HYPERURICEMIA AMONG HYPERTENSIVE PATIENTS ATTENDING CARDIAC CLINICS IN DAR ES SALAAM

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A DISSERTATION SUBMITTED IN (PARTIAL) FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF MASTER OF MEDICINE (INTERNAL MEDICINE) AT KAIRUKI UNIVERSITY

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CERTIFICATION

The undersigned certifies that he has read and hereby recommends for submission a Dissertation entitled ***''HYPERURICEMIA AMONG HYPERTENSIVE PATIENTS ATTENDING CARDIAC CLINICS IN DAR ES SALAAM''*** in fulfillment of the degree of Master of Medicine in Internal Medicine of Kairuki University.

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DECLARATION AND COPYRIGHT

I, **Cellina Joseph Kaupunda**, hereby declare that this dissertation is my original work and has not been presented and will not be presented to any other university for similar or any other degree award.

Signature

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Date

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DEDICATION

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ABSTRACT

Background: Hypertension, characterized by systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg, is a significant global health concern. In sub-Saharan Africa, it poses a growing public health challenge. Elevated uric acid levels have been associated with an increased risk of hypertension. Hyperuricemia, defined as abnormally high serum uric acid (UA) levels, predominantly exists as urate ions. The thresholds for hyperuricemia are >360 µmol/L for females, >420 µmol/L for males, and 5.5 mg/dL for youth (<18 years old). However, in Tanzania, specifically in Dar es Salaam, the prevalence and impact of hyperuricemia among p hypertensive patients remain unreported. Early detection and appropriate intervention could serve as effective preventive measures against hyperuricemia.

Objective: This study aims to assess the distribution of hyperuricemia among hypertensive patients attending cardiac clinics in Dar es Salaam**.**

Methodology: A descriptive cross-sectional study was conducted among hypertensive patients attending cardiac clinics in Dar es Salaam. Structured questionnaires were used to record demographic data. Blood pressure and anthropometric measurements were taken using standard methods. Blood samples were collected for serum uric acid test.

Results: In this study of 381 hypertensive patients from three regional referral hospitals, the majority were female (68.0%), married or cohabiting (60.4%), with no formal or primary education (70.9%), and from a lower income bracket (56.7%). The median age was 63 years, with a range of 28 to 88 years. The median duration since hypertension diagnosis was 3 years, with a BMI median of 26.1. Notably, 40.4% were overweight, and 29.7% were obese. Hyperuricemia was found in

42.5% of participants, with a median value of 335.4. A higher occurrence of elevated uric acid levels was observed in individuals over 45 years of age, nearly doubling from 30% to 49.7% in those aged 65 and above. Males exhibited a higher proportion of normal uric acid levels (68.9%) compared to females (52.1%). Additionally, a slight increase in normal uric acid levels was noted among the middle-income group versus the low-income group, and a significant association was found between alcohol consumption and higher uric acid levels. Individuals with Grade 1 hypertension had a higher incidence of elevated uric acid status compared to those with normal or high-normal blood pressure. The multivariable logistic regression has shown that being 65 or older, female, and consuming alcohol are all significant predictors of the hyperuricemia, with each group showing a higher adjusted odd ratio (AOR).

Conclusion: The study highlights a significant correlation between hyperuricemia and factors such as age, gender, and alcohol consumption, family history of hypertension among hypertensive patients. Females, older individuals, and those who consume alcohol are particularly at risk for higher uric acid levels, which could have implications for managing hypertension.

Recommendations: These findings suggest the need for targeted interventions and lifestyle modifications to address the risks associated with elevated uric acid levels in hypertensive populations.

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LIST OF ABBREVIATIONS

DBP	Diastolic blood pressure
SBP	Systolic blood pressure
HBP	High Blood Pressure
CVD	Cardiovascular Disease
SUA	Serum Uric Acid
HUA	Hyperuricemia
URAT	Uric Acid Transporter
CKD	Chronic kidney disease
SPSS	Statistical Package for Social Science
KU	Kairuki University
FHoKD	Family History of Kidney Disease
HoKD	History of Kidney Disease
FHoHT	Family History of Hypertension
HoHT	History of Hypertension
MSS	Musculoskeletal System

DEFINITION OF TERMS

Hypertension in adults is defined as the systolic blood pressure readings of equal or more than 140 mmHg and/or the diastolic blood pressure readings of equal or more than 90 mmHg on at least two separate readings.[1]

A known hypertensive patient is someone who reported a history of hypertension that was also confirmed from the patient's file

Hyperuricemia is defined as a plasma (or serum) urate concentration >405 umol/L (6.8 mg/dl). Females>360umol/L (6mg/dl) and males >420umol/L (7mg/dl)

Body Mass Index (BMI) It is defined as ratio of body weight in kilograms divided by the square of the height in metres $(kg/m2)^2$.

Cardiac clinic is defined as a healthy facility where services are provided by a cardiologist.

Social economic status is referring to the social standing or class of an individual or group, often measured as a combination of factors such as income, education and occupation.

Low social economic status means less access to financial, educational, social, and health resources.

Medium social economic status means medium access to financial, educational, social, and health resources

High social economic status means high access to financial, educational, social, and health resources.

CHAPTER ONE

INTRODUCTION

1.1 Background

Hypertension is defined as a systolic blood pressure of ≥140 mmHg and/or diastolic blood pressure of ≥ 90 mmHg on the average of two or more readings taken at each of two or more visits after initial screening.[1] Hypertension increases the conditions such as coronary heart disease (CHD), congestive heart failure (CHF), ischemic and hemorrhagic stroke, renal failure, and peripheral arterial disease hypertensive nephrosclerosis, and hypertensive heart disease.[1,3]

1.1.1 Classification of hypertension

Hypertension is classified into several categories based on blood pressure readings.[4] Optimal blood pressure is defined as systolic <120 mmHg and diastolic <80 mmHg.[4] Normal blood pressure ranges from systolic 120-129 mmHg and/or diastolic 80-84 mmHg.[4] High normal blood pressure is characterized by systolic 130-139 mmHg and/or diastolic 85-89 mmHg.[4]Grade 1 hypertension is defined as systolic 140-159 mmHg and/or diastolic 90-99 mmHg, while Grade 2 hypertension ranges from systolic 160-179 mmHg and/or diastolic 100-109 mmHg.[4] Grade 3 hypertension is identified by systolic ≥180 mmHg and/or diastolic ≥110 mmHg.[4] Additionally, isolated systolic hypertension is defined as systolic ≥140 mmHg with diastolic <90 mmHg.[4] Understanding these categories is crucial for diagnosing and managing hypertension effectively.

1.1.2 Epidemiology of hypertension

Hypertension is the leading cause of morbidity and mortality worldwide, affecting an estimated 1.20 billion adults aged 30-79 years.[5] In sub-Saharan Africa, the prevalence of hypertension is rising, contributing to increased cardiovascular-related deaths, especially in regions with high illiteracy rates.[5] The prevalence in Africa ranges from 25% to 35% among adults aged 25 to 64 years, increasing with age.[5] In Tanzania, studies have shown a growing prevalence of hypertension in both rural and urban populations, with alcohol consumption, obesity, and smoking as main risk factors.[6] For instance, studies in Arusha and Morogoro reported hypertension rates of 25.7% and 45%, respectively, with higher odds among males, older adults, and those with obesity.[7,8]

1.1.3 Etiological type of hypertension

Hypertension is categorized into primary (essential) and secondary types.[1] Primary hypertension, which accounts for about 95% of cases, has no specific underlying cause but is linked to factors like renal dysfunction, endothelial dysfunction, and lifestyle factors such as obesity, excessive salt intake, tobacco and alcohol use, and physical inactivity.[1] Secondary hypertension, making up 5-10% of cases, often occurs in individuals under 30 and results from conditions like renal disease, endocrine disorders, and certain medications.[1,9] Additionally, malignant hypertension, isolated systolic hypertension, and resistant hypertension can fall under either primary or secondary categories.[1,9,10]

1.1.4 Management of hypertension

Many people with hypertension are asymptomatic, so regular blood pressure measurement is essential.[1] Symptoms can include headaches, blurred vision, chest pain, dizziness, dyspnea, nausea, vomiting, anxiety, confusion, buzzing in the ears, nosebleeds, and an irregular heart rate.[1] Regular monitoring helps in early detection and management.[1,6] Non-pharmacological treatment of hypertension involves lifestyle modifications such as weight reduction through exercise, smoking cessation, and reduced alcohol intake.[1,6] The WHO recommends initial pharmacological treatment with thiazides, ACE inhibitors/ARBs, or long-acting dihydropyridine calcium channel blockers.[1,3] The target blood pressure goal is 140/90 mmHg or lower for all patients without comorbidities.[12]

1.1.5 Hyperuricemia

Hyperuricemia is defined as an abnormally high level of uric acid in the blood,[13] with serum concentrations exceeding 360 µmol/L (6 mg/dL) in females, 420 µmol/L (7 mg/dL) in males, and 5.5 mg/dL in individuals under 18 years old. Normal serum uric acid levels range from 155-257 µmol/L (2.6-6.8 mg/dL) in adult females and 208-428 µmol/L (3.5-7.2 mg/dL) in adult males. [13]

1.1.6 Epidemiology of hyperuricemia

Asymptomatic hyperuricemia affects up to 21% of the general population and 25% of hospitalized patients, with increased uric acid levels often preceding gout symptoms by 10-15 years. [14] The incidence of hyperuricemia is rising globally, particularly in economically privileged and emerging nations, and is notably high among Pacific islanders.[15] Men have higher rates than women, as estrogen provides

protection against hyperuricemia, but women can develop high serum uric acid levels after menopause. [16,17]

1.1.7 Etiology of hyperuricemia

Increased uric acid production can result from a purine-rich diet, including alcohol, certain meats, and seafood, as well as errors in purine metabolism like HPRT deficiency and PRPP synthetase overactivity.[18] Fructose metabolism, particularly from high-fructose corn syrup and sugary sodas, also contributes to hyperuricemia. Conditions causing cell breakdown, such as lymph proliferative and myeloproliferative diseases and intense exercise, can elevate uric acid levels.[19,20] Decreased excretion of uric acid can be due to kidney disease, acidosis, hypovolemia, certain medications, and endocrine disorders like hyperparathyroidism and hypothyroidism. [18]

Uric acid, an abnormal byproduct of purine metabolism, results from the breakdown of adenine and guanine, components of DNA, RNA, and ATP, and is implicated in hyperuricemia.[21] The kidneys process uric acid through filtration, reabsorption, secretion, and post-secretory reabsorption,[22] eliminating about 70% daily, with the rest excreted through the intestines.[22] Hyperuricemia arises from increased uric acid production, decreased excretion, or both.[18] The body's urate levels depend on the balance between dietary purine intake, internal urate production (e.g., cell turnover), and urate excretion via urine or the gastrointestinal tract. [13]

1.1.8 Management of hyperuricemia

According to the American College of Rheumatology (ACR) mild to moderate asymptomatic hyperuricemia (typically< 535 µmol/L) is managed conservatively through lifestyle and dietary modifications, such as weight reduction, reduced alcohol consumption, increased hydration, limiting fructose-containing beverages, and avoiding purine-rich foods like red meats and shellfish.[7] Severe asymptomatic hyperuricemia, if exceed 650 µmol/L, consider pharmacological treatments include urate-lowering drugs like Allopurinol, Probenecid, which increases uric acid secretion, and Rasburicase, which converts uric acid to allantoin.[7] Conservative management is preferred unless specific indications like chronic kidney disease or recurrent uric acid kidney stones are present.[1,3]

1.1.9 Complications of hyperuricemia

The complications of chronic high serum uric acid are cardiovascular disorders like hypertension, heart failure, atrial fibrillation, limb ischemia, left ventricular hypertrophy, gout, renal diseases such as nephrolithiasis, and renal nephropathy, which may result in chronic kidney disease.[1,3]

1.1.10 Pathophysiology of Hyperuricemia in Hypertension

The increase in serum uric acid in hypertension may be due to decreased renal blood flow, which stimulates urate reabsorption.[23] Hypertension also leads to microvascular disease, causing local tissue ischemia. Uric acid affects blood vessels in two stages: initially, it activates the renin-angiotensin system and downregulates nitric oxide production, leading to vasoconstriction and improved blood pressure with uric acid reduction. [24]

In the second stage, known as uric acid-mediated arteriolosclerosis, vascular smooth muscle cells absorb uric acid, triggering the synthesis of growth factors and protein-1, causing vascular wall thickening and loss of compliance.[25] This process results in chronic sodium-sensitive hypertension, which is not reversed by late uric acid reduction. Ischemia also increases uric acid production and inhibits urate secretion due to lactate release. [26]

Other factors contributing to the association between uric acid and hypertension include alcohol abuse,[27] lead intoxication,[28] obesity, insulin resistance, and diuretic use.[29] Ischemia-induced uric acid production may explain elevated uric acid levels in conditions like preeclampsia[30] and congestive heart failure.[31] These factors highlight the complex interplay between uric acid and hypertension.

