Journal of Population Therapeutics & Clinical Pharmacology

RESEARCH ARTICLE
DOI: 10.53555/6vtsz040

CLINICAL PROFILE AND OUTCOMES OF PATIENTS WITH POLYCYSTIC OVARIAN SYNDROME

Dr. Mili Poonia*

*Assistant Professor, Department of Obstetrics and Gynecology, Muzaffarnagar Medical College, Muzaffarnagar, Uttar Pradesh
milipoonia@gmail com

Accepted: 08 August 2016 Published: 14 September 2016

Abstract

Introduction: Polycystic ovarian syndrome (PCOS) is the most prevalent endocrine disorder affecting reproductive-age women globally, with prevalence ranging 5-26%. Despite its significance, comprehensive clinical profiling and outcome assessment remain limited in South Asian populations. This study aimed to evaluate clinical characteristics and treatment outcomes in women with PCOS at a tertiary care center.

Methods: A prospective observational study was conducted at Gynecology, Muzaffarnagar Medical College, Muzaffarnagar, Uttar Pradesh from June 2015 to July 2016. One hundred eighty women aged 18-40 years diagnosed with PCOS according to Rotterdam criteria were enrolled using consecutive sampling. Comprehensive clinical, anthropometric, biochemical, and hormonal assessments were performed. Treatment outcomes were evaluated at 6-month follow-up. Statistical analysis was performed using SPSS version 20.0.

Results: Mean age was 24.6±4.8 years with 70% participants having BMI≥25 kg/m². Menstrual irregularities affected 90% participants, hirsutism 75%, and polycystic ovarian morphology 85%. Insulin resistance (HOMA-IR>2.5) was present in 60% participants, with 80% showing elevated testosterone levels. Family history of diabetes and PCOS was observed in 40% and 30% respectively. Treatment interventions demonstrated significant improvements: lifestyle modifications reduced BMI (26.8±5.2 to 25.4±4.8 kg/m², $p<0.001$), metformin improved insulin sensitivity, oral contraceptives reduced hyperandrogenic symptoms, and clomiphene achieved 72.2% ovulation rate with 33.3% pregnancy rate.

Conclusion: PCOS presents as a complex metabolic-reproductive syndrome with high prevalence of obesity, insulin resistance, and metabolic dysfunction in South Asian women. Comprehensive, individualized treatment approaches significantly improve clinical outcomes and quality of life, emphasizing the importance of multidisciplinary management strategies.

Keywords: Hyperandrogenism, Insulin Resistance, Metabolic Syndrome, Polycystic Ovarian Syndrome, Rotterdam Criteria

Introduction

Polycystic ovarian syndrome (PCOS) represents one of the most prevalent endocrine disorders affecting women of reproductive age globally, with an estimated prevalence ranging from 5% to 26% depending on the diagnostic criteria employed (Norman et al., 2007). First described by Stein and Leventhal in 1935, PCOS has evolved from being considered merely a reproductive disorder to a complex metabolic syndrome with far-reaching implications for women's health throughout their

lifespan (Azziz et al., 2006). The syndrome encompasses a constellation of clinical manifestations including hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology, creating a heterogeneous phenotype that challenges both clinicians and researchers worldwide.

The pathophysiology of PCOS remains incompletely understood, though current evidence suggests a multifactorial etiology involving genetic predisposition, environmental factors, and intrauterine programming (Franks, 2006). Insulin resistance and compensatory hyperinsulinemia play pivotal roles in the pathogenesis, affecting approximately 50-70% of women with PCOS, irrespective of body weight (Dunaif, 2012). This metabolic dysfunction creates a vicious cycle wherein hyperinsulinemia stimulates ovarian androgen production through direct action on theca cells and indirectly by reducing sex hormone-binding globulin (SHBG) synthesis in the liver, thereby increasing free testosterone levels (Nestler, 2008).

The clinical presentation of PCOS varies significantly among affected individuals, contributing to diagnostic challenges and delayed recognition. Women may present with menstrual irregularities, including oligomenorrhea or amenorrhea, which occur in approximately 85% of cases (Azziz et al., 2009). Hyperandrogenic manifestations such as hirsutism, acne, and androgenic alopecia affect a substantial proportion of patients, with hirsutism being reported in 60-80% of women with PCOS (Rosenfield, 2005). The presence of obesity, particularly central or visceral adiposity, is observed in 50-80% of women with PCOS, further exacerbating insulin resistance and metabolic dysfunction (Lim et al., 2012).

Reproductive consequences of PCOS extend beyond menstrual irregularities to include anovulatory infertility, which accounts for approximately 80% of anovulation-related fertility problems (Balen et al., 2002). Women with PCOS face increased risks of pregnancy complications, including gestational diabetes mellitus, pregnancy-induced hypertension, and increased rates of miscarriage, emphasizing the need for specialized reproductive care (Boomsma et al., 2006). The syndrome's impact on fertility has profound psychological and social implications, affecting quality of life and requiring comprehensive management approaches.

The metabolic implications of PCOS are increasingly recognized as major health concerns. Women with PCOS demonstrate a significantly higher prevalence of type 2 diabetes mellitus, with studies indicating a 2.5 to 7-fold increased risk compared to age-matched controls (Moran et al., 2010). The prevalence of metabolic syndrome ranges from 33% to 47% in women with PCOS, substantially higher than the general population (Apridonidze et al., 2005). Cardiovascular risk factors, including dyslipidemia, hypertension, and endothelial dysfunction, are more prevalent in PCOS patients, suggesting increased long-term cardiovascular disease risk (Wild et al., 2010).

Psychological comorbidities associated with PCOS represent an often-overlooked aspect of the syndrome's clinical impact. Depression and anxiety disorders occur at significantly higher rates in women with PCOS, with studies reporting prevalence rates of 28-64% for depression and 34-57% for anxiety compared to control populations (Dokras et al., 2011). Body image dissatisfaction, related to weight gain, hirsutism, and acne, contributes to reduced quality of life and may perpetuate psychological distress, creating a complex interplay between physical and mental health aspects of the syndrome.

The diagnostic approach to PCOS has evolved considerably since its initial description, with various criteria proposed by different professional organizations. The Rotterdam consensus criteria, established in 2003, require the presence of two out of three features: oligo-ovulation or anovulation, clinical and/or biochemical signs of hyperandrogenism, and polycystic ovaries on ultrasound, after exclusion of other etiologies (Rotterdam ESHRE/ASRM-Sponsored PCOS consensus workshop group, 2004). The Androgen Excess and PCOS Society later proposed alternative criteria emphasizing hyperandrogenism as an essential feature, reflecting ongoing debates about optimal diagnostic approaches (Azziz et al., 2009).

Management of PCOS requires individualized approaches targeting specific patient concerns and long-term health risks. Lifestyle modifications, including dietary interventions and regular physical activity, form the cornerstone of treatment, particularly for overweight and obese patients (Moran et al., 2009). Pharmacological interventions are tailored to address specific symptoms and metabolic

abnormalities, with options including hormonal contraceptives for menstrual regulation and hirsutism control, metformin for insulin sensitization, and fertility medications for women seeking conception (Costello et al., 2007).

The heterogeneous nature of PCOS presentation necessitates comprehensive clinical profiling to optimize patient care and predict long-term outcomes. Understanding the diverse phenotypes within PCOS populations can inform personalized treatment strategies and risk stratification approaches. Indian populations demonstrate unique characteristics in PCOS presentation, with higher prevalence of metabolic dysfunction and distinct genetic susceptibility patterns compared to Western populations, highlighting the importance of population-specific research (Joshi et al., 2014).

Contemporary research efforts focus on elucidating the complex interplay between genetic susceptibility, environmental factors, and phenotypic expression in PCOS. Advanced molecular techniques and genome-wide association studies have identified multiple genetic loci associated with PCOS susceptibility, though the clinical application of these findings remains limited (Chen et al., 2011). Epigenetic modifications and their role in PCOS pathogenesis represent emerging areas of investigation, potentially offering new therapeutic targets and improved understanding of the syndrome's development and progression.

