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Review Article

Periodontal diseases- A brief review

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ABSTRACT

Periodontal diseases consists of a wide range of inflammatory conditions which causes degeneration of Periodontium and affects all supporting structures of teeth such as gingiva, periodontal ligament, cementum and alveolar bone etc. followed by teeth loss. WHO had reported about 10-15% of the world population is suffering from severe periodontal condition. It is complex infectious disease caused by aggressive microbial growth on teeth. The main aim of this study is to provide systemic update on periodontal disease regarding its stages, occurrence, pathophysiology, diagnosis, treatment and management. The pathophysiology of periodontal disease is associated with dental plaque, microbial biofilm formation and immunogenicity of the host cell. The severity of this disease depends upon risk factors and chronological stages. Prevention is attained by daily maintenance of oral hygiene. Various surgical and non-surgical treatments are available to control the formation of microbial biofilm. Daily maintenance and periodic management of this disease control worsening of condition and shows definite improvement in oral health.

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1. Background

Periodontitis is an infection of Periodontium. Whereas, the word 'Perio' means gingiva and other tissues surrounding teeth, 'dont' mean tooth and 'itis' means inflammation, So the whole term "Periodontitis" indicates chronic inflammation of gingiva periodontal ligaments, alveolar bone and dental cementum. According to World Health Organization (WHO) it is widely spreadable chronic disease around the world.² It begins with accumulation of plaque around teeth which form microbial biofilms with bacteria followed by localized inflammation of gingiva. Negligence of this situation causes chronic condition of periodontal disease. At this stage damage of periodontal structure occurs by baleful byproducts and enzyme from periodontal bacteria such as leucotoxins, collagenase, fibrinolysis and other Bacteroids spp.: B. intermedius and B. gingivalis, fusiform organisms: Actinobacillus actinomycetemcomitans, Wollina recta and Eikenella spp.; Porphyromonas gingivalis, Taneerella for synthesis and

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various bacilli and cocci; spirochetes; and amoebas and trichomonads. $^{3-5}$

By maintaining good oral hygiene, it can be reversed at initial stage but if plaque is not removed at this stage then formation of tartar or calculus occur which is not removable by using tooth brush or floss. Because of this tartar, bacteria start attacking deeper tissues due to which periodontal ligaments around teeth gets degraded and leads to resorption of alveolar bone. A space between gingiva and tooth occurs which is referred as Periodontal Pocket and this condition is mainly known as periodontitis or periodontal disease. The severity of this disease depends upon microbial plaque formation.

The screening and examination of this disease has been done by various methods, which results in detection of severity of periodontal ligament. Several tests are there to diagnose the periodontal disease such as radiograph technique, hematological screening, laser treatment, tissue engineering, etc. To control the progression of disease there are many treatment options available (surgical as well as non-surgical) depend upon the chronology of disease. The

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maintenance of this disease is done by intensive care and by sustaining good oral hygiene. ^{8,9}

2. Stages

There are mainly four stages in periodontal diseases which includes different clinical sign & symptoms and radiological screening are given as follows: ^{10–12}

2.1. Gingivitis

It is the only stage when periodontitis can be reversible. At this stage the plaque formation around teeth occurs. There are mainly few painless symptoms seen at this stage such as bad breath, swollen reddish gums and bleeding while brushing and flossing. It can be reversed by maintaining good oral hygiene and regular checkups. Generally, 1-2 mm clinical attachment loss, less than 15% of bone loss around root, probing depth 4mm or less occurs.

2.2. Early stage

It is the second stage of periodontal disease. It is manageable by oral hygiene but not reversible. At this stage, the infection starts spreading to surrounding tissues and starts degrading it. Symptoms at this stage include inflammation of gums, severe bad breath, and bleeding during brushing or flossing, spacing between teeth become evident and will gradually increase. Here, 3-4 mm clinical attachment loss, less than 15-33% of bone loss around root, probing depth 5mm or less occurs.

2.3. Moderate stage

Like second stage moderate stage cannot be reversed. Same symptoms as moderate stage occurs but space between teeth and recessions of gums are more evident. Treatment like deep cleaning, scaling and flap surgeries can be done at this stage. Around5 mm or more clinical attachment loss, 33% of tooth loss of four teeth or less, with complex issues such as probing depth 6 mm or more, Class II-III furcations, and/or moderate ridge defects.

