Research Article

Prevalence of gingival recession in smokers and non-smokers - A cross-sectional study

S. Nandhana[1], Deepa Gurunathan[2]*, M. Thamaraiselvan[3]

ABSTRACT

Introduction: Gingival recession is associated with oral exposure of the root surface due to a displacement of the gingival margin apical to the cementoenamel junction. Recession defects are one of the most common findings in periodontal problems affecting individuals of almost all ages to some degree. Smoking is one of the most important risk factors for periodontitis that affects the prevalence, extent, and severity of disease. The aim of this study is to assess whether smoking has an impact on gingival recession. **Materials and Methods:** Twenty-seven smokers and 21 non-smokers who reported to outpatient were examined and their periodontal status is estimated using a pro forma containing patient details, their chief complaint, smoking habits, loss of attachment, and probing depth with a column for patient consent. **Results:** According to data analysis, smokers group had a mean gingival recession of 25%, whereas non-smokers had 13%. $P = 0.009$ was obtained by unpaired independent sample *t*-test and it shows that the results obtained in this study were statistically significant. **Conclusion:** Based on the results found in this study, we conclude that smoking has its own effects on periodontium. However, further, researches should be conducted to establish the harmful effects of smoking on gingiva, thereby enhancing tobacco cessation to prevent further complications.

KEY WORDS: Gingival recession, Marginal gingiva, Periodontitis, Pockets, Root exposure

INTRODUCTION

Gingival recession is the most common and undesirable condition of the gingiva. Gingival recession in anterior teeth usually creates an esthetic problem and anxiety about tooth loss among the patients. Surgical treatments such as free graft and pedicle flap are indicated when the gingival recession causes functional or esthetic problems.[1] The gingival recession can be defined as migration of the gingival margin apical to the cementoenamel junction and exposure of the root surface.[2] The presence of supragingival calculus and cigarette smoking was strongly associated with the incidence and prevalence of localized and generalized gingival recession.[3] Gingival recession is also associated with dentin hypersensitivity, root caries, abrasion, cervical wear, erosion due to exposure of the root surface to the oral environment and an increase in accumulation of dental plaque.[4] The etiology of gingival recession is based on multiple factors such as excessive or inadequate teeth brushing, destructive periodontal disease, tooth malposition, alveolar bone dehiscence, high muscle attachment, frenum pull, and occlusal trauma.[5] Iatrogenic factors such as orthodontic and prosthodontic treatment can also lead to gingival recession.[6] However, among these factors, smoking is one of the most important factors associated with gingival recession. The chemicals present in cigarettes affect the function of neutrophils, thereby preventing the elimination of periodontal pathogens causing recession. It can also stimulate oxygen species release and oxidative stress-mediated tissue damage.[7] Increasing evidence displays smoking as a major risk factor for periodontal disease, affecting the prevalence, extent, and severity of disease. In addition, smoking may influence the clinical outcome of non-surgical and surgical therapy, as well as the long-term success of implant placement.[8] The response of the microcirculation to plaque accumulation appears to be altered in smokers

Access this article online	
Website: jprsolutions.info	ISSN: 0975-7619

[1]Department of Undergraduate Student, Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, Tamil Nadu, India, [2]Department of Pedodontics, Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, Tamil Nadu, India, [3]Department of Periodontics, Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, Tamil Nadu, India

***Corresponding author:** Deepa Gurunathan, Department of Pedodontics, Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, 162, Poonamallee High Road, Chennai - 600 077, Tamil Nadu, India. Phone: +91-9994619386. E-mail: drgdeepa@yahoo.co.in

Received on: 18-08-2018; Revised on: 25-09-2018; Accepted on: 23-10-2018

when compared with non-smokers.[9] With developing inflammation, increases in gingival blood vessels were lower in smokers than in non-smokers.[10] In addition to this, the oxygen concentration level in healthy gingival tissues appears to be lower in smokers than in non-smokers, although this condition is reversed in the presence of moderate inflammation.[11] Of great interest is the observation that former smokers have less risk for periodontitis than current smokers, but more risk than non-smokers, and that the risk for periodontitis decreased as the number of years since smoking cessation increased.[12] A studies have shown[9,13] that smoking exhibited a decrease in several pro-inflammatory cytokines, chemokines, certain regulators of T-cells, and natural killer cells, thereby reflecting its immunosuppressant effects which lead to gingival recession and periodontitis.[13] This study has been performed to analyze whether smoking has an impact on gingival recession.

