South American Publishing

SAP Dentistry. 2024; 2:107
doi: 10.62486/dent2024107

REVIEW

Periodontal pathogens in the appearance and progression of chronic periodontitis

Patógenos periodontales en la aparición y progresión de la periodontitis crónica

Rosa María Montano-Silva[1], Yoneisy Abraham-Millán[1], Jennifer de la Caridad Romero-Amaro[1]

[1]Facultad de Ciencias Médicas Isla de la Juventud. Isla de la Juventud, Cuba.

Cite as
Montano-Silva RM, Abraham-Millán Y, Romero-Amaro J de la C. Periodontal pathogens in the appearance and progression of chronic periodontitis. SAP Dentistry. 2024; 2:107. https://doi.org/10.62486/dent2024107

Publication History
Submitted: 22-11-2023
Revised: 16-03-2024
Accepted: 03-11-2024
Published: 04-11-2024

Corresponding Author
Rosa María Montano-Silva

ABSTRACT

Periodontal diseases are considered a public health problem due to their high prevalence worldwide, so the authors set out to describe the role of periodontal pathogens in the onset and progression of chronic periodontitis. The documentary analysis method was used and a total of 18 bibliographies were reviewed. Current scientific evidence supports the association between periodontal pathogens and the development of periodontitis, demonstrating that bacteria such as Porphyromonas gingivalis, Tannerella forsythia and Treponema denticola play a crucial role in the pathogenesis of periodontal disease by triggering inflammatory responses and promoting the destruction of tooth-supporting tissues.

Keywords: Periodontal Pathogens; Chronic Periodontitis; Onset; Progression.

RESUMEN

Las enfermedades periodontales son consideradas un problema de salud pública debido a su alta prevalencia a nivel mundial, por lo que las autoras se trazaron como objetivo describir el papel de los patógenos periodontales en la aparición y progresión de la periodontitis crónica. Se utilizó el método de análisis documental y se revisaron un total de 18 bibliografías. La evidencia científica actual respalda la asociación entre los patógenos periodontales y el desarrollo de la periodontitis, demostrando que bacterias como Porphyromonas gingivalis, Tannerella forsythia y Treponema denticola, desempeñan un papel crucial en la patogénesis de la enfermedad periodontal al desencadenar respuestas inflamatorias y promover la destrucción de los tejidos de soporte de los dientes.

Palabras clave: Patógenos Periodontales; Periodontitis Crónica; Aparición; Progresión.

© 2024; The Authors. This Open access article is distributed under the Creative Commons Attribution 4.0 license (https://creativecommons.org/licenses/by/4.0), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

INTRODUCTION

Oral diseases are highly prevalent. Worldwide, 3,5 billion people suffer from them, and they are closely related to non-communicable diseases, thus placing a considerable burden on health, society, and the economy.[1] Although improvements have been seen in some countries, the burden of poor oral health persists, especially among the most vulnerable members of society. The economic burden of poor oral health and the direct and indirect costs of oral diseases amount to US$545 billion worldwide,[2] making them one of the most costly areas of health care. In addition to pain, discomfort, and a lack of well-being and quality of life, oral health problems cause school and work absenteeism,[3] leading to learning deficits and productivity losses.

The estimated prevalence of periodontitis is around 30 %, while the most severe cases range from 6 % in Canada to 50 % in Brazil. In Spain, 85 % and 94 % of the population over 35 years of age has some gum problem, and between 15 % and 30 % suffer from periodontitis, which is severe in 5-11 % of cases. Periodontitis is estimated to be prevalent in 60-80 % of the population in Cuba.

According to the World Health Organization (WHO) definition of health, periodontal health is a state free of inflammatory periodontal disease, allowing the individual to function normally and avoid the consequences (mental or physical) of having or having had the disease. Periodontal health is based on the absence of disease, but not only in patients who have not had the disease; it also includes patients who have had a history of successful treatment of gingivitis and periodontitis or other periodontal conditions, capable of maintaining their teeth without signs of clinical gingival inflammation.[4,5,6]

Periodontitis is considered an inflammatory disease that affects the tissues of the periodontium. It has multiple causes, with periodontopathogenic bacteria commonly (and opportunistically) found in the biofilm present in the oral cavity as the primary etiological factor. Biofilm is defined as a group of microorganisms that create an ideal ecological niche for survival and development.[4,5,6]

Considering the elements described above, the authors set this research's objective to explain the role of periodontal pathogens in the onset and progression of chronic periodontitis.

