REVIEW ARTICLE

EFFECTS OF NASAL PATHOLOGIES ON OBSTRUCTIVE SLEEP APNEA

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Summary: Increased airway resistance can induce snoring and sleep apnea, and nasal obstruction is a common problem in snoring and obstructive sleep apnea (OSA) patients. Many snoring and OSA patients breathe via the mouth during sleep. Mouth breathing may contribute to increased collapsibility of the upper airways due to decreased contractile efficiency of the upper airway muscles as a result of mouth opening. Increased nasal airway resistance produces turbulent flow in the nasal cavity, induces oral breathing, promotes oscillation of the pharyngeal airway and can cause snoring.

Key words: *OSA; Allergic rhinitis; Anatomical abnormalities; Rhinitis; Treatment*

Introduction

Many abnormalities of the nose and pharynx can cause or aggravate snoring and sleep apnea. Nasal septal deviation, nasal polyps, inferior turbinate hypertrophy and rhinitis can cause nasal airway obstructions. Adenoid hypertrophy, nasopharyngitis and nasopharyngeal tumors can cause nasopharyngeal airway narrowing (6).

Epidemiological studies have found relationships between a history of nasal stuffiness and snoring (8) and between measured nasal airflow and a history of snoring (3), but attempts to find a linear correlation between nasal obstruction and sleep-disordered breathing have been less successful (2, 30, 34). However, a weak correlation between nasal resistance measured by means of posterior rhinomanometry and the severity of sleep apnea has recently been reported (15).

Several studies have shown that increased nasal resistance may induce sleep-related breathing disorders (SRBD) and cause disturbed sleep (7, 8); however, contradictory results have also been obtained (12).

Pathophysiology

Three interacting factors play a critical role in obstruction of the upper airway in OSAS: the muscle activity of the dilators of the pharyngeal airway, the negative pressure generated during inspiration, which opposes the activity of the dilators, and the structural anatomy of the airway (7, 31).

Mouth opening, independent of the breathing route, is believed to increase the collapsibility of the upper airways as a result of a decrease in the contractility of the upper airway muscles (20).

The biologic basis for nasal obstruction as a cause of sleep-disordered breathing lies in the effect of nasal breathing on resistance and flow velocity, which affects the pressure differential between the atmosphere and the intrathoracic space (34). Partial or complete obstruction can occur when the intrathoracic negative pressure generated by the inspiratory muscles pulls on the compliant soft tissue in the upper airway, sucking the airway closed (26). It is worth pointing out that the nose accounts for half of the total respiratory system resistance (24). Nasal obstruction leads to various breathing disturbances, including an increase in sleep apnea–hypopnea and a reduced nocturnal lowest SpO_2 (23, 33).

Although the primary site of obstruction in patients with OSAS is believed to be the oropharyngeal–hypopharyngeal region (Fig. 1), numerous studies examining the effects of experimentally-induced nasal obstruction on OSAS have indicated a positive and significant association (7, 23, 32, 36). Therefore, nasal obstruction constitutes a predisposing factor for OSAS (1, 3, 23, 32, 36). Effects of nasal pathologies on OSA are summarized in Fig. 2.

Effects of Allergic Rhinitis on OSA

The prevalence rate of allergic rhinitis is increasing by 3.5 % per decade and is estimated to be 15–25 % in Europe (29).

In general, allergic rhinitis constitutes a pathologic condition characterized by a transient increase in nasal airway resistance due to mucosal swelling (16, 18, 22, 25) and therefore theoretically constitutes a risk factor for OSAS.

