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Circulating Levels of GLP-1 and AGEs in a Group of Iraqi Women with Polyendocrine Metabolic Ovarian Syndrome: A Case-Control Study

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Abstracts:

Background: Polyendocrine metabolic ovarian syndrome (PMOS) is a common condition that affects women of reproductive age. It is usually associated with hormonal and metabolic disorders. However, the precise relationship between these derangements and the development of PMOS is unclear.
Objectives: To evaluate the circulatory levels of Glucagon-Like Peptide-1 (GLP-1) and Advanced Glycation End-products (AGEs) in PMOS and to explore the possible application of these factors as potential diagnostic biomarkers.
Methods: A case-control study was conducted at the College of Science, University of Baghdad, between Nov. 2024 and Feb. 2025. The study involved 90 Iraqi women aged 18-45 years, divided into two groups: 50 women who had recently been diagnosed with PMOS and 40 healthy women as controls. Hormonal profiles and metabolic parameters (GLP-1 and AGEs) were measured in all participants.
Results: Women with PMOS had an elevated hormonal profile, characterized by higher concentrations of LH, an LH/FSH ratio, testosterone, and Anti-Müllerian Hormone (AMH), along with a considerable drop in FSH compared to the control group. They also showed significant elevations in metabolic parameters (AGEs and GLP-1) compared to the control group. GLP-1 and AGEs had an area under the curve (AUC) of 1.000, with 100% sensitivity and 100% specificity, indicating their diagnostic value.
Conclusions: The increase in GLP-1 and AGEs reveals hormonal-metabolic dysregulation, indicating a high degree of correlation in the complexity and progression of the disease. The high diagnostic accuracy of GLP-1 and AGEs in PMOS, may suggest that they are potential biomarkers for diagnosing the disease.
Keywords: Advanced Glycation End Products; Glucagon-Like Peptide-1; HOMA-IR; Hyperandrogenism; Polyendocrine Metabolic Ovarian Syndrome.

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Introduction

Polyendocrine Metabolic Ovarian Syndrome (PMOS) is one of the most prevalent endocrine conditions affects approximately 6-20% of women of reproductive age, depending on the applied diagnostic criteria, and is a key cause of decreased reproductive function. PMOS has devastating effects on physical and mental health, infertility, and anovulation (1, 2). The diagnostic criteria for PMOS have been reported according to the Rotterdam/European Society of Human Reproduction and Embryology (ESHRE) guidelines, which include hyperandrogenism (HA), ovulatory dysfunction, and/or polycystic ovary morphology (3). Recently, PMOS has become well known as a complex metabolic and endocrine disease with major metabolic effects, such as insulin resistance (IR), uncontrolled lipid metabolism, a higher risk of type 2 diabetes mellitus (T2DM) and heart problems, in addition to its reproductive symptoms (4). Insulin resistance (IR), affecting nearly half of lean females and up to three-quarters of obese women with this syndrome, is a significant pathogenic feature (5, 6). IR exacerbates hyperinsulinemia, which in turn worsens HA, preventing follicular development and leading to long-term metabolic issues such as obesity, dyslipidemia, T2DM, and cardiac disease (7-9). Therefore, early IR detection is essential for improving reproductive and metabolic outcomes in women with PMOS (10).

Hormones related to metabolism, especially glucagon-like peptide (GLP-1), are important because they are involved in regulating blood sugar levels through a series of complex metabolic processes. After eating, the gastrointestinal L-cells release GLP-1, which increases the amount of insulin that depends on glucose levels, which in turn helps to release the glucagon hormone. As a result, it slows down stomach emptying and makes one feel full (11, 12). Therefore, disturbances in GLP-1 levels are closely related to obesity and T2DM, and increasing evidence links them to the development of PMOS (13). However, many previous studies have shown wide variations in GLP-1 levels; some studies have indicated low levels, whereas others have not. Furthermore, some studies have suggested elevated GLP-1 levels in conjunction with clinical symptoms of the disease (14, 15). GLP-1 has emerged as a significant pathophysiological factor and potential therapeutic target in PCOS due to its simultaneous adverse effects on glucose metabolism and reproductive function.

