ISSN: 3062-3405

Annals of Orthodontics and Periodontics Specialty
Volume 3, Page No: 28-31
Available Online at: aopsj.com

Review Article

Assessing the Psychosocial and Functional Impact of Periodontal Disease

Monica Macrì[1]*, Giuseppe D'Albis[1], Vincenzo D'Albis[1], Anna Antonacci[2], Antonia Abbinante[2], Riccardo Stefanelli[3], Francesco Pegreffi[3,4], Felice Festa[1]

1. Department of Innovative Technologies in Medicine & Dentistry, University "G. D'Annunzio" of Chieti-Pescara, 66100 Chieti, Italy.
2. Department of Interdisciplinary Medicine, University of Bari "Aldo Moro", 70121 Bari, Italy.
3. Department for Life Quality Studies, University of Bologna, 40064 Bologna, Italy.
4. Department of Biomolecular Sciences, University of Urbino Carlo Bo, 61029 Urbino, Italy.

***E-mail** ✉ m.macri@unich.it

Abstract

The function and appearance of patients are negatively impacted by periodontitis and gingivitis, which also have a direct impact on their oral health-related quality of life (OHRQoL). Periodontitis has been associated with several systemic problems, such as poor pregnancy outcomes, cardiovascular diseases, type 2 diabetes mellitus (DM), respiratory disorders, fatal pneumonia in hemodialysis patients, chronic renal disease, and metabolic syndrome. The present review study aimed to investigate the impact of periodontal disease on patients' quality of life. We searched for studies of patients with non-alcoholic fatty liver disease in the Medline, Pubmed, Embase, NCBI, and Cochrane databases. Analysis was performed on management choices, etiology, and incidence. As of right now, it is evident that gingival inflammation is brought on by dental plaque, a microbial biofilm, and that several systemic and oral variables affect the degree and intensity of the inflammation. Furthermore, the dental plaque biofilm and the host's immune-inflammatory response interact in a complex way, as plaque builds up more rapidly in inflamed gingival locations than in non-inflamed sites. It is important to remember that not every inflammatory site will progress to periodontitis. Gingivitis should still be treated with appropriate topical therapeutic intervention to prevent loss of attachment and periodontal tissue deterioration. In the future, objective analytical methods such as transcriptome characterization or epigenetic alteration classification may be used to diagnose gingival disorders.

Key words: Periodontics, Periodontal debridement, Quality of life, Cardiovascular diseases, Diabetes mellitus

How to cite this article: Macrì M, D'Albis G, D'Albis V, Antonacci A, Abbinante A, Stefanelli R, et al. Assessing the Psychosocial and Functional Impact of Periodontal Disease. Ann Orthod Periodontics Spec. 2023;3:28-31. https://doi.org/10.51847/OjyFxHjTxv

Received: 14 May 2023; **Revised:** 19 August 2023; **Accepted:** 24 August 2023

Introduction

Although it does not directly cause tooth loss, preventing gingivitis is an important step in preventing periodontitis. Epidemiologic data have shown that the most common type of periodontal disease, plaque-induced gingivitis, is common in dentate populations of all ages. It is caused by bacterial plaque accumulation at and below the gingival margin, which results in an inflammatory response of the gingival tissues [1-4]. There are important debates on the clinical boundaries for distinguishing between pathologic and normal inflammation because the early transition from healthy to plaque-induced

gingivitis cannot be observed clinically [5]. However, as plaque-induced gingivitis progresses into more severe forms of the condition, clinical signs and symptoms emerge. Gingivitis caused by plaque begins at the gingival margin and may extend to the rest of the gingival unit. Patients with established forms may experience symptoms like halitosis, gingival enlargement and redness, blood in the saliva, and bleeding when brushing their teeth [5-7]. The intensity of clinical signs and symptoms varies from person to person [3] and from one dental site to another. Erythema, edema, and bleeding are common clinical signs of plaque-induced gingivitis in addition to pain, enlargement, and bleeding [2]. The degree of plaque-induced gingivitis is influenced by the tooth and root structure, restorative and endodontic factors, and other dental-related concerns. In most cases, radiographic analysis and attachment level probing are unable to identify the loss of supporting structures in patients with plaque-induced gingivitis. Histopathologic changes include the extension of the rete ridges into the gingival connective tissue, blood vessel vasculitis adjacent to the junctional epithelium, the progressive degradation of the collagen fiber network with changed collagen types, cytopathologic alterations in local fibroblasts, and the emergence of inflammatory/immune cellular infiltrates [4, 8]. According to OHIP-14 (oral health impacts the quality of life), untreated periodontal disease will eventually lower the quality of life, even though recent research indicates that the bacterial phylotypes associated with gingivitis and periodontitis differ. Seven categories are used to categorize the fourteen OHIP items: handicaps, social impairment, bodily and psychological discomfort, physical and psychological disability, and functional limitation [5].
The present review study aimed to investigate the impact of periodontal disease on patients' quality of life.

