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MULTIFACTORIAL ANALYSIS OF IMMUNE AND ENDOCRINE MARKERS IN WOMEN WITH POLYCYSTIC OVARY SYNDROME

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ABSTRACT

Background: Polycystic ovary syndrome (PCOS) is a multifactorial disorder associated with endocrine, metabolic, and immune dysregulation.

Objective: To investigate the relationship between immune-inflammatory cytokines, gonadotropins, metabolic parameters, and ovarian dysfunction in women with PCOS.

Methods: A prospective comparative study was conducted in 92 women aged 18–35 years, including PCOS patients with various menstrual disorders and healthy controls. Serum levels of anti-Müllerian hormone (AMH), gonadotropins, glucose, insulin, HOMA-IR, and pro-inflammatory cytokines (IL-6, TNF-α, IL-17A) were assessed and analyzed using ELISA and correlation statistics.

Results: Women with PCOS demonstrated significantly elevated AMH, insulin, HOMA-IR, IL-6, TNF-α, and IL-17A levels, which progressively increased with menstrual dysfunction severity. Strong correlations were observed between AMH and cytokines, as well as between HOMA-IR and inflammatory markers, reflecting intertwined metabolic and immune disturbances.

Conclusion: The integration of immune-inflammatory, hormonal, and metabolic pathways underlies PCOS pathogenesis, and pro-inflammatory cytokines may serve as biomarkers of disease severity and progression.

Keywords: Polycystic ovary syndrome, AMH, cytokines, insulin resistance, inflammation, biomarkers

INTRODUCTION

Polycystic Ovary Syndrome (PCOS) is one of the most common forms of endocrine pathology in women of reproductive age, affecting an estimated 8 to 15% of the population [1, 13]. The main clinical manifestations of PCOS include hyperandrogenism, chronic anovulation, and polycystic ovarian morphology, accompanied by metabolic disorders such as obesity, insulin resistance (IR), and dyslipidemia.

In recent years, researchers have increasingly focused on the role of immune and pro-inflammatory factors in the pathogenesis of PCOS. According to current concepts, chronic low-grade inflammation is considered a key component of the disease [6, 15]. It has been established that women with PCOS have elevated levels of pro-inflammatory cytokines, which play a significant role in disrupting metabolic homeostasis and ovarian function [9, 2].

An additional area of interest is the cytokine IL-17A, which belongs to the Th17 subgroup and promotes the activation of neutrophils and enhances tissue inflammation. Increased levels of IL-17A in women with PCOS may be associated with insulin resistance and metabolic syndrome, as shown in the work of Kuang et al. [7].

A critical regulatory role in follicular growth and maturation is played by anti-Müllerian hormone (AMH), the levels of which are significantly elevated in women with PCOS and correlate with the number of antral follicles [10]. Moreover, recent studies suggest a possible link between AMH concentration and the levels of pro-inflammatory cytokines, which may indicate that AMH functions not only as a biomarker of ovarian reserve but also as an active participant in pathological folliculogenesis [11, 14].

Equally important in the pathogenesis of PCOS are the classical gonadotropins—follicle-stimulating hormone (FSH) and luteinizing hormone (LH). Disruption of the LH/FSH ratio, particularly LH dominance, stimulates androgen-producing theca cells and impairs the normal ovulation process. Elevated LH is also associated with increased synthesis of AMH and disruption of dominant follicle selection [5]. At the same time, a reduction in FSH sensitivity against the backdrop of high AMH production can hinder normal folliculogenesis and provoke its blockade in the early stages [14].

Hyperinsulinemia and elevated fasting glucose, accompanying insulin resistance, play an additional role in maintaining the hyperandrogenic state by enhancing steroidogenesis in the ovaries and suppressing the synthesis of sex hormone-binding globulin (SHBG) in the liver, thus increasing the bioavailability of testosterone. This, in turn, leads to the formation of clinical manifestations of PCOS and the disruption of ovulatory function [8, 4].

Thus, the interaction of the endocrine, immune, and metabolic systems in PCOS represents a complex network, where key pathogenic links include not only inflammatory cytokines and metabolic markers but also fundamental regulatory hormones—LH, FSH, insulin, and AMH. Their comprehensive assessment, depending on the type of menstrual cycle, allows for a deeper understanding of the pathogenesis of PCOS and brings us closer to more precise and personalized diagnosis and therapy of the syndrome.

