

# Hormonal Profile Results in Women with Menstrual Cycle Phase Dysfunction

Yuldasheva M. A., Shukurov F. I.\*

Tashkent State Medical University, Tashkent, Uzbekistan

**Abstract Background.** Infertility is one of the most prevalent gynecological problems among women of reproductive age, with hormonal and ovulatory dysfunctions being key etiological factors. Hormonal imbalance during different phases of the menstrual cycle negatively affects folliculogenesis, ovulation, and endometrial receptivity. **Objective.** To evaluate hormonal profile indicators in women with menstrual cycle phase dysfunction and assess their association with reproductive outcomes. **Materials and Methods.** A total of 120 women aged 18–40 years were included and divided into four groups: follicular phase dysfunction (n=30), ovulatory phase dysfunction (n=30), luteal phase dysfunction (n=30), and a control group of healthy women (n=30). Serum levels of FSH, LH, estradiol, estrone, estriol, progesterone, total and free testosterone, and DHEA-S were measured using ECLIA. **Results.** Women with follicular phase dysfunction showed elevated FSH and reduced estradiol and progesterone levels; ovulatory phase dysfunction was characterized by increased LH, testosterone, and DHEA-S; luteal phase dysfunction demonstrated marked reduction of progesterone and estradiol, indicating luteal insufficiency. The control group had values within physiological ranges. Correlation analysis revealed a significant association between estradiol levels and dominant follicle diameter and endometrial thickness ( $r=0.61-0.68$ ;  $p<0.01$ ). ROC analysis identified estradiol ( $>85$  pg/ml) and progesterone ( $>3.8$  ng/ml) as highly diagnostic cut-off values for predicting ovulation and implantation. **Conclusion.** Hormonal profile alterations significantly affect reproductive function. Estradiol, progesterone, testosterone, and DHEA-S serve as reliable biomarkers for assessing ovulation and implantation potential.

**Keywords** Infertility, Menstrual cycle, Hormonal dysfunction, Estradiol, Progesterone, Testosterone, Endometrial receptivity

## 1. Introduction

Infertility is one of the most prevalent gynecological problems among women of reproductive age, with prevalence rates estimated at 10–20% across different countries [1]. According to the World Health Organization (WHO), female factors—particularly hormonal and ovulatory dysfunctions—account for at least half of all infertility cases and represent key pathogenetic causes [2,3].

In the physiology of the menstrual cycle, the precise coordination of the follicular, ovulatory, and luteal phases plays a critical role. Hormonal imbalances at any phase can negatively influence ovulation, folliculogenesis, and endometrial receptivity [4]. For example, estrogen deficiency in the follicular phase may restrict follicular development and prevent dominant follicle formation [5]. Disruption of luteinizing hormone (LH) secretion during the ovulatory phase can lead to anovulation, while insufficient progesterone levels in the luteal phase result in endometrial hypoplasia,

reduced vascularization, and diminished implantation potential [6–8].

Hormonal dysfunctions often reflect only endocrine alterations and may not fully capture the morphofunctional changes in ovulation and endometrial receptivity. Therefore, in addition to hormonal profiling, echographic and Dopplerometric parameters, as well as metabolomic analyses, are considered essential for comprehensive evaluation [9,10].

Recent studies have highlighted that estradiol, estrone, estriol, progesterone, testosterone fractions, and dehydroepiandrosterone sulfate (DHEA-S) serve as key biomarkers that determine ovulatory processes, endometrial proliferation, and implantation efficiency [11–13]. Identifying alterations in the hormonal profile is crucial not only for understanding the pathogenesis but also for enabling individualized prognosis and the selection of personalized therapeutic strategies [14–16].

Moreover, hormonal profile analysis allows for the assessment of the diverse clinical manifestations of follicular, ovulatory, and luteal phase dysfunctions and their impact on reproductive function [17,18]. This contributes to a deeper understanding of infertility pathogenesis and provides opportunities to develop highly accurate diagnostic and

\* Corresponding author:

prof.farxadshukurov@gmail.com (Shukurov F. I.)