Results and Discussion

Epidemiology

The study of epidemiology examines how biology, heredity, the physical and social environments, and individual behavior impact population health and disease. Recent advances in research have brought about a fundamental transformation in our understanding of periodontal diseases. As recently as the mid-1960s, the following principles were included in the widely used model for the epidemiology of periodontal diseases: [3] Gingivitis frequently progressed to periodontitis, resulting in a loss of bone support and eventually tooth loss; periodontitis susceptibility increased with age and was the main cause of tooth loss after the ages of 35 to 55. It was believed that everyone was approximately equally susceptible to severe periodontitis. This disease model has been reevaluated since its inception due to advancements in our knowledge of periodontal diseases [3]. As far as we now know, periodontitis is not age-dependent and does not show a linear progression.

Furthermore, the distribution and severity of the disease are strongly influenced by host vulnerability and risk factors. In the never-ending quest for risk factors for damaging periodontal diseases, several epidemiological studies have been searching for correlations. Analytical epidemiology aims to identify disease risk factors, quantify the strength of those relationships, and determine the causality of those relationships [2]. Comprehending risk factors can assist doctors in formulating causal theories, which can then be applied to the development of therapeutic approaches.

Risk factors

By statistically rescaling and stratifying populations to exclude the influence of confounding variables, risk factor analysis has enabled the identification of independent risk factors. These behavioral factors, which include alcohol and tobacco use, are among the various but modifiable risk factors for periodontal disease [8, 9]. They also include diseases and adverse conditions like osteoporosis, osteopenia, metabolic syndrome, diabetes mellitus, and low levels of calcium and vitamin D in the diet. Many patients with periodontal disease today receive therapy that includes treating these modifiable risk factors. Periodontal disease is also influenced by genetic factors, which allows for the early detection and prevention of the illness in particular individuals [10]. It is obvious how hereditary factors play a part in aggressive periodontitis. In the general population, there is currently no solid evidence to support the strong hypothesis that genetic variables (i.e., individual genes) may be associated with chronic adult periodontitis. Identifying the genetic markers associated with chronic periodontitis is important because genetic factors may assist in identifying individuals who are at risk of acquiring the condition. Obesity, diabetes, Smoking, and osteoporosis in postmenopausal women are among the systemic risk factors for periodontal disease that are likely to influence the majority of patients receiving treatment in clinics and dental offices. To treat patients with

periodontal disease, it is now essential to identify and manage risk factors [11]. Arthritis, COVID-19, and cardiovascular disease are among the conditions linked to periodontitis that cause inflammation in the body. The low systemic inflammatory burden associated with periodontitis is the primary factor supporting the biological plausibility of these associations. Several systemic disorders have been linked to periodontitis, including poor pregnancy outcomes6, type 2 diabetes, respiratory illnesses, pneumonia mortality in hemodialysis patients, chronic renal disease, and metabolic syndrome. Severe chronic periodontitis may be linked to the early stages of atherosclerosis due to endothelial and microvascular dysfunctions.

Symptoms and signs

Gum bleeding during brushing or flossing, bad breath, shifting or loose teeth, receding gums, red, tender, or swollen gums, plaque or tartar buildup on the teeth, tooth loss, chewing pain, an unpleasant taste in the mouth, and an inflammatory response throughout the patient's body are some of the symptoms, though they can vary based on the disease's stage.

Treatments

Procedures that are predictable in terms of infection control include surgical periodontal therapy (SPT), non-surgical periodontal treatment (NSPT), decreasing probing pocket depth (PPD), and raising clinical attachment level (CAL) [12]. Effective plaque control is crucial for preserving periodontal health, according to data on tooth-brushing behavior [13]. Brushing your teeth lowers the number of teeth with periodontal pockets, per an 11-year study. There was a correlation between the number of teeth with PPD under 4 mm and how often you cleaned your teeth [14]. The least costly preventative treatment and the most successful way to reduce plaque and gingivitis scores was daily interdental brushing or flossing. Several systematic evaluations have comprehensively demonstrated the clinical efficacy of scaling and root planning (SRP), considered the gold standard non-surgical treatment for periodontitis [15]. It is still possible to preserve and cure even badly damaged teeth. Plaque reduction and SRP may reduce gingival bleeding on probing in around 45% of locations. The effectiveness of SRP may vary depending on the patient's age, medical history, area, tooth type, and degree of periodontal disease. Non-molar teeth displayed a higher PPD reduction with SRP than molars [6]. However, NSPT might not be sufficient to restore periodontal health in patients with severe periodontitis [6, 7]. It has been suggested that other treatments such as lasers, antiseptics, systemic antibiotics, host modulators, and probiotics can increase the effectiveness of SRP [16]. The primary indication for regeneration treatments was deep intrabony defects. Guided tissue regeneration and enamel matrix derivatives showed comparable PPD reduction and CA gains. Additionally, irregularities in furcation may increase the risk of tooth loss.

