Open Access *Journal of the Faculty of Medicine Baghdad (J Fac Med Baghdad)* Vol.xx. No.x 2026
Print ISSN: 0041-9419, Online ISSN: 2410-8057 Research Article

Evaluation of Serum Interleukin-19 level in Women with Polyendocrine Metabolic Ovarian Syndrome

Tharwa H. Hasan Al-Tai[1]*, Jalal A.Al-Saedi[2], Hayfaa S. AL-Hadithi[3], Tariq G. Nadeem[4]

[1]*Clinical Communicable Diseases Research Unit,College of Medicine,University of Baghdad, Baghdad, Iraq.*
[2]*Medical Skills Laboratory, College of Medicine, University of Baghdad, Baghdad, Iraq.*
[3]*Department of Microbiology, College of Medicine, University of Baghdad, Baghdad, Iraq*
College of Medicine , Ashur University, Baghdad, Iraq
[4]*Centre of Excellence in Molecular Biology, University of the Punjab, Lahore, Pakistan.*

***Corresponding Author:** tharwaaltai@comed.uobaghdad.edu.iq*

Abstract

Background: Polyendocrine Metabolic Ovarian Syndrome (PMOS) typically affects women aged 15 to 50. According to recent studies, immune cells and hormonal imbalances have a role in the pathogenesis of PMOS. The causes and effects of chronic inflammation, particularly in women with PMOS, have been the focus of research.

Objectives: To better understand the immunological profile of PMOS patients through investigating the role of IL-19 blood levels as a biomarker or inflammatory mediator and examining its possible involvement in the pathophysiology of the syndrome.

Cases and Methods: This is a case-control study conducted between December 2024 and March 2025. Sixty women with PMOS were diagnosed at Kamal Al-Samarrai Specialized Hospital in Baghdad, Iraq. 30 healthy women attending private laboratories were used as a control group, matched for age (18-36) to the cases. The blood samples were centrifuged and allowed to clot at room temperature. The serum was then obtained using the enzyme-linked immunosorbent test (ELISA) for use in the human IL-19 detection method.

Results: The mean age was 26.0±4.90 years for the patients and 25.3±4.45 years for the controls. The mean Body Mass Index (BMI) of the control group was 21.9±2.01 kg/m² compared to 28.9±3.62 kg/m² for the patients in PMOS women, the mean serum IL-19 was 113.2±61.56 pg/ml, significantly higher than that of the control group (6.2±4.28 pg/ml).

Conclusion: The inflammatory environment of PMOS seems to be significantly influenced by interleukin-19. The higher blood IL-19 levels in the PMOS cases suggest that it may have a role in the pathophysiology of the syndrome, potentially connected to follicular dysfunction and insulin resistance. IL-19 may be a potential biomarker for tracking the persistent low-grade inflammation linked to PMOS, providing fresh perspectives on tailored diagnostic and treatment approaches.

Keywords: BMI; FSH; Luteinizing hormone (LH); IL-19; Polyendocrine Metabolic Ovarian Syndrome (PMOS).

Received: 17 April. 2026, Revised:23 May 2026, Accepted:08 June 2026, Published Online: June 2026

***Citation:** Al-Tai THH, A. Al-Saedi J, S. AL-Hadithi H, Nadeem TG. Evaluation of serum Interleukin-19 level in women with Polyendocrine Metabolic Ovarian Syndrome. J Fac Med Baghdad. https://doi.org/10.32007/jfacmedbaghdad3321*

Introduction:

Polyendocrine Metabolic Ovarian Syndrome (PMOS) typically affects women aged 15 to 50 (1,2). According to recent studies, immune cells and hormonal imbalances have a role in the pathogenesis of PMOS. The causes and effects of chronic inflammation, particularly in women with PMOS, have been the focus of research. Understanding the elements that contribute to the pathophysiology of PMOS, which impact the hormonal and other metabolic systems, is essential for managing and treating PMOS patients. The connection between the immune system and PMOS has recently been discovered, and research in this area is continuously growing (3). Ovarian impairment in women with PMOS has been linked to chronic inflammation. By releasing different inflammatory cytokines (IL-6, IL-10, IL-18, MIF, and TNF-a), the macrophages contribute to this inflammation (4). There is scientific support for investigating IL-19 in PMOS since it is an important immunomodulator in the IL-10 family. IL-19 becomes a significant mediator that may impact the ovarian and systemic inflammatory milieu, serving as a novel diagnostic biomarker because PMOS is a chronic inflammatory disorder linked to follicular arrest and insulin resistance (5).

