

**HUNGER IN THE MIDST OF PLENTY:
THE MANY TANGLES OF DIABETES**

BY

PROF ROSEMARY TEMIDAYO IKEM

413th INAUGURAL LECTURE

PREAMBLE

The Vice-Chancellor, Deputy Vice-Chancellors - Academic, Administration, and Research, Innovation & Development; the Registrar; the University Librarian and the Bursar; Members of the Governing Council; the Provosts of the College of Health Sciences and the Postgraduate College; Deans, Directors, and Heads of Departments; esteemed Members of Senate; my colleagues; staff and the great students of Obafemi Awolowo University; beloved family and cherished friends; distinguished ladies and gentlemen.

I am deeply honoured to stand before you today to deliver the 413th inaugural lecture. It is a significant milestone in any academic's career, and for me, it is both a moment of reflection and a declaration of intent.

In my view, an inaugural lecture is not merely the marking of a personal milestone, but a public commitment to inquiry, integrity, and the lifelong pursuit of knowledge that serves both scholarship and society.

In medicine, we speak of science and service, evidence and empathy, these dualities are not opposites, but serve as complementary pillars of a tradition that has shaped our discipline for millennia. From the ancient temples of healing in Greece and Egypt, to the scholarly halls of Islamic medicine, to the modern teaching hospitals of today, medicine has always been both a science and an art.

Medicine is a tradition passed not just through textbooks, but through hands, hearts, and generations. An inaugural lecture honours that lineage, a moment to step into the stream of those who taught, healed, and discovered before us, and to carry it forward with purpose.

As I reflect on my own journey from the wards to the lecture halls, from late-night research questions to early morning clinical rounds I'm reminded that we do not work in isolation. We stand on the shoulders of giants, and we share the weight of responsibility that comes with this profession.

My own journey began not with certainty, but with curiosity a desire to understand how the body works, how disease can be a disrupt factor, and how compassion can often be as potent as any prescription. Over the years, that curiosity has taken many forms: the clinician's careful listening, the researcher's restless questioning, and the teacher's quiet encouragement.

Who am I and what are my names? I am Rosemary Temidayo IKEM. My middle name has made many people wonder where I am from, I am a Nigerian and proudly so with ties to the 3 major tribes and the rest is a story for another day.

Like most people who studied Medicine, I have always wanted to be a doctor. I gained admission into the University of Jos medical school after a stint of 3 months in the Federal School of Arts & Science Lagos. Like most medical students, what we needed to learn was enormous to us but we persevered and graduated. Having completed the mandatory NYSC, I worked in the Specialist Hospital Kano as a medical officer but my thirst for knowledge made me apply for residency training in different Teaching Hospitals. The story was that I received my interview letter on a Friday, a day after the interview in OAUTHC had taken place, (a letter from UCH also arrived about the same time but the interview had taken place a week prior) there were no emails, or mobile phones then, and how many had landlines in their homes? I decided to brave it and travelled from Kano to Ile Ife, a place I only heard of.

I met nice accommodating people, who when they heard my determination organised an interview for only myself. I am indeed very grateful to Prof Roger Makanjuola (CMD) and late Dr Ige (CMAC), late Prof A. Adeyemo and the others in the panel. So, my journey from a medical officer into residency training commenced in OAUTHC, where I met good people of the heart who were ready to impart knowledge and train us (Emeritus Prof A. Akinsola, Profs MO. Balogun, GE. Erhabor, DA. Ndububa, A. Ajayi, A. Akintomide, and B. Adamolekun). After the completion of residency and passing my examination with defence of my dissertation, I was then awarded the FMCP (EDM) of the NPMCN. While we were considering whether to continue to stay in this semi urban town, I went to work one day and my supervising consultant Prof MO Balogun gave me the key to the office am still occupying and that answered my question. Thank you, sir, for being a channel which God has used to bring a resolution to my concern. Since then, I have also acquired other fellowships, Fellow Endocrine and Metabolism Society of Nigeria (FEMSON), Fellow American College of Endocrinology (FACE), Fellow American College of Physicians (FACP), Fellow Nigeria Academy of Medicine (FNAMed) in my area of specialisation and subspecialty)

Upon my appointment as a Lecturer I in the Department of Medicine, Obafemi Awolowo University, in the year 1999, I had the privilege of being the first female academic to join the Department. In due course, I became its first female Head of Department and, later, its first female Professor dated back to 2010. I am particularly delighted that several other women have since joined the Department, and their number continues to grow.

I had intended to deliver this inaugural lecture a few years ago just before the Covid-19 pandemic; however, life presented a number of challenges along the way. I remain deeply grateful to God for the strength to overcome them and to live beyond those moments. As the Igbo

adage wisely reminds us, “*Anytime you wake up is your morning.*” Indeed, everything in God’s creation is in its rightful place”, we may make our plans, but ultimately, we submit to the will of God. And so, today is that day.

This inaugural lecture is the 413th in the University and the 5th from the Department of Medicine and is titled “**Hunger in the Midst of Plenty: The Many Tangles of Diabetes.**” It is a particularly significant occasion, as it is delivered by the Department’s first female academic Professor. Adding to the uniqueness of the moment, I also celebrated my birthday just yesterday.

INTRODUCTION

WHAT IS ENDOCRINOLOGY?

Endocrinology is the study of hormones and the endocrine glands and organs.

Hormones are chemical substances or messengers that are produced by the endocrine glands or organs and travel to other parts of the body through the blood or fluids surrounding the cells. An endocrinologist is a medical doctor who specialises in the diagnosis and management of disorders of the endocrine system (Fig 1) the network of glands that produce hormones which regulate metabolism, growth, reproduction, and many other body functions.

In simple terms, an endocrinologist treats diseases caused by hormonal imbalance or dysfunction.

Common conditions managed by endocrinologists include but not limited to Diabetes mellitus and other disorders of glucose metabolism; Thyroid disorders; Pituitary disorders; Adrenal disorders; Calcium and bone disorders; Growth and pubertal disorders, Reproductive and Obesity and metabolic endocrine disorders.

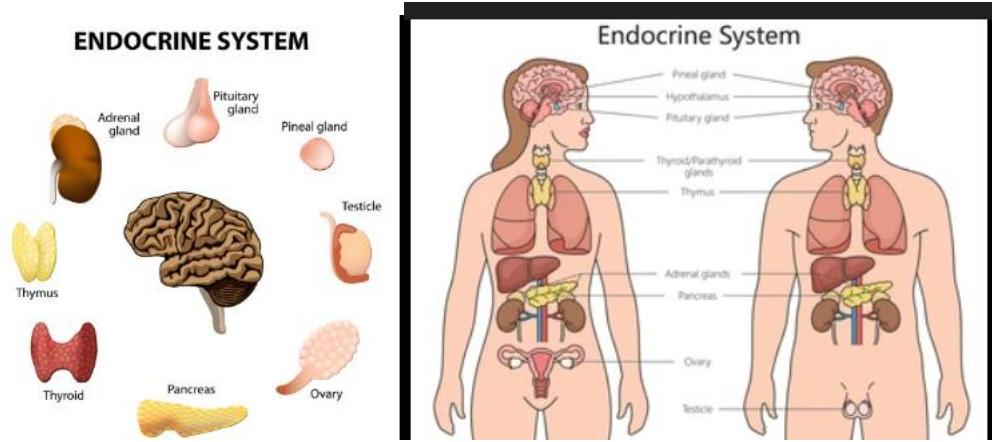


Figure 1: The Endocrine System

This subspecialty of Internal Medicine is referred to as Endocrinology, Diabetes, and Metabolism (EDM). However, it also encompasses Nutrition and for this reason, I may refer to it as EDM and N. As we proceed, you will understand why Nutrition rightfully deserves to be mentioned and included.

PATTERN OF ENDOCRINE DISEASES IN NIGERIA/OAUTHC

Globally, diabetes mellitus is recognized as the most prevalent endocrine disorder in adults. In Nigeria, the burden of endocrine disease is similarly dominated by diabetes, (Fig 2) with tertiary centre data consistently showing that it accounts for the largest share of endocrine morbidity, surpassing all other endocrine disorders (Ogbera, A. O., & Ekpebegh, C. (2014). Using standardized classification systems such as the International Classification of Diseases, Tenth Revision (ICD-10), diabetes emerges as the foremost endocrine condition, reflecting not only its rising prevalence but also its wide-ranging clinical, economic, and societal impact. Data from Ile Ife Endocrine register shows a similar pattern. This inaugural lecture will focus on Diabetes

Mellitus, the major endocrine disorder that has been central to my research endeavours, academic work, and scholarly contributions.

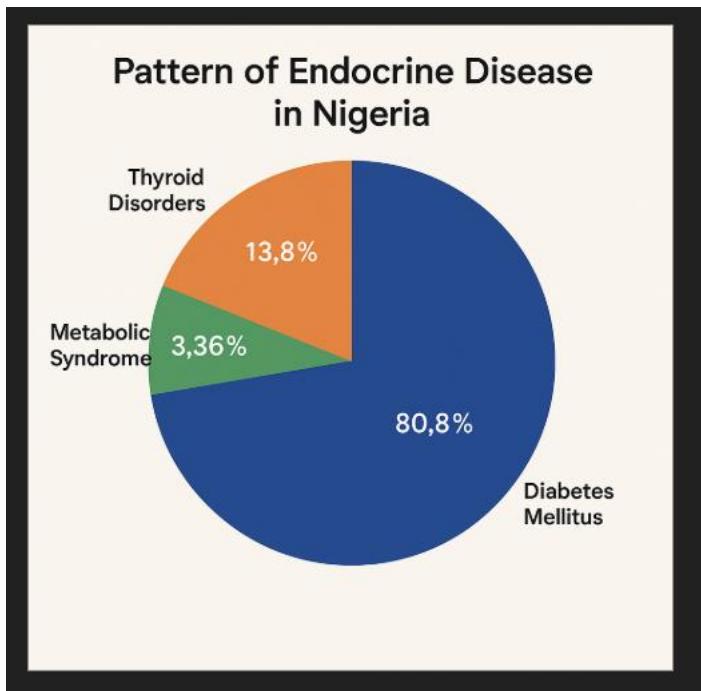


Figure 2: Pattern of Endocrine Disease in Nigeria

HISTORICAL PERSPECTIVE ON THE ORIGINS OF DIABETES MELLITUS

Mr Vice chancellor sir,

To appreciate the full burden of diabetes in the present day, it is instructive to look back at how this condition was recognized across different civilizations and centuries. The earliest known reference to a diabetes-like illness is found in the *Ebers Papyrus*, an ancient Egyptian medical text dating to around 1550 BC. It describes a disorder marked by excessive urination, one of the hallmark features of what we now identify as diabetes mellitus (Bryan, 2010).

Several centuries later, between the 5th and 6th centuries BC, Ayurvedic physicians in India observed that the urine of certain individuals was unusually sweet and attracted ants. They termed this condition *Madhumeha*, meaning “honey urine”, a remarkably perceptive clinical observation for its time (Krall, 2002).

In the 2nd century AD, Aretaeus of Cappadocia, a Greco-Roman physician, provided one of the earliest comprehensive descriptions of diabetes. He depicted it as “a melting down of the flesh and limbs into urine,” vividly capturing the severe weight loss and polyuria that remain diagnostic features till today (Papadakis & McPhee, 2019).

It was not until the 17th century that the condition acquired the full name by which it is known today. Thomas Willis, an English physician, noted the distinctive sweetness of the urine and appended the term *mellitus* Latin for “honey” to differentiate it from *diabetes insipidus*, an unrelated disorder that also causes excessive thirst and passage of large volumes of dilute urine (Williams & Pickup, 2004). These historical milestones highlight not only the remarkable continuity of clinical observation across cultures and eras but also the gradual evolution of our understanding of diabetes mellitus as a systemic progressive disease, a concept that continues to inform clinical practice and research today.

Diabetes mellitus(simply, **diabetes**) is a disease in which the body is unable to properly control the level of **glucose (sugar)** in the blood, leading to an excessive amount of sugar being passed out in the urine. As a result, the kidneys produce **large volumes of urine**.

In major Nigerian languages, diabetes is commonly referred to as:

- **Igbo:** *Oria mamiri*
- **Yoruba:** *Arun itò suga (Àtògbe)*
- **Hausa:** *Ciwon sukari*

(Adapted from Wikipedia, 2025; Google Translate, 2025)

Definition and classification of Diabetes Mellitus

Diabetes mellitus is a chronic metabolic disorder characterized by persistent hyperglycaemia resulting from defects in insulin secretion, insulin action, or both. The condition leads to disturbances in carbohydrate, protein, and fat metabolism, and is associated with dysfunction and long-term damage affecting multiple organ systems (ADA 2014).

Classification of Diabetes Mellitus

The classification of diabetes mellitus has evolved considerably over the decades, reflecting advances in our understanding of its causes, clinical features, and metabolic characteristics. The World Health Organization (WHO) published its first formal classification system in the mid-20th century, and subsequent revisions have continued to refine the categories.

- 1965 WHO Classification:

The earliest WHO system categorized diabetes based on the age at diagnosis. Four groups were identified:

- Infantile or childhood onset (0–14 years)
- Young onset (15–24 years)
- Adult onset (25–64 years)
- Elderly onset (65 years and above)

In addition, WHO recognized several descriptive forms of the disease, including juvenile-type, brittle, insulin-resistant, gestational, pancreatic, endocrine, and iatrogenic diabetes (WHO, 1965).

- 1985 WHO Update:

The 1985 revision introduced two major etiological classes:

- Insulin-Dependent Diabetes Mellitus (IDDM) corresponding to what is now known as Type 1 diabetes, and
- Non-Insulin-Dependent Diabetes Mellitus (NIDDM) corresponding to Type 2 diabetes.

Although the terms Type 1 and Type 2 were not formally used in that document, the distinction between insulin dependence and non-insulin dependence was emphasized.

A third category, Malnutrition-Related Diabetes Mellitus (MRDM), was also introduced to describe forms of diabetes associated with undernutrition in tropical regions (WHO, 1985).

- 1999 WHO Revision:

In 1999, WHO recommended that the classification should reflect both aetiological types and clinical stages of diabetes. The term MRDM was dropped at the time due to insufficient evidence to support its existence as a distinct entity (WHO, 1999).

