Pulmonary Diseases and Periodontal Health- A Review

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Abstract

The oral cavity has a multitude of functions in relation to daily life. The poor health has the potential of affecting the quality of life. In recent years, efforts have been made to recognize oral health as an integral part of overall health. The anatomical continuity between the lungs and the oral cavity makes the latter a potential reservoir of respiratory pathogens. The micro-organisms may enter the lung by inhalation, but the most common route of infection is aspiration of what pneumologists have long referred to as oropharyngeal secretions. There is a fair evidence exists between respiratory disorders and poor oral hygiene & good evidence exist that improved oral health and professional oral health care reduce the progression or occurrence of respiratory diseases among high risk adults. Dental plaque can act as a reservoir for respiratory infections. Microorganisms can be released from the dental plaque

into salivary secretions, which are then aspirated into lower respiratory tract to cause respiratory diseases like pneumonia and chronic obstructive pulmonary disease. Periodontal diseases and respiratory diseases including bronchial asthma have an inflammatory nature thus mandates a positive correlation between these. This paper discusses the relation between the periodontitis, its influence on the incidence of respiratory disorders and underlines the importance of improving oral hygiene among patients who are at risk.

Key words: Chronic Periodontitis, COPD, Bronchial Asthma, antiaesthmatic medications.

Introduction

A chronic respiratory infection is a constant potential source of infection and has been considered as a separate risk factor for respiratory diseases. Evidences dictate a bi-directional relationship between periodontitis and respiratory diseases [1].

Periodontitis affects individuals of all ages but it is more common in elderly patients [2]. The anatomical continuity between the lungs and the oral cavity makes the latter a potential reservoir of respiratory pathogens. Yet an infective agent must defeat sophisticated immunological and mechanical defence mechanisms to reach the lower respiratory tract. The defence mechanisms are so efficient that, in healthy patients, the distal airway and lung parenchyma are sterile, despite the heavy bacterial load (106 aerobic bacteria and107 anaerobic bacteria per millilitre) found in the upper airway. An infection occurs when the host's defences are compromised, the pathogen is particularly virulent or the inoculum is overwhelming.[1]The micro-organisms may enter the lung by inhalation, but the most common route of infection is aspiration of what pneumologists have long referred to as oropharyngeal secretions. Therefore, it is possible that oral micro-organisms might infect the respiratory tract.[3]

Dental plaque can act as a reservoir for respiratory infections. Microorganisms can be released from the dental plaque into salivary secretions, which are then aspirated into lower respiratory tract to cause pneumonia. It is reported that 30 to 40% of all cases of aspiration pneumonia, necrotizing pneumonia or lung abscess show the presence of anaerobic bacteria. Organisms like P.gingivalis, Bacteroides gracilus, Bacteroides oralis, Eikenella corrodens, Fusobacterium nucleatum, Actinobacillus actinomycetemcomitans, Peptostreptococcus, clostridium and actinomyces have been cultured from infected lung fluids. Most of these organisms are associated with the pathogenesis of periodontal disease too. Oral bacteria may also play a role in the exacerbation of chronic obstructive pulmonary disease (COPD).[4] Therefore, there are high chances of periodontal disease lading to exacerbation of respiratory disease especially when the host defence is compromised.

2. History

The concept of focal infection has been known since antiquity. The term oral sepsis used by Hunter was replaced by the term focal infection in 1911 by Frank Billing, He defined Focus of infection as “circumscribed area of tissue infected with pathogenic organism “. Renewed attention has been focused on oral sepsis and its relationship with systemic factors. An old concept is being examined in new height.[1]

Respiratory diseases are responsible for a significant number of deaths and considerable sufferings in human respiratory tract infections. Respiratory tract infections such as Pneumonia and exacerbation of chronic obstructive pulmonary diseases involve the aspiration of bacteria from the oropharynx into the lower respiratory tract [5].

3. Chronic periodontitis

Chronic periodontitis is an inflammatory disease caused by the plaque microorganisms, resulting in progressive destruction of tissues that support the teeth [6]. Periodontitis is characterized by periods of exacerbation with periods of remission. In chronic periodontitis

there is increased local microbial burden that initiates local inflammation resulting in local tissue destruction.

4. Dental plaque – Reservoir of respiratory pathogens:

Lack of attention to oral hygiene can result in an increase in the mass and complexity of dental plaque, which may foster bacterial interactions between indigeneous plaque bacteria and respiratory pathogens such as P. aeruginosa and enteric bacilli [7]. These interactions may result in colonization of dental plaque by respiratory pathogens. Respiratory pathogens that establish in dental plaque may be difficult to eradicate. Respiratory pathogens also colonize in the oral cavity of patients with teeth or denturers than edentulous patients not wearing dentures [3].

