Smoking And Periodontal Health

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Abstract : Tobacco smoking is very common with cigarettes being the main product smoked. Most smokers start the habit as teenagers, with the highest prevalence in the 20-24 year old age group. Socioeconomic differences also exist with higher smoking in the lower socioeconomic groups. [Nanavati B et al NJIRM 2013; 4(4) : 129-134]

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Introduction: Smoking is associated with a wide spectrum of disease including stroke, coronary artery disease, peripheral artery disease, gastric ulcer, cancers of mouth, larynx, oesophagus, pancreas, bladder and uterine cervix. Smoking is also a major cause of chronic obstructive pulmonary disease and a risk factor for low birth weight babies.[1]

Smoking is an important environmental risk factor for periodontitis[2.] Periodontitis (pyorrhoea) is defined of the supporting tissues of the teeth. Usually a progressively destructive change leading to loss of bone and periodontal ligament. An extension of inflammation from gingiva into the adjacent bone and ligament[3] Smoking has been associated with an increased prevalence and severity of periodontal diseases, and there is evidence that a greater proportion of patients with periodontitis are smokers[4]. Smoking has been shown to reduce the effectiveness of both non-surgical and surgical treatment[5]. Quitting smoking may have beneficial effects on periodontal status[6.]

Toxicity Of Tobacco Smoke: Cigarette smoke is a very complex mixture of substances with over 4000 unknown constituents. These include carbon monoxide, hydrogen cyanide, reactive oxidizing radicals, a high number of carcinogens and the main psychoactive and addictive molecule – nicotine[7]. Tobacco smoke has a gaseous phase and solid phase which contains tar droplets. The tar and nicotine yields of cigarettes have been reduced due to physical characteristics of filters[8.] There has been a little change in tar and nicotine content of the actual tobacco and dose an individual receives is largely dependent upon the way in which they smoke[8.]

The patient exposure to tobacco smoke can be measured in a number of ways, including interviewing the subject and biochemical analysis[9]. These include exhaled carbon monoxide in the breath and cotinine (metabolite of nicotine) in saliva, plasma, serum or urine[10]. Cotinine measurements are more reliable in determining a subject's exposure to tobacco smoke because the half life is 14-20 hours compared with shorter half life of nicotine which is 2-3hours[11]. The mean plasma and salivary cotinine concentration of regular smokers are approximately 300ng/ml and urine concentrations are about 1500ng/ml. Non smokers have plasma/saliva concentration under 2ng/ml. While smokers have a plasma concentration of 24.3ng/ml[12].

Inhalation of tobacco smoke allows very rapid absorption of nicotine into the blood and transport to the brain, which is faster than an intravenous infusion. Nicotine in tobacco smoke from most cigarettes is not well absorbed through oral mucosa as it is in ionized form with a pH of 5.5. Cigar and pipe smoke is more alkaline, which allows good absorption of un-ionized nicotine through the buccal mucosa.

When a cigarette is smoked, nicotine-rich blood passes from the lungs to the brain in 7 seconds. After that, there is immediate release of neurotransmitters along with hormones, which are responsible for nicotine's effects. These constituents modify host response in periodontitis [7].Inhalation of tobacco smoke allows very rapid absorption of nicotine into the blood and transport to the brain, which is faster than an intravenous infusion[13.] Spilling a high concentration of nicotine onto the skin can cause intoxication or even death,

since nicotine passes into the bloodstream readily following dermal contact.[15]

Classification Of Smokers:

A. The *criteria* established by Centres for Disease Control and Prevention, defines

i. "Current Smoker "as those who had smoked ≥100 cigarettes over their lifetime and smoked at the time of the interview.

ii. "Former Smokers "had smoked ≥100 cigarettes in their lifetime but were not currently smoking.

iii. "Non Smokers "not smoked ≥100 cigarettes in their lifetime.

B. According to number of cigarettes smoked /day smokers can be classified as :

I. Heavy smokers-smoked ≥20 cigarettes/day
Light smokers-smoked ≤19 cigarettes/day

Effect Of Smoking On Prevalence And Severity Of Periodontal Disease.

