Am J Transl Res 2026;18(4):3322-3330
www.ajtr.org /ISSN:1943-8141/AJTR0170797

Original Article

Comparison of glucolipid metabolic profile, thyroid hormone levels, and reproductive endocrine function in polycystic ovary syndrome patients with/without metabolic syndrome

Xiaoting Li, Mei Wang

Department of Gynecology, Shanghai Pudong New Area People's Hospital, Shanghai 201299, China

Received December 16, 2025; Accepted February 26, 2026; Epub April 15, 2026; Published April 30, 2026

Abstract: Objective: To compare the differences in glucose and lipid metabolism profiles, thyroid hormone levels, and reproductive endocrine function between polycystic ovary syndrome (PCOS) patients with and without metabolic syndrome (MS). Methods: This retrospective study included 160 PCOS patients treated in Shanghai Pudong New Area People's Hospital from January 2020 to December 2021. Patients were divided into two groups based on whether they had MS: a PCOS group without MS (n=105) and a PCOS group with MS (n=55). Clinical and laboratory parameters were compared between the two groups. Results: The prevalence of MS in PCOS patients was 34.38%. Compared with the PCOS group, the PCOS+MS group exhibited significantly elevated levels of fasting plasma glucose, 2-hour postprandial glucose, fasting insulin, homeostasis model assessment of insulin resistance, total cholesterol, triglycerides, low-density lipoprotein cholesterol, and thyroid-stimulating hormone (all $P<0.05$), while high-density lipoprotein cholesterol levels were significantly decreased ($P<0.05$). No significant differences were found in free triiodothyronine, free thyroxine, or key reproductive hormones (testosterone, estradiol, follicle-stimulating hormone, luteinizing hormone) between the groups (all $P>0.05$). Conclusion: MS is common in PCOS patients and is associated with increased glucose and lipid metabolism disorders and elevated TSH, but not with changes in the reproductive endocrine profile. Early screening for MS in PCOS patients is recommended to facilitate timely management of metabolic and thyroid abnormalities.

Keywords: Metabolic syndrome, polycystic ovary syndrome, glycolipid metabolism, thyroid hormones, reproductive endocrine function

Introduction

Polycystic ovary syndrome (PCOS) is a common clinical disorder. Patients with PCOS often present with infrequent or absent ovulation and polycystic ovary morphology. Many patients are overweight or obese and have insulin resistance, which increases the risk of type 2 diabetes. PCOS is a heterogeneous disease with diverse clinical manifestations [1, 2]. Clinically, it mainly manifests as menstrual irregularities, infertility, and amenorrhea, and can also lead to insulin resistance (IR), obesity, and lipid metabolism abnormalities, thereby increasing the risk of type 2 diabetes, metabolic syndrome (MS) and cancer [3, 4].

Patients with PCOS and MS often have insulin resistance. Insulin resistance not only affects glucose metabolism but may also lead to problems such as hyperandrogenemia and obesity. When the body is in a state of insulin resistance, it secretes large amounts of insulin to maintain normal blood glucose levels, which may lead to changes in the body's glucose metabolism [5, 6]. Research data show that patients with the disease studied in this article often have abnormal lipid metabolism [7]. Patients with PCOS lack normal metabolic capacity, and if left untreated for a long time, it will affect their reproductive health. Evidence suggests that improving metabolic abnormalities can help improve treatment outcomes in these

https://doi.org/10.62347/OSPS1409

patients [8]. Therefore, clinicians should pay attention to the metabolic levels of these patients and develop targeted treatment plans [9]. Existing research data indicates a close link between PCOS and multiple sclerosis. Experimental results have shown that genetic factors and tissue dysfunction can influence both conditions [10]. These studies suggest that understanding the mechanisms of action of these two diseases can help clinicians treat patients with both conditions. Some researchers have studied patients with PCOS and multiple sclerosis who also have cardiovascular disease and found that these patients have a higher risk of developing diabetes than PCOS patients, and their prognosis is often poorer [10, 11]. Data suggest that the mechanisms of action of these two diseases are similar in terms of reproductive endocrine disorders such as insulin resistance [12, 13]. Therefore, early screening and appropriate treatment of PCOS patients with MS can help improve treatment outcomes. This study aims to analyze the effects of MS on glucose and lipid metabolism indicators, thyroid hormone levels, and reproductive endocrine function in patients with polycystic ovary syndrome.

Methods and materials

Study subjects

This study was conducted using a retrospective analysis method. The cases collected were patients with polycystic ovary syndrome who received treatment at Shanghai Pudong New Area People's Hospital from January 2020 to December 2021. A total of 160 cases were included in this study.