2.4. Advanced stage

Last stage of periodontal disease; wherein 50-90% of loss of periodontal tissues occurs. Also other symptoms like swollen gums that ooze of pus, cold sensitivity, loosening of teeth, painful chewing and severe halitosis occurs. If left untreated it causes more spaces or gaps between teeth and gums, gum recession, patient needling dentures, and other health problems that can be worst. Treatment includes regular checkups, cleaning and maintaining good oral hygiene can help halt the progression of Periodontitis. Secondary Occlusal trauma, severe ridge defects, bite collapse, pathologic migration of teeth, less than 20 remaining teeth (10 opposing pairs) seen.

3. Types of Periodontitis 15-18

3.1. Gingivitis

As described above, gingivitis is inflammation of gums and can be reversed by maintaining oral hygiene.

3.2. Chronic periodontitis

In this type of periodontal disease, symptoms may include chronic inflammation of gums, severe bad breath, and bleeding during brushing or flossing occurs. Loss of epithelial tissue, bone and ligaments which is not reversible.

3.3. Aggressive periodontitis

It can be present in localized or generalized forms, both are early onset form of chronic periodontal inflammatory disease, typical manifesting between puberty and early third decade of life. The symptoms are same as chronic periodontitis.

3.4. Necrotizing ulcerative gingivitis

It is mainly occurring in people who are suffering from malnutrition, immune suppressive and HIV. Necrosis means death of cell or living tissue. It mainly occurs due to deficiency of nourishment needed by people to remain healthy.

3.4.1. Peri-implant mucositis

It is associated with inflammation of soft tissue surrounding dental implants with no sign of bone loss. Symptoms included red or tender gums around implants, bleeding while brushing.

3.5. Systemic chronic periodontitis

This type of chronic periodontal disease happens in patient who have systemic syndrome. Inflammation of gums occurs due to systemic disease such as Diabetes, Heart disease, Respiratory disease, etc.

4. Occurance & Epidemiology

Periodontal disease is mostly prevalent in adults but it may also occur in children and adolescents. ²⁰ Prevalence of periodontal diseases depends upon the level of dental plaque formation and gingival tissue destruction. Site specificity is one of the key features for chronic and aggressive periodontitis. The severity of this disease depend upon depth of periodontal pocket i.e. attachment loss and bone loss of tooth. ²¹

The epidemiology of periodontitis may vary across masses substantially. Frequently used parameters to collect data for occurrence of this disease are clinical attachment loss and probing depth of periodontal pockets which

Stages Of Gum Disease

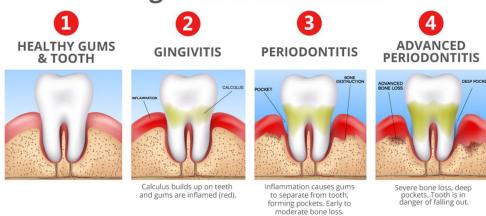


Fig. 1: Various phases of periodontal diseases ¹³

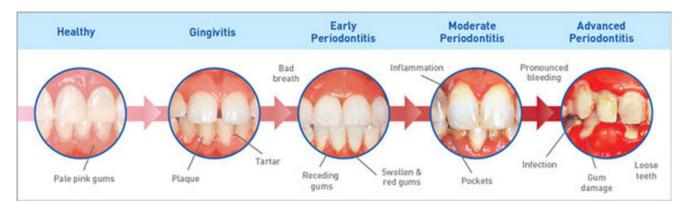


Fig. 2: Various phases of periodontal diseases 14

was first introduced by US centers for Disease Control Prevention and American Academy of Periodontology. ²²

Various studies were conducted by researchers to find out prevalence rate in different countries. Globally 10-15% of populations are suffering from tooth loss due to periodontal disease. 23 A study was conducted in Gautemala by Dowsett et al in 2001 reported that out of 122 patients, attachment loss was found about 3 and 6 mm in 100% and 56% of overall individuals respectively.²⁴ Afterwards in 2003, another clinical study by A. Baelum et al. reported that out of 359 patients of rural Thai population, 92% were diagnosed with higher prevalence of periodontitis due to genetic variation within age group of 30-32 years individuals. 25 Also in 2017, another study was conducted in South India among 1000 individuals which showed prevalence rate of chronic periodontitis among different strata of sample populations (Table 1). ²⁶ Similarly, another clinical study conducted by researchers in 2018 about prevalence rate of periodontitis among different age group of people of South India (Figure 4). 27

Table 1: Prevalence rate in different strata of sample population

Population	Periodontitis prevalence (n=1000)	Prevalence percentage n (%)
Male	252	42.4
Female	171	42.1
Hypertension	19	44.2
Diabetes Type-II	25	43.9
Cigarette smoking	53	44.9
Alcohol consumption	60	39.7
Pan chewing	35	43.2

