Nature and Science of Sleep

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ORIGINAL RESEARCH

Prevalence and Associative Analysis of Thyroid Dysfunction in Patients with Obstructive Sleep Apnea Hypopnea Syndrome: A Cross-Sectional Study

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Objective: Obstructive sleep apnea-hypopnea syndrome (OSAHS) is closely associated with hypothyroidism. This study aimed to evaluate the prevalence and correlation of hypothyroidism in patients with OSAHS.

Methods: This investigation included a cohort of 1693 patients newly diagnosed with OSAHS, all of whom underwent polysomnography and thyroid function assessments.

Results: The study cohort comprised 1693 OSAHS patients (60.4% male). No significant differences in thyroid-stimulating hormone (TSH) or free thyroxine (FT4) levels were observed across OSAHS severity groups ($p = 0.670$ and $p = 0.307$). Notably, patients with overt hypothyroidism demonstrated significantly lower lowest oxygen saturation ($LSpO_2$) levels than their euthyroid counterparts ($p = 0.035$). The analysis did not reveal any significant correlations between TSH/FT4 levels and the apnea-hypopnea index, mean oxygen saturation ($MSpO_2$), or $LSpO_2$. Nevertheless, TSH levels were positively correlated with female sex and age ($p = 0.001$), while no correlation was found with body mass index. After controlling for sex and age, TSH was positively correlated with apnea-hypopnea index ($p = 0.002$) and negatively correlated with both $MSpO_2$ and $LSpO_2$ ($p = 0.001$). In contrast, FT4 levels exhibited a negative correlation with apnea-hypopnea index ($p = 0.016$) and positive correlations with $MSpO_2$ and $LSpO_2$ ($p = 0.030$ and $p = 0.018$). The overall prevalence rates of hypothyroidism and overt hypothyroidism within the study population were determined to be 13.7% and 3.8%, respectively, with no significant differences observed across the severity categories of OSAHS ($p = 0.166$ and $p = 0.193$). Subclinical hypothyroidism was identified in 9.9% of the patients, with a notably lower prevalence in those with severe OSAHS compared to those with milder forms of the condition ($p = 0.004$).

Conclusion: In patients with OSAHS, 13.7% had hypothyroidism, with 3.8% being overt hypothyroidism and 9.9% being subclinical hypothyroidism. Thyroid function parameters are associated with the severity of OSAHS and are influenced by sex and age.

Keywords: sleep apnea, obstructive, hypothyroidism, prevalence, correlation

Introduction

Obstructive sleep apnea-hypopnea syndrome (OSAHS) is a common sleep-related breathing disorder characterized by recurrent episodes of partial or complete obstruction of the upper airway during sleep, resulting in interruptions in breathing and hypoventilation.[1] The prevalence of OSAHS is significant, affecting approximately 936 million individuals globally between the ages of 30 and 69, with an estimated 176 million cases reported in China, reflecting a prevalence rate of 23.6%.[2] Recognized risk factors for OSAHS include obesity, advanced age, and male sex.[3,4] OSAHS has the potential to result in functional deterioration or even failure of various organ systems.[5]

Received: 7 January 2025
Accepted: 4 June 2025
Published: 23 June 2025

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Hypothyroidism (HT), which includes both overt (OH) and subclinical (SCH) forms, is characterized by a hypometabolic state resulting from either absolute or relative deficiency of thyroid hormones.[6] Recent studies indicate a bidirectional relationship between OSAHS and HT. A large cross-sectional study involving 5515 participants found a prevalence of 9.47% for HT in the general population of the United States, with regression analyses revealing significant associations between HT and sleep apnea.[7] Notably, 25–30% of patients with primary HT also exhibit comorbid OSAHS,[8,9] which can exacerbate the severity of the disease[10] and increase cardiovascular morbidity and mortality.[4,11] While the potential for OSAHS to induce thyroid dysfunction is theoretically plausible, current clinical evidence remains inconclusive regarding its role as a causative factor in the development of HT. Although SCH is more prevalent in populations with OSAHS, the clinical implications of this association on disease progression warrant further investigation.

This study aims to conduct a retrospective analysis of the prevalence of HT among patients diagnosed with OSAHS at a single medical center. Additionally, the study will compare the demographic characteristics and specific clinical data of patients exhibiting varying degrees of OSAHS alongside those with HT, thereby examining the relationship between HT and OSAHS.