MATERIALS AND METHODS

Ethical Consideration

This study was performed following the ethical approval of the institution (SIMATS) and conducted following after obtaining informed consent from the subjects participating in this study. The participants were explained about the clinical evaluation before proceeding with the study.

Sample Size

A total of 48 male patients who reported to Saveetha Dental College, Chennai, aged between 35 and 50 years, of which 27 were smokers and 21 were non-smokers. Patients were randomly selected based on the following criteria.

This study included male patients over 35 years of age. Patients who had undergone scaling or any periodontal treatment were not included in this study. Teeth with periodontal pockets were excluded from this study. Based on the habit of smoking, patients were divided into two groups; Group 1: Smokers and Group 2: Non-smokers.

Clinical Examination

The clinical examination was carried out by making the patient sit in the dental chair and gingival recession was evaluated done using mouth mirror and William's probe. A tooth was noted to have gingival recession if it has a probing depth of 1–3 mm with loss of attachment.

The percentage of gingival recession in an individual was calculated by taking the percentage of number of teeth with recession from the number of teeth present in the corresponding individual. Statistics was performed by taking the mean value of recession in smokers and non-smokers by categorizing non-smokers as Group 1 and smokers as Group 2.

Statistics was obtained using independent unpaired sample *t*-test.

Evaluation Sheet

PATIENT NAME :

AGE :

GENDER :

ADDRESS :

OCCUPATION :

CHIEF COMPLAINT :

SMOKER / NON- SMOKER :

PERIODONTAL FINDINGS

LOSS OF ATTACHMENT :

18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28
48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38

PROBING DEPTH :

18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28
48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38

DIAGNOSIS :

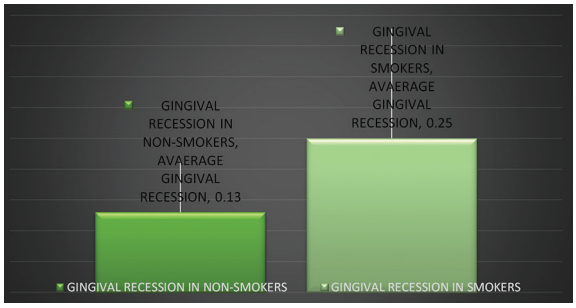
PATIENT CONSENT :

RESULTS

From the statistical analysis, of 21 non-smokers, 13% were diagnosed with gingival recession, and of 27 smokers, 25% were diagnosed with gingival recession.

Group statistics

Recession	Groups	*n*	Mean	Standard deviation	Standard error mean
	1.00	21	0.13	0.086	0.019
	2.00	27	0.25	0.178	0.034



DISCUSSION

Smoking is a known risk factor for many diseases and more and more studies suggest that smoking adversely affects periodontal health.[14] Smokers have been associated with deeper pockets and greater attachment loss, increased radiographic evidence of furcation involvement, and increased alveolar bone loss. Several clinical and epidemiological studies indicate that cigarette smoking has harmful effects on the response

to a variety of non-surgical[15] and surgical procedures including modified Widman flap surgery,[16] guided tissue regeneration,[17] dental implants,[18] and supportive periodontal treatment.[19] Smoking also adversely affects the neutrophils and macrophages, which are crucial as gingival immunocompetent cells. Especially, smoking impairs neutrophils chemotaxis and/or phagocytosis.[20] It has an immunosuppressive effect on the host, severely affecting host-bacterial interactions, and this change may be due to changes in the composition of subgingival plaque. It also provides a conducive environment for some of the periodontopathic species in the plaque and may be one reason why smoking is a risk factor in periodontal disease development.[21] It exerts a strong, chronic, and dose-dependent suppressive effect on gingival bleeding on probing. Smokers displayed less marked gingival inflammatory reaction when compared to non-smokers. The decline in the clinical inflammatory signs in smokers can be attributed to the cotinine, a nicotine metabolic by-product which has a peripheral constrictive action on gingival blood vessels.[22] By way of the vascular and immunological response of the body, smoking is thought to bring its changes in the periodontium. The effects of smoking on periodontium are reflected through the histologic and morphological changes in the gingiva.

