Case 12. Watchful Waiting

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History

A 62-year-old man presents with several years of snoring and, more recently, progressive daytime sleepiness over months. He reports that he has multiple awakenings throughout the night, some of which he recalls being associated with choking/gasping during sleep. He reports a need to urinate 1–2 times/night. There are also reports of witnessed breathing pauses noted by his bedpartner. He feels unrefreshed upon awakening and fatigued throughout the day. He rarely wakes up with a headache. He does wake up with a dry mouth frequently. His Epworth Sleepiness Scale score is 12/24. His sleep schedule is similar on workdays and days off. He has a lights out time of 10:00–10:30 PM, a short sleep latency of a "few minutes", and a final rise time of 6:00 AM. The patient takes a daily nap in his recliner chair for about 15–20 min every afternoon or evening.

His prior medical and surgical histories are notable for gastroesophageal reflux disease (GERD) confirmed on esophagogastroduodenoscopy (EGD), hypertension, hyperlipidemia, chronic rhino sinusitis, and left knee replacement. Family history is notable for coronary artery disease, with both his father and older brother having

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suffered myocardial infarctions. He is a nonsmoker. Alcohol consumption is less than one drink per week. He has two caffeinated beverages in the morning.

His medications include omeprazole, amlodipine, losartan, atorvastatin, fluticasone nasal spray.

Examination

On physical exam, his blood pressure is elevated at 151/100 mmHg, pulse = 96 beats/min, afebrile, oxyhemoglobin saturation on room air = 96%; height = 72 in., weight = 264 lb; body mass index (BMI) = 35.9 kg/m^2. His modified Mallampati score is 3; cardiac, pulmonary, extremity, and neurological exam are all normal.

Investigations

Due to a high suspicion of sleep-disordered breathing, i.e. obstructive sleep apnea (OSA), he is scheduled for an in-laboratory split-night study. The baseline/diagnostic portion of the study demonstrates severe OSA with an apnea-hypopnea index (AHI = 64.6/h) using the American Academy of Sleep Medicine (AASM) criteria of hypopnea scoring (i.e., 3% desaturation or arousal). The oxygen saturation nadir was 78%, and for ~41 min of the 143 min sleep time during the diagnostic portion the oxygen saturation was <90%. Positive airway pressure (PAP) therapy was initiated at a pressure of 4 cm H_2O. Central respiratory events emerged and persisted for the remainder of the titration portion of the study—pressure range of 4–10 cm H_2O. The AHI on PAP therapy ranged from 18.1 to 98.9/h—all central apneic events. Figure 12.1 is a sample of the findings from the baseline portion of the study (Panel A) and after initiation of PAP treatment (Panel B). The differential diagnoses included concurrent OSA and central sleep apnea (CSA), secondary to another CSA disorder such as congestive heart failure or a medication or substance. However, in the setting of concurrent OSA and CSA, the central AHI is ≥5 events/h during the diagnostic polysomnogram (PSG). Given that there were no central respiratory events during the diagnostic PSG, *a diagnosis of treatment emergent central sleep apnea (TECSA) was made*. The patient was prescribed auto titrating PAP therapy, as he was not able to return for a full titration PAP.

At the patient's follow up visit ~4 months later, he reports that he is no longer snoring, that his sleep is less fragmented, and that he wakes up refreshed. The patient's PAP adherence reports demonstrate an average adherence of 6 h and 38 min/night. PAP therapy was used every night and adherence of ≥4 h/night was seen 93% of the time. His 95th percentile pressure was 9.8 cm H_2O. His leak was 4.2 L/min, suggesting a good seal with the mask interface. His overall AHI was 6.5/h (decreased from 64.6/h). His central apnea index (CAI) decreased to 4.1 events/h, and his obstructive respiratory event index 1.4/h, suggesting that his OSA was treated and TECSA had resolved.

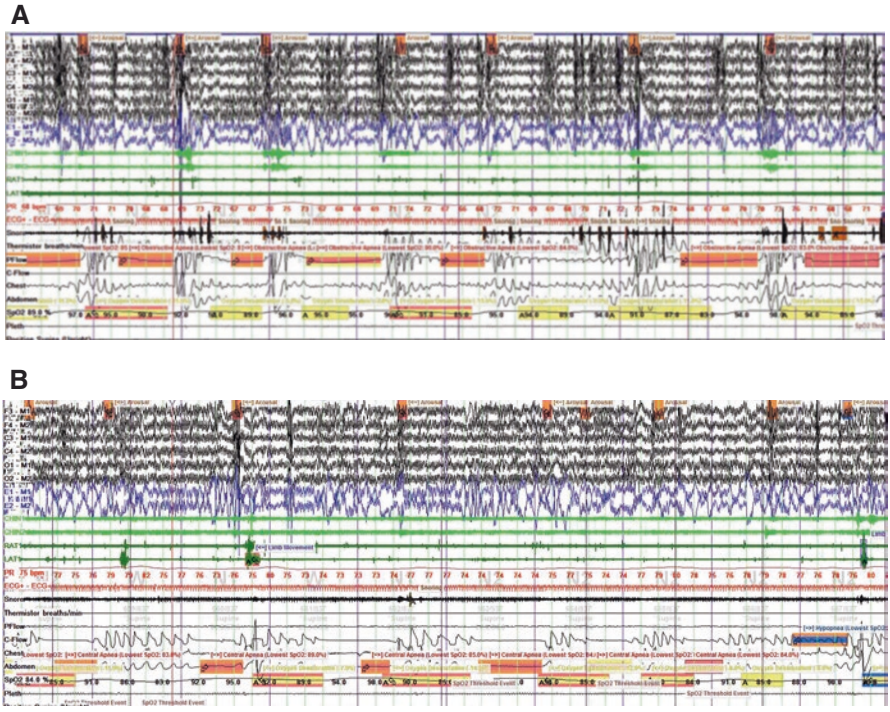

Fig. 12.1 (**A**, **B**) Split-night in-laboratory study

Final Diagnosis

Obstructive sleep apnea with treatment emergent central sleep apnea

Discussion

TECSA is defined by the following criteria according to the International Classification of Sleep Disorders—Third Edition [1]: (1) the presence of predominantly OSA on diagnostic PSG; (2) resolution of obstructive events with PAP therapy without a backup rate; (3) the emergence or persistence of central respiratory events on PAP therapy with a central AHI ≥5/h and central events are ≥50% of the total apneas and hypopneas; and (4) the CSA is not better explained by another CSA disorder. While, the overall prevalence of TECSA is estimated to be between 5% and 20%, there is uncertainty regarding the true prevalence given that central and obstructive hypopneas are not differentiated on most sleep studies. Demographic factors associated with TECSA include male sex, older age, lower body mass index,

congestive heart failure, coronary artery disease, and opioid use. Moreover, baseline PSG and PAP titration factors can also be associated with TECSA. For example, more severe OSA, a high baseline CAI or mixed apnea index, high arousal index, a split-night study, aggressive PAP titration, high residual AHI, and lower totals sleep time and/or sleep efficiency are reported to be associated with TECSA. The most common treatment of TECSA is watchful waiting. Between 54% and 86% of TECSA cases resolve within 2–3 months of PAP use [2]. This approach requires close monitoring of symptoms, encouraging PAP adherence, and optimizing treatment of comorbid conditions associated with CSA (e.g., heart failure, opioid use). For those with persistent symptoms or an AHI ≥15/h on follow-up, alternate therapies such as bi-level pap therapy in spontaneous timed mode (BPAP-ST) or adaptive servo-ventilation (ASV) can be considered.

References

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