This study was conducted in accordance with the Declaration of Helsinki. The study has been approved by the Ethics Committee of Shanghai Pudong New Area People's Hospital and complies with the relevant regulations and ethical guidelines for retrospective studies. Since this retrospective study only used deidentified patient data and would not cause potential harm or impact on patient care, informed consent was exempted.

The diagnosis of MS was based on the International Diabetes Federation (IDF) criteria (2005) [14]. Central obesity was defined as a waist circumference ≥80 cm in Chinese women plus any two of the following: fasting blood glucose ≥5.6 mmol/L or a previous diagnosis of type 2 diabetes; blood pressure ≥130/85 mmHg or currently receiving treatment for hypertension; high-density lipoprotein cholesterol (HDL-C) <1.29 mmol/L; triglycerides ≥1.7 mmol/L.

Inclusion criteria: (1) Based on relevant Chinese diagnostic criteria, patients were determined to have polycystic ovary syndrome (meeting at least two of the Rotterdam criteria: polycystic ovary morphology, biochemical hyperandrogenemia, and anovulation/ovulation disorders) [15]; (2) Newly diagnosed patients who had not previously received treatment for PCOS or metabolic disorders; (3) Complete clinical and laboratory data, including baseline demographic data, glucose and lipid profiles, thyroid hormones, and reproductive hormones. Exclusion criteria: (1) Based on clinical diagnosis and medication records, the patient had thyroid disease (hypothyroidism, hyperthyroidism, thyroiditis) or was currently using thyroid medications (e.g., levothyroxine). (2) Laboratory and imaging examinations confirmed severe organ dysfunction (hepatic, renal, or cardiovascular failure). (3) Clinical and pathological examinations diagnosed the patient with other endocrine disorders (Cushing's syndrome, hyperprolactinemia) or malignant tumors. (4) The patient had recently used hormone therapy (such as oral contraceptives, glucocorticoids) within 3 months prior to enrollment.

Based on whether they had MS, the 160 patients were divided into a PCOS group without MS (n=105) and a PCOS group with MS (n=55). This study was approved by the hospital ethics committee. This study used an observational design, dividing patients into two groups based on the presence or absence of metabolic syndrome. No external control group was set up because the main comparison was between PCOS patients with and without metabolic syndrome.

Data collection

Blood samples were collected from patients during the follicular phase, between 8:00 AM and 10:00 AM. For amenorrhea women, samples could be collected at any time after confirming no pregnancy; for normal women, samples were collected on days 2 to 5 of the menstrual cycle.

Collected blood samples were centrifuged at 4°C, 3000×g for 10 minutes. The serum was separated and stored at -80°C.

Glucose and lipid metabolism indicators: Fasting plasma glucose (FPG) and 2-hour postprandial glucose (2h PG) were measured using the glucose oxidase method (Roche Cobas 8000, Roche Diagnostics, Mannheim, Germany). Intra-assay and inter-assay coefficients of variation (CV) were <2.5% and <3.0%, respectively. Fasting insulin (FINS) was quantitatively analyzed using electrochemiluminescence immunoassay (Elecsys insulin assay, Roche Diagnostics).

Lipid profile: After patient serum samples were collected, the concentrations of total cholesterol (TC), triglycerides (TG), HDL-C, and low-density lipoprotein cholesterol (LDL-C) were measured. Biochemical kits capable of detecting these indicators were selected, and serum samples were analyzed using a biochemical analyzer.

Thyroid hormones and reproductive hormones: Free triiodothyronine (FT3), free thyroxine (FT4), and thyroid-stimulating hormone (TSH) were detected using a chemical method kit manufactured by Siemens, Germany. The intra-assay CVs for these three indicators were 3.2%, 2.8%, and 2.5%, respectively. Our laboratory's normal reference ranges are: FT3 2.3-4.2 pg/mL, FT4 0.89-1.76 ng/dL, and TSH 0.55-4.78 mU/L. When measuring testosterone (T), estradiol (E2), follicle-stimulating hormone (FSH), and luteinizing hormone (LH) in this experiment, the international reference standard (World Health Organization (WHO) FSH/LH standard) was used. Reproductive hormone testing was performed in the early follicular phase (days 2-5 of the menstrual cycle). For amenorrhea women, testing was performed after excluding pregnancy.

Missing data (primary outcome measure n=0) were excluded from the analysis; no imputation was performed.

Statistical analysis

Statistical analysis was performed after data collection. For continuous variables, the Shapiro-Wilk test was used to assess their normality. Normally distributed data were expressed as mean ± standard deviation and compared using an independent samples t-test; non-normally distributed data were expressed as median (interquartile range) and compared using a Mann-Whitney U test. Categorical data were expressed as frequency (%) and compared using a chi-square test. The effect size (Cohen's d) of the primary outcome measure was calculated. This study used binary logistic regression analysis to explore independent risk factors associated with MS in the PCOS population. A two-sided P<0.05 was considered statistically significant.

