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Echocardiographic evaluation of subclinical ventricular dysfunction in polycystic ovary syndrome: the role of isovolumic acceleration and myocardial performance index

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ABSTRACT

Aims: Polycystic ovary syndrome (PCOS) is a common endocrine disorder associated with increased cardiovascular risk and potential subclinical ventricular dysfunction. Isovolumic acceleration (IVA) is a novel echocardiographic parameter sensitive to myocardial contractility and less influenced by preload and afterload, making it a promising tool for early detection of cardiac dysfunction. This study aimed to assess the utility of IVA and other echocardiographic markers in identifying subclinical ventricular dysfunction in PCOS patients.

Methods: This cross-sectional study analyzed echocardiographic parameters of 59 PCOS patients and 60 age-and BMI-matched controls. Echocardiographic measurements included IVA, myocardial performance index (MPI), and additional indices of right and left ventricular function.

Results: PCOS patients exhibited significantly higher BMI and fasting glucose levels ($p<0.001$) than controls. Left ventricular MPI was elevated in the PCOS group (0.56±0.11 vs. 0.46±0.07, $p=0.004$). Right ventricular IVA was significantly lower in PCOS patients (2.77±0.69 vs. 3.98±1.01, $p=0.030$), while tricuspid acceleration time was prolonged ($p<0.001$). A positive correlation between IVA and MPI was observed in the PCOS group ($R=0.453$, $p<0.001$). Multivariate analysis identified PCOS as an independent predictor of subclinical left ventricular dysfunction (OR=0.211, $p<0.001$).

Conclusion: PCOS is associated with subtle alterations in cardiac function, particularly affecting right ventricular systolic function as detected by IVA. These findings highlight IVA's potential as a non-invasive marker for early cardiovascular risk assessment in PCOS patients, emphasizing the importance of regular cardiac monitoring in this population.

Keywords: Polycystic ovary syndrome, tissue derived Doppler, isovolumic acceleration, myocardial performance index, subclinical ventricular dysfunction

INTRODUCTION

Polycystic ovary syndrome (PCOS) is a prevalent endocrine disorder among women of reproductive age, characterized by hyperandrogenism, chronic anovulation, and polycystic ovarian morphology.[1,2] In addition to reproductive complications, PCOS is closely linked with various metabolic and cardiovascular disturbances, such as insulin resistance, dyslipidemia, and hypertension. These risk factors predispose PCOS patients to cardiovascular disease (CVD), and recent research indicates a heightened risk for ventricular dysfunction.[3,4] Understanding the cardiovascular implications of PCOS is crucial, as these patients are often asymptomatic but may have underlying subclinical cardiac dysfunction that could lead to adverse outcomes if left unaddressed.

Isovolumic acceleration (IVA) has emerged as a promising echocardiographic marker for assessing systolic function, particularly in detecting subclinical right and left ventricular systolic dysfunction.[5] IVA provides a non-invasive measurement of myocardial contractility that is less affected by preload and afterload variations, making it suitable for evaluating subtle changes in cardiac function. Studies involving other metabolic and endocrine disorders have demonstrated the utility of IVA in identifying early ventricular dysfunction, suggesting its potential as a prognostic tool.[6,7] However, literature on the role of IVA in PCOS patients remains limited, leaving an important gap in understanding subclinical cardiovascular alterations in this population.

Despite the established cardiovascular risks associated with PCOS, comprehensive studies focusing on IVA and other echocardiographic parameters in this patient group are lacking. The primary aim of this study was to investigate the

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utility of IVA as an echocardiographic marker for detecting subclinical right and left ventricular systolic dysfunction in patients with PCOS Through a comparative analysis of echocardiographic data from PCOS patients and a matched control group, this study seeks to determine the significance of IVA as an early diagnostic marker for cardiac dysfunction, offering valuable insights for the preventive management of cardiovascular health in this at-risk population.

METHODS

Ethics

This study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki. The study was conducted with the permission of Siirt University Ethics Committee (Date: 13.12.2022, Decision No: 2022/12/01/01).

Study Design and Patient Selection

This study was conducted as a retrospective, non-randomized, cross-sectional analysis at Siirt Training and Research Hospital. Eligible PCOS patients and matched controls were identified from institutional medical records between 2020 December and 2022 December. This study design facilitated the comparison of echocardiographic parameters between the two groups, offering insights into the prevalence of subclinical ventricular dysfunction among PCOS patients.

