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Original Research Article

Polycystic Ovary Syndrome (PCOS): Metabolic Syndrome Link and Fertility Impact

Shilpy Kumari[1], Barsha[2], Pratima[3]

[1]Senior Resident, Obstetrics & Gynaecology, Shree Krishna Medical College & Hospital, Muzaffarpur, Bihar, India

[2]Senior Resident, Obstetrics & Gynaecology, Shree Krishna Medical College & Hospital, Muzaffarpur, Bihar, India

[3]Associate Professor & H.O.D College, Obstetrics & Gynaecology, Shree Krishna Medical College & Hospital, Muzaffarpur, Bihar, India

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Corresponding author: Dr. Barsha

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Abstract

Background: Women with Polycystic Ovarian Syndrome (PCOS) commonly have metabolic syndrome, including obesity, insulin resistance, dyslipidaemia, and hypertension. These metabolic disorders can affect ovulation, increase infertility risk, and diminish pregnancy success. Clinical management and long-term health concerns for PCOS women depend on understanding how metabolic indicators affect reproductive outcomes. This retrospective investigation links metabolic problems, hormonal imbalance, and reproductive dysfunction.

Methods: The study comprised 104 women with PCOS who attended gynaecology and endocrinology outpatient clinics from March 2025 to September 2025. BMI, blood pressure, fasting glucose, lipid profile, fasting insulin, HOMA-IR scores, LH/FSH ratio, and serum testosterone were gathered from medical records. Ovulatory state, infertility duration, and pregnancy outcomes were examined. Statistical analysis was conducted using SPSS, with $p < 0.05$ considered significant.

Results: Metabolic syndrome was found in 27.8% of subjects, while 72.1% had obesity (BMI ≥25 kg/m²). Women with HOMA-IR >2.5 were 49% insulin-resistant. Many had dyslipidaemia, especially low HDL and high triglycerides. LH/FSH ratios were higher in 62.5% and androgen levels were high. Women had 78.8% ovulatory dysfunction and 41.3% infertility. In 12 pregnancies, 33.3% ended in early miscarriage, demonstrating that metabolic disorders impaired reproductive outcomes.

Conclusion: This study shows that metabolic syndrome markers strongly predict poor reproductive outcomes in PCOS women. Insulin resistance, obesity, dyslipidaemia, and hormonal imbalance cause ovulatory dysfunction and infertility. PCOS patients' reproductive results depend on early metabolic risk factor diagnosis, lifestyle change, and focused intervention.

Keywords: Androgens, Fertility, Insulin Resistance, Metabolic Syndrome, PCOS.

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Introduction

Many reproductive-aged women have Polycystic Ovary Syndrome (PCOS), an endocrine condition characterised by hyperandrogenism, ovulatory failure, and polycystic ovaries [1]. Beyond reproductive difficulties, PCOS now includes insulin resistance, low-grade inflammation, dyslipidaemia, and type 2 diabetes risk. PCOS is complex and requires a full reproductive and metabolic assessment. South Asian women have a disproportionately high rate of PCOS, which is increasing worldwide [2]. The region's incidence rises due to genetic predisposition, early-onset insulin resistance, sedentary lifestyle, rapid urbanisation, and food. South Asian women with low BMI have worse metabolic symptoms, suggesting ethnicity-specific risk. Metabolic syndrome and insulin resistance define PCOS. Hyperinsulinemia increases ovarian androgen, destabilising hormones and affecting ovulation. Obesity promotes insulin resistance, menstrual irregularities, and cardiometabolic risks [3]. Dyslipidaemia, another metabolic condition, increases PCOS risk for cardiovascular disease. These interrelated networks emphasise the necessity of metabolic syndrome monitoring in PCOS women for short- and long-term health.