1.1.11 Relationship between hyperuricemia and uric acid

The relationship between uric acid and hyperuricemia is well-established, as hyperuricemia is defined by elevated levels of uric acid in the blood. Uric acid is the end product of purine metabolism, and its accumulation can lead to the formation of monosodium urate crystals, which are responsible for conditions such as gout and urolithiasis[32]. Several factors contribute to hyperuricemia, including genetic predisposition, dietary habits, renal function, and the use of certain medications[32,33].

The author's opinion is that addressing hyperuricemia in hypertensive patients is crucial due to its association with increased cardiovascular risk, chronic kidney disease, and metabolic syndrome[34]. Despite the clear need for intervention, there may be a lack of targeted treatments for this group due to various reasons, such as

limited awareness among healthcare providers, insufficient screening practices, and the complexity of managing multiple comorbidities simultaneously[33].

To address these gaps, the study emphasizes the importance of routine screening for hyperuricemia in hypertensive patients and advocates for integrated management strategies that consider both hypertension and elevated uric acid levels. This approach could lead to better overall health outcomes and reduce the burden of associated complications

1.2 Problem statement

Globally, approximately 21% of adults are affected by hyperuricemia, which is linked to conditions such as gout, cardiovascular diseases, and chronic kidney disease.[32] In Africa, the prevalence of hypertension is rising, with 30-50% of adults affected in various regions.[32] However, specific data on hyperuricemia prevalence in hypertensive populations is limited, particularly in Tanzania. In Dar es Salaam, the high prevalence of hypertension is not matched by comprehensive data on hyperuricemia, hindering effective public health responses.

Hyperuricemia can lead to severe health issues, including heart failure, hypertension, atrial fibrillation, gout, and renal illnesses.[34,35] This not only affects individuals' health but also burdens the healthcare system and reduces patients' quality of life. Contributing factors include lack of awareness among healthcare providers and patients, limited access to healthcare, poor dietary habits, rising obesity rates, sedentary lifestyles, and weak healthcare infrastructure.

Failing to address hyperuricemia in hypertensive patients may lead to increased heart problems and kidney failures, escalating healthcare costs and mortality rates. To alleviate this problem, regular screening of uric acid levels in hypertensive patients, education to increase awareness, integrated treatment approaches, promotion of healthy lifestyles, and improved healthcare access are recommended. This study aims to fill the gap in data and provide actionable insights for better management of hyperuricemia among hypertensive patients in Dar es Salaam.

1.3 Rationale

Hyperuricemia is linked to serious health complications such as cardiovascular disease, gout, and renal illness, with heightened risks in hypertensive patients. Understanding this relationship is crucial for developing effective treatment strategies and improving patient outcomes.

The rising prevalence of hypertension in Tanzania, particularly in urban areas like Dar es Salaam, is concerning due to its association with hyperuricemia and related complications. Limited awareness and research on this issue hinder effective public health interventions.

Identifying hyperuricemia in hypertensive patients allows for early intervention and better disease management, reducing the risk of complications. Integrating hyperuricemia management into hypertension care can improve patient health, inform healthcare policies, and reduce healthcare costs.

1.4 Research Question

1.4.1 Broad research question

What is a prevalence of hyperuricemia among hypertensive patients attending cardiac clinics in Dar es Salaam, and how is it distributed across various demographic, lifestyle, socioeconomic, and clinical factors, including the identification of associated risk factors?

1.4.2 Specific research questions

1. What is the prevalence of hyperuricemia among hypertensive patients attending cardiac clinics in Dar es Salaam?
2. How is hyperuricemia distributed by age and gender among hypertensive patients attending cardiac clinics in Dar es Salaam?
3. How is hyperuricemia distributed by risk factors such as smoking, alcohol consumption, and obesity among hypertensive patients attending cardiac clinics in Dar es Salaam?
4. How hyperuricemia distributed by socioeconomic status among hypertensive patients attending cardiac clinics in Dar es Salaam?
5. How is hyperuricemia distributed by the grade of hypertension among hypertensive patients attending cardiac clinics in Dar es Salaam?
6. What factors are associated with hyperuricemia among hypertensive patients attending cardiac clinics in Dar es Salaam?

1.5 Objectives

1.5.1 Broad Objective.

To determine the distribution of hyperuricemia among hypertensive patients attending cardiac clinics in Dar es Salaam.

1.5.2 Specific Objectives

1. To determine the prevalence of hyperuricemia among hypertensive patients attending cardiac clinics in Dar es Salaam.
2. To determine the distribution of hyperuricemia by age, gender among hypertensive patients attending cardiac clinics in Dar es Salaam.
3. To determine the distribution of hyperuricemia by risk factors (smoking, alcohol consumption and obesity) among hypertensive patients attending cardiac clinics in Dar es Salaam.
4. To determine the distribution of hyperuricemia by social economic status such as low or high social economic status among hypertensive patients attending cardiac clinics in Dar es Salaam.
5. To determine the distribution of hyperuricemia by grade of hypertension among hypertensive patients attending cardiac clinics in Dar es Salaam.
6. To determine the distribution of hyperuricemia according to anti-hypertensive drugs used among hypertensive patients attending cardiac clinics in Dar es Salaam.
7. To identify the factors associated with hyperuricemia among hypertensive patients attending cardiac clinics in Dar es Salaam.

CHAPTER TWO

LITERATURE REVIEW

2.1 Overview of the prevalence and associated factors of hyperuricemia among hypertensive patients attending cardiac clinics.

The literature on hyperuricemia among hypertensive patients provides valuable insights into its prevalence, social demographic factors, clinical factors, and the impact of antihypertensive medications. Studies from various regions, including South Asia,[36] Pakistan,[37] Bangladesh,[38] Southwest China,[39] Korea,[40] Assam (India),[41] Cameroon,[42] Angola,[43] Seychelles,[44] Nigeria, [45] and Kenya,[46] highlight the diverse epidemiology of hyperuricemia. This review underscores the need for localized research to better understand and manage hyperuricemia in different populations.

Prevalence rates of hyperuricemia among hypertensive patients vary significantly, ranging from 9.1% in men in Bangladesh[38] to 84% in Pakistan.[37] These variations highlight the importance of regional studies to accurately assess the burden of hyperuricemia. Social demographic factors such as age and gender are consistently associated with hyperuricemia, with higher prevalence rates observed in males and older age groups. [37,38] These demographic variables are crucial for assessing the risk of hyperuricemia in hypertensive patients. Clinical factors, including higher Body Mass Index (BMI), metabolic syndrome, and elevated serum uric acid (SUA) levels, are significantly associated with hyperuricemia. Comprehensive clinical management is essential to mitigate the risk of hyperuricemia in hypertensive patients.

2.2 Prevalence of hyperuricemia among hypertensive patients

The prevalence of hyperuricemia among hypertensive patients has been studied in various regions, revealing significant variations. In South Asia, a 2021 study at Birat Hospital reported a prevalence of 25%.[36] Similarly, a study conducted at the Pakistan Emirates Military Hospital found a strikingly high prevalence of 84% among hypertensive patients.[37] In Bangladesh, a study involving 255 adults found a lower prevalence, with 9.1% in men and 10.3% in women.[38] Meanwhile, a 2014 study in Southwest China reported 18.2% prevalence among hypertensive patients. [39]

Further studies have shown diverse prevalence rates in different populations. In Assam, India, a study from 2021-2022 found that 20% of hypertensive patients were hyperuricemic.[41] In Cameroon, a 2012 study reported a prevalence between 28.7% and 34.9% among newly diagnosed, treatment-naïve hypertensive patients.[42] In Angola, a 2017 study found a 25% prevalence among hypertensive patients.[43]

Additional research in the Seychelles and Nigeria has provided further insights. A 2004 study in the Seychelles reported a prevalence of 8.7% in women and 35.2% in men.[44,45] In Nigeria, a 2020 study found a 46.9% prevalence among hypertensive patients.[45] In Kenya, a 2016 study reported 44% prevalence among hypertensive patients. [46] These studies highlight the significant burden of hyperuricemia in hypertensive populations across different regions.

Given the varying prevalence rates and the lack of specific data from Tanzania, it is crucial to conduct a study to ascertain the prevalence of hyperuricemia among

hypertensive patients in this region. Understanding the local epidemiology of hyperuricemia will provide valuable insights into its impact on the Tanzanian population and help inform targeted healthcare strategies and interventions. This study will fill a critical gap in the literature and support efforts to improve the management and prevention of hyperuricemia and its associated complications in hypertensive patients in Tanzania.

2.3 Distribution of hyperuricemia level by age, gender among hypertensive patients.

The literature reveals several social demographic factors associated with hyperuricemia among hypertensive patients. In the South Asia study conducted in 2021, it was found that males had a higher prevalence of hyperuricemia (30.8%) compared to females (14.8%). Additionally, patients over 60 years of age had a significantly higher prevalence (41.7%). These findings suggest that gender and age are critical demographic factors influencing the prevalence of hyperuricemia in hypertensive populations.

In the Pakistan Emirates Military Hospital study, the mean age of participants was 54.79 years, with a higher prevalence of hyperuricemia among males (32%) compared to females (68%).[37] This study further supports the notion that age and gender are significant demographic factors. Similarly, the Bangladesh study reported a prevalence of 9.1% in men and 10.3% in women, indicating that gender differences exist in the prevalence of hyperuricemia, although the rates were closer compared to other studies.[38]

The Southwest China study from 2014 also highlighted gender differences, with a prevalence of 21.5% in males and 16.2% in females.[39] This study, along with the findings from the Seychelles study, which reported a prevalence of 35.2% in men and 8.7% in women, underscores the consistent pattern of higher prevalence in males across different regions.[44] These studies collectively indicate that gender is a significant demographic factor associated with hyperuricemia among hypertensive patients.[39,44]

2.4 Distribution of hyperuricemia by socioeconomic status among hypertensive patients.

Socioeconomic status, particularly income levels, significantly influences the prevalence of hyperuricemia. Individuals with lower income levels (e.g., $0 - $500 per month) often face barriers to accessing healthcare and maintaining a healthy diet, which can lead to higher rates of hyperuricemia[47]. Poor dietary habits, such as increased consumption of purine-rich foods and sugary beverages, are more common in low-income groups due to affordability and availability[48]. Conversely, those with higher income levels (e.g., above $2000 per month) might have better access to healthcare and healthier food options, but they may also indulge in lifestyle choices that increase hyperuricemia risk, such as higher alcohol consumption and rich diets[49]. Middle-income individuals (e.g., $501 - $2000 per month) often fall between these extremes, with varying influences based on their specific circumstances [47]. These income-related disparities highlight the need for targeted public health interventions to address the unique challenges faced by different socioeconomic groups in managing hyperuricemia.

2.5 Distribution of hyperuricemia by risk factors (smoking, alcohol consumptions and obesity) among hypertensive patients.