As a low-grade chronic inflammatory condition, PMOS is becoming more widely acknowledged. However, the creation of accurate diagnostic biomarkers is hampered by the lack of understanding of the precise molecular processes connecting immune mediators to ovarian dysfunction, which complicates the development of effective treatment strategies for PMOS (6). Despite extensive research on pro-inflammatory cytokines in PMOS, the function of interleukin-19 (IL-19) remains largely unexplored. Clinical information about its serum levels and its role in the pathophysiology of PMOS is lacking, especially in the local clinical setting. By examining IL-19 as a possible novel biomarker, the analyzed data the current study work aims to close this gap. There is a noticeable scarcity of data regarding the role of IL-19. According to recent reviews, the anti-inflammatory family of IL-10, to which IL-19 belongs, remains under-investigated in PMOS pathogenesis (7).

The main causes of PMOS are hyperandrogenism and ovulatory failure, while the precise reasons for this condition are unknown. Lifestyle variables, including food and exercise habits, as well as geographic location, influence the incidence of this ailment. Hirsutism, acne, irregular menstrual periods, anovulation, endometrial cancer, ovarian enlargement, infertility, type 2 diabetes, and cardiovascular diseases are all consequences of PMOS, which is brought on by hyperandrogenism and insulin resistance (8). PMOS arises in the ovaries as a result of both environmental and genetic factors. The way of life also contributes to these illnesses. Women with PMOS need to use oral contraceptives as the follicle stimulating hormone condition causes an imbalance in the sex hormones progesterone and estrogen, which can interfere with a woman's menstrual cycle (9).

Ovarian ultrasonography can be used to diagnose PMOS. Conversely, slim PMOS individuals have much greater levels of luteinizing hormone (LH) and LH to follicle-stimulating hormone (FSH) ratios. This suggests that the most significant mechanism in lean PMOS individuals may be neuroendocrine disorders. Although the exact cause of PMOS is unknown, a complex interplay of genetic and environmental variables is thought to be the cause. (10,11). Women who are infertile have elevated levels of anti-FSH antibodies. As LH and FSH have identical structures, functions, and secretion sites, the same antibodies are effective against both hormones (12). The usual ratio of 1:1 suggests that blood levels of FSH and LH can be compared, but in women with PMOS, the ratio rises to 2 or 3 (13). Pretreatment with oral contraceptives is done to balance the LH/FSH ratio prior to ovulation in order to control the induction of ovaries (14). The clinical definition of insulin resistance (IR) is the inability of a known amount of exogenous or endogenous insulin to increase glucose uptake and utilization in an individual as much as it does in a normal population (15). As peripheral tissues are less sensitive to the effects of insulin in an insulin-resistant state, larger amounts of the hormone are needed to maintain normoglycemia, which leads to the development of compensatory hyperinsulinemia (16).

Interleukin-19 (IL-19), a cytokine that belongs to the IL-10 family, is primarily generated by macrophages and epithelial cells, and has a role in regulating immune responses in inflammatory settings. It has been seen to be involved in regulating the expression of molecules associated with inflammation and fibrosis, such as IL-6, TNF, and TGF, and it communicates through the IL-20R1/IL-20R2 complex (17). The IL-19 genetic locus, which shares a gene cluster with IL-10, is located on human chromosome 1q32 (18). Monocytes and epithelial cells release IL-19 in response to proinflammatory stimuli. IL-19 then intensifies the proinflammatory aspect, by establishing a positive feedback loop, motivating these cells to further intensify their reaction. They will continually produce the cytokine as soon as they are activated during the inflammatory process (18,19). Human monocytes were initially reported to express IL-19, and human B and T cells also exhibit basal levels of IL-19. Lipopolysaccharides (LPS) and granulocyte colony-stimulating factor stimulation can upregulate IL-19 in monocytes, B cells, and T cells. IL-4 and IL-13 can only produce IL-19 in these cells after they have been pretreated with LPS. Although basal levels of IL-19 are not found in nonimmune cells, TNFα and other inflammatory stimuli can cause them (19). IL-19 interacts with both immune (macrophages, T cells, and B cells) and non-immune (endothelial cells, brain-resident glial cells, etc.) cells; however,

it is preferentially expressed in monocytes, macrophages, and T and B lymphocytes (20).

The aim of the study was to better understand the immunological profile of Polyendocrine Metabolic Ovarian Syndrome (PMOS) patients through investigating the role of IL-19 blood levels as a biomarker or inflammatory mediator and examining its possible involvement in the pathophysiology of PMOS.

