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RESEARCH ARTICLE

AN UPDATE ON NOVEL ASSOCIATION BETWEEN OBSTRUCTIVE SLEEP APNOEA AND PERIODONTITIS

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ABSTRACT

Obstructive sleep apnoea syndrome (OSAS) is a chronic multifactorial sleep-related breathing disorder characterized by a partial or total obstruction of the upper airways resulting in a reduction of airflow during sleep. Undiagnosed OSA is closely associated with consequential health problems, including diabetes and cardiac related disorders. Periodontitis is characterized by local bacterial infection within tooth supporting structures which is previously unrecognized but vastly prevalent and clinically relevant disease associated with OSA. It also adversely impacts cardiovascular health by increasing the production of cytokines, promoting atherosclerosis, which is also highly associated with OSA. Further well-structured studies are needed to explore the role of these biomarkers on the progression of Obstructive Sleep Apnoea both from the research and clinical perspective.

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INTRODUCTION

Obstructive sleep apnoea syndrome (OSAS) is a chronicmultifactorial sleep-related breathing disorder characterized by a partial or total obstruction of the upper airways resulting in a reduction of airflow during sleep. This leads to lower oxygen saturation and sleep disruption. Excessive daytime sleepiness, lack of concentration and snoring are common symptoms of OSAS.[1]The prevalence based on Wisconsin cohort study involving middle-aged adults 30–60 years of age, is 2% in females and 4% in males (Young, 1993). According to a survey conducted on a semiurban Indian population (2017),[2] it was found that around 6.2% among total sample were diagnosed with high-risk OSAS and 33.5% of the obese population were at high risk of OSA. OSAS has been associated with an increased risk for the development of cardiovascular disorders such as hypertension, heart failure, coronary heart disease, atherosclerosis and stroke.[3]Studies have suggested that OSAS could have a role in the activation of various inflammatory processes through hypoxia and oxidative stress-induced reperfusion injury.[4]Periodontitis is a chronic disease resulting from the interactions of the host's defence mechanisms with the pathogenic microorganisms characterized by the destruction of teeth supporting tissue that is clinically evident as pocket formation, the progressive loss of periodontal attachment, the loss of alveolar bone, and eventually tooth loss.[5] Risk factors for periodontal disease are age, poor oral hygiene, smoking, obesity, genetic pattern, systemic conditions like diabetes, arthritis, obesity, cardiovasculardisease, osteoporosis and low birth weight deliveries.As Periodontitis also induces systemic inflammation and oxidative stress,Obstructive sleep apnoeais another one of these conditions associatedwith an inflammatory response. Mouth breathing and intermittent decrease in oxygenation of tissues during sleep together with deteriorated daily life comfort may create a predisposition to periodontal disease in OSAS.[6] Charles Tremblay et al. [7] in 2016 stated a probable link between periodontitis and OSA syndrome via plasma and salivary inflammatory markers, and once again emphasized the need for more studies to clarify the exact relationship between these two conditions.

Relationship between sleep apnoea and pro-inflammatory cytokines: Evidence suggests that both periodontitis and OSAS are associated with systemic inflammation and cardiovascular disease.

Alarmingly prevalence of periodontitis in patients with OSAS was four-fold higher than the national average. Seo et al.[8] reported higher prevalence of OSAS in patients with periodontitis, supporting the hypothesis that OSAS and periodontitis are associated. Interleukin (IL)-1β and IL-33 are associatedwith both acute and chronic inflammation. Increased levels of IL-1β were detected in gingivaltissue, saliva and gingival crevicular fluid (GCF) of patients with periodontitis. The prominent biologic effect of IL-33 is the initiation of T-helper 2 cytokines,16 and it also possesses anti-inflammatory activity.[9] Significant upregulation of levels of Pro-inflammatory cytokines like TNF-α, IL-1β and inIL-6have been seen in serum patients with obstructive sleep apnoea.[10,11]Nizam et al,[12]discovered that there is a lower concentration of the serum levels of proMMP-9 in the severe OSAS group compared to the control group, but there is no statistically significant difference between the mild-moderate OSAS group and the control group. The authors concluded that there is no pathophysiological link between the severity of OSAS and the periodontal clinical status mediated by the products of neutrophils and MMPs, The recent meta-analysis (MA) showed that there is an increase in levels of inflammatory markers in subjects with OSA including CRP, TNF α, IL-6, IL-8, ICAM, VCAM and Selectins. This effect is positively influenced by severity ofOSA.[13]

