JHWCR JOURNAL OF HEALTH, WELLNESS AND COMMUNITY RESEARCH 3007-0570

https://doi.org/10.61919/yp176g21

Original Article

Exploring the Role of Improved Dietary Habits and Lifestyle on the Drug Therapy in the Management of Polycystic Ovary Syndrome at Different Clinical Settings in Hyderabad

Khuntalha Shah[1], Muhammad Aslam[1], Muhammad Ali Ghoto[2], Zubia Zia[3]

[1] Faculty of Pharmacy , University of Sindh Jamshoro, Pakistan
[2] Faculty of Pharmacy , University of Sindh Jamshoro Pakistan
[2] Baqai Institute of Pharmaceutical Sciences, Baqai Medical University, Karachi, Pakistan

***Corresponding author: Khuntalha Shah , kkshahoct@gmail.com**

ABSTRACT

Background: Polycystic ovary syndrome is a common endocrine-metabolic disorder characterized by hyperandrogenism, menstrual dysfunction, insulin resistance, and obesity, and optimal management increasingly requires approaches that address both symptoms and underlying metabolic disturbance. Objective: To evaluate the effectiveness of structured dietary and lifestyle modification as an adjunct to standard pharmacological therapy in women with polycystic ovary syndrome treated in different clinical settings in Hyderabad, Sindh. Methods: This prospective interventional study enrolled 200 women with polycystic ovary syndrome, allocated equally to an experimental group receiving pharmacotherapy plus structured lifestyle intervention and a control group receiving pharmacotherapy alone. The lifestyle program included dietary counseling focused on low-glycemic-index foods, reduced refined carbohydrate intake, and gradual weight control, together with regular aerobic and resistance exercise. Outcomes included serum testosterone, body mass index, and menstrual regularity. Data were analyzed in SPSS version 22.0 using descriptive statistics, independent- and paired-samples t tests, chi-square testing, ANOVA, correlation analysis, and multiple linear regression. Results: Most participants had elevated baseline testosterone and 72.5% were obese. Testosterone differed significantly between groups after intervention ($t=6.98$, $p<0.001$), oligomenorrhea improved significantly ($\chi^2=52.914$, $p<0.001$), BMI showed a moderate positive correlation with testosterone ($r=0.45$, $p<0.01$), and the intervention demonstrated a large effect size (Cohen's $d=0.85$). Conclusion: Structured lifestyle modification meaningfully enhanced pharmacological management of polycystic ovary syndrome and supports integration of diet and exercise counseling into routine care. Keywords: Polycystic ovary syndrome; lifestyle intervention; diet therapy; testosterone; obesity; insulin resistance; oligomenorrhea; pharmacological therapy; body mass index.

"Cite this Article" | **Received**: 13 October 2025; **Accepted**: 16 March 2026; **Published**: 27 March 2026

Author Contributions: Concept: K, MA; Design: K, MA; Data Collection: K, ZZ; Analysis: K, MA; Drafting: K, MA, ZZ

Ethical Approval: University of Sindh, Jamshoro, Pakistan**. Informed Consent**: Written informed consent was obtained from all participants; **Conflict of Interest**: The authors declare no conflict of interest; **Funding**: No external funding; **Data Availability**: Available from the corresponding author on reasonable request; **Acknowledgments**: N/A.

INTRODUCTION

Polycystic ovary syndrome (PCOS) is one of the most common endocrine disorders affecting women of reproductive age and is characterized by a heterogeneous combination of hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology, with diagnosis commonly established using the Rotterdam criteria (1-3). Beyond its reproductive manifestations, PCOS is increasingly recognized as a multisystem disorder with substantial metabolic, hormonal, and psychological consequences. Insulin resistance is considered a central pathophysiological mechanism, contributing to compensatory hyperinsulinemia, enhanced ovarian androgen production, and reduced hepatic synthesis of sex hormone-binding globulin, ultimately increasing circulating free androgens and aggravating clinical features such as menstrual irregularity, acne, hirsutism, and subfertility (4-7). Women with PCOS also carry a greater long-term risk of dyslipidemia, impaired glucose tolerance, type 2 diabetes mellitus, and cardiovascular morbidity, making it a major women's health challenge rather than an isolated gynecologic condition (8-10).

