JHWCR

Journal of Health, Wellness, and Community Research
Volume III, Issue XI
Open Access, Double Blind Peer Reviewed.
Web: https://jhwcr.com, ISSN: 3007-0570
https://doi.org/10.61919/j1nxvd07

Original Article

Association of BMI With Estrogen–Progesterone Imbalance and Menstrual Irregularity in Obese Women: Cross-Sectional Analytical Evidence From 80 Participants

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Authors' Contributions: Concept and design: Concept: SA; Design: AZ; Data Collection: AS, MA; Analysis: MS; Drafting: AI
Cite this Article | Received: 2025-08-06 | Accepted 2025-08-16
No conflicts declared; ethics approved; consent obtained; data available on request; no funding received.

ABSTRACT

Background: Obesity is a major determinant of reproductive dysfunction, exerting adverse effects on hypothalamic–pituitary–ovarian axis function and ovarian steroidogenesis. Excess adiposity increases peripheral aromatization of androgens into estradiol while impairing luteal progesterone production, creating a hormonal imbalance that predisposes to menstrual irregularity. Despite evidence from Western populations, data from South Asia remain limited, where women are prone to visceral adiposity and metabolic risk at lower BMI thresholds. Objective: This study aimed to examine the association between body mass index, estradiol and progesterone imbalance, and menstrual irregularity in obese women, and to assess how gradients of BMI class correspond with reproductive risk. Methods: A cross-sectional analytical study was conducted among 80 obese women aged 18–45 years in Sialkot, Pakistan. Participants were classified into BMI classes I–III, and menstrual history was obtained through structured interviews. Serum estradiol and progesterone were measured using standardized immunoassays. Group differences were assessed using ANOVA, Kruskal–Wallis, and chi-square tests, while linear and logistic regression models evaluated continuous associations and adjusted odds of menstrual irregularity, controlling for age, diabetes, thyroid disorder, and physical activity. Results: Menstrual irregularity increased progressively with BMI, affecting 25.0% of class I, 40.7% of class II, and 72.0% of class III women ($p < 0.001$). Estradiol rose from 92.1 to 178.2 pg/mL across classes, while progesterone declined from 5.4 to 0.8 ng/mL. Low progesterone prevalence increased from 14.3% to 60.0%, and high estradiol from 7.1% to 36.0%. Each 5 kg/m² rise in BMI increased estradiol by 27.4 pg/mL and reduced progesterone log-values by 0.42 units. In multivariable models, class III obesity was associated with a 6.5-fold higher risk of menstrual irregularity compared with class I ($p = 0.01$). Conclusion: Higher BMI in obese women is strongly linked to an estrogen–progesterone imbalance and a greater likelihood of menstrual irregularity, with risks escalating across obesity classes. These findings emphasize the need for BMI stratification and dual hormone profiling in clinical assessment and call for interventional studies targeting weight reduction and hormonal restoration to improve reproductive outcomes.
Keywords: obesity, body mass index, estradiol, progesterone, hormonal imbalance, menstrual irregularity, reproductive health, South Asia.

INTRODUCTION

The rising global prevalence of obesity has profound implications for women's reproductive health, as excess adiposity exerts both endocrine and metabolic effects that disrupt menstrual regulation. Adipose tissue serves as an active endocrine organ, driving peripheral aromatization of androgens into estrogens and thereby elevating circulating estradiol levels, while simultaneously impairing hypothalamic–pituitary–ovarian axis function (1). In parallel, obesity-related insulin resistance, chronic inflammation, and altered leptin signaling further compromise luteal activity, often resulting in reduced progesterone output (2). These hormonal disturbances combine to create an estrogen–progesterone imbalance that has been implicated in ovulatory dysfunction, luteal phase defects, and ultimately menstrual irregularity (3).