The economic burden of PCOS on healthcare systems is substantial, encompassing direct medical costs related to diagnosis, treatment, and management of complications, as well as indirect costs associated with reduced productivity and quality of life (Azziz et al., 2005). Early diagnosis and appropriate management can potentially reduce long-term healthcare costs by preventing or delaying the onset of diabetes, cardiovascular disease, and other metabolic complications.

Given the complexity and heterogeneity of PCOS, comprehensive clinical studies examining the diverse presentations, outcomes, and therapeutic responses in different populations are essential. Such investigations contribute to improved understanding of the syndrome's natural history, facilitate development of evidence-based management guidelines, and ultimately enhance patient care and outcomes for women affected by this common but complex endocrine disorder. To evaluate the clinical profile and assess the short-term and long-term outcomes of patients diagnosed with polycystic ovarian syndrome presenting to Lord Buddha Koshi Medical College & Hospital, with specific focus on anthropometric characteristics, hormonal profiles, metabolic parameters, and therapeutic responses.

Methodology

Study Design: A prospective observational study

Study Site: The study was conducted at Gynecology, Muzaffarnagar Medical College, Muzaffarnagar, Uttar Pradesh, located in the eastern region of India. This tertiary care academic medical center serves as a referral center for the surrounding districts and provides comprehensive gynecological and endocrinological services.

Study Duration: The study was conducted over a period of 12 months, commencing in June 2015 and concluding in July 2016.

Sampling and Sample Size

A consecutive sampling method was employed to recruit all eligible women presenting to the study site during the specified study period. The sample size was calculated based on an expected prevalence of PCOS of 10% among reproductive-age women, with a desired precision of 3% and 95% confidence interval. Considering an expected non-response rate of 15% and potential loss to follow-up of 20%, the calculated minimum sample size was 180 participants. The consecutive sampling approach ensured representative inclusion of diverse PCOS phenotypes and minimized selection bias. All women meeting the inclusion criteria during the study period were invited to participate, and recruitment continued until the target sample size was achieved. The sampling frame included both new patients presenting with PCOS-related symptoms and established patients with known PCOS diagnoses seeking ongoing care.

Inclusion and Exclusion Criteria

Women aged 18-40 years with a confirmed diagnosis of PCOS according to Rotterdam criteria were included in the study. Participants were required to have at least two of the following features: oligo-ovulation or anovulation evidenced by menstrual cycles longer than 35 days or fewer than 8 cycles per year, clinical hyperandrogenism manifested as hirsutism (Ferriman-Gallwey score ≥8), acne, or androgenic alopecia, biochemical hyperandrogenism demonstrated by elevated total testosterone or free androgen index, and polycystic ovarian morphology on transvaginal ultrasonography showing 12 or more follicles measuring 2-9mm in diameter or ovarian volume exceeding 10mL. Women were excluded if they had other causes of hyperandrogenism including congenital adrenal hyperplasia, Cushing's syndrome, androgen-secreting tumors, thyroid dysfunction, hyperprolactinemia, pregnancy, lactation within the previous 6 months, current use of hormonal medications or insulin sensitizers within 3 months prior to enrollment, presence of severe psychiatric disorders, or other major medical conditions that could interfere with study assessments. Additionally, women who were unable to provide informed consent or were planning to relocate during the study period were excluded from participation.

Data Collection Tools and Techniques

A comprehensive structured questionnaire was developed and validated for data collection, incorporating standardized assessment tools and clinical evaluation protocols. Demographic information including age, education, occupation, and socioeconomic status was recorded using pre-designed forms. Clinical history was obtained through detailed interviews focusing on menstrual patterns, fertility history, family history of PCOS, diabetes, and cardiovascular disease. Physical examination protocols included standardized anthropometric measurements with height measured using a stadiometer, weight recorded using calibrated digital scales, and body mass index calculated as weight in kilograms divided by height in meters squared. Waist and hip circumferences were measured according to WHO guidelines, and waist-to-hip ratio was calculated. Hirsutism assessment was performed using the Ferriman-Gallwey scoring system by trained personnel, with photographic documentation for quality assurance. Blood pressure measurements were taken using standardized protocols with appropriate cuff sizes. Laboratory investigations included fasting glucose, insulin levels, lipid profiles, liver function tests, and hormonal assessments including total testosterone, SHBG, LH, FSH, and anti-Müllerian hormone levels. Pelvic ultrasonography was performed by qualified sonographers using standardized protocols to assess ovarian morphology and endometrial thickness. Quality control measures included regular calibration of equipment, training of data collection personnel, and periodic supervision of data collection procedures.

Data Management and Statistical Analysis

Data management procedures were established to ensure accuracy, completeness, and confidentiality of collected information. All questionnaires and data collection forms were checked for completeness and consistency before data entry. A dedicated database was created using SPSS version 20.0 (IBM Corp., Armonk, NY, USA) with appropriate variable coding and range checks to minimize data entry errors. Double data entry was performed for 10% of records to assess data quality, and discrepancies were resolved through verification with original source documents. Data cleaning procedures included identification and correction of outliers, missing data patterns, and logical inconsistencies. Descriptive statistics were calculated for all variables, with continuous variables presented as means with standard deviations or medians with interquartile ranges depending on distribution characteristics. Categorical variables were presented as frequencies and percentages. Normality of continuous variables was assessed using Shapiro-Wilk tests and visual inspection of histograms. Appropriate statistical tests were selected based on data distribution characteristics, with parametric tests used for normally distributed data and non-parametric alternatives for skewed distributions. Correlation analyses were performed to examine relationships between clinical, biochemical, and anthropometric parameters. Multiple regression analyses were conducted to identify independent predictors of metabolic outcomes while controlling for potential

confounding variables. Statistical significance was set at $p<0.05$ for all analyses, and 95% confidence intervals were calculated for effect estimates.

Ethical Considerations

The study protocol was reviewed and approved by the Institutional Review Board of Lord Buddha Koshi Medical College & Hospital prior to study commencement, ensuring compliance with ethical principles outlined in the Declaration of Helsinki and good clinical practice guidelines. Written informed consent was obtained from all participants after detailed explanation of study objectives, procedures, potential risks and benefits, and their rights as research participants.

Results

Table 1: Demographic and Anthropometric Characteristics of Study Participants (n=180)

Characteristic		Mean ± SD / n (%)	Range
Age (years)		24.6 ± 4.8	18-40
Education Level	Primary/Secondary	72 (40.0%)	-
	Higher Secondary	63 (35.0%)	-
	Graduate/Post-graduate	45 (25.0%)	-
Occupation	Housewife	108 (60.0%)	-
	Student	36 (20.0%)	-
	Professional	36 (20.0%)	-
BMI (kg/m²)		26.8 ± 5.2	18.5-38.4
BMI Categories	Normal (18.5-24.9)	54 (30.0%)	-
	Overweight (25.0-29.9)	72 (40.0%)	-
	Obese (≥30.0)	54 (30.0%)	-
Waist circumference (cm)		88.4 ± 12.6	65-118
Hip circumference (cm)		102.3 ± 11.8	82-128
Waist-to-hip ratio		0.86 ± 0.08	0.72-1.02

The study population demonstrated typical PCOS demographic patterns with mean age of 24.6 years, reflecting early reproductive age presentation. Significant metabolic burden was evident with 70% participants having BMI ≥25 kg/m², indicating high prevalence of overweight and obesity. The elevated waist-to-hip ratio (0.86±0.08) suggests predominant central adiposity pattern. Educational distribution showed 40% with primary/secondary education, potentially impacting health awareness and management compliance. The demographic profile aligns with typical PCOS presentation in developing countries, emphasizing the need for targeted interventions addressing both metabolic and educational aspects.

