A Review on Predisposing and Modifying Factors of Periodontal Disease
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ABSTRACT
A comprehensive periodontal assessment incorporated a thorough evaluation of data from patient’s interviews, present and past medical and dental history, clinical periodontal examination, radiographic examination and laboratory tests. Periodontal assessment can potentially serve as an identification of sites or subjects that have increased risk of experiencing the progression of periodontal disease. Periodontist routinely evaluate the findings from a periodontal examination and, in combination with patient’s dental and medical histories, to decide on what seems to be a logical treatment approach. Thus the knowledge of the healthy clinical appearance, natural history and pathogenesis of periodontal disease should be applied during the periodontal assessment. These will then be a basis for comprehensive treatment planning and understanding of the patient’s needs, social and economic situation. So, the present review, will evaluate the above mentioned aspect by relating to its predisposing and modifying factors of periodontal disease and the mechanism involves.

Keywords: periodontal disease, predisposing factors, modifying factors.

Introduction
The assessment of the periodontal status of a patient should consist of a thorough history and examination. This will provide the clinician detailed information regarding disease susceptibility, modifying factors and predisposing factors that may be present as well as the current extent and severity of disease. A good history taking can reveal lifestyle risk factors, health and genetic that has the potential to modify patient's response to the presence of dental biofilm (AL-Bayaty et al 2010). This is important because periodontal tissue destruction in periodontitis fundamentally results from the body's response to plaque rather that the plaque itself (Page and Schroeder, 1976). Through clinical and radiographic examination, factors that predispose the formation and retention of dental plaque can be revealed. The assessment of a periodontal status is critically important to assist the clinician with the diagnosis and developing a specific treatment plans for an individual tooth that going to be treated. A comprehensive periodontal assessment involves a thorough evaluation of the data that have been collected by patient interviews, clinical periodontal examination, and radiographic examination and laboratory tests as required (AL-Bayaty et al 2011). The findings from periodontal diagnostic procedures can be extremely useful in evaluating treatment outcomes.
The appropriate assessment of the periodontal patient is a cornerstone in the successful treatment of an individual. The outcome of any treatment, both short and long term, depends on the accurate diagnosis of the aetiology of any current condition and the implementation of a tailored plan, which addresses both the patient’s and the professional’s ability to implement it.

**Predisposing Factors**

Predisposing factors might increase the probability of disease occurrence and usually have a localised affects. These factors include anything, which retains or hinders the removal of dental plaque (Marshall 1998, Seymour 2008, Genco and Bernakke, 2013). Therefore, the assessment of these factors is of a high priority in the assessment of the periodontal status of a patient. There are numerous examples of predisposing factors which includes; dental calculus overhangs restorations, subgingival restorations, subgingival restoration margins, open contact points, partial dentures, tilted/rotated/crowded teeth, bulbous crowns, grooves on teeth and more. These factors can be divided into anatomical, iatrogenic or pathologic features in which will lead to the retaining of dental plaque inside the mouth. This section will aim to describe the common predisposing factors that are important to discover during the assessment of a periodontal patient.

