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Impact of Thyroid Status on Pro-Inflammatory Marker Profile in Women with Diminished Ovarian Reserve

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Abstract Diminished ovarian reserve (DOR) is increasingly recognized as a multifactorial condition influenced not only by age but also by endocrine and inflammatory mechanisms [1,2]. Thyroid dysfunction, particularly hypothyroidism, has been associated with systemic inflammation, which may contribute to impaired reproductive potential [3,4]. ***Objective:*** To evaluate systemic inflammatory markers in women with DOR according to thyroid status (hypothyroidism or euthyroidism), with a focus on C-reactive protein (CRP), complement components C3 and C4, and ceruloplasmin. ***Methods:*** A comparative cross-sectional study was conducted including 80 women of reproductive age diagnosed with diminished ovarian reserve. The study group consisted of 40 women with DOR and clinical or subclinical hypothyroidism, while the second group included 40 women with DOR and normal thyroid function. Serum levels of CRP, complement components C3 and C4, and ceruloplasmin were measured using standardized laboratory methods [5,6]. Statistical analysis was performed using Student's t-test in SPSS v26.0, and statistical significance was defined as $p < 0.05$. ***Results:*** Both groups demonstrated significantly elevated levels of CRP, complement C3 and C4, and ceruloplasmin, indicating the presence of systemic inflammation. However, women with DOR and hypothyroidism exhibited significantly higher concentrations of all assessed inflammatory markers compared with euthyroid women with DOR ($p < 0.05$) [7]. ***Conclusion:*** DOR is associated with a pronounced pro-inflammatory profile that is further exacerbated by hypothyroidism [8–10]. These findings highlight the potential role of thyroid-related systemic inflammation in reproductive dysfunction and support the inclusion of inflammatory marker assessment in the clinical evaluation of women with DOR.

Keywords Hypothyroidism, Diminished ovarian reserve, C-reactive protein, Complement C3, Complement C4, Ceruloplasmin, Female infertility

1. Introduction

Hypothyroidism is one of the most common endocrine disorders among women of reproductive age. According to epidemiological studies, its prevalence reaches approximately 4–5% in the general population, while subclinical hypothyroidism occurs in up to 5–7% of women [4,16]. Overt hypothyroidism occurs in approximately 2–4.5% of women, and autoimmune thyroiditis represents one of the leading causes of thyroid dysfunction in this population [2,4,7,16]. The highest detection rates are observed between 20 and 45 years of age, which makes thyroid disorders particularly relevant in the context of female reproductive health.

Thyroid dysfunction is known to affect multiple physiological systems, including reproductive function, metabolic regulation, and immune homeostasis [17]. Thyroid hormones play an essential role in the regulation of the hypothalamic–pituitary–ovarian axis. Deficiency of thyroid hormones may disrupt gonadotropin secretion, impair folliculogenesis, and reduce ovulatory potential, ultimately contributing to DOR and infertility [2,6,22]. Several studies have demonstrated that thyroid autoimmunity and hypothyroidism are associated with decreased ovarian reserve markers, including reduced anti-Müllerian hormone (AMH) levels and altered follicular development [1,10,21].

Ovarian reserve reflects the quantity and quality of ovarian follicles and represents an important predictor of female reproductive potential [18,19]. DOR has traditionally been associated with aging; however, growing evidence suggests that endocrine disorders, metabolic disturbances, and immune-inflammatory mechanisms may also contribute to premature decline of ovarian function [20,23,24].

Systemic inflammation has emerged as a potential factor influencing ovarian physiology. Inflammatory mediators may affect the ovarian microenvironment, granulosa cell function, and follicular maturation, thereby contributing to impaired reproductive outcomes [9,23]. Biomarkers such as CRP, complement components C3 and C4, and ceruloplasmin reflect activation of inflammatory and immune pathways and

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may serve as indicators of systemic inflammatory burden [5,8,25].

Hypothyroidism has been associated with increased production of inflammatory mediators and activation of complement pathways [5,7]. Complement proteins play an important role in innate immunity and inflammatory signaling and may contribute to tissue damage and immune-mediated alterations in reproductive organs [8].

