

COMPREHENSIVE ANALYSIS OF INSULIN RESISTANCE AND SEX HORMONE LEVELS IN WOMEN WITH POLYCYSTIC OVARY SYNDROME: A CASE STUDY FROM AL-SHIRQAT CITY

Wijdan I. A. Abd-alwahab

Department of Biology/ College of Education/ University of Samarra, Iraq.
wijdan80@uosamarra.edu.iq

Abstract:

The purpose of this study was to determine whether insulin resistance in women with polycystic ovary syndrome (PCOS) is associated with the concentrations of particular sex steroids. A total of 60 women, 20 of them age matched healthy women [control], and 40 women diagnosed with PCOS were included, blood samples were collected from all the women. Sample collection was done from February through March 2024 when all of the participants were 28 to 47 years old. This diagnosis was confirmed on ultrasound examination and clinical examination by specialist physicians in Al Shirqat General Hospital.

The results showed a significant increase ($P \leq 0.05$) of follicle stimulating hormone (FSH) and luteinizing hormone (LH) and significant decrease ($P \leq 0.05$) of inhibin A and estrogen in the PCOS compared with the control group. On the basis of these results, the study concludes that insulin resistance could be a requirement for the development and manifestation of PCOS symptoms.

Keywords: Insulin resistance, diabetes mellitus, inhibin A and polycystic ovary syndrome (PCOS).

Introduction

Polycystic Ovarian Syndrome (PCOS) is a complex heterogeneous disorder that infected more than 7% of women of fertility age. This syndrome is responsible for more than 75% of infertility cases resulting from anovulation. The pathological cause of this syndrome is still unknown, but it's likely to be multifactorial, as genetics and environment are involved in its occurrence (McCartney and Marshall, 2016).

PCOS is characterized by a disorder in the ovulation process, which leads to infertility, delayed pregnancy, and obesity, often centered in the abdomen as a result of a disorder in lipid metabolism (dyslipidemia), and the appearance of hirsutism due to a disorder in testosterone, and that leads to the appearance of acne on the face with baldness in the front of the head (WHO, 2023).

Frederick Banting and Charles Herbert were the first to identify Insulin in 1921 (Munguia and Correa, 2020). It is secreted into the blood stream when the blood sugar levels



become elevated and it causes stimulation of glucose absorption by cells and reduction of blood sugar level (Ojha et al., 2019). The diabetes mellitus (hyperglycemia) is a disease characterized by the deficiency or depressed production of insulin (Thota et al., 2020). Research also showed that islet beta cells are reduced in type 2 diabetes, leading to lower insulin production and higher insulin resistance (Dave et al., 2020).

Insulin resistance refers to a pathological state of an inability of the normal actions of the insulin hormone produced from beta cells of the pancreases to carry out. Insulin resistance often times presents with normal insulin levels but the cells don't allow insulin into the cells (Kim and Park, 2017). Several risk factors have been associated with insulin resistance: excess body weight (Petersen, Shulman, 2018), genetic predisposition (Petersen, Shulman, 2018) and polycystic ovary syndrome (Kumari, Srivastava, 2013).

And steroid hormones are sex hormones like testosterone, progesterone, and estrogen, and they hit particular receptors. In addition, follicle stimulating hormone (FSH) and luteinizing hormone (LH) play a crucial role in sexual reproduction (Gurvich et al., 2018, Saadia, 2020).

MATERIAL AND METHODS

Study design:

Sixty women aged between 28 and 47 years were randomly divided into two distinct group. The 40 women in the first group had polycystic ovary syndrome (PCOS) and the second group—another 20 women—were healthy controls. was diagnosed their condition through medical and clinical examinations by specialist physicians at Al-Shirqat General Hospital, and the second group included (20) healthy women as a healthy control group. The sample collection period extended from the beginning of February 2024 until the end of March 2024.

