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Research Article

Role of Insulin Resistance and Obesity in the Pathogenesis of Menstrual Irregularities in Women with Polycystic Ovary Syndrome. Cross-Sectional Study

Shazia Saeed[1], Rozina Khan[2], Rizwana Naz[3], Shaista Gul[4], Zubia Bugti[5], Fozia Jan[6]

[1]Associate Professor, Gynecology Unit-I, Bolan Medical College, Quetta, Pakistan
[2]Associate Professor, Gynecology Unit-IV, Bolan Medical College, Quetta, Pakistan
[3]Assistant Professor, Gynecology Unit-I, Bolan Medical College, Quetta, Pakistan
[4]Associate Professor, Department of Histopathology, Bolan Medical Complex Hospital, Quetta, Pakistan
[5]Assistant Professor, Gynecology Unit-I, Sandeman Provincial Hospital, Quetta (SPHQ), Pakistan
[6]Associate Professor, Gynecology Unit-II, Bolan Medical College, Quetta, Pakistan
Corresponding Author: Fozia Jan, Associate Professor, Gynecology Unit-II, Bolan Medical College, Quetta, Pakistan, Email: drfjjsahibzada28@gmail.com

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ABSTRACT

Background: Polycystic ovary syndrome (PCOS) is a widespread endocrine-metabolic disorder among women at the age of reproductive age, and it is often linked with menstrual cycle disruptions, obesity, and insulin resistance. Such metabolic abnormalities could play a major role in ovulatory dysfunction and abnormal menstrual cyclicity.

Objective: to identify whether insulin resistance and weight gain have a role in the pathogenesis of menstrual irregularities in women with PCOS.

Methods: This was a cross-sectional study, which was carried out at the Department of Gynecology, Bolan Medical Complex Hospital, Quetta, between January 2024 and January 2025. A total of 150 women aged 18–35 years diagnosed with PCOS according to Rotterdam criteria were included. Biochemical data, anthropometric, and clinical data were recorded. Measures taken were body mass index (BMI), waist circumference, fasting blood glucose, fasting serum insulin, and Homeostatic Model Assessment of Insulin Resistance (HOMA-IR). Menstrual cycles were grouped into regular, oligomenorrhea, amenorrhea, polymenorrhea, and irregular, unpredictable cycles.

Results: Among 150 women, menstrual irregularities were found in 102 (68.0%), whereas 48 (32.0%) had comparatively regular menstrual cycles. Oligomenorrhea was the most common abnormality, observed in 69 (46.0%) women, followed by amenorrhea in 18 (12.0%). Women with menstrual irregularities had significantly higher BMI (31.2 ± 4.8 vs 26.1 ± 3.9 kg/m²), waist circumference (95.8 ± 9.1 vs 85.6 ± 7.8 cm), and HOMA-IR (4.42 ± 1.51 vs 2.71 ± 1.09) compared with women having regular cycles ($p < 0.001$). Menstrual irregularities were present in 85.5% of obese women and 81.5% of women with HOMA-IR >2.5. Predictive factors of menstrual dysfunction were obesity and insulin resistance.

Conclusion: Menstrual irregularities among women with PCOS are closely linked with insulin resistance and obesity, which seem to be major factors in the pathogenesis of ovulatory and menstrual dysfunction.

Keywords: Polycystic ovary syndrome; insulin resistance; obesity; menstrual irregularities; HOMA-IR; oligomenorrhea; amenorrhea

INTRODUCTION

Polycystic ovary syndrome (PCOS) is a primary cause of menstrual dysfunction, anovulatory infertility, and hyperandrogenic symptoms in women of reproductive age worldwide and is among the most prevalent endocrine and metabolic disorders[1]. Recent international consensus suggests that PCOS is prevalent in reproductive-aged women at a rate of about 10–13%, and this varies significantly among ethnic and regional groups. The syndrome is now being established not merely as a reproductive disorder, but also as a complicated metabolic disorder with long-term consequences on endocrine, cardiovascular, and reproductive health. Among the most common presenting complaints in PCOS women are menstrual abnormalities, including oligomenorrhea, amenorrhea, and irregular cycles, which are usually indicative of ovulatory dysfunction[2].