5. Mechanism of action of oral bacteria in the pathogenesis of respiratory diseases

The mechanism of action could be aspiration of oral bacteria, colonization of dental plaque by respiratory pathogens causing Pneumonia into the lungs [8],[9].Flowchart follows

Alteration of the mucosal surface by salivary enzymes in periodontitis

⇩

Leading to an increase in adhesion

⇩

Colonization of respiratory pathogens

⇩

Destruction of salivary pellicles on pathogenic bacteria,
by periodontal disease associated enzymes

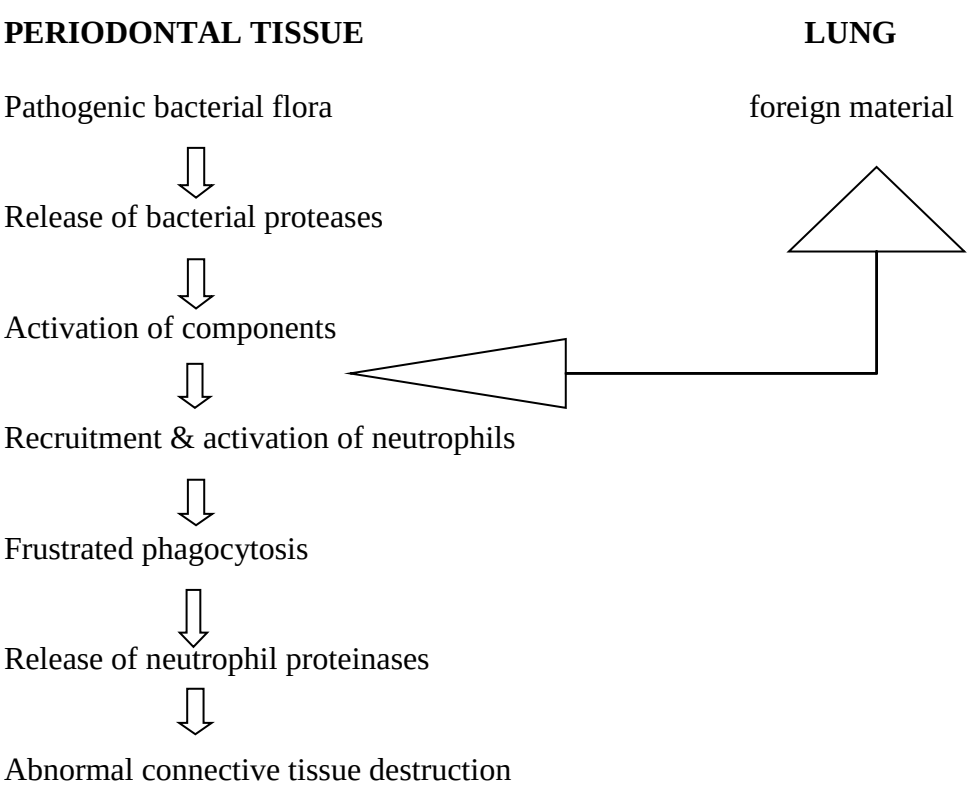
⇩

Facilitating infiltration of the epithelium by respiratory pathogens.

PERIODONTITIS EMPHYSEMA.

According to Moodley A et al (1994) Pulmonary emphysema and Periodontitis share a similar mechanism of tissue destruction. In Periodontitis & Emphysema degranulation of neutrophil occurs during attempted phagocytosis (so called frustrated phagocytosis) which

release proteolytic enzymes. Destruction of the pulmonary alveoli and the periodontal attachment results due to degradation of proteins from connective tissues [10].



PNEUMONIA AND PERIODONTAL DISEASE

Pneumonia is an infection of the lungs caused by bacteria, viruses, fungi, or mycoplasma and is broadly categorized as either community- acquired or hospital acquired pneumonia.[11]

Community-acquired bacterial pneumonia is caused primarily by inhalation of infectious aerosols or by aspiration of oropharyngeal organisms. Streptococcus pneumoniae and Haemophilus influenzae are most common, although numerous other species may be found, including anaerobic bacteria. Antibiotic therapy is highly successful in resolution of most cases of community-acquired bacterial pneumonia.[12]

