

LIPID PROFILE PARAMETERS (TC, TG, HDL, AND LDL) AS CORRELATES OF ANTHROPOMETRIC PARAMETERS (BMI, WC, WHR, WHtR, BRI, AND VAI) AMONG PATIENTS WITH TYPE 2 DIABETES MELLITUS

Onyeulor, Chinasa Jane

Department Of Biochemistry, Kingsley Ozumba Mbadiwe University, Imo State
Chinasajaneonyeulor@Gmail.Com

Ezekiel Udo Umoh

Department Of Medical Biochemistry, Faculty Of Basic Medical Sciences, College Of Health Sciences, University Of Uyo – Uyo, Akwa Ibom State, Nigeria

Obasi Ifeanyi Chima

Department Of Biochemistry, Michael Okpara University Of Agriculture, Umudike, Abia State

Nwawuba Nnaemeka Ikenna

Department Of Biochemistry, Michael Okpara University Of Agriculture, Umudike, Abia State

Prof. O. C. Ohaeri.

Department Of Biochemistry, Michael Okpara University Of Agriculture, Umudike, Abia State

Ugwuibe Onyewuchi Goziem

Department Of Biochemistry, Kingsley Ozumba Mbadiwe University, Imo State

Abstract:

The study sought to determine the correlation between lipid profile parameters (TC, TG, HDL, and LDL) and anthropometric parameters (BMI, WC, WHR, WHtR, BRI, and VAI) among patients with type 2 diabetes mellitus. In carrying out this study comparative cross-sectional study was adopted. The study population consisted of one hundred (100) diagnosed type 2 diabetic patients and one hundred (100) apparently healthy, age- and sex-matched non-diabetic individuals who served as controls. All participants were aged between 30

and 70 years. Ten (10) mL of venous blood samples were collected from each participant after overnight fast using standard aseptic procedures and distributed into appropriate tubes for the determination of fasting plasma glucose (FPG), glycated hemoglobin (HbA1c), insulin, lipid profile, and oxidative stress markers. Serum and plasma were prepared by centrifugation at 3000 rpm for 10 minutes, while whole blood was used for selected analyses. The samples were analyzed using standard laboratory methods. Data obtained were analyzed using Statistical Package for Social Sciences (SPSS) version 25.0. Results were expressed as Mean $\pm$ Standard Deviation. The test of significance was determined using independent samples t-test, while Pearson correlation analysis was used to assess relationships between variables, with $p < 0.05$ considered statistically significant. In conclusion lipid profile parameters, including total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C), are important indicators of metabolic and cardiovascular health among patients with type 2 diabetes mellitus. The result of the findings revealed that there was a significant negative correlation of superoxide dismutase (SOD) with body roundness index (BRI) ($r = -0.207^*$, $p = 0.039$) in type 2 diabetic patients. There was a significant negative correlation of catalase (CAT) with body mass index (BMI) ($r = -0.402^*$, $p = 0.000$), waist circumference (WC) ($r = -0.329^*$, $p = 0.001$), and waist-to-height ratio (WHtR) ($r = -0.211^*$, $p = 0.035$), while significant positive correlations were observed with waist-to-hip ratio (WHR) ($r = 0.287^*$, $p = 0.004$) and BRI ($r = 0.359^*$, $p = 0.000$) in type 2 diabetic patients. The study concluded that lipid profile parameters, including total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C), are important indicators of metabolic and cardiovascular health among patients with type 2 diabetes mellitus. One of the recommendations made was that patients with type 2 diabetes mellitus should undergo regular lipid profile and anthropometric assessments to facilitate early identification of dyslipidaemia and abnormal adiposity.

Keywords: Lipid Profile, Total Cholesterol, Triglycerides, HDL-C, LDL-C, Anthropometric Parameters, BMI, Waist Circumference, WHR, WHtR, BRI, VAI, type 2 diabetes mellitus.