4.1. Risk factor

There are two kind of risk factor in case of periodontal disease out of which one is modifiable and another is non-modifiable. ²⁸ Cigarette smoking is one of the vital modifiable risk factor for chronic periodontal disease. Higher progression of microbial film is severe in smokers than nonsmokers and more worsen chronic condition occurs

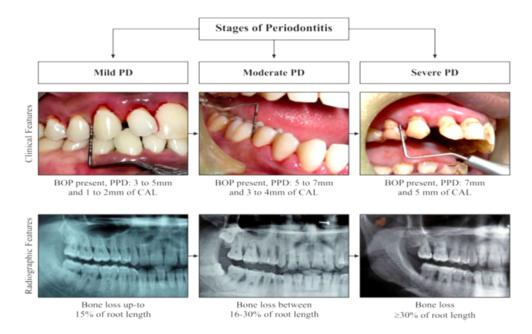


Fig. 3: Clinical photographs illustrating the severity of the disease (BOP: Bleeding on probing; PPD: probing pocket depth; CAL: Clinical attachment loss). Reprinted with permission. ¹⁹ Copyright 2019. Journal of Control Release.

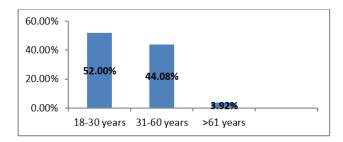


Fig. 4: Prevalence rate in different age group of people

due to habit of smoking. ²⁹ Most prevalent systemic disease is diabetes mellitus predispose to periodontitis. In diabetic patient, prevalence of periodontitis occurs more readily as compared to other immunocompromised patients. ³⁰ Stress is also another consideration as immunosupression and necrotizing ulcerative gingivitis occurs mainly due to stress. ³¹ There are many risk factors associated with this disease are given as (Table 2).

5. Pathophysiology

5.1. The dental plaque or calculus

Periodontitis and gingivitis are mainly initiated with dental plaque. There are around 150 species of microbes are found in single person and overall 800 different types of species of microbes have been identified in calculus of tooth. The species include Gram negative anaerobic bacteria, spirochete and even virus. The imbalance between

Table 2: Types of risk factor in periodontitis

Modifiable risk factor	Non-modifiable risk factor
Microorganisms (specific pathogen)	Osteoporosis
Smoking	Some hematological disorders
Poorly controlled diabetes mellitus	History of periodontitis
Stress	Age
Poor self-care	Gender
Untreated human	Race
immunodeficiency virus or acquired immunodeficiency	
syndrome	
Oral effects of some metabolism	Genetic disorders
Local factors	Bone level
Obesity	Drug-induced disorders
Improper diet	Some host response
Chronic inflammation	Bone levels
Some host responses	Normal hormonal
_	variations (e.g.
	pregnancy)

these microbes forms 'pathogenic unit' in case of chronic periodontal disease. ³²

5.2. Microbial biofilms

As we discussed earlier, microbial biofilm initiates gingivitis. The progression of microbial biofilm depends upon dysbiotic ecological changes in baleful byproducts



Fig. 5: Dental plaque or calculus. Reprinted with permission. ³³ Copyright 2013. International Journal of Medical Dentistry.

and enzyme which results in degradation of periodontal tissue. Microbial biofilms are kind of matrix fixed with different microbial species colony, sticking with each other on tooth surface. ³⁴ There are seven stages of plaque biofilm formation are given as (Table 3).

5.3. Immunogenicity

Not only microbial films are responsible for pathogenesis of periodontal disease but also host cells immune system is responsible for degradation of periodontal ligaments. ³⁶ The balance between microbial biofilm and host cell is lost due to which remarkable variance in both dental plaque and host immunity system occurs, which results in increment of inflammatory cells leads to degradation of periodontal tissue and bone. ³⁷ Therefore decrease of anti-inflammatory cells such as neutrophils, lymphocytes, granulocytes, etc. due to chronic persistence of microbial biofilms which results in severity of alveolar bone resorption by osteoclast and leads to degradation of ligament fibers followed by chronic periodontitis. ³⁸

6. Periodontal Screening and Examination 40,41

The dental examination starts with an extra cellular and intra cellular oral examination of soft and hard tissues. The periodontal examination includes these following steps as follows:

6.1. General description like

- 1. Quantitative assessment of oral hygiene and presence of calculus deposits.
- 2. Presence of gingival inflammation and recession.
- 3. Tooth migrating and related problems.
- 4. Identification of local periodontal risk factors.

- 6.2. Periodontal screening using basic periodontal examination and recordings
- 6.3. Detailed examination of ligament degradation and periodontal pocket depth as:
 - Probing depth of periodontal pocket, attachment loss and recession
 - 2. Bleeding.
 - 3. Suppuration.
 - 4. Furcation involvement.
 - 5. Mobility of tooth.

