Thyroidectomy for Goiter Improves Obstructive Sleep Apnea

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ABSTRACT

Objectives:
Evaluate the effects of total thyroidectomy for euthyroid goiter causing tracheal compression on the apnea hypopnea index (AHI) in patients with obstructive sleep apnea (OSA). Demonstrate a relationship between tracheal compression and OSA.

Methods:
A retrospective study of 8 patients with euthyroid goiter causing tracheal compression who had moderate or severe OSA. Between January 2004 and December 2007, 8 patients with these conditions were treated in a community hospital by the author. At least one compressive symptom (dysphagia, dyspnea, and/or orthopnea) was reported by all patients. Computed tomography of the neck and chest was used to confirm the extent of goiter and tracheal compression. OSA was confirmed with preoperative polysomnography. Total thyroidectomy was performed. Postoperative polysomnography was obtained after 90 days. Outcome measures were changes in compressive symptoms and AHI using paired t test.

Results:
All 8 patients reported symptomatic control of compressive symptoms after thyroidectomy. 7 of 8 patients demonstrated postoperative improvement of AHI. The mean postoperative AHI decreased significantly from 52.1 to 36.6 ($P < 0.05$).

Conclusions:
Total thyroidectomy for goiter causing tracheal compression can significantly improve symptoms and AHI in those patients who have OSA. Evaluation of patients with OSA should include screening for tracheal compression from goiter.

INTRODUCTION

Obstructive sleep apnea (OSA) syndrome is a common disabling condition that may affect 5% or more of the adult population.[1] Apnea is thought to occur during sleep because of periodic obstruction of the upper airway, particularly in the retropalatal or retroglossal regions of the pharynx.[2] Although systemic obesity is a risk factor for OSA, neck circumference is more closely correlated with the presence of OSA.[3] By providing a bypass of all upper airway obstruction in the neck, pharynx, and above, tracheostomy has been shown to consistently eliminate OSA. [4] Despite remaining the gold standard for surgical treatment of OSA, tracheostomy is poorly tolerated or is accepted by few patients.[5]

Besides OSA, goiter is a common condition known to cause obstruction of breathing by direct compression of the trachea.[6] Thyroid goiters have long been known to cause symptoms of dyspnea, obstructive dysphagia, and orthopnea by direct compression of the trachea and esophagus.[6] Total thyroidectomy for goiter has been demonstrated to be an effective treatment to significantly reduce compressive symptoms.[6] Two case series have been reported in which a total of 3 OSA patients with goiter causing tracheal compression underwent thyroidectomy and had substantial improvement of OSA.[7,8]

The objective of the current study is to demonstrate a relationship between tracheal compression from goiter and OSA. First, preoperative polysomnography was used to establish the possible presence of OSA in patients presenting with euthyroid goiter and compressive symptoms. Then, after thyroidectomy was performed to relieve tracheal compression, postoperative polysomnography was used to assess the effects of relieving tracheal compression on OSA.

METHODS & MATERIALS

This was a retrospective review of patients presenting with euthyroid goiter causing tracheal compression who had moderate or severe OSA. All patients presented to the author in a community setting between January 2004 and December 2007. Patients who had presented with goiter causing at least one compressive symptom (obstructive dysphagia, dyspnea, and/or orthopnea) were considered for inclusion in the study.

Evaluation of the goiter and tracheal compression included comprehensive history, complete head and neck examination, and serum TSH. Computed tomography (CT) with intravenous contrast of the neck and chest was performed with the LightSpeed Plus (GE Healthcare, Chalfont St. Giles, UK). CT was used to confirm the size of the thyroid and presence of tracheal compression.

Patients were additionally screened for OSA by assessment of daytime somnolence, fatigue, and reported snoring. Nocturnal polysomnography was performed in suspected cases unless OSA was previously diagnosed. Apnea was scored as $\leq$ 20% airflow from baseline for $\geq$ 10 seconds; hypopnea was scored as $\leq$ 70% airflow from baseline for $\geq$ 10 seconds with $\geq$ 4% drop in SaO2. Patients with thyroid compression and OSA (AHI > 10) confirmed by preoperative polysomnography were included in this retrospective review.