Patients and Methods

This research was carried out at the Sleep Center of the First People's Hospital of Yunnan Province. The study population consisted of patients who were initially diagnosed with OSAHS through polysomnography (PSG) using the Philips Respironics Alice 6 system at the aforementioned hospital between 2018 and 2021, and who also underwent thyroid function assessments. Eligible participants were individuals aged 18 years or older who satisfied the diagnostic and severity criteria for adult OSAHS as specified in the International Classification of Sleep Disorders, 3rd edition (ICSD-3).[1] Exclusion criteria encompassed: 1) individuals with severe organic diseases, including heart failure, hepatic failure, or renal failure; 2) individuals with chronic respiratory conditions such as chronic obstructive pulmonary disease, bronchial asthma, or interstitial lung disease; 3) individuals with a history of thyroid disorders or newly diagnosed hyperthyroidism; 4) individuals with known autoimmune diseases; 5) pregnant individuals; and 6) individuals with incomplete data. This study received approval from the Institutional Review Board (IRB)/Ethics Committee of the First People's Hospital of Yunnan Province (Approval No.: KHLL2023-KY002) and adhered rigorously to the ethical principles outlined in the Declaration of Helsinki. Given that the study utilized de-identified data obtained from existing electronic medical records and presented no more than minimal risk to participants, the requirement for informed consent was waived.

Study Design

This research utilized a retrospective descriptive design to gather data. The demographic characteristics, PSG parameters, and thyroid function indices of eligible patients were obtained through a review of medical records. In accordance with the laboratory testing standards implemented at our facility, which employs chemiluminescent immunoassay techniques, OH was defined as elevated thyroid-stimulating hormone (TSH) levels exceeding 4.2 mIU/L, in conjunction with decreased free thyroxine (FT4) levels below 12 pmol/L. Conversely, SCH was identified by elevated TSH levels while maintaining normal FT4 levels.

Statistical Analysis

Statistical analyses were performed utilizing the SPSS 26.0 software package. Normally distributed data are presented as the mean ± standard deviation (SD), with differences among the three groups assessed through one-way analysis of variance (ANOVA). The Least Significant Difference (LSD) method was employed when variances were homogeneous, while the Games-Howell method was applied in heterogeneous variances. Results are reported as the median (P25, P75) for skewed data, and intergroup comparisons were conducted using the Kruskal–Wallis *H*-test. Categorical data are expressed as the number of cases (percentage), with group comparisons executed via the Chi-square test. Spearman's rho correlation coefficient was used to evaluate the correlation between continuous variables. A p-value of less than 0.05 was deemed indicative of statistically significant findings.

Results

From January 2018 to December 2021, a total of 2161 patients were screened, of which 1693 were ultimately incorporated into the study. The study cohort was comprised of 60.4% males, with a mean age of 49.4 ± 13.8 years, a body mass index (BMI) of 26.6 (23.9, 30.5) kg/m², and an apnea-hypopnea index (AHI) of 32.1 (16.1, 60.7). Figure 1 provides a summary of the patient flow throughout the study.

Table 1 delineates the demographic characteristics and prevalence of thyroid disorders among patients categorized by OSAHS severity. The mean TSH level across the entire study cohort was 2.2 (1.4, 3.2) mIU/L, while the mean FT4 level was recorded at 14.3 ± 3.6 pmol/L. In the cohort with mild OSAHS, there was a notable predominance of female patients and a lower BMI. Conversely, the group with severe OSAHS exhibited a higher proportion of male patients and a greater incidence of hypertension. TSH and FT4 levels did not show significant variation among patients with differing severities of OSAHS. The prevalence of OH across all patients was found to be 3.8%, with no significant differences identified among the three OSAHS severity groups. In contrast, the prevalence of SCH was 9.9%, which was significantly elevated in patients with mild and moderate OSAHS compared to those with severe OSAHS. The overall prevalence of OH and SCH within the study population was 13.7%, with no significant differences noted among the three OSAHS severity categories.

Table 2 delineates the demographic characteristics and clinical profiles of all patients categorized by thyroid function status (euthyroid, SCH, and OH). The groups classified as OH and SCH exhibited a notably higher prevalence of female patients and an older age demographic in comparison to the euthyroid group. Patients diagnosed with OH presented with more pronounced respiratory disturbances, as indicated by significantly elevated AHI scores and reduced lowest oxygen saturation ($LSpO_2$) levels relative to those in the SCH group. Nevertheless, no statistically significant differences were identified in AHI or mean oxygen saturation ($MSpO_2$) between patients with OH and the euthyroid controls.

