Journal of Oral Hygiene & Health
ISSN: 2332-0702
Yoshida, J Oral Hyg Health 2022, 10:12
Mini Review
Open Access

Dental Infection Affects the Body Parts by Inherent Diseases

Kelhal Yoshida*

Department of dentistry, Mount Kenya University, Kenya

Abstract

The pathogenesis and progression of several systemic disorders, including cardiovascular disease, bacterial pneumonia, diabetes mellitus, and low birth weight, may be influenced by mouth infections, particularly periodontitis. This review's objective is to assess the present position of oral infections, particularly periodontitis, as a contributing cause of systemic disorders. There have been three hypothesised mechanisms or pathways that connect oral infections to secondary systemic effects: I the metastatic spread of infection from the oral cavity as a result of transient bacteraemia; (ii) the effects of circulating oral microbial toxins; and (iii) the inflammation brought on by immunological injury brought on by oral microorganisms. Three variables, including shared risk factors, sub gingival biofilms functioning as gram-negative bacteria reservoirs, and the periodontium acting as a reservoir of inflammatory mediators, may contribute to periodontitis' ability to influence the host's susceptibility to systemic disease. The odontogenic systemic disorders listed above are supported by proposed evidence and mechanisms.

Keywords: Systemic disorders; Periodontitis; Hypothesised mechanisms; Oral microbial toxins

Introduction

According to the focal infection idea, which was put forth in the late 19th and early 20th centuries, sepsis "foci" were to blame for the beginning and development of a number of inflammatory disorders, including appendicitis, arthritis, and peptic ulcers [1]. The prevalence of the localised infection theory led to widespread therapeutic eventuation in the oral cavity. The notion was disproved and widely disregarded for many years because numerous teeth were taken in the absence of illness, offering little relief from symptoms. A more accurate evaluation of the significance of oral focused infection has been made possible by recent advancements in the classification and identification of oral bacteria and the understanding that some germs are typically found only in the oral cavity [2]. In particular in immunocompromised hosts like patients with cancer, diabetes, rheumatoid arthritis, or receiving corticosteroid or other immunosuppressive therapy, it has become increasingly evident that the oral cavity can act as the site of origin for dissemination of pathogenic organisms to distant body sites. Numerous epidemiological studies have revealed that systemic disorders may be influenced by mouth infections, particularly marginal and apical periodontitis.

The only no shedding surface in the body is the tooth, and dental plaque contains as many as 1011 germs per milligram me. Approximately 200 species (in apical periodontitis) and more than 500 species (in marginal periodontitis) have been found in complex micro floras that are linked to human endodontic and periodontal diseases [3]. Gram-negative rods are the most typical isolates of these illnesses, which are primarily anaerobic in nature. As a result of these micro floras' near anatomical proximity to the bloodstream, bacteraemia and the systemic transmission of bacterial by-products, components, and immune complexes can be made easier.

Bacteraemia

It is commonly known that dental operations such root scaling, endodontic therapy, periodontal surgery, and tooth extraction can cause bacteraemia. Through the lysis-filtration of blood samples and subsequent aerobic and anaerobic incubation, bacteraemia following dental extraction, third-molar surgery, dental scaling, endodontic treatment, and bilateral tonsillectomy has been examined. Bacteraemia was seen in all of the patients who had dental extractions, 70% of patients who had dental scaling, 55% of patients who had third-molar surgery, 20% of patients who had endodontic therapy, and 55% of patients who had bilateral tonsillectomy [4, 5]. More commonly than facultative anaerobic bacteria, anaerobes were identified. Another study with 735 kids receiving therapy for severe dental decay discovered that 9% of the kids had bacteraemia that could be detected prior to receiving dental care. Additionally, a number of conservative and hygiene measures, such as tooth brushing, raised the prevalence of bacteraemia from 17 to 40%. Surgery and anaesthesia procedures raised the likelihood of bacteraemia by 15 to 97%. In a recent investigation, Debelian used phenotypic and genetic techniques to identify the root canal as the assumed source of bacteria that were discharged into the circulation before and after endodontic therapy [6, 7]. 26 patients with single-rooted teeth suffering from asymptomatic apical periodontitis had their root canals sampled for microbes. Patients had blood taken from them during and 10 minutes after endodontic therapy. There were anaerobic microorganisms in every root canal. Since oral bacteria frequently spread into the bloodstream, as was already said, it is possible for the heart, lungs, and peripheral blood capillary system to become infected within a minute of an oral treatment.

