Unexpected Resolution of Obstructive Sleep Apnea after Nasal Surgery in a Patient Nonadherent to Continuous Positive Airway Pressure Therapy

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Sleep Sci 2023;16:127–130.

Abstract

Continuous positive airway pressure (CPAP) is recommended as first-line therapy in cases of moderate-to-severe obstructive sleep apnea (OSA). Continuous positive airway pressure (CPAP) prevents upper airway obstruction and abolishes apnea or hypopnea events during sleep. But up to 50% of OSA patients may be nonadherent to CPAP due to various causes, including nasal obstruction. Nasal surgery may improve CPAP compliance in some OSA patients but is not regarded as OSA therapy. Here, I report a patient non-adherent to CPAP whose sleep apnea resolved unexpectedly after nasal surgery to increase adherence to CPAP.

Keywords

- continuous positive airway pressure
- adherence
- obstructive sleep apnea
- nasal surgery

Introduction

Sleep apnea is a common disease that lowers the quality of life, causes daytime sleepiness, and is a risk factor for various cardiovascular diseases.[1] Continuous positive airway pressure (CPAP) therapy, acting like an air splint, prevents obstruction or narrowing of the upper airway during sleep and is endorsed as first-line therapy for moderate-to-severe sleep apnea. Adequate CPAP therapy improves the quality of life, daytime sleepiness, and reduces the risk of cardiovascular diseases such as hypertension, heart failure, and atrial fibrillation.[2] The generally accepted goal of CPAP compliance is set to at least an average of 4 hours a day use in more than 70% of the total days recommended. However, CPAP compliance is limited, and it is known that up to 50% of CPAP users are noncompliant.[3] Nasal obstruction is one of the reasons for nonadherence, and nasal surgery may make it easier to use CPAP.[4] Here, I report a male patient, nonadherent to CPAP, who underwent nasal surgery to improve his compliance to CPAP and whose sleep apnea resolved unexpectedly.

Case Report

A 38-year-old man presented with complaints of snoring, apnea, and sleep choking. He had a history of uvulopalatopharyngoplasty due to sleep apnea. He was 188 cm tall and weighed 102 kg, and his body mass index (BMI) was 28.8 kg/m^2. He was taking medication for hypertension and thyroid hormone after total thyroidectomy. He had back pain and history of depression. His Epworth sleepiness scale (ESS) was 8, and he did not take naps. He was a shift worker, working three shifts a day, and his schedule changed every five days. He usually drank approximately four cups of coffee daily, and an alcoholic beverage three times a week besides smoking one pack of cigarettes a day. In the polysomnography (PSG), sleep latency was 11.5 minutes, sleep efficiency 80%, waking after sleep onset 18%, and arousal index was

received
February 22, 2022
accepted
May 31, 2022

DOI https://doi.org/10.1055/s-0043-1767758.
ISSN 1984-0659.

Thieme Revinter Publicações Ltda., Rua do Matoso 170, Rio de Janeiro, RJ, CEP 20270-135, Brazil