The distribution of hyperuricemia among hypertensive patients varies significantly with alcohol consumption and smoking status. Studies have shown that hypertensive patients who consume alcohol are more likely to develop hyperuricemia. For instance, a study in Chongqing, China, reported that 35% of hypertensive patients who consumed alcohol had hyperuricemia, compared to 20% of non-drinkers. [39] This indicates a higher prevalence of hyperuricemia among those who drink alcohol, particularly in those who consume it excessively. The data suggests that reducing alcohol intake could be a crucial step in managing hyperuricemia among hypertensive patients.

Similarly, smoking status also influences the prevalence of hyperuricemia among hypertensive individuals. Research from the Saku study in Korea found that 25% of current smokers with hypertension had hyperuricemia, compared to 18% of non-smokers.[40] Although the difference is less pronounced than with alcohol consumption, it still highlights smoking as a contributing factor. These findings emphasize the importance of lifestyle modifications, such as reducing alcohol consumption and smoking cessation, in the management of hyperuricemia among hypertensive patients**.**

Additionally, other factors such as body mass index (BMI), physical activity, and dietary habits also contribute to the prevalence of hyperuricemia in hypertensive patients. Overall, these studies underscore the multifactorial nature of hyperuricemia and the importance of considering lifestyle factors in managing

hypertensive patients.[37] The literature reveals several clinical factors associated with hyperuricemia among hypertensive patients. In the South Asia study conducted in 2021, it was found that patients with higher BMIs (34.5%) were more likely to have hyperuricemia.[36] This study highlighted the significant association between BMI over 25 kg/m² and hyperuricemia[36] Similarly, the Pakistan Emirates Military Hospital study reported a high prevalence of hyperuricemia (84%) among hypertensive patients, emphasizing the clinical importance of monitoring serum uric acid levels in this population.[36,]

2.6 Distribution of hyperuricemia by grade of hypertension among hypertensive patients.

In the Bangladesh study, there was a positive correlation between blood pressure and serum uric acid (SUA) levels, with an increasing trend for both prehypertension and hypertension as SUA levels rose in the quartiles.[38] This indicates that elevated SUA levels are clinically significant in the progression of hypertension. The Southwest China study from 2014 also noted a significant frequency of hyperuricemia among urban hypertensive patients, suggesting that lifestyle and environmental factors may play a role in its prevalence.[39]

The Korea Cohort Study found that the relationship between serum uric acid (UA) and blood pressure (BP), hyperuricemia, and hypertension varied by age group.[40] For men under 60, hyperuricemia increased the relative risk of hypertension by nearly 30%. Additionally, the Assam, India study from 2021-2022 reported that 20% of hypertensive patients were hyperuricemic, with a significant presence of hyperuricemia in individuals with metabolic syndrome.[41] Serum uric acid levels were

significantly correlated with factors related to metabolic syndrome, such as dyslipidaemia and stage 2 hypertension, [41].

2.7 Distribution of hyperuricemia according to anti-hypertensive drug used among hypertensive patients.

The literature reveals that certain antihypertensive medications are associated with an increased risk of hyperuricemia. Diuretics, particularly thiazide diuretics, are commonly used to manage hypertension but are well-documented to elevate serum uric acid (SUA) levels. This occurs because diuretics increase uric acid reabsorption and decrease its excretion, leading to higher SUA levels. Studies have shown that up to 70% of patients on diuretics may develop hyperuricemia, highlighting the significant impact of these medications on uric acid metabolism [50–52].

The distribution of hyperuricemia among hypertensive patients varies significantly depending on the antihypertensive drugs they use. Studies have shown that certain medications, such as diuretics and beta-blockers, are associated with a higher prevalence of hyperuricemia. For instance, research indicates that 40% of hypertensive patients using diuretics develop hyperuricemia, compared to 25% of those on beta-blockers. Conversely, medications like calcium channel blockers and losartan, an angiotensin II receptor blocker, have been found to have uricosuric properties, which help lower uric acid levels. Hypertensive patients on calcium channel blockers or losartan have a lower prevalence of hyperuricemia, with only 15% and 10% respectively, compared to those on diuretics or beta-blockers. This variation underscores the importance of considering the impact of antihypertensive

drugs on uric acid levels when managing hypertensive patients, particularly those at risk for hyperuricemia. [51]

2.8 Factors associated with hyperuricemia among hypertensive patients

An association between increased SUA and hypertension has been reported in some epidemiological studies.

A cross section study done in 2020 by Chen, Hong Yuan3 at el**,** to Survey on uric acid in Chinese subjects with essential hypertension[53]. The aim was to explore the prevalence and determinants of HUA in Chinese hypertensive adults[53]. The prevalence of hyperuricemia in Chinese hypertensive patients was 38.7%[53]. Female sex, aging (≥65 years), and low eGFR were independent predictors of HUA[53]. HUA was lower among the patients who were taking losartan, valsartan, and Nifedipine. Western region residents, new-onset hypertension, longer hypertension duration, aspirin use, higher FBG, TG, LDL-C levels and BMI were potential risk factors for hyperuricemia[53].

Study of serum uric acid levels in essential hypertension was conducted in India in 2022 by Anita B, Sudam Kr, Dr. Musuku M at el. It was hospital based cross sectional study, to determine the prevalence and association of hyperuricemia with diagnosed cases of essential hypertension[54]. The Sample size was 235 cases with hypertensio; 83.8% were in stage I hypertension, while 16.2% were in stage II hypertension[54]. The Prevalence of hyperuricemia was found to be 27.7% among cases with hypertension[54]. Mean serum uric acid levels were significantly associated with an increase in systolic and diastolic blood pressure[54].

Another cross sectional study done in 2019 by N. Ali ,S.Mahmood2, F. Islam at el.[38]. to evaluate the association of SUA with hypertension among Bangladeshi adults. A total of 255 participants were recruited. The Prevalence of hyperuricemia was 9.1% in males and 10.3% in females[38]. An increasing trend for hypertension and prehypertension was found in both genders, with increasing SUA levels in the quartiles[38]. SUA levels in the quartiles were positively correlated with blood pressure[38].

The observational cross sectional study was conducted in the department of Medicine in a tertiary care hospital in Assam, India from June 2021 to May 2022 by Sarma Bhargab ,Doley Rimamoni, Brahma Bhaskar at el[41]. A total of 150 patients who were diagnosed as hypertensive[41] were enrolled, of which 20% cases were found to be hyperuricemic. Out of which 67% of the cases with metabolic syndrome had hyperuricemia[41]. Serum uric acid level correlated significantly with variables like BMI, Dyslipidemia, stage 2 hypertension, which are components of metabolic syndrome[41].

Another cross-sectional health examination survey was done in Seychelles 2004 by Conen D,V Wietlisbach, P and Bovet, atel to investigate the prevalence of hyperuricemia and the association between uric acid levels and the various cardiovascular risk factors in a developing country with high average blood pressures[44]. It recruited 1011 subjects aged 25 to 64 years[44]. The study reported a prevalence of hyperuricemia being 35.2% in men and 8.7% in women[44] . Serum uric acid was strongly related to serum triglycerides in men as well as in women ($r = 0.73$ in men and $r = 0.59$ in women, $p < 0.001$).

In 2014 a multistage, stratified sampling was conducted by Zhang Y, Feng N, X.Huang at el,to assess the prevalence of hyperuricemia and its associated risk factors among hypertensive patients in Southwest China[39]. It included 3505 hypertensive people aged 50–79 years[39]. The study population, approximately 18.2% of all hypertensive participants had hyperuricemia(638/3505),with a prevalence rate of 21.5% in men and 16.2% in women ($p < 0.05$)[39]. The prevalence of hyperuricemia among hypertensive patients in urban adults was high, while levels of awareness were extremely low[39].

Another case-control study was conducted at Moti Lal Nehru Medical College, Allahabad India 2016 by M A.Mishra, P.Gupta,A.Gupta,at el to determine the prevalence and association of hyperuricemia with newly diagnosed essential hypertension[55].Fifty newly diagnosed hypertensive patients aged >40 years as case and 50 age normotensive subjects as controls were included in the study[55]. A total of 13 (26%) subjects among cases and 3(6%)subjects in control groups were hyperuricemic (Odds ratio 5.50; $p <0.05$) [55]. The Prevalence of hyperuricemia in patients with essential hypertension was significantly higher than in the normal population[55]. Hyperuricemia was significantly associated with newly diagnosed essential hypertensive[55].

Few studies have evaluated the link between hyperuricemia and cardiovascular disease in sub-Saharan Africa. In 2012 Douala, Cameroon (Central Africa)Community-based cross-sectional study was done by Kamdema, M.Douallaa,F. Lekpa at el to assess the prevalence of and factors associated with hyperuricemia among newly diagnosed treatment-naïve hypertensive patients in

sub-Saharan Africa[42]. 839 newly diagnosed treatment-naïve hypertensive patients were enrolled 427 women and 412 men[42].The prevalence of hyperuricemia was 31.8% % (95% confidence interval [CI] 28.7—34.9) and did not differ by sex (132 women vs. 135 men; P = 0.56) [42]. It was common among newly diagnosed treatment-naïve hypertensive patients in sub-Saharan Africa and is associated with increasing age, obesity and family history of hypertension[42].

Another cross-sectional study was carried out in Ekit State University Teaching Hospital, Nigeria on 2020 by A**.**Oladapo A. E.Ajayi, Adebayo at el**.**[45] To determine Serum uric acid and left ventricular hypertrophy in hypertensive patients[45]**.** Total of 542 participants included 271 hypertensive cases and 271 non-hypertensive persons obliged as controls[45]. The prevalence of hyperuricemia was 46.9% among the hypertensive and 11.1% among the controls (p-value < 0.0001)[45]. Among the hypertensive patients, LVH was present in 39.3% of those with hyperuricemia and in 28.0% of those with normal SUA levels (p = 0.003). [45] .The results indicated that serum uric acid is positively correlated with hypertension and a reliable indicator of LVH in study population[45].

In additional to that Cross sectional study was conducted in 2016 at Moi Teaching and Referral Hospital, Eldoret Kenya by C.Sylvia **,**F Some,S Kimaiyo at el[46].among hypertensive patients to determine the prevalence and risk factors of hyperuricemia. A total of 275 patients were enrolled[46]. Prevalence of hyperuricemia was 44 %. Factors associated with hyperuricemia included high Body Mass index (p 0.036), low Glomerular Filtration Rate (P<0.0001) and dyslipidemia (p<0.0001)[46].

CHAPTER THREE

METHODOLOGY

3.1 Study Design

This is a cross-sectional descriptive study among hypertensive patients attending cardiac clinics in Dar es Salaam.

3.2 Study Area

This study was conducted in Dar es Salaam. The city is situated on a natural harbor on the East African coast at 6°48' S and 39°17' E.[56] It consists of three regional referral hospitals (Amana RRH, Mwananyamala RRH, and Temeke RRH) and five municipalities: Kinondoni, Ilala, Temeke, Ubungo, and Kigamboni. [56] In 2024, Dar es Salaam will have 8,161,000 residents. [56] Tanzania's principal economic growth engine, Dar es Salaam is a center for commerce, industry, administration, and fishing. [56]

3.3 General Population

All adult hypertensive patients in Dar–es–Salaam

3.4 Target Population

All adult hypertensive patients who attended cardiac clinics in Dar es Salaam

3.5 Study Population

Randomly selected hypertensive patients who attended cardiac clinics in Dar es Salaam

3.6 Eligibility Criteria

3.6.1 Inclusion Criteria

All hypertensive patients aged 18 years and above attending hypertensive clinics in DSM.