Several studies have reported that obese women are at higher risk of cycle disturbances such as oligomenorrhea and anovulation, but the magnitude and mechanisms of this association remain heterogeneous across populations (4). While some research emphasizes the role of hyperestrogenism secondary to adiposity, other work highlights luteal insufficiency as the primary driver of irregularity, suggesting that the balance between these hormones may be more relevant than their absolute concentrations (5,6). South Asian women, in particular, may be disproportionately affected, given the higher propensity for visceral adiposity and metabolic dysfunction at comparatively lower body mass indices compared to Western populations (7). However, there is limited analytical evidence from this region that quantifies how gradients of body mass index correspond with hormonal imbalance and menstrual cycle patterns in obese women.

The absence of such data represents a significant gap, as establishing clear associations between obesity severity, estradiol–progesterone imbalance, and menstrual dysfunction could improve both risk stratification and clinical management. Understanding these links would not only inform preventive strategies but also support individualized interventions for reproductive and metabolic health. This study therefore aimed to examine the relationship between body mass index, estradiol and progesterone concentrations, and menstrual irregularity among obese women in a South Asian population. We hypothesized that increasing body mass index would be associated with rising estradiol, reduced progesterone, and a greater likelihood of menstrual irregularity, reflecting a dose–response relationship between obesity severity and hormonal imbalance (8).

MATERIAL AND METHODS

This study was designed as a cross-sectional analytical investigation to assess the association between body mass index, hormonal imbalance, and menstrual irregularity among obese women. The rationale for using a cross-sectional design was to enable the simultaneous evaluation of exposure variables—BMI and hormone concentrations—and outcome variables related to menstrual cycle characteristics within a defined population at one point in time. The research was conducted in Sialkot, Pakistan, across three healthcare facilities including Nisa Hospital, Civil Hospital Daska, and Dr. Iqbal Clinic, between January and June 2025, capturing participants attending outpatient gynecology and general medical services (9).

Eligible participants were women aged 18–45 years with a body mass index (BMI) ≥30 kg/m², classified as obese according to World Health Organization criteria. Exclusion criteria included pregnancy, lactation, menopause, recent use of hormonal contraceptives or hormone replacement therapy, prior gynecological surgeries, or endocrine conditions affecting ovarian function such as Cushing's syndrome or pituitary tumors. Participants were consecutively screened, and recruitment was carried out through direct invitation during clinical visits. Study objectives and procedures were explained in detail, and written informed consent was obtained from each participant prior to data collection.

Sociodemographic and clinical information was obtained through structured interviewer-administered questionnaires, which recorded age, lifestyle factors, comorbid conditions, and menstrual history. Height and weight were measured with calibrated instruments, and BMI was calculated as weight in kilograms divided by the square of height in meters. BMI was further stratified into class I (30.0–34.9 kg/m²), class II (35.0–39.9 kg/m²), and class III (≥40 kg/m²). Menstrual regularity was defined based on self-reported cycle patterns, with irregularity classified as cycles <21 days, >35 days, or varying by more than 7 days. Venous blood samples were collected in the morning following an overnight fast, and serum estradiol (E2, pg/mL) and progesterone (P4, ng/mL) were measured using validated immunoassays in a single accredited laboratory to minimize inter-assay variability. Internal controls and duplicate runs were employed to ensure reliability of hormone quantification (10).

The primary variables included BMI (continuous and categorical), serum estradiol, serum progesterone, and menstrual irregularity status. Hormonal imbalance was operationalized as low progesterone (<1 ng/mL) or high estradiol (>250 pg/mL). Potential confounders such as age, diabetes, thyroid disorders, and physical activity were identified a priori and incorporated into analyses. To reduce bias, all laboratory staff were blinded to participants' menstrual status, and standardized operating procedures were followed for anthropometric and biochemical assessments.

