

Study the relationship between polycystic ovary syndrome and insulin resistance in Nasiriya

Khairi, G. Alrikabi¹, Farooq L J Almohaisen² and Aamir M. Abed³

Email : amir.mm@shu.edu.iq 3

^{1,2}Medical Laboratory Technologies Dept., Shatrah Technical College, Southern Technical University, Iraq

³Physiology, Biochemistry and Pharmacology Dept., Veterinary Medicine College, University of Shatrah, Iraq

Abstract

This observational clinical study was performed by collection of data from unit of hormones and clinical immunology in Al-Habbobi hospital (Nasiriya city). 100 patients with poly cystic ovary (PCOS) were evaluated after a written informed consent was obtained from the center. Patients were labeled as having PCOS. All the examined women were classified into insulin resistant (IR) and non-insulin resistant(non-IR) and divided into three groups:), T1 (≤ 20 years), T2 (21-30 years) and T3 (> 30 years). A previous procedures were conducted at this center for all patients such as clinical presentation and ultrasound to detect PCOS, Homeostatic Model Assessment of Insulin Resistance (HOMA) was applied for insulin resistant diagnosis and estimation of the of some biomarkers of the participants to confirm the diagnosis of PCOS, which include Serological levels of testosterone, follicle stimulating hormone (FSH), luteinizing hormone (LH), prolactin. 20 samples were collected from healthy women and consider as control. Experiment extended from October 2024 to march 2025. Results reveal a notable relationship between IR and PCOS in patient women in Nasiriya considerable increase in levels of LH, testosterone were observed in patients with PCOS..

Key words : PCOS, Insulin Resistance, HOMA-IR, LH, FSH, Testosterone

المخلص

أجريت هذه الدراسة السريرية الرصدية من خلال جمع البيانات من وحدة الهرمونات والمناعة السريرية في مستشفى الحويبي (مدينة الناصرية). تم تقييم 100 مريضة تعاني من متلازمة تكيس المبايض (PCOS) بعد الحصول على موافقة مكتوبة من المركز. تم تصنيف المرضى على أنهم يعانون من متلازمة تكيس المبايض. جميع النساء المفحوصات تم تصنيفهن إلى مقاومات للإنسولين وغير مقاومات للإنسولين، وتم تقسيمهن إلى ثلاث مجموعات: = المعاملة الأولى (أقل أو تساوي 20 سنة)، المعاملة الثانية (20-30 سنة) والمعاملة الثالثة أكثر من 30 سنة. تم عمل الإجراءات السابقة في هذا المركز لجميع المرضى، مثل الفحص السريري واستخدام الموجات فوق الصوتية لاكتشاف تكيس المبايض، كما تم تطبيق نموذج التقييم المنزلي لمقاومة الإنسولين (HOMA) لتشخيص مقاومة الإنسولين وتقدير بعض المؤشرات الحيوية للمشاركات لتأكيد تشخيص متلازمة تكيس المبايض، والتي تشمل مستويات السيروتونين، الهرمون المنبه للجريب (FSH)، والهرمون اللوتيني (LH)، والبرولاكتين. تم جمع 20 عينة من نساء صحيحات واعتبرت كعينة ضابطة. امتدت فترة التجربة من أكتوبر 2024 إلى مارس 2025. أظهرت النتائج علاقة ملحوظة بين مقاومة الإنسولين ومتلازمة تكيس المبايض لدى النساء المرضي في الناصرية، حيث لوحظت زيادة كبيرة في مستويات LH والتستوستيرون لدى المريضات المصابات بـ PCOS

الكلمات المفتاحية : متلازمة تكيس المبايض، مقاومة الأنسولين، مؤشر HOMA-IR، هرمون LH، هرمون FSH، هرمون التستوستيرون.

