Section: Pediatrics

Original Research Article

ASSOCIATION BETWEEN UNDERACTIVE THYROID FUNCTION AND SLEEP APNOEA: A CROSS-SECTIONAL COMPARATIVE STUDY

S.Kavitha[1], R.Sivagami[2]

[1]Associate Professor, Department of Physiology, Stanley Medical College, Tamilnadu, India.
[2]Associate Professor, Department of Pharmacology, Stanley Medical College, Tamilnadu, India.

Received : 12/04/2026
Received in revised form : 04/06/2026
Accepted : 19/06/2026

Keywords:
Airway Obstruction; Hypersomnolence; Polysomnography; Sleep Apnea Syndromes.

Corresponding Author:
Dr. S.Kavitha,
Email: drkavitha2010@yahoo.co.in

DOI: 10.47009/jamp.2026.8.3.256

Source of Support: Nil,
Conflict of Interest: None declared

Int J Acad Med Pharm
2026; 8 (3); 1457-1460

ABSTRACT

Background: Obstructive sleep apnoea (OSA) is a common cause of excessive daytime sleepiness. Hypothyroidism contributes to sleep-disordered breathing due to upper airway obstruction and blunted ventilatory chemosensitivity. **Objective:** This study aimed to assess sleep-disordered breathing in newly-diagnosed hypothyroid patients by analysing and comparing respiratory parameters with age and sex-matched euthyroid subjects. **Materials and Methods:** Thirty newly diagnosed hypothyroid patients and 30 age- and sex-matched euthyroid controls underwent polysomnography to assess sleep-disordered breathing using airflow monitors of the nasal and oral cavity, respiratory effort gauges placed around the chest and abdomen, and pulse oximetry. **Results:** Respiratory parameters including Apnoea–Hypopnoea Index (AHI) (32.07 ± 23.18 in cases vs 1.63 ± 1.50 in controls) and lowest oxygen saturation (81.23 ± 8.34 in cases vs 92.63 ± 2.28 in controls) showed a very highly significant difference between hypothyroid patients and euthyroid controls ($P < 0.001$). **Conclusion:** Hypothyroidism is significantly associated with sleep-disordered breathing, increased Apnoea–Hypopnoea Index, and nocturnal oxygen desaturation. Early diagnosis and treatment of hypothyroidism may help reduce complications related to obstructive sleep apnoea.

INTRODUCTION

Sleep is an essential physiological process required for normal physical, metabolic, neurocognitive, and psychological functioning. Sleep disturbances adversely affect quality of life, daytime performance, cognition, mood, and cardiovascular health. Sleep-disordered breathing (SDB) represents a spectrum of respiratory abnormalities occurring during sleep, ranging from habitual snoring to obstructive sleep apnoea (OSA), nocturnal hypoventilation, and respiratory failure. Obstructive sleep apnoea is characterized by recurrent episodes of complete or partial upper airway obstruction during sleep, resulting in apnoea or hypopnoea associated with intermittent hypoxaemia, hypercapnia, fragmented sleep, and excessive daytime sleepiness. These repeated episodes lead to impaired concentration, reduced work efficiency, mood disturbances, neurocognitive dysfunction, and increased risk of cardiovascular and metabolic disorders. Significant sleep abnormalities have been demonstrated in hypothyroid patients before treatment, many of which improved following thyroid hormone replacement therapy.[1] Excessive daytime sleepiness and nocturnal oxygen desaturation have been shown to be strongly associated with polysomnographic abnormalities in OSA patients.[2] Obstructive sleep apnoea has been reported to alter sleep stage transition dynamics and contribute to sleep fragmentation and instability.[3] Elevated TSH levels may influence sleep regulation and slow-wave sleep mechanisms.[4]

Sleep-disordered breathing is increasingly recognized as a major public health problem because of its high prevalence and associated morbidity. Population-based studies have estimated that OSA affects approximately 9–38% of the adult population, with higher prevalence among obese individuals and males. Untreated OSA contributes significantly to systemic hypertension, coronary artery disease, stroke, insulin resistance, obesity, depression, impaired daytime alertness, and sleep-related accidents.[5,6]

Respiration during sleep is regulated by a complex interaction between neural, metabolic, endocrine, and mechanical factors. Sleep is associated with reduction in upper airway muscle tone, diminished ventilatory drive, and reduced responsiveness to hypoxia and hypercapnia. These physiological alterations predispose susceptible individuals to upper airway collapse during sleep. Conditions

ISSN (O): 2687-5365; ISSN (P): 2753-6556

affecting airway anatomy, respiratory muscle function, ventilatory control, or neuromuscular coordination can therefore precipitate sleep-disordered breathing.

Hypothyroidism is one of the common endocrine disorders associated with secondary obstructive sleep apnoea. It is characterized by reduced circulating thyroid hormone levels and elevated serum thyroid stimulating hormone (TSH). Thyroid hormones play an important role in metabolic regulation, respiratory drive, neuromuscular function, and maintenance of upper airway patency. Respiratory abnormalities in hypothyroidism include hypoventilation, impaired ventilatory response to hypoxia and hypercapnia, respiratory muscle weakness, obesity, mucopolysaccharide deposition in upper airway tissues, and reduced neural output to upper airway dilator muscles.