Despite increasing recognition of the relationship between thyroid dysfunction, inflammation, and reproductive disorders, the inflammatory profile in women with diminished ovarian reserve according to thyroid status remains insufficiently studied. Therefore, further investigation is needed to clarify the interaction between endocrine dysfunction and inflammatory activation in women with reduced ovarian reserve.

The aim of the study: to compare the profiles of these markers in women with DOR under conditions of normal and impaired thyroid function.

2. Materials and Methods

Study design and participants

A comparative cross-sectional study was conducted at the Diyor Medical Centre, Tashkent, between January 4, 2024, and December 30, 2024. During the study period, 200 women underwent clinical examination, of whom 80 met the predefined inclusion and exclusion criteria and were enrolled in the study.

The study included women of reproductive age (18–37 years) with confirmed DOR. The diagnosis of DOR was established according to accepted clinical criteria, including reduced AMH (< 1.1 ng/mL) levels and/or decreased antral follicle count (AFC).

The participants were divided into two groups. Group 1 consisted of 40 women diagnosed with DOR in combination with clinical or subclinical hypothyroidism. Group 2 included 40 women with DOR and normal thyroid function (euthyroid status).

Hypothyroidism was diagnosed based on serum thyroid-stimulating hormone (TSH) and free thyroxine (fT4) concentrations, in accordance with international endocrine guidelines.

Inclusion criteria were: female sex, age 18–37 years, confirmed diminished ovarian reserve, and availability of complete clinical and laboratory data.

Exclusion criteria comprised pregnancy, polycystic ovary syndrome, other endocrine disorders (including diabetes mellitus and overt hyperprolactinemia), acute infectious or inflammatory diseases, chronic systemic diseases, and autoimmune disorders unrelated to thyroid pathology. Women who had received hormonal or immunomodulatory therapy within three months prior to enrollment were also excluded.

Sampling was performed using a consecutive sampling approach, whereby eligible patients presenting to the clinic during the study period were invited to participate.

The sample size was determined based on previously published studies investigating inflammatory markers in women with diminished ovarian reserve. A minimum of 36 participants per group was required to detect statistically significant differences with a statistical power of 80% and a significance level of 0.05.

Data collection instruments

Clinical and laboratory data were collected using standardized diagnostic protocols. The laboratory panel included markers of systemic inflammation and immune response such as C-reactive protein (CRP), complement components C3 and C4, and ceruloplasmin.

Serum CRP, complement C3, complement C4, and ceruloplasmin concentrations were measured using standardized biochemical and immunoturbidimetric assays. Thyroid function parameters, including TSH and free T4, were measured using enzyme-linked immunosorbent assay (ELISA) methods.

All laboratory measurements were performed using certified reagents and calibrated equipment in accordance with the manufacturer's instructions. All laboratory assays were performed in duplicate. Internal quality control procedures were applied to ensure the validity, accuracy, and reproducibility of the laboratory results.

Procedures

Eligible participants were identified during routine clinical consultations at the medical center. After screening for eligibility, women who met the inclusion criteria were invited to participate in the study.

Venous blood samples were collected from each participant during the early follicular phase of the menstrual cycle (days 2–4) in order to minimize hormonal variability. Blood samples were obtained by trained medical personnel using standard phlebotomy techniques.

All samples were processed and analyzed in an accredited clinical laboratory according to standardized operating procedures. Clinical and laboratory data were recorded in structured case report forms and subsequently transferred to an electronic database for statistical analysis.

Participants were recruited during routine outpatient consultations at the medical center.

Ethical considerations

The study protocol was reviewed and approved by the Ethics Committee of Tashkent State Medical University (Approval No. TSMU-EC-2024-05). All procedures were conducted in accordance with the ethical principles outlined in the Declaration of Helsinki. Participation in the study was voluntary, and the research was conducted anonymously. The confidentiality and anonymity of personal information were strictly maintained throughout the study, and all collected data were used exclusively for scientific purposes.

Data analysis

Statistical analysis was performed using IBM SPSS Statistics software (version 26.0). Descriptive statistics were used to summarize the study data. Quantitative variables were expressed as mean ± standard deviation (SD). The

Student's t-test for independent samples was applied to compare continuous variables between the two study groups, as this test is appropriate for assessing differences in means between independent groups. A p-value < 0.05 was considered statistically significant. The Student's t-test for independent samples was used to compare continuous variables between groups, as this test is appropriate for evaluating differences in mean values between two independent populations with approximately normally distributed data.