Table 3 illustrates the Spearman's ρ correlations among demographic characteristics, thyroid function indices, and PSG parameters across the entire patient cohort. The AHI exhibited significant correlations with sex ($r = -0.299$; $p = 0.001$), age ($r = -0.121$; $p = 0.001$), and BMI ($r = 0.380$; $p = 0.001$), while no significant associations were found with thyroid function indices. TSH demonstrated significant correlations with sex ($r = 0.193$; $p = 0.001$) and age ($r = 0.086$; $p = 0.001$). FT4 was correlated with age ($r = -0.094$; $p = 0.001$) but not with sex. Following adjustments for sex and age differences, as presented in Table 4, TSH retained significant correlations with AHI ($r = 0.075$; $p = 0.002$), $MSpO_2$ ($r = -0.116$; $p = 0.001$), and $LSpO_2$ ($r = -0.101$; $p = 0.001$). Likewise, FT4 demonstrated significant associations with AHI ($r = -0.059$; $p = 0.016$), $MSpO_2$ ($r = 0.053$; $p = 0.030$), and $LSpO_2$ ($r = 0.057$; $p = 0.018$). Furthermore, BMI exhibited a strong correlation with AHI ($r = 0.350$; $p = 0.001$) but did not show significant relationships with TSH or FT4.

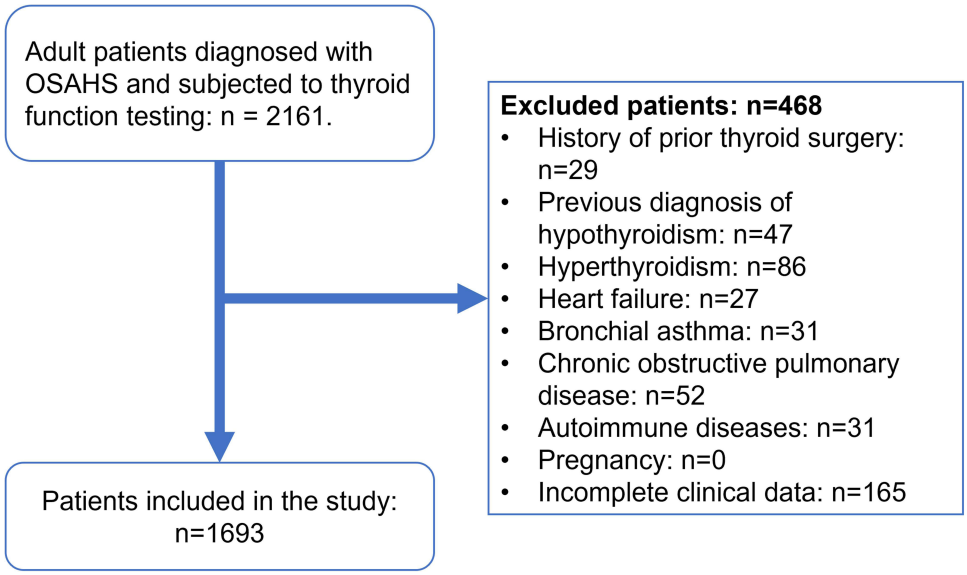

Figure 1 The chart of patient flow through the study.
Abbreviations: OSAHS, obstructive sleep apnea-hypopnea syndrome.

https://doi.org/10.2147/NSS.S515819

Table 1 Demographic Characteristics and Prevalence of Thyroid Disease Among All Patients Stratified by the Severity of OSAHS