Recently, advanced glycation end products (AGEs) have received interest as potential contributors to the etiology of this syndrome, in addition to IR and incretin instability. When proteins, lipids, and nucleic acids are glycated in a manner that does not require the presence of enzymes, reactive chemicals known as AGEs are generated. Hyperglycemia, oxidative stress, and consumption of glycotoxins through food all contribute to the acceleration of this process (16, 17). AGEs can activate pro-inflammatory and oxidative pathways through receptor binding. This triggers the development of a vascular injury, tissue damage, and disturbances in the endocrine system, which in turn contribute to the development of PMOS (18). There is a correlation between higher concentrations of AGEs in women with the syndrome and infertility, increased androgen production, and changes in the function of granulosa and theca cells, which ultimately leads to a reduction in folliculogenesis and ovulatory failure (19, 20). Moreover, they may occur even without obesity or poor glycemic regulation, which explains the critical role of AGEs in PMOS independently of metabolic comorbidities (21).

Increased AGE formation due to GLP-1 dysfunction and IR, is associated with reproductive and metabolic disorders in PMOS. However, the specific impact of these parameters on the progression of the syndrome remains unclear. By examining and addressing this knowledge gap, innovative biomarkers, therapeutic strategies, and insights into metabolic dysregulation and reproductive outcome pathways can be developed. Therefore, this study aimed to measure the serum levels of GLP-1 and AGEs in Iraqi women with PMOS to determine their roles in the etiology of the disease and to explore the diagnostic accuracy of GLP-1 and AGEs levels as potential biomarkers for Iraqi women with PMOS.

Cases and Methods

Study design and participants: This case-control study was conducted at the Chemistry Department, College of Science, University of Baghdad. Participants were recruited from the Al-Alwawiya Maternity Teaching Hospital in Baghdad, during the period from Nov. 2024 to Feb. 2025. In total, 90 women aged 18 - 45 years were included and classified into two groups. The first group comprised 40 healthy female controls, all of whom were clinically normal and free from any known systemic illnesses. The control group comprised women who underwent routine check-ups at the same hospital. Controls were screened by a specialist and confirmed to be free of chronic systemic disorders. The second group comprised 50 women with newly diagnosed PMOS. PMOS diagnosis was based on the Rotterdam criteria, necessitating the presence of at least two of the following: clinical and/or biochemical hyperandrogenism (HA), oligo- or amenorrhea, and polycystic ovarian morphology observed via ultrasound examination (3). All participants provided written informed consent before sample collection and completed a structured questionnaire regarding their demographics, medical and family history, lifestyle habits, and weight.

The study protocol was approved by the Ethics Committee of the College of Science, University of Baghdad (Ref. CSEC/1024/0071 issued on October 13, 2024).

Exclusion Criteria: Participants with obesity who have body mass index (BMI ≥ 30 kg/m²), hypertension, hyperprolactinemia, cardiovascular

disease, renal disease, diabetes mellitus, thyroid dysfunction, or tumors were excluded. Women who were pregnant, lactating, smokers, or users of medications and dietary supplements were also excluded to avoid interference in the study results of patients with PMOS.

Blood Collection and Processing: All participants underwent a comprehensive clinical evaluation, including anthropometric measurements. On day 2 of the menstrual cycle, approximately 5 mL of venous blood was collected in anticoagulant and gel-separator tubes. In cases of amenorrhea, blood samples were collected after clinical evaluation by a specialist gynecologist and exclusion of pregnancy, at a standardized time point determined by the treating physician to ensure consistency among participants. The first tube was used to detect the percentage of glycosylated hemoglobin (HbA1c) in the blood. The other tubes were allowed to clot and then centrifuged at 4000 rpm for 10 min to obtain serum. The serum was divided into two portions: one was used for Fasting Blood Glucose (FBG), serum insulin, LH, FSH, testosterone, and Anti-Müllerian Hormone (AMH), and the other was stored at -20 °C for subsequent analyses of GLP-1 and AGEs.

