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# Reversibility of Obstructive Sleep Apnea Following Levothyroxine Therapy in Newly Diagnosed Hypothyroidism: A Retrospective Polysomnographic Study

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## ABSTRACT

**Background**: Hypothyroidism is a recognized but often underappreciated contributor to obstructive sleep apnea (OSA). Proposed mechanisms include upper airway mucopolysaccharide deposition, soft tissue edema, respiratory muscle dysfunction, and impaired ventilatory drive, yet the extent to which OSA reverses once thyroid hormone levels are corrected is not well established.

**Objectives**: To determine the prevalence and severity of OSA among adults newly diagnosed with hypothyroidism who presented with sleep-disordered breathing, and to characterize change in polysomnographic parameters once biochemical euthyroidism was achieved with levothyroxine.

**Methods**: This retrospective cohort study was conducted in the Department of Pulmonary Medicine at a tertiary care center between May 2024 and December 2025. Adults with newly diagnosed hypothyroidism and symptoms suggestive of sleep-disordered breathing underwent baseline overnight polysomnography before starting levothyroxine. Patients with chronic obstructive pulmonary disease, obesity hypoventilation syndrome, craniofacial abnormalities, prior CPAP use, or previously treated OSA were excluded. Repeat polysomnography followed the achievement of biochemical euthyroidism, and change in apnea-hypopnea index (AHI), oxygen desaturation index (ODI), and OSA severity category was analyzed. Results: Of 84 patients who underwent baseline polysomnography, OSA was diagnosed in 57 (67.8%): mild in 5, moderate in 25, and severe in 27. Two patients with severe OSA were later excluded from outcome analysis, leaving 55 evaluable patients. Mean AHI fell from 41.2 ± 16.2 to 13.2 ± 9.1 events/hour after levothyroxine ($p < 0.001$), and mean ODI fell from 42.9 ± 13.4 to 12.1 ± 3.2 ($p < 0.001$); mean TSH declined from 37.4 ± 4.5 to 2.9 ± 0.6 mIU/L. Twenty-six patients (47.3%) achieved complete resolution of OSA, 18 (32.7%) showed partial improvement, and 11 (20.0%) had persistent disease with no meaningful change or worsening. Resolution was concentrated in patients with mild-to-moderate disease at baseline.

**Conclusions**: In this cohort, correcting hypothyroidism with levothyroxine was followed by substantial improvement in polysomnographic parameters, and this was most consistent among patients who began with mild or moderate OSA; those with severe disease had a more mixed response. The findings support screening for thyroid dysfunction in patients presenting with sleep-disordered breathing and suggest hypothyroidism should be regarded as a modifiable, though not universally reversible, contributor to OSA.

***Keywords***: *obstructive sleep apnea; hypothyroidism; levothyroxine; polysomnography; apnea-hypopnea index; oxygen desaturation index.*

## INTRODUCTION

Obstructive sleep apnea (OSA) is a common sleep-related breathing disorder marked by recurrent upper airway collapse during sleep, producing intermittent hypoxemia, sleep fragmentation, and excessive daytime somnolence. Contemporary reviews frame OSA as a heterogeneous condition in which upper airway anatomy, neuromuscular compensation, arousal threshold, ventilatory control stability, lung volume, and obesity interact to drive recurrent obstruction [1]. Left untreated, it carries well-documented cardiovascular, metabolic, neurocognitive, and quality-of-life consequences [2].

While obesity remains the dominant risk factor, endocrine disease can contribute meaningfully to OSA pathogenesis. Hypothyroidism has long been linked to sleep apnea through upper airway soft tissue infiltration, mucopolysaccharide deposition, macroglossia, pharyngeal edema, impaired respiratory muscle performance, and blunted ventilatory drive [3--5]. These mechanisms are biologically plausible and, at least in principle, reversible with thyroid hormone replacement.

The published evidence on this question, however, is not entirely consistent. Takeuchi and colleagues assessed 156 patients referred for suspected sleep apnea and found primary hypothyroidism to be uncommon in this unselected clinic population, although mean apnea duration correlated with TSH and lower free triiodothyronine tracked with longer apnea episodes [6]. Older cohort and treatment studies — Rajagopal et al., Grunstein and Sullivan, Pelttari et al., Jha et al., and Resta et al. among them — reported that sleep-disordered breathing can improve once hypothyroidism is corrected, but noted that reversibility is often incomplete [3--5,7,8]. More recently, Pancholi et al. found polysomnography-confirmed OSA in 74% of 100 hypothyroid patients, with severe OSA accounting for 40.5% of cases and a higher OSA burden among treatment-naïve patients than among those already on thyroid replacement [9].

