ASSESSMENT OF INFLAMMATORY AND OXIDATIVE STRESS MARKERS IN HYPERTENSIVE PATIENTS ATTENDING THOMAS ADEWUMI UNIVERSITY TEACHING HOSPITAL, OMUARAN KWARASTATE.

BY

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ABSTRACT

Hypertension is the leading risk factor for stroke, heart failure, among others which contribute greatly to morbidity and mortality worldwide. It has been projected that 1.28 billion adults aged 18 years and older are hypertensive and a higher burden in lower middle income countries such as Nigeria. This study aims at investigating the association between inflammatory and oxidative stress biomarkers with hypertension severity among patients at Thomas Adewumi University Teaching Hospital. A case-control design was employed, involving 100 participants,50 hypertensive patients (cases) and 50 normotensive individuals (controls) aged 18 to 65 years. Data collection included use of structured questionnaires, and laboratory analyses of inflammatory markers, oxidative stress markers, and albumin, levels. Laboratory tests were conducted using ELISA and spectrophotometric methods, and data were analyzed with SPSS v25. Results revealed that hsCRP and TAC levels significantly differed between hypertensive and normotensive groups ($p < 0.001$). Among hypertensives, hsCRP levels showed a strong positive correlation with hypertension stage ($\rho = 0.565$, $p < 0.001$), while no significant correlation was found for MDA, TAC, globulin, or albumin. Regression analyses indicated that age, male gender, smoking, and sugary snack intake were significant predictors of elevated hsCRP ($R^2 = 0.344$, $p < 0.001$). Multivariate analysis identified age, smoking, sugary food consumption, hsCRP, and TAC as independent predictors of hypertension severity ($R^2 = 0.781$, $p < 0.001$). The findings suggest a substantial link between systemic inflammation and hypertension progression, particularly marked by elevated hsCRP. The compensatory rise in TAC may reflect the body's adaptive response to oxidative stress. These insights highlight the potential of using inflammatory and oxidative biomarkers as adjunct diagnostic and prognostic tools in hypertension management.
Keywords: hsCRP, TAC, MDA, Hypertensive, Normotensive, Inflammatory markers, Oxidative markers.

DECLARATION

This is to declare that this research project titled Assessment of Inflammatory and Oxidative Stress Markers in Hypertensive Patient Attending Thomas Adewumi Teaching Hospital, Omuaran Kwara state carried out by me David Olabisi Joshua, in the Department of Medical Laboratory Science, Faculty of Basic Medical and Health Sciences, Thomas Adewumi University Oko-Irese, Kwara State, is solely the result of my work except where acknowledged as being derived from other person(s) work or resources

05-08-2025

Signature: _____ **Date:** _____

CERTIFICATION

I declare that this project report is my original work and has not been previously submitted to any other institution of higher learning.

I further certify that all the sources cited or quoted are duly acknowledge by means of comprehensive list of references.

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DEDICATION

I dedicate this project to God Almighty my creator, my strong pillar, my source of inspiration, wisdom, knowledge and understanding. I also dedicate this work to my parents; Late Dr. and Mrs. David for supporting me all the way and their support has made sure that I give it all it takes to finish that which I have started. To all my siblings, who have brought me to where I am today. Thank you. My love for you all can never be quantified. God bless you.

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER ONE

INTRODUCTION

1.0 Background of the study

High rise in blood pressure also called hypertension is a public health problem worldwide. It has been projected that 1.28 billion adults aged 18 years and older are hypertensive and a higher burden in lower middle income countries such as Nigeria (WHO, 2021). Only about (54%) of people with hypertension have been diagnosed with it; only 42% are on treatment and only one out of five people have their hypertension well controlled (WHO, 2023). Statistics like these calls for improved education on how to live with such condition. Hypertension is the leading risk factor for stroke, heart failure, chronic kidney disease among others which contribute greatly to morbidity and mortality worldwide. Research findings have postulated that hypertension is a complex disease which arises from multiple determinants such as genetic predisposition, lifestyle and environmental factors. Over the years, research has shown that inflammation as well as other factors have been noted to be crucial in supporting this disease (Forrester *et al.,* 2018). Inflammation as seen by increase in levels of high sensitivity C- reactive protein (hsCRP) alongside pro inflammatory cytokines like Interleukin-6 (IL-6) make the situation worse by causing vascular injury hence leading to hypertension. Similarly, to this concept is oxidative stress which occurs when there is an imbalance between pro-oxidants and antioxidants, thereby leading to production of some reactive oxygen species (ROS) which further worsen vascular processes leading to high blood pressure (Harrison *et al.,* 2021). Therefore, assessing inflammatory and oxidative stress markers in people with high blood pressure gives an idea of how the disease worsens and points to possible treatment which goes beyond just numbers. Such studies are especially required in settings like Thomas Adewumi university Teaching Hospital

Omu Aran because there is little information available about these markers in hypertensive patients.

1.1 Statement of the Problem

Although doctors routinely attempt to reduce blood pressure in people with hypertension, the hidden role that inflammation and oxidative stress play in this condition rarely get the same attention. Drugs like angiotensin converting enzyme inhibitors (ACEIs) and beta blockers can knock those numbers down nicely, but they can also miss the chronic irritation and free-radical damage that wear on blood vessels and push the disease over the edge (Schiffrin *et al.,* 2020). There is virtually no local research that has looked at these inflammatory and oxidative markers in hypertensive patients. This creates a huge gap in our knowledge. Without this localized data, clinicians may be less likely to appreciate how these forces play out in each individual illness and may therefore pass on a more personalized plan which goes beyond just treatment, including the biomarker tests. This study aims at assessing these markers at Thomas Adewumi university Teaching Hospital and see what light they can shed on patient care (Adeloye *et al.,* 2019).

1.2 Justification of the study

This study is so vital because the study is focused on a poorly studied area of management of hypertension as well as inflammation and oxidative stress. Doctors treat high blood pressure by giving drugs that only reduce blood pressure and this method does not consider the silent inflammatory and oxidative process that causes high blood pressure (Harrison *et al.,* 2021). But if researchers consider measuring things like hsCRP and malondialdehyde (MDA), they might be able to understand how the above-mentioned processes cause the disease in hypertensive patients 18 years and above.

A positive result from the result of the experiment will then make clinicians incorporate assessment of biomarkers into their standard work-up and the care of patients will move from a

one-fit-all to a more personalized care, and so early identification of elevated inflammatory and oxidative biomarkers will be like adding another red light that will warn doctors and patients of an elevated risk of complications and with time take preventive measures that will make their health improve in the long run (Forrester *et al.,* 2018).

1.3 Research Aim and Objectives.

The aim of this study is to determine the level of inflammatory and oxidative stress markers among hypertensive patients 18 years and above in Thomas Adewumi university Teaching Hospital.

Objectives:

1.To determine the blood pressure of the study participants.

2.To determine the level of inflammatory markers (hsCRP and Albumin) among hypertensive patients.

3.To determine the level of oxidative stress markers (MDA and TAC) among hypertensive patients.

4.To evaluate the effects of lifestyle factors (such as diet, physical activity, smoking) on the level of these biomarkers.

1.4 Research questions.

1. What are the level of inflammatory and oxidative stress markers among hypertensive patients 18 years and above in Thomas Adewumi university Teaching Hospital?

2. How do these biomarkers correlate with the severity of hypertension?

3. What is the effect of lifestyle factors on the level of these biomarkers among hypertensive patients?

4. How does the differences in the level of inflammatory markers and oxidative stress marker improves hypertension management strategies.

1.5 Significance of the Study.

This study will show precisely how inflammation and oxidative stress markers appear in hypertensive patients. By looking at patients at Thomas Adewumi university Teaching Hospital, this study will provide information gotten from the study which will help clinicians treat patients more according to what they need. Furthermore, the results might help to understand how biomarkers like hsCRP and MDA could be followed and steered changes in medication or life style (Griendling *et al.,* 2016).

Moreover, the results of this research work could help to formulate public health plans looking for easy and non-invasive tests so hypertension is found and treated early on, which would go a long way in reducing the long-term risks of cardiovascular diseases among hypertensive patients (Munzel *et al.,* 2017).

Research hypothesis:

- **Null Hypothesis (H_0):** There is no significant difference in the levels of inflammatory (hsCRP, Albumin) and oxidative stress markers (MDA, TAC) between hypertensive patients and normotensive individuals attending Thomas Adewumi University Teaching Hospital.
- **Alternative Hypothesis (H_1):** There is a significant difference in the levels of inflammatory (hsCRP, Albumin) and oxidative stress markers (MDA, TAC) between hypertensive patients and normotensive individuals attending Thomas Adewumi University Teaching Hospital.

1.6 Scope of the Study.

The study was carried out on hypertensive patients of 18 years of age and above who presents at Thomas Adewumi university Teaching Hospital for measurement of high sensitivity C- reactive protein (hsCRP), malondialdehyde (MDA) and Total antioxidant capacity (TAC) and their

correlation with the severity of hypertensive disease will be determined. The effect of lifestyle which could induce these variables, which includes diet, physical activities will also be assessed.

CHAPTER TWO

LITERATURE REVIEW

2.1 Hypertension.

Hypertension, also known as high blood pressure, is one of the major preventable health problem that leads to the burden of cardiovascular diseases around the world. According to the American College of Cardiology and American Heart Association, as it is defined now, hypertension settles in when there is persistent rise in the systolic blood pressure (SBP) at or above 140 mmHg, or when diastolic pressure stays at or above 90 mmHg, a threshold which has been years in the making through research (Whelton *et al.,* 2019). When blood pressure stays high for long, the organs such as the heart, brain, kidneys and blood vessels experience an invisible and cumulative damage which eventually leads to life-threatening events such as heart attacks, strokes, heart failure and chronic renal failure (Boehm *et al.,* 2020).

Hypertension can be primary or (essential hypertension) and secondary hypertension. Primary hypertension accounts for nine out of ten cases and develops slowly over many years; it has no single cause, but genes, diet, lack of exercise and stress are thought to play a role. Secondary hypertension, which accounts for 5-10% of cases, occurs as a consequence of other medical conditions, such as renal disease, endocrine disorders, or use of certain medications, corticosteroids, nonsteroidal anti-inflammatory drugs (NSAIDs) and oral contraceptives (Chrysant and Chrysant, 2020).

2.1.1 Prevalence and Impact of hypertension around the world.

1.28 billion adults worldwide live with hypertension, and 2 out of 3 of these live in low- and middle-income countries (WHO, 2021). Because it shows little to no symptoms for a long time, health professionals refer to it as the "silent killer". That lack of awareness contributes significantly to 18 million deaths each year from cardiovascular diseases worldwide (WHO,

2021). Unhealthy diets, little exercise, tobacco and heavy drinking are the main factors driving SSmore people into high blood pressure (WHO, 2021). When hypertension finally gets picked up, the organs such as the heart and kidneys may have already been affected.

In Sub-Saharan Africa the numbers are even more alarming with as many as 46% of adults there having high blood pressure (Adeloye *et al.,* 2021). The fast pace of urbanization and lifestyle changes have had a huge impact on that figure. According to a recent survey in rural Nigeria and Kenya and urban Tanzania and Namibia 19.3% of people in rural Nigeria, 21.4% in rural Kenya, 23.7% in urban Tanzania and 38.0% in urban Namibia carried the same diagnosis (Addo *et al.,* 2024).

Cardiovascular diseases continue to remain the main cause of mortality in many countries in Africa, a huge public-health problem in the continent (Addo *et al.,* 2024). In Nigeria, Shypertension has changed from being a 'silent killer' to an epidemic, especially among the fast-growing cities where modern lifestyle is taking root. A recent meta-analysis shows that the national rate has risen from roughly 8.6% in 1995 to 32.5% in 2020, an increase that adds around 23 million new cases and takes the total from about 4.3 million to 27.5 million people (Adeloye *et al.,* 2024). Now, western-style eating, sitting for long hours, stress, and other urban habits now push an estimated 30 to 40 per cent of adults over 18 into hypertensive territory across the country (Ogunlana *et al.,* 2021).

Despite the huge rise in prevalence, however, awareness, treatment and control rates remain low, with only 29% of hypertensive people aware of their condition, 12% being treated, and just 2.8% of people controlling their blood pressure (Adeloye *et al.,* 2024).

A 2014 survey in Ilorin, Kwara State, revealed that Hypertension stages 1 and 2 were present in Abu-Saeeds work and of the 270 people tested stages 1 and 2 while about half 134 (49.6%) had systolic and diastolic levels classed as pre-hypertensive a higher rate than many other reports.

Abu-Saeed *et al.,* (2014) added that about 14.9% of the sample had never been diagnosed with high blood pressure Abu-Saeed *et al.,* 2014.

Hypertension is generally more prevalent in cities than in the countryside, and Nigerians lead because their diets now consist largely of processed food loaded with salt and unhealthy fats. But as more people move out of rural areas into towns, that gap is narrowing; they pick the habits up and the disease follows.

Generally, speedy urbanization, everyday economic stress, and inadequate access to clinics for early checks and long-term care causes the large numbers of high-blood-pressure patients in Nigeria (Adeloye *et al.,* 2021). Low awareness, poor health literacy, and beliefs in medicine also prevent people from getting assistance when they need it. High-blood-pressure medications are available, but they are expensive and not always available, especially in rural Nigeria (Ogunlana *et al.,* 2021).

2.2 Pathophysiology of Hypertension.

High blood pressure does not result from one problem, but rather, it is the result of a combination of issues that disrupts how our body normally regulates blood pressure. These systems include: the renin-angiotensin-aldosterone system (RAAS), the sympathetic nervous system (SNS), the kidneys, and the inner layers of cells that line our blood vessels.