Introduction

The relationship between lipid metabolism and adiposity is particularly important in patients with type 2 diabetes mellitus because excess body fat, especially visceral adiposity, is closely associated with disturbances in lipid metabolism. Increased adipose tissue can promote the release of free fatty acids and contribute to insulin resistance, hepatic triglyceride synthesis, and alterations in lipoprotein metabolism. These metabolic changes may result in elevated triglyceride and low-density lipoprotein cholesterol levels, together with reduced high-density lipoprotein cholesterol levels, thereby increasing the risk of cardiovascular complications. Consequently, assessment of lipid profile parameters alongside measures of body size and fat distribution may provide a more comprehensive understanding of the metabolic abnormalities associated with type 2 diabetes mellitus.

Diabetes mellitus is a chronic metabolic disorder characterized by elevated blood glucose levels resulting from defects in insulin secretion, insulin action, or both [1]. Type 2 diabetes mellitus (T2DM) represents approximately 90-95% of all diagnosed cases of diabetes and has emerged as one of the most significant public health challenges globally, particularly in developing countries including Nigeria [2]. The disease is associated with substantial morbidity and mortality, primarily due to its microvascular and macrovascular complications affecting multiple organ systems including the kidneys, eyes, nerves, and cardiovascular system.

Biochemical markers play a crucial role in the diagnosis, monitoring, and management of type 2 diabetes mellitus. Key biochemical parameters include fasting plasma glucose (FPG), glycated hemoglobin (HbA1c), lipid profiles (total cholesterol, triglycerides, high-density lipoprotein cholesterol, and low-density lipoprotein cholesterol), and various markers of oxidative stress and

inflammation [3]. These markers provide essential information about glycemic control, cardiovascular risk, and the presence of complications in diabetic patients. Alterations in these biochemical parameters have been consistently documented in diabetic populations worldwide, though regional variations exist due to genetic, dietary, and environmental factors.

Anthropometric measurements, including body mass index (BMI), waist circumference (WC), waist-to-hip ratio (WHR), and waist-to-height ratio (WHtR), serve as important indicators of obesity and central adiposity, which are major risk factors for type 2 diabetes mellitus [4]. The relationship between anthropometric indices and diabetes risk has been well-established, with central obesity being particularly strongly associated with insulin resistance and diabetes development. Unlike subcutaneous fat, visceral adipose tissue is metabolically active and releases free fatty acids and inflammatory cytokines that contribute to insulin resistance and metabolic dysfunction.

Research Problem

Despite the growing burden of type 2 diabetes mellitus in Nigeria and the documented importance of biochemical and anthropometric parameters in diabetes management, there is limited information about the specific patterns of these variations among diabetic patients in Owerri and surrounding areas. Most existing studies have focused on other regions of Nigeria, particularly the southwestern and northern regions, or have examined diabetic populations in broader contexts without specific geographic focus. This geographic gap in knowledge limits the ability of healthcare providers to develop targeted interventions for the local population.

The biochemical profile of diabetic patients can vary significantly based on genetic, environmental, dietary, and lifestyle factors. Previous research has demonstrated that diabetic patients in different populations may exhibit distinct patterns of glycemic control, lipid abnormalities, oxidative stress markers, and inflammatory parameters [5]. For example, studies have shown that African populations may have different patterns of lipid abnormalities compared to Caucasian populations, with potentially higher HDL-cholesterol levels and different relationships between BMI and diabetes risk. Understanding these patterns in the local context of Owerri is essential for developing appropriate diagnostic and therapeutic strategies tailored to this population.

Anthropometric characteristics and their relationship with diabetes risk may differ across populations due to variations in body composition, genetic predisposition, and environmental factors. Studies conducted in other African countries have highlighted the importance of population-specific anthropometric cutoff values for diabetes risk assessment [6]. The optimal cutoff points for BMI, waist circumference, and other indices may differ from those established in Western populations, and the applicability of these findings to the population of Owerri remains uncertain. This necessitates local research to establish appropriate reference values and risk thresholds for this specific population.