Hospital-acquired (nosocomial) bacterial pneumonia has a very high morbidity and mortality rate. Approximately 20% to 50% of patients with nosocomial pneumonia die. The incidence

of nosocomial pneumonia is highest in severely ill patients such as those in intensive care units or on ventilatory support. More than half of patients on mechanical ventilation for several days are vulnerable to acquire pneumonia. Although nosocomial pneumonia is most often caused by gram-negative microorganisms, many cases are the result of infection by anaerobic bacteria, including those typically found in the subgingival environment.[13]

Several studies have reported that hospitalized subjects tend to have poorer oral hygiene than matched ambulatory non hospitalized control subjects. Lack of attention to oral hygiene in hospitalized patients results in increase in the quantity and complexity of dental plaque. Bacterial interactions may result in colonization of plaque by respiratory pathogens. These organisms may be shed into saliva and reach the distal part of the respiratory tree.[14]

BRONCHIAL ASTHMA AND PERIODONTAL DISEASE

Asthma is a chronic inflammatory disease of the respiratory system characterized by being hyper-responsive and episodic, reversible symptoms of air flow obstruction. The prevalence of asthma has been increasing across all age, gender, and racial groups and is found to be higher among children than adults. Many cells and cellular elements play a role in asthma, in particular, eosinophils, T lymphocytes, neutrophils and epithelial cells.[15]

In susceptible individuals, the inflammation causes recurrent episodes of coughing, wheezing, chest tightness and difficult breathing, especially at night and in the early morning; however, asthma is a disease with many faces.Periodontal disease has been known as an inflammatory disease with a reaction to bacterial plaque causing chronic inflammation, gingival bleeding, increasing pocket depth, and ultimately, alveolar bone loss. In fact, bacterial antigens irritate the immune response of the host leading to the effects of the disease.[16]

As in asthma, the immune response is the mechanism involved in the pathogenesis and progression of the disease. Although the disease is mostly associated with adults, a significant portion is seen in children and young adults as well.[17]

Moreover, studies examining the association between periodontal diseases and asthma have reported varying results. Hyyppa et al.,[18] McDeera et al.,[19] Shashikiran etal.[20]and Stensson et al.[21] revealed that asthmatics have poorer periodontal health than the control population, whereas Bjerkeborn et al. and Eloot et al. did not find any difference in the prevalence of periodontal disease in asthmatics. An association between asthma and periodontal disease may involve either pathological activation of the immune and inflammatory process, the side effect of the asthma medications, or the interaction between the two. Hyyppa¨ et al. suggested that gingivitis in asthmatic children could be explained by an altered immune response and the dehydration of alveolar mucosa due to mouth breathing.[18] The concentration of IgE in gingival tissue is found to be elevated in patients with asthma, which can also cause periodontal destruction. Interaction between bacterial and immunological factors are found to be the main cause of destruction of periodontal tissue. Saliva undoubtedly impacts this interaction through its protective mechanism. Since many asthma drugs modify salivary secretion in a significant percentage of patients, the periodontal health of these patients may be affected negatively. McDeera et al.[19] suggested that children with asthma have more calculus than normal children. Higher prevalence of calculus in asthmatic children is thought to be due to an increase in the levels of calcium and phosphorous in sub maxillary and parotid saliva. This can also contribute to an increase in periodontal problems in asthmatics.[20]

CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD) AND PERIODONTAL DISEASE

COPD is a condition in which chronic obstruction to airflow occurs, with excess production of sputum as a result of chronic bronchitis and/or emphysema. The most important established risk factor for COPD is a history of prolonged cigarette smoking. Several reports suggest that potential respiratory pathogens, which cause COPD, colonize the mouth of high risk subjects, for example, those in intensive care units and nursing home residents. Significantly, results from preliminary trials demonstrated that alteration to oral hygiene significantly reduces the rate of lower respiratory tract infection in institutionalized subjects. These studies suggest that mouth may serve as important reservoir in lower respiratory tract infection.[21]

In a systemic review of the evidence, Scannapieco et.al [22] in 2003 conduced the interventions used to improve oral hygiene, such as mechanical tooth brushing and chemical antimicrobial rinses, have the potential to decrease the risk of nosocomial pneumonia in high risk patients, such as those in intensive care units or those on ventilators. This conclusion was based on at least 5 randomized controlled intervention trials that showed consistent positive effects of anti-infective oral therapy on the rate of norsocomial pneumonia.

