Epidemiology of Periodontal Disease- A biomolecular review

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ABSTRACT

Introduction: Periodontitis is one of the most common chronic oral disease throughout the world. It is also one of the major cause contributing towards tooth loss in India. Periodontitis is an inflammatory condition of the periodontium characterized by attachment loss, alveolar bone loss, recession and pocket formation. Clinical attachment loss is a hallmark of periodontitis. Periodontal disease progression Model: The continuous disease progression model was later replaced by Burst theory. Classification of periodontal diseases: American Academy of Periodontics' classification of 1999 is widely accepted and is used for most of the academic and research purposes. Etio-pathogenisis: The inflammatory process includes the stimulation of fibroblasts by IL-1 and the secretion of matrix metalloproteinases (MMPs of which collagen is the most prominent) by polymorphonuclear neutrophils. MMPs are responsible for increased collagen breakdown, activation of cytokines and chemokines, exacerbating the destructive process. Etiology of periodontal disearse: The etiologic factors are divided into local & systemic. Important local factors contributing are plaque, calculus, malocclusion, faulty restorations. Main systemic factors are nutritional deficiencies, physical & mental disabilities, metabolic, endocrine disorders & genetic disorders. Epidemiology of Periodontal Disease: it is studied under agent, host & environmental factors. The important agent factors are bacterias such as A.actinomycetamcomitans, P.gingivalis,T. Host Factors responsible are age, gender, education, occupation , income & genetics. Main environmental factors are saliva, urbanization, lifestyle factors. Conclusion: Periodontitis is very old to mankind. It has affected all the races in the world with India having a high prevalence rate. Socioeconomic status should be considered and people should be made aware about the oral health care.

Keywords: Periodontitis, clinical attachment loss, epidemiology, periodontal disease

Search Strategy: Medline, Pubmed, Ebsco, Google Scholar were searched using the following terms in different combinations. Periodontal Disease, Periodontitis, etiology, epidemiology.

INTRODUCTION

CLINICAL RELEVANCE TO INTERDISCIPLINARY DENTISTRY:

- The article explains in detail all the associated factors with periodontitis on molecular level and comprehensive review on epidemiology of periodontitis.
- The article would help correlate the given concepts of periodontology with that while understanding clinical lesions such as pockets, loss of attachment
- The concepts are reviewed keeping abreast with all the factors associated with periodontitis , its causes and epidemiology bringing them together in one article.

Periodontitis is one of the most common chronic oral disease throughout the world. It is also one of the major cause contributing towards tooth loss in India. Periodontitis progresses in cyclical phases of exacerbation, remission and latency, a phenomenon that is closely linked to the effectiveness of the host immune response.[1] Periodontitis accumulates over the years in an individual thus exhibited at an elderly age. Therefore, certain indicator age groups are identified by World Health Organization for inter-country comparisons and to assess the impact of oral health systems on periodontal health. The essential age groups comprise15 to 19, 35 to 44, and 65 to 74 year olds.[2] .Periodontitis is a debilitating condition as pain, discomfort and general malaise are the associated features. Periodontitis is an inflammatory condition of the periodontium characterized by attachment loss, alveolar bone loss, recession and pocket formation. Clinical attachment loss is a hallmark of periodontitis. The periodontium consists of gingiva, periodontal fibres, cementum and alveolar bone.[3]Junctional epithelium is a cuff-like band of stratified squamous epithelium that is continuous with the sulcular epithelium and completely encircles the tooth. It is non keratinized, upto 15-20 cells in thickness and 0.25-1.35 mm in length.[4] Although a lot of information on periodontitis is available on web but the purpose of this review was to compile studies on periodontitis related to all its epidemiologic factors.

Methods: Medline, Pubmed, Ebsco, Google Scholar were used to search articles.