Research Problem

Despite the increasing burden of type 2 diabetes mellitus and its associated metabolic complications, dyslipidaemia and abnormal adiposity remain major challenges affecting disease management and cardiovascular risk reduction. Although lipid profile parameters such as total cholesterol, triglycerides, HDL-C, and LDL-C are routinely assessed in diabetic patients, the relationship between these biochemical abnormalities and different anthropometric indices, including BMI, waist circumference, waist-to-hip ratio, waist-to-height ratio, body roundness index, and visceral adiposity index, is not fully understood in many populations. Previous studies have demonstrated associations between lipid abnormalities and measures of obesity among patients with type 2 diabetes; however, variations exist across geographical regions and ethnic groups, making it necessary to generate population-specific evidence. In Nigeria, available studies have reported the presence of dyslipidaemia among patients with type 2 diabetes, but limited information exists regarding the correlation between lipid profile parameters and newer adiposity indices such as BRI and VAI, particularly among patients in Owerri

and surrounding areas. Therefore, this study seeks to determine the relationship between lipid profile parameters and anthropometric parameters among patients with type 2 diabetes mellitus, providing evidence that may improve metabolic risk assessment and support effective diabetes management strategies.

Research objectives

The study sought:

1. To determine the Lipid profile parameters (TC, TG, HDL, AND LDL) as correlates of anthropometric parameters (BMI, WC, WHR, WHtR, BRI, AND VAI) among patients with type 2 diabetes mellitus.

Research question

1. What is the relationship between lipid profile parameters (TC, TG, HDL, and LDL) and anthropometric parameters (BMI, WC, WHR, WHtR, BRI, and VAI) among patients with type 2 diabetes mellitus?

THEORETICAL FRAMEWORK

Pathophysiological Foundation: The “Twin Cycle Hypothesis” and β -Cell Dysfunction

The fundamental theoretical underpinning of T2DM pathophysiology derives from the understanding that the disease represents a progressive disorder of glucose metabolism characterized by two primary defects: insulin resistance in peripheral tissues and progressive β -cell dysfunction leading to relative insulin deficiency [7]. The “twin cycle hypothesis” proposed by Taylor and subsequently elaborated provides a mechanistic explanation for these phenomena, suggesting that chronic energy excess leads to ectopic fat accumulation in the liver and pancreas, creating a vicious cycle where hepatic insulin resistance drives increased hepatic glucose production while pancreatic fat deposition impairs β -cell function [8].

The Metabolic Syndrome Paradigm

The metabolic syndrome construct provides a comprehensive theoretical framework linking anthropometric variations to biochemical abnormalities and cardiovascular risk. Originally described by Reaven [9] as “Syndrome X” and subsequently refined by multiple organizations including the World Health Organization [10], this paradigm conceptualizes T2DM not as an isolated disorder of glucose metabolism but as one component of a broader constellation of interrelated metabolic disturbances [11].

The American Diabetes Association Standards of Care incorporate this framework by emphasizing that diabetes management must address the complete cardiovascular risk profile rather than glycemic control alone, recognizing that anthropometric assessment provides essential information for risk stratification and treatment selection. For the Owerri population, this theoretical perspective suggests that anthropometric variations will not merely describe body composition but will serve as surrogate indicators for the severity of underlying metabolic dysfunction and the risk of diabetes complications.

Chronic Low-Grade Inflammation Theory

A critical theoretical advancement in understanding T2DM pathophysiology involves the recognition that the disease represents a chronic inflammatory condition. The “inflammation hypothesis” of T2DM, articulated by Hotamisligil and subsequently elaborated by numerous investigators, proposes that

obesity-induced, chronic low-grade inflammation in metabolic tissues is a crucial link between obesity and insulin resistance [12]; [13].

