ORIGINAL RESEARCH PAPER

Oral Medicine

TOBACCO AND PERIODONTAL HEALTH: EVIDENCE BASED COMPREHENSIVE REVIEW

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Manas Shukla Associate Professor Department of Periodontics Rama Dental College, Hospital and Research Centre, Kanpur.

ABSTRACT

The effects of tobacco use on the population's oral health are alarming, and there is a clear link between tobacco use and oral health outcomes of the same. Oral cancer and periodontal disease are among the most serious oral health problems associated with tobacco use. It has been estimated that smoking accounts for half of all periodontal disease and 91% of oral cancers among males, and 59% of oral cancers among females.In this review article, the author shall present an evidence-based comprehensive understanding of the role of tobacco in periodontal health and disease, including gingivitis, etiology and pathogenesis of periodontal disease, response to periodontal therapy and refractory disease.

Introduction

There is overwhelming evidence that tobacco usage produces harmful effects in the mouth. Tobacco use has many negative effects on a person's oral health. All forms of tobacco use, both smoking and use of smokeless tobacco can significantly affect a person's oral health.

A number of oral diseases, including oral cancer, oral mucosal lesions, periodontal disease and impaired healing after periodontal treatment, gingival recession, and coronal and root caries have been enumerated with tobacco use as a risk factor. Oral cancer and periodontal disease are among the most serious oral health problems associated with tobacco use. Oral cancer is perhaps the most significant threat to a tobacco user's oral health.

The magnitude of the effect of tobacco on occurrence of oral disease is high; and the risk declines with increasing time after smoking cessation, with users having many times the risk as non-users. It has been estimated that smoking accounts for half of all periodontal disease and 91% of oral cancers among males, and 59% of oral cancers among females. Also, tobacco use leads to additional consequences for persons with oral cancer and periodontal disease (Winn DM, 2002).

A number of tobacco-related oral health conditions can be prevented from progressing, and some can even be reversed with proper knowledge of tobacco and oral health, as dentists are in a position of early diagnosis of most of the reversible conditions at the preventable stage (Johnson NW & Bain CA, 1999).

The role of smoking in periodontal diseases has been extensively studied for many years. Smoking has been identified as a major risk factor for periodontitis. The first reports relating periodontal diseases to smoking were evident in the 1950s. A major breakthrough was provided by three separate studies by Bergstrom J, Feldman RS, and Ismail AL in the year 1983.

1. Effects of smoking on prevalence and severity of periodontal disease:

i) Gingivitis

Controlled studies in human models of experimental gingivitis have shown that both, gingival inflammation and bleeding on probing in response to plaque accumulation are reduced in smokers compared with non-smokers (Bergstrom and Preber, 1986).

A number of cross-sectional studies have consistently suggested that smokers have a decreased expression of clinical inflammation in presence of plaque accumulation, as compared to non-smokers (Bergstrom and Preber, 1986; Haber & Kent, 1993).

Smoking adversely affected the blood flow to the gingiva, and there was thus reduced bleeding on probing; Clarke and Carey noted reduced blood flow up to2 to 3 hours after smoking 1 cigarette. These effects which suggest a lower bleeding propensity for smokers are due to the effect of nicotine exerting local vasoconstriction on peripheral circulation. Smoking does not increase the amount of plaque but there is definitely more calculus deposit on the teeth in smokers (Bergstrom, 2006).

ii) Periodontitis

Multiple cross-sectional and longitudinal studies have demonstrated that pocket depth, attachment loss and alveolar bone loss are more prevalent and severe in smokers compared with non-smokers.

In a systematic critical appraisal of the available literature conducted by Bergstrom J in 2006, in which 129 articles were selected, almost all the studies: cross-sectional, case-control and cohort - showed significant association between smoking and impaired periodontal health, using probing pocket depth and clinical attachment loss as the most frequently used end points. Almost 95% of the cohort studies in this review provide evidence of association between smoking and disease onset and progression, thus suggesting that deterioration of periodontal health condition over time is greater in smokers than non-smokers. The NHANES III survey in US on 12,000 patients showed that smokers were four times as likely to have periodontitis as persons who had never smoked. Results showed that 9.2% individuals had periodontitis, these represented almost 15 million cases of periodontitis in the U.S. Former smokers were 1.68 times more likely to have periodontitis than persons who had never smoked. Dose response relationship was observed between cigarettes smoked per day and the odds of having periodontitis. In subjects smoking 9 or fewer cigarettes per day, the odds for having periodontitis were 2.79, as against subjects smoking 31 or more cigarettes per day, having 6 times more risk. Similarly, other studies have shown consistent findings. The odds ratio for periodontitis in current smokers ranges from 1.5 to 7.3, depending on the observed severity of periodontitis (In Carranza FA, 2006).

iii) Severity of Periodontitis

Rate of periodontal destruction increased in smokers, and smoking was significantly associated with higher prevalence of moderate and severe periodontal disease (Albandar JM, 2000). Over a 10-year period, bone loss was reported to be twice as rapid in smokers as non-smokers and proceeds more rapidly even in the presence of excellent plaque control (In Carranza FA, 2006).

