Risk Factors for Periodontal Diseases

Risk: is the 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.

Systemic risk factors: may be modifiable or non-modifiable:

1. Modifiable risk factors: are usually environmental or behavioral in nature.

2. Non modifiable risk factors: are usually intrinsic to the individual and therefore not easily changed. They are also known as determinants.

❖ Modifiable risk factors:

Tobacco Smoking :

Is a well-established risk factor for periodontitis, a direct relationship exists between smoking and the prevalence of periodontal disease. This association is independent of other factors such as oral hygiene or age. Many studies comparing the response to periodontal therapy in smokers, previous smokers, and nonsmokers have shown that smoking has a negative impact on the response to therapy. However, former smokers respond similarly to 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.

- **Psychological factors :**

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 the 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.

Obesity :

Obesity is one of the most significant health risks of modern society, and is now recognized as a major health concern in both developed and developing countries. Conditions associated with obesity, e.g. ‘‘the metabolic syndrome’’, a clustering of dyslipidemia and insulin resistance may exacerbate periodontitis. Obesity has been postulated to reduce blood flow to the periodontal tissues & promoting the development of periodontal disease. Furthermore, obesity may enhance immunological or inflammatory disorders, which might be the reason, also obese subjects tend to exhibit escalating poor periodontal status relative to non-obese individuals.

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 not 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 indirect.

Pregnancy, puberty:

Pregnancy associated gingivitis is an 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 means, 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 also are drug used for treatment of cardio-vascular conditions such as hypertension & angina pectoris, some of these drugs can induce gingival enlargement such as

(Nifedipine) which 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 .

❖ Non-modifiable risk factors :

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.

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 and sever destruction to the periodontium with early loss of primary & permanent teeth . 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 severe 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 severe periodontitis.

Aging :

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 as the following:

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 in 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 e.g. 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 the disease that does not result from impaired neutrophil 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, gender may be a demographic factor, which may interfere with the effects of other factors and it must be controlled for investigating the disease.

- **Ethnicity :**

The level of attachment loss is also influenced by race/ethnicity, although the exact role of this factor is not fully understood. Certain racial/ethnic groups, particularly subjects of African and Latin American background, have a higher risk of developing periodontal tissue loss than other groups.

Human Immunodeficiency Virus (HIV) /Aquired Immunodeficiency Syndrome (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 that 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.

❖ The local anatomic risk factors:

There are local contributing risk factors to the etiology of periodontal diseases including (**Anatomic &Iatrogenic)** factors. These factors may facilitate bacterial plaque accumulation & retention or they interfere with plaque removal (Retentive factors) which include the following:

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 and 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 accessible to oral hygiene procedures or professional instrumentation.

❖ Local predisposing factors :

Biofilm and Calculus :

The microbial challenge presented by bacterial biofilm and calculus is the most important local factor in periodontal diseases. Therefore, in most cases, having a favorable prognosis depends on the ability of the patient and the clinician to remove these etiologic factors. Calculus by itself does not contribute directly to gingival inflammation. Like other retentive factors such as open crown margin or an overhanging restoration, calculus retains dental plaque, which contributes to gingival inflammation.

Iatrogenic Factors :

Deficiencies in the quality of dental restorations or prostheses are contributing factors to gingival inflammation and periodontal destruction. Inadequate dental procedures that contribute to the deterioration of the periodontal tissues are referred to

as iatrogenic factors. Iatrogenic endodontic complications that can adversely affect the periodontium include root perforations, vertical root fractures and endodontic failures that may necessitate tooth extraction. Characteristics of dental restorations and removable partial dentures that are important to the maintenance of periodontal health include the location of the gingival margin for the restoration, the space between the margin of the restoration and the unprepared tooth, the contour of the restorations, the occlusion, materials used in the restoration, the restorative procedure itself and the design of the removable partial denture. These characteristics are related to the etiology of periodontal disease.

