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Review Article

Ocular disorders associated with obstructive sleep apnea: A review

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ABSTRACT

Recently there has been upsurge in the cases of Obstructive Sleep Apnea (OSA) with increased prevalence of obesity in the general population. It has been continuously proved that OSA and metabolic syndrome go hand in hand and hence OSA predisposes an individual to a series of cardio-vascular disorders like ischemic heart disease, myocardial infarction, arrhythmia etc. In this article we have emphasized the possibility of ocular involvement in OSA patients. Several studies have shown ocular associations like floppy eye lid syndrome, glaucoma, dry eye syndrome, papilledema, keratoconus, non-arteritic anterior ischemic optic neuropathy etc. Through this review, we would like to highlight the ophthalmological associations of OSA, their pathogenesis and outcome with the treatment of OSA.

1. Introduction

Sleep disorders have become quite common these days. About 1 out of 4 men and 1 out of 10 women have sleep disorder of one type or the other. Sleep apnoea is one such common disorder. Sleep apnea was first time discovered in 1965 while monitoring abnormal breathing pattern during sleep in Pickwickian syndrome.[1] It is of 3 types i.e. central, obstructive and mixed. Central sleep apnoea is when the defect is in the respiratory centre causing decreased respiratory drive and hence ventilation. Obstructive sleep apnoea [OSA] involves the repetitive collapse of the pharyngeal airways while sleeping resulting into temporary reduced and shallow breathing i.e. hypopnoea or transient complete cessation i.e. apnoea. Mixed sleep disorder is characterised by events of central and mixed apnea.

According to the various studies prevalence of OSA is between 2-10% of the adult population and it is among the common chronic respiratory diseases.[2,3]

Clinically OSA is defined as at least 5 apnoea-hypopnoea events per hour of sleep.[4] OSA is graded into 3 categories i.e. mild, moderate and severe according to the apnoea-hypopnoea index [AHI]. Mild OSA is 5-15 events of AHI per hour, moderate OSA is 15-30 per hour and severe will be more than 30 events per hour.[5]

The treatment of OSA includes behavioural modifications, continous positive airway pressure therapy [CPAP], medicine or surgery. Untreated patients may have long-term clinical consequences including cardiovascular disorders, neurocognitive dysfunction, metabolic dysfunction, respiratory failure and daytime hypersomnolence.[6,7] In OSA, long-term hypoxia of various ocular tissues may induce several ocular disorders. In this review, we would like to highlight the ophthalmological

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associations of OSA, their pathogenesis and outcomes with the treatment of OSA.

2. Epidemiology

OSA is more common in men than women. Almost 13% of men and 6 % of women aged 30-70 years suffer from moderate to severe grades of OSA.[8] Other researchers reported high prevalence of symptomatic OSA ranging from 22% to 24% in men and 9% to 17% in women.[9,10] It has been reported that 82% of men and 93% women having moderate to severe obstructive sleep apnea remain undiagnosed.[11] The serious outcomes of OSA are cardiovascular and neurological disorders like heart attack and stroke. OSA is found in 60%-70% of patients having stroke or ischemic heart disease.[12] It was reported that several ophthalmic diseases are associated with OSA: floppy eye syndrome, primary open angle glaucoma, optic neuropathy, keratoconus, papilledema etc.[13] Glaucoma is the leading cause of blindness worldwide. Prevalence of OSA in patients of primary open glaucoma is about 2% as in the general populations.[14] Mojon DS reported prevalence of glaucoma in OSA approximately 4 times higher than the expected population rate.[15] However, Hirunwiwtkul P et al. 2010 reported higher prevalence of POG (20%) with OSA.[14]

3. Clinical Course of OSA

Patients of OSA usually present with excessive daytime sleepiness, persisting fatigue, irritability, nocturia, memory loss, morning headaches.[16,17] The symptoms of OSA are missed most often. It is the patient's attendants who complaint of snoring, choking, snorting or the respiratory cessation while sleeping. Early diagnosis of OSA is quite important as if left undiagnosed it might result in decreased quality of life, hampered productivity, increased chances of RTA related injuries and further increasing mortality.[18–20]

4. Risk Factors

OSA is a multifactorial disorder. Risk factors include anatomical as well as non-anatomical. A certain amount of anatomical impairment of upper airways is essential for OSA to occur.[21,22] This is the reason, why most treatment modalities target anatomical abnormalities. Certain lumen shapes of pharynx, increased length and narrow airways at single or multiple sites have been identified in patients of OSA, which can cause pharyngeal collapse while sleeping.[23–26] The structures acting as contributory factors for crowding of airways and further collapse are genioglossus, soft palate, lateral wall of pharynx and epiglottis which all are dilatory muscles.