Pathophysiology of menstrual irregularities in PCOS is multifactorial and includes the hypothalamic-pituitary-ovarian axis, ovarian androgen excess, inability to achieve follicular

maturation, and chronic anovulation[3]. Nevertheless, there is increasing evidence to indicate that insulin resistance and obesity may be considered as some of the most significant factors contributing to the onset and the intensity of such reproductive disturbances. Hyperinsulinemia increases the ovarian androgen production, decreases the hepatic production of sex hormone-binding globulin, and aggravates the hyperandrogenic internal environment, thus disrupting the normal folliculogenesis and ovulatory cyclicity. Consequently, the menstrual dysfunction in PCOS is beginning to be considered a symptom of the overall endocrine-metabolic rift as opposed to a pure gynecological defect[4,5].

Insulin resistance is discussed as a key biological characteristic of PCOS and can be evident in both obese and non-obese females, although its clinical manifestation is usually more significant in women with excess adiposity[6]. Obesity and especially central obesity exacerbate insulin resistance and lead to a vicious cycle of metabolic and reproductive dysfunction. Growth of adipose tissue enhances inflammatory signals, changes in adipokine release, and deterioration in insulin sensitivity, which can negatively affect ovarian steroidogenesis and the menstrual cycle. These mechanisms usually lead to more serious menstrual abnormalities, higher ovulatory dysfunction, and worse reproductive outcomes in obese women with PCOS. The role of lifestyle-related weight gain is thus becoming increasingly accepted as a significant disease manifestation modulator and symptom burden in PCOS[7,8].

Clinically, most women with PCOS present to doctors with complaints of menstrual problems, although the metabolic abnormalities that accompany this condition may be either underdiagnosed or underassessed. It is particularly relevant in low- and middle-income environments where obesity, sedentary lifestyle, and delayed endocrine evaluation are becoming popular among young women. Discovering the association between metabolic dysfunction and menstrual abnormality is thus critical in enhancing early detection, risk stratification, and comprehensive care of women with PCOS[9,10].

Whereas other studies have shown that insulin resistance, obesity, and PCOS have a relationship, local clinical evidence that specifically measures their role in menstrual irregularities is scarce. This relationship is of interest because menstrual dysfunction is frequently a surface clinical sign of underlying metabolic pathology and possibly a marker of increased long-term susceptibility to infertility, impaired glucose tolerance, and cardiometabolic disease in women[11].

Therefore, the present study was conducted to assess the role of insulin resistance and obesity in the pathogenesis of menstrual irregularities among women with PCOS and to determine the extent to which these metabolic factors are associated with abnormal menstrual patterns in affected women[12].

MATERIALS AND METHODS

This cross-sectional study was conducted in the Department of Gynecology, Bolan Medical Complex Hospital, Quetta, Pakistan, over a period of one year from January 2024 to January 2025. The research was formulated to determine the contribution of insulin resistance and obesity in the pathogenesis of menstrual irregularities in women with polycystic ovary syndrome (PCOS).

A total of 150 women of reproductive age were enrolled in the study using a non-probability consecutive sampling technique. The sample of women aged between 18 and 35 years with a diagnosis of PCOS based on the Rotterdam criteria was used. PCOS was diagnosed when two out of three of the following requirements were met: oligo/anovulation, clinical or biochemical evidence of hyperandrogenism, and polycystic ovarian morphology on pelvic ultrasonography. The study was only included with those women who were willing to participate and provided informed written consent.

