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ORIGINAL RESEARCH

The Influence of Subclinical Hypothyroidism on Endocrine and Metabolic Characteristics in Patients with Polycystic Ovary Syndrome

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Objective: To analyze the influence of subclinical hypothyroidism (SCH) on endocrine and metabolic characteristics in patients with polycystic ovary syndrome (PCOS).

Methods: A total of 198 cases of PCOS patients admitted to our hospital from January 2022 to December 2023 were selected for the study. According to the presence or absence of SCH, patients were divided into the SCH group (n = 40) and the non-SCH group (n = 158). Differences in demographic characteristics, thyroid hormones, sex hormones, and glucose and lipid metabolism were analyzed between the two groups. Additionally, the correlation between SCH and various endocrine and metabolic indicators in PCOS patients was assessed.

Results: There was no statistically significant difference in demographic characteristics between the two groups ($P > 0.05$). Compared to the non-SCH group, the SCH group had significantly higher levels of serum luteinizing hormone (LH), prolactin (PRL), triglycerides (TG), total cholesterol (TC), fasting insulin (FINS), and homeostasis model assessment of insulin resistance (HOMA-IR) ($P < 0.05$), Conversely, the SCH group had significantly lower levels of serum follicle-stimulating hormone (FSH), estradiol (E2), thyroid-stimulating hormone (TSH), free triiodothyronine (FT3), and free thyroxine (FT4) ($P < 0.05$). Correlation analysis indicated that LH, FSH, E2, PRL, TSH, TG, TC, FINS, HOMA-IR were positively correlated with PCOS complicated by SCH, while FT3, FT4 were negatively correlated.

Conclusion: SCH in PCOS patients is associated with endocrine and metabolic dysfunction, primarily affecting thyroid hormone levels, sex hormones, and glucose and lipid metabolism. Assessing thyroid function is essential for the comprehensive evaluation and management of PCOS patients.

Keywords: subclinical hypothyroidism, polycystic ovary syndrome, endocrine, metabolic characteristics

Introduction

Polycystic ovary syndrome (PCOS) is a common endocrine and metabolic disorder in women of childbearing age, with an incidence of about 6–10%.[1] The etiology of PCOS is complex and is primarily associated with insulin resistance, which affects glucose and lipid metabolism and increases the risk of cardiovascular diseases.[2] Clinically, PCOS presents with heterogeneous symptoms, including menstrual irregularities, acne, hirsutism, and infertility.

Subclinical hypothyroidism (SCH) refers to a type of endocrine and metabolic disorder characterized by elevated thyroid-stimulating hormone (TSH) levels with normal thyroid hormone concentrations, often occurring without overt clinical symptoms.[3] The reported incidence of SCH in the general population ranges from 4–10%.[4] However, studies have shown that SCH is more prevalent in PCOS patients, with a reported prevalence of 20–35%,[5] significantly higher than in healthy women.[6] These results suggest a close relationship between PCOS

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and SCH, as both conditions share similarities in etiology, clinical symptoms, and pathophysiology, particularly regarding endocrine disturbances and metabolic dysfunction.[7]

Despite this association, the endocrine and metabolic characteristics of PCOS patients with SCH remain controversial, and their potential interactions require further investigation. This study aims to evaluate the impact of SCH on endocrine and metabolic parameters in PCOS patients and to explore the relationship between SCH and metabolic dysfunction in PCOS patients. By providing a comprehensive analysis, this study seeks to enhance the understanding of SCH in PCOS and contribute to improving diagnostic and therapeutic strategies in clinical practice.

