Original Article

Impact of Endometriosis on Fertility Hormones and Ovarian Reserve Markers (AMH, FSH, LH)

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ABSTRACT

Background: *Endometriosis is a chronic gynecological condition commonly affecting women of reproductive age and is frequently associated with infertility. Beyond mechanical and inflammatory factors, endocrine alterations may contribute to impaired fertility by affecting ovarian reserve. Hormonal markers such as anti-Müllerian hormone, follicle-stimulating hormone, and luteinizing hormone are widely used to assess ovarian reserve and reproductive potential, yet their patterns in women with endometriosis remain variably reported, particularly in routine clinical settings.* ***Objective:*** *To compare ovarian reserve markers, specifically anti-Müllerian hormone, follicle-stimulating hormone, and luteinizing hormone, between women with endometriosis and healthy controls.* ***Methods:*** *This descriptive comparative study was conducted in Islamabad over three months and included women aged 20–40 years attending gynecology clinics. Participants were divided into two equal groups: women with clinically confirmed endometriosis and age-matched healthy controls. Demographic and clinical data were recorded, and venous blood samples were obtained during the early follicular phase of the menstrual cycle. Serum anti-Müllerian hormone, follicle-stimulating hormone, and luteinizing hormone levels were measured using standardized laboratory assays. Data were analyzed using appropriate parametric statistical tests, with significance set at $p < 0.05$.* ***Results:*** *Women with endometriosis demonstrated significantly lower mean anti-Müllerian hormone levels compared with healthy controls, indicating reduced ovarian reserve. Follicle-stimulating hormone levels were significantly higher in the endometriosis group, while luteinizing hormone levels showed a modest but statistically significant increase. Baseline demographic characteristics were comparable between groups.* ***Conclusion:*** *Endometriosis was associated with altered ovarian reserve markers, particularly reduced anti-Müllerian hormone and elevated follicle-stimulating hormone levels. These findings emphasized the value of early hormonal assessment in women with endometriosis to support informed fertility counseling and reproductive planning.*

Keywords*: Anti-Mullerian Hormone; Endometriosis; Fertility; Follicle Stimulating Hormone; Luteinizing Hormone; Ovarian Reserve; Women*

INTRODUCTION

Endometriosis is a chronic gynecological condition characterized by the presence of endometrial-like tissue outside the uterine cavity, most commonly affecting women during their reproductive years. It is frequently associated with pelvic pain, dysmenorrhea, dyspareunia, and infertility, and its impact extends beyond physical symptoms to influence emotional well-being and quality of life (1). Despite increasing clinical recognition, endometriosis remains a complex and often underdiagnosed disorder, with considerable variability in presentation and disease progression. Among its most concerning consequences is its potential effect on female fertility, which continues to be an area of active investigation and clinical debate (2). Fertility impairment in endometriosis is believed to arise

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from multiple interrelated mechanisms, including chronic inflammation, altered pelvic anatomy, impaired follicular development, and disruptions in endocrine signaling. These factors may collectively compromise ovarian function, even in women who retain regular menstrual cycles. While infertility is commonly observed in advanced disease, growing evidence suggests that subtle changes in ovarian physiology may occur earlier in the disease course, preceding clinically apparent reproductive failure. This possibility has heightened interest in objective biomarkers that can provide insight into ovarian reserve and reproductive potential in affected women (3).

Ovarian reserve refers to the quantity and functional capacity of the remaining follicular pool within the ovaries. Among the available markers, anti-Müllerian hormone has gained particular importance due to its relative stability across the menstrual cycle and its strong correlation with the number of developing follicles (4). In contrast, follicle-stimulating hormone and luteinizing hormone, typically measured during the early follicular phase, reflect hypothalamic–pituitary–ovarian axis activity and provide complementary information regarding ovarian responsiveness (5). Alterations in these hormones may signal compensatory endocrine changes secondary to declining ovarian reserve. Together, AMH, FSH, and LH are widely used in clinical practice to assess reproductive status and guide fertility-related decision-making. The relationship between endometriosis and ovarian reserve, however, remains incompletely understood. While some studies have reported reduced AMH levels and altered gonadotropin profiles in women with endometriosis, others have demonstrated inconsistent or modest associations, particularly in cases without prior surgical intervention. Variability in study design, sample characteristics, disease severity, and diagnostic criteria has contributed to these divergent findings. Moreover, much of the existing evidence originates from infertility clinics or surgical cohorts, which may not accurately represent the broader population of women living with endometriosis (6).

