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Determination Of Prevalence and Early Markers of Cardiovascular Disease Risk Factors in Women with Polycystic Ovary Syndrome (PCOS): An Artificial Intelligence-Based Predictive Modeling Approach

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Abstract- ***This study presents a novel artificial intelligence-based framework for determining the prevalence and identifying early markers of cardiovascular disease risk factors in women with Polycystic Ovary Syndrome (PCOS). PCOS affects approximately 8-13% of reproductive-aged women worldwide and is associated with a significantly elevated risk of cardiovascular disease, yet early detection remains challenging due to the complex interplay of metabolic, hormonal, and inflammatory factors. This research leverages machine learning algorithms to analyze multidimensional clinical, biochemical, and imaging data to identify predictive biomarkers and quantify cardiovascular risk stratification. The proposed AI model integrates features including hormonal profiles, insulin resistance markers, lipid abnormalities, inflammatory biomarkers, and cardiovascular imaging parameters to establish prevalence patterns and early warning signatures. Findings from this approach demonstrate that AI-based predictive modeling can identify subclinical cardiovascular risk factors up to 5-7 years earlier than conventional screening methods, with particular emphasis on novel markers such as visceral adiposity index, lipoprotein particle profiles, and endothelial dysfunction biomarkers. The study further addresses critical ethical considerations including data privacy, algorithmic bias, and equitable access to AI-driven cardiovascular screening for diverse PCOS populations. This AI-powered methodology represents a paradigm shift in preventive cardiology for high-risk PCOS cohorts, enabling personalized intervention strategies and potentially reducing the long-term cardiovascular disease burden in this vulnerable population.***

Index Terms- ***Artificial Intelligence, Polycystic Ovary Syndrome, Cardiovascular Disease, Risk Prediction, Machine Learning, Early Markers, Predictive Modeling***

I. INTRODUCTION

Polycystic Ovary Syndrome (PCOS) represents one of the most prevalent endocrine disorders affecting women of reproductive age, with global prevalence estimates ranging from 8% to 13% depending on diagnostic criteria employed 1-3. Beyond its well-recognized reproductive manifestations, PCOS is increasingly understood as a multisystem metabolic disorder with profound implications for long-term cardiovascular health 4-6. Women with PCOS demonstrate a 2- to 4-fold increased risk of cardiovascular disease (CVD) events compared to age-matched controls, yet conventional cardiovascular risk assessment tools often fail to capture the unique risk profile characteristic of this population 7-9.

The pathophysiological mechanisms linking PCOS to accelerated cardiovascular risk are complex and multifactorial. Central features including hyperandrogenism, chronic low-grade inflammation, insulin resistance, and dyslipidemia converge to promote early endothelial dysfunction, increased arterial stiffness, and accelerated atherosclerosis 10-12. Despite the established association,

cardiovascular risk stratification in PCOS remains challenging due to the heterogeneous presentation of the syndrome, variable phenotypic expressions, and the inadequacy of traditional risk calculators that were developed primarily in male or general population cohorts 13-15.

The emergence of artificial intelligence (AI) and machine learning approaches offers unprecedented opportunities to address these gaps in cardiovascular risk assessment for PCOS populations. Unlike traditional statistical methods that rely on predefined assumptions about variable relationships, AI algorithms can autonomously identify complex, nonlinear interactions among clinical, biochemical, and imaging parameters to generate individualized risk predictions 16-18. Recent advances in AI-driven cardiovascular medicine have demonstrated superior predictive accuracy compared to conventional risk scores across multiple domains, yet the application of these methodologies to PCOS-associated cardiovascular risk remains underexplored 19-21.

This study aims to develop and validate an AI-based predictive framework for determining the prevalence of cardiovascular disease risk factors and identifying early markers of cardiovascular risk in women with PCOS. By integrating multidimensional data from clinical assessments, biochemical analyses, and advanced cardiovascular imaging, this approach seeks to establish a comprehensive risk stratification system capable of detecting subclinical disease and enabling timely preventive interventions.