2.2.1 Renin-angiotensin-aldosterone system (RAAS) dysregulation.

The RAAS system has an important role in regulating blood pressure by regulating sodium concentration, volumetric fluid balance, and vascular tone. When the kidneys sense low sodium content, they begin to synthesize renin that converts angiotensinogen into angiotensin I which is released into the liver. With the aid of an enzyme known as angiotensin-converting enzyme (ACE), ‘Angiotensin I’ is converted into ‘Angiotensin II’. The lungs are mainly responsible for this final conversion (Chiong *et al.,* 2020).

The main function of Angiotensin II is to cause constriction of vessels and an increase in blood pressure. In addition, it also stimulates the release of aldosterone from the adrenal glands which goes on to stimulate the release of sodium reabsorption. Hence, both sodium and water retention is increased by aldosterone which causes an increase in blood volume. Thus, the product of this system amplifies the ability for blood pressure to increase (Hall *et al.,* 2020). Individuals who have been diagnosed with hypertension have an overactive RAAS, which leads to chronic fluid retention, increased systemic vascular resistance, and vasoconstriction which causes constant high blood pressure.

2.2.2 Sympathetic nervous system (SNS) over activity.

The SNS represents another important regulator of cardiovascular function by mediating the "fight-or-flight" response through increasing heart rate and contractility of the heart and causing vasoconstriction. In the setting of hypertension, the chronic activation of the SNS with obesity causes increased peripheral vascular resistance and blood pressure. Stress, obesity, and sleep apnea has been associated with greater SNS activity, which exacerbates the course of hypertension (Brouwers *et al.,* 2021).

2.2.3 Kidney Function and Sodium Retention.

Relationship between Kidneys and Long term Blood Pressure Modulation. Kidney help modulate blood pressure in two main ways; sodium and water balance. An example would be that in many people with hypertension, the sodium handling by the kidneys is suboptimal, such that they retain sodium, increase plasma volume, and lead to high blood pressure (Hall *et al.,* 2020). The other instance would be that chronic kidney disease is often associated with, or causes hypertension and creates a vicious cycle that only serves to compound the problem (i.e. increasing kidney injury leads to mounting blood pressures which in turn increases further renal injury).

2.2.4 Endothelial Dysfunction and Nitric Oxide (NO) Deficiency.

Blood vessels release a number of vasoactive substances, including nitric oxide (NO), which is a vasodilator. In people with hypertension, oxidative stress and inflammation cause a decrease in the levels of Nitric Oxide, leading to difficulty in promoting adequate blood vessel dilation. This impairment in the release of the fuel for NO enhanced vascular resistance and worsens hypertension (Drummond *et al.,* 2019).

2.3 Epidemiology of Hypertension.

It is estimated that hypertension affects 30 to 40 % of adults in Nigeria which places a greater burden on the public health than ever before. There is also strong evidence that age, gender, socio-economic status, and geographic location are determinants for hypertension among Nigerians (Adebayo *et al.,* 2020). Urbanization, along with obesity, a sedentary lifestyle, and poor diet are also driving increase in hypertension cases in Nigeria.

2.3.1 Urban vs. Rural Hypertension.

The adoption of a sedentary lifestyle and a western eating habit in cities have led to an increased prevalence of hypertension in Nigeria's urban areas as compared to rural areas. The consumption of processed foods is more prevalent amongst city dwellers, which leads to an increased take of sodium resulting high blood pressure (Ogunlana *et al.,* 2021). On the other hand, it used to be the case that rural households adhered to a traditional diet that consisted of mostly fruits, vegetables, and grains, low in salt and fats. These diets are changing due to increased rural-urban migration and modern lifestyle changes which are leading to an increased in the number of cases of hypertension in rural areas too (Adeloye *et al.,* 2021).

2.3.2 Gender and Age Related Differences in Hypertension.

Men have a greater risk of developing hypertension at a younger age as compared to women, although postmenopausal women are more affected due to an increase in hormonal changes (Gates *et al.,* 2020). The impact of age also cannot be understated as it increases the likelihood of being diagnosed with hypertension; this poses a serious challenge to Nigeria as its population is aging rapidly and the healthcare system will be expected to accommodate the demand for hypertension services (Adebayo *et al.,* 2020).

2.4 Inflammation and Hypertension.

The importance of inflammation is much better understood in relation to its effects on hypertension and its progression. Chronic low-grade inflammation causes vasculature to become stiffer, promotes atherosclerosis and induces endothelial dysfunction, all of which contribute to the development of hypertension (Tsimikas, 2022).

2.4.1 Role of High Sensitivity C-Reactive Protein (hscrp).

One inflammatory marker that is often overlooked, but has a direct impact on hypertension is high sensitivity c-reactive protein (hsCRP). HsCRP is a marker of system inflammation as it is secreted by the liver after receiving signals from other cytokines such as interleukin-6 (IL-6). The presence of hscrp indicates that there is system inflammation in the body, and the more hscrp present, the greater the risk of cardiovascular complications, especially in hypertensive patients. The hypothesis goes that inflammation causes increased stiffness of arteries and damages blood vessels which in turn causes an increase in blood pressure (Wang *et al.,* 2021).

Just as high concentrations of sodium and water are linked to increased blood pressure and their values predict lower future cardiovascular outcomes in patients once thought to have controlled hypertension. Han *et al.,* (2020) reported that high blood pressure is associated with an increased

cardiovascular disease risk of 35% due to high levels of hsCRP. Furthermore, hsCRP predicts the severity of hypertension with high accuracy.

2.4.2 Other inflammatory biomarkers in hypertension.

Apart from hscrp, it is not only one inflammatory factor that is linked to hypertension, Interleukin-6 (IL-6), as well as tumor necrosis factor-alpha (TNF-α) and interleukin-1 beta (IL-1β) are also linked to hypertension. These cytokines are involved in promoting oxidative stress, endothelial dysfunction, and remodeling of blood vessels and therefore play a vital role in hypertension (Zhao *et al.,* 2022).

In particular, Increased IL-6 levels are associated with increased vascular inflammation as well as endothelial dysfunction which results in increased vascular resistance.

2.5 Hypertension as a result of oxidative stress.

Oxidative stress is an imbalance between the production of reactive oxygen species (ROS) and the body's antioxidant defenses (Drummond *et al.,* 2019). When more ROS is produced than the body can manage, there is an excess of ROS production which leads to the destruction of body components such as lipids, proteins, and DNA. This leads to damage of the endothelium, vascular inflammation, and vasodilation (Drummond *et al.,* 2019).

2.5.1 Oxidative stress markers in hypertension.

The most important consequence of oxidative stress is lipid peroxidation; one of the biomarkers of lipid peroxidation is malondialdehyde (MDA). Hypertensive patients present increased MDA contents, which means that the oxidative damage is enhanced; moreover, it has been demonstrated that the levels of MDA are not only correlated with the degree of hypertension, but also predict future cardiovascular events as heart attacks or strokes (León-Pinto *et al.,* 2023).

2.5.2 Antioxidant Defenses and TAC

Total Antioxidant Capacity (TAC) is a powerful antioxidant that protects cells from oxidative damage. Hypertensive patients usually present decreased contents of TAC, which means that the antioxidant defenses are not adequate to counteract the oxidative stress. That reduced defense system enhances the damage due to oxidative stress and therefore, it leads to an increase in the damage to the blood vessels which increases blood pressure (López-Jaramillo *et al.,* 2022). For these reasons, it is possible to consider that the use of antioxidants could be a possible treatment in hypertensive patients.

2.6 The Use of Biomarkers in the Management of Hypertension.

Hypertension presents as its main biomarkers hsCRP, IL-6, MDA and TAC, which provide us with fundamental information about the pathophysiological mechanisms responsible for the appearance of hypertension and also for its clinical management. For example, using hsCRP it has been possible to know the cardiovascular risk in hypertensive patients. It has been demonstrated that hs-CRP is not only able to predict cardiovascular mortality, but also the progression and degree of hypertension (Wang *et al.,* 2021).

If we measure some inflammatory markers or oxidative stress factors, we will be able to determine the presence of the disease at an early stage of its development and therefore to treat it in its initial phases. With these data we will be able to know better what degree of inflammation and damage is present in the patients and therefore adjust their treatment accordingly. However, it is complicated to standardize the measurement of these biomarkers and obtain reference ranges in different populations (León-Pinto *et al.,* 2023).

2.7 Genetic and environmental factors in hypertension.

2.7.1 Genetic factors.

Hypertension is a disease with multiple factors involved and part of them are genetic. There is strong proof of the heritability of hypertension, since it has been studied in twins, families and also through the genome-wide association studies which have estimated that the genetic factors involved vary from 30-50% of the phenotypic variance of blood pressure (Ehret and Caulfield 2019). This means that if we have family members with hypertension our probability of having it increases a lot and if both parents are hypertensive the risk is even greater.

Hypertension is associated with multiple genes, including the angiotensinogen (AGT), angiotensin-converting enzyme (ACE) and angiotensin II type 1 receptor (AGTR1) genes. The impact of these genes and the renin-angiotensin-aldosterone system (RAAS) is great because greater activity of these genes will result in increased activity of the RAAS leading to vasoconstriction, increased sodium retention and hypertensive blood pressure levels (Ehret and Munroe, 2021).

Additionally, other genes that lead to hypertension are the sodium/potassium ATPase (ATP1A1) and epithelial sodium channel (ENaC) which are responsible for the handling of sodium by the kidneys. Variants of these genes are associated with the excessive reabsorption of sodium which leads to increased blood volume and elevated blood pressure. Polymorphisms in genes encoding for proteins involved in vascular function and oxidative stress, such as nitric oxide synthase (NOS3) and superoxide dismutase (SOD2), have also been associated with increased blood pressure, particularly when associated with endothelial dysfunction (Munroe *et al.,* 2022).

2.7.2 Gene-environment interactions.

Although genetics plays a large role in hypertension, there are also other factors that may play a role in helping to mask the genetic tendency. This combination of inherited factors and other environmental factors such as nutrition, exercise and stress is known as gene-environment

interaction. An example would be people who have certain polymorphisms in RAAS genes and are therefore prone to developing hypertension with increased consumption of salt, which is common in many countries with low and middle income (Lifton *et al.,* 2020).

Hypertension can also result from epigenetic changes that modify the expression of genes without changing the genetic sequence itself. Some maternal factors, such as diet and prenatal stress, as well as early life nutrition, could lead to changes in DNA methylation and histone changes within the fetus which are associated with the regulation of blood pressure. This form of biological programming can permanently affect a person's blood pressure for the rest of their life (Sharma *et al.,* 2021).

2.7.3 Environmental and Lifestyle Factors.

The following environmental factors contribute to set up and worsen hypertension.

Diet: Hypertension remains one of the biggest chronic diseases in the world today caused by a number of different factors but with diet being one of the main factors that lead to hypertension. Sodium leads to retention of fluids which increases blood volume and causes heightened pressure on the blood vessels. On the other hand, potassium, magnesium and calcium rich foods help to lower blood pressure. This is the DASH diet which leads to better sodium excretion and improved endothelial function (Siervo *et al.,* 2020).

Physical activity: The lack of physical activity is also associated with hypertension. Exercise helps to control blood pressure as it improves the function of the heart, reduces resistance of the vessels and improves the insulin sensitivity. Even mild exercises like brisk walking or cycling contributes towards lowering blood pressure in hypertensive people (Pescatello *et al.,* 2019).

Obesity: Obesity is a strong risk factor for the development of hypertension. Increased body weight leads to insulin resistance, activation of the sympathetic nervous system and

activation of the renin-angiotensin-aldosterone system (RAAS) which all lead to an increase in blood pressure. A reduction in excess body weight leads to a decrease in blood pressure in hypertensive and normotensive individuals (Hall *et al.,* 2020).

Alcohol consumption: The risk of developing hypertension is increased by chronic heavy alcohol consumption. Alcohol causes an increase in blood pressure by stimulating the sympathetic nervous system and impairing baroreceptor function. Improved cardiovascular outcomes and a decreased risk of hypertension have been associated with reducing alcohol intake (Roerecke *et al.,* 2018).

Stress: Chronic psychological stress leads to hypertension through sustained activation of the sympathetic nervous system and the hypothalamic-pituitary-adrenal axis (HPA), both of which lead to increased cortisol secretion and vasoconstriction, and sodium retention. Chronic stress leads to hypertension which can be mitigated by mind-body practices such as yoga, guided imagery and cognitive-behavioral therapy (Steptoe and Kiviak 2020).

2.8 Emerging therapies targeting inflammation and oxidative stress in hypertension.

In recent years, advances in the understanding of the role of inflammation and oxidative stress in hypertension have led to the development of new therapies targeting these processes.

2.8.1 Anti-inflammatory therapies.

Given the strong association between chronic low-grade inflammation and hypertension, it is conceivable that treatment with anti-inflammatory medications could be used to reduce blood pressure and the risk of cardiovascular events. Statins are used to treat hypercholesterolemia but they also have anti-inflammatory properties which have been shown in several clinical trials, including lowering of hsCRP and a reduction in the release of pro-inflammatory cytokines such

as IL-6 and TNF-α. It has been proposed that statins provide additional cardiovascular protection in hypertensive patients, particularly in those with high inflammation levels (Ridker *et al.,* 2019). Another approach is to investigate the use of tocilizumab and other IL 6 receptor antagonists which block the inflammatory effects of IL-6. Some early clinical trials have shown that inhibition of IL-6 may reduce blood pressure in hypertensive patients with high inflammatory statuses but more research is required to see if these therapies have any long-term benefits or risks (Zhao *et al.,* 2022).

