© DEC 2025 | IRE Journals | Volume 9 Issue 6 | ISSN: 2456-8880
DOI: https://doi.org/10.64388/IREV9I6-1713113

Evaluation of Lipid Profile Alterations and Their Clinical Significance Among Women with Polycystic Ovarian Syndrome in Kogi State, Nigeria.

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Abstract- ***Polycystic Ovarian Syndrome (PCOS), a prevalent endocrine disorder among women of reproductive age and is frequently accompanied by metabolic disturbances that considerably elevate long-term cardiovascular risk. Among these abnormalities, dyslipidemia is one of the most common and clinically significant; however, evidence from sub-Saharan Africa, particularly Nigeria, remains limited. This study therefore evaluated lipid profile alterations and their clinical significance, with a focus on their contribution to metabolic and cardiovascular disease risk among women with PCOS in Kogi State, Nigeria. A cross-sectional analytical design was employed, involving 110 women diagnosed with PCOS and 40 age-matched controls without PCOS to make a total of 150 samples. Fasting blood samples were obtained for the assessment of lipid parameters, while anthropometric measurements were recorded to calculate cardiometabolic indices, including the Visceral Adiposity Index (VAI) and Lipid Accumulation Product (LAP). Statistical analyses comprised group comparisons and correlation analyses to explore associations between lipid parameters and adiposity-related indices was employed. Women with PCOS exhibited a significantly more atherogenic lipid profile than controls, characterized by elevated total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and triglycerides (TG), alongside significantly reduced high-density lipoprotein cholesterol (HDL-C). Notably, the mean HDL-C level was markedly lower in the PCOS group (1.14 ± 0.39 mmol/L) compared with controls (1.47 ± 0.21 mmol/L; $p < 0.001$), consistent with the characteristic dyslipidemia which is associated with insulin resistance and hyperandrogenism in PCOS. Correlation analyses revealed that TG levels were moderately and positively associated with both body mass index (BMI) ($r = 0.420$, $p < 0.001$) and waist circumference (WC) ($r = 0.455$, $p < 0.001$), whereas HDL-C display significant inverse correlations with BMI ($r = −0.315$, $p = 0.001$) and WC ($r = −0.340$, $p < 0.001$). In contrast, LDL-C demonstrated no statistically significant association with BMI or WC. As expected from lipid physiology, total cholesterol showed a strong positive correlation with LDL-C ($r = 0.835$, $p < 0.01$), while LDL-C was moderately inversely correlated with HDL-C ($r = −0.548$, $p < 0.01$). BMI also correlated positively with TC ($r = 0.413$, $p < 0.01$) and LDL-C ($r = 0.546$, $p < 0.01$), and negatively with HDL-C ($r = −0.402$, $p < 0.01$), strengthening the role of adiposity in lipid dysregulation. LAP, an index derived from waist circumference and triglycerides, demonstrated a very strong positive correlation with TG ($r = 0.720$, $p < 0.01$), underscoring its sensitivity as a marker of visceral fat–related dyslipidemia. Generally, dyslipidemia particularly elevated TC, LDL-C, and TG was highly prevalent among women with PCOS in Kogi State, Nigeria and was closely associated with indices of visceral adiposity and elevated markers of myocardial injury or stress (cTnI, CK-MB, and myoglobin). These findings indicate a substantial burden of subclinical cardiometabolic risk among women with PCOS in this population. Incorporating simple, non-invasive adiposity indices such as VAI and LAP alongside with conventional lipid profiling may improve early risk categorization. Routine cardiometabolic screening and targeted preventive interventions are therefore recommended to extenuate the progression to expressed cardiovascular disease in women with PCOS.***

Keywords: Polycystic Ovarian Syndrome, Dyslipidemia, Lipid Profile, Visceral Adiposity Index, Cardiometabolic Risk.

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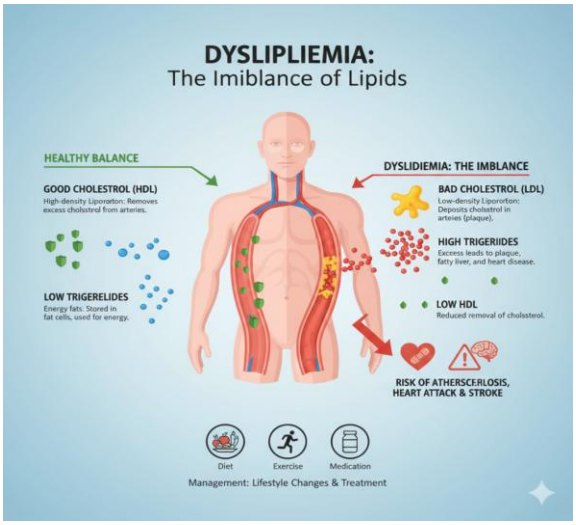
DOI: https://doi.org/10.64388/IREV9I6-1713113

I. INTRODUCTION

1.1 Background of the study

Lipid profile alterations, which can also be called *dyslipidemia*, it is refer to abnormal levels of lipids (fats) and lipoproteins (fat-carrying proteins) in the blood focusing on the pathophysiology and specific alterations. Dyslipidemia is a major, modifiable risk factor for Atherosclerosis and subsequent Cardiovascular Disease (CVD), including myocardial infarction and stroke (Berberich & Hegele, 2022). Dyslipidemia is an abnormal amount of lipids (*fats*) in the blood, such as high total cholesterol, high LDL (*"bad"*) cholesterol, low HDL (*"good"*) cholesterol, or high triglycerides. It is a significant risk factor for cardiovascular diseases, like atherosclerosis, and is often caused by genetics, diet, and lifestyle factors such as obesity and a sedentary lifestyle. Diagnosis is made through a blood test called a lipid profile, and management typically involves lifestyle changes and, when necessary, medication (Pappan *et al.,* 2024). Dyslipidemia refers to abnormal levels of lipids in the bloodstream, which poses a significant risk factor for cardiovascular diseases (CVD). Dysregulation in these lipid levels, whether due to genetic predispositions or lifestyle factors, can lead to atherosclerosis and other CVD complications. Diagnosis often relies on lipid profile tests, with recommended target levels for optimal CV health. Treatment strategies work to mitigate risks by targeting specific lipid abnormalities, emphasizing lifestyle modifications, and considering comorbidities to individualize care. Given the multifaceted nature of dyslipidemia management, a multidisciplinary approach is essential for comprehensive patient care (Blasco & Ascaso, 2019; Pappan *et al.,* 2024).



(Ige *et al.,* 2024).

1.2 Pathophysiology and Clinical Significance of Dyslipidemia

Dyslipidemia is defined by quantitative or qualitative abnormalities in the plasma lipids, encompassing high concentrations of Total Cholesterol (T-CHOL), Low-Density Lipoprotein Cholesterol (LDL-CHOL), and Triglycerides (TG), and/or low concentrations of High-Density Lipoprotein Cholesterol (HDL-CHOL) (Mach *et al.,* 2018). It stands as a major modifiable risk factor that initiates and accelerates the development of Atherosclerosis, the underlying pathology of most Cardiovascular Disease (CVD) events, such as myocardial infarction and ischemic stroke (Ference *et al.,* 2017).

1.3 Histtory and Background

Knowing the signs and symptoms of dyslipidemia is essential for prompt intervention and preventing related complications. As dyslipidemia frequently advance silently, routine lipid screening (especially in high-risk populations) remains a cornerstone for early detection and effective management. Clinicians should consider the broader clinical context, including family history and risk factors, to guide appropriate interventions and reduce the burden of cardiovascular diseases associated with dyslipidemia. Appropriate social history include tobacco use or specific details about diet. Past medical history is vital in identifying patients needing primary prevention versus secondary prevention if statin therapy requires initiation. Lastly, family history is important to identify familial hypercholesterolemia (Pappan *et al.,* 2024).

