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Original Article

Correlation of Hormonal Profile with Pelvic Ultrasonographic Findings in Anovulatory Infertility

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ABSTRACT

Background: Anovulatory infertility is commonly associated with endocrine dysregulation and abnormal ovarian morphology, particularly in women with polycystic ovarian syndrome. Hormonal assays provide functional information about gonadotropin activity, ovarian reserve, and thyroid-related endocrine influence, while pelvic ultrasonography demonstrates follicular burden, dominant follicle development, ovarian morphology, and endometrial response. Objective: To evaluate the association between hormonal profile and pelvic ultrasonographic findings among women with anovulatory infertility. Methods: This cross-sectional analytical study included 100 women aged 18–40 years presenting with infertility and suspected anovulation at Gillani Ultrasound Center. Hormonal parameters included follicle-stimulating hormone, luteinizing hormone, anti-Müllerian hormone, and thyroid-stimulating hormone. Pelvic ultrasonographic parameters included right and left antral follicle count, total antral follicle count, endometrial thickness, dominant follicle status, and PCOS morphology. Data were analyzed using descriptive statistics and independent-samples t-tests, with $p \leq 0.05$ considered statistically significant. Results: Mean age was 30.25±5.16 years and mean infertility duration was 3.53±1.51 years. PCOS morphology was present in 51.0%, irregular cycles in 51.0%, and dominant follicles were absent in 51.0%. Women with PCOS had significantly higher LH, AMH, TSH, and total AFC, while FSH was higher in women without PCOS (all $p<0.001$). Conclusion: Combined hormonal and pelvic ultrasonographic assessment demonstrated clinically meaningful endocrine-ultrasonographic differences in women with anovulatory infertility, particularly according to PCOS status. Keywords: Anovulatory infertility; hormonal profile; pelvic ultrasonography; anti-Müllerian hormone; antral follicle count; polycystic ovarian syndrome.

INTRODUCTION

Infertility is a clinically and psychosocially significant reproductive health problem, commonly defined as failure to achieve pregnancy after 12 months of regular unprotected intercourse. Its burden extends beyond biological impairment because delayed conception often produces emotional distress, social pressure, marital strain, and reduced quality of life, particularly in settings where motherhood carries strong cultural and familial expectations. Global estimates indicate that infertility affects a substantial proportion of couples during their reproductive years, with female-related factors contributing to a considerable share of cases (1). Accurate identification of the underlying cause is therefore essential because infertility is not a single disease entity but a clinical outcome arising from endocrine, ovarian, tubal, uterine, cervical, male-factor, or multifactorial disturbances. Standardized definitions and diagnostic categories are important for clinical decision-making, research comparability, and appropriate treatment planning (2).

Anovulatory infertility represents an important and potentially treatable contributor to female infertility. It occurs when follicular recruitment, maturation, dominant follicle selection, or ovulation is disrupted, usually because of abnormalities within the hypothalamic-pituitary-ovarian axis or associated endocrine systems. Polycystic ovary syndrome is among the most frequent causes of anovulatory infertility and is typically associated with menstrual irregularity, altered gonadotropin dynamics, increased anti-Müllerian hormone levels, excess small antral follicles, and polycystic ovarian morphology on ultrasonography (3). Other endocrine abnormalities, including thyroid

dysfunction and hyperprolactinemia, may also interfere with gonadotropin-releasing hormone pulsatility, follicular maturation, luteal function, and endometrial receptivity, thereby contributing to irregular cycles and impaired conception. Because these mechanisms may overlap in the same patient, isolated clinical assessment is often insufficient for defining the dominant pathway of reproductive dysfunction.

Hormonal profiling provides functional information about ovarian reserve, follicular activity, and endocrine regulation. Follicle-stimulating hormone and luteinizing hormone are commonly assessed to evaluate early follicular phase gonadotropin patterns, while anti-Müllerian hormone reflects the pool of growing pre-antral and small antral follicles and is widely used as a marker of ovarian reserve. Anti-Müllerian hormone has particular relevance in anovulatory infertility because it correlates with antral follicle count and may be elevated in women with polycystic ovarian morphology due to increased follicular mass (4). Thyroid-stimulating hormone and prolactin assessment can further identify endocrine disturbances that may impair ovulation through indirect effects on the hypothalamic-pituitary-ovarian axis. However, hormonal values alone do not fully describe ovarian morphology, follicular development, or endometrial response, and their interpretation is strengthened when assessed alongside pelvic ultrasonographic findings.

