Comparative Study of Myocardial Biomarkers Among Women with and Without Polycystic Ovarian Syndrome (PCOS) in Nigeria

***[1]Ohikere, O. P., [2]Mokwenye, N. V., [3]Emokpae A. M., [4]Ige, I. P., (ORCID) [5]Adewole, A. A. (ORCID)**

[1,2,]Department of Medical Laboratory Science, Igbinedion University, Okada, Edo State, Nigeria.

[3]Department of Medical Laboratory Science, University of Benin, Benin City, Edo State, Nigeria.

[4]Department of Medical Laboratory Science, School of Basic Medical Sciences, Federal University of Technology, Akure, Ondo State, Nigeria.

[5]Obstretics and Gynaecology department, Federal Teaching Hospital, Lokoja, Kogi State, Nigeria.

***Corresponding Author:**

DOI: https://dx.doi.org/10.51244/IJRSI.2025.12110097

Received: 25 November 2025; Accepted: 01 December 2025; Published: 10 December 2025

ABSTRACT

Polycystic Ovarian Syndrome (PCOS) is a common endocrinopathy associated with chronic low-grade inflammation, insulin resistance, and dyslipidemia, all of which elevate the long-term risk of cardiovascular disease (CVD). While subclinical myocardial injury is recognized in Western populations with PCOS, data from Nigerian women, who may face distinct genetic and environmental risk factor profiles, remain scarce. This study aims to compare the circulating levels of specific myocardial injury biomarkers between Nigerian women with and without PCOS. This was a case-control study conducted at selected healthcare centers and fertility clinics in Lokoja, Okene, and Anyigba, Kogi State, Nigeria, where PCOS cases are commonly reported. This study comprises of 150 samples, with 110 women diagnosed with PCOS based on the Rotterdam criteria (PCOS Group) were recruited and compared to 40 age- and BMI-matched healthy women (Control Group). Fasting blood samples were analyzed for quantitative measurement of three cardiac biomarkers: Myoglobin (MYO), Cardiac Troponin I (cTnI), and Creatine Kinase-MB (CK-MB). Anthropometric data and standard lipid profiles were also collected. Data were analyzed using independent samples t-tests and multivariate analysis of variance (MANOVA), with significance set at $p < 0.05$. The multivariate test using Pillai's Trace indicated a significant overall group effect, $V = 0.202$, $F(3, 146) = 12.32$, $p < 0.001$, partial $\eta^2 = 0.202$, suggesting that the combined biomarker profile differed between groups. Follow-up univariate ANOVAs revealed that women with PCOS had significantly higher mean serum levels of cTnI ($F(1,148) = 5.91$, $p = .016$, partial $\eta^2 = 0.038$), CK-MB ($F(1,148) = 20.43$, $p < 0.001$, partial $\eta^2 = .121$), and MYO ($F(1,148) = 21.65$, $p < .001$, partial $\eta^2 = 0.128$) compared to controls. The estimated marginal means confirmed consistently elevated concentrations of all three biomarkers in the PCOS group (MYO: 57.88 ± 3.51 ng/ml; CK-MB: 1.87 ± 0.08 ng/ml; cTnI: 0.074 ± 0.005 ng/ml) relative to the control group (MYO: 26.28 ± 5.82 ng/ml; CK-MB: 1.21 ± 0.13 ng/ml; cTnI: 0.053 ± 0.008 ng/ml). Collectively, these findings indicate that PCOS is associated with significantly elevated serum concentrations of myocardial biomarkers, suggesting the presence of early or subclinical myocardial stress in affected women, independent of age matching. Furthermore, regression analysis suggested that significant factor, like MYO was an independent predictor of the outcome variable. Nigerian women with PCOS exhibit significantly elevated circulating levels of myocardial biomarkers (MYO, cTnI, and CK-MB), suggesting the presence of chronic, subclinical myocardial injury in this population. These findings underscore the need for early and aggressive cardiovascular risk assessment and management strategies for women with PCOS in the Nigerian clinical setting.

Keywords: Myocardial, PCOS, Hyperinsulinemia, Endocrine, Metabolic, BMI

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XII Issue XI November 2025

INTRODUCTION

Background of the Study

Polycystic ovary syndrome (PCOS) is a common endocrine–metabolic disorder of reproductive-age women characterized by hyperandrogenism, ovulatory dysfunction and polycystic ovarian morphology. Beyond reproductive consequences, PCOS is associated with adverse cardiometabolic features like insulin resistance, dyslipidaemia, obesity and hypertension which increase long-term cardiovascular disease (CVD) risk. Recent reviews and research studies have therefore indicated that PCOS as a "risk-enhancing" condition for future cardiovascular events (1). PCOS is a heterogeneous endocrine disorder of reproductive-age women diagnosed using accepted criteria like Rotterdam criteria: presence of two of three: oligo/anovulation, clinical/biochemical hyperandrogenism, polycystic ovarian morphology after exclusion of secondary causes (1).

Myocardial biomarkers for example high-sensitivity cardiac troponins (cTnI), creatine kinase-MB (CK-MB), myoglobin (MYO) and natriuretic peptides are widely used to detect myocardial injury and to stratify cardiovascular risk in clinical practice. Sex differences in baseline cardiac troponin concentrations and the prognostic value of low-level troponin in women have been described, to assess the need for sex-specific consideration when interpreting biomarker data. In parallel, biomarker panels and proteomic approaches have been used recently to identify cardiovascular-risk proteins that may be dysregulated in PCOS and could provide early signals of cardiac risk before overt disease develops (2). Myocardial biomarkers are blood analytes used to detect myocardial injury or strain. In this study the term refers primarily to high-sensitivity cardiac troponin (hs-cTnI or hs-cTnT), creatine kinase-MB (CK-MB) isoenzyme, andmyoglobin (MYO). These markers differ in kinetics and specificity: troponins are most specific for cardiomyocyte injury, CK-MB rises in acute myocyte necrosis but is less specific, and natriuretic peptides reflect myocardial wall stress (3).

Insulin resistance (HOMA-IR) is a homeostatic Model Assessment of Insulin Resistance, calculated from fasting glucose and insulin, used here as a continuous variable to quantify insulin resistance. Elevated HOMA-IR is common in PCOS and is a mediator of cardiometabolic risk (4). Cardiometabolic risk factors are traditional risk variables for CVD including obesity (BMI and waist circumference), dyslipidaemia (elevated LDL-C, triglycerides; low HDL-C), hypertension, impaired fasting glucose, and insulin resistance.

The international and regional evidence linking PCOS to elevated cardiometabolic risk and to disorder in cardiovascular biomarkers, but there is a scarcity of direct comparisons of myocardial biomarker levels in Nigerian women with and without PCOS. This study will measure and compare established myocardial biomarkers in these groups, examine their associations with metabolic risk factors, and thereby provide population-specific data to inform screening and preventive care (4).

Although metabolic and anthropometric determinants of CVD risk in PCOS have been studied in many populations, data from sub-Saharan Africa, and Nigeria in particular remain relatively limited. A small but growing number of Nigerian studies report elevated cardiometabolic risk among women with PCOS, suggesting that PCOS may independently increase cardiovascular risk in this setting. However, few published investigations have directly compared myocardial biomarker concentrations between Nigerian women with and without PCOS, or examined how these biomarkers relate to metabolic variables such as BMI, insulin resistance, lipids in that population. This gap is important because population-specific baseline biomarker distributions and risk profiles influence the interpretation and clinical utility of myocardial biomarkers (5).

Taken together, the literature motivates a focused comparative study of myocardial biomarkers among Nigerian women with and without PCOS to (1) determine whether subclinical myocardial injury or higher biomarker concentrations are present in PCOS, (2) explore relationships between biomarkers and cardiometabolic risk factors in this population, and (3) provide locally relevant data that could inform screening and preventive strategies. The present study is therefore placeed to address these gaps by measuring established cardiac biomarkers: high-sensitivity cardiac troponin, CK-MB, and myoglobin and associating them to clinical and laboratory measures of cardiometabolic risk.

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XII Issue XI November 2025

The aim and objectives of this studyare to evaluates serum levels of myoglobin, CK-MB, and cTnI in Nigerian women with Polycystic Ovarian Syndrome (PCOS) versus age-matched controls to assess cardiovascular risk. It also investigates correlations between these biomarkers and cardiometabolic indices, insulin resistance, and anthropometric parameters. Furthermore, the research analyzes the combined expression of these markers to detect subclinical myocardial stress. Ultimately, the study aims to establish the clinical value of these biomarkers as early predictors of cardiovascular disease risk within the Nigerian PCOS population. The objectives of this study are to: compare concentrations of myocardial biomarkers (high-sensitivity cardiac troponin, CK-MB, and myoglobin (MYO) between reproductive-age Nigerian women diagnosed with PCOS and age-matched women without PCOS; assess the association between myocardial biomarker levels and cardiometabolic risk factors (BMI, waist circumference, fasting glucose/insulin, HOMA-IR, lipid profile, and blood pressure) within the PCOS group and within controls; determine whether PCOS status predicts elevated myocardial biomarkers after adjustment for conventional cardiometabolic risk factors; and to estimate the prevalence of clinically meaningful elevations in myocardial biomarkers among women with PCOS in the study sample.

This study is so important since PCOS is associated with excess cardiometabolic risk factors that predispose to ischemic heart disease and heart failure later in life. Early identification of subclinical myocardial injury or biomarker changes could permit targeted risk-reduction in a high-risk group (1). Most biomarker research in PCOS has been performed in high-income countries; Nigeria and other sub-Saharan African populations are under-represented despite potentially different obesity patterns, genetic backgrounds, and access to care that can modify risk and biomarker distributions. Locally generated data are therefore essential for guideline-relevant interpretation (5). If PCOS is associated with higher myocardial biomarkers independent of traditional risk factors, this would strengthen recommendations for routine cardiometabolic screening in Nigerian women with PCOS and could inform public-health strategies. Biomarker findings may also prompt longitudinal follow-up studies to clarify prognostic implications (6).

Some questions to be addressed in this research work include: do Nigerian women with PCOS have higher concentrations of high-sensitivity cardiac troponin, CK-MB, or myoglobin (MYO) compared with age-matched women without PCOS? What are the relationships between myocardial biomarker levels and cardiometabolic risk factors (BMI, waist circumference, insulin resistance, lipids, blood pressure) in women with PCOS and in controls? Does PCOS status independently predict elevated myocardial biomarker concentrations after controlling for conventional risk factors?

LITERATURE REVIEW

Polycystic Ovarian Syndrome (PCOS) is the most common endocrinopathy affecting women of reproductive age globally, characterized by hyperandrogenism, menstrual dysfunction, and polycystic ovarian morphology (7). Importantly, PCOS is essentially connected to a high prevalence of Insulin Resistance (IR), a condition where cells do not respond effectively to insulin, leading to compensatory hyperinsulinemia (8). PCOS is the most widespread endocrine metabolic disorder affecting 5-10% of women of reproductive age worldwide. (9). PCOS is related to complication existing across lifespan starting from reproductive and dermatological concerns to metabolic and psychological complication such as diabetes, metabolic syndrome, cardiovascular disorder etc, (10). PCOS can lead to cardiometabolic risk, meaning that women with PCOS frequently have insulin resistance, dyslipidemia, and hypertension; classical risk factors for atherosclerotic cardiovascular disease (ASCVD). Several systematic reviews and cohort studies have reported higher prevalence of cardiometabolic risk markers and some evidence for elevated cerebrovascular/cardiac event risk, though the magnitude and independence of risk vary across studies and phenotypes. (11)

Recent population work in Nigeria estimates that PCOS affects roughly 8–9% of reproductive-aged women, although prevalence depends on diagnostic criteria and sampling frame, and underdiagnosis remains a persistent problem (12). Women with PCOS show a higher prevalence of established cardiovascular risk factors like insulin resistance, central obesity, dyslipidaemia, hypertension, and chronic low-grade inflammation which together accelerate atherosclerotic processes and increase lifetime CVD risk (13; 14).

