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# Etiology and Risk factors for periodontal diseases

**Periodontal diseases;** are the most prevalent and multi-factorial diseases that involved hard and soft dental tissues, Bacterial colonization, and immune responses of the host. Periodontal inflammation like other pathologic process in the body involves the immune system with a multifactorial etiology, affecting a large population worldwide. Plaque, being the primary etiologic agent, a constitutional factor, probably of genetic origin, may play a part. Comprises a group of inflammatory conditions of the supporting tissues of the teeth most often includes gingivitis & periodontitis, they are the two basic forms of periodontal disease, and each has a variety of forms.

**Gingivitis:** It is inflammation of the gingiva in which the junctional epithelium remains attached to the tooth at its original level. .It is characterized by areas of redness and swelling, and there is a tendency for the gingiva to bleed easily. Gingivitis is limited to the epithelium and gingival connective tissues. It is important to note that there is no tissue recession or loss of connective tissue or bone. The occurrence of gingivitis is wide spread in the population. Studies have shown, the prevalence of gingivitis in adults was reported to exceed 75% and even to approach 100% in some populations. It is a reversible condition & has been extensively studied in a model system referred to as experimental gingivitis in man by Löe et al (1965).

**Periodontitis :** it is a common, chronic inflammatory disease(inflammation of the supporting tissues of the teeth( cementum, bone, periodontal ligament) leading to permanent destruction of these tissues caused by the accumulation of bacterial matrix at the gingival line . It is characterized by clinical attachment loss , periodontal pocketing & alveolar bone loss .It is irreversible disease can cause a breakdown of the periodontium resulting in loss of tissue attachment and destruction

of the alveolar bone. It is characterized by gingival tissue separation from the tooth, which forms a periodontal pocket and can lead to bone and tooth loss. In a Periodontal diseases (PD) a wide number of microorganisms play an important role in periodontal disease

**Periodontitis is the extension of the inflammatory process from the gingiva into the connective tissue and alveolar bone that supports the teeth**. The progression of periodontitis involves the destruction of connective tissue attachment at the most apical portion of a periodontal pocket. Sever periodontitis, which may result in tooth loss, is found in 5-20% of most adult populations

**Gingivitis** may progress to **periodontitis** when it is left untreated & with the presence of other contributing factors beside the essential factor (dental plaque ), back to healthy situation.

In some individuals gingivitis may not progress to periodontitis for a long period of time even if it is not treated & this condition is called **contained gingivitis** & this is depending on host response & pathogenicity of bacteria.

**Risk**: Probability that an individual will develop a specific disease in a given period of time

**Risk factors:** can be defined as characteristics or factors that when present increases the risk that an individual will get the disease. It is important to make the distinction that risk factors are associated with a disease but do not necessarily cause the disease.

Risk factors may be environmental, behavioral, or biological, that when present increase the likelihood that an individual well develop the disease.

**Risk Determinant:** should be reserved for risk factors that cannot be modified.

**Risk indicators**: are *probable* or *putative* risk factors that have been identified in cross-sectional studies but not confirmed through longitudinal studies

**Risk predictors/markers**: although associated with increased risk for disease, do not cause the disease. These factors also are identified in cross-sectional and longitudinal studies

**Categories of Risk Elements for Periodontal Disease**

**Risk Factors**

Tobacco smoking

Diabetes

Pathogenic bacteria and microbial tooth deposits

Pregnancy, puberty

Medication

Hematological Disorders

**Risk Determinants/Background Characteristics**

Genetic factors

Age

Gender

Socioeconomic status

Stress

**Risk Indicators**

Human immunodeficiency virus (HIV)/acquired immunodeficiency syndrome (AIDS)

Osteoporosis

**Risk Markers/Predictors**

Previous history of periodontal disease

Bleeding on probing

# Risk Factors for Periodontal Disease

**Tobacco Smoking :** tobacco smoking is a major risk factor for increasing the prevalence & severity of periodontal destruction. It was found that the increased risk for periodontitis in smoker was 2.5- 7 times greater than nonsmoker.

The smokers appear have less gingival inflammation and less bleeding in the gingiva may be explained by decreased gingival vascularity, which includes decreased vascular density, reduced lumen area of gingival vessels (increased vasoconstriction ).

Studies suggested that nicotin increases rate of proliferation of gingival epithelium which can contribute to the reduction of inflammatory clinical signs in the gingival tissues. These are the physiological effects of smoking on the etiology of periodontal disease. With Decreased gingival crevicular fluid flow.