Biochemical assay: Body Mass Index (BMI) was calculated by dividing the weight (kg) by the height squared (m^2) (22). Serum levels of FBG, insulin, LH, FSH, testosterone, and AMH were determined using electrochemiluminescence immunoassay (ECLIA) on Roche Cobas E411 and Hitachi immunoassay analyzers in Germany. HbA1c levels were estimated using high-performance liquid chromatography (HPLC). HOMA-IR was estimated using the following formula: HOMA-IR = (fasting insulin × fasting glucose / 22.5) (23). Serum concentrations of GLP-1 and AGEs were quantified using enzyme-linked immunosorbent assay (ELISA) with a commercially available kit (ELK Biotechnology, USA) according to the manufacturer's instructions.

Sample Size: Sample size calculation was performed using G*Power (version 3.1.9.7). The calculation was based on a two-tailed independent-samples t-test, assuming a medium-to-large effect size (Cohen's d = 0.8), significance level of $\alpha = 0.05$, statistical power of 0.96, and allocation ratio (N2/N1) of 0.8. The analysis indicated a required total sample size of 90 participants, with 50 and 40 participants in groups 1 and 2, respectively.

Statistical Analysis: All statistical analyses were performed using SPSS version 26 (IBM Corp., Armonk, NY, US). Results are presented as mean ± standard deviation (SD). An independent-samples t-test was used to compare the group means. The normality of the data distribution was assessed using the Shapiro-Wilk test. A $p < 0.050$ was considered statistically significant, and $p < 0.001$ was considered highly significant. Pearson's correlation analysis was used to assess the association between the study variables and the PMOS cohort. Receiver operating characteristic (ROC) curve analysis was used to determine the sensitivity, specificity, and best cutoff values for the biomarkers studied.

Results

Women with PMOS exhibit severe hormonal dysregulation, as evidenced by considerably higher testosterone and LH levels, an increased LH/FSH ratio, and a significant drop in FSH hormone concentration. AMH concentrations were higher in the PMOS group than in the controls ($P<0.001$), indicating enhanced ovarian follicular activity associated with the syndrome. FBG, insulin, and HbA1c levels were significantly higher in women with PMOS than in healthy women. The severity of the condition was determined using the HOMA-IR scale, which showed a significant difference ($P<0.001$) when comparing PMOS women with their healthy counterparts. The GLP-1 and AGEs levels were significantly elevated in the PCOS group compared to those in the healthy controls ($P< 0.001$), as shown in **Table 1**.

Table 1: Mean ± SD of demographic, hormonal and biochemical variables of the PMOS women and healthy controls

Variables	Groups		*P* value
	Control (n=40)	PMOS (n=50)	
Age (years)	28.3 ± 6.59	26.7 ± 5.71	0.240
BMI (kg/m^2)	26.9 ± 1.94	27.5 ± 1.53	0.095
LH (mIU/mL)	5.6 ± 1.36	8.9 ± 1.72	< 0.001**
FSH (mIU/mL)	9.5 ± 1.66	5.5 ± 1.31	< 0.001 **
LH/FSH ratio	0.6 ± 0.19	1.7 ± 0.48	< 0.001 **
Testosterone (ng/mL)	0.3 ± 0.11	1.3 ± 0.38	< 0.001 **
AMH (ng/mL)	4.5 ± 1.28	9.9 ± 1.83	< 0.001 **
FBG (mg/dL)	86.1 ± 10.29	92.0 ± 8.17	0.004 **
HbA1c (%)	5.4 ± 0.64	5.8 ± 0.51	0.003 **
Insulin (mIU/mL)	6.4 ± 2.25	11.0 ± 3.27	< 0.001 **
HOMA-IR	1.3 ± 0.60	2.5 ± 0.80	< 0.001 **
GLP-1 (ng/mL)	18.2 ± 0.89	11.3 ± 2.20	< 0.001 **
AGEs (ng/mL)	10.6 ± 2.57	5.3 ± 0.69	< 0.001 **

*$P < 0.05$ and **$p < 0.001$.

