A Case of Sleep Apnea Related Nocturnal Heart Block

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Introduction

Obstructive sleep apnea (OSA) is characterized by recurrent episodes of partial or complete upper airway obstruction leading to cessation of breathing while asleep. Obstructive sleep apnea is associated with significant cardiovascular morbidity and mortality. It is a risk factor for arrhythmias ranging from asymptomatic sinus bradycardia to sudden cardiac death. Nocturnal heart block often occurs in patients with OSA. The exact mechanisms underlying the link between OSA and cardiac arrhythmias are not well delineated, but patients with OSA may develop nocturnal heart block as a consequence of the autonomic effects of recurrent apneas with subsequent oxygen desaturation, cyclic fluctuations in sympathovagal tone, and cardiac hemodynamic changes. In 1982 Miller et al reported a 9-13% prevalence of heart block in patients with OSA, while Guilleminault et al reported that almost 18% of OSA patients developed bradycardic arrhythmias.

History of Present Illness

A 52-year-old male with a history of end stage renal disease on hemodialysis, diabetes type 2, and peripheral arterial disease, presented to the Emergency Department (ED) with a non-healing left foot ulcer. An electrocardiogram (EKG) done in the ED was consistent with 2nd degree AV block with 2:1 conduction, and probable Mobitz I. There was an underlying 1st degree AV block with a PR interval of approximately 280msec. An MRI of the left foot revealed osteomyelitis. A left foot wound vac was placed, and patient was discharged for outpatient bone biopsy and Infectious Disease follow-up. Outpatient cardiology follow-up was also requested to address any potential arrhythmias and the need for further evaluation and treatment. Upon outpatient consultation with cardiologist, a sleep study was ordered. The sleep study revealed severe OSA, with an Apnea Hypopnea Index (AHI) of 73/hr, worse in the supine position and associated with oxygen desaturation down to 50% from baseline 98%. CPAP was unable to resolve OSA completely, so bilevel positive airway pressure (BPAP) was administered with successful resolution of OSA, and stabilization of oxygen saturation at 96-98%. Continuous EKG done during the sleep study revealed 3rd degree atrioventricular block with junctional escape rhythm (heart rate 64bpm) during apneic episodes, that improved to patient’s baseline 1st degree heart block with therapeutic BPAP. Patient was prescribed nocturnal BPAP to treat sleep apnea and apnea-associated heart block, and pacemaker was deferred.

Discussion

In 2003, Roche et al investigated the relationship between obstructive sleep apnea and cardiac arrhythmias in a prospective cohort study, and found that nocturnal paroxysmal asystole was significantly more prevalent in OSA patients, and the number of episodes of bradycardia and pauses increased with the severity of the sleep apnea. Almost all bradycardic events occurred in patients with severe OSA, and prolonged desaturation. In 1998 Koehler et al reported that the degree of bradycardia appeared to be related to the severity of hypoxemia. Several previous studies suggested that the administration of supplemental oxygen attenuated the bradycardia, but a study by Maeno et al indicated that OSA can provoke heart block even without oxygen desaturation.

The interaction between sleep disordered breathing, the autonomic nervous system, and cardiovascular function is complicated. As the duration of sleep apnea progresses, negative intrathoracic pressure from the continuous effort to breath against a closed airway intensifies, and results in increasing vagal tone. Simultaneous blood pressure and stroke volume fluctuations trigger baroreceptors and intracardiac mechanoreceptor reflexes, further enhancing vagal stimulation. Early morning increase in sympathetic activity and rapid eye movement (REM) sleep, both associated with prolonged apneas and more severe desaturations, may also trigger arrhythmic events in OSA. CPAP treatment has been shown to ameliorate the blunted daytime cardiac parasympathetic tone associated with repeated nocturnal stimulation.

It has been speculated that the majority of OSA-related nocturnal arrhythmias can be reversed by therapeutic positive airway pressure (PAP) treatment. If the underlying cardiac conduction system lacks pathological abnormality and there is no significant arrhythmia or heart block occurring during non-apneic periods, nocturnal OSA related heart block may be treated effectively with PAP. In a 1995 study by Becker et al, of 239 patients had OSA related bradyarrhythmias, and 16 of them had successful treatment with CPAP. Harbison et al showed abolition of rhythm disturbances with CPAP in seven of eight patients with nocturnal arrhythmias. The reversibility of apnea related nocturnal arrhythmias by PAP has been demonstrated in the majority of patients with OSA, with a favorability of 72–80% abolishment of nocturnal heart block. This raises the possibility that PAP may reduce the need for cardiac pacemakers in those patients with heart block.
occurring exclusively during OSA without obvious pathological abnormality.

Considering the high prevalence of OSA in heart block patients, a polysomnogram should be ideally offered to patients suffering from nocturnal heart block, with trial application of positive airway pressure to abolish apneas and apnea-associated heart block.

**Figure Legends**

**Figure 1**: Baseline electrocardiogram during apnea showing high degree heart block with junctional escape rhythm. Blue arrows: P waves with a regular P-to-P interval (0.6 seconds) marching independent of QRS complexes.

**Figure 2**: Therapeutic BPAP has been administered showing resolution of high degree heart block, with a return to patient’s baseline 1st degree heart block.

**REFERENCES**


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