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Dr. Aishhwarrya Umeshchandara G
Senior Resident, Department of Endocrinology, St John's Medical College, Bangalore, Karnataka, India

Dr. Sonali Appaiah
Associate Professor, Department of Endocrinology, St John's Medical College, Bangalore, Karnataka, India

Dr. Vageesh Ayyar
Professor, Department of Endocrinology, St John's Medical College, Bangalore, Karnataka, India

Dr. Ganapathi Bantwal
Professor, Department of Endocrinology, St John's Medical College, Karnataka, India

Dr. Belinda George
Professor and Head of Department of Endocrinology, St John's Medical College, Bangalore, Karnataka, India

Corresponding Author:
Dr. Aishhwarrya Umeshchandara G
Senior Resident, Department of Endocrinology, St John's Medical College, Bangalore, Karnataka, India

Comparative analysis of cardiac autonomic function in women with and without PCOS

Aishhwarrya Umeshchandara G, Sonali Appaiah, Vageesh Ayyar, Ganapathi Bantwal and Belinda George

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Abstract
Introduction: Polycystic ovary syndrome (PCOS) is characterized by hyperandrogenism, anovulation, and metabolic dysfunction, with adiposity and insulin resistance contributing to long-term cardiometabolic risk. Autonomic imbalance has been proposed as an additional mechanistic link, but data remains inconclusive. Given the ethnic variation in adiposity distribution and autonomic adaptation, evaluating autonomic function in early, untreated PCOS is clinically relevant.
Aim: To compare cardiac autonomic function between treatment-naïve women with PCOS and healthy controls using standardized autonomic testing, and to examine associations between autonomic indices, adiposity, metabolic parameters, and serum testosterone.
Methodology: This cross-sectional study included 30 women with PCOS and 30 age-matched controls. Anthropometry, body fat percentage, lipid profile, and serum testosterone were assessed. Cardiac autonomic function was evaluated using CANWin® implementing Ewing's protocol (30:15 ratio, Valsalva ratio, deep-breathing E:I ratio) and HRV spectral measures (Low frequency power (LF), high frequency power(HF) and ratio LF/HF).
Results: PCOS participants exhibited significantly higher BMI, waist-hip ratio(WHR), and body fat percentage (all $p<0.001$). However, autonomic function indices, including 30:15 ratio, Valsalva ratio, E:I ratio, LF, HF, and LF/HF, were comparable between groups (all $p>0.05$). Within the PCOS group, serum testosterone showed a positive association with HF power ($r = 0.40$, $p = 0.031$), while LF/HF ratio correlated negatively with HDL cholesterol ($r = -0.39$, $p = 0.02$). No significant correlations were found with BMI, WHR and body fat.
Conclusion: Despite greater adiposity and metabolic derangements, treatment-naïve Indian women with PCOS demonstrated preserved cardiac autonomic function. The positive association of testosterone with vagal indices and the HDL-LF/HF relationship suggest early compensatory autonomic modulation rather than overt sympathovagal imbalance. These findings support the possibility that autonomic dysfunction may emerge later in the disease course or differ across ethnic phenotypes.

Keywords: PCOS, Autonomic dysfunction, testosterone

Introduction

Polycystic ovary syndrome (PCOS) is the most common endocrine disorder among women of reproductive age, characterized by persistent anovulation, hyperandrogenism, and polycystic ovarian morphology. It is frequently associated with metabolic disturbances, including dyslipidemia, insulin resistance, and obesity, all of which may compound risks for cardiovascular disease later in life. Recent advances have underscored the importance of the autonomic nervous system (ANS) in regulating metabolic and reproductive function. The ANS, comprising the sympathetic and parasympathetic branches, is pivotal in maintaining homeostasis and is increasingly recognized for its role in the pathophysiology of PCOS [1]. Clinical and experimental studies have demonstrated that women with PCOS frequently display autonomic dysfunction, specifically increased sympathetic activity and decreased vagal tone, measurable by tools such as heart rate variability assessments [2]. These alterations not only influence hormonal regulation but also may predispose to higher cardiometabolic risk seen in this patient group. Besides autonomic dysregulation, body composition plays a crucial role in the heterogeneity of PCOS clinical manifestations.