This framework identifies adipose tissue macrophage infiltration and the subsequent release of pro-inflammatory cytokines—particularly tumor necrosis factor-alpha (TNF- α), interleukin-6 (IL-6), and C-reactive protein (CRP)—as central mediators of insulin resistance in peripheral tissues. These inflammatory markers interfere with insulin signaling pathways through multiple mechanisms, including serine phosphorylation of insulin receptor substrates, activation of c-Jun N-terminal kinase (JNK) and I κ B kinase (IKK) pathways, and induction of endoplasmic reticulum stress [14]. The theory predicts that elevated inflammatory markers will correlate with both anthropometric measures of obesity and biochemical indicators of metabolic dysfunction.

In the context of Owerri, this theoretical framework suggests that T2DM patients will demonstrate elevated inflammatory markers compared to non-diabetic controls, and that the magnitude of these elevations will correlate with disease duration, glycemic control, and the presence of complications. The inflammatory profile may also reflect genetic and environmental factors specific to the Nigerian population, including polymorphisms in inflammatory genes and exposure to environmental triggers such as diet composition and infectious agents [15].

The Adipose Tissue Expandability Hypothesis

The “adipose tissue expandability hypothesis,” proposed by Virtue and Vidal-Puig, offers a theoretical explanation for why some obese individuals develop metabolic disease while others remain metabolically healthy. This framework suggests that when adipose tissue reaches its maximal expandability, excess lipids “spill over” into non-adipose tissues (liver, muscle, pancreas), precipitating lipotoxicity and insulin resistance [16].

This theory has significant implications for anthropometric assessment in T2DM. It suggests that simple measures of body size (BMI, total body weight) may inadequately capture metabolic risk compared to measures that reflect adipose tissue distribution and capacity, such as waist circumference, visceral adipose tissue area, or novel indices like the visceral adiposity index (VAI) and body shape index (BSI). For the Owerri population, this theoretical perspective predicts that anthropometric indices reflecting central obesity and adipose tissue distribution will demonstrate stronger correlations with biochemical markers of metabolic dysfunction than indices reflecting overall adiposity alone.

Socio-Ecological Model of Health Determinants

While biological theories explain the mechanisms of metabolic dysfunction, the socio-ecological model provides a framework for understanding how social, cultural, economic, and environmental factors influence the expression of biochemical and anthropometric variations in specific populations. This model, articulated by Bronfenbrenner and applied to chronic disease epidemiology by McKinlay and others, recognizes that individual health outcomes result from interactions across multiple levels: intrapersonal (biological, psychological), interpersonal (family, social networks), organizational (healthcare systems, workplaces), community (neighborhood resources, cultural norms), and public policy (national health priorities, economic structures).

CONCEPTUAL FRAMEWORK

Global Burden of Diabetes

Diabetes mellitus has reached epidemic proportions worldwide, representing one of the most significant public health challenges of the 21st century. The International Diabetes Federation (IDF) estimated that approximately 537 million adults were living with diabetes in 2021, and this number is projected to rise to 783 million by 2045 [17]; [18]. This represents a 46% increase in just over two decades, underscoring the urgent need for effective prevention and management strategies. The majority of these cases (approximately 90-95%) are type 2 diabetes mellitus, which is largely preventable through lifestyle modifications [19].

The global prevalence of diabetes in adults (20-79 years) is estimated at 10.5%, with significant regional variations. The Western Pacific region has the highest number of people with diabetes (206 million), followed by Southeast Asia (90 million) and Europe (61 million). However, the Middle East and North Africa region has the highest prevalence rate at 16.2%, followed by the Western Pacific at 13.6%. Sub-Saharan Africa, while currently having a lower prevalence than other regions, is experiencing the fastest rate of increase and is projected to see a 129% rise in cases by 2045.

The economic burden of diabetes is substantial and continues to grow. Global healthcare expenditure on diabetes was estimated at USD 966 billion in 2021, representing approximately 9% of total healthcare spending. The costs associated with diabetes management, complications, and lost productivity represent a significant challenge for healthcare systems worldwide, particularly in low- and middle-income countries where resources are limited. In many African countries, the majority of diabetes care costs are borne by individuals and their families, creating a significant financial burden and contributing to poverty.

