powerful indicator of rapidly evolving lethal atherosclerotic disease whether determined by ECG or ECHO.[15]

Presence of LVH has important clinical implications in hypertensive patients as it is associated with increase in the incidence of heart failure, ventricular arrhythmias, death following

myocardial infarction, decreased left ventricular ejection fraction, sudden cardiac death, and a cerebrovascular event. The increase in cardiovascular risk has been directly related to the degree of increase in left ventricular mass. [16]

The prevalence of LVH observed in this study was higher when compared to the findings of 57.3% and 57.1% by Ihab B Abdalrahman et al in Sudan [33] and Armario in Spain [28] respectively. In these two studies, Left ventricular mass was indexed to body surface area which has been shown to miss mild LVH, while in our study indexing to height$^{2.7}$ was used. While the difference could be due to criteria used in LVH diagnosis all studies documented high prevalence of ECHO-LVH among hypertensive patients.

Using ECG criteria, Left ventricular hypertrophy according to Sokolow-Lyon and Cornel criteria was found in 47.5% of study participants. This is comparable to the prevalence of 48% in Democratic Republic of Congo [37] but higher compared to the findings of 27.5% , 31.0%, 35% and 33.3% in Kenyatta Hospital[36], Nigeria [30], Ghana [31] and South Africa[35] respectively. With the fact that ECG has high specificity in detecting LVH and tends to detect more severe forms of Left Ventricular hypertrophy, this high prevalence means majority of participants could be at high risk for cardiovascular disease.

In this study, prevalence of hypertensive retinopathy was 56.7%. Most of the patients presented with grade 1 and 2 retinopathy according to Keith-Wegener and Barker classification. Other studies in black population have documented the comparable high prevalence. In Khartoum and Elshaab hospitals in Sudan [33] prevalence of 67% was reported, and Ayodele et al reported prevalence of 70% in Nigeria [30] The high prevalence may be due to racial factor. A higher prevalence of retinopathy has been reported more frequently in black than caucasian patients. The high prevalence is explained in large part by the higher levels of hypertension among blacks.[24] Grade 3 and 4 hypertensive retinopathy was observed in 9.2% and 2.1% of all participants respectively. The comparable prevalence of advanced retinopathy of 7.4% was reported by Ayodele in Nigeria[30]. Advanced retinopathy is usually seen in patients with severe forms of blood pressure. Its occurance in our setting implies that

significant proportion of our hypertensive patients presents with severe forms which has to be taken care to prevent events associated with severe hypertension like hypertensive encephalopathy and acute renal failure.

Renal insufficiency was found in 30% of study population. This prevalence is comparable to the 26% by Nasheeta in South Africa black population[35] and the recent findings of 36.7% in Sudan by Abdalrahman et al [33] . But it is higher when compared to a study by Addo 4.1% in Ghana[31] and Ayodele 18.2% in Nigeria[30]. The study population in our study differs from these two other studies in that Addo studied stable civil workers who are hypertensive and Ayodele studied only outpatient hypertensive subjects.

Cea-Calvo in Spain reported a higher prevalence of renal insufficiency 45.1% compared to our study. But the study population was > (55) years old who are both hypertensive and diabetic. This may explain the higher prevalence compared to our findings.

The higher prevalence of CKD stage 4 (8.8%) and stage 5 (6.2%) in this study has also been reported elsewhere. Salako[34] in a study of newly diagnosed hypertensive patients, found 11.85% patients in stage 4. This shows the burden of renal insufficiency among hypertensive patients in our setting.

Stroke was detected in 9.2% of the study population. While this figure is comparable to 6.6% and 8.9% reported in Kassala Sudan and by Ayodele in Nigeria 8.9%[30, 32] , it was higher than 2.8% reported by Addo in Ghana[31]. The difference in stroke prevalence in our study compared to that by Addo in Ghana can be explained by fact that Addo studied healthy civil servants.

In this study, target organ damage was more prevalent among patients who had uncontrolled blood pressure measured on the day of study, with statistical significant difference in prevalence of LVH, retinopathy and renal insufficiency. Although the design of this study cannot allow assessing blood pressure control, it has been shown in other studies by Nasheta Peer et al [35] in South Africa and Shirfkan et al in Iran[39], that patients with uncontrolled blood pressure have high prevalence of TOD compared to those with controlled blood pressure.

These studies concluded that, tight control of blood pressure will reduce the prevalence of target organ damage among hypertensive patients.