DEVELOPMENT

Periodontitis is defined as a chronic, multifactorial inflammatory disease associated with a dysbiotic biofilm characterized by progressive destruction of the tissues supporting the teeth. Its main clinical manifestations include:[4,5,6]

- Redness.
- Swelling.
- Bad breath.
- Sensitivity to cold.
- Bleeding.
- Tooth mobility.
- Gingival recession.
- Periodontal pocket formation.
- Masticatory dysfunction.
- Tooth loss.

The oral cavity is a favorable environment for developing microorganisms, providing adequate moisture, temperature, pH, and nutrients for their growth. Moisture promotes biofilm formation and the exchange of ions and nutrients. Bacteria generally require a neutral pH, and in the mouth, the pH range is between 6,75 and 7,25, so any alteration of this range affects or favors the growth of particular species.[7]

Molecular biology methods have identified between 800 and 1 000 species of bacteria isolated from the oral cavity. It has been established that around 50 species are etiological agents of periodontal disease. All these microorganisms progressively damage the structures of the periodontium, initially causing minor lesions in a continuous process, where the chronic inflammatory response becomes harmful, the lesion becomes more severe, generating a clinical picture known as periodontitis, where the supporting structures of the tooth deteriorate, which can lead to tooth loss.[7,8]

Several periodontal pathogenicity factors allow these microorganisms to act at the periodontal level:[9]

1. Structural elements:
    - Adhesins: contribute to adhesion, aggregation, and congregation.
    - Endotoxins activate the alternative complement pathway and macrophages, causing tissue damage and bone resorption.
    - Capsules and immunoglobulin (Ig)-binding surface proteins with anti-opsonic and phagocytosis-blocking effects.
    - Flagella and related structures promote subepithelial penetration of bacteria.
    - Smooth coagulase: creates a fibrin coating around bacteria and protects them from phagocytic action.

2. Exotoxins: especially leukotoxins, which destroy polymorphonuclear cells, and epitheliotoxins, which promote subepithelial penetration.

3. Exoenzymes: these include impedins, which reduce the host's defenses (proteases), and aggressive enzymes, which cause tissue destruction (collagenase, hyaluronidase, alkaline phosphatase, chondroitin sulfate).

4. Metabolites: hydrogen sulfide, ammonia, amines, and fatty acids.

5. Immunoregulatory compounds, such as cytotoxins (which polyclonally activate B lymphocytes or inhibit their proliferation),

those that activate suppressor T lymphocytes, those that inhibit or activate the chemotaxis of polymorphonuclear cells, those that inhibit the intracellular destruction of bacteria, and those that stimulate lysosomal degradation.

Although there are certain variations related to geographical area, ethnicity, or race, the strictly anaerobic bacteria that make up a bacterial flora called the red complex are Aggregatibacter actinomycetemcomitans, Porphyromonas gingivalis, Tannerella forsythia, and Treponema denticola. Other bacteria among the leading infectious agents include Prevotella intermedia and Fusobacterium nucleatum.[10,11,12]