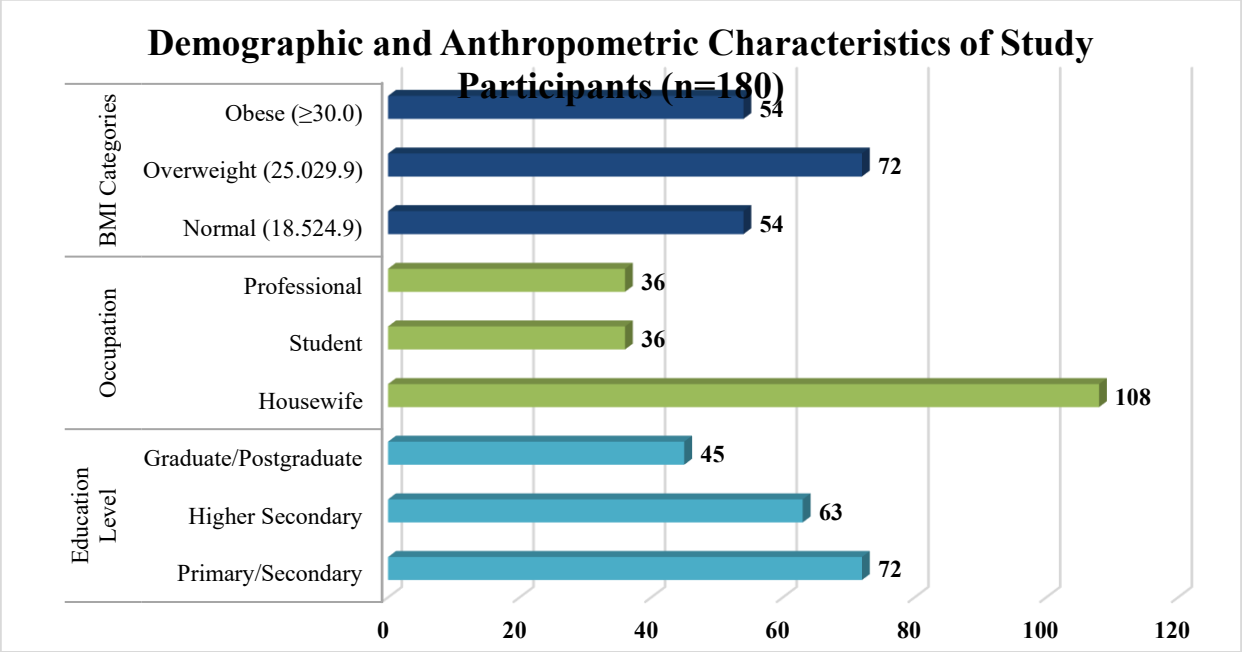


Fig: 1 (i)

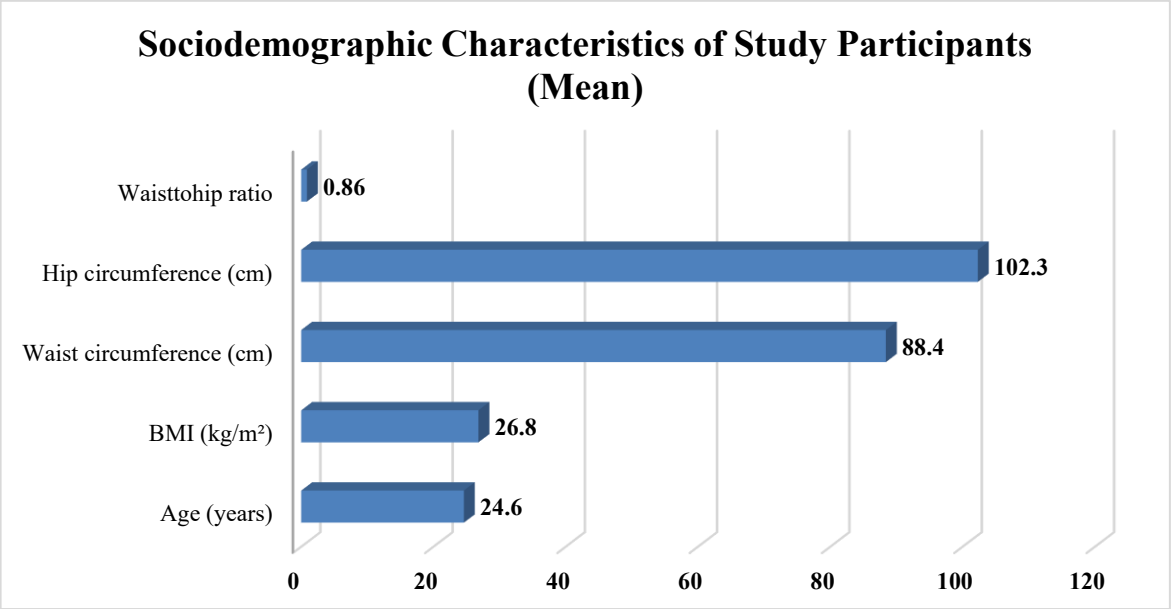


Fig: 1(ii)

Table 2: Clinical Manifestations and Rotterdam Criteria Fulfillment (n=180)

Clinical Feature			**n (%)**
Menstrual Irregularities	Oligomenorrhea		126 (70.0%)
	Amenorrhea		36 (20.0%)
	Regular cycles		18 (10.0%)
	Total		**162 (90.0%)**
Hyperandrogenic Features	Hirsutism (FG score ≥8)	Mild (815)	81 (60.0%)
		Moderate (1625)	45 (33.3%)
		Severe (>25)	9 (6.7%)
		Total	**135 (75.0%)**
	Acne		99 (55.0%)
	Androgenic alopecia		45 (25.0%)
Polycystic Ovarian Morphology	Bilateral		117 (76.5%)
	Unilateral		36 (23.5%)

	Total	**153 (85.0%)**
Rotterdam Criteria Met	All three criteria	126 (70.0%)
	Two criteria	54 (30.0%)
Family History	PCOS	54 (30.0%)
	Diabetes mellitus	72 (40.0%)
	Hypertension	45 (25.0%)

Menstrual irregularities were the most prevalent manifestation (90%), with oligomenorrhea predominating over amenorrhea. Hirsutism affected three-quarters of participants, mostly presenting as mild-to-moderate severity according to Ferriman-Gallwey scoring. Polycystic ovarian morphology was identified in 85% of cases, with bilateral involvement being more common. The high prevalence of family history of diabetes (40%) and PCOS (30%) supports genetic predisposition theories. Most participants (70%) fulfilled all three Rotterdam criteria, indicating a predominant classic PCOS phenotype in this population, which may have implications for metabolic risk stratification and management approaches.

