

## Effect of Smoking on Gingival Recession

Seyed Ali Banihashemrad\*, Kazem Fatemi\*, Mohammad Hasan Najafi\*

### ABSTRACT

**Background:** Gingival recession refers to apical migration of the gingival margin. 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 was to assess the effects of smoking on gingival recession.

**Methods:** Fifty smokers and 55 non-smokers took part in this cross-sectional study. Gingival recession on buccal and palatal surfaces of all teeth was measured. In addition, the relationship between the gingival recession and the following factors was evaluated: educational level, age, number of cigarettes used per day, plaque control methods, the age at which smoking started and plaque index. The data were analyzed using t-test and the coefficient of correlation.

**Results:** According to data analysis, the smoker subjects had a significantly greater mean recession than non-smokers. 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.

**Conclusion:** The results of this study indicated that smoking causes gingival recession, possibly via alteration in immune response and topical changes such as decreasing gingival circulation.

**Keywords:** Gingival recession, periodontitis, smoking.

**Received:** October 2007

**Accepted:** March 2008

Dent Res J 2008; 5(1):1-4

### Introduction

Gingival recession is defined as the exposure of root surface and apical migration of the gingival margin. Recession defects are one of the most common findings in periodontal problems affecting individuals of almost all ages to some degree. Recession exposes the underlying cementum, often leading to hypersensitivity, plaque retention, root caries and loss of esthetics. It may have various etiologies, including faulty tooth brushing methods, tooth malposition, friction from soft tissue, gingival inflammation, abnormal frenum attachment and iatrogenic dentistry.<sup>1</sup> Gingival recession is an intriguing and complex phenomenon. Patients frequently are disturbed by recession owing to sensitivity and esthetics.<sup>2</sup> Cigarette smoking and the presence of supragingival calculus were the factors most significantly associated with localized and generalized recession.<sup>3</sup> Increasing evidence points to smoking as a major risk factor for periodontal disease, affecting the prevalence, extent, and sever-

ity 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.<sup>1</sup> The response of the microcirculation to plaque accumulation appears to be altered in smokers when compared with non-smokers.<sup>4</sup> With developing inflammation, increases in gingival blood vessels were lower in smokers than in non smokers.<sup>3</sup> In addition, the oxygen concentration 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.<sup>5</sup> 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.<sup>6</sup> The purpose of this study was to evaluate the effect of smoking on gingival recession.

\*Assistant Professor, Department of Periodontics, School of Dentistry and Dental Research Center, Mashhad University of Medical Sciences, Mashhad, Iran.

Correspondence to: Seyed Ali Banihashemrad, Assistant Professor, Department of Periodontics, School of Dentistry and Dental Research Center, Mashhad University of Medical Sciences, Mashhad, Iran. E-mail: Banihashemrad@yahoo.com