All body surfaces contain more than 1013 microorganisms, although the bloodstream and underlying tissues are often sterile. A physical barrier made up of the surface epithelium, defensins, which are host-derived peptide antibiotics in the oral mucosal epithelium, an electrical barrier that reflects the Eh difference between the host cell and the microbial layer, an immunological barrier made up of antibody-forming cells, and the reticulo endothelial system are some of the barriers to bacterial penetration from dental plaque into the tissue in

***Corresponding author:** Kelhal Yoshida, Department of dentistry, Mount Kenya University, Kenya

Received: 01-Dec-2022, Manuscript No: JOHH-22-83402, **Editor assigned:** 03-Dec-2022, PreQC No: JOHH-22-83402(PQ), **Reviewed:** 17-Dec-2022, QC No: JOHH-22-83402, **Revised:** 22-Dec-2022, Manuscript No: JOHH-22-83402(R), **Published:** 29-Dec-2022, DOI: 10.4172/2332-0702.1000352

Citation: Yoshida K (2022) Dental Infection Affects the Body Parts by Inherent Diseases. J Oral Hyg Health 10: 352.

the oral cavity (phagocyte barrier). These barrier mechanisms cooperate normally to prevent and get rid of invading germs. The frequency and severity of both acute and chronic infections can increase when this state of equilibrium is upset by an overt breach in the physical system (such as trauma), the electrical system (such as hypoxia), or the immune system (such as through neutropenia, AIDS, or immunosuppressive therapy). Only a tiny number of primarily facultative bacterial species enter the bloodstream under normal dental and oral health conditions [8, 9]. But with poor oral hygiene, the amount of bacteria colonising the teeth, particularly supragingival, could raise 2- to 10-fold, thereby introducing more germs into tissue and the bloodstream and raising the occurrence and severity of bacteraemia.

Periodontal disease affects susceptibility to systemic disease

Periodontal disease, by far the most prevalent oral infection, is the subject of the majority of studies examining the connection between oral infections and systemic illnesses. A series of illnesses that cause inflammation and damage to the teeth's attachment system are together referred to as periodontal disease. Bacteria present in dental plaque, primarily gram-negative rods, have been identified as the suspected pathogens of periodontal disease in roughly 10 different species. The three gram-negative bacteria that are most frequently linked to periodontitis are Actinobacillus actinomycetemcomitans, Porphyromonas Gingivalis, and Bacteroides forsythus. In addition to destroying the alveolar bone and periodontal ligament, periodontitis lesions also show gingival inflammation [10]. Periodontal pockets are created as a result of bone loss and junctional epithelium migration to the apex.

Page suggested that periodontitis may have three effects on the host's susceptibility to systemic disease: by sharing risk factors, by functioning as a reservoir for gram-negative bacteria in sub gingival biofilms, and by acting as a source of inflammatory mediators in the periodontium. People who are at high risk for periodontitis may also be at high risk for other systemic illnesses including cardiovascular disease due to the same risk factors [11, 12]. Smoking, stress, ageing, race or ethnicity, and male gender are a few of the environmental risk factors and indications that both periodontitis and systemic disorders, such cardiovascular disease, share. Although they have not yet been carried out, studies showing genetic factors common to osteoporosis, premature labour, cardiovascular disease, and periodontitis may be useful.

Sub gingival biofilms are a significant and on-going source of microorganisms. They offer readily accessible reservoirs of LPS and other gram-negative bacteria with easy access to the circulation and periodontal tissues. An intravascular coagulation, an inflammatory cell infiltrate in the artery walls, vascular smooth muscle growth, and vascular fatty degeneration are all caused by a systemic challenge with gram-negative bacteria or LPS [13, 14]. LPS increases the production of thromboxane, interleukin-1 (IL-1), and tumour necrosis factor alpha (TNF-), as well as the expression of endothelial cell adhesion molecules. These changes cause platelet adhesion and aggregation, the development of lipid-rich foam cells, and deposits of cholesterol and cholesterol esters.

Cytokine storage in the periodontium. In periodontitis, elevated tissue quantities of prostaglandin E2, the proinflammatory cytokines TNF-, IL-1, and gamma interferon, as well as other cytokines, are present. As a result, the periodontium can act as a replenishing reservoir for the release of these mediators, which can reach the bloodstream and cause and sustain systemic effects [15]. As well as delaying fibrinolysis, IL-1 promotes thrombosis and coagulation. Platelet adhesion and aggregation, the development of lipid-rich foam cells, and cholesterol deposition can all be triggered by IL-1, TNF-, and thromboxane. Preterm labour and low birth weight babies may be caused by the same mediators that are released by a periodontal disease.