2.8.2 Antioxidant therapies

The aim of antioxidant therapy is to reduce oxidative stress by quenching ROS and restoring the balance between pro-oxidants and antioxidants. Because of its ability to decrease oxidative stress and improve endothelial function, N-acetylcysteine (NAC), which is a precursor of glutathione, reduces oxidative stress in hypertensive patients and improves endothelial function by preventing ROS-induced vascular injury, which in turn reduces blood pressure (Vaziri *et al.,* 2020).

Yet another antioxidant that has been shown to reduce blood pressure is Coenzyme Q10 (CoQ10). It also improves mitochondrial function and decreases oxidative injury to the vascular tissues, making it useful for treating hypertension (Tomasetti *et al.,* 2021).

2.8.3 Targeting the Gut Microbiome

Recent research has suggested that the gut microbiome may be a critical determinant of blood pressure because of its influence on inflammation and oxidative stress, as well as metabolic effects. In addition, there is a relationship between dysbiosis and high blood pressure. In animal and human studies, probiotics and prebiotics, which result in increased beneficial gut bacteria, have been shown to improve blood pressure control (Marques *et al.,* 2018).

2.9 Future Directions in Hypertension Research and Management

The possibilities of hypertension are looking very bright as the molecular and genetic causations are being unveiled and they do open doors for personalized medicine. One possibility would be to synthesize therapies for an individual based on his or her genetic composition. For example, in RAAS gene polymorphisms, those individuals will likely respond better to RAAS inhibitors on the other hand those individuals with some genetic variants associated with oxidative stress will respond better to antioxidant therapies (Munroe *et al.,* 2022).

Furthermore, the discovery of new biomarkers will more likely improve the diagnosis of hypertension and the classification of the associated risks. The use of inflammatory markers (hsCRP, IL-6), oxidative stress markers (MDA, TAC), and even some genetic markers will improve the assessment and, and ultimately the classification of the individual's risk for cardiovascular complications and enable targeted more efficient therapeutic intervention (León-Pinto *et al.,* 2023).

2.9.1 Artificial Intelligence (AI) in Hypertension Management

Bringing AI and machine learning into the world of hypertension management can improve care as well as outcomes for patients. From a patient's genetic profile, clinical data, and even their lifestyle information, algorithms can determine if a patient is on track to develop hypertension, and what treatments to offer them.

Blood pressure monitoring is being considered with the use of wearables that would constantly monitor and provide information on blood pressure changes and even predict the onset of early hypertensive complications (Topol, 2021).

CHAPTER THREE

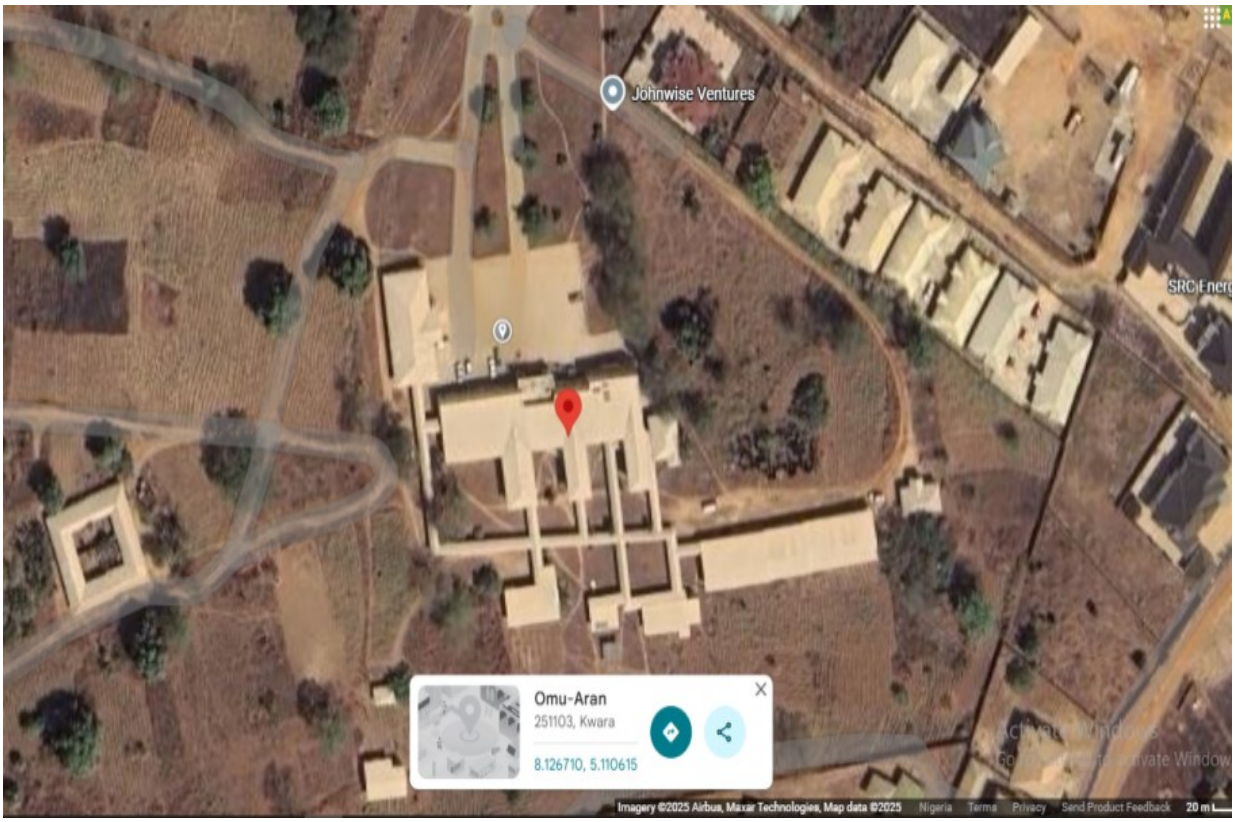
METHODOLOGY

3.1 Research Design

This research work adopted case control design method for the study of inflammatory and oxidative stress markers in hypertensive patients using Thomas Adewumi university Teaching Hospital as the study Centre. This design method was chosen because it is possible to study hypertensive patients (cases) and non-hypertensive patients (controls) in one setting such that it will enable the evaluation of association between some biomarkers and lifestyle factors and the severity of hypertension, this design is also helpful in studying the effect of these markers (inflammatory and oxidative stress) as exposures and the hypertension as an outcome by comparing the patient population with and without the disease.

3.2 Study Location

The study was conducted at Thomas Adewumi University Teaching Hospital Omuaran, Kwara state. It was chosen for this study because the hospital is proximate to some hypertensive patients in the community and surrounding areas. Also the medical Centre treats hypertensive patients which makes it a suitable Centre for recruiting participants for the study and data collection.



8°07'35.3"N 5°06'38.9"E

Figure 1: Geographical location of Thomas Adewumi University Teaching Hospital Omuaran, kwara- state.

3.3 Study Population

The study population for this research consisted of hypertensive patients between the ages of 18 to 65 years who visited Thomas Adewumi University Teaching Hospital for the treatment of hypertension. Both male and female hypertensive patients were recruited for this study so as not to have female or male biased results. The hypertensive patients for this study will be from different strata of the economy and are representative of hypertensive patients in the area.

3.4 Inclusion and Exclusion Criteria Cases

Inclusion criteria (Cases)

1. Patients aged between 18 and 65 years with hypertension.
2. Participants who have a history of diagnosis of hypertension which can be staged as either 1, 2 or 3.
3. Participants who have not taken any anti-inflammatory or antioxidant therapy in the preceding three months prior to this study which may induce changes in biomarker levels.
4. Participants who have voluntarily consented to participate in the research by signing the consent form.

Inclusion criteria (Controls)

1. Participants aged between 18 and 65 years who do not have hypertension as an ailment and a history of ever being hypertensive.
2. Participants who have not been on anti-inflammatory or antioxidant medications in the last 3 months.
3. Participants who have provided written informed consent.

Exclusion criteria (for both Cases and Controls):

1. Individuals with chronic diseases such as diabetes, chronic kidney disease etc. which will confound the result of this study.
2. Pregnant women (this is because pregnancy associated hypertension and other physiological changes may cause changes in the biomarker levels).
3. Individuals with acute infections, surgeries etc.

3.5 Sample Size Determination.

The total number of participants for this case-control study are 100 participants, comprising of 50 hypertensive patients as cases and 50 non hypertensive individuals as controls. The sample size of this study was calculated using Cochran's formula for sample size determination in health research

$n = Z^2.\frac{p(1-p)}{\{e^2\}}$ (Grace *et al.,* 2017)

Where:

- n is the required sample size,
- Z is the Z-value for a 95% confidence interval (1.96),
- p is the estimated proportion of hypertensive patients in the population assumed to be 0.5
- e is the margin of error, set at 0.098.

$$n = 1.96^2 \times \frac{0.5(1-0.5)}{0.098^2}$$

$n = 100$(respondent)

The number of respondents was deemed sufficient to detect significant associations between the levels of biomarkers and the severity of hypertension regardless of possible non-responses or withdrawals that may occur during the study period.

3.6 Data Collection Methods.

Blood Pressure measurement:

Measurement of blood pressure were taken using calibrated sphygmomanometers Patients with hypertension (cases) will be staged as stage 1, 2 or 3 hypertension according to the American Heart Association classification standard. To obtain accurate measurements, patients were assessed three times while at rest and the mean was computed. Non-hypertensive patients (controls) had their blood pressure measured to verify their control status.

Questionnaires:

Both cases and controls were interviewed using a structured questionnaire to get the respondents age, gender, income level as well as their lifestyle behaviors such as diet, smoking, physical activity and alcohol consumption This comparison were made on the level of inflammatory and oxidative stress markers in hypertensive patients or the controls. The questionnaire content, including the types of information collected and response option is summarized in the Table 1 below.

Table 1: summary of data collection instrument

Question	HYPERTENSIVE	NORMOTENSIVE
SECTION 1: DEMOGRAPHIC INFORMATION		
Age		
Gender		
Marital Status		
Educational Level		
Occupation		
SECTION 2: MEDICAL HISTORY		
Have you been diagnosed with hypertension?		
Do you have a family history of hypertension?		
Have you been diagnosed with diabetes?		
Have you been diagnosed with any chronic kidney disease?		
SECTION 3: LIFESTYLE FACTORS		
Do you smoke?		
Do you consume alcohol?		
How often do you exercise?		
SECTION 4: DIETARY HABITS		
How many meals do you eat per day?		
Do you consume fruits and vegetables daily?		
How often do you eat high-salt foods?		
Do you consume sugary drinks or snacks?		
SECTION 5: STRESS AND SLEEP PATTERNS		
How many hours of sleep do you get per night?		
Do you often feel stressed?		
Have you experienced significant life changes recently?		
SECTION 6: MENTAL HEALTH		
In the last month, how often have you felt nervous and stressed?		
In the last month, how often have you felt confident about handling personal problems?		
SECTION 7: MEDICATION ADHERENCE		
Do you take your prescribed medications regularly?		
Do you experience any side effects from medications?		
SECTION 8: KNOWLEDGE AND AWARENESS		
Are you aware of the complications of untreated hypertension?		
Have you received counseling on hypertension management?		
If yes, who provided the counseling?		
Do you know your target blood pressure range?		
SECTION 9: CLINICAL AND BIOCHEMICAL DATA COLLECTION.		
Systolic/ Diastolic (mmHg)		
HsCRP (mg/l)		
MDA Levels (mmol/l)		
TAC Levels		
Total protein (mmol/l)		
Albumin (mmol/l)		
Globulin Level (mmol/l)		
Body measurement: Body weight (Kg)		
Body measurement: Body height (cm)		
Body measurement: Waist circumference (cm)		
Body measurement: BMI		

Blood Sample Collection:

Blood samples were obtained (10 ml) from each participant via venipuncture by trained medical personnel. These blood samples were processed for serum and plasma separation, stored at -10°C and subsequently analyzed for inflammatory and oxidative stress markers in cases and controls groups.

Inflammatory Marker:

High sensitivity C-reactive protein (hsCRP) measurement was done using enzyme linked immunosorbent assay (ELISA) kits Oxidative Stress Marker: Measurement of Malondialdehyde (MDA) using spectrophotometric methods to determine lipid peroxidation

Other Marker:

Albumin level and Globulin level was measured using standard clinical chemistry methods to determine the overall health status and nutritional status of cases and controls groups.

3.7 Laboratory Analysis

Collected blood samples were processed in a certified laboratory (University of Ilorin Teaching Hospital) and analyzed using the following procedures:

Inflammatory Marker:

High sensitivity C- reactive protein (hsCRP) measurement was done using enzyme linked immunosorbent assay (ELISA) kits. All assays were done in duplicates for accuracy and precision of results Concentration will be expressed in mg/L.

Oxidative Stress Markers:

The levels of Malondialdehyde (MDA) was measured by thiobarbituric acid reactive substances (TBARS) assay which is a well-known method for measuring lipid peroxidation and Total antioxidant capacity (TAC/ T-AOC) was measured using colorimetric assay.

Other Marker:

Albumin levels and Total protein were measured by colorimetric assay; it is a well-known biochemical method for albumin measurement in serum (g/dL). Globulin was estimated using simple subtraction method (Total Protein-Albumin= Globulin) in g/dl.

3.8 Data analysis

SPSS version 25.0 statistical package was used for data analysis. The following statistical methods will be used in this study.