Received: Sep. 16, 2025; Accepted: Oct. 12, 2025; Published: Oct. 31, 2025

Published online at <http://journal.sapub.org/ajmms>

prognostic models [19,20].

Therefore, in this study, hormonal profile parameters were evaluated in women with menstrual cycle phase dysfunctions, with the aim of determining their impact on reproductive function and their clinical-prognostic significance.

## 2. Purpose of the Research

To assess the hormonal profile parameters in women with menstrual cycle phase dysfunctions and evaluate their association with reproductive function.

## 3. Materials and Methods

This study included a total of 120 women of reproductive age (18–40 years). All participants were stratified into four groups based on their hormonal status and menstrual cycle characteristics: Group I – women with follicular phase hormonal dysfunction (n = 30), Group II – women with ovulatory phase hormonal dysfunction (n = 30), Group III – women with luteal phase hormonal dysfunction (n = 30), and the comparison group – healthy women with physiological menstrual cycles (n = 30).

Inclusion criteria were reproductive age (18–40 years), failure to conceive for at least 12 months, laboratory and clinical confirmation of hormonal imbalance, and provision of written informed consent for participation in the study. Exclusion criteria included organic pathologies of the reproductive organs (endometriosis, uterine fibroids, tubal-peritoneal factors), severe somatic and endocrine diseases (diabetes mellitus, thyroid disorders), infectious diseases, and a history of long-term hormonal therapy.

For all women, blood samples were collected according to the respective phase of the menstrual cycle. The following parameters were determined: gonadotropins (FSH and LH), estrogen fractions (estradiol, estrone, estriol), progesterone, testosterone fractions (total and free), and dehydroepiandrosterone sulfate (DHEA-S). Additionally, the LH/FSH ratio was assessed. All hormonal measurements were performed using electrochemiluminescence immunoassay (ECLIA) on an Immulite 2000 XPI analyzer (Siemens,

Germany).

The obtained data were processed using SPSS version 25.0. Student's t-test was applied for parametric variables, while the  $\chi^2$  test was used for nonparametric data. Correlations between hormonal parameters and reproductive indicators were evaluated using Pearson's correlation analysis. In all cases, a p-value of <0.05 was considered statistically significant.

## 4. Results

Analysis of the results showed that in women of Group I with follicular phase hormonal dysfunction, mean FSH levels were  $10.2 \pm 0.7$  IU/L, LH –  $6.1 \pm 0.5$  IU/L, and the LH/FSH ratio –  $0.6 \pm 0.05$ . Estradiol concentration was  $38.5 \pm 4.1$  pg/mL, estrone –  $26.2 \pm 3.8$  pg/mL, estriol –  $4.3 \pm 0.7$  ng/mL, progesterone –  $0.7 \pm 0.1$  ng/mL, total testosterone –  $0.42 \pm 0.08$  ng/mL, free testosterone –  $2.3 \pm 0.4$  pg/mL, and DHEA-S –  $1.7 \pm 0.3$   $\mu$ g/mL. These findings reflect diminished ovarian reserve and impaired folliculogenesis, accompanied by reductions in estrogen and progesterone, which indicate decreased ovulatory activity.