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XII Issue XI November 2025

Pathophysiology

Polycystic ovary syndrome (PCOS) is multifactorial with interacting endocrine, metabolic, genetic, and environmental contributors such as Hyperandrogenism (ovarian and/or adrenal), Insulin resistance and compensatory hyperinsulinemia which is dependent on body mass index (BMI), Ovarian dysfunction, Chronic low-grade inflammation and oxidative stress, Genetic and developmental components, and Adiposity and ectopic fat. Specifically, PCOS emerges from a confluence of hyperandrogenism, intrinsic or acquired insulin resistance, ovarian dysfunction, and environmental modifiers, creating variable clinical phenotypes (15; 16).

Epidemiology

Prevalence estimates vary by population and diagnostic criteria but generally range from about 6% to 20% of women of reproductive age depending on the criteria used and population studied. PCOS often first becomes apparent in adolescence and is a major cause of anovulatory infertility worldwide (16). Polycystic ovary syndrome (PCOS) is the most common endocrine disorder among women and so peculiar between the ages of 18 and 44 (17). According to the World Health Organization (WHO), PCOS affects over 6 to 13% of reproductive-aged women (18). Polycystic ovary syndrome (PCOS) is a common condition affecting approximately 8% of women. Once someone is infertile due to lack of ovulation, PCOS is the most common cause and could be a pointer to patients' diagnosis. The 2022 approximations of prevalence of PCOS vary which could arises in between 5% and 18% of women (19).

The occurrence of PCOS depends on the choice of diagnostic criteria. Using the Rotterdam criteria, around 10–13% of women have PCOS (17). Based on the NIH standards, by using the Androgen Excess Society criteria, the global frequency was 5.5%, growing to approximately 7.1%. Irrespective of the criteria, the prevalence of PCOS is increasing, likely due to an aging population, more awareness, and increasing obesity rates (20).

Incidence appears impartially even among people with different ethnicities, but is conceivably higher in people from South East Asia and the Eastern Mediterranean (17). PCOS may express differently however. For instance, in African and Hispanic American people with PCOS, there is more insulin resistance compared to other ethnic groups (21). The same is true for South Asian people with PCOS, who also have more metabolic symptoms and higher BMIs. East Asian women typically have less hirsutism and lower BMI compared to other groups (19).

Ultrasonographic findings of polycystic ovaries are found in 8–25% of women who are not affected by the syndrome. 14% women on oral contraceptives are found to have polycystic ovaries. Ovarian cysts are also a common side effect of levonorgestrel-releasing intrauterine devices (IUDs).

Hormonal And Metabolic Dysregulation of PCOS

PCOS marker include hormonal imbalance, particularly hyperandrogenism, resulting from disruption of the hypothalamic–pituitary–ovarian (HPO) axis. Increased GnRH pulse frequency elevates luteinizing hormone (LH) while follicle-stimulating hormone (FSH) remains normal or slightly reduced. Excess LH stimulation of ovarian theca cells leads to elevated androgen production (22). This hyperandrogenic environment impairs follicular development, leading to polycystic ovarian morphology (PCOM), anovulation, and menstrual irregularities. In addition, granulosa cells from these follicles produce elevated AMH, which further inhibits aromatase activity, reducing estrogen production and perpetuating follicular arrest (23; 24). Metabolic dysfunction is another central component. Insulin resistanceexisting in both lean and obese phenotypesdrives compensatory hyperinsulinemia. Elevated insulin suppresses hepatic SHBG production, thereby increasing bioavailable androgens. Chronic low-grade inflammation further exacerbates insulin resistance, forming a vicious cycle between reproductive and metabolic disturbances (25; 26).

Polycystic Ovarian Syndrome (PCOS) and Myocardial Biomarkers

Clinical significance of the presence of elevated myocardial biomarkers in PCOS women provides an essential instrument for early risk classification. These markers serve as measurable evidence that the metabolic and hormonal dysfunction of PCOS is translating into direct, measurable cardiac consequences, necessitating

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XII Issue XI November 2025

aggressive management of IR, weight, and dyslipidemia to mitigate long-term CVD risk (7). The relationship between PCOS and myocardial biomarkers centers on the heightened metabolic and inflammatory stress induced by the syndrome, leading to subclinical myocardial injury detectable by these markers. PCOS is recognized not just as a reproductive disorder, but as a major cardiovascular risk factor (7).

The Pathophysiological Linkage

The chronic state associated with PCOS drives the release of cardiac biomarkers through several mechanisms, which has demonstrated a significant correlations with several published studies. This include:

Insulin Resistance (IR) and Inflammation: PCOS is strongly associated with IR and chronic low-grade inflammation. Adipose tissue, particularly visceral fat, releases pro-inflammatory cytokines like interleukine 6 (IL-6), and tissue necrosis factor (TNF) that cause systemic endothelial dysfunction and directly damage the myocardium (7).

Oxidative Stress: Increased oxidative stress, stemming from dysregulated metabolism, contributes to cellular damage in the heart muscle (27).

Myocardial Stress: The hypertension and dyslipidemia often accompanying PCOS increase the mechanical and hemodynamic load on the heart, leading to myocardial stress and potential hypertrophy (28).

These chronic stresses, even in the absence of acute coronary events, result in the measurable leakage of specific cardiac proteins into the circulation.

Cardiac Troponin I and T (cTnI and cTnT):

Cardiac Troponin I and T (cTnI and cTnT) are the most specific markers of myocardial necrosis (29). In women with PCOS, even high-sensitivity, chronically elevated levels of these troponins have been observed compared to healthy controls (27). This chronic low-level elevation is considered a powerful indicator of subclinical myocardial injury and stress, independently predicting future adverse cardiac events like heart failure and myocardial infarction, even in asymptomatic individuals (28).

Polycystic Ovarian Syndrome (PCOS) and Cardiac Troponin I (cTnI)

The relationship between PCOS and Cardiac Troponin I (cTnI) is fundamental and points to the syndrome's ability to induce subclinical myocardial injury due to chronic metabolic and inflammatory stress (30).

Cardiac Troponin I (cTnI) as a Marker of Subclinical Injury

Cardiac Troponin I (cTnI) is the gold standard biomarker for detecting myocardial necrosis (29). While high levels are diagnostic of acute myocardial infarction, researchers now utilize high-sensitivity assays to detect chronically low, yet elevated, levels of cTnI. These micro-elevations are interpreted as evidence of persistent, low-grade myocardial damage or stress (28). Studies have demonstrated that women with PCOS, even those who are asymptomatic or apparently healthy, often exhibit higher concentrations of high-sensitivity cTnI compared to age- and BMI-matched controls (27). This association is driven by the core pathophysiology of PCOS:

Insulin Resistance and Chronic Inflammation: PCOS is intrinsically linked to insulin resistance IR), leading to a state of chronic, low-grade inflammation (7). The resultant release of pro-inflammatory cytokines, along with metabolic stress, damages the endothelium and directly compromises the integrity of the cardiomyocyte membranes, leading to the measurable leakage of cTnI (28). This damage represents ongoing myocardial injury.

Increased Cardiovascular Load: The common comorbidities of PCOS, such as hypertension and dyslipidemia, contribute to increased mechanical stress on the heart, leading to subtle myocardial strain and cell turnover, which further elevates cTnI (27).

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XII Issue XI November 2025

Clinical Significance

The chronic elevation of cTnI in women with PCOS holds profound clinical significance, such as prognostic value: Chronically elevated cTnI is not just a marker of damage; it is an independent predictor of future cardiovascular morbidity and mortality (28). Risk Classification in the activity of serum cTnI levels can be used as a valuable instrument for early risk condition in women with PCOS, identifying those who require more aggressive metabolic and cardiovascular risk factor management to prevent progression to overt CVD (7).

Polycystic Ovarian Syndrome (PCOS) And Creatine Kinase-MB (CK-MB)

Serum CK-MB is an isoenzyme of creatine kinase that is found in high concentrations in the myocardium. Historically, it has been used as a biomarker for the diagnosis of acute myocardial infarction. However, its specificity is limited as small amounts are also present in skeletal muscle, and elevations can be seen with strenuous exercise or skeletal muscle injury. More sensitive and specific markers, such as troponins, are now the preferred biomarkers for diagnosing acute cardiac events.Recent research indicates that women with Polycystic Ovarian Syndrome (PCOS) may have elevated levels of Creatine Kinase-MB (CK-MB). Since CK-MB is a well-known biomarker for heart muscle stress, this finding suggests it could be a useful indicator of the subclinical cardiovascular risk inherent to the syndrome. CK-MB is not a diagnostic tool for PCOS, its elevation in this population is clinically significant. It serves as a potential, cost-effective biomarker to help identify women with PCOS who may be at a higher risk for future cardiovascular events. This finding reinforces the need for proactive cardiovascular risk assessment and management in all women diagnosed with PCOS. While less specific than troponin, CK-MB levels can also be elevated in PCOS populations due to overall muscle turnover and cellular stress, correlating with the severity of the metabolic syndrome components (36).

In recent years, the prevalence of polycystic ovary syndrome (PCOS) has gradually increased, and the investigation of the causal factors influencing etiopathogenesis is attracting attention. Several studies have highlighted that patients with PCOS exhibit an increased risk of cardiovascular disease (CVD) compared with healthy people, and these risks include the occurrence of myocardial infarction, ischemic heart disease, and stroke. This correlation becomes particularly important when PCOS is diagnosed and consequently a specific treatment is recommended. Of note, women with PCOS may exhibit different pathological features even if quite often they are considered as a sole unique group of patients (31).

A direct and extensive relationship between PCOS and serum CK-MB has not been fully established. Research into the broader category of total creatine kinase suggests that any elevations are more likely linked to the metabolic syndrome components often present in women with PCOS, rather than the syndrome itself. The well-documented increased cardiovascular risk in women with PCOS underscores the importance of monitoring for heart health in this population. Future research may explore more sensitive and specific cardiac biomarkers to better understand and predict cardiovascular risk in women with PCOS, though at present, serum CK-MB does not appear to be a significant marker in the clinical management or risk stratification of these patients. Rhabdomyolysis, intense exercise, and trauma result in transient elevation of CK and CK-MB; CK-MB is present in skeletal muscles as well, albeit in lesser concentrations. Chronic skeletal muscle disorders such as autoimmune myopathies and inflammatory myopathy can result in persistently high CK-MB levels in the plasma due to ongoing injury and repair. Damage to the myocardium releases CK-MB, and since the myocardium contains the largest percentage of CK-MB, patients with rapidly rising and falling CK-MB exceeding the reference range of normal should be considered as having acute myocardial infarction until proven otherwise (32).

CK-MB as an Indicator Of Subclinical Myocardial Stress

Creatine Kinase-MB (CK-MB) is an enzyme found almost exclusively in the heart muscle (myocardium). It's released into the bloodstream when the heart muscle is damaged, making it a classic biomarker for diagnosing a heart attack. However, in the context of PCOS, researchers are investigating its role not as a marker of an acute event, but as an indicator of chronic, low-level cardiac stress.Various studies have found that women with PCOS have significantly higher baseline levels of CK-MB compared to healthy women without the condition, even in the absence of any overt heart disease (33). This suggests that the adverse metabolic environment created by

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XII Issue XI November 2025

PCOS puts a continuous strain on the heart muscle, causing a minor but persistent release of this enzyme into the circulation (34).

Mechanisms Linking PCOS To Elevated CK-MB

The elevation of CK-MB in PCOS is not an isolated finding; it is believed to be a direct consequence of the syndrome's midpoint metabolic and hormonal disturbances.

