Review Article

Unmasking Cardiovascular Risk in Non-Obese Polyendocrine Metabolic Ovarian Syndrome: The Emerging Role of Novel Biomarkers

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Received: 03-07-2026; **Revised:** 21-09-2026; **Accepted:** 26-09-2026; **Published online:** 20-10-2026.

ABSTRACT

Polyendocrine Metabolic Ovarian syndrome (PMOS) is a common endocrine disorder associated with significant long-term cardiovascular disease (CVD) risk. While obesity is a known factor, non-obese (lean) PMOS women face substantial risk driven by the interplay of hyperandrogenism, inherent insulin resistance, visceral adiposity, and oxidative stress. Lean patients exhibit endothelial dysfunction and a 1.3-fold higher risk of ischemic heart disease independent of BMI. Conventional markers like hs-CRP and VAI provide limited risk stratification. However, proteomics has identified Discoidin, CUB, and LCCL domain-containing protein 2 (DCBLD2) as a specific biomarker for incident CVD in PMOS. High DCBLD2 levels strongly predict coronary artery disease and myocardial infarction, particularly in young, non-obese women, independent of traditional metabolic risk. DCBLD2 functions through angiogenesis and Epithelial-Mesenchymal Transition (EMT). Integrating this novel marker into clinical assessments allows for earlier identification and personalized prevention strategies. Ultimately, a paradigm shift is required to prioritize early cardiovascular screening and biomarker-driven risk stratification in the lean PMOS population

Keywords: Polycystic Ovary Syndrome; Cardiovascular Diseases; Cardiometabolic Risk Factors; Biomarkers; Insulin Resistance; Atherosclerosis; Body Mass Index.

INTRODUCTION

Polyendocrine Metabolic Ovarian Syndrome (PMOS) is the most common endocrine and metabolic illness affecting women of childbearing age worldwide.[1,2] This complex and heterogeneous condition is generally characterized by a combination of clinical or biochemical hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology (PCOM).[1,3] The estimated prevalence of PMOS has been found to vary between 5% and 20% in the global population, depending largely on the particular set of criteria employed for diagnosis and the population under investigation.[1-3] In a particular geographic area, such as India, the prevalence has been found to vary between 8.2% and 22.5% in adolescents and young women.[1,2]

The criteria for the diagnosis of PMOS have seen a radical shift through a series of international consensus meetings. The NIH (National Institutes of Health) criteria in 1990 included both hyperandrogenism and oligo-ovulation for diagnosis.[1,3,4] Later, the 2003 Rotterdam Consensus Meeting broadened the spectrum by requiring at least two of the three main features.[1-4] The 2006 criteria of the Androgen Excess and PMOS Society (AE-PMOS) included hyperandrogenism as a crucial criterion, which should be associated with either ovulatory dysfunction or PCOM.[1,4,5]

These criteria have been used to identify four PMOS phenotypes. Phenotype A (hyperandrogenism, ovulatory dysfunction, PCOM) is the most severe and prevalent (50-60%).[1,6,7] Phenotype B consists of hyperandrogenism and ovulatory dysfunction without PCOM (20-30%).[1,6,7] Phenotype C (ovulatory PMOS) is characterized by hyperandrogenism, PCOM, and regular menses (10-15%).[1,6,7] Phenotype D is non-hyperandrogenic and characterized by ovulatory dysfunction with PCOM (5-10%).[1,6,7] Although Phenotypes A and B have more insulin resistance and metabolic disturbances, all hyperandrogenic phenotypes are linked to an increased risk of cardiovascular disease.[1,3,6]

In addition to the reproductive manifestations of infertility and menstrual dysfunction, PMOS is also linked to long-term cardiometabolic derangements, such as insulin resistance, dyslipidemia, and hypertension.[1,8,9] One of the most important aims of ongoing research is to reveal the "silent" cardiovascular risk in non-obese patients with PMOS, who are often neglected in clinical settings.[1,9] The importance of monitoring non-obese patients is that the risk of cardiovascular events, such as high blood pressure, ischemic heart disease, and cerebrovascular accidents, often exists independently of Body Mass Index (BMI).[10-12] Lean PMOS women also have a 1.3-fold increased risk of ischemic heart disease and cerebrovascular events compared to their matched non-PMOS controls.[13] This group of patients is often neglected due to the "insidious onset" of symptoms and conventional diagnostic biomarkers that may appear normal.[9] In this context, we sought to critically evaluate the independent relationship between non-obese PMOS and cardiovascular disease (CVD) and to describe the development of new biomarkers that may indicate early subclinical cardiovascular risk. The link between PMOS and cardiovascular risk is fueled by a complex and multifactorial "vicious cycle" of hyperandrogenism (HA), insulin resistance (IR), and abdominal adiposity.[2,3,6] As reported in the previous literature, In this vicious cycle hyperandrogenism, which is genetically or epigenetically inherited, leads to

