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Original article

Elevated Serum Ferritin and Insulin Resistance in Libyan Women with Polycystic Ovary Syndrome: A Retrospective Case-Control Study

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Polycystic Ovary Syndrome (PCOS), Insulin Resistance, Serum Ferritin, Metabolic Dysfunction, Libyan Women.

A B S T R A C T

Polycystic Ovary Syndrome (PCOS) is a complex endocrine disorder associated with metabolic and hormonal disturbances, including insulin resistance (IR) and altered iron metabolism. This retrospective case-control study aimed to evaluate serum ferritin levels and insulin resistance in 196 Libyan women (98 PCOS cases and 98 controls) attending the Tripoli Fertility Center. Anthropometric, biochemical, and hormonal parameters were assessed, including body mass index (BMI), waist circumference (WC), lipid profile, and hormonal levels. Results revealed significantly higher BMI, WC, and IR (HOMA-IR) in the PCOS group compared to controls ($p < 0.001$). Elevated ferritin levels were also observed in PCOS patients ($p = 0.001$), with a weak but significant correlation between ferritin and IR ($r = 0.104$, $p = 0.01$). The PCOS group exhibited a more atherogenic lipid profile, with lower HDL-cholesterol ($p < 0.0001$) and higher LDL-cholesterol ($p < 0.0001$). Hormonal analysis showed elevated luteinizing hormone (LH) and a reduced FSH/LH ratio ($p < 0.0001$), consistent with PCOS pathophysiology. intricate relationship between metabolic dysfunction, hormonal imbalances, and altered iron metabolism in women with PCOS.

Introduction

Polycystic Ovary Syndrome (PCOS) is a complex endocrine disorder affecting 6–20% of women of reproductive age worldwide. It is characterized by chronic anovulation, hyperandrogenism, hyperinsulinemia, and insulin resistance (IR) [1]. The pathophysiology of PCOS involves a combination of endocrine, metabolic, genetic, and environmental factors [2]. Insulin resistance, present in 65–95% of women with PCOS, plays a central role in hyperandrogenism by increasing ovarian androgen secretion and reducing hepatic sex hormone-binding globulin (SHBG) production [1, 3]. Hyperinsulinemia further exacerbates hyperandrogenism by disrupting gonadotropin levels, leading to elevated androgen production [4]. Importantly, IR is independent of obesity but is worsened by it, and it is closely associated with metabolic disturbances such as dyslipidemia, impaired glucose tolerance, and an increased risk of type 2 diabetes mellitus [1, 3].

Serum ferritin, a marker of iron storage and inflammation, is elevated in women with PCOS and is linked to metabolic dysfunction [5]. Elevated ferritin levels correlate with fasting glucose, serum insulin, and triglyceride levels, suggesting a role in insulin resistance and metabolic dysregulation [5]. The increase in ferritin levels is driven by oxidative stress and chronic inflammation, both of which are prevalent in PCOS, and may exacerbate IR through mechanisms involving hepatic dysfunction and systemic inflammation [6–10]. In Libya, cultural, dietary, and genetic factors may uniquely influence iron metabolism and its relationship with IR in women with PCOS [11]. A 2024 study by Ben-Issa et al. highlighted the role of dietary management in addressing PCOS symptoms and metabolic outcomes among Libyan women attending the Tripoli Infertility Center [12]. The current study aimed to evaluate serum ferritin levels and the insulin resistance index, measured using the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR), in both study groups to explore the association between serum ferritin levels and insulin resistance in Libyan women with PCOS.

Methods

A retrospective case-control study included 196 Libyan women (98 cases and 98 controls) who attended the outpatient department of obstetrics and gynecology at the Tripoli Fertility Center, Tripoli, Libya, between 2012 and 2023. All participants were diagnosed with PCOS according to the 2003 Rotterdam Diagnostic

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Criteria [1]. Participants were included if they were aged 20–49 years, diagnosed with PCOS based on the 2003 Rotterdam criteria, and attended the PCOS clinic of the infertility outpatient department (OPD). Exclusion criteria included a recent history of blood transfusion or iron supplementation, heavy menstrual bleeding, chronic inflammatory conditions (e.g., diabetes, asthma, tuberculosis), or anemia (hemoglobin <11 g/dL) [2].