Materials and Methods

Study Design and Participants

A total of 198 cases of PCOS patients admitted to our hospital from January 2022 to December 2023 were selected for the study. According to the presence or absence of SCH, the patients were divided into the SCH group and the non-SCH group. Diagnostic criteria:[8] (1) PCOS diagnosis refers to the criteria in the literature: Rare occurrence of ovulation or absence of ovulation; Symptoms related to hyperandrogenism (hirsutism, acne, and alopecia) or hyperandrogenemia; Ovaries showing polycystic changes. Any two of the above three criteria can confirm the diagnosis of PCOS. (2) The diagnostic criteria for SCH refer to the criteria in the literature,[9] that is, an increase in TSH, but normal levels of free triiodothyronine (FT3) and free thyroxine (FT4) in the blood, and excluding pituitary and hypothalamic lesions. The following criteria must be met for patients to be eligible for inclusion in this study: (1) Meet the above diagnostic criteria; (2) Aged 18–45 years; (3) Able to cooperate with the tests; (4) Complete clinical data. Exclusion criteria: (1) Menstrual disorders, polycystic ovaries, or sex hormone abnormalities caused by other reasons; (2) Hyperthyroidism or hypothyroidism; (3) Presence of other acute or chronic underlying diseases; (4) Females who have had their first menstruation within 3 years; (5) Females who have used drugs that can affect the study results in the past 3 months; (6) Patients diagnosed with clinical hypothyroidism; (7) Pregnancy. This was approved by the institutional medical ethics committee and obtained informed consent from the patients or their families. The differences in demographic characteristics, thyroid hormones, sex hormones, and glucose and lipid metabolism between the two groups were observed, and the correlation between PCOS with SCH and endocrine and metabolic indicators was analyzed.

Physical Examination

All physical examinations were conducted by the same researcher to ensure consistency. The main questionnaire collected medical history and physical characteristics, including age, age at menarche, parity, number of deliveries, and the duration of PCOS and SCH. Anthropometric measurements, including weight, height, hip circumference, and waist circumference, were recorded for all patients. The body index (BMI) was calculated using the formula: BMI = weight (kg)/height $(m)^2$. Similarly, the waist-to-hip ratio (WHR) was calculated as: WHR = waist circumference (cm)/hip circumference (cm). These measurements were used to assess body composition and metabolic risk factors in patients with PCOS and SCH.

Thyroid Hormone and Sex Hormone Index Detection

All patients were fasted overnight for 10–12 hours and had fasting blood collection on the next morning at 8:00 on the 3rd-5th day of the menstrual cycle (no restriction on the testing date for amenorrhea). The German Roche Elecsys 2010 electrochemiluminescence equipment was used for the determinations. The sex hormone indicators included luteinizing hormone (LH), testosterone (T), follicle-stimulating hormone (FSH), estradiol (E2), prolactin (PRL), etc. The thyroid hormone indicators included TSH, FT3, FT4, etc. Each indicator was measured 3 times, and the mean value of the 3 results was taken as the final result.

Glucose and Lipid Metabolism Index Detection

All patients were fasted overnight for 10–12 hours and had fasting blood collection on the next morning at 8:00 on the 3rd-5th day of the menstrual cycle (no on the testing date for amenorrhea). The German Roche Elecsys 2010 electrochemiluminescence equipment was used for the determinations. The lipid indicators included triglyceride (TG), total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C). The glucose indicators included fasting blood glucose (FBG), fasting insulin (FINS), oral glucose tolerance test (OGTT),

and homeostasis model assessment of insulin resistance (HOMA-IR), etc. Each indicator was measured 3 times, and the mean value of the 3 results was taken as the final result. HOMA-IR = FBG × FINS/22.5. The OGTT was performed 1 hour and 2 hours after ingesting 75 g of glucose powder.

Statistical Methods

SPSS 25.0 statistical software was used for data analysis. Categorical data were expressed as frequency (n) and percentage (%). If the sample size was ≥40 and the theoretical frequency (T) ≥ 5, the chi-square (χ^2) test was used. If the sample size was ≥ 40, but 1 ≤ T < 5, the chi-square test with continuity correction was used. When the sample size was < 40 or T < 1, Fisher's exact test was used. For continuous variables, normally distributed measurement data were expressed as mean ± standard deviation (SD) and analyzed using the *t*-test. Non-normally distributed measurement data were expressed as median (25th percentile, 75th percentile) and analyzed using the Mann–Whitney *U*-test. Pearson correlation analysis was used to analyze the correlation between PCOS with SCH and various indicators. $P < 0.05$ was considered statistically significant.

