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Serum Sortilin Levels as a Metabolic Biomarker in Polycystic Ovary Syndrome: A Case-Control Study from Southern Iraq

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ABSTRACT

Background: *Polycystic Ovary Syndrome (PCOS) is a prevalent endocrine-metabolic disorder affecting women of reproductive age, characterized by ovulatory dysfunction, hyperandrogenism, and polycystic ovarian morphology. Emerging evidence suggests that sortilin, a VPS10-domain receptor protein involved in lipid metabolism and insulin signaling, may play a role in PCOS pathophysiology through its association with metabolic dysregulation.* ***Objective:*** *This study aimed to assess serum sortilin levels in women with PCOS compared to healthy controls and to examine the association between sortilin and metabolic-hormonal parameters, including insulin resistance, testosterone levels, and body mass index (BMI).* ***Methodology:*** *A case-control study was conducted involving 120 female participants (60 PCOS patients and 60 healthy controls, aged 18–45 years) recruited from Basra Women and Children Teaching Hospital between September 2025 and January 2026. PCOS diagnosis was established using the Rotterdam criteria. Serum sortilin and biochemical markers were quantified using ELISA and automated analyzers.* ***Results:*** *Serum sortilin levels were significantly elevated in PCOS patients compared to controls (4.57 ± 0.98 vs. 2.56 ± 0.86 ng/mL, $p < 0.001$). A significant association was observed between sortilin and insulin resistance ($p = 0.042$), but not with BMI ($p = 0.558$). ROC analysis demonstrated excellent diagnostic performance (AUC = 0.943; sensitivity = 85%; specificity = 86.7%; cut-off = 3.650 ng/mL).* ***Conclusion:*** *Elevated serum sortilin may serve as a promising metabolic biomarker for PCOS, particularly reflecting insulin resistance rather than anthropometric measures. These findings suggest sortilin's potential utility in identifying metabolic dysfunction in PCOS and warrant further validation in larger, multicenter cohorts.*

Keywords: Polycystic Ovary Syndrome; Sortilin; Insulin Resistance; Biomarkers; Metabolic Disorders; Case-Control Studies.

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INTRODUCTION

Polycystic Ovary Syndrome (PCOS) is one of the most prevalent-complex endocrine-metabolic disorder witch developing in femalesnin reproductive age. It is mainly characterized by irregular ovulation, hyperandrogenism, and polycystic ovarian morphology. Clinically, it presents with symptoms such as menstrual irregularity, infertility, acne, and excessive hair growth. The diagnosis is usually based on the Rotterdam criteria, which require the presence of at least two of these features after excluding other related conditions (1, 2). The prevalence of this syndrome is different worldwide depending on diagnostic criteria and population studied. In general, it is estimated

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to affect around 6–20% of women of reproductive age. In Middle Eastern countries, including Iraq and neighboring regions, the prevalence is considered relatively high compared to many Western populations, This may be related with genetic, environmental, and lifestyle causes (3-5). PCOS is not only a reproductive disorder but is also strongly linked to metabolic disturbances Because most women that suffering from this syndrome have features that related with of metabolic disturbance such as insulin sensitive , obesity, dyslipidemia, and an increased risk of diabetes type 2 and cardiovascular disease, (6, 7). Although the Pathophysiology of this syndrome still unknown but some hypothesis may explain the possible mechanism are the changes that occur in some reproductive hormones, like luteinizing-hormone (LH),), dehydro-epiandrosterone (DHEA), follicle-stimulating- hormones (FSH, increased the ratio of LH/FSH, Elevated androgen levels, including DHEA this disturbances may effect on abnormal development of ovarian follicles and associated with clinical signs of hyperandrogenism These hormonal changes interact with metabolic disturbances, especially insulin resistance, which is worsens the function of ovary (8-10). Recently some metabolic and inflammatory biomarker that have a role in PCOS pathophysiology was studied, One of these biomarkers is sortilin, a protein encoded by the SORT1 gene. It is a type I transmembrane protein with a molecular weight of approximately 100 kDa and belongs to the mammalian VPS10 receptor family, which is involved in post-Golgi protein trafficking(11). Sortilin plays an important role in several metabolic processes, including lipid metabolism, glucose regulation, and insulin signaling (12). The relation between Sortilin and this syndrome is not fully understood until now. But because of insulin resistance is have a great role and a key feature of PCOS and because of Sortilin have a role in metabolic regulation it may contribute in the development of the condition (13, 14). So because of this background, this study aimed to assess serum sortilin levels in women with PCOS relative to healthy females and to study the relationship between Sortilin and metabolic-hormonal parameters such as testosterone, insulin resistance, and BMI, and to better understand the pathophysiology of this syndrome.