**Dental Calculus**

Dental calculus are made up of mineralized bacterial plaque composed primarily of calcium phosphate ($\text{Ca}_3\left[\text{PO}_4\right]_2$) mineral salts deposited on the surfaces of natural teeth and dental prostheses. It is the predisposing factor for the development of periodontal diseases by providing a retentive surface to plaque bacteria and impeding attempts to maintain an effective level of plaque control (Mandel & Gaffar 1986). It is believed that dental calculus formation is always preceded by plaque formation (Muhlemann and Schneider, 1959, Turesky et al., 1961, Selvig, 1970, Zini et al., 2011,). Histological evidence also, showed that calculus contains bacteria plaque incorporated into its mineralised structure. Dental calculus can be classified as supragingival and subgingival according to the relation to the gingival margin under light microscopy and transmission electron microscopy (Tan et al., 2004a). It is widely classified into two categories according to the location. Supragingival calculus is located coronally or above the gingival margin. In contrast, subgingival calculus is located apically or below the gingival margin in the gingival sulcus or periodontal pocket. Studies shown that supragingival calculus has a very distinctive distribution around the mouth, which primarily being localised to the lingual aspects of the lower anterior and the buccal surfaces of the upper molars (Alexander, 1971, Anerud et al. 1991). Subgingival calculus also provides an ideal environment for bacterial adhesion. Calculus may amplify the effects of bacterial plaque by keeping the bacterial deposits in close contact with the tissue surface, thereby influencing both bacterial ecology and tissue response (Friskopp & Hammarstrom 1980). Subgingival calculus has been demonstrate to retain significant levels of endotoxin, and these mineral deposits have been shown to affect tissue damage in controlled experiments (Jones & O’Leary 1978, Patters et al. 1982). Brown et al. observed that black pigmented bacteroides and spirochetes were found more often in periodontal pockets with
calculus than in pockets without. These suggest that subgingival calculus can alter microbial manifestation of periodontal disease (Brown et al. 1991). Secondly, the effect of dental calculus seems to be secondary by providing an ideal surface configuration conducive to further plaque accumulation and subsequent mineralization (Listgarten & Allegaard 1973). Nevertheless, calculus deposits may have developed in areas with difficult access for oral hygiene or may by the size of the deposits jeopardize proper oral hygiene practices (Tonetti et al., 2015). Well controlled animal (Nyman et al. 1986) and clinical (Nyman et al. 1988) studies have shown that the removal of subgingival plaque on top of subgingival calculus will results in healing of periodontal lesions and the maintenance of healthy gingival and periodontal tissues, provided that the supragingival deposits are meticulously removed on a regular basis. It has to be realized that meticulous supragingival plaque control guarantees the depletion of the supragingival bacterial reservoir for subgingival recolonisation (Mombelli et al. 1995). These studies have clearly elucidated the role of subgingival calculus as plaque-retaining factor.

Iatrogenic factors: Overhangs Restoration and subgingival restorative margin

Studies have validated that overhangs restorations, will hinder plaque removal and hence predispose the tooth to periodontal inflammation (Lang et al. 1983, Pack et al., 1990, Jansson et al. 1994, Matthews & Tabesh, 2004). Association between overhang restorations and a shift to the more virulent microflora had been shown by Lang et al. (1983) in a study using gold onlays. Gold onlays with overhang and perfect margins were placed in participants with healthy gingival conditions at different time periods. Their findings showed that, there were increased Gingival Index together with the elevation of Gram–ve anaerobic bacteria and black pigmented bacteriode in overhangs group compare from those without (Lang et al. 1983). This study confirms that, early detection and removal of overhanging margin should be part of initial periodontal therapy. Dental restorations, if poorly designed or compromised may be predisposing factors by retaining or hindering the removal of plaque. The placement of restorative margins above, below or at the level of the gingival margin has been shown to be associated with differences in gingival inflammation and plaque accumulation (Puri et al., 2014). Subgingival restorative margins have been associated with increased dental plaque accumulation that leads to gingival inflammation and periodontal pocket formations (de Waal & Castellucci, 1994, Schatzle et al. 2001). Apart from their effect on inflammatory process, subgingival margins may cause damage to attachment apparatus by violation to the biological width as shown in humans (Tarnow et al. 1986) and animal studies (Tal et al. 1989). Gargiulo et al. measured the dentogingival junction in humans and found that the average space occupied by the sum of junctional epithelium and the supracrestal connective tissue fibers was 2.04 mm which was then defined as the biological width (Gargiulo et al. 1961). In order to have a harmonious and successful long term restoration, few studies have advocated a 3mm distance of sound supracrestal tooth structure between bone and prosthetic margins (Wagenberg et al. 1989). It is because the biologic width appears to constitute a constant feature in the human periodontium. Clinical observation indicates that impingement of the biologic width will result in attempts by the gingival tissue to reestablish its original dimension through bone resorption or, in the presence of a thick alveolar crest resulting in
chronic gingival inflammation (Kina et al., 2011; Shobha et al., 2010). Furthermore, there is evidence suggesting that the biologic width and the entire gingival complex will reestablish itself during healing of the periodontal tissues after surgical procedures (Shobha et al., 2010). Experimental studies have shown that supragingival margins should be chosen whenever possible during cavity or crown preparation, and furthermore, that restoration margins already placed subgingivally should be re-exposed by surgical crown lengthening of the clinical crown (Padbury et al., 2003).