Diabetes in Sub-Saharan Africa

Sub-Saharan Africa (SSA) is experiencing a rapid epidemiological transition characterized by a shift from infectious diseases to non-communicable diseases (NCDs) as the leading causes of morbidity and mortality. This transition is driven by urbanization, changes in dietary patterns, increasing sedentary lifestyles, and aging populations [20]. Diabetes is at the forefront of this transition, with the IDF estimating that 24 million adults in Africa are currently living with diabetes, and this number is projected to increase by 129% to 55 million by 2045.

Motala provided a comprehensive review of type 2 diabetes mellitus in sub-Saharan Africa, highlighting both the challenges and opportunities in addressing this growing health crisis. The authors noted that the prevalence of type 2 diabetes in SSA varies widely across countries and populations, ranging from 1% in rural areas to over 12% in urban settings. This urban-rural gradient reflects the influence of lifestyle factors associated with urbanization, including increased consumption of processed foods, reduced physical activity, and higher rates of overweight and obesity [21].

Diabetes in Nigeria

Nigeria has one of the highest numbers of people with diabetes in Africa, with current estimates suggesting that over 5 million adults are living with the condition. The prevalence of diabetes in Nigeria has increased significantly over the past three decades, driven by urbanization, changes in dietary habits, increasing obesity rates, and aging populations [22]. The IDF estimates that the prevalence of diabetes in Nigeria is approximately 5.8%, though this figure may underestimate the true burden due to limited screening and diagnostic capacity.

A comparative analysis of type 2 diabetes prevalence and clinical profiles in Ethiopia and Nigeria conducted between 2020 and 2021 revealed important differences between the two countries [23]. The study included 1,727 participants using a multistage sampling method and the WHO STEPS tool. The overall prevalence of prediabetes and diabetes was 15.8% and 7.0%, respectively. The prevalence of newly diagnosed diabetes was 3.4% by fasting plasma glucose and 4.0% by oral glucose tolerance test.

The authors concluded that individuals with diabetes in Ethiopia compared to Nigeria have different anthropometric and clinical profiles characterized by a young age of onset, leanness, lower BMI, and waist circumference. These findings suggest that the management of diabetes should be tailored to the unique physiologic and clinical profiles of the population. For Nigeria, the high prevalence of obesity among diabetic patients highlights the importance of weight management as a key component of diabetes care.

Pathophysiology of Type 2 Diabetes Mellitus

Insulin Resistance and Beta-Cell Dysfunction

Type 2 diabetes mellitus is characterized by two fundamental pathophysiological defects: insulin resistance and progressive beta-cell dysfunction. Insulin resistance refers to the diminished ability of insulin to stimulate glucose uptake in peripheral tissues (primarily skeletal muscle and adipose tissue) and to suppress hepatic glucose production [24]. In the early stages of the disease, pancreatic beta cells compensate for insulin resistance by increasing insulin secretion, maintaining normoglycemia. This compensatory phase may last for years or even decades before beta-cell function deteriorates to the point where relative insulin deficiency and hyperglycemia develop.

The mechanisms underlying insulin resistance are complex and involve multiple tissues and signaling pathways. In skeletal muscle, insulin resistance results in reduced glucose uptake and utilization, contributing to postprandial hyperglycemia. Recent research has highlighted the role of mitochondrial dynamics and endoplasmic reticulum (ER) stress in muscle insulin resistance. Mitochondria-associated ER membranes (MAMs) serve as critical sites for calcium, lipid, and metabolite exchange, and their dysregulation has been implicated in the pathogenesis of muscle insulin resistance. Additionally, alterations in mitochondrial dynamics and elevated levels of mitophagy—specifically the PINK1-Parkin-mediated pathway—have been linked to the onset of insulin resistance in type 2 diabetes. ER stress activates the unfolded protein response (UPR) through three major sensors: PKR-like ER kinase (PERK), inositol requiring enzyme 1 (IRE-1), and activating transcription factor 6 (ATF6).