Cases and Methods: This is a case-control study conducted between December 2024 and March 2025. Sixty women with Polyendocrine Metabolic Ovarian Syndrome (PMOS) were diagnosed at Kamal Al-Samarrai Specialized Hospital in Baghdad, Iraq. This hospital provides (In vitro fertilization (IVF) and infertility treatment services. Thirty healthy women attending private laboratories were used as a control group, matched for age (18-36) with the cases. Every participant underwent a vein puncture to obtain 5 ml blood samples, during the early follicular phase (days 2–5) of the menstrual cycle. The blood samples were centrifuged and allowed to clot at room temperature. The serum was then obtained using the enzyme-linked immunosorbent test (ELISA) in order to be used in the human IL-19 detection method. The human interleukin-19 ELISA kit (ELK Biotechnology CO., LTD) is a high-sensitivity sandwich (catalog number: ELK3187). Additionally, evaluated were luteinizing hormone (LH), follicle-stimulating hormone (FSH), and HOMA-IR measurement of insulin resistance (IR) for each group.

Patient selection: Two of the three criteria for PMOS increased androgen, oligo- or anovulation, and radiological evidence of Polyendocrine Metabolic Ovarian Syndrome were used to diagnose them as patients. Individuals with androgen-secreting tumors, diabetes mellitus, hyperprolactinemia, steroids, or psychiatric medications were not included.

Ethical agreement: Every participant (patient group and control group) provided written informed consent. Additionally, the study was approved by the Research Ethics Committee at Kamal Al-Samarrai Specialized Hospital and the University of Baghdad's Clinical Communicable Diseases Research Unit, College of Medicine Laboratories.

Statistical Analysis: The Statistical Package for Social Sciences (SPSS version 20.0 for Windows, SPSS, Chicago, IL, USA), was used to conduct the statistical analyses. The mean and standard deviation were used to display the data. The means of the two groups were compared using the Student's t-test. The three mean differences of the three groups were examined using a one-way ANOVA. A *p-value* of less than 0.5 was deemed significant.

Results:

The characteristics of the two study groups are summarized in **Table 1**. The mean age was 26.0±4.90 years for the patients and 25.3±4.45 years for the controls, ($P > 0.05$). The mean Body Mass Index (BMI) of the control group was 21.9±2.01 kg/m² compared to 28.9±3.62 kg/m² for the patients, ($P < 0.05$).

Compared to the controls, the PMOS women had higher serum levels of FSH and LH, which were both statistically significant, ($P < 0.05$). The mean IL-19 of the level control group was 6.2±4.28 pg/mL, compared to 113.2±61.56 pg/mL for the PMOS group, ($P < 0.05$), **(Table 2)**.

Table 1: Demographic characteristics of the study groups

Demographic variables	Group		P -value
	Patient	Control	
	Mean ± SD	Mean ± SD	
Age (year)	26.0 ± 4.90	25.3 ± 4.45	0.532
BMI (kg/m^2)	28.9 ± 3.62	21.9 ± 2.01	0.005*

Student's t-test; *: Significant

Table 2: Mean ± SD of serum biochemical variables and inflammatory markers in the study groups

Hormonal and immunological variables	Group		P value
	Patient	Control	
	Mean ± SD	Mean ± SD	
FSH (mIU/mL)	175.3 ±102.55	1.7 ± 2.29	0.005*
LH (mIU/mL)	407.8 ±185.29	0.4 ± 0.30	0.005*
IL-19 (pg/mL)	113.2 ± 61.56	6.2 ± 4.28	0.005*

Student's t-test; *: Significant

The mean age of the three groups differ significantly ($P < 0.010$), with the mean age of PMOS with IR is 27.4±4.42, that of PMOS without IR is 23.8±4.85, and that of the control group is 25.3±4.45, **Table 3**. The difference is most remarkable between PMOS without IR and PMOS with IR, but not between then and the control group. The BMIs of the three groups differed significantly ($P < 0.005$).

Table 3: Mean ± SD of demographic variables for the study groups

Demographic Variables	PMOS with IR	PMOS without IR	Control	P- value
	Mean ± SD	Mean ± SD	Mean ± SD	
Age (year)	27.4 ± 4.42	23.8 ± 4.85	25.3 ± 4.45	0.010*
BMI (kg/m^2)	29.8 ± 4.28	27.6 ±1.62	21.9 ± 2.01	0.005*

One-Way ANOVA test; *: Significant

There was a substantial difference in mean FSH values across the three groups (P=0.005), **Table 4**. PMOS without IR did not differ from PMOS with IR, but both groups differed from the controls. The

LH of the three groups differed significantly (*P*=0.005); PMOS without IR did not differ from the PMOS with IR, but both groups differed from the controls. There was a significant difference between the three groups as far as IL-19 (p = 0.005) is concerned. PMOS without IR did not differ from PMOS with IR, but both differed from the controls.

