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A Diplomate of the American Board of Ophthalmology, he is also a Fellow of the American Academy of Ophthalmology, the American College of Surgeons, the International College of Surgeons, the Royal College of Ophthalmology, the Association for Research in Vision and Ophthalmology, and the New York Academy of Medicine. He is a member of more than 35 scientific and medical societies, and serves on numerous medical, scientific, and editorial boards.

Dr Ritch has co-authored or edited nine textbooks and over 1,500 papers, book chapters, articles, and abstracts. He has presented nearly 700 lectures worldwide. In 1985, he founded the Glaucoma Foundation and has served as its Secretary, Medical Director, and Chairman of the Scientific Advisory Board. In 1994, he initiated the Optic Nerve Rescue and Regeneration Think Tank, which has attracted numerous researchers from other fields into glaucoma research. He also co-founded the New York Glaucoma Research Institute, the alt.support.glaucoma Internet newsgroup, the New York Glaucoma Support and Education Group, and the World Glaucoma Patient Association. He was one of the three organizers of the first annual World Glaucoma Day in 2008.

Dr Ritch has trained over 130 clinical and research fellows. The international training program that he established at the New York Eye and Ear Infirmary has hosted over 40 International Council of Ophthalmology fellows and more than 100 observers from over 40 countries. Over the last 30 years, he has dedicated much of his time to working at an international level, organizing conferences and symposia, training fellows and observers, and helping to establish residency and teaching programs. He has made fundamental contributions to the modernization of ophthalmology in many countries in Asia and Latin America.

Dr Ritch has received numerous awards during the course of his career, including the Louis Rudin Award for research in ophthalmology, the Albion O Bernstein, MD Award of the Medical Society of the State of New York for outstanding contributions to medicine, the Leadership in Education in Ophthalmology (LEO) Award, the Association for Research in Vision and Ophthalmology (ARVO) Distinguished Service Award, the Maghraby International Achievement Award and the Jose Rizal International Medal.

Dr Ritch received his BA cum laude from Harvard College and an MA in cell biology from Harvard University. He received his MD from Albert Einstein College of Medicine and, after an internship at St Vincent's Medical Center and a residency at Mount Sinai School of Medicine, received fellowships in glaucoma from the Heed Foundation and the National Institutes of Health.

Obststructive sleep apnea (OSA) or obstructive sleep apnea syndrome (OSAS) is an under-recognized disorder with important systemic implications. It is characterized by repeated episodes of upper airway obstruction during sleep, combined with daytime sleepiness. OSAS is the most severe form of intermittent upper airway obstruction. Milder forms range from primary snoring and upper airway resistance syndrome to OSA. Upper airway obstruction is exacerbated by obesity, a large tongue, small airways, a large neck, and the use of alcohol and/or sedatives. The normal physiologic balance by autoregulatory mechanisms to maintain homeostasis during sleep is upset, leading to hypoxia and sympathetic activation.

OSAS influences many facets of physiologic function, affecting the pulmonary, cardiovascular, and cerebrovascular systems. Sympathetic variations result in fluctuations in blood pressure and heart rate. Rapid eye movement (REM) sleep is marked by greater autonomic and cardiovascular instability. Autoregulation of blood flow maintains tissue perfusion to meet demand during times of cardiovascular fluctuation. When intact, the changes during REM sleep will be counterbalanced to maintain blood flow. When there is autonomic dysregulation, pathological changes ensue. OSAS is associated with pulmonary hypertension, endothelial dysfunction, coagulation abnormalities, myocardial infarction, cardiac arrhythmia, congestive heart failure, stroke, cardiac-related mortality, and all-cause mortality. Vasoconstrictors, such as endothelin-1, are increased, exacerbating vascular dysregulation. Hypoxia and subsequent reperfusion can lead to oxidative stress and inflammation, evidenced by increased levels of inflammatory markers and reactive oxygen species.

Associations of OSAS with ocular disease include glaucoma, non-arteritic ischemic optic neuropathy (NAION), intracranial hypertension with disc edema, floppy eyelid syndrome, retinal vascular tortuosity, and central serous chorioretinopathy. The association with NAION is very strong, prospective studies having shown OSAS in 71 % and 89 % of patients.^{1,2} NAION is also present upon awakening in the majority of affected patients.³

Elevated intraocular pressure (IOP) remains the most important known risk factor for glaucomatous damage and the only one proven to beneficially affect disease progression when modified. Disorders associated with reduced ocular blood flow and

ischemia, collectively termed vascular risk factors—such as migraine, Raynaud’s phenomenon, atrial fibrillation, and reduced nocturnal blood pressure—have been implicated in glaucoma. Through hypoxia-mediated damage to blood vessels and their compensatory mechanisms, OSA may alter blood flow to the optic nerve head and, in combination with other predisposing factors, lead to decreased ocular perfusion pressure.

A potentially easily modifiable systemic risk factor, OSA has been increasingly associated with glaucoma, independent of IOP. In general, the severity of OSA has correlated with the severity of the glaucoma. In studies sized from 30 to over 200 patients, the association ranged from 5.7 % to 27 %.⁴⁻⁸ Sergi et al. performed prospective polysomnography on 51 consecutive OSA patients and 40 age-matched controls, observing normal tension glaucoma in 5.9 % of patients (three of 51) in the OSA group versus 0 % in the control group.⁵ In another study, glaucoma was detected in 27 of 100 consecutive patients with moderate-to-severe OSA, a number far exceeding what would be expected by chance.⁷ The association of OSA with normal-tension glaucoma tends to be stronger than that with high-tension glaucoma. Other studies have correlated OSA with optic disc parameters, visual field defects, and electrophysiologic abnormalities.

OSA is a highly treatable disorder, the standard therapy being continuous positive airway pressure (CPAP). Oral appliances are an alternative. CPAP restores normal cardiorespiratory parameters, reduces blood pressure, and improves abnormal biomarkers—such as endothelin-1, nitric oxide, and interleukins. There are no prospective studies on the use of CPAP in slowing the progression of glaucoma.

A recent retrospective analysis of over 2 million patients belonging to a medical plan database, including 156,000 (6.9 %) patients with a diagnosis of sleep apnea, has suggested a lack of association between both high- and normal-tension glaucoma and OSA with or without CPAP treatment.⁹ Limitations included a lack of associated ophthalmic information, for example on the severity of sleep apnea.⁹

At this point, there is increasing evidence of an association, perhaps even a very strong one, between OSA and glaucoma. IOP-independent mechanisms stemming from episodic hypoxia may be the link between OSA and glaucoma. These include perfusion pressure, autonomic dysfunction, ischemia, inflammation, oxidative stress, mitochondrial dysfunction, and hypercapnea. Prospective studies with larger numbers of patients are needed to correlate these with the severity of sleep apnea in particular, but also with subgroups of glaucoma patients. Since OSA is a treatable disorder with a high degree of treatment success, we need to be cognizant of this common but largely underdiagnosed disorder as a potentially modifiable mechanism, in addition to IOP, to counteract the development and progression of glaucoma. ■

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