Hedin *et al.*[23] stated that smoking is detrimental to periodontal health as it worsens the oral hygiene status and depresses the host's defense mechanism. Smoking tobacco affects the oral environment, inflammatory response; the gingival tissues and its vasculature response of the immune system and the homeostasis and healing potential of the periodontal connective tissues.

It is evident that gingival recession is more prevalent in patients with periodontal diseases and smokers. The higher rate of gingival recession in smokers compared to non-smokers in our study is in accordance with other studies. The significant relationship between gingival recession and both age and duration of smoking in our study is consistent with the study was done by Müller *et al.*[24] and Bokor-Bratic.[25] In a study was done by Checchi *et al.*[26] comparing Norway academicians and tea laborers, gingival recession was more frequent in laborers, which implies that it is less frequent in educated people such as those included in our study. The higher rate could be due to a fall in gingival crevicular fluid, less bleeding on probing, and also fewer gingival blood vessels, which is common during smoking. The other reason could be an increase in colonization of periodontal pathogens both in shallow and deep periodontal pockets. Alteration in immune response such as altered neutrophil chemotaxis, phagocytosis, and an increase in the production of prostaglandin E2 by monocytes in response to lipopolysaccharide is also a contributing factor. However, rapid tissue destruction caused in the immune system as the effect of smoking mechanism is still unclear and yet to be discovered.[26] Further studies are needed to define the effects of tobacco use on the immune response and tissue destruction in periodontitis.

A cross-sectional study containing 50 smokers and 55 non-smokers of age 21–36 conducted by Kazem Fatami *et al.* on effect of smoking on gingival recession revealed that gingival recession was greater in smokers (1.12 mm) compared to non-smokers (0.36 mm), which was significant ($P < 0.001$). This study also showed that there was a positive relationship between gingival recession and the number of cigarettes used per day, duration of use, and plaque index. There was a negative significant relationship between gingival recession and plaque control method, as well as educational level.[27]

CONCLUSION

From the results obtained in our study, we conclude that smoking has some deleterious effects on gingiva which leads to gingival recession which may eventually can lead to periodontal diseases. We also conclude that smoking plays a major role in causing gingival recession when compared to other factors that contribute to gingival recession in non-smokers. However, periodontal problem caused by the influence of smoking is reversible on cessation of smoking. Thus, smoking cessation programs should be an integral component of periodontal education and therapy.

Recommendations

It is essential to conduct research with greater bigger sample size and to evaluate gingival recession among current smokers, former smokers, and non-smokers are recommended.

REFERENCES

1. Thamaraiselvan M, Elavarasu S, Thangakumaran S, Gadagi JS, Arthie T. Comparative clinical evaluation of coronally advanced flap with or without platelet rich fibrin membrane in the treatment of isolated gingival recession. J Indian Soc Periodontol 2015;19:66-71.
2. Banihashemra SA, Fatemi K, Najafi MH. Effect of smoking on gingival recession. Dent Res J 2008;5:1-4.
3. Susin C, Haas AN, Oppermann RV, Haugejorden O, Albandar JM. Gingival recession: Epidemiology and risk indicators in a representative urban Brazilian population. J Periodontol 2004;75:1377-86.
4. Tugnait A, Clerehugh V. Gingival recession-its significance and management. J Dent 2001;29:381-94.
5. Kundapur PP, Bhat KM, Bhat GS. Association of trauma from occlusion with localized gingival recession in mandibular anterior teeth. Dent Res J (Isfahan) 2009;6:71-4.
6. Greenwell H, Fiorellini J, Giannobile W, Offenbacher S, Salkin L, Townsend C, *et al.* Oral reconstructive and