Descriptive Statistics:

Descriptive statistics mean and standard deviation (SD) was used to summarize the demographic characteristics, lifestyle factors, blood pressure levels and biomarker concentrations in cases and controls.

Correlation Analysis:

Pearson correlation was used to evaluate the relationships between inflammatory (hsCRP), oxidative stress markers (MDA and TAC), albumin levels, globulin levels and blood pressure readings in hypertensive group (cases).

Comparison of Cases and Controls:

Independent t-tests or Mann-Whitney U tests was used to compare the hsCRP, MDA, Albumin, Globulin and TAC levels between cases (hypertensive patients) and controls (non-hypertensive individuals).

Regression Analysis:

Multiple linear regression analysis was used to evaluate the effects of lifestyle factors (for example; diet, smoking, physical activity) on the hsCRP, MDA and albumin levels, globulin levels. In addition, the regression analysis was adjusted for possible confounding variables (for example; age, gender).

Statistical Significance:

All analyses were done with 95% confidence interval and $p < 0.05$ value will be considered as statistically significant.

3.9 Ethical considerations

Ethical Approval: The study protocol was submitted to Ministry of Health Research Ethics Committee for approval. The study follows the principles presented in the Declaration of Helsinki.

Informed Consent: Written informed consents were taken from participants after explaining the aims of the study, how the study will be carried out and any possible risks or benefits. Participants had the right to withdraw from the study at any time without any disadvantage.

Confidentiality: All personal identifiers were deleted from the data that was collected during the study. Participants' data were stored in a password protected database of the Thomas Adewumi University only by authorized personnel.

Data Safety: Biological samples were handled according to biosafety regulations to protect the participants and the laboratory personnel. All biological samples were disposed of according to the related guidelines after the analysis is done

CHAPTER FOUR

4.0 RESULTS

This chapter presents the collated data derived from the analysis of demographic, lifestyle, clinical, and biochemical data collected from 50 hypertensive and 50 normotensive individuals. The results are systematically produced in tabular and written formats to address the research questions concerning the levels of inflammatory and oxidative stress markers, their correlation with hypertension severity, the influence of lifestyle factors, and comparative differences between the study groups.

4.1. Demographic and Clinical Characteristics of Study Participants

A total of 100 participants were included in the study, comprising 50 hypertensive individuals and 50 normotensive individuals. The demographic and key clinical characteristics of the overall cohort, and disaggregated by hypertension status, are summarized in Figure 2- 8 below.

The two study groups (hypertensive and normotensive) were largely comparable across key demographic variables including age, gender distribution, marital status, educational level, and occupation. This demographic similarity suggests that any observed differences in biomarkers or health outcomes are less likely to be confounded by these baseline characteristics. As expected, the hypertensive group exhibited significantly elevated systolic and diastolic blood pressures (mean systolic 201.24 ± 29.13 mmHg and diastolic 130.64 ± 15.68 mmHg) compared to the normotensive group (mean systolic 115.14 ± 9.42 mmHg and diastolic 70.00 ± 6.16 mmHg), clearly depicting their respective clinical statuses.

Mean Age of the Participants

The overall cohort exhibited a mean age of approximately 41.59 years. Mean ages for the hypertensive and normotensive groups were 41.74 years and 41.44 years, respectively. Age is a well-established confounder influencing both the prevalence and severity of hypertension, as well as the physiological levels of inflammatory and oxidative stress markers.

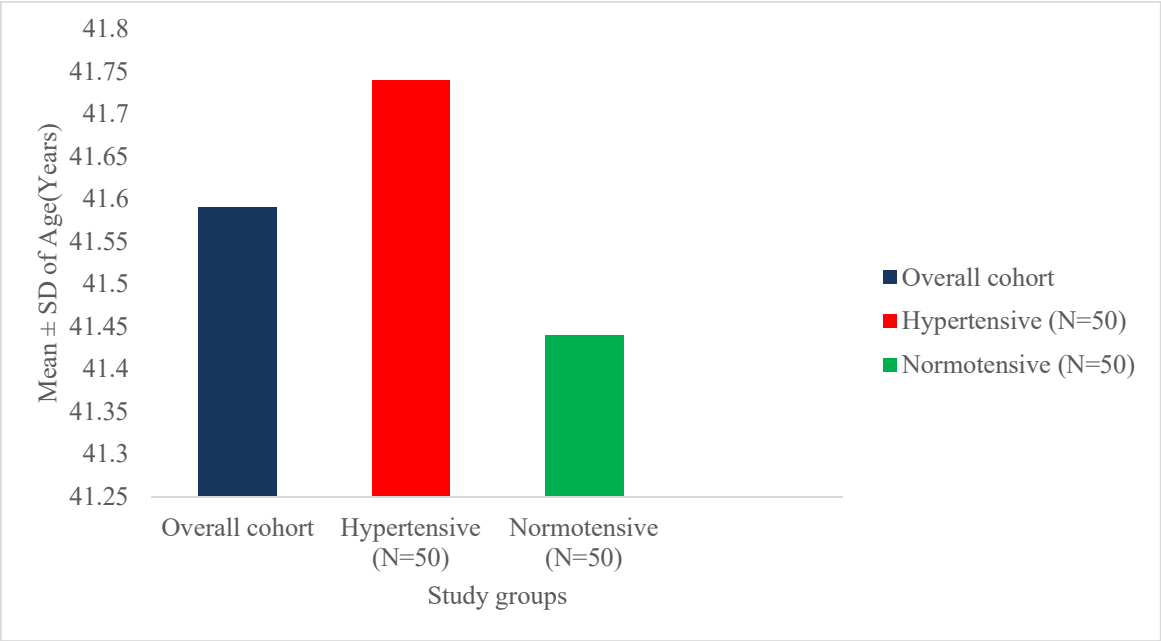


Figure 2: Mean Age of the participant

Gender Distribution of the Participants

The overall cohort (N=100) comprised approximately 52% males and 48% females. Distinct patterns emerged within the subgroups: the hypertensive group (N=50) showed a male predominance (approximately 56% male), while the normotensive group (N=50) exhibited a female predominance (approximately 52% female). This suggests an uneven gender distribution between the cases and controls. Gender is recognized as a biological variable that can influence both the manifestation of hypertension and the basal levels and responses of inflammatory and oxidative stress markers.

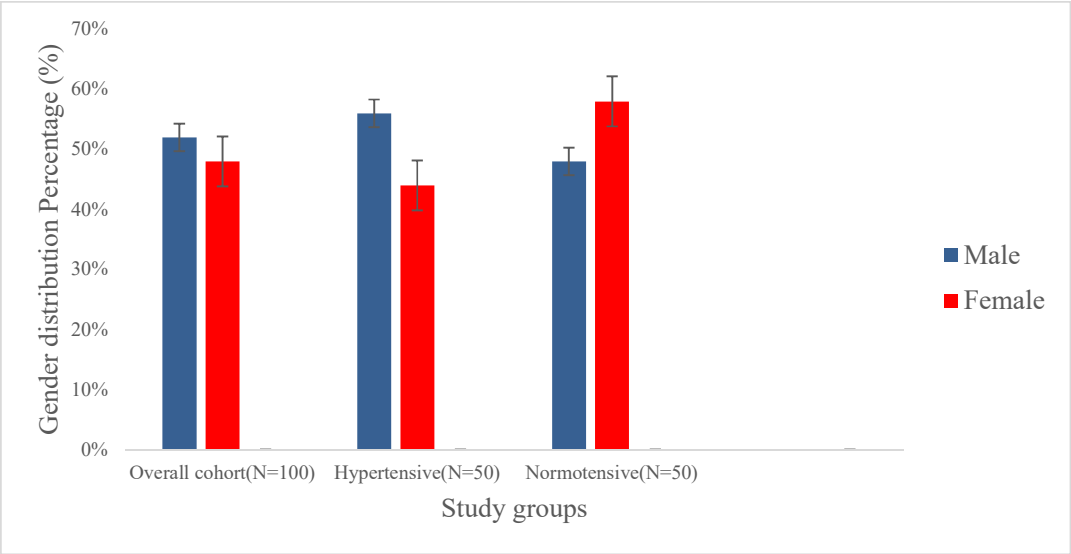


Figure 3: Gender Distribution of the participants across study groups

Marital Status Distribution

Across the overall cohort, married individuals constituted the largest proportion (approximately 38%), followed by single (27%), divorced (22%), and widowed (13%) participants. A similar proportional distribution was observed within both the hypertensive and normotensive groups, indicating a high degree of comparability in marital status between the two cohorts. Marital status, as a social determinant of health, can be associated with varying levels of psychosocial stress and health-seeking behaviors, which may indirectly influence inflammatory and oxidative stress pathways.

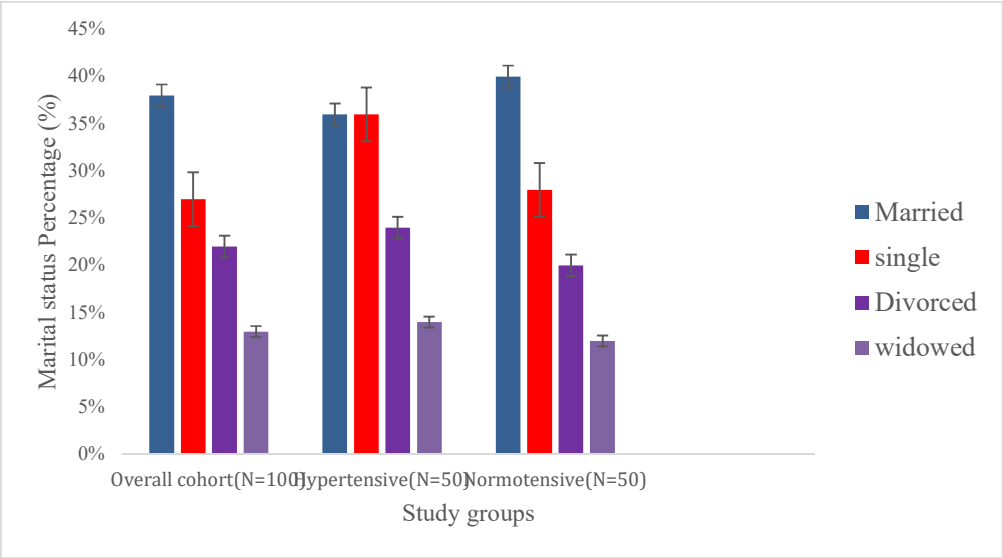


Figure 4: Marital status distribution across study groups

Educational Level Distribution

Secondary education was the most prevalent level across the overall cohort (approximately 37%), the hypertensive group (36%), and the normotensive group (38%). Primary education followed consistently (20%), with tertiary education making up a proportion (35%), and a minor percentage reporting no formal education (around 7%). The distribution of educational levels appears highly consistent across both study groups. Educational attainment often correlates with socioeconomic status, health literacy, and access to health information, all of which can impact lifestyle choices, adherence to treatment, and physiological markers.

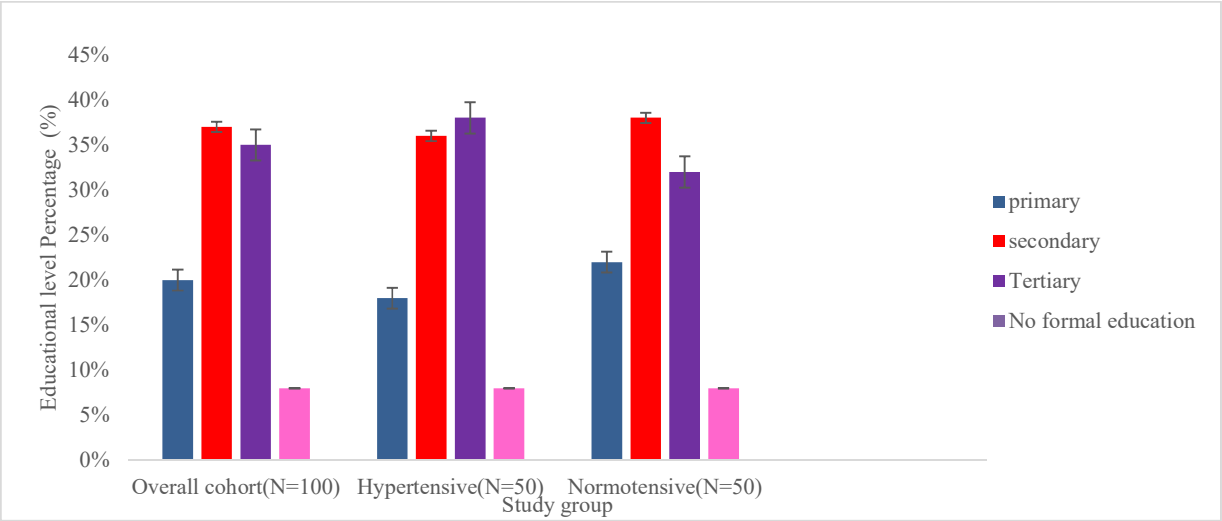


Figure 5: Educational Level percentage distribution across study groups

Occupation of Participants

This presents the occupational distribution of participants by percentage across the overall cohort, hypertensive, and normotensive groups. Consistently, the most represented occupations in all groups were Engineers (approximately 28%), Clerks (20%), and Drivers (16%). Business owners (12%) and Farmers (12%) comprised smaller, but similar, proportions. The occupational distribution appears notably consistent between the hypertensive and normotensive cohorts. Occupational characteristics can expose individuals to varying levels of physical activity, psychological stress, and environmental factors, all of which may influence systemic inflammation and oxidative stress.