Among the non-anatomical risk factors, obesity is very important. So, neck circumference is measured on routine basis in the clinics and is being used as a predictor for risk assessment of OSA. Increased obese population has lead to increased prevalence of OSA also. Almost 50-60% patients with obesity and metabolic syndrome also suffer from OSA,[27,28] which might act as a confounding factor in increasing the cardiovascular burden, also seen in patients of metabolic syndrome & diabetes.[29] Other risk factors include male gender, alcohol [causing upper respiratory tract muscle depression], short neck, physiological variations or anatomical defects e.g. adenoid hypertrophy, retrognathia, macroglossia, reduced upper respiratory size, [either due to obesity or other pathologies associated with excessive fat deposition in soft tissue of pharynx], cardiovascular risk factors including insulin resistance, lipid dysregulations and essential hypertension.[30] Previous studies have demonstrated that OSA is associated with various cardio-vascular events like myocardial infarction, arrhythmias, heart failure & ischemic stroke.[31–33]

5. Metabolic Alterations in OSA

OSA has been found to be associated with several metabolic derangements. Most importantly it has been found to be associated independently with insulin resistance, showing that it may be have an important role in development of diabetes mellitus type 2 and also metabolic syndrome (MS). MS is a group of metabolic derangements predicting risk of developing type II diabetic mellitus and atherosclerotic cardiovascular disease. MS includes obesity, insulin resistance, dyslipidemia and hypertension. Syndrome Z was the term that was given to the association of OSA & MS earlier. It has also been said that OSA by itself is a "metabolic disorder" and is one of the component of MS.[34] A case control study has reported that patients of OSA with AHI>15 were associated with increased prevalence of MS.[35] An another study demonstrated that OSA patients were six times more at risk to develop MS than those without OSA with BMI, smoking and age adjusted. But this study showed that no independent association is present with insulin resistance and OSA.[36] Various cross-sectional studies have suggested link of presence and severity of OSA and presence of glucose intolerance, insulin resistance & diabetes.[35,37–46] There are other studies based on large population, which have shown the association of severity of OSA determined by AHI and the magnitude of glucose intolerance and that of insulin resistance.[47–52] Several studies have stated that OSA has a propensity to increase risk of cardiac disease through above stated cardio-metabolic de-arrangements.[31,53] It has associations with the diseases like ischemic stroke, arrhythmias, and heart failure. So, OSA is an entity that may affect the underlying cardiac disease's prognosis.

6. Pathophysiology of OSA

The repetitive episodes of apnoea –hypopnoea seen in OSA cause hypercapnia & hypoxia. This hypoxia in return causes systemic and pulmonary vasoconstriction due to activation of sympathetic nervous system. This repetitive episodic intermittent hypoxia & the re-oxygenation simulates the ischemic-reperfusion theory and may lead to free radical generation, increased levels of homocysteine and cysteine. Various studies have confirmed association of OSA with increased oxidative stress markers.[54–58] Sleep deprivation via neuro-humoral pathways also activate systemic inflammation.[59–61] Inflammation induced vascular endothelial injury [VEC] damages coronary arteries leading to cardiac ischemia.[33,62] There is release of various proteins in VEC injury which act as precursors of cardio-vascular disorders. These can be used as biomarkers of cardio-vascular disease even in patients of OSA without symptoms e.g. protein YKL-40, MMR-9.[5,63] Also hypoxia in the adipose tissues induces lipolysis, mitochondrial damage, decreased adiponectin levels and increased leptin levels which can cause cardio-metabolic dysfunction.