- Aggregatibacter actinomycetemcomitans: a facultative anaerobic, Gram-negative bacillus. Leukotoxin is one of the main virulence factors that can destroy the host's immune tissues. It is a large pore-forming toxin and belongs to the family of repeated toxin-like bacteriocins (RTX). Leukotoxin A (LtxA) production is considered the main pathogenic component in the progression of periodontitis. Lymphocyte function-associated antigen (LFA)-1 (CD11a/CD18), Mac-1 (CD11b/CD18), and α X β 2 (CD11c/CD18) act as receptors for LtxA (CD18 harbors the primary binding site for LtxA), which explains the selective death of leukocytes. Secreting LtxA induces cytolysis of target cells, deactivating the host's immune defenses and allowing escape from immune surveillance.[10,11,12]
- Porphyromonas gingivalis is a periodontal pathogen that predominantly expresses a series of potential virulence factors involved in the pathogenesis of periodontitis. It is a pigmented Gram-negative anaerobic bacillus that forms black colonies on blood agar, successfully colonizing the oral epithelium and forming part of the subgingival microbiome, widely recognized as a contributor to the development of periodontal infections along with other oral pathogens. Virulence factors include fimbriae, cysteine proteases, hemagglutinins, and lipopolysaccharides (LPS), which, together with their many interactions with the host immune system, strongly support its potency as a pathogen.[13,14,15,16]
- Tannerella forsythia: a fusiform-shaped, strictly anaerobic, immobile bacillus present in periodontal disease. Due to its virulence factor and ability to survive in optimal conditions in the periodontal pocket, it can stimulate the activation of inflammatory mediators, which progressively damage the periodontium with the chronicity of the disease. It has a mechanism for evading the host response, either through its S layer or through the activation of apoptosis by the lipoprotein BflP, which reduces the presence of leukocytes and lymphocytes.[17]
- Treponema denticola: This is a spirochete identified in various forms of gingivitis, with a notable presence in root canal infections and acute apical abscesses. Its presence has been quantified, representing almost 50 % of the polymicrobial population in subgingival plaque in patients with periodontitis and less than 1 % in healthy patients, considering it a commensal bacterium at the gingival sulcus level. Its ability to adhere to cells and extracellular components through adhesins such as the Msp protein gives it the first step in colonization and possible invasion. The presence of motility due to periplasmic flagella allows it to invade and reach deeper areas. Its status as a strict anaerobe will enable it to grow in infected, necrotic root canals, generating acute apical abscesses at a distance.[18]
- Prevotella intermedia: Gram-negative anaerobic bacillus. This microorganism needs iron for growth and virulence acquisition; it also can increase the activity of degradative enzymes in the progression of periodontitis. Hemoglobin is found in the crevicular fluid of sites with periodontal disease, making it a possible source of iron for Prevotella intermedia.[19]
- Fusobacterium nucleatum: an elongated, anaerobic Gram-negative bacillus that plays a vital role in the colonization process of red bacteria.[20] It is a common resident of the oropharyngeal microbiota in humans and is an acute pathogen in periodontal diseases. Of the oral anaerobes, it is also the most likely to cause extraoral infections. Its metastatic infections can involve the brain, liver, joints, and heart valves. The coaggregation capacity of this bacterium allows it to act as a microbial "bridge" between early and late colonizers during biofilm formation, which is why it is considered a key microorganism in the formation of bacterial plaque.[21]

Understanding the mechanisms by which these microorganisms interact with the immune system and cause tissue damage is essential for developing new therapeutic strategies for controlling the bacterial load in the periodontium and preventing the progression of periodontal disease.

CONCLUSIONS

Current scientific evidence supports the association between periodontal pathogens and the onset and progression of chronic periodontitis. It demonstrates that bacteria such as Porphyromonas gingivalis, Tannerella forsythia, and Treponema denticola play a crucial role in the pathogenesis of chronic periodontitis in adults by triggering inflammatory responses and promoting the destruction of the tissues supporting the teeth.

FINANCING

The authors received no funding for the development of this article.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

AUTHORSHIP CONTRIBUTION

Conceptualization: Rosa María Montano-Silva, Yoneisy Abraham-Millán, Jennifer de la Caridad Romero-Amaro.

Research: Rosa María Montano-Silva, Yoneisy Abraham-Millán, Jennifer de la Caridad Romero-Amaro.
Data curation: Rosa María Montano-Silva, Yoneisy Abraham-Millán, Jennifer de la Caridad Romero-Amaro.
Formal analysis: Rosa María Montano-Silva, Yoneisy Abraham-Millán, Jennifer de la Caridad Romero-Amaro.
Methodology: Rosa María Montano-Silva, Yoneisy Abraham-Millán, Jennifer de la Caridad Romero-Amaro.
Writing - original draft: Rosa María Montano-Silva, Yoneisy Abraham-Millán, Jennifer de la Caridad Romero-Amaro.
Writing - revision and editing: Rosa María Montano-Silva, Yoneisy Abraham-Millán, Jennifer de la Caridad Romero-Amaro.

