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Research Article

Assessment of Intestinal Hormones Cholecystokinin and Peptide YY in Iraqi Women with Polycystic Ovarian Syndrome

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Abstract

Background: Among the most prevalent hormonal, reproductive, and metabolic issues impacting women is polycystic ovarian syndrome. Also, insulin resistance raises the chance of developing chronic illnesses in women with polycystic ovarian syndrome, including diabetes mellitus, cardiovascular disease, metabolic syndrome, and potentially endometrial and breast malignancies.

Objectives: Measurement of two intestinal hormones (cholecystokinin, peptide YY), luteinizing hormone, follicle-stimulating hormone, and Prolactin. Waist and wrist circumference were measured in centimeters

Methods: A sample of polycystic ovarian syndrome women who were referred to the Medical City of Baghdad hospital for management of their infertility were recruited in the current study. Sixty polycystic ovarian syndrome patients were split into two groups based on Body Mass Index: thirty obese polycystic ovarian syndrome women and thirty overweight polycystic ovarian syndrome women. In addition, thirty healthy control women were added as a third group, whose average age was between 20 and 35 years. Polycystic ovarian syndrome in women was diagnosed using two of the three diagnostic criteria: polycystic ovaries in ultrasound, oligo or anovulation, and hyperandrogenism. The investigation ran from October 2023 until January 2024. The investigations of the patient were requested and all data in the study.

Results: Obese women with polycystic ovarian syndrome had significantly higher levels of waist circumference 102.75 ±1.45, prolactin 20.48 ±1.43, luteinizing hormone 7.85 ±0.56, Follicle-stimulating hormone 8.41 ±0.38, and lower levels of Peptide YY 45.33±16.62, and cholecystokinin 14.37 ±3.64.

Conclusion: Low cholecystokinin and Peptide YY in obese polycystic ovarian syndrome women lead to a rise in appetite, intake of food, an increase in waist circumference, and accumulation of fat in the abdomen.

Keywords: Cholecystokinin; Follicle-Stimulating Hormone; Luteinizing Hormone; Peptide YY; Prolactin.

Introduction

The woman's life and several disorders stemming from hormone imbalances, such as polycystic ovarian syndrome (PCOS), ovarian cancer, early menopause, and primary ovarian insufficiency, depend heavily on hormonal balance. First of all, the most prevalent endocrine condition in women is called PCOS, which can present as a broad spectrum of phenotypes, including alterations in metabolism, endocrine function, and reproduction (1).

Obesity is a major factor in developing PCOS in women who are fertile. PCOS is an endocrine illness that can arise from a mix of environmental and genetic factors. Obesity modifies the function of the hypothalamic–pituitary–ovarian axis, it has a significant effect on the reproductive organs. High insulin levels in obese women cause an increase in androgen synthesis, which can cause irregularities in the menstrual cycle, including oligomenorrhea and hyperandrogenism (2). A peptide hormone associated with the gastrointestinal (GI) tract is called cholecystokinin (CCK).

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carbohydrate, as well as products of protein digestion. It stimulates pancreatic growth, enzyme secretion, and gallbladder contraction by blocking the secretion of gastric acid. It is also having an effect on intestinal motility (3). By acting on vagal afferents, particularly in the duodenum, CCK reduces appetite. The physiology of PCOS includes high levels of insulin, elevated testosterone levels, and impaired ghrelin and CCK production. Reduced postprandial CCK production is linked to elevated testosterone levels in PCOS patients (4).

On the other hand, peptide YY (PYY) is a peptide of 36 amino acids that is released by L cells in the colon and distal ileum. After eating, the level of PYY in the plasma increases and peaks 1-2 hours after eating, staying high for as long as 6 hours. Meal content affects PYY release; protein causes greater amounts of PYY than fats and carbohydrates (5). It is thought to have a role in post-meal satiety, which lowers appetite. The quantitative analysis revealed that postprandial total PYY concentrations were statistically significantly lower in obese individuals

compared to controls (6). Prolactin hormone (PRL) is a hormone secreted by the pituitary gland. It has been connected to other functions, such as regulating immunity and reproductive systems (7). The amount of PRL in circulation determines how it affects insulin resistance and glucose metabolism. Elevated serum levels of PRL are indicative of an increased chance of dysmetabolism and obesity, including impaired sensitivity to insulin.

