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Advances in Periodontal Disease and its Effects on Carotid Intima-Media Thickness

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INTRODUCTION

Periodontal disease is a series of bacterial infections, such as gingivitis and periodontitis, that affect tooth-supporting tissues and is associated with exacerbations of several lung diseases. Lung diseases such as pneumonia, Chronic Obstructive Pulmonary Disease (COPD), asthma, tuberculosis, COVID-19, and bronchiectasis contribute to poor quality of life and significantly lower mortality. The relationship between periodontitis and pulmonary effects is an important issue and deserves further attention. Many indigenous bacteria coexist in the oral cavity and lungs. However, alterations in the normal microbiota due to oral disease, age, lifestyle, or dental procedures may contribute to altered aspiration of oral periodontal bacteria into the lungs and altered inflammatory responses. Similarly, periodontal disease is associated with a longitudinal decrease in vital capacity. Several studies have suggested a possible beneficial effect of periodontal therapy by reducing the frequency of exacerbations, reducing the risk of respiratory adverse events and morbidity, and improving pulmonary function. Periodontal diseases such as gingivitis and periodontitis are mainly caused by plaque. Several antiplaque and antimicrobial agents have been successfully incorporated into toothpastes and mouthwashes to control plaque biofilms and prevent and treat gingivitis and periodontitis. The purpose of this article is to review basic and clinical research, especially research published in the last 5 years, to demonstrate the anti-plaque, anti-gingivitis properties of some common compounds found in toothpastes and mouthwashes. Common active ingredients in toothpastes and mouthwashes included in this review are Chlorhexidine, Cetylpyridinium Chloride, Sodium Fluoride, Stannous Fluoride, Stannous Chloride, Zinc Oxide, Zinc Chloride, and the two herbs Licorice and Curcumin. We believe this comprehensive overview provides dentists and the general public with useful and up-to-date information about the leading oral care products on the market for daily use.

DESCRIPTION

Periodontitis and osteoporosis are common inflammatory skeletal diseases that pose significant public health problems in aging societies. Both periodontal disease and osteoporosis are bone diseases closely related to inflammation and aging. There is ongoing interest in whether systemic skeletal diseases such as osteoporosis increase alveolar bone loss in periodontitis. A review of the literature published over the past 25 years indicates that low whole-body Bone Mineral Density (BMD) is associated with alveolar bone loss, but recent evidence suggests clinical attachment loss. The other parameters of periodontitis have also been suggested. Inflammation and its effects on bone remodelling play an important role in the pathogenesis of osteoporosis and periodontitis and may serve as a central mechanistic link between these diseases. Increased cytokine production and an increased inflammatory response inhibit osteoclast bone formation and exacerbate osteoclast bone resorption, resulting in net bone loss. With aging, accumulation of oxidative stress and cellular senescence cause progression of osteoporosis and worsening of periodontitis.

CONCLUSION

Vitamin D deficiency and smoking are joint risk factors and may mediate the association between osteoporosis and periodontitis by increasing oxidative stress and impairing the host inflammatory response. Given the association with focal bone loss, regular dental examinations and intraoral radiographs serve as

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cost-effective screening tools for decreased whole body BMD and increased fracture risk. Conversely, patients whose fracture risk exceeds the surgical threshold develop severe periodontitis and are at increased risk of tooth loss. Various treatments for osteoporosis approved by the Food and Drug Administration have shown promising results in treating periodontitis. Understanding the molecular mechanisms underlying their connections will shed light on potential therapeutic strategies that may facilitate joint management of systemic and local bone loss.