Results

Patient Demographics

Among a total of 198 PCOS patients, 40 were included in the SCH group, accounting for 20.20% (40/198), while the remaining 158 were included in the non-SCH group, accounting for 79.80% (158/198). There were no statistically significant differences in age, age at menarche, parity, PCOS duration, and SCH duration between the two groups ($P > 0.05$), but there were statistically significant differences in BMI and waist-to-hip ratio (WHR) ($P < 0.05$). The BMI and WHR of the SCH group were higher than those of the non-SCH group ($P < 0.05$) (Table 1), indicating that SCH can affect the BMI and WHR of PCOS patients.

The study compared FT3, FT4, thyroid hormone levels and sex hormone indices between the two groups and found that there were no statistically significant differences in serum T levels between the two groups ($P > 0.05$), but there were statistically significant differences in serum LH, FSH, E2, PRL, TSH, FT3, and FT4 levels ($P < 0.05$). The serum TSH, LH and PRL levels of the SCH group were higher than those of the non-SCH group ($P < 0.05$), while the serum FSH, E2, levels were lower than those of the non-SCH group ($P < 0.05$) (Table 2), indicating that SCH can affect the thyroid hormone and sex hormone levels of PCOS patients.

Table 1 Comparison of Indicators Between the Two Groups

Parameter	SCH Group (n = 40)	Non-SCH Group (n = 158)	t	*P* value
Age (years)	26.31 ± 5.74	26.75 ± 5.85	0.434	0.666
Age at menarche (years)	13.74 ± 2.18	14.12 ± 2.25	0.975	0.334
Parity (times)	1.14 ± 0.38	1.26 ± 0.35	1.824	0.073
Gravidity (times)	0.78 ± 0.25	0.74 ± 0.15	0.931	0.357
PCOS duration (years)	3.45 ± 0.52	3.53 ± 0.44	0.833	0.409
SCH duration (years)	1.21 ± 0.24	1.15 ± 0.18	1.6	0.116
BMI (kg/m^2)	24.65 ± 5.67	20.75 ± 4.98	3.975	< 0.001
WHR	0.91 ± 0.48	0.68 ± 0.39	2.731	0.009
LH (mU/mL)	10.45 ± 1.05	9.78 ± 1.02	3.628	< 0.001
T (nmol/L)	1.28 ± 0.81	1.34 ± 0.65	0.413	0.681
FSH (μIU/mL)	5.62 ± 0.85	6.04 ± 0.91	2.773	0.007
E_2 (nmol/L)	147.32 ± 20.52	176.85 ± 31.74	7.184	< 0.001
PRL (μIU/mL)	430.56 ± 65.42	263.52 ± 56.32	14.819	< 0.001
TSH (μIU/mL)	6.67 ± 1.51	3.15 ± 1.72	3.774	< 0.001
FT_3 (nmol/L)	5.37 ± 0.55	5.28 ± 0.79	6.344	< 0.001
FT_4 (nmol/L)	16.82 ± 1.53	16.42 ± 1.35	11.926	< 0.001

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Table 2 Comparison of Thyroid Hormone and Sex Hormone Indices Between the Two Groups

Group	LH (mU/mL)	T (nmol/L)	FSH (μIU/mL)	E_2 (nmol/L)	PRL (μIU/mL)	TSH (μIU/mL)	FT_3 (nmol/L)	FT_4 (nmol/L)
SCH (n=40)	10.45 ± 1.05	1.28 ± 0.81	5.62 ± 0.85	147.32 ± 20.52	430.56 ± 65.42	2.16 ± 1.41	4.51 ± 0.65	13.77 ± 1.23
Non-SCH (n=158)	9.78 ± 1.02	1.34 ± 0.65	6.04 ± 0.91	176.85 ± 31.74	263.52 ± 56.32	3.15 ± 1.72	5.28 ± 0.79	16.42 ± 1.35
t	3.628	0.413	2.773	7.184	14.819	3.774	6.344	11.926
P	< 0.001	0.681	0.007	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001

Table 3 Correlation Analysis Between PCOS With SCH and Various Indices

Index	PCOS with SCH	
	r	*P* value
LH	0.504	0.026
FSH	0.482	0.035
E_2	0.598	0.001
PRL	0.636	0.001
TSH	0.605	0.001
FT_3	−0.522	0.020
FT_4	−0.627	0.001
TG	0.640	0.001
TC	0.539	0.012
FINS	0.571	0.001
HOMA-IR	0.640	0.001

