



Serum Levels of Interleukin-36 α and Interleukin-1 α In Iraqi Women with Polycystic Ovary Syndrome

Fatima Hussein^{1,*}, Esraa Ahmed Abdul Qader², Riyam Ahmed Saber²

¹ Department of Biotechnology, Institute of Genetic Engineering and Biotechnology for Postgraduate Studies, University of Baghdad , Jadriya , Baghdad, Iraq.

² Department of Medical Laboratory Technology, College of Medical Technology ,University of Alfarahidi, Al-Qadisiyah, Baghdad, Iraq.

Article's Information	Abstract
Received: 12.04.2025 Accepted: 09.10.2025 Published: 15.09.2026	This study aimed to estimate the serum levels of interleukin-1 alpha (IL-1 α) and interleukin-36 alpha (IL-36 α) in women with polycystic ovary syndrome (PCOS) and to examine their relationship with age and body mass index (BMI). A total number of collected specimens are 75, which were 45 patients and 30 healthy control women participated in this study from different private clinics in Baghdad/ Iraq, for the period starting December 2023 to February 2024. The age ranges for both patients and controls was 18–40 years, with mean ages of 25.60 ± 6.38 years for patients and 29.44 ± 11.61 years for controls. BMI was determined for all participants, revealing a statistically highly significant difference between the PCOS group (27.53 ± 4.72) and the control group (24.02 ± 2.84). The results showed significantly elevated levels of IL-1 α (567.0 ± 59.0 pg/ml vs. 196.56 ± 21.56 pg/ml) and IL-36 α (511.0 ± 99.4 ng/L vs. 175.82 ± 41.67 ng/L) in the PCOS group compared to the control group. A positive correlation was found between IL-1 α and IL-36 α . IL-36 α was not significantly correlated with age or BMI, while IL-1 α was not significantly correlated with age but showed a significant positive correlation with BMI ($r = 0.356$, $P = 0.013$). This suggests that the higher IL-1 α levels may be partly associated with the higher BMI in the PCOS group and may not be due to PCOS alone. Furthermore, levels of luteinizing hormone (LH), follicle-stimulating hormone (FSH), prolactin (PRL), and testosterone (T) were significantly elevated in women with PCOS.
Keywords: Polycystic ovary syndrome, Interleukin-36 alpha, Interleukin-1 alpha, Body mass index, Follicle-stimulating hormone.	

<http://doi.org/10.22401/ANJS.29.3.06>

*Corresponding author: fatma.hussein1206a@sc.uobaghdad.edu.iq



This work is licensed under a [Creative Commons Attribution 4.0 International License](https://creativecommons.org/licenses/by/4.0/)

1. Introduction

Polycystic ovarian syndrome (PCOS), a metabolic and endocrine condition, is one of the most prevalent reasons for infertility due to anovulation and hyperandrogenism [1]. The prevalence of polycystic ovaries in the general population is estimated to range between 20% and 33%, and they are often detected through ultrasound or other pelvic imaging methods [2]. The primary characteristics of this syndrome include oligo/anovulation and disruptions in androgen hormones, resulting in varying degrees of hirsutism, weight gain, acne, and amenorrhea. Overweight or being obese with a body mass index

(BMI) exceeding 30 kg/m² is a substantial risk factor for women in their 30s who develop PCOS [3], in addition to being a risk factor for heart disease, type 2 diabetes, high blood pressure, and a number of malignancies. Genetics, metabolism, hormones, lifestyle, dietary patterns, and psychological behavior all contribute to causing obesity and PCOS, which are complex disorders [4]. It has been shown that PCOS patients have a persistent low-grade inflammatory condition, which includes abnormal proinflammatory cytokine levels and excessive leukocyte counts [5]. In PCOS, hormonal alterations promote interleukin-1 alpha (IL-1 α) production

through activation of M1 macrophages and neutrophils; this elevated IL-1 α not only contributes to inflammation in the reproductive system but also disrupts follicular development and endometrial repair by inhibiting estradiol secretion and correlating with androgen excess [6,7]. IL-1 α is strongly expressed by a variety of cell types, such as T lymphocytes cells, fibroblasts, hepatocytes, keratinocytes, macrophages, and dendritic cells [8]. Numerous skin cells, including endothelial cells, dermal fibroblasts, and epidermal keratinocytes, as well as immune cells, such as macrophages, monocytes, dendritic cells, T lymphocytes, and B lymphocytes, express interleukin-36 (IL-36) cytokines [9]. IL-36 α may contribute to ovulatory dysfunction and insulin resistance, which are core features of PCOS pathophysiology; moreover, it may be associated with chronic low-grade inflammation, increased ovarian androgen production, and elevated luteinizing hormone (LH) levels, warranting further experimental studies to elucidate its mechanistic role [10]. Recent studies suggest that IL-36 α and IL-1 family cytokines may play different roles in obesity and insulin resistance depending on the tissue involved, their circulating levels, and whether the evidence comes from animal or human studies [11, 12]. The study goal was to estimate the serum level of IL-1 α and IL-36 α in PCOS women and to examine their relationship with age and BMI.