Pelvic ultrasonography is a noninvasive and clinically practical method for assessing ovarian morphology, antral follicle count, dominant follicle development, ovarian volume, and endometrial thickness. These sonographic parameters provide structural and functional evidence of follicular activity and endometrial response during the menstrual cycle. In infertility evaluation, ultrasonography helps identify polycystic ovarian morphology, absence of a dominant follicle, altered antral follicle distribution, and abnormal endometrial thickness, all of which may reflect underlying endocrine dysfunction (5). Ultrasound assessment is especially valuable when interpreted in relation to hormonal findings, because the combination can clarify whether biochemical abnormalities are accompanied by measurable ovarian or endometrial changes. In women with suspected anovulation, increased antral follicle count with elevated anti-Müllerian hormone, altered luteinizing hormone patterns, and absent dominant follicle formation may provide a more clinically coherent picture than any single marker alone (6).

Despite the recognized role of both hormonal assays and pelvic ultrasonography in infertility assessment, their combined interpretation remains inconsistently reported in routine clinical studies, particularly in local diagnostic settings where patient presentation may be delayed and access to advanced infertility workup may be limited. Many studies describe hormonal abnormalities or ultrasound findings separately, but fewer present integrated evidence showing how specific hormonal parameters correspond with follicular count, dominant follicle formation, endometrial thickness, and polycystic ovarian morphology in women with anovulatory infertility. This creates a practical knowledge gap because clinicians require evidence that links biochemical and imaging findings in a way that can support early diagnostic classification and individualized management.

Therefore, the present study aimed to evaluate the correlation between hormonal profile and pelvic ultrasonographic findings among women with anovulatory infertility. The study specifically assessed the relationship of follicle-stimulating hormone, luteinizing hormone, anti-Müllerian hormone, thyroid-stimulating hormone, and prolactin with ultrasonographic parameters including antral follicle count, dominant follicle status, endometrial thickness, ovarian morphology, and presence of polycystic ovarian morphology. The study was guided by the hypothesis that abnormal hormonal profiles are significantly associated with altered pelvic ultrasonographic findings in women with anovulatory infertility.

MATERIALS AND METHODS

This cross-sectional analytical study was conducted at Gillani Ultrasound Center to evaluate the association between hormonal profile and pelvic ultrasonographic findings among women presenting with infertility and suspected anovulation. The study design was selected because the objective was to assess the relationship between biochemical hormonal markers and sonographic reproductive parameters measured during the same diagnostic evaluation rather than to determine temporal causality or treatment response. A total of 100 eligible women were enrolled through convenience sampling after assessment against predefined eligibility criteria.

Women aged 18–40 years who presented with infertility, irregular menstrual cycles, suspected anovulation, or clinically indicated hormonal assessment were included. Participants with pelvic malignancy, congenital uterine abnormalities, previous ovarian surgery, history of oophorectomy, prior major pelvic surgery, or severe systemic illness were excluded to reduce the likelihood that structural pathology, surgical alteration, or serious comorbidity would independently distort ovarian morphology, follicular assessment, or endocrine interpretation. Eligible

participants were approached during clinical evaluation, informed about the study purpose and procedures, and enrolled after written informed consent. Demographic and clinical information, including age, type of infertility, duration of infertility, menstrual history, and relevant reproductive history, was recorded using a structured data collection approach.

Hormonal evaluation included follicle-stimulating hormone, luteinizing hormone, anti-Müllerian hormone, thyroid-stimulating hormone, and prolactin. These variables were selected because they represent key functional domains of anovulatory infertility: gonadotropin regulation, ovarian reserve, thyroid-related endocrine influence, and prolactin-related ovulatory suppression. Follicle-stimulating hormone and luteinizing hormone were used to assess gonadotropin patterns related to follicular recruitment and ovulatory dysfunction. Anti-Müllerian hormone was used as a marker of ovarian reserve and follicular pool. Thyroid-stimulating hormone and prolactin were included to identify endocrine disturbances that may interfere with menstrual cyclicity and ovulation. Hormonal findings were interpreted in relation to pelvic ultrasonographic parameters rather than as isolated diagnostic endpoints.

