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Review of Obstructive Sleep Apnea and the Retina

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ABSTRACT

Obstructive sleep apnea (OSA) has negative effects on the retinal vascular system, which was the focus of this review's attempt to synthesize the available research. Using the phrases sleep apnea syndrome, obstructive sleep apnea, retina, vascular tortuosity, central serous chorioretinopathy, diabetes mellitus, and subfoveal choroidal thickness, two independent researchers searched the MEDLINE/PubMed database. Patients with OSA have more tortuous blood vessels than those without OSA, less parafoveal and peripapillary artery density, and a higher incidence of retinal vein occlusions. OSA is more common in people with central serous chorioretinopathy and people who don't respond well to intravitreal anti-VEGF (-vascular endothelial growth factor) treatment for macular edema. OSA may aggravate diabetic maculopathy, increasing the risk of diabetic retinopathy, proliferative diabetic retinopathy, and macular edema. Macular choroidal thickness variations are contentious. OSA is a common syndrome with several vascular alterations throughout the body. The most impacted ocular structures are the retina and choroid, which are predominantly affected by vascular alterations. New noninvasive technologies, such optical coherence tomography and optical coherence tomography angiography, may be able to shed more light on the ophthalmological effects of OSA and provide a better understanding of retinal structures.

Keywords: Retina, sleep apnea syndrome, optical coherence tomography

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INTRODUCTION

Obstructive sleep apnea (OSA) is a chronic breathing disorder that affects sleep that is characterized by recurrent partial or complete cessations of airflow caused by upper airway obstruction. This causes sleep fragmentation, intermittent hypoxia, and hypercapnia, which in turn causes increased sympathetic nervous system activity. [1] OSA is underdiagnosed, however when all OSA severities are taken into account, the prevalence in So Paulo, Brazil, is 32.8% of the adult population. [2].

Anatomically impaired or collapsible upper airways, poor reactivity of the upper airway dilator muscles during sleep, a low respiratory arousal threshold, and an oversensitive ventilatory control system are only a few of the pathophysiological characteristics of OSA (high loop gain). [3] Patients with OSA may snore loudly and continuously, have gasping fits while they sleep, be drowsy, obese, and have a larger neck circumference. [4] OSA

Palpebral laxity, dry eye, and posterior segment abnormalities are a few ophthalmological conditions that have been linked to OSA. [5] The inner carotid arteries give rise to the retinal and choroidal vasculature, which is a terminal microvascular system. OSA and vascular conditions like diabetes mellitus and systemic arterial hypertension might alter the retina's anatomy significantly and permanently.

Recent years have seen the development of several methods for studying retinal structures, including optical coherence tomography (OCT) and OCTangiography (OCT-A), which both allow for quick in vivo histological analysis of vascular and retinal structures without the need for intravenous contrast [6,7]. Research into the specifics of the retina's architecture and vasculature may yield fresh information about the role of the retina in OSA. The goal of this study was to review the literature on OSA complications that affect the retinal vascular system.

METHODS

Two independent researchers searched the MEDLINE/PubMed database for articles published up to December 2019 using the terms "sleep apnea syndrome," "obstructive sleep apnea," "retina," "vascular tortuosity," "central serous chorioretinopathy," "diabetes mellitus," and "subfoveal choroidal thickness." We gathered articles on the adult human retina's ophthalmological

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structures. There were no restrictions on dates or languages. Additionally, primary report and review article reference lists were looked through. Publications about glaucoma and optic neuritis, duplicates, those without an abstract, answers to other articles, and case reports were all disregarded. 24 publications were chosen for this review, 24 of which were fully reviewed by the researchers based on the inclusion and exclusion criteria. The authors of the included articles were not contacted.

RESULT

Numerous diseases, such as diabetes mellitus, inflammatory bowel disease, hypoxia, advanced age, hypertension, and an elevated body mass index, can lead to pathologically increased retinal vascular tortuosity. [8] Pathological vascular alterations are anticipated because OSA is a systemic illness that affects the vascular system. By contrasting 9 patients with OSA with 7 controls who did not have OSA, Mohsenin et al [8] found that there was a considerably higher level of vascular tortuosity in OSA. When breathing stops during OSA, hypoxemia and hypercapnia develop as a result, which causes hyperlactatemia, lactic acidosis, and a subsequent drop in arterial oxygen and rise in arterial carbon dioxide levels. [9]

The authors hypothesized that endothelial dysfunction, sporadic increases in arterial blood pressure, increased venular pressure, increased intracranial pressure, and impaired cerebral autoregulation were responsible for multifactorial modifications to the retinal vascular pattern.