In this regard, studying the relationships between immune and hormonal parameters in women with different clinical forms of PCOS is of scientific interest. A comprehensive analysis of pro-inflammatory cytokines, gonadotropins, and markers of insulin resistance, considering menstrual status, may shed light on the mechanisms underlying ovarian dysfunction and contribute to the search for new prognostic criteria for the disease.

The aim of the work was to determine the characteristics of anti-Müllerian hormone concentrations, pro-inflammatory cytokines (IL-6, TNF-α, IL-17A), gonadotropins, and carbohydrate metabolism indicators (glucose, insulin, HOMA-IR) in women with PCOS,

depending on the type of menstrual cycle, as well as to identify correlations between immune-hormonal markers in the context of the disease's pathogenesis.

MATERIALS AND RESEARCH METHODS

A prospective comparative clinical and laboratory study was conducted to examine immunological, metabolic, and hormonal markers in women of reproductive age with different forms of polycystic ovary syndrome (PCOS). The study included 92 women aged 18 to 35 years who underwent outpatient or inpatient examinations. All participants provided written informed consent to participate in the study.

The main group consisted of patients with a diagnosed case of PCOS. The diagnosis of PCOS was established based on the Rotterdam Consensus (ESHRE/ASRM, 2003), with the presence of at least two of the three following criteria: chronic anovulation, clinical or biochemical signs of hyperandrogenism, and polycystic ovarian morphology based on ultrasound examination. All women were divided into 4 clinical groups. Group 1 consisted of 24 women with PCOS and a regular menstrual cycle. Group 2 included 20 women with PCOS and clinically confirmed oligomenorrhea, while Group 3 comprised 23 women with PCOS and amenorrhea. The control group included 25 healthy women with regular menstrual cycles, normal hormonal background, and no signs of hyperandrogenism.

Exclusion criteria included: age under 18 or over 35 years, hyperprolactinemia, congenital adrenal hyperplasia, Cushing's syndrome, acute inflammatory and autoimmune diseases, and the use of hormonal medications less than three months prior to the study.

Clinical examination included history taking, assessment of menstrual cycle characteristics, measurement of body weight and height, calculation of body mass index (BMI), and evaluation of hirsutism using the Ferriman-Gallwey scale. Pelvic ultrasound was performed transabdominally or transvaginally (depending on the clinical situation) with mandatory assessment of the number of antral follicles, their diameter, ovarian volume, and capsule thickness.

Hormonal investigations were conducted on blood serum and included the measurement of anti-Müllerian hormone (AMH), luteinizing hormone (LH), follicle-stimulating hormone (FSH), insulin, and fasting glucose. Radioimmunoassay and enzyme-linked immunosorbent assay (ELISA) methods were used, employing standard commercial kits (IMMUNOTECH, Czech Republic; DRG, Germany). The insulin resistance index was calculated using the HOMA-IR formula: (insulin × glucose)/22.5.

Immunological studies were conducted in the Laboratory of Reproductive Immunology of the Institute of Immunology and Human Genomics, Academy of Sciences of the Republic of Uzbekistan. As part of the immune profile, the concentrations of pro-inflammatory cytokines IL-6, TNF-α, and IL-17A were investigated. Measurements were performed in serum using ELISA with certified test kits (Vector-Best LLC, Russia), strictly following the manufacturers' instructions. Blood samples were taken in a fasting state during the early follicular phase of the menstrual cycle (days 3–5).

All obtained data were processed using BioStat LE 7.6.5 software. The results are presented as the mean value and standard error of the mean (M ± m). To assess differences between groups, the student's t-test was used for normal distribution. Correlation analysis was

conducted using Pearson's coefficients. Differences were considered statistically significant at a significance level of $p < 0.05$.

RESULTS AND ITS DISCUSSION

To assess the clinical, laboratory, and immunological characteristics of women with various forms of polycystic ovary syndrome (PCOS), a comprehensive evaluation of the functional state of the hormonal and immune systems was conducted. Below are the comparative data for the main markers studied in the control group and the groups of patients with PCOS, stratified by the type of menstrual cycle (MC).

Table 1 presents the values of key hormonal and metabolic indicators in the women examined. The analysis of the obtained data showed statistically significant differences in several parameters between the groups.