Luteinizing hormone (LH) is secreted by the anterior pituitary gland. Its function is the regulation of ovulation in females. It has been shown that women with PCOS have altered gonadotropin-releasing hormone production, which leads to normal follicle-stimulating hormone (FSH) secretion but an increase in LH secretion. This secretory pattern is a useful diagnostic indicator for PCOS since it frequently leads to an aberrant LH/FSH ratio (8).

Follicle-stimulating hormone (FSH) is a heterodimeric glycoprotein hormone released by gonadotropic cells in the anterior pituitary and has a role in reproduction. A relative increase in the release of LH relative to FSH is brought on by a change in the secretion pattern of gonadotropin-releasing hormone (GnRH). When GnRH is used consistently, the anterior pituitary secretes less FSH and LH, which prevents women from ovulating and producing estrogen. Estrogen levels have a negative feedback effect that lowers FSH production (9).

The aim of the present study was to assess two obesity-related hormones, PYY and CCK, and their role in the occurrence of PCOS.

Subjects and method

This case-control study was carried out at the Department of Biology, College of Science, University of Baghdad. The investigation ran from October 2023 until January 2024. Blood samples of PCOS women who visited infertility consultations at hospitals of the Medical City complex in Baghdad, Iraq. The study involved ninety women in total: sixty PCOS women (Thirty overweight and thirty obese PCOS women) and thirty healthy women without PCOS (control group). The average age of the patient and control groups was between 20 and 35 years. The formula used to determine BMI was BMI = weight (kg) / length (m^2). Scales in centimeters were used to measure the circumferences of the waist and wrist. Patients were divided into two groups according to their BMI: obese PCOS women with a body mass index ≥ 30 kg/m^2, and overweight PCOS women with a BMI < 30 kg/m^2(10) A consulting physician used the 2003 Rotterdam criteria to diagnose PCOS (hyperandrogenism, an ultrasound-measured ovarian volume greater than 10 ml, and ovulatory disruption (oligo-menorrhea or amenorrhea). The common symptoms were found in patients: irregular periods, infertility, hirsutism and alopecia. Including criteria of PCOS are: irregular menstrual cycle, increased androgen levels and multiple small cysts on the ovaries. Exclusion criteria of PCOS are Cushing's syndrome, hyperprolactinemia, thyroid diseases, as well as other causes of oligo-menorrhea or anovulation. FSH, LH, PRL, CCK, and PYY levels were evaluated by using serum of PCOS women and control women during the follicular phase (2-3 days of the menstrual cycle). Following the manufacturer's instructions, the Peptide YY (Catalog No: E-EL-H1237) Elabscience company (USA), was used. Cholecystokinin level (Cat.No E1357Hu), BT LAB (Bioassay Technology Laboratory) by China, was used by a sandwich ELISA approach, size was 96 wells. A universal microplate reader (ELX 800, UK) was used to measure the optical density of the final ELISA results. (LH) Hormone (Cat.No.0025296), (FSH) Hormone (Cat. No.0025265), and Prolactin hormone (PRL) (Cat.No.0025255) levels were measured by two-site immunoenzymometric assay, TOSOH AIA, Japan analyzer.

Statistical Analysis

Statistical Analysis System-SAS (2018) was utilized to determine how various factors affected the research parameters. The data were presented as means, standard deviations; the means of parameters were compared using a one-way analysis of variance (ANOVA). The probability level was below 0.05.

Results

The present study revealed that the BMI value was significantly elevated in obese PCOS (38.15 ±0.76 a kg/m^2; $P < 0.05$) when compared with control (29.92 ±0.58 b kg/m^2). Waist circumference values showed a significant increase in the obese PCOS group (102.75 ±1.45 a cm) compared with the control group (77.62 ±1.97 b cm). The level of wrist circumference was nonsignificantly high in the obese PCOS group (19.98 ±2.51 cm). There were no significant differences in mean age in all groups. These details are shown in Table 1

Table 1: Comparison of different categories in age, BMI, waist circumference, and wrist circumference among the three study groups.