.Smoking suggest an imbalance between bacterial challenge & host response which may due to changes in the composition of subgingival plaque with increase in the number & /or virulence of pathogenic organisms

The microbiological effect of smoking; in the etiology of periodontal disease include increase colonization of shallow periodontal pockets by periodontal pathogens & increase levels of periodontal pathogens in deep periodontal pocket. Smoker may have higher level of *Tannerella forsythia, P. gingivalis* and & *Treponema denticola.* It has been found that smoke derived aryl hydrocarbons and bacterial LPS may act additively to inhibit bone formation, which may explain why periodontal bone loss is greater and bone healing is less successful in smokers than non smokers with periodontal infections.

The immunological effect of smoking; 1. Nicotine causes decrease immune response and impair PMNs chamotaxis and phagocytosis.

2. Increase the production of TNF- alpha, IL-1 alpha &IL6 these immune mediators are known to lead to more sever destructive inflammation in the periodontal tissue.

3. Reduction in the serum concentration of Immunoglobulin as IgG2 which is essential in the protection against periodontal infection. Also smoking decrease the level of salivary IgA antibodies.

The risk of periodontal disease increases with the number of cigarettes smoked per day. Smoking cigars and pipes carries the same risks as smoking cigarettes. Exposure to secondhand smoke may also be associated with an increased risk for developing periodontal disease. Fortunately, when smokers quit, their periodontal health gradually recovers to a state comparable to that of nonsmokers.

**Diabetes mellitus**.

Diabetes is a modifiable factor in the sense that though it cannot be cured, it can be controlled. Studies have been done which suggest that poorly controlled diabetics

respond less successfully to periodontal therapy relative to well-controlled and non-diabetics It is an important disease from periodontal standpoint . It is a complex metabolic disease characterized by chronic hyperglycemia. Uncontrolled diabetes (chronic hyperglycemia ) is associated with many problems as reduction in the defense mechanism ( neutrophil dysfunction , Impairment of chemotaxis & phagocytosis ), atherosclerosis & reduce normal gingival blood flow, increased susceptibility to infections including periodontitis & poor wound healing . Diabetes mellitus does not cause gingivitis or periodontal pocket, but it alters the response of periodontal tissues to local factor . Diabetic patients with poor oral hygiene may have very sever gingival inflammation , deep periodontal pockets , rapid bone loss & frequent periodontal abscesses , which is an important feature of periodontal disease in diabetic patients. The mechanism responsible for increasing the risk of sever periodontal destruction in uncontrolled diabetic patients unclear but it is likely to be related an increased susceptibility to infection, an impaired immune response, poor wound healing &increase collagenase activity.

**Dental plaque and oral hygiene**

The primary etiological factor in the development & initiation of periodontal disease is dental plaque. Dental plaque is a bacterial aggregation on the teeth or other solid structures in the oral cavity.

Dental calculus which mineralized dental plaque is consider as secondary etiological factor of periodontal disease & it is covered with dental plaque so it serve as reservoir for bacterial plaque (retentive factor) .

.Oral hygiene measures are very important and can favorably influence the ecology of the microbial flora in shallow-to moderate pockets, but it does not affect host response. Oral hygiene alone has little effect on subgingival microflora in deep pockets and personal oral hygiene practices among health professionals have been shown to be unrelated to periodontitis in these individuals.

Comprehensive oral hygiene programs are effective in preventing or reducing the level of gingival inflammation in children and adults. These programs, however, may not be

viable in preventing sever periodontitis and it may be difficult to achieve a satisfactory level of oral hygiene in the general population to prevent chronic periodontitis and periodontal tissue loss effectively.

**Specific microorganisms**

Although there is sufficient evidence that accumulation and maturation of a plaque biofilm is necessary for the initiation and progression of periodontal diseases, studies show that bacterial species colonizing the gingival pocket play variable roles in the pathogenesis of these diseases and may therefore possess different levels of risk of periodontal tissue loss The subgingival microflora in periodontitis can harbor hundreds of bacterial species but only a small number has been associated with the progression of disease and considered etiologically important Of all of the various microorganisms that colonize the mouth, there are *Aggregatibacter actinomycetemcomitans ( A.a).*

*Porphyromonas gingivalis (P. gingivalis).*

*Prevotella intermedia (P. intermedia).*

*Tannerella forsythia (T.Forsythia*)

These microorganisms produce endotoxins that will cause severe periodontal destruction

**Pregnancy, puberty:**

Pregnancy associated gingivitis is inflammation of the gingival tissues associated with pregnancy . This condition is accompanied by increase in steroid hormones in crevicular fluid & increase in levels of ( *Prevotella intermedia* microorganism ) which use the steroids as growth factors . The increase in sex hormones may exaggerate the inflammatory response to dental plaque which means small amount of plaque may lead to gingivitis. Puberty is also accompanied by an exaggerated response of the gingiva to local irritation . As adulthood is approached, the severity of the gingival inflammation diminishes even when local factors persist.

