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The Emergence of Central Sleep Apnea after Surgical Relief of Nasal Obstruction in Obstructive Sleep Apnea

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CASE REPORTS

By the current definition, complex sleep apnea (CompSA) refers to the emergence of central sleep apnea (CSA) during the treatment of obstructive sleep apnea (OSA) with continuous positive airway pressure (CPAP). However, new-onset CSA has been described with use of other treatments for OSA, including tracheostomy, maxillofacial surgery, and mandibular advancement device. We present a patient with CSA beginning after endoscopic sinus and nasal surgery for nasal obstruction in the setting of mild OSA. This case highlights the importance of non-PAP mechanisms in the pathogenesis of CompSA.

Keywords: Complex sleep apnea, obstructive sleep apnea, central sleep apnea, surgery for obstructive sleep apnea, surgical relief of nasal obstruction

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By definition, complex sleep apnea (CompSA) refers to the emergence of central sleep apnea during the use of continuous positive airway pressure (CPAP) for treatment of obstructive sleep apnea (OSA).[1] This disorder occurs in fewer than 10% of patients with OSA and is often self-limited.[2] The appearance of central sleep disordered breathing has also been seen with non-PAP treatments for obstructive sleep apnea, including tracheostomy, maxillomandibular advancement surgery, and mandibular advancement device.[3-7] We describe a patient with OSA and new-onset central sleep apnea after nasal and sinus surgery.

REPORT OF CASE

A 43-year-old male presented with snoring, frequent nighttime awakenings, and daytime fatigue. In addition, for the previous 5 years he experienced chronic sino-nasal symptoms of congestion, sinus pressure, and postnasal drip without appreciable improvement despite use of nasal saline irrigation and topical nasal steroids. Persistent radiographic evidence of sinus disease had also been noted. He had no other cardiac or neurological problems and was not taking opioid narcotics. Physical examination was pertinent for a body mass index of 31.25 kg/m^2, a modified Mallampati class 3 airway, and S shaped septal deviation resulting in partial nasal obstruction. Head and neck, neurological, and cardiopulmonary examinations were normal. Epworth Sleepiness Scale score was 6/24. Attended diagnostic polysomnography was performed recording electroencephalogram, electrooculogram, electromyogram, electrocardiogram, nasal thermistor sensor, nasal pressure transducer, inductance plethysmography (chest and abdomen), and pulse oximetry. Respiratory events were scored using the American Academy of Sleep Medicine (AASM) criteria, with apneas scored in the setting of thermal sensor excursion reduction ≥ 90% of baseline; hypopneas defined as a decrease in flow (using the nasal pressure transducer) by 30%, accompanied by a drop in oxyhemoglobin saturation ≥ 4%; and respiratory effort related arousals (RERAs) scored when a sequence of breaths ≥ 10 sec was associated with increasing respiratory effort or flattening of the nasal pressure waveform and followed by arousal (but not meeting criteria for apnea or hypopnea).[8] Polysomnogram revealed overall mild obstructive sleep apnea (apnea hypopnea index [AHI] of 5.8/h, central apnea index of 0.2/h) with apneas and hypopneas occurring almost exclusively in supine sleep (supine AHI 28.9/h, lateral AHI 1.4/h). In addition, RERAs were noted with a frequency of 14.9/h. Sleep was fragmented with an arousal index of 46.6/h. Nadir of oxyhemoglobin saturation during the study was 87%. He did not undergo a trial of nasal CPAP.

Due to his concomitant sinus and nasal symptoms, he underwent an uncomplicated bilateral endoscopic sinus surgery (frontal sinusotomy, total ethmoidectomy, maxillary antrostomy, sphenoidotomy) and septoplasty. Four months after surgery his nasal congestion improved, but nighttime sleep fragmentation and excessive daytime sleepiness both worsened. He had not gained any weight and did not have any cardiopulmonary symptoms. A follow-up attended diagnostic polysomnogram demonstrated severe sleep apnea (AHI 30.2/h) with respiratory events still confined largely to sleep in the supine position (supine AHI 79.0/h, lateral AHI 4.6/h). Most of the respiratory events were now central in nature, with central apnea index of 24.5/h, obstructive apnea index of 0.0/h, mixed apnea index of 1.3/h, and obstructive hypopnea index of 4.4/h (Figure 1). However, RERAs had increased to 22.7/h. Sleep fragmentation was also more severe than in the preoperative study, with an arousal index of 67.2/h. Subsequent titration with CPAP was successful in resolving both obstructive and central sleep disordered breathing at a pressure of 11 cm H_2O. Excessive daytime sleepiness resolved with use of CPAP; downloadable data demonstrated excellent compliance and no residual sleep apnea.

DISCUSSION

To our knowledge, this is the first reported case of the emergence of central sleep disordered breathing after surgery to relieve nasal obstruction. Designation of "CompSA" as a distinct disease entity has been controversial, as the term probably encompasses a heterogeneous collection of processes.[9,10] Implicated in the pathogenesis are changes in carbon dioxide regulation. Chronic upper airway resistance results in increased ventilatory response to carbon dioxide; introduction of CPAP may then cause a drop of pCO_2 below the apneic threshold and result in the emergence of central apnea.[11,12] In addition, several factors such as CPAP undertitration, CPAP overtitration, and sleep disruption from CPAP itself may be involved in the appearance of central apnea activity on PAP.[10] Arousals may play a critical role in the generation of central sleep apnea due to increased chemosensitivity to pCO_2 in wake compared to sleep, reduction of upper airway resistance upon arousal, and increased neural output to respiratory centers in wake.[13-15]

Other surgical interventions, namely tracheostomy and maxillomandibular advancement, have resulted in the development of central sleep apnea postoperatively.[3,4] The proposed mechanism in these cases is the persistence of an augmented response to pCO_2 due to chronic obstructive sleep apnea resulting in high loop gain and subsequently, central sleep apnea after ventilatory load is decreased.[3,4] Central sleep apnea resolves in these patients over time, as seen in the vast majority of CPAP emergent central sleep apnea cases.[2-4] However, in our patient, the development of central sleep disordered breathing was accompanied by an increase (rather than decrease) in obstructive respiratory indices and arousals postoperatively. As it is known that ventilation increases after arousal from NREM sleep, we speculate that increased upper airway resistance contributing to compromised sleep continuity in the setting of high loop gain resulted in ventilatory overshoot, repetitive decrements in pCO_2 levels below the apneic threshold, and emergence of central apneas.[13-18] Resolution of central apneas on CPAP was likely due to the clearance of residual airway obstruction. Additionally, a decrease in chemoresponsiveness is expected with use of CPAP.[19]

One could argue that the mechanism of central sleep disordered breathing in this case parallels that seen in both CPAP undertitration and increased sleep disruption after the introduction of PAP in a vulnerable subject. Furthermore, a recent study found that when compared to patients with purely obstructive sleep apnea, patients with primarily obstructive but also central sleep apneas demonstrated a significantly smaller CO_2 reserve.[20] In patients with this tendency, it could be surmised that acute changes such as an increase in arousals or reduction in arterial CO_2 tension after relief of airway obstruction (either by PAP or surgical intervention) are sufficient to induce central apneas. Therefore, our case underlines not only the importance of factors such as sleep fragmentation and ventilatory instability in the pathogenesis of CompSA but also the need to delineate the various causes of a similar polysomnographic finding and recognizing patient heterogeneity. Furthermore, it shows limitations of surgical relief of obstruction at the nasal level in obstructive sleep apnea and the need for close clinical follow-up and postoperative polysomnogram in cases of persistent symptoms.

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DISCLOSURE STATEMENT

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