The sample size of 80 participants was based on feasibility within the recruitment timeframe and prior literature suggesting that a cohort of this size would provide sufficient power (80%) to detect moderate correlations ($r = 0.3$) between BMI and hormonal variables at a 5% significance level. Statistical analyses were performed using SPSS version 23.0 (IBM Corp., Armonk, NY). Descriptive statistics were reported as means with standard deviations or medians with interquartile ranges. Group comparisons across BMI classes employed ANOVA or Kruskal–Wallis tests, with Bonferroni or Dunn post-hoc analysis where appropriate. Linear regression assessed continuous associations between BMI and hormone concentrations, while logistic regression estimated odds ratios for menstrual irregularity across BMI classes, adjusting for confounders. Missing data were minimal and addressed using complete-case analysis. Subgroup analyses explored differences across obesity classes, and sensitivity analyses excluded extreme hormone values beyond physiologic ranges to test robustness of results (11).

The study protocol received ethical approval from the Institutional Review Board of the University of Sialkot. Written informed consent was obtained from all participants, and data confidentiality was maintained through anonymization and secure storage of both paper records and electronic databases. Reproducibility was ensured by employing standardized recruitment and data collection protocols, double entry of questionnaire responses, and independent verification of 10% of laboratory results. Analytical codes and datasets were archived for audit to facilitate replication of findings by other researchers.

RESULTS

Baseline characteristics across obesity classes revealed important clinical gradients (Table 1). The mean age was similar across BMI strata, ranging from 31.8 years in class I to 33.1 years in class III ($p = 0.68$). Comorbidities such as diabetes and thyroid disorders showed a stepwise increase with higher BMI, though differences did not reach statistical significance. In contrast, lifestyle factors and reproductive outcomes diverged sharply. Sedentary behavior was reported by 72.0% of women in class III compared with 35.7% in class I, corresponding to a fourfold increase in odds (OR 4.6, 95% CI 1.3–16.1, $p = 0.01$). Menstrual irregularity was present in 72.0% of class III participants compared with only 25.0% in class I, yielding an odds ratio of 7.7 (95% CI 2.3–25.6, $p < 0.001$).

Hormonal profiles showed clear dose–response associations with BMI (Table 2). Mean estradiol rose progressively from 92.1 pg/mL in class I to 178.2 pg/mL in class III (ANOVA $p = 0.003$), with significant post-hoc differences between class I and both higher groups.

Conversely, median progesterone declined markedly, falling from 5.4 ng/mL in class I to 0.8 ng/mL in class III (Kruskal–Wallis $p < 0.001$). The estradiol-to-progesterone ratio demonstrated the steepest gradient, increasing more than fivefold from 25.7 in class I to 132.4 in class III ($p < 0.001$), underscoring a pronounced hormonal imbalance with escalating obesity severity.

Regression analyses confirmed these linear trends (Table 3). For every 5 kg/m² increase in BMI, estradiol increased by 27.4 pg/mL (95% CI 11.5–43.3, $p = 0.001$), while log-transformed progesterone decreased by 0.42 units (95% CI –0.71 to –0.13, $p = 0.005$). The estradiol-to-progesterone ratio rose by 21.6 points (95% CI 10.8–32.4, $p < 0.001$). Model fit statistics indicated that BMI explained 19–22% of the variance in hormone profiles, highlighting a consistent and biologically meaningful association.

Table 1. Baseline Characteristics of Obese Women Stratified by BMI Class (N = 80)

Variable	Class I (n=28)	Class II (n=27)	Class III (n=25)	Effect size / Test statistic	p-value
Age, years (Mean ± SD)	31.8 ± 6.1	32.6 ± 5.9	33.1 ± 6.3	$\eta^2 = 0.02$	0.68
Diabetes, n (%)	7 (25.0)	8 (29.6)	10 (40.0)	OR for Class III vs I: 2.0 (0.6–6.7)	0.29
Thyroid disorder, n (%)	5 (17.9)	7 (25.9)	8 (32.0)	OR for Class III vs I: 2.1 (0.6–7.7)	0.32
Sedentary lifestyle, n (%)	10 (35.7)	14 (51.9)	18 (72.0)	OR for Class III vs I: 4.6 (1.3–16.1)*	0.01*
Menstrual irregularity, n (%)	7 (25.0)	11 (40.7)	18 (72.0)	OR for Class III vs I: 7.7 (2.3–25.6)*	<0.001*