Pooled and cross-sectional evidence adds further nuance. A meta-analysis by Zhang et al. reported a modest excess of hypothyroidism among patients with OSA [10], whereas a later meta-analysis by Xiong et al. found no consistent association between thyroid dysfunction and OSA-hypopnea syndrome once heterogeneity across studies was accounted for [11]. Cross-sectional data from Bruyneel et al. likewise found a comparatively low prevalence of newly identified thyroid disease among patients with moderate-to-severe OSA [12], while a systematic review by Sorensen et al. concluded that the upper airway and ventilatory effects of thyroid hormone deficiency are mechanistically well established even though population-level prevalence estimates of OSA in hypothyroid cohorts vary considerably [13].

Genetic evidence has begun to weigh in on causality. Zhao et al., using bidirectional Mendelian randomization together with GEO-based bioinformatic analysis, found that genetically predicted hypothyroidism was associated with increased OSA risk, while genetically predicted OSA showed no reciprocal causal effect on hypothyroidism [14]. A second, independently conducted two-sample Mendelian randomization analysis by Lu et al. reported findings consistent with this direction of effect, lending some corroboration to a causal role for hypothyroidism in OSA risk [15].

Given this mixed but generally supportive body of evidence, the present study was designed to characterize the prevalence and severity distribution of OSA among newly diagnosed hypothyroid adults presenting with sleep-disordered breathing, and to describe how polysomnographic parameters change once euthyroid status is achieved with levothyroxine.

## MATERIALS AND METHODS

### Study Design and Setting

This retrospective cohort study was conducted in the Department of Pulmonary Medicine at a tertiary care center between May 2024 and December 2025, using clinical and polysomnographic data from adults newly diagnosed with hypothyroidism who presented with symptoms of sleep-disordered breathing.

### Study Population

Eligible patients were 18 years or older, had newly diagnosed hypothyroidism, reported symptoms suggestive of sleep-disordered breathing (snoring, witnessed apneas, excessive daytime sleepiness, or non-restorative sleep), and underwent baseline overnight polysomnography before starting levothyroxine. Patients were excluded if they had chronic obstructive pulmonary disease, obesity hypoventilation syndrome, craniofacial abnormalities, prior CPAP therapy, or previously diagnosed and treated OSA.

### Study Protocol

Eighty-six patients with elevated TSH met eligibility criteria; two declined consent for baseline polysomnography and were excluded, leaving 84 who underwent baseline testing. All patients received levothyroxine replacement and were followed until biochemical euthyroidism was achieved, at which point repeat polysomnography was performed. Two patients from the severe-OSA subgroup were subsequently excluded from outcome analysis — one lost to follow-up, and one who did not achieve euthyroid status despite treatment optimization.

### Definitions and Outcomes

OSA was defined by the apnea-hypopnea index (AHI) on overnight polysomnography, scored according to standard American Academy of Sleep Medicine criteria [16]. Consistent with those criteria, severity was classified as mild for AHI 5-

14 events/hour, moderate for AHI 15-29 events/hour, and severe for AHI ≥30 events/hour. Biochemical euthyroidism was defined as normalization of serum TSH to within the institutional laboratory reference range, broadly consistent with treatment targets described in American Thyroid Association guidance on levothyroxine replacement [17]. The primary outcome was change in AHI after levothyroxine and achievement of euthyroid status; secondary outcomes were change in ODI, change in OSA severity category, and the proportion of patients with complete resolution, partial improvement, persistent disease, or worsening.

**Statistical Analysis**

Continuous variables are summarized as mean ± standard deviation and categorical variables as counts and percentages. Paired baseline and post-treatment polysomnographic parameters were compared using paired t-tests, with a two-sided p-value <0.05 considered significant.

## RESULTS

**Baseline Cohort**

Of 86 eligible patients, 84 consented to baseline polysomnography and were included in the baseline analysis: 33 men and 51 women, mean age 48.2 ± 7.3 years. Baseline testing identified OSA in 57 patients (67.8%) — 5 mild, 25 moderate, and 27 severe.

