

Periodontal Disease Risk Factors

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Local and Systemic Risk Factors

Local Risk Factors

- **Anatomical risk factors**
 - Enamel pearls
 - Root grooves
 - Furcations
 - Gingival recessions
- **Tooth position**
 - Malalignment
 - Crowding
 - Tipping
 - Migration
 - Occlusal forces
- **Iatrogenic risk factors**
 - Restorative overhangs and margins
 - Partial dentures
 - Orthodontic appliances

Systemic Risk factors

- **Modifiable**
 - Specific microbiota
 - Smoking
 - Diabetes mellitus
 - Oral Hygiene
 - Stress
 - Obesity
 - Immunodeficiency (such as that occurring with systemic disease)
 - Certain medications
 - Diet (poor nutrition)
 - Osteoporosis
 - Other Systemic Diseases
- **Non-modifiable**
 - Age
 - Genetics
 - Hormonal influences (such as those related to pregnancy)

Systemic Risk Factors

Often termed – ‘Environmental’ or
‘Modifiable’ risk factors

Specific Microbiota

Introduction

- Koch's postulates (Haffajee & Socransky 1994)
- Three species identified as causative factors for periodontitis (Aa, Pg, Tf)
- But many studies have found such species in periodontally healthy subjects (Lamell *et al.* 2000)
- Certainly an antimicrobial approach to treatment and maintenance regimes are the most successful strategies for periodontal disease treatment (Herrera *et al.* 2002)

Smoking

Introduction

- 13 million smokers in the UK
- 4000 potentially harmful substances in tobacco smoke
- More than 325 papers have shown a relationship between smoking and periodontitis
- Regarded as a major risk factor attributable to current or former smoking
- A wealth of epidemiological, clinical and *in vitro* studies have provided irrefutable evidence that smoking negatively impacts periodontal health (Bergstrom 1998; Albandar *et al.* 2000 Tomar & Asma, 2000)

Historical Data

- When first discovered to be linked to periodontal disease smoking was thought to be:
 - Linked to effects upon plaque
 - Then thought to be local affects on areas exposed to smoke
 - Today more inclined to hypothesise it affects the periodontium by general influence on the body

How Does Smoking Affect Periodontal Disease?

- Smoking results in changes in the vascular, inflammatory, immune response and healing of periodontal tissues (Palmer 1999):
 - Increased numbers of functionally compromised peripheral blood mononuclear phagocytes
 - Impaired angiogenesis
 - Depressed lymphocytes, IgA, IgG, prostaglandins
 - Deleterious effects on neutrophils and antibody production
 - Inhibit fibroblast functions (Collagen production) that could impair wound healing
 - Negative effects on cytokine/growth factor functions

Features of Periodontitis in Smokers

- Lack of correlation with oral hygiene
- Increased prevalence and severity of disease
- Increased calculus
- Increased tooth loss (Krall *et al.* 1997)
- Greater mean attachment loss (Haffajee & Socransky 2001)
- Greater bone loss (Grossi 1995)
- Deeper pockets (Bergstrom 2000)
- Fibrotic Gingiva
- Limited gingival redness/oedema relative to disease severity (Bergstrom & Preber 1986)
- Greater pocketing anterior maxilla
- Greater extent and severity of anterior gingival recession
- Increased bleeding following smoking cessation
- Increased risk of unfavourable response to therapeutic intervention

Epidemiological Data

- Odds ratios for developing periodontal disease as a result of smoking range from 2.5 (Bergstrom 1989), 3.97 for current smokers and 1.68 for former smokers (Tomar and Asma 2000) and 3.25 for light smokers to 7.28 for heavy smokers (Grossi *et al.* 1995)
- Smoking is a stronger risk factor than any of the 5 most suspected bacterial pathogens (Stoltenberg 1993)
- Periodontal treatment tends to be less likely to be successful in smokers than in non-smokers (Bostram *et al.* 1998; Ah *et al.* 1994)
- 51% of periodontitis in 19-30 year olds associated with smokers and 32% of periodontitis in 31-40 year olds associated with smokers (Haber 1993)

Dose Response Data

- Response to periodontal therapy related to numbers of cigarettes smoked (Kaldahal 1996)
- The negative effect of smoking has been shown to be dose dependent and to be particularly marked in younger individuals
- Long-term studies have shown that smoking is associated with the recurrence of periodontitis during periodontal maintenance