Periodontal Disease Progression Model:

a. Burst theory: By Goodson et al 1982 & Socransky 1984: It states that periodontal tissue apparently has the capacity to repair them. Periodontitis progresses in a series of relatively short acute burst of rapid tissue destruction followed by some tissue repair and long periods of remission.[5]

b. Continuous progressive model: Belief that gingivitis once developed will progress into the periodontium leading to loss of attachment, bone loss & eventually loss of tooth.[6]

c. Epidemiologic wheel: It views that development of host as intertwined with the environment & it recognizes that the host develops from a genetic core that is modifiable to varying degree by the biologic physical & social environment to which host is exposed. [6]

Implant dental restoration has become a permanent and promising result for the management of restoring missing natural dentition in routine clinical practice. Dental implants are reported to have high long term cumulative mean implant survival and success rates of 94.6% ± 6% and 89.7% ± 10.2% after mean post functional loading periods of 13.4 years and 15.7 years, respectively.13 Thus, it has been reported that the annual global dental implant market estimated at around 12-18 million implants sold. In Europe alone, the annual market has been estimated at 5.5-6 million. The number of implants placed per year has increased exponentially and will probably continue to rise as treatment protocols become more predictable and successful over time. Despite of high success in survival rates of dental implants, failures do occur and implant-supported prosthesis may require a substantial periodontal and prosthodontic maintenance over time. . However, there are various factors, which has positive (peri-implant health) and negative effect (peri-implant diseases) on peri-implant tissues.

Classification of periodontal diseases:

The term scurvy was coined by Fauchard in 1723 for the periodontal diseases. In 1920 Gottleib classified periodontal diseases into four groups: Schmutz-pyorrhoae, alveolar atrophy or diffuse atrophy, paradental-pyorrhoae and occlusal trauma. American Academy of Periodontology (AAP) in 1986 classified periodontitis into juvenile periodontitis, pre-pubertal, local juvenile, generalized juvenile, adult periodontitis, Acute Necrotizing Ulcerative Gingivitis/Periodontitis and refractory

periodontitis.AAP in 1989 added a new category of periodontitis with systemic diseases. AAP classification of 1999 is widely accepted and is used for most of the academic and research purposes.[7]

Etiopathogenisis:

The inflammatory process includes the stimulation of fibroblasts by IL-1 and the secretion of matrix metalloproteinases (MMPs of which collagen is the most prominent) by polymorphonuclear neutrophils. MMPs are responsible for increased collagen breakdown, activation of cytokines and chemokines, exacerbating the destructive process. Collagen production is inhibited by the reduced activity of fibroblasts in response to TNF-α. TNF-α is responsible for increased osteoclastic activity resulting in bone resorption. The lymphocytes release antibodies as protective mechanisms but also activate the osteoclasts, resulting in bone loss. T-lymphocytes secrete receptor activator of nuclear factor kappa-B ligand (RANKL) which is involved in osteoclast activity and therefore bone resorption. These destructive inflammatory mediators are inhibited by the secretion of osteoprotegerin and tissue inhibitors of metalloproteinases (TIMPs) [8],[9],[10]

Measuremment of Periodontitis:

Basic clinical measures for periodontitis are clinical attachment loss and probing depth. The clinical attachment loss is a diagnostic gold standard for periodontitis.[11]

Local Etiological Factors for Periodontitis:

1. Plaque & Calculus: Plaque harbors specific microorganisms responsible for periodontal diseases. The nutrient to the microorganisms attached to tooth surface through plaque is supplied through the fluid filled channels surrounding plaque thus rendering plaque pathogenic. The mineralization of plaque results in formation of calculus which is present subgingivally as well as supragingivally and is a plaque retentive factor. The calculus attaches itself snugly to the cemental irregularities and itself being rough harbors plaque. There is no epidemiologic relationship between plaque and periodontitis but the amount of plaque mass retained over it for a long duration which is pathogenic for periodontitis.[12]

2. Food Impaction: Food impaction is the process of wedging food between teeth especially in the proximal surface. The food particles thus impacted impinge onto the periodontal tissues such as gingiva and acts as agent in the development of periodontitis. The food impaction depending on direction is either vertical or horizontal. Plunger cusp are those cusps which forcefully wedge food between teeth.[3]