Pelvic ultrasonography was performed using a high-resolution grayscale ultrasound system according to standardized scanning procedures. Ultrasonographic assessment included ovarian morphology, right antral follicle count, left antral follicle count, total antral follicle count, presence or absence of a dominant follicle, endometrial thickness, and presence or absence of polycystic ovarian morphology. Antral follicle count was recorded separately for both ovaries and then summed to obtain total antral follicle count. Dominant follicle status was categorized as present or absent according to sonographic evidence of follicular maturation during the follicular phase assessment. Endometrial thickness was measured as a sonographic marker of endometrial response. Polycystic ovarian morphology was recorded when ultrasound findings were consistent with multiple small follicles and altered ovarian morphology suggestive of PCOS. All measurements were recorded on the data collection sheet immediately after assessment to reduce transcription error.

The main exposure variables were hormonal parameters, including follicle-stimulating hormone, luteinizing hormone, anti-Müllerian hormone, thyroid-stimulating hormone, and prolactin. The main outcome variables were pelvic ultrasonographic findings, including right antral follicle count, left antral follicle count, total antral follicle count, dominant follicle status, endometrial thickness, ovarian morphology, and polycystic ovarian morphology status. Additional clinical variables included age, infertility duration, infertility type, and menstrual cycle pattern. Infertility type was categorized as primary or secondary infertility. Menstrual cycle pattern was categorized as regular or irregular. Dominant follicle and PCOS status were treated as categorical variables, while age, duration of infertility, hormonal levels, antral follicle counts, and endometrial thickness were treated as continuous variables.

Potential sources of bias were addressed through predefined inclusion and exclusion criteria, standardized recording of demographic and reproductive variables, uniform documentation of hormonal and sonographic parameters, and exclusion of participants with previous ovarian surgery, oophorectomy, pelvic malignancy, congenital uterine abnormality, severe systemic illness, or major pelvic surgery. These exclusions were applied to reduce confounding from structural, surgical, or systemic factors that could independently affect ovarian reserve, follicular morphology, or endometrial appearance. Because convenience sampling was used, the findings were interpreted as clinic-based associations rather than population-level prevalence estimates. Age, infertility duration, menstrual cycle pattern, infertility type, and PCOS status were considered clinically relevant variables during interpretation because each may influence the relationship between hormonal profile and ultrasonographic findings.

Data were entered and analyzed using IBM SPSS Statistics version 27. Continuous variables were summarized using mean, standard deviation, minimum, and maximum values. Categorical variables were summarized using frequencies and percentages. Group comparisons between women with and without polycystic ovarian morphology were conducted using independent-samples t-tests for continuous hormonal and ultrasonographic variables. Pearson correlation analysis was planned to assess relationships between normally distributed continuous hormonal variables and continuous ultrasonographic parameters, including antral follicle count and endometrial thickness. For categorical outcomes such as dominant follicle status and PCOS status, group-based comparisons were considered more appropriate than simple linear correlation. Statistical significance was set at $p \leq 0.05$. Effect sizes were reported where group comparisons were performed to support interpretation of the clinical magnitude of observed differences in addition to statistical significance.

Data integrity was maintained by structured data entry, review of recorded values for completeness, and verification of continuous variables for plausible biological ranges before analysis. Participant confidentiality was protected by anonymizing study records and restricting access to collected data. The study was conducted in accordance with ethical principles for human participant research and followed the rules and regulations of the ethical committee of GC University, Faisalabad. Written informed consent was obtained from all participants before enrollment. Participants were informed about the study procedures, confidentiality of their information, voluntary participation, and their right to withdraw at any stage without penalty.

RESULTS

A total of 100 women with infertility and suspected anovulatory dysfunction were included in the analysis. The mean age of the participants was 30.25±5.16 years, with an age range of 20–38 years. The mean duration of infertility was 3.53±1.51 years, ranging from 1.10 to 6.90 years. The mean follicle-stimulating hormone level was 7.00±2.36 mIU/mL, while the mean luteinizing hormone level was 9.26±4.42 mIU/mL. The mean anti-Müllerian hormone level was 4.82±2.57 ng/mL, and the mean thyroid-stimulating hormone level was 2.96±1.70 µIU/mL. On pelvic ultrasonography, the mean right antral follicle count was 11.41±6.41 and the mean left antral follicle count was 11.88±6.36, producing a mean total antral follicle count of 23.29±12.76. Mean endometrial thickness was 8.33±2.29 mm, indicating measurable variation in endometrial response across the study population.