The correlations between GLP-1 and AGEs levels and other clinical and biochemical indicators in women with PMOS are shown in **Table 2**. A significant weak positive correlation was noted between GLP-1 and LH (r=0.295, p=0.037). No statistically significant correlations were observed between the other parameters in the PMOS group.

Table 2: Pearson correlation analysis of GLP-1 and AGEs levels with other indicators in women with PMOS

Parameter's	GLP-1 (ng/ml)		AGEs (ng/ml)	
	r	p	r	p
Age (years)	0.150	0.299	0.232	0.105
BMI (kg/m^2)	-0.194	0.177	0.114	0.432
LH (mIU/mL)	0.295*	0.037	-0.214	0.136
FSH (mIU/mL)	0.046	0.751	-0.125	0.386
LH/FSH ratio	0.146	0.312	0.005	0.974
Testosterone (ng/mL)	0.050	0.732	0.229	0.110
AMH (ng/mL)	0.025	0.866	-0.018	0.901
FBG (mg/dL)	-0.232	0.104	0.006	0.969
HbA1c %	-0.232	0.105	0.001	0.997
Insulin (mIU/mL)	0.007	0.963	-0.097	0.501
HOMA-IR	-0.063	0.664	-0.092	0.527

*p < 0.05 and **p< 0.001.

The ROC curve was used to evaluate the area under the curve (AUC) for assessing the diagnostic efficacy of GLP-1 and AGEs in patients with PMOS (**Figure 1 and Table 3)**. The AUC values for GLP-1 and AGEs were 1.000 for the control and PMOS patient groups, indicating a strong ability to differentiate between control and PMOS patient groups. The corresponding cutoff values were established at > 15.831 and > 6.992, yielding a sensitivity of 100% and a specificity of 100% for GLP-1 and AGEs, respectively.

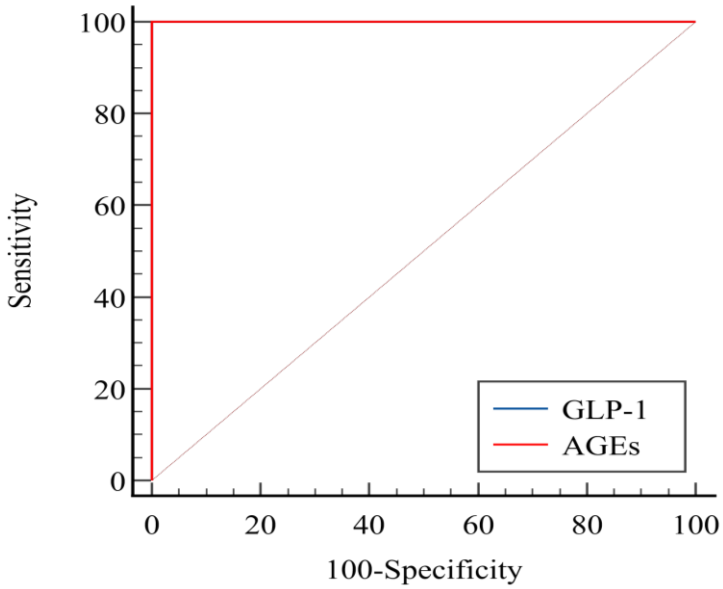

Figure 1: The ROC of GLP-1 and AGEs in control vs. PMOS patients

Table 3: The ROC curve data of GLP-1 and AGEs, in control vs. PMOS patients

Variable(s)	AUC (CIs)	Sensitivity %	Specificity %	Cut off value	Youden index
GLP-1 (ng/ml)	1.000 (0.960 to 1.000)	100	100	> 15.831	1.000
AGEs (ng/ml)	1.000 (0.960 to 1.000)	100	100	> 6.992	1.000