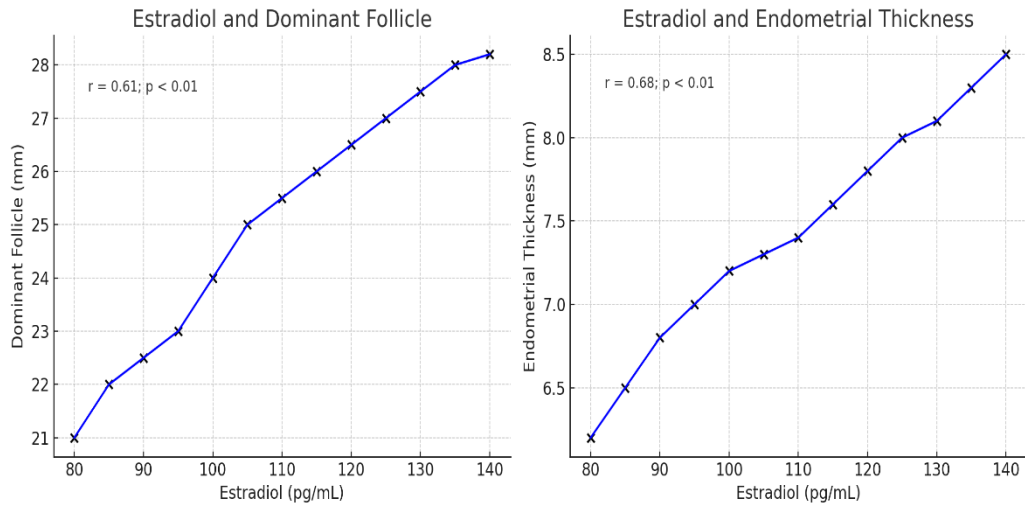
In Group II (ovulatory phase dysfunction), FSH was  $8.9 \pm 0.8$  IU/L, LH –  $17.5 \pm 1.1$  IU/L, and the LH/FSH ratio –  $1.6 \pm 0.2$ . Estradiol ( $E_2$ ) levels were  $110.3 \pm 8.8$  pg/mL, estrone ( $E_1$ ) –  $53.4 \pm 6.9$  pg/mL, estriol ( $E_3$ ) –  $6.8 \pm 1.2$  ng/mL, and progesterone –  $1.1 \pm 0.2$  ng/mL. Total testosterone was  $0.66 \pm 0.09$  ng/mL, free testosterone –  $3.7 \pm 0.5$  pg/mL, and DHEA-S –  $2.3 \pm 0.4$   $\mu$ g/mL. Relatively elevated LH, testosterone, and DHEA-S levels suggested androgenization during ovulation, impaired follicular development, and a risk of hyperandrogenemia.

In Group III (luteal phase dysfunction), mean FSH was  $9.8 \pm 0.6$  IU/L, LH –  $10.3 \pm 0.7$  IU/L, and the LH/FSH ratio –  $1.0 \pm 0.1$ . Estradiol was  $60.2 \pm 5.9$  pg/mL, estrone –  $39.8 \pm 4.4$  pg/mL, estriol –  $5.1 \pm 0.9$  ng/mL, progesterone –  $4.2 \pm 0.6$  ng/mL, total testosterone –  $0.37 \pm 0.07$  ng/mL, free testosterone –  $2.1 \pm 0.3$  pg/mL, and DHEA-S –  $1.6 \pm 0.2$   $\mu$ g/mL. The most prominent hormonal alterations in this group were significantly decreased progesterone and estradiol, reflecting luteal phase insufficiency and impaired implantation potential.

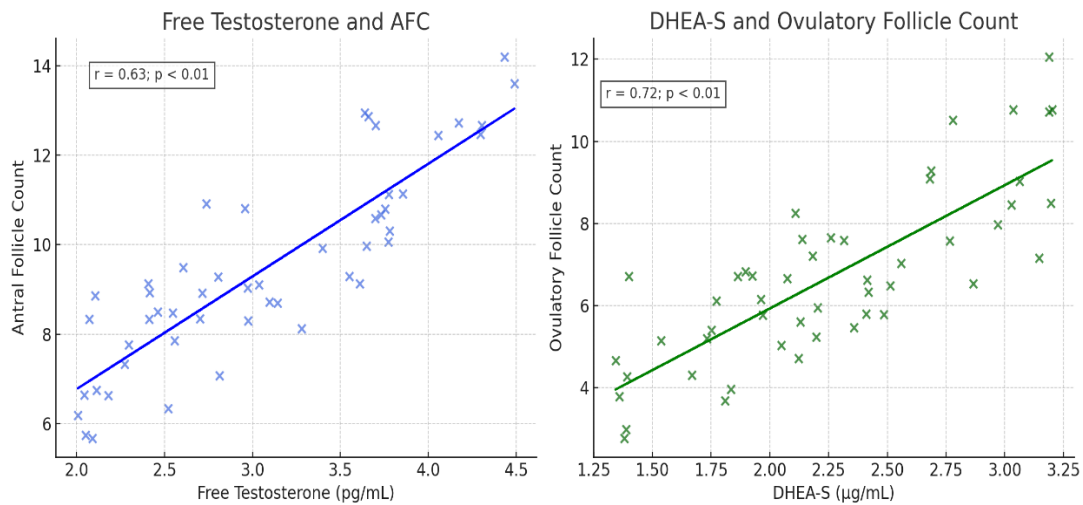
**Table 1.** Serum hormonal parameters in women with menstrual cycle phase dysfunctions (M  $\pm$  m)