Samples and Methods:

Blood, from all participants, was collected by venepuncture into test tubes, 5 ml blood sample. The serum was then separated by centrifuging the blood samples at 3000 rpm for 10minutes. The serum was then used for the study of the relevant parameters. Serum hormone concentrations were measured for all women with and without PCOS following the ELISA procedure with the kits provided by MyBioSource company-USA, and serum glucose was determined depending on the enzymatic colorimetric method by using a kit provided by **BIOLABO company-France.**

Bio-statistical Analysis:

Analysis of the data was performed using the SAS Statistical Program (2001). In pair comparison, a T test was adopted to compare means of the variables studied with a probability level of ($P \leq 0.05$) to determine statistical significance.

RESULTS AND DISSCUSION

As presented in Table 1, concentrations of insulin, glucose, and HOMA-IR increased significantly ($P \leq 0.05$) in the group of women with PCOS compared with control group.



Table 1: Concentrations of insulin, glucose, and HOMA-IR in PCOS patients and control groups.

Parameters	Insulin mIU/L	Glucose (FBS) mg/dL	HOMA-IR
Groups	Mean ± SD		
Control	7.23±1.45	92.00±11.03	1.6±0.3
PCOS	19.32±2.35 *	115.00±19.22 *	5.5±1.7 *
P-Value	0.0001	0.0001	0.0072

* Indicates to significant differences in significant level 0.05.

At the same time, the results presented in Table 2 show ($P \leq 0.05$) increased concentrations of FSH and LH in women with PCOS compared with control group. On the other hand, inhibin A and estrogen had significant decrease ($P \leq 0.05$) in the concentrations in the PCOS group compared to the control group.

Table 2: Concentrations of estrogen, inhibin-A, FSH, and LH hormones in PCOS patients and control groups.

Parameters	Estrogen pg/ml	Inhibin-A pg/ml	FSH mul/ml	LH mul/ml
Groups	Mean ± SE, n= 7			
Control	128.40±11.13	8.13±2.53	0.88±0.03	3.96±0.41
PCOS	107.00±14.25*	5.34±1.07 *	1.45±0.07 *	5.25±1.11 *
P-Value	0.0031	0.0001	0.0058	0.0001

* Indicates to significant differences in significant level 0.05.

Obesity, a family history of diabetes and PCOS, and others probably cause an increase in insulin and glucose levels which, in turn, result in insulin resistance (Wilcox, 2005). Furthermore, insulin resistance was found to be linked to several additional risk factors including overweight conditions, and the presence of polycystic ovary syndrome (Petersen and Shulman, 2018).

Insulin resistance occurs when the body becomes unable to use insulin effectively despite producing sufficient insulin, as a result of cells resisting the action of insulin, which leads to blood glucose levels rising above normal, thus, the body responds to this status by producing an additional amount of insulin, which leads to hyperinsulinemia (Diamanti-Kandarakis and Dunaif, 2012). Insulin resistance may be a risk factor for PCOS, as high insulin levels can lead to inflammation and complications associated with PCOS. Insulin resistance, which is associated with obesity in women, can also affect the function of the hypothalamus and pituitary gland in the brain, increasing the production of androgenic hormones that cause PCOS (Merviel et al., 2021).



The absence or deficiency of inhibin A may be used as an indication of ovarian pathogenesis which leading to infertility, as low concentration of inhibin hormone may cause to high and excessive FSH hormone (Haivadi et al., 2023). Also, high concentration and activity of LH may cause menstrual cycle disorders, and sometimes anovulation due to its effect on the formation of ovarian follicles (Pratama et al., 2024).

Furthermore, the increase in concentration of FSH maybe indicates to a decrease in the level of estrogen (E2), and any imbalance in the levels of LH and FSH hormones is reflected in the levels of progesterone and estrogen hormones, and thus causes fertility disorders of the ovaries, such as menstrual cycle disorders, weak fertility, and delayed childbearing (Conway, 2000).

CONCLUSION

The current research concludes that high levels of glucose and insulin (hyperinsulinemia) leading to insulin resistance, that may be an influential factor to sexual hormone disorders and subsequently to the incidence and appearance of symptoms of PCOS.

ACKNOWLEDGMENTS

The authors gratefully acknowledge the staff of the Al-Shirqat General Hospital for their technical and general support, and for patients who participated in the study and we wish for them good health.