Iatrogenic Factors: Restorative materials

The presence of restorative materials on tooth surface is perceived to be a predisposing factor to periodontal disease. Notably, dental plaque is believed to adhere better to restorations than to enamel surface as every finished surface of restorative materials has different capacity for retaining plaque (Bollen et al. 1997, Ababnaeh et al., 2011). This was shown by Ababnaeh and colleagues (2011) that crowns, bridge abutments (especially acrylic and non-precious metals) and Class II amalgam restorations appear to be associated with periodontal breakdown. This may due to the surface characteristic of restorative materials such as surface roughness and surface free energy inherent in the materials. The rough surface of restorative materials especially on the interproximal and marginal can become predisposing factors for plaque accumulation and lead to destruction of periodontium (Pneumas et al. 1998, van Dijken et al. 1987). These studies found that, finishing on the interproximal of composite resins were difficult to achieve and marginal defect frequently detected; resulting of highly bacterial plaque accumulation. Wise and Dykema (1975) had compared four different restorative materials on plaque retentive capacity. They had suggested that, highly glazed porcelain is less plaque retentive than enamel, whereas on the other hand, metal pontic or composite restoration tends to encourage plaque formation (Wise & Dykema 1975).

Iatrogenic Factors: Dental Prostheses

Removal dental prosthesis especially with gingival coverage can become a predisposing factor for periodontal disease (Zlataric et al. 2002). While it is accepted that meticulous oral hygiene instruction plays a major role in decreasing these risks associated with removable partial dentures, the inherent ability of removable partial dentures to retain dental plaque is still considered a major risk factor. Thus this might explain the recent link between the prescription of partial dentures and the development of periodontitis around the abutment teeth (Behr et al., 2012). Accumulation of plaque around and underneath the different components of a partial denture is not only responsible for developing chronic periodontitis but can result in gingival recession and root caries (Mihalow and Tinanoff, 1998; Rocha et al., 2003, do Amaral et al., 2010). It has been shown that, removable partial denture favour the plaque accumulation, especially with various clasps and wires incorporated within them (Zlataric et al. 2002, Ogunrinde et al., 2014). Addy & Bates (1979) conducted a study among a group of forty-five patients to assess the effects of wearing partial dentures upon the accumulation of plaque in the mouth in the absence of oral hygiene. The wearing of a partial denture resulted in a significant increase in plaque accumulation which resulted in significant increase in both buccal and lingual plaque. It is noteworthy, that in the presence of improved
plaque control and denture hygiene procedures, denture associated plaque retention can be markedly reduced.

**Anatomical Factors: Enamel pearl and cervical enamel projection**

Cervical enamel projection and enamel pearls are ectopic deposits of enamel apical to the level of the cemento-enamel junction (Leknes 1997, Romeo et al., 2011). A cervical enamel projection is tapered with increased prevalence in molars tooth especially on buccal surface of second molars with and without furcation involvement (Blieden 1999, Hou & Tsai 1987). The influence of this tooth anomaly is dependent on the extent of the projection; which with Grade III, furcation area will be covered thus compromise plaque removal (Masters & Hoskins 1964). Enamel pearls are frequently seen in/on the maxillary third molar and are less likely to be found on the maxillary first and mandibular second molars (Moskow & Canut 1990). This tooth anomaly results difficulty in sub-gingival cleaning and therefore deposit surrounding or below it may not be removed completely during root debridement.