Hormones	Group I (n=30)	Group II (n=30)	Group III (n=30)	Control group (n=30)	p
FSH (IU/L)	$10.2 \pm 0.7^{**}$	$8.9 \pm 0.8^{**}$	$9.8 \pm 0.6^{**}$	$6.1 \pm 0.5$	<0.01
LH (IU/L)	$6.1 \pm 0.5$	$17.5 \pm 1.1^{**}$	$10.3 \pm 0.7^{**}$	$7.4 \pm 0.6$	<0.01
LH/FSH ratio	$0.6 \pm 0.05$	$1.6 \pm 0.2^{**}$	$1.0 \pm 0.1$	$1.2 \pm 0.09$	<0.01
Estradiol (pg/mL)	$38.5 \pm 4.1^{**}$	$110.3 \pm 8.8^{**}$	$60.2 \pm 5.9^{**}$	$198.4 \pm 10.2$	<0.01
Estrone (pg/mL)	$26.2 \pm 3.8^{**}$	$53.4 \pm 6.9^{**}$	$39.8 \pm 4.4^{**}$	$75.6 \pm 5.2$	<0.01
Estriol (ng/mL)	$4.3 \pm 0.7^*$	$6.8 \pm 1.2^*$	$5.1 \pm 0.9^*$	$9.1 \pm 1.2$	<0.01
Progesterone (ng/mL)	$0.7 \pm 0.1^{**}$	$1.1 \pm 0.2^{**}$	$4.2 \pm 0.6^{**}$	$13.2 \pm 1.0$	<0.01
Total testosterone (ng/mL)	$0.42 \pm 0.08^*$	$0.66 \pm 0.09^*$	$0.37 \pm 0.07$	$0.54 \pm 0.06$	<0.05
Free testosterone (pg/mL)	$2.3 \pm 0.4^*$	$3.7 \pm 0.5^*$	$2.1 \pm 0.3$	$3.4 \pm 0.6$	<0.05
DHEA-S ( $\mu$ g/mL)	$1.7 \pm 0.3^*$	$2.3 \pm 0.4^*$	$1.6 \pm 0.2^*$	$2.9 \pm 0.3$	<0.05

**Note.** \* p < 0.05; \*\* p < 0.01; \*\*\* p < 0.001 – statistically significant differences compared with the control group.



**Figure 1.** Correlation between estradiol levels, dominant follicle size, and endometrial thickness in women included in the study



**Figure 2.** Correlation between free testosterone, AFC, and ovulatory follicles in women included in the study

In the control group, all parameters remained within physiological limits: FSH –  $6.1 \pm 0.5$  IU/L, LH –  $7.4 \pm 0.6$  IU/L, LH/FSH ratio –  $1.2 \pm 0.09$ , estradiol –  $198.4 \pm 10.2$  pg/mL, estrone –  $75.6 \pm 5.2$  pg/mL, estriol –  $9.1 \pm 1.2$  ng/mL, progesterone –  $13.2 \pm 1.0$  ng/mL, total testosterone –  $0.54 \pm 0.06$  ng/mL, free testosterone –  $3.4 \pm 0.6$  pg/mL, and DHEA-S –  $2.9 \pm 0.3$  µg/mL ( $p < 0.01$ ).

Results of multiparametric correlation analysis demonstrated that hormonal dysfunction in women had a clear and multifaceted impact on key reproductive parameters. The analysis revealed significant and clinically relevant correlations between estrogen levels (particularly estradiol and estrone), testosterone, and dehydroepiandrosterone sulfate (DHEA-S) with ovulatory dynamics, endometrial thickness, and the number of ovarian follicles.