As was already indicated, numerous articles have suggested that a number of clinically significant systemic disorders may be influenced by mouth infections, particularly periodontitis [16]. The most thorough research has been done on endocarditis. It appears that endocarditis can be caused by dental operations and mouth infections according to recognised epidemiological standards. To assert a causative relationship between oral infections and other systemic disorders, however, requires further data.

Cross-sectional and longitudinal epidemiological studies can reveal connections but not causes. Periodontal and systemic disorders may frequently coexist without having a cause-and-effect relationship if some forms of periodontal disease just represent an oral manifestation of a systemic disorder or share etiological characteristics with systemic diseases.

Conclusion

As a result, more study is necessary before it is possible to say for sure whether oral infections have the capacity to harm other parts of the body. The criteria for establishing causation between periodontal disease and broader illnesses were laid out by Slots. These criteria also suggest the paths that additional study in this field should go in the future. Retrospective research should show that the prevalence and incidence of the systemic disease in question are significantly higher in periodontitis patients than in periodontal healthy ones; prospective research should show that the onset of the systemic disease occurs after the onset of periodontitis; intervention research should show that the removal or treatment of periodontitis reduces the incidence of the medical disease; and finally, both the systemic disease's causative microorganisms should belong to the same species (research on pathogenic mechanisms).

It is likely that there is a causal link between periodontal disease and systemic disease if the aforementioned requirements may be met. However, given the wealth of knowledge currently available, it appears reasonable to assert that maintaining good oral health is crucial for both preventing oral disease and maintaining good general health.

References

1. Aldridge JP, Lester V, Watts TL, Collins A, Viberti G, et al. (1995) Single-blind studies of the effects of improved periodontal health on metabolic control in type 1 diabetes mellitus. J Clin Periodontol 22:271–275.
2. Asikainen S, Alaluusua S (1993) Bacteriology of dental infections. Eur Heart J 14:43–50.
3. Beck JD, Garcia RI, Heiss G, Vokonas PS, Offenbacher S (1996) Periodontal disease and cardiovascular disease. J Periodontol 67:1123–1137.
4. Beighton D, Radford JR, Naylor MN (1990) Protease activity in gingival crevicular fluid from discrete periodontal sites in humans with periodontitis or gingivitis. Arch Oral Biol 35:329–335.
5. Boon NA, Fox KAA (1995) Disease of the cardiovascular system. In: Edwards C R W, Bouchier I A D, Haslett C, Chilvers E R, editors. Davidson's principles and practice of medicine. 17th ed New York, NY: Churchill Livingstone 191–312.
6. Byrne J, Ellsworth C, Bowering E, Vincer M (1993) Language development in low birth weight infants: the first two years of life. J Dev Behav Pediatr 14:21–27.
7. Chauncey HH, Muench ME, Kapur KK, Wayler AH (1984) The effect of the loss of teeth on diet and nutrition. Int Dent J 34:98–104.

8. Childs WC, Gibbons R J (1990) Selective modulation of bacterial attachment to oral epithelial cells by enzyme activities associated with poor oral hygiene. J Periodontal Res 25:172–178.

9. Christianson RE, van den Berg BJ, Milkovich L, Oechsli FW (1981) Incidence of congenital anomalies among white and black live births with long-term follow-up. Am J Public Health 71:1333–1341.

10. Clinton SK, Fleet JC, Loppnow H, Salomon RN, Clark BD, et al. (1991) Interleukin-1 gene expression in rabbit vascular tissue in vivo. Am J Pathol 138:1005–1014.

11. Cunha BA (1986) Hospital-acquired pneumonias: clinical diagnosis and treatment. Hosp Physician 22:12–17.

12. Dasanayake AP (1998) Poor periodontal health of the pregnant woman as a risk factor for low birth weight. Ann Periodontol 3:206–212.

13. Debelian GJ, Olsen I, Tronstad L (1998) Anaerobic bacteremia and fungemia in patients undergoing endodontic therapy: an overview. Ann Periodontol 3:281–287.

14. DeStefano F, Anda RF, Kahn HS, Williamson DF, Russell CM (1993) Dental disease and risk of coronary heart disease and mortality. Br Med J 306:688–691.

15. Donowitz GR, Mandell GL (1990) Acute pneumonia. In: Mandell GL, Douglas RG, Bennett JE, editors. Principles and practice of infectious diseases. New York, NY: Churchill Livingstone 540–555.

16. Drangsholt MT (1998) A new causal model of dental diseases associated with endocarditis. Ann Periodontol 3:184–196.

17. Durack DT, Beeson PB, Petersdorf RG (1973) Experimental bacterial endocarditis. 3. Production and progress of the disease in rabbits. Br J Exp Pathol 54: 142–151.