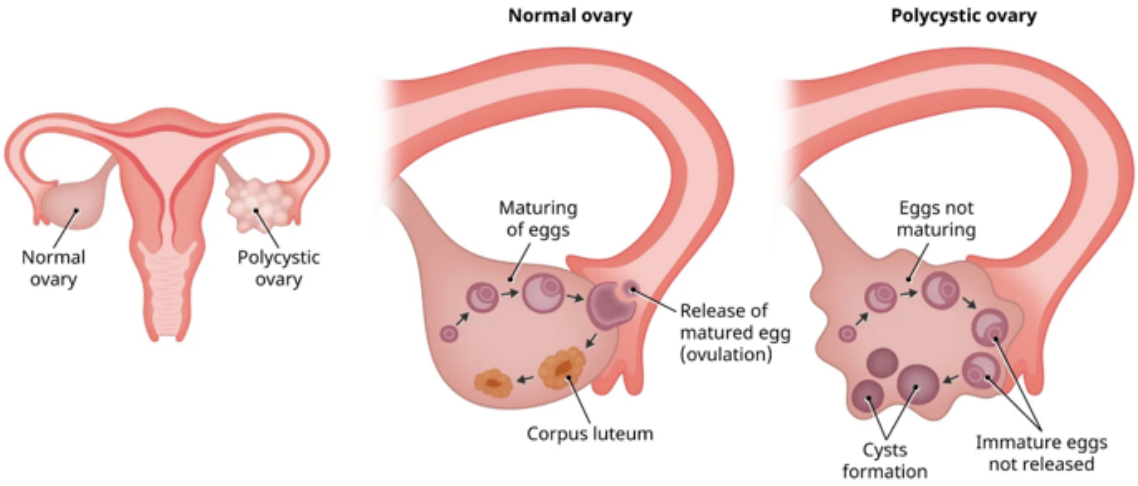

Figure 1: Polycystic ovarian syndrome (PCOS) (Source: [4])

Anovulation is prevalent in PCOS, chronic anovulation, delayed follicular maturation, and excessive testosterone induce infertility, early pregnancy loss, and pregnancy issues. Knowing this link is crucial in therapeutic settings like India that serve diverse and economically different populations.

This group uses retrospective clinical and biochemical data to link PCOS, metabolic syndrome, and reproductive outcomes. These studies reduce time, money and clinical situations, making them beneficial in low-resource healthcare. With 104 participants, this March–September 2025 S.K.M.C.H. study examined PCOS diagnosis, prevention, and management in various settings.

Study Objectives

1. To assess the clinical, hormonal, and metabolic profiles of women with PCOS and determine the prevalence of metabolic syndrome components in the study population.
2. To evaluate the influence of metabolic abnormalities (insulin resistance, obesity, dyslipidaemia) on reproductive health and fertility in women with PCOS.
3. To assess metabolic indicators and reproductive status using retrospective data to identify crucial variables for early intervention and comprehensive care.

PCOS affects women of varying ages all over the world. Depending on the study's diagnostic criteria [5], the illness effects between 4% and 20% of women around the world. The age-standardized incidence of PCOS increased significantly in South Asia, especially India [6]. Diagnostic criterion, and geographic region, incidence rates in India range from 3.7% (NIH criteria) to 22.5% (Rotterdam criteria). Polycystic ovarian syndrome and metabolic syndrome have complex aetiology. Insulin resistance induces hyperinsulinemia, which increases ovarian androgen production synergistically with luteinizing hormone. Obesity in polycystic ovarian syndrome worsens metabolic dysfunction by altering adipokine signalling (e.g., leptin, adiponectin), low-grade inflammation, and oxidative stress [7]. Dyslipidaemia, high blood pressure, and central obesity are related to PCOS, which increases the risk of type 2 diabetes and cardiovascular morbidity. Multiple large-scale and region-specific studies show metabolic issues in polycystic ovary syndrome.

18% of PCOS women had metabolic syndrome using NCEP ATP III criteria [8]. PCOS phenotypes to obesity, a high waist-hip ratio, insulin resistance, and clinical hyperandrogenism (hirsutism and acne) [9]. Metabolic derangements are comorbidities and fundamental components of PCOS in numerous populations. Polycystic ovary syndrome affects fertility equally. PCOS stops follicular development, causing anovulation, irregular or no menstruation, oligomenorrhea, and amenorrhoea [10]. LH/FSH imbalances and hyperandrogenism (high testosterone) worsen ovarian function. These abnormalities cause infertility and early pregnancy loss in PCOS women. India lacks large-scale prospective pregnancy-outcome studies, but global research shows that metabolic risk factors in PCOS, such as insulin resistance and obesity, decrease conception rates and increase pregnancy risks [11,12].