**Medications**

Gingival enlargement is a well-known consequence of the administration of some drugs as anticonvulsants (Phenytoin or Dilantin ), immunosuppressants

(Cyclosporine) & Ca channel blockers (Nifedipine) . In general the overgrowth of the gingiva start as painless enlargement of the interdental papilla & extend to the marginal gingiva , then as the condition progresses, the marginal & papillary enlargements unite together & may cover the clinical crown & may interfere with the occlusion. It has been shown that the presence of dental plaque will increase the chance of development of the enlargement & plaque removal may limit the severity of the lesion , but it does not prevent the overgrowth . Also the gingival enlargement may be dose-related which mean if the physician reduce the dose of the drug without affecting the systemic condition of the patient , this may reduce the enlargement or use an alternative medication that does not cause gingival enlargement as a side effect . Other factor that may influence the gingival enlargement is the continuous use the drug for a long period of time (the duration) which may result in recurrence of the lesion even if it is treated surgically.

The first drug induced gingival enlargement is Phenytoin (Dilantin Na) which is anticonvulsant drug used in the treatment of epilepsy . Gingival enlargement occur in 50-65% of patients receiving the drug.

The other drug is Cyclosporine A which is immunosuppressive agent used to prevent organ transplant rejection. The enlargement occurs in 30% of patients receiving this drug.

Ca channel blockers are drug used for the treatment of cardio-vascular conditions such as hypertension & angina pectoris . Some of these drugs can induce gingival enlargement. Nifedipine is one of the most commonly used drug that induced the enlargement in about 20% of the patients. More recent are Amlodipine & Verapamil which also induced gingival enlargement .

**Hematological Disorders**

Hemorrhagic gingival overgrowth with or without necrosis is a common early manifestation of acute leukemia. Patients with chronic leukemia may experience

similar but less severe periodontal changes. Chemotherapy or therapy associated with bone marrow transplantation may also adversely affect the gingival health .

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**Risk Determinants/Background Characteristics for Periodontal Disease**

**Genetic factors**

There is evidence that genetic differences between individuals may explain why some patients develop periodontal disease & other do not. Genetic factors may play an important role in determining the nature of the host response & may affect the function of phagocytic immune cells or the structure of the epithelia or connective tissue . One of these diseases is **papillon - Lefevre syndrome** which is a rare hereditary disease characterized by hyperkeratotic skin lesion in the palms, soles , knees & elbows & sever destruction to the periodontium with early loss of primary & permanent teeth . Other disease is sever periodontitis which has familial aggregation (which mean seen in one family ).Some immunological defects are associated with aggressive periodontitis. Many studies have been done to demonstrate that periodontal disease was linked to genetics and they found the following:

1. A specific inteukin-1 (IL-1) genotype has been associated with sever chronic periodontitis
2. Neutrophil abnormalities are under genetic control.
3. Genetics play a role in regulating the titer of protective $IgG_2$ antibody response to *A. actinomycetemcomitans* in patients with sever form of periodontitis.

**Age**

Aging is associated with an increased incidence of periodontal disease. However it has been suggested that the increased level of periodontal destruction observed with aging is the result of cumulative destruction rather than a result of increased rates of destruction. With aging a number of changes take place in the periodontal tissues.

a. Arteriosclerosis (reduction in arterial blood supply).

b. The gingiva become more fibrotic &less keratinized.

c. The periodontal fiber bundles become thicker with decrease in cellularity.

d. Osteoporosis of alveolar bone.

From these changes, it was concluded that inflammation develops more rapidly & wound healing proceeds more slowly in old than young individuals with the same susceptibility to periodontal disease . Young persons have better chance of repair than old persons, but when young person was susceptible to have periodontal disease more than old person which was non susceptible, then the opposite is true & age has no influence in this condition . for ex . Rapidly destructive forms of periodontal disease occur in young are usually associated with defect in host response (deficient neutrophil function) while the elderly have slowly progressive form of hil function.