Table 4: Mean ± SD of hormonal and immunological variables in the study groups

Hormonal and immunological variables	PMOS without IR			P value
	PMOS with IR	PMOS without IR	Control	
	Mean ± SD	Mean ± SD	Mean ± SD	
FSH (mIU/mL)	174.3 ± 99.02	176.7 ±109.80	1.7 ± 2.29	0.005*
LH (mIU/mL)	417.2 ±188.81	393.8 ±182.96	0.4 ± 0.30	0.005*
IL-19 (pg/mL)	107.2 ± 53.03	122.1 ± 72.81	6.2 ± 4.28	0.005*

One-Way ANOVA test; *= Significant

Discussion:

PMOS is one of the most prevalent endocrine conditions (21). In this study, the women with (PMOS) are older an average than the controls. corroborate with those of Rudnicka *et al.* (22). The current study is in agreement with the studies by Papalou *et al.* (23). The statistically higher mean BMI among the PMOS cases than the controls in the current study is consistent with the findings of Liu *et al.* (24). The statistically markedly higher levels of FSH (mIU/mL) and LH (mIU/mL) in the PMOS group than the healthy controls is in agreement with the findings of Liu *et al.*, Esteves *et al.* (24,25). In this study, PMOS patients had considerably higher levels of serum IL-19 than the controls. The ratio of estrogen to progesterone affects the immune system. As PMOS patients have low progesterone levels due to oligo-ovulation or anovulation, their immune systems may be overstimulated by high estrogen, which could result in the development of autoantibodies (21,26).

The clinical definition of insulin resistance (IR) centers on the subnormal biological response of peripheral tissues to a known quantity of either exogenous or endogenous insulin, failing to achieve the expected glucose uptake and utilization observed in a healthy population. To counteract this reduced peripheral sensitivity and maintain normoglycemia, the pancreas must over secrete insulin, leading to compensatory hyperinsulinemia (27). In the present study, a key demographic finding was that the average age of patients with PMOS with IR was higher than both PMOS patients without IR and the healthy control group. This age-related divergence warrants careful consideration. While advanced age within the adolescent cohort appears to correlate with manifest IR in study sample, literature confirms that beta-pancreatic cells in teenage girls with PMOS possess a robust compensatory capacity, responding to early-stage IR by elevating insulin secretion. Consequently, these young patients are exposed to chronically elevated levels of circulating insulin. Corroborate with those of Houston and Templeman (28). The current study is in agreement with the study by Meczekalski *et al.* and Ahmed *et al.* found an increase in insulin levels due to its profound effects on ovarian function. In addition to its traditional metabolic pathways, insulin exerts strong stimulatory effects on cell division, regulates growth processes, and contributes significantly to both normal ovarian function and the development of PMOS (29,30). In this study elevated blood insulin levels, observed in our group, may directly accelerate ovarian dysfunction, affecting steroidogenesis and follicular growth. Within the ovaries, insulin functions synergistically as a co-gonadotropin agreement with the findings of Tehrani and Amiri (31). The current study the diagnosing and quantifying insulin resistance in this age group (18-36) presents a significant clinical challenge. Puberty itself is inherently associated with a transient physiological state of insulin resistance, making it difficult to determine the true prevalence of insulin resistance among adolescent girls with PMOS. Despite this diagnostic overlap, our findings suggest that IR and hyperinsulinemia manifest during the nascent stages of the syndrome; thus, IR is undeniably a feature in adolescents with PMOS, rather than a late-stage sequela. corroborate with those of Carreau and Baillargeon (32).

The significantly higher BMI of PMOS patients with IR than that of PMOS patients without IR and the control group in this study is consistent with earlier research Ahmed *et al.*, Gholinezhad *et al.* and Al-Jefout *et al.* (33,34,35). The considerably lower FSH in PMOS patients with IR than in PMOS patients without IR and the control group in this study is consistent with prior research Urbano *et al.*. (36). IL-19 was statistically markedly greater in PMOS patients without IR in this study compared to PMOS patients with IR and the control group. The fact that IL-19 has been shown to have a strong protective effect in a number of chronic inflammatory disorders may be the cause of the rise in IL-19 concentration (37).

The current study is in agreement with the studies by Azuma and Nishiyama The interplay between IR and hyperinsulinemia in PMOS is well-documented to induce a state of low-grade systemic inflammation. The significant increase of IL-19 exclusively in the PMOS-IR subgroup indicates that this cytokine may either be upregulated in response to compensatory hyperinsulinemia or directly contribute to the exacerbation of peripheral insulin insensitivity. This finding opens a new avenue of discussion regarding the role of monocyte-derived cytokines in modulating ovarian tissue inflammation

and metabolic pathways, suggesting that IL-19 could serve as a valuable biomarker for metabolic phenotyping in young patients with (38).

