Periodontitis Classification, Clinical Features and Diagnosis

Subjects: Dentistry, Oral Surgery & Medicine | Others

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Periodontitis is a clinical condition where there is chronic inflammation of the periodontium, resulting in the loss of the periodontal ligament and damage to the surrounding alveolar bone. Various microorganisms have been linked to periodontitis. Chronic inflammation is responsible for various complications which are seen in periodontitis cases. For better treatment and diagnosis, it is important to understand the association of periodontitis with other systemic diseases.

Keywords: periodontitis ; oral ; infection ; mechanisms

1. Introduction

Periodontitis is a clinical condition where there is chronic inflammation of the periodontium, resulting in the loss of the periodontal ligament and damage to the surrounding alveolar bone. At first, the teeth loosen, and at an advanced stage, there may be tooth loss. Periodontitis occurs due to an imbalance in the oral microbiota's natural balance and host resistance (dysbiosis), and has been linked to various systemic conditions. In adults, dysbiosis may be the cause of periodontitis, which can lead to inflammatory changes that affect the bone and connective tissue [1].

Periodontitis is one of the most common inflammatory conditions that involves the oral cavity and it has also been linked to cancer [2]. Bacterial plaque is the cause of periodontitis in susceptible individuals. Improper oral hygiene and tobacco consumption, combined with genetically- and disease-associated disturbances of host defenses, are the main factors associated with periodontitis [1]. Predisposing conditions are those that delay or prevent plaque removal and are dependent on the body's immune response [3]. These factors include supra- and subgingival calculus, anatomical abnormalities with regard to the shape or position of the teeth, and iatrogenic factors, including restorative overhangs and subgingival margins [3]. Stress, smoking habits, diabetes mellitus, and systemic diseases are modifying factors that are responsible for the progression of the disease [3]. The nature and course of the inflammatory response is also altered.

Next to the gut, the oral cavity is the second-largest microbiota, harboring almost 700 different bacteria [4]. Thus, the oral microbiome is the leading source of periodontitis, wherein bacterial pathogens generate an inflammatory response that marks the damage of connective tissues [5][6]. The Gram-negative bacteria responsible for causing periodontitis include *Aggregatibacter actinomycetemcomitans*, *Porphyromonas gingivalis*, *Prevotella intermedia*, and *Tannerella forsythia* [7]. Dental plaque is an excellent example of how most naturally-occurring bacteria grow on surfaces as biofilm [7]. *Porphyromonas gingivalis* is a Gram-negative anaerobic bacteria that has been detected in 85.75% of subgingival plaques of chronic periodontitis cases [6].

The important role of periodontal infection as a risk factor for the development of cancer has been a cause of concern. High levels of periodontal pathogens may also predispose patients to gastric cancers as a result of being mediated by systemic infection and inflammation [8]. The Joint European Federation of Periodontology/American Academy of Periodontology Workshop in November 2012 also addressed the risk of periodontitis and its potential to cause the development of cancer. The workshop proposed that future studies should aim to fulfil the Bradford Hill or equivalent criteria [9].

2. Classification of Periodontitis

Periodontitis is usually seen in adults, but children and adolescents may also present with it. After much debate, the term 'adult periodontitis' was replaced with the term 'chronic periodontitis' [10]. The terms "chronic" and "aggressive" periodontitis were first introduced at the 1999 World Workshop for the Classification of Periodontal Diseases and Conditions [11]. Interestingly, the previously described types, i.e., "chronic" and "aggressive", are now included under one single category of "periodontitis." A consensus conference was later held in 2017 to update the earlier 1999 classification.

According to earlier descriptions, in addition to earlier described types, two other different types of destructive periodontal diseases, i.e., periodontitis of systemic disease and necrotizing periodontal diseases, were also mentioned [12].

According to the European Federation of Periodontology report, the staging of the disease needs to consider: the (i) severity, (ii) complexity, and (iii) scoring [13]. Furthermore, there are a few essential goals described by researchers [13]. The primary goal is to classify the severity and extent of destroyed tissue because of the disease [13]. This is done with the help of radiological investigation. Regarding the second goal, clinicians should aim at assessing the complexity involved in controlling the disease and the long-term duration of this management [13]. The stages are then scored, and the severity score is primarily calculated based on interdental attachment loss. The case's complexity is what determines the complexity score. Grading a periodontitis patient involves estimating the future risk of periodontitis progression and the possible responsiveness to standard therapeutic interventions [13]. In addition, direct and indirect evidence also need evaluation [13].