Results

Comparison of demographic data

In this study, there were 55 patients in the PCOS with MS group and 105 patients in the PCOS group. The basic information of the two groups was compared and analyzed. The results showed no significant differences between the two groups in terms of smoking history, education level, age, etc. (all P>0.05). See **Table 1**.

Comparison of clinical characteristics

There were no significant differences observed in body mass index, duration of PCOS, or menstrual cycle length between the two groups (all *P*>0.05). Waist circumference, systolic blood pressure, heart rate, and diastolic blood pressure were significantly higher in the PCOS with MS group (all *P*<0.05). See **Table 2**.

Comparison of glucose metabolism indicators

The levels of FPG, 2h PG, FINS, and HOMA-IR were significantly higher in the PCOS with MS group than those in the PCOS group. The results for the former were 5.18±0.45 mmol/L, 9.23±2.37 mmol/L, 17.52±6.34 μU/mL, and 5.38±1.07, respectively, while those for the latter were 4.39±0.26 mmol/L, 6.58±1.24 mmol/L, 10.38±4.96 μU/mL, and 3.35±0.50, respectively. The differences were statistically significant (P<0.05). See **Figure 1** and **Table 3** for details.

Comparison of lipid metabolism indicators

Compared with the PCOS group alone, the PCOS with MS group had significantly higher levels of TC, TG, and LDL-C (all P<0.05). In terms of HDL-C, the level in the PCOS with MS group was significantly lower than that in the PCOS group (P<0.05) (**Table 4**).

Table 1. Comparison of demographic data

Demographic Data	Simple PCOS group (n=105)	PCOS+MS group (n=55)	t/χ^2	P
Age (years)	28.41±4.07	28.38±4.63	0.041	0.967
Ethnicity (Han/Other) [n (%)]	96 (91.43)	52 (94.5)	0.156	0.693
Educational level (high school or below/junior college or above) [n (%)]	45 (42.9)	30 (54.55)	1.980	0.159
Residence (Urban/Rural)	68 (64.8)	30 (54.5)	1.587	0.208
Smoking history [n (%)]	8 (7.6)	8 (14.55)	1.924	0.165
Drinking history [n (%)]	15 (14.3)	13 (23.6)	2.186	0.139
Previous Pregnancy [n (%)]	38 (36.2%)	15 (27.3%)	1.296	0.255

Table 2. Comparison of clinical characteristics

Clinical Data	Simple PCOS group (n=105)	PCOS+MS group (n=55)	t	P	Cohen's d	95% CI
Body mass index (kg/m^2)	24.33±3.72	24.10±4.05	0.363	0.717	-	-
Waist Circumference (cm)	78.29±6.54	90.12±7.86	10.128	<0.001	1.62	(1.24, 2.00)
Systolic Blood Pressure (mmHg)	118.52±10.27	132.85±11.53	8.033	<0.001	1.29	(0.94, 1.64)
Heart Rate (beats/min)	72.47±8.12	76.84±9.33	3.070	0.003	0.49	(0.17, 0.81)
Diastolic Blood Pressure (mmHg)	75.36±7.83	84.62±8.94	6.763	<0.001	1.08	(0.74, 1.42)
Duration of PCOS (months)	16.82±4.20	17.17±4.63	0.485	0.629	-	-
Menstrual Cycle Length (days)	45.64±11.36	48.49±12.72	1.444	0.151	-	-

Note: PCOS: polycystic ovary syndrome. Data are presented as mean ± SD and compared using independent samples t-test, unless otherwise noted. Cohen's d effect sizes were interpreted as small (≥0.2), medium (≥0.5), and large (≥0.8).