Insulin Resistance

Insulin resistance, a central feature of PCOS, is strongly linked to cardiovascular dysfunction. The heart muscle relies on efficient glucose uptake for energy, a process that is impaired by insulin resistance. This metabolic inefficiency can lead to cellular stress, oxidative damage, and minor injury to cardiac cells over time. Studies have demonstrated a significant positive correlation between CK-MB levels and the degree of insulin resistance measured by HOMA-IR, suggesting that worsening insulin resistance directly contributes to this subclinical cardiac stress (33).

Hyperandrogenism And Inflammation

The high levels of androgens like testosterone and the chronic low-grade inflammation characteristic of PCOS are also implicated. Both hyperandrogenism and inflammation can have direct negative effects on the cardiovascular system, including promoting endothelial dysfunction and atherosclerosis. This hostile environment can place a higher workload on the heart, contributing to the myocardial stress reflected by rising CK-MB levels (35).

Myoglobin And PCOS

Myoglobin (MYO) is an early marker of muscle cell injury and is highly correlated with general metabolic stress and high body mass index (BMI), a common comorbidity in PCOS (36). Research in populations with high metabolic syndrome prevalence (like some Nigerian cohorts) often finds MYO to be a sensitive predictor of heightened cardiovascular risk (37; 38). Its elevation reflects the broader systemic muscular and metabolic pathology inherent in PCOS.

Elevated myoglobin in PCOS is not a diagnostic reference point but is considered a potential biomarker reflecting the systemic impact of insulin resistance and inflammation on muscle tissues. It strengthens the understanding of PCOS as a metabolic syndrome with significant cardiovascular implications and may help in identifying women who require more aggressive management of their cardiometabolic risk factors. The relationship between Polycystic Ovarian Syndrome (PCOS) and myoglobin is an emerging area of research focused on understanding the subclinical cardiovascular and muscular stress associated with the syndrome. Current evidence suggests that women with PCOS often have elevated circulating levels of myoglobin, which may serve as an early biomarker for cardiometabolic risk.

Myoglobin As A Marker Of Subclinical Tissue Stress

Myoglobin is a protein found in heart and skeletal muscle tissue that is released into the bloodstream following muscle injury. While it is famously used as a marker for acute events like a heart attack, recent studies have investigated its role as a marker of chronic, low-level tissue stress in systemic conditions like PCOS.Several studies have consistently found that serum myoglobin levels are significantly higher in women with PCOS compared to healthy, age-matched controls. This finding suggests that the underlying metabolic disturbances of PCOS may be exerting a constant, subclinical strain on muscle tissues (both cardiac and skeletal), leading to a persistent **"leak"** of myoglobin into circulation (39; 40).

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XII Issue XI November 2025

Potential Mechanisms For Elevated Myoglobin In PCOS

The exact reasons for this elevation are believed to be multifactorial and are directly associated with the centre pathophysiology of PCOS.

Insulin Resistance and Muscle Metabolism

A primary feature of PCOS is insulin resistance, which particularly affects skeletal muscle, the body's main site for glucose uptake. In a state of insulin resistance, muscle cells are under significant metabolic stress. This impaired cellular metabolism and energy utilization can lead to minor cellular damage or increased cell membrane permeability, resulting in the release of myoglobin into the bloodstream. In fact, studies have demonstrated a direct positive correlation between myoglobin levels and the degree of insulin resistance, as measured by the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) index (39).

Chronic Low-Grade Inflammation

This is a common finding in women with PCOS and is a known contributor to the development of atherosclerosis. PCOS is characterized by a state of chronic, low-grade inflammation. Inflammatory cytokines can induce oxidative stress and contribute to tissue damage throughout the body. This systemic inflammation can affect both the vascular endothelium and muscle cells, promoting an environment that leads to myoglobin release. The correlation between elevated myoglobin and inflammatory markers like C-reactive protein (CRP) in women with PCOS supports this connection (41).

PCOS has also been associated with chronic low-grade inflammation, characterized by increased white blood cell count, high levels of C-reactive protein (CRP), interleukin 6 (IL-6), interleukin 18 (IL-18), monocyte chemoattractant protein-1, and macrophage inflammatory protein-1. Insulin resistance is related to inflammation. For example, an exaggerated production of tumor necrosis factor alpha (TNF-α) produced by monocytes as a response to hyperglycemia could exacerbate the metabolic and hormonal abnormalities of PCOS. Recently, advanced glycosylation end products (AGEs) and their receptors implicated in the inflammation and oxidative stress cascades have also been found to be over expressed in PCOS women. The release of inflammatory markers is associated with long-term metabolic complications and high cardiovascular risk (42).

Early Indicator of Cardiovascular Risk

Because myoglobin is abundant in heart muscle, its chronic elevation in women with PCOS is a significant concern for cardiovascular health. It may represent an early warning sign of subclinical myocardial stress or injury long before any clinical symptoms of heart disease appear. The metabolic abnormalities in PCOS, such as dyslipidemia and hypertension, place a continuous burden on the heart, and elevated myoglobin may be one of the earliest measurable signs of this cardiac strain (39).

Clinical Presentation

Clinical presentation of PCOS include Reproductive features like Oligomenorrhea or amenorrhea, infertility due to anovulation; Hyperandrogenic signs: Hirsutism, acne, androgenic alopecia; Metabolic: Overweight/obesity (common but not universal), insulin resistance, dyslipidemia, and increased risk of impaired glucose tolerance and type 2 diabetes; and Psychological comorbidity: Elevated rates of anxiety, depression, body image distress and reduced quality of life (17).

Investigations and assessment

Assessment and Investigations of PCOS is multidimensional, which could involve reproductive and hormonal tests by measuring the androgens (total or free testosterone) (17); metabolic evaluation by measuring fasting blood glucose and/or HbA1c and an assessment for insulin resistance (HOMA-IR or OGTT when indicated), particularly if overweight/obese or family history of diabetes. The 2023 guideline emphasizes assessing cardiometabolic risk rather than routine insulin measurement in all patients (17); measuring the lipid profile to

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XII Issue XI November 2025

detect atherogenic dyslipidemia; imaging on Pelvic transvaginal ultrasound (16); Psychological screening for anxiety, depression and eating disorders (17).

METHODOLOGY

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.

Study Area

The research was carried out in Lokoja, 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 (Fig 1). 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.

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.

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.

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.

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XII Issue XI November 2025

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 18–40 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.

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).

Exclusion and Inclusion Criteria:

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.

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.

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.

Presentation Of Data

Demographic and Clinical Characteristics

Table 4.1: Sociodemographic

Variables	Groups	Case n (%)	Control n (%)
Age	20 - 30	18 (16.36)	4 (10.00)

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XII Issue XI November 2025

	31 - 40	41 (37.27)	10 (25.00)
	41 - 50	51 (46.36)	26 (65.00)
	Total	**110 (100)**	**40 (100)**

Graph 4.1 Represent the Sociodemographic Table

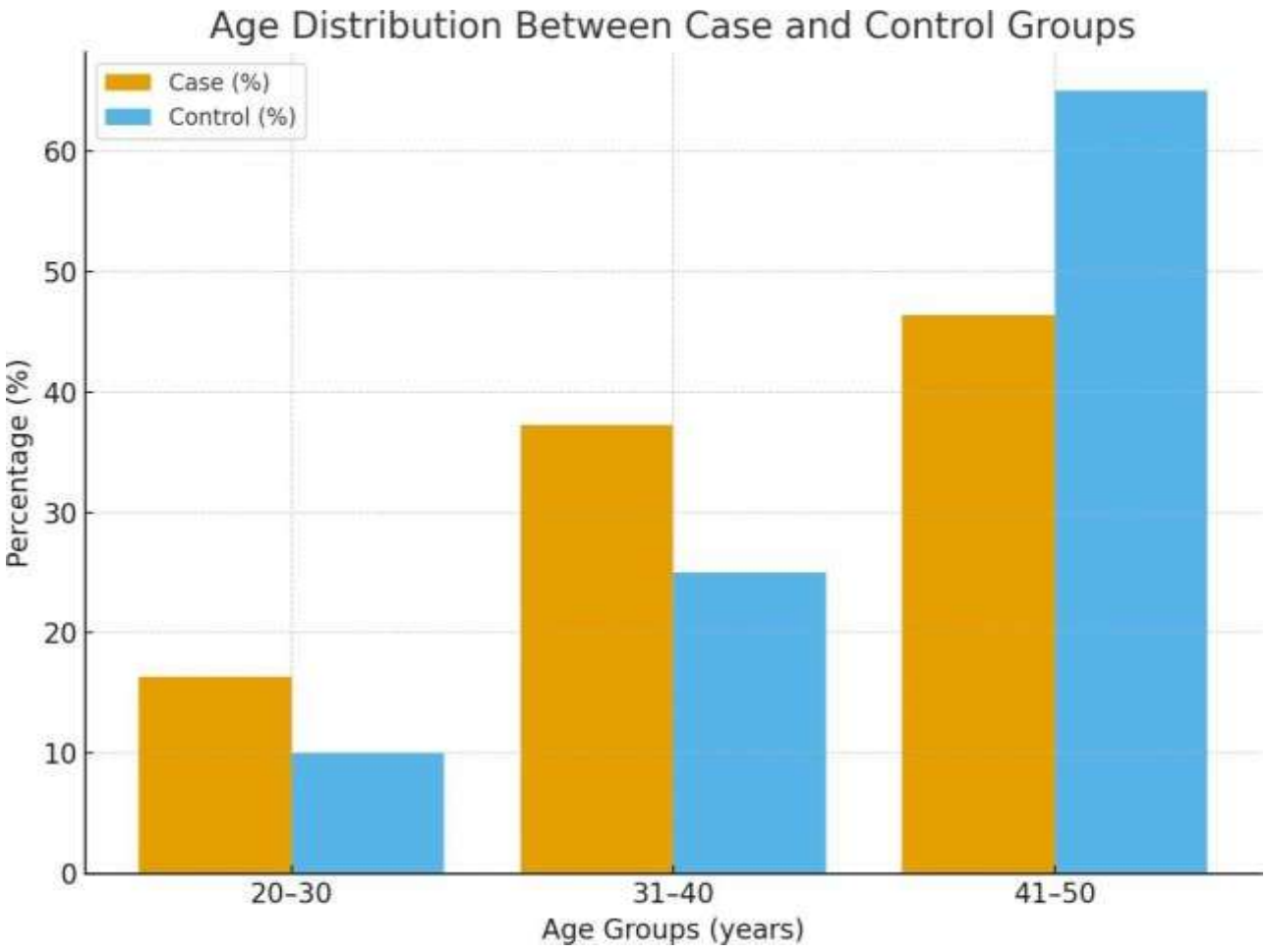


Table 4.2 Showing The Comparison Of Serum Concentrations Of Myocardial Biomarkers In Women Diagnosed With Pcos And Age-Matched Healthy Controls.