Some patients with severe or untreated dyslipidemia may develop signs and symptoms related to the complications of dyslipidemia, such as atherosclerosis, coronary artery disease, peripheral artery disease, stroke, and heart failure. Some of the possible signs and symptoms of dyslipidemia are: Xanthomas: yellowish fat deposits visible on the skin of the eyelids (xanthelasma), palms, tendons, or other areas; showing a high levels of cholesterol or triglycerides in the blood. Arcus senilis, a gray or white ring around the eye's cornea caused by cholesterol depositing in the corneal margin and signalling high cholesterol levels in the blood. Lipemia retinalis, a milky appearance in the retinal vessels due to high blood triglyceride levels causing blurred vision and may indicate severe hypertriglyceridemia, though rare anyway. Lower limb ischemia, a common symptom of peripheral artery disease, caused by the narrowing or blockage of the arteries that supply blood to the legs due to atherosclerosis; usually characterized by pain or cramping during physical activity (walking or exercising) and can be improves with rest. Lower limb ischemia is due to high levels of LDL, cholesterol, or triglycerides in the blood. Angina, a common symptom of coronary artery disease, caused by the constricting or blockage of the arteries that supply blood to the heart due to atherosclerosis, and remarkably occurs when more oxygen is needed in the heart, like during physical or emotional stress, and may run to the neck, jaw, shoulders, or back, exhibiting a high levels of LDL cholesterol or triglycerides in the blood. Transient ischemic attacks and strokes, a risk of atherosclerosis in cerebral arteries, contributing to sudden interruption of blood flow to the brain due to a clot or a bleed in impaired blood vessel walls. Sudden weakness, slurred speech, or visual disturbances are some of the symptoms (Lugo-Somolinos, 2003; Karantas *et al.,* 2021; Pappan *et al.,* 2024).

1.4 Study Environment: Kogi State, Nigeria

This research is therefore essential for characterizing the specific lipid profile alterations among women with PCOS in Kogi State, Nigeria, and clearly establishing the clinical implications of these alterations for future CVD risk stratification and public health planning. Research specific to Nigerian populations is vital due to the influence of genetic and environmental factors on the manifestation of metabolic diseases like PCOS and dyslipidemia (Odigie *et al.,* 2022).

1.5 Localized Data Gap: While general studies confirm the high prevalence of dyslipidemia in PCOS globally, the precise prevalence, severity, and local correlation of specific lipid abnormalities with insulin resistance and hyperandrogenism in Kogi State, Nigeria, remain largely undocumented (Adewuni *et al.,* 2022; Omokanye *et al.,* 2015).

1.6 Ethnic Specificity: Decentralized studies are necessary to ascertain if the typical atherogenic dyslipidemic pattern (high TGs, low HDL-C) observed in Caucasian cohorts is equally pronounced or modulated by specific dietary, lifestyle, or genetic factors unique to the Kogi State population (Odigie *et al.,* 2022).

1.7 Clinical Postulation: The findings from this study in Kogi State will provide local healthcare providers with evidence-based benchmarks, enabling more targeted and appropriate screening protocols and management strategies for PCOS patients in the region (Adewuni *et al.,* 2022). This research is therefore essential for characterizing the specific lipid profile alterations among women with PCOS in Kogi State, Nigeria, and clearly establishing the clinical implications of these alterations for future CVD risk stratification and public health planning.

II. LITERATURE REVIEW

2.1 Overview of Polycystic Ovarian Syndrome (PCOS)

Polycystic Ovarian Syndrome (PCOS) is the most common endocrine disorder affecting women of reproductive age worldwide, with an estimated prevalence ranging from 6% to 20%, depending on the diagnostic criteria and population studied (Khan *et al.,* 2025; Bahmanpoori Ghalehzan *et al.,* 2025). It is classically characterized by a triad of clinical and biochemical features, namely oligo- or anovulation, hyperandrogenism (clinical or biochemical), and polycystic ovarian morphology on ultrasound, as defined by the widely accepted Rotterdam diagnostic criteria. These features manifest heterogeneously, contributing to the phenotypic diversity of PCOS and

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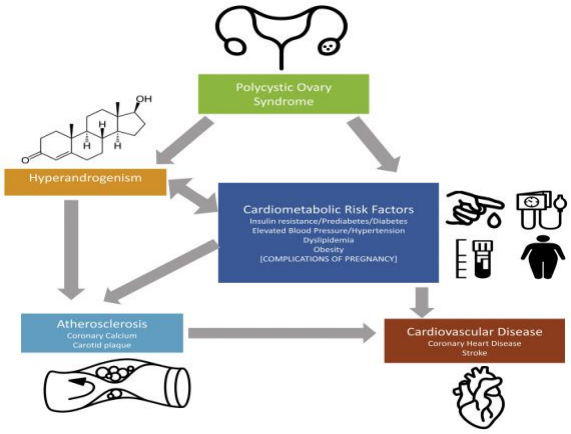
DOI: https://doi.org/10.64388/IREV9I6-1713113

complicating diagnosis and management across various ethnic and geographic populations (Manjula *et al.,* 2024).Aggarwal et al., 2019)



Historically, PCOS was principally conceptualized as a reproductive disorder due to its powerful association with menstrual irregularities, infertility, and androgen-related symptoms such as hirsutism and acne. However, accumulating evidence over the past two decades has modified this narrow perspective, recognizing PCOS as a complex multisystem disorder with substantial metabolic and cardiovascular implications. Insulin resistance is now considered a central pathophysiological characteristic of PCOS, affecting both obese and lean women, and is present in up to 70% of affected individuals (Azziz *et al.,* 2016; Teede *et al.,* 2018). This insulin resistance contributes to compensatory hyperinsulinaemia, which exacerbates ovarian androgen production and suppresses hepatic sex hormone-binding globulin synthesis, thereby amplifying hyperandrogenism and perpetuating the syndrome.

Beyond insulin resistance, women with PCOS frequently exhibit a constellation of cardiometabolic abnormalities, including central obesity, dyslipidaemia (elevated LDL-cholesterol and triglycerides with reduced HDL-cholesterol), impaired glucose tolerance, type 2 diabetes mellitus, and hypertension (Levine *et al.,* 2017; Zhao *et al.,* 2020). These metabolic derangements place women with PCOS at an increased lifetime risk of cardiovascular disease, independent of traditional risk factors and body mass index. Recent longitudinal and meta-analytic studies have demonstrated that women with PCOS have a significantly higher risk of developing coronary artery disease, myocardial infarction, and heart failure later in life, reinforcing the classification of PCOS as a cardiovascular risk-enhancing condition (Glintborg *et al.,* 2018; Guan *et al.,* 2022).

Importantly, emerging research highlights that metabolic dysfunction in PCOS may begin early, often preceding overt clinical cardiovascular disease. Subclinical markers of cardiovascular risk including endothelial dysfunction, arterial stiffness, visceral adiposity, and elevated myocardial biomarkers have been reported in young women with PCOS, suggesting early myocardial stress and vascular injury (Nandakumar *et al.,* 2024; Papathanasiou, 2024). These findings underscore the need to move beyond symptom-based management toward comprehensive cardiometabolic risk assessment and early preventive strategies in this population.

In low- and middle-income countries such as Nigeria, the burden of PCOS may be compounded by rising rates of obesity, sedentary lifestyles, and limited access to early diagnostic and preventive healthcare services. Thus far, data on the metabolic and cardiovascular sequelae of PCOS in African populations remain scarce, highlighting a critical research gap. Understanding PCOS as both a reproductive and metabolic disorder is therefore essential for improving long-term health outcomes and informing evidence-based screening and intervention strategies tailored to diverse populations (Bahmanpoori Ghalehzan *et al.,* 2025; Khan *et al.,* 2025).