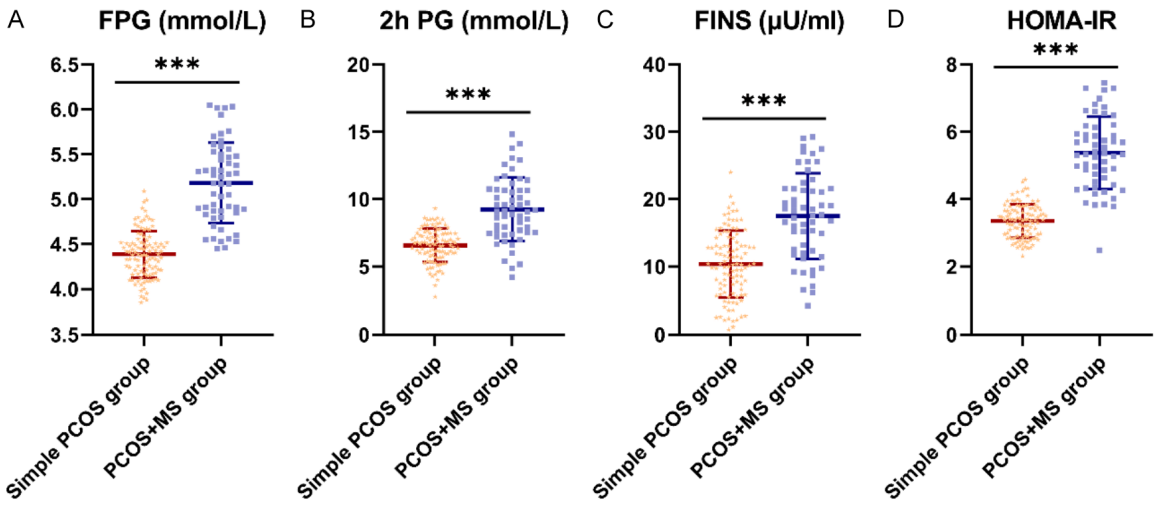

Figure 1. Comparison of glucose metabolism indexes between the two groups. A: FPG (mmol/L); B: 2h PG (mmol/L); C: FINS (μU/ml); D: HOMA-IR. Note: FPG: fasting plasma glucose; 2h PG: 2-hour postprandial glucose; FINS: fasting insulin; HOMA-IR: homeostatic model assessment of insulin resistance. ***: *P*<0.001.

Comparison of thyroid hormone levels

Compared with the PCOS group, there were no significant differences in FT3 and FT4 levels between the groups (P>0.05). Comparing the TSH levels, the latter was significantly higher than the former (P<0.05). See **Figure 2**.

Comparison of reproductive endocrine function indicators

Compared with the PCOS group, there were no significant differences in serum testosterone, E2, FSH or LH levels between the groups (all P>0.05, **Table 5**), indicating no MS-

Table 3. Cohen's d value of glucose metabolism indicators

Indicator	PCOS+MS group vs. PCOS group alone	Cohen's d	95% CI
FPG (mmol/L)	5.18±0.45 vs. 4.39±0.26	2.34	(1.82, 2.86)
2h PG (mmol/L)	9.23±2.37 vs. 6.58±1.24	1.68	(1.21, 2.15)
FINS (μU/ml)	17.52±6.3 vs. 10.38±4.96	1.42	(0.98, 1.86)
HOMA-IR	5.38±1.07 vs. 3.35±0.50	3.12	(2.45, 3.79)

Note: FPG: fasting plasma glucose; 2h PG: 2-hour postprandial glucose; FINS: fasting insulin; HOMA-IR: homeostatic model assessment of insulin resistance. Cohen's d effect sizes were interpreted as small (≥0.2), medium (≥0.5), and large (≥0.8).

Table 4. Comparison of lipid metabolism indicators

Lipid metabolism indexes	Simple PCOS group (n=105)	PCOS+MS group (n=55)	t	P
TC (mmol/L)	4.84±0.91	5.23±1.19	2.169	0.033
TG (mmol/L)	1.32±0.31	2.26±0.56	11.574	<0.001
HDL-C (mmol/L)	1.52±0.29	1.30±0.34	4.247	<0.001
LDL-C (mmol/L)	2.49±0.25	3.06±0.34	11.032	<0.001

Note: TC: total cholesterol; TG: triglycerides; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol.

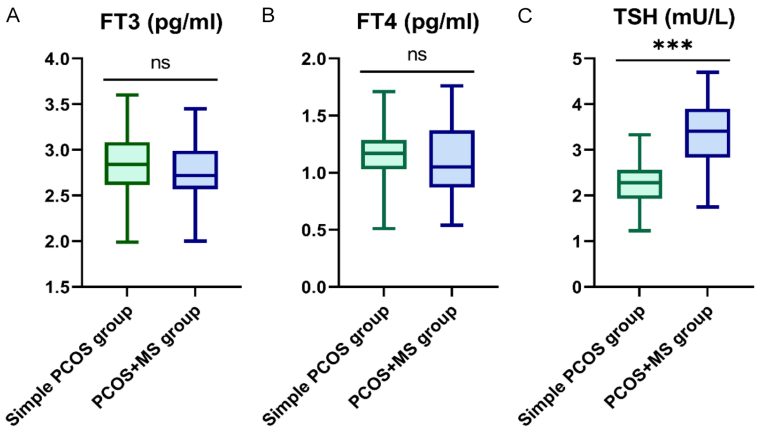

Figure 2. Comparison of thyroid hormone levels. A: FT3 (pg/ml); B: FT4 (pg/ml); C: TSH (mU/L). Note: FT3: free triiodothyronine; FT4: free thyroxine; TSH: hypersensitive thyrotropin; ns: no significant difference; ***: P<0.001.

associated changes in reproductive hormone profiles.