2. Materials and Procedures

2.1. Patients and the collecting of control

The study involved 45 women patients with PCOS, ranging in age from 18 to 40, and 30 healthy individuals as control subjects. The patients were attendants seeking treatment at various private clinics in Baghdad between December 2023 and February 2024. All cases of PCOS were diagnosed by professional gynecologists based on clinical findings, in accordance with standard diagnostic criteria. A diagnosis required the presence of at least two of the following three signs [13]: (i) irregular or absent ovulation, (ii) clinical or biochemical signs of hyper androgenism (e.g., hirsutism, alopecia, or acne), and (iii) polycystic ovarian morphology confirmed by transvaginal ultrasound. These cases were identified in comparison with a control group. Our study was approved by Alfarahidi University, college of Medical Technology, Department of Medical Laboratory Technology, (Ethical No.:203188 on 15/12/2023). On the other hand, ethical consent was obtained from all subjects, and they were provided with comprehensive information about our study, including the purpose, procedure, benefits, and confidentiality of the research.

2.2. Exclusion criteria

Exclusion criteria included the use of hormonal contraception or fertility medications within the three to four months prior to enrollment, as well as any medication known to interfere with follicular development or hormonal levels. Additionally, patients with hyper prolactinemia, thyroid dysfunction, congenital adrenal hyperplasia, or those with oligomenorrhea or amenorrhea due to causes other than PCOS were excluded.

2.3. Collection of specimens

A 5 ml disposable syringe and tubes containing gel were used to draw blood from each woman in the PCOS and healthy control groups. To extract serum, the samples were centrifuged at 5000 rpm for five minutes after being allowed to clot for ten minutes at room temperature (20–25°C). To prevent frequent freezing and thawing, the serum was divided into two Eppendorf tubes following centrifugation. After that, the tubes were stored at –20°C for 2 months until analysis. The samples were subjected to only one freeze–thaw cycle before analysis, and all samples were analysed in a single batch on the same day.

2.4. Laboratory techniques

Using the ELISA technique and following the respective manufacturers' protocols, the levels of IL-36 α and IL-1 α in serum were measured in a microplate reader at a wavelength of 450 nm. Detailed information regarding the two ELISA kits is provided in Appendices 1 and 2. Additionally the electrochemiluminescence immunoassay method (RocheHitachiCobase411) was used during the menstrual cycle's third day to assess the reproductive hormones (LH, FSH, testosterone, and prolactin) associated with PCOS. The same blood samples collected on the third day of the menstrual cycle were used to measure serum IL-36 α and IL-1 α levels by ELISA. The manufacturer's instructions were followed in both situations, and the body mass index (BMI) was calculated using the formula: BMI = body weight (kg) / height squared (m²) [14].

2.5. Statistical analysis

The data in this study were analyzed using IBM SPSS V26 and Microsoft Excel. The results are presented as median and mean $\pm$ SD. The medians and means of the two study groups were compared using the Mann-Whitney U test and the independent t-test, respectively. The linear relationship between two continuous variables was evaluated using the Pearson correlation coefficient. Significant and very significant were the probability values less than 0.05 and 0.01, respectively.