75% of periodontitis cases were due to smoking among smokers

Smokers have

1. Deeper probing depths and a large number of deep pockets
2. More attachment loss including more gingival recession.
3. More alveolar bone loss. More tooth loss
4. Less gingivitis and less bleeding on probing
5. More teeth with furcation involvement

Smoking And Gingival Inflammation: Pindborg (1947) was one of the first investigators to study the relationship between smoking and periodontal disease. Early studies showed that smokers had higher levels of periodontitis but they also had poorer levels of oral hygiene[16] and higher levels of calculus. Later studies which took account of oral hygiene status and employed more sophisticated statistical analyses showed that smokers had more disease regardless of oral hygiene [17].Smoking may result in lower resting Gingival crevicular fluid flow rate[18] and episode of smoking may produce a transient increase in GCF flow rate[19]. The reduced bleeding on the other hand is probably due to long-term effects on the inflammatory lesion. Histological comparisons of the lesions from smokers and non smokers and non-smokers have shown fewer blood vessels in the inflammatory lesions of smokers [20]. It is pertinent to note that gingival bleeding on probing has been shown to increase within 4-6 weeks of quitting smoking[21].
In gingival inflammation, there is dilatation of capillaries and increase of blood flow. Inflammatory infiltrate also increases, which clinically brings about enlargement of gingival. Followed by an increase in lymphocytes and macrophages.

When the patient is a smoker, there is reduction in clinical signs of gingivitis. Heavy smokers may have greyish discoloration and hyperkeratosis of the gingiva. An increased number of keratinized cells have been found in smokers. Smoking is a major risk factor in the development of forms of aggressive periodontitis that are characterized by generalized and localized rapid loss of periodontal support.

Smoking And Oral Microorganisms : Studies have failed to demonstrate a difference in the rate of plaque accumulation of smokers compared with non-smokers, suggesting that if an alteration in microbial challenge in smokers exists, it is due to a qualitative rather than quantitative alteration in the plaque[22]

Several studies have shown that smokers harbor more bacterial species which are associated with periodontitis including Porphyromonas gingivalis, Actinobacillus actinomycetum comitans, Bacteriodes forsythus[23], Prevotella intermedia, Peptostreptococcus micros, Fusobacterium nucleatum, Campylobacter rectus[24,] Staphylococcus aureus, Escheria coli and Candida albicans [25], than in non smokers. Smokers may have a higher proportion of sites harboring these putative periodontal pathogens, in particular the palatal aspects of the maxillary teeth and the upper and lower incisor regions. Cross sectional data from the large Erie county study population demonstrated that the proportion of subjects' positive for Actinobacillus actinomycetum comitans, Porphyromonas gingivalis and

Bacteriodes[26] forsythus was significantly higher among current smokers than former or never smokers. Current smokers were 3.1 times more likely to be infected with Actinobacillus actinomycetum comitans or 2.3 times more likely to be positive for Bacteriodes forsythus than former or never smokers.

Of particular interest is that smokers do not respond to mechanical therapy as well as non-smokers and this is associated with increased levels of Bacteriodes forsythus, Actinobacillus actinomycetum comitans and Porphyromonas gingivalis remaining in the pocket after therapy in the smoking group when compared with non-smokers.

Smoking And Host Response : Smoking has a profound effect on the immune and inflammatory system[27]. Smokers have an increased number of leukocytes in the systemic circulation, but fewer cells may migrate into the gingival crevice/pocket. Smoking is associated with chronic obstructive pulmonary disease[28] and many of the mechanisms indicated are paralleled in findings related to periodontal disease.