3.6.2 Exclusion Criteria

1. Patients with a known history of diabetes mellitus.
2. Patients with known history of renal impairment.
3. Patients with known history of malignancy and patients on chemotherapy.
4. Patients with known history of gout and patients on hyperuricemic drugs**.**

3.7 Sample Size Estimation

The Kish and Leslie formula used in cross-sectional studies[57]

$$n = \frac{z^2.p.(1-p)}{\varepsilon^2}$$

Where

Z — 1.96 for 95% CI

ε — margin of error (precision): for this study taken as 0.05

P — expected proportion with characteristic of interest. For this study p value = 44% was obtained from a previous study. [46]

$$n = \frac{1.96^2 * 0.44.(1-0.44)}{0.05^2} = 378$$

The estimated minimum sample size was approximately 380 ± 10

Justification of formula

A similar study was done by Sylvia C Mibey in 2016 at Moi Teaching and Referral Hospital, Eldoret, Kenya; on the prevalence of hyperuricemia among Patients with hypertension[46]. This was due to the socioeconomic similarities in the area where the study was coducted,which influenced both the study design and the operational definition of hypertension. Thus, usage of the same sample size formula ensured the validity results and minimized bias and wastage of resources.

3.8 Sampling Techniques

Stratified sampling technique was used in this study. The strata were regional referral hospitals; Amana, Mwananyamala and Temeke Regional referral hospitals. The proportionate sample were drawn from each hospital according to the following formula below.

- The monthly hypertensive patients seen in Amana RRH = ARRHc
- The monthly hypertensive patients seen in Mwananyamala RRH = MRRHc
- The total number of hypertensive patients seen in Temeke RRH = TRRHc

$$Sample\ recruited\ at\ ARRH\ = \frac{ARRHc}{\text{ARRHc} + \text{MRRHc} + \text{TRRHc}} * Sample\ size(381)$$

$$Sample\ recruited\ at\ MRRHc = \frac{MRRHc}{\text{ARRHc} + \text{MRRHc} + \text{TRRHc}} * Sample\ size(381)$$

$$Sample\ recruited\ at\ TRRHc\ = \frac{TRRHc}{\text{ARRHc} + \text{MRRHc} + \text{TRRHc}} * Sample\ size(381)$$

The number of samples recruited at ARRH, MRRH, and TRRH were 135, 107 and 139 hypertensive patients respectively

Several small pieces of papers were made with marks "Yes/No" separately on each piece of papers. These pieces of papers were folded and put into a jar. Potential participants were asked to pick one piece of papers. For those who selected a piece of paper with a mark "yes" were selected for potential participation in the study.

3.9 Data Collection

Data collection is comprised of sociodemographic, clinical, and laboratory variables. The principal investigator and a trained research assistant (nurse) collected data from study participants from selected hospitals**.**

Sociodemographic and clinical variables

The data collection tool for this study was a questionnaire. This questionnaire was comprised of social demographics, clinical characteristics, and patient medical and treatment history.

The social demographic information included gender, age, residence, and marital status, level of education, occupation, and employment status as for sociodemographic data. The socioeconomic data were asked from the participants as a numeric estimation of their monthly income, which were later categorized into low, middle, and high income based on the categorization by the previous studies.[47,49]

Clinical characteristics include history of hypertension, family history of hypertension, history of type of antihypertensive drug, history of stroke, ischaemic

heart disease, family history of kidney disease history of kidney disease and history consumption of tobacco and alcohol, history of musculoskeletal disease and history of joint disease.

Body Weight and Height Measurements

The research assistant, under the supervision of the Principal Investigator, was responsible for accurately measuring participants' body weight and height. The process began with measuring body weight using standardized RGZ-160 weighing scale, manufactured in Changzhou, China.

Participants were instructed to remove their shoes and any heavy clothing to ensure an accurate measurement. They stood on the scale with their feet close together and arms at their sides. The research assistant ensured that the participants were standing still and balanced before recording the weight to the nearest 0.5 kg.

Next, the height measurement was conducted using a height measuring rod. Participants were asked to stand on a flat surface parallel to the measuring scale. They were instructed to stand straight, look directly forward, and keep their feet together. The research assistant ensured that the participants' heels, buttocks, shoulders, and the back of their heads were touching the vertical surface of the measuring rod. The height was then recorded to the nearest 0.5 cm.

Body Mass Index was calculated using a person's height and weight, using the formula:

BMI = weight in kg/Height in m^2

The BMI parameters were used per the 2023 WHO guideline.

Underweight < 18.5

Normal weight -18.5–24.9,

Overweight-25-29.9,

Obese >30kg/ m^2

Blood pressure measurement

Blood pressure (BP) was measured using an aneroid sphygmomanometer (Spengler, Spengler SAS, Cachan, France). Participants were seated comfortably in a chair with their back supported and their arm at heart level. BP was measured in both arms during the initial screening, and the arms with the higher BP was used for subsequent measurements. The cuff was wrapped snugly around the upper arm, and the stethoscope was placed over the brachial artery. The cuff was inflated until the pulse was no longer audible, then slowly deflated while listening for the first appearance of the pulse sound (systolic pressure) and the disappearance of the pulse sound (diastolic pressure). The following definition was adopted for this study. Hypertension: persistently elevated BP of ≥ 140 mmHg for systolic or 90 mmHg for diastolic or both, measured at least twice, with a minimum of five minutes of rest between readings, to ensure accuracy after the initial screening or those receiving treatment for hypertension. The choice of 140/90 mmHg as a cut-off point is based on the World Health Organization on the classification of hypertension[4].

Serum uric acid test

A professional phlebotomist, under the supervision of the Principal investigator, was responsible for taking venous blood samples from study participants. Using a 5CC syringe, at least 2.5ml of blood was drawn from each participant. The collected blood samples were then transported to Kairuki Hospital for serum uric acid (SUA) measurement, ensuring that the transit time was less than 12 hours. Upon arrival,

the blood samples were allowed to clot and then centrifuged to separate the serum. The serum was stored at −20°C until analysis. The concentration of SUA was determined calorimetrically using a commercially available uric acid kit (uricase-peroxidase method). SUA was measured using a Mindray BS 240 analyzer, manufactured in Guangdong, China, in 2017.

The grading system for hyperuricemia with levels provided in micromoles per liter (μmol/L) with reference to standard guidelines such as American College of Rheumatology (ACR).[7]

Grade	**uric acid level (μmol/L)**
Normal	200-420(male)
	140-360(female)
Mild hyperuricemia	421-535
Moderate hyperuricemia	536-650
Severe hyperuricemia	>650

3.10 Data Analysis

IBM SPSS Statistics (Statistical Package for the Social Sciences), developed by IBM Corporation in Armonk, New York, USA, was used for data analysis. Data entry was performed by the principal investigator (PI) into SPSS. The data were then cleaned to address any missing entries, forgotten data, and incorrect entries.

Descriptive data for continuous variables, such as age and BMI, were summarized using measures of central tendency (median) and dispersion (range). For categorical variables, such as gender and education level, frequencies and percentages were calculated to provide a clear overview of the distribution within

the study population. Categorical data were presented in tabular form using frequency tables that displayed the counts and percentages of each category. Textual descriptions accompanied these tables and figures to enhance clarity and understanding.

Bivariate analysis was conducted using the chi-square test to examine significant differences between groups for categorical variables. Variables with a p-value less than 0.25 were considered significantly different and were subsequently included in the multivariable logistic regression model to identify independent associations with hyperuricemia while controlling for other confounders.

Multivariable logistic regression was performed to control for confounding variables and identify independent predictors of hyperuricemia. Variables with a p-value less than 0.05 were considered statistically significant. Adjusted odds ratios (aORs) and 95% confidence intervals (CIs) were reported to quantify the strength and precision of the associations.

3.11 Study variables

3.11.1 Dependent Variable

- Hyperuricemia

3.11.2 Independent Variables

The independent variables of this study were categorized into Social demographics and the patient's lifestyle risk history.

- Social demographic: Age group: <45, 45-54, 55-64, & 65+, gender, marital status, occupation, education level, income.

- Lifestyle and risk history: BMI, history of alcohol consumption, history of smoking, history of hypertension, family history of hypertension, family history of kidney disease, history of stroke, history of heart disease, history of kidney disease, history of MMS and history of joint disease.

3.12 Conceptual framework

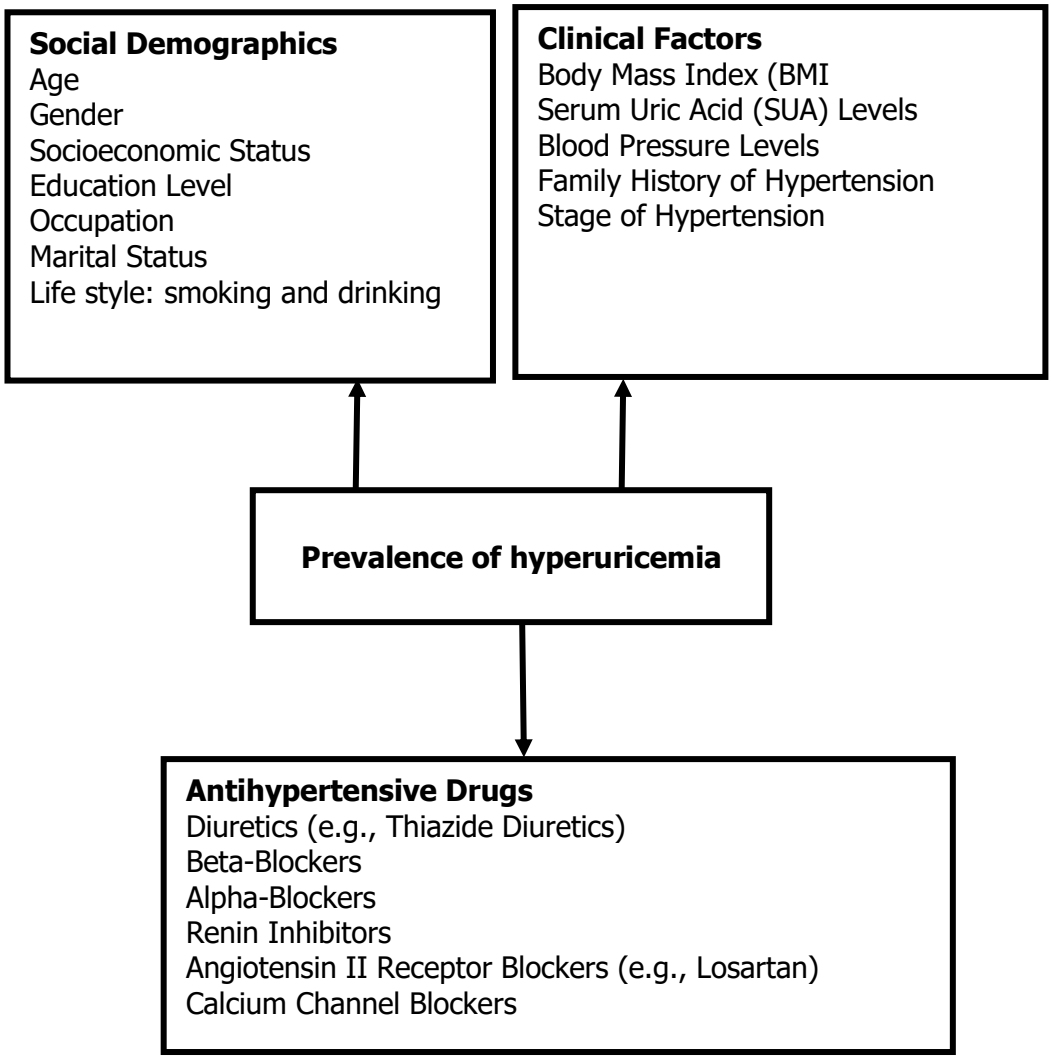

Figure 1: Conceptual framework, developed after literature review

3.13 Dissemination of results

The research result findings will be disseminated to the KU library, the Ministry of Health, association of physicians of Tanzania through research report, scientific conferences, and publications**.**

CHAPTER FOUR

ETHICAL CONSIDERATION

Ethical clearance was obtained from the Research Committee of Kairuki University (KU). Permission to conduct the study was obtained from the medical officer in charge of the selected regional hospitals. The purpose and procedures of the study were explained to the participants. All participants signed an Informed consent form in Swahili. Participants were informed of the possibility of experiencing mild pain during sample withdrawal (phlebotomy), which resolved immediately after the procedure. Information that was obtained during this study was treated with the highest degree of confidentiality. All investigation results were communicated back to the patients, and those participants who were identified were referred for further management. At the end of this study, the result was disseminated to KU, the Ministry of Health, and other places, and at the end of this study, it was published.