Rapid damage to the periodontal ligament and alveolar bone could occur in periodontitis. The extent of tissue destruction in periodontitis is generally determined by the surface of the dental plaque, host defense, and the linked risk factors. A vital characteristic of periodontitis is specific to the site of involvement, the loss of the specific periodontal pockets and their attachments, and the loss of bones that are not uniform throughout the teeth. Actual evidence does not support a different pathophysiology for chronic and aggressive periodontitis. Accordingly, the definition of a case of periodontitis largely depends on both the extent of the disease (number of affected teeth) and the severity of the disease (severity of depth of pocket, loss of clinical attachment, and loss of alveolar bone in the affected tooth) [13]. Further epidemiological studies are needed to know the etiology of the disease and its prevalence and risk factors for proper planning of adequate treatments and prevention programs.

3. Clinical Features of Periodontitis

The most important aspect in the management of periodontal disease is to diagnose the condition as early and accurately as possible, as extensive damage to the periodontal bone and soft tissue may be difficult to tackle. In the early stages of periodontal disease, patients hardly complain of any pain. Patients may complain of bleeding while brushing, but seldom is pain described. Clinical features of periodontitis include gum redness, changes in structure and swelling, and bleeding from the gum area. There is periodontal pocketing and attachment loss, often there is bad taste or odor, and in the most advanced forms there is tooth loss [14]. Weakening of the dental support can cause severe pain due to abscesses or tooth loss. Compared to other inflammatory conditions, periodontitis may sometimes be present without any pain [15]. Painless presentation explains the fact about why the detection may be delayed and that severe periodontitis is the main cause of tooth loss in adults [15].

The absence of pain sensation is due to the changes in periodontal nociception with regard to painless periodontal conditions [15]. Bacterial virulence, host response, altered innervation of the affected part, and suppression of the inflammation may be main factors that determine pain sensation [15].

In some cases, the disease progresses at a slower rate and there is minimal risk of periodontal function loss, while in others it may progress faster. In addition, some gingival sites within the same individual are more liable to the development of chronic periodontitis than others [16]. It was found that race/ethnicity, level of poverty, and education were associated with increased chances of developing periodontitis [16].

4. Diagnosis of Periodontitis

At present, the diagnosis of periodontal diseases is mainly based on radiographic and clinical examinations of periodontal tissues. Much is needed for the early detection, ascertainment of the severity, and prognosis of the disease, as the present tolls may be inadequate. Using clinical parameters such as bleeding on probing, probing depth and clinical attachment level, and radiographic analysis of supporting bone, proper diagnosis can be made [17][18]. Additional evidence, such as medical and family history and specific medical characteristics, also helps with diagnosis. The diagnostic process also depends on the individual skill of the examiner. Furthermore, the examination is to be repeated at regular intervals to monitor the course of the disease.

It is necessary to examine the oral cavity, measure the depth of pockets, and obtain proper radiographs to know the severity of the disease. Periodontitis biomarkers, such as lactoferrin, hemoglobin, and leukocytes in the saliva, may help in diagnosis. A research study investigated IL-1β, IL-6, MMP-8, and IL-10 levels in healthy as well as periodontitis patients

and found that IL-1β and IL-6 concentrations were significantly higher in periodontitis patients [19]. This proves the importance of investigating the underlying biomarkers.

Once diagnosed, the healthcare practitioner should immediately remove the etiological factors (microbial biofilm on the surface of teeth and gums) and advise the patient about the different risk factors involved. The reversible risk factors can be well controlled. Hence, successful management of the disease should always aim at providing prior and necessary information to the patient about its possible association with any other systemic disease [20].