Studies *in vitro* have shown a direct inhibition of neutrophil and monocyte-macrophage defensive functions by high concentrations of nicotine that may be achieved in patients using smokeless tobacco[29]. Patients were examined with refractory periodontitis and a high proportion of smokers were found in this diagnostic group[30]. These investigators demonstrated abnormal PMN phagocytosis associated with a high level of cigarette smoking. The PMN is a fundamental defense cell in the periodontal tissue. There is constant traffic of PMNs from the gingival vasculature through the connective tissue and junctional epithelium into the gingival sulcus/pocket. The PMN contains a powerful battery of enzymes including elastase and other collagenases that have been implicated in tissue destruction in periodontitis and pulmonary disease. There is decreased PMN migration into the oral cavity of smokers[31]. Subsequently, PMNs harvested from the gingival sulcus of smokers were shown to have reduced phagocytic capacity compared to PMNs from non-smokers [32].

The effect of smoking on lymphocyte function and antibody production is very complex, with the various components having the potential to cause immunosuppression or stimulation. Smoking appears to affect both T cell subsets report different findings of either reduced, increased or no change in number of CD4T cells [33]. Smoking appears to affect both B and T cell function, inducing functional unresponsiveness in T cells. It has been reported that serum IgG levels in smokers may be reduced[34] with depression of IgG2, particularly in some racial groups[35]. Reported levels of serum IgA and IgM classes are variable and IgE may be elevated [36].

Effects Of Cigarette Smoking Healing And Periodontal Therapy: Smoking has been identified as an important cause of impaired healing in orthopedic surgery, plastic surgery, dental implant surgery[37] and in all aspects of periodontal treatment including non-surgical treatment, basic periodontal surgery, regenerative periodontal surgery and mucogingival, plastic periodontal surgery[38].

In non-surgical treatment, smoking is associated with poorer reductions in probing depth and gains in clinical attachment. The poorer reductions in probing depths and gains in attachment level amount to a mean of approximately 0.5 mm. Much of this may be due to less recession of the marginal tissues in smokers as there is less edema and more fibrosis in the gingiva[39]. However, the response following periodontal surgery is more complex and involves an initial inflammatory reaction followed by organization of the clot, formation of granulation tissue consisting of capillary buds and fibroblasts laying down collagen. The surgical flaps have to revascularize and the epithelial attachment has to reform on the surface. In regenerative surgery there also has to be formation of a connective tissue attachment and cementogenesis. Tobacco smoke and nicotine undoubtedly affect the microvasculature, the fibroblasts and connective tissue matrix, the bone

and also the root surface itself. It has been shown in *in vitro* studies that fibroblasts are affected by nicotine in that they demonstrate reduced proliferation, reduced migration and matrix production and poor attachment to surfaces[40]. The root surfaces in smokers are additionally contaminated by products of smoking such as nicotine and cotinine and these molecules may affect the attachment of cells[40]. Smoking has a direct effect on bone and is an established risk factor in osteoporosis. It has also been proposed that it may have a direct effect on bone loss in periodontitis[41] and it undoubtedly delays healing of bone in fracture wound repair. It is not surprising therefore that tobacco smoking has been implicated in poorer responses to periodontal surgical treatment.

Several studies have shown that implant success rates are reduced in smokers[37], whereas other studies have shown no effect[42].

Periodontal Effects Of Tobacco Cessation: All patients should be assessed for smoking status and given advice to quit the habit. About 70% of people who smoke would like to quit and should be assisted. They should be referred to specialist cessation services if the treating practitioner does not feel confident in this area. They can be advised about nicotine replacement therapy. People's success with quitting is considerably improved using nicotine replacement therapy and drugs such as buproprion hydrochloride.

References:

1. Mary B. Meyer, Bruce S. Jonas, James A TonasciaPerinatal events associated with maternal smoking during pregnancy. American journal of epidemiology. 1976 1103(5):464-476.
2. Glossary of periodontal terms, American academy of periodontology, 2004
3. Palmer et al 2005 Mechanisms of action of environmental factors – tobacco smoking. J Clin Periodontol. 2005;32 Suppl 6:180-95
4. Tomar SL, Asma S . Smoking-attributable periodontitis in the United States: findings from NHANES III. National Health and Nutrition Examination Survey. J Periodontol 2000 71:743–751.
5. Labriola et al. 2005, Bergstrom 2006, Heasman et al. 2006
6. Bergstrom et al. 2000, Brothwell 2001, Johnson & Hill 2004, Preshaw et al. 2005, Thomson et al. 2007). Journal of Periodontology August 2000, Vol. 71, No. 8, Pages 1338-1347
7. Benowitz Pharmacology of nicotine: addiction and therapeutics. Annual Reviews in Pharmacology Toxicology 1996 36, 597-613
8. Benowitz Health and public policy implications of the low yield cigarette. New England Journal of Medicine 1989 320, 1619-1621
9. Scott, D.A., Palmer, R.M. & Stapleton, J.A.. Validation of smoking status in clinical research into inflammatory periodontal disease: a review. Journal of Clinical Periodontology 2001 28, 712-722.
10. Wall, M.A., Johnson, J., Jacob, P. & Benowitz, NL. Cotinine in the serum, saliva and urine of nonsmokers, passive smokers and active smokers. American journal of Public Health 1988 78, 699-701.
11. Jarvis, M.J., Russell, M.A.H., Benowitz, N.L. & Feyerabend, C. Elimination of cotinine from body fluids: Implications for non-invasive measurement of tobacco smoke exposure. American Journal of Public Health 1988 78, 696-698.
12. Venzo teneggi : correlation and predictive performance of saliva and plasma nicotine conc on tobacco withdrawal induced carving, British journal of clinical pharmacology, October 2002Volume 54, Issue 4, pages 407–414
13. Benowitz, N.L. Pharmacological aspects of cigarette smoking and nicotine addiction. New England Journal of Medicine 1988 319, 1318-1330
14. Jarvis, M.J., Russell, M.A.H., Benowitz, N.L. & Feyerabend, C. Elimination of cotinine from body fluids: Implications for non-invasive measurement of tobacco smoke exposure. American Journal of Public Health 1988 78, 696-698