Limitations:

A primary limitation of this study is the relatively small and specific sample size. While this sample was intentionally selected to facilitate an in-depth and rigorous analysis of the relationship between Interleukin-19 and PMOS, it may restrict the immediate generalizability of the findings to broader population. The study was conducted within a tightly defined therapeutic setting. Consequently, the dataset lacks the clinical and demographic diversity required to capture the full spectrum of Polyendocrine Metabolic Ovarian Syndrome (PMOS) variations across different populations. As an exploratory insight into the role of IL-19, these findings serve as a foundational step. However, translating these insights into widespread, scalable therapeutic applications remains limited until validated by future research utilizing larger, multi-center datasets.

Conclusion:

The inflammatory environment of Polyendocrine Metabolic Ovarian Syndrome seems to be significantly influenced by interleukin-19. The higher blood IL-19 levels in the PMOS cases suggest that it may have a role in the pathophysiology of the syndrome, potentially connected to follicular dysfunction and insulin resistance. IL-19 may be a potential biomarker for tracking the persistent low-grade inflammation linked to PMOS, providing fresh perspectives on tailored diagnostic and treatment approaches.

Authors' declaration:

We confirm that all the Figures and Tables in the manuscript belong to the current study. Besides, the figures and images, which do not belong to the current study, have been given permission for re-publication attached to the manuscript. The approval of ethical considerations is signed by the authors-Ethical Approval: at Kamal Al-Samarrai Specialized Hospital and the University of Baghdad's Clinical Communicable Diseases Research Unit, College of Medicine, Laboratories ethics committee accepted the project under code number **(3) in 20/11/2024).**

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

Funding: No financial support or grant was received for conducting this study.

Data availability: Data supporting the findings of this study are available from the corresponding author upon reasonable request.

Authors' contributions:

Study conception & design: **(**Tharwa H. Hasan Al-Tai**).** Literature search:(Tharwa H. Hasan Al-Tai**).** Data acquisition**: (**Tharwa H. Hasan Al-Tai**).** Data analysis & interpretation: **(**Hayfaa S. AL-Hadithi**).** Manuscript preparation: **(**Tharwa H. Hasan Al-Tai**).** Manuscript editing & review: (Tariq G. Nadeem & Jalal A. Al-Saedi**)**

AI declaration: No artificial intelligence tools were used in the design, analysis, or writing of this manuscript.

References

1. *Zhang J, Zhu Y, Wang J, et al. Global burden and epidemiological prediction of polycystic ovary syndrome from 1990 to 2019: a systematic analysis from the Global Burden of Disease Study 2019. PloS one. 2024;19(7):e0306991. https://doi.org/10.1371/journal.pone.0306991.*

2. *Teede HJ, Khomami MB, Morman R,et al. Polyendocrine metabolic ovarian syndrome, the new name for polycystic ovary syndrome: a multistep global consensus process. The Lancet. 2026;407(10545):2329-2339. https://doi.org/10.1016/S01406736(26)00717-8*

3. *Wang J, Yin T, Liu S. Dysregulation of immune response in PCOS organ system. Front Immunol. 2023;14:1169232. https://doi.org/10.3389/fimmu.2023.1169232.*

4. *Khashchenko E, Vysokikh M, Uvarova E, et al. Activation of systemic inflammation and oxidative stress in adolescent girls with polycystic ovary syndrome in combination with metabolic disorders and excessive body weight. Journal of Clinical Medicine. 2020; 9(5): 1399. https://doi.org/10.3390/jcm9051399.*

5. *Al-Taei OMS and Al-Harmooshee MBH. Genetic polymorphisms and immune cytokine profiles in the cellular pathogenesis of polycystic ovary syndrome among Iraqi women. Cellular and Molecular Biology.2025; 71(9), 56-62. https://doi.org/10.14715/cmb/2025.71.9.7.*

6. *Rudnicka E, Suchta K, Grymowicz M, et al. Chronic Low-Grade Inflammation in Pathogenesis of PCOS. Int J Mol Sci. 2021;22(7):3789. https://doi.org/10.3390/ijms22073789.*

7. *Vasyukova E, Zaikova E, Kalinina O, et al. Inflammatory and Anti-Inflammatory Parameters in PCOS Patients Depending on Body Mass Index: A Case-Control Study. Biomedicines. 2023;11(10):2791. https://doi.org/10.3390/biomedicines11102791.*

8. *Singh S, Pal N, Shubham S, et al.et al.. Polycystic ovary syndrome: etiology, current management, and future therapeutics. Journal of clinical medicine. 2023; 12(4):1454. https://doi.org/10.3390/jcm12041454.*

9. *Akpang N, Kwiatkowski J, Zaborowska L, et al. Autoantibodies targeting the hypothalamic-pituitary-ovarian axis in polycystic ovary syndrome:*

emerging key players in pathogenesis?. International Journal of Molecular Sciences. 2025; 26(9): 4121. https://doi.org/10.3390/ijms26094121.