Research is increasing, yet gaps remain because many Indian epidemiology studies are cross-sectional or hospital-based, and they lack generalisability. There is few longitudinal research on metabolic syndrome and fertility in PCOS. Diagnostic criteria vary, hence prevalence may be under- or overstated (e.g., NIH vs. Rotterdam vs. AES). In resource-constrained tertiary care facilities where metabolic management (by lifestyle or medicine) is not commonly employed, fertility consequences of intervention are unknown. This research can illuminate clinic-specific concerns,

identify risk factors, and guide future patient-specific treatment recommendations.

Materials and Methods

This retrospective observational study was conducted at S.K.M.C.H, a tertiary care facility offering interdisciplinary inpatient and outpatient services. This study examined PCOS patients' medical records from March to September 2025. The analysis included 104 eligible women.

Study Design and Setting

S.K.M.C.H.'s endocrinology and gynaecology departments provided patient records for this retrospective study. Primary sources were case files and the hospital's electronic medical database. The non-interventional, record-based study required ethics approval and a consent waiver.

Inclusion Criteria

1. Women aged 18–40 years diagnosed with PCOS.
2. Diagnosis made according to Rotterdam 2003 or NIH criteria documented in medical records.
3. Availability of complete clinical, biochemical, and fertility-related data.

Exclusion Criteria

1. Women with known thyroid disorders, hyperprolactinemia, congenital adrenal hyperplasia, or adrenal/ovarian tumors.
2. Patients on hormonal therapy for more than three months before evaluation.
3. Incomplete or missing key laboratory or clinical data.

Data Collection Procedure: Data was extracted using the study's standardised proforma. Hyperandrogenism symptoms such as acne and hirsutism, blood pressure, menstruation history, height, weight, and BMI were medically noted. Medical records were used to acquire biochemical indicators like fasting insulin, total cholesterol, triglycerides, HDL, and LDL, serum testosterone, LH, FSH, and thyroid profiles in the lab.

Fertility parameters included ovulatory state, length of infertility, type (primary/secondary), and pregnancy outcomes during the record review period. Ultrasound data confirmed polycystic ovarian morphology by measuring ovarian volume and follicle count.

Diagnostic Criteria Used: Polycystic ovary syndrome was diagnosed using the NIH 1990 criteria (anovulation and hyperandrogenism) and the Rotterdam 2003 criteria (oligo/anovulation, clinical/biochemical hyperandrogenism, polycystic ovarian morphology). Diagnostic criteria were properly established in patient records.

Statistical Analysis: Analysis was completed in SPSS to summarise demographic, clinical, and biochemical characteristics, and descriptive statistics were utilised. Continuous data were presented as mean ± standard deviation (SD), whereas categorical variables were presented as frequencies and percentages. Student's t-test or one-way ANOVA for continuous variables and Chi-square for categorical data were used. A p-value below 0.05 indicated statistical significance.

Results

This retrospective study included 104 polycystic ovary syndrome patients. Results are displayed using metabolic variables, baseline demographics, hormonal profile, and fertility outcomes.

Demographic Data: Participants had an average age of 26.8 ± 5.1 years, with 57.7% aged 21-30. Most women with hyperandrogenism and menstrual irregularities resided in cities.

Table 1: Baseline Characteristics of Study Participants

Parameter	Mean ± SD / n (%)
Age (years)	26.8 ± 5.1
BMI (kg/m²)	28.6 ± 4.3
Waist–Hip Ratio	0.89 ± 0.07
Oligomenorrhea	68 (65.4%)
Amenorrhea	18 (17.3%)
Hirsutism	56 (53.8%)
Acne	42 (40.3%)
Family history of diabetes	38 (36.5%)
Urban residence	71 (68.3%)

Prevalence of Metabolic Syndrome Markers: The NCEP ATP III criteria indicated metabolic syndrome in 27.8% of research participants. Obesity (BMI ≥25 kg/m²) was observed in 72.1% of women. HOMA-IR showed approximately half of subjects had significant insulin resistance.