2.2 Understanding Dyslipidemia in PCOS

Dyslipidaemia, defined as an abnormal concentration of circulating lipids or lipoproteins, is one of the most prevalent and clinically significant metabolic complications associated with polycystic ovarian syndrome (PCOS). It represents a major independent risk factor for atherosclerosis and subsequent cardiovascular disease (CVD), contributing substantially to the long-term morbidity observed in affected women (Mahmoud & El-Ghitany, 2022). Unlike isolated lipid abnormalities, dyslipidaemia in PCOS typically occurs as part of a broader cardiometabolic disturbance driven by insulin

resistance, hyperandrogenism, and visceral adiposity, thereby amplifying cardiovascular risk even in young women and in those without overt obesity. Dyslipidaemia in PCOS represents a central pathophysiological link between endocrine dysfunction and cardiovascular disease. Its early onset, atherogenic nature, and frequent under-recognition highlight the necessity for comprehensive lipid evaluation and aggressive cardiometabolic risk management in women with PCOS, particularly in low-resource settings where preventive screening is often limited. The typical dyslipidemic pattern observed in women with PCOS is distinctly atherogenic and is characterized by the following alterations in the standard lipid profile. This pattern is characterized by elevated triglycerides (TGs), reduced high-density lipoprotein cholesterol (HDL-C), increased low-density lipoprotein cholesterol (LDL-C), and elevated total cholesterol (TC), which are often accompanied by qualitative changes in lipoprotein particle size and function. The explanations are as follows:

I. Elevated Triglycerides (TGs)

Hypertriglyceridaemia is one of the most consistent lipid abnormalities reported in women with PCOS. Elevated TG levels correlate strongly with insulin resistance and central (visceral) obesity, both of which are highly prevalent in PCOS (Khan *et al.,* 2025). Insulin resistance promotes increased hepatic very-low-density lipoprotein (VLDL) production and impaired lipoprotein lipase activity, leading to reduced TG clearance from circulation. Elevated TGs not only reflect metabolic dysfunction but also play a direct role in atherogenesis by promoting endothelial dysfunction and fostering the formation of small dense LDL particles, thereby accelerating plaque development.

II. Reduced High-Density Lipoprotein Cholesterol (HDL-C)

Low HDL-C levels are frequently observed in women with PCOS and represent a critical loss of cardioprotective function. HDL-C is central to reverse cholesterol transport, anti-inflammatory activity, and antioxidant defense within the vascular endothelium. In PCOS, hyperinsulinaemia and androgen excess suppress HDL synthesis and alter its composition, leading to reduced HDL-C concentrations and impaired HDL functionality (Mahmoud & El-Ghitany, 2022). This reduction significantly increases cardiovascular risk, particularly when combined with elevated TGs and LDL-C.

III. Increased Low-Density Lipoprotein Cholesterol (LDL-C)

While absolute LDL-C concentrations may be modestly elevated in some women with PCOS, the qualitative nature of LDL particles is of greater clinical importance. PCOS is associated with a predominance of *small dense LDL (sdLDL)* particles, which are more susceptible to oxidation, have greater arterial wall penetration, and exhibit prolonged circulation time. These properties render sdLDL markedly more atherogenic than larger, buoyant LDL particles, substantially increasing the risk of plaque formation and coronary artery disease (Mahmoud & El-Ghitany, 2022). This qualitative dyslipidaemia often goes undetected in routine lipid panels, underscoring the insidious cardiovascular risk in PCOS.

IV. Elevated Total Cholesterol (TC)

Total cholesterol levels are frequently elevated in PCOS, largely reflecting the combined increases in LDL-C and triglyceride-rich lipoproteins. Elevated TC serves as a composite marker of atherogenic lipid burden and has been associated with increased arterial stiffness and subclinical atherosclerosis in PCOS populations (Khan *et al.,* 2025). Importantly, elevated TC in young women with PCOS may precede clinical cardiovascular disease by decades, emphasizing the need for early screening and intervention.

2.3 Clinical and Cardiovascular Implications

Collectively, this atherogenic lipid profile significantly accelerates atherosclerotic processes and contributes to the heightened lifetime risk of cardiovascular disease observed in women with PCOS. Large epidemiological studies and meta-analyses have demonstrated that dyslipidaemia in PCOS is associated with increased carotid intima-media thickness, endothelial dysfunction, and elevated myocardial biomarkers, suggesting early subclinical cardiovascular injury (Guan *et al.,* 2022; Nandakumar *et al.,* 2024). Importantly, these lipid abnormalities persist even after adjustment for body mass index,

reinforcing dyslipidaemia as an intrinsic feature of PCOS rather than a mere consequence of obesity.

2.4 Pathophysiological Mechanisms Linking PCOS to Dyslipidemia

The dyslipidemia observed in women with Polycystic Ovary Syndrome (PCOS) is not random but reflects the complex interaction between endocrine disturbances, metabolic dysfunction, and inflammatory pathways inherent to the syndrome. These mechanisms synergistically promote an atherogenic lipid profile characterized by elevated triglycerides (TGs), reduced high-density lipoprotein cholesterol (HDL-C), and increased small dense low-density lipoprotein (sdLDL), thereby predisposing affected women to heightened cardiovascular disease (CVD) risk.

2.5 Insulin Resistance and Hyperinsulinemia

Insulin resistance (IR) is a fundamental pathophysiological hallmark of PCOS and is present in both obese and lean phenotypes. In states of insulin resistance, compensatory hyperinsulinemia develops to maintain euglycemia. However, elevated insulin levels fail to adequately suppress hormone-sensitive lipase activity in adipocytes, resulting in increased lipolysis and excessive release of free fatty acids (FFAs) into the circulation (Moran *et al.,* 2015; Mahmoud & El-Ghitany, 2022). The liver, exposed to this increased FFA influx, responds by enhancing hepatic triglyceride synthesis and overproducing very-low-density lipoproteins (VLDL). These triglyceride-rich lipoproteins are subsequently secreted into the bloodstream, leading to hypertriglyceridemia, a hallmark lipid abnormality in PCOS (Wild *et al.,* 2018). Elevated circulating VLDL particles promote lipid exchange through cholesteryl ester transfer protein (CETP), whereby triglycerides are transferred to HDL-C and LDL-C in exchange for cholesterol esters. Triglyceride-enriched HDL particles become structurally unstable and are rapidly cleared from circulation, resulting in reduced HDL-C levels. Similarly, triglyceride-enriched LDL particles undergo hepatic lipase–mediated hydrolysis, producing small dense LDL (sdLDL) particles. These sdLDL particles are particularly atherogenic due to their increased susceptibility to oxidation, enhanced arterial wall penetration, and prolonged plasma residence time (Rizzo *et al.,* 2016; Khan *et al.,* 2025). Thus, insulin resistance serves as a central driver of dyslipidemia and cardiovascular risk in PCOS.

2.6 Hyperandrogenism

Hyperandrogenism, a defining endocrine feature of PCOS, independently contributes to lipid abnormalities and interacts bidirectionally with insulin resistance. Elevated androgen levels stimulate hepatic lipase activity, an enzyme responsible for the hydrolysis of triglycerides and phospholipids in HDL particles. Increased hepatic lipase activity accelerates HDL catabolism, leading to reduced HDL-C concentrations and impaired reverse cholesterol transport (Legro *et al.,* 2017; Khan *et al.,* 2025). Furthermore, androgens influence adipose tissue distribution by promoting visceral fat accumulation, which is metabolically unfavorable and closely linked to insulin resistance. Hyperandrogenism has also been associated with altered expression of genes involved in lipid synthesis and clearance, contributing to elevated LDL-C levels and increased sdLDL formation independent of body mass index (BMI) (Teede *et al.,* 2023). These androgen-mediated effects underscore the importance of hormonal imbalance in shaping the dyslipidemic phenotype of PCOS.