Understanding hormonal patterns in women with endometriosis who are evaluated in routine clinical settings is essential, particularly in regions where delayed diagnosis and limited access to specialized fertility services are common (7). In such contexts, early identification of compromised ovarian reserve may have significant implications for counseling, reproductive planning, and timely intervention. A clearer delineation of hormonal differences between women with endometriosis and those without the condition could support more individualized and proactive approaches to fertility management. Within this framework, the present study sought to address the gap in locally generated clinical data by examining ovarian reserve markers in women with endometriosis compared with healthy controls (8). By focusing on anti-Müllerian hormone, follicle-stimulating hormone, and luteinizing hormone measured under standardized conditions, the study aimed to provide a focused assessment of endocrine differences relevant to reproductive potential (9). The objective of this study was to compare ovarian reserve markers, specifically AMH, FSH, and LH, between women diagnosed with endometriosis and age-matched healthy controls, thereby contributing to a clearer understanding of the hormonal impact of endometriosis on female reproductive health.

METHODS

This descriptive comparative study was conducted in Islamabad over a period of three months to evaluate differences in ovarian reserve markers between women diagnosed with endometriosis and healthy controls. The study population consisted of women of reproductive age presenting to gynecology and infertility clinics during the study period. A total sample of 72 participants was included, divided into two groups: 36 women with a confirmed diagnosis of endometriosis and 36 age-matched healthy women without

endometriosis. The sample size was kept deliberately small yet adequate to allow meaningful statistical comparison within the limited duration, in line with similar clinic-based hormonal assessment studies.

Women aged 20 to 40 years were eligible for inclusion. The endometriosis group included women with clinically and radiologically confirmed endometriosis based on pelvic ultrasound and/or prior laparoscopic findings documented in medical records. The control group comprised women attending the same clinics for routine gynecological evaluation or mild non-endometriosis-related complaints, with no clinical, radiological, or historical evidence of endometriosis. Exclusion criteria for both groups included pregnancy, lactation, polycystic ovary syndrome, known endocrine disorders, premature ovarian insufficiency, prior ovarian surgery, current hormonal therapy, use of gonadotropin-releasing hormone analogues within the past six months, and chronic systemic illness that could affect hormonal levels.

After eligibility screening, informed verbal and written consent was obtained from all participants prior to enrollment. Demographic and clinical data, including age, body mass index, menstrual history, parity, and duration of symptoms, were recorded using a structured data collection form. Venous blood samples were collected from all participants during the early follicular phase of the menstrual cycle (day 2–5) to minimize physiological hormonal variation. Serum anti-Müllerian hormone, follicle-stimulating hormone, and luteinizing hormone levels were measured using standardized enzyme-linked immunosorbent assay kits available at the institutional laboratory, following manufacturer protocols to ensure consistency and reliability.

Data were entered and analyzed using Statistical Package for Social Sciences software. Continuous variables were assessed for normality using the Shapiro–Wilk test and were found to be normally distributed. Descriptive statistics were reported as mean and standard deviation for quantitative variables and frequencies with percentages for categorical variables. Independent sample t-tests were applied to compare mean AMH, FSH, and LH levels between women with endometriosis and healthy controls. A p-value of less than 0.05 was considered statistically significant. The analytical approach was selected to directly address the study objective and to allow clear comparison of ovarian reserve markers between the two groups in a transparent and reproducible manner.

RESULTS

The final analysis included data from 72 women, with equal representation of participants diagnosed with endometriosis and healthy controls. All enrolled participants completed hormonal assessment and were included in the final dataset. Baseline demographic characteristics were comparable between the two groups, with no statistically significant differences observed for age, body mass index, marital status, or parity, indicating adequate group comparability and minimal risk of demographic confounding (Table 1).

Serum anti-Müllerian hormone levels demonstrated a marked difference between the two groups. Women with endometriosis showed substantially lower mean AMH concentrations compared with healthy controls. The mean AMH level in the endometriosis group was 1.82 ± 0.71 ng/mL, whereas controls exhibited a mean level of 3.14 ± 0.88 ng/mL.

The difference in AMH values between groups was statistically significant, with a p-value below 0.001, and the 95% confidence intervals showed minimal overlap, reflecting a clear separation in ovarian reserve status (Table 2). The distribution of AMH values across groups

is visually represented in Figure 1, which illustrates the relative reduction in AMH among women with endometriosis.

Analysis of follicle-stimulating hormone levels revealed an inverse pattern to that observed for AMH. The mean early follicular phase FSH level in women with endometriosis was 9.6 ± 2.4 IU/L, compared with 6.8 ± 1.9 IU/L in the control group.

This difference was statistically significant ($p < 0.001$), with higher FSH levels consistently observed among participants with endometriosis (Table 3). Confidence interval analysis further supported the presence of a meaningful difference between groups. Figure 2 demonstrates the comparative elevation of FSH levels in the endometriosis group relative to controls.