3. Results and Discussion

Polycystic ovarian syndrome (PCOS) is one of the most common endocrine and metabolic conditions, affecting 5–15% of women who are of reproductive age [15]. This study was focused on estimating the serum levels of IL-1 α and IL-36 α in PCOS women and their relation with age and BMI. This study indicates that the age mean $\pm$ SD of PCOS patients was 25.60 ± 6.38 , ranging from 18 to 40 years. Agarwal et al. [16] noted that the majority of women with PCOS (80%) were under the age of 40, whereas only 20% were over 40. Thus, PCOS frequency and severity seem to rise with middle age. Kasim and Saadoon [17] revealed that 64% of PCOS cases were prevalent in individuals aged 18 to 26 years. In addition, Al-Saffar [18] and Al-Shattawi [19] reported that the mean age of the PCOS group and the control group did not vary statistically significantly. While Alfatlawi [20] showed that the age mean $\pm$ SD of PCOS patients was 40.1 ± 3.66 , ranging from 35 to 60 years. The results presented in table 1 show that

there was a highly significant difference in the BMI means $\pm$ SD among studied case groups of PCOS (27.53 ± 4.72) as compared with the control groups (24.02 ± 2.84). Figure 1: The occurrence of overweight was more common among women with PCOS compared to their matched controls. These results agreed with [21], which explained that the BMI of the infertile women was significantly increased compared to the control group. As well as, compared to their corresponding control group, women with PCOS had a significantly greater prevalence of overweight and obesity [22]. According to another research study, the average BMI distribution of females with PCOS and the control group did not significantly vary [23]. Obesity is a common feature in PCOS women; the relation between adiposity with menstrual disturbance and hyperandrogenic status in PCOS is confirmed by data that detect an improvement in these parameters with weight loss [24].

Table 1: BMI (Kg/m²) distribution by the studied group

	Patient	Control	P-value [¥]
BMI	27.53 ± 4.72	24.02 ± 2.84	0.002**
Data presented as Mean $\pm$ SD, ¥ Independent t-test was used to test between groups, ** highly significant ($P \leq 0.01$)			

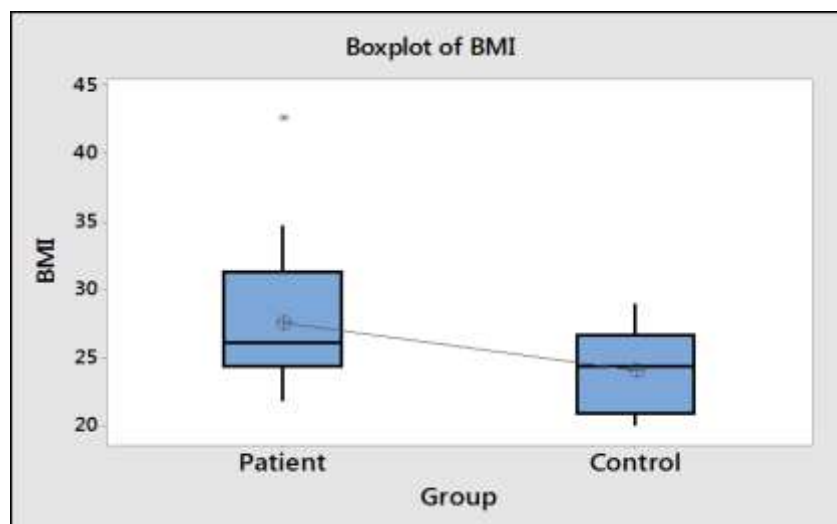


Figure 1: An analogous boxplot showing the patients' and controls' BMI levels

According to Table 2, the results indicated that PCOS patients had considerably higher levels of LH, FSH, testosterone, and prolactin than control. One study reported higher levels of prolactin, LH, and FSH in

women with PCOS compared with the control group [25]. These findings are consistent with previous research indicating that hormonal dysregulation is a hallmark feature of PCOS [26].

Table 2: Reproductive hormone concentrations between studied groups

Parameters	Patient	Control	P-value [¥]
LH (mIU/ml)	10.52±0.45	8.38±0.44	$P \leq 0.05^*$
FSH (mIU/ml)	7.80±0.35	6.11±0.26	$P \leq 0.05^*$
Testosterone (ng/ml)	0.74±0.13	0.06±0.01	$P \leq 0.001^{**}$
Prolactin (ng/ml)	16.22±2.81	12.20±1.35	$P \leq 0.05^*$

Data presented as Mean ± SD, ¥ Independent t-test was used to test between groups, ** highly significant ($P \leq 0.01$), * significant ($P \leq 0.05$)

Since this study evaluated two parameters, IL-36 α and IL-1 α , these findings suggest that they may be associated with the inflammatory profile of PCOS. One antagonist, IL-36 receptor antagonist (IL-36RN), and three agonists, IL-36 α , IL-36 β , and IL-36 γ , are members of the IL-36 cytokine family, which is a subfamily of the IL-1 cytokine family and plays a role in the functional control of immunological and inflammatory responses [27]. In this study, IL-36 α was assessed in serum; Table 3 revealed IL-36 α that highly significantly increased in patients (511.0 ± 99.4 ng/L) than in the control group (175.82 ± 41.67 ng/L), Figure 2. The results of the current study were inconsistent with [10], which found that in comparison to the control group, the PCOS patients'