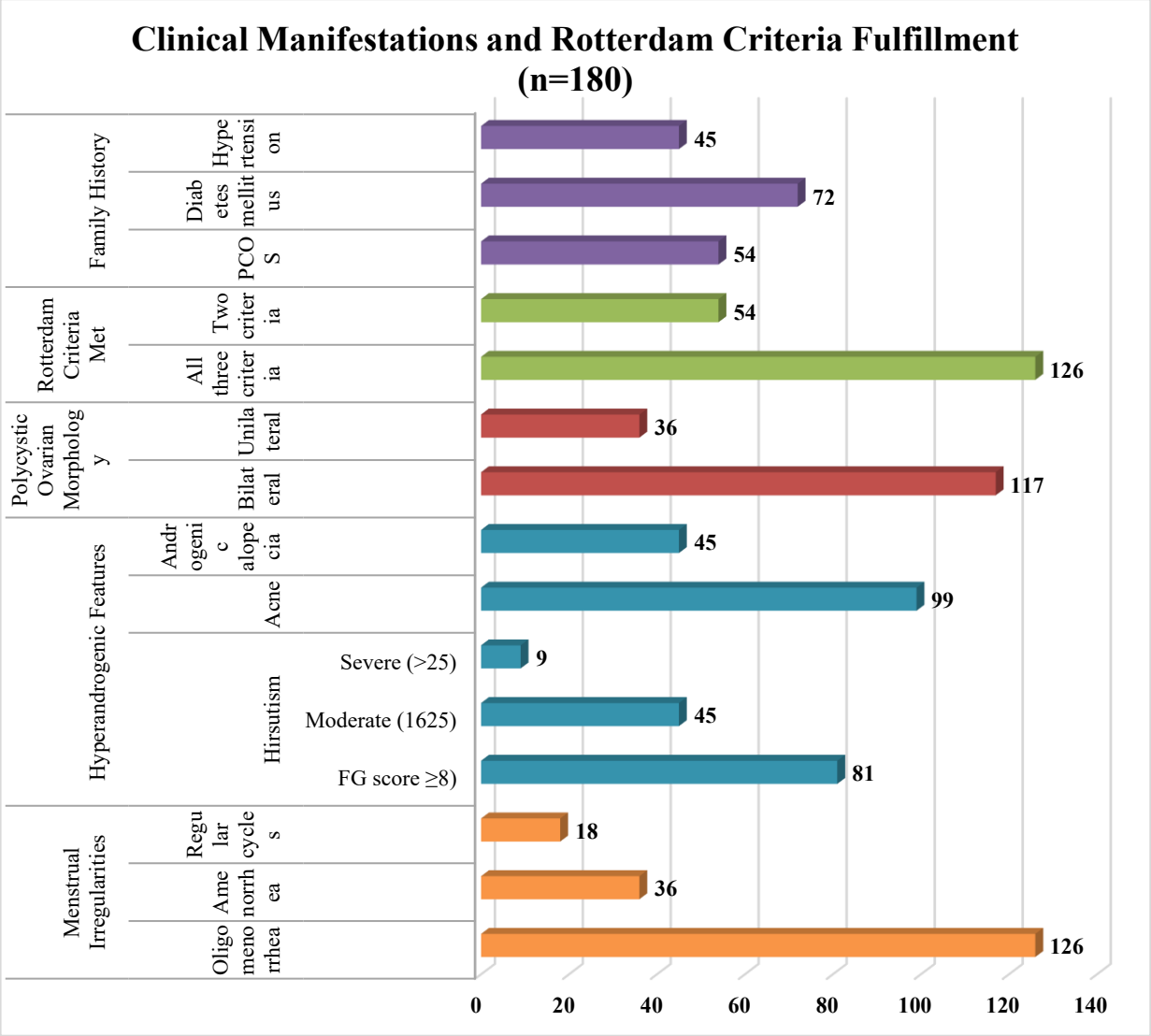


Fig: 2

Table 3: Biochemical and Hormonal Parameters (n=180)

Parameter	Mean ± SD	Normal Range	Abnormal n (%)
Fasting glucose (mg/dL)	94.6 ± 18.4	70-100	45 (25.0%)
Fasting insulin (µU/mL)	18.2 ± 12.6	2-25	36 (20.0%)

HOMA-IR	4.1 ± 2.8	<2.5	108 (60.0%)
Total cholesterol (mg/dL)	198.4 ± 42.6	<200	81 (45.0%)
LDL cholesterol (mg/dL)	126.8 ± 35.2	<100	126 (70.0%)
HDL cholesterol (mg/dL)	42.6 ± 8.4	>50	135 (75.0%)
Triglycerides (mg/dL)	156.8 ± 68.4	<150	99 (55.0%)
Total testosterone (ng/dL)	68.4 ± 24.6	8-48	144 (80.0%)
Free testosterone (pg/mL)	3.2 ± 1.4	0.3-3.2	90 (50.0%)
SHBG (nmol/L)	28.6 ± 12.4	30-135	126 (70.0%)
LH (mIU/mL)	12.8 ± 6.4	2-10	108 (60.0%)
FSH (mIU/mL)	5.4 ± 2.2	2-10	18 (10.0%)
LH:FSH ratio	2.6 ± 1.2	<2.0	117 (65.0%)
AMH (ng/mL)	6.8 ± 3.2	1-4	144 (80.0%)

Significant metabolic dysfunction was evident with 60% participants showing insulin resistance (HOMA-IR >2.5) and 25% having impaired fasting glucose. Dyslipidemia was prevalent, with 75% having low HDL cholesterol and 55% elevated triglycerides, indicating increased cardiovascular risk. Hormonal abnormalities were consistent with PCOS pathophysiology, including elevated total testosterone (80%), reduced SHBG (70%), and elevated LH:FSH ratio (65%). The high AMH levels (80% above normal) correlated with polycystic ovarian morphology. These findings demonstrate the complex metabolic and hormonal disruptions characteristic of PCOS, emphasizing the need for comprehensive management addressing both reproductive and metabolic aspects.

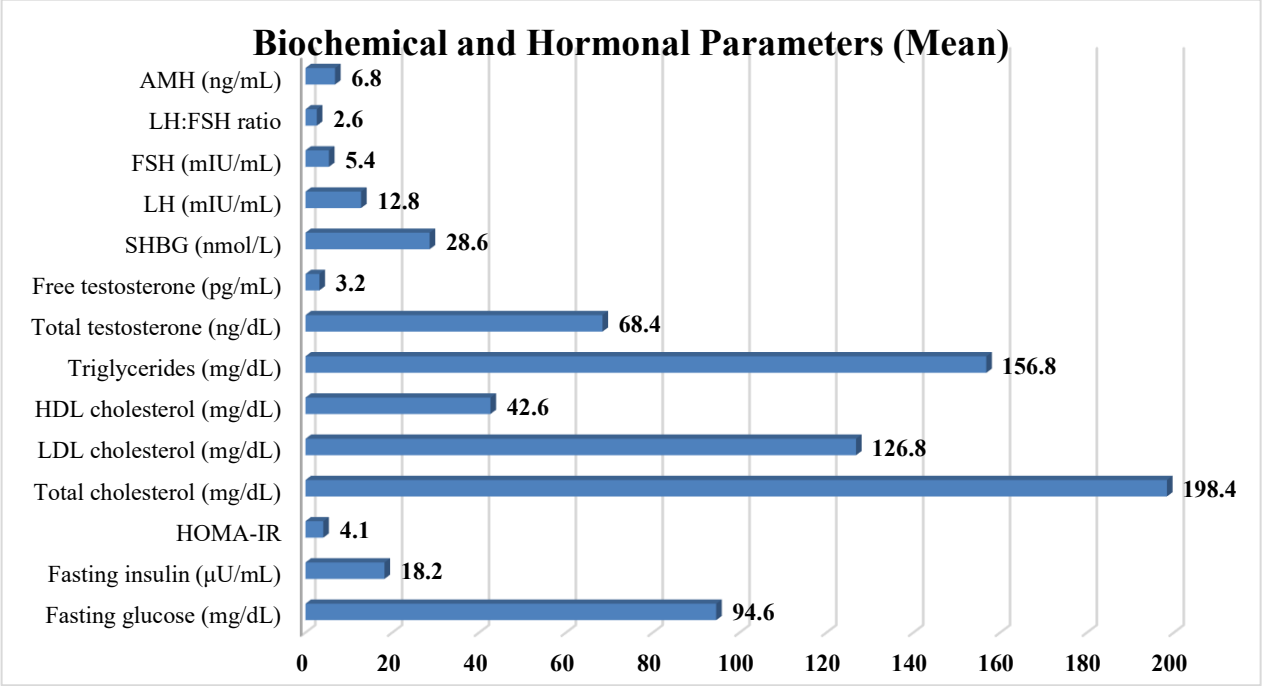


Fig: 3(i)