3. Results

Hormonal profile

Comparative analysis of the hormonal profile demonstrated statistically significant differences between Group 1 and Group 2 for the majority of the evaluated parameters. The results of intergroup comparisons are summarized in Table 1.

Patients in Group 1 (DOR with hypothyroidism) exhibited markedly elevated levels of anti-thyroid peroxidase antibodies (anti-TPO), with a mean concentration of 143.50 ± 18.85 IU/mL, which was significantly higher than that observed in Group 2 (14.05 ± 1.04 IU/mL; $p < 0.001$). These changes were accompanied by significantly increased serum TSH levels (6.97 ± 0.61 vs. 1.76 ± 0.08 mIU/L; $p < 0.001$) and a pronounced reduction in free thyroxine concentrations (0.76 ± 0.02 vs. 1.30 ± 0.02 ng/dL; $p < 0.001$), indicating overt thyroid dysfunction of autoimmune origin.

Assessment of the hypothalamic–pituitary–gonadal axis revealed significantly higher prolactin levels in Group 1 compared with Group 2 (33.62 ± 1.83 vs. 22.29 ± 1.45 ng/mL; $p < 0.001$), along with increased luteinizing hormone concentrations (7.97 ± 0.75 vs. 5.27 ± 0.50 mIU/mL; $p = 0.004$). In contrast, follicle-stimulating hormone levels did not differ significantly between groups ($p = 0.843$), suggesting predominant dysregulation of ovarian function rather than pituitary reserve depletion.

The steroid hormone profile was characterized by reduced estradiol levels in Group 1 compared with Group 2 (67.78 ±3.75 vs. 78.22 ±3.35 pg/mL; $p = 0.041$). Progesterone concentrations during the early follicular phase of the menstrual cycle (days 3–5) were significantly lower in Group 1 (0.28 ±0.04 vs. 0.51 ± 0.05 ng/mL; $p < 0.001$), reflecting impaired folliculogenesis and early luteinization. However, progesterone levels measured during the luteal phase (day 21) did not differ significantly between groups ($p = 0.198$).

Table 1. Comparative characteristics of hormonal profile parameters in the study groups

Parameter	Group 1: DOR with Hypothyroidism (Mean ±SD), n = 40	Group 2: DOR with Euthyroidism (Mean ±SD), n = 40	*p*-value
Anti-TPO antibodies (IU/mL)	143.50 ±18.85	14.05 ±1.04	<0.001
TSH (mIU/L)	6.97 ±0.61	1.76 ±0.08	<0.001
Free T4 (ng/dL)	0.76 ±0.02	1.30 ±0.02	<0.001
Prolactin (ng/mL)	33.62 ±1.83	22.29 ±1.45	<0.001
LH (mIU/mL)	7.97 ±0.75	5.27 ±0.50	0.004
FSH (mIU/mL)	9.11 ±1.08	8.85 ±0.78	0.843
Estradiol (pg/mL)	67.78 ±3.75	78.22 ±3.35	0.041
Progesterone (days 3–5), ng/mL	0.28 ±0.04	0.51 ±0.05	<0.001
Progesterone (day 21), ng/mL	5.88 ±0.53	7.06 ±0.74	0.198
Total testosterone (ng/mL)	1.68 ±0.09	1.31 ±0.10	0.007
DHEA-S (μg/mL)	2.20 ±0.12	1.82 ±0.08	0.011
17-hydroxycorticosteroids (ng/mL)	0.93 ±0.06	0.77 ±0.04	0.032
AMH (ng/mL)	0.57 ±0.05	0.98 ±0.08	<0.001
Inhibin B (pg/mL)	4.75 ±0.82	14.14 ±1.55	<0.001
Vitamin D (ng/mL)	11.35 ±0.82	20.18 ±0.74	<0.001

Note: Differences were considered statistically significant at p < 0.05.

