Case Report

Syncope in Hypoxemic Respiratory Arrest

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Abstract

This is a case report of an 81 year-old female with severe obstructive sleep apnea and hypoxemia presenting with recurrent episodes of unexplained syncope. Treatment with continuous positive airway pressure eliminated the syncopal episodes.

ABBREVIATIONS

OSA: Obstructive Sleep Apnea; CPAP: Continuous Positive Airway Pressure; SDB: Sleep Disordered Breathing; EEG: Electroencephalography

CASE PRESENTATION

An 81 year-old female with a history of paroxysmal atrial fibrillation, hypertension, and hypercholesterolemia presented for sleep evaluation due to recurrent episodes of apparent syncope over the preceding 18 months, unresolved despite an extensive workup. The patient had no recall of these episodes, which most occurred at home, and were witnessed by family members. The episodes were described as a sudden loss of consciousness while sitting quietly or lying down, becoming unarousable for up to 3 minutes, often triggering a call to EMS. There were no specific triggers, no seizure activity or incontinence present. The patient would resume consciousness after the events, and described no pain, nausea, or other associated symptoms. The last episode was preceded by a loud snort after she apparently dozed off while watching television. During multiple hospital emergency admissions due to loss of consciousness, extensive neurological and cardiac evaluations, including several electrocardiograms and Holter monitoring were non-revealing. A pacemaker was implanted due to concerns regarding potential bradyarrhythmias despite the negative cardiac evaluation. A sleep evaluation was requested in order to complete her work-up. Her sleep history revealed long-term snoring. There were no reports of other sleep related problems. The history was limited by the fact that she was widowed and slept alone despite sharing a home with her son. On physical exam, she presented as a thin and fragile elderly lady, with normal vital signs, body mass index of 25 kg/m², normal oropharyngeal exam, clear chest to auscultation, regular rate and rhythm, 2/6 systolic murmur at the left upper sternal border and no lower extremity edema. Resting oxygen saturation during wakefulness was 93%, compatible with a PaO2 of 75 mm Hg seen on previous arterial blood gases. An overnight sleep study was performed.

The detailed sleep study findings are demonstrated in (Figure 1). Loud snoring with frequent obstructive apneas (apnea-hypopnea index was 60 events/hour) and severe oxygen desaturation to below 60% were documented shortly after sleep onset due to prolonged obstructive apneas, which lasted an average of 30 seconds. Oxygen levels failed to recover to normal levels in-between events as a result of recurrent apneas. After 23 minutes of sleep recording, emergent treatment with Continuous Positive Airway Pressure (CPAP) was implemented. After CPAP initiation, oxygen saturation levels progressively increased. The titration was continued to 14 cmH2O. At this pressure level, respiratory events and snoring were mostly eliminated. No arrhythmias were detected.

The dramatic oxygen desaturation seen in this patient in association with severe obstructive sleep apnea (OSA) was the most likely etiology of her recurrent episodes of loss of consciousness. Impending respiratory arrest was witnessed by the technicians during the night, associated with the severe and sustained oxygen desaturation shortly after sleep onset, shown by the arrows in (Figure 1). During CPAP application, the patient was mostly asleep based on electroencephalographic (EEG) monitoring. No abnormal EEG activity, including slowing or spikes suggestive of seizure activity were seen. This patient was sent home on CPAP. She is currently 86 years-old and has been followed for 5 years with excellent CPAP compliance. One recurrent episode of loss of consciousness occurred while temporarily off CPAP due to an upper airway infection, and in the past 2 years, no further episodes have occurred.

DISCUSSION

There has been a suggestion that hypoxemia in OSA may elicit cardiac vagal reflex and sympathetic activation in the peripheral vasculature, and one report in the literature describes severe...

hypoxemia leading to cardiopulmonary arrest during a sleep study in a non-obese patient [1,2]. We believe that the syncopal episodes in our patient were related to similar mechanisms. In predisposed individuals, the triggering of a vagal reflex leads to bradycardia, a drop in blood pressure, and loss of consciousness, and results in vasovagal syncope [3,4]. In a report of 11 patients with sleep disordered breathing (SDB), including both OSA and upper airway resistance syndrome, and vasovagal syncope, the authors hypothesized that the intermittent hypoxia, sleep fragmentation, and large variations of intra thoracic pressures resulting from respiratory efforts in these patients may lead to chronic adaptations in the autonomic nervous system [5]. Situations of strong stimulation of the sympathetic system may trigger a secondary vagal hyper reactivity, potentially leading to recurrent vasovagal episodes [5]. Furthermore, parasympathetic/vagal hyperactivity during apneas may induce long sinus pauses and second degree atrio-ventricular blocks in some patients [6,7]. Apnea-related sympathetic hyperactivity may persist into the daytime [8], partly accounting for the increased incidence of high blood pressure in some patients with OSA. The parasympathetic hyper reactivity in these patients may also be present during the daytime, detected by Valsalva or Mueller maneuvers [9,10]. Finally, atrial stretch during repeated respiratory efforts equivalent to Mueller maneuvers during airway obstruction may lead to the release of atrial natriuretic peptide, which can result in reductions in blood volume and a reduction of blood pressure [11].

Treatment of SDB with CPAP has been demonstrated to improve the sympathovagal balance [12-14]. The effectiveness of this treatment for improving daytime syncopal episodes in our patient, as well as in the 11 patients with vasovagal syncope described earlier [11] bolsters the hypothesis that this improvement persists into the day. Longer follow up and randomized controlled studies in the future may further elucidate the use of CPAP as a means of treating syncope in the face of SDB. The case here reported highlights the necessity of broadening the differential diagnosis of unexplained syncopal episodes and respiratory arrests to include life-threatening sleep related respiratory abnormalities.
REFERENCES