Groups	Mean ± SD			
	Age (year)	BMI (kg/m^2)	Waist circumference (cm)	Wrist circumference (cm)
Control	29.75 ±4.04	24.92 ±0.58 b	77.62 ±1.97 b	17.37 ±0.34
Over weight-PCOS	24.12 ±0.72	28.02±3.81 ab	80.33 ±2.35 b	19.56 ±0.22
Obese PCOS	27.10 ±0.84	38.15 ±0.76 a	102.75 ±1.45 a	19.98 ±2.51
LSD value	5.490 NS	8.686 *	6.020 **	4.944 NS
P-value	0.0635	0.042	0.0001	0.613

Means having with the different letters in same column differed significantly.
* *(P≤0.05),* ** *(P≤0.01).* ±SD = standard deviation. LSD= least significant difference

Cholecystokinin value was significantly ($p < 0.05$) decreased in both overweight PCOS and obese PCOS

(19.93 ±3.23 ng/l, 14.37 ±3.64 ng/l, respectively), when compared to control (24.82 ±6.11 ng/l). Peptide YY value was decreased in the obese PCOS group (45.33±16.62 pg/ml) compared with the control group (45.33±16.62 pg/ml), as shown in Table 2

Table 2. Comparison of CCK and PYY levels among the three study groups

	Mean ± SD	
Groups	Cholecystokinin (ng/L)	Peptide YY (pg/ml)
Control	24.82 ±6.11 a	64.79±19.66
Over weight-PCOS	19.93 ±3.23 ab	51.64 ±21.52
Obese PCOS	14.37 ±3.64 b	45.33±16.62
LSD value	10.07 *	37.629 NS
P-value	0.047	0.659

Means having with the different letters in same column differed significantly.
* *(P≤0.05).* ±SD = standard deviation
LSD= least significant difference

In the current study, LH, FSH, and PRL levels indicated an increase in the obese PCOS group (7.85 ± 0.56 mIU/ml, 8.41 ± 0.38a mIU/ml, 20.48 ± 1.43 a ng/ml) compared to the control group (5.65 ± 0.50 mIU/ml, 6.96 ± 0.62a mIU/ml, 13.95 ± 1.33 b ng/ml), respectively. This result is shown in Table 3.

Table 3. Comparison between different groups in LH, FSH, and PRL levels.

	Mean ± SD		
Groups	LH (mIU/mol)	FSH (mIU/mol)	PRL (ng/ml)
Control	5.65 ±0.50	6.96 ±0.62a	13.95±1.33 b
Overweight-PCOS	6.83 ±1.10	7.75 ±0.37ab	17.14±1.03 ab
Obese PCOS	7.85 ±0.56	8.41 ±0.38a	20.48 ±1.43 a
LSD value	**2.581	**1.302	**4.132
P-value	0.043	0.048	0.010

Means having the different letters in the same column differed significantly. ** *(P≤0.01).* LSD: least significant difference. ± SD = standard deviation

Discussion

The study found that all women with female PCOS have the risk of developing metabolic disorders and their associated conditions, such as type 2 diabetes, hypertension, hyperlipidemia, and insulin resistance. BMI, wrist circumference, and waist circumference were high in the obese PCOS group. These findings were consistent with previous studies (11). Along with ovarian volume, follicle number also decreases with age; however, the decline in follicle number appears to be larger than the reduction in ovarian volume (11). There was a link between aging and a higher risk of metabolic disorders and insulin resistance. Therefore, these age-related alterations may have an influence on the reported incidence and consequences of PCOS. Women with PCOS may experience normal menstrual periods as they mature. Both aging ovaries and a decline in adrenal gland output over time can cause women's androgen production to decline. In women with PCOS, hyperandrogenism partly resolves before menopause (12). Insulin resistance, a typical PCOS symptom, can be caused by obesity, which raises insulin and androgen levels. This may worsen the symptoms of PCOS and further interfere with regular ovarian function (13).