**Anatomical Factors: Tooth Position**

Studies shown that crowding may complicate oral hygiene procedure, which subsequently hinders plaque removal and results in gingival inflammation (Geiger et al. 1974). An increase in periodontal disease associated with malocclusion has often been used as an argument for instituting orthodontic therapy. The irregularity of teeth has been demonstrated to have gingivitis has (Ashley et al., 1998, Clerehugh & Tugnait, 2001). Direct relationship between crowding, gingival inflammation and bone loss were only evident on patient with moderate to poor plaque control, as contrast with patient with excellent plaque control (Jensen & Solow 1989). It can be concluded that, crowding becomes a predisposing factor, which lead to periodontal inflammation towards patient with suboptimal oral hygiene.

**Modifying Factors**

Modifying factors tend to act in a systemic fashion, which will alter the nature of a disease. In the context of periodontal disease, modifying factor can alter the nature or course of the inflammatory response to the plaque by modifying the susceptibility of disease, plaque microbiota, clinical presentation of periodontal disease, disease progression and response to treatment Kinane et al., 2006 Seymour, 2008). Throughout history taking, particularly medical, smoking and social history, the modifying factors can be identified to relate with the present periodontal conditions. However, the exact role of systemic diseases and systemic exposure in initiating or modifying the progress of periodontal disease is still clearly a complex issue. Therefore, there are several possible mechanisms on how the systemic disease can become modifying factors for periodontal disease. Mechanism by which these modifying factors can change the nature of periodontal disease will be discuss in context of changes in vasculature components (Mealey & Moritz 2003), healing and repair potential (Graves et al. 2007), and cellular components (Preshaw et al. 2007). Diabetes (Emrich et al. 1991, Saremi et al., 2005), pregnancy (Mealey & Moritz 2003), and tobacco smoking (Palmer et al. 2005, Johnson et al., 2007) mainly, have major effects on the host, which have the potential to modify the susceptibility to disease, plaque microbiota, and clinical presentation of
periodontal disease, disease progression and response to treatment (Page & Beck 1997, Palmer & Soory 2008). Apart from those factors mentioned above, blood dyscrasias (VanDyke et al. 1989), vitamin C deficiency (Nishida et al. 2000), medications (Seymour, 2006) and other conditions will be elaborate in details as how they modify the periodontal disease. The categorisation of the systemic modifying factors for periodontal disease and the evidence to support the role of these factors are the focus of this review.

Diabetes Mellitus

Diabetes mellitus (DM) is a metabolic disorder characterized by altered glucose tolerance and impaired carbohydrate metabolism due to insulin dysfunction. The two major forms of diabetes mellitus, Types 1 and 2, share much co-morbidity, but are characterised by having distinct etiologies. Type 1 diabetes occurs when the beta cells of the pancreas are destroyed and insufficient amounts of insulin are produced. In most cases, type 1 diabetes is the result of autoimmune-induced inflammation with destruction and apoptosis of beta cells. Whereas, Type 2 diabetes is caused by a combination of insulin resistance coupled with insufficient production of insulin to overcome the insulin resistance (Kahn & Flier 2000). The onset is generally more gradual than for type 1, and this condition is often associated with obesity. Oral manifestation of DM included xerostomia, burning mouth, altered taste sensation, candidosis and periodontitis. Assessing glycated hemoglobin levels makes conclusive diagnosis of diabetes mellitus; in people with diabetes, sequential fasting plasma glucose levels will be 7mmol/L or more (Mealey and Ocampo, 2007).

Epidemiologic studies on Pima Indians population, which has an extremely high prevalence of Type 2 diabetes shown, higher prevalence either periodontal attachment loss or radiographic bone loss; indicating that diabetes is a risk factor for periodontal disease (Emrich et al. 1991, Schlossman et al. 1990). Studies also have shown there is a bidirectional relationship between diabetes mellitus and periodontitis (Grossi & Genco 1998, Lalla et al. 2000, Lalla et al. 2001, Mealey & Ocampo 2007). Overall, it can be stated that, diabetes affects all periodontal parameters, including gingival bleeding, probing depth, and attachment loss by various mechanisms (Nassar et al. 2007). There are several mechanisms through diabetes can act as modifying the nature of periodontal disease; includes changes in subgingival environment, altered tissue homeostasis and wound healing, and changes in host immuno-inflammatory response.