3. Overhanging Margins of Restorations: After the restoration is complete the clinician must check for overhanging margins of restorations, which makes the filling bulbous and the subgingival margin which rests on the gingiva or may impinge onto it and cause pain, gingival inflammation, bleeding gums or even formation of a pocket and reduced bone height. The surface of the restorations must be polished so that it does not encourage plaque retention. Some of the restorations have corrosive products which injure or destruct the tissue.[13]

4. Traumatic Occlusion: Parafunctional habits such as clenching, grinding, foreign body chewing habits such as pencil, bobby pin creates traumatogenic forces which create occlusal stresses exceeding physiologic limit which leads to cemental tear, resorption of roots, bone resorption , widening of periodontal ligament and tooth mobility. According to Carranza there is no relation between bruxism and periodontitis thus there is no established relationship between traumatic occlusion and periodontitis.[3]

5. Design Of Removable Partial Dentures: The removable partial dentures or orthodontic appliances if designed faulty can exert unnecessary pressure and forces onto the periodontium and may cause continuous irritation to underlying tissue and sometimes act as an area for food lodgement and nidus for bacterial growth thus producing periodontitis.[14]

6. Malocclusion: Malalignment such as crowding results in improper tooth brushing and retention of plaque and formation of calculus. Deepened interdental cols and lack of attached gingiva width, grooves on palatal aspect of incisors, enamel pearls, accentuated approximal concavities, poor relationship between arches predisposes towards periodontitis.[14],[15]

7. Extraction of impacted third molars: Deep impacted mandibular third molars when removed may lead to periodontal defects at the distal end of adjacent second molar. In a study done by Sammartino G et al the best treatment of reducing second molar periodontal defect bovine porous bone mineral (BPBM) with collagen membrane(CM) showed best outcome when compared with BPBM alone.[16] ,[17]

8. Habits and self inflicted injuries: Self inflicted oral mutilations is defined as deliberate harm to one's own body without suicidal intention. Stewart and

Kernohan suggested a classification system for self inflicted gingival injuries.

1. Type A: injuries are superimposed on a pre existing condition , such as herpetic lesions or localized gingival infection. [17]

2. Type B: injuries are secondary to established habits, such as finger sucking or nail biting

3. Type C: injuries have unknown or complex etiologies. These would include injuries due to psychological problems. The etiology of self inflicted injuries includes emotional disturbances, anxiety and stress. [3],[4]

10. Tooth brush trauma: excessive and over enthusiastic toothbrushing leads to abrasion and gingival recession. [3]

12. Radiation Therapy: Intense/high dose of radiations disturbs normal alveolar pattern. Periodontal attachment loss and tooth loss are greater in cancer patients treated with high dose unilateral radiation as compared to non radiated control side of dentition. Radiation treatment results in mucositis. Salivary production is permanently impaired. Xerostomia results in greater plaque accumulation and reduced buffering capacity. [20]

13. Oral hygiene status: Poor oral hygiene is directly related to gingival and periodontal disease due to plaque accumulation. Brushing twice daily and using fluoridated toothpaste and floss is a normal regime to maintain good oral hygiene.[7],[21]

14. Tobacco users have 67% greater chances to loose teeth than non smokers. And three times more likely to get acute periodontitis. Smoking alters neutrophil chemotaxis, phagocytosis and oxidative bursts. It can also increase secretion of tumor necrosis factor alpha, prostaglandin E 2, Neutrophil collagenase and elastase in gingival crevicular fluid. [14]

15. Chemical irritation: The irritants such as tobacco in the chewable and smoking form and alcohol has a dehydrating effect on gingiva thus making it more permeable to passage of irritants such as nicotine thus lowering it's resistance and making it more susceptible to necrotizing ulcerating gingivitis.[3],[22]

Systemic Etiologic Risk Factors for Periodontitis:

1. Nutritional factors: Periodontal disease is associated with an increased production of reactive oxygen species which, if not buffered sufficiently, will cause damage to host tissues and cells. Antioxidant nutrients such as ascorbic acid (Vitamin C), beta carotene, Alpha Tocopherol(vitamin E) are important buffers of reactive oxygen species which are found in fruits, vegetables, grain and seeds. Severe Vitamin C deficiency may cause scurvy- related periodontitis. [20],[23]

2. Physical & Mental disabilities: Physical disabilities makes a person affected with it with lower neuromotor skills and thus poor maintenance of oral hygiene and thus rendering him more prone to gingival and periodontal disease. [24]

3. Xerostomia : Xerostomia will lead to retention as well as lower clearance of food particle from mouth and reduced ability of natural oral clearance of food by the action of tongue, lips and cheeks thus increased chances of periodontitis.[25]

4. Drug induced disorders: Dilantin sodium used in treatment of epilepsy leads to gingival inflammation & enlargement. Chronic bismuth intoxication shows ulcerative gingivostomatitis.[25]

5. Metabolic conditions: Endocrine adjustments takes place during pregnancy, puberty, menopause. There is generalized tissue enlargement with discoloration. bleeding and mulberry like swelling and papillae become bulbous. There is adverse effect on periodontal structure during the course of pregnancy.[26]

6. Endocrinal disturbances: diabetes mellitus, hyperparathyroidism and hyperthyroidism. The glucose level of saliva and blood in diabetic patients is more than normal which has the potential to alter the microflora towards more pathogenic bacterias. Due to polymorphonuclear leucocytes deficiencies in diabetes which results in impaired chemotaxis, defective phagocytosis thus making the diabetic individual more susceptible to periodontal infection. Most of the studies show statistically significant relationship between severe gingival inflammation and loss of attachment in diabetic group as compared to normal individuals which is more when there is increase in glucose level. Periodontal disease in diabetics presents with very severe gingival inflammation, deep periodontal pockets, rapid bone loss, frequent periodontal abscesses, greater loss of attachment, increased bleeding on probing and increased tooth mobility.[27]

1,25(OH)2D3 has an important role in maintaining calcium and phosphate level in blood. Its deficiency leads to increased secretion of parathyroid hormone leading to rickets and osteomalacia. In Mice, vitamin D receptor (VDR)-mediated induction of osteoblast

RANKL may account for enhanced bone resorption. Osteoporosis results in lowered density in jaw bones leading to altered trabecular bone pattern and rapid alveolar bone resorption followed by invasion of periodontal pathogens. Periodontal infection increases the systemic release of proinflammatory cytokines, which accelerate systemic bone resorption. Hypopitutarism exhibits with crowding of teeth, enlarged gingiva, delayed resorption of deciduous teeth, delayed formation and eruption of teeth. Hyperpitutarism exhibits spacing of lower teeth-jaw size. Thus people with poor metabolic control have higher frequency of gingivitis and periodontitis.[28]

7. Hematologic disorders: There is occurrence of hemorrhagic gingival overgrowth with or without necrosis as a common early manifestation of acute leukemia. Chemotherapy associated with bone marrow transplantation also adversely affect gingival health. In diseases affecting leukocytes such as agranulocytosis, cyclical neutropenia, Chediak Higashi Syndrome and congenital neutropenia severe alveolar bone loss has been reported with earlier loss of teeth. [29]

8. Immune system disorder: leukocyte disorder, antibody disorders, HIV infection, persons on immunosuppressive drugs: Bacterial infections acts as a stimulus for systemic acute phase response resulting in increased production of acute phase proteins like C Reactive protein(CRP), macroglobulin and Serum amyloid. The bacterial endotoxins also stimulate the local host inflammatory mediators which finally results in serum antibody response to bacteria. With the onset of periodontitis the host immune response comes into action. It plays an important role in the overall activity. It includes the activation of leukocytes, neutrophils, T-lymphocytes and plasma cells. Release of antibodies, lipopolysaccharides and chemical inflammatory mediators that include cytokienes, chemokines and C-reactive proteins. The most powerful stimulant for host cell response are lipopolysaccharides which are present on gram negative bacterial cell walls. Initially because of the increased presence of neutophils, cytokienes are released by neutrophils and macrophages. Chemical mediators released include tumor necrosis factor alpha (TNF-α), Interleukin- 1 (IL-1) and prostaglandins. Thus if the immune system is compromised it will be a risk factor for developing periodontitis. HIV infected patients exhibit Kaposi's sarcoma, necrotizing ulcerative gingivitis, necrotizing ulcerative stomatitis, necrotizing ulcerative periodontitis which results in inflammation and enlargement of gingiva. [3]