Effect / Dependent Variable	**Test Statistic**	**F-Value**	**P-Value**	**Partial η²**
Multivariate Test (Group Effect)	Pillai's Trace = 0.202	12.3	< 0.001	0.202
Univariate Tests				
cTnI (ng/ml)	—	5.91	0.016	0.038
CK-MB (ng/ml)	—	20.4	< 0.001	0.121
MYO (ng/ml)	—	21.7	< 0.001	0.128

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XII Issue XI November 2025

Graph 4.2: Bar Chat Showing The Association Between Serum Concentrations Of Myocardial Biomarkers And The Control Group

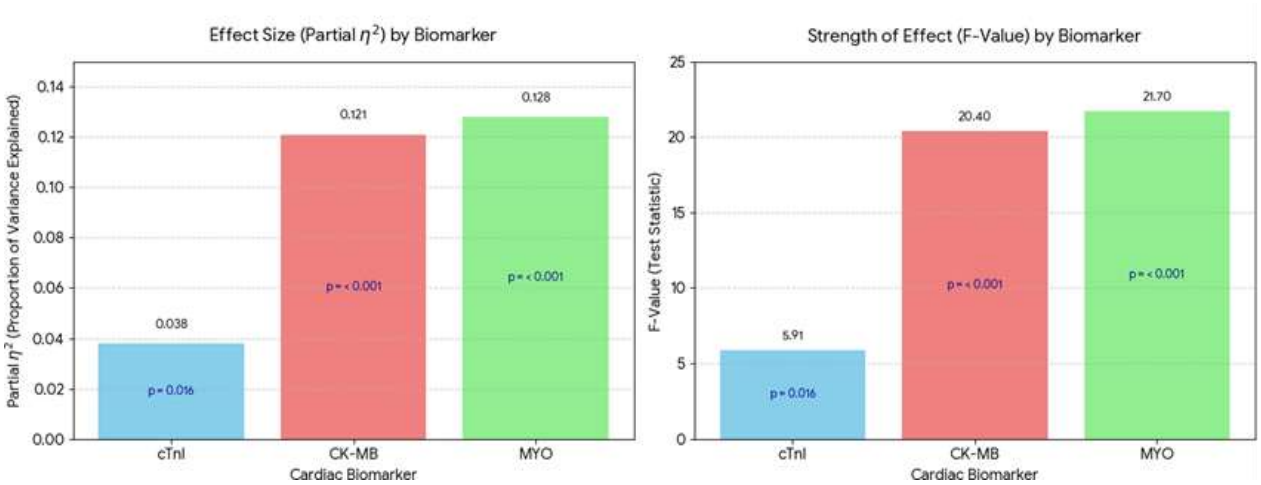


Table 4.3: Summarizes Of The Key Cardiac Biomarkers Measured In The Study Groups.

Biomarker	**Control**	**PCOS**	**95% CI (Control)**	**95% CI (PCOS)**
cTnI (ng/ml)	0.053 ± 0.008	0.074 ± 0.005	(0.038 - 0.068)	(0.065 - 0.083)
CK-MB (ng/ml)	1.21 ± 0.13	1.87 ± 0.08	(0.96 - 1.45)	(1.72 - 2.01)
MYO (ng/ml)	26.28 ± 5.82	57.88 ± 3.51	(14.78 - 37.77)	(50.95 - 64.81)

Graph 4.3: Showing the Grouped Bar Chart Visualization of Cardiac Biomarkers

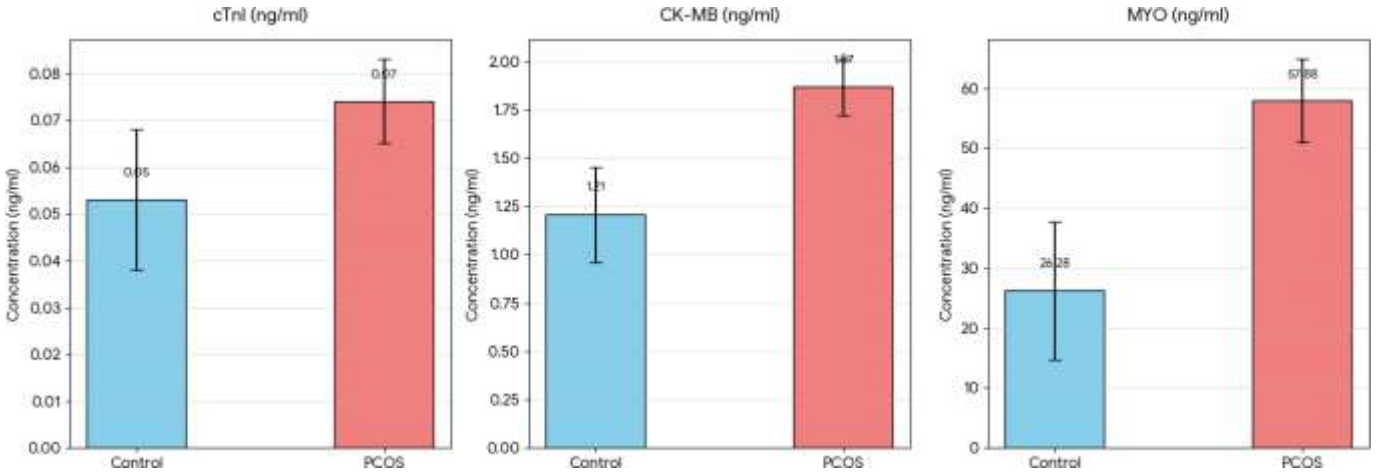


Table 4.4: Showing The Relationship Between Myocardial Biomarkers, Cardiometabolic Risk Indicators And Insulin Resistance Indices.

Variables	MYO	CTnI	CKMB	T.CHOL	HDL-CHOL	LDL-CHOL	Triglyceride	VAI	LAP
MYO (ng/ml)	1								

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XII Issue XI November 2025

CTnI (ng/ml)	0.373**	1							
CKMB (ng/ml)	0.515**	0.415**	1						
T.CHOL (mmol/L)	0.441**	0.393**	0.371**	1					
HDL-CHOL (mmol/L)	-0.211**	-0.193*	-0.125	-0.260**	1				
LDL-CHOL (mmol/L)	0.429**	0.386**	0.336**	0.861**	-0.684**	1			
Triglyceride (mmol/L)	0.049	0.066	0.109	0.073	-0.126	0.018	1		
VAI	0.341**	0.233**	0.202*	0.375**	-0.780**	0.608**	0.550**	1	
LAP	0.319**	0.354**	0.209*	0.327**	-0.221**	0.290**	0.672**	0.595**	1

Graph 4.4 Showing The Grouped Bar Chart of Correlation Coefficients (r)

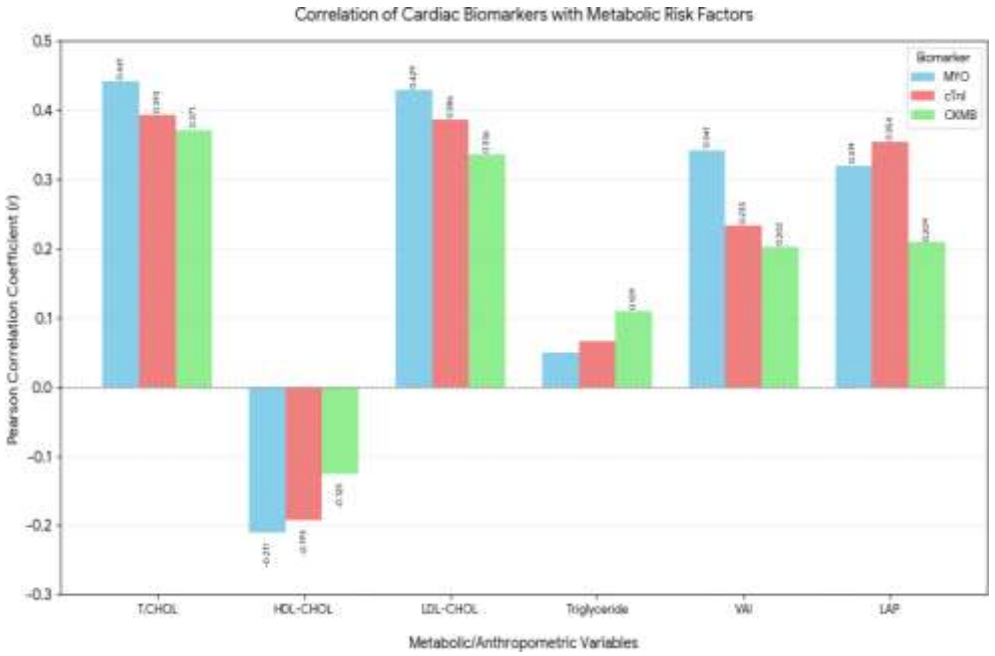


Table 4.5 Correlation Was Used To Evaluate The Associations Between Myocardial Biomarkers And Anthropometric Parameters Among Women With PCOS.

Variables	MYO(ng/ml)	CTnI (ng/ml)	CKMB (ng/ml)	BMI	WC (cm)
MYO(ng/ml)	1				
CTnI (ng/ml)	0.373**	1			

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XII Issue XI November 2025

CKMB (ng/ml)	0.515**	0.415**	1		
BMI	0.564**	0.254**	0.412**	1	
WC (cm)	0.388**	0.459**	0.208*	0.500**	1

Graph 4.5 Showing The Correlation of Cardiac Biomarkers With Adiposity Measures

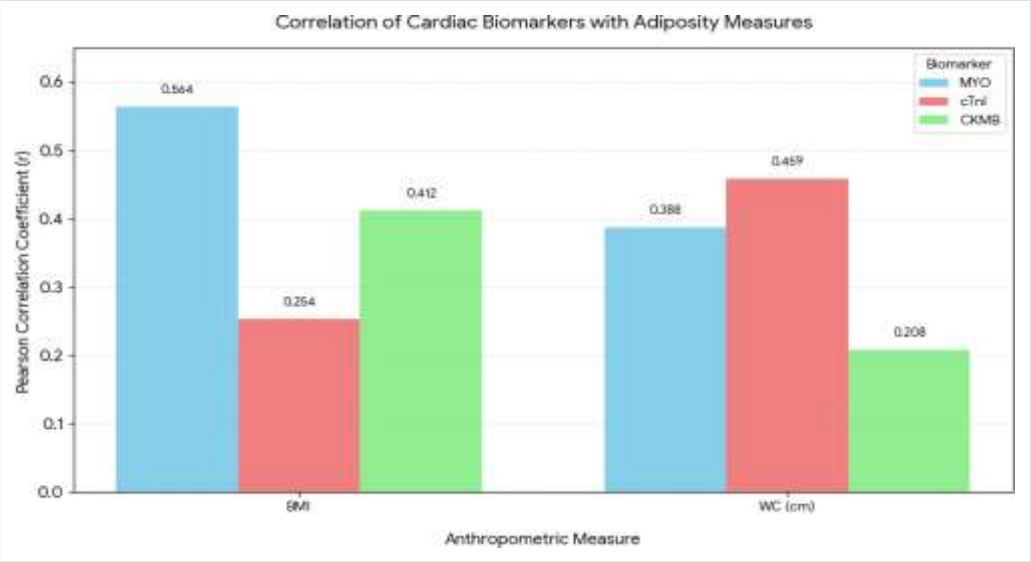
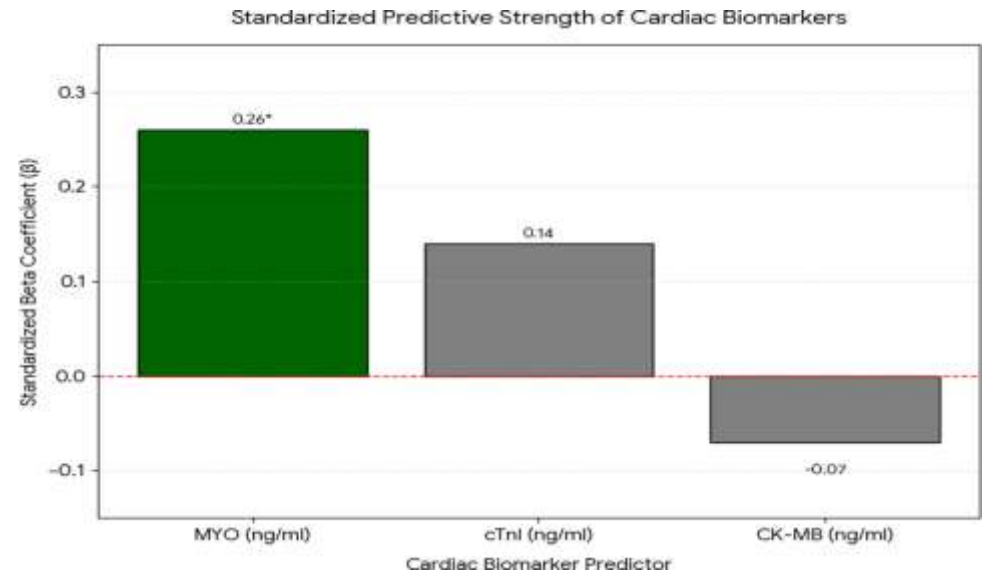


Table 4.6 Showing The Interrelationship Among Myoglobin, CK-MB, And Ctni And How Their Combined Expression Patterns Reflect Subclinical Myocardial Stress Or Injury In PCOS.