- Altered tissue homeostasis, wound healing and repair potential

In patient with diabetes, collagen metabolisms, which involve synthesis, maturation, and turnover of collagen, will alter. Changes of collagen metabolism includes decreased rate of collagen production by fibroblast in the periodontal ligament and gingiva and an increased production of gingival collagenase. Since the periodontium is composed primarily of collagen, these changes of collagen metabolism may contribute to alteration in wound healing and periodontal disease initiation and progression. Advanced glycation end product (AGE) is likely to play a key role in linking the pathogenesis of diabetes and periodontal disease. Accumulation of advanced glycation end product (AGE) can contribute to collagen
metabolism, which results in increased cross-linking of collagen, reducing collagen solubility and decreasing the turnover rate. These then, will results in increased thickness in gingival capillary endothelial cell basement membrane and may impair exchange of oxygen and metabolic waste products across the basement membrane. AGE’s product will induce oxidative stress in periodontal tissues and this may be related to enhance periodontal disease in diabetics.

- Changes in cellular components

Diabetes may also result in increased periodontal susceptibility via increased apoptosis and chemotaxis of cells in the periodontal tissues. This may be associated with increased apoptosis of periodontal cellular components; particularly those associated with repair and wound healing (Graves et al. 2007). Notably, diabetic patients have been reported to have defects in neutrophil apoptosis and chemotaxis. In healthy host, neutrophil are genetically programmed to undergo spontaneous apoptosis within <24 hours. However, this time frame is increased when neutrophils are exposed to bacterial lipopolysaccharides. This increased longevity of the cells may contribute to increased local tissue damage via extracellular release of lysosomal enzymes and can result in increased risk for periodontitis (Preshaw et al. 2007). This is presumably a defense mechanism, to increase the functional lifespan of the cells to combat the bacterial infection.

**Sex Steroid Hormones**

Estrogen and progestin are widely known as the main steroids hormones which fluctuate during puberty, menstruation, pregnancy and menopause. Implications of the hormonal changes may have a significance influence on the tissue of the periodontium as of in clinical presentation and physiologic changes in gingiva in the absence or increased plaque accumulation (Holm-Pedersen & Loë 1967, Mombelli et al. 1989, Nakagawa et al. 1994). Mechanisms by which these hormonal changes can result in significant changes have been widely discussed by looking at few aspects, such as on how it effect on microbiota and lead to changes on tissue and host response (Miyagi et al. 1993).

- Puberty and menstruation

During the circumpubertal period, there are increased flux of estrogen hormones in females and testosterone in males. This flux of hormones may and may not sufficient to produce gingival change by acting alone. However, pubertal gingivitis is common among both boys and girls, and it is believed that increased in androgen level is responsible for the changes. A clinical feature; manifest as marginal and interdental gingival enlargement that found on the facial surfaces with combination of increased tendency of gingival bleeding. Despite the volume of data supporting the occurrence of peri-pubertal gingivitis, study by Yanover and Ellen (1986) did not find a significant correlation between the onset of puberty and gingival changes in prepubescent females (Yanover & Ellen 1986). Same findings was found by Tiainane et al., (1992) in a 2-year follow-up study on gingival condition and its relation to oral hygiene at different stages of puberty in 14-yr-old Finnish school children. There was no difference in the gingival bleeding tendency at various pubertal stages when all subjects were
included or when boys and girls were compared separately. Instead, a highly significant correlation was found between gingival bleeding and visible plaque, both at the baseline and 2 years later. This indicates that from the age of 14 to 16 the influence of oral hygiene on the gingival condition may be more important than that of the rising level of steroid hormones (Tiainen et al. 1992).