IL-36 α levels were considerably lower. As well as, inflammatory markers have been studied in women with PCOS, indicating that chronic inflammation may play a role in ovarian dysfunction and reproductive abnormalities [28]. Additionally, elevated levels of IL-36 are not an inherent characteristic in women with PCOS, but they can serve as a valuable clinical marker for monitoring and managing the condition [29]. Thus, most global and current studies improve the importance of IL-36 α in diagnosing many diseases, one being PCOS. The serum level of IL-36 α may have a potential role as an immune-related parameter in PCOS among Iraqi women.

Table 3: Groups test of some immune parameters

	Patient	Control	P-value [¥]
IL-36 α (ng/L)	511.0 ± 99.4	175.82 ± 41.67	0.001**
IL-1 α (pg/mL)	567.0 ± 59.0	196.56 ± 21.56	0.001**

Data presented as Mean ± SD, ¥ Independent t-test was used to test between groups, ** highly significant ($P \leq 0.01$)

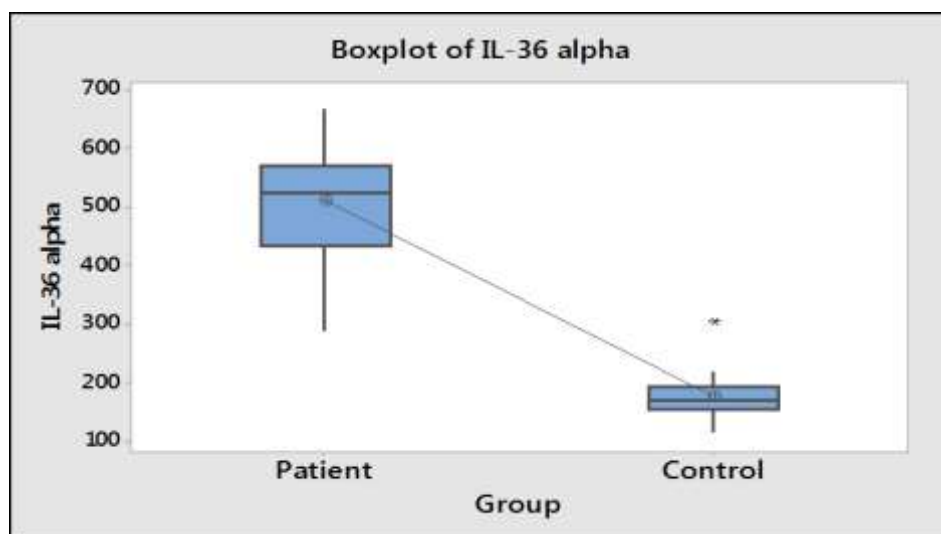


Figure 2: IL-36 α levels in a boxplot that compares patients and control

Another immunological metric assessed in this investigation was IL-1 α ; the results in table 3 indicated that IL-1 α recorded a highly significant difference between the patients with PCOS (567.0 ± 59.0 pg/mL) and the control (196.56 ± 21.56 pg/mL), figure 3. In our study, the increasing in IL-1 α may related to the short of ovulate in PCSO women which match a study carried out by Zangeneh et al. and Hlail et al, [7,30], which revealed that the serum level of IL-1 α higher in the PCOS group compared to the control group. In contrast to the present study, Moin

et al. [31] revealed no variation in the IL-1 α levels between non-obese PCOS participants and controls. Also, Aboeldalyl et al. [32] discovered that the presence of inflammation has therapeutic benefits and has been linked to the pathophysiological process underlying PCOS and its consequences. A study by Rostamtabar et al. [33] discovered that chronic low-grade inflammation and IL-1 family mediators may contribute to the pathophysiology of PCOS.