Discussion

Consistent with multiple studies, our findings indicate elevated hormonal levels, including serum LH, testosterone, and AMH, along with an increased LH/FSH ratio in women with PMOS relative to healthy controls (24-26). Other studies have shown that abnormal regulation of these hormones may be related to PMOS (27). The elevation in LH concentration signifies dysregulation of the hypothalamic-pituitary axis, in which augmented GnRH pulses preferentially enhance LH secretion over FSH, thereby increasing ovarian androgen production (28). Other studies have emphasized that BMI is not correlated with an increased LH/FSH ratio, as the LH/FSH ratio is the same in women with a normal BMI (29). A higher level of testosterone in the blood confirms the hyperandrogenic environment linked to PMOS in women. Another study found that serum testosterone and its free form levels were significantly increased in hirsute women and were

positively correlated with glucose levels and BMI among women with PMOS (30-32).

An increase in serum AMH concentration has been suggested as a biomarker for both follicular excess and inherent ovarian dysfunction associated with the syndrome (33). The LH/FSH ratio, which has been used as a diagnostic tool in the past but is not always as reliable as AMH, still demonstrates how gonadotropin levels change in the body when a patient has PMOS. A study on Iraqi women showed that a significantly high level of hormonal alteration, including AMH, was found in women with PMOS compared to healthy controls (34). The distinctive hormonal changes observed in the current study underscore the pivotal role of the pituitary-hypothalamic-ovarian axis as the most prominent feature of polycystic ovary syndrome.

The results of the current study show that changes occurred at both the hormonal and metabolic levels, and that women with PMOS had significantly higher levels of HbA1c, HOMA-IR, GLP-1, and AGEs than healthy women. These findings support the hypothesis that PMOS is not only an organic dysfunction but also a complicated hormonal and metabolic disorder. Elevated FBG and HbA1c levels indicate an imbalance in glucose metabolism and long-standing regulatory disturbances. Research on PMOS has reported a very high correlation between HbA1c and the syndrome, which implies that blood sugar control should be considered as part of the treatment protocol for PMOS (35).

HbA1c is a widely recognized method for determining the amount of sugar one has been exposure over the past two to three months. The increasing number of individuals with PMOS indicates that their tolerance to glucose is deteriorating, which can lead to the development of T2DM (36). HbA1c is sensitive and specific for diagnosing prediabetes in young women with PMOS (37). Recent studies on PMOS have demonstrated a disruption in glucose metabolism, regardless of the presence or absence of obesity. This aligns with the findings of our study, which indicates an alternative perspective on the underlying mechanisms of disease development and progression (38). Recent results suggest that controlling the metabolic profile, particularly blood glucose and HbA1c levels, is essential for the comprehensive clinical management of this syndrome.

One of the key pathophysiological characteristics is IR, with prevalence rates ranging from 50% to 70%, depending on ethnicity, BMI, and diagnostic criteria (39). On a molecular basis, compensatory hyperinsulinemia occurs when post-receptor insulin signaling is impaired. This condition is exacerbated by HA and chronic inflammation. It maintains anovulation and hyperandrogenic features, which continue to worsen metabolic issues and increase ovarian androgen production (40). It is crucial to note that IR is a condition found in both obese and lean PMOS phenotypes. However, it is more likely to be more intense in the former and, to a certain degree, supports the definition of insulin resistance as an intrinsic defect in the latter.

GLP-1 plays a critical role in glucose metabolism and insulin regulation, and its elevated levels in the present study may reflect this. This may also represent a compensatory response to the underlying IR. While some studies have reported reduced GLP-1 levels in PMOS, the increased levels observed in the present study may reflect an adaptive mechanism to maintain glucose homeostasis (41). This is further supported by the therapeutic efficacy of GLP-1 receptor agonists, which have been shown to improve metabolic parameters, reduce body weight, and decrease androgen levels in patients with PMOS (42-44). The information presented above highlights the importance of GLP-1 as the main mediator between metabolic and reproductive dysfunction in PMOS, making it a viable therapeutic target.