Table 2. Indicators of systemic inflammation and immune response in the study groups

Parameter	Group 1: DOR with Hypothyroidism (Mean ±SD), n = 40	Group 2: DOR with Euthyroidism (Mean ±SD), n = 40	*p*-value
C-reactive protein (mg/L)	30.25 ±1.58	13.53 ±0.94	<0.001
Ceruloplasmin (mg/dL)	84.55 ±4.59	51.35 ±2.14	<0.001
Complement C3 (g/L)	6.76 ±0.64	2.57 ±0.30	<0.001
Complement C4 (g/L)	1.87 ±0.19	0.60 ±0.08	<0.001

Note: Differences were considered statistically significant at p < 0.05.

In addition, patients in Group 1 demonstrated significantly higher concentrations of total testosterone (1.68 ± 0.09 vs. 1.31 ± 0.10 ng/mL; $p = 0.007$), dehydroepiandrosterone sulfate (2.20 ± 0.12 vs. 1.82 ± 0.08 μg/mL; $p = 0.011$), and 17-hydroxycorticosteroids (0.93 ± 0.06 vs. 0.77 ± 0.04 ng/mL; $p = 0.032$), indicating relative hyperandrogenism and increased adrenal steroidogenic activity. Markers of ovarian reserve were significantly reduced in patients with hypothyroidism. AMH levels were markedly lower in Group 1 compared with Group 2 (0.57 ± 0.05 vs. 0.98 ± 0.08 ng/mL; $p < 0.001$). A similar pattern was observed for inhibin B, with concentrations more than threefold lower in Group 1 (4.75 ± 0.82 vs. 14.14 ± 1.55 pg/mL; $p < 0.001$), confirming substantial depletion of functional ovarian reserve.

Furthermore, patients in Group 1 exhibited a pronounced deficiency of vitamin D (11.35 ± 0.82 vs. 20.18 ± 0.74 ng/mL; $p < 0.001$), which may further aggravate endocrine, immune, and reproductive dysfunction.

Inflammatory and immunological markers

Intergroup analysis demonstrated significantly higher levels of all assessed inflammatory and immunological markers in Group 1 compared with Group 2 (Table 2).

CRP concentrations were significantly elevated in patients with DOR and hypothyroidism (30.25 ± 1.58 mg/L) compared with euthyroid patients (13.53 ± 0.94 mg/L; $p < 0.001$). Similarly, ceruloplasmin levels were markedly higher in Group 1 (84.55 ± 4.59 vs. 51.35 ± 2.14 mg/dL; $p < 0.001$).

Complement system analysis revealed a more than 2.5-fold increase in complement component C3 levels in Group 1 compared with Group 2 (6.76 ± 0.64 vs. 2.57 ± 0.30 g/L; $p < 0.001$). Complement component C4 demonstrated a comparable pattern, with significantly higher concentrations in Group 1 (1.87 ± 0.19 vs. 0.60 ± 0.08 g/L; $p < 0.001$).

Collectively, these findings indicate a pronounced systemic inflammatory and immune activation profile in women with DOR in the presence of hypothyroidism.

4. Discussion

The main finding of the present study is that women with DOR and hypothyroidism exhibit significantly higher levels of systemic inflammatory markers compared with euthyroid women with DOR. In particular, concentrations of CRP, complement components C3 and C4, and ceruloplasmin were markedly elevated, indicating enhanced inflammatory and immune activation in the presence of thyroid dysfunction.

Systemic inflammation has increasingly been recognized as an important contributor to reproductive disorders and ovarian aging. Elevated CRP levels reflect activation of the acute-phase inflammatory response and have been associated with impaired folliculogenesis, altered granulosa cell function, and reduced oocyte competence [9,14,23]. Previous clinical and experimental studies indicate that inflammatory mediators can disrupt ovarian microenvironment homeostasis and negatively affect follicular development and oocyte maturation [11,22]. Our results are consistent with earlier reports demonstrating increased systemic inflammatory activity in women with thyroid dysfunction and reproductive disorders [5,7,17].

Activation of the complement system represents another important component of innate immunity that may influence reproductive physiology. Complement components C3 and C4 play a key role in inflammatory signaling and immune regulation. Excessive activation of the complement cascade may contribute to local ovarian inflammation, endothelial dysfunction, and disturbances in follicular fluid composition, ultimately impairing oocyte maturation and embryo quality [8,18]. Previous studies have suggested that autoimmune thyroid diseases may promote complement activation and inflammatory tissue injury, potentially affecting ovarian tissue and reproductive capacity [1,21].

