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CASE REPORTS

Hypoglossal nerve stimulation impact on a patient with obstructive sleep apnea and heart failure

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A 75-year-old man with body mass index of 30.5 kg/m^2 and severe obstructive sleep apnea (OSA) with an apnea-hypopnea index (AHI) of 72 events/h was referred for upper airway stimulation (UAS) therapy. Past medical history was significant for cardiovascular disease including congestive heart failure due to ischemic cardiomyopathy with impaired left ventricular function and ejection fraction of 35%. Following evaluation of clinical and polysomnographic data, he was an appropriate candidate for UAS and underwent uncomplicated implantation. Three months postoperatively, polysomnography showed a titrated AHI of 0 events/h. Follow-up cardiac evaluation revealed ejection fraction increase to 47% since implantation. No interval change in medical management or body mass index had occurred. Given the high prevalence of OSA in patients with cardiovascular disease, UAS may become an important adjunct in the comprehensive multidisciplinary treatment of heart failure in patients with OSA. Further clinical studies are required to investigate the impact of UAS on treatment and prognosis of heart disease.

Keywords: obstructive sleep apnea, upper airway stimulation, cardiovascular disease, congestive heart failure

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INTRODUCTION

Obstructive sleep apnea (OSA) is a respiratory disorder that disrupts spontaneous ventilation during sleep due to airway collapse and resulting hypoxemia. Risk factors include elevated body mass index, anatomic obstruction, and inadequate motor tone of the upper airway dilator muscles and/or the tongue. It is increasingly recognized as an independent risk factor for cardiovascular disease, stroke, seizures, diabetes, cancer mortality, and perioperative morbidities.[1]

Conservative treatment for OSA includes weight reduction and positional therapy. Continuous positive airway pressure (CPAP) has become the primary treatment for moderate to severe OSA and has been shown to improve sleep apnea by inhibiting disordered breathing events. However, intolerance to CPAP is frequent and adherence rates are only about 40%.[1] Clinical studies have shown that hypoglossal nerve stimulation by a surgically implanted device can relieve obstruction of the upper airway by causing relaxation of the dilator muscles, thus resulting in improvement of OSA.[2]

Use of CPAP has been shown in some studies to mitigate the cardiovascular risks, emphasizing the importance of recognizing OSA and maintaining close follow-up to ensure adherence to CPAP therapy.[3,4] Nevertheless, the impact of upper airway stimulation (UAS) therapy on ameliorating the cardiovascular risk due to OSA has not yet been extensively studied. Specifically, its effect on management and prognosis of heart failure remains unknown.

In this report, we describe a patient with OSA and heart failure due to ischemic cardiomyopathy who had significant improvement in functional status and myocardial contractility 1 year following UAS therapy.

REPORT OF CASE

A 75-year-old man with body mass index of 30.5 kg/m^2, severe OSA (apnea-hypopnea index [AHI] of 72 events/h) and Epworth Sleepiness Scale of 7 was referred for UAS therapy. He had difficulty adhering to CPAP treatment, which he was unable to tolerate due to sleep disruption. Past medical history was significant for cardiovascular disease, including congestive heart failure due to ischemic cardiomyopathy with impaired left ventricular function and ejection fraction of 35% by echocardiography. In addition, he had persistent atrial fibrillation and had received an implantable defibrillator 4 years previously, following atrioventricular node ablation. His functional status was New York Heart Association Class III. His preoperative AHI (72 events/h) was outside of the standard recommendations for UAS. However, given his failure to adhere to conservative measures, cardiovascular risk of untreated sleep apnea, and anatomic candidacy for UAS on drug-induced sleep endoscopy, he was deemed to be an appropriate candidate for UAS in an off-label indication outside of standard criteria. He underwent uncomplicated implantation.[2]

Three months postoperatively, the patient and his bed partner reported no sleep apnea events. He described his sleep as "more refreshing" and reported overall improvement in quality of sleep and daytime fatigue. His body mass index had not changed. Polysomnography showed a titrated AHI of 0 events/h at 1.8 V and 4% oxygen desaturation index of 1.5. He further titrated his

device to 2 V at a bipolar (+-+) configuration and had a repeat 2-night home sleep study 2 months later showing 4% respiratory event index of 3.9 and 2.3 events/h on respective nights. At 1-year follow-up, the patient reported continuous improvement in quality of sleep with usage of the device at 2 V. Cardiac evaluation revealed that ejection fraction had increased to 47% since implantation and chronic anemia had resolved. Functional class had improved to New York Heart Association Class I. No interval change in his medical management had occurred.