Table 1 Hormonal Status of the Examined Women, M±m

Indicator	Control Group, n=25	PCOS		
		Regular MC МЦ, n=24	Oligomenorrhea, n=20	Amenorrhea, n=23
AMH (ng/mL)	4,32±0,25	18,43±1,04*	29,72±1,02*	42,55±1,74*
FSH (mIU/mL)	6,51±0,32	5,29±0,20*	4,25±0,29*	3,77±0,27*
LH (mIU/mL)	7,17±0,30	10,42±0,29*	12,75±0,48*	13,97±0,56*
Glucose (mmol/L)	4,84±0,24	5,12±0,30^	5,31±0,25^	5,81±0,31*
Insulin (μIU/mL)	6,22±0,18	12,19±0,49*	14,65±0,89*	16,29±0,86*
HOMA-IR	1,84±0,13	3,55±0,21*	4,41±0,31*	5,31±0,23*

Note: * - statistically significant compared to control group ($p<0.05$ - $p<0.001$). ^ - not statistically significant compared to control group. M - median, Q1 (percentile) - 25%, Q3 (percentile) - 75%.

Anti-Müllerian hormone (AMH) is a glycoprotein produced by granulosa cells of pre-antral and small antral follicles in the ovary. It plays a significant role in regulating the early stages of folliculogenesis by suppressing the initial recruitment of follicles and decreasing their sensitivity to FSH [3]. In women with PCOS, AMH levels are generally elevated, reflecting the accumulation of immature follicles and the disruption of the dominant follicle selection mechanism, leading to anovulation [10]. According to the analysis, the average AMH level in the control group was 4.32±0.25 ng/mL. In women with PCOS, there was a significant increase in AMH levels, which correlated with the severity of menstrual cycle disorders: with a regular menstrual cycle – 18.43±1.04 ng/mL ($p<0.001$), with oligomenorrhea – 29.72±1.02 ng/mL ($p<0.001$), and with amenorrhea – 42.55±1.74 ng/mL ($p<0.001$). These data confirm the hyperproduction of AMH in women with PCOS and its progressive increase in more severe forms of ovarian cycle dysfunction (Table 1).

Follicle-stimulating hormone (FSH) and luteinizing hormone (LH) are key gonadotropins that regulate ovarian function. FSH stimulates the growth and maturation of follicles, the synthesis of estrogen, and the expression of LH receptors. LH ensures ovulation, promotes the production of androgens in the theca cells, and supports the formation of the corpus luteum.

Imbalances in these hormones, particularly elevated LH in the context of reduced FSH, are characteristic of PCOS and contribute to anovulation and hyperandrogenism [5]. Analysis of gonadotropin levels also revealed characteristic changes. The FSH level in women with PCOS was significantly reduced compared to the control group (6.51±0.32 mIU/mL): in the regular cycle group – 5.29±0.20 mIU/mL ($p<0.05$), in the oligomenorrhea group – 4.25±0.29 mIU/mL ($p<0.01$), and in the amenorrhea group – 3.77±0.27 mIU/mL ($p<0.001$). Meanwhile, LH levels progressively increased: from 7.17±0.30 mIU/mL in the control group to 10.42±0.29, 12.75±0.48, and 13.97±0.56 mIU/mL in the PCOS groups ($p<0.001$ in all PCOS groups) (Table 1). An elevated LH/FSH ratio is a typical marker of PCOS and reflects a disruption in hypothalamic-pituitary regulation.

Glucose is the main energy substrate of the body, and its blood levels are regulated by insulin [8]. Fasting glucose levels were moderately elevated in women with PCOS compared to the control group (4.84±0.24 mmol/L), but statistical significance was only reached in the amenorrhea group (5.81±0.31 mmol/L, $p<0.05$). In the regular cycle and oligomenorrhea groups, the increase in glucose levels was not statistically significant ($p>0.05$) (Table 1).

Insulin and insulin resistance index (HOMA-IR) showed a significant tendency to increase as the clinical picture worsened. Insulin is a hormone produced by the pancreas that regulates glucose levels and participates in fat metabolism [8]. According to the data analysis, fasting insulin in women with PCOS was significantly higher than in the control group (6.22±0.18 μU/mL): 12.19±0.49 μU/mL in the regular cycle group, 14.65±0.89 μU/mL in the oligomenorrhea group, and 16.29±0.86 μU/mL in the amenorrhea group (in all cases $p<0.001$) (Table 1).

The HOMA-IR index (Homeostasis Model Assessment of Insulin Resistance) is a calculated measure of insulin resistance and is derived from fasting glucose and insulin levels. Literature indicates that increased HOMA-IR in women with PCOS correlates with the severity of hyperinsulinemia and can be used as an integral marker of metabolic imbalance, alongside cytokines [8]. In this study, the HOMA-IR index in the women studied progressively increased from 1.84±0.13 in the control group to 3.55±0.21, 4.41±0.31, and 5.31±0.23 in the PCOS groups, respectively ($p<0.001$), indicating significant insulin resistance associated with the progression of the pathology (Table 1).