7. Systemic Disorders Associated with OSA

Common systemic associations with OSA are cardiovascular disorders, stroke & transient ischemic attacks, depression, daytime sleepiness, fatigue, irritability, [they are symptoms of OSA not systemic disorders] insulin resistance and thrombosis etc.[11,64] Hanis M et al reported that 21-41% patients of OSA had depression. The systemic hypertension, pulmonary hypertension, nocturnal cardiac dysrhythmias, ventricular failure and myocardial infarction are common cardiovascular disorders associated with OSA.[65,66] It has been reported that about 40% of diagnosed patients of OSA had systemic hypertension. The apneic episodes of OSA may lead to sympathetic overstimulation, oxidative stress, endothelial damage and platelet activation; all contribute to cardiovascular disorders.[67,68]

8. Ophthalmic Associations

OSA involves various systems of body and eye is not an exception. The most common eye events that have shown association with OSA are floppy eyelid syndrome, glaucoma, non-arteritic anterior ischemic optic neuropathy, central serous retinopathy, retinal vein occlusions, papilloedema, diabetic retinopathy, papillary conjunctivitis, recurrent corneal erosions, keratitis, corneal punctate epitheliopathy, dry eye syndrome and keratoconus. Since OSA usually remains undiagnosed for a long time and some of the ophthalmological associations have irreversible visual outcome too. So, it becomes important for regular ophthalmic consultation, after the patient is diagnosed for OSA. Main underlying mechanisms remain the same i.e. hypoxia and mechanical injuries. Pathogenesis of individual entities has been described below.

8.1. Floppy eyelid syndrome

It is a condition where eyelids become everted easily with slight upward traction or spontaneously and become elastic and flaccid. In literature, one study suggested that 96% of the patients who had floppy eyelid syndrome [FES] were also associated with OSA[69] and the prevalence of OSA patients with FES varies from 2-25.8%.[69,70]

FES is usually seen in middle aged, male gender and overweight subjects. Because of the eversion of the upper eyelid, cornea gets exposed during sleep which contributes to ocular surface disorders e.g. dry eye, corneal ulceration, scarring, exposure keratopathy, neovascularisation and infection which might latter cause corneal perforations as well.[71–73] As the eyelid is everted and the conjunctiva is irritated by being rubbed against the pillow while sleeping continuously, it may further cause chronic papillary conjunctivitis. When eyelids undergo constant mechanical trauma, it might lead to lid oedema, meibomitis, ptosis, dermatochalasis, ectropion and trichiasis. Also repeated trauma to the exposed cornea may also result in keratoconus.[72,73]

Histological findings also indicate common mechanisms of FES & OSA. OSA is associated with pharyngeal collapse, which in turn is due to connective tissue compromise against the increased thickness of neck.

Two theories have been proposed for FES development in OSA; mechanical stress & transient tissue ischemia. FES histologically has increase in elastolytic metalloproteinase enzymes, hence leading to decreased elastin fibres of the tissue, which is associated with chronic inflammation of the lid. Also patients of FES experience symptoms often on the side they usually sleep on, correlating with the mechanical stress theory of FES. The events of hypoxia, transient tissue ischemia occurring secondarily in apneic episodes have been found to cause vessel damage and chronic lid inflammation.[74]

Acar et al. in a study showed low schirmer-I & tear break up time readings and increased corneal staining in patients with OSA and the changes in the values corroborated with the severity of OSA.[75] Acar et al. also demonstrated that all the above mentioned impaired findings showed significant improvement in moderate & severe cases of OSA after around 18 months of treatment with CPAP.[76]

As FES & ocular surface disease can impact patient's vision and quality of life, the treatment specific to FES which targets the associated ocular surface disease should be advised and patient is advised to not sleep on their side or face. Lubricants and artificial tears are advised to ensure ocular surface health. Surgical intervention involves lid tightening surgery to shorten upper eyelid. So, it is very crucial for the physician to ask the patients with OSA about these symptoms at every routine visit.