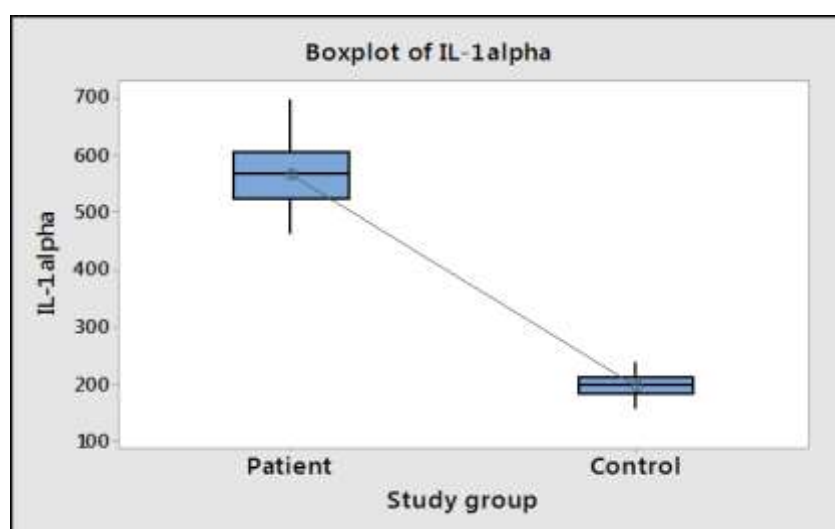


Figure 3: Comparable IL-1 α level boxplot across patients and controls

On another hand, the current study's Table 4 demonstrates a substantial difference ($P < 0.001$) in the serum levels of IL-36 α and IL-1 α , figure 4. The strong positive correlation may suggest a potential association between these inflammatory pathways .IL36 α may control IL-1 α in a feedback loop, with

primary keratinocytes quickly producing IL-1 α in response to IL36 α stimulation. Interestingly, IL-1 α induction was associated with increased IL-36 α expression [34]. In Iraq there was no previous research that reported.

Table 4: Pearson Correlation among Study Parameters and Their Relationship with Demographic Factors				
Variable	Statistics	IL-1 α	Age	BMI
IL-36 α (ng/L)	correlation	0.866	-0.248	0.249
	P-value	0.001**	0.090	0.088
IL-1 α (pg/mL)	correlation	-	0.212	0.356
	P-value	-	0.149	0.013

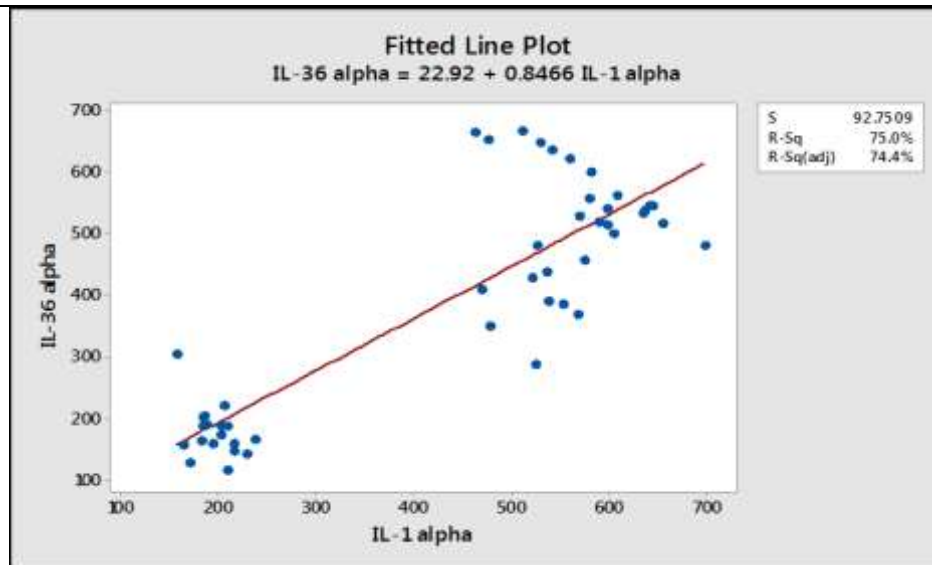


Figure 4: correlation between IL-36α and IL-1α

Furthermore, IL-36α was not significantly associated with age or BMI. Similarly, IL-1α was not significantly associated with age, but a significant positive correlation was observed between IL-1α and BMI ($r = 0.356$, $P = 0.013$), as shown in Table 4. These results are compatible with some that other researchers documented. Saleh et al. [35] elucidated that, based on age, PCOS women's IL-1α levels do not significantly vary from those of the control group. Moreover, Eroglu and Cakmakliogullari [10] confirmed that there was no association between the IL-36α level and BMI ($P > 0.05$). Additionally, in healthy obese women, the concentration of IL-1 was higher compared to their healthy lean counterparts; however, no such difference was observed when comparing obese and lean women with PCOS [36]. However, the findings of the present study consistent with those reported by [37], who identified IL-1α as one of the key adipokines regulating inflammatory processes in obese individuals. They suggested that dysfunctional adipose tissue contributes significantly to systemic inflammation through the release of cytokines such as IL-1α. This may support the possible association between increased BMI and IL-1α levels observed in the present study. A notable limitation of this study is the sample size of the PCOS group. While PCOS is a recognized condition, a larger patient cohort would strengthen the generalizability of the findings. Furthermore, the underlying causes and optimal treatment strategies for PCOS remain elusive. Future research with larger and more diverse patient populations is warranted to solidify the observed associations between IL-1α, IL-36α with PCOS.