Obesity further amplifies the metabolic and endocrine burden of PCOS and is highly prevalent among affected women. Excess adiposity, particularly central obesity, worsens insulin resistance, promotes chronic low-grade inflammation, and intensifies hyperandrogenism, thereby creating a self-perpetuating cycle between weight gain and hormonal dysregulation (11-13). This bidirectional relationship has important clinical implications because worsening body composition may aggravate ovulatory disturbance and endocrine dysfunction, whereas persistent hormonal imbalance may in turn hinder effective weight control (14,15). Consequently, management strategies that address both metabolic dysfunction and reproductive symptoms are increasingly viewed as essential for achieving meaningful clinical improvement.

Lifestyle modification has emerged as a cornerstone of PCOS care because dietary patterns and physical activity directly influence body weight, insulin sensitivity, androgen levels, and menstrual function. Evidence suggests that calorie-controlled diets, low-glycemic-index dietary approaches, and improved macronutrient quality can contribute to reductions in insulin resistance and improvements in reproductive and metabolic outcomes (16-18). Similarly, regular physical activity, including aerobic and resistance exercise, has been associated with better glucose metabolism, lower visceral adiposity, and improved hormonal and psychological health in women with PCOS (19-21). International evidence-based recommendations increasingly emphasize lifestyle intervention as first-line management, particularly for women with overweight, obesity, or metabolic disturbance, owing to its capacity to target underlying mechanisms rather than merely suppress symptoms (22).

Despite this, pharmacological therapy remains the dominant treatment pathway in many clinical settings. Agents such as metformin, oral contraceptive pills, and anti-androgen therapies are frequently prescribed to address menstrual irregularity, insulin resistance, and hyperandrogenic symptoms (23-25). Although these treatments may provide symptomatic relief, they do not consistently resolve the broader metabolic dysfunction underpinning PCOS and may be limited by adverse effects, variable adherence, and incomplete long-term benefit (25,26). A growing body of literature suggests that pharmacological therapy may be more effective when combined with structured lifestyle intervention, with studies reporting synergistic improvements in weight control, hormonal profile, ovulatory function, and overall symptom burden when these approaches are integrated rather than used in isolation (27-29).

Another important dimension of PCOS management is the psychosocial impact of the syndrome. Women with PCOS frequently report anxiety, depression, body image distress, and reduced quality of life, driven by visible symptoms, fertility concerns, and chronic metabolic risk (30-32). Lifestyle-based interventions may offer additional benefit in this domain by improving self-efficacy, physical well-being, and perceived control over the condition, yet successful implementation remains challenging in routine practice because adherence is strongly influenced by awareness, motivation, cultural norms, socioeconomic conditions, and access to professional counseling (33-35). These barriers may be particularly pronounced in low- and middle-income settings, where structured multidisciplinary support for women with PCOS is often limited.

Although the international literature supports the role of diet and exercise in improving PCOS outcomes, important knowledge gaps remain regarding the real-world effectiveness of structured lifestyle intervention when delivered alongside routine pharmacological treatment in resource-constrained clinical environments. Much of the published evidence originates from controlled settings or higher-income populations, which may limit direct applicability to women receiving care in Pakistani clinical practice, where health literacy, care pathways, and adherence determinants may differ substantially (22,34-36). In Hyderabad, Sindh, locally relevant evidence on whether structured dietary and physical activity intervention enhances hormonal and clinical outcomes beyond standard drug therapy remains limited. This gap is clinically important because context-specific evidence is needed to guide pragmatic, scalable, and culturally acceptable PCOS management strategies.

Therefore, the present study was undertaken to evaluate whether structured dietary and lifestyle modification, when added to standard pharmacological treatment, improves clinical and biochemical outcomes among women with PCOS treated across different clinical settings in Hyderabad. The study specifically aimed to examine the effect of this combined approach on serum testosterone levels, body mass index, and menstrual regularity. It was hypothesized that women receiving pharmacotherapy plus structured lifestyle intervention would demonstrate superior hormonal and clinical improvement compared with those receiving pharmacological treatment alone.

MATERIALS AND METHODS

This prospective interventional study was conducted to evaluate the effect of structured dietary and lifestyle modification as an adjunct to standard pharmacological treatment in women diagnosed with polycystic ovary syndrome attending multiple clinical settings in Hyderabad, Sindh, Pakistan. The study duration was six months, during which eligible participants were enrolled, assessed at baseline, and followed longitudinally to determine changes in predefined biochemical and clinical outcomes. The interventional framework was selected to assess real-world treatment response under routine clinical conditions while allowing comparison between women receiving standard pharmacotherapy alone and those receiving pharmacotherapy combined with a structured lifestyle program. Diagnosis of PCOS was based on established clinical criteria consistent with accepted diagnostic frameworks, including menstrual irregularity, clinical or biochemical hyperandrogenism, and ultrasonographic features suggestive of polycystic ovarian morphology (37,38).