All these parameters are measured on six sites per tooth such as mesiobuccal, buccal, distobuccal, mesiolingual, midlingual and distolingual. And all these readings are recorded in periodontal chart.

7. Diagnosis 45-47

Diagnosis of periodontal disease has been done by following investigations are given as:

7.1. Radiograph

- 1. Periapical radiograph, Bitewing radiographs, Panoramic X-ray or combination of all these is used to diagnose the prognosis of patients.
- Radiograph provides detailed information about patient's tooth condition. The degree of bone loss and depth of periodontal pocket can be assessed by using Radiograph and also pattern and amount of bone loss.

7.2. Vitality test

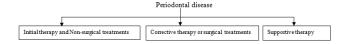
1. Electric Pulp tester or Thermal stimuli is used to diagnose the pulp vitality of tooth.

7.3. Other tests

- 1. Full hematological screening.
- 2. Blood glucose level test.
- 3. INR or microbial plaque sampling

8. Treatment and Management

Treatment plan for periodontal disease are divided into three phases as follows:



8.1. Initial therapy

This therapy is given at initial stage of gingivitis to control the microbial plaque formation and identify any modifiable

Table 3: Stages of microbial biofilm formation ³⁵

No.	Stages	Features
1	Pellicle formation	Occurs by adsorption of Host and bacterial molecules, salivary glycoprotein on tooth surface.
2	Transport	Occurs via natural salivary flow, transport of bacteria such as Neisseria, Streptococcus sanguis, S. oralis, S. mitis and Actinomyces to the pellicle occurs.
3	Long range interactions	This stage leads to reversible adhesion with Vander Wall's and electrostatic forces between microbial cell surface and the pellicle.
4	Short range interactions	This stage leads to irreversible interaction between microbial cell surface and pellicle.
5	Co-aggregation	Increased micro flora diversity due to co-adhesion of new microbes over already attached microbes.
6	Multiplications	Multiplication of adhered bacteria on tooth surface lead to severity of periodontal disease.
7	Detachments	Detachment of colonies to the new site for confluent growth.

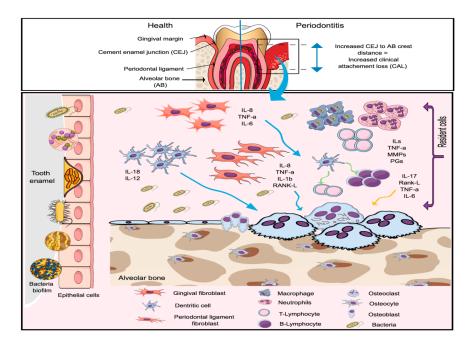


Fig. 6: Immune response in periodontal disease. Reprinted with permission from ref. ³⁹ Copyright 2017. Journal of Clinical Periodontology.

Table 4: Types of radiograph and their parameters

Radiograph types	Parameters
Periapical radiograph	 Long cone parallel technique.
	 Good clarity of images as compared to horizontal radiograph.
	 Time consuming process.
Horizontal bitewings radiograph	• Use for caries detection.
	 Alveolar crest can be visualized.
	 Provides good quality of image for bone loss.
Vertical bitewing radiograph	 Shows 90° angle bitewing film image.
	 Better quality of image for extensive bone loss
Panoramic radiograph	 All teeth seen in one image or film.
	 Newer machine generated for good quality of images.
	 Details are much fine as compared to intraoral radiographs.

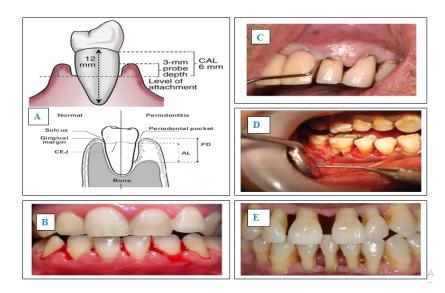


Fig. 7: A: Probing depth of periodontal pocket., ⁴²B: Gingival bleeding. ⁴³, C: Suppuration. ⁴³, D: Furcation involvement. ⁴⁴ E: Mobility of tooth ⁴⁴

risk factor. ⁴⁸ Giving advice to the patients regarding oral hygiene technique, cessation of habits like smoking, alcohol consumption, chewing pan masala, etc. and also doctors gives instruction regarding type of toothbrush to be use, use of interdental aids, dentifrices or mouthwash, etc. ⁴⁹ If periodontal risk factors like Diabetes mellitus identified then patient should be advised accordingly. The therapy is revaluated after 8-12 weeks because 6 weeks is minimum period for healing of tissue or periodontal ligaments. The initial treatment includes following therapies:

8.1.1. Tooth brushing

Manual and electronic toothbrush are available to remove dental plaque. Robinson et al in 2005 reported in this study that oscillating, rotating, powered toothbrush shows more efficiency in removing dental plaque. ⁵⁰

8.1.2. Interdental cleaning

An effective toothbrush can clean only 65% of tooth surface but do not remove overall dental plaque, so interdental cleaning is also necessary to clean the microbial biofilms such as dental floss, tape and powered flossing device. When interdental papillae completely embrasure then dental floss and tape are advised to the patient which helps to improved periodontal clinical outcomes. ⁵¹ (Figure 8)

8.1.3. Adjunctive pharmacological agent

Many Pharmaceutical aids have been added into mouthwashes and toothpaste to increase the efficiency of the product. Widely used agent like Chlorhexidine Gluconate is considered as gold standard anti-plaque/anti-gingivitis agent. ⁵² It is mainly added into mouth wash, gel or toothpaste. There are various examples of adjunctive

aids are given as (Table 5).

8.2. Non-surgical treatment: 53,54

At initial stage of gingivitis, the treatment may be less aggressive as given as:

8.2.1. *Scaling*

Scaling helps to remove calculus and microbial biofilms from gums. It may be operated by using hand instruments or by ultrasonic device.

8.2.2. Root planning

Root planning helps to smooth the root surface and also inhibiting further buildup of tartar. It also removes baleful byproducts to reduce the inflammation and increase healing of attachment of gums to tooth surface.

8.2.3. Antibiotics

Topical or Oral antibiotics are used to control the formation of microbial biofilms. Topical antibiotics such as insertion or gels or implants etc. are inserted in gingival sulcus or in periodontal pockets. However, Oral antibiotics eliminate infection caused by bacteria on gums and teeth surfaces.

8.3. Corrective therapy or surgical treatments 55–58

There are several surgical treatments to treat periodontal disease (Table 6).

8.4. Supportive therapy

This therapy is suggested for prevention of recurrence of disease and also sustenance of periodontal health.⁵⁹

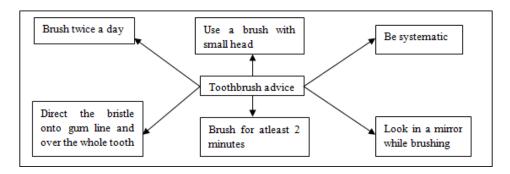


Fig. 8: Tooth brushing advice

Table 5: Adjunctive pharmacological agents

Compounds	Pharmacological aids
Bisguanidine	Chlorhexidine gluconate
Enzyme	Protease, dextranase
Quaternary ammonium compounds	Cetyl pyridinium chloride
Phenols	Triclosan
Essential oils	Thymol, eucalyptol
Metal ions	Zinc, stannous fluoride
Oxygenating agents	Peroxide

Table 6: Examples of surgical Treatments

No.	Surgical Treatments	Features
1.	Flap surgery	 Pocket reduction surgery.
		 Incision on gum tissue for better healing.
2.	Soft tissue grafting	 Removal of small tissue from palate.
		 Use for reducing gum recession.
3.	Bone grafting	 Bone grafting of small fragment from own, synthetic or donated bone.
		 Helps in tooth loss problems and regrowth of natural bone.
4.	Tissue-stimulating proteins	• Applying gel to a diseased tooth root.
		Gel helps in developing tooth enamel and stimulates growth of bone and tissue.
5.	Reparative surgery	 Modified windman flap technique.
		• Use for better access and direct vision to root surface of
		debridement.
6.	Resective surgery	Gingivectomy.
		 Removal and reshaping of tissue occur.
7.	Regenerative surgery	 Regeneration of Periodontium.
		 Regrowth of bone that destroyed by bacteria.
		 Example as tissue regeneration or enamel matrix derivation.
8.	Other surgery	 Occlusal adjustment.
		• Endodontitis.
		 Extraction of microfilm.
		• Fixed/ Removal prosthodontics.
		• Implants.
		Host modulation therapy. Out to the disconnection of the disconnec
		• Orthodontics.
		• Laser surgery.
		• Tissue engineering.