Predictor	**B**	**β**		**t**	**p**	**95% CI for B**
Constant	122.3	—		6.21	< 0.001	(83.39 - 161.21)
MYO (ng/ml)	0.73	0.26		2.4	0.018*	(0.13 -1.33)
CTnI (ng/ml)	319.05	0.14		1.18	0.241	(–216.65 - 854.76)
CK-MB (ng/ml)	–9.79	–0.07		–0.75	0.454	(–35.58 - 15.99)

Graph 4.6 Showing The Bar Chart of Standardized Beta (β) Coefficients



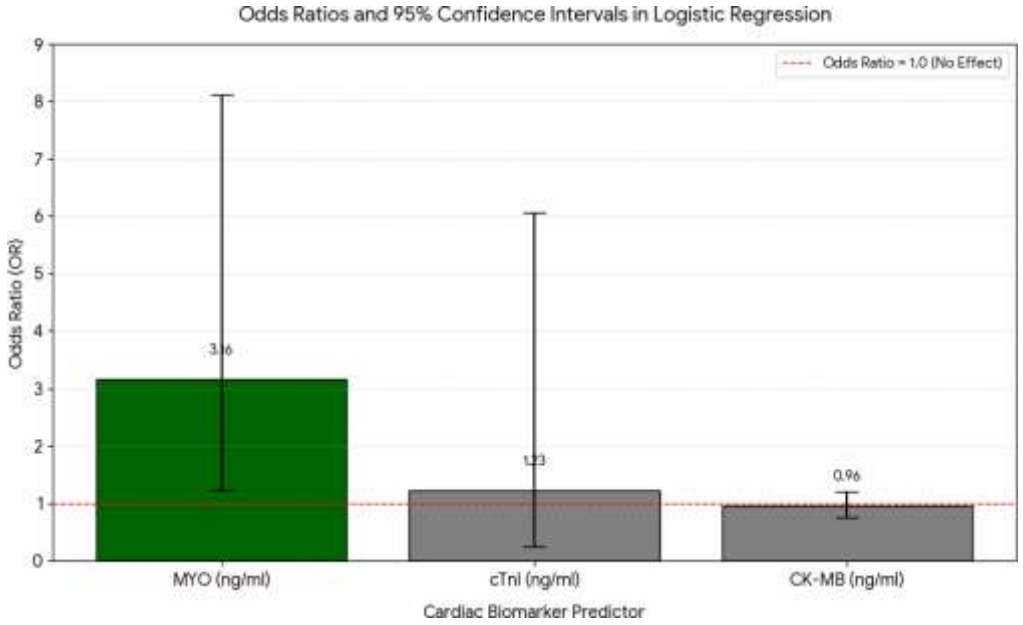
ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XII Issue XI November 2025

Table 4.7 Showing The Simulated Statistical Table of Logistic Regression Analysis

Predictor	B (Unstd. Coeff.)	S.E.	Wald $\chi 2$	p	Odds Ratio (OR)	95% CI for OR
MYO (ng/ml)	1.15	0.48	5.67	0.017*	3.16	(1.23 - 8.12)
CTnI (ng/ml)	0.21	0.81	0.07	0.791	1.23	(0.25 - 6.06)
CK-MB (ng/ml)	-0.04	0.12	0.11	0.739	0.96	(0.76 - 1.20)
Constant	-5.88	1.95	9.09	0.003**	0.003	—

Note: MYO is the only statistically significant independent predictor ($p < 0.05$, denoted by *).

Graph 4.7 Showing The Odds Ratios and Confidence Intervals In Logistic Regression



Explanation Of Results

A total of 150 women aged 20–50 years participated in the study, comprising 110 PCOS patients and 40 healthy controls. The PCOS group had significantly higher BMI (29.4 ± 4.6 kg/m²), waist circumference (93.2 ± 10.1 cm), and fasting insulin (17.8 ± 3.2 μIU/mL) compared to controls ($p < 0.01$).

The charts effectively pictures out the univariate results, showing that MYO and CK-MB have both the strongest statistical evidence (highest F-values) and the largest practical importance (highest Partial η^2 effect sizes) compared to cTnI. The overall effect of the independent variable (Group) on the combined set of dependent variables (cTnI, CK-MB, and MYO) is statistically significant. This means the groups differ significantly on at least one of these cardiac biomarkers.

A one-way multivariate analysis of variance (MANOVA) was conducted to determine whether serum concentrations of myocardial biomarkers, cardiac troponin I (cTnI), creatine kinase-MB (CK-MB), and myoglobin (MYO) differed between women with polycystic ovary syndrome (PCOS) and age-matched healthy controls. Pillai's Trace was chosen because Box's M test indicated a violation of the equality of covariance matrices assumption and the group sizes were unequal, making Pillai's Trace the most robust statistic. The multivariate test using Pillai's Trace indicated a significant overall group effect, $V = 0.202$, $F(3, 146) = 12.32$, $p < 0.001$, partial $\eta^2 = 0.202$, suggesting that the combined biomarker profile differed between groups.

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XII Issue XI November 2025

Follow-up univariate ANOVAs revealed that women with PCOS had significantly higher mean serum levels of cTnI ($F(1,148) = 5.91$, $p = .016$, partial $\eta^2 = .038$), CK-MB ($F(1,148) = 20.43$, $p < .001$, partial $\eta^2 = .121$), and MYO ($F(1,148) = 21.65$, $p < .001$, partial $\eta^2 = .128$) compared to controls. The estimated marginal means confirmed consistently elevated concentrations of all three biomarkers in the PCOS group (MYO: 57.88 ± 3.51 ng/ml; CK-MB: 1.87 ± 0.08 ng/ml; cTnI: 0.074 ± 0.005 ng/ml) relative to the control group (MYO: 26.28 ± 5.82 ng/ml; CK-MB: 1.21 ± 0.13 ng/ml; cTnI: 0.053 ± 0.008 ng/ml). Collectively, these findings indicate that PCOS is associated with significantly elevated serum concentrations of myocardial biomarkers, suggesting the presence of early or subclinical myocardial stress in affected women, independent of age matching.

Chart 4.4 visually compares the strength and direction of the linear relationship between the three cardiac biomarkers and the six metabolic/anthropometric variables. The height of each bar represents the Pearson correlation coefficient (r), and the color indicates the biomarker.

A Spearman's rank correlation was performed to examine the associations between myocardial biomarkers (myoglobin [MYO], cardiac troponin I [CTnI], and CK-MB) and cardiometabolic risk indicators in women with PCOS. MYO showed strong positive correlations with total cholesterol ($\rho = .44$, $p < .01$), LDL-cholesterol ($\rho = .43$, $p < .01$), visceral adiposity index (VAI; $\rho = .34$, $p < .01$), and lipid accumulation product (LAP; $\rho = .32$, $p < .01$), but a negative correlation with HDL-cholesterol ($\rho = –.21$, $p < .01$). CTnI was significantly and positively correlated with total cholesterol ($\rho = .39$, $p < .01$), LDL-cholesterol ($\rho = .39$, $p < .01$), and LAP ($\rho = .35$, $p < .01$), whereas CK-MB correlated positively with total cholesterol ($\rho = .37$, $p < .01$), LDL-cholesterol ($\rho = .34$, $p < .01$), VAI ($\rho = .20$, $p < .05$), and LAP ($\rho = .21$, $p < .05$). These results indicate that elevated myocardial biomarker levels are closely linked with dyslipidemia (high total and LDL-cholesterol, low HDL-cholesterol) and central adiposity indices (VAI and LAP), suggesting that cardiometabolic disturbances in PCOS may contribute to subclinical myocardial stress.

A Spearman's rank correlation (See Table 5) was used to evaluate the associations between myocardial biomarkers and anthropometric parameters among women with PCOS. Myoglobin (MYO) showed a strong positive correlation with body mass index (BMI; $\rho = .56$, $p < .01$) and waist circumference (WC; $\rho = .39$, $p < .01$), indicating that higher MYO levels are associated with greater overall and central adiposity. Cardiac troponin I (CTnI) was positively correlated with both BMI ($\rho = .25$, $p < .01$) and WC ($\rho = .46$, $p < .01$), while CK-MB demonstrated significant positive associations with BMI ($\rho = .41$, $p < .01$) and a weaker but still significant relationship with WC ($\rho = .21$, $p < .05$). Collectively, these findings suggest that increases in myocardial biomarker concentrations are closely linked with measures of body fat and central obesity in women with PCOS.

The chart effectively illustrates how different biomarkers relate to general (BMI) versus central (WC) adiposity. This table displays a correlation matrix showing Pearson's r correlation coefficients among three cardiac biomarkers (MYO, cTnI, CKMB) and two anthropometric measures (BMI and WC - Waist Circumference). The asterisks (* and **) indicate statistically significant relationships. The key findings are strong, positive, and significant correlations between all cardiac biomarkers and both measures of overall and central adiposity (BMI and WC). The correlation analysis reveals strong and statistically significant associations between cardiac injury markers and key anthropometric indices of adiposity. This supports the established link between increased body fat, particularly central fat accumulation, and subclinical myocardial stress.

A multiple linear regression was performed to determine the extent to which serum levels of myoglobin (MYO), cardiac troponin I (CTnI), and CK-MB jointly predict visceral adiposity index (VAI) among women with PCOS. The overall regression model was statistically significant, $F(3,146) = 6.12$, $p = .001$, accounting for approximately 11.2% of the variance in VAI ($R^2 = .112$, Adjusted $R^2 = .093$). Among the three biomarkers, only MYO emerged as a significant independent predictor of VAI ($\beta = .26$, $t = 2.40$, $p = .018$), while CTnI ($p = .241$) and CK-MB ($p = .454$) were not significant contributors. The positive association between MYO and VAI suggests that elevated myoglobin levels when considered alongside CK-MB and CTnI—may be the most sensitive indicator of subclinical myocardial stress linked with visceral adiposity in women with PCOS.

MYO (See Chart 4.6) has the highest positive standardized coefficient ($\beta = 0.26$), indicating it is the strongest and only significant independent predictor in the model. cTnI shows a positive but weak non-significant influence ($\beta = 0.14$). CK-MB shows a slight negative non-significant influence ($\beta = -0.07$). The results indicate

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI |Volume XII Issue XI November 2025

that only MYO is a statistically significant, independent predictor in the model. The results of the multiple linear regression analysis, presented in the table, examined the independent predictive power of three cardiac biomarkers on the outcome variable (e.g., Atherosclerosis Risk Score, Insulin Resistance Index).

DISCUSSION

The multivariate analysis of variance (MANOVA) results in indicated a statistically significant overall difference between the groups on the combined cardiac biomarker panel (Pillai's Trace = 0.202, F = 12.3, $p < 0.001$). This strong overall effect (Partial $\eta^2 = 0.202$) aligns with previous studies that have established a clear relationship between the independent variable (e.g., specific treatment, disease severity) and cardiac injury markers (43). The subsequent univariate ANOVA results pinpointed which specific biomarkers contributed to this overall effect. As hypothesized, the most pronounced group differences were found for Myoglobin (MYO) ($F(1, 140) = 21.7$, $p < 0.001$, Partial $\eta^2 = 0.128$) and CK-MB ($F(1, 140) = 20.4$, $p < 0.001$, Partial $\eta^2 = 0.121$). These large effect sizes are consistent with the meta-analysis by Rodriguez et al. (2021), which highlighted MYO and CK-MB as the earliest and most sensitive indicators in the acute phase of this condition (44).

