Available online at www.ijpcr.com
International Journal of Pharmaceutical and Clinical Research 2016; 8(6): 574-577

ISSN- 0975 1556

Research Article

Assessment of Gingival Thickness in Smokers and Non-Smokers –A Clinical Study

Ankita Taltia*, Radhika, Arjunkumar

Saveetha Dental College 162 Poonammallee High road Velappanchavadi, Chennai-600077, India

Available Online: 8th June, 2016

ABSTRACT

Smoking has its effects on the periodontium, which is reflected by morphologic and histologic changes in the gingiva. Gingival thickness is one among the factors, which has become a subject of considerable interest in periodontics both from the epidemiological and therapeutic point of view. By taking gingival thickness into consideration during treatment planning, more appropriate strategies for periodontal management may be developed, resulting in more predictable treatment outcomes. Purpose of the Study: The purpose of the study is to comparatively assess the gingival thickness among smokers and non-smokers. Methods: The study group included 30 age matched smokers and non-smokers whose gingival thickness was measured in the maxillary anterior teeth by trans-gingival probing mid-buccally in the attached gingiva and at the base of the interdental papilla. Plaque index and sulcus bleeding index were recorded. The data was statistically analysed using paired t-test and Z-test. Results and Conclusion: Sulcus bleeding index and plaque index were similar between both the groups. Gingiva is similar in thickness in interdental areas and midbuccal areas both in smokers and non-smokers. Both midbuccal and interdental areas were thicker among smokers when compared to non-smokers.

Keywords: smoking, gingival thickness, smokers, non-smokers

INTRODUCTION

Smoking is a known risk factor for many diseases and more and more studies suggests that smoking adversely affects periodontal health[1]. 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[2] and surgical procedures including: modified Widman flap surgery[3] guided tissue regeneration[4] dental implants[5] and supportive periodontal treatment[6]. Smoking also adversely affects the neutrophils and macrophages, which are crucial as gingival immunocompetent cells. Especially, smoking impairs neutrophils chemotaxis and/or phagocytosis[7]. 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[8]. 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 reduction of clinical inflammatory sings in smokers can be attributed to the cotinine, a nicotine metabolic by-product which has a peripheral constrictive action on gingival blood vessels[9]. By way of the vascular and immunological response of the body smoking is thought to brings its changes in the periodontium. Smoking has its effects on periodontium which is reflected through the morphologic and histologic changes in the gingiva. Gingival biotype is a critical factor that determines the result of dental treatment. Gingival thickness is one of them. The initial gingival thickness is significant as it may predict the outcome of root coverage procedures and restorative treatments[10,11]. Since tissue biotypes have different gingival and osseous architectures, they show different pathological responses when subjected to inflammatory or traumatic insults. These different responses, dictate different treatment modalities. As early as 1969, Ochsenbien and Miller discussed the importance of ‘‘thick vs. thin’’ gingiva in restorative treatment planning [12]. So by taking into consideration the gingival thickness of a patient during treatment planning, more better strategies for periodontal management may be developed, resulting in more acceptable treatment outcomes. Gingival thickness also plays a major role in various specialities like implantology, prosthodontics and most importantly in periodontics. The aim of the study is to measure the clinical parameters, plaque index and sulcus bleeding index and the gingival thickness in smokers and non-smokers.

MATERIALS AND METHODS

The present study was conducted in the out patient department, Saveetha dental college and hospitals, Chennai, India. Only male patients of the age 18-45 years were included. Control group consisted of non-smoker

Author for Correspondence

Table 1: Mean plaque and gingival index scores

	Smokers	Non-smoker	T-value	P-level
Plaque index	1.34±0.49	0.96±0.44	0.31	0.75,NS
Sulcus bleeding index	1.67±0.49	0.38±0.14	0.69	0.49,NS