Recent evidences highlighting the relationship between Obstructive Sleep Apnoea and Periodontitis: Gunaratnam et al. (2009)[14]was the pioneer to establish an inflammatory link between periodontitis and OSA. They noted a high incidence of periodontal disease insubjects with OSA (77% to 79%) but could not assert a causallink due to a significant absence of correlation of the measures. Nevertheless, they suggested that periodontitis may be oneof the co-factors involved in the association between apnoeaand cardiovascular disease, or that a pre-existing OSA couldworsen the presence and severity of periodontal disease. The results of preliminary study by Seo WH et al stated that 17.5% of the participants had periodontitis, 46.6% had OSA and 60.0% who were diagnosed with periodontitis had OSA. Authors identified old age, male gender, current smoking status and mouth breathing during sleep as risk factors for periodontitis.[8]In a recent study by Nizam et al, he concluded that biomarker salivary IL-6 is significantly related to the severity of OSAS. This study emphasized that there is a marked change in the presence of particular oral and periodontal microorganisms in the subgingival plaque; suggesting that OSAS has a connection with the development ofperiodontal inflammation.[15] Latorre et al conducted a study to identify the association between periodontal disease and OSA in adults with systemic comorbidities. One hundred and ninety-nine individuals (107 women and 92 men) underwent polysomnography with a mean age of 49.9 years were recruited. They described a significant link between periodontitis and mild OSA and this association was more pronounced in women with hypertensive cardiomyopathy. Furthermore, periodontitis was associated with severe OSA in men who showed any oftwo comorbidities such as hypertension or hypertensive cardiomyopathy.[16] Gamsiz-Isk et al. [17]compared periodontitis prevalence between controls and patients with OSA by assessing clinical periodontal parameters and gingival crevicular fluid (GCF) levels of interleukin (IL-1β), tumor necrosis factor (TNF-α), and high- sensitive C-reactive protein (hs - CRP); serum hs - CRP was also sampled. He observed that PI, GI, PD, CAL, BoP, PD ≥4 mm isremarkablyhigher in the OSAS group. Periodontitis prevalence is 96.4% in the OSAS group compared to 75% in the non-OSAS group. They concluded higher prevalence ofperiodontitis and higher levels ofGCF IL - 1β and serum hs - CRP in patients with OSA. In latest systematic review by Daneila Lembo (2021)[18] which assessed 5 case-control studies, and 5 cross-sectional with sample size comprising of sample size 50 to 29,284 subjects concluded that there is low evidence ofa possible association between OSAS and periodontitis. The pathophysiological mechanism, cause-effect, or dose-response relationship are still unclear. Further studies are needed and should use a precise classification of OSAS subjects, while the new classification of periodontitis from the World Workshop ofChicago 2017 should be used for the periodontal assessment. Nazanin Khodadadi in his contemporaneous meta-analysis (2022), tried to find out whether periodontitis is associated with OSA severity as there are conflicting results on the association for the same. According to the results, periodontitis has a direct association with OSA. Also, periodontitis has been shown to be associated with mild-to-moderate OSA, but not with severe OSA. Further studies are warranted to elucidate the mechanisms of these associations.[19]

Future Scope of Research and Current Treatment Modalities: Carra et al.[20]recently published a study stating treatment with a facial mask with/without a humidification device could be a novel therapy for OSA. Even if the link between mouth breathing associated with OSA and periodontitisis still debated to this day, this device opens severalavenues of research. The limitation of the study was that they failed to observe a difference in masticatory function between individuals using these devicesand those who did not use them. In light of recent studies, we could integratenew OSA front-line detection techniques into clinicalevaluation. According to Ahmad et al,[21] along with promotion of oral hygiene he suggested that the STOP questionnaire (Appendix 1, supplementalmaterial) be included in the dental examination. As arapid and effective method, this self-reported questionnaireidentifies patients at risk for sleep apnoea without, however,providing a categorical diagnosis. A combined alliance and possible exchange of results between the dentist and thefamily physician would then be necessary in order to carryout a more objective examination such as polysomnographyor polygraphy.

CONCLUSION

There is low evidence of a possible association indicating higher levels of markers of systemic inflammation in patients with Periodontitis associated with Obstructive Sleep Apnoea. The pathophysiological mechanism, cause-effect, or dose-response relationship are still unclear. The many limiting factors and the difficulty in recruiting high numbers of participants preclude scientist and clinicians from drawing any conclusions. Further well-structured studies are needed to explore the role of these biomarkers on the progression of Obstructive Sleep Apnoea both from the research and clinical perspective.

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