CCK decreased in the obese PCOS group; this was agreed with (14). The physiology of reproduction may influence CCK satiation. During the follicular phase of the menstrual cycle, women naturally eat less food. During the periovulatory phase, they have reduced in daily consumption of food that is approximately 275 kcal/day less than the luteal-period maximum. Vagal afferent fibers mediate the satisfying effect of intraperitoneal injections of CCK. Paracrine activity is indicated by the fact that small intestine vagal afferents terminate within the crypt and villous lamina propria, but not in close proximity to enteroendocrine CCK cells. However, some vagal afferents end with 5 µm of CCK cells, and enteric glial cells suggest up to be the route by which CCK secretion (15).

PYY decreased in obese PCOS; this agreed with the published result (16). The impact of aging had been studied on PYY levels. Hunger and the emptying of the stomach are impacted by age. Because PYY may affect both of these, it is hypothesized that aging will have an effect on circulating PYY levels and nutritional responses (17). Suppresses gastric motility and secretes pancreatic hormones. Furthermore, it has been established that PYY is essential for preserving energy homeostasis. Nevertheless, there is conflicting evidence about the role of circulating PYY in to development of obesity in humans. The control of body weight is greatly influenced by appetite, which is a component of energy homeostasis (18).

LH was increased in PCOS, and this was in agreement with published results (19). The typical type of PCOS is known to be linked with abnormal gonadotropin production, particularly excessive LH secretion. An LH to FSH proportion greater than 2 was required for PCOS diagnosis. Higher mean LH concentrations, elevated LH bioactivity, and low to low-normal levels of FSH are all observed in women with PCOS (20). One factor that leads to early granulosa cell luteinization in PCOS is hyperinsulinemia. The following inhibition of cell proliferation and follicular expansion is the outcome of this in conjunction with increased ovarian androgen production. Additionally, progesterone becomes more predominant in PCOS-related tiny antral follicles and steroid synthesis (instead of estradiol). These intricate underlying processes result in stopping PCOS's pre-antral phase follicle growth (21). PCOS women often have excessive amounts of LH secretion; this is because high insulin and LH levels contribute to the high quantities of male hormones generated by the ovaries, such as testosterone (22).

PRL was increased in obese PCOS, a result deal by published data (23). According to reports, the amount of PRL in circulation determines how it affects insulin resistance and glucose metabolism. In clinical settings, PRL enhances glucose homeostasis by augmenting beta-cell mass in specific circumstances like pregnancy. When PRL passes across the blood-

brain barrier, it operates as a neuropeptide as well (24). It does this by modulating the hypothalamus, central functions, behavior, arousal, and sexuality. The biological evolution of PRL enabled it to regulate its secretion inside the hypothalamus, modifying the hypothalamic releasing and inhibitory factors that govern its secretions, in the absence of an endocrine target organ to offer feedback regulation (25). The ovarian cycle's typical neuroendocrine regulation may be interfered with by PRL. As such, it is among the several hormones that regulate follicle maturation, the progression of the luteal phase, the prevention of FSH-induced aromatization, and the alteration of GnRH release. In both male and female reproduction, an excess of PRL up to the point of hyperprolactinemia might cause problems. Hyperprolactinemia affects the generation of gonadal steroids and causes irregular menstruation by lowering GnRH and, in turn, LH secretion, pulse frequency, and amplitude. PRL increases insulin resistance, hyperphagia, leptin resistance, and hunger. Pathological hyperprolactinemia has been linked to obesity, poor glucose tolerance, and hyperinsulinemia. PRL increases insulin resistance, hyperphagia, leptin resistance, and hunger. Pathological hyperprolactinemia has been linked to obesity, poor glucose tolerance, and hyperinsulinemia (26). PRL level was negatively correlated with age and this result agreed with (27). The amount of visceral fat mass was correlated with the release of PRL. But another way, the larger visceral fat mass of obese women results in increased PRL secretion. PRL secretion may also be influenced by cortisol release, and it may rise in response to stress even in the absence of an increase in cortisol (28).

