CLINICAL VIGNETTE

Can't Sleep, Can't See: Sleep Disordered Breathing and Non-arterial Ischemic Optic Neuropathy

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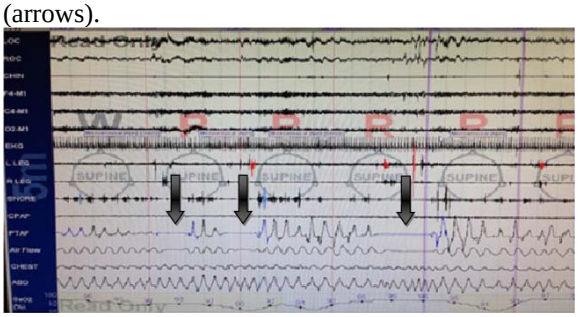
A 56-year-old man was referred for evaluation for sleep disordered breathing. He was encouraged by his wife to seek evaluation for a long history of snoring and periods of breathing cessation during sleep. He reported significant excessive daytime sleepiness with an Epworth Sleepiness Scale of 4. The Beck Depression Inventory was also 4.

The patient was recently diagnosed with non-arterial ischemic optic neuropathy (NAION) by neuroophthalmology upon evaluation for vision loss. Other past history was notable for allergic rhinitis with a distant history of facial trauma requiring nasal reconstruction, varicella zoster, and well-controlled depression. Medications included sertraline, finasteride, as needed zolpidem, and cetirizine with pseudoephedrine. Prednisone at 20mg daily was recently started for NAION. He was married and was employed as a lawyer. He did not use tobacco and occasionally consumed 2 servings of alcohol and denied any other recreational substances.

Vital signs in clinic revealed a temperature of 97.2, blood pressure of 118/79 mmHg, pulse of 86 beats per minute, respiratory rate of 18 breaths per minute, and an oxygen saturation of 98% breathing ambient air. His height was 6 feet 2 inches, and weight was 207 pounds with a body mass index of 26.6. On exam he was comfortable sitting on an exam table. Head and neck exam was unremarkable including normal nasal mucosa. The remainder of the exam was also unremarkable.

Review of laboratory values including a complete blood count and metabolic panel were normal. Thyroid stimulating hormone, erythrocyte sedimentation rate and rheumatoid factor were also normal. A polysomnogram revealed an apnea hypopnea index of 19 events per hour which increased to 72 during rapid eye movement (REM) sleep (Figure 1). This normalized with application of continuous positive airway pressure (CPAP) therapy titrated to 9 cmH2O. Periodic limb movements associated with significant sleep disturbance improved, but did not completely resolve, with CPAP at 9 cmH2O.

Figure 1: Polysomnogram with obstructive apneas (arrows).



Discussion

Sleep apnea syndromes (SAS) are, as the name implies, nocturnal breathing disorders that occur only during sleep. These patients suffer from cessation of air flow. Etiologies include collapse of a narrowed upper airway or loss of central drive for respiration when voluntary respiratory efforts cease as they fall asleep. The apneas are terminated by micro-arousals, which are accompanied by sympathetic surges, allowing the patient to resume respiration until airflow is again interrupted. Untreated SAS are associated with increased cardiovascular events including stroke, myocardial infarction, arrhythmia and hypertension as well as increased all cause mortality.

SAS occur quite commonly in the general population with prevalence of 9% in men and 2% in women. Prevalence increases dramatically up to 50% or more in specific populations with underlying cardiovascular disease, particularly with a history of congestive heart failure and stroke. Interestingly, there is a very high incidence of SAS in patients with NAION. NAION is a common cause of unilateral, painless visual loss in the elderly. The loss in vision is acute and permanent. Symptoms are most often

noted upon awakening. Its incidence rate is 2.3-10.2 per 100,000 in the population greater than 50 years old[1,2]. The prevalence of SAS in the patients with NAION has been reported to be between 71-89%[3,4]. Approximately 30% of patients with NAION will continue to suffer further visual loss over several weeks. Recurrence occurs in 6.4% in the ipsilateral eye and 14-24% in the contralateral eye[5,6,7]. Risk factors for NAION include diabetes, hypertension, hyperlipidemia, atherosclerosis and small optic cup to disc ratio. More recent studies added SAS, specifically, obstructive sleep apnea (OSA) as a risk factor for NAION.

NAION results from a decrease in perfusion of posterior circulation of the globe of the eye, primarily involving the posterior ciliary artery supplying the optic nerve head. Ischemia results in edema of the optic disc which may further compromise blood flow. The damage on the optic nerve head causes defects to occur in the patient's visual acuity, visual field or both. The patient may also develop peripheral scotomas or quadrantic visual field loss without compromise of visual acuity. OSA is postulated to increases the risk of NAION via several mechanisms. First, optic nerve vascular dysregulation may occur during apneic episodes with associated wide fluctuations in blood pressure between very low levels and the hypertensive range. These swings superimposed on atherosclerotic vessels ultimately result in periods of hypoperfusion of the optic nerve. Second, impaired auto-regulation of the microcirculation supplying the optic nerve head may result from repetitive apneic episodes. Third, direct damage to optic nerve may occur from repetitive hypoxemic insults related to cessation of airflow. Finally, intracranial pressure may increase with airway obstruction and increased respiratory effort worsening papilledema compromising blood flow to the posterior ciliary artery[3,8,9].

It is interesting that most patients with OSA did not have a clinical history suggestive of a sleep related breathing disorder in Palombi's study of 27 consecutive patients with NAION. The mean Epworth sleepiness score was only 7 (normal <10) and only 53% of patients reported snoring. Yet overnight polysomnography confirmed OSA in 89%, using a threshold AHI of 15/hr[3].

Unfortunately, there is no proven effective treatment for NAION. Antiplatelet therapy with aspirin is generally recommended with a possible decrease in risk of development of NAION in the contra-lateral eye[10]. CPAP therapy was evaluated in a very small group of patients with NAION and OSA by Behbehani et al which did not alter the in progression of NAION[11].

Conclusion

The list of end organs negatively impacted by SAS continues to grow with ongoing investigation. Suspicion for presence of OSA should be high in patients presenting with acute visual loss related to NAION. Although there is paucity of data supporting OSA treatment to improve outcomes in NAION, treatment remains important to reduce overall cardiovascular risk and to improve quality of life.

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