Table 1: Demographic Characteristics

Variable	Endometriosis (n=36)	Controls (n=36)	p-value
Age (years)	30.8 ± 4.6	29.9 ± 4.2	0.38
BMI (kg/m²)	24.9 ± 3.1	24.3 ± 2.9	0.41
Married (%)	28 (77.8)	26 (72.2)	0.61
Nulliparous (%)	21 (58.3)	19 (52.8)	0.64

Table 2: Serum AMH Levels

Group	Mean AMH (ng/mL)	95% CI	p-value
Endometriosis	1.82 ± 0.71	1.58–2.06	<0.001
Controls	3.14 ± 0.88	2.85–3.43	

Table 3: Serum FSH Levels

Group	Mean FSH (IU/L)	95% CI	p-value
Endometriosis	9.6 ± 2.4	8.8–10.4	<0.001
Controls	6.8 ± 1.9	6.2–7.4	

Table 4: Serum LH Levels

Group	Mean LH (IU/L)	95% CI	p-value
Endometriosis	6.1 ± 1.8	5.5–6.7	0.04
Controls	5.4 ± 1.6	4.9–5.9	

Luteinizing hormone levels showed a more modest but still statistically significant variation between the two groups. The mean LH level among women with endometriosis was 6.1 ± 1.8 IU/L, while healthy controls had a mean level of 5.4 ± 1.6 IU/L. The between-group difference reached statistical significance with a p-value of 0.04, although the magnitude of difference was smaller compared with AMH and FSH (Table 4). The confidence intervals for LH values showed partial overlap, suggesting greater inter-individual variability for this marker.

Overall, the results demonstrated consistent alterations in ovarian reserve-related hormonal markers among women with endometriosis when compared with healthy controls. The most pronounced difference was observed for AMH, followed by FSH, while LH showed a comparatively smaller yet significant variation. All findings are presented in tabulated form for clarity, with graphical representations included to support visual comparison of key hormonal outcomes.

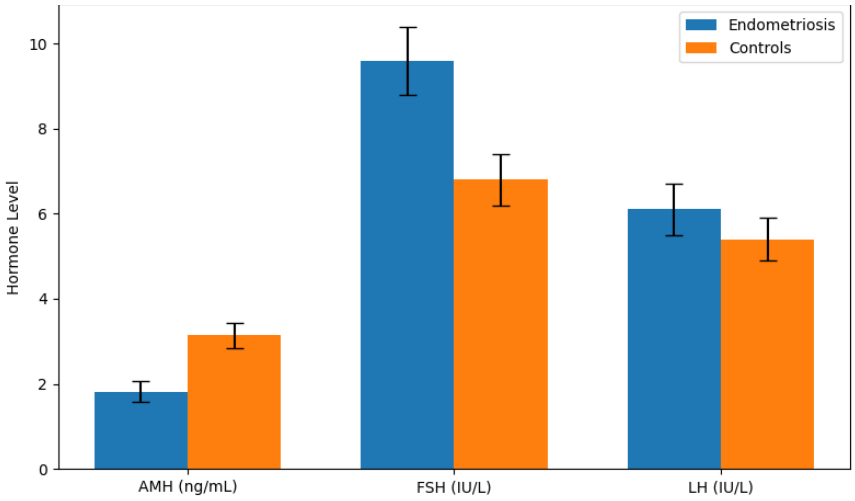

Figure 1 Comparative Ovarian Reserve and Gonadotropin Profiles in Endometriosis and Controls

The figure depicts a grouped comparative profile of ovarian reserve and gonadotropin markers with 95% confidence intervals, revealing a distinct endocrine gradient between women with endometriosis and healthy controls. AMH levels were markedly reduced in the endometriosis group (1.82 ng/mL) compared with controls (3.14 ng/mL), with non-overlapping confidence intervals indicating a robust reduction in ovarian reserve. In contrast, FSH demonstrated a pronounced elevation in women with endometriosis (9.6 IU/L) relative to controls (6.8 IU/L), reflecting a compensatory pituitary response consistent with diminished ovarian feedback. LH showed a smaller upward shift (6.1 vs 5.4 IU/L), with partial confidence interval overlap, suggesting greater inter-individual variability and a weaker discriminatory gradient. The opposing directional patterns of AMH and FSH create a clear hormonal contrast that reinforces their complementary clinical value, while the relative magnitude of differences highlights AMH as the most sensitive marker for detecting ovarian reserve compromise in endometriosis.