- 2019 WHO Classification: With the new classification of diabetes, the spectrum of diabetes subtypes and the role of precision medicine in tailoring treatment strategies based on genetic, phenotypic, and environmental factors (Table 1). This invites discussion on evolving perspectives on diabetes classification.

Table 1 – 2019 WHO Classification of Diabetes

Type of diabetes	Description
Type 1 diabetes	β-cell destruction (mostly immune-mediated) and absolute insulin deficiency; onset most common in childhood and early adulthood

Type 2 diabetes	Most common type, various degrees of β -cell dysfunction and insulin resistance; commonly associated with overweight and obesity
Hybrid forms of diabetes	
Slowly evolving, immune-mediated diabetes of adults	Similar to slowly evolving type 1 in adults but more often has features of the metabolic syndrome, a single GAD autoantibody and retains greater β -cell function
Ketosis-prone type 2 diabetes	Presents with ketosis and insulin deficiency but later does not require insulin; common episodes of ketosis, not immune-mediated
Other specific types	
Monogenic diabetes - Monogenic defects of β -cell function - Monogenic defects in insulin action	Caused by specific gene mutations, has several clinical manifestations requiring different treatment, some occurring in the neonatal period, others by early adulthood. Caused by specific gene mutations; has features of severe insulin resistance without obesity; diabetes develops when β -cells do not compensate for insulin resistance
Diseases of the exocrine pancreas	Various conditions that affect the pancreas can result in hyperglycaemia (trauma, tumour, inflammation, etc.)

Endocrine disorders	Occurs in diseases with excess secretion of hormones that are insulin antagonists
Drug- or chemical-induced	Some medicines and chemicals impair insulin secretion or action, some can destroy β -cells
Infection-related diabetes	Some viruses have been associated with direct β -cell destruction
Uncommon specific forms of immune-mediated diabetes	Associated with rare immune mediated diseases
Other genetic syndromes sometimes associated with diabetes	Many genetic disorders and chromosomal abnormalities increase the risk of diabetes
Unclassified	Describes diabetes that does not clearly fit into other categories. This category should be used temporarily when there is not a clear diagnostic category especially close to the time of diagnosis
<p>Hyperglycaemia first detected during pregnancy</p>	
DM in pregnancy	Type 1 or type 2 diabetes first diagnosed during pregnancy
Gestational DM	Hyperglycaemia below diagnostic thresholds for diabetes in pregnancy

- 2025 IDF/WHO Update:

The most recent classification (IDF & WHO, 2025) reintroduces Malnutrition-Related Diabetes Mellitus (MRDM) as a recognized category, now formally designated as Type 5 Diabetes Mellitus (T5DM). This form is characterized by diabetes occurring in individuals with a history of undernutrition, most commonly in low- and middle-income countries.

The new classification of diabetes broadens the spectrum of recognized subtypes and highlights the role of precision medicine in tailoring treatment to genetic, phenotypic, and environmental factors. This shift invites further discussion on evolving perspectives in diabetes classification, particularly with the inclusion of malnutrition-related diabetes (MRDM) in the 2025 framework. These successive revisions reflect the dynamic and evolving understanding of diabetes as a heterogeneous group of metabolic disorders, shaped by both genetic and environmental influences.

Diabetes is a chronic health condition that affects how the body converts food into energy.

Normally, the body breaks down most of the food we eat into sugar (glucose), which is then released into the bloodstream to provide energy for daily activities.

Paradoxically, despite having an excess of glucose circulating in the blood, individuals living with diabetes often experience **weakness, fatigue, unexplained weight loss, and increased appetite** at the time of diagnosis. This paradox has led to the descriptive phrase “**hunger in the midst of plenty.**”

EPIDEMIOLOGY OF DIABETES MELLITUS

Global and Regional Epidemiology of Diabetes

Global Burden (Fig 3)

Diabetes is a rapidly growing global public health problem, especially in low- and middle-income countries. In 2025, about 589 million adults worldwide nearly one in nine were living with diabetes, many of them undiagnosed, which hinders early prevention and effective care (IDF, 2025). By 2050, prevalence is projected to rise to 853 million adults, underscoring the escalating global burden of the disease.

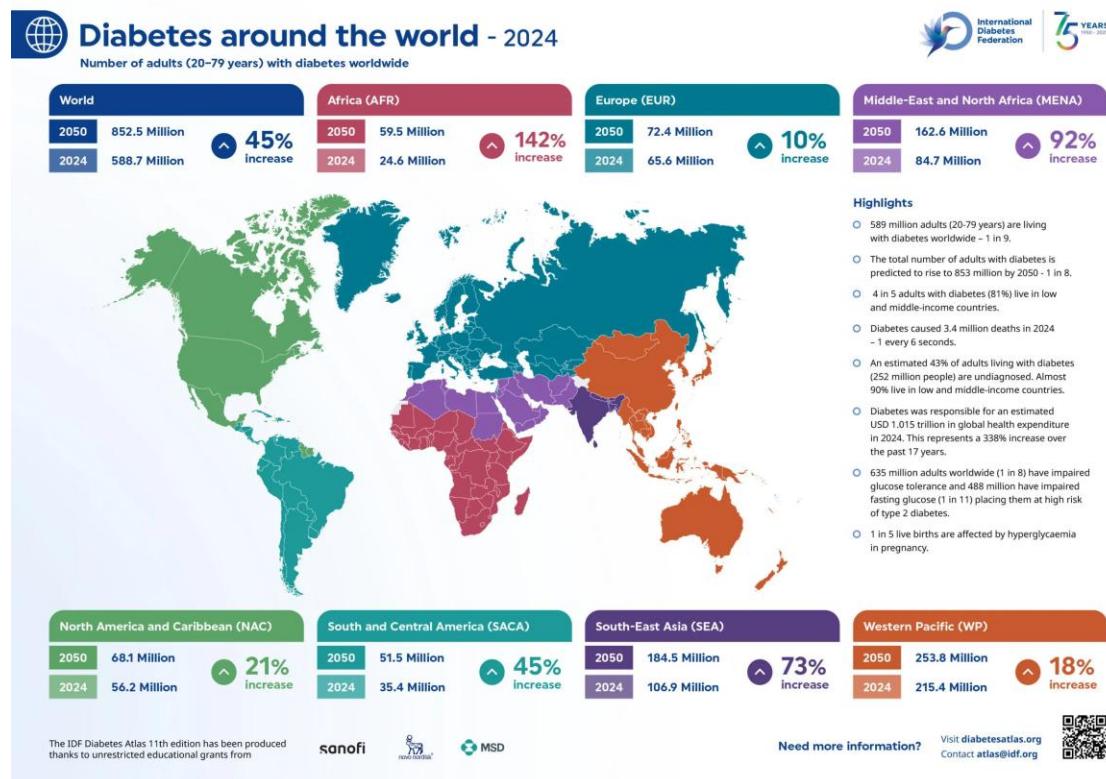


Figure 3: Map of Diabetes around the world

Regional Burden (Africa) (Table 2)

Across Africa, Diabetes prevalence is steadily increasing, largely due to urbanization, sedentary lifestyles, dietary transitions, and limited healthcare access. The continent has the highest proportion of undiagnosed diabetes globally, with about 54% of affected individuals unaware of

their condition (IDF, 2025). Although rates remain lower in rural areas, urban populations are disproportionately affected, underscoring the metabolic health impact of modernization.

Table 2: Estimated number of people living in Africa with diabetes, prevalence and deaths due to diabetes (Source: IDF, Diabetes Atlas 2021, 10th edition, 2022)

Adult population in Africa	2021	2030	2045
Aged 20–79 years	527m	696m	1.05b
Diabetes (20–79 years)	2021	2030	2045
Regional prevalence	4.5%	4.8%	5.2%
Age-adjusted comparative prevalence	5.3%	5.5%	5.6%
Number of people with diabetes	24m	33m	55m
Number of deaths due to diabetes	416,000	-	-

m=million, b=billion

Within Africa, Nigeria is facing a rapidly growing diabetes epidemic. IDF estimates indicate an adult prevalence of about 3.0–3.7%, translating to approximately 3–3.6 million adults aged 20–79 years living with diabetes in 2024, reflecting a significant and rising national health burden. (Table 3)

Table 3: Number of people living with diabetes in Africa (IDF Atlas 10th edition)

Top five countries for number of people with diabetes (20–79 years)	2011	2021
South Africa	1.9m	4.2m
Nigeria	3.1m	3.6m
United Republic of Tanzania	472 900	2.9m
Ethiopia	1.4m	1.9m
Democratic Republic of the Congoⁱ	730 700	1.9m

ⁱbased on extrapolation from similar countries

m=million b = billion

Burden and Prevalence of Diabetes in Nigeria

Systematic reviews and meta-analyses suggest that the true burden may be considerably higher.

Key findings include:

- 1992 national Survey (Akinkugbe et al.) : prevalence 2.2%
- 2018 meta-analysis (Uloko et al.) : pooled prevalence 5.77% (Fig. 4) with

Regional variations: South-West averages 5.5% (Adeloye et al., 2018)

- Recent review (Olamoyegun et al., 2024): estimated prevalence of 7% (95% CI: 5–9%), suggesting roughly 1 in 14 adults are affected

Additionally, nearly half of adults with diabetes remain undiagnosed or untreated, and an estimated 9.4 million Nigerian adults have impaired glucose tolerance, placing them at high risk of developing type 2 diabetes mellitus (T2DM), which accounts for over 90% of all cases. These data highlight a growing burden of diabetes in Nigeria, particularly in the Southwestern region including Ile-Ife, and underscore the urgent need for enhanced early detection, preventive strategies, and health-system strengthening to reduce morbidity and mortality.

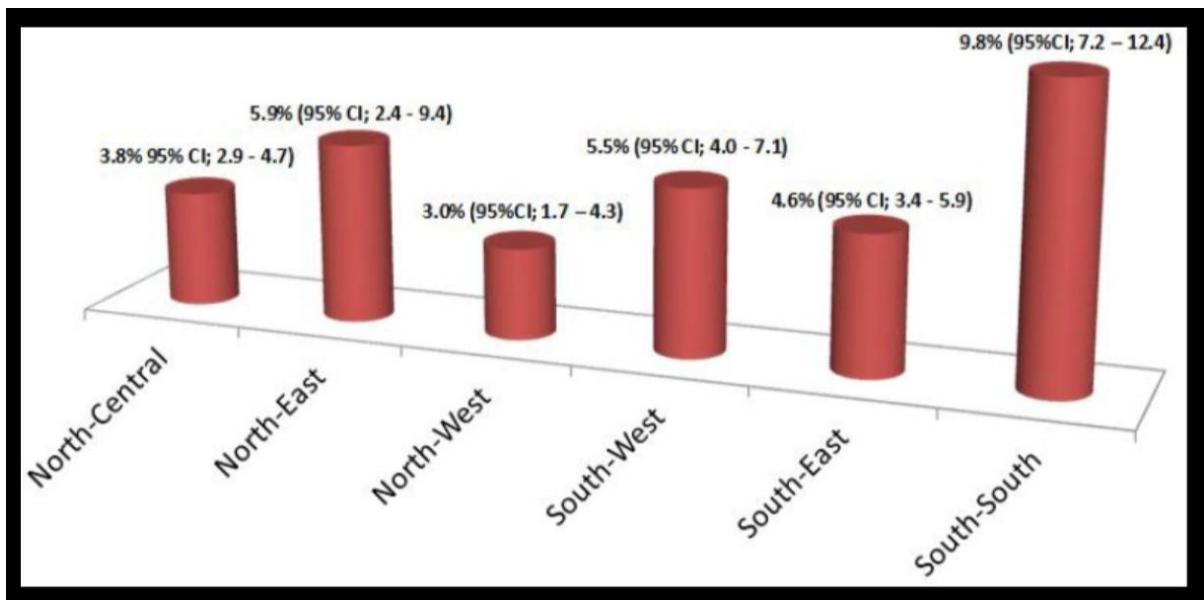


Figure 4: Overall prevalence of DM in Nigeria (Uloko et al 2018)

Current Diagnostic Criteria for Diabetes Mellitus

The diagnosis of diabetes mellitus is based on established biochemical thresholds that reflect persistent hyperglycaemia. According to the World Health Organization (WHO, 2023) and the International Diabetes Federation (IDF, 2025), the diagnostic criteria are as follows:

- Fasting Plasma Glucose (FPG):
 - Normal: ≤ 5.5 mmol/L (≤ 99 mg/dL)
 - Prediabetes: 5.6–6.9 mmol/L (100–125 mg/dL) — also referred to as impaired fasting glucose (IFG)
 - Diabetes Mellitus: ≥ 7.0 mmol/L (≥ 126 mg/dL) on two separate occasions
- Glycated Haemoglobin (HbA1c):

- Normal: < 5.7%
- Prediabetes: 5.7–6.4%
- Diabetes Mellitus: $\geq 6.5\%$
- Random Plasma Glucose (RPG):
 - A random blood glucose level ≥ 11.1 mmol/L (≥ 200 mg/dL) in the presence of classic symptoms of diabetes such as polyuria, polydipsia, and unexplained weight loss is diagnostic of diabetes mellitus.

These criteria provide a standardized framework for the early identification and classification of individuals at risk, enabling timely intervention and prevention of complications.

These diagnostic thresholds are not just numbers; they represent a vital opportunity for early detection and prevention. In our setting, where many people rarely undergo routine medical screening, diabetes often remains undiagnosed until complications arise. By the time a patient presents with symptoms, organ damage may already have occurred. Hence, Diabetes is also often referred to as a **silent killer**, because many people remain unaware of their condition for years.

Symptoms may be subtle or absent until complications develop. By the time these complications become apparent, they can impose a significant physical, emotional, and socioeconomic burden on affected individuals and their families.

This underscores the need for greater public awareness, community-based screening, and strengthened primary healthcare systems. Early identification of prediabetes and prompt management can significantly reduce the future burden of diabetes in Nigeria particularly here in Ile-Ife and the South-West region, where the prevalence continues to rise.

PATHWAYS OF COMPLICATIONS OF DIABETES MELLITUS

Mr. Vice Chancellor, Sir,

The metaphor, “hunger in the midst of plenty” captures the paradox of diabetes complications.

The body has an abundance of glucose circulating in the blood, yet the tissues and cells cannot use it effectively (due to insulin deficiency or resistance). This leads to a state of cellular starvation in the midst of systemic abundance. Because insulin is either deficient or resisted, glucose cannot be used effectively for energy. This “misplaced plenty” forces cells to seek alternative metabolic routes. These routes are not innocuous, they create toxic by-products, metabolic stress, and tissue damage, which underlie the microvascular and macrovascular complications of diabetes.