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post-receptor insulin resistance due to the inhibition of the intracellular insulin-activated pathway for glucose uptake.[6] This insulin resistance is then compensated for by hyperinsulinemia, which directly stimulates theca cells of the ovaries to secrete more androgens, thus perpetuating the cycle of hormonal imbalance.[2,6]

METHODS

This narrative review was conducted through a literature search of PubMed / MEDLINE, Scopus and Google Scholar using the key terms including "PMOS", "non obese PMOS," "cardiovascular risk," "DCBLD2," and "subclinical atherosclerosis," covering publications from 2015 to 2025. Relevant peer reviewed original studies, meta analyses, and review articles in English were included, with emphasis on non-obese or lean PMOS phenotypes. The 25 references cited in this review were selected to represent the breadth of current evidence across pathophysiology, conventional biomarkers and emerging proteomic findings in this population.

PATHOPHYSIOLOGY

The evidence provided from the review shows that, Hyperinsulinemia also has a catalytic effect on the development of abdominal visceral adiposity due to changes in lipid metabolism and a reduction in the expression of protective proteins such as adiponectin.[2,3] Hyperandrogens also preferentially promote the accumulation of visceral adipose tissue over subcutaneous adipose tissue, regardless of the patient's overall body weight, thus creating a pro-atherogenic state.[3,6] Moreover, it is further supported by other literature which highlights this adipose tissue dysfunction also leads to a chronic state of low-grade systemic inflammation.[14,15] Immune cells, such as macrophages and T-lymphocytes, are activated and migrate into visceral adipose tissue and the kidneys, releasing pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6).[2,14,15]

One of the research article shows that the role of Oxidative stress is a focal point as a contributing factor to cardiovascular injury in PMOS.[2,16] Hyperglycemia and hyperinsulinemia increase the production of Reactive Oxygen Species (ROS), which overpowers the body's antioxidant capacity, especially superoxide dismutase (SOD) and glutathione peroxidase (GPx).[2,16] High ROS levels result in lipid peroxidation, which is reflected by high levels of malondialdehyde (MDA), a marker of early endothelial dysfunction and arterial stiffness.[2,5,16] The oxidative injury to the endothelium of the blood vessels inhibits the release of nitric oxide, resulting in vascular hypertrophy and decreased compliance, ultimately culminating in an increased risk of hypertension and myocardial infarction.[15,16] In summary, the complex interplay between hormonal imbalance, inflammation, and oxidative stress creates a biological milieu that renders young women with PMOS susceptible to subclinical atherosclerosis from a young age.[5,6,15]

NON-OBESE PMOS: A DISTINCT CARDIOMETABOLIC PHENOTYPE

The lean PMOS population is of utmost importance to monitor because the risk of cardiovascular disease is often present independently of BMI.[1,9,10] Although obesity is recognized as an aggravating factor, evidence from many studies has shown that lean women with PMOS have a substantial number of cardiometabolic risk factors that cannot be accounted for by the presence of obesity alone.[4,10,13] For instance, lean women with PMOS have been shown to have a 37% increased risk of developing hypertension compared with healthy controls, even after adjusting for BMI.[10,13] In addition, lean Korean women with PMOS had a 1.3-fold increased risk of ischemic heart disease and cerebrovascular events compared with those without PMOS.[13]

One of the characteristic features of the non-obese form of PMOS is the presence of inherent insulin resistance.[4,17,18] Insulin clamp tests have revealed that women with PMOS, even those who are of normal weight, have a decreased sensitivity to insulin, which is associated with an impaired serine phosphorylation of the insulin receptor substrate-1 (IRS-1) molecule.[5,17,18] Additionally, lean patients with PMOS are prone to the accumulation of intra-abdominal fat, even in the presence of a normal BMI, with increased subcutaneous adipocyte size and decreased plasma levels of adiponectin.[8,17,19] This particular fat distribution abnormality is associated with increased levels of the visceral adiposity index (VAI) and an elevated waist-to-hip ratio, which are direct indicators of metabolic abnormalities.[19-21]