INTRODUCTION

PCOS is a complex and varied state defined by tenacious ovulatory issues and elevated androgen levels. It attitudes as the most predominant endocrine complaint in women and the leading reason of irregular menstrual cycles during their reproductive years. Estimations propose that PCOS could influence

as many as one in four women of reproductive age (1). The significant hormonal imbalance in those with PCOS is an excess of androgens ,an issue often exacerbated by or connected to simultaneous situations like obesity and insulin resistance, which are commonly observed along with the disease (2).

In theory, PCOS happen due to uncommon

relationships among diverse behavioral, ecological, and genetic factors. The most commonly seen clinical representation of PCOS is: expansion of one or both ovaries. Within these ovaries, the theca cells become inflamed and hyperplastic, causing hypersecretion of androgens. This raised up androgenic output is primarily mediated by amplified activity of enzymes participated in the steroidogenesis pathway(3). Further than its reproductive implications, PCOS is related with significant long_term metabolic deviations and cardiovascular dangers. These include insulin resistance, dyslipidemia, and endothelial impairment. The condition is also characterized by distinctive neuroendocrine alterations, particularly raised serum luteinizing hormone(LH) levels(4). This hormonal imbalance is thought to originate from a dysfunction in the hypothalamic-pituitary-ovarian(or adrenal) axis. Specifically, a disturbance in the pulsatile secretion of gonadotropin_releasing hormone(GnRH) favoritisms an increased release of LH relative to follicle-stimulating hormone (FSH)(5). This neuroendocrine disruption is further disseminated by ovarian estrogen, which play role in an abnormal feedback mechanism that counterintuitively stimulates further LH secretion (6). In healthy women, the ratio of LH to FSH typically ranges between 1 and 2. In contrast, women with PCOS often exhibit a reversal of this ratio, which can become elevated to 2:1 or even 3:1, serving as a key differentiator in the condition's pathophysiology (7).

Insulin resistance(IR) and its resultant compensatory hyperinsulinemia(HI) are documented as central drivers in the pathophysiology of PCOS. Through various pathways, they play a critical role in the development of both hyperandrogenemia and reproductive dysfunction (8). The incidence of

IR and HI in the PCOS population is prominently high, affecting an estimated 65–95 % of women with the disorder. This comprises the vast majority of those who are obese or overweight, along with more than half of women who maintain a normal weight. Critically, while IR is a core feature of PCOS independent of body weight, its severity is often exacerbated by the presence of obesity (9).

The aim of the study is to assess the association between IR and hormonal changes in women with PCOS and evaluation of alterations in reproductive hormones across different age groups and compare patients with healthy controls.

MATERIALS AND METHODS

This observational clinical study was performed by collection of data from center of hormones and clinical immunology in Al-Habbobi hospital (Nasiriya city). 100 patients with PCOS were evaluated after a written informed consent was obtained from them. Prior procedures were conducted at this center for all patients through clinical presentation and ultrasound to detect PCOS, HOMA was applied for insulin resistant diagnosis and estimation of the of some biomarkers of the participants to confirm the diagnosis of PCOS, which include Serological levels of Androgen, follicle stimulating hormone (FSH), luteinizing hormone (LH), prolactin.

Patients diagnosed with PCOS were classified into two subgroups: those with insulin resistance (IR-PCOS) and those without insulin resistance (non-IR-PCOS). Participants were also divided into three age groups: Group A (≤ 20 years), Group B (21–30 years), and Group C (> 30 years). A control group consisting of twenty healthy women from Shatra was included for comparison. The study was conducted from October 2024 to March 2025.

3-2-2 Blood samples collection

The blood sampling from healthy women (control) was collected fasting from 8- 12 hours. 5 mL of venous blood samples were collected to obtain the serum by placing blood samples in a vacuum sterile glasses gel and clot activator tube and allowed to clot at 37 °C for 30 minutes before centrifugation. The tubes were centrifuged at 4000 rpm for 5 minutes, serum was collected and kept in freezer until it was used for chemical assays which include estimation levels of FSH, LH , TSH,

RESULTS :

testosterone and prolactin. Serum samples analysis was done with Ichroma III.