MATERIALS AND METHODS

Study Design and Population

This case-control study was conducted to evaluate the serum sortilin levels and associated endocrine-metabolic parameters in women with Polycystic Ovary Syndrome (PCOS). A total of 120 female participants, aged between 18 and 45 years, were enrolled in the study. The cohort was divided into two equal groups: 60 patients diagnosed with PCOS and 60 healthy, age-matched individuals who served as the control group.

Participant Recruitment and Selection Criteria

Participants in the patient group were recruited from women attending the Basra Women and Children Teaching Hospital over a five-month period, extending from September 2025 to January 2026. Relevant demographic and clinical data were obtained through direct, structured interviews with each participant.

The diagnosis of PCOS was established based on the revised Rotterdam criteria [1]. To ensure the validity of the findings, rigorous inclusion and exclusion criteria were applied. Individuals with conditions that could potentially confound the study outcomes were excluded. Specifically, the exclusion criteria encompassed the presence of cancer, hepatic or renal diseases, thyroid disorders, diabetes mellitus, and Cushing's syndrome. Furthermore, women who were pregnant, those currently using hormonal medications, and individuals diagnosed with other

gynecological conditions unrelated to PCOS—such as ovarian cysts, endometriosis, or ovarian tumors—were also excluded from the study.

Blood Sampling and Processing

Fasting venous blood samples were collected from all participants under standardized conditions. The collected whole blood was allowed to clot and subsequently centrifuged at [insert specific speed, e.g., 3000 rpm] for [insert specific time, e.g., 10 minutes] at [insert specific temperature, e.g., 4°C] to separate the serum. The obtained serum was aliquoted into two separate portions to facilitate distinct analytical procedures.

Biochemical and Hormonal Analyses

The first portion of the serum was utilized immediately for the quantification of routine biochemical and hormonal parameters. The lipid profile, including total cholesterol, low-density lipoprotein (LDL), high-density lipoprotein (HDL), and triglycerides, was evaluated. Hormonal assessments included the measurement of luteinizing hormone (LH), follicle-stimulating hormone (FSH), dehydroepiandrosterone (DHEA), total testosterone, and prolactin. Additionally, metabolic markers such as random blood sugar (RBS) and fasting insulin levels were determined. To ensure the proper exclusion of confounding conditions, supplementary tests, including serum urea, creatinine, thyroid-stimulating hormone (TSH), and liver function tests (e.g., AST, ALT), were performed. All routine biochemical and hormonal analyses were executed using fully automated clinical chemistry and immunoassay analyzers, specifically the Cobas Integra 400 plus system and the Cobas e411 analyzer (Roche Diagnostics, Basel, Switzerland), following the manufacturer's protocols.

Quantification of Serum Sortilin

The second portion of the serum was immediately frozen and stored at −20°C until further analysis. Serum sortilin concentrations were quantified using a commercially available enzyme-linked immunosorbent assay (ELISA) kit (E7987Hu), in strict accordance with the manufacturer's instructions.

Statistical Analysis

All the data were systematically analyzed by using SPSS software (version 26.0). in the other hand The Shapiro–Wilk test was used to assess data normality to guide the selection of appropriate statistical methods. Data were presented as mean ± standard deviation. The Student’s t-test was used when data were normally distributed. When the data were not normally distributed, the Mann–Whitney U test was used. The Welch’s t-test was also used when variances were unequal, especially in analyzing biomarkers such as sortilin. The correlation of Spearman’s rank was used to assess the link between levels of circulating sortilin and hormonal variables (LH, FSH, prolactin, testosterone, DHEA). ROC analysis was using to evaluate The diagnostic performance of sortilin as a potential biomarker. The area under the curve (AUC) was calculated, and optimal cut-off values were determined to estimate sensitivity, specificity, and diagnostic accuracy. The data considered statistical significance if p-value less than 0.05.