A structured data collection form was used to record detailed information, including medical history, physical examination findings, hormonal levels, and anthropometric measurements such as body mass index (BMI), height, weight, and waist circumference. PCOS diagnosis was based on the 2003 Rotterdam criteria, requiring at least two of the following: menstrual irregularities (amenorrhea: absence of menstrual cycles for >6 months; oligomenorrhea: menstrual cycles lasting >35 days to 6 months) [3], clinical and/or biochemical hyperandrogenism (e.g., acne, oily skin, hirsutism), or polycystic ovaries on ultrasonography.

Anthropometric measurements included BMI, calculated as weight (kg) divided by height (m^2), with overweight defined as BMI >25 kg/m^2 and obesity as BMI >30 kg/m^2, according to World Health Organization (WHO) criteria [13]. Waist circumference was measured at the level of the umbilicus, with <88 cm considered normal and ≥88 cm classified as abnormal [14]. Pelvic ultrasound was performed to assess ovarian morphology and identify polycystic ovaries [4].

Laboratory investigations were conducted during the early follicular phase (day 2 of menstruation) in a fasting state, including complete blood count (CBC), thyroid-stimulating hormone (TSH), serum prolactin, follicle-stimulating hormone (FSH), luteinizing hormone (LH), testosterone, serum ferritin levels, and the insulin resistance index (HOMA-IR). The study adhered to ethical guidelines, with informed consent obtained and approval from the institutional review board of the Tripoli Fertility Center.

Quantitative demographic and clinical data with normal distribution were expressed as the mean ± standard deviation and analyzed by t-test, Pearson correlation, $P < 0.05$ was considered to indicate statistical significance. Statistical Analysis was performed using IBM SPSS Statistics 25.0.

Results

Demographic data

This study was performed on 196 infertile women divided into two groups: 98 women with and 98 women without the criteria of PCOS. The two groups were compared concerning age, BMI, and serum hormonal levels. The mean age was found to be 32.54±6.22 years in the case group while 34.43±6.2 with a range from 20 to 49 years in both groups. The age distribution of the study population is illustrated in Figure 1.

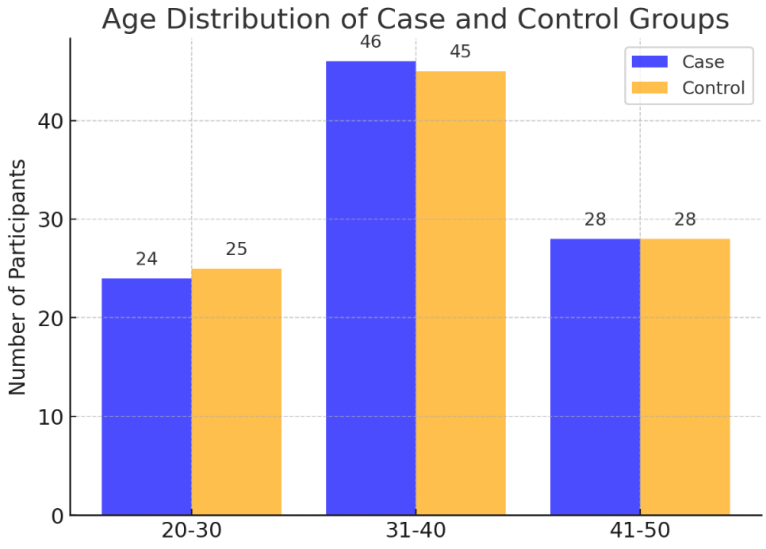

Figure 1 Age distribution of study population.

Regarding BMI, the present study showed that The Case group had a significantly higher prevalence of morbid obesity (71.42% vs. 1.02%) and abnormal WC (>88 cm: 61.22% vs. 34.69%) compared to the Control group. In contrast, the Control group had a higher proportion of participants with a normal BMI (84.69% vs. 4.08%) and normal WC (<88 cm: 65.30% vs. 38.77%). As demonstrated in table 1.

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Table 1 Demographic profile of PCOS patients.