Smoking Cessation

- The reduction in the effectiveness of non-surgical periodontal treatment in periodontal patients indicates that smoking cessation therapy should be offered to smokers requiring such treatment (Preshaw *et al.* 2005; Heasman 2006)
- Cost-benefit analysis - adding smoking cessation to concept of periodontitis prevention will enable cost savings (Braegger 2005)
- Smoking cessation advice may increase abstinence rates (Carr 2006)

Managing Smokers

- Although the improvements in smokers are less than those for non-smokers, it is important to recognize that smokers DO benefit from therapies of all types
- Smoking compromises but DOES NOT prevent healing
- It is the extent of improvement and predictability that is reduced

Diabetes

Introduction

- Loe suggested that periodontitis is the sixth complication of diabetes
- Diabetes is a modifiable factor in the sense that though it cannot be cured, it can be controlled
- The bulk of the evidence indicates that there is a direct relationship between diabetes mellitus and periodontal disease
- Most reports indicated that subjects with diabetes have increased prevalence, extent, severity and progression of periodontal diseases (Kinane *et al.* 2006)
- In general no difference in impact has been determined between type 1 and type 2 diabetes mellitus

Literature

- A review of the literature by Kinane found considerable evidence to suggest that diabetes and periodontitis have a direct relationship (Kinane and Chestnutt 1997)
- Studies have shown a relationship between poor glycemic control and periodontal disease parameters (Guzman *et al.* 2003; Tsai *et al.* 2002; Tervonen *et al.* 1994, Cutler *et al.* 1999, Graves *et al.* 2006))
- Cross sectional studies on Pima Indians, a group displaying the highest prevalence of type-2 diabetes in the world, show an odds ratio of 2.8 to 3.4 for developing periodontal disease in type-2 diabetics compared to non-diabetics (Emrich *et al.* 1991)
- Similarly, longitudinal studies have shown increased risk of ongoing periodontal destruction in diabetics as compared to non-diabetics with an odds ratio of 4.2

How does Diabetes Influence Periodontal Disease?

- The prevalence, severity and extent of periodontitis have been shown to increase with poorer metabolic control of diabetes as well as with the onset of diabetes at a younger age
- Factors contributing are:
 - Degree of glycaemic control
 - Age of onset
 - Duration of the disease
- Prevalence and severity has been attributed to the reduced function of their neutrophils and to the formation of advanced glycosylation endproducts (AGEs) that lead to the oversecretion of inflammatory mediators such as interleukin-1, tumor necrosis factor-alpha and prostaglandin E2
- It has also been attributed to:
 - Dysregulation of polymorph function
 - Altered collagen regulation
 - Microvascular damage

Treatment Implications

- Studies have been done which suggest that poorly controlled diabetics respond less successfully to periodontal therapy (Westfelt *et al.* 1996; Tervonen & Karjalainen, 1997)
- Owing to the evidence it is recommended that controlling diabetes should be a decisive factor in any periodontal treatment

Oral Hygiene

Introduction

- In a seminal series of studies, Loe *et al.* established the causal relationship between plaque and gingivitis
- The evidence for oral hygiene (plaque) being a risk factor for periodontitis is based on evidence drawn from randomized controlled trials that examined the cause–effect relationship of plaque on gingivitis
- Clinical studies have shown the improvement in surrogate measures of periodontitis resulting from improved plaque control
- Long-term studies have shown greater tooth loss in patients with poorer oral hygiene

(Stabholz 2010)

Stress

Introduction

- Clinical observations and epidemiologic studies suggest that stress, depression and anxiety are potential factors that may affect periodontal disease
- The exact impact of stress on the periodontium has not yet been clearly elucidated
- When analyzing reports that examine the stress–periodontal disease associations in humans, it should be borne in mind that such studies lack a uniform method to define and quantify stress and thus make it difficult to compare results and draw conclusions
- Studies have demonstrated that individuals under psychological stress are more likely to develop clinical attachment loss and loss of alveolar bone (Hugoson *et al.* 2002; Mawhorter & Lauer 2001; Pistorius *et al.* 2002; Wimmer *et al.* 2002)
- One possible link in this regard may be increases in production of IL-6 in response to increased psychological stress (Kiecolt-Glaser *et al.* 2003)
- Perhaps the relationship is simply due to the fact that individuals under stress are less likely to perform regular good oral hygiene and prophylaxis (Croucher *et al.* 1997)