RESULTS

The comparison of age, body mass index, insulin resistance, and lipid profile between the two groups is shown in Table 1. This research show no correlation in the age between the two groups (24.47 ± 5.67 years in cases vs. 25.82 ± 5.03 years in controls, P = 0.170).in the other hand, BMI have high correlation in the case group relative to the control (31.13 ± 8.29 kg/m² vs. 23.76 ± 2.80 kg/m², P = 0.0001). in the same way insulin resistance also have a high correlation in the case group(3.11 ± 1.93) versus to controls (1.87 ± 0.76, P = 0.0001). Regard to parameters of lipid profile, total cholesterol levels were the same in the two groups (151.61 ± 38.83 mg/dL in cases vs.

151.63 ± 26.35 mg/dL in controls, P = 0.935). Triglyceride levels were higher in the case group (105.97 ± 56.11 mg/dL) compared to controls (85.81 ± 32.89 mg/dL); however, this difference did not reach statistical significance (P = 0.095). The concentration of Low-density lipoprotein (LDL) was significantly more in patients (105.21 ± 31.33 mg/dL) in relevant to controls (91.17 ± 22.68 mg/dL, P < 0.05). In contrast, high-density lipoprotein (HDL) concentrations were significantly less in the patients (46.70 ± 9.98 mg/dL) compared to the healthy participant (54.86 ± 10.63 mg/dL, P < 0.05).

Table 1: Basic features of study sample .

Variable	Case (n = 60)	Control (n = 60)	P value
Age (years)	24.47 ± 5.67	25.82 ± 5.03	0.170*
BMI (kg/m²)	31.13 ± 8.29	23.76 ± 2.80	0.0001**
IR	3.11 ± 1.93	1.870 ± 0.760	0.0001**
Total Cholesterol (mg/dL)	151.61 ± 38.83	151.63 ± 26.35	0.935*
Triglycerides (mg/dL)	105.97 ± 56.11	85.81 ± 32.89	0.095*
LDL (mg/dL)	105.21 ± 31.33	91.17 ± 22.68	0.031**
HDL (mg/dL)	46.70 ± 9.98	54.86 ± 10.63	0.0001**

The hormonal profile and Sortilin levels of the study groups are presented in Table 2. The concentrations of Luteinizing hormone (LH) were higher in the case group than in controls (14.99 ± 12.34 vs. 6.98 ± 5.63, P = 0.0001). In contrast, follicle-stimulating hormone (FSH) were significantly lower in case than in control (5.45 ± 2.09) (6.63 ± 2.77, P = 0.009). No significant difference was observed in prolactin levels between the two groups (17.48 ± 5.81 vs. 17.62 ± 9.80, P = 0.283). Similarly, DHEA levels showed no statistically significant variation between cases and controls (238.10 ± 139.43 vs. 219.38 ± 67.19, P = 0.642). Otherwise, the levels of testosterone were markedly more in the case group versus to controls (42.04 ± 24.40 vs. 21.94 ± 10.31, P = 0.0001). moreover, levels of Sortilin were high between patients relative to the controls (4.57 ± 0.98) (2.56 ± 0.86, P = 0.0001).

Table 2: Sortilin and Hormone levels in the study population.

Hormones	Case (n = 60)	Control (n = 60)	P value
LH	14.99 ± 12.34	6.98 ± 5.63	0.0001
FSH	5.45 ± 2.09	6.63 ± 2.77	0.009
Prolactin	17.48 ± 5.81	17.62 ± 9.80	0.283
Testosterone	42.04 ± 24.40	21.94 ± 10.31	0.0001
DHEA	238.10 ± 139.43	219.38 ± 67.19	0.642
Sortilin	4.57 ± 0.98	2.56 ± 0.86	0.0001

The comparison of Sortilin levels according to BMI, insulin resistance, and testosterone is shown in Table 3. Sortilin levels didn't have correlation between individuals with normal and high BMI (4.47 ± 1.09 vs. 4.60 ± 0.96, P = 0.558).In the other hand women that have high level of insulin resistance showed high level of sortilin (4.72 ± 1.01) relative to those with normal insulin resistance (4.20 ± 0.81), and that was have statistically significant (P = 0.042). depending on the testosterone and Sortilin levels there were different that showed between the two groups (P = 0.001). individuals with normal testosterone have a littel higher sortilin (4.64 ± 1.03) than to the women with high testosterone (4.37 ± 0.81).