15. Lockhart LP. Nicotine poisoning (letter). Br Med J 1933 1:246247
16. Grossi, S.G., Zambon, J.J., Ho, A.W. et al. Assessment of risk for periodontal disease. I. Risk indicators for attachment loss. Journal of Periodontology 1994 65, 260-267.
17. Ismail A.I., Burt, B.A. & Eklund, S.A. Epidemiologic patterns of smoking and periodontal disease in the United States. Journal of the American Dental Association 1983 106, 617-621.
18. James, J.A., Sayers, N.S., Drucker, D.B. & Hull, P.S. Effects of tobacco products on the attachment and growth of periodontal ligament fibroblasts. Journal of Periodontology 1999 70, 518- 525.
19. Persson, L., Bergstrom, J., Gustafsson, A. & Asman, B. Tobacco smoking and gingival neutrophil activity in young adults. Journal of clinical periodotology 1999 26, 9-13.
20. Mclaughlin, W.S., Lovat, F.M., Macgregor, I.D. & Kelly, P.J. The immediate effects of smoking on gingival fluid flow. Journal of clinical periodontology 1993 20, 448-451
21. Rezavandi, K., Palmer, R.M., Odell, E.W., Scott, D.A. & Wilson, R.F. Expression of E-Selectin and ICAM-1 in gingival Tissues of smokers and non-smokers with periodontitis. Journal of Oral Pathology and Medicine 2001 31, 59-64.
22. Bergstrom, J. Cigarette smoking as a risk factor in chronic periodontal disease. Community Dentistry and Oral Epidemiology 1989 17, 245-247.
23. Zambon, J.J., Grossi, S.G., Machtei, E.E., Ho, A.W., Dunford, R. & Genco, R.J. Cigarette smoking increases the risk for subgingival infection with periodontal pathogens. Journal of Periodontology 1996 67, 1050-1054.
24. Van Winkelhoff. A.J., Bosch-Tijhof, C.J., Winkel, E.G. & van der Reijden, W.A. Smoking affects the subgingival microflora in periodontitis. Journal of Periodontology 2001 72, 666-671.
25. Kamma, J.J., Nakou, M. & Baehni, P.C. Clinical and microbiological characteristics of smokers with early onset periodontitis. Journal of Periodontal Research 1999 34, 25-33.
26. Haffajee, A.D. & Socransky, S.S. . Relationship of cigarette smoking to the subgingival microbiota. Journal of Clinical Periodontology 2001b 28, 377-388.
27. Barbour, S.E., Nakashima, K., Zhang, J.B. et al. Tobacco and smoking: environmental factors that modify the host response (immune system) and have an impact on periodontal health. Critical Reviews in Oral Biology & Medicine 1997 8, 437-460.
28. Barnes, P.J. Chronic obstructive pulmonary disease. New England Journal of Medicine 2000 343, 269-280.
29. Pabst, M.J., Pabst, K.M., Collier, J.A. et al. Inhibition of neutrophil and monocyte defensive functions by nicotine. Journal of Periodontology 1995 66, 1047-1055.
30. MacFarlane, G.D., Herzberg, M.C., Wolff, L.F. & Hardie, N.A. Refractory periodontitis associated with abnormal polymorphonuclear leukocyte phagocytosis and cigarette smoking. Journal of Periodontology 1992 63, 908-913. Machuca, G., Khoshfeiz, 0., Lacalle, J.R., Machuca, C. & Bullon, P. The influence of general health and socio-cultural variables on the periodontal condition of pregnant women. Journal of Periodontology 1999 70, 779-785.
31. Eichel, G. & Shahrik, H.A. (1969). Tobacco smoke toxicity: loss of human oral leucocyte function and fluid cell metabolism. Science 1969 166, 1424-1428.
32. Kenny, E.B, Kraal, J.H., S.R. & Jones, J. The effect of cigarette smoke on human oral polyorphonuclear leukocytes. Journal of Periodontal Research 1977 12, 227-234.
33. Loos, B.G., Roos, M.T., Schellekens, P.T., van der Velden, U. & Miedema, F. Lymphocyte numbers and function in relation to periodontitis and smoking, Journal of periodontology 2004 75, 557-564.
34. Quinn, S.M., Zhang, J.B., Gunsolley, J.C., Schenkein, H.A. & Tew, J.G. The influence of smoking and race on adult periodontitis and serum 1gG2levels. Journal of Periodontology 1998 69, 171-177.
35. Quinn, S.M., Zhang, J.B, Gunsolley, J.C., Schenkein, J.G., Influence of smoking and race on immunoglobulin G subclass concentrations in early-onset periodontitis patients . Infection and immunity 1996 64, 2500-2505.

36. Burrows, B., Halonen, M., Barbee, R.A. & Lebowitz, M.D. The relationship of serum immunoglobulin E to cigarette smoking. American Review of Respiratory Disease 1981 124, 523-525
37. Bain, c.a. & moy, p.k. The association between the failure modifying factors: diabetes, puberty, pregnancy and the menopause and tobacco smoking • 193 of dental implants and cigarette smoking. International journal oral & maxillofacial implants 1993 8, 609-615.
38. Preber, H. & Bergstrom, J. The effect of non-surgical treatment on periodontal pockets in smokers and non-smokers. Journal of Clinical Periodontology 1986 13, 319-323.
39. Biddle, A., Palmer, R.M., Wilson, R.F. & Watts, T.L.P. Comparison of periodontal probing measurements in smokers and non-smokers. Journal cif Clinical Periodontology 2001 28, 806-812.
40. Raulin, L.A., McPherson, J.C., McQuade, M.J. & Hanson, B.S. The effect of nicotine on the attachment of human fibroblasts to glass and human root surfaces in vitro. Journal of Periodontology 1988 59, 318-325.
41. Bergström J, Eliasson S, Preber H. Cigarette smoking and periodontal bone loss. J Periodontol. 1991 Apr;62(4):242-6. Erratum in: J Periodontol 1991 Dec;62(12):809.
42. Minsk L, Polson AM, Weisgold A, Rose LF, Sanavi F, Baumgarten H, Listgarten MA. Outcome failures of endosseous implants from a clinical training center. Compend Contin Educ Dent. 1996 Sep;17(9):848-50, 852-4, 856 passim.

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