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Reversible exacerbation of obstructive sleep apnea by α1-adrenergic blockade with tamsulosin: A case report

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Abstract

Obstructive sleep apnea (OSA) is characterized by repeated involuntary closure of the pharyngeal airspace during sleep. Normal activity of the genioglossus (GG) muscle is important in maintaining airway patency, and inhibition of GG activity can contribute to airway closure. Neurons in the hypoglossal motor nucleus (HMN) regulate GG activity. Adrenergic tone is an important regulator of HMN neuronal excitability. In laboratory models α_1-adrenergic antagonists inhibit HMN neurons and GG activity, suggesting that α_1-adrenergic antagonism might adversely affect patients with OSA. To date there has been no report of such a case. Case Summary: The patient was a 67-year old man with a 27-month history of obstructive sleep apnea. Diagnostic polysomnography demonstrated a baseline apnea-hypopnea index (AHI) of 21.3 and a trough oxygen saturation of 84%. Treatment with continuous positive airway pressure (CPAP) was initiated. The AHI in year 1 averaged 1.0 ± 0.1 (mean ± SD) and 0.8 ± 0.1 in year 2. Other medical conditions included hypertension controlled with losartan and benign prostatic hypertrophy not well controlled by finasteride monotherapy. The α_1-adrenergic receptor antagonist tamsulosin 0.4 mg daily was added. Shortly after initiation of tamsulosin, subjective sleep quality deteriorated. Significant surges in obstructive events, apneic episodes, and AHI were also recorded, and nocturnal airway pressure was frequently sustained at the CPAP device maximum of 20 cm H_2O. Tamsulosin was discontinued. CPAP parameters and sleep quality returned to the pre-tamsulosin baselines within 10 days. These findings suggest that α_1-adrenergic blockade with tamsulosin may exacerbate sleep-disordered breathing in susceptible patients.

Keywords

AHI; CPAP; obstructive sleep apnea; prostatic hypertrophy; tamsulosin; alpha-adrenergic antagonist