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Why Dentists Should Treat Periodontal Disease in Patients with Respiratory Disease? The Impact of Oral Health on Fragile Patients

Raffele B[1]*, Luciano Z[2] and Sharock E[3]

[1]Hospital of Lovere, Bergamo, Italy

[2]Private practise in Milan, Italy

[3]Private practise in Pavia, Italy

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***Corresponding author:** Borgia Raffele, MD, PhD, Hospital of Lovere, Bergamo, Italy, Email: dorina.lauritano@unimib.it

Abstract

The correlation between periodontal disease (PD) and respiratory diseases (RDs) has been investigated for a long time. Some authors suggested that PD could affect the progression of RDs, and pathogens in the oral biofilm could be inhaled causing lower respiratory tract diseases.

There are many infectious agents responsible of RDs: bacteria, mycoplasma, fungi, viruses and parasites. We can classify two kinds of infectious agents: commensal agents and nosocomial agents. Commensal agents are resident on oropharynx mucosa. Nosocomial agents are usually bacteria that are not commensal, but come from the external environment. They both may cause RDs. Even if the lower respiratory airways are sterile, inhaled microorganism coming from oropharynx could contaminate them. Oropharynx bacteria may play an important role in pathogenesis of RDs with several mechanism of action.

Some epidemiologic studies and reviews have stressed the potential role of periodontal pathogens in development and progression of RDs. In fact poor oral hygiene may be a significant risk factor for RDs especially in high-risk group as hospitalized and institutionalized patients. A better oral health may help the controlling of RDs. It should be necessary a strict oral hygiene recall in risk group and institution of oral hygiene courses for caregivers in long-term care residences.

Keywords: Pulmonary Infection; Periodontitis; Oral Biofilm; Nosocomial Pneumonia; Oral Hygiene

Introduction

The correlation between periodontal disease (PD) and respiratory diseases (RDs) has been investigated for a long time. Scannapieco and other authors firstly suggested that PD could affect the progression of RDs and pathogens in the oral biofilm could be inhaled causing lower respiratory tract diseases [1-4]. RDs include serious respiratory diseases such as chronic obstructive pulmonary disease, characterized by chronic obstruction to the passage of air in the lung tree, with an excess production of expectorate, chronic bronchitis and emphysema. Respiratory diseases (RDs) are responsible of high morbidity and mortality all over the world. RDs are ubiquity present in world population and in the United States are the fourth cause of death [5].

Microbiota of RDs

There are many infectious agents responsible of RDs: bacteria, mycoplasma, fungi, viruses and parasites. These microorganisms are becoming more and more resistant to antibiotics, so RDs, refractory to systemic therapy, will assume a great importance in the next years. We can classify two kinds of infectious agents: commensal agents and nosocomial agents. Commensal agents are resident on oropharingeal mucosa (Streptococcus pneumonia and Haemophilus influenza, Chlamydia pneumonia, Candida albicans, Mycoplasma pneumonia, Legionella pneumophila). These bacteria can become virulent when the subject has impaired immunity conditions or is debilitated, and can cause various forms of pneumonia. Nosocomial agents are usually bacteria that are not commensal, but come from the external environment. These are Gram-negative bacilli (Escherichia coli, Klebsiella pneumonia, Serratia sps, Enterobacter sps, Pseudomonas aeruginosa and Staphylococcus aureus) [3].

Pathogenesis of RDs

Even if the lower respiratory airways are sterile, inhaled microorganism coming from oropharynx could contaminate them. The mucociliary transport and secretions from the bronchial tree, as well as the cough reflex, maintain the sterility of the lower airways. These mechanical actions are associated with immune-mediated activity of the epithelium of the respiratory tract secretions (humoral immunity, polymorph nuclear leukocytes and cell mediated immunity). These secretions include surfactant, immunoglobulins and complement. Oropharynx bacteria may play an important role in pathogenesis of RDs with several mechanism of action. The first step is inhalation of periodontal pathogens such as “red complex” bacteria in respiratory tract. Subsequently the salivary enzymes associated with PD may change antigenic epithelial surface receptors of respiratory tract mucosa, modifying the adhesion for respiratory pathogens. Then salivary enzymes may destroy bacteria pellicle favouring the adhesion and colonization of inhaled pathogenic bacteria. Finally periodontal cytokines may promote infection by respiratory pathogens.

Periodontal Disease (PD)

Periodontal disease (PD) affects about half of the adult population all over the world[6]. PD is caused by bacterial infection inducing an inflammatory response with progressive destruction of the periodontal tissues and finally the lost of teeth. Smoking, alcohol consumption, and systemic conditions such as diabetes, osteoporosis, malnutrition and stress are considered additional risk factors both for PD and RDs. The oral pathoges may cause burning sensations of mouth such as burning mouth syndrome also [5-7].