In particular, estradiol levels showed a moderately strong positive correlation with ovulatory cycle dynamics, namely the development of the dominant follicle and the effectiveness of ovulation ( $r = 0.61$ ;  $p < 0.01$ ). This finding indicates that in women with ovulatory phase hormonal dysfunction, decreased folliculogenesis and impaired oocyte

maturation are closely linked to reduced estradiol concentrations. Furthermore, low estrogen levels negatively influenced endometrial receptivity and thickness, thereby compromising the formation of a trophic environment necessary for successful implantation (Figure 1).

The concentrations of testosterone and dehydroepiandrosterone sulfate (DHEA-S) demonstrated a moderate positive correlation with ovulatory activity and the number of follicles ( $r = 0.43$ – $0.56$ ;  $p < 0.01$ ). Specifically, patients with lower testosterone and DHEA-S levels exhibited a reduced number of antral follicles and impaired development of ovulatory follicles. This condition can be explained by diminished ovarian reserve, hypoandrogenemia, and trophic alterations of the ovarian stroma (Figure 2).

Furthermore, a decline in DHEA-S and testosterone levels below the physiological range was associated with impaired follicular development and a poorer reproductive prognosis.

Correlation analysis between endometrial thickness and hormonal biomarkers also revealed a moderate and statistically significant association. In particular, estradiol demonstrated a correlation coefficient of  $r = 0.68$ ; ( $p < 0.01$ )

with endometrial thickness. This indicates that patients with higher estradiol levels had greater endometrial proliferation and vascularization, thereby creating a more favorable environment for implantation. Conversely, women with reduced estrogen and androgen levels were more likely to present with endometrial hypoplasia and diminished receptivity.

The overall analysis demonstrated that ovulatory cycle dynamics, endometrial thickness, and follicle count are directly influenced by complex hormonal and metabolomic interactions. These multiparametric correlation results provide a deeper understanding of the pathogenesis of infertility, while also offering a strong scientific basis for the development of individualized prognostic models and personalized therapeutic strategies.

The relationships identified between hormonal parameters and reproductive outcomes, as well as their prognostic potential, were further evaluated using correlation analysis, ROC analysis, and the calculation of cut-off values. This approach provided the opportunity to develop evidence-based strategies that meet modern requirements for individualized diagnosis and prognosis.

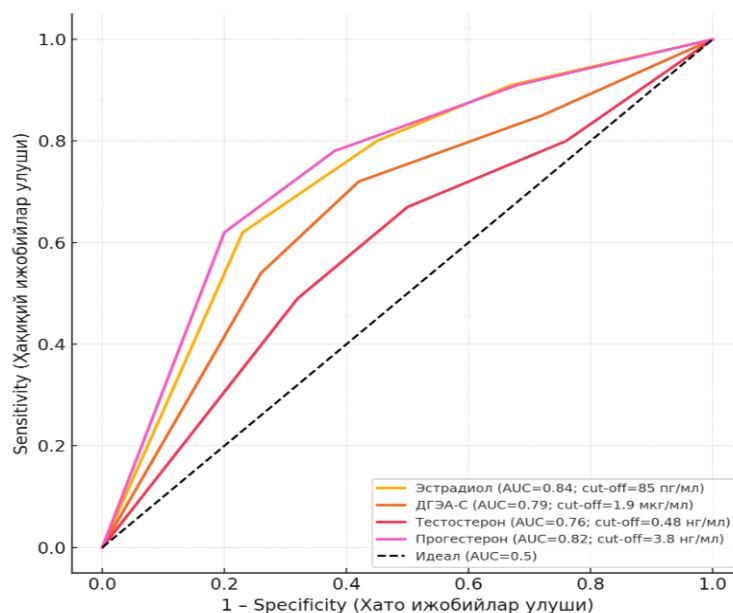
Correlation analysis revealed that estradiol, testosterone, and DHEA-S levels showed moderate to strong significant associations with ovulatory dynamics (dominant follicle diameter and follicle count) and endometrial thickness. Specifically, estradiol correlated with follicle diameter ( $r = 0.61$ ,  $p < 0.01$ ) and endometrial thickness ( $r = 0.68$ ,  $p < 0.01$ ), while testosterone and DHEA-S were significantly correlated with AFC ( $r = 0.43$ – $0.56$ ,  $p < 0.01$ ). These results demonstrate the substantial prognostic value of these hormones for reproductive function and implantation success. Alongside other diagnostic tools, ROC analysis was conducted to evaluate the diagnostic and prognostic significance of hormonal and echographic parameters. According to ROC analysis, the individualized cut-off value for estradiol in predicting ovulation was determined to be 85 pg/mL (AUC = 0.84; 95%