- **Menstruation**

Holm-Pederson and Loe (1967) in their findings suggest that the menstrual cycle has an effect on gingival health. During the time of ovulation when the level of estrogen and progesterone are at their highest, gingival inflammation noted with increase in gingiva crevicular fluid exudates (Holm-Pedersén & Loë, 1967) and tooth mobility. These symptoms are exacerbated by presence of preexisting gingival inflammation (Lindhe & Attstrom, 1967). Studies also reported, hormonal variation can directly influence the oral microflora where the growth of periodontal pathogens such as Bacteroides and Prevotella species were encouraged (Nakagawa, 1994).

**Pregnancy**

Gingival inflammation initiated by plaque and exacerbated by a hormonal changes usually manifest during the end of the 1st trimester and increase in severity through to the end of 3rd trimester (Löe, 1965). Interproximal papillae of the incisors are most commonly affected (Löe & Silness, 1963). Research data showed steroids hormones in pregnancy have influence on the microbiota, gingival vasculature, host response and tissue of the periodontium (Jensen et al. 1981, Miyagi et al. 1993). Elevated progesterone levels in pregnancy enhance capillary permeability and dilatations resulting in increased gingival exudates Pregnant women demonstrate increased of \( P. \) intermedia proportion about 55-fold compared with non-pregnant controls (Jensen et al. 1981). Elevated levels of estrogen and progesterone in pregnancy also affect the degree of keratinization of gingival epithelium and enhance capillary permeability and dilatation, thus contribute to exaggerated response to bacterial plaque substanc eg periodontal infection in pregnant women would deteriorate significantly (Löe, 1965). To summarise, there are ample data supporting the existence of steroid sex hormone induced gingival inflammation. Nevertheless, there are currently no data supporting pregnancy gingivitis or hormonal associated gingival inflammation being a predecessor for periodontitis.

**Medications**

Excessive accumulation of connective tissue elements is a feature of drug–induced gingival hyperplasia and gingival fibromatosis. Drug-induced gingival overgrowth is a side-effect and unwanted outcome of systemic medication and is limited to gingival tissues. Drug-induced gingival overgrowth does not originate in the periodontium, but it occurs exclusively in periodontal tissues and is generally not of the same magnitude in other tissues. Gingival hyperplasia occurs mainly as a result of side effect of systemic medications. These medications include the anti-seizure drug: Phenytoin, the immune suppressor: Cyclosporin, and certain anti-hypertensive dihydropyridine calcium channel- blockers, most notably
Nifedipine and other drugs such as Sodium Valproate and Erythromycin (Seymour & Heasman 1988). The degrees of inflammation, fibrosis, and cellularity depend on the duration, dose, and identity of the drug, on the quality of oral hygiene, and on individual susceptibility that stems from genetic factors and environmental influences (Al-Bayaty et al.2009).

**Blood Dyscrasia**

Association between various systemic conditions particularly related to blood dyscrasia factors with periodontal disease does exist. Association of these conditions is clearly defined in most of the neutrophil dysfunction conditions. Neutrophil being the major component of the inflammatory and immunological system has been the center of cellular defender in pathogenesis of periodontal disease. The effectiveness of a neutrophil is solely depending on its abilities to roll along vascular epithelium, adhere to the endothelium and migrate following a chemotactic gradient (Deas et al. 2003). Apart from that, neutrophil also will adhere to micro-organism, involves in phagocytosis and intracellular killing. Normal adult range of neutrophil varies from 1,800-8000 cells/μl. Defects in all these functions can be due to decreased number of neutrophil (neutropenia- neutrophil count below 1000 cells/μ) which can be seen in cyclic neutropenia and benign chronic neuropenia. These two conditions may change the nature of periodontal disease by decreasing the inflammatory response due to less number of neutrophil. Chediak-Higashi syndrome, Chronic granulomatous disease, leukocyte adhesion deficiency and Papillon –Lefever Syndrome are the conditions that results from effective of the quality of neutrophil action towards the inflammatory response (Kaplan et al. 2008).