8.2. Glaucoma

Glaucoma is described to be a chronic progressive optic neuropathy associated with visual field defects & cupping of the optic nerve. It is currently 2^{nd} most common cause of blindness worldwide & has shown positive association with OSA.[77,78] Studies have shown association of OSA with both normal tension glaucoma (NTG) and open angle glaucoma (OAG).[30,69,79] A meta-analysis study comprising 3 case control studies found OR of 2.46 for association between OSA & glaucoma, while same meta-analysis in cohort study demonstrated OR of 1.43.[80] Various such case reports have shown such association. One of these reports showed that a 60-year-old patient of NTG who had progressive worsening of visual field in spite of controlled intra-ocular pressure with 2 anti-glaucoma drops and surgical intervention; after patient was diagnosed with OSA, and the treatment with nasal CPAP started, the patient visual field defects remained stabilised for 3.5 years of follow up.[81]

2 theories have been proposed for mechanism of glaucomatous changes of optic nerve in OSA i.e. mechanical and vascular theories. The mechanical theory says that the raised intraocular pressure [IOP] in OSA causes glaucomatous damage. Mechanical factors include being supine position, raised IOP related to obesity, raised nocturnal intracranial pressure, trabecular meshwork and lamina cribosa fibre depletion. Vascular theory includes predisposition of optic nerve to ischemia, repeated hypoxic episodes causing its damage, oxidative stress & inflammation.[80–82] Due to the decreased perfusion and potential for ischaemic changes, optic nerve head in OSA becomes more sensitised to mechanical damage further[80] However, there are many confounding factors in OSA and glaucoma, which are associated with poor perfusion to optic nerve head such as obesity, hypertension and diabetes.

Various studies have shown thinning of retinal nerve fibre layer [RNFL] which is a feature of early glaucomatous optic neuropathy, related with OSA.[83,84] Shiba et al. demonstrated that nasal RNFL & AHI have a negative co-relation.[85] Visual loss and severity of OSA has also been shown to be co-related in various studies.[83,86,87] Ulusoy et al in cross-sectional study showed that CPAP treated patients of OSA showed decreased IOP as compared to those who had untreated with OSA. Also untreated patients of OSA showed higher cup disc ratios.[88]

8.2.1. Non-Arteritic Anterior Ischemic Syndrome (NA-AION)

NA-AION is described as sudden loss of vision, usually unilateral, painless and occurs mostly upon waking.[89] Eye examination reveals characteristic RAPD (if unilateral) and fundus abnormalities.[90,91] If bilateral NA-AION has developed, there will be absent RAPD, but both pupils show sluggish reactivity to light.

Stein et al. in his cohort study found 16% increased risk of developing NA-AION in patients who had untreated OSA as compared to those without OSA.[92] Other studies have demonstrated the prevalence of OSA in patients with NA-AION to be 71-89%. A meta-analysis study reported that NA-AION patients are at five times more risk to OSA than the general population.[89,91,93]

Predisposing factors to NA-AION include obesity, hypertension, atherosclerosis and diabetes. It also involves anatomical risk factors e.g. small optic cup & small optic nerve head.[94] So, patients taking multiple antihypertensive drugs are especially at risk for developing this ischemic disease. As the vision loss is irreversible, patient has only single normal eye i.e. contralateral eye. There is 15% risk of involvement of other eye over a period of 5 years. So, this demands urgent attention to alleviate the risk factors to avoid involvement of the contralateral eye in known cases of NA-AION with OSA.[91]

Pathogenesis of NA-AION is proposed to be vascular. It is thought to be developed due to ischemia of the short posterior ciliary artery supplying the optic nerve. Ischemia causes oedema at the site of nerve exit from eye causing further compression and hence vascular compromise.

Archer et al. found that the above mentioned 16% increased risk of developing NA-AION in patients with OSA was not present in those treated by CPAP, which further establish the relation between the two entities.[89]

8.3. Central serous chorio-retinopathy (CSCR)

It is the serous detachment of the neuro-sensory retina in the macular region typically seen in middle aged males. It presents as mild decrease in visual acuity, decreased contrast sensitivity, visual distortion and affects color vision.

Huon LK et al. in a meta-analysis found significant association between OSA & CSCR.[79] Brodie et al. showed no significant association between the two.[95]

Both OSA & CSCR share common mechanism i.e. elevated serum cortisol levels and sympathetic tone causing vasospasm and endothelial cell dysregulation. This dysregulation in CSCR increases vascular permeability of choroidal vessels, hence fluid leaks from retinal capillaries causing oedema by building osmotic pressure gradient and pulling of fluid in choroid from RPE.[96] CSCR is self resolving usually with most of the patients resuming their pre-disease visual acuity within 3 months.[97]

There are lacunae of study to ascertain the association of CSCR &OSA, so further studies are required.