Comparative studies examining both autonomic function and precise body composition parameters (using methodologies such as bioelectrical impedance or DEXA) between PCOS cohorts and normal females are essential for elucidating the complex interplay of metabolic,

hormonal, and neuroregulatory factors [3, 4, 5]. Clarifying these relationships may inform early identification of at-risk groups and the development of targeted therapeutic strategies.

Briefing about Autonomic Function Tests

Autonomic function in study participants was assessed using a comprehensive protocol comprising both cardiovascular reflex and heart rate variability (HRV) analyses. These methods offer objective quantification of sympathetic and parasympathetic regulation, crucial for understanding autonomic alterations in PCOS.

Heart Rate Variability (HRV) Analysis [6, 7, 8]

HRV quantifies beat-to-beat variations in cardiac rhythm. Evaluation was conducted in both time and frequency domains:

Low Frequency (LF) Power (0.04-0.15 Hz): Indicates combined sympathetic and parasympathetic modulation, with sympathetic predominance in normalized units.

High Frequency (HF) Power (0.15-0.40 Hz): Primarily reflects parasympathetic (vagal) activity.

LF/HF Ratio: Represents sympathovagal balance; higher ratios suggest relative sympathetic dominance.

SDNN (Standard Deviation of Normal-to-Normal Intervals): Measures overall HRV and global autonomic activity.

RMSSD (Root Mean Square of Successive Differences): Sensitive marker of short-term vagal (parasympathetic) activity.

NN50 and pNN50: Number and proportion (%) of successive RR intervals differing by more than 50 ms, also indices of parasympathetic tone.

Deep Breathing Test: Evaluates cardiovagal function by determining heart rate changes during paced deep breathing. The expiration-inspiration (E:I) ratio is the ratio of longest RR interval during expiration to the shortest RR interval during inspiration averaged over 6 cycles of respiration.

Valsalva Maneuver: Assesses both sympathetic and parasympathetic branches through heart rate and blood pressure responses to forced expiration against a closed airway. The Valsalva ratio helps detect abnormal autonomic reflexes.

Response to Standing (Orthostatic Test): Measures changes in heart rate and blood pressure when rising from supine to standing. The ratio of longest RR interval at 30th beat to shortest RR interval at 15th beat (30:15 ratio) and the magnitude and recovery speed of blood pressure indicate both baroreflex function and autonomic control of cardiovascular adaptation to posture.

BP Response to Standing: On standing, a healthy response involves a minor drop in systolic and diastolic pressures, quickly restored by baroreceptor-mediated sympathetic activation. Persistent or exaggerated drops may indicate autonomic insufficiency.

Handgrip Test: Assesses sympathetic function by recording the rise in diastolic blood pressure during sustained handgrip contraction. A normal response is an increase of ≥16 mmHg; smaller increments suggest impaired sympathetic vasoconstriction.

Collectively, these tests enable robust characterization of both branches of the autonomic nervous system and their perturbations in PCOS.

Methodology

The research was carried out in the Department of Endocrinology, St. John's Medical College and Hospital, Bangalore with IEC clearance. Patients attending endocrinology OPD, meeting the inclusion criteria, were recruited in the study. After informed consent, participants were subjected to history taking and clinical examination. Relevant investigation were done. Normal healthy individuals were recruited as control group.

Aim: Of the study was to compare autonomic function in PCOS women and normal females and also to compare body composition in PCOS and normal females.

Women >18 years, fulfilling criteria for PCOS as per Rotterdam criteria. Newly diagnosed PCOS or diagnosed PCOS but off treatment for ≥ 3 months were included in the study. The controls were age matched.

Women previously diagnosed as PCOS and on treatment or those with severe virilization or history of drug intake (Phenytoin, valproate, antipsychotics and other drugs causing weight change) were excluded from the study.