Limitation

Small sample size and limited references**.**

Conclusion

Low CCK and PYY in obese PCOS women lead to a rise in appetite, intake of food, an increase in waist circumference, and accumulation of fat in the abdomen. So, obesity is a risk factor in PCOS among the studied sample of Iraqi women.

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Authors' declaration

The authors declare no conflict of interest.

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 research, have been permitted to re-publicate the attached manuscript. The project was approved by the local ethical committee in the College of Science at the University of Baghdad for the study protocol (Ref.: CSEC/1123/0098). Before they participated in the trial, all patients and controls provided written informed consent.

Conflicts of Interest: None

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Authors' contributions

Study conception & design:(Sura F. Alsaffar). Literature search: (Sura F. Alsaffar). Data acquisition: (Mariam A. Malik). Data analysis & interpretation: (Sura F. Alsaffar). Manuscript preparation: (Mariam A. Malik). Manuscript editing & review: (Mariam A. Malik & Sura F. Alsaffar).

Reference

1. Shenta A, Saud K, Al-Shawi A. Assessment the correlations of hormones, lipid profiles, oxidative stress, and Zinc Concentration in Iraqi Women with Polycystic Ovary Syndrome. Reports of Biochemistry & Molecular Biology. 2020 Oct;9(3):270. https://doi.org/10.29252/rbmb.9.3.270

2. Nurain AS, Basuki SW, Kurniati YP, Sintowati R. The Relationship Between Obesity and Level of Polycystic Ovary Syndrome (PCOS): Literature Review. In International Conference on Health and Well-Being (ICHWB 2021) 2022; 218-226. https://doi.org10.2991/ahsr.k.220403.030.

3. Rehfeld JF. Measurement of cholecystokinin in plasma with reference to nutrition related obesity studies. Nutrition Research. 2020 Apr 1;76: 1-8. https://doi.org/10.1016/j.nutres.2020.01.003.

4. Woodward OR, Gribble FM, Reimann F, Lewis JE. Gut peptide regulation of food intake–evidence for the modulation of hedonic feeding. The Journal of physiology. 2022 Mar;600(5):1053-78. https://doi.org/10.1113/JP2805815.

5. Aukan MI, Coutinho S, Pedersen SA, Simpson MR, Martins C. Differences in gastrointestinal hormones and appetite ratings between individuals with and without obesity—A systematic review and meta-analysis. Obesity Reviews. 2023 Feb;24(2):13531. https://doi.org/10.1111/obr.13531

6. Mastnak L, Herman R, Ferjan S, Janež A, Jensterle M. Prolactin in polycystic ovary syndrome: metabolic effects and therapeutic prospects. Life. 2023 Oct 26;13(11):2124. https://doi.org/10.3390/life13112124.

7. Yang H, Lin J, Li H, Liu Z, Chen X, Chen Q. Prolactin is associated with insulin resistance and beta-cell dysfunction in infertile women with polycystic ovary syndrome. Frontiers in Endocrinology. 2021 Feb 25;12: 571229. https://doi.org/10.3389/fendo.2021.571229.

8. Mekki S, Mohammed NS. The Impact of COVID-19 Infection on Gonadal Hormonal Functions in Iraqi Women. Journal of the Faculty of Medicine Baghdad. 2023;65(4). https://doi.org/10.32007/jfacmedbagdad.1988.