Table 3: Sortilin level in base of BMI, Insulin resistance, and Testosterone in PCOS women.

Parameters	Sortilin	P value
BMI	**Normal (n = 14)** : 4.47 ± 1.09	0.558
	High (n = 46) : 4.60 ± 0.96	
Insulin resistance	**Normal (n = 17)** : 4.20 ± 0.81	0.042
	High (n = 43) : 4.72 ± 1.01	
Testosterone	**Normal (n=45)** : 4.64 ± 1.03	0.001
	High (n=15) : 4.37 ± 0.81	

The correlation analysis between sortilin and hormonal parameters is presented in Table 4. There is no significant correlation between prolactin, testosterone, DHEA, LH, FSH and Sortilin level ($P > 0.05$), but there is a strong positive link was observed between testosterone and DHEA ($r = 0.682$, $P < 0.001$), ($r = 0.499$, $P < 0.001$).

Table 4: Correlation of biomarkers and hormones in PCOS women.

		Prolactin	Testosterone	DHEA	LH	FSH
Sortilin	R	-0.104	0.025	0.026	0.154	0.205
	P value	0.428	0.848	0.841	0.241	0.117
Prolactin	R		0.173	0.093	-0.203	-0.149
	P value		0.185	0.481	0.119	0.257
Testosterone	R			0.682	0.188	0.255
	P value			0.000	0.151	0.049
DHEA	R				0.080	0.172
	P value				0.545	0.188
LH	R					0.499
	P value					0.000

Figure 1 The ability of Sortilin to distinguish between PCOS patients and healthy ones was evaluated using (ROC) curve analysis. The diagnostic value of Sortilin demonstrated high accuracy (AUC = 0.945). At a cut-off value 0f 3.650, it was highly sensitive and highly specific with an overall accuracy of 85.8% (Table 5 and Figure 1).

Table 5: Diagnostic value of Sortilin.

AUC	P value	CI	Cut-off value	Sensitivity	Specificity	Accuracy
0.943	0.0001	0.905-0.980	3.650	85%	86.7%	85.8%

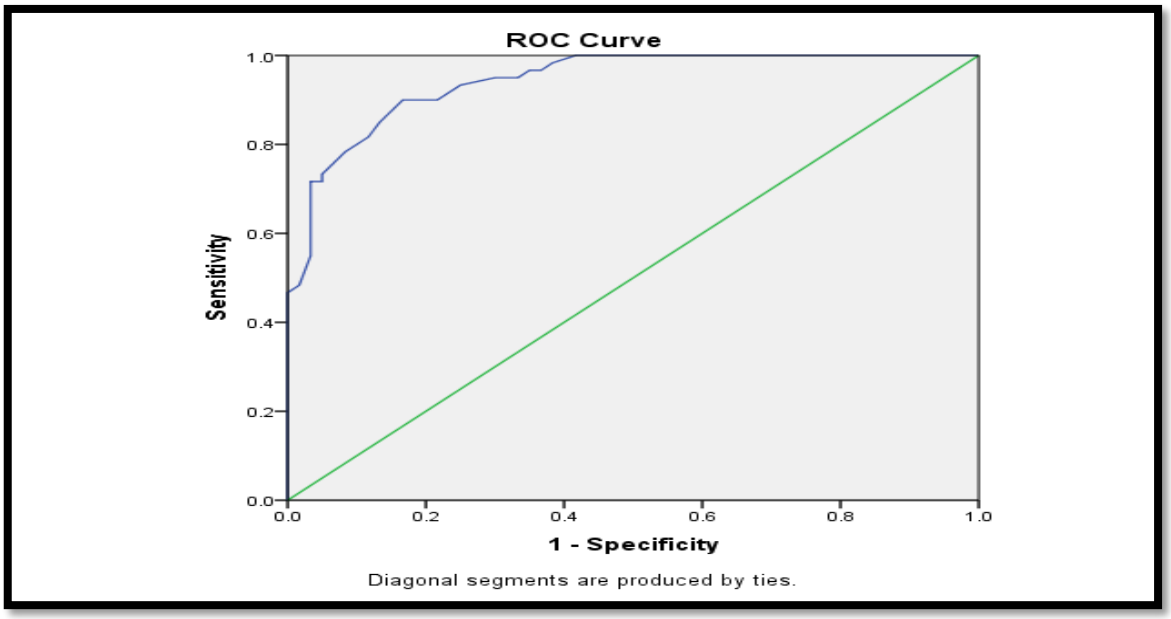

Figure 1: ROC curve for Sortilin.