4. Conclusions

The findings of the present study demonstrate that pro-inflammatory cytokines (IL-36α and IL-1α) are significantly elevated in women with PCOS. These findings suggest that IL-36α and IL-1α may be associated with the inflammatory profile of PCOS. Importantly, the elevated IL-1α levels may be partly influenced by the higher BMI observed in the PCOS group, whereas IL-36α was not significantly associated with BMI or age. The significant positive correlation between IL-36α and IL-1α further supports their potentially interconnected roles in the inflammatory processes. In addition, this study also revealed elevated levels of testosterone, FSH, LH, and prolactin in PCOS patients, which are likely associated with clinical manifestations of hyperandrogenism.

Acknowledgments: The authors thank Dr. Ali Hussein and Dr. Mohammed Al-Dabbagh for their valuable comments.

Conflicts of Interest: The authors declare no conflicts of interest regarding this work.

Funding Statement: No fund was received for this work.

Appendix (1) IL-36 α ELISA kits

Parameter	IL-36α ELISA Kit
Full kit name	Human Interleukin-36 Alpha, IL1F6 ELISA Kit
Manufacturer	BT LAB (Bioassay Technology Laboratory), China
Catalog number	E5524Hu
Lot number	Not specified in the provided kit instructions
Detection range	10–2000 ng/L
Sensitivity	4.65 ng/L
Intra-assay CV	4.4–6.7%
Inter-assay CV	<10%
Sample dilution	No dilution
Number of technical replicates	Not specified for samples; standards were prepared in duplicate

Appendix (2) IL-1α ELISA kits

Parameter	IL-1α ELISA Kit
Full kit name	Enzyme-linked Immunosorbent Assay Kit for Interleukin 1 Alpha (IL1α)
Manufacturer	Cloud-Clone Corp., USA
Catalog number	SEA071Hu
Lot number	Not specified in the provided kit instructions
Detection range	7.8–500 pg/mL
Sensitivity	<2.7 pg/mL
Intra-assay CV	<10%
Inter-assay CV	<12%
Sample dilution	Diluted by PBS; optimal dilution should be determined according to sample concentration
Number of technical replicates	Duplicate readings for standards, controls, and samples; duplication is recommended but not required

References

- [1] Eiras, M. C.; Pinheiro, D. P.; Romcy, K. A. M.; Ferriani, R. A.; Reis, R. M. D.; Furtado, C.L.M.; "Polycystic ovary syndrome: the epigenetics behind the disease". *Reprod. Sci.*, 29(3): 680-94, 2022.
- [2] Sirmans, S. M.; Pate, K. A.; "Epidemiology, diagnosis, and management of polycystic ovary syndrome". *Clin. Epidemiol.*, 6: 1-13, 2013.
- [3] Peña, A.S.; Witchel, S.F.; Hoeger, K.M.; Oberfield, S.E.; Vogiatzi, M.G.; Misso, M.; Garad, R.; Dabadghao, P.; Teede, H.; "Adolescent polycystic ovary syndrome according to the international evidence-based guideline". *BMC Med.*, 18(1): 72, 2020.
- [4] Ibraheem, Q.A.; Al Obaidy, L.H.A.; Nasir, G.A.; Al-Obaidi, M.T.M.; "Fat Mass and Obesity Association gene Polymorphism in PCOS Iraqi Women". *Baghdad Sci. J.*, 17(3): 1103-1112, 2020.
- [5] Kuang, H.; Duan, Y.; Li, D.; Xu, Y.; Ai, W.; Li, W.; Wang, Y.; Liu, S.; Li, M.; Liu, X.; Shao, M.; "The role of serum inflammatory cytokines and berberine in the insulin signaling pathway among women with polycystic ovary syndrome". *PLoS. ONE.*, 15(8): e0235404, 2020.
- [6] Silva, J. R.; Lima, F. E.; Souza, A. L.; Silva, A. W.; "Interleukin-18 and TNF-α systems in ovarian follicles and their roles during follicular