Variable	Case N(%)	Control N(%)
Body Mass Index		
<18.5 (underweight)	1(1.02)	12(12.24)
18.5-24.9 (normal)	4(4.08)	83(84.69)
25-29.9(overweight)	6(6.12)	1(1.02)
30-34.9(obese)	17(17.34)	1(1.02)
>35(morbid obese)	70(71.42)	1 (1.02)
Waist circumference (WC)		
<88cm	38(38.77)	64(65.30)
>88cm	60(61.22)	34(34.69)

The present study compared anthropometric measures, including weight, waist circumference, waist-to-hip ratio (WHR), and body mass index (BMI), between women with polycystic ovary syndrome (PCOS) and healthy controls using paired T-tests. The results revealed significant differences in weight, waist circumference, and BMI between the two groups. The Case group had a significantly higher mean weight (94.4562 kg vs. 80.9418 kg, $p < 0.001$), larger waist circumference (93.1531 cm vs. 80.1224 cm, $p < 0.001$), and higher BMI (36.9221 kg/m² vs. 19.9204 kg/m², $p < 0.001$) compared to the Control group. However, there was no significant difference in waist-to-hip ratio (WHR) between the groups ($p = 0.818$), as shown in table 2.

Table 2 Comparison of Anthropometric Measures Between PCOS Cases and Controls

Variable	Group	N	Mean	Std. Deviation	Mean Difference	95% CI	p-value
Weight (kg)	Case	98	94.4562	19.53556	13.51439	8.65688 to 18.37189	<0.001*
	Control	98	80.9418	14.58833			
Waist (cm)	Case	98	93.1531	12.93310	13.03061	9.21178 to 16.84945	<0.001*
	Control	98	80.1224	14.14743			
Waist Circumference	Case	98	0.8037	0.12352	0.00357	-0.02705 to 0.03419	0.818
	Control	98	0.8001	0.09149			
BMI (kg/m²)	Case	98	36.9221	6.40440	17.00173	15.58072 to 18.42274	<0.001*
	Control	98	19.9204	3.10021			

** Significant p- value (< 0.05).*

The PCOS group exhibited significantly higher insulin resistance (HOMA-IR) ($p < 0.0001$), consistent with the metabolic dysfunction commonly associated with PCOS. In terms of lipid profile, the PCOS group had significantly lower HDL-cholesterol ($p < 0.0001$) and higher LDL-cholesterol ($p < 0.0001$), indicating a more atherogenic lipid profile, although no significant differences were observed in total cholesterol ($p = 0.161$) or triglycerides ($p = 0.117$). Additionally, the PCOS group showed significantly higher levels of iron ($p < 0.00001$) and ferritin ($p = 0.001$), suggesting altered iron metabolism. Hormonal profiles also differed significantly, with the PCOS group demonstrating higher LH levels ($p < 0.00001$) and a lower FSH/LH ratio ($p < 0.0001$), consistent with the characteristic hormonal imbalance of PCOS. However, no significant differences were found in FSH ($p = 0.139$), estradiol (E2) ($p = 0.135$), prolactin ($p = 0.081$), or TSH ($p = 0.626$) using paired t test. as demonstrated in table 3.

Table 3. Laboratory Parameters in PCOS cases and controls

Laboratory parameters	Case	Control	p-value
IR HOMA	3.78±0.90	1.61±0.65	0.0001*
FBS	101.77±24.76	97.32±26.06	0.222
HbA1c	5.64±1.15	5.40±0.77	0.128
Total cholesterol (mg/dl)	177.51±332.40	.170.74±33.88	0.161
HDL-cholesterol (mg/dl)	53.07±25.16	60.16±15.84	0.0001*
LDL-cholesterol (mg/dl)	115.61±45.06	60.34±41.71	0.0001*
TG (mg/dl)	129.75±62.50	116.14±85.38	0.117
Ferritin (ng/ml)	52.42±35.06	37.92±23.75	0.001*
Iron	72.05±37.58	8.01±5.08	0.00001*