DISCUSSION

The PCOS is the most complex metabolic endocrine condition that effects on females on their reproductive ages(15). In the current research the average age of female that diagnosed with PCOS was in there mid-twenties, these results are align with multiple studies in Iraq and other countries, this reflect the demographic characteristics of females that get medical care for their reproductive and gynecological problems on this age(16, 17)

In the present study, PCOS patients exhibited significantly higher BMI and insulin resistance compared to controls. These findings are in agreement with previous studies conducted in Iraq and populations of Middle East (18-20). this indicating that insulin sensitive and body mass index are the major factors that effect on this syndrome (21)

Additionally, the current research showed significantly high LH and testosterone concentrations, with an elevated LH/FSH ratio in PCOS patients. These findings are consistent with the classical endocrine pattern of PCOS and have been widely documented in regional studies(22-24), moreover, this study have been showed that Sortilin witch is a VPS10-domain receptor involved in intracellular protein trafficking (25, 26), showed elevated in their levels in women with this syndrome This elevation is consistent with emerging evidence that Sortilin is strongly associated with metabolic dysregulation, particularly insulin resistance and altered lipid metabolism(27, 28)

Interestingly, there were no observed correlation between sortilin and reproductive hormones in both PCOS and control groups. This may indicate that sortilin is not directly involved in the hypothalamic–pituitary–ovarian axis. Instead, it may be more related to metabolic processes in the body. This idea is supported by previous studies showing that sortilin is mainly involved in lipid metabolism and insulin-related pathways rather than hormone secretion.(29, 30)

ROC analysis in this study revealed that sortilin has excellent diagnostic performance with high sensitivity and specificity, indicating its potential utility as a biomarker for PCOS. This aligns with recent findings reporting that circulating sortilin may serve as a promising indicator of metabolic risk in reproductive disorders, although further large-scale multicenter studies are still required to validate its clinical

Overall, the findings of this study support the concept that PCOS is a complex reproductive–metabolic disorder rather than endocrine disease. Additionally, the high levels of sortilin and its good diagnostic performance highlight its potential role in metabolic biomarker rather than a reproductive hormones regulator.

Limitation of the study

this study has some limitations. It was done in only one center in southern Iraq, so the results may not apply to other populations. The sample size was small, especially in subgroup analysis, which may have affected the results. Also, because it is a case-control study, it cannot show cause and effect, and the biomarkers were measured only once, so changes over time were not studied. In addition, the study did not examine how these biomarkers are linked to PCOS at the molecular level.

CONCLUSION

This study provides compelling evidence that serum sortilin functions as a metabolic biomarker in PCOS, reflecting the underlying pathophysiological mechanisms of metabolic dysregulation rather than serving as a reproductive hormone regulator. The strong association between elevated sortilin and insulin resistance, coupled with its lack of correlation with anthropometric measures, suggests that sortilin may serve as a sensitive indicator of metabolic dysfunction in PCOS independent of body weight.

This distinction is clinically significant, as it highlights the heterogeneity of PCOS phenotypes and the importance of identifying metabolic risk beyond traditional anthropometric assessment. Future research should investigate the mechanistic relationship between sortilin and insulin resistance pathways in PCOS, potentially exploring therapeutic targeting of sortilin-mediated metabolic dysfunction. Additionally, longitudinal studies examining the temporal relationship between sortilin elevation and the development of metabolic complications would provide valuable insights into its role in PCOS progression. These findings collectively support the reconceptualization of PCOS as a complex reproductive-metabolic disorder, emphasizing the need for integrated metabolic assessment and intervention strategies in clinical management.