Parameters	All Patients n=1693(100%)	OSAHS-Mild 5≤AHI < 15 n=382(22.6%)	OSAHS-Moderate 15≤AHI < 30 n=411(24.3%)	OSAHS-Severe AHI ≥ 30 n=900(53.2%)	*p* value
Sex (male/female)	1023/670 (60.4/39.6%)	154/228 (40.3/59.7%)	213/198 (51.8/48.2%)[a]	656/244 (72.9/27.1%)[ab]	*<0.001*
Age(years)	49.4±13.8	49.8±13.6	51.2±14.2	48.3±13.6[b]	*0.002*
BMI (kg/m^2)	26.6(23.9,30.5)	24.2(21.7,27.6)	25.7(23.3,29.2)[a]	28.0(25.2,31.3)[ab]	*<0.001*
AHI (n)	32.1(16.1,60.7)	9.5(6.9,12.0)	21.4(18.0,25.7)[a]	58.6(41.7,79.0)[ab]	*<0.001*
TSH (mIU/L)	2.2(1.4,3.2)	2.2(1.4,3.4)	2.2(1.4,3.4)	2.2(1.4,3.1)	0.670
FT4(pmol/L)	14.3±3.6	14.4±3.4	14.5±3.3	14.2±3.9	0.307
Diabetes	219(12.9%)	41(10.7%)	54(13.1%)	124(13.8%)	0.328
Hypertension	640(37.8%)	121(31.7%)	149(36.3%)	370(41.1%)[a]	*0.005*
OH (TSH > 4.2 mIU/L, FT4 < 12 pmol/L)	64(3.8%)	12(3.1%)	11(2.7%)	41(4.6%)	0.193
SCH (TSH > 4.2 mIU/L)	168(9.9%)	46(12%)	53(12.9%)	69(7.7%)[ab]	*0.004*
OH+ SCH	232(13.7%)	58(15.2%)	64(15.6%)	110(12.2%)	0.166

Notes: [a]Compared with the mild OSAHS group, $P < 0.05$; [b]Compared with the moderate OSAHS group, $P < 0.05$. Significance values are displayed in italics.
Abbreviations: BMI, body mass index; AHI, apnea-hypopnea index; TSH, thyroid-stimulating hormone; FT4, free thyroxine; SCH, subclinical hypothyroidism; OH, overt hypothyroidism.

Table 2 Comparative Analysis of Demographic Characteristics and Clinical Parameters Stratified by Thyroid Function Status

Parameters	Euthyroid n=1461(86.3%)	OH n=64(3.8%)	SCH n=168(9.9%)	*p* value
Sex (male/female)	924/537 (63.2/36.8%)[a]	29/35 (45.3/54.7%)	70/98 (41.7/58.3%)	*<0.001*
Age(years)	48.8±13.5[b]	51.9±13.4	53.4±15.5	*<0.001*
BMI (kg/m^2)	27.6±5.6	28.2±5.0	26.8±5.6	0.859
TSH (mIU/L)	2.0(1.3,2.7)[ab]	6.9(5.2,23.9)	5.2(4.6,6.5)	<0.001
FT4(pmol/L)	14.7(12.3,16.6)[ab]	9.5(6.7,11.1)	15.3(13.6,16.7)[a]	<0.001
AHI(n)	33.0(16.4,61.4)[b]	43.1(18.0,73.3)[b]	24.1(13.6,44.4)	*<0.001*
$MSpO_2$(%)	91(88,93)	90(87,92)	91(88,92)	0.294
$LSpO_2$(%)	76(65,81)[a]	75(58,78)	75(67,81)[a]	*0.035*

Notes: [a]Compared with the OH group, $P < 0.05$; [b]Compared with the SCH group, $P < 0.05$. Significance values are displayed in italics.
Abbreviations: BMI, body mass index; AHI, apnea-hypopnea index; TSH, thyroid-stimulating hormone; FT4, free thyroxine; SCH, subclinical hypothyroidism; OH, overt hypothyroidism; $MSpO_2$, mean oxygen saturation; $LSpO_2$, lowest oxygen saturation.

Table 3 Spearman's ρ Correlations Between Demographic Characteristics, Thyroid Function Indicators, and PSG Parameters Among All Patients

rho/p	Sex		Age (years)		BMI (kg/m^2)		AHI (n)		$MSpO_2$ (%)		$LSpO_2$ (%)	
TSH(mIU/L)	0.193	*0.001*	0.086	*0.001*	0.004	0.881	−0.024	0.317	0.003	0.887	0.003	0.897
FT4(pmol/L)	0.014	0.562	−0.094	*0.001*	0.025	0.309	−0.017	0.479	−0.003	0.897	0.011	0.653
AHI (n)	−0.299	*0.001*	−0.121	*0.001*	0.380	*0.001*						

Note: Significance values are displayed in italics.
Abbreviations: PSG, polysomnographic; BMI, body mass index; AHI, apnea-hypopnea index; TSH, thyroid-stimulating hormone; FT4, free thyroxine; $MSpO_2$, mean oxygen saturation; $LSpO_2$, lowest oxygen saturation.