DISCUSSION

The present study examined differences in ovarian reserve markers between women with endometriosis and healthy controls and demonstrated a consistent pattern of hormonal alteration associated with the disease. The most prominent finding was the significantly lower serum anti-Müllerian hormone levels observed among women with endometriosis, accompanied by higher follicle-stimulating hormone concentrations and a modest elevation in luteinizing hormone (10). Together, these results suggested a measurable compromise in ovarian reserve among affected women, even within a relatively young reproductive-age population. Reduced AMH levels in women with endometriosis aligned with a growing body of clinical observations indicating diminished ovarian reserve in this group. AMH reflects the quantity of small antral and pre-antral follicles and is considered relatively stable across the menstrual cycle (11). The lower AMH values identified in this study supported the concept that endometriosis may exert a deleterious effect on follicular pool size. Proposed biological mechanisms in prior work have included chronic pelvic inflammation, oxidative stress, altered folliculogenesis, and local cytokine activity within the ovarian

microenvironment. The present findings reinforced these interpretations by demonstrating a clear separation in AMH values between cases and controls, despite comparable demographic characteristics. The observed elevation in FSH levels among women with endometriosis further supported the notion of impaired ovarian reserve. Increased early follicular phase FSH is commonly interpreted as a compensatory pituitary response to declining ovarian feedback. The combination of low AMH and elevated FSH has been described as a more robust indicator of reduced ovarian reserve than either marker alone. In this context, the findings suggested that endocrine adaptations associated with endometriosis may be detectable even before overt infertility or advanced reproductive aging becomes clinically evident. The modest but statistically significant increase in LH levels added further nuance, indicating subtle alterations in hypothalamic–pituitary–ovarian axis dynamics rather than gross endocrine disruption (12).

Comparison with earlier clinic-based studies revealed broad consistency, particularly regarding the direction and magnitude of AMH and FSH changes. Some investigations have reported more pronounced LH disturbances, while others have found no significant difference, suggesting that LH alterations may be more sensitive to disease stage, symptom severity, or timing of hormonal assessment (13). The relatively small difference in LH levels observed in this study may therefore reflect early or moderate disease impact, inter-individual variability, or the restrictive sampling window used to minimize hormonal fluctuation. The implications of these findings were clinically relevant, particularly in counseling women with endometriosis regarding fertility potential and reproductive planning (14). Early identification of reduced ovarian reserve may assist clinicians in tailoring fertility management strategies, optimizing the timing of conception attempts, or considering fertility preservation in selected cases. The results also underscored the importance of incorporating objective hormonal assessment into routine evaluation rather than relying solely on symptom burden or imaging findings. Several strengths supported the validity of this study. Age-matched controls and strict inclusion and exclusion criteria reduced the likelihood of confounding from other endocrine or gynecological conditions. Hormonal measurements were standardized and obtained during the early follicular phase, enhancing comparability across participants. The use of multiple ovarian reserve markers provided a more comprehensive assessment than reliance on a single hormone (15).

Nonetheless, certain limitations warranted consideration. The relatively small sample size and short study duration limited generalizability and precluded subgroup analysis based on disease severity or lesion location. The descriptive design did not allow for causal inference, and longitudinal changes in hormonal profiles could not be assessed (16). Radiological and clinical diagnosis, although reflective of routine practice, may have introduced some heterogeneity in disease characterization. Additionally, ovarian reserve markers were not correlated with direct fertility outcomes such as time to conception or assisted reproductive success, which would have strengthened clinical interpretation (17). Future research would benefit from larger, multicenter designs incorporating longitudinal follow-up to clarify the trajectory of ovarian reserve decline in endometriosis. Stratification by disease stage, prior surgical intervention, and symptom duration may further elucidate differential hormonal patterns. Integration of biochemical markers with functional reproductive outcomes would also enhance translational relevance. In summary, the findings provided coherent evidence that endometriosis was associated with altered ovarian reserve markers, most notably reduced AMH and elevated FSH levels. While cautious interpretation was warranted, the results contributed to an evolving understanding of the endocrine dimension of endometriosis and supported the need for proactive reproductive assessment in affected women (18).

CONCLUSION

This study demonstrated that women with endometriosis exhibited significantly lower anti-Müllerian hormone levels and higher follicle-stimulating hormone levels compared with healthy controls, indicating a compromised ovarian reserve. Subtle alterations in luteinizing hormone were also observed. These findings highlighted the importance of early hormonal assessment in women with endometriosis to support timely fertility counseling and informed reproductive planning.

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DECLARATIONS

Ethical Approval
Ethical approval was by institutional review board of Respective Institute

Informed Consent
NA

Conflict of Interest
The authors declare no conflict of interest.

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Authors' Contributions
Concept: AI; Design: AI, NS; Data Collection: AI, LM, RA, SKN, HAA; Analysis: NS, LM; Drafting: AI, NS

Data Availability
The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Acknowledgments
Not applicable.

Study Registration
Not applicable.