In conclusion, this study found high prevalence of markers of organ damage among hypertensive patients. Early detection of Hypertension and strict Blood pressure control will help to reduce TOD in the hypertensive population.

4.1.0 STUDY LIMITATIONS

1. This study was done at a tertiary level hospital; hence included a selected group of patients may be with advanced stage of hypertension. The results of this study may not be generalized to the community.
2. The cross section nature of the study could not allow assessing control of hypertension among study subjects so it was difficult to relate blood pressure control with presence of target organ.
3. Due to limited funds, diagnosis of stroke was clinical.
4. In this study, we did not separate inpatients from outpatients. So it was not possible to compare these two populations.
5. In this study, we did not collect information on heart failure or other clinical heart diseases. So we are not able discuss clinical heart disease beyond LVH

CHAPTER FIVE

5.0.0 CONCLUSION

This study showed a high prevalence of target organ damage among hypertensive patients admitted in medical wards or attending medical outpatient clinic at Muhimbili National Hospital. Left ventricular hypertrophy by Echocardiogram was the most prevalent target organ damage followed by hypertensive retinopathy, renal insufficiency and stroke in that order.

5.1.0 RECOMMENDATIONS

1. With high prevalence of markers of hypertension organ damage, early workup to identify presence or absence of organ damage is recommended in all hypertensive patients.
2. Strict blood pressure control is recommended to prevent organ damage. Treatment with all antihypertensive medications that results in significant BP reductions also reduces fatal and nonfatal hypertensive complications and slows down the progression to organ failure. Uses of drugs that antagonize Renin-Angiotensin-Aldosterone system (ACE inhibitors and ARB's) have provided compelling proof of organ protection.
3. Further studies to investigate treatment adherence, control, quality of hypertensive care and other factors associated with high prevalence of Target organ damage are recommended.

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APPENDICES

QUESTIONNAIRE.

Questionnaire No. …… Patient Contact…….

A. SOCIODEMOGRAPHIC CHARACTERISTICS

1. Name ……2. Hospital reg. ……..

3. Age ……..

4. Sex a) Male b) Female

5. Level of Education

a) Primary Education b.) Secondary Education c) Vocational training

d)University e) No Formal education

6. Marital Status

a) Single b) Married/cohabiting c) Divorced/Separated d) Widow/widower

7. Are you Hypertensive? a) Yes b.) No

8. Duration since diagnosis of hypertension ……. (months)
9. Are you using anthypertensives? a)Yes b)No
10. Duration on antihypertensives …….. months
11. Use of anthypertensives a) Regular b) irregular

B. CARDIOVASCULAR RISK FACTORS

1. Are you Diabetic? a) Yes b) No
2. If Diabetic, Duration since diagnosis …….. (months)
3. Are you on treatment for Diabetes a) Yes b) No
4. If yes, a) Diet b) Hypoglycemic drugs c) Insulin d) others (specify)……..
5. Treatment a) Regular b) Irregular
6. Have you ever smoked cigarettes? a)Yes b) No **(Go to 11)**
7. A re you current cigarette smoker? a) Yes b)No
8. If yes for how long …… months
9. How many cigarettes per day …….
10. If stopped, When did you stop smoking ……Months ago
11. Do you take alcohol a)Yes b) No

    If yes

12. Have you ever felt that you should cut down on your drinking? a)Yes b) No
13. Have people annoyed you by criticizing your drinking? a) Yes b) No
14. Have you ever felt bad or guilty about your drinking? a) Yes b) No
15. Have you ever had a drink first thing in the morning to steady your nerves or get rid of a hangover? a)Yes b) No
16. Conclusion: excessive alcohol use a)Yes b)No

PHYSICAL ACTIVITY

INTERNATIONAL PHYSICAL ACTIVITY QUESTIONNAIRE

The questions will ask you about the time you spent being physically active in the **last 7 days**. Please answer each question even if you do not consider yourself to be an active person. Please think about the activities you do at work, as part of your house and yard work, to get from place to place, and in your spare time for recreation, exercise or sport.

Think about all the **vigorous** activities that you did in the **last 7 days**. **Vigorous** physical activities refer to activities that take hard physical effort and make you breathe much harder than normal. Think *only* about those physical activities that you did for at least 10 minutes at a time.