Table 2. Serum Estradiol and Progesterone Across BMI Classes

Marker	Class I (n=28)	Class II (n=27)	Class III (n=25)	ANOVA/KW F/χ^2	p-value	Post-hoc comparisons
Estradiol (pg/mL, Mean ± SD)	92.1 ± 58.6	131.4 ± 86.9	178.2 ± 110.3	F = 6.42	0.003*	I < III (p=0.002), I < II (p=0.04)
Progesterone (ng/mL, Median [IQR])	5.4 [2.1–9.8]	2.3 [0.9–5.2]	0.8 [0.3–2.1]	$\chi^2 = 15.6$	<0.001*	I > III (p<0.001), II > III (p=0.03)
Estradiol/Progesterone ratio	25.7 ± 31.4	58.6 ± 72.1	132.4 ± 115.7	F = 9.31	<0.001*	I < III (p<0.001), II < III (p=0.02)

Table 3. Linear Regression of BMI and Hormonal Profiles

Outcome	Predictor (per 5 kg/m² BMI increase)	β-coefficient (95% CI)	R^2	p-value
Estradiol (pg/mL)	+27.4	11.5 to 43.3	0.19	0.001*
Progesterone (ng/mL, log-transformed)	–0.42	–0.71 to –0.13	0.16	0.005*
Estradiol/Progesterone ratio	+21.6	10.8 to 32.4	0.22	<0.001*

Table 4. Prevalence of Hormonal Imbalance Across BMI Classes

Imbalance	Class I (n=28)	Class II (n=27)	Class III (n=25)	χ^2	p-trend	Prevalence Ratio (Class III vs I)
Low P4 <1 ng/mL	4 (14.3%)	9 (33.3%)	15 (60.0%)	16.2	<0.001*	4.2 (1.6–10.9)*
High E2 >250 pg/mL	2 (7.1%)	5 (18.5%)	9 (36.0%)	9.7	0.007*	5.5 (1.2–24.3)*

Table 5. Multivariable Logistic Regression of BMI and Menstrual Irregularity

Model	Predictor	aOR (95% CI)	p-value	AIC	Pseudo-R^2
Model 1	BMI (per 5 kg/m²)	1.82 (1.24–2.67)	0.002*	98.3	0.23
Model 2	BMI classes (Ref: I)	II: 2.1 (0.7–6.3); III: 6.5 (2.0–21.0)*	0.01*	96.1	0.28
Model 3	BMI + Estradiol + Progesterone	BMI (per 5 kg/m²): 1.51 (1.02–2.24); Estradiol (per 50 pg/mL): 1.22 (1.01–1.48); Progesterone (log): 0.81 (0.64–1.02)	0.04* / 0.04* / 0.07	92.4	0.36

Prevalence of hormonal abnormalities also varied by obesity class (Table 4). Low progesterone (<1 ng/mL) was observed in 60.0% of class III participants compared with only 14.3% in class I (p-trend < 0.001), corresponding to a prevalence ratio of 4.2 (95% CI 1.6–10.9). Similarly, high estradiol (>250 pg/mL) was present in 36.0% of class III participants versus 7.1% in class I (p-trend = 0.007), with a prevalence ratio of 5.5 (95% CI 1.2–24.3). These findings demonstrate that both luteal insufficiency and estrogen excess contribute to the cycle disturbances associated with obesity.