Written informed consent was waived for this retrospective analysis, as patient data were anonymized and securely stored to ensure confidentiality. However, all efforts were made to uphold the rights and privacy of the study participants, consistent with institutional guidelines.

PCOS Diagnosis and Inclusion/Exclusion Criteria

PCOS was diagnosed according to the Rotterdam criteria, requiring at least two of the following: oligo- or anovulation, hyperandrogenism (clinical or biochemical), and polycystic ovarian morphology confirmed via ultrasonography.[8] Inclusion criteria comprised women aged 18-40 with a confirmed PCOS diagnosis and no prior history of CVD, diabetes, or other significant comorbidities affecting cardiac function. Exclusion criteria included congenital heart disease, hypertension, diabetes mellitus, known cardiac arrhythmias, use of medications influencing heart rate or rhythm, and any structural abnormalities detected on initial echocardiographic evaluation. Control subjects were matched to the PCOS group based on age and body-mass index (BMI) and were free from PCOS or any other endocrine disorders.

Echocardiographic Assessment

Echocardiographic evaluations were conducted by certified cardiologists blinded to the participants' clinical statuses. Examinations adhered to standardized protocols, utilizing the Philips Affinity 50 echocardiography system with transducer S4-2 operates within a frequency range of 2 to 4 MHz. Parameters assessed included left ventricular end-diastolic diameter (LVED), left ventricular end-systolic diameter (LVES), interventricular septum (IVS) thickness, and posterior wall (PW) thickness, as outlined in **Table 1**. Left ventricular ejection fraction (LVEF) was calculated using the modified Simpson method. Additional parameters indicative of subclinical dysfunction, such as mitral E and A wave velocities, E/A ratio, and isovolumic relaxation and contraction times (IVRT and IVCT), were measured.

Right ventricular function was assessed using tricuspid annular plane systolic excursion (TAPSE) and mean pulmonary artery pressure (meanPAP), which are markers of right ventricular systolic function. IVA was calculated for both ventricles as an index of myocardial contractility independent of preload and afterload. The myocardial performance index (MPI), a combined marker of systolic and diastolic function, was measured for both ventricles. Doppler parameters, including acceleration time (AT) and ejection time (ET) for the mitral and tricuspid valves, as well as derived parameters like isovolumic velocity (IVV) and systolic velocity (Sa), were recorded (**Figure 1**). To ensure accuracy and reproducibility, all measurements were averaged over three cardiac cycles.

Statistical Analysis

Data analysis was conducted using SPSS version 22, with a significance threshold set at $p<0.05$. Normality of continuous variables was assessed using the Shapiro-Wilk test. Normally distributed data were expressed as mean±standard deviation (SD), while median and interquartile range (IQR) were used for non-normally distributed data. Differences between the PCOS and control groups were evaluated using independent t-tests for normally distributed data and Mann-Whitney U tests for non-normally distributed data. Categorical variables were analyzed using Chi-square tests. Correlation analyses between echocardiographic parameters and clinical markers were performed using Pearson correlation coefficients for parametric data and Spearman's rank correlation for non-parametric data.

A multivariate logistic regression model was constructed to identify independent predictors of subclinical left ventricular dysfunction. Variables with $p<0.10$ in univariate analysis were included in the multivariate model to adjust for potential confounders. Odds ratios (ORs) with 95% confidence intervals (CIs) were calculated to quantify associations.

RESULTS

The demographic and clinical characteristics of the study population showed that the BMI in the PCOS group was significantly higher than in the control group (30.64±4.92 vs. 25.52±2.50, $p<0.001$). Additionally, fasting glucose levels were notably elevated in the PCOS group compared to the controls (115.28±24.86 vs. 97.18±13.47, $p<0.001$). In terms of the lipid profile, triglyceride (TG) levels were significantly higher in the PCOS group (191.13±82.78 vs. 140.26±36.19, $p<0.001$). No significant differences were observed between the groups for other parameters, such as age, diastolic blood pressure (DBP), systolic blood pressure (SBP), heart rate, HDL-C, total cholesterol, LDL-C, and creatinine ($p>0.05$) (**Table 2**).