Logistic regression analysis

The logistic regression analysis identified several significant predictors for MS in patients with PCOS (**Table 6**). Increased waist circumference, HOMA-IR scores, TG levels, TSH levels and lower HDL-C levels were associated with an increased risk of MS among PCOS patients (all $P<0.05$).

Discussion

PCOS has a certain incidence rate among women. It can occur when women experience endocrine disorders due to various reasons. This condition can lead to other metabolic-related syndromes, resulting in a unique symptom state. Without effective intervention, it can lead to cardiometabolic abnormalities. This study investigated changes in metabolic and endocrine levels in patients with PCOS and MS. The results indicated that patients in this study exhibited abnormalities in glucose metabolism and blood lipid levels, as well as abnormalities in TSH, but no significant abnormalities in reproductive hormones. It can be considered that MS is a modifying factor that leads to endocrine and glucose metabolism abnormalities in PCOS patients.

The study data showed a close relationship between the presence of MS and abnormal glucose metabolism levels in the disease studied in this paper. These patients often have high fasting blood glucose and high FINS levels. This indicates that insulin resistance is a common feature of the two diseases [16, 17]. We observed significantly elevated HOMA-IR and FINS levels in the PCOS with MS group , further supporting the previous finding that insulin resistance synergistically worsens when these conditions coexist [18]. Notably, the Cohen's

Table 5. Comparison of reproductive endocrine function indicators

Reproductive endocrine function indexes	Simple PCOS group (n=105)	PCOS+MS group (n=55)	t	P
T (nmol/L)	1.55±0.41	1.63±0.55	0.961	0.339
E2 (pmol/L)	151.46±85.12	173.84±91.26	1.541	0.125
FSH (IU/L)	6.56±1.15	6.73±1.82	0.619	0.538
LH (IU/L)	10.15±3.78	9.86±3.15	0.486	0.628

Note: T: testosterone; E2: estradiol; FSH: follicle-stimulating hormone; LH: luteinizing hormone.

Table 6. Logistic regression analysis of risk factors for MS in PCOS patients

Variable	β	SE	Wald	OR (95% CI)	P
Waist Circumference (cm)	0.352	0.118	10.122	1.426 (1.157-1.766)	0.001
HOMA-IR	1.245	0.301	17.123	3.472 (1.925-6.262)	<0.001
TG (mmol/L)	2.104	0.456	21.294	8.192 (3.351-20.023)	<0.001
HDL-C (mmol/L)	-1.893	0.521	13.201	0.151 (0.054-0.419)	<0.001
TSH (mU/L)	0.876	0.287	9.312	2.401 (1.367-4.217)	0.002

Note: HOMA-IR: homeostatic model assessment of insulin resistance; TG: triglycerides; HDL-C: high-density lipoprotein cholesterol; TSH: thyroid-stimulating hormone.

coefficient for HOMA-IR was 3.12, indicating a large effect size and underscoring that MS substantially exacerbates insulin resistance in PCOS patients, potentially leading to glucose and lipid metabolism disorders and long-term metabolic risks. Clinical evidence suggests that insulin resistance is a hallmark of metabolic syndrome, which leads to a vicious cycle [18]. Consequently, to improve this condition, the pancreas begins to secrete insulin, which can lead to hyperinsulinemia [19]. When this condition occurs, it stimulates the ovaries to produce related hormones, resulting in PCOS and an inability to maintain normal endocrine levels [20]. In this case, we find that patients' glucose metabolism also changes, thus leading to a higher incidence of diabetes in these patients compared to other types.

We studied and analyzed these cases to identify patients with two comorbidities who had poor lipid metabolism. When we tested the lipid levels of these patients, we found that their lipid profiles were poor. Abnormal lipid levels increase the risk of atherosclerosis. Data show that women with abnormal lipid levels have a higher risk of PCOS than women with normal lipid levels, and abnormal androgen levels also increase their risk of the disease [21]. Androgens can regulate lipid levels in atherosclerosis and significantly increase total cholesterol levels, increasing the incidence of cardiovascular disease. This observation is consistent with the study by Olejarz et al., who emphasized that endocrine disorders, including PCOS, are associated with specific lipoprotein changes that promote the development of atherosclerosis [22]. When metabolic syndrome is present, it can trigger other pathological changes in the body. In these cases, the body cannot metabolize fat properly, which may lead to an imbalance in the secretion of adipokines [23]. High levels of pro-inflammatory cytokines in the body can negatively affect hepatic lipid metabolism, leading to increased LDL-C and decreased HDL-C levels [24]. This mechanism, known as the "vicious cycle of metabolic inflammation", prevents the body from metabolizing lipids properly. Therefore, patients with the disease studied in this paper have a higher risk of cardiovascular disease. Our observation of dyslipidemia in PCOS patients with MS is supported by previous studies that showed similar lipid abnormality patterns in the context of metabolic inflammation [25].