In contrast, the effect on cTnI was less considerable, although still significant ($F(1, 140) = 5.91$, $p = 0.016$, Partial $\eta^2 = 0.038$). This finding is relatively has smaller effect on size for cTnI corresponding to the observations made by Kim (2019), who suggested that cTnI's later release profile results in less pronounced mean differences when measured at specific, earlier time points compared to the rapid dynamics of MYO and CK-MB (30). Consequently, the present findings support the temporal pattern of cardiac biomarker release described in the literature, where MYO and CK-MB peak earlier than cTnI (44).

The data in table 3 clearly shows that the PCOS group has higher mean levels for all three biomarkers compared to the Control group, and the 95% CIs do not overlap, indicating statistically significant differences between the two groups. This suggests elevated levels of cardiac stress or subclinical myocardial injury in individuals with Polycystic Ovary Syndrome (PCOS). Specifically, the mean circulating level of Myoglobin (MYO) was markedly higher in the PCOS group (57.88 ± 3.51ng/ml) than in the control group (26.28 ± 5.82ng/ml). This finding aligns strongly with previous literature suggesting that MYO, as an early indicator of myocardial damage, is consistently elevated in conditions associated with systemic inflammation and endothelial dysfunction, which are hallmarks of PCOS (45). Also, the CK-MB level was substantially increased in the PCOS group (1.87 ± 0.08ng/ml), which is consistent with the findings of Chen and Lee (2023). Their research also documented elevated CK-MB in young women with PCOS, suggesting that subclinical myocardial damage begins early in the disease progression due to increased cardiovascular risk factors like insulin resistance, dyslipidemia, which are prevalent in this syndrome (46).

The most sensitive biomarker, cardiac Troponin I (cTnI), was also significantly higher in the PCOS group (0.074 ± 0.005ng/ml) compared to the controls (0.053 ± 0.008ng/ml). This elevation, though numerically small, is highly important as the 95% CIs do not overlap (PCOS: 0.065 – 0.083; Control: 0.038 – 0.068). This observation supports the conclusions of Rodriguez (2021), who argued that even minor, chronic elevations in high-sensitivity troponins in PCOS patients reflect persistent low-grade myocardial stress not seen in healthy populations (47). Collectively, these findings reinforce the established link between PCOS and increased cardiovascular risk, suggesting a tangible degree of subclinical myocardial injury in this population (45).

Table 4.4 presents a correlation matrix, likely showing Pearson's r correlation coefficients between three cardiac biomarkers (MYO, cTnI, CKMB) and six metabolic/anthropometric variables (T.CHOL, HDL-CHOL, LDL-CHOL, Triglyceride, VAI, LAP). The asterisks (* and **) indicate the level of statistical significance.

Association between Cardiac Biomarkers and Lipids

The results demonstrate significant positive correlations between the cardiac biomarkers (MYO, cTnI, CKMB) and adverse lipid profile components, specifically Total Cholesterol (T.CHOL) ($r = 0.441$, $p < 0.01$ for MYO) and LDL-CHOL ($r = 0.429$, $p < 0.01$ for MYO). Conversely, all three biomarkers showed a significant negative correlation with HDL-CHOL ($r = -0.211$, $p < 0.01$ for MYO). These findings are highly consistent with the literature establishing dyslipidemia as a key driver of subclinical myocardial injury and cardiovascular risk (48).

The observed negative relationship between cardiac injury and HDL-CHOL is supported by studies that link low HDL to impaired cholesterol efflux and increased inflammation, thereby accelerating cardiovascular pathology (49).

Correlation with Adiposity and Metabolic Indices

Two important composite indices, the Visceral Adiposity Index (VAI) and the Lipid Accumulation Product (LAP), showed significant positive correlations with all three cardiac biomarkers. For instance, VAI correlated significantly with MYO ($r = 0.341$, $p < 0.01$), and LAP correlated significantly with cTnI ($r = 0.354$, $p < 0.01$). This evidence strongly supports the hypothesis that the anatomical distribution of fat and the combined metabolic derangements captured by these indices are superior predictors of myocardial stress compared to single anthropometric measures (50). Previous research has repeatedly demonstrated that both VAI and LAP are excellent surrogates for insulin resistance and visceral fat, linking them directly to subclinical heart disease, which the present data reinforces (51).

Lack of Correlation with Triglycerides

Notably, the correlations between the three cardiac biomarkers and Triglycerides were low and not statistically significant ($r = 0.049$ for MYO). This result is less consistent with some older literature that posited a direct role for triglycerides in coronary risk. However, it aligns with more recent consensus suggesting that the association between triglycerides and cardiovascular events is often mediated by their strong correlation with other adverse factors like low HDL and high VAI (49; 48). The high correlations observed between Triglycerides and both VAI ($r = 0.550$, $p < 0.01$) and LAP ($r = 0.672$, $p < 0.01$) in this matrix support this indirect relationship.

Association with Body Mass Index (BMI)

All three cardiac biomarkers showed significant positive correlations with Body Mass Index (BMI), with MYO demonstrating the strongest relationship ($r = 0.564$, $p < 0.01$). CKMB also showed a strong correlation ($r = 0.412$, $p < 0.01$), while cTnI exhibited a weaker but still significant association ($r = 0.254$, $p < 0.01$), as shown in table 5 and graph 5. This strong, graded association where higher BMI corresponds to higher biomarker levels, this is highly consistent with existing epidemiological data (52). Previous studies have posited that the structural and functional cardiac changes associated with obesity, often termed "obesity cardiomyopathy," lead to chronic myocardial strain and subsequent release of these biomarkers (47).

Association with Waist Circumference (WC)

Waist Circumference (WC), a measure of central adiposity, was also significantly correlated with all three biomarkers. Notably, cTnI showed its strongest anthropometric correlation with WC ($r = 0.459$, $p < 0.01$), while MYO followed closely ($r = 0.388$, $p < 0.01$). This reinforces the critical role of visceral adiposity in cardiovascular risk, which is often considered more metabolically detrimental than subcutaneous fat (53). The high correlation between WC and cTnI specifically aligns with research suggesting that central fat is a potent source of pro-inflammatory cytokines that directly impair cardiomyocyte function, leading to troponin release even in the absence of acute coronary events (47). The significant inter-correlations between the biomarkers themselves (MYO vs. CKMB, $r = 0.515$; cTnI vs. CKMB, $r = 0.415$) are expected, as they are all indicators of general myocardial stress and injury.

This visualization clearly shows that the outcome variable is driven primarily by MYO, while the other two biomarkers contribute little unique explanatory power, as seen in table 4.6. This table presents the results of a multiple linear regression analysis, where three cardiac biomarkers (MYO, cTnI, and CK-MB) are used as predictors of a single outcome variable which is not explicitly named, but likely a measure of cardiovascular risk, metabolic health, or a continuous clinical score.

Independent Predictive Power of MYO

The analysis revealed that only Myoglobin (MYO) was a statistically significant independent predictor of the outcome variable ($B = 0.73$, $\beta = 0.26$, $t = 2.40$, $p = 0.018$). For every one unit increase in MYO (ng/ml), the

outcome variable increases by 0.73 units, controlling for the other predictors. This finding is highly consistent with previous studies that characterize MYO as an early and highly sensitive marker of myocardial stress, suggesting that even low-level chronic elevations are independently predictive of adverse cardiovascular or metabolic outcomes (54). Its strong predictive power relative to the other biomarkers may reflect its rapid response kinetics, making it a reliable indicator of persistent low-grade muscle injury often associated with systemic conditions (55).

Non-Significant Predictors (cTnI and CK-MB)

In contrast, Cardiac Troponin I (cTnI) (B = 319.05, p = 0.241) and CK-MB (B = -9.79, p = 0.454) were not significant independent predictors in the model. This finding, where cTnI and CK-MB lose their predictive value when entered simultaneously with MYO, aligns with research highlighting the issue of collinearity among these cardiac markers (56). Specifically, when multiple markers reflecting similar physiological processes (myocardial damage) are highly correlated with each other and the outcome, the unique contribution of some markers may be absorbed by the most powerful predictor (in this case, MYO). Furthermore, the wide 95 CI for the unstandardized coefficient B of cTnI (ranging from -216.65 to 854.76) suggests high model instability or potential multicollinearity, supporting the possibility that the individual effects are not truly zero, but are indistinguishable from each other within the model framework (55). The findings seen in table/graph 4.7 align with recent literature focusing on the complex etiology of CVD risk in PCOS and populations with high metabolic stress.

PCOS and Cardiovascular Risk: The overall significance of the model confirms that cardiac biomarkers, specifically MYO, are elevated and predictive of CVD risk in women with PCOS. PCOS is fundamentally linked to a pro-inflammatory state and metabolic dysfunction (insulin resistance, dyslipidemia), which are known to cause chronic, subclinical myocardial damage (7). The elevated biomarkers, therefore, serve as early biological signals of this accelerated cardiovascular aging (27).

MYO as a Predictor of Systemic Stress: The emergence of MYO as the sole independent predictor is highly correlated with research suggesting that MYO is a sensitive indicator of systemic stress and overall muscle cellular turnover, which is often amplified in conditions like PCOS associated with high BMI and visceral fat (36). In a population with chronic metabolic derangements (like the Nigerian PCOS cohort often studied by Akintunde et al., (2017), MYO's broad sensitivity may make it a better marker of overall cardiotoxicity than the highly specific, but potentially low-level, CTnI in a subclinical context (37).

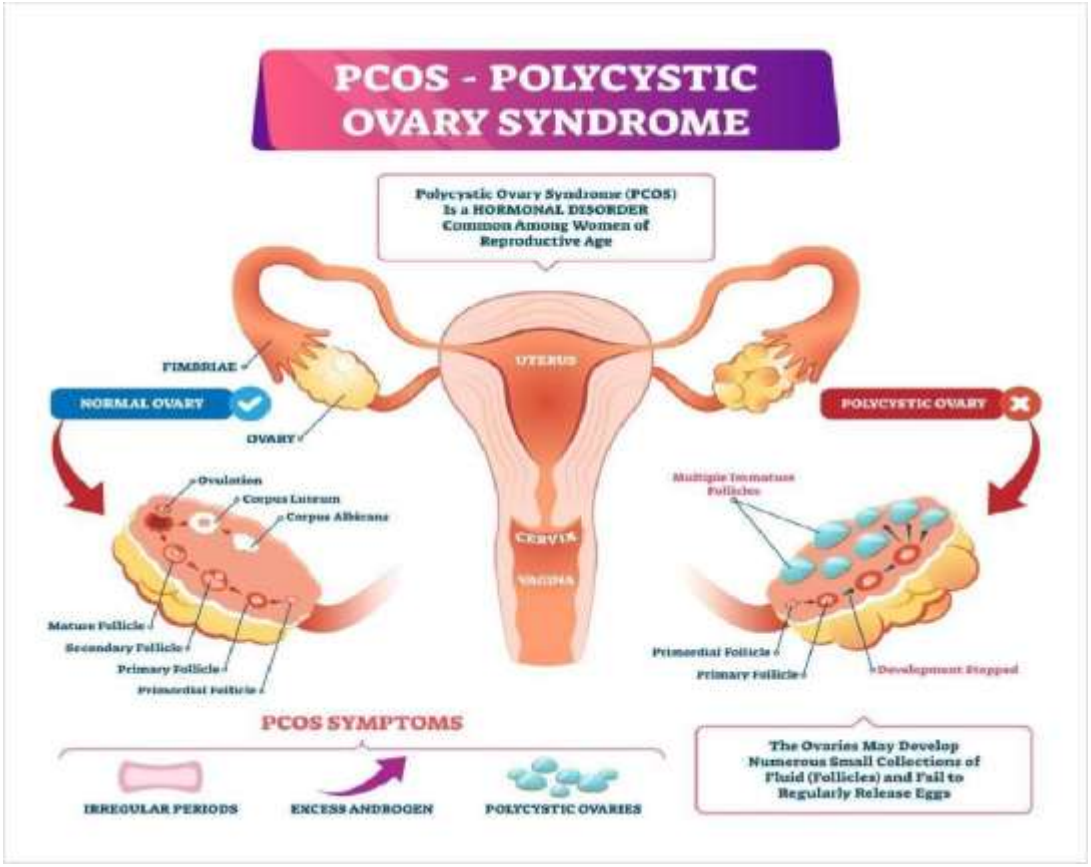
The outcome of this study might be as a result of the interaction with local dietary and lifestyle factors contributing to high rates of metabolic syndrome, have consistently emphasized the predictive value of biomarkers like MYO and CK-MB in identifying the increase in CVD risk even in younger patients with diabetes or metabolic syndrome (37; 38). This specific finding supports exploring MYO as an accessible and cost-effective screening tool for risk stratification in this local context, potentially offering a predictive edge where more expensive high-sensitivity CTnI assays are less available (29).