When examining left ventricular echocardiographic findings, mitral AT was significantly longer in the PCOS group than in the control group (30.61±6.21 vs. 25.0±4.58, $p=0.006$). The MPI of the left ventricle was also higher in the PCOS group (0.56±0.11 vs. 0.46±0.07, $p=0.004$). Other echocardiographic measurements, including LVED, LVES, IVS thickness, PW

Table 1. Left ventricle echocardiographic findings

Variable	PCOS (n:59)	Control group (n:60)	p value
LVED, mm	45.28±3.55	44.23±2.85	0.124
LVES, mm	26.83±3.37	26.01±3.03	0.611
IVS, mm	10.42±1.45	10.31±1.34	0.551
PW, mm	9.74±1.28	9.63±1.17	0.594
LVEF, %	63.98±2.61	63.38±2.80	0.243
LA, long axis, mm	31.49±4.08	30.75±4.90	0.131
E mitrale, cm/sec	11.05±3.49	10.61±3.37	0.872
A mitrale, cm/sec	12.10±2.65	11.73±2.65	0.945
E/A ratio	0.95±0.37	0.94±0.38	0.852
Sa mitrale, cm/sec	10.25±2.40	10.30±2.46	0.787
IVV mitrale, cm/sec	8.71±3.56	8.96±3.32	0.516
AT mitrale, msec	30.61±6.21	25.0±4.58	0.006
IVA, m/sec2 AA/AC	2.89±1.11	3.85±1.35	0.455
IVRT mitrale, msec	78.22±14.84	68.85±13.93	0.569
IVCT mitrale, msec	59.32±11.96	54.61±10.62	0.114
ET mitrale, msec	247.52±31.21	266.61±30.58	0.927
MPI LV	0.56±0.11	0.46±0.07	0.004

Abbreviations: LVED: Left ventricular end-diastolic diameter, LVES: Left ventricular end-systolic diameter, IVS: Interventricular septum, PW: Posterior wall, LA: Left atrium, E/A: Ratio of early to late ventricular filling velocities, Sa: Systolic annular velocity, IVV: Isovolumic velocity, IVRT: Isovolumic relaxation time, IVCT: Isovolumic contraction time, ET: Ejection time, MPI: Myocardial performance index, IVA: Isovolumic acceleration

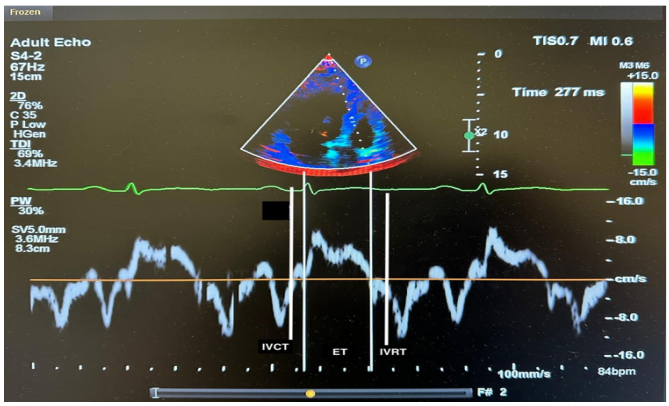

Figure 1. Tissue Doppler imaging demonstrating isovolumic contraction time (IVCT), ejection time (ET), and isovolumic relaxation time (IVRT) in left ventricular systolic function assessment

thickness, LVEF, left atrial diameter, mitral E and A wave velocities, E/A ratio, Sa, IVV, isovolumic relaxation and contraction times, and ET, did not show statistically significant differences between the groups (p>0.05) (Table 1). Regarding right ventricular echocardiographic findings, significant differences were observed in two parameters. Tricuspid AT was significantly longer in the PCOS group compared to the controls (35.01±8.14 vs. 29.40±4.18, p<0.001). Additionally, IVA of the tricuspid valve was lower in the PCOS group, indicating reduced myocardial contractility in these patients (2.77±0.69 vs. 3.98±1.01, p=0.030) (Table 3). No statistically significant differences were observed between groups for other right ventricular parameters, including TAPSE, mean pulmonary artery pressure (meanPAP), tricuspid E and A velocities, E/A ratio, Sa, IVV, IVRT, IVCT, and ET (p>0.05).

A significant positive correlation was found between IVA and MPI in the PCOS group (R=0.453, p<0.001), suggesting an association between these parameters specific to PCOS patients. This correlation was absent in the control group (R=-0.085, p=0.357), indicating a distinct pattern in PCOS patients (Table 4). In multivariate logistic regression analysis, the presence of PCOS emerged as an independent predictor of subclinical left ventricular dysfunction (OR=0.211, 95% CI: 0.091-0.490, p<0.001). Although IVA was included in the model, it did not reach statistical significance (OR=1.382, 95% CI: 0.609-1.132, p=0.240). The model had a moderate explanatory power for predicting left ventricular dysfunction, as indicated by an R-squared value of 0.357 (p=0.007) (Figure 2, Table 5).