1. During the **last 7 days**, on how many days did you do **vigorous** physical activities like heavy lifting, digging, aerobics, or fast bicycling? _____ **Days per week**

☐ No vigorous physical activities → ***Skip to question 3***

2. How much time did you usually spend doing **vigorous** physical activities on one of those days? _____ **hours per day** _____ **minutes per day**

☐ Don't know/Not sure

Think about all the **moderate** activities that you did in the **last 7 days**. **Moderate** activities refer to activities that take moderate physical effort and make you breathe somewhat harder

than normal. Think only about those physical activities that you did for at least 10 minutes at a time.

3. During the **last 7 days**, on how many days did you do **moderate** physical activities like carrying light loads, bicycling at a regular pace, or doubles tennis? Do not include walking.

_____ **Days per week**

☐ No moderate physical activities → ***Skip to question 5***

4. How much time did you usually spend doing **moderate** physical activities on one of those days? _____ **hours per day** _____ **minutes per day**

☐ Don't know/Not sure

Think about the time you spent **walking** in the **last 7 days**. This includes at work and at home, walking to travel from place to place, and any other walking that you might do solely for recreation, sport, exercise, or leisure.

5. During the **last 7 days**, on how many days did you **walk** for at least 10 minutes at a time?

_____ **Days per week**

☐ No walking → ***Skip to question 7***

6. How much time did you usually spend **walking** on one of those days?

_____ **Hours per day** _____ **minutes per day**

☐ Don't know/Not sure

The last question is about the time you spent **sitting** on weekdays during the **last 7 days**. Include time spent at work, at home, while doing course work and during leisure time. This may include time spent sitting at a desk, visiting friends, reading, or sitting or lying down to watch television.

7. During the **last 7 days**, how much time did you spend **sitting** on a **week day**?

_____ **Hours per day** ____**minutes per day**

☐ Don't know/Not sure

CONCLUSION: Level of physical activity……

C. PHYSICAL EXAMINATION

1. EYE EXAMINATION

Corneal Arcus a) Present b) absent **xanthelasma** a) Present b) absent

2. ARTHROPOMETRY

Height ……. Cm Weight ……. BMI ……. Kg/m^2

3. CARDIOVASCULAR EXAMINATION

BP 1st reading ……. BP 2nd reading …….. Average BP …… mmHg

Apex beat

Location ……. Character: Heave a) present b) absent

Left parastenal heave ……. a) present b) Absent

4. CENTRAL NERVOUS SYSTEM EXAMINATION

FUNDOSCOPY FOCAL NEUROLOGICAL DEFICIT

Retinopathy

a)present b) absent a)present b)absent

GRADE

D. LABORATORY TESTS

1. Fasting Serum triglyceride.....mol/L **2**. Fasting LDL..... mmol/L
3. Fasting HDL..... mmol/dL **4**. Fasting Total Cholesterol..... mmol/dl
5. Serum createnine ..... mmol/L **6**. eGFR (using cockroft and Gaut) .....
7. Urine Albumin
1. Present a)+1 b) +2 c) +3/+4
2. Absent

E. ECHOCARDIOGRAM

1. Mass Index .....

   Conclusion LVH:

   a)Present b) Absent

F.ELECTROCARDIOGRAM

1. Sokolow-lyon criteria (sum of voltage) .....
2. Cornell criteria (sum of voltage) .....
3. Conclusion LVH

   a) Present b) Absent

DODOSO LA KISWAHILI

Namba ya dodoso …… simu ya mgonjwa ……..

A.TAARIFA ZA MGONJWA

1. Jina …….2. Namba ya usajili . 3. Umri……

Jinsia a) Mme b) Mke

4. Elimu

a.Elimu ya msingi b) Elimu ya sekondari c)Elimu ya ufundi

d) Chuo kikuu e) Hajaenda shule

5. Hali ya ndoa

a)Hajaoa/hajaolewa b) Ameoa/wanaishi pamoja c)Ameachika/ametengana d) Mjane

6. Una shinikizo la damu? a) Ndio b). Hapana
7. Muda tangu shinikizo la damu lijulikane ……..(miezi)
8. Unatumia dawa za shinikizo la damu? a) Ndio b). Hapana
9. Muda tangu uanze kutumia dawa za shinikizo la damu…….. (miezi)