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Declarations

Ethical approval

Approval to conduct this study was obtained before the start of sample collection. The study was carried out in collaboration with Southern Technical University and the Basra Health Department. Approval for the study was done by the research committee (approval number 2119) on September 2, 2025, following the Helsinki Declaration guidelines, and all participants provided written informed consent.

Competing interests: The author declare that they have no competing interests

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Author Contribution

Conceptualization, TADA and MAM; methodology, TADA, MAM and WFM; validation, TADA, MAM and WFM; formal analysis, TADA; investigation, TADA, MAM and WFM; resources, TADA; data curation, TADA; writing – original draft preparation, TADA and MAM; writing – review and editing, all authors; visualization, TADA; supervision, MAM; project administration, MAM; funding acquisition, TADA, MAM and WFM. All authors have read and agreed to the published version of the manuscript..

Availability of data and material: The datasets used and analyzed during the current study are available from the corresponding author on reasonable request.

REFERENCES

1. Fahs D, Salloum D, Nasrallah M, Ghazeeri G. Polycystic Ovary Syndrome: Pathophysiology and Controversies in Diagnosis. Diagnostics. 2023;13(9):1559.
2. Rosenfield RL, Ehrmann DA. The Pathogenesis of Polycystic Ovary Syndrome (PCOS): The Hypothesis of PCOS as Functional Ovarian Hyperandrogenism Revisited. Endocrine Reviews. 2016;37(5):467-520.
3. Alkhezi F, AlNemash N, AlMutairi J, Saleh S, AlMutairi M, Saleh S, et al. Prevalence of and Risk Factors Associated with Polycystic Ovary Syndrome Among Female University Students of Health Sciences in a Middle Eastern Country. Women's Health Reports. 2024;5(1):whr.2023.0176.
4. Alam Z, Alseari S, Alameemi M, Alzaabi M, Alkhoori R, Östlundh L, et al. Prevalence of polycystic ovary syndrome among infertile women in the Gulf Cooperation Council (GCC) countries: A systematic review and meta-analysis. Heliyon. 2024;10(24):e40603.
5. Abbood AH, Majeed Hameed R, Ghazi Al Safi W. Neuregulin 4 in Polycystic Ovarian Syndrome (PCOS) Phenotypes: A Key Role or Standby. Rep Biochem Mol Biol. 2023;12(3):359-65.
6. Salari N, Nankali A, Ghanbari A, Jafarpour S, Ghasemi H, Dokaneheifard S, et al. Global prevalence of polycystic ovary syndrome in women worldwide: a comprehensive systematic review and meta-analysis. Arch Gynecol Obstet. 2024;310(3):1303-14.
7. Helvaci N, Yildiz BO. Polycystic ovary syndrome as a metabolic disease. Nature Reviews Endocrinology. 2025;21(4):230-44.
8. Kambale T, Sawaimul KD, Prakash S. A study of hormonal and anthropometric parameters in polycystic ovarian syndrome. Annals of African medicine. 2023;22(1):112-6.
9. 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. Front Endocrinol (Lausanne). 2021;12:571229.
10. Yucel A, Noyan V, Sagsoz N. The association of serum androgens and insulin resistance with fat distribution in polycystic ovary syndrome. European journal of obstetrics, gynecology, and reproductive biology. 2006;126(1):81-6.
11. Mitok KA, Keller MP, Attie AD. Sorting through the extensive and confusing roles of sortilin in metabolic disease. Journal of lipid research. 2022;63(8):100243.
12. Su X, Chen L, Chen X, Dai C, Wang B. Emerging roles of sortilin in affecting the metabolism of glucose and lipid profiles. Bosnian journal of basic medical sciences. 2021;22(3):340.
13. Çalışkan Burgucu H, Yarar Z, Can M, Karadeniz Y, Yerlikaya Aydemir FH, Karaköse M, et al. CIMT as a sensitive indicator of cardiovascular risk in PCOS: a case-control study of sortilin and sclerostin. BMC Womens Health. 2026;26(1).
14. Alarslan P, Doruk M. Serum Sortilin Levels as a Biomarker for Metabolic and Hormonal Dysregulation in Polycystic Ovary Syndrome. Journal of personalized medicine. 2025;15(2).
15. Yokyongsakul M, Humart P, Techatraisak K, Tanmahasamut P, Rattanachaiyanont M, Indhavivadhana S, et al. Anthropometric Measurements to Predict Metabolic Syndrome in Thai Women With Polycystic Ovary Syndrome: A Retrospective Study. Journal of Obstetrics and Gynaecology Research. 2026;52(3):e70255.
16. Jalil P, Shnawa B, Ahmed M. Association of free testosterone, glucose level and obesity among women with polycystic ovary syndrome in Soran city, Kurdistan-Iraq. Clínica e Investigación en Ginecología y Obstetricia. 2023;50(4):100903.
17. AlMekhlafi SA, Campo-Redondo MS. Association between body image satisfaction and depressive symptoms among young women in the UAE with Polycystic Ovary Syndrome (PCOS) and Non-PCOS: A