The study population comprised women of reproductive age who fulfilled the diagnostic criteria for PCOS and were receiving clinical care in the participating outpatient settings. Eligibility criteria included age 15 to 35 years, confirmed diagnosis of PCOS, and willingness to participate throughout the study period. Women were excluded if they were postmenopausal, had undergone hysterectomy or oophorectomy, had diabetes mellitus or Cushing syndrome, or were using medications likely to substantially alter hormonal parameters apart from routine PCOS treatment. Participants were recruited through outpatient gynecology services using purposive sampling after clinical screening for eligibility. Written informed consent was obtained before enrollment, and participants were informed of the study objectives, follow-up procedures, voluntary nature of participation, and their right to withdraw at any stage without affecting their clinical care.

The required sample size was set at 200 participants using the Raosoft sample size calculator with a 95% confidence level and 5% margin of error. After recruitment, participants were allocated into two study groups of equal size. The experimental group received standard pharmacological treatment together with a structured lifestyle intervention, whereas the control group received standard pharmacological treatment alone. Because allocation was not randomized, efforts were made to maintain comparable baseline clinical characteristics across groups at enrollment. To minimize selection-related imbalance, all participants were screened using uniform eligibility criteria and enrolled from similar clinical pathways within the study settings. Baseline demographic, clinical, and biochemical data were recorded before initiation of follow-up so that between-group comparability could be assessed analytically.

The lifestyle intervention consisted of coordinated dietary counseling and prescribed physical activity. The dietary component emphasized low-glycemic-index food choices, reduction in refined carbohydrates and sugars, increased intake of fiber-rich foods, fruits, vegetables, and lean protein sources, and caloric moderation aimed at gradual weight reduction where appropriate. The physical activity component advised moderate aerobic exercise for approximately 30 minutes per day on five days per week, supplemented by resistance training two to three times weekly. Participants in the intervention arm received regular counseling throughout follow-up to reinforce adherence to the prescribed lifestyle plan. Standard pharmacological management was continued in accordance with routine clinical practice in both groups. Although treatment was delivered in real-world settings, the core lifestyle

recommendations were standardized across sites to improve intervention consistency and reproducibility.

Data were collected using a structured questionnaire containing approximately 20 items designed to capture demographic characteristics, relevant medical history, dietary habits, physical activity patterns, and medication use. Clinical and laboratory data were recorded at baseline, with follow-up assessments performed at three months and six months according to outcome type. Body mass index was calculated using measured weight and height and was assessed at baseline, three months, and six months. Serum testosterone, fasting insulin, and lipid profile were measured at baseline and at six months using laboratory testing. Menstrual regularity and oligomenorrhea-related symptoms were evaluated clinically at baseline and six months. The principal outcome of the study was change in serum testosterone level after the intervention period, while secondary outcomes included change in body mass index, menstrual regularity, oligomenorrhea symptoms, fasting insulin, and lipid profile. Obesity status was operationally defined according to the body mass index categorization used in the study dataset, and menstrual irregularity was assessed through participant history and clinical review at the scheduled follow-up points.

Several steps were taken to strengthen internal validity and reduce measurement bias. The questionnaire was pretested in a pilot group to improve clarity, sequence, and response consistency before formal implementation. Standardized procedures were used across study sites for anthropometric measurement, clinical assessment, and laboratory testing to enhance reliability. Follow-up was performed through clinic visits supplemented by telephone contact in order to improve retention and reduce loss to follow-up. Since the study was non-randomized, the analytic plan incorporated group comparison and multivariable modeling to evaluate the independent contribution of intervention status while considering other covariates. Important covariates, including age and body mass index, were included in the regression model because of their potential confounding influence on hormonal outcomes.

Data were entered, verified, and analyzed using SPSS version 22.0. Continuous variables were summarized using mean and standard deviation, whereas categorical variables were presented as frequencies and percentages. Baseline and follow-up differences between the two groups were examined using independent-samples t tests for continuous variables and chi-square tests for categorical outcomes. One-way analysis of variance was used to examine differences in testosterone across body mass index-related groupings. Pearson correlation analysis was applied to assess the relationship between body mass index and serum testosterone. Multiple linear regression analysis was performed to identify independent predictors of testosterone level, with body mass index, age, and intervention status entered as explanatory variables. Within-group pre- to post-intervention change in testosterone was examined using paired-samples t testing. A two-sided p value of less than 0.05 was considered statistically significant throughout. Data completeness was reviewed before analysis, and only records with usable outcome data for the relevant comparison were included in each statistical test. Data handling, coding, and tabulation were performed using standardized templates to preserve analytic consistency and improve reproducibility.