9. Psychological or emotional factors : Stress, depression and anxiety are considered potential risk factors to affect periodontal condition as these factors are risk indicators to take up health impairing habits such as smoking, tobacco chewing, alcoholism, poor oral hygiene status and the pathophysiological factors that lead to higher glucocorticoid and catecholamine level which affects the immunological profile thus making the individual susceptible to periodontitis.[11] stress related habits such as pencil biting are detrimental to periodontal health. [30]

10. Obesity: Obesity raises the susceptibility to infection and postoperative complications . It seems that high lipid diet disrupts normal immune response to P. gingivalis. Repeated P. gingivalis bacteraemias result in repeated endothelial/macrophage invasion which causes chronic inflammatory state, exacerbates atherosclerosis and periodontitis through homotolerance induction. Tolerance induction is responsible for the role of obesity in the faster progression of periodontal diseases. [31]

Epidemiology Of Periodontal Diseases:

The epidemiology is studied under the triad of Agent, Host and Environment factors.

The Agent Factors:

1. Periodontal microorganisms: Bacterias by their periodontal pathogenicity are categorized by Socransky and Haffajee using a color classification. The orange and red complexes denoting the most pathogenic bacterias, heavy plaque harbours higher proportion of green and orange complexes and lighter plaque harboured orange, yellow and purple bacterial complexes. These bacterias are associated with gingival inflammation, recession and pocket depth. The red complex includes Tannerella forsythia, porphyromonas gingivalis, Treponema denticolae.[32]

2. Bacterias such as A.actinomycetamcomitans, P.gingivalis,T.forsythia, P.intermedia,Fusobacterium nucleatum and peptostreptococcus micros are markers of destructive disease.

3. Bacterias associated with chronic gingivitis are:

a. Predominant gram positive species: S.Sanguis,S.mitis,S.intermedius,S.oralis,A.viscosus, A.naeslundii and peptostreptococcus micros.

b. Gram negative microorganisms:

F.nucleatum,P.intermedia,V.parvula,Hemophilus,Capnocytophaga,Campylobacter spp. [9],[33]

4. Pregnancy associated gingivitis: there is increase in steroid hormones in crevicular fluid and increase in levels of P.intermedia which uses steroids as growth factor.[14]

5. Chronic periodontitis: plaque from sites of chronic periodontitis shows presence of elevated levels of spirochetes, anaerobic gram negative bacterial species. Bacteria most often cultivated include P.gingivalis, B.Forsythus, P.intermedia,C.rectus, Eikenella Corrodens,F.nucleatum,A.actinomycetamcomitans, P.micros, Treponema,Eubacterium spp. [5]

6. Bacterias associated with localized aggressive periodontitis: 90% of total microbiota are A.actinomycetamcomitans, other organisms are P.gingivalis, E.corrodens, C.rectus, F.nucleatum, B.capillus,Eubacterium Brachy, capnocytophaga spp., spirochetes including herpes viruses EBV-1 and HCMV.[5]

7. Necrotizing periodontal disease: Characterized by necrosis of marginal gingiva tissue and interdental papilla associated with pain, malodour, lymphadenopathy, fever and malaise. The microorganisms associated are P.Intermedia and spirochetes. [9]

8. Abscess of periodontium: it may occur in the absence of periodontitis due to foreign object injury or endodontic problem. The microbiota associated is F.nucleatum, P.intermedia, P.gingivalis, P.micros and B.forthysus. [9], [33]