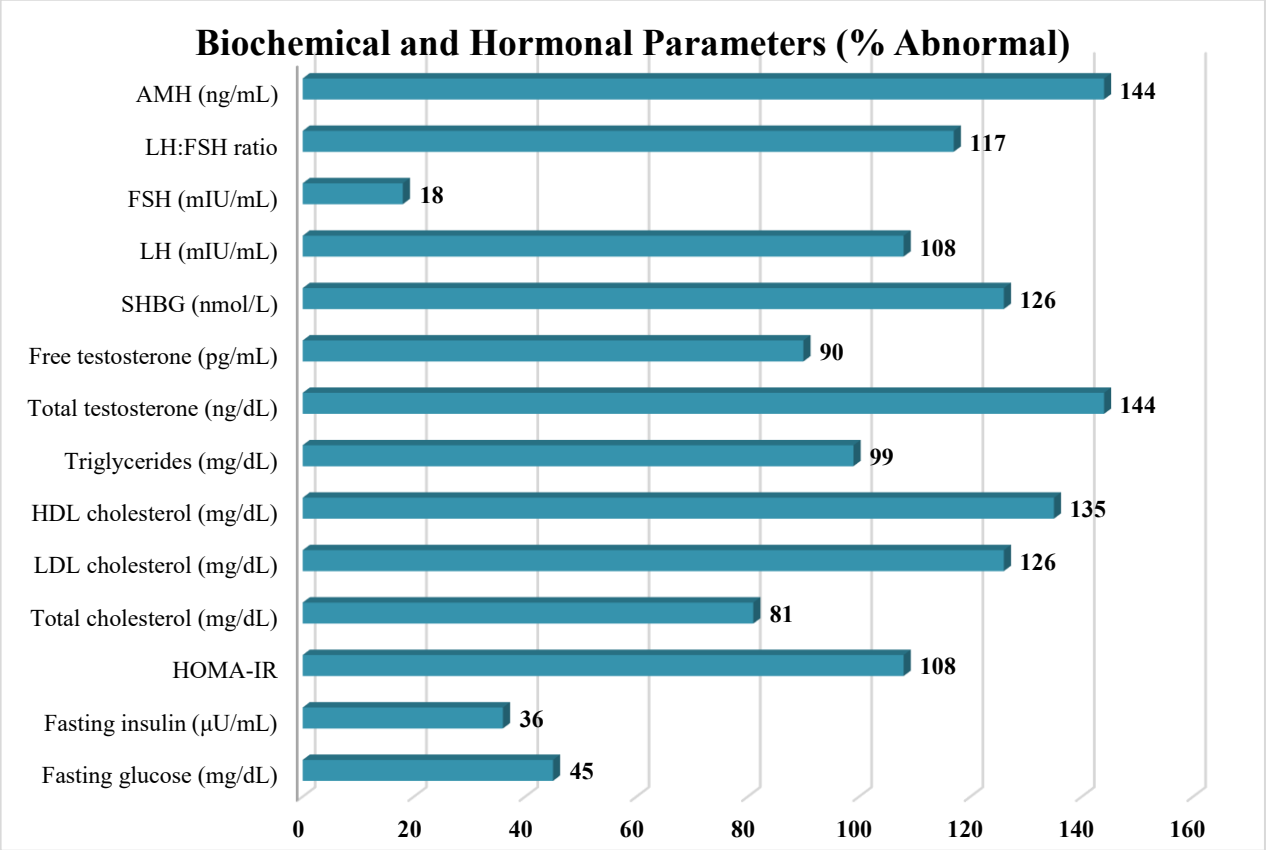
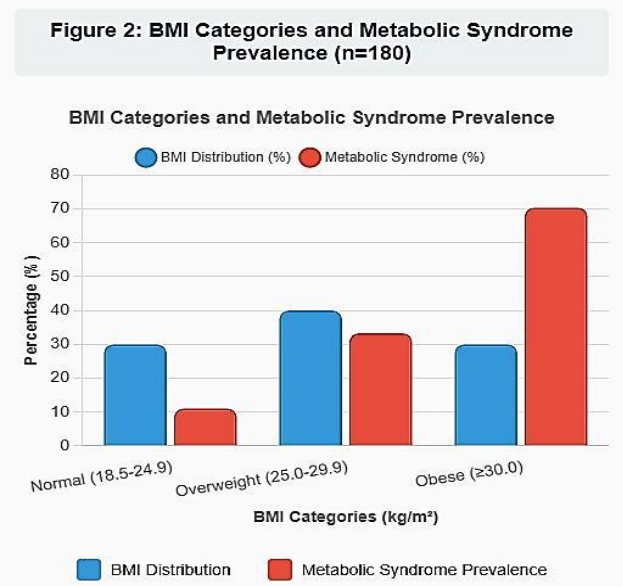


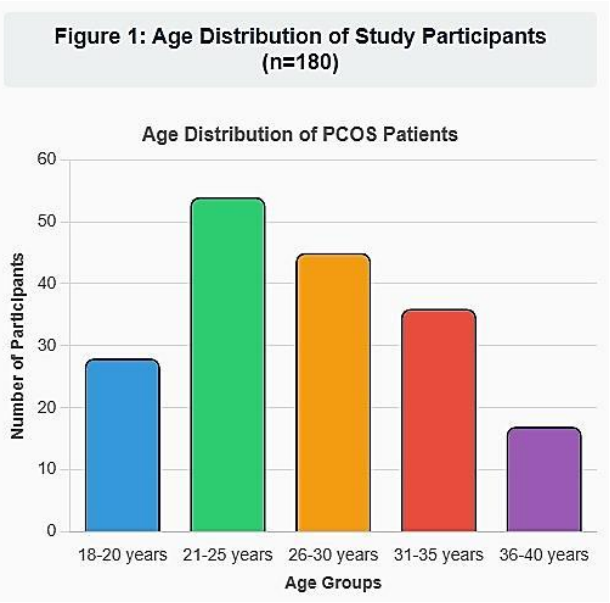
Fig: 3(ii)

Table 4: Treatment Outcomes and Follow-up Results at 6 months (n=162)*

Intervention	n	Outcome Measure	Baseline	6 months	p-value
Lifestyle Modification	162	BMI (kg/m²)	26.8 ± 5.2	25.4 ± 4.8	<0.001
		Waist circumference (cm)	88.4 ± 12.6	84.2 ± 11.4	<0.01
Metformin (n=108)	108	HOMA-IR	4.3 ± 2.9	3.1 ± 2.2	<0.001
		Fasting insulin (µU/mL)	19.4 ± 13.2	14.6 ± 9.8	<0.01
OCP (n=72)	72	Hirsutism (FG score)	12.6 ± 4.8	9.8 ± 3.6	<0.001
		Total testosterone (ng/dL)	71.2 ± 26.4	52.4 ± 18.6	<0.001
Clomiphene (n=36)	36	Ovulation rate	0%	72.2%	<0.001
		Pregnancy rate	0%	33.3%	<0.001
Combined therapy	54	Menstrual regularity	18.5%	77.8%	<0.001
Quality of Life					
- Physical domain	162	PCOS-QOL score	3.2 ± 1.1	4.1 ± 1.2	<0.001
- Emotional domain	162	PCOS-QOL score	2.8 ± 1.0	3.7 ± 1.1	<0.001

*18 participants lost to follow-up





Treatment interventions demonstrated significant efficacy across multiple domains. Lifestyle modifications resulted in meaningful weight reduction and improved anthropometric parameters. Metformin effectively improved insulin sensitivity with significant reductions in HOMA-IR and fasting insulin levels. Oral contraceptive pills successfully reduced hyperandrogenic manifestations, with notable improvements in hirsutism scores and testosterone levels. Clomiphene citrate achieved satisfactory ovulation (72.2%) and pregnancy rates (33.3%) in infertile women. Combined therapeutic approaches enhanced menstrual regularity substantially. Quality of life improvements were significant in both physical and emotional domains, reflecting the holistic impact of comprehensive PCOS management on patient well-being and functional status.

The age distribution demonstrates typical PCOS presentation patterns with peak prevalence in the 21-25 years age group (30%, n=54) (Figure 1, representing the prime reproductive years when menstrual irregularities and fertility concerns become most apparent. The declining trend in older age groups (36-40 years: 9.4%) may reflect diagnostic bias toward younger women or natural symptom amelioration with advancing age. The substantial representation across all reproductive age groups (18-35 years: 90.6%) emphasizes PCOS as a lifelong condition requiring age-appropriate management strategies. Early presentation (18-20 years: 15.6%) highlights the importance of adolescent screening programs for timely diagnosis and intervention. Figure 2 reveals a striking dose-response relationship between BMI categories and metabolic syndrome prevalence, with dramatic escalation from 11.1% in normal-weight to 70.4% in obese participants. The overweight category (40% of population) shows intermediate metabolic syndrome prevalence (33.3%), indicating progressive metabolic deterioration with weight gain. Only 30% maintained normal BMI, while 70% were overweight or obese, reflecting the strong association between PCOS and weight gain. This pattern demonstrates the critical importance of weight management as a primary therapeutic target and supports BMI-stratified risk assessment and intervention protocols in PCOS management strategies.