Women were not included when they suffered other endocrine or metabolic conditions that may affect the way menstruation works, such as thyroid dysfunction, hyperprolactinemia, Cushing syndrome, congenital adrenal hyperplasia, or previously diagnosed diabetes mellitus. Pregnant, lactating, or having a history of ovarian surgery or hormonal drug use, insulin-sensitizing agents, or oral contraceptive pills within the last three months were also excluded to reduce the number of confounding factors.

Upon enrolment, a comprehensive clinical assessment of every participant was conducted. The data was documented on the following variables: age, marital status, menstrual history, period of menstrual complaints, history of weight gain, and symptoms indicative of PCOS such as hirsutism and acne. Menstrual pattern was assessed in detail and categorized into regular cycles, oligomenorrhea (menstrual cycles longer than 35 days), amenorrhea (absence of menstruation for more than three consecutive months), polymenorrhea (cycles shorter than 21 days), or irregular, unpredictable menstrual cycles.

Standard clinical methods were used to acquire anthropometric measures. The measurement of body weight was in kilograms, and height was in meters, with the help of a calibrated weighing scale and stadiometer, respectively. The body mass

index (BMI) was computed by dividing the weight in kilograms by the square of height in meters (kg/m ^{2}). Participants were classified as normal weight (BMI <25 kg/m²), overweight (BMI 25–29.9 kg/m²), or obese (BMI ≥30 kg/m²). Measuring central obesity also included measuring waist circumference at the mid position between the lower costal margin and the iliac crest with non-stretchable measuring tape. The circumference of the waist greater than 88 cm was regarded as high.

Pelvic ultrasonography was performed on all participants to verify the ovarian morphology and to aid the diagnosis of PCOS, where necessary. Furthermore, biochemical analysis was done on fasting venous blood samples following an 8–12-hour overnight starvation. Laboratory tests comprised the fasting blood glucose and the fasting serum insulin. The Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) was used to estimate insulin resistance, and it was computed with the help of the following formula:

$$\text{HOMA-IR} = \frac{\text{Fasting Insulin } (\mu IU/mL) \times \text{Fasting Blood Glucose } (mg/dL)}{405}$$

HOMA-IR above 2.5 was considered insulin resistant.

The data obtained were coded and analysed with the Statistical Package of Social Sciences (SPSS) version 26.0. Quantitative variables (age, BMI, waist circumference, fasting glucose, fasting insulin, and HOMA-IR) were reported as mean and standard deviation. Qualitative variables that included obesity levels, insulin resistance levels, and menstrual irregularity patterns were presented in terms of frequency and percentage. Comparisons of continuous variables across groups were performed with the help of the independent sample t-test, and the associations between the categorical variables were evaluated with the help of the chi-square test. Multivariate logistic regression analysis was done to determine independent predictors of menstrual irregularities. A p-value that was lower than 0.05 was considered significant.

The study was ethically approved by the Institutional Ethical Review Committee of Bolan Medical Complex Hospital, Quetta. The study followed all institutional ethical requirements, and informed consent was obtained in written form by all the participants before the study's inclusion.

RESULTS

A total of 150 women diagnosed with polycystic ovary syndrome (PCOS) were included in the study. The age of the participants was 26.4 years of age, with a standard deviation of 4.7 (18-35 years of age). Among all participants, 102 (68.0%) presented with menstrual irregularities, while 48 (32.0%) had comparatively regular menstrual cycles despite fulfilling the Rotterdam diagnostic criteria for PCOS. In this population study, therefore, menstrual disturbance was the most common reproductive complaint. Women with anomalous menstrual cycles had an obviously worse anthropometric and metabolic phenotype than women with more regular cycles. The average body mass index (BMI) of the irregular menstruation group was 31.2 ± 4.8 kg/m^2, and 26.1 ± 3.9 kg/m^2 of the regular-cycle group. Similarly, the waist circumference, fasting serum insulin, and HOMA-IR levels were found to be significantly elevated in the group of women with abnormal menstrual cycles, which suggested a strong metabolic correlation with cycle disruption. Table 1 gives these baseline comparisons.