It is like a city flooded with food supplies but whose people cannot access them, the tissues of the body are surrounded by sugar but starved of its proper use. In trying to cope, cells divert excess glucose into alternative biochemical pathways. These detours are far from harmless; they generate toxic by-products that trigger a cascade of damage, ultimately leading to the well-known complications.

1. The Polyol Pathway (the Sorbitol–Fructose Trap): In the setting of excess glucose, cells that do not depend on insulin for glucose entry (nerves, retina, kidneys) allow glucose to enter freely. Instead of going through normal glycolysis, excess glucose is shunted into the polyol pathway, where it is reduced to sorbitol (an alcoholic sugar) by the enzyme aldose reductase. Sorbitol accumulates, because it is poorly diffusible, it leads to an Osmotic stress (cell swelling, particularly in lens → cataract formation). This represents one of the situations where cells are overwhelmed by "too much of what they cannot properly use."

2. Advanced Glycation End Products (AGEs): Glucose in excess undergoes a non-enzymatic glycation binding of proteins, lipids, and nucleic acids. These AGEs crosslink with

structural proteins (collagen, basement membranes), making tissues stiff, amplifying oxidative stress and inflammation and making them dysfunctional. This leads to thickened capillary basement membranes, endothelial dysfunction, and impaired healing that is a "toxic sweetness" that corrodes tissues over time.

3. Protein Kinase C (PKC) Activation: Hyperglycaemia increases diacylglycerol (DAG) levels, which activate PKC. This causes Increased vascular permeability, altered blood flow (via endothelin ↑, nitric oxide ↓), enhanced angiogenesis (abnormal, leaky vessels in retinopathy). This pathway demonstrates how excess glucose hijacks normal signalling, creating "false alarms" that damage the vasculature.

4. Hexosamine Pathway: Excess glucose is diverted into the hexosamine pathway, leading to abnormal glycosylation of proteins involved in gene expression. Again, the abundance of glucose becomes misused, rewriting the genetic instructions of the cell in harmful ways. "The Tangles of Diabetes in the Midst of Plenty" is not poetic, it captures the essence of diabetic complications. (Fig 5)

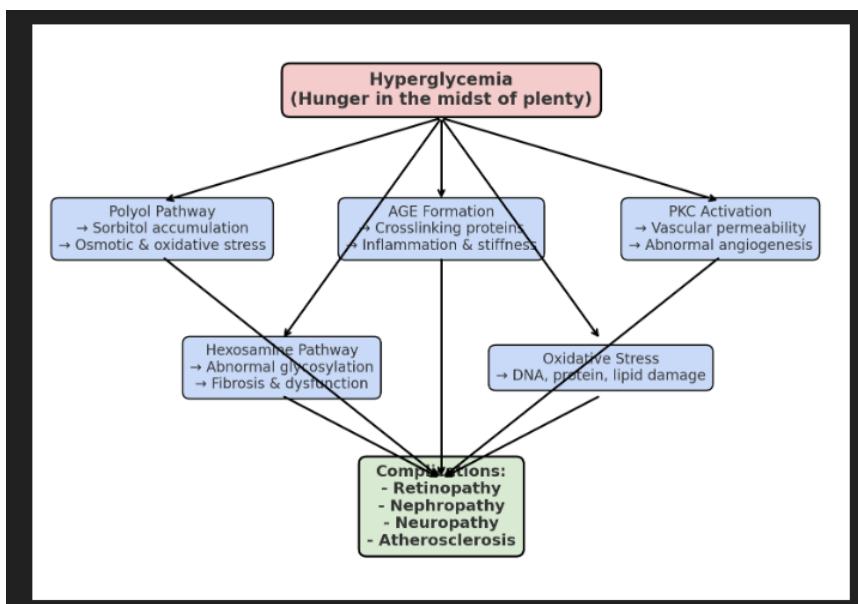


Figure 5: Pathways of DM Complications

Types of Diabetic Complications

The complications of DM can be acute or chronic. The long- term complications of diabetes can be broadly categorized into two groups: microvascular and macrovascular complications.

Microvascular complications include diabetic retinopathy, nephropathy, and neuropathy, while macrovascular complications include cardiovascular diseases such as coronary artery disease, stroke, and peripheral arterial disease conditions to which people living with diabetes are two to four times more susceptible. (IDF, 2025; World Heart Federation, n.d.).

The burden of diabetes is largely driven by its microvascular complications. Diabetic retinopathy results from chronic retinal vascular damage and remains a leading cause of preventable blindness in working-age adults (Centers for Disease Control and Prevention, 2024). Diabetic nephropathy, arising from sustained hyperglycaemia, progresses to chronic kidney disease and may culminate in end-stage renal failure. Diabetic neuropathy leads to pain, sensory loss, and weakness, particularly in the lower limbs, predisposing to foot ulcers, infections, and limb amputation. Beyond vascular injury, diabetes also worsens outcomes in infectious diseases such as tuberculosis and COVID-19, contributing significantly to overall morbidity and mortality.

MY CONTRIBUTIONS TO KNOWLEDGE

Clinical Practice: The Human Encounter - At the heart of medicine is the patient encounter - a sacred space shaped not only by clinical data, but by stories, fears, and hopes. I have come to understand that a physician's greatest instrument is not technology, but the doctor's presence, the capacity to witness suffering and respond with both competence and compassion.

Research: My research has been guided by questions that matter across the laboratory, the clinic, and interdisciplinary collaborations. I have learned that medicine must never become complacent; we owe our patients and students a commitment to curiosity and continual inquiry. In framing this inaugural lecture, I have sought to examine diabetes as far more than a disorder of glucose. My work explores its systemic effects on the heart, kidneys, eyes, and nervous system, and its intersection with obesity, hypertension, and dyslipidaemia, with particular attention to complications, risk factors, and opportunities for improved care. Diabetes may begin with numbers, but it does not end there, it is a systemic disease that rewrites the body's story, revealing its true burden in the tangled complications that affect function, mobility, and the psychological fabric of life.

My milk tooth in research was cut as a Registrar in cardiology unit when I was given an assignment by one of my supervising consultants - Prof A.A Ajayi on a new medication for treatment of heart failure which then led to the publication of the article titled "A three- year clinical review of the impact of angiotensin converting enzyme inhibitors on the intra hospital mortality of congestive heart failure in Nigerians "(Adewole, A.D, **Ikem, R.T**, et al (1996). We examined the outcomes of patients with congestive heart failure (CHF), comparing those treated with angiotensin-converting enzyme (ACE) inhibitors to those who did not receive such therapy. Our aim was to assess the real-world impact of these drugs on patient survival within the hospital setting. Drawing from a retrospective analysis conducted between 1992 and 1994 in Nigeria, we observed a steady rise in the use of ACE inhibitors particularly enalapril and captopril over the study period. Notably, patients who received these medications consistently demonstrated lower mortality rates than those managed with conventional therapies alone. An additional observation of clinical interest was the association between hyponatremia and

increased mortality. In several cases, the correction of hyponatremia with ACE inhibitor therapy suggested a favourable neurohormonal response. These findings not only reaffirm the central role of ACE inhibitors in the management of heart failure but also called attention to the pressing need for their wider adoption in routine clinical practice especially in Nigeria and among Black African populations where the burden of heart failure remains significant.

This experience set the stage for my extensive collaborations with colleagues across my unit, department, and the College of Health Sciences in advancing the management of diabetes.

1. DIABETES MELLITUS AND OBESITY

Obesity is a global health concern and a major risk factor for non-communicable diseases (NCDs), requiring multidisciplinary management.

In terms of definition, obesity is a medical condition characterised by excessive accumulation of body fat that increases the risk of various health complications. It is typically defined using the **Body Mass Index (BMI)**, where:

- **BMI $\geq 30 \text{ kg/m}^2$** indicates obesity
- **BMI $25\text{--}29.9 \text{ kg/m}^2$** is classified as overweight

In simple terms, it is when a person has too much body fat, which can lead to health problems like diabetes, heart disease, and joint pain. It usually happens when people consume more calories than they burn through activity and exercise.

Causes of Obesity:

1. **Genetic factors** – Family history influences metabolism and fat storage.
2. **Dietary habits** – High-calorie intake, processed foods, and sugary drinks contribute.

3. **Physical inactivity** – Sedentary lifestyles reduce calorie expenditure.
4. **Hormonal and metabolic disorders** – Conditions like hypothyroidism and insulin resistance.
5. **Psychological factors** – Stress, emotional eating, and lack of sleep can contribute.

Health Risks of Obesity:

- Type 2 diabetes
- Hypertension and cardiovascular diseases
- Stroke
- Certain cancers (e.g., breast, colon)
- Osteoarthritis and joint problems

One of our studies explored the perception of body size and the rising prevalence of overweight and obesity among Nigerian university undergraduates (**Ikem, R.T et al.**, 2010). The findings revealed a notable mismatch between actual body weight and self-perception. Many students either underestimated or overestimated their weight status. This misperception, coupled with lifestyle factors such as poor dietary habits and physical inactivity, contributed to a significant prevalence of overweight and obesity within this population. These results point to an urgent need for targeted health education and awareness initiatives focused on body image, nutrition, and the long-term health risks associated with obesity. By addressing these gaps early, we can foster healthier behaviours and empower young adults to make informed choices about their well-being.

In another one of our studies, (**Ikem, R.T et al.**, 2001) we investigated the prevalence of obesity among Nigerian patients living with type 2 diabetes mellitus (T2DM). The research focused on

key anthropometric parameters including body mass index (BMI), waist-to-hip ratio (WHR), and other body measurements to examine their association with the onset and progression of diabetes. The study, which involved 178 participants most of whom were females with a mean age of 60.7 years revealed a high prevalence of both generalised obesity ($BMI > 30 \text{ kg/m}^2$) and abdominal obesity.

These findings were particularly concerning, as central obesity emerged as a common feature among many newly diagnosed diabetic patients, underscoring its role as a major risk factor for insulin resistance and other metabolic complications. The study reinforced the critical need for early anthropometric assessments in diabetes care, as well as the promotion of lifestyle modifications aimed at reducing abdominal adiposity. Addressing these risk factors early offers a valuable opportunity to improve outcomes and reduce the burden of obesity-related complications in our diabetic population.

In trying to see what drives obesity in Nigerians with type 2 diabetes we studied “Relationship between Serum Leptin and Adiponectin Levels in Obese Nigerians with Type 2 Diabetes Subjects” (**Ikem R.T 2023**), and observed that obese patients showed elevated leptin and reduced adiponectin levels, reflecting adipose dysfunction, insulin resistance, and the metabolic imbalance underlying type 2 diabetes.

Leptin, is a hormone predominantly secreted by adipose (fat) tissue, it plays a central role in regulating appetite, energy expenditure, and metabolic processes (Fig 6). It exerts its effects by acting on the hypothalamus, where it suppresses hunger and promotes the utilization of energy, thereby contributing to the maintenance of body weight and metabolic balance. In the context of obesity, however, leptin's regulatory role becomes impaired. Although leptin levels are elevated in obese individuals a condition known as hyperleptinaemia the body's response to the hormone

is often blunted. This phenomenon, referred to as leptin resistance, results in a failure of the brain to recognize satiety signals, leading to increased food intake, progressive weight gain, and the development of metabolic disturbances such as insulin resistance and type 2 diabetes mellitus (T2DM).

Clinically, elevated leptin levels in obesity reflect increased fat mass, yet this elevation does not translate to enhanced metabolic regulation due to the presence of leptin resistance. This resistance has been strongly associated with the pathophysiology of obesity, metabolic syndrome, and T2DM.

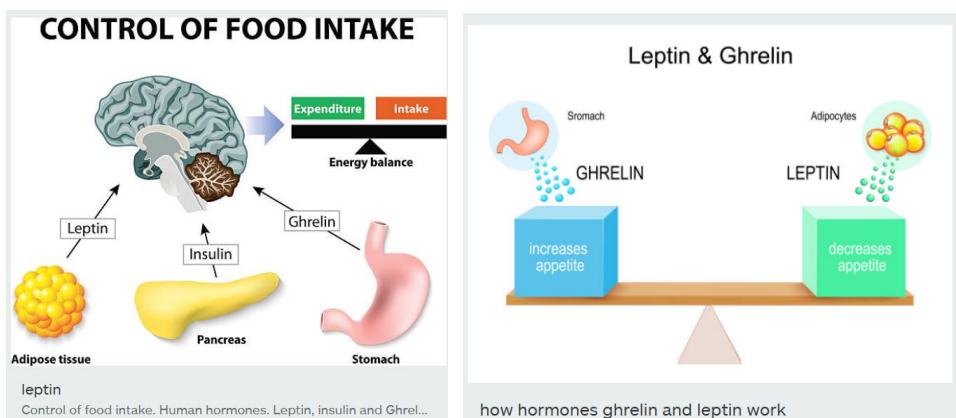


Figure 6: Effect of Leptin on food intake

In another of our study of obese Nigerians living with T2DM, we examined two key hormones secreted by fat tissue leptin (Ajani GOD... **Ikem RT** 2018) and adiponectin (Ezekpo OO, **Ikem RT**, 2022) and their links to cardiometabolic risk (Table 4 & Figure 7). These hormones, often called *adipokines*, act as messengers between fat, metabolism, and the cardiovascular system. We found the serum leptin levels were high, and, as expected, they rose in tandem with measures of body fat and insulin. Adiponectin, on the other hand, tended to be lower, but where present it was

associated with a more favourable lipid profile, especially higher HDL cholesterol in those with diabetes.

Importantly, when we compared obese individuals with and without diabetes, the overall levels of leptin and adiponectin were not very different. This suggests that it is obesity rather than diabetes alone that is the stronger driver of these adipokine patterns.

In summary: in obese Nigerians with type 2 diabetes, leptin rises with adiposity, adiponectin aligns with the “good cholesterol,” and together they underscore how obesity, more than blood sugar, shapes the hormonal landscape that fuels cardiometabolic risk.