Bilateral, completion, or unilateral thyroidectomy was performed through a standard transverse cervical incision. Bilateral laryngeal electromyography was performed intraoperatively with the NIMS endotracheal tube system (Medtronic ENT, Jacksonville, FL). In cases with possible parathyroid gland compromise, frozen section biopsy and ipsilateral autotransplantation to the sternocleidomastoid muscle was performed. All patients were instructed to have their continuous positive airway pressure (CPAP) device available during the perioperative period.

Patients were evaluated in the immediate postoperative period for airway compromise, hematoma, and vocal fold paresis. The changes in compressive symptoms were reported by patients in the immediate postoperative period and after 4 to 8 weeks. After 90 days, postoperative polysomnography was performed. The preoperative and postoperative AHI were then compared. Statistical analysis was performed using the paired t test.

Detailed informed consent for inclusion in this retrospective review was obtained from all patients. The study design was approved by the River Region Health System institutional review board.

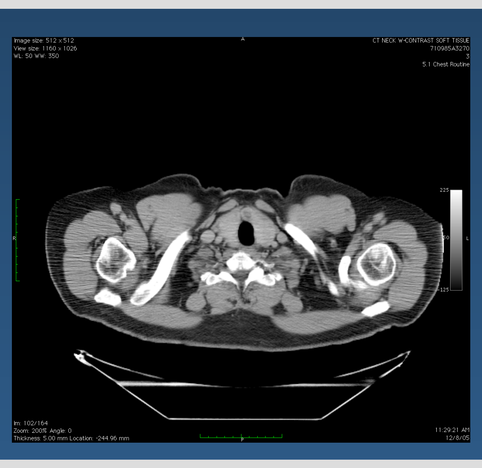

Fig. 1 Preoperative CT scan axial image of patient 1, goiter and mild tracheal compression (Preop AHI 56).

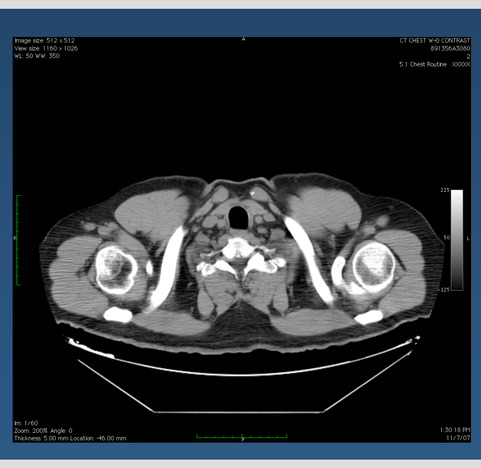

Fig. 2 Postoperative CT scan axial image of patient 1, with relief of tracheal compression (Postop AHI 39).

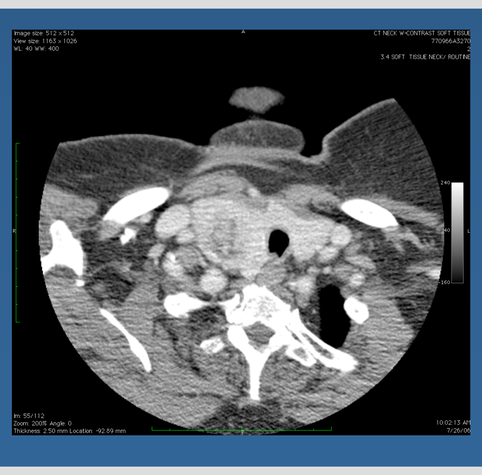

Fig. 3 Preoperative CT scan axial image of patient 2, moderate tracheal compression (Preop AHI 39 → Postop AHI 8).

Fig. 4 Preoperative CT scan axial image of patient 3, minimal tracheal compression (Preop AHI 16 → Postop AHI 1).