10. Matumizi ya dawa a) muda wote b) Si muda wote

B.VIASHIRIA HATARI VYA MAGONJWA YA MISHIPA YA DAMU NA MOYO

1. Una kisukari? a) Ndio b). Hapana
2. Kama una kisukari , muda tangu kisukari kijulikane ……. miezi)
3. Unatibiwa kisukari a) Ndio b) Hapana
4. Kama ndio a) Masharti ya chakula b). Vidonge c) Insulini d) Nyingine ……
5. Matibabu ya kisukari a). Muda wote b). Si muda wote
6. Umewahi kuvuta sigara? a) Ndio b). Hapana
7. Kwa sasa unavuta sigara? a) Ndio b). Hapana
8. Kama ndio, kwa muda gani? …….. 10. Sigara ngapi kwa siku ……
9. Kama umeacha, kwanini ?……
10. Unakunywa pombe? a) Ndio b). Hapana

Kama ndio

11. Umewahi kufikiria kupunguza unywaji wako? a) Ndio b) Hapana
12. Watu wanachukizwa na kupinga tabia yako ya unywaji wa pombe? a) Ndio b) Hapana
13. Umewahi kujisikia vibaya au kujihukumu kutokana na unywaji wako? a) Ndio b)Hapana
14. Umewahi kunywa pombe asubuhi kama kitu chako cha kwanza kufanya ili ujisikie vizuri au uondokane na mninginio? a) Ndio b) Hapana
15. Hitimisho: Unywaji kupita kiasi a) Ndio b). Hapana

DODOSO LA KIMATAIFA LA MAZOEZI YA MWILI

Katika maswali haya, utaulizwa kuhusu muda unaotumia kushughulisha mwili katika siku saba zilizopita.Tafadhali,jibu kila swali hata kama unajichukulia kua si mtu unayeushughulisha mwili wako.Fikiria shughuli unazofanya kazini, nyumbani na shambani, kutoka sehemu moja kwenda nyingine, na unavuotumia muda wako wa ziada kwa mapumziko, mazoezi au michezo.

Fikiria kuhusu shughuli nzito sana ulizofanya katika siku 7 zilizopita. shughuli nzito sana zinamaanisha shughuli inayotumia nguvu nyingi na inayokufanya uheme kwa nguvu kuliko kawaida. Fikiria tu shughuli zote ulizofanya kwa angalau dakika 10 kila wakati.

1. Katika **siku 7 zilizopita**, siku ngapi ulifanya shughuli nzito sana kama kunyanyua vitu vizito, kulima, kukimbia au kuendesha baiskeli kwa haraka?

_____ **siku kwa wiki**

☐ Hakuna shughuli nzito sana → ***Nenda swali la 3***

2. Muda gani huwa kwa kawaida ulikua unatumia katika kufanya shughuli nzito sana katika siku hizo?

_____ **Masaa kwa wiki**

_____ **Dakika kwa wiki**

☐ Sijui/sina hakika

Fikiria shughuli za wastani ulizofanya katika **siku 7 zilizopita.** shughuli za wastani ni zile zinazotumia nguvu za wastani na zinakufanya uheme angalau kwa nguvu kidogo kuliko kawaida.Fikiria tu shughuli zote za mwili ulizozifanya angalau kwa dakika 10 kila wakati.

3. Katika **siku 7 zilizopita**, siku ngapi ulifanya shughuli ya wastani kama kubeba mizigo myepesi, kuendesha baiskeli katika mwendo wa kawaida, au kucheza tenesi? Kutebea hakuhusiki.

_____ siku kwa wiki

☐ Hakuna mazoezi ya wastani ***Nenda swali la 5***

4. Muda gani kwa kawaida ulikua unatumia kufanya shughuli za wastani? Katika moja ya siku hizo?

_____ Masaa kwa siku

_____ dakika kwa siku

☐ Sijui/sina hakika

Fikiria muda uliotumia kutembea katika **siku 7 zilizopita**. Hii ina jumuisha kazini na nyumbani, kutembea ukisafiri kutoka sehemu moja kwenda nyingine au kutembea kwa makusudi ya kujifurahisha, michezo, mazoezi au burudani.

5. Katika **siku 7 zilizopita**, siku ngapi ulitembea kwa angalau dakika 10 kwa siku?

_____ **siku kwa wiki**

☐ sikutembea → ***Ruka swali hili***

5. Muda gani kwa kawaida huwa unautumia kutembea katika siku hizo?

_____ **masaa kwa siku**

_____ **dakika kwa siku**

☐ Sijui/sina hakika

Swali la mwisho ni kuhusu muda uliotumia kukaa katika siku za katikati ya wiki katika **siku 7 zilizopita**. Jumuisha muda uliotumia kazini, nyumbani, wakati ukijisomea, na wakati wa burudani. Hii inajumuisha muda uliotumia kukaa kwenye meza, kutembelea rafiki, kusoma au kukaa/ kulala ukiangalia television.