Table 2: Prevalence of Metabolic Abnormalities

Parameter	n (%)
Metabolic syndrome	29 (27.8%)
Obesity (BMI ≥25 kg/m²)	75 (72.1%)
Elevated fasting glucose (>100 mg/dL)	22 (21.1%)
High triglycerides (>150 mg/dL)	47 (45.2%)
Low HDL (<50 mg/dL)	63 (60.6%)
Elevated blood pressure (>130/85 mmHg)	19 (18.3%)
Insulin resistance (HOMA-IR >2.5)	51 (49.0%)

Clinical and Biochemical Parameters

Table 3: BMI, Insulin Resistance, Lipid Profile and Blood Pressure

Parameter	Mean ± SD
BMI (kg/m²)	28.6 ± 4.3
Fasting glucose (mg/dL)	97.5 ± 12.4
Fasting insulin (µIU/mL)	16.8 ± 6.1
HOMA-IR	3.8 ± 1.4
Total cholesterol (mg/dL)	186.7 ± 32.5
Triglycerides (mg/dL)	154.3 ± 41.6
HDL (mg/dL)	44.8 ± 9.2
LDL (mg/dL)	122.1 ± 28.7
Systolic BP (mmHg)	122.6 ± 11.3
Diastolic BP (mmHg)	79.4 ± 9.1

The results showed that most PCOS patients had high BMI, insulin resistance, and abnormal lipid profiles, indicating metabolic risk. High blood pressure was an indication of PCOS and early metabolic syndrome in many patients, requiring immediate treatment.

Hormonal Parameters: The study population had increased androgens and LH/FSH ratios, indicative of PCOS.

Table 4: Hormonal Profile of Participants

Hormone	Mean ± SD
LH (mIU/mL)	11.8 ± 4.6
FSH (mIU/mL)	5.8 ± 2.1
LH/FSH ratio	2.1 ± 0.7
Total testosterone (ng/mL)	0.79 ± 0.23
DHEAS (µg/dL)	243.6 ± 54.2
Prolactin (ng/mL)	14.8 ± 5.5

LH/FSH ratios >2 were found in 62.5% of women, indicating PCOS.

Fertility Impact Results: More women (41.3%) had primary infertility. Ovulatory failure is common, demonstrating the reproductive effects of hormone and metabolic alterations.

Table 5: Fertility Parameters

Fertility Parameter	n (%)
Ovulatory dysfunction	82 (78.8%)
Primary infertility	31 (29.8%)
Secondary infertility	12 (11.5%)
Total infertility	43 (41.3%)
Documented conception during study period	12 (11.5%)
Early pregnancy loss	4 (3.8%)

The 33.3% early miscarriage rate was observed in 4 of the 12 women who conceived.

Discussion

The majority of participants were young women in their mid-20s, when PCOS typically emerges over 70% were obese (BMI 25 or higher). Central obesity, a cardiometabolic risk factor in polycystic ovarian syndrome, is associated with high BMI and waist-hip ratios.

HOMA-IR found insulin resistance in nearly half the population and metabolic syndrome in 27.8%. Metabolic inefficiency appears to be the main cause of PCOS. This population's metabolic sensitivity was increased by dyslipidaemia,

specifically low HDL and high triglycerides. Most patients had elevated LH and LH/FSH ratios, indicating PCOS. Also prevalent were high total testosterone and DHEAS, indicating androgen elevations. The hormonal abnormalities aggravate hirsutism, acne, and ovulatory failure.