RESULTS

8 patients with thyroid goiter and compressive symptoms presented to the author between January 2004 and December 2007. All patients reported at least 2 pillow orthopnea and some degree of dysphagia to solids. 3 patients reported nocturnal dyspnea independent of position. 2 patients were known to have had OSA prior to their evaluation for goiter by the author. The remaining 6 patients had histories of snoring and/or daytime somnolence and were evaluated with polysomnography prior to surgery. All patients with compressive goiter studied by the author were found to have either moderate or severe OSA.

TSH was found to be normal in all 8 patients. CT imaging of the neck and chest with intravenous contrast was obtained in all patients to confirm the extent of the goiter and the degree of tracheal compression. Minimal, mild, or moderate tracheal narrowing and distortion was noted in all patients (**Figure 1-4**).

Thyroid surgery consisted of total thyroidectomy in 5 patients, completion unilateral thyroidectomy in 1 patient previously operated, and unilateral thyroidectomy with isthmustectomy in 2 patients with asymmetric unilateral enlargement. Pathology consisted of multinodular goiter in 7 patients. One specimen contained diffuse Hashimoto's thyroiditis.

Patients were monitored for complications, and there was no perioperative airway compromise, hypocalcemia, or hematoma. 1 patient developed right vocal cord paresis 2 weeks after surgery that resolved after 14 weeks. After thyroidectomy, all 8 patients reported symptomatic control of compressive symptoms, with resolution of orthopnea, dysphagia, and dyspnea. Patients did not have significant weight change in the perioperative period. Patients were placed on long term thyroid hormone replacement as serologically indicated.

7 of 8 patients demonstrated postoperative improvement of AHI. 2 patients had ostensible cures of OSA with a final AHI of 8 and 1, respectively. The preoperative and postoperative AHI were compared with the paired t test. Statistical analysis was performed using Excel (Microsoft, Redmond, WA). The mean postoperative AHI decreased significantly from 52.1 to 36.6, $p < 0.05$ (**Figure 5**). In terms of percentage improvement, there was a 45% improvement of mean AHI (**Figure 6**). Post-hoc power analysis determined that beta = 0.19, with an estimated 82 cases necessary to achieve statistical power (beta > 0.8).

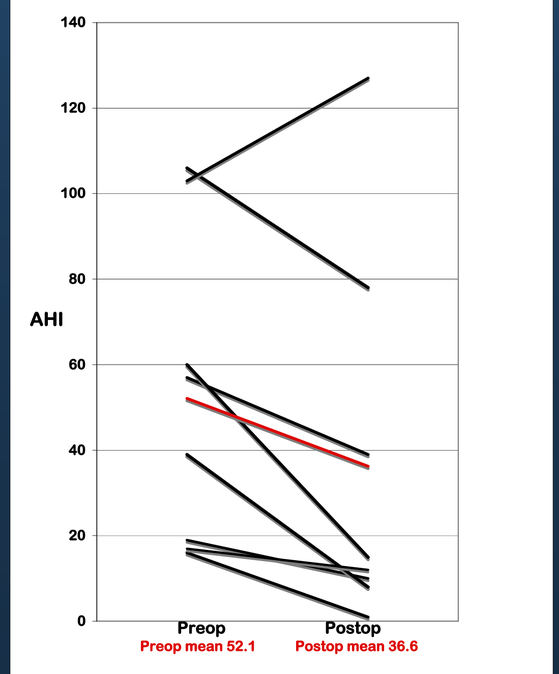

Fig. 5. Comparison of preoperative and postoperative AHI after thyroidectomy ($p < 0.05$).