CI: 0.74–0.91; sensitivity 80%; specificity 77%). Women with estradiol levels below this threshold exhibited significantly lower ovulation efficiency and folliculogenesis indices.

For DHEA-S, the cut-off value for identifying follicular reserve and ovulation was 1.9  $\mu\text{g/mL}$  (AUC = 0.79; sensitivity 72%; specificity 74%). For testosterone, the cut-off value for predicting ovulation and follicle count was established at 0.48 ng/mL (AUC = 0.76). Progesterone, in turn, showed prognostic significance for luteal phase efficiency, with a cut-off value of 3.8 ng/mL (AUC = 0.82; sensitivity 78%; specificity 80%) (Figure 3).

The identified individual cut-off values provide a reliable scientific basis for the early diagnosis of hormonal dysfunctions, individualized assessment of ovulation and endometrial receptivity, formulation of effective treatment strategies, and the development of reproductive prognosis. To further strengthen the reliability and clinical significance of the findings, the associations between hormonal dysfunction and reproductive parameters were also evaluated using multivariate and adjusted analyses. This allowed for the determination of independent and integrated effects of multiple factors simultaneously within statistical models.

In the adjusted analyses (multivariate linear regression), the primary hormones (estradiol, progesterone, testosterone, DHEA-S) along with demographic (age, BMI) and clinical factors (somatic comorbidities, IVF history) were analyzed in relation to ovulation markers (dominant follicle diameter, endometrial thickness, and antral follicle count). Results indicated that estradiol was identified as the strongest independent predictor influencing ovulation efficiency and endometrial thickness ( $\beta = 0.37$ ;  $p < 0.001$ ). DHEA-S and free testosterone levels were significant independent predictors of follicle count and ovulation indices ( $\beta = 0.29$  and  $\beta = 0.21$ ;  $p < 0.01$ , respectively). Progesterone demonstrated a direct effect on endometrial receptivity and implantation efficiency ( $\beta = 0.26$ ;  $p < 0.01$ ).



**Figure 3.** ROC analysis of hormonal biomarkers in predicting ovulation and implantation

Even after adjusting for confounding variables such as age, obesity, dietary habits, and other background factors, the impact of hormonal dysfunction remained statistically significant (adjusted  $R^2 = 0.44$ ). At the same time, certain factors (e.g., obesity) were found to partially modify the effects of estrogens and androgens, which should be considered in individualized reproductive prognostication. Overall, the results of the multivariate analysis highlight that infertility pathogenesis and individual prognosis should not rely solely on a single hormonal parameter, but rather on the combined influence of multiple hormones and clinical factors. In practice, this supports the development of personalized therapeutic approaches and dynamic monitoring models tailored to each patient.

Thus, hormonal dysfunctions observed across different menstrual cycle phases in infertile women represent one of the key pathogenetic mechanisms affecting reproductive function. Comprehensive assessment and dynamic monitoring of cycle-specific hormonal profiles provide a fundamental basis for individualized therapy and prognosis. Combined hormonal–metabolomic analysis, supported by echographic and clinical parameters, serve as modern biomarkers for the accurate and reliable evaluation of fertility.

## 5. Discussion

The results of this study confirm that hormonal dysfunctions in different phases of the menstrual cycle play a decisive role in the pathogenesis of female infertility. Our data demonstrated that follicular phase disorders were associated with reduced estradiol and progesterone levels, impaired folliculogenesis, and diminished ovarian reserve, while ovulatory phase dysfunctions were characterized by excessive luteinizing hormone (LH) and hyperandrogenemia, and luteal phase insufficiency was marked by critically low progesterone and estradiol values. These findings are consistent with previous reports highlighting the central role of endocrine and ovulatory disorders in the etiology of infertility [1,3,4,7,11,12,13].