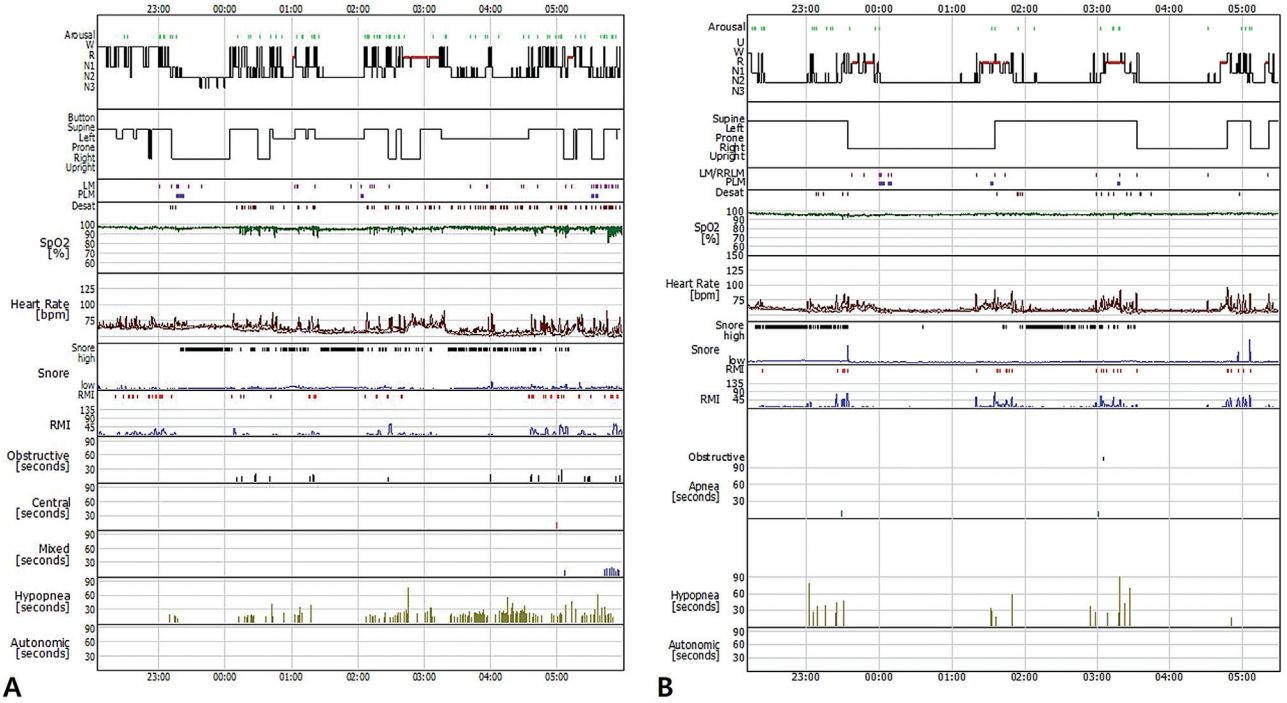

Fig. 1 Polysomnographic hypnograms during the diagnostic study (**A**) and follow-up study after nasal surgery (**B**).

34.1/h. He slept 36.5% of the total sleep time in supine position. He was diagnosed with moderate obstructive sleep apnea with a total apnea-hypopnea index (AHI) of 27.8/h (apnea index 5.2/h, hypopnea index 22.6/h, supine AHI 41.6/h, REM AHI 20/h) (►**Fig. 1A**). At the request of the patient, auto positive airway pressure (APAP) was started with minimum 4 cmH_2O and maximum 10 cmH_2O; then, a fixed pressure of 8 cmH_2O was prescribed after a month. The patient was successfully acclimatized initially (average daily usage of 6 hours 41 minutes, 81% of device usage in 30 days, device AHI 0.5/h), but then complained of problems such as mask fitting, dry mouth, and occasional nasal obstruction. Education and training on mask fitting, and humidification failed to solve the problem. Expiratory pressure relief mode adjustment and retrial of APAP did not improve symptoms and compliance (average daily usage of 2–3hours, 46–63% of device usage days, device AHI 0.4–0.7/h). After 1 year of CPAP therapy, I referred him to the ear, nose, and throat (ENT) department due to nasal obstruction, with the goal of improving CPAP adherence. He was diagnosed with septal deviation with inferior turbinate hypertrophy and underwent nasal surgery with septoplasty and submucosal inferior turbinoplasty. After the surgery, he stopped CPAP therapy and visited the outpatient clinic 4 months after surgery. At that time, his weight and BMI were slightly reduced to 96 kg and 27.1 kg/m^2. He reported no more snoring or apnea. The ESS was 10, and unlike 2 years prior, he took a nap for 1 to 2 hours only on weekends. Other than that, there was no difference in the amount or frequency of drinking, smoking, and consumption of caffeinated beverages. In PSG, sleep latency was 4 minutes, sleep efficiency 94.2%, waking after sleep onset 5%, arousal index 14/h. He slept 50% of the total sleep time in a supine position and total AHI was 3/h (apnea index, 0.3/h, hypopnea index 2.7/h, supine AHI 5.8/h, REM AHI 8.2/h) (►**Fig. 1B**).