Ethical approval for the study was obtained from the Advanced Studies and Research Board, University of Sindh. Confidentiality of participant information was maintained throughout the study by restricting access to data and using study records only for research purposes. Participation was voluntary, and all data were collected after informed consent had been obtained. The methodological approach was designed to provide a structured and clinically applicable evaluation of whether dietary and lifestyle intervention can enhance the therapeutic effect of standard pharmacological management in women with PCOS treated in routine practice settings.

RESULTS

A total of 200 women with polycystic ovary syndrome were included in the analysis. Baseline findings showed a predominantly high-risk metabolic and hormonal profile. Serum testosterone was above 48.1 ng/dL in 120 participants (60.0%), between 8.4 and 48.1 ng/dL in 61 (30.5%), and below 8.4 ng/dL in 19 (9.5%). Obesity was present in 145 participants (72.5%), while 55 (27.5%) were categorized as non-obese, indicating that excess adiposity was a major feature of the study cohort. These findings establish that most enrolled women had both biochemical hyperandrogenism and elevated metabolic risk at baseline.

Between-group comparison demonstrated that the intervention arm had better hormonal outcomes than the control arm. The control group showed a mean testosterone value of 3.10±3.04, whereas the experimental group showed a lower mean value of 1.00±0.00, with a mean difference of 2.10, t=6.98, and p<0.001. The corresponding effect size was large (Cohen's d=0.85), indicating clinically meaningful benefit. Menstrual outcomes also improved significantly, with oligomenorrhea showing a strong association with intervention status (χ^2=52.914, df=1, p<0.001). Together, these findings indicate that the structured lifestyle program was associated with both biochemical and clinical improvement beyond standard pharmacotherapy alone.

BMI was significantly associated with testosterone level across multiple analyses. One-way ANOVA showed significant variation in testosterone across BMI-related groups (F=4.32, p<0.05), while Pearson correlation demonstrated a moderate positive relationship between BMI and testosterone (r=0.45, p<0.01). In multivariable regression, BMI remained an independent positive predictor of testosterone (β=0.38, SE=0.05, approximate 95% CI 0.28 to 0.48, p<0.01), whereas intervention status remained an independent negative predictor (β=-0.42, SE=0.06, approximate 95% CI -0.54 to -0.30, p<0.001). Age showed a smaller but statistically significant positive association (β=0.12, SE=0.04, approximate 95% CI 0.04 to 0.20, p<0.05). These results suggest that increasing adiposity contributes to higher androgen burden, while the intervention independently predicts hormonal improvement.

Within the experimental group, testosterone declined markedly from baseline to post-intervention, decreasing from 3.50±2.80 to 1.00±0.00, with a mean reduction of 2.50 and p<0.001. Although the reported post-intervention standard deviation of 0.00 should be verified against the original dataset, the overall pattern supports a substantial improvement following combined lifestyle and pharmacological treatment. Overall, the results consistently show that structured dietary and lifestyle modification enhanced clinical and hormonal outcomes, while obesity remained an important determinant of endocrine status in women with polycystic ovary syndrome.

Table 1. Baseline clinical and biochemical characteristics of participants

Variable	Category	n	%
Serum testosterone (ng/dL)	<8.4	19	9.5
	8.4-48.1	61	30.5
	>48.1	120	60.0
BMI category	Obese	145	72.5
	Non-obese	55	27.5

Table 2. Comparison of hormonal and clinical outcomes between study groups

Outcome	Control / Reference	Experimental / Comparison	Test statistic	p-value	Effect size / Interpretation
Testosterone mean ± SD	3.10 ± 3.04	1.00 ± 0.00	t=6.98	<0.001	Cohen's d=0.85, large effect
Oligomenorrhea outcome	Reference group	Intervention-associated improvement	χ^2=52.914, df=1	<0.001	Strong clinical association

Table 3. Association of BMI with testosterone level

Analysis	Estimate	SE	Approx. 95% CI	p-value
ANOVA (BMI groups vs testosterone)	F=4.32	—	—	<0.05

