EDITORIAL

Putting Some Teeth into It: Connecting Periodontitis with Sleep Apnea

Commentary on Sanders et al. Periodontitis and sleep disordered breathing in the Hispanic Community Health Study/Study of Latinos. SLEEP 2015;38:1195–1203.

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Researchers have noted for decades that there is a higher prevalence of periodontal disease among those with cardiovascular disease (CVD). The disorders share many common risk factors: obesity, age, smoking, male sex, diabetes, hypertension and low socioeconomic status (SES). Thus epidemiologically, this association was difficult to differentiate from confounding factors.[1] More recently studies have demonstrated a biological plausibility for the association, in the form of inflammation. The oral microbiome has been identified within atherosclerotic plaques with gingival organisms isolated on atheroma culture.[2] With the transient bacteria, innate immunity activation, and inflammatory cascade-triggered bacterial antigens, periodontitis promotes atherosclerosis.[3] The metabolic syndrome with its hyperglycemia and insulin-resistance state is also highly associated with periodontitis. This association has been hypothesized to be driven by oxidative stress and inflammation characteristic of both disorders.[4] As CVD and the metabolic syndrome, and their associated risk factors (namely obesity, male sex), have an established strong relationship with obstructive sleep apnea (OSA), a connection of OSA with periodontitis must be considered.

Chronic periodontitis is a highly prevalent illness within the US (46% of the adult population, 9% is severe).[5] Hispanics have the highest prevalence of this disease (63% of adults ≥ 30 years) compared to other ethnic/racial groups.[5] Periodontitis is characterized by local infection with oral pathogens within tooth supporting structures leading to destruction of periodontal connective tissue, ligaments and alveolar bone. Without treatment, periodontitis ultimately leads to tooth loss. Dental pathogens create a biofilm within plaques producing local inflammation and leading to elevation of systemic inflammatory biomarkers such as C-reactive protein (CRP). Periodontitis manifestation depends on both the bacterial pathogen (biofilms, transient bacteremia) and the host response (inflammation).[6] The host response may be modulated by smoking, stress and chronic inflammatory illnesses.

In this month's issue of *SLEEP*, Sanders and colleagues, identify a novel association of chronic periodontitis with obstructive sleep apnea within the Hispanic population.[7] The association of severe periodontitis with OSA was strongest in those < 55 years of age, and was independent of established periodontitis and OSA risk factors (i.e., obesity, smoking, diabetes, male sex, and tooth loss). An AHI > 0 had an increased odds of periodontitis, with 11× greater prevalence in those with AHI > 15 compared to zero.[7] Although others have investigated this relationship,[8–11] no previous study had such a large cohort (N = 12,469) with high quality objective sleep and periodontal data. All eligible subjects underwent NHANES standard, calibrated periodontal exams as well as home sleep studies, therefore not relying on administrative coding and potential misclassification. The study was epidemiologically rigorous, recruiting by random sampling to reflect the population, and not biased by clinic referral base, surveillance bias, or health care system factors. Thus, with the addition of this study to the literature, the finding of an association of OSA and chronic periodontitis in younger adults appears robust.

The underlying mechanism explaining this association is not clear, nor is the direction of the association. Severe periodontitis may reflect greater inflammatory burden and resulting exuberant host response to bacterial plaques among those with OSA. As with other cardiovascular and metabolic diseases, both OSA and periodontitis pathophysiology involves oxidative stress, chronic inflammation and an elevated C-reactive protein (CRP) levels. Sanders et al. did not find evidence that CRP mediated the relationship of OSA and periodontitis.[7] Efforts to improve CV outcomes by treating periodontitis have not borne out, although given the limited evidence, no conclusions can be drawn.[12] It is possible that the shared inflammatory pathways, common risk factors, and disease clustering for both OSA and periodontitis explain the association, despite attempts to control for known confounders. However, the more pronounced association in younger adults, prior to atherosclerotic disease manifestations, makes this less likely.

Dry mouth (xerostomia) commonly experienced by most snorers and mouth breathers with OSA may explain the association with periodontitis. Xerostomia may exacerbate the bacterial overgrowth and plaque formation, as bacterial clearance is impaired. If so, treating OSA with nasal CPAP and/or improving nasal airflow may be expected to reduce periodontitis by resolving xerostomia. Similarly, CPAP therapy should resolve much of the hypoxemia and oxidative stress of OSA that may contribute to periodontitis pathology, reducing periodontitis severity. Future studies looking at dental outcomes after initiating CPAP therapy are warranted. Better dental hygiene and treating periodontitis seems unlikely to improve OSA. Investigations of periodontal therapy and cardiovascular and diabetes outcomes have been disappointing to date.[13,14] Yet missing teeth has been found to be predictive of cardiovascular events, with ≥ 9 lost teeth increasing risk of CV death.[15]

Submitted for publication June 2015
Accepted for publication June 2015
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The high prevalence of periodontitis among those with OSA has implications for OSA therapy as well. Dental disease and especially posterior tooth loss limit treatment options, preventing utilization of mandibular advancement devices (MAD). This may be a larger problem in lower SES groups, with more smoking, obesity, and reduced access to preventative dental care, and as a result more prevalent severe periodontitis and tooth loss.[16] Thus, due to oral health disparities, lower SES subjects with OSA may be poor candidates for MAD. The association of OSA with periodontitis is most pronounced in younger adults, those who often prefer MAD to CPAP. Improving the dental health and reducing tooth loss, may allow for greater therapeutic options for those with OSA.

In summary, periodontitis is a previously unrecognized but highly prevalent and clinically relevant disease associated with OSA. Periodontitis adversely impacts cardiovascular health, promoting atherosclerosis, which is also highly associated with OSA. The consequences of periodontitis, namely tooth loss, may limit OSA therapy choices. Sleep and dental health disparities may worsen the burden of CV disease among low SES groups. Future research is necessary to determine if treating OSA may improve periodontitis.

CITATION

Billings ME. Putting some teeth into it: connecting periodontitis with sleep apnea. *SLEEP* 2015;38(8):1153–1154.

DISCLOSURE STATEMENT

Dr. Billings has received research support from Philips-Respironics.

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