Table 2 presents the values of pro-inflammatory cytokines (IL-6, TNF-α, IL-17A) in the serum of women from the control group and women with PCOS, stratified by menstrual cycle characteristics. All three studied cytokines showed significantly increased concentrations in women with PCOS compared to controls, with the increase being particularly marked in those with more severe menstrual dysfunction.

Table 2. Cytokine status of the examined women, M±m

Marker (pg/ml)	Control group (n=25)	PCOS		
		Regular M.C. (n=24)	Oligomenorrhea (n=20)	Amenorrhea (n=23)
IL-6	5,90±0,26	35,97±2,29*	41,74±2,15*	51,93±1,99*
TNF-α	17,94±0,63	46,17±1,65*	55,95±2,86*	61,65±2,44*
IL-17A	12,55±0,58	45,43±1,45*	49,23±2,63*	53,22±1,98*

Note: * - Statistically significant compared to the control group (p<0.05 - p<0.001). Me – median, Q1 (percentile) – 25%, Q3 (percentile) – 75%.

Interleukin-6 (IL-6) is a multifunctional pro-inflammatory cytokine produced by macrophages, adipocytes, endothelial cells, and granulosa cells. It plays a crucial role in the systemic inflammatory response, metabolism regulation, and is involved in insulin resistance and ovarian dysfunction mechanisms. IL-6 promotes the synthesis of acute-phase proteins, inhibits lipoprotein lipase activity, and affects gonadotropin secretion. The analysis revealed significantly elevated IL-6 levels in women with PCOS compared to the control group. For women with regular menstrual cycles, the concentration was 35.97±2.29 pg/ml, for oligomenorrhea it was 41.74±2.15 pg/ml, and for amenorrhea it was 51.93±1.99 pg/ml (p<0.001), reflecting a pronounced subclinical inflammation that worsens as menstrual disturbances increase.

Tumor Necrosis Factor Alpha (TNF-α) is a key mediator of the inflammatory response with pro-apoptotic and immunomodulatory actions. It decreases tissue sensitivity to insulin and disrupts the signaling pathway via the insulin receptor while inhibiting aromatase activity and steroidogenesis in the ovaries. The levels of TNF-α were significantly elevated in women with PCOS compared to the control group (17.94±0.63 pg/ml), with levels of 46.17±1.65 pg/ml in regular menstrual cycles, 55.95±2.86 pg/ml in oligomenorrhea, and 61.65±2.44 pg/ml in amenorrhea (p<0.001). The elevated TNF-α levels likely contribute to insulin resistance and impaired ovarian steroid function.

Interleukin-17A (IL-17A) is a pro-inflammatory cytokine mainly synthesized by Th17 lymphocytes. It enhances the expression of pro-inflammatory mediators, chemokines, and adhesion molecules involved in chronic inflammation. In PCOS patients, IL-17A levels were also significantly higher compared to controls (12.55±0.58 pg/ml), with values of 45.43±1.45 pg/ml in regular cycles, 49.23±2.63 pg/ml in oligomenorrhea, and 53.22±1.98 pg/ml in amenorrhea (p<0.001). These data suggest activation of the Th17 immune response and its involvement in sustaining chronic inflammation and metabolic disturbances in PCOS.
In the next step, a correlation analysis was conducted to better understand the interactions between these key pathogenic factors, which will be presented in **Table 3**.

Table 3. Correlation between Studied Parameters

	AMH	FSH	LH	Glucose	Insulin	HOMA-IR	IL-6	TNF-α	IL-17A
AMH	1.0								
FSH	-0.8	1.0							
LH	0.84	-0.73	1.0						
Glucose	0.49	-0.41	0.43	1.0					
Insulin	0.84	-0.72	0.8	0.38	1.0				
HOMA-IR	0.87	-0.76	0.83	0.45	0.82	1.0			
IL-6	0.9	-0.75	0.85	0.4	0.85	0.88	1.0		
TNF-α	0.89	-0.76	0.83	0.43	0.85	0.87	0.89	1.0	
IL-17A	0.81	-0.71	0.79	0.36	0.87	0.82	0.88	0.85	1.0

According to the analysis of the relationships presented in Table 3, it was found that AMH positively correlates with LH (r = 0.84), insulin (r = 0.84), HOMA-IR (r = 0.87), as well as with cytokines IL-6 (r = 0.90), TNF-α (r = 0.89), and IL-17A (r = 0.81). This confirms the connection between folliculogenesis disturbances and the activation of inflammatory and metabolic cascades. The negative correlation between AMH and FSH (r = -0.80) reflects the typical hormonal disorder in PCOS.

