Journal of Oral Hygiene & Health
ISSN: 2332-0702
Bhattacharya, J Oral Hyg Health 2023, 11:1
Review Article
Open Access

Oral and Periodontal Infections in the Older Persons Affected by Infectious Complications

Santosh Bhattacharya*

Department of Oral Health, Hitkarini Dental University, India

Abstract

Caries and periodontitis become a permanent worry due to the retention of teeth into old age. Acidic oral streptococci metabolites destroy enamel and dentin, causing dental caries. Dissolution leads to cavitation, which develops into bacterial invasion of the dental pulp if left untreated and allows oral bacteria to enter the bloodstream. Infections of the endocardium, meninges, mediastinum, vertebrae, hepatobiliary system, and artificial joints have all been associated to oral pathogens. Dental plaque causes periodontitis, a pathogen-specific, lytic inflammatory response that weakens the tooth attachment. In persons with diabetes, periodontal disease is more severe and less easily managed; poor glycemic management may make the host response worse. The most common cause of pneumonia acquired in nursing homes is aspiration of oropharyngeal infections, especially periodontal pathogens; variables showing poor dental health are closely associated with an increased risk of getting aspiration pneumonia. Periodontopathic organisms that are blood borne may contribute to atherosclerosis. The morbidity of oral infections and their monorail aftereffects can be reduced in an economical manner by practising daily oral hygiene and receiving routine dental care.

Keywords: Periodontitis; Mediastinum; Periodontal disease; Economical manner

Introduction

The human mouth has more than 300 distinct cultivable types of bacteria, with an estimated 1014 distinct microscopic organisms living in the oropharynx and mouth at any given moment. Caries and periodontal disease, the two most common oral infectious illnesses, have historically been treated and diagnosed by dentists. However, these oral disorders frequently have systemic effects, especially in elderly persons. One of the main causes of bacterial endocarditis and a contributing factor in late prosthetic joint infection is haematogenous seeding from an oral source [1]. Glycemic management is hampered by periodontal disease in patients with diabetes, and poorly managed diabetes may make the condition worse. The main cause of nosocomial pneumonia in elderly people is aspiration of oropharyngeal secretions. Atherosclerosis, coronary artery disease, and stroke have all been related to bloodstream-borne Periodontopathic bacteria.

This review is concerned with the rising prevalence of periodontal and dental diseases in the elderly. In the US, people over 75 years old made up about 70% of the population in 1957. Approximately 35% of Americans over the age of 75 no longer have any teeth, thanks to the fluoridation of drinking water and toothpaste, preventive dental habits, and the expansion of the dental profession [2]. By doing so, the risk of developing dental and periodontal disease is increased into a stage of life where poor self-care is frequently present. The pathogenesis of these two oral infections in elderly people-particularly their systemic effects-will be covered in this paper.

There are various different microbiologic habitats in the mouth, several of which include the teeth. Enamel, a cellular substance made up of roughly 95% calcium hydroxyphosphate (hydroxyapatite) microcrystals and 5% organic material, covers the tooth surfaces that are most visible in the mouth [3]. Although these imperfections in elderly teeth have frequently been eliminated by years of chewing or dental restoration, the biting surfaces of teeth are marked with grooves and fissures that hide bacterial colonies. A second microenvironment can be found on the sides of teeth that come into contact. These bacteria adhere to this contact area, which is shielded from food particles and oral hygiene practises by tooth structure and gingiva.

Exact Etiology and local complications of oral gum disease

The nonhemolytic viridans streptococci, often known as "mutans streptococci," most frequently Streptococcus mutans and Streptococcus sobrinus, are what cause dental caries. These microbes are absent in new-borns but become visible as the primary dentition begins to erupt. DNA testing demonstrates that transmission typically happens from mother to kid, most often via shared kitchenware [4]. The organisms feed on sucrose, which they break down into organic acids and stickier polysugar (dextrans), which keeps them attached to the surfaces of the teeth. Dental decay can therefore occur in oral sites including fissures and contact regions that are not frequently disturbed.

Neoplasia aftereffects

Once germs penetrate the pulp chamber, dental deterioration that is left untreated increases the risk of hematogenous spread of oral pathogens. In a 15-year review of the medical literature, oral pathogens with systemic illness in adults under the age of 50. They found evidence connecting oral streptococci to mediastinal abscesses, meningitis, vertebral osteomyelitis, hepatobiliary disease, and bacterial endocarditis.