***10.** Pratama G, Wiweko B, Asmarinah, et al. Mechanism of elevated LH/FSH ratio in lean PCOS revisited: a path analysis. Scientific reports. 2024; 14(1): 8229. https://doi.org/10.1038/s41598-024-58064-0.*

***11.** Zheng L, Yang L, Guo Z, et al. Obesity and its impact on female reproductive health: unraveling the connections. Frontiers in endocrinology.2024; 14,1326546. https://doi.org/10.3389/fendo.2023.1326546.*

***12.** Daghestani MH, Daghestani MH, Warsy A, et al. Adverse effects of selected markers on the metabolic and endocrine profiles of obese women with and without PCOS. Frontiers in endocrinology. 2021;12:665446. https://doi.org/10.3389/fendo.2021.665446.*

***13.** Kyrou I, Karteris E, Robbins T, et al. Polycystic ovary syndrome (PCOS) and COVID-19: an overlooked female patient population at potentially higher risk during the COVID-19 pandemic. BMC Medicine. 2020; 18:220. https://doi.org/10.1186/s12916-020-01697-5.*

***14.** Gao J, Mai Q, Zhong Y, et al. Pretreatment with oral contraceptive pills in women with PCOS scheduled for IVF: a randomized clinical trial. Human Reproduction Open. 2024; 2024(2): hoae019. https://doi.org/10.1093/hropen/hoae019.*

***15.** Calcaterra V, Verduci E, Cena H, et al. Polycystic Ovary Syndrome in Insulin-Resistant Adolescents with Obesity: The Role of Nutrition Therapy and Food Supplements as a Strategy to Protect Fertility. Nutrients. 2021; 13(6):1848. https://doi.org/10.3390/nu13061848.*

***16.** Zeitler FS and Nadeau KJ. Insulin Resistance: Childhood Precursors and Adult Disease e-book. USA, Totowa(NJ): Humana Press;2020. p.356. https://doi.org/10.1007/978-3-030-25057-7.*

***17**. Rayi MM, Monteiro RC, Ramachandrayya S. Role of serum interleukin-19 (IL-19) levels in acne vulgaris: a comprehensive case control study. Archives of Dermatological Research. 2025; 317(1): 759. https://doi.org/10.1007/s00403-025-04263-2.*

***18.** Mohammed HM, Qurtas DS, Meran AD. Serum interleukin-19 levels in acne vulgaris patients of varying clinical severity in Erbil City. Cureus. 2023;15(11). https://doi.org/10.7759/cureus.48939.*

***19.**Azuma YT, Nakajima H, Takeuchi T. IL-19 as a potential therapeutic in autoimmune and inflammatory diseases. Curr Pharm Des. 2011;17(34):3776-80.https://doi.org/10.2174/138161211798357845.*

***20**.Leigh T, Scalia RG, Autieri MV. Resolution of inflammation in immune and nonimmune cells by interleukin-19.Am J Physiol Cell Physiol. 2020 ;319(3):C457-C464.https://doi.org/10.1152/ajpcell.00247.2020*

***21.** Al-Musawy SH, Al-Saimary IE, Flaifil MS. Levels of cytokines profile in polycystic ovary syndrome. Medical Journal of Babylon. 2018 Apr 1;15(2):124-8. https://doi.org/10.4103/mjbl.mjbl_32_18..*

***22.** Rudnicka E, Kunicki M, Suchta K, et al. Inflammatory Markers in Women with Polycystic Ovary Syndrome. Biomed Res Int. 2020 4;2020:4092470. https://doi.org/10.1155/2020/4092470.*

***23.** Papalou O, Livadas S, Karachalios A, et al. White blood cells levels and PCOS: direct and indirect relationship with obesity and insulin resistance, but not with hyperandogenemia. Hormones (Athens). 2015;14(1):91-100. https://doi.org/10.14310/horm.2002.1563.*

***24.** Liu S, Hong L, Lian R, et al. Transcriptomic analysis reveals endometrial dynamics in normoweight and overweight/obese polycystic ovary syndrome women. Frontiers in Genetics.2022;13:874-887. https://doi.org/10.3389/fgene.2022.874487.*