Glycolipid Metabolism Indices

There were no statistically significant differences in serum HDL-C, LDL-C, fasting blood glucose (FBG), OGTT 1-hour, and OGTT 2-hour levels between the two groups ($P > 0.05$), but there were statistically significant differences in serum TG, TC, fasting insulin (FINS), and homeostasis model assessment of insulin resistance (HOMA-IR) levels ($P < 0.05$) (Table 2). The serum TG, TC, FINS, and HOMA-IR levels of the SCH group were higher than those of the non-SCH group, indicating that SCH can affect the glycolipid metabolism indices of PCOS patients.

Correlation Analysis Between PCOS with SCH and Various Indices

As shown in Table 3, correlation analysis revealed positive correlations between LH, FSH, E2, PRL, TSH, TG, TC, FINS, HOMA-IR, and PCOS with SCH, and negative correlations between FT3, FT4, and PCOS with SCH, indicating that PCOS with SCH is associated with endocrine and glycolipid metabolism abnormalities.

Discussion

Patients with PCOS often exhibit a series of endocrine abnormalities such as obesity, insulin resistance, and metabolic dysfunction.[10] The relationship between PCOS and another type of endocrine disorder, SCH, has increasingly become a focus in clinical research.[11] After the occurrence of SCH, the body's metabolic capacity significantly decreases, leading to menstrual disorders, PCOS, and infertility through various mechanisms.[12] The energy and glucose and lipid metabolism balance within the body mainly relies on thyroid hormones. When there are abnormal changes in thyroid function, a series of metabolic abnormalities can occur. Therefore, it is necessary to determine the presence of hypothyroidism during the diagnosis of PCOS patients.

In this study, among the 198 PCOS patients, 40 cases were combined with SCH, accounting for 20.20%, which is consistent with the study by Elkind-Hirsch,[13] but lower than the study by Łagowska.[14] The different conclusions in different studies may be related to differences in sample populations and age distributions. Johnson[15] found that PCOS

patients had increased BMI and WHR after developing SCH. The BMI and WHR in the combined SCH group were higher than those in the non-combined SCH group ($P<0.05$), consistent with the above study, suggesting that the occurrence of SCH in PCOS patients can lead to obesity. The reason for this may be that after the occurrence of SCH, the metabolic rate of PCOS patients decreases significantly, resulting in a significant reduction in calorie burning and energy accumulation, leading to an increase in BMI and WHR.[16]

In this study, patients in the SCH group had significantly higher LH levels compared to the non-SCH group, which may be related to the progression of PCOS. Previous studies have shown that obesity is an important factor contributing to the severity of PCOS, and it may lead to elevated LH levels by affecting the hypothalamus-pituitary-ovary axis.[10] We observed that patients in the SCH group had higher BMI and higher LH levels, which is consistent with more severe PCOS. Moreover, studies have shown that obesity is closely associated with a slight increase in TSH levels.[17] This increase may be due to the effects of obesity-related hormones (such as leptin) on the hypothalamus-pituitary-thyroid axis. Additionally, the slight elevation in TSH levels in obese patients may reflect an adaptive change in metabolic function.

Thyroid hormones and sex hormones are common reference indicators for clinical judgment of endocrine abnormalities.[18] In this study, serum LH and PRL levels were significantly higher in the SCH group compared to the non-SCH group, while FSH, E2, TSH, FT3, and FT4 levels were significantly lower. These findings are consistent with previous research,[19] further suggesting that SCH is associated with the altered endocrine profiles in PCOS patients. The observed abnormalities in sex hormone levels (LH, PRL, FSH, and E2) may be attributed to reduced thyroid hormone levels, which impair aromatase production and activity, a key enzyme involved in estrogen synthesis.[20] Consequently, this could lead to a decrease in FSH, E2, and LH levels, disrupting normal ovarian function. Furthermore, low FT4 levels may negatively regulate the hypothalamic-pituitary-thyroid axis, leading to increased TSH production, and the hypothalamic-pituitary-gonadal axis can interact with each other, leading to increased PRL secretion.[21] Additionally, changes in the gonadotropin-releasing hormone regulatory environment could result in abnormal TSH production, which further interferes with thyroid axis function, leading to decreased FT3 and FT4 levels.[22,23]