After obtaining preliminary data from cases and controls like age, blood pressure, weight, height, clinical signs of androgenisation (acne, hirsutism), waist circumference, hip circumference, waist hip ratio (WHR), BMI, participants were subjected to a series of autonomic function test and body composition analysis (using a bio-impedance analysis).

Waist circumference was measured as the circumference of the abdomen midway between the highest point of the iliac crest and the bottom of ribcage. Hip circumference was measured widest circumference over the greater trochanters. The subject's height was measured to the nearest millimetre by a wall-mounted stadiometer and weight was measured with a weighing machine to the nearest kg.

Fasting blood samples were drawn in follicular phase (cycle days 2-8) in patients with a cycle length shorter than 3 months. Patients with cycle length >3 months had the blood samples drawn on a random cycle day.

Baseline serum testosterone levels, fasting blood glucose, plasma insulin(HOMA-IR) HbA1C, fasting lipid profile and other investigations as per standard of care was done in cases only.

Autonomic function tests included: Blood-pressure response to standing, Blood-pressure response to sustained handgrip, immediate heart-rate response to standing, heart-rate response to valsalva manoeuvre, heart-rate (R-R interval) variation during deep breathing.

Heart rate variability (HRV) analysis were performed using CANwin technology based on EWING's protocol.

Recording of HRV: Short-term HRV recording was performed using lead II electrocardiogram (ECG), following the standard procedure as per the recommendation of Task Force. The data acquisition was performed using a diabetes risk analyser (CANwin) data acquisition system which used TachoCardioGram (TCG) to conduct a battery of six tests and a Windows based CANWin software.

Raw ECG wasfiltered using a band pass filter (2-40 Hz). HRV analysis of the RR tachogram was performed for frequency

domain (by power spectral analysis using fast Fourier transformation) and time domain measures using the software.
The frequency domain indices include low frequency (LF; 0.04-0.15 Hz), high frequency (HF; 0.15-0.4 Hz), total power (TP), LF in normalized units (LFnu), HF in normalized units (HFnu) and the ratio of LF to HF (LF-HF ratio).

Recording of other CAFT (Cardiac- autonomic function test)
The CAFT included HR and BP response to 5 min of active standing, HR response to deep breathing at the rate of 6 breaths/min with inspiratory and expiratory cycles for 5 s each, and DBP response to isometric hand grip (IHG) at one-third of maximum voluntary capacity for 3 min.
Body composition analysis was done using bio-impedance with specific reference to fat mass percentage in PCOS and control cohort.

Comparison was made between the cases and control
Statistical analysis was performed using SPSS version 22.0. Data entry was completed in Microsoft Excel and all statistical tests were two-tailed. Continuous variables with normal distribution are presented as mean ± standard deviation, while non-normally distributed variables are described as median (interquartile range). Categorical variables are expressed as frequencies (percentages). The independent sample t-test was used for group comparison of continuous variables with normal distribution, while the Mann-Whitney U test was applied for skewed data. Proportions were compared using Chi-square test or Fisher's exact test, as appropriate. Correlation between continuous variables was estimated using Pearson's correlation coefficient. All statistical tests were interpreted at the 0.05 significance level.