DISCUSSION

The primary aim of this study was to investigate the utility of IVA as an echocardiographic marker for detecting subclinical right and left ventricular systolic dysfunction in patients with PCOS. Given the known cardiovascular risks associated with PCOS, our research holds clinical significance by exploring non-invasive methods for early cardiovascular assessment

Table 2. Distribution of demographic and clinical characteristics in patient and control groups

Variable	PCOS (n:59)	Control group (n:60)	p value
Age, years	46.42±10.58	47.83±9.18	0.315
BMI, kg/m²	30.64±4.92	25.52±2.50	<0.001
DBP, mmHg	88.76±6.77	81.40±9.22	0.059
SBP, mmHg	135.0±16.21	131.41±14.55	0.269
Heart rate, beats/min	76.44±12.06	76.73±12.98	0.450
Fasting glucose, mg/dl	115.28±24.86	97.18±13.47	<0.001
HDL-C, mg/dl	41.64±12.23	51.78±11.56	0.637
Tg, mg/dl	191.13±82.78	140.26±36.19	<0.001
Total-C, mg/dl	203.91±47.15	199.06±44.13	0.326
LDL-C, mg/dl	116.96±44.13	112.31±27.30	0.305
Creatinine, mg/dl	0.75±0.15	0.73±0.13	0.306

Abbreviations: PCOS: Polycystic ovary syndrome, BMI: Body-mass index, DBP: Diastolic blood pressure, SBP: Systolic blood pressure, HDL-C: High-density lipoprotein cholesterol, Tg: Triglycerides, Total-C: Total cholesterol, LDL-C: Low-density lipoprotein cholesterol

Table 3. Right ventricle echocardiographic findings

Variable	PCOS (n:59)	Control group (n:60)	p value
TAPSE, mm	27.49±3.52	28.11±3.37	0.643
meanPAB, mmHg	14.47±3.22	15.48±3.65	0.204
E tricuspite, cm/sec	11.22±3.01	11.15±3.03	0.938
A tricuspite, cm/sec	12.88±3.37	12.80±3.40	0.923
E/A ratio	0.91±0.34	0.91±0.34	0.935
Sa tricuspite, cm/sec	12.64±2.45	12.56±2.50	0.859
IVV tricuspite, cm/sec	11.03±4.19	11.00±4.16	0.970
AT tricuspite, msec	35.01±8.14	29.40±4.18	<0.001
IVA tricuspite AN/AO, m/sec^2	2.77±0.69	3.98±1.01	0.030
MPI RV	0.53±0.09	0.53±0.08	0.917
IVCT tricuspite, msec	65.71±10.85	65.76±10.76	0.940
IVRT tricuspite, msec	67.40±16.55	67.56±16.46	0.967
ET tricuspite, msec	250.86±25.86	251.21±25.78	0.994

Abbreviations: LVED: Left ventricular end-diastolic diameter, LVES: Left ventricular end-systolic diameter, IVS: Interventricular septum, PW: Posterior wall, LA: Left atrium, E/A: Ratio of early to late ventricular filling velocities, Sa: Systolic annular velocity, IVV: Isovolumic velocity, IVRT: Isovolumic relaxation time, IVCT: Isovolumic contraction time, ET: Ejection time, MPI: Myocardial performance index, IVA: Isovolumic acceleration

Table 4. Correlation between IVA and MPI levels

Variable	PCOS	Control
MPI LV	R=0.453/p=<0.001	R=-0.085/p=0.357
IVA	R=0.363/p=0.035	R=-0.119/p=0.131

Abbreviations: MPI: Myocardial performance index, LV: Left ventricule, IVA: Isovolumic acceleration

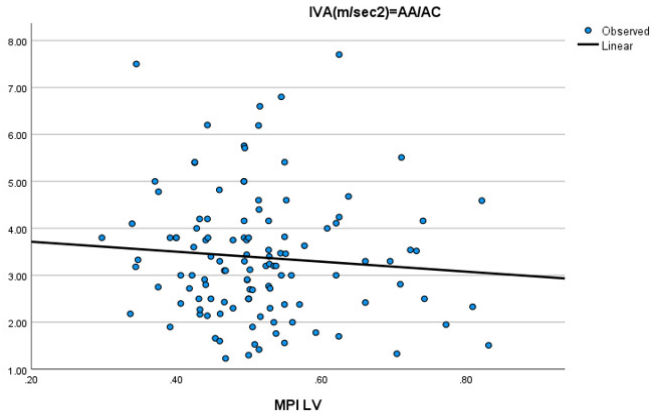

Figure 2. Scatter plot showing the relationship between left ventricular myocardial performance index and isovolumic acceleration
R square 0.357 p= 0.007