Several mechanisms have been proposed to explain the association between hypothyroidism and OSA. Increased upper airway obstruction and a higher prevalence of sleep-disordered breathing have been demonstrated among hypothyroid individuals compared to euthyroid controls.[5] Mucinous oedema and soft tissue infiltration involving the tongue and pharyngeal structures contribute to upper airway narrowing in hypothyroid patients.[6] Respiratory muscle weakness occurring in hypothyroidism further impairs ventilation and contributes to respiratory dysfunction during sleep.[7] A proportion of patients diagnosed with OSA may actually have secondary sleep apnoea due to undiagnosed hypothyroidism, highlighting the importance of thyroid evaluation in sleep apnoea patients.[8]

The relationship between hypothyroidism and sleep apnoea has also been supported by clinical and polysomnographic studies. Nocturnal hypoxia adversely affects cognition, executive functions, judgement, and reaction time, emphasizing the neuropsychological consequences of sleep-disordered breathing.[9] Thyroxine replacement therapy has been shown to significantly reverse sleep-disordered breathing and nocturnal oxygen desaturation in patients with primary hypothyroidism.[10] Significantly increased apnoeic index, oxygen desaturation, and arousal index have been demonstrated in hypothyroid patients.[9,10] These findings emphasize the reversible nature of respiratory dysfunction associated with thyroid hormone deficiency and underline the clinical importance of early diagnosis.

Despite growing evidence supporting the association between hypothyroidism and sleep-disordered breathing, many patients remain undiagnosed because symptoms such as fatigue, daytime sleepiness, poor concentration, lethargy, and weight gain are often attributed solely to hypothyroidism itself. In addition, data evaluating respiratory polysomnographic parameters among newly diagnosed untreated hypothyroid patients remain limited, particularly in the Indian population. Most previous studies have focused either on sleep architecture or on treated hypothyroid patients, while fewer studies have specifically evaluated respiratory polysomnographic abnormalities such as Apnoea–Hypopnoea Index and nocturnal oxygen desaturation in newly diagnosed overt hypothyroid individuals.

The present study was therefore undertaken to evaluate sleep-disordered breathing in newly diagnosed overt hypothyroid patients by analysing respiratory polysomnographic parameters and comparing them with age- and sex-matched euthyroid controls. The study specifically aimed to assess Apnoea–Hypopnoea Index and nocturnal oxygen saturation among hypothyroid patients and thereby determine the association between underactive thyroid function and obstructive sleep apnoea.

Aim & objective of the study: The aim of this study was to assess sleep-disordered breathing in newly-diagnosed hypothyroid patients by analysing and comparing respiratory parameters with age and sex-matched euthyroid subjects.

MATERIALS AND METHODS

The cross-sectional study was conducted [] after getting approval from the Institutional Ethical Committee [Ethical no].

Thirty newly diagnosed overt hypothyroid patients not on medical therapy of both sexes, age group between 18 to 50 years, and with regular night time sleep habits were selected and 30 age. Sex matched controls were selected for comparison with the cases. Patients excluded from the study were those with irregular sleep routine, nightshift workers, and individuals with neurological, psychiatric, respiratory, cardiovascular, or other systemic illnesses affecting sleep, and those taking medications known to alter sleep or respiratory function.

Informed verbal and written consent were obtained from the participants, and polysomnography (PSG) was conducted. Patients diagnosed with hypothyroidism were enrolled for the study to evaluate respiratory polysomnographic parameters associated with sleep-disordered breathing. International 10-20 system was utilized for recording EEG. Respiratory belts were placed around the thorax and abdomen. Airflow sensor-nasal prongs connected to a pressure transducer was used for measuring nasal flow Oxygen saturation was monitored using a pulse oximeter probe placed on a finger. Scoring and analysis of the recorded data was done using the criteria of Rechtschaffen and Kales and with scoring updates from the American Academy of Sleep Medicine.[11,12]

RESULTS

Age and sex distribution were similar between the groups, while body mass index and serum TSH levels

ISSN (O): 2687-5365; ISSN (P): 2753-6556

were significantly higher in hypothyroid patients [Table/Fig 1].

Hypothyroid patients exhibited significantly increased Apnoea–Hypopnoea Index and reduced nocturnal oxygen saturation compared with controls, suggesting greater sleep-disordered breathing [Table/Fig 2].