The categorical profile of the study population showed a near-balanced distribution of primary and secondary infertility. Primary infertility was present in 48 women, while secondary infertility was reported in 52 women. Menstrual cycle pattern was similarly distributed, with 49 participants reporting regular cycles and 51 reporting irregular cycles. Dominant follicle formation was absent in 51 participants and present in 49 participants, indicating that slightly more than half of the study population showed sonographic evidence consistent with impaired follicular maturation at the time of assessment. Polycystic ovarian morphology was present in 51 participants and absent in 49 participants, showing that PCOS-related morphology was common among women evaluated for anovulatory infertility.

Table 1. Baseline Clinical, Hormonal, and Ultrasonographic Characteristics of Participants (n=100)

Variable	n	Minimum	Maximum	Mean ± SD
Age, years	100	20.00	38.00	30.25±5.16
Duration of infertility, years	100	1.10	6.90	3.53±1.51
Follicle-stimulating hormone, mIU/mL	100	3.05	11.57	7.00±2.36
Luteinizing hormone, mIU/mL	100	3.03	17.75	9.26±4.42
Anti-Müllerian hormone, ng/mL	100	1.01	8.99	4.82±2.57
Thyroid-stimulating hormone, µIU/mL	100	0.56	5.99	2.96±1.70
Right antral follicle count	100	2.00	22.00	11.41±6.41
Left antral follicle count	100	3.00	22.00	11.88±6.36
Total antral follicle count	100	5.00	44.00	23.29±12.76
Endometrial thickness, mm	100	5.10	13.00	8.33±2.29

Table 2. Distribution of Infertility Type, Menstrual Pattern, Dominant Follicle Status, and PCOS Status (n=100)

Variable	Category	Frequency	Percentage
Infertility type	Primary infertility	48	48.0
	Secondary infertility	52	52.0
Menstrual cycle pattern	Regular cycle	49	49.0
	Irregular cycle	51	51.0
Dominant follicle status	Absent	51	51.0
	Present	49	49.0
Polycystic ovarian morphology / PCOS status	Absent	49	49.0
	Present	51	51.0

Independent-samples t-test analysis demonstrated marked hormonal and ultrasonographic differences between women with and without polycystic ovarian morphology. Women without PCOS had significantly higher follicle-stimulating hormone levels than women with PCOS, with a mean difference of 3.90 mIU/mL (95% CI: 3.37 to 4.42; $p<0.001$; Cohen's $d=2.92$). In contrast, women with PCOS had substantially higher luteinizing hormone levels, with a mean difference of −7.59 mIU/mL when PCOS-absent participants were compared with PCOS-present participants (95% CI: −8.48 to −6.69; $p<0.001$; Cohen's $d=-3.37$). Anti-Müllerian hormone also differed strongly between groups, with women with PCOS showing a mean AMH level of 7.10±1.18 ng/mL compared with 2.46±0.96 ng/mL in women without PCOS, producing a mean difference of −4.64 ng/mL (95% CI: −5.07 to −4.21; $p<0.001$; Cohen's $d=-4.30$). Thyroid-stimulating hormone was also higher among women with PCOS,

with a mean difference of −2.97 µIU/mL (95% CI: −3.30 to −2.65; p<0.001; Cohen's d=−3.63). The largest group difference was observed for total antral follicle count, which was markedly higher in women with PCOS than in those without PCOS, with a mean difference of −23.94 follicles (95% CI: −25.64 to −22.24; p<0.001; Cohen's d=−5.59). These findings indicate that PCOS status was associated with a distinct biochemical and sonographic pattern characterized by higher LH, AMH, TSH, and total AFC, together with lower FSH.

Table 3. Comparison of Hormonal and Ultrasonographic Parameters Between Women with and Without PCOS

Variable	PCOS Absent (n=49), Mean ± SD	PCOS Present (n=51), Mean ± SD	Mean Difference*	95% CI	t	df	p-value	Cohen's d
Follicle-stimulating hormone, mIU/mL	8.99±1.47	5.09±1.19	3.90	3.37 to 4.42	14.596	98	<0.001	2.92
Luteinizing hormone, mIU/mL	5.40±1.40	12.98±2.84	−7.59	−8.48 to −6.69	−16.836	98	<0.001	−3.37
Anti-Müllerian hormone, ng/mL	2.46±0.96	7.10±1.18	−4.64	−5.07 to −4.21	−21.492	98	<0.001	−4.30
Thyroid-stimulating hormone, µIU/mL	1.44±0.57	4.41±1.01	−2.97	−3.30 to −2.65	−18.135	98	<0.001	−3.63
Total antral follicle count	11.08±3.44	35.02±4.97	−23.94	−25.64 to −22.24	−27.917	98	<0.001	−5.59

*Mean difference calculated as PCOS absent minus PCOS present.