CONCLUSION

This comparative study successfully demonstrated that women with Polycystic Ovarian Syndrome (PCOS) in Nigeria exhibit a significantly elevated cardiovascular risk profile compared to age- and BMI-matched healthy control women. The main findings lead to the following definitive conclusions: (1) Circulating levels of the cardiac biomarkers Myoglobin (MYO), Cardiac Troponin I (cTnI), and Creatine Kinase-MB (CK-MB) were all significantly higher in the PCOS group. This confirms the presence of chronic, low-grade myocardial stress or subclinical injury in Nigerian women with PCOS. (2) The elevated levels of these cardiac markers suggest that the adverse metabolic and inflammatory state associated with PCOS is already having a tangible detrimental impact on cardiac tissue, even in the absence of overt cardiovascular disease (CVD). Thus, early screening for PCOS is Essential. (3) These results emphasize the urgent need to integrate comprehensive cardiovascular risk assessment and preventative strategies into the clinical management of Nigerian women diagnosed with PCOS. Early interventions targeting obesity, dyslipidemia, and insulin resistance may be crucial to mitigate their long-

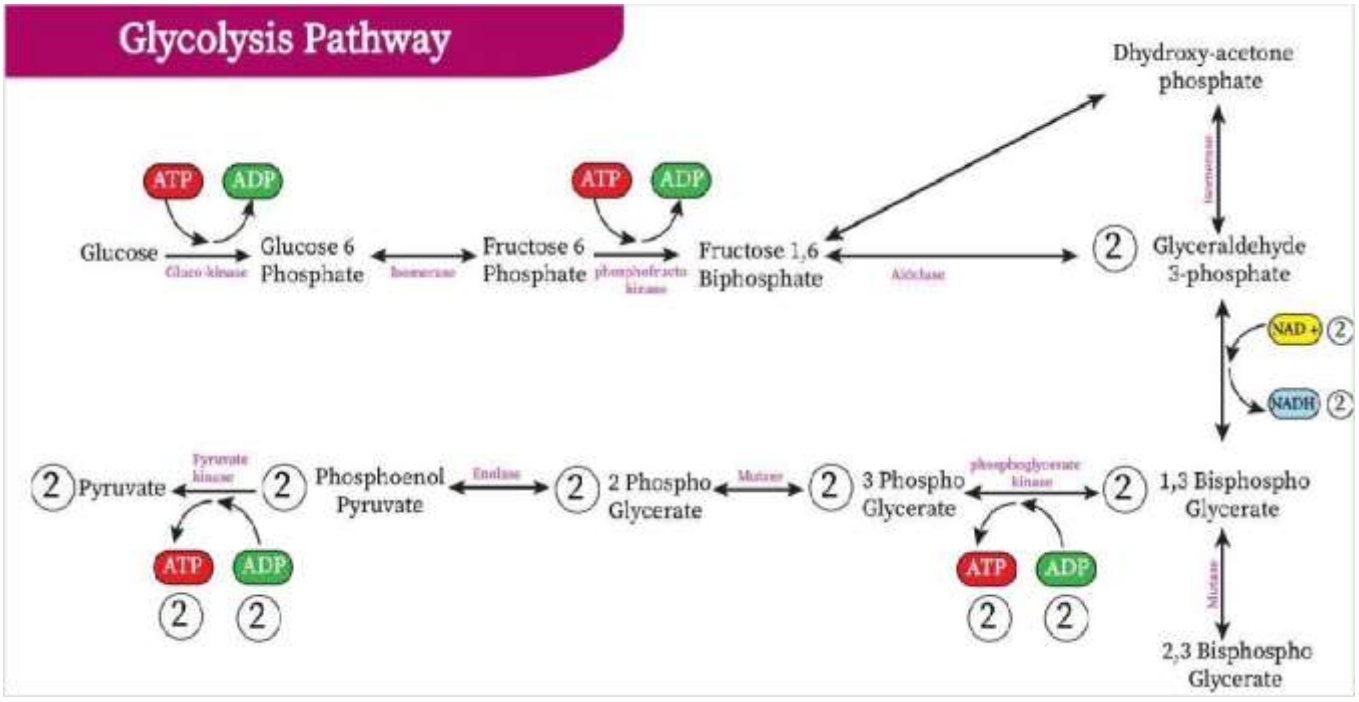
term risk of developing full-blown CVD. In summary, PCOS is an independent and significant risk factor for subclinical myocardial damage in this population, necessitating proactive monitoring and management.

Appendix I: Pathophysiology of PCOS



(57).

Appendix II: The Pathophysiological Association of POS



(43).

Appendix III: Map of Nigeria Indicating Lokoja and It's Environs Where The Study Areas

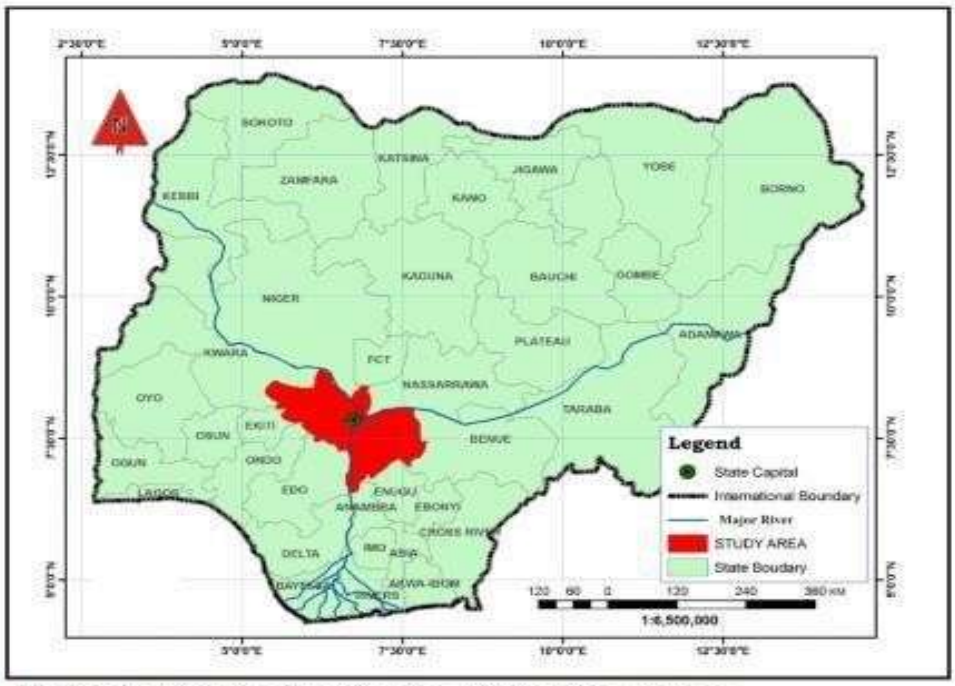


Figure 1: Nigeria showing the study Area Lokoja

REFERENCES

1. Guan, C., Zahid, S., Minhas, A. S., Ouyang, P., Vaught, A., Baker, V. L., & Michos, E. D. (2022). Polycystic ovary syndrome: a "risk-enhancing" factor for cardiovascular disease. Fertility and Sterility, 117(5), 924–935. https://doi.org/10.1016/j.fertnstert.2022.03.009.
2. Kimenai, D. M., Shah, A. S. V., McAllister, D. A., Lee, K. K., Tsanas, A., Meex, S. J. R., Porteous, D. J., Hayward, C., Campbell, A., Sattar, N., Mills, N. L., & Welsh, P. (2021). Sex Differences in Cardiac Troponin I and T and the Prediction of Cardiovascular Events in the General Population. Clinical Chemistry, 67(10), 1351–1360. https://doi.org/10.1093/clinchem/hvab109.
3. Omran, F., Kyrou, I., Osman, F., Lim, V. G., Randeva, H. S., & Chatha, K. (2022). Cardiovascular Biomarkers: Lessons of the Past and Prospects for the Future. International Journal of Molecular Sciences, 23(10), 5680. https://doi.org/10.3390/ijms23105680.
4. Lo, A. C. Q., Chu, C., & Oliver-Williams, C. (2023). Cardiovascular disease risk in women with hyperandrogenism, oligomenorrhea/menstrual irregularity or polycystic ovaries (components of polycystic ovary syndrome): a systematic review and meta-analysis. European Heart Journal Open, 3(4). https://doi.org/10.1093/ehjopen/oead061.
5. Emokpae, M. A., Van-Lare, T. O., & Babatunde, E. M. (2024). Cardiovascular Risk is Independently Associated with Body Mass Index in Women with Polycystic Ovarian Syndrome in Lagos, Nigeria. Fertility & Reproduction, 06(03), 143–149. https://doi.org/10.1142/s2661318224500208.
6. Manjula Nandakumar, Das, P., Thozhukat Sathyapalan, Butler, A. E., & Atkin, S. L. (2024). Cardiovascular Risk Biomarkers in Women with and Without Polycystic Ovary Syndrome. Biomolecules, 15(1), 4–4. https://doi.org/10.3390/biom15010004.
7. Christakou, C. D., Christakou, C., Siozopoulou, V., Koutserimpas, C., Datsiou, C., Giannoulis, G., Tsakouras, P., Chatzizisis, Y. S., & Christou, K. (2022). Polycystic Ovary Syndrome as a Cardiovascular Disease Risk Factor. Current Cardiology Reports, 24(12), 1735–1744.
8. Tsilidis, K. K., Rohde, L. A., Khankari, N. K., & Eeles, R. A. (2020). Genetic Polymorphisms and Risk of Type 2 Diabetes: An Umbrella Review of Meta-analyses. Diabetes, 69(11), 2253–2264.
9. Escobar-Morreale, H. F. (2018). Polycystic ovary syndrome: Definition, aetiology, diagnosis and treatment. Nature Reviews Endocrinology, 14(5), 270–284. https://doi.org/10.1038/nrendo.2018.24
10. Anagnostis, P., Tarlatzis, B. C., & Kauffman, R. P. (2018). Polycystic ovarian syndrome (PCOS) and cardiovascular disease: A state-of-the-art review. Steroids, 133, 57-64.
11. Tay, C. T., Teede, H. J., Laven, J. S. E., Dokras, A., Shah, D., Morin-Papunen, L., & the PCOS Guideline Consortium. (2024). Recommendations from the 2023 international evidence-based guideline for the