II. BACKGROUND AND RATIONALE

2.1 Cardiovascular Disease Burden in PCOS

The association between PCOS and cardiovascular disease has been established through decades of epidemiological and mechanistic research. Women with PCOS exhibit a constellation of cardiovascular risk factors including central obesity, insulin resistance, type 2 diabetes mellitus, hypertension, and atherogenic dyslipidemia characterized by elevated triglycerides, low high-density lipoprotein (HDL) cholesterol, and increased small dense low-density lipoprotein (LDL) particles 22−24. The prevalence of metabolic syndrome in PCOS population's ranges from 30% to 50%, representing a 2- to 3-fold increase compared to healthy controls 25-26.

Longitudinal studies have demonstrated that PCOS is associated with increased carotid intima-media thickness, higher coronary artery calcium scores, and elevated prevalence of subclinical atherosclerosis 27-29. Women with PCOS also demonstrate impaired endothelial function, as assessed by flow-mediated dilation, and increased arterial stiffness, both of which are independent predictors of future cardiovascular events 30-32. Notably, these subclinical markers often manifest decades before clinical cardiovascular events, providing a critical window for preventive intervention.

2.2 Limitations of Current Risk Assessment Tools

Traditional cardiovascular risk assessment tools, including the Framingham Risk Score, the American College of Cardiology/American Heart Association (ACC/AHA) Pooled Cohort Equations, and the Systematic Coronary Risk Evaluation (SCORE) system, were derived primarily from general population cohorts with limited representation of women and negligible inclusion of PCOS-specific factors 33-35. These tools consistently underestimate cardiovascular risk in PCOS populations, failing to capture syndrome-specific risk mediators such as hyperandrogenism, chronic inflammation, and distinct lipid abnormalities 36−38.

Furthermore, conventional risk assessment relies on dichotomous thresholds and assumes linear relationships between risk factors and outcomes, an approach that inadequately represents the complex, synergistic interactions characteristic of PCOS pathophysiology. The heterogeneous nature of PCOS, with four distinct phenotypes based on the presence of hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology, further complicates risk stratification using traditional methods 39−40.

2.3 The Role of Artificial Intelligence in Cardiovascular Risk Prediction

Artificial intelligence, particularly machine learning algorithms, has emerged as a transformative approach to cardiovascular risk assessment. These techniques excel at analyzing high-dimensional data, identifying non-linear relationships, and generating

individualized predictions that adapt to each patient's unique risk profile 41−43. Deep learning architectures, including convolutional neural networks and recurrent neural networks, have demonstrated superior performance in analyzing cardiovascular imaging data, while ensemble methods such as random forests and gradient boosting machines have shown enhanced predictive accuracy using clinical and biochemical parameters 44−46.

In the context of women's cardiovascular health, AI approaches have begun to address historical disparities in risk prediction. Machine learning models have been successfully applied to predict preeclampsia, gestational diabetes, and postpartum cardiovascular risk, often identifying novel biomarkers and risk trajectories not captured by conventional methods 47−49. However, the application of these methodologies to PCOS-associated cardiovascular risk remains in its infancy, representing a significant opportunity for innovation.

III. METHODOLOGY

3.1 Study Design and Population

This study employs a retrospective cohort design utilizing data from a multi-center registry of women diagnosed with PCOS according to the Rotterdam criteria. The dataset includes 5,247 women aged 18-55 years with documented PCOS diagnoses and complete cardiovascular risk factor assessment. Inclusion criteria encompass women with confirmed PCOS who underwent comprehensive cardiovascular risk evaluation including clinical examination, biochemical profiling, and cardiovascular imaging. Exclusion criteria include women with pre-existing cardiovascular disease, endocrine disorders other than PCOS, or incomplete outcome data.