The comparative findings show that women with PCOS had a clearly different endocrine-ultrasonographic profile from women without PCOS. The most clinically prominent difference was observed in total antral follicle count, which was more than three times higher in the PCOS group than in the non-PCOS group. Anti-Müllerian hormone showed a similarly strong group separation, with mean AMH in the PCOS group approximately 4.64 ng/mL higher than in the non-PCOS group. Luteinizing hormone was also substantially elevated in the PCOS group, while follicle-stimulating hormone was higher in women without PCOS. The direction of these findings is consistent with a PCOS-related anovulatory pattern in which increased follicular pool and altered gonadotropin balance are reflected by elevated AMH, increased AFC, higher LH, and reduced FSH relative to women without PCOS. The higher mean thyroid-stimulating hormone level observed in the PCOS group also suggests that thyroid-related endocrine variation may coexist with anovulatory patterns in this study population, although causal inference cannot be made from the cross-sectional design.

Overall, the results indicate that women presenting with anovulatory infertility had substantial variability in hormonal profile and pelvic ultrasonographic parameters. PCOS was present in just over half of the participants and was associated with significantly higher LH, AMH, TSH, and total AFC, whereas women without PCOS had significantly higher FSH. Dominant follicle formation was absent in 51% of participants, and menstrual irregularity was reported by 51%, supporting the clinical relevance of combined endocrine and ultrasonographic assessment in this population. The available results support significant group-level associations between PCOS status and hormonal-ultrasonographic markers; however, direct correlation coefficients between individual hormonal variables and ultrasound parameters should be added in the final manuscript to fully address the stated correlation objective.

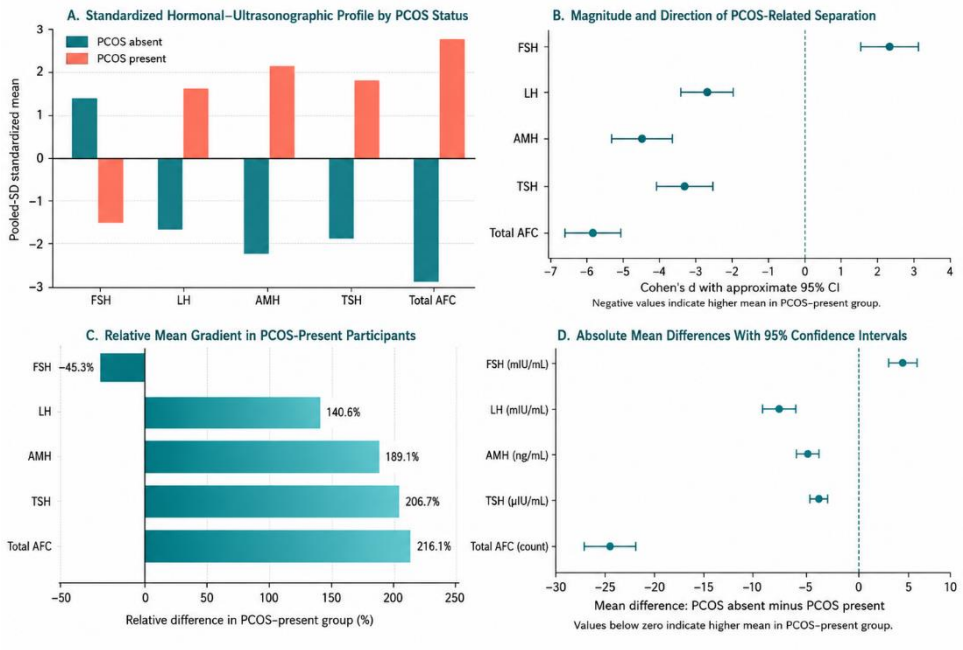

Figure 1 Integrated Endocrine–Ultrasonographic Differentiation by PCOS Status in Women With Anovulatory Infertility. Panel A presents pooled-SD standardized mean profiles for hormonal and ultrasonographic variables. Panel B shows Cohen's d with approximate 95% confidence intervals, where

negative values indicate higher means in the PCOS-present group. Panel C displays relative mean gradients in the PCOS-present group compared with the PCOS-absent group. Panel D presents absolute mean differences with 95% confidence intervals calculated as PCOS absent minus PCOS present.