Table 1. Baseline Clinical and Metabolic Characteristics of Women with PCOS According to Menstrual Pattern

Variable	Menstrual Irregularities (n = 102)	Regular Cycles (n = 48)	p-value
Age (years)	26.8 ± 4.5	25.6 ± 4.9	0.148
BMI (kg/m²)	31.2 ± 4.8	26.1 ± 3.9	<0.001
Waist Circumference (cm)	95.8 ± 9.1	85.6 ± 7.8	<0.001
Fasting Blood Glucose (mg/dL)	98.4 ± 11.6	90.7 ± 9.4	<0.001
Fasting Insulin (µIU/mL)	18.3 ± 5.7	12.1 ± 4.3	<0.001
HOMA-IR	4.42 ± 1.51	2.71 ± 1.09	<0.001
Obesity (BMI ≥30 kg/m²), n (%)	65 (63.7%)	11 (22.9%)	<0.001
Central Obesity (>88 cm), n (%)	79 (77.5%)	19 (39.6%)	<0.001

The menstrual abnormalities pattern of the current study also highlighted the prevalence of ovulatory dysfunction in PCOS women. Out of 150 participants, oligomenorrhea was the most common menstrual disorder and was identified in 69 (46.0%) women. This was succeeded by amenorrhea in 18 (12.0%), irregular, unpredictable cycles in 10 (6.7%), and polymenorrhea in 5 (3.3%) women. A comparatively regular menstrual cycle was reported by only 48 (32.0) of the participants. BMI and HOMA-IR increased progressively with increasingly worsening menstrual phenotypes. Women who had amenorrhea were the ones who had the highest mean BMI (33.4 ± 4.7 kg/m ^{2}) and HOMA-IR (5.01 ± 1.58), then those who had irregular,

unpredictable cycles and those with oligomenorrhea. This trend is very much indicative that increased menstrual dysfunction is linked to the extent of metabolic disturbance. These findings are summarized in Table 2.

Table 2. Distribution of Menstrual Patterns and Their Metabolic Profile in Women with PCOS

Menstrual Pattern	Frequency, n (%)	Mean BMI (kg/m²)	Mean HOMA-IR
Regular cycles	48 (32.0%)	26.1 ± 3.9	2.71 ± 1.09
Oligomenorrhea	69 (46.0%)	30.6 ± 4.3	4.18 ± 1.34
Amenorrhea	18 (12.0%)	33.4 ± 4.7	5.01 ± 1.58
Polymenorrhea	5 (3.3%)	28.9 ± 3.7	3.52 ± 1.01
Irregular unpredictable cycles	10 (6.7%)	31.8 ± 4.5	4.46 ± 1.29

When the relationship between insulin resistance, obesity, and menstrual dysfunction was examined more closely, both factors showed strong and statistically significant associations. Among women with HOMA-IR >2.5, 88 out of 108 (81.5%) had menstrual irregularities, compared with only 14 out of 42 (33.3%) women whose HOMA-IR was ≤2.5. Similarly, menstrual disturbance was markedly more common in women with obesity (BMI ≥30 kg/m²), where 65 out of 76 (85.5%) had abnormal cycles, compared with 37 out of 74 (50.0%) among non-obese participants. Menstrual abnormalities were also clearly related to central obesity. Multivariable logistic regression demonstrated that BMI ≥30 kg/m² and HOMA-IR >2.5 were the strongest independent predictors of menstrual irregularities in women with PCOS. Women with elevated HOMA-IR had more than fivefold higher odds of menstrual dysfunction, while obesity nearly quadrupled the risk. These association data are shown in Table 3.