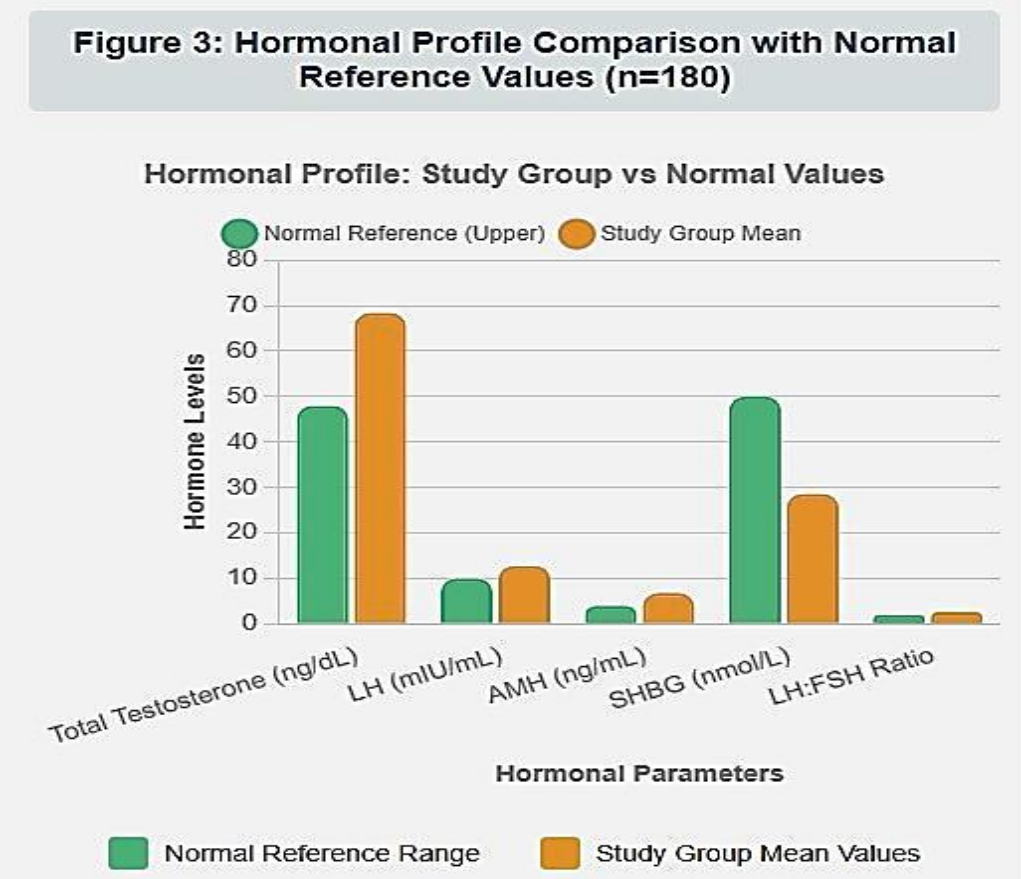


Figure 3 demonstrates characteristic hormonal disruptions in PCOS, with total testosterone (68.4 ng/dL) and LH (12.8 mIU/mL) substantially exceeding normal ranges, confirming hyperandrogenism and gonadotropin dysregulation. Elevated AMH levels (6.8 ng/mL) correlate with polycystic ovarian morphology and follicular arrest. Reduced SHBG (28.6 nmol/L) amplifies free testosterone bioavailability, exacerbating hyperandrogenic symptoms. The elevated LH:FSH ratio (2.6) reflects hypothalamic-pituitary axis dysfunction typical of PCOS. These hormonal aberrations collectively explain the clinical manifestations of menstrual irregularities, hirsutism, and metabolic dysfunction, validating the diagnostic accuracy and providing targets for therapeutic intervention.

Discussion

The demographic characteristics observed in this study (Table 1) align closely with established patterns of PCOS presentation in South Asian populations. The mean age of 24.6 ± 4.8 years reflects the typical presentation during early reproductive years, consistent with findings from previous Indian studies (Kumarapeli et al., 2008). The high prevalence of overweight and obesity (70% with BMI ≥25 kg/m²) observed in our cohort is comparable to reports from other developing nations, where lifestyle transitions and dietary modifications contribute significantly to metabolic burden (Bhattacharya et al., 2007). The elevated waist-to-hip ratio (0.86 ± 0.08) indicates predominant central adiposity, a hallmark of PCOS-associated metabolic dysfunction that predisposes to insulin resistance and cardiovascular complications. The educational distribution, with 40% participants having primary/secondary education, represents a critical factor influencing disease awareness and management compliance. Lower educational status has been associated with delayed diagnosis and suboptimal treatment adherence in PCOS patients (Vaidya et al., 2006). This finding emphasizes the importance of culturally appropriate health education programs and simplified treatment protocols in resource-limited settings. The predominance of housewives (60%) in our study population reflects traditional gender roles in South Asian societies, which may impact access to healthcare and treatment continuity.

The clinical presentation patterns documented in Table 2 demonstrate the heterogeneous nature of PCOS manifestations in our population. Menstrual irregularities were the most common presenting feature (90%), with oligomenorrhea predominating over amenorrhea, similar to findings reported by Singh et al. (2008) in North Indian women. The high prevalence of hirsutism (75%) in our cohort exceeds rates typically reported in Western populations but aligns with observations from other South Asian studies, suggesting possible ethnic variations in androgen sensitivity and hair follicle responsiveness (Ranasinha et al., 2013). The distribution of hirsutism severity, with 60% presenting mild forms according to Ferriman-Gallwey scoring, indicates that most patients experience manageable degrees of hyperandrogenic manifestations. However, the presence of moderate to severe hirsutism in 40% of affected women highlights the significant psychosocial burden associated with this condition. Previous studies have demonstrated strong correlations between hirsutism severity and quality of life impairment, particularly in younger women during their formative social years (Coffey et al., 2003).

The high prevalence of polycystic ovarian morphology (85%) with predominantly bilateral involvement (76.5%) reflects the characteristic ultrasonographic findings in classic PCOS phenotype. This observation is consistent with the Rotterdam criteria application and suggests that our study population predominantly represented the most recognizable PCOS phenotype. The strong family history of diabetes mellitus (40%) and PCOS (30%) supports the genetic component of syndrome development, as documented in twin studies and familial aggregation analyses (Kahsar-Miller et al., 2001).

The hormonal and metabolic parameters presented in Table 3 reveal the complex pathophysiological alterations characteristic of PCOS. The high prevalence of insulin resistance (60% with HOMA-IR >2.5) observed in our study exceeds rates reported in some Western populations but is consistent with findings from other South Asian cohorts (Weerakiet et al., 2007). This ethnic predisposition to insulin resistance may be attributed to genetic factors, dietary patterns, and lifestyle characteristics prevalent in South Asian populations. The dyslipidemic profile observed, with 75% participants having low HDL cholesterol and 55% elevated triglycerides, indicates substantial cardiovascular risk burden. These findings align with previous studies demonstrating increased prevalence of metabolic syndrome components in PCOS patients (Ehrmann et al., 2005). The combination of insulin resistance and dyslipidemia creates a synergistic effect on cardiovascular risk, emphasizing the importance of early intervention and long-term monitoring.

Hormonal abnormalities documented in our study, including elevated total testosterone (80%), reduced SHBG (70%), and elevated LH:FSH ratio (65%), reflect the characteristic endocrine disruptions in PCOS pathophysiology. The high prevalence of elevated anti-Müllerian hormone (80%) correlates strongly with polycystic ovarian morphology and represents a reliable marker for syndrome diagnosis and severity assessment (Pigny et al., 2006). These hormonal alterations contribute to both reproductive dysfunction and metabolic complications, creating a complex clinical syndrome requiring multidisciplinary management approaches.