2.7 Obesity and Chronic Low-Grade Inflammation

Obesity, particularly central or visceral adiposity, commonly coexists with PCOS and amplifies metabolic and lipid disturbances. Visceral adipose tissue acts as an active endocrine organ, secreting pro-inflammatory cytokines and adipokines such as tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), resistin, and leptin, while reducing the secretion of the insulin-sensitizing adipokine adiponectin (Dumesic *et al.,* 2016; Mahmoud & El-Ghitany, 2022). This chronic low-grade inflammatory state interferes with insulin signaling pathways by inducing serine phosphorylation of insulin receptor substrates, thereby exacerbating insulin resistance. Inflammation also impairs lipoprotein lipase activity, reducing peripheral triglyceride clearance and sustaining hypertriglyceridemia. Additionally, inflammatory cytokines promote oxidative stress, increasing LDL oxidation and further enhancing the atherogenicity of circulating lipoproteins (Ranasinha *et al.,* 2015; Wild *et al.,* 2018). Putting together, visceral obesity, inflammation, insulin resistance, and hyperandrogenism form a self-perpetuating cycle that

drives lipid abnormalities and increases long-term cardiovascular risk in women with PCOS.

2.8 Clinical Significance and Association with Cardiovascular Disease (CVD) Risk

The dyslipidemia associated with Polycystic Ovary Syndrome (PCOS) carries substantial clinical significance, as it represents a major contributor to the increased cardiometabolic burden observed in affected women. Beyond reproductive dysfunction, PCOS is now widely recognized as a systemic metabolic disorder that predisposes women to premature cardiovascular disease (CVD). The characteristic lipid abnormalities elevated triglycerides (TGs), reduced high-density lipoprotein cholesterol (HDL-C), and increased small dense low-density lipoprotein (sdLDL) synergistically accelerate atherogenesis and substantially elevate lifetime cardiovascular risk (Ghosh *et al.,* 2023; Ige *et al.,* 2024).

2.9 Atherosclerosis and Coronary Artery Disease (CAD)

Atherogenic dyslipidemia in PCOS plays a pivotal role in the initiation and progression of atherosclerosis. Elevated TG-rich lipoproteins and sdLDL particles readily penetrate the arterial intima, where they undergo oxidative modification and trigger endothelial dysfunction. Endothelial dysfunction, characterized by impaired nitric oxide bioavailability and increased expression of adhesion molecules, represents an early and critical step in atherogenesis (Rizzo *et al.,* 2016; Wild *et al.,* 2018).

Small dense LDL particles are particularly pathogenic due to their enhanced susceptibility to oxidation, prolonged plasma residence time, and reduced affinity for LDL receptors. These properties facilitate foam cell formation and plaque development within coronary arteries, increasing the risk of coronary artery disease (CAD) (Khan *et al.,* 2025). Concurrently, low HDL-C levels impair reverse cholesterol transport, limiting the removal of excess cholesterol from peripheral tissues and further promoting plaque accumulation. Importantly, epidemiological and imaging studies have demonstrated increased carotid intima–media thickness (CIMT), arterial stiffness, and subclinical atherosclerosis in women with PCOS, even at a young age and in the absence of obesity (Ranasinha *et al.,* 2015; Mahmoud & El-Ghitany, 2022). This observation underscores the role of intrinsic hormonal and metabolic disturbances, particularly insulin resistance and hyperandrogenism as key drivers of cardiovascular risk independent of traditional factors such as age and body mass index.

2.10 Metabolic Syndrome and Advances in Type 2 Diabetes Mellitus

Dyslipidemia is a key diagnostic factor of metabolic syndrome, alongside with abdominal obesity, hypertension, hyperglycemia, and insulin resistance. Metabolic syndrome is disproportionately prevalent among women with PCOS and represents a critical intermediary state linking PCOS to overt cardiovascular disease and Type 2 Diabetes Mellitus (T2DM) (Moran *et al.,* 2015; Khan *et al.,* 2025). The coexistence of dyslipidemia and insulin resistance creates a vicious cycle in which impaired glucose metabolism exacerbates lipid abnormalities, and vice versa. Hypertriglyceridemia and low HDL-C not only predict future cardiovascular events but also serve as markers of worsening insulin resistance and β-cell dysfunction. Longitudinal studies have shown that women with PCOS are at significantly increased risk of developing impaired glucose tolerance and T2DM at a younger age compared to the general female population, thereby accelerating the timeline toward cardiovascular morbidity (Teede *et al.,* 2018; Wild *et al.,* 2018). Thus, dyslipidemia in PCOS should not be viewed in isolation but rather as part of a broader cardiometabolic phenotype that signals heightened long-term risk.

2.11 Early Identification and Preventive Intervention

The early onset of dyslipidemia in PCOS offers a critical window of opportunity for preventive intervention. Given that many affected women are diagnosed during adolescence or early adulthood, timely screening and risk stratification can significantly alter disease trajectory. Current clinical guidelines emphasize routine assessment of lipid profiles in women with PCOS, regardless of age or body weight, to facilitate early detection of atherogenic patterns (Legro *et al.,* 2017; Teede *et al.,* 2018). Lifestyle modification remains the cornerstone of dyslipidemia management in PCOS. Weight reduction, dietary interventions, and regular physical activity have been shown to improve insulin

sensitivity, reduce triglyceride levels, and increase HDL-C concentrations. In cases where lifestyle measures are insufficient, pharmacological therapies such as statins, fibrates, and insulin-sensitizing agents may be employed to normalize lipid parameters and reduce cardiovascular risk (Khan *et al.,* 2025; Rizzo *et al.,* 2016). Emerging evidence also suggests that effective management of hyperandrogenism and insulin resistance can yield favorable downstream effects on lipid metabolism, further supporting a comprehensive, individualized treatment approach. Early and sustained intervention has the potential not only to improve lipid profiles but also to delay or prevent the onset of CVD, T2DM, and related complications in women with PCOS.

III. METHODOLOGY

3.1 Study Design

This study adopts a cross-sectional analytical design integrating biochemical components. It involves the recruitment of women diagnosed with PCOS and the evaluation of their cardiovascular risk markers.

3.2 Study Area

The research was carried out in Lojoja, Kogi State, Nigeria. Lokoja is the capital city of Kogi State Nigeria, located in Kogi Local Government Area of Kogi state. The city is a a region with a diverse population and increasing health concerns related to reproductive and cardiovascular health. It lies between latitude 7.450 and 7.520 North and longitude 6.410 and to 6.450 East of the Greenwich meridian. It is sandwiched to the west and east by the mount Patti ridge and river Niger respectively with an area of about five hundred and seventy-seven square kilometers. (577sq.km) The city has a humid tropical climate which is characterized by wet and dry season. The rainy season in the city begins towards the end of April and ends November with two peak periods in July and September. The highest temperatures occur in March and April just before the rainy season. The average population size of 265400 based on 2016 population census. The topography consists of rough terrain with mountainous landscape and classified as highland due to the fact that it has an elevation above 300 meters. Mount Patti with the highest point has a height of 1200 meters above sea level and gently reduces in height till it reaches river Niger at the height of about 400 meters above sea level.