Logistic regression analysis identified several independent risk factors for MS in PCOS patients. The highest odds ratio was for elevated triglycerides, suggesting that hypertriglyceridemia may be a core driver of MS in this population. Increased waist circumference, higher HOMA-IR, lower HDL-C, and elevated TSH were also significantly associated with MS risk, highlighting the interrelationships between obesity, insulin resistance, dyslipidemia, and thyroid dysfunction in the pathogenesis of MS among PCOS patients.

The observed association between elevated TSH and MS in PCOS patients may involve multiple interrelated mechanisms. Chronic low-grade inflammation is a common feature of PCOS and MS. It may impair thyroid hormone signaling through cytokines such as interleukin-6 and tumor necrosis factor-α, which alter deiodinase activity and pituitary feedback. In addition, insulin resistance may dysregulate the hypothalamic-pituitary-thyroid axis, further elevating TSH levels. These pathways suggest that metabolic inflammation and hormonal cross-actions contribute to thyroid dysfunction in this population, warranting further molecular-level investigation. Our results are consistent with those of Dai et al., who reported that subclinical hypothyroidism in PCOS patients is associated with metabolic abnormalities, suggesting a bidirectional relationship between the two [26]. This effect may be achieved through multiple processes, such as metabolic abnormalities. Inflammatory factors in the body, such as interleukin-6, can affect the thyroid gland, causing thyroid hormone conversion. This factor also regulates pituitary feedback [27, 28]. When the body is in a state of insulin resistance, it affects the secretion of thyroid hormones, leading to thyroid axis dysregulation, which in turn affects the body's metabolic levels. Roos et al. proposed a similar mechanism, finding that even in people with normal thyroid function, the thyroid function is associated with components of metabolic syndrome, suggesting a close interaction between the two [29, 30].

Analysis of the results of this study reveals that there are differences in metabolic levels and thyroid-stimulating hormone levels among different groups of samples. Comparison of hormones such as estradiol and testosterone showed that there were no significant differences in these indicators among different groups of samples. It can be concluded that for the disease studied in this study, the presence of MS does not significantly affect its sex hormones and does not cause significant abnormalities in hormone secretion levels. The results of this study have certain significance for clinical work, indicating that MS does not significantly interfere with the secretion level of sex hormones in the body, but it does significantly interfere with the secretion level of thyroid hormones [31]. This finding is consistent with the meta-analysis results of Zhou et al., which concluded that metabolic syndrome does not significantly change the sex hormone levels in PCOS patients, but may affect reproductive function through other pathways [32]. When analyzing this issue, starting from the physiological background helps us to carry out research. For women, MS can have a certain impact on their reproductive health. When women suffer from this disease, ovarian function will be affected, and in severe cases, it will also affect pregnancy outcomes. MS may affect women's reproductive health through factors other than regulating serum hormone levels, such as changing follicular fluid composition and inducing ovarian inflammation. These changes cannot be reflected by detecting serum hormone levels [33]. It can be argued that normal hormone levels in female patients do not necessarily mean normal reproductive function.

This study has limitations. The sample size of 160 patients selected in this study is relatively small, which may lead to bias in the results. Therefore, it is necessary to expand the sample size and clarify whether there are differences between different patient groups. Small sample size may result in the analysis results not representing rare phenotypes, which may make the conclusions of this study lack universality. Future studies will include factors such as geographical location and ethnicity to analyze their impact on the content of this study. This study used a cross-sectional research method and lacked longitudinal data, making it difficult to determine whether there is a causal relationship between different factors. Future studies will consider using a multicenter research method, including a larger sample size and more analytical factors, and exploring disease progression and causal relationships through longitudinal design. When evaluating the results, drug strategies and lifestyle indicators should be included to determine whether they can improve the patient's condition. Furthermore, although we used logistic regression analysis to identify factors associated with MS in patients with PCOS, this study did not consider several potential confounding factors, including genetic polymorphisms related to insulin resistance, dietary patterns, physical activity levels, and body fat distribution patterns. Future prospective studies should incorporate these variables to better elucidate the independent role

of elevated TSH in metabolic syndrome in PCOS patients.

Conclusion

Patients with PCOS have a higher risk of MS, which may lead to various clinical manifestations, including dyslipidemia and insulin resistance. These patients often have thyroid dysfunction but normal reproductive hormone levels. This study suggests that early screening and effective interventions in these patients can improve their endocrine and metabolic levels. Clinicians can employ various scientifically sound intervention strategies, including pharmacological and lifestyle interventions as needed, to maintain stable metabolic levels, reduce the probability of developing diabetes, and decrease the incidence of cardiovascular disease. Future research should explore targeted therapies (e.g., GLP-1 analogs and selenium supplementation) to address thyroid dysfunction associated with MS in PCOS patients.