Epidemiological studies had shown that the prevalence & severity of periodontal disease increase with age & a high incidence of Periodontal disease was found after the age of 40 years . Age by itself has no influence on the periodontal tissues, but the older the age , the longer the time interval & the more the chance of periodontal tissues to be exposed to local factor (accumulative effect of age) , also there is declined host defense mechanism & high incidence of systemic diseases & drug intake which may adversely affect the periodontal health.

It has been demonstrated that the mean annual rate of bone loss among the initially 70 years old subjects was 0.28 mm compared to 0.07 on the 25 years old individual.

**gender**

Numerous studies reported higher periodontal destruction among males compared to the female population .The reasons for these sex differences are not clear, but they are thought to be related to the ignorance of oral hygiene, which is usually observed among males . However, the relationship observed between sex and the disease is not apparent and is not considered as strong and consistent. Thus, sex may be a demographic factor, which may interfere with the effects of other factors and it must be controlled for investigating the disease.

**Socioeconomic status (SES )**

Multitudes of disease conditions are associated with socioeconomic status, and cause/effect is plausible. Generally, those who are better educated, wealthier, and live in more desirable circumstances enjoy better health status than the less educated and poorer segments of society. Periodontal diseases are no different and have been related to lower SES. The ill effects of living in deprived circumstances can start early in life. Gingivitis and poor oral hygiene are clearly related to lower SES, This can be

attributed to decreased dental awareness and decreased frequency of dental visits compared with more educated individuals of higher SES. But the relationship between periodontitis and SES is less direct

**Psychological stress**

Studies have demonstrated that individuals under psychological stress are more likely to develop clinical attachment loss and loss of alveolar bone

One possible link in this regard may be increased glucocorticoid secretion that can depress immune function increased insulin resistance, increases in production of IL-6 in response to increased psychological stress and potentially increased risk of periodontitis. Another study suggests that host response to P. gingivalis infection may be compromised in psychologically stressed individuals also the relationship is simply due to the fact that individuals under stress are less likely to perform regular good oral hygiene and prophylaxis

**Risk Indicators for Periodontal Disease**

**HIV \ AIDS:**

It has been stated that the immune dysfunction ( immunosuppression) associated with human immunodeficiency virus (HIV) infection & acquired immunodeficiency syndrome (AIDS) increases susceptibility to periodontal disease .Those patients often had severe periodontal destruction characterized by necrotizing ulcerative periodontitis

**Osteoporosis**

Many of the studies conducted to date suggest there is a relationship between skeletal osteoporosis and bone loss to the extent that postmenopausal osteoporosis may result in dental osteopenia involving the jaws, and particularly the mandible. Osteoporosis was significantly associated with severe alveolar crestal bone loss and the prevalence of periodontitis cases in postmenopausal women. During menopause, estrogen deficiency will reduce bone mineral density, some women may develop menopausal gingivostomatitis

# Risk Markers/Predictors for

# Periodontal Disease

## Previous History of Periodontal Disease

A history of previous periodontal disease is a good clinical predictor of risk for future disease. Patients with the most severe existing loss of attachment are at the greatest risk for future loss of attachment. Conversely, patients currently free of periodontitis have a decreased risk for developing loss of attachment compared with those who currently have periodontitis

## Bleeding on Probing

Bleeding on probing is the best clinical indicator of gingival inflammation. bleeding on probing coupled with increasing pocket depth may serve as an excellent predictor for future loss of attachment. Lack of bleeding on probing does appear to serve as an excellent indicator of periodontal health.

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**The local anatomic risk factors**: include

1-**Furcation anatomy**: the entrance of bifurcations or trifurcations is restricted enough to limit the access for mechanical root instrumentation. Also the presence of concavities in the furcal aspects of molar roots will limit instrumentation as well.

2- **Cervical enamel projections** (CEP) : These are tooth developmental deformities of the CEJ found on molars . The enamel is projected toward the entrance of the furcation & this projection may responsible for furcation invasion & localized severe bone loss around the tooth.

3-**Palatogingival grooves** (PGG) : These are tooth developmental deformities of maxillary central & lateral incisors . They begin in lingual pits & extend vertically onto root surfaces & may extend to the root apex & are associated with increased gingival inflammation & plaque accumulation.

4- **Root Morphology***:* The mesial root surface of the maxillary first premolar presents with a pronounced concavity which may not be accessable to oral hygiene procedures or professional instrumentation.

The **iatrogenic risk factors (faulty dentistry)** include many examples as :

* Overhang margins of proximal restorations.

* Open or loose contacts of crowns & fillings.

* Poorly designed or fitted prosthesis ….ex . Lingual bar is better than full coverage to keep the cleansing action of saliva & massage from tongue.