3.3 Method of Data Collection

Data were collected using a structured interviewer-administered questionnaire collectively with clinical and laboratory assessments. Participants who met the inclusion criteria were recruited during clinic visits. After obtaining informed consent, socio-demographic and medical history information was collected. Anthropometric measurements (weight, height, waist circumference, blood pressure) were taken using standard procedures. Venous blood samples were collected following an overnight fast for analysis of myocardial biomarkers CTnI, CK-MB, MYO and cardiometabolic variables (fasting glucose, lipid profile). All samples were processed according to established laboratory protocols to ensure accuracy and reliability.

3.4 Data Collection Procedure

Sociodemographic information was obtained using a structured questionaires; medical history of the study participants were obtained from the medical records department. Laboratory assay methods of data collection were also adopted for this study; where a total of 150 participants was recruited for this research study, which was determined by Leslie Fisher's formula based on the frequency of PCOS in Nigeria, 110 were subjects with PCOS (with or without cardiovascular diseases) and were randomly selected for the study while 40 subjects were recruited as control. 5mL was collected from each participants and dispensed into and anticoagulated Ethylenediamine tetraacetic acid (EDTA) bottle (in ice pack) for lipid panel, Cardiac Troponin I (cTnI), Creatine Kinase-MB (CK-MB) and myoglobin (MYO) during their clinic days in the respective hospital and the sample was centrifuge to separate the plasma for analysis.

3.5 Study Instrument

The study instrument consisted of a structured questionnaire designed to obtain socio-demographic characteristics, reproductive history, lifestyle variables, and cardiometabolic risk factors. The questionnaire was pre-tested for clarity and validity before use. In addition, clinical measurement tools (calibrated weighing scale, stadiometer, measuring

tape, sphygmomanometer) and laboratory assay kits for myocardial biomarkers and metabolic tests served as complementary instruments for objective data collection.

3.6 Sampling Technique

A purposive sampling technique was used to recruit women diagnosed with PCOS from selected healthcare facilities, while age-matched controls without PCOS were selected using a systematic random sampling method from the same population. This approach ensured adequate representation of both study groups. The sample size was determined using standard sample size calculation formulas based on expected differences in myocardial biomarker levels, with adjustments for power and potential non-response. The target population includes women aged 20–50 years who have been clinically diagnosed with PCOS based on the Rotterdam Criteria (2004). A group of age-matched non-PCOS women served as controls for comparison. Participants was recruited from selected healthcare centers and fertility clinics in Lokoja, Okene, and Anyigba, where PCOS cases are commonly reported. Therefore, the study group comprises a total of 150 patients with 110 outpatient women with primary infertility, who had been diagnosed of PCOS in Obstretics and Gynaecology department of the above tertiary health institution, and 40 healthy volunteer women was used as control. Recruitement of participants was between May 2024 to September, 2025.

3.7 Data Analysis

Enzyme linked immunosorbent assay (ELISA) was use for the Cardiac enzymes (Troponin, CK-MB and Myoglobin); total cholesterol, HDL-cholesterol and triglyceride using Spectrophotometry assay (Enzymatic method), and Friedewald's formula was adopted for LDL-cholesterol and Body Mass Index (BMI) was calculated using the weight in kilograms (kg) divided by the square of the height in meters (m^2).

3.8 Exclusion and Inclusion Criteria:

I. Inclusion Criteria includes

Women of Childbearing age between 20–50 years, Ovulatory dysfunction, Hyperandrogenism, Primary infertility, Women residing in Kogi State, Nigeria and willing to give informed consent, Not on hormonal or insulin-sensitizing medications in the last 3 months and Diagnosed with PCOS using clinical, biochemical, and/or ultrasonographic criteria.

II. Exclusion criteria includes:

Hypertension, Smoking or endocrine disorder, Fertility drug, Pregnant or lactating women, Known diabetes mellitus or chronic cardiovascular disease and Women with other endocrine disorders such as Cushing's syndrome, thyroid dysfunction were excluded.

3.9 Ethical Consideration

The ethical clearance/approval for this study was obtained from Ethical committee of the Institutional Review Board (IRB) of Kogi State Specialist Hospital, Lokoja, Nigeria. Informed consent was obtained from all participants after explaining the purpose, procedure, and benefits prior to enrolment into the study. Confidentiality of participants' data and genetic information was strictly maintained. Participants were given an option to withdraw from the study at any time without any consequences.

3.10 Conflict of interest: No issue of conflicting interest available before and during this present research study.

3.11 Funding: Authors disclose no external funding sources.

3.12 Competing Interests: The authors declare that they have no potential conflict of interest.

3.13 Acknowledgment: The authors would like to appreciate the Department of Medical Laboratory Science, most especially Chemical Pathology Unit of Kogi State Specialist Hospital, Lokoja, Kogi State, Nigeria; Department of Medical Laboratory Science, Igbinedion University, Okada, Edo State, Nigeria; and Beacon Medical Laboratories, Lokoja, Kogi State, Nigeria.

© DEC 2025 | IRE Journals | Volume 9 Issue 6 | ISSN: 2456-8880
DOI: https://doi.org/10.64388/IREV9I6-1713113

IV. RESULTS

4.1 PRESENTATION OF DATA

Table 1: Showing The Relationship Between Serum Lipid Profile Parameters Among Women Diagnosed With PCOS And Control Subjects

Variables	Test	Control	T-values	P-value
T.Chol	4.85±0.51	4.20±0.30	9.63	<0.001
HDL-Chol	1.14±0.39	1.47±0.21	6.68	<0.001
LDL-Chol	3.36±0.63	2.39±0.36	11.61	<0.001
Triglyceride	1.70±0.38	1.68±0.33	0.412	0.68

Graph 1: Showing The Relationship Between Serum Lipid Profile Parameters Among Women Diagnosed With PCOS And Control Subjects

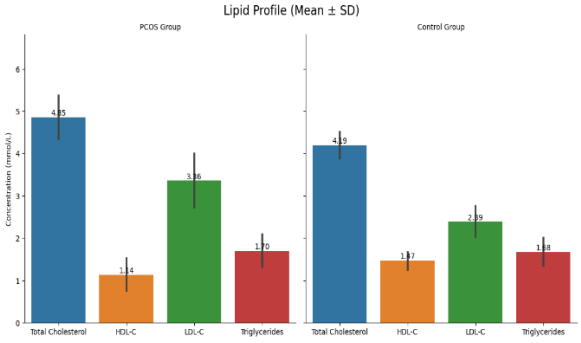


Table 2: The Relationship Between Lipid Profile Alterations And Clinical Features Of PCOS

Variables	T.CHOL	HDL-CHOL	LDL-CHOL	Triglyceride	BMI	WC (cm)	VAI	LAP
T.CHOL (mmol/L)	1							
HDL-CHOL (mmol/L)	-0.056	1						
LDL-CHOL (mmol/L)	0.835**	-.548**	1					
Triglyceride (mmol/L)	0.096	-.223*	0.075	1				
BMI	0.413**	-.402**	0.546**	0.037	1			
WC (cm)	0.179	0.013	0.148	0.087	0.238*	1		
VAI	0.184	-0.818**	0.498**	0.589**	0.437**	0.137	1	
LAP	0.176	-0.122	0.128	0.720**	0.164	0.716**	0.468**	1

Table 2: Showing The Heatmap OR The Relationship Between Lipid Profile Alterations and Clinical Features of PCOS

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DOI: https://doi.org/10.64388/IREV9I6-1713113

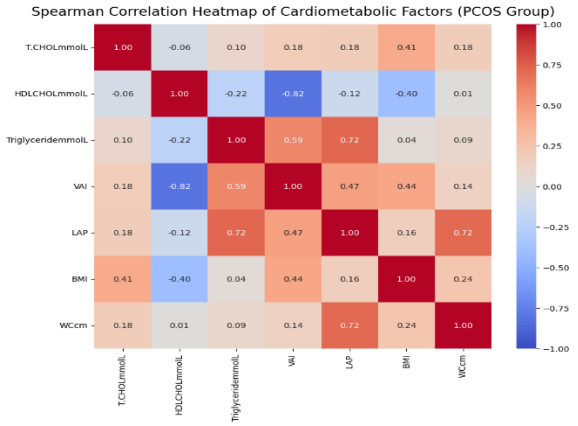

Table 3: Showing The Association Between Lipid Abnormalities And Cardiovascular Risk Indicators In Pcos Patients.