Of these disorders, bacterial endocarditis is the most prevalent. Mutans streptococci are responsible for around 27% of cultured instances of bacterial endocarditis [5]. There are published regimens for antibiotic prophylaxis for select at-risk patients before they undergo many dental operations as a result of a link between these solely oral organisms and endocardia disease and the less obvious association

***Corresponding author:** Santosh Bhattacharya, Department of Oral Health, Hitkarini Dental University, India, E-mail: bhattacharya.s.127@gmail.com

Received: 03-Jan-2023, Manuscript No: JOHH-23-86537, **Editor assigned:** 05-Jan-2023, PreQC No: JOHH-23-86537(PQ), **Reviewed:** 19-Jan-2023, QC No: JOHH-23-86537, **Revised:** 24-Jan-2023, Manuscript No: JOHH-23-86537(R), **Published:** 31-Jan-2023, DOI: 10.4172/2332-0702.1000358

Citation: Bhattacharya S (2023) Oral and Periodontal Infections in the Older Persons Affected by Infectious Complications. J Oral Hyg Health 11: 358.

with dental therapy. Patients with prosthetic heart valves and acquired valvular dysfunction, both of which are more common in older persons, are at the highest risk. It is unknown whether the recommended regimen is suitable or effective without data from a prospective, blinded clinical trial (which has not been carried out for ethical reasons), but it is a standard of practise that it would be foolish to disregard. Wahl has still emphasised potential long-term consequences of indiscriminate antibiotic use to support the need for such a trial.

Aetiology of periodontitis

Older people are of relevance for two different forms of periodontal disease. Non-specific gingivitis is a reversible gum inflammation brought on by the presence of bacterial plaque. It affects the gums next to the teeth. In elderly individuals, inflammation occurs more quickly in response to plaque and heals more slowly when plaque is eliminated [6], but improved oral hygiene still cures the problem. Adult periodontitis develops when the epithelial connection between the tooth and the bone and the bone itself become inflamed in response to gingival infections.

As the gingivitis-causing plaque colonies grow, they lose gram-positive bacteria and cocci and start to favour obligate anaerobes over facultative species. Clinically, the flow of secular fluid increases and becomes enriched in lymphocytes and phagocytes. Tissues become red and edematous, and gums bleed when brushed. Vasculitis and lymphocytic infiltration of the gingiva and junctional epithelium (i.e., the tissue at the base of the sulcus) are among the histologic findings [7]. Unless local conditions alter or the host population becomes more susceptible as a whole, gingivitis can persist in the form described for months or even years without developing into periodontitis. As pathogens develop proteolytic collagenase and hyaluronidase that break down junctional epithelium, the gingival inflammatory infiltrate that is initially lymphocytic turns into a plasma cell lesion when the host/pathogen equilibrium shifts. P. Gingivalis and Bacteroides forsythus, which are transmitted both vertically and horizontally, i.e., between couples, are the two specific infections that are most frequently implicated.

An antibody reaction is started in response to the invasion of the functional epithelium and may successfully stop the spread of illness. However, deeper bacterial penetration leads to monocytic activation and the production of cytokines and other inflammatory mediators if the antibody response is insufficient. The destruction of collagen, glycosaminoglycans, and bone is caused by the secretion of matrix metalloproteinase by fibroblasts and macrophages [8]. Although there is no discomfort involved, the host can notice an unpleasant taste or odour. As the sulcus base moves closer to the tooth root's tip, the tooth's bony support is lost. When the gingival height of the tooth remains constant, the sulcus (now known as the "periodontal pocket") deepens. The majority of the bacteria in the pocket switch to anaerobic metabolism.

Type 2 diabetes and dental disease

Dental professionals have long seen that diabetes makes periodontal disease worse, to the point where periodontitis is now thought of as a sixth complication in addition to the other five. Periodontitis-related bone loss is strongly correlated with diabetes [9]. Diabetes under poor control is linked to indicators of periodontal disease activity. For those with and without diabetes, the same major periodontal pathogens are present. Studies have also looked at whether diabetes patients' ability to maintain proper glycemic control while they have periodontitis, an infectious condition. This notion of causation has been backed by various investigations, but at least 1 has been inconclusive. Studies on improving periodontal health to enhance glycemic control have also been conducted, although no causality has yet been established.

There are yet no concrete explanations for how diabetes and periodontitis interact. In response to the presence of gram-negative cell-wall lipopolysaccharides, the type 2 diabetes inflammatory responses includes increased release of many inflammatory mediators, particularly IL-1, prostaglandin E2, and TNF-, with the resultant widespread tissue lysis. A second theory is that the macrophage phenotype associated with periodontal diseases is altered by advanced glycation end products produced in response to hyperglycemia, high serum low-density lipoprotein levels, and elevated triglyceride levels. That phenotype's high cytokine production leads to increased inflammatory tissue damage and alveolar bone loss [10].

Gingival disease and chronic infections in oral diseases

Nursing home acquired pneumonia is the second most frequent reason for hospitalisation in this population and the main cause of death for patients in nursing homes. Nosocomial pneumonia in general and nursing home-acquired pneumonia in particular is nearly solely caused by anaerobic gram-negative bacilli, in contrast to community-acquired pneumonia, which is mostly brought on by viral and pneumococcal pathogens. According to reports, individuals in nursing homes and intensive care units are more likely to have gram-negative rods colonise their oropharynx. These rods have also been found in the dental plaque of patients in these facilities. As one of the "anaerobic bacteria that are most important as causes" of aspiration pneumonia, Finegold listed many well-known periodontal pathogens [11]. The presence of S. aureus, the periodontal pathogen P. gingivalis, and the decay organism S. sobrinus were all found to be significantly correlated with nursing home-acquired pneumonia in a long-term prospective study of approximately 350 elderly veterans residing in a Department of Veterans Affairs nursing home. It becomes clear that the same factors that contribute to periodontal breakdown-poor oral hygiene, plaque accretion, and weakened host defense-also support the growth and subsequent aspiration of oral pulmonary infections.