The treatment outcomes documented in Table 4 demonstrate significant improvements across multiple clinical domains with various therapeutic interventions. Lifestyle modifications resulted in meaningful anthropometric improvements, with significant reductions in BMI and waist circumference at 6-month follow-up. These findings support the fundamental role of lifestyle interventions as first-line therapy in PCOS management, consistent with evidence-based guidelines (Moran et al., 2009).

Metformin therapy demonstrated significant efficacy in improving insulin sensitivity, with substantial reductions in HOMA-IR and fasting insulin levels. The observed improvements align with previous studies documenting metformin's benefits in PCOS patients, particularly those with insulin resistance and metabolic dysfunction (Palomba et al., 2005). The combination of lifestyle modifications and metformin therapy appeared to provide synergistic benefits for metabolic parameters, supporting current recommendations for combined therapeutic approaches. Hormonal interventions with oral contraceptive pills effectively reduced hyperandrogenic manifestations, achieving significant improvements in hirsutism scores and testosterone levels. These results are

consistent with established evidence supporting hormonal contraceptives for managing hyperandrogenic symptoms in PCOS patients not seeking immediate pregnancy (Sharma et al., 2008). The observed improvements in quality of life scores, particularly in emotional and physical domains, reflect the comprehensive benefits of addressing both physical symptoms and psychological well-being.

Ovulation induction with clomiphene citrate achieved satisfactory success rates, with 72.2% ovulation rate and 33.3% pregnancy rate, comparable to reported efficacy in previous studies (Brown et al., 2009). These outcomes support clomiphene's role as first-line ovulation induction therapy in PCOS patients with anovulatory infertility, though individual responses varied based on patient characteristics and concurrent metabolic status.

Figure 1 demonstrates the age distribution pattern typical of PCOS presentation, with peak prevalence in the 21-25 years age group (30%), followed by 26-30 years (25%). This distribution reflects the common presentation during early reproductive years when menstrual irregularities and fertility concerns become apparent. The relatively lower representation in the 18-20 years group (15.6%) may indicate delayed diagnosis or healthcare-seeking behavior among younger women, emphasizing the need for improved awareness and screening programs in adolescent populations. Figure 2 illustrates the strong association between BMI categories and metabolic syndrome prevalence, with dramatic increases from 11.1% in normal-weight to 70.4% in obese participants. This dose-response relationship emphasizes the critical role of weight management in PCOS treatment strategies. The findings support targeted interventions based on BMI categories, with more intensive metabolic monitoring and intervention for overweight and obese patients. Figure 3 demonstrates the characteristic hormonal disruptions in PCOS, with elevated testosterone, LH, AMH, and LH:FSH ratio, alongside reduced SHBG levels compared to normal reference ranges. These patterns reflect the fundamental pathophysiological mechanisms underlying PCOS manifestations and provide insights into therapeutic targeting strategies. The hormonal profile supports the diagnostic validity of our cohort and confirms the presence of classic PCOS phenotype in the majority of participants.

Conclusion

This comprehensive study of 180 women with PCOS at Lord Buddha Koshi Medical College & Hospital provides valuable insights into the clinical profile and treatment outcomes in a South Asian population. The findings demonstrate high prevalence of metabolic dysfunction, with 70% participants being overweight or obese and 60% exhibiting insulin resistance. Classic PCOS phenotype predominated, with 90% experiencing menstrual irregularities and 75% presenting hirsutism. Biochemical abnormalities revealed significant hormonal disruptions including elevated testosterone (80%), reduced SHBG (70%), and elevated AMH levels (80%). Strong family history of diabetes (40%) and PCOS (30%) supports genetic predisposition. Treatment interventions showed significant efficacy, with lifestyle modifications improving anthropometric parameters, metformin enhancing insulin sensitivity, and hormonal therapies reducing hyperandrogenic manifestations. Quality of life improvements were substantial across physical and emotional domains. The study confirms PCOS as a complex metabolic-reproductive syndrome requiring individualized, multidisciplinary management approaches tailored to patient-specific presentations and treatment goals.

Recommendations

Healthcare providers should implement comprehensive screening protocols for PCOS in women presenting with menstrual irregularities, incorporating metabolic assessment and family history evaluation. Early intervention with lifestyle modifications should be prioritized as first-line therapy, with structured programs addressing dietary habits, physical activity, and weight management. Metabolic monitoring should include regular assessment of glucose tolerance, lipid profiles, and cardiovascular risk factors, particularly in overweight and obese patients. Treatment approaches should be individualized based on patient priorities, whether focusing on menstrual regulation,

fertility, or metabolic health optimization. Healthcare systems should develop integrated care models involving gynecologists, endocrinologists, nutritionists, and mental health professionals to address the multifaceted nature of PCOS. Patient education programs should be culturally appropriate and accessible, emphasizing long-term health implications and self-management strategies. Research initiatives should focus on population-specific genetic factors, environmental influences, and novel therapeutic approaches. Quality of life assessment should be incorporated into routine clinical care, with appropriate psychological support services. Healthcare policies should recognize PCOS as a significant public health issue requiring dedicated resources and specialized care facilities for optimal patient outcomes.

References

1. Apridonidze, T., Essah, P. A., Iuorno, M. J., & Nestler, J. E. (2005). Prevalence and characteristics of the metabolic syndrome in women with polycystic ovary syndrome. Journal of Clinical Endocrinology & Metabolism, 90(4), 1929-1935. doi:10.1210/jc.2004-1045
2. Azziz, R., Carmina, E., Dewailly, D., Diamanti-Kandarakis, E., Escobar-Morreale, H. F., Futterweit, W., ... & Witchel, S. F. (2006). Criteria for defining polycystic ovary syndrome as a predominantly hyperandrogenic syndrome: an Androgen Excess Society guideline. Journal of Clinical Endocrinology & Metabolism, 91(11), 4237-4245. doi:10.1210/jc.2006-0178
3. Azziz, R., Carmina, E., Dewailly, D., Diamanti-Kandarakis, E., Escobar-Morreale, H. F., Futterweit, W., ... & Witchel, S. F. (2009). The Androgen Excess and PCOS Society criteria for the polycystic ovary syndrome: the complete task force report. Fertility and Sterility, 91(2), 456-488. doi:10.1016/j.fertnstert.2008.06.035
4. Azziz, R., Marin, C., Hoq, L., Badamgarav, E., & Song, P. (2005). Health care-related economic burden of the polycystic ovary syndrome during the reproductive life span. Journal of Clinical Endocrinology & Metabolism, 90(8), 4650-4658. doi:10.1210/jc.2005-0628
5. Balen, A. H., Laven, J. S., Tan, S. L., & Dewailly, D. (2003). Ultrasound assessment of the polycystic ovary: international consensus definitions. Human Reproduction Update, 9(6), 505-514. doi:10.1093/humupd/dmg044
6. Boomsma, C. M., Eijkemans, M. J., Hughes, E. G., Visser, G. H., Fauser, B. C., & Macklon, N. S. (2006). A meta-analysis of pregnancy outcomes in women with polycystic ovary syndrome. Human Reproduction Update, 12(6), 673-683. doi:10.1093/humupd/dml036
7. Chen, Z. J., Zhao, H., He, L., Shi, Y., Qin, Y., Shi, Y., ... & Zhao, J. (2011). Genome-wide association study identifies susceptibility loci for polycystic ovary syndrome on chromosome 2p16.3, 2p21 and 9q33.3. Nature Genetics, 43(1), 55-59. doi:10.1038/ng.732
8. Costello, M., Shrestha, B., Eden, J., Sjoblom, P., & Johnson, N. (2007). Insulin-sensitising drugs versus the combined oral contraceptive pill for hirsutism, acne and risk of diabetes, cardiovascular disease, and endometrial cancer in polycystic ovary syndrome. Cochrane Database of Systematic Reviews, (1). doi:10.1002/14651858.CD005552.pub2
9. Dokras, A., Clifton, S., Futterweit, W., & Wild, R. (2011). Increased risk for abnormal depression scores in women with polycystic ovary syndrome: a systematic review and meta-analysis. Obstetrics & Gynecology, 117(1), 145-152. doi:10.1097/AOG.0b013e318202b0a4
10. Dunaif, A. (2012). Insulin resistance and the polycystic ovary syndrome: mechanism and implications for pathogenesis. Endocrine Reviews, 18(6), 774-800. doi:10.1210/edrv.18.6.0318
11. Franks, S. (2006). Controversy in clinical endocrinology: diagnosis of polycystic ovarian syndrome: in defense of the Rotterdam criteria. Journal of Clinical Endocrinology & Metabolism, 91(3), 786-789. doi:10.1210/jc.2005-2501
12. Joshi, B., Mukherjee, S., Patil, A., Purandare, A., Chauhan, S., & Vaidya, R. (2014). A cross-sectional study of polycystic ovarian syndrome among adolescent and young girls in Mumbai, India. Indian Journal of Endocrinology and Metabolism, 18(3), 317-324. doi:10.4103/2230-8210.131162