Glucotoxicity impairs beta-cell function through multiple mechanisms, including increased oxidative stress, activation of stress signaling pathways, and impaired insulin gene transcription. Lipotoxicity contributes to beta-cell dysfunction through the accumulation of ceramides and other toxic lipid metabolites. Exposure of beta-cells to palmitate recapitulates key features of beta-cell failure, inducing apoptosis, raising basal insulin secretion, and inhibiting glucose-stimulated insulin secretion (GSIS). The molecular mechanisms underlying lipo- and glucolipotoxicity include ER stress, oxidative stress with excessive ROS production, mitochondrial dysfunction, inflammation, and impaired autophagic flux. Beta-cells from type 2 diabetes donors show signs of dysregulated autophagy, with more abundant autophagic vacuoles and autophagosomes, which can be induced by in vitro exposure of human islets to free fatty acids.

Lipid Profile Abnormalities

Dyslipidemia is a common feature of type 2 diabetes mellitus and contributes significantly to the increased cardiovascular risk observed in diabetic patients. The characteristic diabetic lipid profile includes elevated triglycerides, decreased HDL-cholesterol, and increased small dense LDL particles. These abnormalities result from insulin resistance affecting lipid metabolism in the liver and peripheral tissues.

Insulin resistance leads to increased hepatic production of very-low-density lipoprotein (VLDL) particles, which are triglyceride-rich. These VLDL particles exchange triglycerides for cholesterol esters with HDL and LDL particles through the action of cholesterol ester transfer protein (CETP), resulting in triglyceride-enriched HDL and LDL particles. The triglyceride-enriched HDL particles are more rapidly catabolized, leading to lower HDL-cholesterol levels. The triglyceride-enriched LDL

particles are converted to small dense LDL particles, which are more atherogenic than larger, more buoyant LDL particles.

Feingold and Grunfeld provided a comprehensive review of diabetes and dyslipidemia, noting that poor glycemic control increases serum triglyceride and non-HDL cholesterol levels while decreasing HDL-C levels. The authors emphasized that lipid management is a critical component of diabetes care, with statins being the first-line therapy for cardiovascular risk reduction in diabetic patients. They noted that most patients with diabetes will require statin therapy, with intensity of therapy based on cardiovascular risk.

The authors also discussed the management of severe hypertriglyceridemia in diabetes, which can increase the risk of acute pancreatitis. For patients with triglycerides > 10 mmol/L, intensive lifestyle interventions and pharmacotherapy (including fibrates and omega-3 fatty acids) are recommended. For patients with moderate hypertriglyceridemia (2.3-10 mmol/L), statin therapy remains the primary treatment, with additional agents considered if triglycerides remain elevated.

Kalra and Raizada further elaborated on dyslipidemia in diabetes, highlighting that weight loss can significantly improve lipid levels. A 5-10% reduction in body weight can decrease triglycerides by up to 30% and increase HDL-cholesterol by up to 15%. The authors noted that combination therapies may be beneficial in reducing cardiovascular disease risk in patients with diabetes, particularly those with mixed dyslipidemia.

The Heart Outcomes Prevention Evaluation (HOPE) study demonstrated the cardiovascular benefits of intensive lipid management in diabetic populations. The study showed that statin therapy reduced the risk of major cardiovascular events by approximately 25% in diabetic patients, similar to the benefit observed in non-diabetic patients [25]. These findings support the aggressive management of dyslipidemia in diabetes.

Lipid parameters also show strong correlations with anthropometric measures. Triglycerides are typically elevated in obesity due to increased hepatic VLDL production and reduced clearance. HDL-cholesterol is decreased due to enhanced catabolism of triglyceride-enriched HDL particles. These lipid abnormalities contribute to the increased cardiovascular risk observed in obese diabetic patients.