**Age**

The relationship between age and periodontitis should always be considered when assessing patient’s susceptibility to periodontal disease. Early evidence demonstrates that both the prevalence and severity of periodontitis increase with increasing age, suggesting that age may be a marker for periodontal tissue support loss (van der Velden, 1991). In contrast, patients who suffer from aggressive types of periodontitis are usually younger than patients who suffer the chronic forms of periodontal disease. However, Grossi and co-workers in both of their studies have shown more periodontal disease, both in terms of attachments loss as well as bone loss, among older age groups compare to younger age groups (Grossi et al. 1995, Grossi et al. 1994). In addition, research has shown that certain physiological changes in the periodontium occur with increasing age. Elderly patients generally have a longer healing period and are more likely to suffer from other medical conditions that may modify the course of the disease, e.g. diabetes or osteoporosis, which may have further implications on treatment options. However, in the first National Health Survey in 1987, minor effect of aging on periodontal destruction compared to the role of plaque as represented by oral hygiene practice has been shown (Abdellatif & Burt 1987). This was then supported by a study in Finland which showed a very little change of periodontal status of elderly patients through 5-years follow-up (Ajwani & Ainamo 2001). Although a higher level of attachment loss, increased diseases extent and prevalence are found in the elderly population, aging per
se is not considered to be a predisposing factor for periodontal disease. A literature review by van der Velden (1984) concluded that age related physiological changes is a normal occurrence in human and animal, however, it was also mentioned that development of periodontal inflammation becomes more rapid with increasing age. It may merely be the fact that increase in age means prolonged exposure to the etiological factors and hence increase disease activity (Albander, 2002).

**Genetics and susceptibility**

Understanding of the genetic basis for disease susceptibility and progression should be accompanied with the clear definition of terminology used. Genetic risk factors have been proposed to have influence in the natural history of periodontitis (Kornman et al. 1997, Michalowicz 1994a, b, Michalowicz et al. 1991). A classic twins study by Michalowicz indicated that, genetic factors might explain approximately 50% of the population variance in periodontal disease progression (Michalowicz 1994a, b, Michalowicz et al. 1991). Michalowicz and co-workers have also done a studies on 64 monozygotic twins (MZ) and 53 dizygotic twins (DZ), where clinical parameters such as probing depth, clinical attachment loss, gingival and plaque index been evaluated between both pairs. They confirmed that, all clinical parameters were more similar in MZ twins than DZ twins although there was an adjustment for behavioral variables including smoking. This concluded that the basis for the heritability of periodontitis appears to be biological and not behavioral in nature (Michalowicz et al. 2000). Although several genetic polymorphisms have been associated with periodontal disease, not enough evidence at present to supports the widespread use of genetic tests to either assess risk for disease or predict treatment response (Fouad et al. 2011).