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FSH (IU/L)	7.52±5.23	6.52±5.49	0.139
LH(IU/L)	10.52± 6.74	3.81±2.41	0.00001*
FSH/LH ratio	1.97± 0.93	1.048± 2.05	0.0001*
Estradiol E2	55.39±78.65	42.72±28.04	0.135
Prolactin (ng/ml)	24.09±39.36	16.77±10.01	0.081
TSH (uIU/ml)	2.45±2.49	2.31±1.47	0.626

** Significant p- value (< 0.05).*

The present study reveals a strong positive correlation between insulin resistance (IR) and the grouping variable (case-control status) (r = 0.810, p < 0.01), indicating significant differences in IR levels between the PCOS and control groups. Additionally, a moderate positive correlation was observed between case-control status and ferritin levels (r = 0.236, p < 0.01), suggesting variations in iron storage between the two groups. However, the correlation between IR and ferritin levels was found to be weak and statistically significant (r = 0.104, p = 0.01), suggesting a slight tendency for higher ferritin levels to be associated with higher IR. As demonstrated in figure (2,3,4).

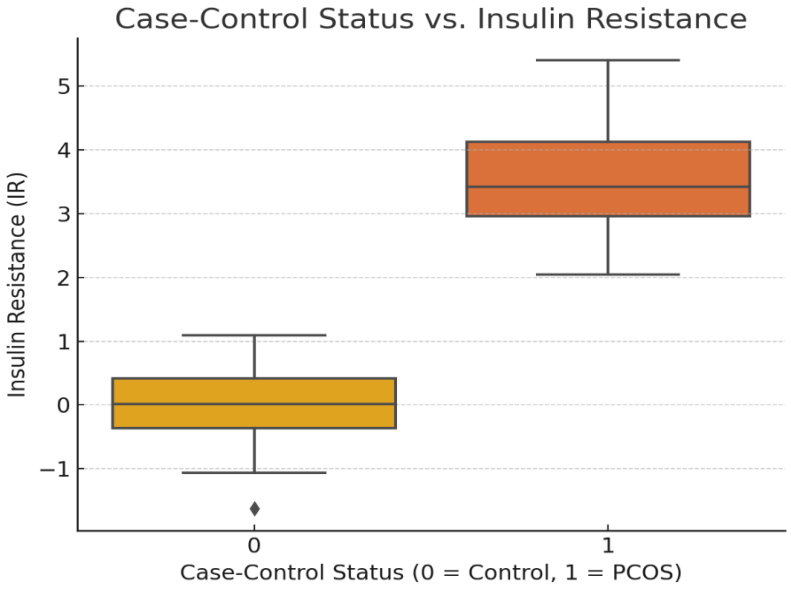

Figure 2 A box plot showing the difference in insulin resistance between the PCOS and control groups

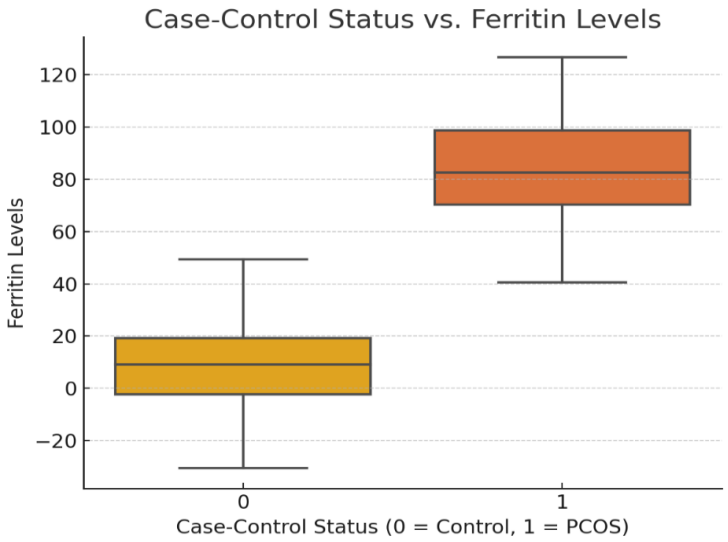

Figure 3. A box plot displaying the variation in ferritin levels between the two groups.