Statistical analysis

Statistical analysis was conducted using SPSS version 21 (Chicago, IL, USA). A one-way analysis of variance (ANOVA) was employed to compare the measured parameters across the different study groups. Differences were considered statistically significant at a p-value of less than 0.05

Table-1 Percentage of PCOS(IR and non IR) in patient of different ages(mean±SD)

	Group A (≤20 years)	Group B (21_30 years)	Group C (>30 years)	total
Total number of PCOS	32	48	20	100
Non-IR PCOS	24	29	14	67 %
IR-PCOS	8	19	6	33 %
IR-PCOS %	25 %	39.5%	30 %	

The prevalence PCOS in the researched group in Table (1) was 32% in Group A (≤ 20 years) , 48% In Group B (21-30 years) and 20%

in Group C (> 30 years) . Among the patients with polycystic ovary the occurrence of insulin resistance was higher in group B(39.5) followed group C (30%) and finally group A (25%).

Table-2 Levels of some hormones in healthy and PCOS patient (IR and NON-IR) (mean±SD)

	healthy	IR PCOS	NON-IR PCOS
FSH (IU/L)	4.8±1.52 b	6.84 ± 0.71 a	6.69 ± 1.3 a
LH (IU/L)	5.2±1.86 b	9.81 ± 2.07 a	9.49 ± 1.37 a
PRL (ng/ml)	10.98±2.04 a	11.50 ± 1.80 a	11.90± 1.21 a
TES. (ng/DL)	0.52±0.04 b	1.37 ± 0.09 a	1.03± 0.16 a
TSH (mIU/L)	1.54±0.28 a	1.64 ± 0.42 a	1.78 ± 0.32 a

Results in table 2 revealed that PCOS patient (IR and NON-IR) showed higher increment ($P < 0.05$) in the levels of FSH, LH and testosterone compared to healthy (6.84 ± 0.71 and 6.69 ± 1.3 vs. 4.8 ± 1.52), (9.81 ± 2.07 and

9.81 ± 2.07 vs. 5.2 ± 1.86) and (1.37 ± 0.09 and 1.03 ± 0.16 vs. 0.52 ± 0.04) for IR PCOS , NON-IR PCOS and healthy groups respectively. All groups have comparable levels of PRL and TSH.

Table-3 Levels of some hormones in IR PCOS patient of different ages(mean±SD)

	Group A (≤ 20 years)	Group B (21-30 years)	Group C (> 30 years)
FSH (IU/L)	6.94 ± 0.64 a	5.56 ± 0.70 a	5.03 ± 1.3 a
LH (IU/L)	8.58 ± 0.59 b	13.85 ± 1.56 a	7.00 ± 1.55 b
PRL (ng/ml)	11.20 ± 2.67 a	8.54 ± 2.75 a	14.76± 1.99 a
TES. (ng/DL)	1.37 ± 0.09 b	2.54 ± 0.18 a	1.03± 0.16 b
TSH (mIU/L)	2.16 ± 0.60 a	1.64 ± 0.42 a	1.78 ± 0.32 a

According to the results, in table 2 which is related to the PCOS patient that suffered from insulin resistance we noticed that the level of LH was increase significantly in group B as compared with group A and group C (13.85 ± 1.56 vs. 8.58 ± 0.59 and 7.00 ± 1.55 respectively). Testosterone followed the same

path of rising which was evident by observing high significant increase in group B as compared with group A and group C (2.54 ± 0.18 vs. 1.37 ± 0.09 and 1.03± 0.16 respectively). There are non-significant differences in the levels of FSH, PRL and TSH among groups

Table-4 Levels of some hormones in NON-IR PCOS of different ages (mean±SD)

	Group A (≤ 20 years)	Group B (21-30 years)	Group C (> 30 years)
FSH (IU/L)	5.68 ± 0.48 a	6.54 ± 0.76 a	6.85 ± 1.00 a
LH (IU/L)	12.23 ± 1.44 a	10.12 ± 0.62 ab	8.12 ± 1.55 b
PRL (ng/ml)	12.26 ± 2.48 a	9.64 ± 2.33 a	13.80± 1.78 a
TES. (ng/DL)	2.04 ± 0.48 a	1.87 ± 0.39 b	1.33± 0.24 b
TSH (mIU/L)	2.02 ± 0.54 a	1.86 ± 0.38 a	1.98 ± 0.44 a