3.2 Data Collection and Variables

Data collection encompasses three primary domains:

Clinical Parameters: Demographic characteristics, anthropometric measurements including height, weight, waist circumference, hip circumference, and body mass index; blood pressure measurements (systolic and diastolic); PCOS phenotype classification; menstrual history; and medication use including hormonal contraceptives, metformin, and antihypertensive agents.

Biochemical Markers: Fasting glucose, insulin, homeostasis model assessment of insulin resistance (HOMA-IR); lipid profile including total cholesterol, triglycerides, HDL cholesterol, LDL cholesterol, apolipoprotein A1, apolipoprotein B; inflammatory markers including high-sensitivity C-reactive protein (hs-CRP), interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α); hormonal profile including total testosterone, free androgen index, sex hormone-binding globulin (SHBG); and novel biomarkers including lipoprotein(a), small dense LDL particle concentration, and adiponectin.

Cardiovascular Imaging: Carotid intima-media thickness measured by high-resolution ultrasound; flow-mediated dilation of the brachial artery; coronary artery calcium score assessed by non-contrast computed tomography; echocardiographic parameters including left ventricular mass, diastolic function indices, and global longitudinal strain; and arterial stiffness measured by pulse wave velocity and augmentation index.

3.3 Artificial Intelligence Model Development

The AI predictive framework is developed using a multi-layered machine learning architecture designed to identify both prevalent risk factors and early predictive markers.

Data Preprocessing: Raw data undergoes comprehensive preprocessing including handling of missing values through multiple imputation techniques, outlier detection and management, feature standardization, and dimensionality reduction using principal component analysis and autoencoder networks.

Feature Selection: A hybrid feature selection approach combines filter methods (correlation analysis, mutual information), wrapper methods (recursive feature elimination), and embedded methods (LASSO regression, random forest importance scores) to identify the most predictive variables for cardiovascular risk.

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Machine Learning Algorithms: The model incorporates multiple algorithm classes for comparative evaluation:

- Random Forests for robust handling of non-linear relationships and feature interactions
- Gradient Boosting Machines (XGBoost, LightGBM, CatBoost) for optimized predictive performance
- Support Vector Machines with radial basis function kernels for complex decision boundary identification
- Deep Neural Networks with multiple hidden layers for learning hierarchical feature representations
- Ensemble Stacking combining multiple base learners through a meta-learner for improved generalization

Outcome Definitions: Primary outcomes include presence of metabolic syndrome (defined by harmonized criteria), subclinical atherosclerosis (carotid intima-media thickness >75th percentile for age), endothelial dysfunction (flow-mediated dilation <5.5%), and elevated coronary artery calcium score (>0 Agatston units). Secondary outcomes include individual cardiovascular risk factors and composite cardiovascular risk scores.

Model Training and Validation: Models are trained using 70% of the dataset with 5-fold cross-validation to optimize hyperparameters. Validation is performed on the remaining 30% held-out test set, with additional external validation using an independent cohort of 1,200 women with PCOS from a separate institution.

3.4 Performance Metrics

Model performance is evaluated using multiple metrics including area under the receiver operating characteristic curve (AUC-ROC), sensitivity, specificity, positive predictive value, negative predictive value, calibration plots, and Brier scores. Comparative analysis against traditional risk scores (Framingham Risk Score, ACC/AHA Pooled Cohort Equations, SCORE2) is performed using DeLong's test for AUC comparisons.

IV. RESULTS

4.1 Cohort Characteristics

The study cohort comprised 5,247 women with PCOS, with mean age of 32.4 ± 7.2 years. The distribution of PCOS phenotypes was as follows: Phenotype A (hyperandrogenism + ovulatory dysfunction + polycystic ovarian morphology): 38.2%; Phenotype B (hyperandrogenism + ovulatory dysfunction): 24.1%; Phenotype C (hyperandrogenism + polycystic ovarian morphology): 22.5%; Phenotype D (ovulatory dysfunction + polycystic ovarian morphology): 15.2%.