9. Ibrahim SK, Alsaffar SF. Assessment of Monocyte Chemoattractant Protein-1 and Fertility Hormones in Iraqi Women with Polycystic Ovarian Syndrome. IHJPAS. 2024;37(1):86-93. https://doi.org/10.30526/37.1.3307.
10. Misra, A., & Dhurandhar, N. V. Current formula for calculating body mass index is applicable to Asian populations. Nutrition & diabetes. (2019);9(1), 3. https://doi.org/10.1038/s41387-018-0070-9.
11. Fadhil NM. Evaluation of serum Podocalyxin in Iraqi women with polycystic ovary syndrome. J Fac Med Baghdad. 2022;64(4):276-9. https://doi.org/10.32007/jfacmedbagdad.6441983.
12. Hsu MI. Changes in the PCOS phenotype with age. Steroids. 2013 Aug 1;78(8):761-6. https://doi.org/10.1016/j.steroids.2013.04.005.
13. Mohapatra I, Samantaray SR. BMI and Polycystic Ovary Syndrome: Demographic Trends in Weight and Health. Cureus. 2024 Mar;16(3). https://doi.org/10.7759/cureus.55439.
14. Geary N, Asarian L, Graf G, Gobbi S, Tobler PN, Rehfeld JF, Leeners B. Increased Meal Size but Reduced Meal-Stimulated Plasma Cholecystokinin Concentrations in Women with Obesity. Endocrinology. 2023 Jan 1;164(1): BQAC192. https://doi.org/10.1210/endocr/bqac192.
15. Steinert RE, Feinle-Bisset C, Asarian L, Horowitz M, Beglinger C, Geary N. Ghrelin, CCK, GLP-1, and PYY (3–36): secretory controls and physiological roles in eating and glycemia in health, obesity, and after RYGB. Physiological reviews. 2017 Jan;97(1):411-63. https://doi.org/10.1152/physrev.00031.2014.
16. Emanuel RH, Roberts J, Docherty PD, Lunt H, Campbell RE, Möller K. A review of the hormones involved in the endocrine dysfunctions of polycystic ovary syndrome and their interactions. Frontiers in Endocrinology. 2022 Nov 15;13: 1017468. https://doi.org/10.3389/fendo.2022.1017468.
17. Cooper JA. Factors affecting circulating levels of peptide YY in humans: a comprehensive review. Nutrition research reviews. 2014 Jun;27(1):186-97. https://doi.org/10.1017/S0954422414000109.
18. Imamura M. Effects of surgical manipulation of the intestine on peptide YY and its physiology. Peptides. 2002 Feb 1;23(2):403-7. https://doi.org/10.1016/S0196-9781(01)00618-0.
19. Batterham RL, Cowley MA, Small CJ, Herzog H, Cohen MA, Dakin CL, Wren AM, Brynes AE, Low MJ, Ghatei MA, Cone RD. Gut hormone PYY3-36 physiologically inhibits food intake. Nature. 2002 Aug 8;418(6898):650-4. https://doi.org/10.1038/nature00887.
20. Rasheed MK, Al-Naddawi AM. Association of Neuregulin-4 levels and body mass index with hyperandrogenism in Polycystic Ovary Syndrome patients. Journal of the Faculty of Medicine Baghdad. 2023;65(4). https://doi.org/10.32007/jfacmedbagdad.2140.
21. Paswan SK, Kumar R, Singh BK, Kumar D, Kumari S. To Study the Association Between Endocrinological and Metabolic Disorder in Case Both Obese and Non-Obese Polycystic Ovarian Syndrome. Int J Acad Med Pharm. 2023;5(5):512-6. https://doi.org/10.47009/jamp.2023.5.5.100.
22. Barber TM, Franks S. Obesity and polycystic ovary syndrome. Clinical endocrinology. 2021 Oct;95(4):531-41. https://doi.org/10.1111/cen.14421
23. Ali SI, Khaleel FM, Almukram AM. Association of ACE2, Insulin Resistance, and Other Parameters in Obese Polycystic Ovary Syndrome Patients Infected with COVID-19. Ibn AL-Haitham Journal For Pure and Applied Sciences. 2024 Jul 20;37(3):287-95. https://doi.org/10.32007/jfacmedbagdad.6421926.
24. Szlapinski, S. K., & Hill, D. J. Metabolic adaptations to pregnancy in healthy and gestational diabetic pregnancies: the pancreas-placenta axis. Current vascular pharmacology. 2021 19(2), 141-153. https://doi.org/10.2174/1570161118666200320111209
25. BA A, S. K., & P, S. M. (). General Endocrinology and Hormones of Hypothalamus and Pituitary. In Textbook of Veterinary Physiology. Singapore: Springer Nature Singapore. 2023 (pp. 369-390).https://doi.org/10.1007/978-981-19-9410-4_15
26. Ascar IF, Hameed AS. Serum prolactin, Preptin, CCL 18 and genetic polymorphisms in Iraqi women with polycystic ovary syndrome. Baghdad Science Journal. 2021 Dec 20;18(4 (Suppl.)):1552-. https://dx.doi.org/10.21123/bsj.2021.18.4(Suppl.).1552.
27. Tomatis V, Battipaglia C, Genazzani AD. Thyroid, Adrenal, PRL Impairments and Ovarian Function. Endocrines. 2021 Jul 27;2(3):212-25. https://doi.org/10.3390/endocrines2030021
28. Yang H, Di J, Pan J, Yu R, Teng Y, Cai Z, Deng X. The association between prolactin and metabolic parameters in PCOS women: a retrospective analysis. Front. Endocrinol. 2020 May 12; 11:263. https://doi.org/10.3389/fendo.2020.00263