**Table 1. Baseline cohort characteristics**

| Characteristic | Value |
|---|---|
| Eligible patients identified | 86 |
| Patients included after consent | 84 |
| Male / female | 33 / 51 |
| Age, years | 48.2 ± 7.3 |
| OSA diagnosed at baseline | 57/84 (67.8%) |
| Mild OSA | 5/57 (8.8%) |
| Moderate OSA | 25/57 (43.9%) |
| Severe OSA | 27/57 (47.4%) |

**Treatment and Follow-up**

All patients with diagnosed OSA received levothyroxine and were followed until biochemical euthyroidism was achieved, which took a mean of 4.6 ± 1.2 months. Two patients with severe OSA were excluded from follow-up analysis for the reasons noted above, leaving 55 patients for outcome assessment.

**Table 2. Baseline and post-treatment clinical and polysomnographic parameters**

| Parameter | Baseline | Post-treatment | p-value |
|---|---|---|---|
| TSH, mIU/L | 37.4 ± 4.5 | 2.9 ± 0.6 | Not calculated |
| BMI, kg/m² | 26.6 ± 2.7 | 24.9 ± 1.8 | Not calculated |
| AHI, events/hour | 41.2 ± 16.2 | 13.2 ± 9.1 | <0.001 |
| ODI | 42.9 ± 13.4 | 12.1 ± 3.2 | <0.001 |

**Overall Polysomnographic Response**

Mean AHI improved from 41.2 ± 16.2 to 13.2 ± 9.1 events/hour after levothyroxine ($p < 0.001$), and mean ODI improved from 42.9 ± 13.4 to 12.1 ± 3.2 ($p < 0.001$). Complete resolution of OSA occurred in 26 patients (47.3%), partial improvement in 18 (32.7%), and persistent disease without meaningful improvement or worsening in 11 (20.0%).

**Table 3. Overall polysomnographic response after levothyroxine therapy**

| Outcome category | Patients, n (%) |
|---|---|
| Complete resolution | 26 (47.3%) |
| Partial improvement | 18 (32.7%) |
| Persistent disease without meaningful improvement or worsening | 11 (20.0%) |
| Total follow-up cohort | 55 (100%) |

**Response by Baseline OSA Severity**

Improvement was most pronounced among patients with mild-to-moderate baseline OSA. Four of five patients with mild OSA achieved complete resolution. Among the 25 patients with moderate OSA, 18 resolved completely, two improved to mild OSA, one improved but remained moderate, three showed no significant change, and one progressed to severe OSA. Among the 25 severe-OSA patients with follow-up data, four resolved completely, two improved to mild OSA, four improved to moderate OSA, eight remained severe despite some improvement, five showed no meaningful change, and two worsened.

**Table 4. Response by baseline OSA severity**

| Baseline severity | Follow-up patients | Complete resolution | Partial improvement | No meaningful improvement / worsening |
|---|---|---|---|---|
| Mild OSA | 5 | 4 | 1 | 0 |
| Moderate OSA | 25 | 18 | 3 | 4 |
| Severe OSA | 25 | 4 | 14 | 7 |
| Total | 55 | 26 | 18 | 11 |

## DISCUSSION

This retrospective polysomnographic study found a high prevalence of OSA among newly diagnosed hypothyroid adults presenting with sleep-disordered breathing. The more striking finding, though, is what happened after treatment: achieving euthyroid status with levothyroxine tracked with marked improvement in both AHI and ODI, and nearly half of the follow-up cohort resolved completely.

These results fit a plausible biological picture. Hypothyroidism can narrow and destabilize the upper airway through mucopolysaccharide deposition, macroglossia, and pharyngeal soft tissue edema, while separately impairing respiratory muscle function and ventilatory drive 3--5. Correcting the underlying thyroid deficiency would therefore be expected to improve both airway patency and ventilatory control during sleep. Because BMI changed only modestly in this cohort, it seems unlikely that weight loss alone explains the improvement; thyroid correction more plausibly made an independent contribution, though the retrospective design does not allow this to be established with certainty.