8.4. Retinal vascular occlusion (RVO)

RVO is the 2^{nd} most common cause of vascular blindness just next to diabetic retinopathy.[79,98] Vascular occlusion involves central retinal vein and branch of the retinal vein, latter being more common. Patients of RVO present with

sudden painless, unilateral vision loss or new visual field defect.

Various studies have shown association between OSA & RVO. Glacet –bernard A et al. and Chou et al. found OSA to be associated with 77% cases of RVO based on nocturnal symptoms [98] Mechanism of RVO with OSA is hypothesised to be hypercoagulability and retinal vessel inflammation due to raised nocturnal intracranial pressure. [99] Also, hypoxia in the apnoeic event causes increased haematocrit values and hence is a predisposing factor in clot formation. [100]

A study demonstrated that regular use of CPAP in OSA patients caused decreased hypercoagulability and hence decreased risk to develop stroke and BRVO.

8.5. Papilledema

It is the swelling of the disc bilaterally involving raised ICP. Various studies have shown OSA and idiopathic intracranial hypertension [IIH] to be associated with each other.

Proposed mechanism is that during apnoeic episodes, the oxygen and carbon dioxide levels in blood decrease and increase respectively. It causes cerebral vasodilation and hence increase in blood volume intracranially. This in turn leads to venous stasis and raised ICP as seen in IIH. [101]

Marcus et al. showed that sleep disordered breathing was very common in cases with IIH but all the patients also had obesity. [102] Another study found that with treatment of OSA, IIH improved symptomatically. [103] Javaheri S et al also showed improvement of papillodema symptoms with OSA treatment. There are other studies available which dismiss their association. [104] One such study was conducted by Thurtell MJ et al found no significant association of severity and prevalence of OSA as compared to the controls having similar body mass index and other demographic variables. [105] Because of the variable results of different studies about association between the two entities, more studies are needed.

8.6. Keratoconus

It is the non- inflammatory ectatic condition of cornea which is characterized by progressive thinning and corneal steepening or protrusion. [106] The prevalence of OSA in keratoconus patients was found to be 18%. [107] Various studies have found that 18-20% of keratoconus patients having obesity are associated with OSA while in general population the prevalence of keratoconus being only 0.054%. [108,109] However, if obesity is removed as a risk factor, non- obese patients of keratoconus were not found to be associated with OSA. [110] Contrary reports have been shown in another study which demonstrated no association between keratoconus & obesity and insignificant chances of occurrence of OSA in keratoconus patients. [111]

9. Management of Obstructive Sleep Apnea

Ocular complications may improve after appropriate management of OSA. McNab AA et al reported recurrence of surgically treated floppy eye syndrome in patients with untreated OSA. [69] Management of OSA includes general measures like weight control, avoidance of alcohol, smoking and sedatives. The nasal continuous positive airway pressure (CPAP) is an effective measure to prevent the collapsibility of the upper airway. [111] A comprehensive lifestyle intervention (CIL) program may significantly improve in obesity and OSA. CIL includes low calorie diet, increase physical activity/ exercise and behavioral modification. [112]

10. Conclusion

As it has already been discussed, hypoxia is the main culprit in the pathogenesis of OSA and it is a well established fact and proven by multiple studies that OSA increases risk of cardiovascular disorders like hypertension, atrial fibrillation, myocardial infarction, sudden death, arrhythmias, ischemic heart diseases. Above discussion indicates that OSA and ophthalmic involvement have close association. Also, several ophthalmic diseases have also shown improvement with the treatment of OSA i.e, CPAP. Almost all the ophthalmic entities described above are chronic in their course and may involve visual worsening or even blinding outcomes like visual field defects in glaucoma, vascular occlusions and NA-AION. So, it becomes even more necessary to diagnose these diseases at right time and proper follow up at regular intervals. It is highly essential that physicians treating OSA patients should consider ophthalmology consultation at time of diagnosis only as OSA itself is underdiagnosed disease. And by the time it is diagnosed it might have affected the eye as well.

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12. Conflict of Interest

The authors declare that there is no conflict of interest.

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