Table 4: Relationship between serum leptin level with indices of obesity

Parameter	Obese DM		Obese non-DM	
	R value	P value	R value	P value
BMI	+0.038	0.776	+0.281*	0.030
WC	-0.25	0.849	+0.237	0.068
Serum insulin	-0.077	0.558	+0.446*	0.0001

r = Spearman's simple correlation coefficient, *p < 0.05 is statistically significant

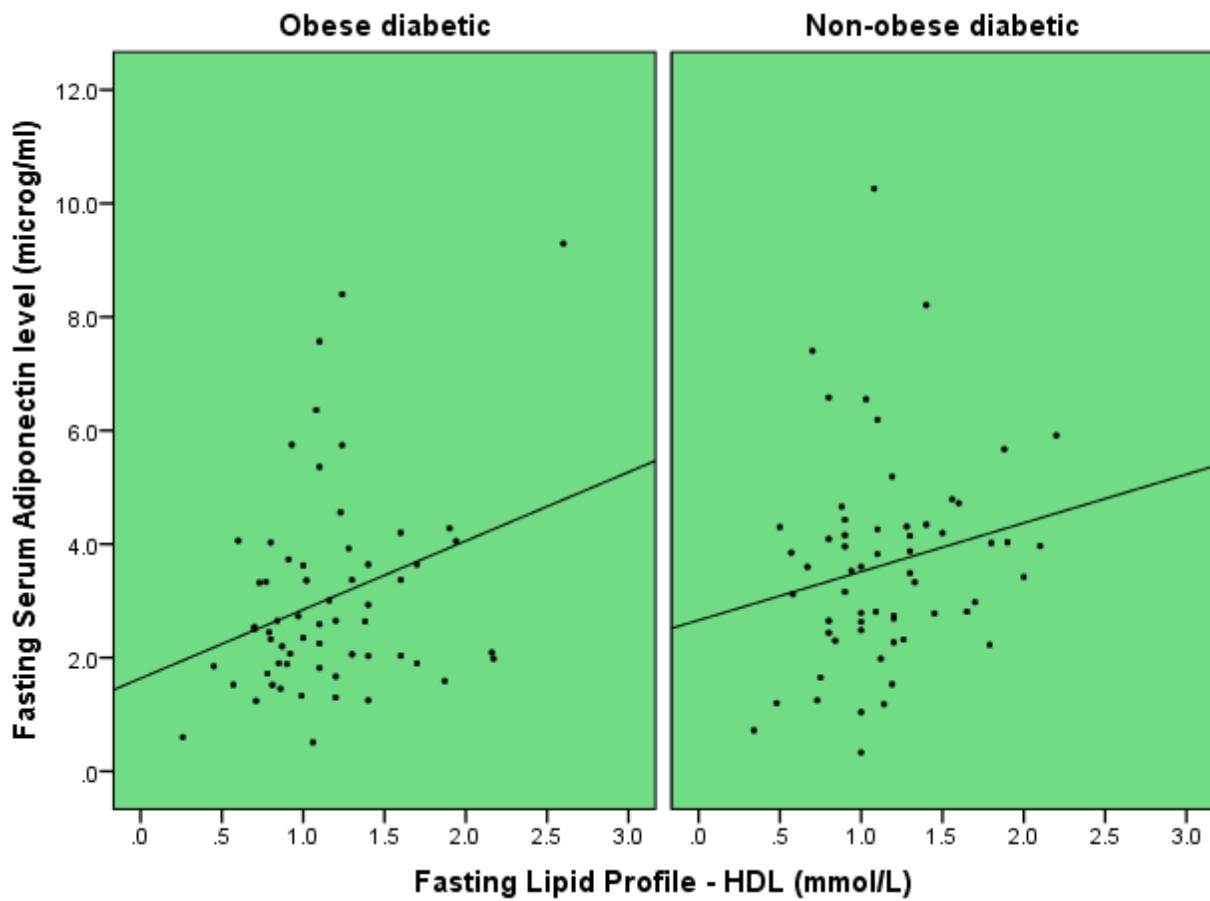


Figure 7: Scattered graph of Serum Adiponectin and Serum Lipid in obese and non obese DM participants

2. THE BURDEN OF COMPLICATIONS OF DIABETES MELLITUS

Indeed, diabetes rarely occurs in isolation. It is the complications that define the burden. It is frequently accompanied by obesity, hypertension, and dyslipidaemia each amplifying its deleterious effects. Together, these factors drive the widespread occurrence of both microvascular and macrovascular complications. Vision is threatened by diabetic retinopathy, renal function declines with nephropathy, sensation diminishes through neuropathy, and the

lower limbs suffer from peripheral arterial disease. These complications stand as stark reminders of the profound toll diabetes exerts on individuals, families, and society at large.

In two cross-sectional studies of type 2 diabetes mellitus conducted at OAUTHC Ile-Ife, Nigeria, we evaluated the prevalence of both microvascular and macrovascular complications (Enikuomehin AC, et al 2020; Ikem RT et al, 2022). The findings revealed a high burden of complications, with diabetic neuropathy, retinopathy, nephropathy, and macrovascular conditions such as peripheral arterial disease and left ventricular hypertrophy. (Tables 5 & 6) Further regression analyses identified advancing age and increased waist circumference as significant predictors of these complications, underscoring the need for targeted risk-factor management in diabetes care.

Table 5: Frequency of microvascular complications in participants

Variables	Total (%) N = 400	p- value	χ^2	df
Neuropathy				
Present	338(82)	0.175	1.84	1
Absent	72(18)			
RETINOPATHY				
No apparent DR	217(54.25)	0.087	10.97	1
Diabetic Retinopathy	183(45.75)			
NEPHROPATHY				
Albumin in urine No -albuminuria	224(56)	0.938	3.07	1
Microalbuminuria	176(44)			
DR - diabetic retinopathy				

Table 6: Distribution of macrovascular complications in participants

Variables	Total 400 (%)	p - value	df	χ^2
Peripheral arterial diseases intermittent claudication				
Present	81(20.25)	0.104	1	2.64
Absent	319 (79.75)			
ABI				
Normal (>0.9)	230(57.5)	0.519	1	
Mild (0.7-0.9)	149(37.25)			
Moderate (0.4-0.69)	21(5.25)			
Severe (<0.4)	0(0)			
Cerebrovascular diseases TIA				
Present	5(1.25)	0.735	1	0.48
Absent	395(98.75)			
Stroke				
Present	16(4)	0.066	1	3.38
Absent	384(96)			
ECG findings for IHD				
Present	37(9.25)	0.753	1	0.10
Absent	363(90.75)			
Central chest pain				
Yes	4(1)	0.920	1	0.10
No	396(99)			

3. THE KIDNEY AND DIABETES

The kidney is both a regulator of glucose and vulnerable target of diabetes. Beyond its familiar role in excretion, the kidney serves as a vital regulator of glucose homeostasis - filtering, reabsorbing, and even producing glucose. In the diabetic state, chronic hyperglycaemia and metabolic disturbances gradually injure renal structures, culminating in diabetic nephropathy. Conversely, when the kidney becomes dysfunctional, glucose regulation worsens.

Diabetic nephropathy (DN) is a common microvascular complication of diabetes and the leading cause of end-stage kidney disease globally. In Nigeria, it ranks third. DN begins with albuminuria, affects 30–40% of people living with diabetes, and increases cardiovascular risk, with rising prevalence in sub-Saharan Africa.

In our study on “What does the presence of hypertension portend in the Nigerian with non insulin dependent diabetes mellitus?” (**Ikem, R. T.**, et al 2001), we investigated the clinical implications of hypertension among Nigerian patients with Non-Insulin-Dependent Diabetes Mellitus (NIDDM), now referred to as type 2 diabetes. The coexistence of hypertension and diabetes significantly elevates cardiovascular risk and contributes to increased morbidity and mortality in this population. In a cohort of 132 Nigerian patients with NIDDM, participants were stratified into hypertensive and normotensive groups for comparative analysis. While no statistically significant differences were observed in age, sex distribution, or body mass index between the two groups, a notable variation was found in the duration of diabetes, which was significantly longer in the hypertensive group. Laboratory findings further revealed that patients with hypertension and diabetes exhibited higher fasting blood glucose levels, elevated serum urea, reduced creatinine clearance, and a greater degree of proteinuria compared to their normotensive counterparts. These findings underscore the association between hypertension and increased end-organ damage in individuals with type 2 diabetes.

The study also highlights that hypertension in Type 2 Diabetes is a double burden in Nigerian patients and the critical need for early detection and aggressive management of hypertension in patients with diabetes. It advocates for enhanced screening protocols, patient education, lifestyle modifications, and the timely initiation of appropriate antihypertensive therapy to mitigate long-term complications and improve overall patient outcomes.

In another study titled “The Prevalence, Pattern, and Clinical Correlates of Proteinuria in Nigerian Patients with Non–Insulin-Dependent Diabetes Mellitus” (**Ikem, R. T.**, Akinsola, A., et al., 2003), we evaluated the occurrence of proteinuria and its clinical associations among Nigerians with type 2 diabetes. Proteinuria was detected in 53.8% of patients and showed significant associations with increasing age, longer duration of diabetes, and elevated systolic blood pressure (Table 7). Its strong correlation with diabetic retinopathy and neuropathy underscores proteinuria as an important marker of end-organ damage in this population.

Table 7: Correlates of Proteinuria in DM Patients

Variables		
	R value	P value
Age	+0. 0.7	. 0.0016
Duration of DM	+0.7	0.001
SBP	+0.5	0 0001
Diabetic retinopathy		< 0.01
peripheral neuropathy		< 0.01

r = Spearman’s simple correlation coefficient, *p < 0.05 is statistically significant

Our study on “Which Factors Actually Influence the Development and Progression of Overt Nephropathy in Nigerian Diabetics?” (Ibrahim, A., **Ikem, R. T.**, et al., 2009), we demonstrated that modifiable factors particularly hypertension and poor metabolic control are key drivers of diabetic nephropathy among Nigerian patients. We therefore, recommended that early

identification and aggressive management of these factors could significantly reduce the burden of diabetic kidney disease.

Using Doppler ultrasound, the study on, “Relationship between Renal Doppler Indices and Renal Function in Type 2 Diabetes” (Eze, C. U., ... **Ikem, R. T.**, 2019), investigated the association between renal Doppler indices (the Resistivity Index (RI) and Pulsatility Index (PI)) and biochemical markers of renal function in patients with type 2 diabetes mellitus. Both RI and PI correlated positively with albuminuria and serum creatinine, and negatively with estimated glomerular filtration rate (eGFR). These results indicate that elevated RI and PI values reflect declining renal function, suggesting that renal Doppler ultrasonography can serve as a non-invasive tool for early detection and staging of diabetic kidney disease.

4. DIABETES AND THE HEART

A large-scale multicentre survey (Akinuemokhan, I.K., ... **Ikem, R.T.**, 2008) involving 2,487 Nigerians with type 2 diabetes examined the prevalence and implications of coexisting hypertension. The findings showed that 58% of the participants had hypertension (newer data now indicates a higher prevalence, Soyoye.....**Ikem, R.T et al 2015**). Patients with hypertension were generally older, had lived with diabetes longer, and had higher body mass indices than their normotensive peers. These observations highlight a critical point: hypertension is more than a companion of diabetes; it is a powerful driver of complications. It amplifies the risk of cardiovascular disease, kidney damage, and premature death, further burdening individuals already facing the challenges of diabetes.

And so, what do we do with this knowledge? First, we need to push for early detection by ensuring that routine blood pressure monitoring becomes a standard, non-negotiable part of diabetes care in every clinic and every consultation.

Secondly, we must not underestimate the power of lifestyle modification - weight control, dietary adjustments, physical activity, all of which play a critical role in both preventing and managing hypertension. And of course, where necessary, we must implement timely and effective antihypertensive therapy, tailored to the needs of each individual. But beyond treatment, these studies call us to something greater - a shift in how we approach care. We must embrace integrated care models, where diabetes and hypertension are not treated in isolation, but as two sides of the same coin managed together, with shared goals and coordinated strategies.

Mr. Vice Chancellor Sir, the high prevalence of hypertension in our population with diabetes is not just a statistic, it is a call to action. If we are to reduce the burden of complications and improve outcomes, we must address this double threat with urgency, with clarity, and with purpose.

In an effort to unravel the cardiovascular burden of diabetes in our population, we studied left ventricular function in Nigerian patients with diabetes, with or without hypertension (Ajayi EA, ...Ikem RT 2011). The findings revealed a progressive decline in left ventricular ejection fraction across the groups, with the most severe impairment observed in those with both diabetes and hypertension. Even normotensive patients with diabetes, who might otherwise be considered at lower risk, showed early signs of cardiac dysfunction compared to controls. These findings were a cause for serious concern. This suggests that diabetes alone can compromise cardiac function and that hypertension acts as an accelerant, further impairing the heart's ability to pump effectively.

But that was not the only part of the story, in a related study (Ajayi, E.A, et al 2010), we turned our attention specifically to normotensive patients with diabetes, those who often slip under the radar of cardiovascular screening protocols. Using echocardiography and exercise treadmill testing, we evaluated their left ventricular geometry and blood pressure response to physical exertion. Interestingly, only 29.7% of these patients had normal cardiac geometry. A notable 17.2% already showed concentric left ventricular remodelling, that is a structural adaptation of the heart associated with increased cardiovascular risk, even in the absence of overt hypertension. Although, none had progressed to left ventricular hypertrophy, but the warning signs were clear.

Furthermore, those with remodelling had a longer duration of diabetes, experienced greater systolic blood pressure rises during exercise, and exhibited increased relative wall thickness, all subtle but significant indicators of early target-organ damage. This suggests that in Nigerian patients with diabetes, cardiac changes often begin early and silently. Cardiovascular compromise may occur without obvious symptoms, and patients can develop masked or exercise-induced hypertension that is missed by routine resting blood pressure measurements. Waiting for symptoms of overt heart disease may be too late. Early, proactive cardiovascular monitoring including echocardiography, stress testing, and attention to exercise-induced blood pressure changes is crucial to prevent serious complications. Our findings make a compelling case for early, aggressive cardiovascular surveillance in patients with diabetes, hypertension and normotensives alike. Only by detecting these changes early can we hope to prevent the tragic outcomes that often follow.

5. THE DIABETIC FOOT

One of the most distressing yet preventable complications of diabetes and one that continues to devastate lives across the globe is the diabetic foot disease. This condition encompasses a range of serious foot complications in people living with diabetes, primarily resulting from i.) neuropathy, ii.) peripheral arterial disease, and iii.) infection. These three factors interact insidiously, often leading to tissue breakdown, ulceration, and in severe cases, amputation.