References

1. Jensen, A.; Gronkjaer, L.; Holmstrup, P.; Vilstrup, H.; Kilian, M. Unique subgingival microbiota associated with periodontitis in cirrhosis patients. Sci. Rep. 2018, 8, 10718.
2. Michaud, D.S.; Fu, Z.; Shi, J.; Chung, M. Periodontal disease, tooth loss, and cancer risk. Epidemiol. Rev. 2017, 39, 49–58.
3. Knight, E.T.; Liu, J.; Seymour, G.J.; Faggion, C.M., Jr.; Cullinan, M.P. Risk factors that may modify the innate and adaptive immune responses in periodontal diseases. Periodontology 2016, 71, 22–51.
4. Deo, P.N.; Deshmukh, R. Oral microbiome: Unveiling the fundamentals. J. Oral Maxillofac. Pathol. 2019, 23, 122–128.
5. Colombo, A.P.V.; Magalhães, C.B.; Hartenbach, F.A.; do Souto, R.M.; da Silva-Boghossian, C.M. Periodontal-disease-associated biofilm: A reservoir for pathogens of medical importance. Microb. Pathog. 2016, 94, 27–34.
6. How, K.Y.; Song, K.P.; Chan, K.G. Porphyromonas gingivalis: An overview of periodontopathic pathogen below the gum line. Front. Microbiol. 2016, 7, 53.
7. Bascones-Martínez, A.; Figuero-Ruiz, E. Periodontal diseases as bacterial infection. Med. Oral Patol. Oral Cir. Bucal 2004, 9, 92–100.
8. Chrysanthakopoulos, N.A.; Reppas, S.A.; Oikonomou, A.A. Association of periodontal disease indices with risk of gastric adenocarcinoma. Ann. Res. Hosp. 2017, 1, 4.
9. Nwizu, N.; Wactawski-Wende, J.; Genco, R.J. Periodontal disease and cancer: Epidemiologic studies and possible mechanisms. Periodontology 2000, 83, 213–233.
10. Armitage, G.C. Development of a classification system for periodontal diseases and conditions. Ann. Periodontol. 1999, 4, 1–6.
11. Demmer, R.T.; Papapanou, P.N. Epidemiologic patterns of chronic and aggressive periodontitis. Periodontology 2000, 53, 28–44.
12. Lindhe, J.; Ranney, R.; Lamster, I.; Charles, A.; Chung, C.P.; Flemmig, T.; Kinane, D.; Listgarten, M.; Löe, H.; Schoor, R.; et al. Consensus report: Periodontitis as a manifestation of systemic diseases. Ann. Periodontol. 1999, 4, 64.
13. Sanz, M.; Tonetti, M. European Federation of Periodontology. Available online: https://www.efp.org/fileadmin/uploads/efp/Documents/Campaigns/New_Classification/Guidance_Notes/report-02.pdf (accessed on 21 September 2022).
14. Gasner, N.S.; Schure, R.S. Periodontal disease. In StatPearls ; StatPearls Publishing: Treasure Island, FL, USA, 2022. Available online: https://www.ncbi.nlm.nih.gov/books/NBK554590/ (accessed on 21 September 2022).
15. Gaurilcikaite, E.; Renton, T.; Grant, A.D. The paradox of painless periodontal disease. Oral Dis. 2017, 23, 451–463.
16. Eke, P.I.; Wei, L.; Thornton-Evans, G.O.; Borrell, L.N.; Borgnakke, W.S.; Dye, B.; Genco, R.J. Risk Indicators for periodontitis in US adults: NHANES 2009 to 2012. J. Periodontol. 2016, 87, 1174–1185.
17. Mittal, S.; Komiyama, M.; Ozaki, Y.; Yamakage, H.; Satoh-Asahara, N.; Yasoda, A.; Wada, H.; Funamoto, M.; Shimizu, K.; Miyazaki, Y.; et al. Gingival bleeding and pocket depth among smokers and the related changes after short-term smoking cessation. Acta Odontol. Scand. 2022, 80, 258–263.
18. Tonetti, M.S.; Sanz, M. Implementation of the new classification of periodontal diseases: Decision-making algorithms for clinical practice and education. J. Clin. Periodontol. 2019, 46, 398–405.
19. Reddahi, S.; Bouziane, A.; Rida, S.; Tligui, H.; Ennibi, O. Salivary biomarkers in periodontitis patients: A pilot study. Int. J. Dent. 2022, 2022, 3664516.
20. Preoteasa, C.T.; Dumitrache, A.; Enache, M.; Iosif, L.; Preoteasa, E. Patient's information on medical aspects. Rom. J. Oral Rehabil. 2018, 10, 36–40.