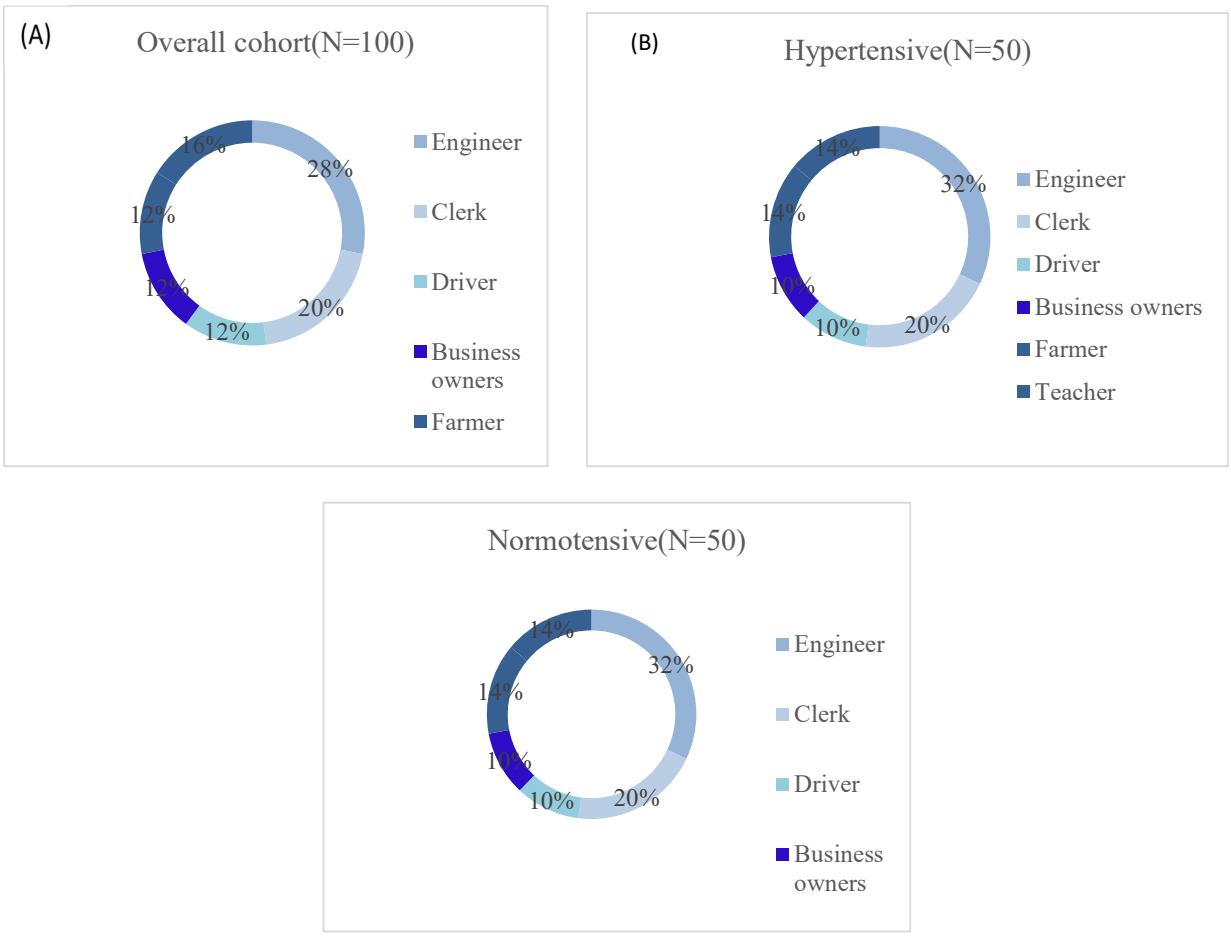


Figure 6: Occupation of participants in percentage.

Study Groups Blood Pressure Characteristics

This clearly depicts the mean systolic and diastolic blood pressure values across the overall cohort, the hypertensive group, and the normotensive group. The hypertensive group exhibited markedly elevated mean systolic blood pressure (approximately 201 mmHg) and diastolic blood pressure (approximately 131 mmHg). In stark contrast, the normotensive control group maintained blood pressure within the healthy range, with mean systolic and diastolic pressures of approximately 115 mmHg and 70 mmHg, respectively. The overall cohort's blood pressure values reflect the combined average of the two distinct groups. This figure serves as a fundamental validation of the study's primary group classification. The substantial and statistically significant differences in blood pressure values between the hypertensive and normotensive groups confirm the successful categorization of participants based on their clinical hypertension status.

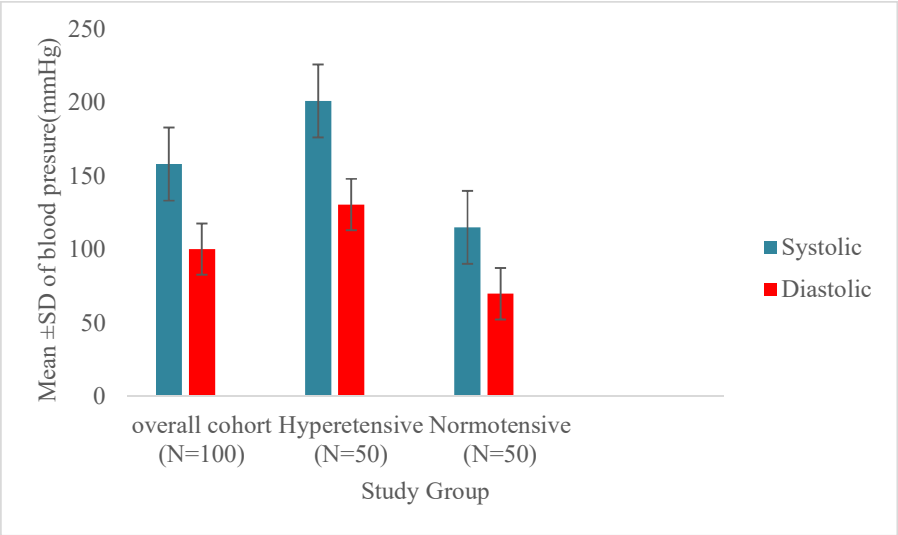


Figure 7: Study groups blood pressure characteristics

4.2. Levels of Inflammatory and Oxidative Stress Markers (and Albumin) in Hypertensive Patients

The summary statistics for hsCRP, MDA, TAC, Globulin, and Albumin in the hypertensive patient group are presented in **Figure 8**, alongside their respective generally accepted reference ranges. Hypertensive patients, as a group, demonstrated an average hsCRP level (6.94 ± 8.65 mg/l) significantly above the upper reference limit (3 mg/l). While the median hsCRP was 2.80 mg/l, the wide range and maximum value of 30.00 mg/l indicate a substantial proportion of hypertensive individuals experiencing elevated systemic inflammation. MDA levels (0.79 ± 0.08 μM) were generally within the reference range but clustered towards its higher end, suggesting ongoing oxidative stress. Interestingly, the mean TAC (6.36 ± 2.45 mM) was substantially higher than its reference range (0.513-1.5 mM), potentially implying a compensatory increase in antioxidant capacity. Globulin levels (30.86 ± 9.77 g/dl) were largely within the normal range. Albumin levels (35.80 ± 6.06 g/dl), on average, were at the lower boundary of the healthy reference range, with some individuals showing concentrations notably below the normal threshold, indicating potential hypoalbuminemia.

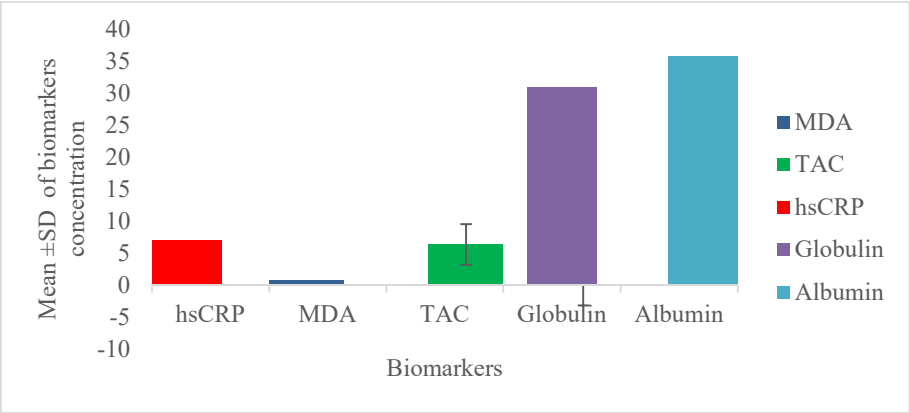


Figure 8: Concentration of various biomarkers in hypertensive cases.

Table 2: Reference range of the biomarkers

Biomarkers	Reference range
HsCRP	1-3 (mg/l)
MDA	0.36-1.24(µM)
TAC	0.5-1.5 (mM)
Globulin	35-50(g/dl)
Albumin	20-35(g/dl)

4.3. Correlation of Biomarkers with Hypertension Severity

Correlation analysis was performed to assess the relationship between biomarker levels and hypertension severity (categorized as Stage 1, Stage 2, and Stage 3) within the hypertensive group. The results are presented in **Figure 9**.

A moderately strong and statistically significant positive correlation was observed only between hsCRP levels and hypertension stage (rho=0.565, p <0.001). This indicates that as hypertension progresses through its stages (from Stage 1 to Stage 3), hsCRP levels significantly tended to increase. No other biomarker (MDA, TAC, Globulin, or Albumin) demonstrated a statistically significant correlation with hypertension stage in this cohort.

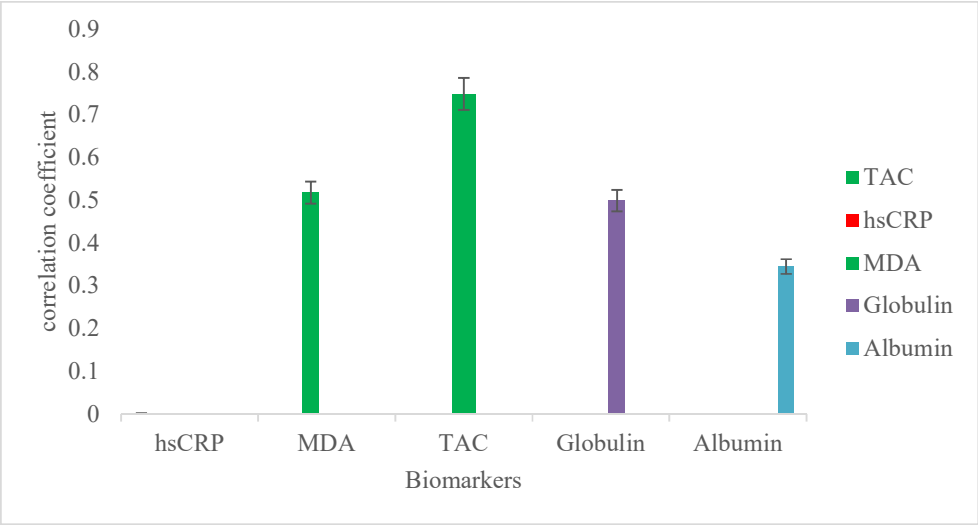


Figure 9: Correlation of biomarkers with hypertension severity.

4.4. Influence of Lifestyle Factors on Biomarker Levels

Multiple linear regression models were employed to identify the significant predictors of each biomarker, including various lifestyle factors (smoking, alcohol consumption, exercise, dietary habits) and demographic variables (age, gender).

Influence of Lifestyle Factors on hsCRP(mg/l)

Smoking status (p <0.001), age (p=0.011), male gender (p=0.009), and consumption of sugary drinks/snacks (p=0.038) were identified as statistically significant positive predictors of hsCRP levels. This indicates that individuals who smoke, are older, are male, or consume sugary drinks/snacks tend to have higher hsCRP, suggesting a heightened inflammatory state. The model explained 34.4% of the variance in hsCRP levels.

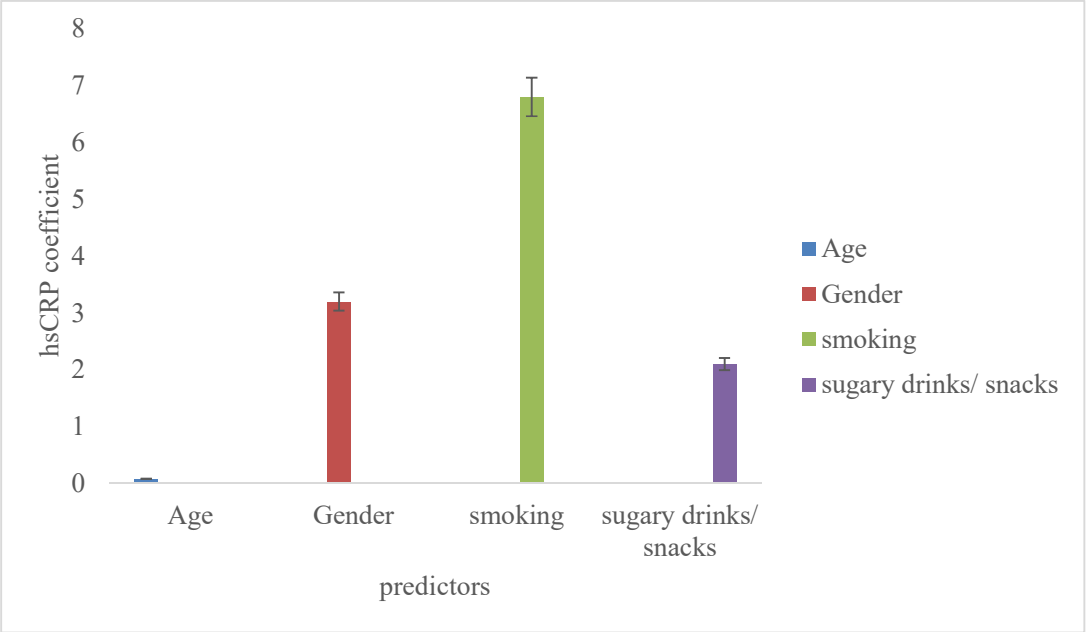


Figure 10: Influence of life style factors on hsCRP

Influence of lifestyle factors on MDA level(µM)

Age was the only statistically significant predictor for MDA levels (p=0.003), showing a negative association, implying that older individuals in this cohort tended to have lower MDA. This counter-intuitive finding warrants further investigation. The model explained 9.0% of the variance in MDA levels.