***25.** Esteves SC and Alviggi C. The role of LH in controlled ovarian stimulation. In: Ghumman S, editor. Principles and practice of controlled ovarian stimulation in ART. New Delhi: Springer;2015. p.171-96. https://doi.org/10.1007/978-81-322-1686-5_16.*

***26.** Khan D and Ansar AS. The immune system is a natural target for estrogen action: Opposing effects of estrogen in two prototypical autoimmune diseases. Front Immunol 2016; 6:635. https://doi.org/10.3389/fimmu.2015.00635.*

***27.** Papaetis GS, Sacharidou A, Michaelides IC, et al. Insulin resistance, hyperinsulinemia and atherosclerosis: insights into pathophysiological aspects and future therapeutic prospects. Current cardiology reviews. 2025;21(1):E1573403X314035. https://doi.org/%20%2010.2174/011573403X314035241006185109*

***28.** Houston EJ and Templeman NM. Reappraising the relationship between hyperinsulinemia and insulin resistance in PCOS. Journal of Endocrinology. 2025; 265(2). https://doi.org/10.1530/JOE-24-0269.*

***29.** Meczekalski B, Niwczyk O, Kostrzak A, et al. PCOS in Adolescents ongoing riddles in diagnosis and treatment. Journal of clinical medicine.2023; 12(3): 1221. https://doi.org/10.3390/jcm12031221.*

***30.** Parker J, Briden L, Gersh FL. Recognizing the Role of Insulin Resistance in Polycystic Ovary Syndrome: A Paradigm Shift from a Glucose-Centric Approach to an Insulin-Centric Model. Journal of Clinical Medicine. 2025; 14(12):4021. https://doi.org/10.3390/jcm14124021.*

***31.** Tehrani FR and Amiri M. Polycystic ovary syndrome in adolescents: challenges in diagnosis and treatment. International journal of endocrinology and metabolism. 2019; 17(3): e91554. https://doi.org/10.5812/ijem.91554.*

32. *Carreau AM and Baillargeon JP. PCOS in Adolescence and Type 2 Diabetes. Curr. Diab. Rep. 2015; 15(1): 564. https://doi.org/10.1007/s11892-014-0564-3.*

33. *Ahmed RS, Aljabar FA, Al-Shakir NM. Impact of Insulin Resistance in PCOS (LH/FSH) in a Group of Woman in Baghdad City. Al-Esraa University College Journal for Medical Sciences. 2025; 6(9):20-30. https://doi.org/10.70080/2790-7937.1045.*

34. *Gholinezhad M, Gholsorkhtabaramiri M, Esmaeilzadeh S, et al. Insulin resistance and adverse metabolic profile in overweight/obese and normal-weight young women with polycystic ovary syndrome. Caspian J Intern Med. 2018; 9(3): 260-267. https://doi.org/10.22088/cjim.9.3.260.*

35. *Al-Jefout M, Alnawaiseh N, Al-Qtaitat A. Insulin resistance and obesity among infertile women with different polycystic ovary syndrome phenotypes. Scientific reports. 2017; 7(1):5339. https://doi.org/10.1038/s41598-017-05717-y.*

36. *Urbano F, Chiarito M, Lattanzio C, et al. Sex hormone-binding globulin (SHBG) reduction: the alarm bell for the risk of non-alcoholic fatty liver disease in adolescents with polycystic ovary syndrome. Children. 2022; 9(11):1748. https://doi.org/10.3390/children9111748.*

37. *Esam D, Abdel-Moneim A, Mahmoud B, et al. Role of nuclear factor kappa B, interleukin-19, interleukin-34, and interleukin-37 expression in diabetic nephropathy. Beni-Suef University Journal of Basic and Applied Sciences. 2022; 11(1), 119. https://doi.org/10.1186/s43088-022-00299-9.*

38. *Azuma YT and Nishiyama K. Interleukin-19 enhances cytokine production induced by lipopolysaccharide and inhibits cytokine production induced by polyI:C in BALB/c mice. J Vet Med Sci. 2020;82(7):891-896. https://doi.org/10.1292/jvms.20-013.*

تقييم مستوى إنترلوكين-19 في مصل الدم لدى النساء المصابات بمتلازمة الغدد الصمية الأيضية للمبياض

ثروه هادي حسن الطائي[1], جلال علوان مجبل[2] ,هيفاء سلمان الحديثي[3], طارق غلام نديم[4]
[1]وحده بحوث الامراض الانتقاليه السريريه،كليه الطب،جامعه بغداد،بغداد،العراق.
[2]مختبر المهارات الطبية، كلية الطب، جامعة بغداد، بغداد، العراق.
[3]فرع الاحياء المجهريه الطبيه،كليه الطب،جامعه بغداد،بغداد،العراق
[3]عميدة كلية الطب،جامعة اشور،بغداد،العراق
[4]مركز التميز في البيولوجيا الجزيئية، جامعة البنجاب، لاهور، باكستان.