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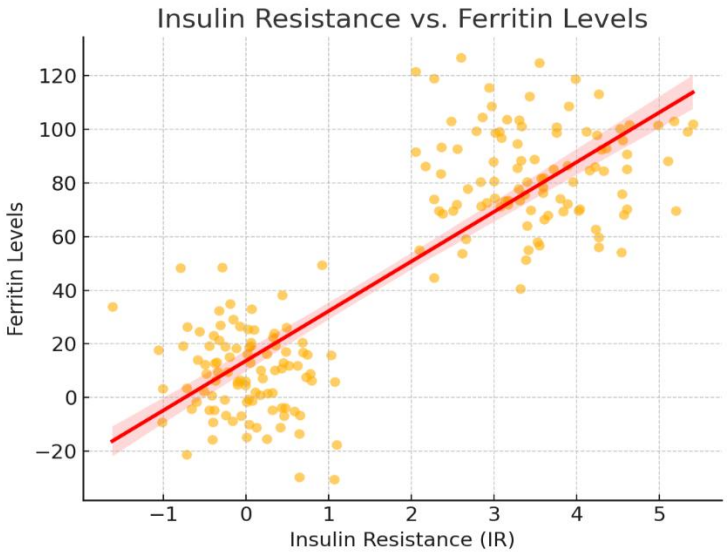

Figure 4. A scatter plot illustrating the correlation between insulin resistance and ferritin levels.

Discussion

The present study highlights significant differences in metabolic, anthropometric, and biochemical parameters between women with PCOS and healthy controls. A strong positive correlation was observed between insulin resistance (IR) and PCOS status ($r = 0.810$, $p < 0.01$), consistent with prior research indicating that IR is a central feature of PCOS, irrespective of obesity status [1,4]. Similar to our findings, a study by Dunaif et al. (2008) reported a significantly higher HOMA-IR in PCOS women compared to controls, reinforcing the notion that IR is intrinsic to PCOS pathophysiology [3]. The higher IR levels in the PCOS group may contribute to the increased metabolic risks associated with the syndrome, including type 2 diabetes and cardiovascular disease [4].

In addition to IR, our study identified a moderate positive correlation between case-control status and ferritin levels ($r = 0.236$, $p < 0.01$). Elevated ferritin levels in PCOS patients may reflect altered iron metabolism, a finding corroborated by previous studies [5, 6]. Excess iron accumulation has been suggested as a potential contributor to oxidative stress and chronic inflammation in PCOS [7, 8]. Furthermore, the weak but significant correlation between ferritin and IR ($r = 0.104$, $p = 0.01$) suggests that while increased iron stores may be associated with insulin resistance, other factors such as chronic inflammation and hormonal imbalances might mediate this relationship [9, 10].

Anthropometric measures in our study demonstrated significantly higher BMI, waist circumference (WC), and weight in the PCOS group compared to controls. These findings align with previous research indicating that PCOS is frequently associated with obesity and central adiposity [11, 12]. Obesity exacerbates IR, contributing to the metabolic dysfunction seen in PCOS [13]. However, our study found no significant difference in waist-to-hip ratio (WHR) between the two groups ($p = 0.818$), suggesting that while abdominal obesity is more prevalent in PCOS, the distribution of fat between the waist and hips may not differ significantly [14].

Lipid profile analysis revealed significantly lower HDL-cholesterol ($p < 0.0001$) and higher LDL-cholesterol ($p < 0.0001$) in the PCOS group, consistent with previous studies [15]. The dyslipidemia observed in PCOS is likely a consequence of both IR and androgen excess, which impair lipid metabolism [16]. However, total cholesterol and triglycerides did not show significant differences between groups ($p = 0.161$ and $p = 0.117$, respectively), similar to findings reported by Wild et al. (2011), who suggested that while PCOS is characterized by a more atherogenic lipid profile, total cholesterol levels may not always be significantly altered [17].

