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RISK FACTORS ALLIED WITH PERIODONTAL DISEASE

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ABSTRACT

Periodontal disease is a chronic inflammatory disease characterised by loss of tooth supporting tissues. Microbial plaque is considered as a primary etiologic factor accountable for the initiation of periodontal disease. The clinical manifestation and progression of the disease is however controlled by anarray of other factors such as immune response or defence mechanism of the individual against the microbial plaque, genetic factors, systemic disease, environmental factors etc. Hence, it can be rightly said that periodontal disease is a multifactorial disease and the liaison between these factors and their effect on the disease progression needs to be understood for suitable treatment of individuals suffering from periodontal disease.

KEYWORDS: periodontal disease, risk factor, plaque, bacteria, healing.

INTRODUCTION

A risk factor is any characteristic, behaviour or exposure with an alliance to a particular disease.[1] The relationship between the risk factor and the particular disease is not necessarily causal in nature. A risk factor acknowledged to be associated with disease from cross sectional or case control studies is known as risk indicator. However, longitudinal studies are fundamental to identify risk factors. Risk marker is a term which indicates a risk factor used to foretell the future course of the disease. Risk factors that cannot be modified are known as determinants.[2]

Periodontal disease is a continuously progressing inflammatory disease which is preceded by gingivitis. In some patients, the disease is restrained to the gingiva which is clinically presented as gingivitis. In susceptible patients, the disease may progress leading to attachment loss and bone resorption, clinically manifesting as periodontitis. Risk factors modify the inflammatory response and thus affect the clinical outcome of periodontal disease.Some risk factors can be modified to reduce the risk of initiation or disease progression such as smoking cessation or improved oral hygiene while others cannot be modified such as genetic factors.[3]

Modifiable risk factors

Oral hygiene level

Microbial plaque is the prime etiologic factor in the initiation and development of periodontal disease.[4] The level of oral hygiene is directly correlated to the amount of plaque present in the oral cavity and is for that reason a factor which affects the prevalence and severity of periodontal disease. Moreover, the level of oral hygiene is also correlated to the frequency of tooth brushing. Several epidemiological studies have shown that the oral hygiene level is directly related to the prevalence of periodontal disease.[5,6] Matthew S. Hopcraft et al 2012 conducted a study to investigate oral hygiene and periodontal disease in Victorian nursing homes. The author concluded that periodontal disease was a common finding in individuals with poor oral hygiene with significant amounts of plaque and calculus.[7] Indian studies have also demonstrated a positive relationship between gingivitis and the amount of plaque and calculus.[8] Despite of several studies accounting for a positive association between oral hygiene and periodontal disease, this topic is still controversial. Merchant et al have stated that other factors such as genetics play a larger role in the initiation of periodontal disease than oral hygiene.[9] Although, after mechanical debridement the clinical signs of periodontal disease such as gingival bleeding or inflammation, periodontal pockets are reduced and there is maintenance of clinical attachment levels.[10]

Smoking

Smoking is a key risk factor for periodontal disease. There is a higher prevalence of periodontal pockets, alveolar bone loss and tooth loss in smokers with periodontal disease. Several longitudinal studies have reported an increased risk for disease progression in

smokers.[11,12] This risk is dose dependent in that former smokers (2 or more years have passed since the individual has quit smoking) experience less attachment loss than current smokers but more than never smokers.

Smoking has an effect on the inflammatory response and the healing response of the individual. Smoking increases the intraoral temperature which increases the initial gingival blood flow followed by a gradual decline in about 5 minutes.[13] Thus ultimately, reduced gingival bleeding is observed in smokers. This is often considered as an alteration in immune response which is thought to be responsible for the increased severity of periodontal disease. With respect to cellular response, smoking has an effect on neutrophils, which are of prime importance in defense mechanisms. Smoking has a dose dependent suppressive consequence on neutrophil chemotaxis and phagocytosis[14,15] and an increased production of matrix metalloproteinase by neutrophils thus contributing to an amplified periodontal destruction.[16] In addition to neutrophils, smoking also reduces the proliferation of T and B-cells resulting in a reduced cellular infiltrate in the periodontal tissues.[17]