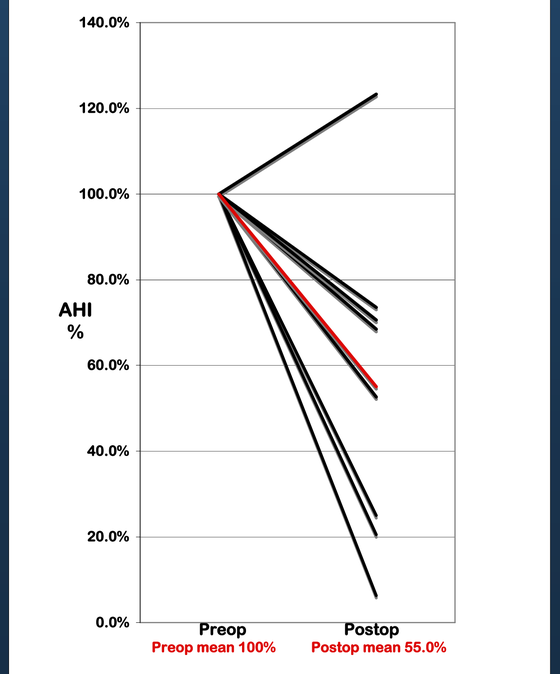

Fig. 6. Postoperative AHI as a percentage of preoperative AHI (mean reduction 45.0%).

DISCUSSION

This retrospective review demonstrates a relationship between compressive thyroid goiter and OSA. All 8 patients with compressive goiter were determined to have preoperative moderate or severe OSA. Total thyroidectomy successfully treated compressive symptoms in these patients. In addition, 7 of 8 patients demonstrated postoperative improvement of OSA after total thyroidectomy. 2 patients were cured of OSA with final AHI < 10. There was a mean improvement in the AHI of 45%, and statistical significance was obtained with $p < 0.05$.

These findings need to be considered in the context of the limitations of this study. First, with a sample size of 8, it would be difficult to generalize the results of the study to larger populations. The study is underpowered and a sample size of 82 would be required to achieve statistical power. Second, variability in polysomnography findings may account for some of the changes in AHI.[9] Night to night changes in AHI can be seen in the same patient depending on the quality of sleep, patient position, and methods of technical scoring. Finally, changes in body mass or levels of circulating thyroid hormone may have contributed to the reduction in AHI.[10]

Schwab and Goldberg have described a model for the pathogenesis of OSA in adults as the closure of the retropalatal and/or retrolingual pharyngeal airway.[2] While various reconstructive procedures of the palate and tongue can sometimes provide a cure, Schechtman and others have shown that they do not match the efficacy of tracheostomy for the control of OSA in adults.[11] Because patients generally reject tracheostomy, first line treatment of OSA in adults is generally nonsurgical.[4]

CPAP overcomes negative airway pressure and is generally accepted as the first line therapy for OSA.[12] Despite its effectiveness, however, Kribbs and others demonstrated that CPAP is often poorly tolerated and provides no lasting reversal of airway obstruction when removed.[13] Weight loss is another effective first line therapy that can provide a permanent reversal of OSA.[14] In patients with morbid obesity who sustain massive weight loss, such as that achieved with gastric bypass, OSA may be eliminated.[14]

The mechanism of systemic weight reduction leading to control of OSA remains unclear. Neck circumference correlates more strongly with the presence of OSA than systemic obesity.[15] Koenig and Thach studied the effects of mass loading to the anterior neck on airway resistance in an animal model.[16] Although endoscopy was used to assess pharyngeal closure, it is notable that the inspiratory resistance was measured from the trachea to the nasal aperture. [16] It is possible that the mass loading on the anterior neck contributed to increased airway resistance by direct deformation of the trachea.

These prior studies support the current findings that thyromegaly and mild to moderate tracheal compression may contribute to the pathogenesis of OSA. The effects of thyroidectomy for goiter on OSA have been documented in only 3 prior patients.[7,8] The results of the current study demonstrate that removal of thyroid goiter and relief of tracheal compression can similarly reduce the severity of OSA, and in some cases provide its cure. Further study of the relationship between tracheal compression and OSA may lead to improved diagnosis and treatment for OSA.

CONCLUSION

OSA may be common in patients with compressive thyroid goiter. Total thyroidectomy to relieve tracheal compression can significantly improve symptoms and AHI in patients with goiter who have OSA. Evaluation of patients with OSA should include screening for tracheal compression from goiter. Compression of the airway at the level of the trachea may be an important contributor to the pathogenesis of OSA. Further study will be necessary to define the relationship between goiter, tracheal compression, and OSA.

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