The pattern here is broadly consistent with earlier work. Jha et al. described reversal of sleep-disordered breathing after thyroxine replacement in primary hypothyroidism, and Resta et al. reported comparable effects of T4 therapy on OSA prevalence and severity in subclinical disease 7,8. Grunstein and Sullivan, reviewing the mechanistic and management literature, cautioned that treatment response can vary considerably, particularly where anatomic or obesity-related contributors coexist 4. The high baseline OSA prevalence seen here also echoes Pancholi et al., who found polysomnography-confirmed OSA in close to three-quarters of hypothyroid patients and a lower prevalence among those already receiving thyroid replacement 9.

One of the more clinically useful observations in this study is that response tracked baseline severity. Complete resolution was common in mild and moderate disease but much less so in severe OSA, where persistent anatomical narrowing, obesity-related airway collapsibility, age-related neuromuscular change, or other non-endocrine factors may account for a larger share of the disease burden. In occasional patients where severe, refractory obstruction coexists with substantial goitrous enlargement, thyroid surgery has been described as an adjunctive option for anatomic upper airway compromise 18, though none of the patients in this cohort required this. Practically, this argues for treating hypothyroidism as a modifiable contributor to OSA severity while still pursuing standard OSA management — including positive airway pressure therapy — for disease that persists after euthyroidism.

More broadly, the findings support checking thyroid function in selected patients with sleep-disordered breathing, particularly when hypothyroid features are present or when OSA severity seems out of proportion to body habitus. That said, population-level data have not consistently supported universal thyroid screening in every OSA patient; reported prevalence of thyroid dysfunction in OSA cohorts varies substantially across studies, and a meta-analysis by Xiong et al. found no consistent association once study heterogeneity was taken into account 6,11,19.

Thyroid assessment, then, is probably best integrated into clinical judgment rather than treated as a substitute for a full sleep evaluation. The Mendelian-randomization work from Zhao et al., corroborated by the independent analysis from Lu et al., supports hypothyroidism as a plausible upstream risk factor for OSA rather than simply a co-occurring condition — although genetic evidence of causality does not, on its own, prove that levothyroxine will reverse established disease in a given patient 14,15.

### Limitations

Several limitations warrant mention. The retrospective design limits causal inference and leaves room for selection bias. The study was conducted at a single tertiary center with a modest sample size, and follow-up was limited to the period immediately after euthyroidism was reached, so the longer-term durability of improvement is unknown. Multivariable modeling and formal correlation testing between baseline TSH, BMI, time to euthyroidism, and magnitude of AHI improvement were not performed. Symptom scores, sleep-architecture measures, neck circumference, levothyroxine dose, adherence data, and comorbidity-adjusted outcomes were not available in the dataset used for this analysis. Finally, there was no contemporaneous untreated control group, so regression to the mean and other temporal effects cannot be fully excluded.

## CONCLUSIONS

Among newly diagnosed hypothyroid patients with OSA, levothyroxine therapy was followed by meaningful improvement in AHI and ODI once biochemical euthyroidism was reached, with the most consistent benefit seen in mild-to-moderate disease and a more heterogeneous picture in severe OSA. Taken together, these findings position hypothyroidism as a clinically relevant and at least partly modifiable contributor to OSA severity, and support routine consideration of thyroid function testing in adults who present with sleep-disordered breathing. Where disease persists despite euthyroidism, standard OSA care pathways should still apply.

### Declarations

- Ethics approval and consent to participate: This study used retrospective clinical and polysomnographic data; informed consent was obtained according to institutional policy and ethics committee approval.
- Funding: No external funding was reported for this study.
- Conflicts of interest: The authors declare no conflicts of interest related to this manuscript.
- Data availability: Aggregate data used to prepare this manuscript are reported within the article tables. De-identified source data may be made available by the corresponding author on reasonable request, subject to institutional and ethics committee requirements.
- Author contributions: Dr. Mani Tiwari contributed to study conception, data acquisition, analysis, and manuscript drafting. Dr. Vishesh Mohan Saxena contributed to data interpretation and critical revision. All authors reviewed and approved the final version before submission.