Discussion

The findings of this study indicate a significant prevalence of HT among patients diagnosed with OSAHS. The majority of these cases were newly identified instances of OH and SCH in individuals without a previous history of thyroid

Table 4 Partial Spearman's ρ Correlations Between Thyroid Function Indices and PSG Parameters, Adjusted for Sex and Age

rho/p	BMI (kg/m^2)		AHI (n)		$MSpO_2$ (%)		$LSpO_2$ (%)	
TSH(mIU/L)	0.034	0.167	0.075	*0.002*	−0.116	*0.001*	−0.101	*0.001*
FT4(pmol/L)	−0.039	0.112	−0.059	*0.016*	0.053	*0.030*	0.057	*0.018*
AHI (n)	0.350	*0.001*						

Note: Significance values are displayed in italics.
Abbreviations: PSG, polysomnographic; BMI, body mass index; AHI, apnea-hypopnea index; TSH, thyroid-stimulating hormone; FT4, free thyroxine; $MSpO_2$, mean oxygen saturation; $LSpO_2$, lowest oxygen saturation.

surgery or current treatment with thyroid hormone therapy. Additionally, the relationship between HT and the severity of OSAHS was found to be moderated by factors such as sex and age.

The existing literature indicates significant variability in the prevalence of HT among patients with OSAHS. For example, a meta-analysis conducted by Zhang et al[10] identified an overall prevalence of OH at 8.12 ± 7.13% and SCH at 11.07 ± 8.49% within this patient population. Additional clinical studies examining the prevalence of thyroid disorders in OSAHS cohorts have reported OH rates ranging from 1.97% to 10%, SCH rates between 7% and 12%, and newly diagnosed OH rates of 0.4% to 2%.[12–15] These variations may be attributed to differences in the inclusion criteria for study populations, methodologies for assessing thyroid hormone levels, and standards for diagnosing OSAHS. In the current study, which excluded most conditions that could potentially influence thyroid function, the prevalence of HT among OSAHS patients was found to be 13.7%, with SCH at 9.9% and OH at 3.8%. Although non-OSAHS control groups were not included in this analysis, the prevalence of OH observed in this study was notably higher than that reported in the general population of China (3.7% compared to 1.03%).[16] Furthermore, the overall prevalence of HT in this cohort surpassed that documented in the general population of the United States (13.5% versus 9.47%).[7]

Previous research has highlighted sex differences in the prevalence of HT and OSAHS, with females demonstrating a greater vulnerability to HT[6,17] and males exhibiting a higher likelihood of developing OSAHS.[5] The current study reveals a notably elevated prevalence of HT among female patients with OSAHS in comparison to their male counterparts, aligning with the findings of Bahammam et al[12] and Wang et al.[18] This phenomenon may be linked to sex-specific hormonal factors and the inherent predisposition of females to autoimmune conditions,[19,20] although the existing evidence remains insufficient. Additionally, thyroid function plays a significant role in influencing PSG parameters in patients with OSAHS. Previous studies have shown that OH diminishes sex-related differences in PSG outcomes among OSAHS patients, while SCH is primarily associated with increased BMI in female patients relative to males.[12] The influence of sex in OSAHS patients with concurrent hypothyroidism constitutes a vital yet underexplored domain of research.

Previous researchers have systematically delineated the bidirectional relationship between HT and OSAHS through comprehensive reviews.[21] Thyroid hormones play crucial roles in glucose, lipid, and protein metabolism, facilitating mitochondrial oxidative processes, promoting pulmonary surfactant maturation, and regulating the transcription of genes encoding myofibrillar and calcium-regulatory proteins in muscle fibers.[22] HT may induce or exacerbate OSAHS through several pathophysiological mechanisms: (1) deposition of mucopolysaccharides and proteins in the upper airway leading to pharyngeal narrowing;[22,23] (2) impaired contractile function of pharyngeal and respiratory muscles[24] coupled with neurological dysfunction, resulting in ventilatory impairment;[22] (3) metabolic syndrome presenting primarily as obesity;[11,25] and (4) attenuated ventilatory response to hypoxia and hypercapnia in the respiratory center, contributing to central apnea.[10] Conversely, OSAHS may promote thyroid tissue damage and hypothyroidism through intermittent hypoxia, sleep deprivation, and sustained sympathetic activation, which collectively enhance oxidative stress and inflammatory responses.[26,27] Pathophysiologically, HT progression would be expected to correlate with OSAHS severity. However, our study found no significant differences in thyroid function parameters (TSH and FT4) across varying OSAHS severity groups, aligning with findings by Ozcan et al[13] and Bruyneel et al.[14] This suggests that thyroid hormone levels may be modulated by multiple confounding factors.