Several reports in which nasal congestion due to allergic rhinitis as a risk factor for OSAS were studied and pro-

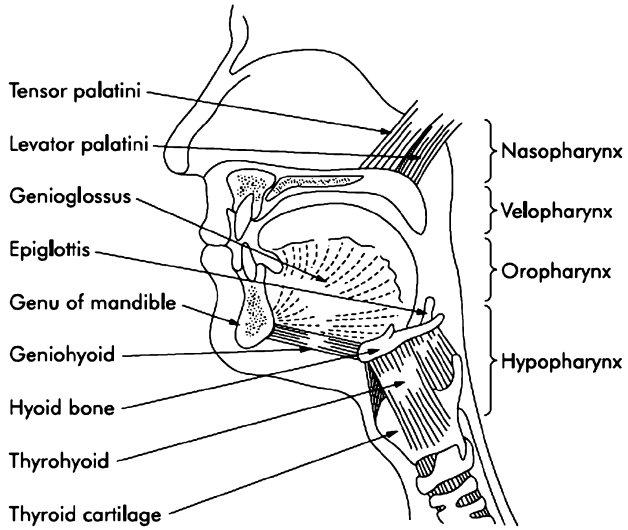

Fig. 1 (11): Anatomical representation of the upper airway and the important muscles controlling airway patency. In patients with apnea, airway collapse typically occurs behind the palate (velopharynx), the tongue (oropharynx), or both.

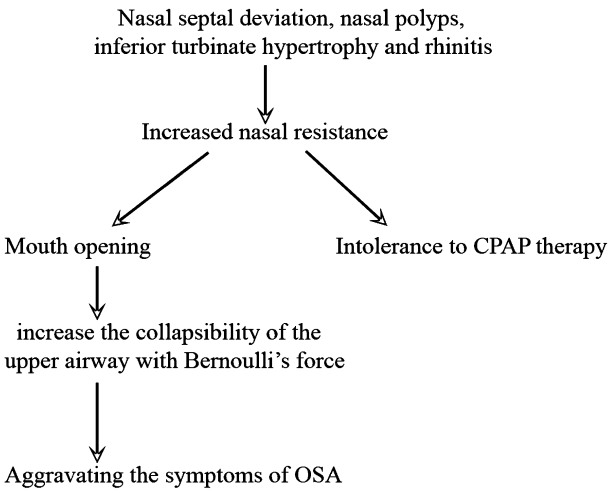

Fig. 2: Effects of nasal pathologies on OSA.

duced different results. Lavie et al. (13) reported on 14 patients with allergic rhinitis and 7 controls. The findings revealed that although allergic rhinitis patients had multiple microarousals from sleep, these arousals were generally associated with non-apneic breathing events.

Duchna et al. (4) found elevated positive allergy skin tests in 75 patients suffering from OSAS but failed to demonstrate significant changes in pathologic sleep parameters such as AI, HI or AHI. In contrast McNicholas et al. (18), in seven patients, found that during the asymptomatic phase of seasonal allergic rhinitis caused by ragweed pollen both the number of obstructive apneas and the duration of the apneas significantly decreased, and that these changes were associated with a significant decrease in mean nasal resistance. In a study of 39 children with habitual snoring, McColley et al. (16) concluded that, in the population studied, the presence of allergy was associated with an increased risk of OSAS. Using the population-based sample of the Wisconsin Sleep Cohort Study, Young et al. (34) reported that participants with nasal congestion due to allergy were 1.8 times more likely to have moderate-to-severe sleep disordered breathing than those without nasal congestion due to allergy. Nevertheless, a linear relationship between nasal obstruction and the severity of sleep-disordered breathing was not found.

Allergic rhinitis may cause daytime fatigue or sleep fragmentation without having effects on breathing. Allergic rhinitis may cause cortical arousal and fragmented sleep. Furthermore, somnolence due to sleep fragmentation may be compounded by daytime use of anti-allergic medication (34). Allergic rhinitis as a major risk factor for OSAS It is ruled out in the data (34).

Effects of Nasal Anatomical Abnormalities on OSA

Nasal structural alterations, septal deviation and/or conchal hypertrophy, or other pathologies lead to obstruction of nasal air flow. A narrow nasal passage may increase the velocity of nasal airflow and exacerbate collapse of the pharyngeal airway by means of Bernoulli's force; this mechanism may therefore aggravate the symptoms of OSA (10).