Acknowledgements

This study was supported by the Key Discipline of Shanghai Pudong New Area Health Commission (No. PWZxk2022-28).

Disclosure of conflict of interest

None.

Address correspondence to: Xiaoting Li, Department of Gynecology, Shanghai Pudong New Area People's Hospital, No. 490, Chuanhuan South Road, Chuansha New Town, Pudong New Area, Shanghai 201299, China. E-mail: lixiaoting@shpdph.com

References

[1] Stańczak NA, Grywalska E and Dudzińska E. The latest reports and treatment methods on polycystic ovary syndrome. Ann Med 2024; 56: 2357737.

[2] Pereira-Eshraghi CF and Vuguin PP. Polycystic ovary syndrome. Pediatr Rev 2024; 45: 363-365.

[3] Dubey P, Reddy S, Sharma K, Johnson S, Hardy G and Dwivedi AK. Polycystic ovary syndrome, insulin resistance, and cardiovascular disease. Curr Cardiol Rep 2024; 26: 483-495.

[4] Benham JL, Goldberg A, Teede H and Tay CT. Polycystic ovary syndrome: associations with cardiovascular disease. Climacteric 2024; 27: 47-52.

[5] Bila J, Dotlic J, Andjic M, Ivanovic K, Micic J, Tulic L, Pupovac M, Stojnic J, Vukovic I and Ivanovic S. Obesity as a part of polycysric ovary syndrome (PCOS)-a review of pathophysiology and comprehensive therapeutic strategies. J Clin Med 2025; 14: 5642.

[6] Genazzani AD and Genazzani AR. Polycystic ovary syndrome as metabolic disease: new insights on insulin resistance. touchREV Endocrinol 2023; 19: 71-77.

[7] Mousa S, Saif A, Fathy M, Mansour M, Abd Elhamid AM, Atef A, Galal M, Saad S and Aboulsoud S. Assessment of early vascular changes in adult females with polycystic ovary syndrome: correlation with insulin resistance. Gynecol Endocrinol 2023; 39: 2210226.

[8] Ashraf N, Qayyum A, Bashir R, Zubair Ahmed S, Shafiq M, Batool M, Sahil M and Kakar MN. Metabolic dysregulation and female infertility: a systematic review of hormonal and reproductive outcomes from recent clinical trials. Cureus 2025; 17: e90887.

[9] Zhao H, Zhang J, Cheng X, Nie X and He B. Insulin resistance in polycystic ovary syndrome across various tissues: an updated review of pathogenesis, evaluation, and treatment. J Ovarian Res 2023; 16: 9.

[10] Mahabamunuge J, Sekula NM, Lepore C, Kudrimoti M, Upadhyay A, Alshowaikh K, Li HJ, Seifer DB and AlAshqar A. The molecular basis of polycystic ovary syndrome and its cardiometabolic correlates: exploring the intersection and its clinical implications-a narrative review. Biomedicines 2025; 13: 709.

[11] Geraci G, Riccio C, Oliva F, Gabrielli D, Colivicchi F, Grimaldi M, Facchinetti F and Unfer V. Women with PCOS have a heightened risk of cardiometabolic and cardiovascular diseases: statement from the experts group on inositol in basic and clinical research and PCOS (EGOI-PCOS) and Italian association of hospital cardiologists (ANMCO). Front Cardiovasc Med 2025; 12: 1520490.

[12] Wang Q, Zhao R, Han C, Huang Z, Bi Y, Zhang X and Shen S. Correlation between thyroid hormone sensitivity and the risk of polycystic ovary syndrome. BMC Endocr Disord 2024; 24: 76.

[13] Palomba S, Colombo C, Busnelli A, Caserta D and Vitale G. Polycystic ovary syndrome and thyroid disorder: a comprehensive narrative review of the literature. Front Endocrinol (Lausanne) 2023; 14: 1251866.

[14] Alberti KG, Zimmet P and Shaw J. Metabolic syndrome--a new world-wide definition. A consensus statement from the international diabetes federation. Diabet Med 2006; 23: 469-480.

[15] Legro RS, Arslanian SA, Ehrmann DA, Hoeger KM, Murad MH, Pasquali R and Welt CK. Diagnosis and treatment of polycystic ovary syndrome: an endocrine society clinical practice guideline. J Clin Endocrinol Metab 2013; 98: 4565-4592.