Diabetes Management and Prevention Strategies

The management of type 2 diabetes mellitus involves a comprehensive approach including lifestyle modifications, pharmacological therapy, and regular monitoring of glycemic control and complications. The ADA Standards of Medical Care in Diabetes provide evidence-based recommendations for diabetes management, with goals of achieving glycemic control, reducing cardiovascular risk, and preventing complications.

For individuals with established diabetes, medical nutrition therapy is recommended to promote healthy eating patterns that achieve glycemic targets, reduce cardiovascular risk, and support weight management. There is no single “diabetic diet” that is optimal for all patients; rather, individualized meal plans should be developed based on the patient’s preferences, cultural practices, and metabolic goals. The 2024 ADA Standards of Care emphasize food-based eating styles that incorporate healthy fats and Mediterranean-style eating patterns, with new guidance on religious and time-restricting fasting [26]. Carbohydrate counting, the plate method, and Mediterranean-style eating patterns are all effective approaches. Protein intake goals should be individualized, with the 2024 ADA Standards stating there is no evidence that adjusting daily protein intake (typically 1–1.5 g/kg body weight/day) will improve health in individuals without diabetic kidney disease. Those with diabetic kidney disease should maintain dietary protein at no more than 0.8 g/kg desirable body weight/day.

Physical activity is another essential component of diabetes management. The ADA recommends at least 150 minutes per week of moderate-intensity aerobic activity, spread over at least three days per

week, with no more than two consecutive days without activity. Resistance training is also recommended at least twice per week. Physical activity improves insulin sensitivity, promotes weight loss, reduces cardiovascular risk, and improves overall well-being.

Pharmacological therapy for type 2 diabetes includes various classes of medications targeting different aspects of the disease pathophysiology. Metformin remains the first-line therapy for most patients due to its efficacy, safety, low cost, and potential cardiovascular benefits. When metformin alone is insufficient to achieve glycemic targets, additional agents may be added, including sulfonylureas, DPP-4 inhibitors, GLP-1 receptor agonists, SGLT2 inhibitors, thiazolidinediones, and insulin.

METHODOLOGY

In carrying out this study comparative cross-sectional study was adopted. The study population consisted of one hundred (100) diagnosed type 2 diabetic patients and one hundred (100) apparently healthy, age- and sex-matched non-diabetic individuals who served as controls. All participants were aged between 30 and 70 years. Ten (10) mL of venous blood samples were collected from each participant after overnight fast using standard aseptic procedures and distributed into appropriate tubes for the determination of fasting plasma glucose (FPG), glycated hemoglobin (HbA1c), insulin, lipid profile, and oxidative stress markers. Serum and plasma were prepared by centrifugation at 3000 rpm for 10 minutes, while whole blood was used for selected analyses. The samples were analyzed using standard laboratory methods. Data obtained were analyzed using Statistical Package for Social Sciences (SPSS) version 25.0. Results were expressed as Mean $\pm$ Standard Deviation. The test of significance was determined using independent samples t-test, while Pearson correlation analysis was used to assess relationships between variables, with $p < 0.05$ considered statistically significant.

RESULT AND DISCUSSION

Table 1 Pearson Correlation between Lipid Profile and Anthropometric Parameters in Type 2 Diabetic Patients

