Clinical Issues: What is the importance of Glaucoma and Obstructive Sleep Apnea Syndrome?

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• Relatively common, increasingly recognized but still under-diagnosed, Obstructive Sleep Apnea Syndrome (OSAS) sufferers experience apneic and hypopneic episodes during sleep.
• OSAS symptoms include daytime somnolence, daytime headaches, difficulty concentrating and memory problems.
• OSAS has been correlated with ocular diseases including glaucoma and it has been identified as a possible risk factor for glaucoma.
• OSAS may alter the blood flow to the optic nerve, thus decreasing perfusion pressure.

Several studies observed a significant proportion of glaucoma patients (5.7 – 27%) suffer from OSAS.9-10 These studies ranged from 30 to over 200 patients and all patients had either a prospective PSG (i.e. after an ophthalmic exam and diagnosis of glaucoma) or were referred for ophthalmic examination directly after a positive PSG for OSAS in a consecutive manner.6-12 In 2007 Sergi and colleagues performed prospective PSG and ophthalmic examinations on 51 consecutive OSA patients (RDI≥10) and 40 age-matched controls, observing normal tension glaucoma (NTG) in 5.9% (3/51) versus 0% in the control group. In another study, 27 of 100 consecutive OSA patients who had a PSG within 2 days of ophthalmic examination (RDI≥15) were found to have glaucoma.9 Lin et al. diagnosed NTG in 12 of 209 patients (5.7%) consecutively diagnosed with OSAS by prospective PSG (RDI≥5).9 Two small studies respectively found OSAS in 3/6 NTG patients and 12/25 OAG patients, and all patients had either a prospective PSG (i.e. after an ophthalmic exam and diagnosis of glaucoma) or were referred for ophthalmic examination directly after a positive PSG for OSAS in a consecutive manner.9

Symptoms of OSAS include daytime somnolence, daytime headaches, difficulty concentrating and memory problems.1 Factors predisposing to OSAS are obesity, male gender, upper airway abnormalities (palate shape), large tongue, large tonsils, a shorter lower than upper normalities (palate shape), large tongue, snoring and enlarged neck girth.2 Most patients with OSAS do not remember waking in the night during an apnea-hypopnea episode.1 OSAS can affect the pulmonary, cardiovascular and cerebrovascular systems. The Joint National Committee on the Prevention, Detection Evaluation, and Treatment of High Blood Pressure has identified OSAS as a treatable cause of secondary hypertension.3

OSAS has been correlated with ocular diseases including glaucoma, non arteritic anterior ischemic optic neuropathy, bilateral disc edema secondary to intracranial hypertension, floppy eyelid syndrome, blepharitis, ptosis, papillary conjunctivitis, filamentary keratopathy, retinal vascular tortuosity and central serous chorioretinopathy.4,4

Elevated intraocular pressure (IOP) is the most common known risk factor for the development and progression of glaucoma. Other risk factors include thin central cornea, family history of glaucoma and optic disc hemorrhages.5 However, the progression of glaucoma can still occur with a low IOP, attributed currently in large part to factors affecting ocular perfusion, including low blood pressure, IOP fluctuation, low intracranial pressure and OSAS.

Relatively common, increasingly recognized but still under-diagnosed, Obstructive Sleep Apnea Syndrome (OSAS) sufferers experience repeated episodes of upper airway obstruction called apneic episodes, or decreases in airflow called hypopneas during sleep with varying severities. Both diagnosis and severity of OSAS can be made with formal polysomnography ( PSG) or with a home sleep test. This analyzes nasal airflow, respiratory effort and oxygen saturation. These variables determine an apnea-hypopnea index ( AHI) and respiratory disturbance index ( RDI). OSAS is defined as >5 AHI/hour, with severity graded as mild, moderate or severe.

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The severity of OSAS was reported to be correlated with the extent of glaucomatous damage.

When applying continuous positive airway pressure ( CPAP) – the most common treatment for OSAS – normalized IOP and restored blood pressure have been observed.

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and colleagues showed that CPAP normalized IOP and restored blood pressure to a normal rhythm. CPAP in patients with OSAS and glaucoma may have a beneficial effect for the glaucoma as well as the OSAS.

It is important when taking a patient history to inquire about snoring and daytime somnolence. Often a patient’s spouse or partner can elucidate if he or she snores or describe moments when he or she stops breathing for a few seconds then resumes snoring. These symptoms are possible indications for OSAS and these patients should consider a formal polysomnography. If diagnosed with OSAS, the patient should seriously consider CPAP treatment to aid glaucoma treatment as well as improve their systemic status.

References

Practical Tips:
IOP assessment with the water drinking test

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Core concepts
• To identify both IOP-dependent and non IOP dependant factors is essential to understand why progression takes place in patients.
• We are currently unable to monitor 24 hour IOP continuously.
• Surrogate measures such as inter-visit IOP variation or diurnal IOP curves, although helpful, are sometimes impractical.
• An alternative to determine IOP fluctuation and peak IOP is the water drinking test (WDT), which is simple to perform and evidence based.
• Peak WDT-induced IOP correlates well with peak diurnal IOP and may help to identify patients with fluctuating IOP peaks outside or even within routine office hours.
• The WDT requires the patient to drink a set volume of water within a short time period (usually 5 minutes).
• Due the diuretic effect of the WDT, care should be taken in individuals with cardiovascular or renal co-morbidities.
• Because of its poor sensitivity, the WDT is no longer acceptable as a provocative test to diagnose glaucoma.
• The WDT is a low cost, low tech practical alternative for cumbersome assessments of diurnal variations and peak IOPs and can highlight the need for further intervention or re-evaluation of current treatment plans.

Well-designed clinical trials provide strong evidence that elevated intracocular pressure (IOP) is a risk factor for the development of glaucoma and for progression of established disease. For example, pooled data from the Ocular Hypertension Treatment Study (OHTS) and European Glaucoma Progression Study (EGPS) suggests that for every 1mmHg higher baseline IOP, there is a relative risk of 1.09 for glaucoma development.1 IOP reduction is a proven strategy to prevent progression from ocular hypertension (OHT) to glaucoma or to slow the rate of glaucoma progression. However, patients with glaucoma progression despite IOP reduction remain a significant challenge. To identify both IOP-dependent and non IOP-depen-