Hayes et al. in the year 1998 also observed that periodontal disease was an independent risk factor for COPD in 1048 adult males in the Veterans Administration Normative aging study, and found a positive correlation between advanced alveolar bone loss and COPD.[23]

A retrospective study conducted by Deo V et. Al.in 2009 to evaluate the association between respiratory diseases and periodontal health status and to co-relate the severity of periodontal disease with that of COPD revealed that the risk of COPD appeared to be significantly elevated when the attachment loss was found to be severe. [1]

MECHANISM OF ACTION OF ORAL BACTERIA IN THE PATHOGENESIS OF RESPIRATORY INFECTION:-

Hematogenous spread and aspiration are the two possible routes exist for oral micro-organisms to reach the lower respiratory tract.s Haematogenous spread of bacteria is an inevitable adverse effect of some dental treatments and may occur even after simple prophylactic procedures. Nonetheless, this route of infection seems rare, and only 2 well documented case reports could be found in the literature. In both cases hematogenous spread was the most likely source of pulmonary infection with periodontal anaerobes.[24]

In contrast, aspiration of material from the upper airway occurs in 45% of healthy subjects during sleep and in 70% of subjects with impaired consciousness. It is probably the main cause of nosocomial infection along with aspiration of gastric contents.[25]

Four important mechanisms have been proposed to explain, how oral bacteria can participate in the pathogenesis of respiratory infection.

1. Oral pathogens may be aspirated into the lung.

2. Enzymes in the saliva, which are associated with periodontal disease, may modify mucosal surfaces to promote adhesion and colonization by respiratory pathogens.

3. Periodontal disease associated enzymes may destroy salivary pellicles on pathogenic bacteria.

4. Cytokines released during periodontal disease process may alter respiratory epithelium to promote infection by respiratory pathogens.[26]

It has been demonstrated that removal of fibronectin from the surface epithelial cells may unmask mucosal surface receptors for respiratory pathogens. Saliva of patients with periodontal disease contains many hydrolytic enzymes capable of degrading fibronectin. These enzymes are derived from bacteria as well as polymorphonuclear leukocytes. P.gingivalis and spirochetes are known to produce proteases that can act on mucosal cells and

favour adhesion of respiratory pathogens. Several studies have shown that certain oral bacteria can breakdown a variety of salivary components such as mucins, which offers a non-specific protection to the host tissues. Many of the pathogenic organisms bind to mucins and are cleared from the oral cavity. Destruction of this protective salivary pellicle may favour colonization of respiratory pathogens.[27]

Cytokines, produced by the host tissue in response to periodontal pathogens, such as IL-1□, IL-1□, IL-6, IL-8 and TNF□ may alter the epithelial cell adhesion molecules. These cytokines get mixed with saliva and contaminate the distal respiratory epithelium. The stimulated respiratory cells may then release other cytokines that recruit inflammatory cells to the site (IL-8). These inflammatory cells release hydrolytic enzymes and other modifying molecules resulting in damage to the epithelium. Such epithelial surfaces are more susceptible to colonization by respiratory pathogens.[28]

Because of the important role, oropharyngeal bacterial colonization plays in the pathogenesis of bacterial pneumonia. Several methods have been proposed to reduce or prevent colonization in susceptible patients, especially those on mechanical ventilation. Selective digestive decontamination (SDD) is one of the methods in which topical antibiotics are used on the mucosal surfaces including oral cavity, to reduce the carriage of pathogenic bacteria to the respiratory tract. Production of proteases by subgingival bacteria and diminish the chances of adhesion of pathogenic bacteria.[29]

CONCLUSION

This review highlights the relationship between periodontal diseases and respiratory diseases. A majority of the scientific literature available suggests that poor oral hygiene thereby leading to periodontal diseases have a positive correlation with the occurrence of respiratory diseases namely pneumonia, bronchial asthma and COPD.The emerging discipline of periodontal medicine will continue to gather higher levels of evidence on these risk

associations and conduct the needed intervention trials. Meanwhile dental clinicians might be highly appraised of the available information on these associations and understand the strengths and weakness of the data. Up-to-date and accurate information on these potential relationships, especially among patients with diagnosed systemic disease or patients with other known risk factors like family histories, smoking or diabetes must be provided.

Dentist have to strengthen their ties and share their findings with medical colleagues. In future, new speciality of periodontal medicine may assist dental clinicians in making diagnosis and treatment modalities which will improve periodontal health in particular and oral health in general and also limit the likelihood of systemic diseases and increased lifespan.

Clinical Significance

The impact of periodontal infection on systemic conditions and diseases has been widely investigated and documented in literature and associated with cardiovascular diseases, diabetes, low birth weight baby, as well as respiratory tract diseases. All interrelated components of respiratory diseases and pulmonary diseases has to be kept in mind while performing oro-dental procedure or the pnemomniologist have to be aware about the periodontal component associated with pulmonary diseases. There should be interdisciplinary approach and consultations in dealing with these problems.

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