- development, oocyte maturation and ovulation". *Zygote.*, 28(4): 270–7, 2020.
- [7] Zangeneh, F. Z.; Naghizadeh, M. M.; Masoumi, M.; "Polycystic ovary syndrome and circulating inflammatory markers". *Int. J. Reprod. Biomed.*, 15(6): 375–82, 2017.
- [8] Voronov, E.; Dotan, S.; Krelm, Y.; Song, X.; Elkabets, M.; Carmi, Y.; Rider, P.; Idan, C.; Romzova, M.; Kaplanov, I.; Apte, R.N.; "Unique Versus Redundant Functions of IL-1alpha and IL-1beta in the Tumor Microenvironment". *Front. Immunol.*, 4, 2013.
- [9] Sachin, K. L.; Arnold, G. C. N.; Towne, J. E.; "Role of IL-36 cytokines in psoriasis and other inflammatory skin conditions". *Cytokine*, 156: 155897, 2022.
- [10] Eroglu, S.; Cakmakliogullari, E. K.; "Decreased serum profile of the interleukin-36a in polycystic ovary syndrome". *Taiwan J Obstet Gynecol.*, 60(6): 1018-1022, 2021.
- [11] Giannoudaki, E.; Hernandez-Santana, Y.E.; Mulfaul, K.; Doyle, S.L.; Hams, E.; Fallon, P.G.; Arimin, M.; Donal, O.S.; Manfred, K.; Andrew, E. H.; Patrick, T. W.; "Interleukin-36 cytokines alter the intestinal microbiome and can protect against obesity and metabolic dysfunction". *Nat Commun.*, 10: 4003, 2019.
- [12] Ballak, D. B.; Stienstra, R.; Tack, C. J.; Dinarello, C. A.; van Diepen, J. A.; "IL-1 family members in the pathogenesis and treatment of metabolic disease: focus on adipose tissue inflammation and insulin resistance". *Cytokine*, 75: 280–290, 2015.
- [13] Jacob, P.C.; Marcelle, I.C.; "Current Guidelines for Diagnosing PCOS". *Diagnostics (Basel)*, 13(6): 1113, 2023.
- [14] Flegal, K. M.; Graubard, B. I.; Williamson, D. F.; Gail, M. H.; "Excess deaths associated with underweight, overweight, and obesity". *JAMA.*, 293(15): 1861-7, 2005.
- [15] Aslam, M.; Batool, M.; Tahir, M.; Asmat, A.; Naeem, S.; Tul, W. U.; Haneef, Y.; Sabir, U.; "An Explanatory Research to Undermine the Knowledge Related Attributes of Polycystic Ovary Syndrome Among Adult Females: Polycystic Ovary Syndrome Among Adult Females". *Pakistan Biomed.J.*, 5(3): 73-77, 2022.
- [16] Agarwal, M.; Sinha, S.; Lohani, P.; Singh, R.; Dureja, S.; "Polycystic Ovarian Syndrome in Aging Women: An Observational Study". *Cureus.*, 14(9): e29776, 2022.
- [17] Kasim, S. F.; Saadoon, S. J.; "The Correlation of BMI and Age to Some Hormonal Indices in Iraqi Women with the Polycystic Ovarian Syndrome". *Int. J. Drug Deliverv. Technol.*, 12(2): 829-833, 2022.
- [18] Ibrahim, M. I.; Al-saffar, J. M.; "Serum level evaluation of interleukin-18 in obese women with polycystic ovary syndrome". *Iraqi J. Sci.*, 59(4B): 1989-1994, 2018.
- [19] Al-Shattawi, S. S. M.; Al-Jumili, E. F.; Al-Azzam, M.A.; "The relationship between obesity and polycystic ovary syndrome in a sample of Iraqi infertile women". *Iraqi J. Biotechnol.*, 7(3): 40-46, 2018.
- [20] Alfatlawi, W. R.; "Study the Effect of Interleukin36 Gamma and AMH in Iraqi Women with PCOS". *Al-Mustansiriyah J. Sci.*, 28(3): 151–156, 2017.
- [21] Al-Taie, F. K. H.; Al-Jawadi, Z. A.; "The Impact of Obesity on Infertile Women with Polycystic Ovaries in Iraq". *Al-Rafidain J. Med. Sci.*, 28(2): 1-9, 2019.
- [22] Alsaadi, Y.L.; Mohamad, B.J.; "Prevalence of hyperandrogenism in Iraqi women with polycystic ovary syndrome". *Iraqi J. Sci.*, 60(12): 2600-2608, 2019.
- [23] Al-Musawy, S. H. H.; Al-Saimary, I. E.; Flaifil, M. S.; "Levels of cytokines profile in polycystic ovary syndrome". *Med. J. Babylon*, 15(2): 124-128, 2018.
- [24] Solomon, C.G.; "The epidemiology of polycystic ovary syndrome prevalence and associated disease risks, endocrinology and metabolism clinics of cohort study". *J. Women Health*, 28(2): 247–263, 1999.
- [25] AL kafhage, F. A.; Abbas, A. N.; Al-Masaoodi, R. A.; Hassan, S.; Al-Shemery, M. K.; "The relationship between hormonal levels and hematological parameters in cystic ovarian syndrome". *J. Med. Life*, 16(6), 2023.
- [26] Wang, K.; Li, Y.; Chen, Y.; "Androgen excess: a hallmark of polycystic ovary syndrome". *Front. Endocrinol*, 14: 1273542, 2023.