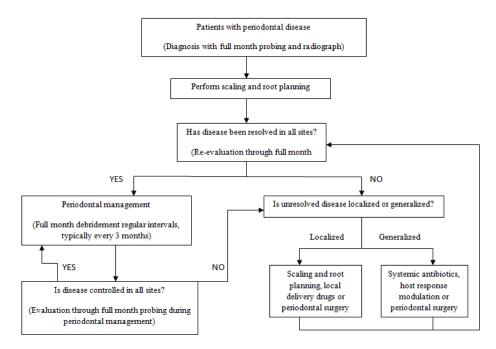


Fig. 9: Decision making for management of periodontitis

This therapy includes regular check-ups of the patient, to monitor the periodontal status and to re-educate to the patient regarding plaque control measures and oral hygiene maintenance. ⁶⁰

Management: Periodontal disease has capacity to control the progression of disease and inhibit the growth of microorganisms. However, the success of therapy for periodontal disease depends upon appropriate management with proper treatments. ⁶¹ The management of periodontal disease consists of removal of supra-gingival and subgingival dental plaque followed by healing in tooth loss. ⁶² In general it takes around 3 months of treatment interval to control the chronicity of periodontal disease. Maintenance period has been customized depends upon severity of disease. Supportive therapy aims long term maintenance of disease, so proper measures are taken to improve the compliances of management by patients to control the disease progression. ⁶³ Management plan for periodontal disease includes Figure 9.

9. Clinical Significance

There has been known direct and indirect impact on overall general health due to oral health. This study helps to improve researcher's knowledge regarding periodontal diseases. Due to rapidly increase of progression and aggressiveness of this disease, patients requires early diagnosis and treatment to prevent from further progression and tissue damage. With the growing advancement in etiopathogenesis of this disease, there have been exponential increments in novel treatment strategies. There are various

treatment modalities available for treatment of periodontal diseases. A thorough diagnosis and treatment planning is important for the management of patient to reduce the chronicity of this disease.

10. Source of Funding

None.

11. Conflict of Interest

None declared.

References

- Preshaw PM, Bissett SM. Periodontitis: Oral Complication of Diabetes. Endocrinol Metab Clin N Am. 2013;42(4):849–67.
- Chapple ILC, Mealey BL, Dyke TEV, Bartold PM, Dommisch H, Eickholz P. Periodontal health and gingival diseases and conditions on an intact and a reduced periodontium: Consensus report of workgroup 1 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. *J Clin Periodontol*. 2018;45(S20):24–9.
- White D, Pitts N, Steele JG. Diseases and related disorders- a report from the adult Dental Health Survey. NHS Information Centre for Health and Social care; 2011.
- Yucel-Lindberg T, Båge T. Inflammatory mediators in the pathogenesis of periodontitis. Expert Rev Mol Med. 2013;15:7–11.
- Abusleme L, Dupuy AK, Dutzan N, Silva N, Burleson JA, Strausbaugh LD, et al. The subgingival microbiome in health and periodontitis and its relationship with community biomass and inflammation. ISME J. 2013;7(5):1016–25.
- Nelson RG, Shlossman M, Budding LM, Pettitt DJ, Saad MF, Genco RJ, et al. Periodontal Disease and NIDDM in Pima Indians. *Diabetes Care*. 1990;13(8):836–40.
- Holand C. Rethinking perio classification for the 21st century. BDJ Team. 2019;6(3):24–7.