DISCUSSION

This cross-sectional analytical study evaluated the association between hormonal profile and pelvic ultrasonographic findings among women with anovulatory infertility. The findings showed that the study population was within the active reproductive age range, with a mean age of 30.25±5.16 years and mean infertility duration of 3.53±1.51 years. Secondary infertility was slightly more frequent than primary infertility, affecting 52.0% of participants, while 51.0% reported irregular menstrual cycles. Dominant follicle formation was absent in 51.0% of women, and polycystic ovarian morphology was present in 51.0%, indicating that more than half of the participants showed clinical or sonographic features compatible with anovulatory dysfunction. These findings are clinically relevant because infertility is a multifactorial reproductive health problem, and ovulatory dysfunction remains one of the major female-related contributors requiring integrated endocrine and imaging evaluation (1,2).

The hormonal profile of the participants demonstrated considerable variability across gonadotropin, ovarian reserve, and thyroid-related markers. Mean follicle-stimulating hormone was 7.00±2.36 mIU/mL, mean luteinizing hormone was 9.26±4.42 mIU/mL, mean anti-Müllerian hormone was 4.82±2.57 ng/mL, and mean thyroid-stimulating hormone was 2.96±1.70 µIU/mL. On ultrasonography, the mean total antral follicle count was 23.29±12.76, with mean right and left antral follicle counts of 11.41±6.41 and 11.88±6.36, respectively. These values demonstrate a broad endocrine-ultrasonographic spectrum among women presenting with suspected anovulatory infertility. The coexistence of elevated follicular reserve markers, increased antral follicle counts, menstrual irregularity, and absent dominant follicle formation supports the biological plausibility of impaired follicular maturation in a substantial proportion of the study population.

A key finding of the study was the marked difference in hormonal and ultrasonographic parameters between women with and without PCOS. Women with PCOS had substantially higher luteinizing hormone, anti-Müllerian hormone, thyroid-stimulating hormone, and total antral follicle count, whereas follicle-stimulating hormone was higher among women without PCOS. The difference in total antral follicle count was particularly large, with mean AFC increasing from 11.08±3.44 in women without PCOS to 35.02±4.97 in women with PCOS. This pattern is consistent with the expected sonographic phenotype of polycystic ovarian morphology, where multiple small antral follicles accumulate without appropriate dominant follicle selection. The very large effect size for total AFC indicates that antral follicle burden was the strongest ultrasonographic separator between PCOS-present and PCOS-absent participants in this dataset.

Anti-Müllerian hormone also showed a strong group-level difference, increasing from 2.46±0.96 ng/mL in women without PCOS to 7.10±1.18 ng/mL in women with PCOS. This finding is biologically coherent because AMH is produced by granulosa cells of pre-antral and small antral follicles and is expected to rise when the small follicle pool is increased. Previous literature supports the role of AMH as a marker of ovarian reserve and as a biochemical correlate of antral follicle count, particularly in women with polycystic ovarian morphology (3). In the present study, the parallel elevation of AMH and total AFC among women with PCOS reinforces the value of interpreting hormonal and sonographic findings together rather than relying on either marker in isolation.

The gonadotropin profile further supports an anovulatory pattern among women with PCOS. Luteinizing hormone was substantially higher in the PCOS-present group, whereas follicle-stimulating hormone was lower compared with women without PCOS. This altered gonadotropin balance is clinically important because adequate FSH stimulation is required for follicular recruitment and maturation, while disproportionate LH activity may contribute to abnormal follicular development and failure of dominant follicle selection. Current clinical reviews describe PCOS as a disorder characterized by menstrual disturbance, ovulatory dysfunction, biochemical or clinical hyperandrogenism, and polycystic ovarian morphology, with gonadotropin and ovarian reserve markers often contributing to the diagnostic picture (4). The current findings are consistent with this framework, although the cross-sectional design limits interpretation to association rather than causation.

Thyroid-stimulating hormone was also higher among women with PCOS than among women without PCOS, with mean values of 4.41±1.01 µIU/mL and 1.44±0.57 µIU/mL, respectively. Thyroid dysfunction can disturb menstrual cyclicity and ovulatory regulation through effects on the hypothalamic-pituitary-ovarian axis, and TSH assessment is therefore commonly included in infertility evaluation. In this study, the higher TSH level in the PCOS group suggests that thyroid-related endocrine variation may coexist with PCOS-related anovulatory

features. However, because thyroid status was assessed cross-sectionally and no multivariable adjustment was performed for age, body mass index, metabolic variables, or medication history, this finding should be interpreted as an observed association rather than evidence of an independent causal effect.