[16] Biernacka-Bartnik A, Kocełak P, Owczarek AJ, Choręza PS, Markuszewski L, Madej P, Puzianowska-Kuźnicka M, Chudek J and Olszanecka-Glinianowicz M. The cut-off value for HOMA-IR discriminating the insulin resistance based on the SHBG level in women with polycystic ovary syndrome. Front Med (Lausanne) 2023; 10: 1100547.

[17] Gusain N, Anjankar AP, Ambad RS, Jha RK and Jha R. Study the correlation between serum caspase-1, IL-10, HOMA-IR, FAI, dyslipidemia, and oxidative stress status in women with polycystic ovary syndrome (PCOS). J Pharm Bioallied Sci 2025; 17: S921-S923.

[18] Houston EJ and Templeman NM. Reappraising the relationship between hyperinsulinemia and insulin resistance in PCOS. J Endocrinol 2025; 265: e240269.

[19] Areloegbe SE, Peter MU, Oyeleke MB and Olaniyi KS. Low-dose spironolactone ameliorates adipose tissue inflammation and apoptosis in letrozole-induced PCOS rat model. BMC Endocr Disord 2022; 22: 224.

[20] Joshi A. PCOS stratification for precision diagnostics and treatment. Front Cell Dev Biol 2024; 12: 1358755.

[21] Wang K, Li Y and Chen Y. Androgen excess: a hallmark of polycystic ovary syndrome. Front Endocrinol (Lausanne) 2023; 14: 1273542.

[22] Olejarz M, Szczepanek-Parulska E and Ruchala M. Lipoprotein alterations in endocrine disorders - a review of the recent developments in the field. Front Endocrinol (Lausanne) 2024; 15: 1354098.

[23] Koleva-Tyutyundzhieva D, Ilieva-Gerova M, Deneva T and Orbetzova M. Metabolic and inflammatory adipokine profiles in PCOS: a focus on adiposity, insulin resistance, and atherogenic risk. Int J Mol Sci 2025; 26: 9702.

[24] Lee HC, Cheng WC, Ma WL, Lin YH, Shin SJ and Lin YH. Association of lipid composition and unsaturated fatty acids of VLDL with atrial remodeling in metabolic syndrome. Sci Rep 2023; 13: 6575.

[25] Nowak-Ciołek M, Stachowiak JJ, Krok K, Sokal J, Malczyk Ż, Skrzyńska K and Zachurzok A. Adolescent PCOS and long-term metabolic risk: insights from triglycerides to high-density lipoprotein cholesterol ratio and high-density lipoprotein cholesterol profiles. Front Endocrinol (Lausanne) 2025; 16: 1579217.

[26] Dai F, Wu Z, Qin S, Wu F, Wang M, Liu X and Liu F. The influence of subclinical hypothyroidism on endocrine and metabolic characteristics in patients with polycystic ovary syndrome. Int J Womens Health 2025; 17: 1019-1026.

[27] Rudnicka E, Suchta K, Grymowicz M, Calik-Ksepka A, Smolarczyk K, Duszewska AM, Smolarczyk R and Meczekalski B. Chronic low grade inflammation in pathogenesis of PCOS. Int J Mol Sci 2021; 22: 3789.

[28] Shi D, Du J, Kang H, Feng L and Liu F. The effect of subclinical hypothyroidism on hormonal and metabolic profiles and ovarian morphology in patients with polycystic ovary syndrome: a cross-sectional study. Gynecol Endocrinol 2024; 40: 2358219.

[29] Ach T, Dhaffar R, Ben Abdessalem F, Saafi W, Halloul I, ElFekih H, Saad G and Hasni Y. Subclinical hypothyroidism in polycystic ovary syndrome: prevalence and impact on metabolic and cardiovascular risk. Clin Med Insights Endocrinol Diabetes 2025; 18: 117955142513-43678.

[30] Roos A, Bakker SJ, Links TP, Gans RO and Wolffenbuttel BH. Thyroid function is associated with components of the metabolic syndrome in euthyroid subjects. J Clin Endocrinol Metab 2007; 92: 491-496.

[31] Abbasi-Ranjbar Z, Sharami SH, Kazemi S, Sayyad-Abdi D and Dalil Heirati SF. The relation between free testosterone and components of metabolic syndrome in women with polycystic ovary syndrome. J Family Reprod Health 2018; 12: 1-7.

[32] Zhou L, Han L, Liu M, Lu J and Pan S. Impact of metabolic syndrome on sex hormones and reproductive function: a meta-analysis of 2923 cases and 14062 controls. Aging (Albany NY) 2020; 13: 1962-1971.

[33] Li XL, Ji YF, Feng Y and Liu SW. Metabolic disparities between obese and non-obese patients with polycystic ovary syndrome: implications for endometrial receptivity indicators. Gynecol Endocrinol 2024; 40: 2312895.