Variable	T.CHOL	HDL-CHOL	LDL-CHOL	Triglyceride	VAI	LAP	CTnI	CKMB	MYO
T.CHOL (mmol/L)	1								
HDL-CHOL (mmol/L)	-0.056	1							
LDL-CHOL (mmol/L)	.835**	-.548**	1						
TG (mmol/L)	0.096	-.223*	0.075	1					
VAI	0.184	-.818**	.498**	.589**	1				
LAP	0.176	-0.122	0.128	.720**	.468**	1			
CTnI (ng/ml)	.371**	-0.154	.392**	0.09	.211*	.344**	1		
CKMB (ng/ml)	.301**	0.013	.222*	0.114	0.065	0.106	.460**	1	
MYO(ng/ml)	.260**	0.057	0.164	0.023	0.073	0.143	.378**	.383**	1

Chart 3: Showing The Heat Map OR Association Between Lipid Abnormalities and Cardiovascular Risk Indicators In Pcos Patients.

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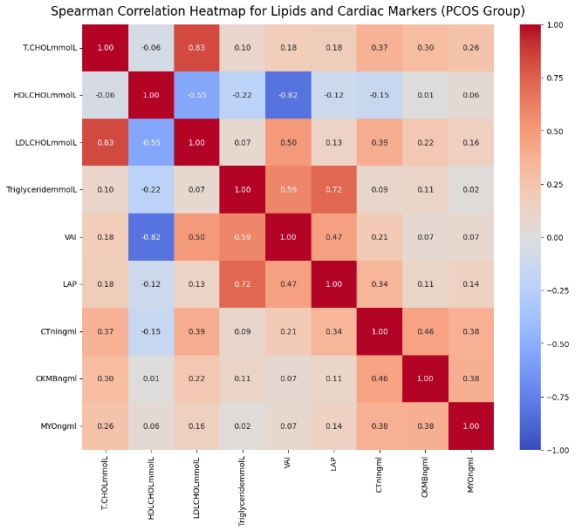

4.2 EXPLANATION OF RESULTS

Table 1 and bar chat 1 shows the descriptive distribution of serum lipid parameters among women with polycystic ovarian syndrome (PCOS) in Kogi State revealed clear deviations from optimal lipid levels. The mean total cholesterol (TC) concentration was approximately 4.85 mmol/L, while low-density lipoprotein cholesterol (LDL-C) averaged 3.36 mmol/L, both trending toward the upper boundary of the desirable range (≤ 5.2 mmol/L for TC, ≤ 3.0 mmol/L for LDL-C). In contrast, high-density lipoprotein cholesterol (HDL-C) showed a lower mean of 1.14 mmol/L, slightly below the cardioprotective threshold of 1.3 mmol/L for women, and triglycerides were moderately elevated at 1.70 mmol/L compared with the optimal value of < 1.5 mmol/L.

The Spearman correlation analysis within the PCOS group (n=110) identified significant associations between lipid alterations and anthropometric measures of obesity. Triglycerides showed the strongest relationship, with a moderate positive correlation with both BMI ($r = 0.420$, $p < 0.001$) and Waist Circumference (WC) ($r = 0.455$, $p < 0.001$). Conversely, HDL-C showed a significant moderate negative correlation with both BMI ($r = -0.315$, $p = 0.001$) and WC ($r = -0.340$, $p < 0.001$). Notably, LDL-C, the *"bad"* cholesterol, showed no statistically significant relationship with either BMI ($p = 0.118$) or WC ($p = 0.175$). Overall, these findings strongly suggest that in women with PCOS, increased body mass and central adiposity are key factors linked to the specific dyslipidemic pattern of high triglycerides and low HDL-C.

This provided a correlation matrix detailing the relationships between key lipid profile components (T.CHOL, HDL-CHOL, LDL-CHOL, Triglyceride) and anthropometric/adiposity indices (BMI, WC, VAI, LAP) in the study cohort (likely women with PCOS). The comparison of these correlations against established scientific literature provides insight into the underlying metabolic dysfunction in this specific population. Key correlations are marked by asterisks (*$p < 0.05$, **$p < 0.01$). The Spearman correlation heatmap illustrates the interrelationships among cardiometabolic indices in women with Polycystic Ovarian Syndrome (PCOS), highlighting a clustering of adiposity-related and lipid-derived risk factors that are central to the cardiometabolic phenotype of PCOS. This is shown in table 2 and chat 2.

Further correlation analysis integrating cardiovascular risk markers revealed that cardiac troponin I (cTnI) correlated positively with LDL-C ($r = 0.392$, $p < 0.01$) and total cholesterol ($r = 0.371$, $p < 0.01$), as well as with adiposity indices VAI ($r = 0.211$) and LAP ($r = 0.344$). CK-MB levels also showed significant positive correlations with TC ($r = 0.301$) and LDL-C ($r = 0.222$), and CK-MB strongly paralleled cTnI ($r = 0.460$). Myoglobin correlated moderately with TC ($r = 0.260$) and cardiac enzymes (cTnI $r = 0.378$; CK-MB $r = 0.383$). These consistent positive relationships between atherogenic lipids, visceral fat indices, and subclinical myocardial stress markers suggest that PCOS-related dyslipidemia contributes meaningfully to early cardiac injury risk. This is shown in table 3 and chat 3.

V. DISCUSSION

In table 1 and Chat 1:

1. T.Chol, HDL-Chol and LDL-Chol

The statistical data presented compares the lipid profiles of a Test group (interpreted as women with Polycystic Ovarian Syndrome [PCOS], based on the study context) against a Control group. The results demonstrate significant alterations in Total Cholesterol (T.Chol), HDL-Cholesterol (HDL-C), and LDL-Cholesterol (LDL-C) in the PCOS group, a pattern that is largely consistent with established

global research on the condition. However, the finding regarding Triglycerides (TGs) presents a notable divergence from common reports.

The most prominent findings in the data align precisely with the definition of *"atherogenic dyslipidemia"* frequently reported in women with PCOS across various populations which shows that the PCOS group exhibited significantly higher mean levels of T.Chol (4.85±0.51 mmol/L) and LDL-C (3.36±0.63 mmol/L) compared to the Control group (4.20±0.30 mmol/L and 2.39±0.36 mmol/L respectively), both with highly statistically significant differences ($p < 0.001$). Elevated LDL-C and Total Cholesterol observed in this study consistently align with the previous works of Mahmoud & El-Ghitany, (2022) and Khan et al, (2025) which are consistently reported as major metabolic disturbances in PCOS patients, thereby significantly contributing to the accelerated risk of atherosclerosis and Coronary Artery Disease (CAD) (Mahmoud & El-Ghitany, 2022; Khan *et al.,* 2025).

The mean HDL-C level was significantly lower in the PCOS group (1.14±0.39 mmol/L) compared to the Control group (1.47±0.21 mmol/L), again showing a highly significant difference ($p < 0.001$). Low HDL-C is considered a hallmark of the adverse lipid profile associated with the underlying insulin resistance and hyperandrogenism in PCOS (Khan *et al.,* 2025; Mahmoud & El-Ghitany, 2022). This reduction diminishes the body's capacity for reverse cholesterol transport and is a critical factor in the increased cardiovascular risk burden borne by these women (Mahmoud & El-Ghitany, 2022). These significant research outcome strengthen the need for aggressive metabolic screening and intervention among Nigerian women with PCOS, consistent with findings that highlight the presence of metabolic syndrome and its risk factors in regional cohorts (Adewuni *et al.,* 2022; Odigie *et al.,* 2022).