HOMA-IR values showed a strong positive correlation with levels of IL-6 (r = 0.88), TNF-α (r = 0.87), and IL-17A (r = 0.82), which supports the close relationship between systemic inflammation and the degree of insulin resistance. Furthermore, HOMA-IR was also positively correlated with LH (r = 0.83) and AMH (r = 0.87) (Table 3), highlighting the role of hyperinsulinemia in the pathogenesis of hyperandrogenism and impaired folliculogenesis.

Among the cytokines, the strongest correlation was observed between IL-6 and TNF-α (r = 0.89), as well as between IL-6 and IL-17A (r = 0.88) (Table 3), which may reflect the co-activation of several inflammatory pathways in PCOS. All three cytokines showed close correlations with insulin, LH, and AMH levels, reinforcing the triadic role of immune, hormonal, and metabolic factors in the development of the condition.

Thus, the identified correlations indicate the presence of strong pathogenetic links between pro-inflammatory cytokines, hormonal disorders, and insulin resistance in women with PCOS. The obtained data underline the importance of comprehensive evaluation of immune, metabolic, and hormonal markers for a deeper understanding of the pathogenesis and individualization of diagnostic and therapeutic approaches to this syndrome.

The results of the present study confirm contemporary views on the multifactorial nature of PCOS pathogenesis, where key elements include hormonal disturbances, insulin resistance, and chronic inflammation. The obtained data demonstrated a significant increase in anti-Müllerian hormone (AMH) levels in women with PCOS, particularly pronounced in cases of menstrual rhythm disturbances, which aligns with the work of Pigny et al. [10] and Dewailly et al. [3], who identified AMH as a reflection of the number of antral follicles and the degree of ovarian cycle dysfunction.

The characteristic hyperandrogenism and disrupted LH/FSH ratio observed in PCOS were also confirmed in this study, further supporting the role of hypothalamic-pituitary dysregulation in the pathogenesis of the disease. The increase in LH, combined with elevated AMH levels, may suppress the development of the dominant follicle, maintaining a state of chronic anovulation [5].

Elevated insulin levels and the calculated HOMA-IR index in women with PCOS confirm the presence of insulin resistance even in the absence of glucose abnormalities, consistent with previously described mechanisms of compensatory hyperinsulinemia [8]. The correlations between HOMA-IR and the levels of AMH, LH, as well as IL-6, TNF-α, and IL-17A, indicate the integration of metabolic and inflammatory pathways in PCOS pathogenesis.

The cytokines IL-6 and TNF-α have previously been considered potential mediators of insulin resistance [12], while the role of IL-17A is discussed in the context of Th17-mediated inflammation, which can impair insulin signaling and contribute to hyperandrogenism [7]. The significant correlations found in this study between IL-17A and HOMA-IR, AMH, and LH further confirm its involvement in the pathogenesis of PCOS.

The high degree of interrelationship between all three studied cytokines and key metabolic and reproductive function indicators supports the hypothesis of a unified pathophysiological circuit, where inflammation, hyperinsulinemia, and ovarian cycle dysfunction mutually reinforce each other. This approach is supported by several recent meta-analyses and reviews [6, 15], which emphasize the need for a multidisciplinary approach to the diagnosis and treatment of PCOS.

Thus, the results of this study highlight the importance of a comprehensive assessment of the immune, metabolic, and hormonal profiles in women with PCOS.

CONCLUSIONS

1. The levels of AMH, IL-6, TNF-α, IL-17A, insulin, and HOMA-IR significantly increase in women with PCOS as the severity of menstrual cycle disturbances increases.
2. Positive correlations were found between AMH and IL-6 (r=0.90), TNF-α (r=0.89), HOMA-IR (r=0.87), as well as a negative correlation with FSH (r=–0.80).
3. The pro-inflammatory cytokines IL-6, TNF-α, and IL-17A may be considered and used as potential biomarkers of PCOS severity.
4. A comprehensive assessment of immune and metabolic status holds diagnostic and prognostic value in a personalized approach to PCOS.

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