Results
The study included 30 women with polycystic ovary syndrome (PCOS) and 30 age-matched controls. The groups were comparable in age (27.7±4.9 years vs 27.8±4.5 years).
Anthropometric assessment revealed that PCOS cases exhibited significantly higher adiposity: mean BMI was 31.9±7.4 kg/m² compared to 24.7±5.3 kg/m² in controls. Similarly, waist circumference (97.7±12.3 cm vs 77.4±11.0 cm), hip circumference (109.4±11.9 cm vs 94.8±9.8 cm), waist-hip ratio (0.88±0.06 vs 0.81±0.06), and body fat percentage (40.9%±4.6% vs 33.3%±6.6%) were significantly higher in cases.
Biochemical analysis within the PCOS cohort revealed the following mean (±SD) values: Total cholesterol: 177.77±27.43 mg/dL, HDL cholesterol: 33.53±9.46 mg/dL (notably low), LDL cholesterol: 119.67±23.32 mg/dL, Triglycerides: 156.60±105.89 mg/dL (moderately elevated with wide variability), Serum testosterone: 0.46±0.21 ng/mL (confirming biochemical hyperandrogenism) The mean duration since onset of irregular menstrual cycles was 20.17±44.48 months.
Cardiac autonomic function parameters, evaluated using heart rate variability (HRV) indices such as the HRV to standing 30:15 ratio, HRV to Valsalva maneuver, HRV during deep breathing (E:I ratio), and normalized low-frequency (LF) and high-frequency (HF) power, did not show significant differences between PCOS cases and controls on application of independent sample t- test and mann-Whitney U test (Table 1).

Among All Subjects (n = 60): CAN Parameters, no significant correlations were observed between any CAN/HRV parameter and BMI, body fat percentage, or waist-hip ratio in the combined cohort.
Among PCOS Cases Only (n = 30) No statistically significant correlations were found between any canonical HRV/CAN parameter and anthropometric variables (BMI, body fat percentage, waist-hip ratio).
Serum testosterone was significantly positively correlated with HF power (r = 0.40, p = 0.031). All other correlations between testosterone and CAN indices were not significant (Table 2).
A significant negative correlation was detected between LF/HF ratio and HDL cholesterol (r = -0.39, p = 0.02). No other significant associations between lipid parameters and CAN indices were observed (Table 3).
Testosterone was significantly positively correlated with hirsutism score (r = 0.45, p = 0.01) and HOMA-IR (r = 0.39, p = 0.034). No statistically significant correlations were seen with BMI, body fat, waist-hip ratio, or lipid values (Table 4).
Waist-hip ratio correlated significantly and positively with triglycerides (r = 0.38, p = 0.04). Body weight significantly correlated with total cholesterol (r = 0.4; p = 0.028) and LDL (r = 0.39, p = 0.033).All other correlations between lipid parameters and anthropometric/metabolic markers were not statistically significant.
HOMA-IR was significantly positively correlated only with serum testosterone (r = 0.39, p = 0.034) and with HF power (r = 0.40, p = 0.031). No other significant correlations with CAN, anthropometry, or lipid parameters were detected (Table 3).

Table 1: CAN parameters comparison between cases and control

CAN Parameters	Cases	Control	P value
HRV to standing (30: 15) ratio (Mean ± SD)	1.24±0.19	1.23±0.17	0.78
HRV to valsalva (Mean ± SD)	1.53±0.28	4.6±16.74	0.32
HRV to deep breathing (E:I ratio) (Mean ± SD)	1.22±0.14	1.27±0.13	0.17
Normalized LF Power (Mean ± SD)	0.49±0.15	0.46±0.15	0.41
Normalized HF power(Mean ± SD)	0.51±0.15	0.54±0.15	0.38
LF power [Median(25^{th} -75^{th} centile)]	95.2(46.82 - 174.4)	118.65(81.42-157.67)	0.31
HF power [Median(25^{th} - 75^{th} centile)]	102.77(53.21 - 153.57)	123.9(95.98-227.22)	0.13
LF/HF ratio [Median(25^{th} - 75^{th} centile)]	1(0.61 - 1.59)	0.74(0.57-1.35)	0.4
SDNN [Median(25^{th} - 75^{th} centile)]	39.54(24.83 - 54.06)	40.39(36.25-47.77)	0.33
RMSSD [Median(25^{th} -75^{th} centile)]	32.67(18.9 - 50.91)	36.42(29.34-53.25)	0.19
NN50 [Median(25^{th} - 75^{th} centile)]	31.5(3.25 - 85)	45.5(24-109)	0.08
pNN50 [Median(25^{th} - 75^{th} centile)]	9.09(0.78 - 22.97)	12.75(6.59-33.67)	0.1
BP response to standing [Median (25^{th} - 75^{th} centile)]	-2(-8 - -2)	0(0-0)	0.22
DBP response to hand grip [Median (25^{th} - 75^{th} centile)]	17(10 - 23)	10(8.5-20)	0.27