During healing, smoking influences the functions of fibroblasts such as proliferation, collagen production and attachment on the root surface.[18] The effect of these functions is reflected in the response of smokers to periodontal therapy which is reported to be negative in soft tissue grafts, regenerative procedures and implants.[19,20]

Diabetes Mellitus

It is an assembly of metabolic disorders in which there are high blood sugar levels over a prolonged period. The disease is characterised by defects in insulin secretion, insulin action or both.[21] The current classification of diabetes is based on pathophysiology of each form of the disease, Type 1(Insulin dependent diabetes), Type 2 (Non-insulin dependent diabetes) and gestational diabetes.[22] Type 2 is more rampant than type 1. Periodontitis has been recognized as a complication of diabetes mellitus due to the consequential hyperglycaemia.

Several epidemiologic studies in diabetic adults have shown an increased prevalence and severity of periodontal disease.[23-26] There are several longitudinal and epidemiological studies on Pima Indians, which is a population with highest prevalence of type 2 diabetes in the world, that show a direct relationship between periodontal disease and diabetes.[24,25]

Patients with poor glycaemic control are at an increased risk of developing periodontal disease. An earlier and a stronger immune and inflammatory response is developed in these patients. Alterations in diabetic patients include endothelial dysfunction due to oxidative stress, reduced nitric oxide production and increased nitric oxide inactivation.[27] Also, immune cells such as neutrophils and macrophages are affected in patients with diabetes. Defective neutrophil functions such as reduced chemotaxis, phagocytosis and increased production of superoxides which increases further the oxidative stress.[28] Monocytes in diabetic patients produce more amounts of tumour necrosis factor alpha when stimulated with lipopolysaccharide compared to non-diabetic patients.

Advanced glycation end products are formed as a consequence of hyperglycaemia. These products are molecules of glucose bound to a protein or lipid, formed in plasma and tissues during the state of hyperglycaemia. There are receptors for these advanced glycation end products on endothelial cells and monocytes, which when combine result in an increased production of inflammatory mediators such as TNF- alpha and interleukin-6.[29] These mediators are to blame for the increased periodontal destruction observed in diabetic patients.

Stress

The psycho-physiological reaction of the organism to a perceived confront or danger is referred to as stress. The notion of stress was first pioneered in life science by Hans Selye in 1936. It is well established that stress can modulate the neural and endocrine systems by the autonomic nervous system pathways, release of neuropeptides and hypothalamic and pituitary hormones.[30] When the body is in a state of stress, there is a noticeable raise of immune cells in the plasma mobilized from lymphoid organs. The release of neuropeptides, which are located in the periodontal tissues in close contact with the vascular plexus, modulate the immune system and causes the release of cytokines. The hypothalamus-pituitary-adrenal (HPA) axis ultimately leads to release of glucocorticoid hormones which have suppressive effects on the number and activity of a variety of inflammatory cells.

Scores of mechanisms have been anticipated which link stress and chronic diseases like periodontal disease such as endocrine changes which take account of the increased amounts of cortisol in Gingival crevicular fluid (GCF), overlook of oral hygiene due to the mental status of the patient, modifications in dietary intake which includes the preference of foods, physical consistency of foods and measure of food eaten, smoking and other detrimental habits, alteration of blood supply and nutrients to the gingival tissues, alteration in salivary flow and components, oral habits and lowered host resistance.[31]

Acute necrotizing ulcerative gingivitis is observed among students at the time of examination and in military personnel.[32] Aggressive periodontitis patients have been reported to be more depressed than individuals with chronic periodontitis or healthy individuals.[33] A systematic review by Peruzzo DC et al 2007 also

demonstrated a positive relationship between stress and periodontal disease.[34]

Tooth factors

Tooth related factors or the local factors that may act as risk factors for the development of periodontal disease include tooth morphology and alignment, form and site of furcation, level and quality of dental restorations, calculus, carious lesions in close proximity to the gingiva, trauma from occlusion, alveolar bone morphology, gingival form and contact between the teeth.[35]

The margins of the restorations should be at or coronal to the gingival margin and care should be taken to avoid overhangs and irregularity in the restorations. Faulty restorations hamper the oral hygiene procedures, act as plaque retentive factor, alter the subgingival microflora and cause overt gingival inflammation and periodontal tissue destruction. Similarly, untreated carious lesions adjacent to the gingival tissues cause gingival inflammation. Calculus also acts as a plaque retentive factor as it is covered with a layer of unmineralized plaque.