CHAPTER FIVE

RESULTS

5.1 Demographic characteristics of the study participants

This study was conducted for 10 months, from November 2023 to August 2024.This study recruited 381 primary hypertensive patients. Figure 1, shows grade of these primary hypertensive patients, with 63(16.5%) were optimal or normal state. Most of the recruited participants, 317(83.2) were using anti-hypertensive drugs.

The proportion of the participants recruited from each hospital was 139(63.5%), 135(35.4%) and 107(28.1%) recruited from Temeke, Amana and Mwananyamala regional referral hospitals, respectively.

Table 1, describes the social demographic and socioeconomic profiles of the study participants. The median participant's age was 63 years, ranging from 28 to 88 years. Most participants, 259(68.0%) were female, 230(60.4%) were married/cohabiting, 270(70.9%) had no-formal or primary school education level. The majority, 216(56.7%) of the study participants were at lower income levels**.**

Table 1:Demographic characteristics among hypertensive patients attending cardiac clinics in Dar es Salaam (N=381).

Variable	N(%)
Age	
<45	30 (7.9)
45-54	82 (21.5)
55-64	114 (29.9)
65+	155 (40.7)
Gender	
Male	122 (32.0)
Female	259 (68.0)
Marital status	
Unmarried	151 (39.6)
Married	230 (60.4)
Occupation	
Unemployed or Retired	212 (55.6)
Employed or Self employed	169 (44.4)
Education	
Primary/informal	270 (70.9)
Secondary	55 (14.4)
College/higher education	56 (14.7)
Income	
Low	216 (56.7)
Middle	164 (43.0)
High	1 (.3)
Alcohol consumption	
No	220 (57.7)
Yes	161 (42.3)
Smoking	
No	316 (82.9)
Yes	65 (17.1)

5.2 Clinical characteristics among hypertensive patients attending cardiac clinics in Dar es Salaam (N=381).

Table 2, Clinical characteristics of the study participants, the study sample consisted of participants with varying body mass index (BMI) categories, duration since hypertension diagnosis, and hypertension grades. The median duration since the diagnosis of hypertension among study participants was 3 years, ranging from less than a year to 15 years. The median BMI was 26.1, ranging from 13.6 to 72.7. Most participants, 154(40.4%), and 113(29.7%) were overweight and obese

respectively. The majority of participants were either overweight or obese. Most had been diagnosed with hypertension for less than four years. Hypertension grades ranged from normal to isolated, with a significant portion having a normal grade or isolated hypertension.

Regarding medical history, a substantial number of participants had no history of hypertension (HT), while a notable proportion had a family history (FH) of hypertension. The prevalence of kidney disease (KD) and stroke was relatively low among the participants, with most having no history of these conditions. Similarly, the majority had no history of heart disease (HD) or musculoskeletal symptoms (MSS).

Table 2: Clinical characteristics among hypertensive patients attending cardiac clinics in Dar es Salaam (N=381).

Variables	N(%)
BMI	
Underweight	9 (2.4)
Normal	105 (27.6)
Overweight	154 (40.4)
Obese	113 (29.7)
Duration since diagnosis	
< 4	252 (66.1)
4+	129 (33.9)
Hypertension grade	
Normal	121 (31.8)
Grade 1	76 (19.9)
Grade 2	41 (10.8)
Grade 3	23 (6.0)
Isolated	120 (31.5)
History of HT	
No	260 (68.2)
Yes	121 (31.8)
FH of HT	
No	211 (55.4)
Yes	170 (44.6)
FH of KD	
No	336 (88.2)
Yes	45 (11.8)
History of stroke	
No	329 (86.4)
Yes	52 (13.6)
History of HD	
No	375 (98.4)
Yes	6 (1.6)
FH of KD	
No	378 (99.2)
Yes	3 (.8)
History of MSS	
No	301 (79.0)
Yes	80 (21.0)

5.3 Distribution of antihypertensive drugs used among hypertensive patients attending cardiac clinics in Dar es Salaam (N=381).

Figure 1 below, the distribution of antihypertensive drug use among the study participant's shows that calcium channel blockers (CCBs) were the most commonly used, with 59.8% of participants taking them. Angiotensin II receptor blockers (ARBs) were also widely used, with 52.5% of participants on this medication. Diuretics were used by 41.7% of the participants, while beta-blockers and angiotensin-converting enzyme inhibitors (ACEIs) were less commonly used, at 10.0% and 5.2%, respectively.

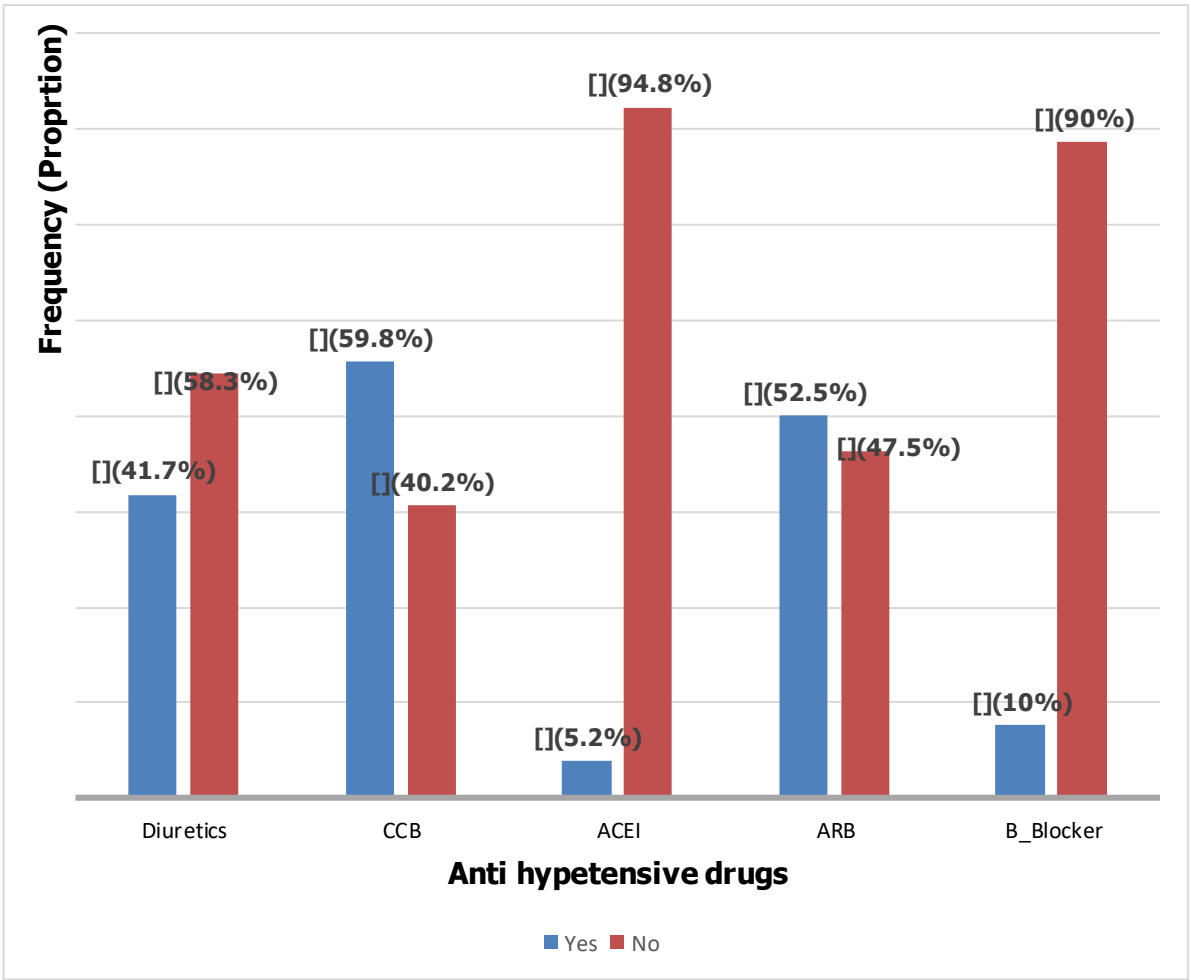

Figure 2. Distribution of anti-hypertensive drugs among hypertensive patients attending cardiac clinics in Dar es Salaam.

5.4 The prevalence of hyperuricemia among hypertensive patients attending cardiac clinics in Dar es Salaam (N=381).

The median uric acid of the study participants was 335.4, ranging from 1.4 to 885.4. Of the total participants, 162(42.5%) were identified with elevated uric acid.

5.5: Distribution of social demographic factors associated with hyperuricemia among hypertensive patients attending cardiac clinics in Dar es Salaam (N=381)

Table 3; show the distribution of Uric Acid (UA) levels across different age groups and genders indicates a trend where the proportion of individuals with high UA levels increases with age. Individual under 45 years have a 30% occurrence of high UA levels, which nearly doubles to 49.7% in those aged 65 and above. This trend is statistically significant, as suggested by the p-value of 0.041. Gender also plays a role, with males showing a higher proportion of normal UA levels (68.9%) compared to females (52.1%), and this difference is highly significant with a p-value of 0.002, indicating a potential influence of gender on UA levels.

Table 3; show the distribution of AU across socioeconomic variables. The distribution of UA levels across different socioeconomic variables indicates a relatively uniform distribution among the unemployed or retired, and employed or self-employed groups, with no significant difference in UA status (p-value 0.698). In terms of income level, there is a slight increase in normal UA levels among the middle-income group compared to the low-income group, but this difference is not statistically significant (p-value 0.389).

Table 3: Bivariate analysis of Social demographic factors associated with hyperuricemia among hypertensive patients attending cardiac clinics in Dar es Salaam (N=381).

Variables	Normal	High	p value
Age group			0.041
<45	21 (70.0)	9 (30.0)	
45-54	55 (67.1)	27 (32.9)	
55-64	65 (57.0)	49 (43.0)	
65+	78 (50.3)	77 (49.7)	
Gender			0.002
Male	84 (68.9)	38 (31.1)	
Female	135 (52.1)	124 (47.9)	
Marital status			0.15
Unmarried	80 (53.0)	71 (47.0)	
Married	139 (60.4)	91 (39.6)	
Occupation			0.698
Unemployed or Retired	120 (56.6)	92 (43.4)	
Employed or Self employed	99 (58.6)	70 (41.4)	
Education			0.914
Primary/informal	157 (58.1)	113 (41.9)	
Secondary	31 (56.4)	24 (43.6)	
College/higher education	31 (55.4)	25 (44.6)	
Income			0.389
Low	121 (56.0)	95 (44.0)	
Middle	98 (59.8)	66 (40.2)	
High	0 (.0)	1 (100.0)	

5.6 Distribution of hyperuricemia level by risk factors among hypertensive patients attending cardiac clinics in Dar es Salaam

The bivariate analysis of the clinical and medical history factors was conducted. There were significant differences in hyperuricemia prevalence across BMI categories and history of hypertension (HT). Specifically, obese participants had a higher proportion of hyperuricemia (54.0%) compared to other BMI categories. There was a significant association between alcohol consumption and higher UA levels, as indicated by the p-value of 0.027. Conversely, smoking does not show a significant correlation with UA status, as reflected by the p-value of 0.316.

Participants with a history of hypertension also had a higher proportion of hyperuricemia (50.4%) compared to those without (38.8%). Other variables, such as duration since diagnosis, family history of hypertension (FH HT), family history of kidney disease, history of stroke, history of heart disease, family history of kidney disease, history of musculoskeletal symptoms (MMS), and history of joint disease, did not show significant differences in hyperuricemia prevalence. Variables with a p-value less than 0.25 were considered for further analysis.