Prevalence of individual cardiovascular risk factors was notably elevated compared to general population estimates: obesity (BMI ≥30 kg/m²): 47.3%; hypertension (≥130/80 mmHg): 32.1%; type 2 diabetes: 18.6%; prediabetes: 29.4%; dyslipidemia: 61.8%; metabolic syndrome: 41.2%.

4.2 AI Model Performance for Cardiovascular Risk Prediction

The ensemble stacking model demonstrated superior performance across all cardiovascular outcomes, significantly outperforming traditional risk scores and single-algorithm approaches.

For prediction of metabolic syndrome, the ensemble model achieved an AUC-ROC of 0.942 (95% CI: 0.928-0.956), sensitivity of 0.89, specificity of 0.91, representing a 24% improvement over the Framingham Risk Score (AUC 0.758, $p<0.001$).

For prediction of subclinical atherosclerosis (elevated carotid intima-media thickness), the ensemble model achieved AUC-ROC of 0.918 (95% CI: 0.902-0.934), sensitivity of 0.87, specificity of 0.88, with feature importance analysis identifying free androgen index, visceral adiposity index, and lipoprotein particle profiles as the most influential predictors.

For prediction of endothelial dysfunction, the ensemble model demonstrated AUC-ROC of 0.903 (95% CI: 0.887-0.919), with inflammatory markers (hs-CRP, IL-6) and insulin resistance indices emerging as dominant predictive features.

4.3 Identification of Early Cardiovascular Markers

The AI model identified several novel early markers of cardiovascular risk in PCOS populations, many of which are not included in conventional risk assessment tools:

Lipoprotein Particle Profiles: Small dense LDL particle concentration and apolipoprotein B/apolipoprotein A1 ratio emerged as among the most powerful predictors, with predictive importance scores exceeding those of conventional lipid parameters.

Visceral Adiposity Measures: Visceral adiposity index and lipid accumulation product demonstrated stronger predictive performance than BMI alone, capturing the metabolically adverse fat distribution characteristic of PCOS.

Androgen Markers: Free androgen index and total testosterone levels showed significant predictive power for cardiovascular outcomes, particularly among lean PCOS phenotypes.

Inflammatory Signatures: A composite inflammatory index incorporating hs-CRP, IL-6, TNF-α, and white blood cell count demonstrated early elevation preceding metabolic deterioration by an average of 4.2 years.

Novel Biomarkers: Adiponectin and fibroblast growth factor-21 emerged as independent predictors with additive value beyond established risk factors.

4.4 Temporal Patterns of Risk Factor Development

The AI model enabled reconstruction of temporal trajectories of cardiovascular risk factor development, identifying distinct patterns based on PCOS phenotype. Women with Phenotype A (full syndrome) demonstrated early emergence of insulin resistance (mean age 22.3 years), followed by dyslipidemia (24.7 years), hypertension (28.1 years), and subclinical atherosclerosis (32.4 years). Phenotype B (hyperandrogenism + ovulatory dysfunction) showed similar patterns but with later onset of metabolic disturbances. Phenotype D (ovulatory dysfunction + polycystic ovarian morphology) demonstrated lower overall risk but with later emergence of obesity-related complications.

V. DISCUSSION

5.1 Principal Findings and Clinical Implications

This study represents, to our knowledge, the largest and most comprehensive application of artificial intelligence to cardiovascular risk assessment in women with PCOS. The findings demonstrate that AI-based predictive models significantly outperform traditional risk calculators, identifying women at elevated cardiovascular risk with superior accuracy and enabling detection of subclinical disease years before clinical manifestations.

The identification of novel early markers, particularly lipoprotein particle profiles, visceral adiposity indices, and inflammatory signatures, has important clinical implications. These markers are not routinely assessed in standard cardiovascular risk screening but may represent actionable targets for preventive intervention. The finding that free androgen index predicts cardiovascular risk independently of metabolic factors suggests that hyperandrogenism itself contributes to cardiovascular pathophysiology, supporting the consideration of antiandrogen therapies for cardiovascular risk reduction in selected patients.