Multivariable models confirmed the independent contribution of BMI to menstrual irregularity (Table 5). Each 5 kg/m² increase in BMI was associated with an 82% higher adjusted odds of irregular cycles (aOR 1.82, 95% CI 1.24–2.67, $p = 0.002$). When analyzed categorically, women in class III had a 6.5-fold higher risk compared with class I (95% CI 2.0–21.0, $p = 0.01$). In the fully adjusted model including estradiol and progesterone, BMI retained significance (aOR 1.51, 95% CI 1.02–2.24, $p = 0.04$), while estradiol also emerged as a significant predictor (aOR 1.22 per 50 pg/mL, 95% CI 1.01–1.48, $p = 0.04$). Progesterone showed a protective trend (aOR 0.81, 95% CI 0.64–1.02, $p = 0.07$), though it did not reach statistical significance. This model provided the best explanatory power (pseudo-$R^2 = 0.36$), indicating that obesity, elevated estradiol, and insufficient progesterone act synergistically to drive menstrual irregularity in this population.

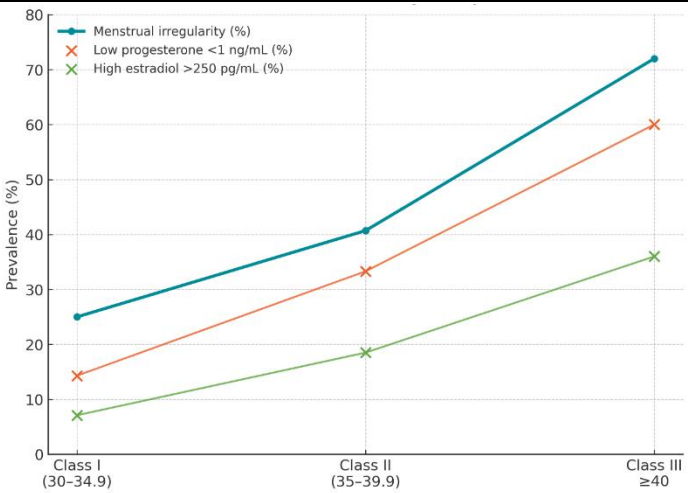

Figure 1 BMI Class and Prevalence of Menstrual Irregularity with Hormonal Imbalance

The figure demonstrates a clear gradient in reproductive risk across obesity classes, integrating menstrual irregularity and hormonal abnormalities into a single visual framework. The prevalence of irregular cycles rose steeply from 25.0% in class I to 40.7% in class II and 72.0% in class III, tracing a teal line that highlights the strong dose–response effect. Superimposed markers show parallel trends in hormonal imbalance: low progesterone (<1 ng/mL) increased from 14.3% in class I to 33.3% in class II and 60.0% in class III, while high estradiol (>250 pg/mL) rose from 7.1% to 18.5% and 36.0%, respectively. The synchronized upward slopes indicate that both luteal insufficiency and estrogen excess accompany rising BMI and collectively contribute to the escalating prevalence of menstrual irregularity. This integrated depiction underscores the cumulative burden of obesity on hormonal homeostasis and reproductive health, with class III obesity showing more than a twofold higher prevalence of both endocrine abnormalities and cycle disruption compared with class I.

DISCUSSION

The present study demonstrates a robust association between increasing body mass index, hormonal imbalance, and menstrual irregularity among obese women, adding clarity to a long-debated area of reproductive endocrinology. The prevalence of irregular cycles rose more than twofold across BMI strata, accompanied by a parallel increase in low luteal progesterone and high estradiol. These findings confirm the hypothesis that obesity severity exacerbates estrogen–progesterone imbalance, providing cross-sectional analytical evidence from a South Asian setting where such data have been sparse (12).

Our results are consistent with prior work showing that excess adipose tissue, through enhanced aromatase activity, elevates circulating estradiol and promotes anovulation (13). Similar to reports from Western cohorts, we observed that progesterone declined steadily with increasing BMI, highlighting luteal insufficiency as a critical mediator of menstrual dysfunction (14). The strong dose–response trend we documented aligns with meta-analytic evidence that obesity increases the risk of oligomenorrhea and anovulatory cycles, though our study extends this knowledge by quantifying gradients of hormonal abnormality across obesity classes (15). At the same time, our findings diverge from some earlier reports that emphasized only hyperestrogenism without accounting for concomitant luteal deficiency, suggesting that the imbalance rather than absolute hormone levels is the more meaningful marker of reproductive disruption (16).