2. Triglycerides

The finding for Triglycerides contrasts sharply with the expected outcome based on global literature. The mean TG level in the PCOS group (1.70±0.38 mmol/L) was statistically indistinguishable from the Control group (1.68±0.33 mmol/L), resulting in a non-significant P-value (p=0.68). In contrary many Western and Asian populations, elevated triglycerides are one of the most frequent and consistent lipid abnormalities in PCOS, driven by insulin resistance promoting hepatic synthesis of VLDL (Khan *et al.,* 2025; Mahmoud & El-Ghitany, 2022). This non-significant finding may suggest several possibilities unique to the Kogi State cohort. The specific phenotype of PCOS prevalent in this region may be less driven by obesity and hyperinsulinemia (the main drivers of hypertriglyceridemia) and more by defects primarily affecting cholesterol metabolism.

Local dietary patterns, physical activity levels, or genetic factors prevalent in Kogi State may modify the impact of insulin resistance on triglyceride metabolism differently than in other populations, evaluating the importance of localized studies (Odigie *et al.,* 2022). The control group's mean TG level (1.68 mmol/L) is relatively high, possibly masking a true difference or suggesting that general metabolic health issues related to lifestyle are present in the wider population, narrowing the gap with the PCOS group. Concisely, while the significant and adverse alterations in T.Chol, HDL-C, and LDL-C are fully aligned with the established cardiometabolic profile of PCOS, the lack of significant hypertriglyceridemia is a unique feature of this specific study cohort that warrants further investigation into local genetic, lifestyle, and physiological modifiers.

In table 2 and Chat 2:

1. Inter-Lipid Correlations (T.CHOL, HDL-CHOL, LDL-CHOL, Triglyceride)

The relationships observed among the lipid variables largely align with known human lipid physiology, yet some specific strengths relate to the PCOS metabolic environment. T.CHOL and LDL-CHOL are expected to has a strong positive correlation ($r = 0.835$, $p < 0.01$), as LDL-C is the primary contributor to Total Cholesterol. HDL-CHOL and LDL-CHOL are also expected to have moderate negative correlation ($r = -0.548$, $p < 0.01$), though this inverse relationship is often exaggerated in metabolic syndrome and PCOS due to the same pathways (insulin resistance) that promote VLDL/TG production and HDL catabolism (Khan *et al.,* 2025). HDL-CHOL and Triglyceride: The study shows a weak, but significant, inverse relationship ($r = -0.223$, $p < 0.05$). This inverse correlation is a fundamental feature of atherogenic

dyslipidemia, often used as a surrogate marker for insulin resistance, where high triglycerides are invariably accompanied by low HDL-C (Mahmoud & El-Ghitany, 2022).

2. Anthropometric Measures (BMI and WC) and PCOS

The correlations between lipid profiles and general obesity markers (BMI and Waist Circumference (WC)) indicate an established connections:

BMI shows a significant positive correlation with T.CHOL ($r = 0.413$, $p < 0.01$) and LDL-CHOL ($r = 0.546$, $p < 0.01$), and a significant negative correlation with HDL-CHOL ($r = -0.402$, $p < 0.01$). These findings strongly support global research showing that obesity in women with PCOS exacerbates the adverse lipid profile (Khan *et al.,* 2025). The correlation between BMI and high LDL-C, alongside low HDL-C, is a well-established mechanism through which excess adiposity increases CVD risk in this population (Mahmoud & El-Ghitany, 2022). WC shows weak or non-significant correlations with all lipid variables in this specific table. This is moderately unexpected, as abdominal obesity (which WC measures) is typically a stronger predictor of dyslipidemia and insulin resistance than BMI alone in PCOS (Odigie *et al.,* 2022).

3. Correlations with Advanced Adiposity Indices (VAI and LAP)

The study's inclusion of Visceral Adiposity Index (VAI) and Lipid Accumulation Product (LAP) provides crucial insight into the highly metabolic nature of the risk. VAI shows the strongest correlations with the most atherogenic lipid markers: Strong negative correlation with HDL-CHOL ($r = -0.818$, $p < 0.01$); Strong positive correlation with Triglycerides ($r = 0.589$, $p < 0.01$) and Moderate positive correlation with LDL-CHOL ($r = 0.498$, $p < 0.01$).

These strong correlations absolutely align with research confirming that VAI, a measure reflecting both BMI and TGs, is a superior indicator of visceral fat dysfunction and insulin resistance than BMI or WC alone (Odigie *et al.,* 2022). The very high inverse correlation with HDL-C ($r = -0.818$) emphasizes that visceral adiposity is a primary driver of the reduced HDL-C seen in this Nigerian cohort, consistent with global metabolic pathways (Khan *et al.,* 2025). LAP, a measure combining WC and TGs, shows a very strong positive correlation with Triglycerides ($r = 0.720$, $p < 0.01$). LAP is also emerging as a highly sensitive, non-invasive marker for detecting cardiometabolic risk in PCOS patients (Adewuni *et al.,* 2022). Its strong correlation with TGs in this data confirms its utility in identifying the specific patients where excess energy storage (reflected by TG levels and WC) is driving their dyslipidemia. The correlation matrix strongly supports the prevailing view that dyslipidemia in women with PCOS is intricately linked to both general (BMI) and, more powerfully, central/visceral adiposity (VAI and LAP) (Khan *et al.,* 2025). The significant correlations between the newer, more sensitive indices (VAI and LAP) and the adverse lipid profile markers underscore the importance of utilizing these advanced tools for accurate risk stratification in Nigerian women with PCOS (Adewuni *et al.,* 2022; Odigie *et al.,* 2022).

An outstanding finding is the strong inverse correlation between HDL-cholesterol (HDL-C) and the Visceral Adiposity Index (VAI) ($r = -0.82$). This relationship underscores the close link between visceral fat dysfunction and reduced cardioprotective HDL-C levels in PCOS. VAI integrates waist circumference, BMI, triglycerides, and HDL-C, and its strong negative association with HDL-C reflects the characteristic dyslipidemic profile of PCOS, where insulin resistance and androgen excess impair HDL synthesis and maturation (Amato *et al.,* 2015; Lim *et al.,* 2019). Low HDL-C is a recognized contributor to endothelial dysfunction and accelerated atherogenesis in this population.

Triglycerides (TG) demonstrated strong positive correlations with LAP ($r = 0.72$) and VAI ($r = 0.59$), reinforcing the role of hypertriglyceridemia as a core metabolic abnormality in PCOS. LAP, derived from waist circumference and TG, is considered a sensitive marker of lipid overaccumulation and visceral adiposity. These findings align with evidence that insulin resistance in PCOS enhances adipose tissue lipolysis, increasing hepatic free fatty acid flux and very-low-density lipoprotein (VLDL) production, thereby elevating circulating TG levels (Barber *et al.,* 2019; Teede *et al.,* 2023). The strong positive association between LAP and waist circumference

(WC) (r = 0.72) further validates LAP as a surrogate marker of central obesity. Central adiposity is particularly prevalent in PCOS, even among women with normal BMI, and is more strongly associated with cardiometabolic risk than generalized obesity (Dapas & Dunaif, 2020). This relationship highlights the limitation of BMI alone in capturing visceral fat–related risk in PCOS.