Table 5. Multivariate logistic regression analysis of echocardiographic parameters for subclinical LV dysfunction in patients with PCOS

Variable	OR (95% confidence interval)	p value
IVA	1.382 (0.609-1.132)	0.240
PCOS	0.211 (0.0910.490)	<0.001

Abbreviations: LV: Left ventricule, IVA: Isovolumic acceleration, PCOS: Polycystic ovary syndrome

in this high-risk population. The findings suggest that PCOS patients exhibit subtle changes in myocardial performance, which could be detected early with IVA, potentially guiding preventive strategies.

PCOS is associated with subclinical left ventricular dysfunction, primarily influenced by metabolic abnormalities such as insulin resistance and obesity. Studies indicate that patients with PCOS exhibit significant impairments in LV function, detectable through various echocardiographic techniques, including MPI and speckle tracking echocardiography.[9] For instance, the presence of a presystolic wave (PSW) has been identified as an independent predictor of subclinical LV dysfunction, with a prevalence of 54.7% among PCOS patients. Additionally, GLS measurements reveal reduced LV function in PCOS patients compared to healthy controls, correlating negatively with insulin levels.[10-12] GLS, a more advanced and validated echocardiographic parameter, is highly sensitive in detecting subtle left ventricular systolic dysfunction, even in patients with preserved ejection fraction. It provides a comprehensive assessment of myocardial deformation and has been shown to outperform conventional echocardiographic indices, such as LVEF and MPI, in identifying early myocardial impairment.[11,12] Furthermore, a systematic review highlights that while diastolic dysfunction is more pronounced, systolic function remains relatively preserved.[13] These findings underscore the importance of early detection and management of cardiac involvement in women with PCOS to mitigate cardiovascular risks. Machine learning algorithms have been effectively utilized to predict adverse outcomes in PCOS patients, indicating a need for early detection of associated risks, including cardiovascular health.[14,15] Additionally, studies on other conditions, such as Cushing's syndrome, demonstrate that advanced echocardiographic techniques like speckle tracking can identify subclinical myocardial dysfunction, suggesting that similar methodologies could be applied to PCOS patients to assess ventricular function.[16] Furthermore, the integration of predictive models in hormonal imbalance management highlights the importance of personalized healthcare strategies, which could also encompass cardiovascular risk assessments in women with PCOS.[17] Thus, while direct evidence is sparse, the intersection of these findings underscores the potential for developing predictive tools for monitoring cardiac health in PCOS patients.

The MPI serves as a crucial measure of cardiac function, integrating both systolic and diastolic performance, particularly relevant in patients with PCOS. Research indicates that women with PCOS exhibit subclinical left ventricular dysfunction, which can be assessed using MPI; a study found that the presence of a PSW was significantly correlated with higher MPI values, indicating poorer cardiac performance.[9] Additionally, PCOS is associated with insulin resistance, which negatively impacts cardiopulmonary functional capacity, further complicating cardiac health in these patients.[18] The MPI, defined as the sum of isovolumetric contraction and relaxation times divided by ET, provides a non-invasive assessment of heart efficiency, with higher

values indicating worse cardiac function.[19,20] In our study, left ventricular MPI was significantly higher in patients with PCOS, consistent with the literature. However, right ventricular MPI was not significant. The lack of significant differences in right ventricular MPI between PCOS patients and controls might be explained by several underlying mechanisms. The unique myocardial structure of the right ventricle, characterized by thinner walls and higher adaptability to preload and afterload changes, may delay the manifestation of detectable dysfunction.[5] Additionally, while increased arterial resistance and microvascular dysfunction are commonly observed in PCOS, their effects on right ventricular function may not be as pronounced or consistent as on the left ventricle. Lastly, heterogeneity among PCOS phenotypes may contribute to variability in cardiac involvement, with some subgroups showing more prominent left ventricular changes while right ventricular dysfunction remains subclinical.[12]