Patients with PMOS have metabolic abnormalities due to high levels of AGEs. The role of AGEs in PMOS has garnered increasing interest from researchers studying female reproductive disorders. AGEs bind to their membrane receptor, RAGE, triggering signaling pathways that cause oxidative stress, inflammation, ovulatory dysfunction, HA, IR, and obesity. AGE products are particularly detrimental to ovarian granulosa cells, affecting cell growth and hormone release (45). Our findings are supported by recent systematic reviews that reported that AGEs are significantly higher in women with PMOS than in controls and are associated with the metabolic and reproductive characteristics of the condition (46). This indicates that AGEs can be considered biomarkers and predictors of oxidative stress and may serve as therapeutic targets for PMOS. These results support a functional and pathological relationship between blood sugar imbalance and inflammatory mediators in the progression and development of PMOS. Conversely, high insulin concentrations and HOMA-IR indicate pathology, reflecting metabolic issues, and are closely associated with hormonal alterations, specifically high androgen levels (47). Along with elevated GLP-1 levels, high concentrations of AGEs are signs of a chronic oxidative environment and seem to be directly linked to the evolution of this syndrome.

ROC curve analysis showed that both GLP-1 and AGEs had outstanding diagnostic performance in distinguishing PMOS from healthy controls. Both biomarkers had AUCs of 1.000, indicating maximal discriminative power in the cohort under study, implying that changes in incretin signaling and glycation-related pathways are closely linked to PMOS pathophysiology. The identified cut-off values (> 15.831 for GLP-1 and > 6.992 for AGEs) showed 100% sensitivity and specificity, highlighting their potential as strong diagnostic predictors. These results confirm that metabolic dysregulation and oxidative stress are the primary characteristics of PMOS, regardless of the classical diagnostic criteria.

The observed perfect diagnostic performance (AUC = 1.000) may be attributed to several factors. Biologically, both GLP-1 and AGEs are closely

linked to the key pathophysiological mechanisms of PMOS, including IR, oxidative stress, and metabolic dysregulation, which may help distinguish affected individuals from healthy individuals. Methodologically, the relatively homogeneous study population and strict inclusion criteria may have reduced the variability and enhanced group separation. In addition, the case-control design is inherently more likely to yield stronger discrimination than population-based designs. From a statistical perspective, the complete separation of values between groups in smaller datasets can overestimate the diagnostic performance. Therefore, although the findings suggest a strong discriminatory potential, they should be interpreted with caution and validated in larger, independent, and more heterogeneous populations.

Limitations

This study has some limitations, including a relatively small sample size and recruitment from a specific Iraqi healthcare center, which may limit the generalizability of the findings. Additionally, the case-control design prevents establishing causality between biomarkers and PMOS. Some lifestyle-related confounding factors were not fully evaluated, and biomarker levels were only measured once. Further larger, prospective research is necessary to validate these results.

Conclusions

GLP-1 and AGEs had high diagnostic accuracy in distinguishing patients with PMOS from controls, as indicated by the ideal AUC values, sensitivity, and specificity at the preset cutoff points. These findings indicate the potential of GLP-1 and AGEs as biomarkers for PMOS diagnosis and risk stratification. However, additional multicenter and longitudinal studies are needed to verify these results, test their replicability, and identify their clinical relevance in routine diagnostic practice.

Authors' declaration

We confirm that all the figures and tables in the manuscript belong to the current study. Authors sign on ethical considerations' approval - Ethical Clearance: The project was approved by the local ethical committee in the College of Science Institutional Ethics Committee at the University of Baghdad, according to code number (CSEC/1-24/0071) on (13/10/2024).

Conflict of Interest:

The authors declare that they have no conflicts of interest.

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Data Availability:

Upon reasonable request, the corresponding author will make the data sets generated and/or analyzed during the current work available.