Table 3. Association of Obesity and Insulin Resistance with Menstrual Irregularities in Women with PCOS

Variable	Menstrual Irregularities n/N (%)	Odds Ratio (95% CI)	p-value
BMI ≥30 kg/m²	65/76 (85.5%)	4.12 (1.95–8.70)	<0.001
Waist Circumference >88 cm	79/98 (80.6%)	3.48 (1.67–7.23)	0.001
HOMA-IR >2.5	88/108 (81.5%)	5.67 (2.49–12.89)	<0.001
Fasting Blood Glucose ≥100 mg/dL	31/39 (79.5%)	2.11 (0.92–4.83)	0.076
Age >25 years	61/84 (72.6%)	1.42 (0.69–2.90)	0.334

Overall, the results of this study demonstrate that menstrual irregularities in women with PCOS are strongly linked with obesity and insulin resistance. The women who had higher BMI, central adiposity, and high HOMA-IR were highly likely to have abnormal menstrual patterns, especially oligomenorrhea and amenorrhea. This evidence confirms the hypothesis that metabolic dysfunction is a significant contributor to the pathogenesis of menstrual disturbances in PCOS and indicates the value of metabolic screening in the early phases of the disease in women.

DISCUSSION

The present study evaluated the relationship between insulin resistance, obesity, and menstrual irregularities in women diagnosed with polycystic ovary syndrome (PCOS) and demonstrated a clear and statistically significant association between metabolic dysfunction and reproductive disturbance[10,11]. The results revealed that women with abnormal menstrual cycles possessed considerably larger body mass index, waist circumference, fasting insulin, and HOMA-IR values than women with rather normal menstrual cycles. The findings are in Favor of the hypothesis that insulin resistance and obesity are significant contributors to the pathogenesis of menstrual dysfunction in PCOS, especially the onset of chronic anovulation and cycle variability[12].

Among the most significant discoveries of this paper was that 68.0% of women with PCOS had menstrual irregularities, and oligomenorrhea was the most frequent menstrual abnormality, followed by amenorrhea[13]. This trend is a clinical anticipation and corresponds with the typical reproductive phenotype of PCOS, with ovulatory dysfunction that is mainly presented as infrequent or absent menstruation. Oligomenorrhea prevalence in the current paper means that impaired follicular maturation and persistent anovulation are still the key menstrual symptoms of PCOS in this group of people. Notably, women having more serious menstrual abnormalities, especially amenorrhea, exhibited the greatest

mean of BMI and HOMA-IR, which may indicate that the increasing metabolic disturbance can be accompanied by more severe menstrual dysfunction[14].

The high correlation between menstrual abnormalities and insulin resistance in the present study is biologically plausible and can be explained by the existing knowledge of PCOS pathophysiology[15]. The effect of insulin resistance is compensatory hyperinsulinemia, which is directly involved in causing ovarian androgen excess through the stimulation of theca cells and an increase in the production of luteinizing hormone-mediated androgen production. Simultaneously, insulin inhibits the hepatic synthesis of sex hormone-binding globulin (SHBG), which raises the number of free androgens in circulation. This hyperandrogenic hormonal milieu interferes with normal follicular growth, negative ovulation, and gives rise to irregular or absent menstrual cycles. The current global PCOS practice still identifies insulin resistance as a key metabolic cause of reproductive dysfunction and chronic disease morbidity[16].

In the current study, women who received an HOMA-IR value of above 2.5 were significantly more likely to experience menstrual irregularities compared to women with lower insulin resistance levels, and the association was statistically significant even with the other variables held constant. The discovery is especially significant as it indicates that insulin resistance is not just a related metabolic defect, but it can be a key pathogenic factor causing menstrual dysfunction in PCOS. Clinically, it justifies the integration of insulin resistance screening among women with cycle abnormalities, particularly those with suspected PCOS[17,18].

The other significant conclusion of the research was that obesity, especially central obesity, had a significant contribution to exacerbating menstrual disruptions[19]. Women who had a BMI of 30 kg/m 2 and high waist circumference had a significant probability of abnormal menstrual cycles as compared to women. These results support the idea that obesity increases the endocrine and metabolic disruptions that already exist in PCOS. The presence of excessive adipose tissue, particularly visceral fat, leads to insulin resistance, release of inflammatory cytokines, disturbed adipokine secretion, and disruption of steroid metabolism, which also may exacerbate ovarian functioning. Recent mechanistic reviews still explain visceral adiposity and adipose tissue dysfunction as important aggravators of insulin resistance and ovulatory dysfunction in PCOS[20].