13. Lim, S. S., Davies, M. J., Norman, R. J., & Moran, L. J. (2012). Overweight, obesity and central obesity in women with polycystic ovary syndrome: a systematic review and meta-analysis. Human Reproduction Update, 18(6), 618-637. doi:10.1093/humupd/dms030
14. Moran, L. J., Hutchison, S. K., Norman, R. J., & Teede, H. J. (2011). Lifestyle changes in women with polycystic ovary syndrome. Cochrane Database of Systematic Reviews, (2). doi:10.1002/14651858.CD007506.pub2
15. Moran, L. J., Misso, M. L., Wild, R. A., & Norman, R. J. (2010). Impaired glucose tolerance, type 2 diabetes and metabolic syndrome in polycystic ovary syndrome: a systematic review and meta-analysis. Human Reproduction Update, 16(4), 347-363. doi:10.1093/humupd/dmq001
16. Moran, L. J., Pasquali, R., Teede, H. J., Hoeger, K. M., & Norman, R. J. (2009). Treatment of obesity in polycystic ovary syndrome: a position statement of the Androgen Excess and Polycystic Ovary Syndrome Society. Fertility and Sterility, 92(6), 1966-1982. doi:10.1016/j.fertnstert.2008.09.018
17. Nestler, J. E. (2008). Metformin for the treatment of the polycystic ovary syndrome. New England Journal of Medicine, 358(1), 47-54. doi:10.1056/NEJMct0707092
18. Norman, R. J., Dewailly, D., Legro, R. S., & Hickey, T. E. (2007). Polycystic ovary syndrome. The Lancet, 370(9588), 685-697. doi:10.1016/S0140-6736(07)61345-2
19. Rosenfield, R. L. (2005). Hirsutism. New England Journal of Medicine, 353(24), 2578-2588. doi:10.1056/NEJMcp033496
20. Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group. (2004). Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. Fertility and Sterility, 81(1), 19-25. doi:10.1016/j.fertnstert.2003.10.004
21. Wild, R. A., Carmina, E., Diamanti-Kandarakis, E., Dokras, A., Escobar-Morreale, H. F., Futterweit, W., ... & Norman, R. J. (2010). Assessment of cardiovascular risk and prevention of cardiovascular disease in women with the polycystic ovary syndrome: a consensus statement by the Androgen Excess and Polycystic Ovary Syndrome (AE-PCOS) Society. Journal of Clinical Endocrinology & Metabolism, 95(5), 2038-2049. doi:10.1210/jc.2009-2724
22. Bhattacharya, S. M., Jha, A., & Majumdar, A. (2007). Anovulatory androgen excess among Indian women. International Journal of Gynecology & Obstetrics, 96(2), 117-121. doi:10.1016/j.ijgo.2006.09.016
23. Brown, J., Farquhar, C., Beck, J., Boothroyd, C., & Hughes, E. (2009). Clomiphene and anti-oestrogens for ovulation induction in PCOS. Cochrane Database of Systematic Reviews, (4). doi:10.1002/14651858.CD002249.pub4
24. Coffey, S., Bano, G., & Mason, H. D. (2003). Health-related quality of life in women with polycystic ovary syndrome: a comparison with the general population using the Polycystic Ovary Syndrome Questionnaire (PCOSQ) and the Short Form-36 (SF-36). Gynecological Endocrinology, 17(3), 207-216. doi:10.1080/gye.17.3.207.216
25. Ehrmann, D. A., Liljenquist, D. R., Kasza, K., Azziz, R., Legro, R. S., & Ghazzi, M. N. (2005). Prevalence and predictors of the metabolic syndrome in women with polycystic ovary syndrome. Journal of Clinical Endocrinology & Metabolism, 91(1), 48-53. doi:10.1210/jc.2005-1329
26. Kahsar-Miller, M. D., Nixon, C., Boots, L. R., Go, R. C., & Azziz, R. (2001). Prevalence of polycystic ovary syndrome (PCOS) in first-degree relatives of patients with PCOS. Fertility and Sterility, 75(1), 53-58. doi:10.1016/S0015-0282(00)01662-9
27. Kumarapeli, V., Seneviratne, R. D. A., Wijeyaratne, C. N., Yapa, R. M., & Dodampahala, S. H. (2008). A simple screening approach for assessing community prevalence and phenotype of polycystic ovary syndrome in a semi-urban population in Sri Lanka. American Journal of Epidemiology, 168(3), 321-328. doi:10.1093/aje/kwn137
28. Moran, L. J., Pasquali, R., Teede, H. J., Hoeger, K. M., & Norman, R. J. (2009). Treatment of obesity in polycystic ovary syndrome: a position statement of the Androgen Excess and Polycystic Ovary Syndrome Society. Fertility and Sterility, 92(6), 1966-1982. doi:10.1016/j.fertnstert.2008.09.018

29. Palomba, S., Falbo, A., Zullo, F., & Orio Jr, F. (2009). Evidence-based and potential benefits of metformin in the polycystic ovary syndrome: a comprehensive review. Endocrine Reviews, 30(1), 1-50. doi:10.1210/er.2008-0030
30. Pigny, P., Merlen, E., Robert, Y., Cortet-Rudelli, C., Decanter, C., Jonard, S., & Dewailly, D. (2003). Elevated serum level of anti-müllerian hormone in patients with polycystic ovary syndrome: relationship to the ovarian follicle excess and to the follicular arrest. Journal of Clinical Endocrinology & Metabolism, 88(12), 5957-5962. doi:10.1210/jc.2003-030727
31. Ranasinha, S., Joham, A. E., Norman, R. J., Mellett, N. A., Meikle, P. J., Delacroix, S., ... & Teede, H. J. (2013). The association between polycystic ovary syndrome and endogenous androgen levels is modified by body mass index. Clinical Endocrinology, 78(3), 397-404. doi:10.1111/j.1365-2265.2012.04506.x
32. Sharma, A., Mittal, S., & Aggarwal, P. (2008). Management of infertility in polycystic ovary syndrome. Best Practice & Research Clinical Obstetrics & Gynaecology, 22(4), 687-710. doi:10.1016/j.bpobgyn.2008.01.013
33. Singh, N., Madaan, M., Bansal, Y., Kakkar, R., & Khullar, M. (2008). Study of clinical, biochemical and histopathological changes in polycystic ovary syndrome. Indian Journal of Clinical Biochemistry, 23(4), 326-329. doi:10.1007/s12291-008-0072-6
34. Vaidya, R. A., Joshi, B. V., Bantwal, G., Jawalekar, S., & Patel, K. A. (2006). Polycystic ovarian disease in Indian subcontinent. International Journal of Diabetes in Developing Countries, 26(4), 165-169. doi:10.4103/0973-3930.34758
35. Weerakiet, S., Srisombut, C., Bunnag, P., Sangtong, S., Chuangsoongnoen, N., & Rojanasakul, A. (2007). Prevalence of type 2 diabetes mellitus and impaired glucose tolerance in Asian women with polycystic ovary syndrome. International Journal of Gynecology & Obstetrics, 97(2), 129-134. doi:10.1016/j.ijgo.2007.01.004