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The 5 Most Common Ocular Manifestations of Obstructive Sleep Apnea

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Editor's Note:

Obstructive sleep apnea (OSA) is associated with numerous comorbid conditions. In many, a causative relationship has either been well established or strongly associated. As the knowledge of sleep-disordered breathing and its consequences continues to grow, so does the list of associated or consequential conditions. The following is part 4 of a 5-part series exploring more recently identified consequences if OSA.

Introduction

Several ophthalmologic conditions have been found to have a clear association with OSA. Similarly, OSA may present with several ocular manifestations. Simple and benign findings, such as drooping of the eyelids, are a common and early sign of sleepiness, whereas other sequelae may herald a more serious underlying condition.

It is important for optometrists, ophthalmologists, and primary care providers to be aware of these physical findings and unusual associations, because they may represent a potentially vision-threatening disorder. Routine screening for sleep-disordered breathing in these conditions is highly recommended.

Floppy Eyelid Syndrome

Floppy eyelid syndrome (FES) is the most common ocular disorder associated with OSA.[1] FES is characterized by papillary conjunctivitis and a rubbery, redundant upper eyelid tissue. Similar to OSA, FES is more prevalent in obese, middle-aged men. Of note, FES frequently presents unilaterally, affecting the eyelid on the side the patient most often sleeps on.

FES is observed in 2%-5% of patients with OSA. However, among patients with FES, the prevalence of OSA approaches 90%.[1,2] The association of OSA and FES was explored in a case/control study by Ezra and colleagues.[3] In this study, 102 patients with FES were compared with 102 matched controls. OSA was identified in 32 of 102 patients with FES (31.4%), compared with only 9 of 102 controls (8.8%).

Glaucoma

Several studies have shown a higher prevalence of both primary open-angle glaucoma and normal-tension glaucoma among patients with OSA.[1,4,5] One study reported the prevalence of OSA was 27% among patients with glaucoma, whereas another concluded that patients with OSA were 4 times more likely to have glaucoma and visual field defects than age-matched controls. Furthermore, glaucoma severity appears to correlate with both the frequency and duration of apneic episodes.

Although the specific etiology of this association is not well described, one proposed mechanism suggests damage to the optic nerve head caused by apnea-induced ischemic events. Similarly, OSA is thought to cause poor autoregulation of the optic nerve vascular endothelium, which elicits further ischemic damage. This may explain the relative lack of intraocular pressure elevation frequently seen in patients with both glaucoma and OSA. In a study by Bendel and colleagues,[4] the authors concluded that OSA should be considered in patients with normal-tension glaucoma without classic risk factors, especially if medical and surgical therapies have failed.

While it seems that there is a clear association between OSA and some forms, or perhaps presentations, of glaucoma, there is ongoing controversy regarding the benefits of treatment. Continuous positive airway pressure (CPAP) remains the most efficacious treatment option for OSA and is thought to resolve the pathophysiologic derangements that result from upper-airway collapse, intermittent desaturations, sleep fragmentation, and sympathetic activation. As such, it should alleviate, or at least improve, glaucoma.

In a study by Kiekens and colleagues,[5] both intraocular pressure and ocular perfusion pressure were measured in patients with OSA before and after 1 month of CPAP therapy.[5] At baseline, untreated OSA was associated with significant fluctuations in intraocular pressure. Not surprisingly, the highest intraocular pressure recordings were noted

during the night. However, CPAP therapy was associated with a significant increase in intraocular pressure from baseline. These pressures tended to decreased 30 minutes after CPAP cessation. Similarly, ocular perfusion pressure significantly decreased during CPAP therapy. The authors concluded that CPAP therapy may cause an additional increase in intraocular pressure with a subsequent reduction in ocular perfusion. Given this, the proper treatment of OSA-associated glaucoma remains unknown.

Papilledema

Papilledema, or optic disc swelling, is a frequent manifestation and hallmark feature of elevated intracranial pressure/intracranial hypertension. Several case series and case reports have found an association between papilledema and untreated OSA.[1,6,7]

Potential mechanisms regarding the role of OSA in papilledema have been proposed. OSA results in forced inspiration against a closed airway, which leads to an increase in venous pressures and impaired venous return. This can produce impaired venous drainage of the cranial vault, with subsequent increases in intracranial pressure. In addition, OSA leads to retention (or impaired elimination) of carbon dioxide. Hypercapnia is known to induce cerebral venous dilation, which produces transient elevations in intracranial pressure and is one of the mechanisms by which OSA induces headaches and headache disorders.

Among patients with OSA, CPAP therapy has been shown to improve or resolve papilledema.[7] Of note, high mean airway pressures may alter the transpleural pressure gradient and further impair venous return, and CPAP therapy should be used with caution in certain individuals, particularly those with organic causes of intracranial hypertension. Among patients with nontraumatic papilledema and normal neuroimaging results, OSA should be suspected and patients should undergo further evaluation.

Nonarteritic Anterior Ischemic Optic Neuropathy

Anterior ischemic optic neuropathy results from impaired perfusion of the optic nerve head, or optic disc. Arteritic anterior ischemic optic neuropathy is almost always associated with underlying giant cell arteritis. Nonarteritic causes are frequently idiopathic. However, there is a strongly suggested association with OSA.[8,9]

Several small studies and case series have found that the incidence of OSA is higher in patients with nonarteritic anterior ischemic optic neuropathy, with a prevalence approximately 2.5 times higher than in controls. In a case/control study of consecutive patients with nonarteritic anterior ischemic optic neuropathy, 71% were found to have underlying OSA, compared with only 18% of controls.[8] The mechanism of action is not well understood, but it is likely that acute and intermittent apnea-induced increases in blood pressure and intracranial pressure, along with intermittent desaturations and nocturnal hypoxemia, result in optic nerve edema and impaired perfusion.

Retinal Vein Occlusion

Retinal vein occlusion is one of the most common nondiabetic causes of blindness. It occurs as a consequence of impaired venous return of the retina and atherosclerotic defects of the feeding arterioles. Several studies have found that retinal vein occlusion is more common among patients with OSA.[10,11]

The prevalence of both conditions rises with increasing age, and both are associated with hypertension, diabetes, and atherosclerosis. Whether a true causative relationship exists or these 2 conditions concomitantly occur in individuals with similar associated comorbid conditions is not well understood. However, there appears to be a clear association.

Most patients with retinal vein occlusion discover vision loss on awakening, and snoring is commonly reported. In a study of 63 consecutive patients with retinal vein occlusion, 30 (47.6%) reported snoring and daytime sleepiness. Polysomnography in these 30 individuals identified OSA in 23 (77%), with a mean apnea/hypopnea index of 21 events/hr. In an intention-to-treat analysis, the authors concluded that even if all 33 untested patients did not have sleep-disordered breathing, the prevalence of OSA would still have been 37%.

Similarly, in a study of 40 consecutive patients with retinal vein occlusion, 37% were found to have evidence of sleep-disordered breathing by overnight pulse oximetry (42% of men and 33% of women).[11]

Conclusions

Several ocular manifestations of OSA have been described, and there appears to be a more than just a chance relationship between OSA and certain ophthalmologic conditions. The conditions with the greatest known association with OSA include FES, glaucoma, papilledema not associated with trauma or a space-occupying lesion, nonarteritic anterior ischemic optic neuropathy, and retinal vein occlusion. Although the prevalence of these conditions among patients with OSA is small, the prevalence of OSA in individuals with these conditions is significantly higher than the general population. As such, patients with these 5 conditions should be queried regarding symptoms suggestive of OSA and referred for further evaluation.

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