In particular, the luteal phase findings of our study, showing significantly decreased progesterone levels, are in line with the concept of luteal phase deficiency as a major factor impairing endometrial receptivity and implantation potential [5,17]. This corresponds with earlier reports that insufficient progesterone production leads to defective secretory transformation of the endometrium, reduced vascularization, and increased implantation failure rates [6,10].

The observed relationship between elevated LH, increased androgens (testosterone and DHEA-S), and impaired ovulatory dynamics reflects the pathogenic mechanisms of hyperandrogenemia in reproductive failure. Similar results have been reported in both clinical and experimental studies, confirming that excessive androgen activity suppresses follicular development, alters gonadotropin feedback, and negatively affects oocyte maturation [8,9,13].

Another important outcome of our study is the identified

correlation between estradiol levels and both follicular development and endometrial thickness ( $r=0.61-0.68$ ,  $p<0.01$ ). This supports the critical role of estrogens in regulating folliculogenesis and preparing the endometrium for implantation [14,18]. Moreover, reduced estrogen levels in infertile women with menstrual cycle dysfunctions explain the frequent findings of thin endometrium, hypoplasia, and impaired implantation rates.

The integration of metabolomic perspectives into reproductive endocrinology also enhances the interpretation of our results. Recent metabolomics-based investigations of follicular fluid have confirmed that altered profiles of steroid hormones, androgens, and metabolic intermediates are directly associated with poor ovarian response and reduced pregnancy outcomes in assisted reproductive technology (ART) cycles [15,16,19,20]. This aligns with our findings, underscoring the importance of comprehensive multiparametric analysis—combining hormonal, echographic, and metabolomic data—for precise diagnosis and prognosis.

The novelty of this study lies in its multiphase and multiparametric evaluation of hormonal dysfunction in infertile women. Unlike most previous studies that focused on single markers or single-cycle phases, our research provides a comprehensive analysis of estradiol, progesterone, androgens, and DHEA-S across different phases of the menstrual cycle, correlating them with ovarian reserve markers and endometrial receptivity. Such an integrated approach provides a more reliable prognostic framework for infertility diagnostics and the development of personalized therapeutic strategies.

Clinically, these findings highlight the necessity of not only routine hormonal testing but also the inclusion of advanced diagnostic approaches such as ROC-analysis and metabolomic profiling to determine individualized cut-off values for predictive markers. For instance, in our study, the estradiol cut-off of 85 pg/mL (AUC=0.84) was a reliable predictor of ovulation, while the progesterone threshold of 3.8 ng/mL (AUC=0.82) allowed accurate estimation of luteal phase competence. These thresholds may serve as practical tools for optimizing preconception counseling, ART protocols, and personalized treatment strategies.

Overall, our study supports the view that infertility in reproductive-aged women is not a result of isolated hormonal changes, but rather a multifactorial condition where endocrine dysfunction, ovarian reserve depletion, and endometrial receptivity impairment interact in a complex manner. Therefore, dynamic monitoring of the hormonal profile across menstrual phases, combined with metabolomic and echographic assessments, represents an evidence-based and clinically valuable approach to improving the outcomes of infertility treatment.

## 6. Conclusions

This study demonstrated that hormonal dysfunction across different phases of the menstrual cycle has a significant

negative impact on female reproductive function. Reduced levels of estrogen and progesterone, combined with elevated LH and androgens, were associated with impaired folliculogenesis, ovulatory dysfunction, and decreased endometrial receptivity. Correlation and ROC analyses confirmed that estradiol, progesterone, testosterone, and DHEA-S serve as highly valuable diagnostic markers for predicting ovulation and implantation outcomes. Therefore, comprehensive evaluation of the hormonal profile represents an essential tool for the early diagnosis of infertility and for guiding individualized therapeutic strategies.

**Conflict of Interest:** The authors declare that there is no conflict of interest regarding the publication of this paper.