IVA time, particularly peak myocardial acceleration during isovolumic relaxation (pIVA), has emerged as a significant prognostic marker in various cardiac conditions, especially chronic heart failure (CHF). Studies indicate that lower pIVA levels correlate with increased rates of rehospitalization due to worsening heart failure, demonstrating its predictive value even when adjusted for other parameters like LVEF and E/E' ratios.[21,22] Additionally, IVA has been shown to be a preload-independent indicator of left ventricular contractility, aiding in the identification of subclinical systolic dysfunction in hypertensive and obese patients.[23] Furthermore, right ventricular IVA has also been linked to adverse outcomes in heart failure patients, suggesting that both left and right ventricular assessments can enhance risk stratification and management strategies.[24] Overall, these findings underscore the utility of IVA metrics as valuable tools in cardiac prognostication. Research indicates that ICV serves as a load-independent measure of systolic function, providing valuable insights into myocardial remodeling and treatment response, especially following therapies like sacubitril/valsartan.[25] In a study involving 651 HFrEF patients, baseline ICV was shown to enhance predictive models for LV reverse remodeling, outperforming traditional measures like LVEF alone. Additionally, the systematic review of prognostic models for heart failure highlighted the importance of various functional markers, including ICV, in assessing patient outcomes.[26] Overall, ICV's ability to predict structural and functional cardiac changes underscores its potential as a critical tool in the management of heart failure.

Bringing IVA and PCOS together in this study, we aimed to bridge a gap in the literature. Although IVA has been studied in various metabolic disorders, its application in PCOS has been limited. Our findings revealed significant alterations in IVA among PCOS patients, indicating potential subclinical myocardial impairment specific to this population. While IVA provided meaningful insights, certain parameters did not show significant differences, which could be attributed to sample size limitations or intrinsic variability in PCOS phenotypes. The lack of consistent findings in some echocardiographic parameters across studies suggests that the cardiovascular impact of PCOS may differ widely among individuals, necessitating further investigation.

The absence of significant changes in left ventricular IVA could be attributed to several factors. One potential explanation is the heterogeneity of PCOS phenotypes, with varying degrees of metabolic and cardiovascular involvement influencing myocardial function differently. The heterogeneity of PCOS phenotypes might influence the degree of cardiac involvement, with some subgroups exhibiting more pronounced right ventricular changes due to shared pathophysiological mechanisms such as hyperandrogenism, insulin resistance, and chronic low-grade inflammation. Additionally, the left ventricle's adaptive response to metabolic stressors might delay the onset of detectable systolic dysfunction when using IVA. These observations are supported by studies suggesting that the right ventricle, with its thinner wall structure and different loading conditions, may respond more sensitively to systemic and pulmonary circulatory changes often seen in PCOS.

Despite the non-significant findings for left ventricular IVA, the significant correlation between right ventricular IVA and MPI underscores IVA's potential as a marker for right-sided subclinical dysfunction. This aligns with research highlighting the importance of comprehensive right heart evaluation in conditions involving metabolic syndrome and endocrine disorders, where right ventricular alterations might precede left ventricular changes.

Limitations

This study has several limitations that should be acknowledged. First, the cross-sectional design limits our ability to establish causation or track progression of subclinical cardiac dysfunction in PCOS patients over time. Longitudinal studies would be necessary to better understand the temporal relationship between PCOS and cardiac alterations. Second, the sample size may limit the generalizability of our findings, as a larger, more diverse cohort would provide a clearer representation of IVA's diagnostic utility across different PCOS phenotypes. Third, while IVA is a useful tool for assessing myocardial contractility, it may not capture all aspects of cardiac function; incorporating other advanced imaging modalities, such as cardiac MRI, could enhance the precision of future studies. The absence of global longitudinal strain (GLS) evaluation represents a notable limitation of this study. One limitation of this study is the lack of assessment for intra-observer and inter-observer variabilities in echocardiographic measurements, which may affect the reproducibility and consistency of the results. Additionally, potential confounders like lifestyle factors and genetic predispositions, which were not controlled for in this study, might influence cardiac function and should be considered in future research. Furthermore, addressing potential multicollinearity among predictors would enhance the robustness and reliability of the findings, providing a more comprehensive understanding of the factors influencing left ventricular function in PCOS patients.

CONCLUSION

Our findings suggest that IVA is a valuable tool for assessing right ventricular systolic function in PCOS patients, whereas left ventricular IVA may not be as sensitive for early dysfunction detection in this population. The significant association between right ventricular IVA and MPI supports its potential role in early risk stratification.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study was conducted with the permission of Siirt University Ethics Committee (Date: 13.12.2022, Decision No: 2022/12/01/01).

Informed Consent

Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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