The detrimental impact of long-term smoking on periodontal and dentate status of older adults:

Older adult smokers are three times more likely to have severe periodontal disease (Beck et al 1990; Locker D and Leake JL1993); and number of years of tobacco use is a significant factor in tooth loss, coronal root caries and periodontal disease (Jette AM, Feldman HA 1993, In Carranza FA, 2006).

Smoking and periodontal disease severity in younger individuals:

Increased severity of generalized aggressive periodontitis is seen in younger patients. Those between 19 and 30 years of age who smoke are 3.8 times more likely to have periodontitis than non-

smokers (Schenkein et al 1995).

Longitudinal studies have shown that young individuals smoking more than 15 cigarettes per day showed highest risk for tooth loss. Smokers are more than 6 times as likely as non-smokers to demonstrate continuous attachment loss (In Carranza FA, 2006).

Effects of cigar and pipe smoking:
Effects are similar to cigarette smoking. Cigar / pipe smoking was associated with increased risk for experiencing tooth loss, and alveolar bone loss, but the elevations in risk are similar in magnitude to those observed in cigarette smokers. Studies have shown that severity of disease is intermediate between the current cigarette smokers and non-smokers (Krall and co-workers, 1999; Albandar JM, 2000).

Smokeless tobacco:
No generalized effects on periodontal disease progression seem to occur. Localized attachment loss and recession are seen at the site of tobacco placement (Robertson et al., 1990; Amaresena and co-workers, 2002).

Former smokers and cessation of smoking:
Former smokers are at less risk than current smokers, but more risk than non-smokers. Risk decreases with increasing number of years since quitting smoking. Effects on the host are reversible with smoking cessation (Tomar SL & Asma S, 2000).

iii) Acute Necrotizing Ulcerative Gingivitis:
Smokers have higher prevalence and severity of ANUG as compared to non-smokers. A similar relationship has been reported between smoking and ANUG-like lesions in HIV infected individuals (Johnson NW & Bain CA, 2002).

2. Etiology and Pathogenesis of periodontal disease:
i) Microflora and host response:
Studies have shown no difference in rate of plaque accumulation between smokers and non-smokers. Most of the studies have also failed to demonstrate any difference in the composition of sub-gingival plaque. One study reported higher subgingival levels of B.forsythus in smokers than non-smokers (Zambon et al., 1996).

Altered host response:
The greater prevalence and severity of disease in smokers might be explained by an altered host response. Smokers demonstrate -

- Downregulation of the immune response to bacterial challenge;
- Functional alterations in chemotaxis, phagocytosis, and oxidative burst of neutrophils;
- Decreased Immupoglobulin (IgG2) and cytokine levels, and lymphocyte counts.
- Increased tissue destructive enzymes – TNF , PGE2, MMP-8.

ii) Physiology:
Smokers show decreased clinical signs of inflammation, and alterations in vascular response and inflammatory response.
Local effects of smoking are:

- Less gingival bleeding and inflammation due to localized vasoconstriction induced by nicotine (Haber and Kent, 1993).
- Oxygen conc. in healthy gingival tissues appears to be less in smokers than in non-smokers, although this condition is reversed in the presence of moderate inflammation.
- Subgingival temperatures are less and recovery from vasoconstriction caused by local anaesthetic administration takes longer in smokers.
- With developing inflammation, increases in GCF flow, bleeding on probing, and gingival blood vessels were less in smokers than non-smokers.
- Smoking hampers the period of regression due to poor healing capacity (In Carranza FA, 2006).

3. Response to periodontal therapy:
Current smokers do not respond as well to periodontal therapy as non-smokers – both, non-surgical and surgical. Decreased levels of clinical attachment gain, and less reduction in pocket depth have been consistently reported. After surgical intervention, healing is impaired as nicotine can impair the attachment of fibroblasts to rot surface in vitro, and affects collagen synthesis and protein secretion. Smoking has also been shown to have a negative impact on the outcome of GTR, and treatment of intrabony defects with bone allografts (Bergstrom J, 2006).

Implants:
Tobacco is detrimental to both the initial and long-term success of dental implants, and smoking cessation can improve implant success rates. Smoking is by far the most important predisposing factor to implant failure (Bain CA, Moy PK, 1993). Significantly greater bleeding index, peri-implant pocket depth, peri-implant inflammation, and radiographically discernible bone loss was noticed around successfully integrated maxillary implants of smokers.

Maintenance therapy:
The detrimental effects of smoking on treatment outcomes are long-lasting and independent of the frequency of maintenance therapy (In Carranza FA, 2006).

4. Recurrent (refractory) disease:
Many smokers become refractory to traditional periodontal therapy, and tend to show more periodontal breakdown after therapy. 90% of patients with refractory disease were smokers (Mac Farlane et al., 1992).

Summary:
Smokers –

- Show increased prevalence and severity of periodontal disease.
- May present with periodontitis at an early age.
- May be difficult to treat with conventional therapy.
- May continue to have progressive and recurrent periodontitis leading to tooth loss.
- Smoking cessation is associated with improved response to periodontal therapy and reverses host changes involved in the development of periodontal disease.

Conclusion:
The epidemic of tobacco use is one of the greatest threats to global health. The contribution of tobacco use to oral diseases is immense, but it is a major preventable factor, which can be controlled through effective intervention through both individual and public health approach.

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