DISCUSSION

The association of OSA with increased cardiovascular risk was previously recognized.[1] While there is some conflicting data on CPAP and its impact on cardiovascular risks, observational studies suggest that patients with severe sleep apnea who undergo treatment with CPAP have reduced fatal and nonfatal cardiovascular events compared to patients that do not adhere to CPAP treatment.[3,4] A large observational study in more than 1,500 men, including 235 with untreated and 372 with treated severe disease in a 10-year follow-up, found that untreated severe OSA independently increased the odds of fatal and nonfatal cardiovascular events (odds ratio 2.87 and 3.17, respectively) compared to those treated with CPAP.[3] Lee et al[3] reported on 105 patients who underwent primary percutaneous intervention for myocardial infarction and found a prevalence of OSA (defined by AHI ≥ 15 events/h) of 65.7%. Patients with OSA and established coronary artery disease who had undergone primary percutaneous intervention treatment and received CPAP therapy have a significantly lower 5-year death rate compared to patients who do not receive CPAP (3% vs 10%).[4]

While CPAP has been shown to mitigate CV effects of OSA, the Sleep Apnea Cardiovascular Endpoints (SAVE) trial, a large randomized controlled trial, showed no significant differences in cardiovascular event incidence in patients treated with CPAP vs patients not treated with CPAP, despite CPAP significantly reducing snoring and daytime sleepiness. However, patients exhibited adherence only 3.3 hours per night and there was a trend toward improved benefit with increased usage.[5]

Studies show that alternative treatments for OSA, such as uvulopalatopharyngoplasty, also contribute to reduction in cardiovascular disease. A large retrospective cohort study of 192,316 patients with newly diagnosed OSA reported that patients who underwent uvulopalatopharyngoplasty had reduced incidence of congestive heart failure compared to those who received no treatment (hazards ratio 1.66 and 0.71, respectively).[6]

Upper airway stimulation is a novel therapy that has demonstrated treatment success OSA in select patients who have failed CPAP therapy.[3,7] The Stimulation Therapy for Apnea Reduction (STAR) trial followed patients for 5 years and measured improvement of AHI, oxygen desaturation index, adverse effects, sleepiness, quality of life, and snoring. AHI response rate (AHI < 20 events/h and > 50% reduction) was 75%. The responder rate was 63% at 5 years. Serious device-related events were reported in 6% of the patients. Improvement in sleepiness (Epworth Sleepiness Scale) and quality of life as measured by the functional outcomes of sleep questionnaire were observed, with normalization of scores increasing from 33% to 78% and 15–67% respectively.[2] However, patients with severe cardiopulmonary disease were excluded from this trial.

Hypoglossal nerve stimulation was Food and Drug Administration approved in 2014 for the treatment of moderate and severe OSA in adults. Favorable selection criteria included a body mass index of 32, limited central apnea, and a favorable pattern of palatal collapse during drug-induced sleep endoscopy. Treatment with upper airway stimulation vs no treatment is estimated to be a cost-effective therapy in the US health care system.[8]

When implanting permanent devices it is important to take into consideration future health care needs of the patient. UAS is compatible with other implanted devices, such as cardiac pacemakers. Furthermore, the updated UAS devices are approved for magnetic resonance imaging of head, neck, and extremities.

A review of the English literature yielded 1 study on the effects of UAS on cardiovascular risk.

Using data from the STAR trial, Dedhia et al[9] noted heart rate variability reduction during sleep in patients successfully treated with upper airway stimulation over a 12-month period. Heart rate variability is a measure of autonomic dysfunction and can monitor changes in cardiovascular control. Increased heart variability is associated with cardiovascular disease and is present during sleep in patients with untreated OSA likely secondary to oxygen saturation fluctuations. Thus, this study may suggest some mitigation of cardiovascular risk if we use heart rate variability as a surrogate marker of disease burden.

It is possible that UAS may be more effective than CPAP in ameliorating cardiovascular risk due to improved adherence. A recent multi-institutional registry publication suggested that average nightly usage of 5.6 hours in 1,017 patients.[10] However, the definitive impact on cardiovascular risks would need to be studied in prospective trials. Furthermore, there is scant information on the effect of OSA treatment on patients with coexisting heart failure. Nakashima et al[7] documented that OSA inhibits the recovery of left ventricular function in patients with acute myocardial infarction following percutaneous coronary intervention. A total of 37 patients with AHI ≥ 15 events/h were identified in a cohort of 86 consecutive patients who underwent primary percutaneous coronary intervention. AHI correlated negatively with left ventricular ejection fraction and regional wall motion.

Our patient demonstrated a significant improvement in functional class and left ventricular ejection fraction following successful treatment with UAS. To our knowledge, similar cases have not been reported to date. Given the high prevalence of OSA in patients with heart failure, UAS may be an important consideration in the comprehensive multidisciplinary treatment of heart failure in patients with OSA who are CPAP intolerant. Further clinical studies are required to investigate this relationship.

ABBREVIATIONS

AHI, apnea-hypopnea index
CPAP, continuous positive airway pressure
OSA, obstructive sleep apnea
SAVE, Sleep Apnea Cardiovascular Endpoints

STAR, Stimulation Therapy for Apnea Reduction
UAS, upper airway stimulation

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DISCLOSURE STATEMENT

All authors have seen and approved this manuscript. The authors report no conflicts of interest.