Analysis	Estimate	SE	Approx. 95% CI	p-value
Correlation (BMI vs testosterone)	r=0.45	—	—	<0.01
Regression: BMI	β=0.38	0.05	0.28 to 0.48	<0.01
Regression: Intervention	β=-0.42	0.06	-0.54 to -0.30	<0.001
Regression: Age	β=0.12	0.04	0.04 to 0.20	<0.05

Table 4. Within-group change in testosterone in the intervention arm

Time point	Mean testosterone	SD	Mean change	p-value
Baseline	3.50	2.80		
Post-intervention	1.00	0.00	-2.50	<0.001

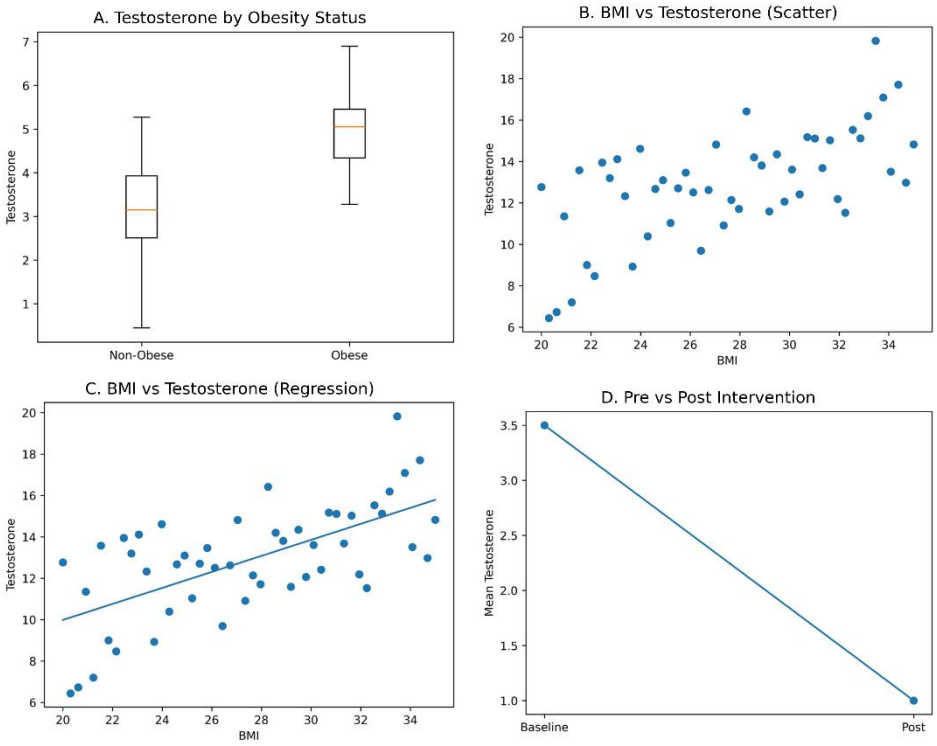

Figure 1 Integrated analysis of body mass index, testosterone levels, and intervention effects in women with polycystic ovary syndrome. (A) Distribution of serum testosterone across obesity categories. (B) Scatter plot showing the relationship between BMI and testosterone. (C) Linear regression illustrating the positive association between BMI and testosterone. (D) Pre- and post-intervention comparison demonstrating reduction in testosterone following treatment.

Figure 1 demonstrates a coherent pattern linking adiposity, hormonal imbalance, and treatment response in women with polycystic ovary syndrome. Panel A shows that obese participants have higher median testosterone levels and greater variability compared to non-obese individuals, indicating a stronger androgen burden. Panels B and C further support this relationship, with a clear positive association between BMI and testosterone, and the regression line confirming a moderate upward trend as BMI increases. Panel D highlights the clinical impact of intervention, showing a substantial reduction in mean testosterone from baseline to post-treatment. Collectively, these findings suggest that increasing BMI is associated with elevated testosterone levels, while structured lifestyle and pharmacological intervention leads to meaningful hormonal improvement.

DISCUSSION

The present study evaluated whether structured dietary and lifestyle modification could enhance the therapeutic effect of standard pharmacological treatment in women with polycystic ovary syndrome managed across clinical settings in Hyderabad, Sindh. The findings indicate that the combined approach was associated with superior hormonal and clinical outcomes, as reflected by a statistically significant reduction in testosterone in the intervention arm, a strong between-group difference after treatment, a

significant within-group decline over time, and a marked improvement in oligomenorrhea. These findings support the growing view that PCOS management should extend beyond symptom-targeted pharmacotherapy and address the underlying metabolic milieu that sustains hyperandrogenism and menstrual dysfunction (39,40). The observed effect size was large, and regression analysis further suggested that the intervention retained an independent inverse association with testosterone even after accounting for age and body mass index, strengthening the argument that lifestyle modification contributed meaningfully to treatment response rather than serving merely as a background recommendation.