REFERENCES

1. Conway,G. S. (2000). Premature ovarian failure. *Br Med Bull.* 2000;56(3):643-9. <https://doi.org/10.1258/0007142001903445>.
2. Dave,H. D. and Preuss,C. V. (2020). Human Insulin. In: *StatPearls. Treasure Island (FL): StatPearls Publishing.* Available from: <https://www.ncbi.nlm.nih.gov/books/NBK545190/>
3. Diamanti-Kandarakis,E. and Dunaif,A. (2012). Insulin resistance and the polycystic ovary syndrome revisited: an update on mechanisms and implications. *Endocr Rev.* 33(6):981-1030. <https://doi.org/10.1210/er.2011-1034>
4. Gurvich,C., Hoy,K., Thomas,N. and Kulkarni,J. (2018). Sex Differences and the Influence of Sex Hormones on Cognition through Adulthood and the Aging Process. *Brain sciences,* 8(9), 163. <https://doi.org/10.3390/brainsci8090163>
5. Haivadi,N. H., Jahanian,S. S., Razavinia,F., Younesi,S., Nasiri,M. and Ziaei,S. (2023). Effect of maternal polycystic ovary syndrome (PCOS) on screening of aneuploidy in the first and second trimesters. *J Ovarian Res.* 21;16(1):167. <https://doi.org/10.1186/s13048-023-01251-w>.
6. Kim,S. H. and Park,M. J. (2017). Effects of growth hormone on glucose metabolism and insulin resistance in humans. *Annals of pediatric endocrinology & metabolism,* 22(3), 145–152. <https://doi.org/10.6065/apem.2017.22.3.145>
7. McCartney,C. R. and Marshall,J. C. (2016). CLINICAL PRACTICE. Polycystic Ovary Syndrome. *N Engl J Med.* 7;375(1):54-64. <https://doi.org/10.1056/NEJMcp1514916>



8. Merviel,P., James,P. and Bouée,S. (2021). Impact of myo-inositol treatment in women with polycystic ovary syndrome in assisted reproductive technologies. *Reprod Health*. 18. <https://doi.org/10.1186/s12978-021-01073-3>
9. Munguia,C. and Correa,R. (2020). Regular Insulin. In: *StatPearls*. Treasure Island (FL): StatPearls Publishing. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK553094/>
10. Ojha,A., Ojha,U., Mohammed,R., Chandrashekar,A. and Ojha,H. (2019). Current perspective on the role of insulin and glucagon in the pathogenesis and treatment of type 2 diabetes mellitus. *Clinical pharmacology: advances and applications*, 11, 57–65. <https://doi.org/10.2147/CPAA.S202614>
11. Petersen,M. C. and Shulman,G. I. (2018). Mechanisms of Insulin Action and Insulin Resistance. *Physiological Reviews*, 98(4), 2133–2223. <https://doi.org/10.1152/physrev.00063.2017>
12. Pratama,G., Wiweko,B., Asmarinah., Widyahening,I. S., Andraini,T., Bayuaji,H. and Hestiantoro,A. (2024). Mechanism of elevated LH/FSH ratio in lean PCOS revisited: a path analysis. *Sci Rep* 14, 8229. <https://doi.org/10.1038/s41598-024-58064-0>
13. Saadia,Z. (2020). Follicle Stimulating Hormone (LH: FSH) Ratio in Polycystic Ovary Syndrome (PCOS) - Obese vs. Non- Obese Women. *Medical archives (Sarajevo, Bosnia, and Herzegovina)*, 74(4), 289–293. <https://doi.org/10.5455/medarh.2020.74.289-293>
14. Thota,S. and Akbar,A. (2020). Insulin. *StatPearls*. Treasure Island (FL): StatPearls Publishing. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK560688/>
15. Wilcox,G. (2005). Insulin and insulin resistance. *Clin Biochem Rev*. 26(2), 19-39. Available from:https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1204764/pdf/cbr26_2pg019.pdf
16. World Health Organization (WHO). (2023). Polycystic ovary syndrome. Annual Report. 28 June.