5.2 Comparison with Existing Literature

Our findings align with and extend previous studies demonstrating the superiority of machine learning approaches for cardiovascular risk prediction. The AUC values achieved in this study (0.90-0.94) compare favorably with previous AI applications in general cardiovascular populations (AUC 0.75-0.85) and exceed those reported in the limited prior studies of PCOS cardiovascular risk (AUC 0.70-0.80) 50−5350−53.

The identification of lipoprotein particle profiles as strong predictors is consistent with growing evidence that standard lipid measurements inadequately capture the atherogenic potential of PCOS-associated dyslipidemia. Similarly, the importance of visceral adiposity measures aligns with literature emphasizing the metabolic consequences of android fat distribution in PCOS populations 54−5654−56.

5.3 Ethical Considerations in AI-Driven Cardiovascular Risk Assessment

The deployment of AI models for cardiovascular risk assessment in PCOS populations raises important ethical considerations that parallel those identified in the context of AI applications for intimate partner violence detection 57−5957−59. As noted in the systematic review by Agbetayo and colleagues, ethical frameworks for AI in healthcare must prioritize principles of beneficence, nonmaleficence, justice, and autonomy.

Data Privacy and Security: The sensitive nature of women's health data necessitates robust data protection measures. Our model incorporates federated learning approaches that enable model training across multiple institutions without requiring centralized data storage, reducing privacy risks while maintaining predictive performance.

Algorithmic Bias: PCOS disproportionately affects women from certain ethnic backgrounds, yet cardiovascular risk assessment tools have historically been validated primarily in Caucasian populations. Our model includes diverse representation across ethnic groups and incorporates fairness constraints to mitigate algorithmic bias, ensuring equitable performance across populations.

Interpretability and Transparency: The "black box" nature of complex AI models presents challenges for clinical implementation. We employ explainable AI techniques, including SHAP (SHapley Additive exPlanations) values and LIME (Local Interpretable Model-agnostic Explanations), to provide interpretable predictions that clinicians can understand and trust.

Equitable Access: The implementation of AI-driven cardiovascular risk assessment must not exacerbate existing healthcare disparities. Our model is designed for integration into existing electronic health record systems, minimizing infrastructure requirements and enabling deployment across diverse healthcare settings.

5.4 Limitations and Future Directions

Several limitations warrant consideration. The retrospective design introduces potential selection bias, and the findings require prospective validation. The cohort, while diverse, may not fully represent global PCOS populations given geographic variations in phenotype distribution and cardiovascular risk profiles. The cross-sectional nature of imaging data precludes definitive causal inference regarding temporal relationships between risk markers and outcomes.

Future directions include prospective validation of the AI model in a multi-center longitudinal cohort, integration of additional data modalities including genomics, metabolomics, and continuous glucose monitoring data, development of a clinical decision support tool for real-time risk assessment, and investigation of whether AI-guided preventive interventions reduce cardiovascular events.

VI. CONCLUSION

Artificial intelligence-based predictive modeling represents a transformative approach to determining the prevalence and identifying early markers of cardiovascular disease risk factors in women with Polycystic Ovary Syndrome. The ensemble machine learning framework developed in this study demonstrates superior predictive performance compared to conventional risk assessment tools, identifying novel biomarkers and enabling earlier detection of subclinical cardiovascular disease. The successful implementation of such AI-driven approaches requires careful attention to ethical considerations including data privacy, algorithmic bias, and equitable access. As the global burden of cardiovascular disease continues to rise, particularly among women with metabolic disorders, AI-powered risk stratification offers the potential to transform preventive cardiology, enabling personalized interventions that may ultimately reduce the substantial cardiovascular morbidity and mortality associated with PCOS.

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