-
- [27] Neurath, M.F.; "IL-36 in chronic inflammation and cancer". *Cytokine Growth Factor Rev.*, 55 : 70–79, 2020.
- [28] Abraham Gnanadass, S., Divakar Prabhu, Y. & Valsala Gopalakrishnan, A.; "Association of metabolic and inflammatory markers with polycystic ovarian syndrome (PCOS): an update". *Arch Gynecol Obstet*, 303, 631–643, 2021.
- [29] Halah A. A., Methaq J. A., Manal, T. A.; Majid, M. M.; "Functional impact of IL-36γ rs28947206/rs28947207 and IL-17 a rs2275913 polymorphisms in polycystic ovary syndrome: Novel insights from the Iraqi population". *Science Direct*, 198, 157106, 2026.
- [30] Hlail, Z. A.; Mohammed, K. I. A.; "Immunological Detection of Interleukin-1α (IL-1α) In Iraqi Women with Polycystic Ovarian Syndrome". *SAR J. Med. Biochem.*, 3(3): 39-42, 2022.
- [31] Moin, A.S.; Sathyapalan, T.; Atkin, S.; Butler, A.; "Inflammatory markers in non-obese women with polycystic ovary syndrome are not elevated and show no correlation with vitamin D metabolites". *Nutrients*, 14(17): 3540, 2022.
- [32] Aboeldaly, S.; James, C.; Seyam, E.; Ibrahim, E.M.; Shawki, H.E.; Amer, S.; "The Role of Chronic Inflammation in Polycystic Ovarian Syndrome-A Systematic Review and Meta-Analysis". *Int. J. Mol. Sci.*, 22(5): 2734, 2021.
- [33] Rostamtabar, M.; Esmailzadeh, S.; Tourani, M.; Rahmani, A.; Bae, M.; Shirafkan, F.; Kiarash, S.; Sajede, S. M.; Soheil, E.; Hamid, R. N.; "Pathophysiological roles of chronic low-grade inflammation mediators in polycystic ovary syndrome". *J Cell Physiol.*, 236(2): 824–38, 2021.
- [34] Yuan, Z.C.; Xu, W.D.; Liu, X.Y.; Liu, X.Y.; Huang, A.F.; Su, L.C.; "Biology of IL-36 Signaling and Its Role in Systemic Inflammatory Diseases". *Front. Immunol.*, 10: 2532, 2019.
- [35] Saleh, H.S.; AlRufaei, I.A.; Jawad, E.S.; "Physiological Change of Interleukin-1 Alpha (IL1α) in Iraqi, Thi-Qar Women with Polycystic Ovarian Syndrome". *Univ. Thi-Qar J. Sci.*, 10(1), 171-174, 2023.
- [36] Mazloomi, S.; Barartabar, Z.; Pilehvari, S.; "The Association Between Increment of Interleukin-1 and Interleukin-6 in Women with Polycystic Ovary Syndrome and Body Mass Index". *J Reprod Infertil.*, 24(1): 26-34, 2023.
- [37] Fain, J. N.; "Release of inflammatory mediators by human adipose tissue is enhanced in obesity and primarily by the nonfat cells: A review". *Mediators Inflamm.*, 2010: 1–20, 2010.
-