Table 2: Among the Cases; Correlation of different CAN parameters with serum testosterone levels

	r value	P value
SBP response to standing	-0.06	0.76
DBP response to hand grip	-0.09	0.63
HRV to standing (30: 15 ratio)	0.02	0.91
HRV to valsalva	0.3	0.16
HRV to deep breathing (E:I ratio)	0.14	0.47
LF power	0.24	0.21
HF power	0.4	0.031
LF/HF ratio	-0.32	0.09
SDNN	0.33	0.08
RMSSD	0.29	0.11
NN50	0.27	0.15
pNN50	0.27	0.14

Table 3: Among the Cases; Correlation of different CAN parameters with lipid parameters, body fat and HOMA-IR

CAN parameters		HDL	LDL	TG	Total cholesterol	Body fat%	HOMA-IR
SBP response to standing	r	-0.12	-0.03	-0.11	-0.21	-0.11	-0.11
	p	0.53	0.87	0.55	0.26	0.39	0.58
DBP response to hand grip	r	-0.04	0.36	-0.12	0.28	0.27	0.35
	p	0.83	0.048	0.52	0.13	0.04	0.055
HRV to standing (30: 15 ratio)	r	0.03	-0.04	-0.13	-0.02	-0.06	0.14
	p	0.87	0.83	0.48	0.92	0.64	0.47
HRV to valsalva	r	-0.07	-0.11	-0.27	-0.11	-0.18	0.21
	p	0.72	0.55	0.14	0.796	0.16	0.27
HRV to deep breathing (E:I ratio)	r	-0.05	-0.11	-0.27	0.16	-0.14	0.06
	p	0.78	0.55	0.15	0.41	0.27	0.76
LF power	r	-0.16	0.03	0.01	0.1	-0.17	0.17
	p	0.39	0.86	0.97	0.6	0.2	0.36
HF power	r	-0.05	-0.01	0.14	0.17	-0.04	0.36
	p	0.77	0.96	0.47	0.37	0.76	0.049
LF/HF ratio	r	-0.39	-0.07	-0.14	-0.24	-0.16	-0.12
	p	0.02	0.69	0.47	0.2	0.22	0.54
SDNN	r	0.12	0.09	-0.02	0.13	-0.18	0.19
	p	0.54	0.63	0.93	0.49	0.17	0.3
RMSSD	r	0.11	-0.01	0.03	0.03	-0.13	0.08
	p	0.57	0.93	0.88	0.87	0.32	0.68
NN50	r	0.11	-0.02	0.07	0.09	-0.2	0.12
	p	0.57	0.91	0.72	0.63	0.12	0.54
pNN50	r	0.12	-0.04	0.03	0.04	-0.17	0.07
	p	0.52	0.85	0.87	0.81	0.19	0.7

Table 4: Among cases, correlation between serum testosterone and anthropometry, lipid parameters, body fat and HOMA-IR

	r value	P value
Anthropometry		
Weight	0.18	0.35
BMI	0.16	0.4
W: H ratio	0.09	0.62
Lipid parameters		
Serum HDL	0.19	0.31
Serum LDL	0.33	0.08
Serum Triglycerides	0.19	0.33
Total cholesterol	0.34	0.06
Others		
Body fat%	0.2	0.3
HOMA -IR	0.39	0.034

Discussion

This study confirms that PCOS cases, compared to matched controls, exhibit greater adiposity, visceral fat, and a pro-atherogenic lipid profile—hallmarks of classical metabolic PCOS phenotype [9]. Despite this, resting cardiac autonomic function, as assessed by robust HRV and BP-based indices, did not differ significantly between groups [10]. This finding, challenges the universality of CAN dysfunction in PCOS and highlights the importance of investigating this disorder in ethnically divergent, well-defined cohorts. Notably, within PCOS, higher serum testosterone was independently associated with increased HF power—signifying greater parasympathetic (vagal) modulation. Studies such as Sathyapalan *et al*. (2023) and Sharma *et al*. (2024) have described comparable HRV and sympathovagal balance in PCOS and controls, particularly in groups without overt obesity or severe metabolic syndrome [11, 12,