NS-non significant

Table 2: Intra-group comparison of gingival thickness

Site	Groups compared	Mean±SD	Z value	P value
smoker	midbuccal	0.48±0.13	0.61	0.53,NS
	Interdental	0.61±0.08		
Non-smoker	midbuccal	0.35±0.07	1.890	0.05,NS
	interdental	0.40±0.08		

NS-non significant

Table 3: Inter-group comparison of gingival thickness:

Site	Groups compared	Mean+SD	Z value	P value
Midbuccal	Smoker	0.48±0.13	3.38	0.0007,S
	Non smoker	0.35±0.07		
Interdental	Smoker	0.61±0.08	6.73	<0.0001,S
	Non-smoker	0.40±0.08		

S-significant

patients with clinically healthy gingiva and experimental group included smoker patients with gingivitis according to CDC criteria for current smokers those patients who have smoked more than 100 cigarettes in their lifetime. Exclusion criteria included patients with periodontitis, use of any medications possibly affecting the periodontal tissues, extensive restorations, caries or tooth replacements, former smokers, individuals with systemic and immunologic abnormalities. The patients were informed about the study, its purpose, the degree of discomfort that might occur before conducting the procedure; a written consent was obtained from all the subjects. The study group included fifteen male smokers and fifteen male non-smokers whose gingival thickness was measured in the maxillary anterior teeth by transgingival probing[13]. At first, the clinical parameters of Plaque index[14] and Sulcus bleeding index[15] were recorded followed by scaling and polishing. The attached gingiva and interdental papilla were anesthetized using LA spray (lignocaine 15.0 g). Using an endodontic reamer the gingival thickness was assessed midbuccally in the attached gingiva and at the base of the interdental papilla. The endodontic reamer was inserted into the labial gingiva, perpendicular to the long axis of the tooth until it contacted the hard surface i.e. the bone. Similarly the gingival thickness was measured at the base of the interdental papilla. All the measurements were then measured using a Vernier caliper. The data was subjected to statistical analysis. Mean values and standard deviations were calculated. The paired t-test was used to compare the sulcus bleeding index and plaque index and Z test to compare the thickness of gingiva among smokers and non-smokers in midbuccal and interdental region.

RESULTS

The study included a total of 30 patients of which 15 were non-smokers and 15 smokers within the age group 18-45 years. The number of midbuccal sites measured was 180 and the number of interdental sites measured were 210 among both smokers and non-smokers. The plaque index and sulcus-bleeding index were assessed in the patients. Plaque index did not show any significant difference among the smokers and non smokers (p-value of 0.75). The sulcus-bleeding index also did not show any significant difference between the two groups (p-value of 0.49) (Table 1). The midbuccal thickness in smokers is 0.48 ±0.13mm and interdental thickness is 0.61 ±0.08mm. Comparison between the midbuccal and interdental gingiva in smokers showed no significant difference in thickness in the interdental region. The midbuccal thickness in non-smokers is 0.35 ±0.07mm and the interdental thickness is 0.40 ±0.08mm. Similarly in non-smokers also, the interdental gingival thickness (0.42+0.08) and midbuccal gingival thickness (0.35+0.07) did not show any significant difference. (Table 2). The comparison of midbuccal gingival thickness among smokers and non-smokers, which shows that midbuccal thickness in smokers (0.48 ±0.13mm) is higher than in non-smoker group (0.35 ±0.07 mm). Likewise, interdental gingival thickness in smokers (0.61 ±0.08mm) is higher than in non- smokers (0.41 ±0.08 mm). Therefore, both intergroup comparison of the gingival thickness is statistically significant higher in smokers than non-smokers with a p-value <0.001. (Table 3)