Margins of restorations (overhanging margins of dental restorations) :

Contribute to the development of periodontal disease by (1) changing the ecologic balance of the gingival sulcus to an area that favors the growth of disease-associated organisms (predominantly gram-negative anaerobic species) at the expense of the health-associated organisms (predominantly gram-positive facultative species) and (2) inhibiting the patient's access to remove accumulated plaque.

The frequency of overhanging margins on proximal restorations has varied in different studies from 16.5% to 75%. Noted that persons with overhanging posterior restorations had an average of 0.22 mm reduced alveolar bone support adjacent to the surfaces with overhangs. The removal of overhangs allows for the more effective control of plaque, thereby resulting in a reduction of gingival inflammation and a little increase in radiographic alveolar bone support. The location of the gingival margin of a restoration is directly related to the health status of the adjacent periodontal tissues. Numerous studies have shown a positive correlation between restoration margins located apical to the marginal gingiva and the presence of gingival inflammation. Subgingival margins are associated with large amounts of plaque, more severe

gingivitis, deeper pockets, and a change in the composition of the subgingival microflora that closely resembles the microflora noted in chronic periodontitis.

Other predisposing factors are iatrogenic factors, which are deficiencies in the quality of dental restorations or prostheses & are contributing factors to gingival inflammation and periodontal destruction. Inadequate dental procedures that contribute to the deterioration of the periodontal tissues are referred to as iatrogenic factors. Characteristics of dental restorations and removable partial dentures that are important to the maintenance of periodontal health include the location of the gingival margin for the restoration, the space between the margin of the restoration and the unprepared tooth, the contour of the restorations, the occlusion, the materials used in the restoration, the restorative procedure itself, and the design of the removable partial denture. These characteristics are described in this chapter as they relate to the etiology of periodontal disease. A more comprehensive review with special emphasis on the interrelationship between restorative procedures and the periodontal health

Contours and open contacts, over contoured crowns and restorations tend to accumulate plaque and handicap oral hygiene measures in addition to possibly preventing the self-cleaning mechanisms of the adjacent cheek, lips, and tongue. Restorations that fail to reestablish adequate interproximal embrasure spaces are associated with papillary inflammation. Pontics in fixed bridges should barely touch the mucosa. Access for oral hygiene is inhibited with excessive pontic-to-tissue contact, thereby contributing to plaque accumulation that will cause gingival inflammation and possibly the formation of pseudopockets.

- **Malocclusion**:

The irregular alignment of teeth as found in cases of malocclusion may facilitate plaque accumulation and make plaque control more difficult.

- ✓ Periodontal complications associated with orthodontic therapy:

Orthodontic therapy may affect the periodontium by favoring plaque retention, by directly injuring the gingiva as a result of overextended bands and by creating excessive forces, unfavorable forces, or both on the tooth and its supporting structures. Orthodontic appliances tend to retain bacterial plaque and food debris, thereby resulting in gingivitis and they are also capable of modifying the gingival ecosystem. An increase in *Prevotella melaninogenica, Prevotella intermedia*, and *Actinomyces odontolyticus* and a decrease in the proportion of facultative microorganisms was detected in the gingival sulcus after the placement of orthodontic bands. More recently, *Aggregatibacter actinomycetemcomitans* was found in at least one site in 85% of children who were wearing orthodontic appliances. By contrast, only 15% of unbanded control subjects were positive for *A. actinomycetemcomitans*.

Categories of Risk Elements for Periodontal Disease

Risk Factors:

- **Tobacco smoking**
- **Diabetes**
- **Pathogenic bacteria and microbial tooth deposits**

Risk Determinants/Background Characteristics:

- **Genetic factors**
- **Age**
- **Gender**
- **Socioeconomic status Stress**

Risk Indicators:

- **Human immunodeficiency virus (HIV)/acquired immunodeficiency syndrome (AIDS)**
- **Osteoporosis**
- **Infrequent dental visits**

Risk Markers/Predictors:

- **Previous history of periodontal disease**
- **Bleeding on probing**