**Funding:** This research was carried out as part of the institutional research activities of Tashkent State Medical University and received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

## REFERENCES

- [1] Abubakirov AN, Adamyan LV, Andreeva EN. Female infertility: diagnosis of the problem and ways to overcome it. *Okhrana materinstva i detsva* [Maternal and Child Health Protection]. 2019; 1(33): 42–47.
- [2] Urupina KV, Kutsenko II, Kravtsova EI. Endometrial factor of infertility in patients of late reproductive age. *Kuban Scientific Medical Bulletin*. 2020; 27(6): 149–163.
- [3] Adamyan LV, Makatsariya AD, Sukhikh GT. *Reproductive Medicine: A Guide for Physicians*. Moscow: GEOTAR-Media; 2021. 864 p.
- [4] Serov VN, Prilepskaya VN, Ovsyannikova TV. Menstrual cycle disorders: a modern view on diagnosis and treatment. *Akusherstvo i Ginekologiya* [Obstetrics and Gynecology]. 2020; (7): 15–21.
- [5] Tyutyunnik VL, Gasparov AS, Davydova YuA. Luteal phase deficiency: pathogenesis, diagnosis, treatment. *Akusherstvo, Ginekologiya i Reproduktsiya* [Obstetrics, Gynecology and Reproduction]. 2019; 13(2): 128–137.
- [6] Gasparov AS, Ovsyannikova TV, Sukhikh GT. Modern approaches to the assessment of hormonal profile in ovulatory disorders. *Rossiyskiy Vestnik Akushera-Ginekologa* [Russian Bulletin of Obstetrician-Gynecologist]. 2021; 21(5): 45–52.
- [7] Radzinsky VE, Ordiyants IM, Savel'yeva GM. *Gynecology: National Guidelines*. Moscow: GEOTAR-Media; 2020. 1056 p.
- [8] Anvarova ShA, Shukurov FI, Tulametova ShA. Innovative methods for solving the problem of female infertility associated with endocrine disorders. *Akusherstvo, Ginekologiya i Reproduktsiya* [Obstetrics, Gynecology and Reproduction]. 2024; 18(5): 706–719.
- [9] Dolgushina NV, Nazarenko TA. Hormonal markers of ovarian reserve: modern possibilities and limitations. *Problemy Reproduktsii* [Reproduction Problems]. 2018; 24(6): 36–43.
- [10] Sukhikh GT, Adamyan LV, Kurtser MA. *Modern Reproductive Technologies: Clinical Guidelines*. Moscow: GEOTAR-Media; 2022. 528 p.
- [11] World Health Organization. Infertility definitions and terminology. Geneva: WHO; 2020. 25 p.
- [12] Vander Borgh M, Wyns C. Fertility and infertility: Definition and epidemiology. *Clin Biochem*. 2018; 62: 2–10.
- [13] Carson SA, Kallen AN. Diagnosis and management of infertility: A review. *JAMA*. 2021; 326(1): 65–76.
- [14] Agarwal A, Aponte-Mellado A, Premkumar BJ, Shaman A, Gupta S. The effects of oxidative stress on female reproduction: a review. *Reprod Biol Endocrinol*. 2012; 10: 49.
- [15] Bracewell-Milnes T, Saso S, Abdalla H, et al. Metabolomics as a tool to identify biomarkers in reproductive medicine. *Hum Reprod Update*. 2017; 23(6): 723–736.
- [16] Gao J, Xiao Y. Metabolomics and its applications in assisted reproductive technology. *IET Nanobiotechnol*. 2023; 17: 399–405.
- [17] Devoto L, et al. The role of progesterone in luteal phase deficiency. *Fertil Steril*. 2016; 105(1): 27–37.
- [18] Vlaisavljević V, et al. Estradiol therapy in ART cycles: mechanisms and outcomes. *Reprod Biomed Online*. 2020; 41(2): 211–220.
- [19] Dogan B, Karaer A, Tuncay G, et al. NMR metabolomics of follicular fluid in advanced maternal age. *J Assist Reprod Genet*. 2020; 37: 321–330.
- [20] Li J, Zhang Z, Wei Y, et al. Metabonomic analysis of follicular fluid in diminished ovarian reserve. *Front Endocrinol*. 2023; 14: 1132621.