To evaluate HT's impact on OSAHS, we compared polysomnographic measures between OSAHS patients with and without HT. Our results demonstrated significantly higher AHI levels and more severe hypoxemia in OH patients. These findings contrast with Wang et al,[18] who reported no differences in PSG parameters (AHI, $MSpO_2$, $LSpO_2$) between comparable groups - a discrepancy potentially attributable to their limited HT sample size (n=27) and failure to stratify OH and SCH subgroups.

The SCH-OSAHS relationship remains unclear. One study found no difference in OSAHS prevalence between SCH patients and controls, nor any correlation between TSH levels and respiratory disturbance indices.[28] Intriguingly, our SCH patients exhibited milder OSAHS than euthyroid counterparts - a novel observation potentially mediated by sex and age factors. Furthermore, SCH's transient nature[29] may explain its modest clinical impact on OSAHS severity.

Spearman correlation analysis conducted on the entire cohort of study participants indicated an absence of significant associations between thyroid function parameters and polysomnographic measures. It is noteworthy that both TSH and the AHI exhibited significant correlations with sex and age. Upon adjusting for these confounding variables, significant correlations were identified between TSH/ FT4 and AHI, $MSpO_2$, and $LSpO_2$, thereby providing enhanced evidence for a pathophysiological connection between these conditions. These results imply that the onset of HT in patients with OSAHS may be linked to the extent of systemic hypoxia,[30] while also underscoring the importance of sex and age as critical confounding factors in research pertaining to thyroid function and OSAHS. Conversely, several studies have documented a lack of significant correlations between TSH/FT4 levels and AHI or $LSpO_2$.[13,14,31] The discrepancies in findings may be attributed to methodological limitations, such as the failure to consider potential confounders (including sex, age, obesity, and comorbid chronic conditions) and insufficient sample sizes, which could introduce considerable bias. The metabolic disturbances associated with HT are known to promote obesity, a well-recognized risk factor for the exacerbation of OSAHS.[25] In our study cohort, patients with OH displayed higher BMI values compared to euthyroid individuals (28.2 ±5.0 vs 27.6±5.6, P=0.859), although this difference did not achieve statistical significance.

The inherent limitations of this retrospective study hinder the evaluation of two essential clinical interactions: (1) the therapeutic effects of thyroid hormone replacement on the progression of OSAHS in patients with HT, and (2) the potential alterations in thyroid function following continuous positive airway pressure (CPAP) therapy in individuals with OSAHS. Prior research has indicated that patients with hypothyroidism receiving thyroid hormone supplementation experience significant reductions in apnea events, oxygen desaturation indices, and episodes of snoring and choking, with these improvements occurring independently of changes in body weight.[9,32] Simultaneously, CPAP treatment has been linked to measurable enhancements in thyroid function among the majority of OSAHS patients,[23,33] collectively suggesting indirect evidence of a bidirectional relationship between OSAHS and HT.

Moreover, studies investigating the prevalence and clinical significance of SCH within OSAHS populations encounter an additional methodological challenge: the potential transient nature of SCH as a physiological state that may resolve spontaneously under varying bodily conditions.[29] This situation necessitates longitudinal studies to differentiate between persistent and transient cases of SCH, thereby ensuring the validity of the research findings. Additionally, while autoimmune thyroiditis is recognized as the primary cause of HT,[6] emerging evidence suggests that shared immunopathological mechanisms—particularly those involving Th1/Th17 cell-mediated inflammatory pathways—may be implicated in both OSAHS and autoimmune thyroiditis.[34,35] A large cross-sectional study involving 920 participants identified lymphocyte count as an independent predictor of OSAHS in conjunction with HT,[36] thereby reinforcing the immunological connection between these conditions. Although our study excluded patients with diagnosed immune disorders, this exclusion criterion may be inadequate given the complexities associated with immune dysregulation. Future investigations should incorporate comprehensive immunophenotyping to reduce bias and clarify the specific mechanistic pathways that link immune markers to the comorbidity of OSAHS and HT.