comparative quantitative cross-sectional study. PLoS One. 2026;21(1):e0333908.

18. Al-Murshedi HH, Al-Tu'ma FJ, Hadi EA, Mohammed BM, Al-Kayat T. Roles of C-peptide and triglyceride as effective indices for insulin resistance investigations in Iraqi women with polycystic ovarian syndrome. J Contemp Med Sci| Vol. 2022;8(3):169-75.

19. Rahmatnezhad L, Moghaddam-Banaem L, Behroozi-Lak T, Shiva A, Rasouli J. Association of insulin resistance with polycystic ovary syndrome phenotypes and patients' characteristics: a cross-sectional study in Iran. Reproductive Biology and Endocrinology. 2023;21(1):113.

20. Uysal E, Tammo O, Soylemez E, Incebıyık M, Filiz D, Alci M. Significance of measuring anthropometric and atherogenic indices in patients with polycystic ovary syndrome. BMC Endocrine Disorders. 2024;24(1):160.

21. Mansour A, Mirahmad M, Mohajeri-Tehrani MR, Jamalizadeh M, Hosseinimousa S, Rashidi F, et al. Risk factors for insulin resistance related to polycystic ovarian syndrome in Iranian population. Scientific reports. 2023;13(1):10269.

22. Ahmed RS-A, Aljabar FAA, Al-Shakir NMM. Impact of Insulin Resistance in PCOS (LH/FSH) in a Group of Woman in Baghdad City. Al-Esraa University College Journal for Medical Sciences. 2025;6(9):20-30.

23. Kheirollahi A, Hamidi H, Vatannejad A. Distribution of polycystic ovary syndrome (PCOS) phenotypes in Iranian women: a cross-sectional study. BMC research notes. 2025;18(1):215.

24. Ates S, Sevket O, Sudolmus S, Dane B, Ozkal F, Uysal O, et al. Different phenotypes of polycystic ovary syndrome in Turkish women: clinical and endocrine characteristics. Gynecological endocrinology : the official journal of the International Society of Gynecological Endocrinology. 2013;29(10):931-5.

25. Andevari AN, Firoozjaee AH, Meftah N, Asciabari HA, Bahri F, Shahandashti NE, et al. The effects of atorvastatin consumption on blood levels of sortilin, glycemic, and lipid indices in type 2 diabetic patients: A randomized clinical trial. International Journal of Diabetes in Developing Countries. 2025:1-8.

26. Namitokov A. Sortilin and its potential role in cardiovascular pathology. The Egyptian Heart Journal. 2024;76(1):78.

27. Alarslan P, Doruk M. Serum sortilin levels as a biomarker for metabolic and hormonal dysregulation in polycystic ovary syndrome. Journal of personalized medicine. 2025;15(2):70.

28. Mousa BA, Al Joborae S. Analysis of Patients with PCOS According to Demographic Factors & Hormonal Assayin Babylon Government in Iraq. Medico Legal Update. 2020;20(2):473-8.

29. Kjolby M, Nielsen MS, Petersen CM. Sortilin, encoded by the cardiovascular risk gene SORT1, and its suggested functions in cardiovascular disease. Current atherosclerosis reports. 2015;17(4):496.

30. Su X, Chen L, Chen X, Dai C, Wang B. Emerging roles of sortilin in affecting the metabolism of glucose and lipid profiles. Bosnian journal of basic medical sciences. 2022;22(3):340-52.