In the study “The diabetic foot at Ile-Ife” (Ikem, I. C. & **Ikem, R. T.** (2004), we examined the clinical profile and burden of diabetic foot disease among patients in Ile-Ife, Nigeria. It revealed that diabetic foot lesions are both common and serious, often resulting from a combination of peripheral neuropathy, poor glycaemic control, structural foot deformities, and peripheral vascular disease. A high prevalence of “foot-at-risk” was particularly noted among older patients with type 2 diabetes, poor glycaemic control, and longer disease duration. These features include neuropathy, callus formation, deformities, and vascular insufficiency. These findings underscore the multifactorial nature of diabetic foot complications and highlight the urgent need for regular foot screening, patient education, and early intervention to prevent ulceration and limb loss.

Overall, the study emphasises that diabetic foot disease contributes significantly to morbidity and carries important implications for clinical practice and health policy in Nigeria.

Diabetic foot disease is not just a medical problem. It is a major social and economic burden, and it is felt on every continent. But its impact is particularly acute in resource-limited settings, where late presentation, inadequate foot care, and limited access to specialist services compound the challenge.

Other evidences from our study about this interplay are as follows:

i. Peripheral neuropathy: Peripheral neuropathy is a major driver of diabetic foot disease.

Because it develops insidiously, significant nerve damage often occurs before symptoms are

recognized, placing patients at high risk of foot ulceration and amputation. In our study, “Screening and Identifying Diabetic Patients at Risk of Foot Ulceration Using the Semmes Weinstein Monofilament” (Figure 8) (Ikem RT & Ikem IC 2009), 117 Nigerian adults with type 2 diabetes were assessed. Peripheral neuropathy was highly prevalent – 79% had a documented history, 96% showed impaired sensory function, and nearly half had loss of protective sensation detected with the monofilament.

This simple, low-cost, and non-invasive tool (Figure 8) revealed that many patients walk on insensate feet, unaware of injuries that may progress silently to ulceration and amputation. The findings confirm peripheral neuropathy as a central risk factor in diabetic foot disease.

Routine neuropathy screening allows early identification of high-risk patients and creates opportunities for effective preventive interventions, including patient education, protective footwear, lifestyle modification, and timely medical care. Prevention of diabetic foot disease must therefore begin with systematic neuropathy screening rather than delayed treatment of established ulcers. Early detection not only prevents amputations but preserves mobility, independence, and dignity for people living with diabetes.



Figure 8: A- The Semmes Weinstein Monofilament; B – Its use; C – Points of touch

Still on diabetic peripheral neuropathy, in a study of 322 diabetic patients “ Descriptive Study of Foot Complications in Diabetic Patients with Symptomatic Peripheral Neuropathy” (**Ikem, R. T, et al (2005)**), we evaluated the prevalence of symptomatic peripheral neuropathy and its link to foot complications. About 19% of participants reported symptoms such as numbness, tingling, cramps, or the characteristic burning sensations in the lower limbs. Beyond discomfort, the most concerning finding was the condition of their feet. Peripheral neuropathy not only diminishes sensation but also compromises the body’s early warning system, minor injuries go unnoticed, pressure points are ignored, and small lesions can rapidly progress to serious, potentially limb-threatening complications. These findings underscore a critical principle: early detection of neuropathy must be an integral part of diabetes care. Clinics need both the right tools and adequately trained staff to identify sensory loss before irreversible damage occurs. Equally important is patient education, teaching individuals with diabetes to inspect and care for their feet daily can be the difference between preserving a limb and losing it.

ii) Peripheral Arterial Disease in Type 2 Diabetes: Peripheral Arterial Disease (PAD) is an under-recognized yet critical vascular complication of type 2 diabetes, playing a major role in limb loss and reduced survival. In people living with diabetes, PAD extends beyond a vascular disorder to become a key pathway leading to non-healing ulcers, infection, amputation, and premature mortality.

In our study, “Does Concomitant Hypertension Increase the Risk of Peripheral Arterial Disease in Nigerians with Type 2 Diabetes Mellitus?” (Adebayo OJ... & **Ikem RT**, 2023), we evaluated 160 Nigerian adults with type 2 diabetes, comparing those with diabetes alone to those with both diabetes and hypertension. Peripheral arterial disease was common in both groups, with no

significant difference, underscoring diabetes itself as the primary driver of arterial damage, independent of coexisting hypertension.

The key factors associated with PAD included advancing age, male sex, central obesity, and elevated high-sensitivity C-reactive protein, highlighting the roles of both traditional cardiovascular risks and systemic inflammation. These results confirm that PAD is a major determinant of delayed wound healing, non-healing foot ulcers, and amputations in diabetic patients, yet it remains frequently underdiagnosed, particularly in resource-limited settings. Simple bedside assessments (Fig 9) such as palpation of foot pulses and measurement of the ankle brachial pressure index using a handheld Doppler are reliable and effective methods for early detection. PAD screening should therefore be an integral component of routine diabetes care, regardless of hypertension status. Early identification enables timely intervention and, when systematically applied, not only prevents limb loss but also extends life.



A

B





C

Figure 9: A- Palpation of Dorsalis Pedis Pulse, B- Hand held Doppler, C- Demonstration of use of hand-held Doppler

Diabetic Foot Ulcer (DFU): Globally, up to 15% of people with diabetes will develop a foot ulcer in their lifetime, and about 85% of diabetes-related lower-limb amputations are preceded by such ulcers, many of which could be prevented. In Nigeria, DFU is a leading cause of non-traumatic lower-limb amputations, accounting for approximately 50–61% of major amputations in tertiary hospitals (Ugwu, E., et al. 2019; Inyang et al., 2023; Hart et al., 2025) and other low- and middle-income countries, diabetic foot ulcers are a leading cause of diabetes-related

hospitalisation, often resulting in prolonged admissions, high morbidity and mortality, and significant psychosocial and economic burdens.

Peripheral vascular disease (PWD) is central to the pathogenesis of diabetic foot ulcers, contributing to poor wound healing, limb loss, and mortality. In our study, “An assessment of peripheral vascular disease in patients with diabetic foot ulcer” (Ikem, R. T., Ikem, I. C., & Adebayo, O., & Soyoye, D. (2010) we evaluated peripheral vascular disease (PWD) in type 2 diabetic patients with and without DFUs by measuring the ankle-brachial index (ABI) a non-invasive indicator of blood flow to the lower limbs. A total of 74 patients were enrolled, of whom 62.2% had foot ulcers. The prevalence of PWD (ABI < 0.9) was much higher among patients with DFUs (76.4%) compared to those without ulcers (13.4%). Further analysis showed that tobacco use, longer duration of diabetes, and higher systolic blood pressure were significantly associated with the presence of DFUs in patients with PWD. The use of the simple tool - hand-held Doppler assessment of ABI is a valuable, simple tool for early detection of PWD, which can help identify limbs at risk and potentially reduce the high rate of amputations in diabetic populations.

Despite its impact, diabetic foot disease often remains overshadowed by a focus on glycaemic control alone. Systematic vascular assessment and early recognition of PWD are therefore essential for limb preservation and survival.



Figure 10: A- DFU B –DM foot gangrene; C – Right below knee amputation

There is a saying in diabetes care: *give it an inch, and it will take a foot*, sometimes quite literally (Fig 10). This simple phrase captures the profound truth that even the smallest neglect in diabetes management can lead to devastating complications. So, what must we do? We must intensify efforts toward early detection, patient education, routine foot examinations, and multidisciplinary foot care services. Community awareness campaigns, primary care engagement, and capacity building for front-line health workers must all be part of the strategy.

In essence, diabetic foot disease is preventable but only if we are vigilant, coordinated, and proactive. I thank the management of OAUTHC, for considering our proposal to start a Diabetic Foot Clinic, where we can assess the feet of all our clients to see those at risk. As stakeholders, let us not wait until the ulcer has formed and amputation becomes inevitable. Let us act early because in the case of diabetic foot disease, early action truly saves limbs, and saves lives.

iii) Microbial Patterns in Diabetic Foot Infections: Implications for Targeted Therapy

Diabetic foot infections are a major cause of prolonged hospitalisation and limb-threatening complications in people with diabetes. In our study, “Characterisation of Bacterial Isolates from Diabetic Foot Infections in Ile-Ife, Southwestern Nigeria” (Ako-Nai, A.K, Ikem, I.C, ... **Ikem**,

R.T., 2006), we examined the bacterial profile of infected diabetic foot ulcers to inform more effective treatment strategies and improve clinical outcomes.

The findings demonstrated a predominance of aerobic organisms, with *Escherichia coli* and *Staphylococcus aureus* being the most frequently isolated pathogens. Of particular concern was the high level of antibiotic resistance observed, underscoring the growing challenge of antimicrobial resistance in diabetic foot care. This study highlighted the importance of culture-guided antibiotic therapy over empirical treatment to improve infection control and limit the emergence of resistance. It also showed that superficial swab samples can reliably identify causative organisms, offering a practical and accessible approach in routine clinical settings. As diabetes increasingly intersects with antimicrobial resistance, these findings emphasise the need for ongoing microbiological surveillance, rational antibiotic use, and the integration of laboratory-guided therapy into diabetic foot management pathways.

The Psychosocial Burden of Diabetic Foot Ulcers: More Than a Physical Wound

Diabetic foot ulcers are often approached as purely physical complications, yet their impact extends far beyond the wound itself. Our research highlights the profound psychological and quality-of-life consequences of diabetic foot disease.

In a preliminary controlled study involving Nigerian adults with diabetes on “Relationship between Depression, Cognitive Function and Quality of Life of Nigerians with Diabetic Foot Ulcers” (Ikem RT, et al 2009) we compared patients with active foot ulcers to those with peripheral neuropathy but no ulceration. Individuals with foot ulcers had significantly poorer outcomes across multiple domains, including physical health, psychological well-being, general health perception, and overall quality of life.

Depression was present in both groups but was more pronounced among patients with active ulcers and showed a strong association with reduced health-related quality of life. Although no clinically significant cognitive impairment was identified, the psychological burden of foot ulcers emerged as a key determinant of patient well-being.

These findings underscore a critical insight: diabetic foot disease is not solely a surgical or dermatological problem but also a mental health challenge. Physical disability, chronic pain, and social isolation contribute to psychological distress, which can further compromise recovery and self-care. Effective diabetic foot management must therefore be holistic. Along side wound care, routine screening for depression, psychosocial support, and integration of mental health services should form an essential part of diabetic foot care. Healing the diabetic foot ultimately requires caring for the whole person.

6. THE SKIN IN DIABETES -An often-overlooked window into systemic diseases.

When we think of diabetes, we often focus on the well-known complications - retinopathy, nephropathy and neuropathy. Yet, one of the most visible and often overlooked manifestations of diabetes lies quite literally on the surface - the skin. Working with my colleagues in Dermatology department, our research on the “Patterns of dermatological disorders among diabetics” (Ezejiofor I O ... **Ikem RT** 2013) has shown that individuals living with diabetes are significantly more prone to a wide range of dermatological conditions (Table 8). This increased vulnerability stems from a combination of immune system dysfunction, poor peripheral circulation, and the broader metabolic disturbances that characterise diabetes.

Table 8: Prevalence of Dermatological disorders among the participants (n=355)

Dermatological disorders	Frequency	Percent
Superficial fungal infections	163	35.4
Pruritus	76	16.7
Diabetic dermopathy	67	14.8
Acanthosis nigricans	22	4.9
Diabetic thick skin	18	4
Bacterial infection	16	3.6
Diabetic ulcer	16	3.6
Skin tag	13	3
Tendinous xanthoma	12	2.6
Vitiligo	10	2.2
Erysipelas like erythema	10	2.2
Sclerederma diabetorum	9	2
Idiopathic gutate hypomelanosis	7	1.6
Insulin lipohypertrophy	4	0.9
Seborrheic dermatitis	2	0.45
Seborrheic keratosis	2	0.45
Herpes zoster	2	0.45
Viral wart	1	0.23
Lichen simplex chronicus	1	0.23
Keloids	1	0.23
Insulin lipoatrophy	1	0.23
Diabetic rubeosis	1	0.23

Table 9: Prevalence of infections among the participants (n=163)

Infections	Frequency(n)	Percent(%)
Fungal Infections		
Tinea Infections		
Tinea corporis	7	1.50
Tinea unguium	8	1.80
Tinea pedis	56	12.30
Cutaneous candidiasis		
Candida intertrigo	40	8.80
Vaginal candidiasis	36	7.90
Candida paronychia	1	0.23
Pityriasis versicolor		
	67	14.80
Bacterial infections		
Furuncles	16	3.60
Viral infection		
Viral wart	1	0.23
Total	232	

Among the most commonly observed skin manifestations are diabetic dermopathy (Table 9), fungal and bacterial infections, chronic itching (pruritus), acanthosis nigricans, and of course, diabetic foot ulcers which, as we have discussed, carry devastating consequences if left unmanaged. These skin conditions often serve as early warning signs of more serious internal derangements, and in some cases, can escalate to severe infections, systemic illness, and hospital admissions if not promptly addressed.

Why is this important? Because early recognition of these dermatological signs offers a valuable opportunity for intervention not just to treat the skin itself, but to signal deeper metabolic issues that may require urgent attention. Effective management of skin complications in diabetes can significantly reduce the risk of infection, improve treatment outcomes, and enhance the overall

quality of life for patients many of whom suffer in silence from these conditions. In essence, the skin can serve as both a mirror and a messenger reflecting the internal struggle of poorly controlled diabetes and offering us a chance to act before more serious complications arise. As clinicians, educators, and researchers, we must ensure that skin health is not treated as an afterthought, but as an integral part of comprehensive diabetic care.

In our study on “Descriptive study of foot complications in diabetic patients with symptomatic peripheral neuropathy” (**Ikem, R. T, et al 2005**), patients with symptomatic neuropathy frequently presented with a constellation of lesions: dry skin, hyperpigmentation, corns and callosities, cracked heels, and fungal infections (Table 10).