Table 3: Influence of lifestyle factors on MDA level(µM)

Predictor	Coefficient	Std. Error	t-statistic	*P-value*
Constant	≈ 0.8	≈ 0.05	≈ 16.0	< 0.001
Age	≈ 0.002	≈ 0.001	≈ -3.0	0.003
All other predictors	(Coefficients varied)	(Non-significant *P-value*s)		

Note: Overall model R^2=0.090, F-Statistic p= 0.003

Influence of lifestyle factors on for TAC (mM)

Consumption of sugary drinks/snacks (p=0.036) was identified as a statistically significant positive predictor for TAC levels. This suggests an association between sugary intake and higher antioxidant capacity, potentially indicating a compensatory response mechanism. The model explained 5.5% of the variance in TAC levels.

Table 4: Influence of lifestyle factors on for TAC (mM)

Predictor	Coefficient	Std. Error	t-statistic	*P-value*
Constant	≈ 5.5	≈ 0.8	≈ 6.8	< 0.001
Sugary Drinks/Snacks (Yes=1)	≈ 0.7	≈ 0.3	≈ 2.1	0.036

All other predictors	(Coefficients varied)	(Non-significant *P-value*s)	

Note: Overall model R^2=0.055, F-Statistic p= 0.036

Influence of lifestyle factors on Globulin (g/dl)

Age was the only significant positive predictor for Globulin levels ($p < 0.001$), indicating that older individuals tended to have higher Globulin concentrations. The model explained 15.0% of the variance in Globulin levels.

Table 5: Influence of lifestyle factors on Globulin (g/dl)

Predictor	**Coefficient**	**Std. Error**	**t-statistic**	***P-value***
Constant	≈ 20.0	≈ 3.0	≈ 6.7	< 0.001
Age	≈ 0.2	≈ 0.01	≈ 4.9	< 0.001
All other predictors	(Coefficients varied)	(Non-significant *P-value*s)		

Note: Overall model R^2=0.150, F-Statistic p=< 0.001

Influence of lifestyle factors on Albumin(g/dl)

Age emerged as a significant negative predictor for Albumin levels (p=0.003), implying that older individuals had lower Albumin. Additionally, being male (p=0.038) was a significant negative predictor, with males tending to exhibit lower Albumin levels compared to females. The model explained 10.4% of the variance in Albumin levels.

Table 6: Influence of lifestyle factors on Albumin(g/dl)

Predictor	**Coefficient**	**Std. Error**	**t-statistic**	***P-value***
Constant	≈ 42.0	≈ 1.8	≈ 23.3	< 0.001
Age	≈ -0.04	≈ 0.02	≈ -3.0	0.003
Gender (Male=1)	≈ -1.7	≈ 0.8	≈ -2.1	0.038
All other predictors	(Coefficients varied)	(Non-significant *P-value*s)		

Note: Overall model R^2=0..104, F-Statistic p= 0.001

4.5. Differences in Biomarkers by Hypertension Stage

Significant differences in biomarker levels across the three hypertension stages (Stage 1, Stage 2, Stage 3) were determined within the hypertensive group. The results are presented in **Table 7.** Only hsCRP levels showed statistically significant differences across hypertension stages (H=8.530, p=0.014). Post-hoc analysis for hsCRP revealed a significant difference between Stage 1 and Stage 3 (p=0.019), indicating that Stage 3 hypertensive patients exhibited significantly higher hsCRP levels compared to those in Stage 1. No other biomarker (MDA, TAC, Globulin, or Albumin) demonstrated significant variation across the hypertension stages.

Table 7: Biomarker Differences Across Hypertension Stages (Hypertensive Group, N=50)

Biomarker	**H-statistic**	***P-value***	**Significant (p < 0.05)?**
hsCRP (mg/l)	8.530	0.014	Yes
MDA level (µM)	0.596	0.742	No
TAC (mM)	0.187	0.911	No
Globulin (g/dl)	1.096	0.578	No
Albumin (g/dl)	2.190	0.335	No

Table 8: hsCRP(mg/l) across Hypertension Stages (Corrected *P-value*s)

Comparison	**Corrected *P-value***
Stage 1 vs. Stage 2	1.000
Stage 1 vs. Stage 3	0.019
Stage 2 vs. Stage 3	0.076

Only hsCRP levels showed statistically significant differences across hypertension stages (H=8.530, p=0.014). Post-hoc analysis for hsCRP (Table 4.5.1) revealed a significant difference

between Stage 1 and Stage 3 (p=0.019), indicating that Stage 3 hypertensive patients exhibited significantly higher hsCRP levels compared to those in Stage 1. No other biomarker (MDA, TAC, Globulin, or Albumin) demonstrated significant variation across the hypertension stages.

4.6. Comparison of Biomarkers between Hypertensive and Normotensive Individuals

Mann-Whitney U tests were performed to compare the mean levels of the biomarkers between the hypertensive and normotensive groups. The results are presented in **Table 8**.

Highly significant differences were observed for hsCRP and TAC levels between the two groups (p <0.001 for both). Hypertensive individuals demonstrated significantly higher hsCRP levels, while normotensive individuals exhibited significantly higher TAC levels. No significant differences were found for MDA, Globulin, or Albumin levels between hypertensive and normotensive individuals.

Table 9: Biomarker Differences between Hypertensive (N=50) and Normotensive (N=50) Groups

Biomarker	**U-statistic**	***P-value***	**Significant (p < 0.05)?**
hsCRP (mg/l)	398.000	< 0.001	Yes
MDA level (µM)	1194.500	0.725	No
TAC (mM)	134.000	< 0.001	Yes
Globulin (g/dl)	1242.000	0.963	No
Albumin (g/dl)	1243.500	0.941	No

4.7. Multivariate Predictors of Hypertension Severity

A multiple linear regression model was constructed to identify the independent factors influencing hypertension severity (hypertensive stage, treated as a continuous numerical variable for this analysis). The overall model was statistically significant (F-statistic≈19.5, $p < 0.001$) and explained a substantial portion of the variance in hypertension severity ($R^2 = 0.781$). The estimated coefficients and *P-value*s for each predictor are shown in **Figure 11**.

The multivariate regression model, which had strong explanatory power ($R^2 = 0.781$), identified Age ($p < 0.001$), Smoking Status ($p < 0.001$), Sugary Drinks/Snacks consumption ($p = 0.038$), hsCRP levels ($p < 0.001$), and TAC levels ($p < 0.001$) as statistically significant independent predictors of hypertension severity**.** Specifically, older age, smoking, and consuming sugary drinks/snacks were associated with increased hypertension severity. Higher hsCRP levels were strongly associated with increased severity, while higher TAC levels were strongly associated with lower severity. Gender, other lifestyle factors (alcohol consumption, exercise, fruits/vegetables intake, high salt foods intake), MDA levels, Globulin levels, and Albumin levels were not identified as significant independent predictors of hypertension severity in this comprehensive model.

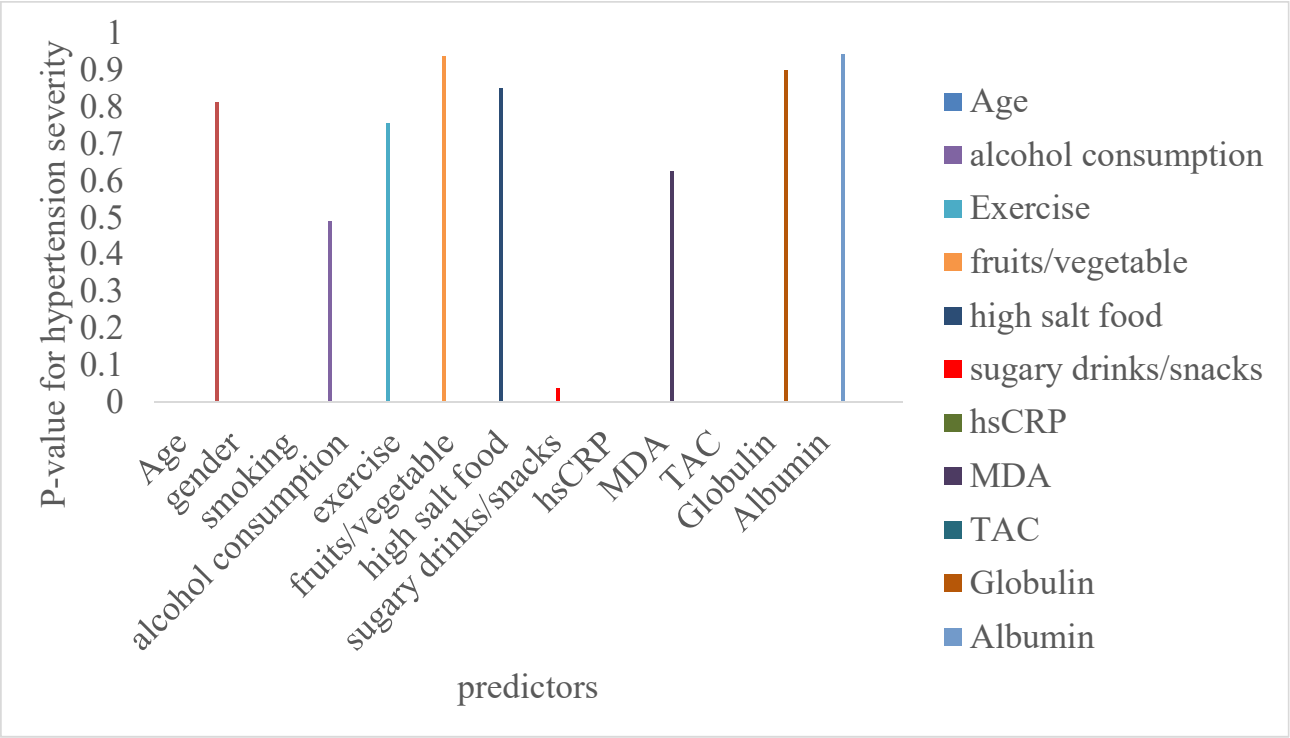


Figure 11: Hypertension severity predictor ($p < 0.001$)

CHAPTER FIVE

DISCUSSION

This study embarked on a comprehensive investigation into the intricate relationships among inflammatory and oxidative stress markers, lifestyle factors, and the severity of hypertension. The findings underscore the multifaceted etiology of hypertension, extending beyond conventional hemodynamic considerations to encompass chronic inflammatory processes and oxidative imbalance.

The most prominent finding is the robust and consistent association of hsCRP with hypertension. Hypertensive individuals in this cohort exhibited significantly higher hsCRP levels compared to their normotensive counterparts. More critically, hsCRP levels demonstrated a direct and significant positive correlation with the increasing stages of hypertension severity, with Stage 3 patients showing markedly higher hsCRP than Stage 1. This observation strongly supports the growing body of evidence that chronic low-grade inflammation plays a pivotal role in the initiation and progression of hypertension (Akinlua *et al.,* 2015; Orji *et al.,* 2022; Udofia *et al.,* 2023). hsCRP, as an acute-phase protein, reflects systemic inflammatory activity that can contribute to endothelial dysfunction, increased arterial stiffness, and altered vascular tone, all pathways implicated in hypertension (Ridker, 1997; Norlander *et al.,* 2018; Harrison *et al.,* 2021). Furthermore, our regression analysis illuminated key upstream drivers of elevated hsCRP, identifying smoking, increasing age, male gender, and consumption of sugary drinks/snacks as significant positive predictors (Orji *et al.,* 2022; Udofia *et al.,* 2023). This highlights how specific lifestyle choices and demographic factors can fuel the inflammatory cascade, thereby potentially exacerbating the hypertensive state.

The analysis of oxidative stress markers offered a more complex narrative. While MDA levels in the hypertensive group were within the broad physiological range, they did not significantly

differentiate between hypertensive and normotensive individuals, nor did they show a clear correlation with hypertension severity. This might indicate that MDA, as a biomarker of lipid peroxidation, may not serve as the most sensitive or specific marker for distinguishing hypertension status or severity in this cohort (Tsiropoulou *et al.,* 2016), or that its levels are influenced by other unmeasured factors. The unexpected inverse relationship between age and MDA in the regression model warrants cautious interpretation and calls for further research, as oxidative stress is typically expected to increase with age. This anomaly could be due to complex adaptive responses or confounding factors not fully captured.