Non-modifiable risk factors

Genetic factors

It is precisely said that periodontal disease is a complex disease with a multifactorial aetiology. The inflammatory response of periodontal tissues to infection is influenced by genetic factors. Aggressive periodontitis is said to have a strong genetic component with higher prevalence within families.[36] Familial aggregation studies have shown a higher frequency of periodontitis among siblings with autosomal dominant mode of inheritance.[37] In addition to aggressive periodontitis, twin studies have also demonstrated that 50% of the expression of chronic periodontitis is modified by genetic factors.

Genetic polymorphisms or single nucleotide polymorphisms have been studied in both chronic and aggressive types of periodontitis. These genes include those affecting the expression of interleukin-1, interleukin-6, tumour necrosis factor, interleukin-10, E-selectins, Fc gamma receptor, CD14, toll like receptors, caspase recruitment domain 15 and vitamin D receptor.[35] Most studies show variable and inconclusive correlations between the presence of disease markers and the tested single nucleotide polymorphisms in both the aggressive and chronic forms of periodontitis. Thus, evidence for the relation between genetic factors and periodontal diseases is poor and large scale comparative studies are required for stronger confirmation.

Host response

Periodontal diseases are chronic inflammatory diseases that are initiated by gram negative tooth associated microbial biofilms that elicit a host response. This disease is multifactorial in nature and the mere presence of gram negative bacteria does not account for the destruction seen.[38] The host immuno-inflammatory response to the bacterial challenge plays an important role in the initiation and progression of periodontal disease. In cases of aggressive periodontitis "primed neutrophils" are responsible for mediating the tissue destruction that is observed in that disease. Thus, deficiencies and abnormalities in polymorphonuclear neutrophils will have effects on periodontium.

Osteoporosis

Osteoporosis is a skeletal disease characterised by diminution in bone mass and micro architectural changes in the bone which leads to an increased bone fragility and increased risk of fracture. Since both osteoporosis and periodontal diseases are bone destructive diseases, it has been hypothesized that osteoporosis could be a risk factor for the progression of periodontal disease.

Several authors showed a positive association between osteoporosis and periodontal disease.[39] Majority of the studies have shown low bone mass to be independently associated with loss of alveolar crestal height and tooth loss.

Aging

Aging is associated with increased incidence of periodontal disease as shown by many epidemiological studies.[40] However, the effect of age is often reduced and even considered negligible after the adjustment of confounding factors such as oral hygiene.

Gender

Studies have consistently shown that periodontal disease is more prevalent in males than females. A higher prevalence of probing depth and attachment loss has been observed in males across different age groups.[41] In addition to poorer oral hygiene observed in males other factors like hormonal and physiological and behavioural differences between males and females contributes to higher prevalence of periodontal disease in males.

Race/ethnicity

Variations in disease expression have been found in different racial and ethnic groups in a given population. Higher prevalence of periodontal disease has been observed in blacks followed by Mexican Americans than in whites. Aggressive form of periodontitis has been more prevalent in African American adolescents and Hispanics as compared to whites. Asians have also been reported to have more bone loss.[41]

CONCLUSION

This paper shows that periodontal disease is a multifactorial disease. Dental plaque and microbial biofilms are the primary etiologic agents for periodontal disease, whereas several other factors have a modifying role in the pathogenesis of periodontal disease. Of all the risk factors, smoking and diabetes are significant risk factors for periodontal destruction. Effective disease

management should take account of detection of risk factors for periodontitis and an assessment of disease risk due to these factors.

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