The hormonal profile in our study confirmed characteristic alterations in PCOS, including significantly higher luteinizing hormone (LH) levels ($p < 0.00001$) and a lower FSH/LH ratio ($p < 0.0001$). These findings are consistent with the classic endocrine features of PCOS, wherein elevated LH secretion contributes to hyperandrogenism and anovulation [18, 19]. However, no significant differences were observed in follicle-stimulating hormone (FSH) ($p = 0.139$), estradiol ($p = 0.135$), prolactin ($p = 0.081$), or thyroid-stimulating hormone (TSH) ($p = 0.626$), which is in agreement with prior studies reporting normal levels of these hormones in PCOS [20, 21].

Overall, our findings align with existing literature on PCOS and reinforce the complex interplay between metabolic dysfunction, hormonal imbalance, and altered iron metabolism in affected women. Future studies with larger sample sizes and longitudinal follow-up are warranted to explore the causative mechanisms

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underlying these associations and their clinical implications. Additionally, population-specific studies, such as those conducted on Libyan women with PCOS [12, 22], can provide insights into genetic and environmental influences on PCOS pathophysiology.

Conclusion

The present study reveals a complex link between metabolic dysfunction, hormonal imbalances, and altered iron metabolism in women with PCOS. Elevated insulin resistance and serum ferritin levels are linked to PCOS, indicating iron dysregulation may exacerbate metabolic complications. The study also highlights the atherogenic lipid profile and hormonal imbalances, emphasizing the need for comprehensive management strategies.

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Conflicts of Interest

The authors declare no conflicts of interest.

References

1. Azziz R, Carmina E, Chen Z, Dunaif A, Laven JSE, Legro RS, et al. Polycystic ovary syndrome. Nat Rev Dis Primers. 2016;2:16057.
2. Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. Fertil Steril. 2004;81(1):19-25.
3. Dunaif A. Insulin resistance and the polycystic ovary syndrome: Mechanism and implications for pathogenesis. Endocr Rev. 1997;18(6):774-800.
4. Witchel SF, Oberfield SE, Peña AS. Polycystic ovary syndrome: Pathophysiology, presentation, and treatment with emphasis on adolescent girls. J Endocr Soc. 2019;3(8):1545-1573.
5. Begum N, Sultana N, Akhter QS, Begum S, Ferdousi S. Serum ferritin as a marker of insulin resistance in polycystic ovary syndrome: A meta-analysis. J Clin Endocrinol Metab. 2021;106(12):e4871-e4882.
6. Luque-Ramírez M, Álvarez-Blasco F, Botella-Carretero JI, Sanchón R, San Millán JL, Escobar-Morreale HF. Increased body iron stores of obese women with polycystic ovary syndrome are a consequence of insulin resistance and hyperinsulinism. J Clin Endocrinol Metab. 2009;94(1):26-32.
7. Kowalska K, Milaszewska P, Nikołajuk A, et al. Iron metabolism in polycystic ovary syndrome: A systematic review and meta-analysis. Nutrients. 2018;10(11):1664.
8. Fernández-Real JM, López-Bermejo A, Ricart W. Cross-talk between iron metabolism and diabetes. Diabetes. 2002;51(8):2348-2354.
9. González F, Rote NS, Minium J, Kirwan JP. Reactive oxygen species-induced oxidative stress in the development of insulin resistance and hyperandrogenism in polycystic ovary syndrome. J Clin Endocrinol Metab. 2006;91(1):336-340.
10. Li X, He T, Yu K, Lu Q, Alkasir R, Guo Y, et al. Markers of iron status in Chinese women with polycystic ovary syndrome and insulin resistance. Int J Clin Exp Med. 2020;8(7):11330-11335.
11. Carmina E, Lobo RA. Polycystic ovary syndrome (PCOS): Arguably the most common endocrinopathy is associated with significant morbidity in women. J Clin Endocrinol Metab. 1999;84(6):1897-1899.
12. Ben-Issa NA, Abofaed M, Alqeeyadi H, Alshahrani M, Alsherif E. Prevalence and Dietary Management of Polycystic Ovary Syndrome among Libyan Women Attending Tripoli Infertility Center. Alq J Med App Sci. 2024;7(1):74-81.
13. Lim SS, Davies MJ, Norman RJ, Moran LJ. Overweight, obesity and central obesity in women with polycystic ovary syndrome: A systematic review and meta-analysis. Hum Reprod Update. 2012;18(6):618-637.
14. Nielsen J, Alfirevic Z. Waist circumference as a measure of central obesity: Practical guidelines. BJOG. 2006;113(10):1117-1124.
15. Sam S, Dunaif A. Polycystic ovary syndrome: Syndrome XX? Trends Endocrinol Metab. 2003;14(8):365-370.
16. Moran LJ, Misso ML, Wild RA, Norman RJ. Impaired glucose tolerance, type 2 diabetes and metabolic syndrome in polycystic ovary syndrome: A systematic review and meta-analysis. Hum Reprod Update. 2010;16(4):347-363.
17. Wild RA, Carmina E, Diamanti-Kandarakis E, et al. Assessment of cardiovascular risk and prevention of cardiovascular disease in women with the polycystic ovary syndrome: A consensus statement by the Androgen Excess and Polycystic Ovary Syndrome (AE-PCOS) Society. J Clin Endocrinol Metab. 2010;95(5):2038-2049.
18. Azziz R, Sanchez LA, Knochenhauer ES, et al. Androgen excess in women: Experience with over 1000 consecutive patients. J Clin Endocrinol Metab. 2004;89(2):453-462.
19. Yao X, Chen X, Wu Y, Liu Y, Zhang X. Pathogenesis of polycystic ovary syndrome: A review. J Endocrinol Invest. 2017;40(11):1137-1149.
20. Kumar P, Arora S. Hormonal profile in polycystic ovary syndrome: A clinical study. J Obstet Gynaecol India. 2018;68(1):58-63.