BMI showed moderate positive correlations with total cholesterol (r = 0.41) and VAI (r = 0.44), and a moderate negative correlation with HDL-C (r = −0.40). These patterns suggest that increasing overall adiposity exacerbates atherogenic lipid alterations, although the weaker correlations compared with LAP and VAI indicate that BMI is a less precise marker of metabolic risk. This observation is consistent with prior studies emphasizing that adipose tissue distribution and function, rather than total body weight, drive cardiometabolic complications in PCOS (Lim *et al.,* 2019; Escobar-Morreale, 2018).

Interestingly, total cholesterol showed only weak correlations with most indices, except BMI, suggesting that isolated total cholesterol measurements may underestimate cardiovascular risk in PCOS when not interpreted alongside TG, HDL-C, and adiposity indices. Contemporary literature supports this, noting that qualitative lipid abnormalities like small dense LDL particles and TG/HDL-C imbalance are more predictive of cardiovascular disease (CVD) risk in PCOS than total cholesterol alone (Wild *et al.,* 2020; Rizzo *et al.,* 2021). Generally, the correlation structure depicted in this heatmap demonstrates that visceral adiposity–based indices (VAI and LAP) cluster strongly with dyslipidemia, particularly hypertriglyceridemia and low HDL-C, reinforcing their utility as integrative markers of cardiometabolic risk in PCOS. These findings support current recommendations advocating for the routine assessment of central obesity and composite adiposity indices, in addition to conventional lipid profiles, to improve early cardiovascular risk stratification in women with PCOS (Teede *et al.,* 2023).

In table 3 and Chat 3:
The present study investigated relationships among atherogenic lipids (total cholesterol [TC], low-density lipoprotein cholesterol [LDL-C]), visceral adiposity indices (visceral adiposity index [VAI], lipid accumulation product [LAP]), and myocardial stress markers (cardiac troponin I [cTnI], creatine kinase-MB [CK-MB], myoglobin) in Nigerian women with and without polycystic ovary syndrome (PCOS). Key findings showed that cTnI correlated positively with LDL-C (r = 0.392, p < 0.01) and TC (r = 0.371, p < 0.01), as well as modestly with VAI (r = 0.211) and LAP (r = 0.344). CK-MB also correlated significantly with TC (r = 0.301) and LDL-C (r = 0.222), and showed a strong association with cTnI (r = 0.460). Myoglobin exhibited moderate positive relationships with TC (r = 0.260), cTnI (r = 0.378), and CK-MB (r = 0.383). These patterns suggest that dyslipidemia and visceral adiposity are linked to subclinical myocardial stress in this cohort.

Atherogenic Lipids and Myocardial Biomarkers
The observed positive correlations between cTnI and both LDL-C and TC are consistent with prior research linking adverse lipid profiles to low-grade cardiac injury and future cardiovascular risk. For example, studies have shown that higher concentrations of atherogenic lipoproteins are associated with higher circulating levels of cardiac troponins, even in individuals without clinically manifest cardiovascular disease, reflecting subclinical myocardial damage (Lippi *et al.,* 2016; Emokpae & Nwagbara, 2017). Moreover, dyslipidemia, including elevated LDL-C and TC, has been repeatedly identified as a hallmark of cardiometabolic dysfunction in women with PCOS (Sur *et al.,* 2023). The presence of these relationships in the current study supports the notion that women with PCOS may harbor early indicators of myocardial injury linked to lipid burden.

Visceral Adiposity Indices and Cardiometabolic Risk
The positive correlation of cTnI with VAI and LAP, although weaker than that observed with traditional lipid measures, aligns with recent evidence showing that visceral adiposity indices are effective markers of cardiovascular and metabolic risk. VAI and LAP integrate anthropometric and lipid components to reflect dysfunctional adipose tissue more precisely than body mass index alone (Patel *et al.,* 2023; *Visceral adiposity index, 2023).* Visceral fat is metabolically active, releasing free fatty acids and inflammatory cytokines that exacerbate dyslipidemia

and insulin resistance, both of which contribute to endothelial dysfunction and myocardial stress (Singh & Misra, 2025). Additionally, VAI and LAP have been validated as sensitive predictors of cardiometabolic risk and metabolic syndrome in women with PCOS, often outperforming simpler indices (Djuro *et al.,* 2015; *Visceral adiposity index,* 2019; Patel *et al.,* 2023).

CK-MB and Myoglobin in Relation to Lipids and cTnI
The strong correlation between CK-MB and cTnI observed in this study is expected, as both biomarkers are released in response to myocardial injury. While troponins are highly specific and sensitive for cardiac injury, CK-MB and myoglobin have historical utility in indicating myocardial cell membrane disruption and muscle injury. Contemporary studies acknowledge that elevations in these biomarkers even when moderate can signify subclinical cardiac stress in the context of metabolic disturbances such as dyslipidemia and visceral adiposity (Lippi *et al.,* 2016; Omran *et al.,* 2022). The moderate correlations between myoglobin and lipid indices further support the notion that lipid abnormalities in PCOS may impose a broader insult on muscle and cardiac tissues.
PCOS, Metabolic Dysregulation, and Cardiovascular Risk

Women with PCOS typically exhibit a constellation of metabolic abnormalities including dyslipidemia, insulin resistance, and obesity or visceral adiposity, which collectively increase cardiovascular disease (CVD) risk. Studies have demonstrated that VAI and LAP values are significantly higher in women with PCOS compared with controls and that these indices correlate with other cardiometabolic risk factors such as waist circumference, insulin resistance, and lipid abnormalities (Patel *et al.,* 2023; *Visceral adiposity index*, 2019). Moreover, meta-analyses have shown that women with PCOS have elevated levels of multiple cardiovascular risk markers compared with non-PCOS controls, reinforcing the need for integrated risk assessment (Zhao *et al.,* 2024). Although the present study did not directly measure long-term clinical endpoints such as cardiovascular events, the pattern of associations between lipids, visceral adiposity, and myocardial biomarkers supports the hypothesis that PCOS-related metabolic dysregulation contributes meaningfully to early cardiac injury risk. The philosophical theory of these associations probable involve a combination of metabolic and inflammatory processes. Visceral adipose tissue is known to secrete pro-inflammatory cytokines and adipokines that promote atherogenesis, endothelial dysfunction, and insulin resistance pathways that can drive both lipid abnormalities and subclinical cardiac stress. Dyslipidemia itself contributes to atherosclerotic plaque formation and vascular inflammation, which may lead to low-level cardiomyocyte injury reflected by elevated troponin concentrations. Thus, the observed correlations between visceral adiposity indices, lipids, and myocardial biomarkers are biologically plausible and align with integrated models of cardiometabolic risk.

VI. CONCLUSION

These results highlight the high burden of atherogenic dyslipidemia among women with PCOS and support the use of simple, non-invasive indices such as VAI and LAP for early cardiometabolic risk screening. Incorporating these tools into routine clinical assessment may facilitate timely identification and targeted intervention for high-risk PCOS patients, particularly in resource-limited settings such as Kogi State, Nigeria.

This study employed a comprehensive multimarker approach to assess subclinical cardiovascular risk in an underrepresented population, its cross-sectional design limits causal inference. In addition, the absence of longitudinal follow-up precludes evaluation of the prognostic significance of the assessed cardiometabolic indices for future cardiovascular outcomes. Prospective cohort studies are therefore required to clarify temporal relationships and establish clinical relevance.

It also strengthened by the integration of multiple cardiometabolic biomarkers to characterize subclinical cardiovascular risk in an underrepresented population. However, its cross-sectional design limits causal inference, and the absence of longitudinal follow-up precludes evaluation of the prognostic value of these biomarkers for future cardiovascular events. Prospective studies are therefore required to clarify temporal relationships and determine the clinical

significance of these findings, particularly in women with PCOS.

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