[13]. This lack of autonomic alteration is in contrast to classic literature from Western populations reporting reduced HRV, heightened LF/HF ratio, or sympathetic predominance in PCOS [14, 15]. But data from South Asian cohorts consistently show less pronounced autonomic dysfunction than Western comparators, possibly due to differences in body composition, genetic influences on autonomic tone, or lifestyle factors [11, 12]. Several Indian and Asian studies have reported neutral or even positive associations between testosterone and vagal indices (e.g., HF power, rMSSD), particularly in less obese or early-stage PCOS [12].

Possible mechanism includes central neuroendocrine adaptation, i.e testosterone might upregulate baroreflex sensitivity or parasympathetic output as a compensatory mechanism against metabolic or inflammatory stress. Western studies may over-represent severe phenotypes, while less obese or ethnically distinct groups may have different neuroendocrine-autonomic set points. Phenotypic Heterogeneity-as PCOS is clinically diverse—milder or non-hyperandrogenic phenotypes, or lower prevalence of overt hypertension/diabetes, could yield average HRV values similar to controls. Measurement Context: Resting, non-provocative HRV may miss latent or stress-induced autonomic impairments which are otherwise demonstrable in challenge protocols or dynamic testing. There is possible reverse Causality i.e higher vagal tone may affect gonadotropin release, indirectly boosting androgen synthesis. These possible hypothesis need further basic research for substantiation.

This contrast suggests complex, potentially compensatory neuroendocrine-autonomic interactions in these women.

Importantly, the only significant CAN-lipid relationship was a negative correlation between LF/HF ratio and HDL cholesterol [16, 17]. This mirrors findings in both Indian and global research showing that higher HDL levels are linked to a more favorable sympathovagal balance [18, 19]. This reflects well-known cardiovascular physiology where higher HDL supports endothelial/parasympathetic pathways, reducing cardiovascular risk.

No direct links were found between HRV and other lipid or anthropometric markers, implying partial independence of metabolic and autonomic pathways.

The strong positive association between HOMA-IR and both serum testosterone and HF power fits established literature underpinning the insulin resistance-hyperandrogenism axis in PCOS [14].

The only anthropometric-lipid association reaching significance was between waist-hip ratio and triglycerides, underscoring the central role of visceral adiposity in dyslipidemia [20, 21].

The robust association of testosterone with HOMA-IR and hirsutism score reiterates its role as a driver of metabolic and clinical hyperandrogenism in PCOS, but not as a simple marker of autonomic derangement.

These findings strongly support the growing view that PCOS cardiometabolic and neuroendocrine risk stratification requires personalized, pathophysiology-oriented approaches, rather than assumptions based on group averages or classic dogma [22].

Conclusion

Indian PCOS cohorts may maintain relatively preserved or even adaptively increased parasympathetic tone, even in the presence of metabolic and androgen excess. This study thus underscores the need for ethnic- and phenotype-specific reference data and highlights the limits of one-size-fits-all models for PCOS cardiovascular risk stratification.

This study supports the concept that, in certain populations, the relationship between hyperandrogenism and autonomic function in PCOS is independent of obesity, insulin resistance, or chronicity of menstrual dysfunction. Rather, intrinsic regulatory factors may play a key role—a hypothesis that warrants future prospective validation.

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Conflict of interest: None.