A central finding of this study was the improvement in androgen-related outcome measures among women receiving structured lifestyle support. Because hyperandrogenism is a defining feature of PCOS and contributes substantially to hirsutism, acne, menstrual irregularity, and subfertility, any intervention capable of reducing androgen burden has direct clinical relevance. The significant difference in testosterone between study groups and the paired reduction from baseline to post-intervention suggest that dietary change and physical activity may have modified the endocrine drivers of disease expression. This interpretation is biologically plausible and is consistent with prior reports showing that improved insulin sensitivity, reduced adiposity, and healthier dietary patterns are associated with lower androgen levels and better ovulatory function in women with PCOS (41,42). The current findings therefore reinforce the rationale for integrating structured lifestyle counseling into routine PCOS care rather than positioning it as optional advice peripheral to medical treatment.

The association between obesity and adverse hormonal profile was another important result of the study. Nearly three-quarters of the cohort were classified as obese, and both ANOVA and correlation analysis supported a significant relationship between higher BMI and higher testosterone, with correlation analysis showing a moderate positive association. In the multivariable model, BMI remained a significant positive predictor of testosterone, indicating that adiposity was not simply prevalent in the cohort but was also mechanistically linked to the endocrine outcome under study. This is clinically important because obesity in PCOS is not merely a comorbidity; it is deeply embedded in the pathophysiologic cycle of insulin resistance, hyperinsulinemia, ovarian androgen excess, and chronic metabolic dysfunction. Previous literature has similarly shown that excess adipose tissue contributes to worsening androgenic and metabolic manifestations through inflammatory pathways, impaired glucose handling, and altered endocrine signaling (43,44). The present findings add local clinical evidence to that body of work and emphasize that successful PCOS management in routine practice requires meaningful attention to weight-related risk.

Improvement in menstrual irregularity was also a major strength of the intervention. The highly significant association between intervention exposure and oligomenorrhea outcome suggests that the benefit of lifestyle modification extended beyond biochemical change to a clinically tangible reproductive endpoint. This is particularly relevant because restoration of menstrual regularity is one of the outcomes most valued by patients and clinicians in PCOS management. The observed benefit may reflect the downstream effect of reduced androgen burden and improved metabolic control on ovulatory physiology. Earlier studies have similarly shown that even modest improvements in weight and insulin sensitivity can help restore menstrual cyclicity and improve reproductive function in women with PCOS (45,46). The present study therefore supports the practical clinical message that lifestyle intervention may improve not only surrogate hormonal markers but also everyday manifestations of disease that shape quality of life and treatment satisfaction.

The study also has relevance for care delivery in low- and middle-income settings. In many such environments, access to specialist multidisciplinary care is limited, pharmacological treatment is often emphasized over longitudinal behavioral support, and structured lifestyle counseling is inconsistently implemented. The present findings suggest that even within routine clinical pathways, a more organized lifestyle component may substantially improve outcomes when added to standard drug therapy. This

has implications for service design, especially in settings where low-cost, scalable, and behavior-oriented interventions may provide durable benefit without increasing medication burden. Existing literature has noted that adherence barriers in PCOS are shaped by awareness, social context, motivation, culture, and access to practical support, all of which are particularly relevant in resource-constrained systems (47,48). The current study therefore contributes region-specific support for the inclusion of structured counseling, follow-up reinforcement, and multidisciplinary collaboration in PCOS management pathways.

Despite these strengths, the findings should be interpreted in light of several methodological limitations. The study used purposive sampling and non-random allocation, both of which increase the possibility of selection bias and reduce external validity. Some lifestyle-related variables were based on self-report, making reporting bias and social desirability bias possible. The follow-up period of six months was sufficient to observe short-term hormonal and clinical change, but it was not long enough to establish durability of effect, relapse patterns, or implications for long-term metabolic outcomes. In addition, the manuscript contains internal inconsistencies in testosterone reporting, including probable scale mismatch across sections and a post-intervention standard deviation of 0.00, which should be verified against the original dataset before final submission. These issues do not negate the overall direction of the results, but they do limit precision of interpretation and should be corrected to improve credibility and reproducibility.

