Periodontal Disease Contributes to Obstructive Sleep Apnea

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Abstract:

Periodontal disease (PD) is associated with obstructive sleep apnea (OSA) by a postulated bi-directional causal-effect relationship of PD and OSA through systemic inflammation. New evidence suggests that periodontal disease may contribute to obstructive sleep apnea through the aspiration of periodontal bacterial pathogens into the lungs, causing a decrease in lung function, which consequently contributes to obstructive sleep apnea. Additionally, xerostomia, which creates an oral environment that aggravates PD, also plays a role in increasing the surface tension of the Upper Airway Lining Liquid (UALL) and resultantly contributes to OSA.

Key words: periodontal disease, obstructive sleep apnea, lung function, COPD, periodontal therapy, xerostomia, Upper Airway Lining Liquid

Periodontal Disease

Periodontal disease (PD) is a chronic, complex, biofilm-based oral bacterial infection affecting the supporting structures of the teeth[1]. It is estimated that approximately 47.2% of the adult U.S. population is infected with PD[1]. The oral cavity is an optimal environment for bacterial growth due to the high humidity and warm temperature[2]. As a result, periodontal biofilm is rich with many harmful aerobic and anaerobic bacteria[1]. These gram negative and spirochete pathogens can enter the blood stream and cause bacteremia, resulting in an inflammatory response in the body[1]. Risk factors for PD include but are not limited to poor oral hygiene, age, xerostomia, stress, lack of sleep, bruxism, cortisol production, and smoking status[3]. PD has been linked to cardiovascular disease, diabetes, stroke, pre-term birth, COPD, cancer, renal failure, and most recently linked to obstructive sleep apnea (OSA)[1]. Conventional treatment of PD includes mechanical debridement and antibacterial therapy[4].

Obstructive Sleep Apnea

OSA is a breathing-related sleep disorder characterized by partial or complete collapse of the upper airway during sleep, causing a reduction (hypopnea) or cessation (apnea) of airflow[5]. OSA is diagnosed by a medical physician through a polysomnogram sleep study, using the apnea-hypopnea index (AHI), where the minimum requirement for the diagnosis of OSA is at least an average of five episodes per hour of cessation of airflow[6]. OSA affects approximately 24% of adults and 1-4% of children[7,8]. OSA causes sleep fragmentation, an increase in sympathetic activity, and a decrease in oxygen saturation in the blood[5]. Risk factors for OSA include, but are not limited to, age, obesity, and craniofacial abnormalities of a high arched palate leading to nasal obstruction and mouth breathing [9]. OSA has been associated with daytime sleepiness, behavioral disorders in children, hypertension, diabetes, cardiovascular disease, myocardial infarction, stroke, and death[9,10]. Routine treatment of OSA includes CPAP, mandibular advancement oral appliances, and surgery[5].

periodontal pathogens and inflammatory cytokines in their saliva compared to healthy individuals[14]. Aspiration of oral bacteria from PD directly into the lungs has been shown to cause respiratory inflammation, a decrease in expiratory volumes, and an overall decrease in lung function, subsequently leading to COPD[25-29].

Moreover, it is important to note that the treatment of PD has been shown to improve symptoms of COPD, where Zhou et al. (2014) conducted a two-year pilot study documenting that 60 patients with COPD had lowered exacerbations, increased forced expiratory volumes, and an overall increase in lung function after periodontal therapy[30].

Xerostomia Contributes to Periodontal Disease and Obstructive Sleep Apnea

Xerostomia is characterized by an individual's feeling of a dry mouth due to qualitative changes in saliva with or without an actual decrease in salivary volume[35,36]. Xerostomia effects upwards of 46% of the population[37]. It may be caused by the anti-cholinergic side-effect of medications, and or by an increase in sympathetic activity in response to bodily stress or pain[38,39]. Changes in salivary composition due to xerostomia has been shown to increase bacterial plaque colonization, which aggravates and contributes to PD[45,36,40-45]. Xerostomia also has a direct effect on OSA through the increased surface tension of the Upper Airway Lining Liquid (UALL), increasing upper airway obstruction.

Kirkness et al.(2005) found no significant difference in surface tension between fluid sampled from under the tongue and the posterior oropharyngeal wall[46]. This demonstrated that swallowing coats the upper airway with saliva, and that saliva is a primary constituent/ surfactant of the UALL. Key findings from Verma et al. (2006) showed that there is a strong inverse correlation between oral mucosal wetness and UALL surface tension[47]. Studies showed that reductions in salivary flow rates or changes in the quality of the saliva cause increased surface tension of the UALL, thereby contributing to upper airway obstruction[48,49-56]. Van de Touw et al. (1997) reported that patients with OSA had significantly higher surface tension of the UALL when compared to controls[51].

Many studies have documented that greater surface tension of the upper airway lining causes the airway to collapse[49,51,53]; yet the inclination to collapse is significantly reduced by the application of a surfactant[52,54,55]. Kirkness et al. (2005) concluded that patients with OSA showed a reduction in respiratory disturbance when a surfactant was administered[52]. Jokic et al. (1998) found that using a surfactant reduced the frequency of obstructive events by up to 42% in OSA patients[55].

Overall, the research shows that salivary surface tension is correlated to obstructions of the airway[48,49-56]. Therefore, xerostomia's change in the quality and composition of saliva, also increases the surface tension of the UALL, and thereby contributes to nocturnal upper airway obstructions.

Conclusions

As discussed above, the scientific literature documents the association of PD and OSA[1,12-14], where it has been postulated that the pathogenicity of PD and OSA includes the production of

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