Due to the absence of comprehensive sleep architecture data, this study did not include the sleep parameters of patients with OSAHS in the final analysis. Patients with OSAHS typically experience chronic sleep fragmentation, disrupted sleep architecture, and sleep deprivation as a result of recurrent nocturnal awakenings.[5] Importantly, the synthesis and secretion of thyroid hormones exhibit distinct circadian rhythms,[37] indicating that prolonged sleep deprivation may disrupt the secretion patterns of TSH and, in turn, impair thyroid function. Epidemiological studies have shown that night-shift workers have significantly elevated serum TSH levels and a higher risk of SCH compared to their day-shift counterparts, with this

risk increasing in proportion to the duration of exposure to shift work.[38] Furthermore, research by Yan et al[39] has indicated that individuals with isolated TSH elevation report poorer nocturnal sleep quality compared to those with normal TSH levels. Mechanistic investigations have revealed that sleep deprivation leads to oxidative stress, which triggers an imbalance between autophagy and apoptosis in thyroid cells, ultimately compromising thyroid histology and function.[40] Collectively, these findings highlight the clinically significant effects of disrupted sleep architecture on thyroid homeostasis, thereby necessitating systematic exploration in future research endeavors.

Conclusion

The current investigation reveals a notably increased prevalence of HT among patients with OSAHS, with a higher vulnerability observed in females compared to males, a finding that aligns with existing clinical epidemiological literature. A significant methodological advantage of this study is its large sample size and the thorough exclusion of confounding comorbidities. Our findings reinforce the clinically relevant association between the severity of OSAHS and thyroid dysfunction, highlighting the necessity for heightened awareness and proactive management by healthcare providers. However, several critical gaps in knowledge remain due to the limitations of the study. Future research should aim to: (1) clarify the natural progression of transient SCH, (2) investigate the pathogenesis of autoimmune thyroiditis, (3) assess disruptions in sleep architecture, and (4) analyze the therapeutic effects of both thyroid hormone replacement and CPAP treatments in OSAHS populations. A systematic examination of these areas will contribute to an evidence-based framework for enhancing clinical management for OSAHS patients with concurrent HT.

Abbreviations

OSAHS, obstructive sleep apnea-hypopnea syndrome; PSG, polysomnography; AHI, apnea hypopnea index; $LSpO_2$, lowest oxygen saturation; $MSpO_2$, mean oxygen saturation; BMI, body mass index; CPAP, continuous positive airway pressure; TSH, thyroid stimulating hormone; FT4, free thyroxine; HT, hypothyroidism; OH, overt hypothyroidism; SCH, subclinical hypothyroidism.

Data Sharing Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Ethical Considerations

This study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of the First People's Hospital of Yunnan Province (Approval No. KHLL2023-KY002). The requirement for informed consent was waived by the committee due to the retrospective nature of the study, which involved analysis of anonymized patient data without any intervention or additional risk. All data were de-identified before analysis and stored on a password-protected institutional server accessible only to the research team.

Acknowledgments

We thank all the participants and staff involved in this study.

Author Contributions

Pinyi Zhou (First Author): Conceptualization, Formal Analysis, Investigation, Writing - Original Draft, Writing - Review & Editing.

Hongmei Li: Investigation, Formal Analysis, Validation, Writing - Original Draft, Writing - Review & Editing.

Jingman Qiu: Investigation, Formal Analysis, Validation, Data Curation, Writing - Original Draft.

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Jingyu Guo: Data Curation, Validation, Visualization, Writing - Original Draft.

Yun Yao: Investigation, Data Curation, Resources, Writing - Original Draft.

Hongyan Li: Formal Analysis, Validation, Data Curation, Writing - Original Draft.

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Yufan Duan: Writing - Original Draft, Investigation, Visualization, Writing - Review & Editing.

Yunhui Lv (Corresponding Author): Conceptualization, Formal Analysis, Supervision, Writing - Review & Editing, Funding Acquisition, Project Administration.

All authors gave final approval of the version to be published and agreed to be accountable for all aspects of the work. All authors reviewed and approved the final manuscript for submission to this journal.

Funding

This research was funded by [National Natural Science Foundation of China], Number: 1760022; Hanfang Expert Workstation in Yunnan Province, NO. 202105AF150022. Yunnan Province "Xing Dian Talent Support Program" Project, NO. XDYC-MY-2022-0013. Kunming Health Science and Technology Talent Cultivation Program and Medical Science and Technology Discipline Thousand Project Reserve Talents, NO. 2024-SW (Reserve-30).

Disclosure

The authors declare that there are no competing interests associated with the paper.

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