Despite these high risks, the non-obese population is often overlooked in clinical practice due to the "insidious onset" and mild symptoms.[9] Because these patients do not have the obvious metabolic clues of obesity, their underlying cardiovascular risk can remain "silent" and unchecked for years.[5] This is problematic because atherosclerosis and endothelial dysfunction begin early in the reproductive years and can progress to become fixed cardiovascular disease later in life.[5,9,22] Thus, recent clinical data have established the importance of annual cardiovascular screening for blood pressure and lipids in all lean PMOS phenotypes.[9,10]

CONVENTIONAL BIOMARKERS

High sensitivity C-reactive protein (hs-CRP) is a major marker used to reveal chronic inflammation in PMOS.[1,14] Meta-analyses have revealed that plasma levels of CRP are significantly increased in women with PMOS, compared to healthy controls, and that these differences are often maintained in non-obese women.[1,5,14] CRP is a sensitive predictor of future cardiovascular events, which indicates the low-grade inflammation that underlies subclinical atherosclerosis.[14]

Atherogenic lipid profiles, particularly an increased level of Low-Density Lipoprotein Cholesterol (LDL-C) and non-High-Density Lipoprotein Cholesterol (non-HDL-C), are common

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to all phenotypes of PMOS.[8,23] Non-HDL-C is increasingly being recognized as a better predictor of risk than LDL-C because it is a precise measure of the total amount of all pro-atherogenic lipoproteins.[8] Women with PMOS also have been found to have lower levels of large HDL particles, which impairs the anti-inflammatory and cholesterol efflux functions of the vascular system.[23]

Anti-Müllerian Hormone (AMH) has been recognized as a surrogate marker that is associated with both reproductive and metabolic severity of PMOS.[2,20] High levels of AMH in PMOS women have been found to have significant positive correlations with BMI, Waist Circumference, and Triglyceride levels, indicating that it is a marker of metabolic health.[20,22] Moreover, the Visceral Adiposity Index (VAI) is a more precise measure of the function of visceral fat than either Waist Circumference (WC) or BMI.[20,21] VAI is strongly associated with insulin resistance and is a useful marker for the identification of "metabolically unhealthy" non-obese patients.[20,21] Although helpful, these traditional markers have limitations in predicting particular long-term outcomes in lean patients, as the findings have remained inconsistent across various ethnic groups and study designs.[22,23]

DCBLD2: AN EMERGING BIOMARKER

A recent study using data from the UK Biobank has found Discoidin, CUB, and LCCL domain-containing protein 2 (DCBLD2) to be a highly specific biomarker promising candidate biomarker warranting further validation for cardiovascular risk in PMOS.[9] In a prospective cohort study of 3,864 participants, women with PMOS and high levels of DCBLD2 had a significantly increased risk of new-onset CVD (Hazard Ratio [HR] = 1.77), coronary artery disease (HR = 2.27), and myocardial infarction (HR = 2.^{08}) compared to non-PMOS women.[9] Notably, these hazard ratios were highest in young, non-obese PMOS patients, who were thus identified as a high priority population for risk management.[9]

DCBLD2 is a neuropilin-like transmembrane protein that is found on chromosome 3 and functions as a membrane receptor for the Vascular Endothelial Growth Factor (VEGF) and semaphoring.[9] Its biological function includes the role of a scaffold for intracellular signaling molecules like Epidermal Growth Factor Receptor (EGFR), Vascular Endothelial Growth Factor Receptor (VEGFR), and insulin receptors.[9,24,25] Apart from its biological functions, DCBLD2 plays a pivotal role in pathological processes like angiogenesis and Epithelial-Mesenchymal Transition (EMT).[24,25] EMT is a biological process by which epithelial cells undergo a transformation to become highly migratory mesenchymal cells, a process that has been identified as a mechanism for vascular wall remodeling and plaque progression in CVD.[24,25]

The genetic and proteomic findings suggest a strong causal link a biologically plausible association between DCBLD2 expression and the “vulnerable” cardiovascular phenotype in PMOS.[9] Proteomic analysis indicated that DCBLD2 expression is strongly disrupted in PMOS, associated with the cardiac inflammation phenotype that is independent of the traditional metabolic risk factors.[9] Moreover, phenotype analysis of the knockout mice indicates that DCBLD2 is associated with important adhesion molecules such as Integrin Subunit Beta-1 (ITGB1), modulating the focal adhesion signaling pathways that facilitate the entry of leukocytes into the vascular tissue.[25] Therefore, DCBLD2 is a unique molecule that provides a strong link between the endocrine disturbance of PMOS and the early anatomical development of atherosclerosis.[9]

