Editorial

Retinal nerve fiber layer thickness alterations in patients with obstructive sleep apnea

Alteraciones del espesor de la capa de fibras nerviosas de la retina en pacientes con apnea obstructiva del sueño

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Obstructive sleep apnea (OSA) is a partial (hypo-apnea) or complete (apnea) interruption of air flow for more than 10 s during sleep. When the number of apneas-hypoapneas per hour of sleep (IAH) exceed five events, it is considered as OSA, a clinical entity. The prevalence of OSA is above 20%.1,2 OSA is deemed to be an independent cardiovascular risk factor for the development of cardiovascular diseases.3 In patients with IAH > 30, the risk of fatal and non-fatal cardiovascular events is 2.8 and 3.5 higher than the healthy population, respectively.4 The intermediate mechanisms which explain said mortality increase include the development of high negative intrathoracic pressure, increased central sympathetic discharge, hypoxemia–hypercapnia and elevation of pulmonary and systemic arterial pressure.5,6 De-oxygenation and re-oxygenation events associated to apnea establish an oxidative stress which contributes to endothelial injury and the promotion of generalized atherosclerosis.7

The retina ganglion cells (RGC) are a peripheral region within the central nervous system (SNC) and, just like other neurons, suffer the above events. Hypoxia and oxidative stress are considered to be significant neuron death factors in multiple retinal pathologies such as retinal ischemia, diabetic retinopathy or glaucoma.8–10

Various studies demonstrate a high prevalence of glaucoma in AOS patients.11–13 In addition, the compromise derived from optic nerve head oxygenation and perfusion in these patients could give rise to glaucomatous optic neuropathy.14,15

The controversy stems from this point: in OSA patients with primary open angle glaucoma and high intraocular pressure (IOP) there is a feasible cause for neuropathy, but what about OSA patients with normotensive glaucoma? Is the neuropathy due to glaucoma or to the systemic perfusion alterations produced by OSA?

Optic coherence tomography (OCT) has been widely validated as a highly useful diagnostic tool in ophthalmology, mainly to assess the thickness of the retina nervous fibre layer (RNFL) in optic nerve pathologies.16,17 Recent studies have demonstrated its usefulness in neurodegenerative diseases such as multiple sclerosis or Parkinson’s.18,19 On the other hand, Karlij demonstrated that the ocular perfusion reduction secondary to hypoxia and vessel spasm can cause RNFL thinning.20

As OSA is characterized by intermittent hypoxia during many years and sleep fragmentation and could be associated to glaucoma and optic nerve dysfunction, it is worthy investigating not only the RNFL thickness differences in OSA patients compared to healthy subjects but also if these changes in OCT could be useful as biomarkers for neuronal damage.


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How would these findings assist the clinician? Would OCT changes correlate with OSA severity? Does OCT alteration influence in the decision to establish treatment? Is OCT stabilized after treatment? Perhaps these questions comprise the truly important points as well as the most difficult ones to answer.

REFERENCES