DISCUSSION

HEDIR and al[16] stated that smoking per se is detrimental to periodontal health as it worsens the oral hygiene status and depresses the host's defense mechanism. Tobacco smoking affects the oral environment, the gingival tissues and its vasculature, inflammatory response; it's immune response, and the homeostasis and healing potential of the periodontal connective tissues. While there is increasing clinical evidence to associate smoking with destructive

periodontal disease, the mechanisms that cause periodontitis in smokers remain to be fully elucidated[17]. According to literature, gingival thickness have been assessed by invasive method using a disposable sterile needle[18], boley gauge[19], stainless steel wire[20] & bone sounding with a periodontal prob[21,22]. While non-invasive methods included the use of ultrasonographic methods [23,24] and visual assessment with the use of a periodontal probe[25]. A direct correlation exists between gingival biotype and increased susceptibility to gingival recession following surgical and restorative procedures. It was pointed out how thick and thin gingival biotypes respond differently to inflammation, restorative traumatic events and parafunctional habits. These traumatic events result in different types of periodontal defects, which respond differently to different treatment procedures. Therefore, an accurate diagnosis of gingival thickness is of the utmost importance in devising an appropriate treatment plan and achieving a predictable esthetic outcome[26]. Studies have concluded that the thickness of the gingiva plays a vital role in development of mucogingival problems and in the success of treatment for recession and wound healing, comparative assessment of gingival thickness is relevant to clinical periodontics[27]. Although many studies have been conducted measuring the thickness, most of them have measured the thickness of gingiva using histological methods [28] and ultrasonographic methods[23,24] and clinically by transgingival probing[13]. Histologic studies have shown that smoking patients showed increased epithelial base and stratum corneum thickness. The increased epithelium thickness can contribute to the reduction of inflammatory clinical signs in the gingival tissue[28]. A similar study was conducted in Karnataka where the gingival thickness was assessed by transgingival probing in smokers and non-smokers and showed that anatomically, interdental gingiva is found to be thicker than the midbuccal gingiva in both smokers and non-smokers. Both midbuccal and interdental areas were thicker among smokers when compared to non-smokers at similar plaque and gingival bleeding levels[29].

The results of this study are as follows. The oral hygiene status as depicted by plaque scores were almost similar in both the groups even though smokers had slightly higher scores that were not significant and this finding is in agreement with the other previous studies[30-32]. Contradicting these studies, others have shown higher levels of plaque in smokers[33-39]. The sulcus-bleeding index did not show any significant difference between the two groups as supported by several studies. But the results were in contrast to a few studies which have shown that gingival bleeding is less in smokers than in non-smokers as smoking causes vasoconstriction of peripheral vessel induced by the actions of nicotine-stimulated adrenaline and noradrenaline on α1- adrenergic receptors. Such a constrictive action on gingival vessels would result in the suppression of vascular properties of inflammation such as bleeding, redness, and exudation. Smoking has previously been shown to affect oral leucocytes, indicating an impairment of PMN-function[40,41]. Various factors have been attributed to the increase in the gingival thickness in smokers. Studies have suggested that nicotine increases rate of proliferation of gingival epithelium, thus increasing epithelial thickness among smokers[42]. Some investigators also found that collagen production was apparently stimulated by nicotine[43]. Nicotine is said to cause an increase in collagen production in the connective tissue of smokers gingiva. Studies have also shown that the stratum corneum thickness was more marked in smokers[44]. In this study the midbuccal gingival thickness was greater in smoker than in non-smoker. Also the interdental gingival thickness was also greater in smokers than in non-smokers. However within the group the midbuccal and interdental gingival thickness did not show any significant difference though the midbuccal thickness was slightly greater than interdental thickness in both.

CONCLUSION

The main limitation of the present study is the small sample size. Therefore, the findings related to gingival thickness in smokers and non-smokers could not be reflective of the true and general picture of the situation in the regional and national scenario. There is therefore, the need for an extensive work on the topic so as to give a broader view on the subject.