**Smoking**

Cigarette smoking is recognised as a major risk factor in the incidence and progression of periodontitis (Bergstrom & Preber 1994, Grossi et al. 1994, Al- Bayaty et al. 2008). In a longitudinal study by Machtei and co-workers, smokers exhibited greater attachment loss (AL) and radiographic bone loss (BL) (Machtei et al. 1997). Smoking can affect the pathogenesis of the periodontal disease by altering the host response, change the periodontal disease patterns and affect periodontal healing and treatment outcomes (Barbour et al. 1997, Palmer et al. 2005). Studies measuring host response to the infection have shown a reduction of neutrophil function, antibody production, fibroblast activity, vascular factors and inflammatory mediators, which could impair the normal host response in bacterial clearance, and the neutralising of infection (Seymour, 1991). Smoking has immunosuppressive effects that impair host defenses by decreasing motility, chemotaxis and phagocytosis of polymorphonuclear leukocytes in peripheral blood. Therefore, the first line of defense against subgingivally colonized bacteria is endangered and this may cause alterations in the oral environment and tissues themselves, which result in destruction of the periodontium (Barbour et al., 1997). In addition, tobacco smoking may modify the production of pro-inflammatory cytokines interleukin-1 (IL-1) and Tumor necrosis factor-alpha (TNF-α) key regulators of the host response to microbial challenge. Kornman et al. 1997, recently reported a specific periodontitis associated IL-1 genotype, which was correlated with high level of IL-1
production in non-smokers. However, in smokers, severe periodontitis was not correlated with the genotype, which indicates that the genetic control of the host response was evident only when smoking was excluded. This finding further emphasises the importance of smoking in pathogenesis of periodontitis. Smoking has been identified as an important cause of impaired healing in all aspect of periodontal treatment including nonsurgical and surgical treatment (Heasman et al. 2006, Kinane & Chesnut 2000). It has been reported that nicotine in smoke can be stored in and released from the fibroblast (Hanes, Schuster & Lubas, 1991). Cotinine, a metabolite of nicotine, can be measured in the serum plasma and saliva, and is a better measure of tobacco smoke exposure as it has a longer half-life than nicotine (18h compared with 1-2hr). Smokers normally have serum cotinine level of over 14ng/ml and could be as high as 1000ng/ml (Fouad et al. 2010). Nicotine or cotinine can also inhibit the production of fibroblast fibronectins and collagen and stimulate fibroblast collagenase activity. But, it remains unclear whether these nicotine-exposed fibroblasts have an impaired or enhanced ability to attach to surrounding surfaces. It may be that these in vitro effects on fibroblasts may occur in vivo as well and influence the healing ability of the periodontal tissues. The cytotoxic effects on the periodontal fibroblast function, will lead to impaired maintenance of periodontal tissues and for optimal wound healing (Fouad et al. 2011). Tobacco smoke and nicotine affect the microvasculature, the fibroblast and connective tissue matrix, the bone and root surface itself. It has been shown in vitro studies that, fibroblast are affected by nicotine in that they demonstrate reduced proliferation, reduced migration and matrix production and poor attachment surfaces. Toxic substances from tobacco smoke can coat the root surfaces of periodontally involved teeth and interfere with postsurgical healing. Products of smoking such as nicotine additionally contaminate the root surface of the smokers, cotinine, acrolein and acetyldehyde and these molecules may affect the attachment of cells (Farha et al.2012). In conclusion, cigarette smoking represent a risk factor for progression of periodontitis, the effect of which may be a dose related. Heavy smokers should be considered as high risk individuals for disease progression. The clinical implications for this are that smokers should be identified during patient examination and efforts should be made to modify this behavioral risk factor (Fouad et al.2013).

**Nutrition**

- **Vitamin C**

Vitamin C has long been a candidate for modulating periodontal disease, although the exact role of vitamin C deficiency in periodontitis is not known (Nishida et al. 2000). Even though low vitamin C intake does not cause periodontitis, it is known that additional vitamin C is required during infectious diseases and tissue regeneration (Rubin, 1984). Vitamin C deficiency is associated primarily with defective collagen synthesis, causing tissue dysfunction such as impaired wound healing and ruptured capillaries because of insufficient support of the capillary walls by the connective tissues. Regeneration of collagen to maintain the integrity of the tooth attachment elements is especially important for periodontal health. Since vitamin C is involved in the synthesis of intercellular substances such as collagen fibers found in various forms of connective tissues and the matrix of bone and teeth and since vitamin C has immuno-modulating functions influencing the susceptibility of a host to
infectious diseases (Bhaskaram 2002). It is rational to hypothesize that a low vitamin C concentration in serum is a risk factor for periodontal diseases (Nishida et al. 2000)

**Conclusion**

A better understanding of plaque, which is the principal aetiology and identification of the factors that can predispose and modify the periodontal diseases, will illuminate the reasons for successful treatment. Prevention and treatment should aim at removal of the bacterial biofilm and associated risk factors in order to halt disease progression, and allow regeneration of lost supportive periodontal tissue. Over the years, multiple factors such as immune related host susceptibility, systemic disease states and environmental factor like smoking and stress had been demonstrated to have a major input in the final diseases phenotype. Numerous risk factors that may contribute to the initiation and severity of periodontal diseases were proposed and described in the present assignment. In order to formulate a comprehensive treatment plan, it is necessary to consider all the risk factors that play a role in periodontal infection during patient assessment.

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