Table 1: Demographic and Thyroid Profile of Study Participants

Variable	Controls	Cases	P-value
Age (years) Mean ± SD	32.23 ± 9.81	31.97 ± 10.27	-
Gender (Male/Female)	15/15	15/15	-
BMI (kg/m²) Mean ± SD	24.30 ± 2.94	28.92 ± 3.04	P < 0.001
Serum TSH (mIU/L) Mean ± SD	2.74 ± 1.03	103.02 ± 28.16	P < 0.001

Table 2: Comparison of Respiratory Polysomnographic Parameters Between Healthy Controls and Hypothyroid Patients

Variable	Controls (Mean ± SD)	Cases (Mean ± SD)	P-value
Apnoea–Hypopnoea Index (AHI)	1.63 ± 1.50	32.07 ± 23.18	<0.001
Lowest Oxygen Saturation (%)	92.63 ± 2.28	81.23 ± 8.34	<0.001

DISCUSSION

SDB is a prevalent but underdiagnosed disorder that ranges from simple snoring to nocturnal hypoventilation and respiratory failure during sleep. Obstructive sleep apnoea (OSA), the most common form of SDB caused by upper airway obstruction, leads to fragmented sleep, impaired sleep quality, and adverse effects on lifestyle, intellectual functions, and social interactions.[1]

Glucose-PET studies have demonstrated that sleep deprivation for 24 hours results in decreased metabolism in the prefrontal and parietal associational areas. This leads to hypometabolism in regions responsible for judgment, impulse control, attention, and visual association compared to primary sensory and motor areas, indicating significant cognitive impairment during sleep-deprived states.[13]

Patients with SDB are at increased risk of developing serious comorbidities, including hypertension, insulin-resistance diabetes, coronary artery disease, obesity, reduced bone and muscle mass, depression, and sleepiness-related accidents.[1,14]

OSA is characterized by recurrent episodes of complete or partial upper airway obstruction during sleep, resulting in apnoea or hypopnoea events associated with intermittent hypoxemia, hypercapnia, and arousals. Persistent respiratory effort against a collapsed airway during these episodes, followed by arousal that restores patency, contributes to sleep fragmentation, breathing instability, and overall SDB.[1,2]

Previous studies have reported a higher prevalence of sleep-disordered breathing among hypothyroid patients. One study found that 74% of hypothyroid patients had OSA on polysomnography, with a significantly higher prevalence among treatment-naïve individuals. Proposed mechanisms include obesity, respiratory muscle weakness, reduced ventilatory drive, and mucopolysaccharide deposition within upper airway tissues, which may increase airway collapsibility during sleep.[1,13]

It has been estimated that a proportion of patients diagnosed with primary OSA may actually have secondary sleep apnoea due to undiagnosed hypothyroidism. Thyroid hormone replacement therapy in such cases often resolves nocturnal oxygen desaturation and apnoeic episodes.[1,3]

In the present study, the Apnoea–Hypopnoea Index (AHI) was markedly elevated in hypothyroid patients (32.07 ± 23.18) compared to euthyroid controls (1.63 ± 1.50), with a highly significant difference (P < 0.001). This indicates a substantially higher burden of obstructive events. Predisposing factors in hypothyroidism include altered ventilatory control, reduced upper airway muscle tone, increased pharyngeal compliance, and obesity, as evidenced by the significantly higher BMI in cases.[1,2]

Severity of OSA is graded by AHI, with values >30 episodes/hour indicating severe disease associated with frequent arterial desaturation and sleep disruption. Cognitive abilities, executive function, judgment, and reaction time show inverse correlations with respiratory parameters such as lowest oxygen saturation and total apnoeic time.[13]

The present study also demonstrated a highly significant reduction in lowest oxygen saturation in hypothyroid patients (81.23 ± 8.34%) compared to controls (92.63 ± 2.28%) (P < 0.001). This nocturnal hypoxia is primarily attributed to recurrent obstructive apnoeic and hypopnoeic events occurring during sleep. Consistent with earlier findings, OSA was reversible with adequate thyroxine replacement in a substantial proportion of hypothyroid cases.[2,3]

Supporting these observations, recent studies have confirmed significantly increased apnoeic index, oxygen desaturation, and arousal index in hypothyroid patients. Recent Mendelian randomization analyses further support the association between hypothyroidism and an increased risk of OSA.[2,3]

Early recognition of SDB in hypothyroid patients and prompt thyroid hormone replacement may reduce respiratory abnormalities during sleep and improve overall health outcomes. Larger studies, particularly in the Indian population, are warranted to further elucidate these associations.

ISSN (O): 2687-5365; ISSN (P): 2753-6556

CONCLUSION

The following conclusions have been derived from the study:

- There is increase in obstructive apnoea and hypopnoea episodes in hypothyroid patients causing SDB resulting in secondary changes in sleep pattern.
- There is a significant decrease in lowest nocturnal oxygen saturation in hypothyroid patient.
- Recording the history of sleep habits must be made mandatory to suspect sleep disorders at clinical level from the patient and partner or attender.

This comparative case-control study can be further followed up with thyroxine therapy to assess the therapy duration for disease reversal, diagnose patients not responding to therapy, and also to assess morbidity and mortality due to SDB in our community.

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ISSN (O): 2687-5365; ISSN (P): 2753-6556