- Savage A, Eaton KA, Moles DR, Needleman I. A systematic review of definitions of periodontitis and methods that have been used to identify this disease. J Clin Periodontol. 2009;36(6):458–67.
- Papapanou PN, Sanz M, Buduneli N, Dietrich T, Feres M, Fine DH. Periodontitis: Consensus report of workgroup 2 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. *J Clin Periodontol*. 2018;89(1):1–8.
- Fine DH, Patil AG, Loos BG. Classification and diagnosis of aggressive periodontitis. J Periodontol. 2018;89:S103–7.
- Dietrich T, Ower P, Tank M, West NX, Walter C, Needleman I, et al. Periodontal diagnosis in the context of the 2017 classification system of periodontal diseases and conditions – implementation in clinical practice. *Br Dent J.* 2019;226(1):16–22.
- Graetz C, Mann L, Krois J, Sälzer S, Kahl M, Springer C. Comparison of periodontitis patients' classification in the 2018 versus 1999 classification. J Clin Periodontol. 2019;46(9):908–17.
- Murakami S, Mealey BL, Mariotti A, Chapple ILC. Dental plaqueinduced gingival conditions. J Clin Periodontol. 2018;45(20):28–35.
- Abbott P. Classification, diagnosis and clinical manifestations of oral diseases. *Endodontitis Topical*. 2004;9(2):899–904.
- Dietrich T, Ower P, Tank M, West NX, Walter C, Needleman I, et al. Periodontal diagnosis in the context of the 2017 classification system of periodontal diseases and conditions – implementation in clinical practice. Br Dent J. 2019;226(1):16–22.
- Herrera D, Retamal-Valdes B, Alonso B, Feres M. Acute periodontal lesions (periodontal abscesses and necrotizing periodontal diseases) and endo-periodontal lesions. *J Periodontol*. 2018;89(1):S85–S102.
- Gher M, Vermino AR. Root morphology- Clinical signifiance in pathogenesis and treatment of dental plaque gingivitis. *J Am Dent Assoc.* 1990;12:36–41.
- Tonetti MS, Sanz M. Implementation of the new classification of periodontal diseases: Decision-making algorithms for clinical practice and education. J Clin Periodontol. 2019;46(4):398–405.
- Rajeshwari HR, Dhamecha D, Jagwani S, Rao M, Jadhav K, Shaikh S, et al. Local drug delivery systems in the management of periodontitis: A scientific review. *J Controlled Release*. 2019;307:393–409.
- Susin C, Haas AN, Albandar JM. Epidemiology and demographics of aggressive periodontitis. *Periodontol.* 2000;29(1):70–8.
- Petersen PE, Ogawa H. The global burden of periodontal disease: Towards integration with chronic disease prevention and control. Periodontol. 2000;60(1):15–39.
- Eke PI, Dye BA, Wei L, Thornton-Evans GO, Genco RJ. Prevalence of Periodontitis in Adults in the United States: 2009 and 2010. *J Dent Res*. 2012;91(10):1449–54.
- Slots J, Neslon M. Systemic Diseases: facts, fallacies and the future. J Sci Res Dev. 2017;13(2):134–9.
- Dowsett SA, Archila L, Segreto VA, Eckert GJ, Kowolik MJ. Periodontal disease status of an indigenous population of Guatemala, Central America. J Clin Periodontol. 2001;72:1790–1800.
- 25. Anusaksathien O, Sukboon A, Sitthiphong P, Teanpaisan R. Distribution of Interleukin- 1β +3954and IL- 1α -889Genetic Variations in a Thai Population Group. *J Periodontol*. 2003;74(12):1796–1802.
- Sundaram NS, Narendar R, Dineshkumar P, Ramesh SB, Gokulanathan S. Evaluation of oral health related quality of life in patient with mild periodontitis among young male population of Namakkal district. *J Pharm Bioallied Sci.* 2013;5(5):30–2.
- Rao S, Balaji SK, Lavu V. Chronic periodontitis prevalence and the inflammatory burden in a sample population from South India. *Indian J Dent Res.* 2018;29(2):254–9.
- Genco RJ, Borgnakke WS. Risk factors for periodontal disease. Periodontol. 2000;62(1):1049–52.
- Drago C. Treatment of Edentulous Patients with Immediate Occlusal Loading, Implant Restorations; 2020.
- Taylor GW, Borgnakke WS. Periodontal disease: associations with diabetes, glycemic control and complications. *Oral Diseases*. 2008;14(3):191–6.
- Wadia R, Chapple I. Periodontal care in general practice: 20 important FAQs - Part two. BDJ Team. 2020;7(1):26–32.