Of particular interest is the relationship between central obesity and menstrual irregularities. The circumference of the waist was significantly increased among women with abnormal cycles, and body fat distribution may be more important than body weight in isolation[5]. Visceral adiposity is metabolically active and is better associated with insulin resistance compared to generalized obesity. This could be the reason why women who have similar BMI values report varying levels of menstrual dysfunction, given their metabolic phenotype. The waist circumference measurement during regular gynecological practice can, therefore, offer clinically useful data to supplement BMI [10-13].

The implications of the findings of this study are also significant to the clinical management of women with PCOS. In most cases, women mainly come with menstrual abnormalities and are only assessed in a gynecological light, where metabolic deviations are not adequately assessed[8]. Nevertheless, the results of this paper have shown that menstrual dysfunction can be an external clinical sign of a more profound metabolic imbalance. Thus, an early diagnosis of obesity and insulin resistance can not only contribute to the explanation of the abnormalities in the menstrual cycle but can also aid in the prevention of such long-term outcomes as infertility, impaired glucose tolerance, type 2 diabetes mellitus, and cardiometabolic disease. The relevance of thorough metabolic risk evaluation and weight-conscious care in PCOS women is highlighted in the current international recommendations as well[17,18].

The current research contributes to the local evidence on the topic, as the information about the metabolic determinants of menstrual dysfunction in PCOS is still insufficient in the tertiary care environment in Quetta, Pakistan[1,15]. Such findings are especially relevant in the context of low- and middle-income populations where a sedentary lifestyle, inadequate diet, and late endocrine examination are becoming a frequent occurrence. They note that it is necessary to change the clinical approach to PCOS to a more integrated reproductive-metabolic model of care[5,12].

Despite the strengths, this study is not devoid of limitations. As a cross-sectional study, it will be capable of establishing a significant association, yet it will not be capable of determining the direct cause[5-10]. The sample size is also limited to the hospital setting, and this might limit the applicability of the findings to the rest of the population. In addition to this, the more advanced endocrine tests, such as serum testosterone, SHBG, LH/FSH ratio, and anti-Mullerian hormone, were not stated, which would have provided a broader hormonal picture of the subjects. However, the study has provided

clinically significant findings that indicate the strong pathogenic role of insulin resistance and obesity in menstrual dysfunction in PCOS women[10-15].

In general, the data indicate that the menstrual irregularities observed in PCOS cannot be considered as isolated gynecological symptoms, but as components of the larger endocrine and metabolic disorder in which obesity and insulin resistance play a significant role in determining disease expression and severity[18-20].

CONCLUSION

This study concludes that insulin resistance and obesity are strongly associated with menstrual irregularities in women with polycystic ovary syndrome. Women who had high BMI, central adiposity, and high HOMA-IR were highly prone to have abnormal menstrual cycles, especially oligomenorrhea and amenorrhea. These results show that metabolic dysfunction is a significant factor in the pathogenesis of menstrual disturbances in PCOS. Obesity and insulin resistance should therefore be regularly measured in clinical examination of women with menstrual irregularities with an aim of improving early diagnosis, specific intervention, and long-term reproductive as well as metabolic outcomes.

Availability of Data and Materials

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Competing Interests

The authors declare that they have no competing interests.

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Authors' Contributions

- **SS**: Data collection, clinical assessment, manuscript drafting.
- **RK**: Data analysis, statistical interpretation.
- **RN**: Literature review, data entry, initial draft.
- **SG**: Results interpretation, manuscript writing.
- **ZB**: Conceptualization, study design, supervision, final approval.
- **FJ**: Study design, patient recruitment, critical review.

All authors approved the final manuscript.

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