Host Factors:

1. Age: periodontitis is a disease which is accumulated over the years and hence chronic in nature. It is rare to find the disease in young people or teenagers. It is established in the age group of 35-44 years of age group which is also chosen by WHO as a monitoring age group for established conditions such as periodontitis. [34],[35]

2. Gender: There is a male predilection for periodontitis as compared to females. The supposed reasons are neglect of oral hygiene in males, female being more concerned about looks, oral hygiene, mouth odour. Also deleterious habits such as smoking, tobacco chewing, alcoholism are more prevalent in males which deteriorates the periodontal condition. [36]

3. Education: Periodontal disease is inversely proportional to education. Higher the education level lower will be the periodontal diseases due to awareness, understanding of disease and readiness to change attitude towards taking up healthy oral habits.[37]

4. Genetic Considerations: several studies show genetic risk factors associated with periodontitis. In chronic periodontitis the phenotype or disease characteristic is evident in third decade while aggressive periodontitis presents in the first and second decade of life. It is not rare to find differences in clinical differentiation of disease such as periodontal disease and similar problems occur in medical genetics in the study of other delayed onset hereditary traits. [38]

5. Socioeconomic Status: Lower socioeconomic status is related to higher periodontal diseases. It has been found in many studies that there is better gingival and periodontal health amongst highly educated group as compared to lower education group.[37]

Environmental Factors:

1. Saliva: Several inorganic and organic factors in saliva are important for bacteria and their products in oral environment. Saliva like GCF contains antibodies that are reactive with indigenous oral bacterial species. Salivary enzymes such as hyaluronidase , lipase, beta-glucuronidase, chondroitin sulfatase are present in increased concentrations in periodontal disease. Proteolytic enzymes in the saliva are generated by both host and oral bacteria which are contributors to the initiation and progression of periodontal disease. To combat these enzymes saliva contains antiproteases that inhibit cystine proteases such as cathepsins and antileucoproteases. Saliva contains coagulation factors that hastens blood coagulation and protects wound from bacterial invasion. Saliva exerts a major role in plaque initiation, maturation and metabolism. Calculus formation, periodontal disease and caries are dependent on salivary flow. An increase in inflammatory gingival disease, rapid tooth destruction are a consequence of decrease salivary gland secretion.[39]

2. Urbanization: Slightly higher in rural areas than in urban areas due to decreased dentist : population ratio in rural areas with that lack of awareness that periodontal disease is not life threatening condition. Inaccessibility to dental products and services.[22]

3. Lifestyle Factors: harmful cultural habits such as use of tobacco, snuff , bidi. Folklore myths. Cleaning teeth with charcoal, ash, brick powder. As suggested by Abel, the lifestyle is measured by means of questions about tobacco-related habits, physical

activity, dietary habits, and alcohol consumption. When the association between the lifestyle and periodontal status was compared, the prevalence of periodontitis had decreased in healthy lifestyle compared with unhealthy lifestyle subjects.[37]

4. Fluorides: Fluorides interfere with bacterial cell adherence to tooth structure.[40]

5. Geographic Areas: Data collected 1980 onwards in WHO global oral data bank

Gingivitis and calculus deposits are more prevalent and severe in low income nations.

High – Chile, Lebanon, Jordan Thailand, Burma, Malaya, Ceylon, India & Trinidad

Intermediate – US (Blacks), Ecuador, Colombia, Ethiopia.

Low – US (White).[34]

Conclusion

Periodontitis is very old to mankind. It affects the periodontium such as gingiva, periodontal ligament, cementum and alveolar bone. Periodontitis has become a public health problem in India especially in rural area. Due to neighborhood deprivation many loose the chance to get treatment for periodontitis. There is still a gap between the needs and medical /dental facilities available to people in remote/ rural areas. Considering the socioeconomic status the people in rural areas should be made aware about oral health problems through dental camps and means to improve oral health should be explained.

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