Table 10: Frequency of foot lesions in symptomatic patients, asymptomatic patients and healthy control

FOOT LESION	SYMPTOMATIC (n = 64)	ASYMPTOMATIC (n = 60)	CONTROLS (n = 25)
Dry skin	48 (75%)	8 (13.3%)	0 (0%)
Callus	8 (12.5%)	4 (6.7%)	2 (8%)
Fungal infection	12 (18.8%)	3 (75%)	2 (8%)
Yellow nail	2 (3.1%)	0 (0%)	0 (0%)
Onycholysis	14 (21.9%)	2 (3.3%)	0 (0%)
Hyperpigmentaion	42 (65.6%)	18 (30%)	5 (20%)
Cracked sole skin	12 (18.8%)	4 (6.7%)	2 (8%)
Ulcer	3 (4.7%)	0 (0%)	0 (0%)
Corns	26 (40.6%)	5 (8.3%)	2 (8%)
Hypopigmentation	4 (6.3%)	5 (8.3%)	3 (12%)
Hallux Valgus	8 (12.5%)	1 (1.7%)	0 (0%)
Harmer toe	3 (4.7%)	1 (1.7%)	0 (0%)
Scabies	0 (0%)	0 (0%)	0 (0%)
Bullosis Diabeticorum	2 (3.1%)	0 (0%)	0 (0%)
Eczema	2 (3.1%)	0 (0%)	0 (0%)
Hallux varus	4 (6.3%)	0 (0%)	0 (0%)
Paronychia	4 (6.3%)	1 (1.7%)	2 (8%)
Pes cavus	4 (6.3%)	1 (1.7%)	0 (0%)

7. GENDER

Gender plays a crucial role in medicine, influencing disease presentation, access to healthcare, treatment outcomes, and research representation. Understanding how gender influences disease expression is essential for delivering targeted and equitable care. Here's an overview of some gender-related issues we saw in our studies.

In a study, on the "Influence of gender on the distribution of type 2 diabetic complications at the Obafemi Awolowo Teaching Hospitals Complex, Ile-Ife, Nigeria" (Enikuomehin, A., ..., & Ikem,

R. T. (2020), we set out to explore gender-specific differences in cardio-metabolic risk factors and diabetes-related complications among individuals living with type 2 diabetes mellitus. The findings revealed that women with type 2 diabetes had a higher prevalence of hypertension and obesity, both of which are critical risk factors for cardiovascular disease. On the other hand, men were more likely to achieve LDL-cholesterol treatment targets, suggesting better lipid control in the male subgroup. Thus, underscoring gender differences in cardio-metabolic risk among patients with Type 2 Diabetes. However, one notable exception stood out: women were more likely to develop moderate to severe diabetic retinopathy, highlighting a potentially higher vulnerability to certain vision-related complications.

Erectile Dysfunction in Men with Type 2 Diabetes: A Common but Overlooked Complication.
As we continue to explore the far-reaching consequences of type 2 diabetes, one complication that often remains under-discussed, yet deeply impactful, is erectile dysfunction (ED). In a study in OAUTHC Ile-Ife titled, “Erectile dysfunction among male type 2 diabetics in a South Western Teaching Hospital, Nigeria”, (Ugwu, T. E., & **Ikem, R. T.** (2016), we examined the prevalence and predictors of ED among male patients living with type 2 diabetes. The results were revealing - an overwhelming 94.3% of participants reported some degree of erectile dysfunction. More than 47% of these men were living with moderate to severe forms of the condition, that significantly affect their quality of life, emotional well-being, and intimate relationships. ED was more prevalent among those with coexisting high blood pressure. Furthermore, men with poorly controlled blood sugar were over seven times more likely to suffer from ED, a reminder that hyperglycaemia does not only damage nerves and vessels but also deeply impacts sexual health. Several independent predictors of erectile dysfunction emerged from our analysis (Table 11):

- Duration of diabetes: For each additional year of living with diabetes, the odds of ED increased by 14%.
- Peripheral arterial disease: Its presence increased the risk of ED by nearly fourfold.
- Autonomic neuropathy: Associated with a threefold increase in the odds of ED.
- Poor glycaemic control: As mentioned, this raised the risk by over seven times.
- And importantly, testosterone deficiency a condition often under-investigated was linked to a sixfold increase in ED risk.

Table 11: Independent Predictors of ED in the Study Population

Variables	OR for ED	95% CI	P value
Age > 60 years	1.17	0.81–1.14	0.063
Duration of DM (5-year increments)	1.14	1.02–1.28	0.024*
Peripheral arterial disease (present)	3.87	1.28–11.67	0.016*
HbA1c \geq 7%	7.12	2.49–20.37	<0.001*
Testosterone < 8 nmol/L	6.63	2.61–16.83	<0.001*
Autonomic neuropathy (present)	3.51	1.82–6.79	<0.001*

DM = diabetes mellitus; ED = erectile dysfunction; HbA1c = glycated hemoglobin.

* = significant with P value < 0.05.

These findings emphasise the need for a holistic approach to diabetes management, one that includes routine screening for sexual dysfunction, especially in patients with long-standing disease, vascular complications, or poor metabolic control. Erectile dysfunction is more than a quality-of-life issue, it is often an early marker of vascular compromise, a signpost pointing to deeper systemic dysfunction. If we did not ask, we would not have found this and if we do not treat, we miss a critical opportunity to intervene. As clinicians, we must initiate the conversation and ask the right questions, while men need to respond freely and honestly. Therefore, integrating sexual health into our chronic disease care models, and recognizing that ED is not just a complication, but a clinical clue to broader cardiometabolic risk.

In addition, the study on “Androgen Deficiency in Aging Male Questionnaire for the Clinical Detection of Testosterone Deficiency in a Population of Black Sub-Saharan African Men with Type 2 Diabetes Mellitus: Is it a Reliable Tool?”, (Ugwu, T. E., & **Ikem, R. T.** (2018) We evaluated if ADAM questionnaire can be used as a first-line screening tool, especially where laboratory resources are limited. The ADAM questionnaire demonstrated very high sensitivity as most men with biochemically confirmed hypogonadism were correctly identified. It helps clinicians identify men who should undergo testosterone testing. Also, sexual symptoms (especially erectile dysfunction and reduced libido - demonstrated better specificity 75.5%) were the most frequently reported and these symptoms strongly influenced ADAM positivity. The study therefore, validates the usefulness of ADAM questionnaire in Nigerian men reinforcing the need for combined clinical and biochemical assessment and highlights hypogonadism as an important but neglected comorbidity in men with diabetes.

In conclusion, these observations indeed, highlight the need for gender-responsive diabetes care one that recognises not only the biological differences, but also the potential variations in access to care, adherence, and health-seeking behaviours that may influence outcomes. In tailoring our management strategies, we must move beyond a one-size-fits-all approach. Gender-specific risk profiling can help us anticipate complications more effectively and design interventions that are both precise and inclusive.

8. THE LUNGS IN DIABETES

Poor glycaemic control in people with diabetes is linked to adverse lung outcomes.

Hyperglycaemia increases inflammation, impairs lung function, and delays recovery from respiratory illnesses. As a systemic disease, diabetes affects the lungs through multiple mechanisms, increasing susceptibility to pulmonary complications, including respiratory infections such as pneumonia and tuberculosis. In our study on “Latent Mycobacterium tuberculosis infection among Type 2 Diabetes Mellitus Patients” (Adewole O.O., ... **Ikem R.T.**, et al., 2014), we found that those with T2DM have increased prevalence of latent TB, making them at increased risk of developing active TB.

Diabetes has also been linked to reduced lung function, with numerous studies including ours “Assessment of lung function parameters in Nigerian males with diabetes mellitus” ,(Kolawole, B. A., Erhabor, G. E., & **Ikem, R.T** 2012). In this cross-sectional study of male Nigerian patients with diabetes, spirometry revealed significantly lower lung function values (FEV₁ and FVC) compared with predicted norms, suggesting a measurable impairment in pulmonary function. This decline is thought to result from diabetes-related changes such as microvascular damage, fibrosis, and altered lung mechanics, which may contribute to restrictive lung disease.

Sleep apnoea is a condition where breathing repeatedly stops and starts during sleep. The commonest type, called obstructive sleep apnoea, (OSA), happens when the airway gets blocked. It is particularly common in people with type 2 diabetes, because both conditions share several risk factors like obesity, insulin resistance, and certain hormonal changes. This is a two-way relationship. Poor sleep from apnoea makes it harder to control blood sugar, while poorly controlled diabetes can, in turn, worsen the apnoea. In our study “Prevalence of obstructive sleep apnoea syndrome in patients with type 2 diabetes mellitus in Nigeria.” (Obaseki DO, ... **Ikem RT, et al** 2013) we found that many of the patients were at high risk of obstructive sleep apnoea.

Using standard screening tools (the Berlin and Epworth questionnaires) we discovered that neck circumference was a strong and independent predictor, even more than age, sex, or blood sugar levels. These findings remind us that good sleep is not a luxury, it is part of good diabetes care. Addressing both conditions together can go a long way in improving heart health and overall wellbeing.

In summary, diabetes can impair lung health through immune, structural, and functional changes, highlighting the importance of good glycaemic control and managing obesity and sleep apnoea to reduce these risks.

9. THE RELATIONSHIP BETWEEN DIABETES AND THE HEPATOBILIARY SYSTEM

In endocrine parlance, the gastrointestinal (GI) system is the largest endocrine organ and it is, indeed. The GI interface include the hepatobiliary system. Thus, we say that the relationship between diabetes and the hepatobiliary system maybe bi-directional. Diabetes predisposes to liver and gallbladder diseases through metabolic and neuropathic mechanisms. In turn, hepatic dysfunction can worsen insulin resistance and metabolic control. This interplay underscores the need for an integrated approach to diabetic care that includes liver and biliary health.

The most common liver disease associated with diabetes is Non-alcoholic Fatty Liver Disease (NAFLD) (Figure 11) . In 2023, an international consensus of Hepatologists agreed to change the terminology, NAFLD to Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) / MASLD). It is characterised by hepatic fat accumulation (steatosis) unrelated to alcohol use. The spectrum ranges from simple steatosis to steatohepatitis (MASH), fibrosis, cirrhosis, and hepatocellular carcinoma. Prevalence: 50–70% of people with T2DM have NAFLD/MASLD

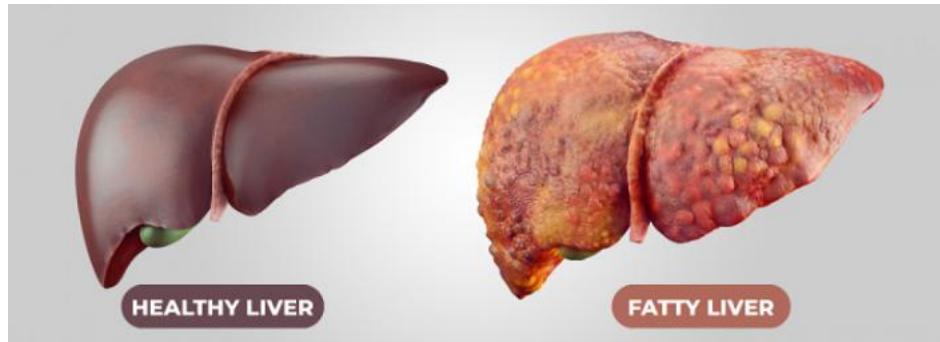


Figure 11: The liver in health and disease (fatty liver)

The Liver:

In our study titled “The relationship between glycaemic control and non-alcoholic fatty liver disease in Nigerian type 2 diabetic patients.” (Afolabi BI, ...**Ikem RT**, et al 2018), the B-mode ultrasound detected NAFLD in 68.8% of participants. The prevalence was significantly associated with higher BMI and poor glycaemic control. Multivariate analysis identified overweight (OR = 6.63), obesity (OR = 11.51), and poor glycaemic control (OR = 3.47) as independent predictors. NAFLD severity also increased with age and BMI (Table 12). The study thus emphasises the need for routine NAFLD screening and better metabolic control in Nigerian T2DM patients to reduce liver-related complications.

Table 12: Logistic Regression for predictors of NAFLD in participants

Variables	B	S.E.	OR (95% CI)	p value
BMI				
Obese	2.443	0.937	11.508 (1.833-72.245)	0.009
Overweight	1.891	0.750	6.626 (1.525-28.797)	0.012
Glycaemic control				
$\text{HbA}_{1c} \geq 7.0\% \text{ (Poor)}$	1.245	0.627	3.473 (1.017-11.864)	0.047
Constant	-7.820	2.919	0.000	0.007

The p values that are statistically significant are in bold.

BMI: body mass index; B: regression coefficient; SE: standard error; OR: odds ratio; CI: confidence interval; significant p ≤ 0.05.

Gall Bladder: While the biliary manifestation that is commonly seen is Gallstones (Cholelithiasis), diabetes increases its prevalence by 1.5–2 times. Clinically, gallstones may remain silent or lead to cholecystitis, cholangitis, or pancreatitis.

In our study on “Gall bladder volume and contractility in type 2 diabetes mellitus” (Ugbaja, C. A., ..., **Ikem, R. T.**, 2015), we examined gallbladder changes in individuals with type 2 diabetes using ultrasonography to calculate the gallbladder contractility index. Patients with diabetic neuropathy had larger gallbladder volumes, reduced contractility, and a higher prevalence of gallstones compared to those without neuropathy and healthy controls. These findings suggest possible autonomic nerve dysfunction and highlight the potential value of ultrasound screening in this population.

Clinical Implications:

- Liver and biliary involvement may be asymptomatic; hence, screening is vital.
- Ultrasound and liver enzyme tests are simple tools for early detection.
- Tight glycaemic and lipid control, weight management, and avoidance of hepatotoxic drugs are essential.
- Recognising MASLD/NAFLD in people with diabetes is important for preventing progression to cirrhosis and liver cancer.

10. DIABETES AND NUTRITION

Diabetes is surrounded by myths that contribute to misinformation and poor management. A common misconception is that people with diabetes must completely avoid carbohydrates; in fact, carbohydrates can be consumed when chosen wisely. Emphasising complex carbohydrates, controlling portion sizes, and understanding the glycaemic index are key to maintaining stable blood glucose levels.

Low-carbohydrate diets can help improve blood sugar control, but extreme restriction is often unsustainable and may cause nutrient deficiencies. Effective diabetes management requires balancing evidence-based guidance with practical and culturally appropriate nutrition advice.