Beyond endocrine disturbances, SCH in PCOS patients is also associated with metabolic dysfunction, particularly in blood glucose and lipid indicators.[24] This study also found that the serum levels of TG, TC, FINS, and HOMA-IR in the PCOS with SCH group were higher than those in the non-combined SCH group, indicating that combined SCH can cause disorders in glucose and lipid metabolism. The underlying mechanism may be related to reduced intestinal glucose absorption due to thyroid hormone deficiency, resulting in higher fasting insulin levels, impaired glucose utilization in the liver, muscle, and adipose tissues, and a decrease in gluconeogenesis and insulin resistance.[25] Furthermore, the decreased FT3 and FT4 levels in the liver and muscle fat directly affect the anti-lipolysis effect of insulin.[26] Moreover, the activity of lipase in patients tends to decrease, affecting the clearance of blood lipids and reducing the excretion of cholesterol and bile acids in blood lipids, ultimately leading to increased blood lipid levels.[27]

Notably, given that both SCH and PCOS are major causes of amenorrhea, their potential etiological relationship cannot be overlooked. Thyroid dysfunction may disrupt the hypothalamic-pituitary-ovarian axis, leading to altered gonadotropin secretion and menstrual irregularities in PCOS patients.[28] Additionally, shared metabolic disturbances, such as insulin resistance and chronic low-grade inflammation, may contribute to the coexistence of these conditions, exacerbating endocrine dysfunction.[29] While our study primarily explores the association between SCH and PCOS-related endocrine and metabolic abnormalities, it does not establish causality. These findings highlight the clinical importance of thyroid function screening in PCOS patients, particularly those with amenorrhea, obesity, or insulin resistance. Further research, including prospective and interventional studies, is needed to determine whether thyroid hormone replacement therapy could improve reproductive and metabolic outcomes in PCOS patients with SCH.

The innovation of this study lies in the observation of the correlation between SCH and PCOS-related endocrine and metabolic indicators. Unlike previous studies that focused on individual metabolic parameters, our study integrates a broad range of hormonal and metabolic markers, providing a more detailed understanding of the SCH-PCOS

relationship. Furthermore, our study highlights the importance of distinguishing between direct thyroid-related effects and obesity-related metabolic disturbances, which has been underexplored in prior research. These findings offer a new perspective on the pathophysiological interplay between SCH and PCOS.

However, this study still has limitations. Firstly, we selected PCOS patients who received treatment in our hospital during a specific time range, which may limit the sample representativeness. Secondly, the retrospective cohort study design cannot completely eliminate potential confounding factors and information bias, despite our efforts to collect comprehensive data for both groups. Additionally, the study setting within a single institution may limit the generalizability of the results to broader populations with diverse clinical backgrounds and healthcare practices. Moreover, the specific mechanisms through which SCH influences metabolism and endocrine function in PCOS patients require further analysis. Future studies should address these limitations by employing prospective, multicenter studies with larger sizes to improve generalizability and reduce potential biases. Additionally, mechanistic studies are needed to elucidate the underlying biological pathways linking SCH and PCOS.

Collectively, this study provides substantial support for the diagnosis and treatment of PCOS, contributing to a better understanding of its endocrine and metabolic implications and offering new directions for future research.

Conclusion

In conclusion, SCH can affect endocrine and metabolic indicators in PCOS patients, mainly manifested as thyroid hormone, sex hormone, and glucose and lipid metabolism. Determining thyroid function is of great significance for PCOS patients. In future clinical work, thyroid-related functional status tests should be conducted in PCOS patients to mitigate endocrine and metabolic disturbances caused by combined SCH, ultimately improving reproductive and metabolic outcomes in this population.

Data Sharing Statement

All data generated or analyzed in this study are included in the present manuscript.

Ethics and Informed Consent Statement

This study was approved by the Ethics Committee of the First Affiliated Hospital of Guizhou University of Traditional Chinese Medicine and obtained informed consent from the patients. In cases where patients were unable to provide consent themselves, consent was obtained from their families in accordance with ethical guidelines.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors have no conflicts of interest to declare.

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