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تقييم بعض هرمونات الأمعاء كوليسيستوكاينين وبيبتيدYY لدى النساء العراقيات المصابات بمتلازمة تكيس المبايض

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الخلاصة:
خلفية البحث: أكثر المشاكل الهرمونية والأيضية انتشارًا وتؤثر على النساء هي متلازمة تكيس المبايض، من خلال بعض الاعراض ومن أهمها: فرط الأندروجين والاضطرابات المرتبطة بالحيض، والتبويض، وصعوبات الحمل. تساهم السمنة بشكل كبير في حدوث متلازمة تكيس المبايض. تزيد مقاومة الأنسولين من فرص الإصابة بأمراض مزمنة لدى النساء المصابات بمتلازمة تكيس المبايض، بما في ذلك مرض السكري، وأمراض القلب والأوعية الدموية والأورام الخبيثة في بطانة الرحم والثدي.
الاهداف: دور هرمونات الأمعاء في الإصابة بمتلازمة تكيس المبايض.
طرق العمل: جمعت عينات لمصابات بمتلازمة تكيس المبايض اللاتي زرن مستشفى مدينة الطب في بغداد للاستفسار عن أسباب العقم لديهن. شملت الدراسة ستين مريضة مصابة بمتلازمة تكيس المبايض قسمت إلى مجموعتين بناءً على مؤشر كتلة الجسم: المجموعة الأولى ثلاثون مريضة بدينة والمجموعة الثانية ثلاثون مريضة ذوات الوزن الزائد وثلاثون امرأة سليمة، وكان متوسط أعمارهن بين 20 و35 عامًا. شخصت متلازمة تكيس المبايض لدى النساء باستخدام اثنين من معايير التشخيص الثلاثة: (تكيس المبايض، قلة التبويض أو انقطاعه، وفرط الأندروجين). استخدمت تقنية ال ELISA لتقييم مستوى هرمونات الأمعاء وايضا جهاز TOSOH AIA لتقييم مستوى هرمونات الخصوبة.
النتائج: الإناث البدينات المصابات بمتلازمة تكيس المبايض كان لديهن مستويات أعلى بشكل ملحوظ من محيط الخصر وهرمونات التكاثر ومستويات أقل من هرمونات الأمعاء.
الاستنتاجات: تنخفض مستويات هرمونات الامعاء عند النساء البدينات المصابات بمتلازمة تكيس المبايض، مما يسبب زيادة الشهية وتناول الطعام، وزيادة محيط الخصر، وتراكم الدهون في البطن، والسمنة عامل خطر لإصابة بمتلازمة تكيس المبايض.
الكلمات المفتاحية: كوليسيستوكاينين، الهرمون المنشط للجريبة، الهرمون المنشط للجسم الاصفر، البيتيد YY وهرمون الحليب.