In contrast, Total Antioxidant Capacity (TAC) emerged as a critical protective factor. Normotensive individuals possessed significantly higher TAC levels than hypertensive individuals, and, strikingly, higher TAC was a significant independent predictor of lower hypertension severity. This suggests that a robust endogenous antioxidant defense system may be crucial in conferring resilience against hypertension development or progression. Such antioxidant-protective trends align with broader African metabolic data, including decreased antioxidant enzymes and elevated oxidative stress markers in Nigerian populations with metabolic risk (Suleiman *et al.,* 2016). Conversely, the diminished TAC observed in hypertensive patients could imply a depletion of antioxidant reserves due to chronic oxidative stress, or a compromised ability to maintain antioxidant balance. The positive association between sugary drink/snack consumption and elevated TAC is an intriguing finding. Rather than suggesting a beneficial effect of sugar, this may represent a compensatory upregulation of antioxidant pathways in response to the pronounced pro-oxidant burden induced by high sugar intake, an adaptive mechanism striving to counteract cellular damage.

Albumin and Globulin levels, while integral components of protein homeostasis, did not demonstrate the same diagnostic or prognostic utility for hypertension in this study as hsCRP and TAC. Although the hypertensive group showed a slightly lower average albumin

concentration (though not statistically significant in direct comparison to normotensives), and both albumin and globulin levels were influenced by age (albumin decreasing, globulin increasing) and gender (albumin lower in males), they did not independently correlate with hypertension severity or distinguish between different hypertensive stages. This indicates that their role as direct biomarkers specifically reflective of hypertension's inflammatory or oxidative stress components, within the observed ranges, may be limited (Shang xun *et al.,* 2025).

The multifactorial regression model for hypertension severity provided a powerful synthesis of these findings. It strongly elucidated that the interplay of demographic factors (age), lifestyle choices (smoking, sugary drink/snack consumption), and key biochemical markers (hsCRP as a pro-inflammatory agent, TAC as a protective antioxidant) collectively accounts for a substantial proportion of the variability in hypertension severity. This holistic view underscores that effective hypertension management must address these interconnected elements rather than focusing solely on blood pressure values.

Despite the valuable insights generated, this study is subject to several methodological limitations that warrant acknowledgment. The inherent nature of a cross-sectional study design means that data were collected at a single point in time. Consequently, this study cannot establish definitive cause-and-effect relationships. While associations are identified (e.g high hsCRP with severe hypertension), it is not possible to conclude whether inflammation causes increased severity or if it is a consequence of the long-standing hypertensive state. Longitudinal studies are required to elucidate causality.

The relatively small sample size of 100 participants (50 hypertensive, 50 normotensive) provides foundational insights but may limit the statistical power to detect subtler associations or generalize the findings broadly to the wider population. Larger, multi-center studies would enhance the robustness and applicability of the results.

Information regarding crucial lifestyle factors such as smoking habits, alcohol consumption, dietary patterns, and perceived stress levels was obtained through self-report. This method is susceptible to inherent biases, including recall bias (inaccurate memory of past behaviors) and social desirability bias (tendency to provide answers perceived as socially acceptable), which could potentially impact the accuracy and reliability of the observed associations. Some lifestyle factors were captured using binary (Yes/No) or broadly categorized responses. While pragmatic for questionnaire design, this simplification may oversimplify the complexity of human behavior and limit the depth of analysis, potentially masking more nuanced dose-response relationships or the impact of specific types or intensities of activities/habits.

CONCLUSION

This study significantly contributes to the understanding of hypertension beyond its hemodynamic manifestations, unequivocally demonstrating the pivotal role of systemic inflammation and a compromised antioxidant capacity. Elevated hsCRP levels are strongly associated with both the presence and increased severity of hypertension, with clear links to modifiable lifestyle factors such as smoking and sugary drink/snack consumption. Conversely, a robust Total Antioxidant Capacity (TAC) appears to confer protection against hypertension severity, being notably higher in normotensive individuals. While Albumin, MDA, and Globulin are essential physiological indicators, their direct utility as distinct markers of hypertension status or severity was not as pronounced in this cohort. The findings collectively underscore that hypertension is a complex, multifactorial disease intimately linked with inflammatory processes and oxidative imbalance, offering promising avenues for refined preventive and therapeutic strategies centered on comprehensive lifestyle modifications and targeted interventions to restore redox homeostasis.

The strong and consistent association of hsCRP with hypertension presence and severity suggests its potential utility as a valuable biomarker in clinical settings. Routine monitoring of hsCRP levels in hypertensive patients, particularly those with higher stages, could serve as a valuable tool for risk stratification, identifying individuals with heightened inflammatory risk who may benefit from more targeted therapeutic strategies, including lifestyle modifications or potentially anti-inflammatory pharmacotherapies.

The robust links identified between smoking, sugary drink/snack consumption, and both systemic inflammation (hsCRP) and hypertension severity provide compelling evidence for intensifying public health campaigns. Aggressive advocacy for smoking cessation and significant reduction in added sugar intake should be prioritized within health education programs. These are crucial modifiable risk factors whose amelioration could substantially

impact the national burden of hypertension and its associated complications.

The observed protective role of higher TAC levels against hypertension severity opens new avenues for preventive strategies. Future research should explore the efficacy of dietary interventions rich in antioxidants (e.g fruits, vegetables, nuts) and the potential therapeutic role of specific antioxidant supplements in maintaining optimal antioxidant balance, thereby mitigating oxidative stress and potentially preventing or ameliorating hypertension.

Effective management of hypertension necessitates a holistic approach that extends beyond pharmacological blood pressure control. Healthcare providers should integrate the assessment and modulation of inflammatory and oxidative stress pathways, particularly through lifestyle counseling, into comprehensive patient management plans. This paradigm shift can empower patients to actively participate in managing the underlying drivers of their condition.

RECOMMENDATION

To firmly establish causal pathways and the temporal sequence of events, future research should adopt a prospective longitudinal design. Tracking changes in biomarker levels and lifestyle behaviors over time, and their subsequent impact on hypertension incidence or progression, would provide invaluable insights. Expanding the sample size and recruiting participants from diverse geographical, socioeconomic, and ethnic backgrounds would enhance the statistical power of the study and improve the generalizability of findings to a broader population.

Conduct intervention studies to directly evaluate the efficacy of targeted lifestyle modifications (e.g structured smoking cessation programs, specific dietary interventions emphasizing reduced sugar intake and increased antioxidant-rich foods, tailored exercise regimens) on modulating hsCRP and TAC levels, and subsequently on blood pressure control and long-term cardiovascular outcomes in hypertensive patients. Future studies should employ a more extensive panel of oxidative stress biomarkers (beyond MDA, such as F2-isoprostanes, oxidized low-density lipoprotein, protein carbonyls) and a comprehensive assessment of endogenous antioxidant defense systems (e.g levels of antioxidant enzymes like superoxide dismutase, catalase, glutathione peroxidase, and key non-enzymatic antioxidants like glutathione, vitamins E and C). This would provide a more holistic understanding of oxidative balance in hypertension. Implement objective measures for lifestyle factors, such as accelerometers for physical activity, or detailed food diaries/biomarkers of dietary intake, to reduce reliance on self-report biases and provide more accurate data.

Research can be conducted on the practical implementation and cost-effectiveness of integrating routine hsCRP and TAC monitoring into hypertension management protocols, evaluating its impact on patient stratification, treatment adherence, and clinical outcomes.

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APPENDIX

APPENDIX I: Demographic and Clinical Characteristics of Study Participants

Characteristic	Overall Cohort (N=100)	Hypertensive Group (N=50)	Normotensive Group (N=50)
Age (Years)	Mean ± SD: 41.59 ± 14.88	Mean ± SD: 41.74 ± 13.91	**Mean ± SD: 41.44 ± 15.89**
	Median: 40.00	Median: 39.50	**Median: 40.00**
	Range: 18-65	Range: 20-65	**Range: 18-65**
Gender (%)	Male: 52%	Male: 56%	**Male: 48%**
	Female: 48%	Female: 44%	**Female: 52%**
Marital Status (%)	Married: 38%	Married: 36%	**Married: 40%**
	Single: 27%	Single: 26%	**Single: 28%**
	Divorced: 22%	Divorced: 24%	**Divorced: 20%**
	Widowed: 13%	Widowed: 14%	**Widowed: 12%**
Educational Level (%)	Secondary: 37%	Secondary: 36%	**Secondary: 38%**
	Tertiary: 35%	Tertiary: 38%	**Tertiary: 32%**
	Primary: 20%	Primary: 18%	**Primary: 22%**
	No formal education: 8%	No formal education: 8%	**No formal education: 8%**
Occupation (%)	Engineer: 28%	Engineer: 32%	**Engineer: 24%**
	Clerk: 20%	Clerk: 20%	**Clerk: 20%**
	Teacher: 16%	Teacher: 14%	**Teacher: 18%**
	Driver: 12%	Driver: 10%	**Driver: 14%**
	Business Owner: 12%	Business Owner: 10%	**Business Owner: 14%**
	Farmer: 12%	Farmer: 14%	**Farmer: 10%**
Systolic BP (mmHg)	Mean ± SD: 158.19 ± 45.47	Mean ± SD: 201.24 ± 29.13	**Mean ± SD: 115.14 ± 9.42**

	Median: 140.00	Median: 198.00	**Median: 114.50**
	Range: 100-249	Range: 146-249	**Range: 100-130**
Diastolic BP (mmHg)	Mean ± SD: 100.32 ± 34.61	Mean ± SD: 130.64 ± 15.68	**Mean ± SD: 70.00 ± 6.16**
	Median: 101.50	Median: 132.00	**Median: 71.00**
	Range: 60-160	Range: 94-160	**Range: 60-79**

APPENDIX II: Summary Statistics of Biomarker Levels in Hypertensive Patients (N=50)

Biomarker	**Mean ± SD**	**Median**	**Range**	**Reference Range**
hsCRP (mg/l)	6.94 ± 8.65	2.80	0.10 - 30.00	1-3 mg/l
MDA level (μM)	0.79 ± 0.08	0.77	0.67 - 0.99	0.36-1.24 μM
TAC (mM)	6.36 ± 2.45	6.75	1.59 - 12.11	0.513-1.5 mM
Globulin (g/dl)	30.86 ± 9.77	31.00	7.00 - 50.00	20-35 g/l
Albumin (g/dl)	35.80 ± 6.06	37.00	13.00 - 46.00	35-50 g/l

Key words: HsCRP- High Sensitive C- reactive protein, **MDA-** Malondialdehyde, **TAC-**Total Antioxidant Capacity

APPENDIX III: Correlation Coefficients between Biomarkers and Hypertension Stage (Hypertensive Group, N=50)

Biomarker	**Hypertension Stage (rho)**	***P-value***
hsCRP (mg/l)	0.565	< 0.001
MDA level (µM)	-0.093	0.518
TAC (mM)	-0.046	0.749
Globulin (g/dl)	0.097	0.499
Albumin (g/dl)	-0.136	0.345

APPENDIX IV: Significant predictor for hsCRP(mg/l)

Predictor	**Coefficient**	**Std. Error**	**t-statistic**	***P-value***
Constant	≈15.9	≈ 5.1	≈ -3.1	0.003
Age	≈ 0.08	≈ 0.03	≈ 2.6	0.011
Gender (Male=1)	≈ 3.2	≈ 1.2	≈ 2.7	0.009
Smoking (Yes=1)	≈ 6.8	≈ 1.3	≈ 5.1	< 0.001
Sugary Drinks/Snacks (Yes=1)	≈ 2.1	≈ 1.0	≈ 2.1	0.038
Other lifestyle factors (non-sig)	(Coefficients varied)	(Non-significant *P-value*s)		

Note: Overall Model $R^2 = 0.344$, F-statistic $p < 0.001$

APPENDIX V: Influence of lifestyle factors on MDA level(µM)

Predictor	**Coefficient**	**Std. Error**	**t-statistic**	***P-value***
Constant	≈ 0.8	≈ 0.05	≈ 16.0	< 0.001
Age	≈ 0.002	≈ 0.001	≈ -3.0	0.003
All other predictors	(Coefficients varied)	(Non-significant *P-value*s)		

Note: Overall model R^2=0.090, F-Statistic p= 0.003

APPENDIX VI: Influence of lifestyle factors on Globulin (g/dl)

Predictor	**Coefficient**	**Std. Error**	**t-statistic**	***P-value***
Constant	≈ 20.0	≈ 3.0	≈ 6.7	< 0.001
Age	≈ 0.2	≈ 0.01	≈ 4.9	< 0.001
All other predictors	(Coefficients varied)	(Non-significant *P-value*s)		

Note: Overall model R^2=0.150, F-Statistic p=< 0.001

APPENDIX VII: Influence of lifestyle factors on Globulin (g/dl)

Predictor	**Coefficient**	**Std. Error**	**t-statistic**	***P-value***
Constant	≈ 20.0	≈ 3.0	≈ 6.7	< 0.001
Age	≈ 0.2	≈ 0.01	≈ 4.9	< 0.001
All other predictors	(Coefficients varied)	(Non-significant *P-value*s)		

Note: Overall model R^2=0.150, F-Statistic p=< 0.001

APPENDIX VIII: Influence of lifestyle factors on Albumin(g/dl)

Predictor	**Coefficient**	**Std. Error**	**t-statistic**	***P-value***
Constant	≈ 42.0	≈ 1.8	≈ 23.3	< 0.001
Age	≈ -0.04	≈ 0.02	≈ -3.0	0.003
Gender (Male=1)	≈ -1.7	≈ 0.8	≈ -2.1	0.038
All other predictors	(Coefficients varied)	(Non-significant *P-value*s)		

Note: Overall model R^2=0..104, F-Statistic p= 0.001

APPENDIX IX: Biomarker Differences Across Hypertension Stages (Hypertensive Group, N=50)