Table 4: Bivariate analysis distribution of hyperuricemia level by risk factors among hypertensive patients attending cardiac clinics in Dar es Salaam (N=381)

Variables	Normal UA	Hyperuricemia	p value
BMI			0.026
Underweight	5 (55.6)	4 (44.4)	
Normal	63 (60.0)	42 (40.0)	
Overweight	99 (64.3)	55 (35.7)	
Obese	52 (46.0)	61 (54.0)	
Duration Since diagnosis			0.178
< 4	151 (59.9)	101 (40.1)	
4+	68 (52.7)	61 (47.3)	
Alcohol consumptions			0.027
No	137 (62.3)	83 (37.7)	
Yes	82 (50.9)	79 (49.1)	
Smoking			0.316
No	178 (56.3)	138 (43.7)	
Yes	41 (63.1)	24 (36.9)	
History of HT			0.033
No	159 (61.2)	101 (38.8)	
Yes	60 (49.6)	61 (50.4)	
FH HT			0.19
No	115 (54.5)	96 (45.5)	
Yes	104 (61.2)	66 (38.8)	
FH kidney disease			0.184
No	189 (56.3)	147 (43.8)	
Yes	30 (66.7)	15 (33.3)	
History of stroke			0.524
No	187 (56.8)	142 (43.2)	
Yes	32 (61.5)	20 (38.5)	
History of heart disease			0.228
No	217 (57.9)	158 (42.1)	
Yes	2 (33.3)	4 (66.7)	
History family kidney disease			0.135
No	216 (57.1)	162 (42.9)	
Yes	3 (100.0)	0 (.0)	
History of MMS			0.307
No	169 (56.1)	132 (43.9)	
Yes	50 (62.5)	30 (37.5)	
History of joint disease			0.153
No	165 (55.6)	132 (44.4)	
Yes	54 (64.3)	30 (35.7)	

5.7 Distribution of hyperuricemia level by hypertensive grade status among hypertensive patients attending cardiac clinics in Dar es Salaam (N=381)

Table 5; shows the distribution of AU across hypertensive grade status. The distribution of uric acid (UA) levels across different grades of hypertension suggests a trend where higher UA levels are often associated with more severe grades of hypertension. Individuals with Grade 1 hypertension show a higher percentage of elevated UA status compared to those with normal or high-normal blood pressure. The p-value of 0.515 indicates that the difference in UA levels across the hypertensive grades is not statistically significant, suggesting that while there is a trend, it may not be strong enough to be considered definitive without further study.

Table 5, show the distribution, of hyperuricemia level by hypertensive grades status among hypertensive patients attending cardiac clinics in Dar es Salaam (N=381)

	UA status		
Variables	**Normal [N(%)]**	**High [N(%)]**	**p-value**
Hypertensive grade status			
Optimal	13 (50.0)	13 (50.0)	
Normal	19 (51.4)	18 (48.6)	
High normal	37 (63.8)	21 (36.2)	
Grade 1 hypertension	50 (65.8)	26 (34.2)	
Grade 2 hypertension	21 (51.2)	20 (48.8)	
Grade 3 hypertension	13 (56.5)	10 (43.5)	
Isolated systolic hypertension	66 (55.0)	54 (45.0)	0.515

5.8 The distribution of hyperuricemia with anti-hypertensive drugs used among hypertensive patients attending cardiac clinics in Dar es Salaam. (N=381)

Table 7, the bivariate analysis of antihypertensive drugs with hyperuricemia status were run, and there was a significant difference in hyperuricemia prevalence among users of different antihypertensive drugs. Diuretic users had a higher proportion of hyperuricemia (50.9%) compared to non-users (36.5%). Similarly, ARB users had a higher proportion of hyperuricemia (49.5%) compared to non-users (34.8%). Conversely, CCB users had a lower proportion of hyperuricemia (37.7%) compared to non-users (49.7%). There were no significant differences in hyperuricemia prevalence among users of ACE inhibitors (ACEI) and beta-blockers. Variables with a p-value less than 0.25 were considered for further analysis.

Table 6: Bivariate analyses of the distribution of hyperuricemia with anti-hypertensive drugs used among hypertensive patients attending cardiac clinics in Dar es Salaam.(N=381)

Variables	Normal UA	High UA	p value
Diuretics			0.005
No	141 (63.5)	81 (36.5)	
Yes	78 (49.1)	81 (50.9)	
CCB			0.021
No	77 (50.3)	76 (49.7)	
Yes	142 (62.3)	86 (37.7)	
ACEI			0.815
No	207 (57.3)	154 (42.7)	
Yes	12 (60.0)	8 (40.0)	
ARB			0.004
No	118 (65.2)	63 (34.8)	
Yes	101 (50.5)	99 (49.5)	
B—Blocker			0.326
No	200 (58.3)	143 (41.7)	
Yes	19 (50.0)	19 (50.0)	

5.9 Multivariable logistic regression for factors associated with hyperuricemia among hypertensive patients attending cardiac clinics in Dar es Salaam (N=381).

Participants aged 65 and older had a significantly higher likelihood of hyperuricemia compared to those under 45, with an adjusted odd ratio (aOR) of 2.613 (95% CI: 1.097—6.224, p = 0.03). Gender was also a significant factor, with females having a higher likelihood of hyperuricemia compared to males (aOR = 2.53, 95% CI: 1.55—4.128, p = 0.001). Additionally, alcohol consumption was associated with an increased likelihood of hyperuricemia (aOR = 1.884, 95% CI: 1.214—2.924, p = 0.005). Marital status did not show a significant association with hyperuricemia in this analysis.

a family history of hypertension (FH HT) was significantly associated with a lower likelihood of hyperuricemia (aOR = 0.642, 95% CI: 0.417—0.988, p = 0.044). Other clinical factors did not show significant associations with hyperuricemia.

The use of calcium channel blockers (CCBs) was significantly associated with a lower likelihood of hyperuricemia. Specifically, participants not using CCBs had a higher likelihood of hyperuricemia compared to those using CCBs (aOR = 1.749, 95% CI: 1.144—2.674, p = 0.01). Other hypertensive drug-related factors did not show significant associations with hyperuricemia.

Table 7: Multivariable logistic regression for factors associated with hyperuricemia among hypertensive patients attending cardiac clinics in Dar es Salaam (N=381).

Variable	95% AOR(Low-UP)	P value
Age group		
<45	Reference	1
45-54	1.236 (0.492—3.108)	0.652
55-64	2.035 (0.842—4.918)	0.115
65+	2.613 (1.097—6.224)	0.03
Gender		
Male	Reference	1
Female	2.53 (1.55—4.128)	0.001
Marital status		
Unmarried	Reference	1
Married	0.955 (0.611—1.492)	0.84
Alcohol consumptions		
No	Reference	1
Yes	1.884 (1.214—2.924)	0.005
BMI		
Underweight	Reference	1
Normal	0.837 (0.209 —3.349)	0.802
Overweight	0.724 (0.184 —2.85)	0.644
Obese	1.508 (0.377 —6.028)	0.561
Duration Since diagnosis		
< 4	Reference	1
4+	1.289 (0.818 —2.032)	0.274
History of HT		
No	Reference	1
Yes	1.461 (0.922 —2.315)	0.107
FH HT		
No	Reference	1
Yes	0.642 (0.417 —0.988)	0.044
FH kidney disease		
No	Reference	1
Yes	0.665 (0.334 —1.324)	0.245
History of heart disease		

CHAPTER SIX

DISCUSSION

The global burden of hypertension presents a significant challenge, impacting an estimated 1.20 billion people.[5] Our research examined the association between hypertension and hyperuricemia within the context of Dar-es-Salaam's cardiac clinics. Remarkably, we found a 42.5% prevalence of hyperuricemia among patients with primary hypertension, accompanied by notably higher serum uric acid levels. These findings underscore the complex interplay between these conditions and set the stage for a deeper discussion on their implications in public health and clinical practice.

The observed prevalence of hyperuricemia varies across different studies, with rates of 44% in Kenya,[46] and ranging from 31.8% to 46.9% in Nigeria, Cameroon, and South Asia. Variations in prevalence may be due to differences in dietary habits, genetic factors, and healthcare access. These findings underscore the importance of regular monitoring of uric acid levels in hypertensive patients to prevent potential complications such as cardiovascular disease and kidney dysfunction. The pathophysiology behind the elevation of serum uric acid in hypertensive patients is multifactorial. It is primarily attributed to reduced renal blood flow, which stimulates urate reabsorption. Hypertension-induced micro vascular disease can also cause tissue ischemia, leading to increased uric acid synthesis.[26] This is further exacerbated by ischemia-driven degradation of ATP to adenine and xanthine, alongside an upsurge in xanthine oxidase activity, culminating in heightened uric acid production and oxidant formation.

Older participants, particularly those aged 65 and above, were more likely to have hyperuricemia compared to younger participants. This finding aligns with other studies that have reported an increased prevalence of hyperuricemia with advancing age, likely due to age-related declines in renal function and increased comorbidities such as hypertension and diabetes.[37] For instance, a study in Korea found similar trends, where older adults had higher uric acid levels compared to younger cohorts. [40] The reasons for this association may include reduced renal clearance of uric acid, increased use of medications that affect uric acid metabolism, and lifestyle factors. The implication of this result is significant for clinical practice, as it suggests that older hypertensive patients should be regularly screened for hyperuricemia to manage and mitigate potential complications such as gout and cardiovascular diseases.

Females in this study were found to have a higher likelihood of hyperuricemia compared to males. The prevalence of females (47.9%) was higher than males (31.1%) in the development of HUA with a statistically significant association ($p = 0.02$). However, contrasting data from Kenya[46] and Seychelles[44] suggest that prevalence rates can significantly vary by region and demographic factors.[39,42]The result of this study is consistent with other research indicating that postmenopausal women are at increased risk for hyperuricemia due to hormonal changes that affect uric acid metabolism. For example, studies have shown that estrogen has a uricosuric effect, which diminishes after menopause, leading to higher uric acid levels in women.[36] Additionally, lifestyle factors and comorbidities such as obesity and hypertension, which are more prevalent in older women, may contribute to this increased risk.[9] The clinical implication of this finding is that healthcare providers

should be particularly vigilant in monitoring uric acid levels in female hypertensive patients, especially those who are postmenopausal, to prevent complications such as gout and cardiovascular diseases.

Alcohol consumption was associated with an increased likelihood of hyperuricemia in this study. This finding suggests that alcohol intake may contribute to elevated uric acid levels, potentially due to the metabolism of alcohol producing uric acid and reducing its excretion. Similar associations have been observed in other studies, such as research conducted in the United States and Japan, which found that alcohol consumption, particularly beer, is linked to hyperuricemia.[20,27,51,52] The reasons for this association include the purine content in alcoholic beverages and the effect of alcohol on renal function. Clinically, this underscores the importance of advising hypertensive patients to limit alcohol intake to manage and prevent hyperuricemia and its complications.

The finding that Body Mass Index (BMI) was not a statistically significant predictor of hyperuricemia in multivariable logistic regression, despite its significance in bivariate analysis, suggests a complex relationship between BMI and hyperuricemia. This discrepancy may arise due to the presence of confounding variables that are accounted for in multivariable analysis but not in bivariate analysis. It's important to consider that while BMI can be an indicator of health status, its role as a predictor of specific conditions like hyperuricemia may vary depending on the interplay with other factors. The positive association between BMI and hyperuricemia levels was confirmed by Pawe Muszyn´ ski et al,2023 in Poland revealed over 80%(n= 705) of the study group had a BMI > 25 kg/m2[58].Also study done in Kenya found there was

significant association between hyperuricemia and BMI (p=0.033).This can be explained by increased production of uric acid. This could be a result of dietary changes and sedentary lifestyles among the population. Adipose tissue in obese individuals produces more uric acid due to increased turnover of nucleotides, which are broken down into uric acid. Also, Obesity may give rise to insulin resistance, even in individuals with normal glucose tolerance tests. Insulin resistance is known to be inversely related to 24 hours of urinary uric acid clearance[59,60]

The study indicates that socioeconomic factors such as income and employment status do not significantly influence serum uric acid levels, aligning with the findings from a Chinese study that highlighted a lack of significant health literacy among asymptomatic hyperuricemia patients.[61] This suggests that while socioeconomic position correlates with serum uric acid levels, it is health literacy that may play a more critical role in managing these levels. Enhancing health literacy could therefore be a key strategy in improving self-management for patients with asymptomatic hyperuricemia, potentially mitigating the risks associated with elevated serum uric acid.