OCT-A is a non-invasive, dye-free diagnostic that uses light dispersion to assess the retinal layers and veins. The contrast between static (retina tissues) and dynamic (vessels) features can be used to determine the vascular plexus map. [10] With increasing OSA severity (as measured by the AHI and Saturation of Peripheral Oxygen [SpO2]), Yuetal [11] used OCT-A to evaluate retinal vascular density in 69 patients with OSA. They discovered that the changes were most pronounced in the peripapillary area due to the vessel caliber and vascular origin. [11]

The second most prevalent retinal vascular condition and a leading cause of blindness is retinal vein occlusion (RVO). [12] RVO has been linked to systemic conditions like arterial hypertension, peptic ulcer disease, cerebrovascular illness, chronic pulmonary hypertension, diabetes, hyperlipidemia, smoking, thyroid problems, and OSA in recent years. [13]

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Macular Choroidal Thickness

The choroid, which controls ocular metabolism, is the most vascularized system in the body and has a variety of vessel sizes. The thickness of the subfoveal choroidal layer can alter in inflammatory illnesses, and systemic oxygen levels regulate the oxygen levels in the choroidal layers. [14] It is unclear how systemic oxygen and carbon dioxide levels affect the choroidal circulation. Increased retinal artery diameter and vascular blood flow brought on by hypoxia and hypercapnia are the pathophysiology that has been hypothesized. [15]

In 84 eyes of 42 patients with OSA and 112 eyes of 56 controls, Ozge et al. [16] discovered that patients with OSA had a considerably thicker choroidat 0.5 to 1.5 mm from the fovea than the control group; additionally, the choroidal thickness was adversely linked with AHI in patients with OSA. The pathogenesis that has been hypothesized is that hypoxemia causes a rise in intracranial pressure, which then causes an increase in choroidal thickness.

Male sex, smoking, hyperopia, pregnancy, systemic arterial hypertension, corticosteroid use, and type A personality are all known risk factors for CSC. OSA is regarded as a risk factor for CSC as well. [17] 48 OSA patients were examined by Brodie et al. [18] who observed no discernible difference in CSC risk between OSA patients and controls. On the other hand, a meta-analysis by Wu et al.32 based on 7,238 patients from 6 studies found that patients with CSC are more likely to develop OSA; however, OSA severities were not separated.

Diabetes, age-related macular degeneration, and other neovascular retinal disorders can all cause an increase in macular thickness. [19] In individuals with macular edema, OSA has been investigated as a factor impacting the response to anti-vascular endothelial growth factor (-VEGF) treatment. Increased VEGF is a result of intermittent hypoxia and hypercapnia. [20]

Diabetic Retinopathy

The most common cause of adult blindness worldwide and a microvascular consequence of diabetes mellitus is diabetic retinopathy (DR). OSA is regarded as a DR risk factor. [21] OSA may exacerbate diabetic maculopathy by boosting the production of inflammatory cytokines as a result of frequent nocturnal desaturation episodes. OSA also raises levels of advanced glycation products and oxidative stress. [22] Based on a review of 99 patients, these authors discovered

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that individuals with diabetes and macular edema had mean AHIs that were considerably greater than those with diabetes alone.

Smith et al [23] evaluated the prevalence of DR in patients receiving CPAP treatment retrospectively and came to the conclusion that DR was considerably less common in the CPAP-compliant group than in the noncompliant group. After analyzing data from 131 patients, Nishimura et al. [23] discovered that DR was strongly and independently correlated with AHI during rapid eye movement sleep.

DISCUSSION

OSA is a frequently occurring syndrome in the adult population that is both underdiagnosed and underappreciated. 2 OSA may cause a variety of systemic effects, most notably vascular alterations. [6–9] OSA may also exacerbate inflammatory condition in conjunction with a number of vascular alterations in the body, including endothelial damage, an increase in systemic arterial pressure, and an increase in carotid artery intima-media thickness. [24] In systemic vascular disorders like diabetes and systemic arterial hypertension, the retina represents a terminal microvascular system with well-established structural changes. Improved understanding of retinal and choroidal apnea alterations, including changes in retinal structure or vasculature, is provided by new noninvasive retinal tests like OCT and OCT-A.

Vascular tortuosity, lower vascular density, and a higher risk of venular occlusion are among the vascular alterations in OSA patients that have been documented in the literature. The variation in choroidal thickness is still debatable; some groups state that OSA patients show no change, while others report weakening or even increasing thickness. More research is required to fully understand the causes of choroidal involvement, which appear to be related to other unexplained factors.

In conclusion, OSA is a common illness that causes numerous systemic vascular alterations. The most impacted ocular structures are the retina and choroid, which are predominantly affected by vascular alterations. OCT and OCT-A, two new noninvasive technologies, could improve our understanding of retinal structures and shed more light on the ophthalmological effects of OSA.

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