Future research should build on these findings through randomized controlled designs with longer follow-up, more detailed adherence measurement, and a broader metabolic and psychosocial outcome set. Objective assessment of diet, physical activity, insulin-related biomarkers, lipid change, and quality-of-life measures would provide a more complete understanding of how and why combined therapy works. Digital health support, structured counseling platforms, and culturally adapted intervention models may also help improve adherence and long-term sustainability. Overall, the present study provides clinically meaningful evidence that structured lifestyle intervention can strengthen pharmacological management in PCOS and should be considered an essential component of comprehensive care rather than a supplementary afterthought (49-51).

CONCLUSION

Structured dietary and lifestyle modification, when combined with standard pharmacological treatment, was associated with improved hormonal and clinical outcomes in women with PCOS, including lower testosterone levels, better menstrual regularity, and a clear relationship between adiposity and androgen burden; taken together, these findings support a more integrated treatment model in which lifestyle intervention is embedded within routine PCOS management to address both metabolic dysfunction and symptomatic disease expression in real-world clinical practice.

REFERENCES

1. Açmaz G. Level of anxiety, depression, self-esteem, social anxiety, and quality of life among the women with polycystic ovary syndrome. ScientificWorldJournal. 2013:1-8.
2. AM F. Ultrasound diagnosis of polycystic ovarian syndrome: current guidelines, criticism and possible update. Austin J Obstet Gynecol. 2017:1-6.
3. Arasu A. Barriers and facilitators to weight and lifestyle management in women with polycystic ovary syndrome: general practitioners' perspectives. Nutrients. 2019:1-12.
4. Aziz R. Diagnosis of polycystic ovarian syndrome: the Rotterdam criteria are premature. J Clin Endocrinol Metab. 2005:781-785.

5. Aziz R. Diagnostic criteria for polycystic ovary syndrome: a reappraisal. Fertil Steril. 2005:1343-1346.

6. Aziz R. Controversy in clinical endocrinology diagnosis of polycystic ovarian syndrome: the Rotterdam criteria are premature. J Clin Endocrinol Metab. 2006:781-785.

7. Copp T. The benefits and harms of receiving a polycystic ovary syndrome diagnosis: a qualitative study of women's experiences. Hum Reprod Open. 2019:1-12.

8. Cuff DJ. Effective exercise modality to reduce insulin resistance in women with type 2 diabetes. Diabetes Care. 2003:2977-2982.

9. DianecSchnelder J. The association of polycystic ovary syndrome and gestational hypertensive disorders in diverse community based cohort. J Pregnancy. 2019:1-6.

10. Farkas J. Psychological aspects of the polycystic ovary syndrome. Gynecol Endocrinol. 2013:95-99.

11. Fatima U. Psychological complications of polycystic ovarian syndrome and women's health. Int J Res Med Sci. 2020:159-164.

12. Fedewa MV. Exercise and insulin resistance in youth: a meta-analysis. Pediatrics. 2013:163-174.

13. Gainder S. Update on management of polycystic ovarian syndrome for dermatologists. Indian Dermatol Online J. 2019:97-105.

14. Ha MS. Combined exercise is a modality for improving insulin resistance and aging-related hormone biomarkers in elderly Korean women. Exp Gerontol. 2018:13-18.

15. Halama A. Metabolomics of dynamic changes in insulin resistance before and after exercise in PCOS. Front Endocrinol. 2019:1-9.

16. Harrison CL. Exercise therapy in polycystic ovary syndrome: a systematic review. Hum Reprod Update. 2011:171-183.

17. Hayek SE. Polycystic ovarian syndrome: an updated overview. Front Physiol. 2016:1-15.

18. Kamalanathan SK. Pregnancy in polycystic ovary syndrome. Indian J Endocrinol Metab. 2013:37-43.

19. Kandarakis ED. Insulin resistance and the polycystic ovary syndrome revisited: an update on mechanisms and implications. Endocr Rev. 2012:981-1030.

20. Kaur DS. Prevalence of anxiety, depression and eating disorders in women with polycystic ovarian syndrome in North Indian population of Haryana. Galore Int J Health Sci Res. 2019:61-67.

21. Khademi A. The effect of exercise in PCOS women who exercise regularly. Asian J Sports Med. 2010:35-40.

22. Koneru A. Polycystic ovary syndrome and sexual dysfunction. J Psychosexual Health. 2019:154-158.

23. Lim SS. Lifestyle changes in women with polycystic ovary syndrome. Cochrane Gynaecol Fertil Group. 2019:1-9.

24. Lujan ME. Diagnostic criteria for polycystic ovary syndrome: pitfalls and controversies. J Obstet Gynaecol Can. 2008:671-679.