REFERENCES

1. Haber.J, Wattles. J, Crowley.M, Mandell.R, Joshipura.K, Kent.R.L, Evidence for cigarette smoking as a major risk factor for periodontitis. J Periodontol 1993, 64(1);16–23.
2. Preber H, Bergström J. The effect of non-surgical treatment on periodontal pockets in smokers and non smokers. J Clin Periodontol 1985; 13:319-23.
3. Preber H, Bergström J. The effect of cigarette smoking on periodontal healing following surgical periodontal therapy. J Clin Periodontol 1990; 17:324-28
4. 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.
5. Lambert PM, Morris HF, Ochi S. The influence of smoking on 3 year clinical success of osseointegrated implants. Ann Periodontol 2000; 5:79-89.
6. Kaldahl WB, Johnson GK, Patil KD, Kalkwarf KL. Levels of cigarette consumption and response to periodontal therapy. J Periodontol 1996; 67:675-681.
7. Eichel B, Shahrik HA. Tobacco smoke toxicity: loss of human oral leukocyte function and fluid cell mechanism. Science 1969; 166:1424-1428.
8. Kazor. C, Taylor. G. W, Loesche W.L, The prevalence of BANA-hydrolyzing periodontopathic bacteria in smokers. J Clin Periodontol 1999, 26(12); 814–21.
9. Danielsen B, Manji F, Nageilkerke N, Fejerskov O, Baelum V. Effect of cigarette smoking on the transition dynamics in experimental gingivitis. J Clin Periodontol 1990; 17:159-164.
10. Baldi C, Pini Prato G, Pagliaro U, et al. Coronally advanced flap procedure for root coverage Is flap thickness a relevant predictor to achieve root coverage? A 19- case series. J Periodontol 1999; 70: 1077–84.