CLINICAL IMPLICATIONS OF DCBLD2 IN NON-OBESE PMOS

The discovery of DCBLD2 has major clinical implications for the "vulnerable" non-obese PMOS population, who lack the typical physical characteristics of metabolic risk.[9] Since the current widely accepted therapies, such as oral contraceptives and metformin, fail to significantly reduce the incidence of CVD in this particular population, a paradigm shift towards prevention is necessary.[9] DCBLD2 can be used as a "sensitive early warning" system to alert young, thin women with advanced cardiac testing before the onset of irreversible vascular damage,[9] represents an exploratory finding with potential future clinical relevance for the non-obese PMOS population. It is important to emphasize that, at present, DCBLD2 has not been evaluated in prospective intervention trials, and no validated clinical assay exists for its routine measurement. Therefore, any clinical application remains premature and contingent on further prospective validation.

Integration of DCBLD2 into the clinical management approach could result in the establishment of gender-targeted prevention strategies. Healthcare professionals could use high expression levels of DCBLD2 as a justification for increased physical examinations and early intervention, even in asymptomatic lean patients.[9] In addition, the involvement of the protein in angiogenesis and EMT indicates its potential as a new target for therapy.[9] Manipulation of the DCBLD2-mediated signaling pathways, including its association with ITGB1 in focal adhesion, could potentially stop the progression of atherosclerosis in hyperandrogenic women.[9] Finally, DCBLD2 provides a means to personalized medicine in PMOS, ensuring that high-risk patients receive the required surveillance to mitigate the lifetime risk of cardiac disease.[9]

DISCUSSION

A critical analysis of the current literature shows that there are substantial gaps in the literature concerning the incidence of long-term CVD events in lean PMOS women.[1,4,12] Most of the current literature is hampered by small sample size, short-term follow-up, or the inability to adequately stratify between obese and non-obese groups.[1,9] Furthermore, there is substantial variability in the definition of outcomes, with some studies considering hypertension as a CVD event while others have considered only fatal outcomes, resulting in conflicting results regarding PMOS as an independent risk factor.[1,11]

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The potential of proteomics to fill these gaps is vast, as it offers a world view of protein changes that are not captured by traditional analyses.[9] Through the identification of proteins such as DCBLD2, it is possible to gain a better understanding of the distinct 'masculinized' adipose and vascular signatures of women with PMOS and how these signatures contribute to cardiac injury.[3,9] Future studies must target a multi-ethnic population to validate these new biomarkers and explore whether their pharmaceutical inhibition can finally reduce the high cardiovascular burden of PMOS.[5,9,22]

CONCLUSION

In conclusion, women with PMOS, especially the young and lean subset, are at a significantly increased risk for major cardiovascular events in the long term than was ever before suspected.[9,10] Although the traditional risk factors of obesity and insulin resistance play a role in this risk, the development of new biomarkers such as DCBLD2 makes it clear that PMOS itself confers a risk of cardiovascular disease independent of overall weight.[9,13] The lean phenotype is currently not taken into account in clinical practice because of the subtle presentation of their metabolic disease, but atherosclerosis in these women gets underway early.[5] There is a pressing need for a change in clinical practice to focus on early detection by new proteomic biomarkers and blood pressure monitoring in all women with PMOS.[9,10]

Source of Support: The author(s) received no financial support for the research, authorship, and/or publication of this article

Conflict of Interest: The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

List of abbreviations

AMH – Anti-Müllerian Hormone

BMI – Body Mass Index

CVD – Cardiovascular Disease

DCBLD2 – Discoidin, CUB, and LCCL Domain-Containing Protein 2

EGFR – Epidermal Growth Factor Receptor

EMT – Epithelial–Mesenchymal Transition

GPx – Glutathione Peroxidase

HA – Hyperandrogenism

HDL-C – High-Density Lipoprotein Cholesterol

HR – Heart Rate

hs-CRP – High-Sensitivity C-Reactive Protein

IL-6 – Interleukin-6

IR – Insulin Resistance

IRS-1 – Insulin Receptor Substrate-1

ITGB1 – Integrin Beta-1

LDL-C – Low-Density Lipoprotein Cholesterol

MDA – Malondialdehyde

NIH – National Institutes of Health

PCOM – Polycystic Ovarian Morphology

PMOS – Polycystic Ovary Syndrome

ROS – Reactive Oxygen Species

SOD – Superoxide Dismutase

TNF-alpha – Tumor Necrosis Factor-Alpha

VAI – Visceral Adiposity Index

VEGF – Vascular Endothelial Growth Factor

VEGFR – Vascular Endothelial Growth Factor Receptor

WC – Waist Circumference

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Conflict of Interest: The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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