BIBLIOGRAPHIC REFERENCES

1. Global, regional, and national incidence, prevalence, and years lived with disability for 354 diseases and injuries for 195 countries and territories, 1990–2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet 2018, 392: 1789–1858. doi:10.1016/S0140-6736(18)32

2. Righolt AJ, Jevdjevic M, Marcenes W, Listl S. «Global-, regional-, and country-level economic impacts of dental diseases in 2015». J. Dent. Res. 2018, 97(5): 501-507. doi:10.1177/0022034517750572

3. Peres MA, Macpherson LMD, Weyant RJ, Daly B, Venturelli R, Mathur PR, et. al. Oral diseases: a global public health challenge. Lancet 2019, 394(10194): 249–60. doi:10.1016/S0140-6736(19)31146-8

4. Sojod B, Périer JM, Zalcberg A, Bouzegza S, El Halabi B, Anagnostou F. Enfermedad periodontal y salud general. EMC - Tratado de medicina 1, 2022; 26(1).

5. Yáñez-Zambrano AI, Alvarado-Solórzano AM. Consideraciones sobre la enfermedad periodontal y su control. Dom. Cien. 2016; 2. ISSN: 2477-8818

6. Gil-Raga L. Enfermedad periodontal y embarazo, influencia de los mediadores inflamatorios y otros factores involucrados. Universidad Cardenal-Herrera-CEU, 2014.

7. Hurtado-Camarena A, Bojórquez-Anaya Y, Montaño-Pérez ML, López-Mendoza JA. Bacterias asociadas a enfermedades periodontales. 2016.

8. Ramos-Perfecto D. Tannerella forsythia: patógeno importante en la periodontitis, integrante del complejo rojo. Odontol. Sanmarquina 2020.

9. Peña-Sisto M, Calzado-Da-Silva M, González-Peña M, Cordero-García S, et. al. Patógenos periodontales y sus relaciones con enfermedades sistémicas. MEDISAN 2012; 16(7).

10. Gholizadeh P, Pormohammad A, Eslami H, et.al. Oral pathogenesis of Aggregatibacter actinomycetemcomitans. Microbial Pathogenesis 2017; 113.

11. Höglund-Åberg C, Kelk P, Johansson A. Aggregatibacter actinomycetemcomitans: Virulence of its leukotoxin and association with aggressive periodontitis. Virulance 2015; 6.

12. Gómez-Bañuelos E, Mukherjee A, Darrahy E, Andrade F. Rheumatoid Arthritis-Associated Mechanisms of Porphyromonas gingivalis and Aggregatibacter actinomycetemcomitans. J. Clin. Med. 2019.

13. Enersen M, Nakano K, Amano A. Porphyromonas gingivalis fimbriae. Journal of Oral Microbiology 2013; 5.

14. Mei F, Xie M, Huang X, Long Y, et al. Porphyromonas gingivalis and Its Systemic Impact: Current Status. Journal Pathogens 2020.

15. Yan-How K, Peng-Song K, Gan-Chan K. Porphyromonas gingivalis: An Overview of Periodontopathic Pathogen below the Gum Line. Front. Microbiol. 2016.

16. Olsen I, Taubman MA, Singhrao SK. Porphyromonas gingivalis suppress adaptative inmmunity in periodontitis, atherosclerosis, and Alzheimer's disease. Journal of Oral Microbiology 2016; 8.

17. Moscoso-Arpi EF. Tannerella forsythia en pacientes con enfermedad periodontal de progresión lenta que acuden a la clínica odontológica de la Universidad Católica de Cuenca. 2019.

18. Ramos-Perfecto D, Ávila-Campos MJ, Lévano-Torres V. Treponema denticola: patógeno en procesos periodontales y

pulpares. Odontol. Sanmarquina 2012.

19. Falcón-Pasapera GS, Falcón-Guerrero BE. Prevotella intermedia y enfermedad periodontal en embarazadas. Rev. Odontol. Basadrina 2020; 4.

20. Mujica-Troncoso C, Castillo-Ruiz M, Daille LK, Fuentevilla IA, Bittner M. Codetection of Periodontal Pathogens in Chilean Patients with Chronic Periodontitis. Rev. Clin. Periodoncia Implantol. Rehabil. Oral 2010; 3.

21. Gutiérrez-de-Velasco AZ, Porra-Lira D. Fusobacterium nucleatum, ¿Un patógeno periodontal promotor de carcinogénesis colorrectal? Rev ADM 2016.