The Role of Dietary Fibre in Diabetes Control

In a study on “A Controlled Comparison of the Effect of a High Fiber Diet on the Glycaemic and Lipid Profile of Nigerian Clinic patients with type 2 Diabetes” (Ikem, R.T, et al 2007.), we evaluated the impact of a high-fibre diet on the glycaemic and lipid profiles of Nigerian patients with type 2 diabetes who were on oral hypoglycaemic agents. Those who received a fibre-rich, calorie adequate diet showed significant improvements in waist circumference, fasting and postprandial glucose levels, total cholesterol, triglycerides, and LDL-C compared to the control group. The study demonstrated that incorporating dietary fibre promotes early attainment of normoglycaemia and improves lipid control. The results reinforce the importance of including fibre recommendations in diabetes management guidelines.

Cassava, Glycaemic Response, and Local Food Choices

Understanding local dietary staples is also crucial in diabetes care. In a study on “Blood glucose response on consumption of cassava varieties (Garri) in healthy Nigerian subjects” (Ogbonna

OC... **Ikem RT.** 2018), we examined blood glucose responses to different cassava varieties, including vitamin A bio-fortified (pruned) cassava. We conducted a single-blind, randomised, cross-over trial in 40 healthy adults. Fasting and postprandial glucose levels were measured after consumption of various cassava preparations. The results showed that the vitamin A bio-fortified cassava produced the lowest postprandial glucose response and glycaemic load compared to conventional varieties. This finding suggests that traditional and bio-fortified local foods can be part of a healthy, culturally appropriate diet, even for individuals at risk of metabolic diseases like diabetes.

Malnutrition in the Elderly with Diabetes

As the global population ages, the number of elderly individuals living with type 2 diabetes mellitus (T2DM) continues to rise. Yet, one important complication often overlooked in this group is malnutrition. In one of our studies “Malnutrition in elderly patients with type 2 diabetes mellitus in a Nigerian tertiary hospital: A cross-sectional study”, we assessed the prevalence and determinants of malnutrition among elderly patients with T2DM (Junaid OA, ...**Ikem RT** et al 2022). The results revealed significantly higher malnutrition rates in the diabetes group compared with non-diabetic control. Key predictors included male gender, albuminuria, and poor glycaemic control. These findings highlight the urgent need to integrate routine nutritional assessment into diabetes care for the elderly. Addressing malnutrition in this population is vital not only to improve quality of life but also to enhance treatment outcomes. This truly, reflects the paradox: ***hunger in the midst of plenty***.

Dispelling Myths with Science

Managing diabetes effectively requires evidence-based approaches balanced nutrition, regular physical activity, appropriate medication, and sustained lifestyle changes. Myths and dietary

fads, though often well-intentioned, can mislead patients and worsen outcomes. It is, therefore, our collective responsibility as clinicians, researchers, and educators to replace misinformation with knowledge grounded in science and adapted to our local realities.

Influence of Knowledge, Attitude, Practice, and Beliefs on Drug Compliance among Nigerian Patients with Type 2 Diabetes Mellitus (Fabiyi, A.K...**Ikem, R.T.** 2002). This study explored how patients' knowledge, attitudes, practices, and beliefs (KAPB) affect medication adherence among Nigerians living with Type 2 Diabetes Mellitus (T2DM). The findings were

1. Knowledge and Compliance: Patients with adequate understanding of diabetes and its complications were more likely to adhere to treatment, while limited awareness contributed to poor compliance.
2. Attitudes and Beliefs: Misconceptions and cultural beliefs such as viewing diabetes as temporary or not serious often led to treatment neglect.
3. Practices and Lifestyle: Unhealthy diets, physical inactivity, and dependence on alternative remedies negatively affected medication adherence.
4. Barriers to Compliance: High drug costs, fear of side effects, and limited healthcare access were major obstacles to consistent treatment.

We therefore concluded that enhancing diabetes education, correcting false beliefs, and improving healthcare accessibility are crucial steps toward better drug compliance and overall disease control among Nigerian T2DM patients.

CONCLUSIONS AND RECOMMENDATIONS

Mr. Vice Chancellor, Sir,

Despite the rising prevalence of diabetes in Nigeria, access to quality care remains severely limited. Nigeria records one of the lowest life expectancies for people living with diabetes, driven by poor availability and affordability of essential medications and supplies, with rural populations bearing the greatest burden. In Ile-Ife and across Southwest Nigeria, diabetes and its complications are increasing, yet care is often delayed by stigma, cultural beliefs, and reliance on self-medication and unregulated herbal remedies.

As medicine advances into an era shaped by artificial intelligence, genomics, and global health challenges, we must remain vigilant. Innovation must not eclipse compassion, and excellence must never replace equity. The heart of medicine, now and always, is human.

The vascular disease in diabetes extends far beyond the heart it is systemic (**Tangles**). Our response must therefore be **multidisciplinary, proactive, and patient-centred**, focusing not only on survival but on quality of life, function, and dignity.

Today's inaugural lecture, *Diabetes – a condition of so much sweetness in the body, yet hunger in the midst of plenty*, underscores diabetes as a silent pandemic shaped by social, cultural, and economic inequities. Addressing its burden demands improved access to care, patient education, public awareness, culturally sensitive interventions, and policies that place equity at the centre of prevention and management.

This is not merely a clinical challenge; it is a public health priority and a moral responsibility.

My Journey of Service - advancing the profession through leadership, mentorship and building Institutions

I have had the privilege of serving both the University and the medical profession in diverse and meaningful capacities. At Obafemi Awolowo University, I have served as Head of the Department of Medicine, Chair departmental examination committees, and examiner across multiple faculties including the Faculty of Pharmacy, Faculty of Science (Department of Microbiology), Faculty of Technology (Department of Computer Science), Faculty of Basic Medical Sciences (Nursing Sciences and Anatomy). I also served as post-UTME invigilator and supervisor on many occasions and member of the Ebola Virus Surveillance Committee (2014). I have also examined for Master's and PhD programs and contributed as a cognate member across several faculties, supporting the academic and administrative development of the University. I was a Principal Investigator on URC grant and co investigator on SIDCAIN grants from IDF and won other awards including IDEG Travel Grant, British Endocrine Society Fellowships, and the 1st Dago Jack Award Lecture (2024).

With a focus on endocrinology and diabetes care, I head the Endocrine Unit at OAU and OAUTHC, where I established the EDM Unit - a national training centre attracting resident doctors from across Nigeria and providing Diabetes and General Endocrine service, now expanded to include a Diabetic Foot Clinic.

My contributions to postgraduate medical education extend nationally. I have mentored and supervised over 30 resident dissertations for fellowship examinations of both the NPMCN and WACP. I have also co supervised resident dissertations in Radiology, Family Medicine and others. I have supervised and currently oversee MD theses for the NPMCN. I served as Faculty Treasurer and immediate past Chairperson of the Endocrine, Diabetes, and Metabolism (EDM) Subspecialty of the Faculty of Internal Medicine at NPMCN, contributing to the development of the EDM curriculum and fellowship training logbook. I continue to serve on the Court of Judges

and Court of Examiners, resource person at Training-of-Trainers courses, and led accreditation panels to teaching hospitals in Nigeria. I have also served as external examiner for MBBS programmes at multiple universities and as a resource person and examiner for foreign medical graduates. I have attended and co organised several conferences within and outside the country. Beyond institutional roles, I have served as Consultant Physician and Endocrinologist at OAUTHC since 1999, as Welfare Officer for the Medical Women Association of Nigeria, Osun State and was a member of Osun State Nigerian Medical Association Advisory Committee on the National Health Insurance Scheme. I have held leadership and mentorship positions in professional associations including EMSON, ACEN, ISE, AACE, and EASD. As a scholar and educator, I have published extensively over 70 papers and book chapters, reviewed for numerous national and international journals, and currently serve as Editor-in-Chief of the Nigerian Endocrine Practice. I have delivered lectures to professional and public audiences on diabetes, cardiovascular health, lifestyle modification, and related complications. Throughout my career, I have remained deeply committed to leadership, scholarship, and the advancement of endocrinology, diabetes care, and medical education in Nigeria, striving always to inspire excellence, as a mentor of future leaders, and leave a lasting impact on the next generation of medical professionals.

APPRECIATIONS

Mr Vice-Chancellor, Sir,

To deliver an inaugural lecture is not merely to look back, it is to look forward; to reaffirm a lifelong commitment to inquiry, mentorship, and care. I am deeply grateful for the mentors who shaped me, the colleagues who continue to inspire me, and the students who keep me both honest

and hopeful. In appreciation: I will start by saying *“abundance thrives in the presence of a grateful heart and I am indeed grateful.* “First to God, almighty the Divine in whom everything that exists, lives, and moves draws its life from this divine source, and to my friends in ECKANKAR, my spiritual path, I offer my profound thanks.

I extend my deep appreciation to Obafemi Awolowo University and the Teaching Hospital for providing an enabling environment to serve, teach, and contribute to the advancement of knowledge.

In my Department of Medicine, I acknowledge with gratitude my teachers who became colleagues and friends:

- Professor GE Erhabor, whom I first made contact with as a resident in his unit he taught me that a ward round is not merely a walk through the ward, but an opportunity to see, listen, and learn to engage meaningfully with patients rather than simply glance at them or their records.
- Professor DA Ndububa: A true model of meticulous clinical excellence where every patient is seen, every detail is considered, and every decision is grounded in careful observation and thoughtful judgment.
- Professor MO Balogun: a guiding mentor who inspired and nurtured my early steps into the world of research.
- Emeritus Professor A. Akinsola: my mentor and father figure, whose unwavering support has been a constant pillar for both me and my family.

My late mentors, Professor A.E. Ohwovoriole and Professor T. Johnson, the doyens of Endocrinology in Nigeria, whose lasting influence continues to shape my work in the field.

To my colleagues - Professors AO Akintomide, BA Kolawole, AA Sanusi, FA Arogundade, MA Komolafe, OO. Adewole, Dr. T Ekwere, and Prof RA Adebayo (HoD). Special thanks to the

committee for this Inaugural Lecture and my other colleagues who if you don't hear your name know that you are in my heart, I say a big thank you.

Teaching has been one of the most fulfilling aspects of my career. Guiding students as they begin their journey in medicine is both an honor and a profound responsibility. To me, teaching is more than transmitting knowledge; it is about shaping judgment, nurturing values, and inspiring purpose. I strive to model the qualities I hope they carry forward: integrity, humility, and a dedication to service.

To my Endocrine "children" - many of whom have returned to establish their own units across Nigeria and making impact globally, including Dr. F.A. Adesina, Dr. D.O. Soyoye, Dr. J. Adebayo, Prof. T. Ugwu, Dr. A. Ojo, Dr. G Ajani, Dr. A.C. Enikuomehin, Dr. O Ala, Dr. F. Owolabi, Dr. T. Yusuff, Dr. D. Amjo, Dr. O. Ezekpo, Dr. T. A. Adetunji, Dr. O. Ogundele, Dr. L Imarhiagbe, Dr. Y. Akuma, and many others - my mentees, and all my students making an impact both in Nigeria and beyond: I am immensely proud of you. Thank you, and may your lights continue to shine brightly. I extend my appreciation to members of my professional societies- ACEN and EMSON.

I am profoundly grateful to colleagues beyond my department, my friends and mentors particularly Prof. M. A. Durosinmi, my research collaborators, my patients especially members of DAN and the countless individuals whose support and influence have enriched my journey. To my parents, the late Elder Andrew Okoro and Retired Major Grace Okoro, whose guidance laid the foundation of my life; my heartfelt appreciation to my siblings - Emilia (late), Helen, Christopher, Faustina, Andrew, Martha, Adeoye, Sophie, and Foster and to my late grandmother, Mama Ijebu, Madame Ayodele Otule, as well as my many in-laws - most especially the late

Diokpa Anthony Ikem, whose life and legacy remain deeply cherished in our hearts, nephews, nieces, and cousins thank you all for being such a blessing in my life.

To our lovely daughters, Anwuli and Ugo who, in their junior secondary school days, typed part of my Part II Fellowship dissertation and learned the basics of computer long before it was common to own one, thank you for your steadfast love and support.

To my dependable sons-in-law and now my sons, Ikenna and Arinze, and my wonderful grandchildren, Chiedu, Adaora, Munachiso, and Adaeze who have brought youthfulness into grandpa and grandma home, you all ever remain my joy.

Finally, to the love of my life, my life partner, and my greatest support, my darling husband, Prof. Innocent (Inno) Ikem, a distinguished professor of Orthopaedic Surgery & Traumatology in this great university I thank you. You have been my steadfast confidant, my strength when I grow weary, and my reminder of perseverance when I feel discouraged. Your unwavering support has made every challenge surmountable, and every achievement more meaningful you said *I can do it, and today I did.*

Mr Vice-Chancellor, distinguished colleagues, esteemed guests, and dear students, thank you for allowing me the privilege to contribute to this noble tradition. To every one of you here both in person and online, I express my deepest gratitude for honouring me today with your presence.

Thank you, and God bless you all. (May the blessings of God be)

REFERENCES

1. Adediran, I. A., **Ikem, R. T.**, & Borisade, M. F. (2004). Fibrinolytic activity in Nigerian diabetics. *Postgraduate Medical Journal*, 60, 610–612.
2. Adewole, A. D., **Ikem, R. T.**, Adigun, A. Q., Akintomide, A. O., Balogun, M. O., & Ajayi, A. A. (1996). A 3-year clinical review of the impact of ACE inhibitors on the intrahospital mortality of congestive heart failure in Nigerians. *Central African Journal of Medicine*, 42(8), 253–255.

3. Adewole OO, Kayode O, Abe O, Kolawole BA, **Ikem RT**, Adewole TO, Erhabor GE. (2014) Latent Mycobacterium tuberculosis infection among Type 2 Diabetes Mellitus Patients. *Nigerian journal of Chest Diseases* 1 (1) 24 – 30.

4. Afolabi, B. I., Ibitoye, B. O., **Ikem, R. T.**, Omisore, A. D., Idowu, B. M., & Soyoye, D. (2018). The relationship between glycaemic control and nonalcoholic fatty liver disease in Nigerian type 2 diabetic patients. *Journal of the National Medical Association*, 110(3), 256–264.

5. Ajayi, A. A., Balogun, M. O., Akintomide, A. O., & **Ikem, R. T.** (2000). Monotherapy with lacidipine in Nigerians with mild to moderate essential hypertension. *Nigerian Journal of Medicine*, 9(3), 104–105.