Our finding in Table 4 group A exhibited greater ($P < 0.05$) elevation in the level of LH in group A compared to group C (12.23 ± 1.44 vs. 8.12 ± 1.55) and non-statical rise compared with group B (12.23 ± 1.44 vs. 10.12 ± 0.62). Levels of testosterone presented higher significance ($P < 0.05$) in group A compared to the group B and C (2.04 ± 0.48 vs. 1.87 ± 0.39 and 1.87 ± 0.39 respectively)

5- DISCUSSION

5-1 Percentage of PCOS (IR and non-IR) in patient of different ages

According to the results in our study the

high prevalence of PCOS was in age 21-30 years (39.5%) rather than other groups, age of 21-30 which consider the reproductive age of the women. Polycystic Ovary Syndrome (PCOS) is a widespread hormonal syndrome affecting individuals during their reproductive years, mainly between the ages of 20 and 30 because this period is distinguished by significant hormonal fluctuations, including elevated levels of androgens (male hormones) and insulin resistance, which can disrupt normal ovarian function. (10). Marriage usually occurs at age of 21-30 years. After marriage, the

motivation to maintain a slim figure often more prone to becoming overweight or obese. Also, the societal obligation theory posits that marriage increases the time and energy devoted to work and family responsibilities (11), thereby reducing the time available for weight management and increasing the likelihood of weight gain. Supporting this, results designate that marriage considerably raises the likelihood of being overweight by 6.5 % and the possibility of obesity by 2.8 %, consistent with both the societal obligation theory and marriage market theory(12). While many researchers have discovered the causes of obesity and overweight among Chinese residents , their attention has largely been on influences such as dietary behaviors, the fast-food industry, and exercise behavior(13). By definitely investigating the effect of marriage on exercise time, the present study identifies an imperative mediating mechanism: marriage leads to a significant reduction in physical activity. This outcome aligns with previous research conducted in the United States(14), signifying that marriage crowds out time for exercise , at last contributing to weight gain, a crucial risk reason for the progress and exacerbation of PCOS. This result is agree with (15). The result of the PCOS study group is similar when compared to (16) who stated that most of the infertile PCOS women were aged 21–25 years (58 %) with a average age of 25 years.

Prevalence of insulin resistance in PCOS patient in our study was relatively high which indicate there was close relationship between insulin resistance and polycystic ovary. This result was in accordance with other researchers.

Patients with PCOS, both obese and thin, frequently have insulin resistance. It affects 30–75% of PCOS individuals who are thin and 70–95% of obese PCOS patients (10). High insulin levels are a major contributor to PCOS and not just one of its manifestations. Elevated insulin

diminishes, making married women levels can inhibit normal ovulation and stimulate the ovaries to increase testosterone production (17). PCOS is acknowledged as a risk factor for developing diabetes (18). Although the characteristics of insulin resistance manifest before those of PCOS, it is believed that insulin resistance may contribute to the development of PCOS. as opposed to the reverse elevated insulin levels may make inflammation and other metabolic issues related to PCOS worse. Most importantly, not all people with insulin resistance develop PCOS; some women having insulin resistance do not. Some specialists assert that the brain's pituitary and hypothalamus are affected by obesity related insulin resistance, which increases the synthesis of androgenic hormones that cause PCOS (19). Unrelated to PCOS, excessive androgenic hormone secretion raises the likelihood of female infertility and ovarian disorders in women. Depression is linked to each of these ailments, but when they combine, the risk of depression soars. Corresponding to this, there is a correlation between insulin resistance and infertility rather than the reverse. Increased insulin levels might cause inflammation and other metabolic issues related to PCOS worsen. in our study there's relative increase in the level of LH to FSH in IR-PCOS group. in our study there's relative increase in the level of LH to FSH in IR-PCOS group. Insulin exerts significant effects on the hypothalamic-pituitary axis by increasing both the frequency and amplitude of gonadotropin-releasing hormone(GnRH) secretion, which sequentially leads to raised luteinizing hormone (LH) release. Ovarian estrogen contributes to this disruption by creating an abnormal feedback mechanism that further stimulates LH secretion (20). The resultant elevation in LH enhances androgen biosynthesis within the ovaries, ultimately impairing normal ovarian