Zwillich et al. (37) induced nasal obstruction using a balloon-fitted nasal cannula in seven normal subjects and noted increases in apneic episodes and unstable sleep.

Olsen et al. (23) suggested that in cases of nasal obstruction such as nasal septal deviation or inferior turbinate hypertrophy, breathing is done via the oral airway and a negative pressure as well as vibration can occur due to the Bernoulli effect at the narrowed oral airway. However, nasal obstruction does not induce OSA and snoring in all people.

Some authors have reported that surgical correction of severe nasal obstruction results in differing degrees of improvement in OSA between individuals, indicating that it is difficult to predict the degree of improvement (2, 14, 17). Ibrahim and Sayed (9) reported that the severity of preoperative nasal obstruction and snoring did not influence the improvement of symptoms after septoplasty. They performed nasal operations (septoplasty, nasal polypectomy) in 96 patients with nasal obstruction and snoring. Series et al. (28) reported that in 4/20 mild OSA patients the RDI improved to a normal value after nasal surgery. These patients had normal cephalometric indices.

Nasal surgery for snoring and OSA patients with nasal obstruction does not always improve sleep apnea. It may still help to relieve airway obstruction but usually has a very positive effect on improving the quality of life and CPAP tolerance in OSA patients (21, 22).

Schonhofer et al. (34) investigated nasal dilators and they found no effect of nasal valve dilatation on sleep-related breathing disorders in patient with moderate to severe OSA and that reduction in nasal resistance does not prevent hypopharyngeal obstruction.

Effects of Chronic Rhinitis and/or Nasal Polyposis on OSA

Whether nasal obstruction due to chronic rhinitis and/or nasal polyposis is a predisposing factor for OSAS is not clear. Epidemiological studies have shown that chronic rhinitis symptoms and increased nasal resistance measured by rhinomanometry are associated with habitual snoring, but a similar association was not demonstrated for OSAS (34, 35). Nevertheless, treating OSAS patients with chronic rhinitis with fluticasone administered intra nasally for one month improved sleepiness, and reduced the apnea/hypopnea index with statistical significance, though only to a minimal degree compared to the placebo (5, 11).

Conclusions

Nasal obstruction therefore appears to have a minor role in the pathophysiology of OSAS. Treating impaired nasal breathing may be beneficial in selected patients. It improves subjective symptoms and sleep quality and may contribute to successful nasal CPAP therapy in OSA patients. Patients with coexisting nasal pathology (e.g. nasal polyposis, deviated septum, turbinate hypertrophy) were seen by an ear, nose and throat (ENT) specialist before nCPAP treatment was started, and nasal obstruction was treated and septoplasty or polypectomy was performed, if necessary (Fig. 3).

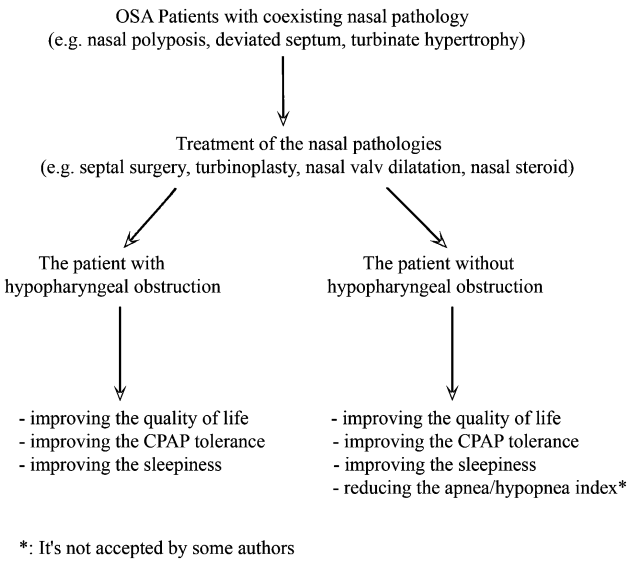

Fig. 3: Effects of treatment the nasal pathologies on OSA patients.

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