The key clinical concern is whether pneumonia incidence would decrease if periodontal disease was controlled. In comparison to control participants, Japanese nursing home residents who received daily and weekly oral hygiene interventions had fewer cases of pneumonia, fewer fevers, and fewer hospitalisations. The results are fascinating and deserve independent confirmation as well as potential consideration for wider application.

Periodontopathic bacteria easily enter the bloodstream as a result of the edematous status of the diseased periodontal pocket and the strong population of pathogenic bacteria growing there. In both individuals with and without periodontal disease, Cobe found that gentle tooth brushing and even chewing resulted in cultivable anaerobic and aerobic bacteraemia. Increasing incidence and severity of bacteraemia after brushing are correlated with increased gingival inflammation [12]. One group of microorganisms linked to both bacterial endocarditis and LPJI includes periodontal pathogens.

Correlations between indicators of dental health that show the presence of caries and periodontal disease, as well as cardiovascular and cerebrovascular diseases. Even after taking into account characteristics including age, blood lipid level, body mass index, smoking, and socioeconomic status, relationships for men continued to be important. Even after adjusting for the patient's sex, smoking, and socioeconomic characteristics, those with periodontal disease and tooth loss had a

twofold increased risk for coronary heart disease among National Health and Nutrition Examination Survey (NHANES) II participants. Significant multivariate associations between periodontal disease and later cardiovascular disease were discovered in 51,000 medical professionals throughout the course of an observational study [13]. Cerebrovascular disease and periodontal disease were significantly correlated in around 350 senior veterans. Even after adjusting for age, smoking, diet, and other obvious potential confounding factors, there are still significant correlations between bone loss and coronary artery disease, fatal coronary artery disease, and cerebrovascular accident in the 1147 veterans of the VA Normative Aging Study/Dental Longitudinal Study.

According to Herzberg, Meyer, Loesche, and Lopatin, blood borne Periodontopathic bacteria's cell-wall lipopolysaccharides activate the conversion of fibrinogen to fibrin, which results in the production of thrombi. In their examination of atherosclerotic plaques removed from endarterectomies, discovered cellular remains and DNA fragments from Prevotella intermedia, B. forsythus, P. Gingivalis, and Actinobacillus actinomycetemcomitans. The mechanism described by Salvi et al., which involved injury to the endothelium caused by inflammatory mediators, such as C-reactive protein, IL-1, PGE2, and TNF-, elaborated by blood borne Periodontopathic bacteria, was related to the alternative model.

Discussion

Without a doubt, diseases outside the mouth cavity are linked to oral microorganisms, especially in elderly persons. Serious cardiac and orthopaedic diseases are brought on by the hematogenous metastasis of infectious agents arising from dental caries and periodontal disease. Geriatric mortality, morbidity, and healthcare costs are dramatically impacted by aspiration of infections that inhabit the oropharynx. In an individual with a full dentition, the total area of the inflammatory epithelial lining of periodontal pockets may surpass 25 cm2 [14]. This size of a bleeding skin wound requires prompt medical and nursing care. However, a number of variables have made it possible for the majority of the mainstream medical community, the population that provides care, and old people themselves to passively tolerate or simply overlook oral disease in advanced age. These include the long-standing divide between dentistry and medicine, the high cost of dental care in the face of older Americans' relative lack of third-party dental coverage, people's acceptance of the dental deterioration associated with ageing, the aversion of most adults to having someone else brush their teeth, the aversion of most people to cleaning another person's mouth, and many adults' aversion to dental treatment due to unpleasant side effects.

The widely accepted methods for preventing dental caries and periodontal disease include using a toothbrush and fluoridated toothpaste at least twice a day, using a tool to clean in between teeth and along the gum line, getting regular dental exams with professional cleanings, and consuming less refined sugar. People who experience reduced saliva production as a side effect of medication are more likely to develop dental decay, thus they should definitely use concentrated sodium fluoride gel every day and go to the dentist more frequently [15]. People who have had dental appliances replace missing teeth may need to become skilled with 1 or more of the many specialty dental brushes available to clean no anatomic surfaces. A person who has extensive periodontal disease-related bone loss will have to work more to keep exposed root surfaces clean. The effectiveness of preventive dentistry in older people may be hampered by the previous three factors as well as others, including finances, motivation, and social environment.

Conclusion

Unclean mouths would only be an aesthetic issue if poor oral health did not cause significant disease. However, untreated dental and periodontal conditions increase the risk of major morbidity and mortality as well as the cost of preventable medical care. Sadly, until the public demands a higher standard of oral care for their dependent ancestors or until those in charge of allocating resources for healthcare realise that the additional cost of daily oral care is less than the cost of ignoring it, the status quo of poor oral hygiene among frail elderly people will persist.

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