Discussion

To ensure adequate treatment and better compliance, it is essential to follow up with CPAP patients regularly, monitor objective efficacy, and address any issues that arise during treatment. Although CPAP is an effective therapy for OSA, nonadherence often occurs in clinics. As CPAP withdrawal led to recurrent respiratory events within 1 night and increased blood pressure, heart rate, and subjective daytime sleepiness within 2 weeks,[5] effort should be made to keep CPAP and improve compliance. Generally accepted risk factors of nonadherence include younger age, obesity, lower socioeconomic status, moderate-to-severe OSA, comorbid insomnia and depression, minimum sleepiness, nasal obstruction, oronasal mask, claustrophobia, high prescribed pressure, the experience of adverse events, initial nonadherence in the first 2 weeks, problems on first-night use, and lack of education.[3,6,7]

Nasal obstruction is prevalent but can be easily overlooked in non-adherent CPAP patients. Septal deviation, turbinate hypertrophy, allergic rhinitis, nasal valve insufficiency, or rhinosinusitis with polyps are common causes of nasal obstruction and they reduce nasal airflow and elevate nasal airway resistance, which may cause snoring, apnea, or hypopnea. Nasal surgery may help to enhance CPAP compliance.[8] The common nasal surgery is septoplasty to straighten the deviated nasal septum, and turbinoplasty to reduce the volume of the inferior turbinate bone. A mechanism has been suggested that nasal surgery can improve sleep apnea by relieving the negative pressure of the intraluminal area within the upper airway through the expansion of the

narrowed nasal passages.[9] Although there is accumulating evidence that upper airway surgery facilitates the use of PAP and relieves the pressure burden by lowering the therapeutic pressure levels,[10,11] the impact of nasal surgery on PAP adherence is controversial.[11] Nasal surgery seems to lower the risk of OSA and affect positively multilevel surgical outcomes in patients with OSA.[12,13]

It may be inferred that OSA was relieved mainly by nasal surgery and some weight reduction. As respiratory events at initial PSG were mainly due to hypopnea, the effect of reducing negative pressure to keep upper airway patency of nasal surgery may be weak to abolish apnea but sufficient to prevent hypopnea events. In this case, OSA was resolved through nasal surgery. There is a possibility that the patient's weight loss may have played a part but, considering that weight loss rarely normalizes AHI and the AHI decreases by 26% when the weight is reduced by 10%,[14] the effect might be limited. The positional effect should also be considered. The patient had supine-predominant OSA (supine AHI/non-supine AHI $\geq$ 2). However, the ratio of supine position to total sleep time was 36.5% in diagnostic PSG and 50.3% in PSG after nasal surgery; thus, the improvement of apnea by position change is not applicable in this case. However, the causal link between nasal surgery and OSA resolution remains uncertain in this case because this inference is based on a single-night follow-up PSG. Though in-laboratory PSG is the gold standard for evaluating and diagnosing OSA, several studies have reported a relatively high night-to-night variability in AHI.[15,16]

The primary treatment for moderate-to-severe OSA is CPAP therapy. When compliance with the CPAP continues to decrease despite optimal pressure, various strategies to improve patient's adherence to CPAP and ultimately increase therapeutic efficacy should be tried: changing mask type, educating the patient on proper mask fitting, adjusting comfort setting, applying humidifier, modifying pressure, prescribing medications, or improving nasal permeability,[3,4,11] etc. As for nonadherent patients with nasal obstructive symptoms, adherence may be augmented by nasal surgery.[4] Although nasal surgery is not a treatment option for OSA, it is necessary to consider follow-up PSG while monitoring the patient's condition because sleep apnea may be attenuated and resolved in some cases.

Conflict of Interests

The author declares that he has no conflict of interests.

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