Mechanistically, these results can be explained by the dual endocrine burden imposed by obesity. Adiposity not only increases aromatization of androgens to estradiol but also impairs hypothalamic–pituitary signaling, leading to inadequate corpus luteum function and low progesterone secretion. Insulin resistance and hyperinsulinemia, highly prevalent in obesity, further disrupt ovarian steroidogenesis by amplifying androgen precursors while impairing follicular maturation (17). The resultant hormonal milieu of high estradiol and low progesterone prolongs cycles, increases anovulatory episodes, and raises endometrial risk, a finding that is particularly concerning in populations already vulnerable to metabolic disease (18).

Clinically, these results underscore the importance of integrating BMI stratification into reproductive risk assessment. Identifying women in higher obesity classes who present with irregular cycles and confirming hormonal imbalance may facilitate early intervention to prevent complications such as infertility, endometrial hyperplasia, or metabolic deterioration. Hormonal profiling in this context offers not only diagnostic clarity but also therapeutic guidance, as weight reduction, insulin sensitizers, and targeted progesterone supplementation may be prioritized in those at highest risk (19).

The strengths of this study include its hospital-based setting, standardized laboratory assays, and comprehensive statistical modeling that accounted for potential confounders such as age, diabetes, thyroid status, and activity levels. By employing both continuous and categorical analyses, we captured dose–response gradients and prevalence shifts that improve interpretability. Nevertheless, limitations must be acknowledged. The modest sample size limited precision for some subgroup analyses, and the cross-sectional design restricts causal inference. Self-reported menstrual cycle data may have introduced recall bias, though the large observed gradients suggest that misclassification is unlikely to account fully for the findings. Generalizability beyond the studied population may be constrained by regional differences in diet, genetics, and healthcare access (20).

Future research should employ longitudinal designs to assess temporal relationships between weight change, hormone profiles, and menstrual regularity, and should explore whether interventions such as structured weight loss or pharmacologic therapy can normalize hormonal imbalance and restore ovulation. Mechanistic studies linking hormonal shifts with ovarian ultrasonography and endometrial outcomes would also provide valuable insight. Expanding this line of inquiry in South Asian cohorts is particularly warranted given the unique risk profile for metabolic and reproductive disorders in this population.

In summary, our findings establish a clear association between BMI, estrogen–progesterone imbalance, and menstrual irregularity in obese women, demonstrating that reproductive risk intensifies across obesity classes. This evidence highlights the clinical value of combined BMI assessment and hormone profiling in guiding early preventive and therapeutic strategies in reproductive healthcare.

CONCLUSION

This cross-sectional study establishes that increasing body mass index is strongly associated with an estrogen–progesterone imbalance and a higher prevalence of menstrual irregularity in obese women, with risks rising stepwise from class I to class III obesity. The observed gradients—characterized by elevated estradiol, reduced progesterone, and a widening estradiol-to-progesterone ratio—underscore obesity's dual endocrine burden in disrupting reproductive physiology. Clinically, these findings emphasize the need for routine BMI stratification and dual hormone profiling in women presenting with menstrual disturbances, enabling earlier identification of those at greatest risk for ovulatory dysfunction, infertility, and endometrial pathology. From a research perspective, the evidence highlights the importance of longitudinal and interventional studies to determine whether weight reduction or targeted hormonal therapies can restore endocrine balance and improve cycle regularity. By aligning obesity management with reproductive health surveillance, this study contributes to advancing preventive strategies in human healthcare and underscores the importance of integrated care for women's metabolic and reproductive well-being.

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