6. Katika **siku 7 zilizopita**, muda gani ulitumia kukaa katika siku za katikati ya wiki.

_____ masaa kwasiku

_____ dakika kwa siku

CONSENT FORM- ENGLISH VERSION

TITLE; PREVALENCE OF TARGET ORGAN DAMAGE AMONG HYPERTENSIVE PATIENTS AT MUHIMBILI NATIONAL HOSPITAL

Greetings! My name is Dr. Tuzo Lyuu, a resident in the department of Internal Medicine. I would like to conduct a study mentioned above as necessary requirement for fulfillment of my postgraduate studies.

Purpose of the study

The aim of the study is to determine prevalence of target organ damage among hypertensive patients at Muhimbili National Hospital. This will help in improving management of hypertensive patients.

How to participate

Patients who meet the inclusion criteria will be recruited into the study.

They will be interviewed using a questionnaire, which will include their social demographic characteristics, medical history, and clinical examination and undergo investigations including urine dipstick, CCP, ECG, ECHO and Lipid profile.

Risks

There is a mild pain and a risk of thrombophlebitis during venopuncture.

Benefits: Patients found to have any problem will be treated and followed up appropriately through our medical wards/Clinics.

Confidentiality:

Any patient's results will not be revealed to anybody except attending doctors and patient himself.

Cost

No payment is requested from you as a fee to participate in the study.

Person to contact in questions or problems:

Dr. Tuzo Lyuu (0784 4839940/0712035620) - Investigator

Dr Patricia Munseri (0754562784) - Lecturer, department of internal Medicine (MUHAS)

I…… have read/been told of the contents of this form and understood its meaning. I agree to participate in this study.

Signature…….. (Participant) Date……

Signature…….. (Researcher) Date……

CONSENT FORM SWAHILI VERSION.

Fomu ya ridhaa ya kushiriki katika utafiti.

UTAFITI KUHUSU IDADI YA WAGONJWA KATIKA HOSPITALI YA TAIFA YA MUHIMBILI AMBAO OGANI ZAO ZIMEHARIBIWA NA UGONJWA WA SHINIKIZO LA DAMU.

Salaam! Jina langu ni Dk. Tuzo Lyuu, mwanafunzi wa shahada uzamili ya Tiba katika idara ya Internal Medicine. Ninafanya utafiti kuhusu idadi ya wagonjwa ambao ogani zao zimeharibiwa na ugonjwa wa shinikizo la damu, kama sehemu ya kukamilisha masomo yangu ya uzamili

Lengo la utafiti.

Lengo la utafiti huu ni kujua idadi ya wagonjwa wenye shinikizo la damu katika Hospitali ya Taifa ya Muhimbili ambao ogani zao zimeharibiwa na ugonjwa wa shinikizo la damu . Hii itasaidia kuboresha matibabu ya wagonjwa wa shinikizo la damu.

Jinsi ya kushiriki.

Wagonjwa wenye sifa za ushiriki, wataingizwa katika utafiti. Taarifa kuhusu umri, elimu, kazi, historia ya ugonjwa, zitakusanywa kwa kutumia dodoso. Pia vipimo kama cha mkojo,CCP,ECG,ECHO na kuangalia mafuta kwenye damu vitafanyika.

Hatari

Maumivu kidogo yanaweza kutokea wakati wakutoa damu kwa ajili ya vipimo.

Faida:

Endapo mgonjwa atakutwa na tatizo, atatibiwa na kufuatiliwa kimatibabu ipasavyo.

Usiri:

Majibu yote ya vipimo vya mgonjwa hayataoneshwa kwa yeyote isipokua madaktari na mgonjwa

Gharama

Hautatakiwa kulipa chochote ili ushiriki katika utafiti huu

Mtu wa kuwasiliana nae utakapohitaji ,maelezo zaidi au ukipata tatizo.

Dr. Tuzo Lyuu (0784 4839940/0712035620) – Mtafiti.

Dr Patricia Munseri (0754562784) - Mhadhiri, idara ya internal Medicine (MUHAS).

Mimi…… Nimesoma/ nimesomewa yaliyomo katika fomu hii na kuelewa maana yake. Ninakubali kushiriki katika utafiti huu.

Sahihi…….. (mshiriki) Tarehe……

Sahihi…….. (Mtafiti) Tarehe……