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Libyan Medical Journal
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21. Kim CH, Kim HK, Bae SJ, Park JY, Lee KU. Association of elevated serum ferritin concentration with insulin resistance and impaired glucose metabolism in Korean men and women. Metabolism. 2011;60(3):414-420.
22. El-Mezughi A, Al-Sharbatti S, Moussa M. Consanguinity and its correlates in the Libyan population. J Biosoc Sci. 2015;47(5):669-681.

المستخلص

متلازمة تكيس المبايض اضطراب غددي معقد يرتبط باضطرابات أيضية وهرمونية، بما في ذلك مقاومة الأنسولين وتغير أيض الحديد. هدفت هذه الدراسة الاستعادية القائمة على المقارنة بين الحالات إلى تقييم مستويات الفيريتين في المصل ومقاومة الأنسولين لدى 196 امرأة ليبية (98 حالة من متلازمة تكيس المبايض و98 حالة من مجموعة الضبط) راجعت مركز طرابلس للخصوبة. تم تقييم المعايير الأنثروبومترية والكيميائية الحيوية والهرمونية، بما في ذلك مؤشر كتلة الجسم، ومحيط الخصر، ومستوى الدهون، ومستويات الهرمونات. أظهرت النتائج ارتفاعًا ملحوظًا في مؤشر كتلة الجسم، ومحيط الخصر، ومقاومة الأنسولين لدى مجموعة متلازمة تكيس المبايض مقارنةً بمجموعة الضبط .($p < 0.001$) كما لوحظ ارتفاع في مستويات الفيريتين لدى مريضات متلازمة تكيس المبايض($p = 0.001$) ، مع وجود ارتباط ضعيف ولكنه مهم بين الفيريتين ومقاومة الأنسولين $r = 0.104$ ، $p = 0.01$ أظهرت مجموعة متلازمة تكيس المبايض مستوى دهنيًا أكثر تصلبًا، مع انخفاض في كوليسترول البروتين الدهني عالي الكثافة) قيمة ($P < 0.0001$) وارتفاع في كوليسترول البروتين الدهني منخفض الكثافة) قيمة .($P < 0.0001$) أظهر التحليل الهرموني ارتفاعًا في هرمون الملوتن وانخفاضًا في نسبة FSH/LH قيمة $P < 0.0001$ ، وهو ما يتوافق مع الفسيولوجيا المرضية لمتلازمة تكيس المبايض. هناك علاقة وثيقة بين الخلل الأيضي، والاختلالات الهرمونية، وتغير استقلاب الحديد لدى النساء المصابات بمتلازمة تكيس المبايض.

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