الخلاصة:

خلفية البحث: تصيب متلازمة الغدد الصمية الأيضية للمبياض (PMOS) عادةً النساء اللواتي تتراوح أعمارهن بين 15 و50 عامًا. ووفقًا لدراسات حديثة، تلعب الخلايا المناعية والاختلالات الهرمونية دورًا في نشأة هذه المتلازمة. وقد ركزت الأبحاث على أسباب وتأثيرات الالتهاب المزمن، لا سيما لدى النساء المصابات بمتلازمة تكيس المبايض. ويُعد فهم العناصر التي تُسهم في الفيزيولوجيا المرضية لهذه المتلازمة، والتي تؤثر على النظام الهرموني والأنظمة الأيضية الأخرى، أمرًا بالغ الأهمية لإدارة وعلاج المريضات المصابات بها.

الأهداف: تحسين فهم الملف المناعي لمرضى متلازمة الغدد الصمية (PMOS) من خلال دراسة دور مستويات إنترلوكين-19 في الدم كعلامة حيوية أو وسيط التهابي وفحص احتمالية دورها في الفيزيولوجيا المرضية لمتلازمة الغدد الصمية .

المنهجية: أُجريت الدراسة بين ديسمبر 2024 ومارس 2025. شُخِّصت ستون امرأة مصابة بمتلازمة المبيض متعدد الغدد الصماء الأيضية في مستشفى كمال السامرائي التخصصي في بغداد، العراق. يُقدّم هذا المستشفى خدمات التلقيح الصناعي وعلاج العقم. استُخدمت ثلاثون امرأة سليمة يترددن على مختبرات خاصة كمجموعة مراقبة، مُطابقة في العمر (18-36 عامًا) مع الحالات. خضعت كل مشاركة لسحب عينة دم بحجم 5 مل عن طريق بزل الوريد، خلال المرحلة الجريبية المبكرة (الأيام 2-5) من الدورة الشهرية. خُضعت عينات الدم للطرد المركزي وتُركت لتتخثر في درجة حرارة الغرفة. ثم جُمع المصل باستخدام اختبار المقايسة المناعية الإنزيمية المرتبطة (ELISA) لاستخدامه في طريقة الكشف عن إنترلوكين-19 البشري. بالإضافة إلى ذلك، تم تقييم الهرمون اللوتيني (LH) والهرمون المنبه للجريب (FSH) وقياس HOMA-IR لمقاومة الأنسولين (IR) لكل مجموعة.

النتائج: بلغ متوسط عمر المرضى 4.90 ±26.0 عامًا، بينما بلغ متوسط عمر المجموعة الضابطة 4.45±25.3 عامًا (P < 0.05). أما متوسط مؤشر كتلة الجسم (BMI) للمجموعة الضابطة فكان 2.01±21.9 كجم/م²، مقارنةً بـ3.62±28.9 كجم/م² للمرضى (P < 0.05). وبلغ متوسط مستوى إنترلوكين-19 في مصل الدم لدى النساء المصابات بمتلازمة الغدد الصمية الأيضية للمبياض (61.56±113.15 بيكوغرام/مل)، وهو أعلى بكثير من مستواه لدى المجموعة السليمة (4.28±6.21 بيكوغرام/مل).

الاستنتاجات: وفقًا لهذه الدراسة، يتأثر الالتهاب المصاحب لمتلازمة الغدد الصمية الأيضية للمبياض (PMOS)بشكل كبير بالإنترلوكين-19 (IL-19). تشير الاختلافات الملحوظة في مستويات IL-19 في الدم بين المرضى ومجموعة المقارنة إلى احتمال وجود دور له في الفيزيولوجيا المرضية للمتلازمة، وربما يرتبط ذلك بخلل وظائف الجريبات ومقاومة الأنسولين. ونتيجة لذلك، يُظهر IL-19 إمكانات واعدة كعلامة حيوية محتملة لتتبع الالتهاب المزمن منخفض الدرجة المرتبط ب بمتلازمة الغدد الصمية الأيضية للمبياض، مما يوفر رؤى جديدة حول أساليب التشخيص والعلاج المُخصصة.

الكلمات المفتاحية: انترلوكين-19 (IL-19)، متلازمة الغدد الصمية الأيضية للمبياض (PMOS)، الهرمون المنبه للجريب (FSH)، الهرمون اللوتيني (LH)، مؤشر كتلة الجسم (BMI).