Blood pressure categories did not show significant associations with serum uric acid levels, as evidenced by their respective p-values =0.515. Similar to the study done in Kenya by et al did not establish an association between blood pressure and hyperuricemia. Baseline serum uric acid levels were not a predictor of future changes in both systolic and diastolic blood pressure. This indicates that rising serum uric acid levels do not negatively impact blood pressure control in adults who are already receiving treatment for hypertension. However study done in Egypt by

Tomader Taha, Abdel Rahman, et al, was found hyperuricemia was significantly and positively associated with systolic blood pressure and diastolic blood pressure[62]. This increase in blood pressure (BP) in hyperuricemia is brought about by several mechanisms. A few worthy mentions are endothelial dysfunction, caused by the decreased production of nitric oxide (NO),[63] systemic inflammation due to lipid oxidation and enhanced platelet aggregation, and expression of the C-reactive protein within the vascular endothelium.[64]The activation of the renin-angiotensin system and vascular smooth muscle proliferation a other important factors that induce hypertension.

A family history of hypertension was associated with a lower likelihood of hyperuricemia in this study. This finding is intriguing and may suggest a potential protective genetic or environmental factor within families with a history of hypertension. It contrasts with some studies that have found a positive association between family history of hypertension and hyperuricemia, possibly due to shared lifestyle factors or genetic predispositions.[23,28,31] The lower likelihood observed in this study could be due to better awareness and management of hypertension within these families, leading to healthier lifestyles and better control of risk factors associated with hyperuricemia. Clinically, this highlights the importance of considering family history in the assessment and management of hyperuricemia risk among hypertensive patients.

The use of calcium channel blockers (CCBs) was linked to a lower likelihood of hyperuricemia, with non-users being more likely to have elevated uric acid levels compared to users. This suggests that CCBs may have a beneficial effect on uric

acid metabolism, possibly by enhancing renal excretion of uric acid. This finding aligns with previous research indicating similar benefits of CCBs on uric acid levels.[65] In contrast, other antihypertensive drugs, such as diuretics, are known to increase uric acid levels.[65] The implication of this finding is significant for clinical practice, suggesting that CCBs might be a preferable choice for hypertensive patients at risk of hyperuricemia. This could guide clinicians in selecting antihypertensive therapy that not only controls blood pressure but also minimizes the risk of hyperuricemia and its associated complications.

CHAPTER SEVEN

STRENGTH AND LIMITATION OF STUDY

A key strength of the study results discussed is the comprehensive identification of significant sociodemographic and clinical factors associated with hyperuricemia among hypertensive patients. This includes the detailed analysis of age, gender, alcohol consumption, family history of hypertension, and the use of calcium channel blockers. By highlighting these associations, the study provides valuable insights that can inform targeted interventions and personalized treatment strategies, ultimately improving patient outcomes and guiding clinical practice in managing hyperuricemia in the hypertensive population.

There are several potential limitations to this study. First, the cross-sectional design limits the ability to establish causality between hyperuricemia and the identified risk factors. Second, the reliance on self-reported data for some variables, such as alcohol consumption and family medical history, may introduce recall bias. Third, the study population is limited to hypertensive patients attending cardiac clinics in Dar es Salaam, which may not be representative of the broader hypertensive population. Finally, the study did not account for dietary factors, which can significantly influence uric acid levels, potentially confounding the results. These limitations should be considered when interpreting the findings and planning future research.

CHAPTER EIGHT

CONCLUSION AND RECOMMENDATION

8.1 Conclusion

This study identified that for every two hypertensive patients, one is likely to have hyperuricemia. Several factors were significantly associated with hyperuricemia: participants aged 65 and older had a higher likelihood compared to those under 45, females were more likely to have hyperuricemia than males, and alcohol consumption increased the likelihood. Clinically, a family history of hypertension was associated with a lower likelihood of hyperuricemia. Regarding hypertensive medications, the use of calcium channel blockers (CCBs) was linked to a lower likelihood of hyperuricemia, with non-users having a higher likelihood compared to users. These findings underscore the importance of considering sociodemographic, clinical, and medication-related factors in managing hyperuricemia among hypertensive patients.

8.2 Recommendation

1. **Regular Monitoring**: Older adults, females, and individuals who consume alcohol should be regularly monitored for hyperuricemia, given their higher risk identified in this study. This can help in early detection and management of elevated uric acid levels.
2. **Lifestyle Interventions**: Patients should be advised to limit alcohol consumption as part of their hypertension management plan to reduce the risk of hyperuricemia. Additionally, promoting healthy lifestyle choices can help mitigate other risk factors.

3. **Medication Management**: Clinicians should consider prescribing calcium channel blockers for hypertensive patients at risk of hyperuricemia, as these medications were associated with a lower likelihood of elevated uric acid levels. This approach can help in managing both hypertension and hyperuricemia effectively.

4. **Public Health Strategies**: Public health initiatives should focus on educating the population about the risks of hyperuricemia and its management, particularly targeting high-risk groups identified in this study.

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APPENDICES

APPENDIX I: RESEARCH DATA COLLECTION INSTRUMENTS.

SPECTRUM OF HYPERURICEMIA AMONG HYPERTENSIVE PATIENTS ATTENDING CARDIAC CLINICS IN DAR-ES-SALAAM

DODOSO NA .....

NO.	QUESTIONS	ANSWER
1.	Study Site	Amana.....1
		Temeke.....2
		Mwananyamala.....3
2.	Residence	
3.	File No	

A. SOCIAL-DEMOGRAPHIC DATA

4.	AGE	YEARS.....
5.	Gender _	MALE.....1
		FEMALE.....2
6.	Marital status	Single.....1
		Married.....2
		Widowed.....3
		Divorced.....4
		Cohabiting.....5

7. Income level _____

8.Occupation

Unemployed.....3

Self-employment/Business.....4

Retired.....5

9. Education

No formal education.....0

Primary Education.....1

Secondary education.....2

College.....3

University.....4

B. MEDICAL HISTORY

10. Duration since Diagnosis of Hypertension

Years.....

11 H/O Hypertension

Yes 1

No 0

12. H/O Stroke

Yes 1

No 0

Yes 1

13. H/O Ischemic Heart Disease No 0

Yes 1

14. H/O Diabetes No 0

Yes 1

15. H/O Kidney disease No 0

Yes 1

16. H/O MSS disease No 0

17. H/O Joint disease

C. FAMILY & SOCIAL

18. History of use of alcohol consumption Yes 1

No 0

19. History of Cigarette smoking Yes 1

No 0

Yes 1

20 History of hypertension in the family No 0

Yes 1

21 History of kidney disease in the family No 0

D.TREATMENT HISTORY

Hypertension drugs [Circle appropriate]

22. DIURETIC Used.....1

Not used.....0

23. Type of diuretics

24. CCB Used.....1

Not used.....0

25. Type of CCB

26. ACEI Used.....1

Not used.....0

27. Type of ACEI

28. ARB Used.....1

Not used.....0

29. Type of ARB

30. B-Blocker Used.....1

Not used.....0

31. Type of B-Blocker

32. Others Used.....1

Not used.....0

33 Specify

E. PHYSICAL EXAMINATION

ANTHROPOMETRICS MEASUREMENTS

34. Height ……CM

35. Weight ……..KG

36. Waist circumference ……CM

37. BMI ……Kg/M ^{2}

F. BLOOD PRESSURE MEASUREMENTS

38 SYSTOLIC BP …… mmHg

39 DIASTOLICBP …… mmHg

40 PULSE RATE ……b/min

G.BIOCHEMISTRY

41 Serum Uric Acid ……µmol/L

MFUMO WA KUHUSISHA HYPERURICEMIA NA WAGONJWA WA SHINIKIZO LA DAMU WANAODHURIA KLINIKI ZA MAGONJWA YA MOYO DAR-ES SALAAM

Namba……..

NA	**SWALI**	**MAJIBU (ZUNGUSHIADUARA)AU JAZA SEHEMU HUSIKA**
1.	Eneo ya utafiti	Amana……..1
		Temeke……2
		Mwananyamala……3
2.	Makazi	
3.	Faili Na.	
DATA YA IDADI YA WATU		
4.	Umri	Miaka……..
5.	Jinsia	MME……..1
		MKE……2
6.	Hali ya Ndoa	Hajaoa/Hajaolewa……1
		Kuoa / kuolewa ……..2
		Mjane/Mgane ……3
		Talaka ……4
		Kuishi pamoja bila ndoa……..5
7.	Kipato	Kidogo……1
		Cha kati……2
		Kikubwa……3

	8.Kazi	Ajira ya muda.....1
		Ajira ya kudumu.....2
		Ukosefu wa Ajira.....3
		Kujiajiri/Biashara..... 4
		Mstaafu 5
9.	Aina ya Elimu	Elimu isiyo rasmi.....1
		Elimu rasmi.....2
10.	Kama ni elimu rasmi,	Msingi.....1
	kiwango cha elimu ni	Sekondari.....2
	kipi?	Chuo.....3
		Chuo Kikuu.....4

B. HISTORIA YA MGONJWA

11.	Muda tangu Utambuzi wa Shinikizo la damu,	Miaka.....
12.	Historia ya shindikizo la damu	Ndiyo 1
		Hapana.....0
13.	Historia ya kiharusi	Ndiyo 1
		Hapana.....0
14.	Historia ya magonjwa ya moyo	Ndiyo 1
		Hapana.....0
15.	Historia ya kisukari	Ndiyo 1
		Hapana.....0

16.	Historia ya magonjwa ya figo	Ndiyo .....1 Hapana.....0
17.	Historia ya magonjwa ya mifupa	Ndiyo .....1 Hapana.....0
18.	Historia ya magonjwa ya viungo	Ndiyo .....1 Hapana.....0

C,HISTORIA YA FAMILIA NA BINAFSI

19.	Historia ya matumizi ya pombe	Ndiyo .....1 Hapana.....0
20.	Kama ndiyo swali la 18 ni kwa muda gani?	Miaka.....
21.	Kama ndiyo swali la18, Kiasi gani kila siku	Kila siku.....1 Kila wiki.....2 Mara chache.....3
22.	Historia ya uvutaji sigara	Ndiyo .....1 Hapana.....0
23.	Kama ndiyo swali la 21, Kiasi gani kila siku	Chini ya pakiti 1.....1 Pakiti1-2.....2 Zaidi ya pakiti 2.....3
24.	Historia ya shindikizo la damu kwenye familia	Ndiyo .....1 Hapana.....0
25.	Historia ya magonjwa ya figo kwenye familia	Ndiyo .....1 Hapana.....0

D. HISTORIA YA MATIBABU

Dawa za shinikizo la damu (zungushia duara anayotumia)

26. Diuretics Natumia.....1

Situmii 0

27. Aina gani ya Diuretics?

28. CCB Natumia.....1

Situmii 0

29. Aina gani ya CCB?

30. ACEI Natumia.....1

Situmii 0

31. Aina gani ya ACEI?

32. ARB Natumia.....1

Situmii 0

33. Aina gani ya ARB?

34. B-Blocker

35. Aina gani ya B-Blocker?

36. Dawa nyinginezo Natumia.....1

Situmii 0

37. Taja

E. **UCHUNGUZI WA KIMWILI, VIPIMO VYA ANTHROPOMETRIC**

38. UREFU Sentimita……

39. UZITO Kilogram…….

40. KIUNO Sentimita……..

41. BMI Kg/M ^{2}

F. VIPIMO VYA SHINIKIZO LA DAMU

42. Shindikizo la damu BP - Systoliki ……..mmHg

43. Shindikizo la damu BP- Diastoliki ……mmHg

44. Mapigo ya moyo (pigo/dakika) ……b/min

G. VIPIMO VYA BIOKEMIA

45. Seramu yuriki acidi …… µmol/L

APPENDIX III: CONSENT FORM [ENGLISH VERSION]

I, Dr CELLINA JOSEPH KAUPUNDA a 3rd year resident at Hubert Kairuki Memorial University, currently pursuing Master of Medicine in Internal Medicine will be conducting a study as part of partial fulfillment of the requirements for the Degree of Master of Medicine.