6. Ajayi, E. A., Balogun, M. O., Akintomide, O. A., Adebayo, R. A., **Ikem, R. T.**, Ogunyemi, S. A., & Oyediji, A. T. (2010). Blood pressure response to an exercise treadmill test, and echocardiographic left ventricular geometry in Nigerian normotensive diabetics. *Cardiovascular Journal of Africa*, 21(2), 103–106.

7. Akinuemokhan, I. K., Ehusani-Anumah, F. O., Ogbera, A. O., **Ikem, R. T.**, Puepet, F., Adeyemi-Doro, A. O., Eregie, A., Fasanmade, O. A., & Ohwovoriole, A. E. (2008). Hypertension in Nigerians with type 2 diabetes: a multicenter survey. *Diabetes International*, 15(1), 17–18.

8. Ako-Nai, A. K., Ikem, I. C., Akinloye, A. O., Aboderin, A. O., **Ikem, R. T.**, & Kassim, O. O. (2006). Characterization of bacterial isolates from diabetic foot infections in Ile-Ife, Southwestern Nigeria. *The Foot*, 16, 158–164.

9. Alebiosu, O. C., Familoni, O. B., Ogunsemi, O. O., Adeleye, J. O., Balogun, W. O., Kolawole, B. A., & **Ikem, R. T.** (2009). Knowledge of diabetes and hypertension care among health care workers in southwest Nigeria. *Postgraduate Medicine*, 121, 1–5.

10. American Diabetes Association. (2014). Diagnosis and classification of diabetes mellitus. *Diabetes Care*, 37(Suppl. 1), S81–S90. <https://doi.org/10.2337/dc14-S081>.

11. American Diabetes Association. (2024). Classification and diagnosis of diabetes: Standards of care in diabetes—2024. *Diabetes Care*, 47(Suppl. 1), S16–S33. <https://doi.org/10.2337/dc24-S002>

12. Awotidebe, T. O., Adedoyin, R. A., Oke, K. I., Ativie, R. N., Opiyo, R., Ikujeyisi, E. O., **Ikem, R. T.**, & Afolabi, M. A. (2017). Relationship between functional capacity and health-related quality of life of patients with type 2 diabetes. *Diabetes & Metabolic Syndrome: Clinical Research & Reviews*, 11(1), 1–5.

13. Centers for Disease Control and Prevention. (2024, May 15). Vision loss and diabetes. <https://www.cdc.gov/diabetes/diabetes-complications/diabetes-and-vision-loss.html>.

14. Dawha, S., Ayoola, O. O., Ibitoye, B. O., **Ikem, R. T.**, & Arogundade, F. A. (2014). An assessment of factors influencing resistivity and pulsatility indices in diabetes mellitus. *Tropical Journal of Nephrology*, 9, 15–22.

15. Enikuomehin, A. C., Soyoye, D. O., Adebayo, J., Kolawole, B. A., & **Ikem, R. T.** (2020). Influence of gender on the distribution of type 2 diabetic complications at the Obafemi Awolowo Teaching Hospitals Complex, Ile-Ife, Nigeria. *African Health Sciences*, 20(1), 294–307. <https://doi.org/10.4314/ahs.v20i1.35>.

16. Eze, C. U., Nwobi, I. C., Agwu, K. K., Aronu, A. E., & **Ikem, R. T.** (2019). Relationship between renal Doppler indices and biochemical indices of renal function in type 2 diabetes mellitus. *Nigerian Journal of Clinical Practice*, 22(7), 917–922.

17. Ezejiofor, O. I., Onayemi, O., Olasode, O. A., & **Ikem, R. T.** (2013,). Patterns of dermatological disorders among diabetics. *Egyptian Dermatology Online Journal*, 9(2), 1–14.

18. Fabiyi, A. K., Kolawole, B. A., Adefehinti, O., & **Ikem, R. T.** (2002). The impact of knowledge, attitude, practice, and beliefs of type 2 Nigerian patients on drug compliance. *Diabetes International*, 12(1), 15–17.

19. Hart, F., & Orupabo, F. (2025). Evolving patterns of lower limb amputations at a Nigerian tertiary hospital: A retrospective study. *International Surgery Journal*, 12(8), 1263–1268.
<https://doi.org/10.18203/2349-2902.isj2025227>.

20. Ibrahim, A., Arogundade, F. A., Sanusi, A. A., **Ikem, R. T.**, Akintomide, A. O., & Akinsola, A. A. (2009). Which factors actually influence the development and progression of overt nephropathy in Nigerian diabetics? *Central African Journal of Medicine*, 55, 28–34.

21. **Ikem, R. T.**, Akinola, N. O., Balogun, M. O., Ohwovoriole, A. E., & Akinsola, A. A. (2001). What does the presence of hypertension portend in Nigerian NIDDM? *West African Journal of Medicine*, 20(2), 127–130.

22. **Ikem, R. T.**, Kolawole, B. A. (2001). Anthropometric indices of newly diagnosed type 2 diabetic Nigeria subjects. *Nigerian Journal of Internal Medicine*, 4(1), 6–8.

23. **Ikem, R. T.**, Kolawole, B. A., Ikem, I. C. (2002). The prevalence, presentation and outcome of diabetic foot lesions in a Nigerian teaching hospital. *Tropical Doctor*, 32, 226–227.

24. **Ikem, R. T.**, Kolawole, B. A. (2002). Diabetes register: an audit of newly presenting patients in diabetes outpatient clinic. *African Journal of Endocrinology and Metabolism*, 3(1), 52–54.

25. **Ikem, R. T.**, Akinsola, A., Balogun, M. O., & Ohwovoriole, A. E. (2003). The prevalence, pattern, and clinical correlates of proteinuria in Nigerian patients with non–insulin dependent diabetes mellitus. *Nigerian Journal of Health Sciences*, 2, 21–24.

26. Ikem, I. C., & **Ikem, R. T.** (2004). The diabetic foot at Ile-Ife. *Nigerian Journal of Surgical Sciences*, 14(1), 13–17.

27. **Ikem, R. T.**, Kolawole, B. A., Olasode, O. (2005). A descriptive study of foot complications in diabetic patients with symptomatic peripheral neuropathy. *African Journal of Neurological Sciences*, 24(1), 7–12.

28. **Ikem, R. T.**, Adesina, O. F., Soyoye, D. O., Adebayo, O. J., & Kolawole, B. A. (2010). Current prospects in DM admission and mortality. *African Journal of Endocrinology and Metabolism*. (June 2010).

29. **Ikem, R. T.**, Kolawole, B. A., Ojofeitimi, E. O., Salawu, A., Ajose, O. A., Abiose, S., & Odewale, F. (2007). A controlled comparison of the effect of a high fiber diet on the glycaemic and lipid profile of Nigerian clinic patients with type 2 diabetes. *Pakistan Journal of Nutrition*, 6(2), 111–116.

30. **Ikem, R. T.**, & Ikem, I. C. (2009). Screening and identifying diabetic patients at risk of foot ulceration: Use of Semmes–Weinstein monofilament. *Diabetes International*, 17(1), 15–17.

31. **Ikem, R. T.**, Ikem, I. C., & Olaogun, M. O. B., Owoyemi, A., & Ola, B. A. (2009). Assessment of limited joint mobility of the hand in black Africans with diabetes mellitus and in non-diabetics. *West Indian Medical Journal*, 58(6), 506–511.

32. **Ikem, R. T.**, Ikem, I. C., Ola, B. A. (2009). Relationship between depression, cognitive function, and quality of life of Nigerians with diabetic foot ulcers: A preliminary controlled study. *Acta Endocrinologia*, 5(1), 75–83.

33. **Ikem, R. T.**, Ikem, I. C., & Adebayo, O., & Soyoye, D. (2010). An assessment of peripheral vascular disease in patients with diabetic foot ulcer. *Foot (Edinburgh, Scotland)*, 20(4), 114–117.

34. Junaid, O. A., Ojo, O. A., Adejumo, O. A., Junaid, F. M., Ajiboye, K. J., Ojo, O. E., Akitikori, T. O., Kolawole, A. B., & **Ikem, T. R. (2022)**. Malnutrition in elderly patients with type 2 diabetes mellitus in a Nigerian tertiary hospital: A cross-sectional study. *Dialogues in Health*, 1, 100019. <https://doi.org/10.1016/j.dialog.2022.100019>.

35. Kolawole, B. A., Abodunde, O., **Ikem, R. T.**, & Fabiyi, A. K. (2004). A test of the reliability and validity of diabetes-specific quality of life scale in a Nigerian hospital. *Quality of Life Research*, 13, 1287–1295.

36. Kolawole, B. A., Mosaku, S. K., & **Ikem, R. T.** (2009). A comparison of two measures of quality of life of Nigerian clinic patients with type 2 diabetes mellitus. *African Health Sciences*, 9, 161–166.

37. Kolawole, B. A., Erhabor, G. E., & **Ikem, R. T.** (2012). Assessment of lung function parameters in Nigerian males with diabetes mellitus. *African Journal of Respiratory Medicine*, 7(2), 20–22.

38. Mosaku, S. K., Kolawole, B. A., Mume, C. O., & **Ikem, R. T.** (2008). Depression, anxiety and quality of life among diabetic patients: A comparative study. *Journal of the National Medical Association*, 100(1), 73–78.

39. Obaseki, D. O., Kolawole, B. A., Gomerep, S. S., Obaseki, J. E., Abidoye, I. A., **Ikem, R. T.**, & Erhabor, G. E. (2013). Prevalence of obstructive sleep apnoea syndrome in patients with type 2 diabetes mellitus in Nigeria. *Nigerian Medical Journal*, 54(6), 59–63.

40. Odetoyin, W. B., Aboderin, A. O., **Ikem, R. T.**, Kolawole, B. A., & Oyelese, A. O. (2008). Asymptomatic bacteriuria in patients with diabetes mellitus in Ile-Ife, southwest Nigeria. *East African Medical Journal*, 85(1), 18–23.

41. Ogbonna, O. C., Fadeiye, E. O., & **Ikem, R. T.** (2018). Blood glucose response on consumption of cassava varieties (Garri) in healthy Nigerian subjects. *Journal of Nutrition and Human Health*, 2(1), 22–27.

42. Ogbera, A. O., & Ekpebegh, C. (2014). Diabetes mellitus in Nigeria: The past, present and future. *World Journal of Diabetes*, 5(6), 905–911. <https://doi.org/10.4239/wjd.v5.i6.905>.

43. Olamoyegun, M. A., Alare, K., Afolabi, S. A., Aderinto, N., & Taiwo, A. (2024). A systematic review and meta-analysis of the prevalence and risk factors of type 2 diabetes mellitus in Nigeria. *Clinical Diabetes and Endocrinology*, 10, Article 43. <https://doi.org/10.1186/s40842-024-00209-1>.

44. Raimi, T. H., Alebiosu, O. C., Adeleye, J. O., Balogun, W. O., Kolawole, B. A., Familoni, O. B., **Ikem, R. T.**, Adesina, O. F., Odusan, O., Oguntona, S. A., Olunuga, T., & Ogunsemi, O. (2014). Diabetes education: strategy for improving diabetes care in Nigeria. *African Journal of Diabetes Medicine*, 22(1), 9–11.

45. Soyoye, D. O., Kolawole, B. A., **Ikem, R. T.**, Ijarotimi, O., & Enikuomehin, A. C. (2015). Metabolic syndrome and component risks: A comparative study of Nigerians with diabetes mellitus and healthy adults. *Nigerian Endocrine Practice*, 10(1–2), 35–41.

46. Soyoye, D. A., **Ikem, R. T.**, Kolawole, B. A., & Adebayo, J. (2016). Prevalence and correlates of peripheral arterial disease in Nigerians with type 2 diabetes. *Advances in Medicine*. Article ID 3529419. <https://doi.org/10.1155/2016/3529419>.

47. The Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. (1997). Report of the expert committee on the diagnosis and classification of diabetes mellitus. *Diabetes Care*, 20, 1183–1197.

48. The Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. (2003). Follow-up report on the diagnosis of diabetes mellitus. *Diabetes Care*, 26, 3160–3167.

49. Ugbaja, C. A., Ayoola, O. O., **Ikem, R. T.**, & Idowu, B. M. (2015). Gall bladder volume and contractility in type 2 diabetes mellitus. *African Journal of Diabetes Medicine*, 23(2), 9–12.

50. Ugwu, T., Ezeani, I., Onung, S., Kolawole, B. A., & **Ikem, R. T.** (2016). Predictors of erectile dysfunction in men with type 2 diabetes mellitus referred to a tertiary healthcare centre. *Advances in Endocrinology*, 2016, Article ID 9753154. <https://doi.org/10.1155/2016/9753154>

51. Ugwu, T. E., **Ikem, R. T.**, Kolawole, B. A., & Ezeani, I. U. (2016). Clinicopathologic assessment of hypogonadism in men with type 2 diabetes mellitus. *Indian Journal of Endocrinology and Metabolism*, 20(5), 667-673. <https://doi.org/10.4103/2230-8210.190554>

52. Ugwu, T. E., Ezeani, I., Onung, S., Kolawole, B., & **Ikem, R. T.** (2016). Predictors of erectile dysfunction in men with type 2 diabetes mellitus referred to a tertiary healthcare centre. *Advances in Endocrinology*, 2016, 1–8. <https://doi.org/10.1155/2016/9753154>.

53. Ugwu, T. E., & **Ikem, R. T.** (2018). Androgen Deficiency in Aging Male Questionnaire for the clinical detection of testosterone deficiency in a population of black Sub-Saharan African

men with Type 2 diabetes mellitus: Is it a reliable tool? Current Diabetes Review, 14(3), 280-285. <https://doi.org/10.2174/1573399812666161228152036>.

54. Uloko, A. E., Musa, B. M., Ramalan, M. A., Gezawa, I. D., Puepet, F. H., Uloko, A. T., et al. (2018). Prevalence and risk factors for diabetes mellitus in Nigeria: A systematic review and meta-analysis. Diabetes Therapy, 9(3), 1307–1316. <https://doi.org/10.1007/s13300-018-0441-1> BioMed Central+1.

55. World Health Organization. (2016). International statistical classification of diseases and related health problems (10th rev.). World Health Organization.

56. World Health Organization. (2019). WHO classification of diabetes. **ISBN:** 978-92-4-151570-2.

57. International Diabetes Federation. (2021). IDF Atlas 2021,10th edition.