References

1. Yu Y, Chen J, Zheng C, Jia X, Liao J, Ren X, Liu Z, Liu H. The role of the autonomic nervous system in polycystic ovary syndrome. Front Endocrinol (Lausanne). 2024;15:1295061. doi:10.3389/fendo.2023.1295061.
2. Choudhary A, *et al*. A comparative study of heart rate variability in women with polycystic ovary syndrome. 2024.
3. Zakeri A, Shafiee G, Kazemi S, *et al*. Body composition analysis in women with polycystic ovary syndrome and matched controls. 2024.
4. Parua S, Dasgupta A, Saha I, Dutta S. Assessing body composition through anthropometry: implications for diagnosing and managing polycystic ovary syndrome (PCOS). Clin Physiol Funct Imaging. 2025. DOI:10.1111/cpf.12905.
5. Zhu S, *et al*. Imaging-based body fat distribution in polycystic ovary syndrome. 2021.
6. Ewing DJ, Clarke BF. Diagnosis and management of diabetic autonomic neuropathy. Br Med J. 1982;285(6346):916-918. DOI:10.1136/bmj.285.6346.916.
7. Shaffer F, Ginsberg JP. An overview of heart rate variability metrics and norms. Front Public Health. 2017;5:258. DOI:10.3389/fpubh.2017.00258.
8. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. Heart rate variability: standards of measurement, physiological interpretation and clinical use. Circulation. 1996;93(5):1043-1065. DOI:10.1161/01.CIR.93.5.1043.
9. Sharma S, Shukla H, *et al*. Study of lipid profile in polycystic ovarian syndrome: a hospital-based case control study. Int J Reprod Contracept Obstet Gynecol. 2021;10(12):4452-4457.
10. Srivastava N, Kumari R, Narayan J. A comparative study of heart rate variability in women with and without PCOS. Healthcare Bulletin. 2024;9(2):125-132.
11. Yildirir A, Kabakci G, Akgul E, *et al*. Heart rate variability in young women with polycystic ovary syndrome. Int J Cardiol. 2006;113(1):102-106.
12. Saxena Y, Sarda N, Phaneendra BV, *et al*. Effect of polycystic ovary syndrome on cardiac autonomic function at rest and during orthostatic stress: a prospective case-control study. BMJ Open. 2022;12:e059183.
13. Kaur A, Doddi S, Deenadayal M, *et al*. Racial and ethnic disparities in polycystic ovary syndrome: the multifactorial role of genes, environment, and healthcare practices. J Endocrinol Invest. 2023;46(7):1455-1466.
14. Verma N, Chopra R, Sahu S, *et al*. Cardiovascular autonomic function in PCOS using heart rate variability among young adults in Delhi NCR, India. J Indian Med Assoc. 2025;123(4):22-31.
15. Saxena Y, Sarda N, *et al*. Cardiovascular autonomic dysfunction in women with polycystic ovary syndrome. Indian J Endocrinol Metab. 2014;18(2):246-252.

16. Yazıcı S, Karachi S, Yilmaz Y, *et al*. Associations between heart rate variability and lipid profile in women with polycystic ovary syndrome. Anatol J Cardiol. 2017;17(5):395-404.
17. Taskin M, Kocoglu G, *et al*. Heart rate variability and lipid levels: a cross-sectional study in women with PCOS. J Obstet Gynaecol Res. 2018;44(7):1392-1400.
18. Mehta P, *et al*. Associations between heart rate variability and lipid profile in women with PCOS. Afr J Biomed Res. 2022;25(3):307-313.
19. Reddy SB, Shankar KV, Chittari S, *et al*. A cross-sectional study of heart rate variability and androgen status in Indian women with PCOS. Int J Reprod Contracept Obstet Gynecol. 2022;11(6):1589-1594.
20. Kelly CJ, Lyall H, Petrie JR, Gould GW, Connell JM, Sattar N. Waist circumference is a key predictor of dyslipidemia in PCOS. Hum Reprod. 2001;16(5):1088-1096.
21. Sirmans SM, Pate KA. Epidemiology, diagnosis, and management of polycystic ovary syndrome. Int J Womens Health. 2014;6:1233-1243.
22. Reaven GM, *et al*. Central neuroendocrine regulation of insulin and cardiovascular risk in PCOS. J Clin Endocrinol Metab. 2016;101(4):1451-1458.

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