- Kato T, Fujiwara N, Kuraji R, Numabe Y. Relationship between periodontal parameters and non-vital pulp in dental clinic patients: a cross-sectional study. BMC Oral Health. 2020;20(1):125–53.
- EKE PI, Borgnakke WS, Albandar JM. Measurement and Distribution of Periodontal diseases. In: Burt and Eklund's Dentistry. Dental Practice, and the Community; 2020. p. 171–88.
- 34. Berglundh T, Donati M. Aspects of adaptive host response in periodontitis. *J Clin Periodontol*. 2005;32(s6):87–107.
- Hajishengallis G. Periodontitis: from microbial immune subversion to systemic inflammation. Nat Rev Immunol. 2015;15(1):30–44.
- Jepsen S, Suvan J, Deschner J. The association of Periodontal diseases with metabolic syndrome and obesity. *Periodontol*. 2000;83(1):3–12.
- Ji S, Choi Y. Microbial and Host Factors That Affect Bacterial Invasion of the Gingiva. J Dent Res. 2020;12:19–62.
- Costalonga M, Herzberg MC. The oral microbiome and the immunobiology of periodontal disease and caries. *Immunol Lett*. 2014;162(2):22–38.
- Meyle J, Dommisch H, Groeger S, Giacaman RA, Costalonga M, Herzberg M. The innate host response in caries and periodontitis. J Clin Periodontol. 2017;44(12):1215–25.
- Gu Y, Han X. Toll-Like Receptor Signalling and Immune Regulatory Lymphocyte in Periodontal Disease. *Int J Mol Sci.* 2020;21(9):3329– 34.
- Chapple ILC, der Weijden FV, Doerfer C, Herrera D, Shapira L, Polak D, et al. Primary prevention of periodontitis: managing gingivitis. J Clin Periodontol. 2015;42(16):S71–5.
- Carra MC, Gueguen A, Thomas F, Pannier B, Caligiuri G, Steg PG, et al. Self-report assessment of severe periodontitis: Periodontal screening score development. J Clin Periodontol. 2018;45(7):818–31.
- Cole E, R-Chaudhuri A, Vaidyanathan M, Johnson J, Sood S. Simplified basic periodontal examination (BPE) in children and adolescents: a guide for general dental practitioners. *Dent Update*. 2014;41(4):328-7.
- Palmer RM, Floyd PD. Periodontology: A clinical approach: Periodontal examination and screening among different groups of Patients. Br Dent J. 1995;178(7):263–8.
- Oh TJ, Eber R, Wang HL. Periodontal diseases in the child and adolescent. J Clin Periodontol. 2002;29(5):400–10.
- Johnson TM, Worthington HV, Clarkson JE, Pericic TP, Sambunjak D, Imai P. Mechanical interdental cleaning for preventing and controlling periodontal diseases and dental caries (Protocol). *Cochrane Database* Syst Rev. 2015;12:21–9.
- 47. Fine DH, Patil AG, Loos BG. Classification and diagnosis of aggressive periodontitis. *J Clin Periodontol*. 2018;45(20):S95–S111.
- Laine ML, Crielaard W, Loos BG. Genetic susceptibility to periodontitis. *Periodontol*. 2000;58(1):37–68.
- Nibali L, D'Aiuto F, Donos N, Griffiths GS, Parkar M, Tonetti MS, et al. Association between periodontitis and common variants in the promoter of the interleukin-6 gene. Cytokine. 2009;45(1):50–4.
- 50. Hine MK. The use of the toothbrush in the treatment of periodontitis. *J Am Dent Assoc.* 1950;41(2):158–68.
- 51. Erbe C, Klees V, Braunbeck F, Ferrari-Peron P, Ccahuana-Vasquez RA, Timm H, et al. Comparative assessment of plaque removal and motivation between a manual toothbrush and an interactive power toothbrush in adolescents with fixed orthodontic appliances: A single-center, examiner-blind randomized controlled trial. Am J Orthod Dentofac Orthop. 2019;155(4):462–72.
- Efstratiou M, Papaioannou W, Nakou M, Ktenas E, Vrotsos IA, Panis V. Contamination of a toothbrush with antibacterial properties by oral microorganisms. *J Dent*. 2007;35(4):331–7.
- Sanz I, Alonso B, Carasol M, Herrera D, Sanz M. J Evid Based Dent Pract. 2012;47(3):18–26.
- Cleland WP. Nonsurgical periodontal therapy. Clin Tech Small Anim Pract. 2000;15(4):221–5.
- Friedman S, Abitbol S, Lawrence H. Treatment Outcome in Endodontics: The Toronto Study. Phase 1: Initial Treatment. *J Endod*. 2003;29(12):787–93.
- Jain N, Jain GK, Javed S, Iqbal Z, Talegaonkar S, Ahmad FJ, et al. Recent approaches for the treatment of periodontitis. *Drug Discovery*

- Today. 2008;13(21-22):932-43.
- 57. Cobb CM. Lasers and the treatment of periodontitis: the essence and the noise. *Periodontology*, 2000;75(1):205–295.
- 58. Nowzari H. Aesthetic osseous surgery in the treatment of periodontitis. *Periodontol* 2000. 2001;27(1):8–28.
- Rohlin M, Susanna A, Ekman A, Klinge B, Larsson G. Chronic Periodontitis -Prevention, Diagnos and Treatment - A systematic review. SBU Syst Rev Summ. 2004;31:239–52.
- Durham J, Fraser HM, McCracken GI, Stone KM, John MT, Preshaw PM. Impact of periodontitis on oral health-related quality of life. *J Dent*. 2013;41(4):370–6.
- 61. Dyke TEV. The Management of Inflammation in Periodontal Disease. *J Periodontol*. 2008;79(8s):1601–8.
- 62. Farzaneh M, Abitbol S, Friedman S. Treatment outcome in endodontics: The Toronto study. Phases I and II: Orthograde retreatment. *J Endod*. 2004;32:493–503.

 Siqueira JF, Rôças IN, Ricucci D, Hülsmann M. Causes and management of post-treatment apical periodontitis. Br Dent J. 2014;216(6):305–12.

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