## REFERENCES


1. Jordan AS, McSharry DG, Malhotra A. Adult obstructive sleep apnoea. Lancet. 2014;383(9918):736-747.
2. Punjabi NM. The epidemiology of adult obstructive sleep apnea. Proc Am Thorac Soc. 2008;5(2):136-143.
3. Rajagopal KR, Abbrecht PH, Derderian SS, Pickett C, Hofeldt F, Tellis CJ, Zwillich CW. Obstructive sleep apnea in hypothyroidism. Ann Intern Med. 1984;101(4):491-494.
4. Grunstein RR, Sullivan CE. Sleep apnea and hypothyroidism: mechanisms and management. Am J Med. 1988;85(6):775-779.
5. Pelttari L, Rauhala E, Polo O, Hyyppa MT, Kronholm E, Viikari J, Kantola I. Upper airway obstruction in hypothyroidism. J Intern Med. 1994;236(2):177-181.
6. Takeuchi S, Kitamura T, Ohbuchi T, Koizumi H, Takahashi R, Hohchi N, Suzuki H. Relationship between sleep apnea and thyroid function. Sleep Breath. 2015;19(1):85-89. doi:10.1007/s11325-014-0966-0.
7. Jha A, Sharma SK, Tandon N, Lakshmy R, Kadhiravan T, Handa KK, Gupta R, Pandey RM. Thyroxine replacement therapy reverses sleep-disordered breathing in patients with primary hypothyroidism. Sleep Med. 2006;7(1):55-61.
8. Resta O, Carratu P, Carpagnano GE, Maniscalco M, Di Gioia G, Lacedonia D, Giorgino R, De Pergola G. Influence of subclinical hypothyroidism and T4 treatment on the prevalence and severity of obstructive sleep apnoea syndrome (OSAS). J Endocrinol Invest. 2005;28(10):893-898.
9. Pancholi C, Chaudhary SC, Gupta KK, Sawlani KK, Verma SK, Singh A, Verma AK, Usman K, Atam V. Obstructive sleep apnea in hypothyroidism. Ann Afr Med. 2022;21(4):403-409. doi:10.4103/aam.aam_134_21.
10. Zhang M, Zhang W, Tan J, Zhao M, Zhang Q, Lei P. Role of hypothyroidism in obstructive sleep apnea: a meta-analysis. Curr Med Res Opin. 2016;32(6):1059-1064.
11. Xiong J, Hu H, Huang Y, Zhang J, He J. Lack of associations between thyroid dysfunction and obstructive sleep apnea-hypopnea syndrome: a meta-analysis. Medicine (Baltimore). 2023;102(35):e36531.
12. Bruyneel M, Veltri F, Poppe K. Prevalence of newly established thyroid disorders in patients with moderate-to-severe obstructive sleep apnea syndrome. Sleep Breath. 2019;23(2):567-573.
13. Sorensen JR, Winther KH, Bonnema SJ, Godballe C, Hegedus L. Respiratory manifestations of hypothyroidism: a systematic review. Thyroid. 2016;26(11):1519-1527.
14. Zhao M, Huang X, Zheng H, Cai Y, Han W, Wang Y, Chen R. Association between hypothyroidism and obstructive sleep apnea: a bidirectional Mendelian randomization study combined with the GEO database. Front Neurol. 2024;15:1420391. doi:10.3389/fneur.2024.1420391.
15. Lu N, Chen B, Liu P, Wang C, Lu Z, Li S. Causal association between hypothyroidism and obstructive sleep apnea: a bidirectional 2-sample Mendelian randomization study. Medicine (Baltimore). 2024;103(42):e40114.
16. Berry RB, Brooks R, Gamaldo CE, Harding SM, Lloyd RM, Quan SF, Troester MM, Vaughn BV. The AASM Manual for the Scoring of Sleep and Associated Events: Rules, Terminology and Technical Specifications. Version 2.6. Darien, IL: American Academy of Sleep Medicine; 2020.
17. Jonklaas J, Bianco AC, Bauer AJ, Burman KD, Cappola AR, Celi FS, Cooper DS, Kim BW, Peeters RP, Rosenthal MS, Sawka AM. Guidelines for the treatment of hypothyroidism: prepared by the American Thyroid Association task force on thyroid hormone replacement. Thyroid. 2014;24(12):1670-1751.
18. Schneider A, Bourahla K, Petiau C, Velten M, Volkmar PP, Rodier JF. Role of thyroid surgery in the obstructive sleep apnea syndrome. World J Surg. 2014;38(8):1990-1994. doi:10.1007/s00268-014-2519-x.

19. Kapur VK, Koepsell TD, deMaine J, Hert R, Sandblom RE, Psaty BM. Association of hypothyroidism and obstructive sleep apnea. Am J Respir Crit Care Med. 1998;158(5 Pt 1):1379-1383.