The absence of a dominant follicle in 51.0% of participants provides clinically meaningful sonographic support for anovulatory dysfunction. Dominant follicle development reflects successful follicular selection and maturation, and its absence may indicate follicular arrest, endocrine imbalance, or cycle-phase variation. The near-equal distribution of dominant follicle presence and absence in this study suggests heterogeneity within the infertile population, with some women showing active follicular maturation and others demonstrating impaired or delayed follicular development. Endometrial thickness also varied across participants, with a mean of 8.33±2.29 mm, indicating differences in endometrial response that may be influenced by ovulatory status and hormonal environment. These findings highlight the value of ultrasound not only for identifying ovarian morphology but also for evaluating functional reproductive markers such as follicular maturation and endometrial development.

The present findings support the clinical utility of combined hormonal and ultrasonographic assessment in women with anovulatory infertility. Hormonal assays provide biochemical evidence of gonadotropin activity, ovarian reserve, and endocrine disturbance, while ultrasonography demonstrates ovarian morphology, follicle burden, dominant follicle status, and endometrial response. When interpreted together, these parameters offer a more complete clinical profile of reproductive dysfunction. This integrated approach is consistent with infertility evaluation pathways in which biochemical and imaging findings are used together to classify ovulatory disorders, identify PCOS-related patterns, and guide further clinical decision-making (5,6). However, the current study did not perform diagnostic accuracy analysis, receiver operating characteristic analysis, or longitudinal follow-up; therefore, the findings should be presented as association-based evidence rather than proof of diagnostic prediction or treatment benefit.

Several limitations should be considered when interpreting these findings. The study was conducted at a single center and used convenience sampling, which may limit generalizability to broader infertile populations. The sample size of 100 participants was adequate for descriptive and group-comparison analysis but may be insufficient for robust subgroup analysis or multivariable modeling. Hormonal levels may fluctuate according to menstrual cycle phase, and the study would be strengthened by clearer reporting of sampling timing, particularly for FSH and LH. Ultrasound assessment is operator-dependent, and reproducibility would be improved by specifying sonographer training, equipment details, probe frequency, route of scanning, and measurement protocol. Important confounders such as body mass index, insulin resistance, hyperandrogenism, medication use, and metabolic status were not presented, although these variables may influence both hormonal and ultrasonographic findings. In addition, prolactin was included in the stated hormonal profile but was not reported in the available results, and future revision should either add prolactin findings or remove it from the analysis framework.

Despite these limitations, the study provides clinically relevant evidence that women with PCOS-related anovulatory infertility show a distinct endocrine-ultrasonographic pattern characterized by higher LH, AMH, TSH, and total AFC, together with lower FSH. The magnitude of group differences, particularly for total AFC and AMH, suggests that ovarian follicle burden and ovarian reserve markers are central features of the observed PCOS phenotype. Future studies should use larger multicenter samples, prespecified hormone sampling protocols, standardized ultrasound definitions, and multivariable models adjusted for age, BMI, metabolic status, and infertility duration. Longitudinal follow-up linking hormonal-ultrasound profiles with ovulation induction response, conception, or pregnancy outcomes would further strengthen the clinical relevance of this integrated assessment approach.

CONCLUSION

This study concluded that hormonal profile was meaningfully associated with pelvic ultrasonographic findings among women with anovulatory infertility. Women with PCOS demonstrated significantly higher LH, AMH, TSH, and total AFC, while FSH was higher among women without PCOS, indicating a distinct endocrine-ultrasonographic pattern linked with polycystic ovarian morphology and impaired ovulatory function. Dominant follicle absence, menstrual irregularity, increased antral follicle count, and elevated AMH collectively support the clinical relevance of evaluating biochemical and sonographic markers together in women presenting with infertility and suspected anovulation. The findings support combined hormonal and pelvic ultrasonographic assessment as a practical approach for characterizing anovulatory infertility, although further multicenter studies

with standardized hormone timing, complete prolactin reporting, metabolic profiling, and longitudinal fertility outcomes are needed before predictive or diagnostic-accuracy claims can be made.

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