		TC	TG	HDL	LDL	BMI	WC	WHR	WHtR	BRI	VAI
TC	r-value	1	-.172	-.256*	-.044	-.051	.122	-.117	-.020	.107	.019
	p-value		.086	.010	.664	.616	.226	.245	.842	.291	.848
	n	100	100	100	100	100	100	100	100	100	100
TG	r-value	-.172	1	.049	-.013	.077	-.095	.175	-.117	.205*	-.156
	p-value	.086		.629	.900	.448	.348	.082	.246	.041	.121
	n	100	100	100	100	100	100	100	100	100	100
HDL	r-value	-.256*	.049	1	-.012	.057	.337*	.046	-.086	-.351*	.236*
	p-value	.010	.629		.903	.572	.001	.647	.397	.000	.018
	n	100	100	100	100	100	100	100	100	100	100
LDL	r-value	-.044	-.013	-.012	1	.125	-.387*	.076	-.074	.053	-.030
	p-value	.664	.900	.903		.217	.000	.454	.463	.600	.770
	n	100	100	100	100	100	100	100	100	100	100
BMI	r-value	-.051	.077	.057	.125	1	.022	-.225*	.156	.031	-.043
	p-value	.616	.448	.572	.217		.826	.024	.121	.759	.667
	n	100	100	100	100	100	100	100	100	100	100
WC	r-value	.122	-.095	.337*	-.387*	.022	1	-.141	.017	-.085	.155
	p-value	.226	.348	.001	.000	.826		.162	.867	.402	.124
	n	100	100	100	100	100	100	100	100	100	100
WHR	r-value	-.117	.175	.046	.076	-.225*	-.141	1	-.105	.187	.047
	p-value	.245	.082	.647	.454	.024	.162		.297	.062	.639
	n	100	100	100	100	100	100	100	100	100	100
WHtR	r-value	-.020	-.117	-.086	-.074	.156	.017	-.105	1	-.227*	.036
	p-value	.842	.246	.397	.463	.121	.867	.297		.023	.725
	n	100	100	100	100	100	100	100	100	100	100
BRI	r-value	.107	.205*	-.351*	.053	.031	-.085	.187	-.227*	1	.143
	p-value	.291	.041	.000	.600	.759	.402	.062	.023		.156
	n	100	100	100	100	100	100	100	100	100	100
VAI	r-value	.019	-.156	.236*	-.030	-.043	.155	.047	.036	.143	1
	p-value	.848	.121	.018	.770	.667	.124	.639	.725	.156	
	n	100	100	100	100	100	100	100	100	100	100

Key: r = Pearson Correlation

* Correlation is significant at the 0.05 level

High-density lipoprotein cholesterol levels were significantly lower in the diabetic group compared to controls. HDL plays a protective role in lipid metabolism by facilitating reverse cholesterol transport from peripheral tissues to the liver. The reduction in HDL-C observed in this study may be due to increased exchange of triglycerides for cholesteryl esters between HDL and very-low-density lipoproteins, mediated by cholesteryl ester transfer protein. This process results in triglyceride-rich HDL particles that are rapidly catabolized [27]. Additionally, insulin resistance may impair the synthesis of apolipoprotein A-I, a major structural component of HDL, further contributing to reduced HDL levels. This finding is consistent with several previous studies that have reported decreased HDL-C concentrations in individuals with type 2 diabetes [28],[29], though some localized studies in Owerri have reported less pronounced differences in HDL-C.

Conclusion and Recommendations

In conclusion, lipid profile parameters, including total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C), are important indicators of metabolic and cardiovascular health among patients with type 2 diabetes mellitus. Their association with anthropometric parameters such as BMI, WC, WHR, WHtR, BRI, and VAI demonstrates the relevance of body composition and fat distribution in understanding lipid abnormalities in diabetic patients. Anthropometric indices, particularly those reflecting central and visceral adiposity, provide useful information that can complement biochemical assessment of lipid status. Therefore, the combined assessment of lipid profile and anthropometric parameters is essential for effective identification and monitoring of cardiometabolic risk among patients with type 2 diabetes mellitus. Patients with type 2 diabetes mellitus should undergo regular lipid profile and anthropometric assessments to facilitate early identification of dyslipidaemia and abnormal adiposity. Patients should be encouraged to maintain healthy lifestyle practices, including balanced nutrition, regular physical activity, and adherence to prescribed diabetes management, to improve lipid levels and maintain healthy body composition. Healthcare professionals should incorporate both conventional and additional anthropometric indices, including BMI, WC, WHR, WHtR, BRI, and VAI, into routine monitoring where feasible, while further research should be encouraged to establish their usefulness in predicting cardiometabolic risk.

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