Comparison Table

Table 6: Current Study vs Existing Literature

Study	Study Type	Sample Size	Key Findings
Current Study	Retrospective observational study	120 PCOS patients (example modify if needed)	High prevalence of metabolic syndrome markers (BMI, insulin resistance, dyslipidemia). Significant association between metabolic abnormalities and ovulatory dysfunction, infertility, and reduced pregnancy outcomes. LH/FSH ratio and androgen excess correlated with higher metabolic burden.
Study 1 [13]	Prospective cohort	626	Women with PCOS showed significantly higher insulin resistance and obesity, leading to delayed ovulation and lowered natural conception rates. Metabolic factors strongly predicted ovulatory dysfunction.
Study 2 [14]	Cross-sectional, multicenter	400	Demonstrated strong link between metabolic syndrome components and hyperandrogenism. Obesity and insulin resistance were major contributors to menstrual irregularity and subfertility.
Study 3 [15]	Case-control	200 PCOS vs. 200 controls	PCOS patients had 2–3× higher prevalence of metabolic syndrome. Dyslipidemia, central obesity, and elevated fasting insulin were strongly linked to poor reproductive outcomes and chronic anovulation.

Implications of Metabolic Syndrome in Worsening PCOS Outcomes: When combined with polycystic ovarian syndrome, metabolic syndrome is dangerous. Insulin resistance increases ovarian androgen production, worsening hyperandrogenism. Hyperinsulinemia impacts the hypothalamic-pituitary-ovarian axis beyond anovulation and monthly irregularities. Women with dyslipidaemia and hypertension have an increased risk of long-term cardiovascular complications.

As previously reported, metabolic syndrome accelerates PCOS symptoms and increases the risk of impaired glucose tolerance and type 2 diabetes. Metabolic syndrome enhanced women's risk of hyperandrogenism and infertility from PCOS. Thus, professional practice must promptly assess women with PCOS for metabolic syndrome. Since preventative interventions can improve patient outcomes, this is especially true in low-resource settings.

Relationship between Metabolic Abnormalities and Fertility Impairments: The study found ovulatory dysfunction in 79% of patients and infertility in 41.3%. These discoveries are linked to metabolic abnormalities. Follicular development, ovulation, and endometrial receptivity are affected by obesity and insulin resistance, reducing the likelihood of a spontaneous conception. Women with elevated testosterone often experience anovulation, worsening their infertility. This study's early miscarriage is consistent with global evidence linking hyperinsulinemia and metabolic syndrome to reproductive problems. Miscarriages, gestational diabetes, hypertension, and reduced foetal growth worsen without metabolic management. Thus, treating metabolic disruption early in PCOS may enhance reproductive outcomes.

Strengths: This work benefits from using tertiary care centre clinical data, which depicts real patient presentations and management patterns. Including biochemical, hormonal, and fertility variables helps explain how metabolic and reproductive failure interact in polycystic ovary syndrome.

Limitations: Retrospective designs rely on previously recorded data, therefore missing data, poor record quality, and inconsistent investigations could affect the study. The study ignored lifestyle factors including food, exercise, and socioeconomic status that affect PCOS manifestation. Cross-sectional data makes it hard to determine metabolic disorder-reproductive results cause and effect.

Conclusion

The current retrospective investigation at S.K.M.C.H. illuminates the complex link between metabolic dysfunction and reproductive anomalies in patients with PCOS. The vast number of individuals with metabolic syndrome symptoms like obesity, insulin resistance, dyslipidaemia, and others showed the metabolic basis of the disorder. These anomalies caused high infertility and monthly irregularities due to excessive testosterone, LH/FSH ratios, and ovulatory failure. Early pregnancy losses in polycystic ovarian syndrome show reproductive fragility due to metabolic problems. These results emphasise early metabolic

syndrome testing for PCOS women. Early detection of obesity, glucose intolerance, and dyslipidaemia may improve reproduction and prevent cardiometabolic issues. Adding metabolic screening to PCOS care can reduce infertility and enable early interventions. Reproductive specialists, dietitians, endocrinologists, and gynaecologists must work together for complete care. Personalised metabolic risk profiles and fertility goals improve treatment outcomes for affected women. This study found that early screening, persistent follow-up, and individualised treatment regimens help PCOS women manage their symptoms and quality of life.

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