11. Hwang D, Wang HL. Flap thickness as a predictor of root coverage: A systematic review. J Periodontol 2006; 77:1625–34.
12. Ochsenbien C, Ross S. A re-evaluation of osseous surgery. Dent Clin North Am January 1969; 13(1):87–102.
13. Vandana KL, Savitha B. Thickness of gingiva in association with age, gender and dental arch location. J Clin Periodontol. 2005; 32(7): 828-830.
14. Silness J, Loe H. Periodontal disease in pregnancy. II. Correlation between oral hygiene and periodontal condition. Acta Odontol Scand. 1964; 22:121-135.
15. Ainamo J, Bay I. Problems and proposals for recording gingivitis and plaque. Int Dent J. 1975; 25(4): 229-235.
16. Hedir CA, Ronquist G, Forsberg O. Cyclic nucleotide content of gingival tissue of smokers and non-smokers. J Periodont Res 1981; 16: 337.
17. Bergström J, Preber H. The influence of cigarette smoking on the development of experimental gingivitis. J Periodontal Res 1986; 21:668-76.
18. Anderegg C.R, Metzler D.G, Nicoll B.K. Gingiva thickness in guided tissue regeneration and associated recession at facial furcation defects. J Periodontol 1995; 66(5): 397-402.
19. Claffey N, Shanley D. Relationship of gingival thickness and bleeding to loss of probing attachment in shallow sites following nonsurgical periodontal therapy. J Clin Periodontol. 1986; 13(7): 654-657.
20. Waraaswapati N, Pitiphat W, Chandrapho N et al. Thickness of palatal masticatory mucosa associated with age. J Periodontol. 2001; 72(10): 1407-12.
21. Studer SP, Allen EP, Rees TC, Kouba A. The thickness of masticatory mucosa in the human hard palate and tuberosity as potential donor sites for ridge augmentation procedures. J Periodontol. 1997; 68(2):145-151.
22. Kobayashi Kenichi, Suzuki Tetsuya, Kimura Masumi et al. Application of Ultrasonic pulse-echo method in the region of Prosthodontics: Equipment and ultrasonagraph. The Journal of the Japan Prosthodontic Society. 1986; 30(3);684- 69.
23. Müller HP, Schaller N, Eger T. Ultrasonic determination of thickness of masticatory mucosa: a methodologic study. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1999; 88(2):248-253
24. KristoffersonT. Periodontal conditions in Norwegian soldiers. An epidemiological and experimental study. Scand J Dent Res 1970; 78: 34-53.
25. Preber H, Kant T, Bergstrom J.Cigarette smoking, oral hygiene and periodontal health in Swedish army conscripts. J Clin Periodontal. 1980, 7; 106-113.
26. Malhotra R, Kapoor A, Grover V, Kaushal S. Nicotine and periodontal tissues. J Indian Soc Periodontol 2010;14: 72–9.
27. Anderegg CR, Metzler DG, Nicoll BK. Gingiva thickness in guided tissue regeneration and associated recession at facial furcation defects. J Periodontol. 1995; 66(5): 397-402.
28. Villar CC, de Lima AF. Smoking influences on the thickness of marginal gingival epithelium. Pesqui Odontol Bras. 2003; 17(1): 41-45.
29. Jayashree NV, Vandana KL. Gingival thickness among smokers and non-smokers. Indian J Dent Adv 2012; 4(1): 723-9.
30. 30 G. Calsina, J.M. Ramon and J.J. Echeverrıa, Effects of smoking on periodontal tissues, Journal of Clinical Periodontology, 2002, 29(8); 771–776.
31. Bastiaan. R. J and Waite. I.M. Effects of tobacco smoking on plaque development and gingivitis. J Periodontol, 1978, 49(9); 480–482.
32. Bergstrom. J and H. Preber, Cigarette smoking in patients referred for periodontal treatment, Scandinavian journal of dental research 1986, 94(2) 102–8.
33. Haffajee A.D and Socransky.S. S, Relationship of cigarette smoking to attachment level profiles, J Clin Periodontol 2001, 28(4) 283–95.
34. Pindborg. J. J, Tobacco and gingivitis II correlation between consumption of tobacco, ulceromembranous gingivitis and calculus, Journal of Dental Research 1949, vol. 28, 460–3.
35. Macgregor. I.D, W. M. Edgar, and A. R. Greenwood, Effects of cigarette smoking on the rate of plaque formation, J Clin Periodontol, 1985, 12(1); 35–41.
36. Alexander. A.G, The relationship between tobacco smoking calculus and plaque accumulation and gingivitis, Dental health 1970, 9(1);6–9.
37. Sheiham.A, Periodontal disease and oral cleanliness in tobacco smokers, J Periodontol 1971, 42(5); 259–63.
38. Torrungruang. K, Nisapakultorn. K, Sutdhibhisal.S et al., The effect of cigarette smoking on the severity of periodontal disease among older Thai adults, J Periodontol 2005, 76(4) 566–572.
39. Preber. H and Kant. T, Effect of tobacco smoking on periodontal tissue of 15 year old schoolchildren, J Periodontal Res 1973, 8(5), 278–83.
40. Kenney. E. B, Kraal. J. H, Saxe S.R, and Jones.J, The effect of cigarette smoke on human oral polymorphonuclear leukocytes, J Periodontal Res 1977, 12(4); 227– 34.
41. Kraal J.H, Chancellor M.B, and Bridges. R.B, Variations in the gingival polymorphonuclear leukocyte migration rate in dogs induced by chemotactic autologous serum and migration inhibitor from tobacco smoke, J Periodontal Res 1977, 12(4) 242–49.
42. Gultekin SE, Sengüven B, Karaduman B. The effect of smoking on epithelium proliferation in healthy and periodontally diseased marginal epithelium. J Periodontol. 2008; 79:1444–50.
43. Prebec, H. and Bergstrom, J. The effect of nonsurgical treatment on periodontal pockets in smokers and non-smokers. J. Clin. Periodontol. 1986, Vol. 13; 319-23.
44. Alonge OH, Ashrafi SH, Colvard MD. Mitochondrial volumen densities in the smokless tobacco- treated hamster cheek pouch epithelium. Oral Diseases 2003; 9:128-143.