Obesity

Introduction

- After poor oral hygiene, it has been suggested that obesity is second only to smoking as the strongest risk factor for inflammatory periodontal tissue destruction
- A positive association between obesity and periodontitis has recently been shown in animal and human epidemiological studies (Saito *et al.* 2001; Wood *et al.* 2003)
- The underlying biological mechanisms for the association of obesity with periodontitis are not well known; however, adipose tissue-derived cytokines and hormones, may play a key role in this link by modulating periodontitis (Nishimura & Murayama 2001)

Immunodeficiency

Introduction

- Conflicting evidence for prevalence and severity of periodontal diseases in patients with AIDS (Winkler & Murray 1987; Chapple & Hamburger 2000)
- Main periodontal disease associated with AIDS are necrotizing periodontal diseases (gingivitis and periodontitis)
- Periodontitis can result in large soft tissue necrosis, no deep pocket formation (unlike chronic periodontitis) and interdental ulceration with exposure of bone
- Main causes of HIV-periodontal destruction:
 - 1) Primed or overactive PMNs.
 - 2) Failure of CD4 lymphocyte and macrophage response.
 - 3) Increase pathogenicity of bacteria in micro-flora due to immunodeficiency

Medications

Introduction

- Gingival hyperplasia/ gingival overgrowth can be caused by:
 - Phenytoin (epileptic medication)
 - cyclosporin (immunosuppressive)
 - nifedipine (calcium-channel blocker to treat cardiac angina and hypertension)

Diet

Introduction

- At the moment there is no evidence to say that diet directly affects the state of the periodontal tissue
- However it is a factor that can influence disease expression
 - eg: Vitamin C deficiency there is less collagen synthesis and thus a reduced turnover and repair when combined with poor oral hygiene then disease expression is more likely

Osteoporosis

Introduction

- A systemic skeletal disease characterized by reduced bone mineral density
- Relationship of osteoporosis to periodontal disease and tooth loss is complex
- Several recent papers have drawn attention to the possible link between osteoporosis and periodontal disease
- This relationship emerged from the fact that both conditions involve bone loss and may share common pathological pathways
- The assumption of an increased risk for periodontal disease in the presence of osteoporosis is based on the hypothesis that osteoporosis results in the loss of bone mineral density throughout the body, including the maxilla and the mandible
- The resulting low density in the jawbones leads to increased alveolar porosity, an altered trabecular pattern and more rapid alveolar bone resorption following infection with periodontal pathogens
- Perhaps best regarded as a risk indicator rather than risk factor

Other Systemic Diseases

Several deficiencies of neutrophil function have been related to periodontal disease. These include:

- Chediak-Higashi syndrome
- Cyclic neutropenias
- Lazy leukocyte syndrome
- Agranulocytosis
- Leukocyte adhesion deficiency syndrome
- Down's syndrome
- Papillon Lefevre syndrome

Deficiencies in collagen production:

- Ehlers-Danlos syndrome

Intrinsic Risk Factors

Often termed - 'Non-modifiable' risk factors

Age

Introduction

- Prevalence and severity of periodontitis increases with age
- However periodontitis as an inevitable consequence of aging is likely incorrect and more likely the cumulative effect of exposure to true risk factors (Papapanou *et al.* 1991)

Genetics

Introduction

- Loe *et al.* (1986) were able to identify 3 subgroups – no progression of periodontal disease, moderate and rapid progression
- This phenomenon that a small population is at risk of the *most severe* forms of disease may suggest that not everybody is equally susceptible
- Although bacterial infection is the etiologic agent in periodontal disease, studies of identical twins suggest 50% of susceptibility to periodontal disease is due to host factors (Michalowicz *et al.* 2000)
- Similarly, indigenous and relatively isolated populations have been shown to develop distinct periodontal of periodontal disease that differ from group to group (Dowsett *et al.* 2001; Ronderos *et al.* 2001)
- Specific Il-1 genotypes have been linked to the presence of pathogenic microorganisms (Socransky *et al.* 2000), and to an increased risk of periodontal diseases in non-smokers (Kornman *et al.* 1997) and smokers (Meisel *et al.* 2002/3)
- Conversely, Meisel *et al.* 2002 have shown results that demonstrate no effect of Il-1 genotype in non-smokers

Hormone Influences

Introduction

- Hormones can affect the state of the periodontal tissues
 - e.g. Puberty, pregnancy and when using oral contraceptives
- Hormone-linked changes in the periodontium have been attributed to changes in the microbiota, immune functions and vascular properties (Mariotti 1994 & 2005)
 - A note on pregnancy: when the mouth is clean and does not have gingivitis there is no evidence to say that the patient will develop gingivitis as a result of pregnancy
 - Pregnancy can however increase the severity of pre-existing gingivitis