Titled; SPECTRUM OF HYPERURICEMIA AMONG HYPERTENSIVE PATIENTS ATTENDING CARDIAC CLINICS IN DAR-ES-SALAAM

I would like to recruit you into my research which is to study if hypertensive patients could have high serum uric acid levels. Thosehypertensive who will meet the inclusion criteria will be recruited into the study and will be interviewed using a questionnaire, which will include their social demographic characteristics and physical examination.

For us to know whether you have hyperuricemia, we will request you to undergo some tests preceding which you shall have to answer a few questions on your pertinent.

Medical history, We will take a blood sample (2mls) to check whether you have high uric acid level.

We will keep all your test results in confidence and keep you informed of the results.

No fee will be required from the participant for the study.

In case of any queries the following may be contacted;

Prof Fredrick Kaijage, Chairperson of the Ethical Review Committee (+255 767 306 421)

Prof Y. Mgonda, Chairperson of Internal Medicine Department

Dr Cellina J. Kaupunda, Post-graduate student, Department of Internal Medicine

Director of Postgraduate Studies and Research Institute, HKMU

Participant declaration

I have read/been explained the contents of this form and understood its purpose; hence, I am willing to participate and give my full cooperation in the study.

Sign: ……

Name: ……

Date: ……

APPENDIX IV: FOMU YA IDHINI [SWAHILI VERSION]

Mimi CELLINA JOSEPH KAUPUNDA. Ni mwanafunzi wa shahada ya uzamili ya magonjwa ya ndani katika chuo kikuu cha Kumbukumbu ya Hubert Kairuki. Nina fanya utafiti kama sehemu ya mahitaji kwa ajili ya kukamilisha shahada hii. Nimekuona leo kwa sababu ninafanya uchunguzi kujua kama watu wazima walio na ugonjwa wa shinikizo la damu huenda wakawa na hyperuricemia.

Mada ya utafiti:- SPECTRUM OF HYPERURICEMIA AMONG HYPERTENSIVE PATIENTS ATTENDING CARDIAC CLINICS IN DAR-ES-SALAAM

Shinikizo la damu kuhusishwa na kuongezeka kwa uric acid kwenye damu, ambayo inaweza

Kusababisha ugonjwa wa figo, ugonjwa wa moyo na kiharusi. Ili kufanya uchunguzi huu. Tutakuuliza maswali kuhusu magonjwa yoyote mengine unayoweza kuwa nayo au unayotibiwa, historia ya jamii yako, madawa yoyote unayotumia na kisha tutakupima Ilitujue kama unano ugonjwa huu wa hyperuricemia na shindikizo la dawa. Tutapima damu ilitujue kiwango ya uric acid kwenye mwili. Tutayaweka matokeo yako kwa njia ya kuheshimu haki yako. Tutakujulisha kuhusu matokeo yako na maana kwa afya yako.

Hakuna ada itakayohitajika kutoka kwa mshiriki kwa ajili ya utafiti.

Mshiriki wa utafiti anaruhusiwa kujiondoa katika utafiti wakati wowote anaotaka.

Ikiwa kuna maswali yoyote wafuatayo wanaweza kuwasiliana;

Prof Fredrick Kaijage, Mwenyekiti wa Kamati ya Mapitio ya Maadili (+255 767 306 421)

Prof Y. Mgonda, Mkuu wa Idara, Magonjwa ya ndani, HKMU

Dr Cellina J Kaupunda, Mwanafunzi wa shahada ya uzamili, Idara ya Magonjwa ya Ndani, HKMU

Mkurugenzi wa utafiti, HKMU

Tamko la mshiriki:

Nimesoma /nimeelezwa ipasavyo yaliyomo katika fomu hii na kuelewa madhumuni yake, hivyo nipo tayari kushiriki na kutoa ushirikiano wangu kamili katika utafiti.

Sahihi: ……..……..……

Jina: ….……..……..……

Tarehe……

Appendix V: Kairuki University ethical clearance certificate for conducting

HUBERT KAIRUKI MEMORIAL UNIVERSITY,
P.O.BOX 65300,
DAR ES SALAAM
Email: cellinakaupunda@gmail.com
PHONE NO: 0754572716
27th FEBRUARY 2024

CHAIRPERSON,
INSTITUTIONAL RESEARCH AND ETHICS COMMITTEE,
HUBERT KAIRUKI MEMORIAL UNIVERSITY,
P.O.BOX 65300
DAR ES SALAAM.

U.F.S
CHAIRPERSON, INTERNAL MEDICINE DEPARTMENT
HKMU.

Forwarded for consideration
27/02/2024

Dear Sir/Madam,

REF: ETHICAL CLEARANCE FOR DISSERTATION RESEARCH WORK.

Kindly refer to the heading above. I am Celina Joseph Kaupunda, an MMed year III student with registration number HK/PG/IM/21/0016 from Internal Medicine Department. I am about to start collecting data for the dissertation research work titled **"Hyperuricemia among primary hypertensive patients attending cardiac clinic in Dar es salaam".** I therefore, kindly request to be given ethical clearance for this important activity.

Attached is my proposal for the mentioned activity.

I hope my request will be considered.

Yours faithfully,

Kaupunda.
.....
Celina J. Kaupunda.

KAIRUKI UNIVERSITY (KU)

70 Chwaku Street
Regent Estate – Mikocheni
P.O. Box 65300,
Dar es Salaam
Tanzania

Tel: +255-22-2700021/4
Email: secvc@ku.ac.tz
Website: www.ku.ac.tz

REF: KU/PT/30.5/474

8 May 2024

Medical Officer In-charge,
Amana Regional Referral Hospital,
Dar es Salaam.

Re: Letter of introduction Dr. CELINA KAUPUNDA (MMED Part 2 – Internal Medicine)

The above named is a MMED postgraduate student specialising in Internal Medicine. As part of fulfilling her MMED programme, she plans to undertake a study titled **"HYPERURICEMIA AMONG PRIMARY HYPERTENSIVE PATIENTS ATTENDING CARDIAC CLINICS IN DAR ES SALAAM"**. This study was reviewed and has been granted with an ethics approval No. **KU/IREC/27.10/444** by the KU Institutional Research Ethics Committee that will be valid for one year with effect from 2nd May 2024.

This letter serves to introduce **Dr. CELINA KAUPUNDA** who will be conducting her study in Dar es Salaam, please accord her with the needed support.
Thank you for your support and cooperation in developing human resources for health in our country.

Regards,

DIRECTOR POSTGRADUATE STUDIES
HUBERT KAIRUKI MEMORIAL UNIVERSITY
P. O. Box 65300, DSM
AND RESEARCH INSTITUTE

Professor Columba Mbekenga, PhD
Director Postgraduate Studies & Research Institute

c. c. Dr. Warles Charles, Ag. Dean, Faculty of Medicine, KU
c. c. Prof. Yassin Mgonda, Chair, Department of Internal Medicine, KU.

General Contact:
Email: info@ku.ac.tz
Contact: +255 659 371 234

Academic:
Email: dvc-academics@ku.ac.tz
Contact: +255 716 999 151

Admissions:
Email: admissions@ku.ac.tz
Contact: +255 769 724 636
+255 659 371 234

Appendix VI: Permission for data collection from Regional referral hospital in Dar es Salaam.

THE UNITED REPUBLIC OF TANZANIA
MINISTRY OF HEALTH

AMANA REGIONAL REFERRAL HOSPITAL

Telegram "HEALTH", DODOMA
Phone No.: +255 026 – 2323267
Email: ps@afya.go.tz

P.O. Box 25411
DAR ES SALAAM
Phone: 022—2861903

REF. NO. MoHCDGEC/ARRH/R.1/VOL II/35

Date: 09/05/2024

Director Postgraduate Studies and
Research Institution,
HKMU,
P.O. B ox 65300,
DAR ES SALAAM.

Re: PERMISSION FOR DATA COLLECTION

Refer to your letter dated 08th May, 2024 which requested us to allow **Dr Celina Kaupunda** to conduct research and collect data in our institution.

We are here to acknowledge your request with the following conditions, that you must submit the results of your research after completion of analysis in order the hospital to make use of data's to solve hospital problems.

Regards.

FOR: MEDICAL OFFICER I/C
AMANA REGIONAL REFERRAL HOSPITAL
P. O. Box 25411
DAR ES SALAAM

Dr. Rose Ntambuto
FOR: MEDICAL OFFICER INCHARGE
AMANA REGIONAL REFERRAL HOSPITAL

JAMHURI YA MUUNGANO WA TANZANIA
WIZARA YA AFYA.
HOSPITAL YA RUFAA YA MKOA YA TEMEKE
Barua pepe:temekerh@afya.go.tz,S.L.P 45232 Dar es Salaam,Simu 0222850007

Kumb. Na. TRRH/RSC/9/10/01/17 **Tarehe: 13/05/2024**

Dkt. Celina Kaupunda
Kaituki University
S.L.P 65300
DAR ES SALAAM.

YAH: OMBI LA KUFANYA UTAFITI "HYPERURICEMIA AMONG PRIMARY HYPERTENSIVE PATIENTS ATTENDING CARDIAC CLINICS IN DAR ES SALAAM." (RESEARCH)

Tafadhali husika na somo tajwa hapo juu.

2. Nimepokea barua yako ya tarehe 08 Mayi, 2024 kuhusu ombi lako la kufanya Utafiti (Research) katika Taasisi yetu, kuhusu **"hyperuricemia among primary hypertensive patients attending cardiac clinics in Dar es salaam."**

3. Ombi lako limekubaliwa, utatakiwa kulipa ada kiasi cha **Tshs. 100,000/=**. Hivyo wasiliana na mhasibu wa mapato wa Hospitali **Ndg. Lusajo Nsajigwa** kwa namba **0717 959495** ili akupatie control Number kwa ajili ya malipo ya ada hii ili uweze kuruhusiwa kufanya utafiti.

4. Asante kwa ushirikiano.

Dkt. Husna Msangi
Kny: MKURUGENZI
HOSPITALI YA RUFAA YA MKOA YA TEMEKE

Nakala: CSCO/INTERNAL MEDICINE - ***Tafadhali hakikisha taarifa ya utafiti inabaki hospitalini***

THE UNITED REPUBLIC OF TANZANIA
MINISTRY OF HEALTH

Telephone Address:

Telephone 022-2760500

Mwananyamala Regional
Referral Hospital,
P.O.Box 61665
Dar es Salaam

RE: NO: MA. 59/240/01/37

DATE: 14th May,2024

Director,
Hurbert Kairuki Memorial University,
P.O.BOX 65300,
DAR ES SALAAM.

RE: DR. CELINA KAUPUNDA - TO CONDUCTI HIS RESEARCH IN MWANANYAMALA REGIONAL REFERRAL HOSPITAL

The captioned subject refers

2. May you be informed that your request to research Titled "***Hyperuricemia among primary hypertensive patients attending cardiac clinics in Dar es Salaam, Tanzania***" ***Start*** **to 2th May,2024,** is asserted.

3. The Institution charges **50,000/=**, as Research fee as per student spent. The payments are to be made upon reporting.

4. May she report to the Administration and HR department head for further instruction.

Thanks.

For: MEDICAL OFFICER INCHARGE
MwananyamalaRegionalReferralHospital
P. O. Box 61665
DAR-ES-SALAAM

Atugonza Kyaruzi
RESEARCH COORDINATOR
FOR: MEDICAL OFFICER INCHARGE
MWANANYAMALA REGIONAL REFERRAL HOSPITAL

COPY:
Heads of Internal Medicine Department - **MWANANYAMALA REGIONAL REFERRAL HOSPITA**

Appendix VII: Plagiarism check score from TURNIT IN