corrective considerations in periodontal therapy. J Periodontol 2005;76:1588-600.
7. Matthews JB, Chen FM, Milward MR, Wright HJ, Carter K, McDonagh A, *et al.* Effect of nicotine, cotinine and cigarette smoke extract on the neutrophil respiratory burst. J Clin Periodontol 2011;38:208-18.
8. Chambrone L, Chambrone D, Lima LA, Chambrone LA. Predictors of tooth loss during long-term periodontal maintenance: A systematic review of observational studies. J Clin Periodontol 2010;37:675-84.
9. Tymkiw KD, Thunell DH, Johnson GK, Joly S, Burnell KK, Cavanaugh JE, *et al.* Influence of smoking on gingival crevicular fluid cytokines in severe chronic periodontitis. J Clin Periodontol 2011;38:219-28.
10. Labriola A, Needleman I, Moles DR. Systematic review of the effect of smoking on nonsurgical periodontal therapy. Periodontol 2000 2005;37:124-37.
11. Wan CP, Leung WK, Wong MC, Wong RM, Wan P, Lo EC, *et al.* Effects of smoking on healing response to non-surgical periodontal therapy: A multilevel modelling analysis. J Clin Periodontol 2009;36:229-39.
12. Sheiham A, Watt RG. The common risk factor approach: A rational basis for promoting oral health. Community Dent Oral Epidemiol 2000;28:399-406.
13. Bergström J, Eliasson S. Noxious effect of cigarette smoking on periodontal health. J Periodontal Res 1987;22:513-7.
14. Taltia A, Arjunkumar R. Assessment of gingival thickness in smokers and non-smokers a clinical study. Int J Pharm Clin Res 2016;8:574-7.
15. Preber H, Bergström J. The effect of non-surgical treatment on periodontal pockets in smokers and non-smokers. J Clin Periodontol 1986;13:319-23.
16. Preber H, Bergström J. Effect of cigarette smoking on periodontal healing following surgical therapy. J Clin Periodontol 1990;17:324-8.
17. Tonetti MS, Pini-Prato G, Cortellini P. Effect of cigarette smoking on periodontal healing following GTR in infrabony defects. A preliminary retrospective study. J Clin Periodontol 1995;22:229-34.
18. Lambert PM, Morris HF, Ochi S. The influence of smoking on 3-year clinical success of osseointegrated dental implants. Ann Periodontol 2000;5:79-89.
19. Kaldahl WB, Johnson GK, Patil KD, Kalkwarf KL. Levels of cigarette consumption and response to periodontal therapy. J Periodontol 1996;67:675-81.
20. Eichel B, Shahrik HA. Tobacco smoke toxicity: Loss of human oral leukocyte function and fluid-cell metabolism. Science 1969;166:1424-8.
21. Kazor C, Taylor GW, Loesche WJ. The prevalence of BANA-hydrolyzing periodontopathic bacteria in smokers. J Clin Periodontol 1999;26:814-21.
22. Danielsen B, Manji F, Nagelkerke N, Fejerskov O, Baelum V. Effect of cigarette smoking on the transition dynamics in experimental gingivitis. J Clin Periodontol 1990;17:159-64.
23. Hedin CA, Ronquist G, Forsberg O. Cyclic nucleotide content in gingival tissue of smokers and non-smokers. J Periodontal Res 1981;16:337-43.
24. Müller HP, Stadermann S, Heinecke A. Gingival recession in smokers and non-smokers with minimal periodontal disease. J Clin Periodontol 2002;29:129-36.
25. Bokor-Bratić M. Effects of smoking on the periodontium. Med Pregl 2002;55:229-32.
26. Checchi L, Daprile G, Gatto MR, Pelliccioni GA. Gingival recession and toothbrushing in an Italian school of dentistry: A pilot study. J Clin Periodontol 1999;26:276-80.
27. Banihashemrad SA, Fatemi K, Najafi MH. Effect of smoking on gingival recession. Dent Res J 2008;5:1-4.

Source of support: Nil; Conflict of interest: None Declared