Biomarker	**H-statistic**	***P-value***	**Significant (p < 0.05)?**
hsCRP (mg/l)	8.530	0.014	Yes
MDA level (µM)	0.596	0.742	No
TAC (mM)	0.187	0.911	No
Globulin (g/dl)	1.096	0.578	No
Albumin (g/dl)	2.190	0.335	No

APPENDIX X: Test for hsCRP(mg/l) across Hypertension Stages (Corrected *P-value*s)

Comparison	**Corrected *P-value***
Stage 1 vs. Stage 2	1.000
Stage 1 vs. Stage 3	0.019
Stage 2 vs. Stage 3	0.076

APPENDIX XI: Biomarker Differences between Hypertensive (N=50) and Normotensive (N=50) Groups

Biomarker	**U-statistic**	***P-value***	**Significant ($p < 0.05$)?**
hsCRP (mg/l)	398.000	< 0.001	Yes
MDA level (µM)	1194.500	0.725	No
TAC (mM)	134.000	< 0.001	Yes
Globulin (g/dl)	1242.000	0.963	No
Albumin (g/dl)	1243.500	0.941	No

APPENDIX XII: Multiple Linear Regression Analysis for Hypertension Severity (Dependent Variable: hypertensive stage numeric)

Predictor	**Coefficient**	**Std. Error**	**t-statistic**	***P-value***
Constant	≈ -0.3	≈ 0.3	≈ -1.0	0.316
Age	≈ 0.05	≈ 0.00	≈ 5.1	< 0.001
Gender (Male=1)	≈ -0.01	≈ 0.05	≈ -0.2	0.814
Smoking (Yes=1)	≈ 0.3	≈ 0.06	≈ 5.0	< 0.001
Alcohol Consumption	≈ -0.02	≈ 0.03	≈ -0.7	0.490
Exercise (Yes=1)	≈ 0.01	≈ 0.05	≈ 0.3	0.755
Fruits/Vegetables (Yes=1)	≈ -0.004	≈ 0.05	≈ -0.08	0.938
High Salt Foods (Yes=1)	≈ -0.01	≈ 0.05	≈ -0.2	0.852
Sugary Drinks/Snacks (Yes=1)	≈ 0.1	≈ 0.05	≈ 2.1	0.038
hsCRP(mg/l)	≈ 0.02	≈ 0.00	≈ 5.0	< 0.001
MDA level(µM)	≈ -0.001	≈ 0.002	≈ 0.5	0.627
TAC (mM)	≈ -0.06	≈ 0.01	≈ 4.8	< 0.001
Globulin (g/dl)	≈ 0.000	≈ 0.002	≈ 0.1	0.902

Albumin(g/dl)	≈ 0.000	≈ 0.003	≈ 0.1	0.945

APPENDIX XIII

QUESTIONNAIRE

INFORMED CONSENT FORM

Dear Respondent,

My name is **DAVID OLABISI JOSHUA**, a final-year student in the **Department of Medical Laboratory Science, Faculty of Basic Medical and Health Sciences, Thomas Adewumi University, Oko-Irese, Kwara State, Nigeria.**

I am conducting a research study titled: **"Assessment of Inflammatory and Oxidative Stress Markers in Hypertensive Patients Attending Thomas Adewumi University Teaching Hospital, Omu-Aran, Kwara State."**

Data obtained from this study will provide valuable insights into the significance of inflammatory and oxidative stress markers in hypertensive patients. The information provided in this questionnaire will be used strictly for research purposes and will be treated with confidentiality. Kindly read the instructions carefully and answer each question as honestly and accurately as possible. If you have any concerns or require clarification, please feel free to ask, and I will be happy to assist.

I hereby confirm that I have been fully informed about the research and its purpose. I voluntarily consent to participate in this study and promise to cooperate throughout the research process.

Researcher's Signature: _____

Respondent's Signatue: _____

INSTRUCTIONS:

- Please do not write your name.
- Tick [[ ]] or write your responses in the spaces provided.

SECTION 1: DEMOGRAPHIC INFORMATION

1. Age: _____

2. Gender:

   [ ] Male [ ] Female

3. Marital Status:

   [ ] Single [ ] Married [ ] Divorced [ ] Widowed

4. Educational Level:

   [ ] No formal education [ ] Primary [ ] Secondary [ ] Tertiary

5. Occupation: _____

SECTION 2: MEDICAL HISTORY

6. Have you been diagnosed with hypertension?

   [ ] Yes [ ] No

7. If yes, how long ago?

   [ ] <1 year [ ] 1–5 years [ ] >5 years

8. Do you have a family history of hypertension?

   [ ] Yes [ ] No

9. Are you currently on antihypertensive medication?

   [ ] Yes [ ] No

10. If yes, specify: _____

11. Have you been diagnosed with diabetes?

    [ ] Yes [ ] No

12. Have you been diagnosed with any chronic kidney disease?

    [ ] Yes [ ] No

13. Are you on any anti-inflammatory or antioxidant drugs?

    [ ] Yes [ ] No

    If yes, specify: _____

SECTION 3: LIFESTYLE FACTORS

14. Do you smoke?

    [ ] Yes [ ] No

    If yes, how many cigarettes per day? _____

15. Do you consume alcohol?

    [ ] Yes [ ] No

    If yes, how often:

    [ ] Occasionally [ ] Weekly [ ] Daily

16. How often do you exercise?

    [ ] Never [ ] Occasionally [ ] Regularly

17. What type of exercise do you engage in? _____

SECTION 4: DIETARY HABITS

18. How many meals do you eat per day?

    [ ] 1 [ ] 2 [ ] 3 [ ] More than 3

19. Do you consume fruits and vegetables daily?

    [ ] Yes [ ] No

20. How often do you eat high-salt foods?

    [ ] Rarely [ ] Occasionally [ ] Frequently

21. Do you consume sugary drinks or snacks?

    [ ] Yes [ ] No

    If yes, how often:

    [ ] Occasionally [ ] Weekly [ ] Daily

SECTION 5: STRESS AND SLEEP PATTERNS

22. How many hours of sleep do you get per night?

    [ ] <4 [ ] 4–6 [ ] 7–8 [ ] >8

23. Do you often feel stressed?

    [ ] Yes [ ] No

24. How do you manage stress? _____

25. Have you experienced significant life changes recently?

    [ ] Yes [ ] No

    If yes, please specify: _____

26. SECTION 6: MENTAL HEALTH

27. In the last month, how often have you felt nervous and stressed?

[ ] Never [ ] Almost Never [ ] Sometimes [ ] Fairly Often [ ] Very Often

28. In the last month, how often have you felt confident about handling personal problems?

[ ] Never [ ] Almost Never [ ] Sometimes [ ] Fairly Often [ ] Very Often

29. SECTION 7: MEDICATION ADHERENCE

30. Do you take your prescribed medications regularly?

[ ] Yes [ ] No

31. If no, why?

[ ] Forgetfulness [ ] Side effects [ ] Cost [ ] Other: _____

32. Do you experience any side effects from medications?

[ ] Yes [ ] No

If yes, describe: _____

SECTION 8: KNOWLEDGE AND AWARENESS

33. Are you aware of the complications of untreated hypertension?

[ ] Yes [ ] No

34. Have you received counseling on hypertension management?

[ ] Yes [ ] No

35. If yes, who provided the counseling?

[ ] Doctor [ ] Nurse [ ] Pharmacist [ ] Other: _____

36. Do you know your target blood pressure range?

[ ] Yes [ ] No

37. Do you know your target blood pressure range?

[] Yes [ ] No

SECTION 9: CLINICAL AND BIOCHEMICAL DATA COLLECTION.

38. Blood pressure measurement;

Systolic(mmHg): _____ Diastolic(mmHg): _____

39. Laboratory analysis results:

i. HsCRP(mg/l): _____

ii. MDA Levels(mmol/l): _____

iii. Total protein(mmol/l): _____

iv. Albumin(mmol/l): _____

v. TAC(mmol/l): _____

40. Body measurement:

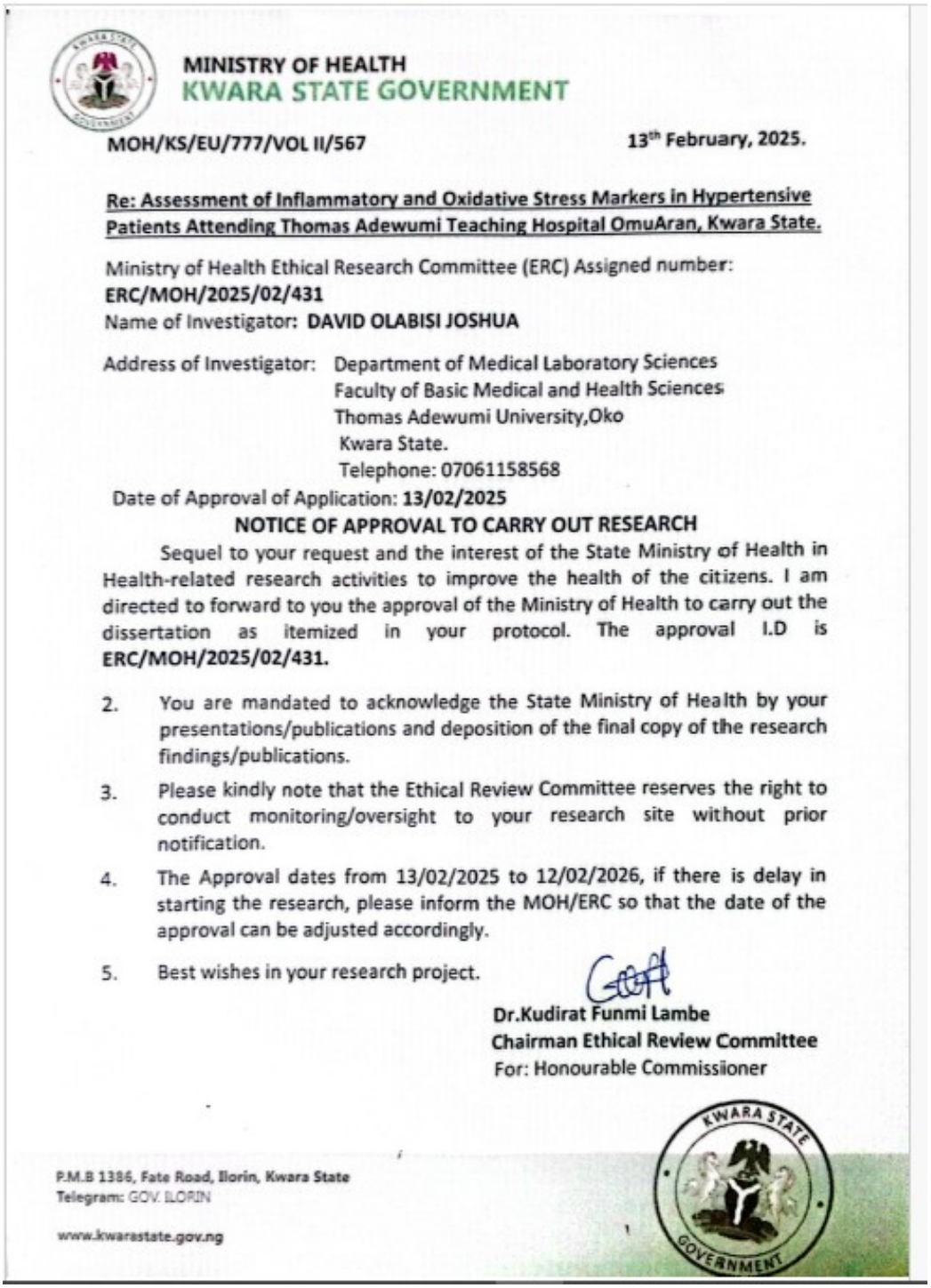
i. Body weight: _____ Kg

ii. Body height: _____ cm

iii. Waist circumference: _____ cm

iv. BMI: _____

APPENDIX XIV



MINISTRY OF HEALTH
KWARA STATE GOVERNMENT

MOH/KS/EU/777/VOL II/567 — 13th February, 2025.

Re: Assessment of Inflammatory and Oxidative Stress Markers in Hypertensive Patients Attending Thomas Adewumi Teaching Hospital OmuAran, Kwara State.

Ministry of Health Ethical Research Committee (ERC) Assigned number:
ERC/MOH/2025/02/431
Name of Investigator: **DAVID OLABISI JOSHUA**

Address of Investigator: Department of Medical Laboratory Sciences
Faculty of Basic Medical and Health Sciences
Thomas Adewumi University,Oko
Kwara State.
Telephone: 07061158568

Date of Approval of Application: **13/02/2025**

NOTICE OF APPROVAL TO CARRY OUT RESEARCH

Sequel to your request and the interest of the State Ministry of Health in Health-related research activities to improve the health of the citizens. I am directed to forward to you the approval of the Ministry of Health to carry out the dissertation as itemized in your protocol. The approval I.D is **ERC/MOH/2025/02/431.**

2. You are mandated to acknowledge the State Ministry of Health by your presentations/publications and deposition of the final copy of the research findings/publications.
3. Please kindly note that the Ethical Review Committee reserves the right to conduct monitoring/oversight to your research site without prior notification.
4. The Approval dates from 13/02/2025 to 12/02/2026, if there is delay in starting the research, please inform the MOH/ERC so that the date of the approval can be adjusted accordingly.
5. Best wishes in your research project.

Dr.Kudirat Funmi Lambe
Chairman Ethical Review Committee
For: Honourable Commissioner

P.M.B 1386, Fate Road, Ilorin, Kwara State
Telegram: GOV. ILORIN

www.kwarastate.gov.ng