In addition to inflammatory activation, oxidative stress appears to represent an important pathophysiological mechanism linking thyroid dysfunction with ovarian impairment. Ceruloplasmin, an acute-phase glycoprotein involved in copper metabolism and antioxidant defense, was significantly elevated in women with DOR and hypothyroidism in our study. Increased ceruloplasmin levels may reflect enhanced oxidative stress and disturbances in metal ion metabolism. Oxidative imbalance is known to accelerate follicular atresia, induce mitochondrial dysfunction, and promote apoptosis of granulosa cells, thereby reducing oocyte quality and reproductive potential [9,10,24].

Thyroid hormones play a central role in metabolic regulation, immune modulation, and tissue homeostasis. Numerous studies have demonstrated that thyroid dysfunction may alter cytokine production, activate inflammatory pathways, and influence immune cell function [4,15,17]. These endocrine–immune interactions are increasingly recognized as a key mechanism linking thyroid disorders with reproductive dysfunction. In particular, hypothyroidism has been associated with altered gonadotropin secretion, impaired ovarian steroidogenesis, and disturbances in follicular development, which may contribute to reduced ovarian reserve [3,6,19].

The findings of the present study support the concept that DOR should not be viewed solely as a consequence of chronological aging but rather as a multifactorial condition influenced by endocrine, metabolic, and immunological factors [20,24]. Identification of systemic inflammatory activation in women with DOR may therefore provide additional insight into disease severity and pathophysiological mechanisms.

Recent meta-analyses further support the relationship between thyroid autoimmunity and ovarian dysfunction. Several studies have demonstrated that autoimmune thyroiditis is associated with reduced AMH levels, diminished ovarian reserve markers, and impaired reproductive outcomes [10,16,25]. Emerging evidence also suggests that chronic inflammation and endocrine disturbances may jointly contribute to accelerated ovarian aging and infertility [12,22].

The novelty of the present study lies in the simultaneous evaluation of multiple inflammatory and oxidative markers —including CRP, complement components C3 and C4, and

ceruloplasmin—in women with diminished ovarian reserve stratified according to thyroid status. To our knowledge, this is among the first studies conducted in a Central Asian population to investigate the interaction between thyroid dysfunction and systemic inflammatory activation in patients with reduced ovarian reserve.

Several limitations should be acknowledged. First, the cross-sectional design of the study limits the ability to establish causal relationships between thyroid dysfunction, systemic inflammation, and ovarian reserve decline. Second, the sample size was relatively modest. Future prospective and longitudinal studies are required to determine whether correction of thyroid dysfunction may reduce systemic inflammatory activation and improve reproductive outcomes in women with DOR.

The novelty of the present study lies in the simultaneous assessment of multiple inflammatory and oxidative stress markers—including CRP, complement C3 and C4, and ceruloplasmin—in women with diminished ovarian reserve stratified according to thyroid status. To our knowledge, this is among the first studies conducted in a Central Asian population investigating the interaction between thyroid dysfunction and systemic inflammatory activation in reproductive endocrinology.

5. Conclusions

Overall, the present findings highlight the potential role of thyroid-associated inflammatory and oxidative pathways in the pathophysiology of diminished ovarian reserve. The results demonstrate that DOR is accompanied by systemic pro-inflammatory and oxidative activation, which becomes significantly more pronounced in the presence of hypothyroidism.

Assessment of inflammatory biomarkers, including C-reactive protein, complement components C3 and C4, and ceruloplasmin, may provide additional insight into disease severity and help identify women at increased risk of reproductive dysfunction. Incorporating these markers into the clinical evaluation of patients with diminished ovarian reserve could improve risk stratification and support more personalized diagnostic and therapeutic strategies aimed at reducing inflammatory burden and optimizing reproductive outcomes.

From a broader clinical and public health perspective, early detection of thyroid dysfunction and associated inflammatory activation in women with reproductive disorders may contribute to improved infertility management and better reproductive health outcomes.

The strengths of this study include the evaluation of several inflammatory and oxidative stress markers and the comparison of patients according to thyroid status. However, certain limitations should be acknowledged, including the cross-sectional design and the relatively small sample size.

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