PD therapy is important to reduce local inflammation and bacteraemia. Recently has been stated that PD appear to increase the risk of cardiovascular disease, pulmonary disease, and preterm and low birth weight [8].

In fact the oral microbiota is constituted by a large number of bacteria species that form a biofilm. This biofilm includes both saprophytes and potentially pathogenic species. PD may be associated to other oral diseases such as oral lichen planus, atrophy of the oro-pharyngeal mucosa and oral lesions of gastroesophageal reflux disease [9-11].

The oral cavity could represent a reservoir of pathogenic bacteria that are infecting the pulmonary tree mucosa. A pathogen is expected to exceed immune defence mechanisms to reach the lower respiratory airways. The immune defence mechanisms are so efficient, that the lower respiratory airways are sterile, despite that in the upper airway bacterial load is huge [12]. The pulmonary tract infection by oral biofilm pathogenic bacteria can occur when the immune system defence is decreased and the pathogens are particularly virulent. The pathogens can enter through inhalation, but the most accepted hypothesis is that infection of the lung mucous is caused by aspiration of oropharyngeal secretions. It is therefore plausible that oral microorganisms may infect pulmonary tract. Infections of the lower respiratory tract can start by the colonization of pathogenic bacteria of the oral and oropharyngeal mucosa [13]. Pathogens are successively mixed in the oral secretions in addition to oral bacteria, to hydrolytic enzymes and proinflammatory cytokines. The content of these secretions may contaminate and cause modifications on epithelial surface of the lower respiratory airways mucosa. Since periodontal disease is characterized by chronic inflammation, inflammatory mediators, released at the level of saliva, can reach the respiratory epithelium. So it is likely that periodontal disease may contribute to the development and progression of RDs. This idea is supported by a study of Terpening, et al. stating that incidence of RDs by oropharyngeal colonization is more frequent in dentate patients and patients wearing denture, than in totally edentulous patients not wearing dentures [14]. In addition another study of Woods et al reported that

periodontal enzymes may cause the loss of fibronectine from the epithelial cell surface, discovering mucosal surface receptors for respiratory pathogen adhesins, and favouring a better adhesion of periodontal pathogens to respiratory mucosa [15].

Oral pathogens may stimulate the oral mucosal cells to release cytokines such as interleukin (IL)-1α, IL-1β, IL-6, IL-8, and TNF-α. Inflammatory cells may be recruited to the lower respiratory airways by oral tissues cytokines. These inflammatory cells damage epithelium making it more susceptible to colonization by respiratory pathogens [16].

Oral Health and Fragile Patients

Poor oral health may be related with nosocomial RDs. It was not established a direct correlation between PD and RDs, however may be that oral colonization by periodontal agents may favour development of pneumonia. The specific pathogenetic mechanism of action is unknown in detail. In addition it is difficult to establish if patients resident in nursing home, present more risk to develop pneumonia due to their poor oral hygiene. Another important problem that must be investigated is if pneumonia or others RDs may be more common in institutionalised patients or vegetative patients. However a recent paper by Barros et al. studying the consequences of edentulism, PD and systemic biomarkers of inflammation on the upset of RDs among subjects with chronic obstructive pulmonary disease, concluded that the risk for RDs-related events may be attributable to both edentulism and elevated serum IL-6 levels [17].

PD Treatment and RD Exacerbations

The mouth is a reservoir for pathogen agents: bacteria, mycoplasma, fungi, viruses and parasites. In debilitated patients, institutionalised or not, oral bacteria may colonize the respiratory airways. In addition the reduction of salivary flow, frequent event in these patients, may increase bacterial loading in the mouth and respiratory tract. Infact analysing the sputum of patients with RD were found increased antibody levels against “red complex” bacteria [18].

Kucukcoskun, et al. found that PD treatment significantly reduced the frequency of RDs exacerbations [19]. It is important to raise awareness among dentists on maintaining oral health in patients with RDs in order to reduce fatal and non-fatal RDs exacerbations.

Conclusion

However, even if the possibility of a direct transmission of pathogens from the mouth of the respiratory tract is very impressive, no studies have demonstrated this correlation [20]. Some authors in a recent review reported that there is wake evidence of a correlation between periodontal disease and RDs [21]. The correlation between PD and RDs is very plausible but remains a speculation. On the contrary some epidemiologic studies and reviews have stressed the potential role of periodontal pathogens in development and progression of RDs [22-24]. In fact poor oral hygiene may be a significant risk factor for RDs especially in high risk group as hospitalized and institutionalized patients. A better oral health may help the controlling of RDs. It should be necessary a strict oral hygiene recall in risk groups, and institution of oral hygiene courses for caregivers in long-term care residences.

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