Authors' contributions:

Study conception & design: (Ahmed K. Atheeb, Saba Z. Hussein). Literature search: (Ahmed K. Atheeb). Data acquisition: (Ahmed K. Atheeb). Data analysis & interpretation: (Ahmed K. Atheeb, Saba Z. Hussein). Manuscript preparation: (Ahmed K. Atheeb). Manuscript editing & review: (Saba Z. Hussein).

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مستويات الببتيد الشبيه بالجلوكاجون-1 و منتجات الغلكزة المتقدمة في الدم لدى مجموعة من النساء العراقيات المصابات بمتلازمة المبيض الأيضية متعددة الغدد الصماء: دراسة الحالات والشواهد

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الخلاصة:
الخلفية: متلازمة المبيض الأيضية متعددة الغدد الصماء (PMOS) هو مرض شائع يصيب النساء في سن الإنجاب، وعادةً ما يرتبط بمضاعفات هرمونية واستقلابية. لكن العلاقة الدقيقة بين هذه المضاعفات وتطور هذه المتلازمة لا تزال غير واضحة.
الاهداف : تقييم مستويات الببتيد الشبيه بالجلوكاجون-1 و منتجات الغلكزة المتقدمة لدى المريضات المصابات بمتلازمة المبيض الأيضية متعددة الغدد الصماء ، واستكشاف امكانية استخدام هذه العوامل كمؤشرات حيوية تشخيصية محتملة.
الطريقة: أُجريت دراسة المقارنة حالات-شواهد في كلية العلوم بجامعة بغداد، في الفترة ما بين تشرين الثاني 2024 وشباط 2025. وشملت الدراسة 90 امرأة عراقية تتراوح أعمارهن بين 18 و45 عامًا، وقُسمن إلى مجموعتين: 50 امرأة تم تشخيص إصابتهن حديثا بمتلازمة المبيض الأيضية متعددة الغدد الصماء ، و40 امرأة سليمة. تم قياس المستويات الهرمونية والمعايير الأيضية (الببتيد الشبيه بالجلوكاجون-1 و منتجات الغلكزة المتقدمة) لجميع المشاركات.
النتائج: أظهرت النتائج أن النساء العراقيات المصابات بمتلازمة المبيض الأيضية متعددة الغدد الصماء لديهن مستوى هورموني مرتفع، يتميز بتركيزات أعلى من الهورمون اللوتيني، ونسبة الهورمون اللوتيني / الهورمون المنبه للجريبات، وهورمون التستوستيرون، والهورمون المضاد للمولر ، وانخفاض ملحوظ في مستوى الهورمون المنبه للجريبات مقارنةً بالمجموعة الضابطة. كما تبين أن النساء المصابات بمتلازمة المبيض الأيضية متعددة الغدد الصماء لديهن ارتفاع ملحوظ في المؤشرات الأيضية (الببتيد الشبيه بالجلوكاجون-1 و منتجات الغلكزة المتقدمة) مقارنةً بالمجموعة الضابطة. وقد بلغت المساحة تحت المنحنى 1.000 لكل من الببتيد الشبيه بالجلوكاجون-1 و منتجات الغلكزة المتقدمة، مع حساسية وخصوصية 100%، مما يجعلهما مؤشرين تشخيصيين مهمين.
الاستنتاجات: يشير ارتفاع مستوى الببتيد الشبيه بالجلوكاجون-1 و منتجات الغلكزة المتقدمة إلى خلل في التنظيم الهرموني الأيضي، مما يدل على وجود ارتباط وثيق بين المرض وتطوره. وقد أظهر كل من الببتيد الشبيه بالجلوكاجون-1 و منتجات الغلكزة المتقدمة دقة تشخيصية عالية في متلازمة المبيض الأيضية متعددة الغدد الصماء ، مما قد يشير إلى فائدتهما كمؤشرات حيوية محتملة لتشخيص المرض.

مفتاح الكلمات: نواتج الغلكزة المتقدمة؛ الببتيد الشبيه بالغلوكاغون-1؛ مقاومة الأنسولين؛ فرط الأندروجينية؛ متلازمة المبيض الأيضية متعددة الغدد الصماء