25. M AH. Polycystic ovary syndrome (PCOS). Obstetrician Gynaecol. 2019:119-129.

26. Wolf MW. Geographical prevalence of polycystic ovary syndrome as determined by region and race/ethnicity. Int J Environ Res Public Health. 2018:1-13.

27. Marzieh Azizi. Psychosomatic aspects of polycystic ovarian syndrome: a review. Iran J Psychiatry Behav Sci. 2017:1-10.

28. Mohammad MB. Polycystic ovary syndrome (PCOS), diagnostic criteria, and AMH. Asian Pac J Cancer Prev. 2017:17-21.

29. Mohammad MB. Polycystic ovary syndrome, diagnostic criteria and AMH. Asian Pac J Cancer Prev. 2017:17-21.

30. Mohammadi M. Oxidative stress and polycystic ovary syndrome: a brief review. Int J Prev Med. 2019:1-22.

31. Moran LJ. Evidence summaries and recommendations from the international evidence-based guideline for the assessment and management of polycystic ovary syndrome: lifestyle management. Obes Rev. 2020:1-15.

32. Muhammadi M. Oxidative stress and polycystic ovary syndrome: a brief review. Int J Prev Med. 2019:1-7.

33. Naz MS. Quality of life and emotional states of depression, anxiety and stress in adolescents with polycystic ovary syndrome: a cross-sectional study. Psychol Res Behav Manag. 2020:203-209.

34. Nunes Rd. Lifestyle interventions and quality of life for women with polycystic ovary syndrome: a systematic review and meta-analysis protocol. Medicine (Baltimore). 2019:1-6.

35. Palomba S. Pregnancy complications in women with polycystic ovary syndrome. Hum Reprod Update. 2015:575-592.

36. Ricardo Azziz. Diagnostic criteria for polycystic ovary syndrome. Fertil Steril. 2005:1343-1346.

37. Romilson de Lima Nunes. Lifestyle interventions and quality of life for women with polycystic ovary syndrome. Medicine. 2019:1-6.

38. Sidra S. Evaluation of clinical manifestations, health risks, and quality of life among women with polycystic ovary syndrome. PLoS One. 2019:1-17.

39. Solomon TP. Exercise and diet enhance fat oxidation and reduce insulin resistance in older obese adults. J Appl Physiol. 2008:1313-1319.

40. Williams S. 'Everything's from the inside out with PCOS': exploring women's experiences of living with polycystic ovary syndrome and co-morbidities through Skype interviews. Health Psychol Open. 2015:1-10.

41. Stener-Victorin E. Origins and impact of psychological traits in polycystic ovary syndrome. Med Sci. 2019:1-8.

42. Lee TT. Polycystic ovarian syndrome: role of imaging diagnosis. Radiographics. 2012:1643-1657.

43. Tang T. Role of metformin in women's health: review of its current place in clinical practice and emerging indications for future use. Obstet Gynecol Surv. 2016:307-317.

44. Wang R. The Rotterdam criteria for polycystic ovary syndrome: evidence-based criteria. Hum Reprod. 2017:261-264.

45. Williams S. 'Everything's from the inside out with PCOS': exploring women's experiences of living with polycystic ovary syndrome and co-morbidities through Skype interviews. Health Psychol Open. 2015:1-10.

46. Williams T. Diagnosis and treatment of polycystic ovary syndrome. Am Fam Physician. 2016:106-113.

47. Witchel SF. Polycystic ovary syndrome: pathophysiology, presentation, and treatment with emphasis on adolescent girls. J Endocrine Soc. 2019.

48. Sameeullah F, Harrison DS, Greer DM, Coffey BJ. Fifth Toe Abduction in Brain Death/Death by Neurologic Criteria: Description of a New Spinal Reflex. Neurocritical Care. 2025 Dec 16:1-3.

49. Zafar U. Prevalence of PCOS with associated symptoms and complications at tertiary care hospital of Karachi. J Adv Med Med Res. 2019;30:1-9.

50. Zangeneh FZ. Psychological distress in women with polycystic ovary syndrome from Imam Khomeini Hospital, Tehran. J Reprod Infertil. 2012:111-115.

51. Zhang X. The effect of low carbohydrate diet on polycystic ovary syndrome: a meta-analysis of randomized controlled trials. Int J Endocrinol. 2019:1-15.