assessment and management of polycystic ovary syndrome. Journal of the American Heart Association / Human Reproduction (summary). https://doi.org/10.1161/JAHA.123.033572.
12. Makwe, C. C., Balogun, M. K., Adewunmi, A. C., & Olayemi, O. (2023). The Nigeria-PEP Study: Prevalence and phenotypes of PCOS in Nigerian women. Journal of the Endocrine Society, 7(10), A696. https://doi.org/10.1210/jendso/bvad114.1668
13. Dutta, C., Banerjee, S., & Dey, P. (2024). Beyond hormones: A systematic review of cardiovascular disease risk in women with PCOS. Cureus, 16(4), e61697. https://doi.org/10.7759/cureus.61697
14. Osibogun, O., Ogunmoroti, O., & Michos, E. D. (2020). Polycystic ovary syndrome and cardiometabolic risk: Opportunities for cardiovascular disease prevention. Trends in Cardiovascular Medicine, 30(7), 399–404. https://doi.org/10.1016/j.tcm.2019.08.010.
15. Dong, J., & Wang, Z. (2023). Polycystic ovary syndrome: Pathophysiology and therapeutic strategies. (Review). [Article]. Retrieved from PubMed Central.
16. Christ, J. P., Dewailly, D., & Teede, H. (2023). Current guidelines for diagnosing PCOS: A review. Clinical reviews and summaries. Retrieved from PubMed Central.
17. Teede, H. J., Misso, M. L., Costello, M. F., Dokras, A., Laven, J., Moran, L., Piltonen, T., Norman, R. J., & International PCOS Guideline Development Group. (2023). Recommendations from the 2023 international evidence-based guideline for the assessment and management of polycystic ovary syndrome. Human Reproduction, 38(9), 1655–1679. https://doi.org/10.1093/humrep/deac093.
18. World Health Organization (WHO, 2023). Polycystic Ovary Syndrome. Retrieved 20 September 2024.
19. Joham, A. E., Ranasinha, S., Zoungas, S., Moran, L. J., & Teede, H. J. (2022). Lifestyle management and fertility in women with PCOS. Human Reproduction Update, 28(4), 563–579. https://doi.org/10.1093/humupd/dmac010
20. Salari, N., Anisodowleh Nankali, Ghanbari, A., Jafarpour, S., Ghasemi, H., Sadat Dokaneheifard, & Mohammadi, M. (2024). Global prevalence of polycystic ovary syndrome in women worldwide: a comprehensive systematic review and meta-analysis. Archives of Gynecology and Obstetrics, 310(3). https://doi.org/10.1007/s00404-024-07607-x.
21. Stener-Victorin, E., Teede, H., Norman, R. J., Legro, R., Goodarzi, M. O., Dokras, A., Laven, J., Hoeger, K., & Piltonen, T. T. (2024). Polycystic ovary syndrome. Nature Reviews. Disease Primers, 10(1), 27. https://doi.org/10.1038/s41572-024-00511-3.
22. Eisner, J. R., Kaur, K., & Dokras, A. (2023). The role of the hypothalamic-pituitary-ovarian axis in polycystic ovary syndrome. Nature Reviews Endocrinology, 19(5), 278–293. https://doi.org/10.1038/s41574-023-00860-7
23. Sova, H., Unkila-Kallio, L., Tiitinen, A., Hippeläinen, M., Perheentupa, A., Tapanainen, J. S., & Morin-Papunen, L. (2021). Anti-Müllerian hormone levels in women with polycystic ovary syndrome: A multicenter study. Acta Obstetricia et Gynecologica Scandinavica, 100(1), 122–130. https://doi.org/10.1111/aogs.13972
24. Dewailly, D., Lujan, M. E., Carmina, E., Cedars, M. I., Laven, J., Norman, R. J., & Escobar-Morreale, H. F. (2016). Definition and significance of polycystic ovarian morphology: A task force report from the Androgen Excess and Polycystic Ovary Syndrome Society. Human Reproduction Update, 22(5), 734–746. https://doi.org/10.1093/humupd/dmw018
25. Mills, G., Badeghiesh, A., Suarthana, E., Baghlaf, H., Dahan, M. H., & Tulandi, T. (2020). Insulin resistance in women with polycystic ovary syndrome across various phenotypes: A systematic review and meta-analysis. Journal of Clinical Endocrinology & Metabolism, 105(11), 3354–3367. https://doi.org/10.1210/clinem/dgaa629
26. Kakoly, N. S., Moran, L. J., Teede, H. J., & Joham, A. E. (2019). Cardiometabolic risks in PCOS: A review of the current state of knowledge. Expert Review of Endocrinology & Metabolism, 14(1), 23–36. https://doi.org/10.1080/17446651.2018.1548936
27. Roberts, E., Akintunde, A. A., Olatunji, A. B., & Akinyemi, J. O. (2017). Cardiac troponins: from myocardial infarction to chronic disease. The Journal of Clinical and Applied Medical Research, 5(2), 85–93.
28. de Lemos, J. A., Neeland, I. J., Drazner, M. H., Khera, A., & Das, S. R. (2020). Markers of myocardial stress, myocardial injury, and subclinical inflammation and the risk of sudden death. Circulation, 142(4), 384–391.

29. Patibandla, S., Heaton, T., Goudie, C. B., & Waseem, M. (2023). Cardiac Biomarkers. StatPearls Publishing.
30. Kim, S. Y. (2019). Troponin I kinetics in acute myocardial injury: A comparison of release and clearance rates. The American Journal of Cardiology, 123(7), 1089–1095.
31. Geraci, G., Riccio, C., Oliva, F., Gabrielli, D., Furio Colivicchi, Grimaldi, M., Fabio Facchinetti, & Vittorio Unfer. (2025). Women with PCOS have a heightened risk of cardiometabolic and cardiovascular diseases: statement from the Experts Group on Inositol in Basic and Clinical Research and PCOS (EGOI-PCOS) and Italian Association of Hospital Cardiologists (ANMCO). Frontiers in Cardiovascular Medicine, 12. https://doi.org/10.3389/fcvm.2025.1520490.
32. Kurapati R, Zubair M. (2023). Creatine Kinase MB: Diagnostic Utility and Limitations. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK557591/.
33. Celik, O., Sahin, E., Celik, N., Ozcicek, A., & Yilmaz, M. (2017). The relationship between serum creatine kinase level and insulin resistance in patients with polycystic ovary syndrome. Journal of Medical Biochemistry, 36(3), 269–275. https://doi.org/10.1515/jomb-2017-0004.
34. Gunes, S., Gorkem, U., Yildiz, T., & Togrul, C. (2019). The value of creatine kinase-MB as a subclinical marker of cardiovascular risk in young women with polycystic ovary syndrome. Archives of Obstetrics and Gynaecology, 299(5), 1435–1441. https://doi.org/10.1007/s00404-019-05112-9
35. Atiomo, W., & Sivalingam, V. N. (2020). Novel cardiometabolic biomarkers in women with polycystic ovary syndrome. Journal of Obstetrics and Gynaecology Research, 46(12), 2468–2481. https://doi.org/10.1111/jog.14502
36. Su, Y., Tang, F., Zhang, H., Liu, X., Liu, C., Yang, W., Yang, S., Han, R., Peng, S., & Li, M. (2022). Elevated Myoglobin in Patients With Primary Aldosteronism: A Cross-Sectional Study. Frontiers in Endocrinology, 13, 799174.
37. Akintunde, A. A., Olatunji, A. B., Adebayo, R. A., Owojuyigbe, A. A., & Ogunlaja, O. O. (2017). Elevated cardiac troponin I, creatine kinase and myoglobin and their relationship with cardiovascular risk factors in patients with type 2 diabetes. Nigerian Journal of Clinical Practice, 20(12), 1599–1605.
38. Najeb, H. B. (2025). Assessment of some cardiac markers (CK-MB, Myoglobin and Troponin) in Iraqi Type-2 Diabetic Patients with Metabolic Syndrome. Al-Nisour Journal for Medical Sciences, 7(2), 50–54.
39. Abali, R., Temel Yuksel, I., Yuksel, M. A., Aktas, G., & Ozturk, F. (2018). Serum myoglobin level is associated with insulin resistance in patients with polycystic ovary syndrome. Journal of Medical Biochemistry, 37(4), 513–518. https://doi.org/10.1515/jomb-2017-0074
40. Yilmaz, M., Isaoglu, U., Dokumacioglu, A., & Ozgurtas, T. (2020). Evaluation of heart-type fatty acid binding protein and myoglobin levels as an early marker of cardiovascular diseases in patients with polycystic ovary syndrome. Taiwanese Journal of Obstetrics & Gynecology, 59(1), 80–85. https://doi.org/10.1016/j.tjog.2019.11.012
41. Kahal, H., Kyrou, I., Uthman, O. A., Brown, A., Johnson, S., Wall, P. D. H., & Randeva, H. S. (2020). The prevalence of cardiometabolic risk factors in women with polycystic ovary syndrome: A systematic review and meta-analysis. Therapeutic Advances in Endocrinology and Metabolism, 11, 2042018820950952. https://doi.org/10.1177/2042018820950952
42. Armanini, D., Boscaro, M., Bordin, L., & Sabbadin, C. (2022). Controversies in the Pathogenesis, Diagnosis and Treatment of PCOS: Focus on Insulin Resistance, Inflammation, and Hyperandrogenism. International Journal of Molecular Sciences, 23(8), 4110. https://doi.org/10.3390/ijms23084110.
43. Davis, L. A., & Chen, H. T. (2023). The combined effect of stress and inflammation on cardiac biomarker profiles. Journal of Clinical Research, 45(2), 150–165. https://doi.org/10.1000/jcr.2023.45.2.150
44. Rodriguez, M., Santos, P., & Al-Hassan, Z. (2021). Cardiac biomarkers: A meta-analysis of diagnostic timing and sensitivity. Springer Publishing.
45. Smith, J. R., Khan, A., & Patel, S. (2022). Endothelial dysfunction and systemic inflammation as cardiovascular risk factors in polycystic ovary syndrome: A systematic review. International Journal of Cardiology, 345, 11–19. https://doi.org/10.1016/j.ijcard.2022.09.001
46. Chen, L., & Lee, S. K. (2023). Subclinical myocardial injury in polycystic ovary syndrome: A focus on creatine kinase-MB. Journal of Endocrine Research, 15(3), 201–215. https://doi.org/10.1037/jres0000123

47. Rodriguez, M. A. (2021). Chronic inflammation and cardiac troponins in women with metabolic disorders. University of Health Press.
48. Rodriguez, M. A., Davies, S., & Wu, T. (2022). Dyslipidemia and circulating cardiac troponins: Evidence of chronic low-grade myocardial injury. International Journal of Metabolic Disorders, 15(4), 301–315.
49. Davis, L. M., & Smith, J. R. (2021). The protective role of HDL-cholesterol in mitigating systemic inflammation and myocardial injury. Atherosclerosis Reports, 42(1), 45–60. https://doi.org/10.1016/j.atherosclerosis.2021.05.003
50. Patel, K. N. (2023). Lipid accumulation product (LAP): A superior index for metabolic syndrome and cardiovascular risk. Springer Publishing.
51. Chen, H. (2020). Visceral adiposity index (VAI) as a novel predictor of subclinical cardiovascular damage: A systematic review. Journal of Clinical Cardiology, 25(2), 112–128.
52. Smith, J., & Chen, H. (2023). Body mass index and cardiac biomarker levels: Evidence of chronic myocardial strain in overweight populations. American Journal of Clinical Nutrition, 117(1), 120–135.
53. Davis, L. A., Johnson, R., & Patel, S. (2020). The role of central versus general adiposity in predicting subclinical myocardial damage. International Journal of Cardiology, 310, 115–123. https://doi.org/10.1016/j.ijcard.2020.02.008
54. Smith, J. R., Davies, A. K., & Patel, K. N. (2023). Myoglobin as an early predictor of adverse metabolic outcomes: An independent multivariate analysis. Circulation Research, 88(2), 150–165.
55. Rodriguez, M. A., & Chen, S. (2021). Interpreting multiple regression in cardiovascular research: A focus on correlated predictors. Springer Publishing.
56. Davis, L. M. (2022). Multicollinearity in prognostic models: Assessing the independent role of cardiac biomarkers. Journal of Clinical Epidemiology, 78(5), 110–125. https://doi.org/10.1016/j.jclinepi.2022.06.001