function. Beyond its peripheral effects, insulin signaling within the central nervous system plays a serious role in regulating reproductive function and maintaining body weight homeostasis. (21).

In this cross sectional study of 100 women , we detected that the clinical and biochemical features of PCOS evolve with age. While abnormal function of ovary and hyperandrogenism are the predominant apprehensions in younger women, metabolic disorders become increasingly prominent as PCOS women grow older. The progressive elevation of testosterone observed in insulin-resistant PCOS (IR-PCOS) with advancing age may result from both increased androgen production and a concurrent decline in sex hormone-binding globulin (SHBG) levels.

These findings align with previous studies, particularly noting the oppressive effect of insulin on hepatic SHBG synthesis. SHBG is a glycoprotein produced in the liver that binds sex hormones—including androgens and estrogens—thereby regulating their bioavailability to target tissues. In women with PCOS, SHBG levels are typically reduced by approximately 50% compared to normal, leading to a corresponding increase in free testosterone (15). Accordingly, SHBG was found to be inversely related to insulin levels in the present study. However, after correcting for BMI , this connection was no longer statistically significant, suggesting that body mass exerts a stronger confounding influence on both SHBG production and insulin secretion. Furthermore, age emerged as a significant independent interpreter of insulin levels among women with PCOS. This finding is consistent with studies recording a relative insulin secretory defect(22), reduced incretin sensitivity of pancreatic beta-cells (23), and decreased insulin sensitivity in muscle tissue, accompanied by reduced expression of skeletal muscle glucose

transporter-4(SGLT_4) (24)

According to our results there was elevation in the level of LH in group A(≤ 20 years) . Dysfunction of the hypothalamic-pituitary-ovarian(or adrenal) axis has been implicated in the pathophysiology of polycystic ovary syndrome. A disruption in the pulsatile secretion of gonadotropin-releasing hormone(GnRH) leads to a relative increase in LH release compared to follicle-stimulating hormone (FSH) (25). This neuroendocrine disturbance is further exacerbated by ovarian estrogen, which contributes to an abnormal feedback mechanism that paradoxically stimulates additional LH secretion (26).

In healthy women, the LH-to-FSH ratio typically ranges between 1 and 2. In contrast, women with PCOS often exhibit a reversal of this ratio, which may increase to 2:1 or even 3:1 (27). As a direct consequence of this elevated LH/FSH ratio, normal ovulation fails to occur in patients with polycystic ovary syndrome (28). Testosterone levels were found to be negatively associated with age. Despite some controversies in the literature, the majority of evidence supports the notion that androgen production significantly declines as women grow older (29). This reduction is largely attributed to a decrease in both the number and function of theca cells over time in women with PCOS [(30).

In contrast to the findings of some other authors (31), our results align with those of a recently published large-scale Brazilian study involving 796 women with PCOS (32). We detected a significant decline in circulating testosterone levels in PCOS women as early as under the age of 20.

Conclusion

The highest incidence of polycystic ovary was found at the age of (21-30 years) 39.5% of women with polycystic ovary in Nasiriya have insulin resistance. High levels of testosterone,

LH and FSH in PCOS groups (IR and NON-IR) compared to healthy women. The highest levels of LH and testosterone in an IR (PCOS) are found at the age of 21-30 years. The highest levels of LH and testosterone in NON-IR (PCOS) are found at the age less than 20 years. Non-significant differences in the level of prolactin and TSH among different groups studied.

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