Conclusion

Dental plaque, a microbial biofilm, is the cause of gingival inflammation, and at this stage, several systemic diseases and oral variables affect the inflammation's degree and intensity. Additionally, the dental plaque biofilm and the host's immune-inflammatory response interact in a complex way since plaque builds up more quickly at inflamed gingival sites than at non-inflamed ones [17]. It is important to remember that not every inflammatory site will develop into periodontitis. But as of right now, there is no scientific proof that tells us which gingivitis locations are most likely to develop into periodontitis. Therefore, to avoid attachment loss and periodontal tissue deterioration, gingivitis must still be treated with the proper local therapeutic intervention. In the future, objective analytical methods like transcriptome characterization or epigenetic alteration classification may be used to diagnose gingival disorders.

Acknowledgments: None

Conflict of interest: None

Financial support: None

Ethics statement: None

References

1. Löe H, Theilade E, Jensen SB. Experimental gingivitis in man. J Periodontol. 1965;36:177-87.
2. Tonetti MS, Chapple IL, Jepsen S, Sanz M. Primary and secondary prevention of periodontal and peri-implant diseases: introduction to, and objectives of the 11th European Workshop on Periodontology consensus conference. J Clin Periodontol. 2015;42 Suppl 16:S1-4.
3. National Center for Health Statistics (US). Periodontal Disease in Adults-United States-1960-1962. 1965.
4. Page RC, Schroeder HE. Pathogenesis of inflammatory periodontal disease. A summary of current work. Lab Invest. 1976;34(3):235-49.
5. Quirynen M, Dadamio J, Van den Velde S, De Smit M, Dekeyser C, Van Tornout M, et al. Characteristics of 2000 patients who visited a halitosis clinic. J Clin Periodontol. 2009;36(11):970-5.
6. Ashurko I, Esayan A, Magdalyanova M, Tarasenko S. Current concepts of surgical methods to increase mucosal thickness during dental implantation. J Adv Pharm Educ Res. 2021;11(3):37-41.
7. Remizova AA, Dzgoeva MG, Tingaeva YI, Hubulov SA, Gutnov VM, Bitarov PA. Tissue dental status and features of periodontal microcirculation in patients with new COVID-19 coronavirus infection. Pharmacophore. 2021;12(2):6-13.
8. Kistler JO, Booth V, Bradshaw DJ, Wade WG. Bacterial community development in experimental gingivitis. PLoS One. 2013;8(8):e71227.
9. Breivik T, Thrane PS, Murison R, Gjermo P. Emotional stress effects on immunity, gingivitis and periodontitis. Eur J Oral Sci. 1996;104(4(Pt 1)):327-34.
10. Centers for Disease Control and Prevention. National diabetes fact sheet: national estimates and general information on diabetes and prediabetes in the United States, 2011. Atlanta, GA: US department of health and human services, centers for disease control and prevention. 2011;201(1):2568-9.
11. Cuff MJ, McQuade MJ, Scheidt MJ, Sutherland DE, Van Dyke TE. The presence of nicotine on root surfaces of periodontally diseased teeth in smokers. J Periodontol. 1989;60(10):564-9.
12. Feres M, Faveri M, Figueiredo LC, Teles R, Flemmig T, Williams R, et al. Group B. Initiator paper. Non-surgical periodontal therapy: mechanical debridement, antimicrobial agents and other modalities. J Int Acad Periodontol. 2015;17(1 Suppl):21-30.
13. Kalf-Scholte SM, Van der Weijden GA, Bakker E, Slot DE. Plaque removal with triple-headed vs. single-headed manual toothbrushes-a systematic review. Int J Dent Hyg. 2018;16(1):13-23. doi:10.1111/idh.12283
14. Joshi S, Suominen AL, Knuuttila M, Bernabé E. Toothbrushing behavior and periodontal pocketing: an 11-year longitudinal study. J Clin Periodontol. 2018;45(2):196-203. doi:10.1111/jcpe.12844
15. Van der Weijden GA, Timmerman MF. A systematic review on the clinical efficacy of subgingival debridement in treating chronic periodontitis. J Clin Periodontol. 2002;29(s3 Suppl 3):55-71. doi:10.1034/j.1600-051X.29.s3.3.x
16. Graziani F, Karapetsa D, Alonso B, Herrera D. Nonsurgical and surgical treatment of periodontitis: how many options for one disease? Periodontol 2000. 2017;75(1):152-88. doi:10.1111/prd.12201
17. Hillam DG, Hull PS. The influence of experimental gingivitis on plaque formation. J Clin Periodontol. 1977;4(1):56-61.