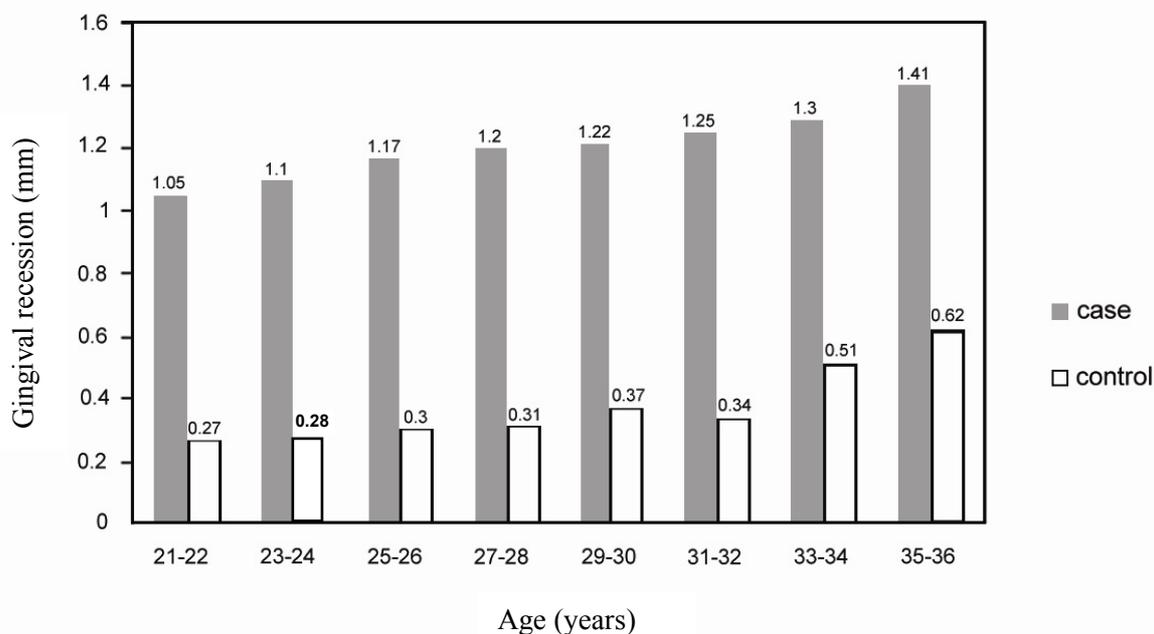
## Materials and Methods

This cross-sectional study involved 105 consecutively selected Mashhad Dental School patients, between the ages of 21-36 (50 smokers as the case group and 55 non-smokers as the control group). The patients in these two groups were matched according to sex, age and oral hygiene status. None of them had received scaling and root planning or periodontal treatments during the previous six months. Patients were regarded as smokers if they smoked more than 10 cigarettes per day. Occasional and former smokers were excluded. Patients with systemic diseases were also not selected. A questionnaire consisting of two parts was prepared for collecting data. Part 1 was composed of data regarding age, sex, education level, plaque control methods (tooth brush, dental floss) and smoking habits. Part 2 was composed of plaque index (O'Leary et al, 1973), (scaled as < 30%, 30-70% and > 70%), and gingival recession rate from CEJ to gingival margin, measured by William's probe in the mid-facial surfaces of all teeth except the third molars. The data were analyzed with t-

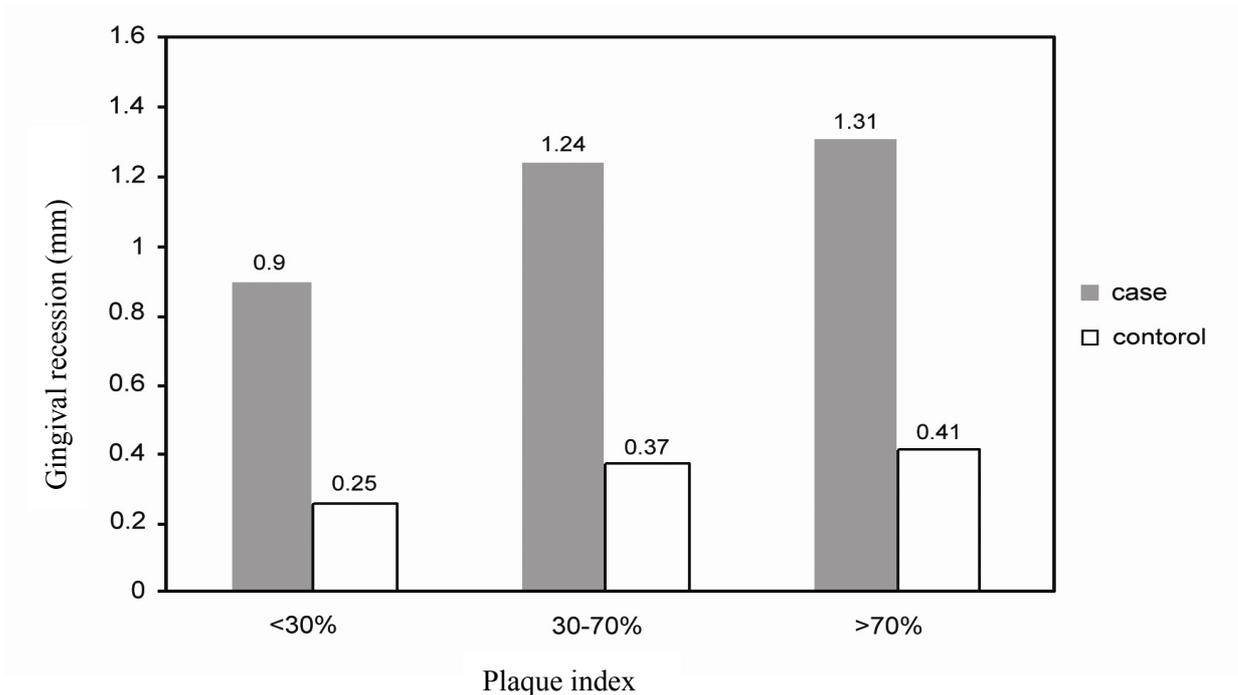
test and the coefficient of correlation was also evaluated.

## Results

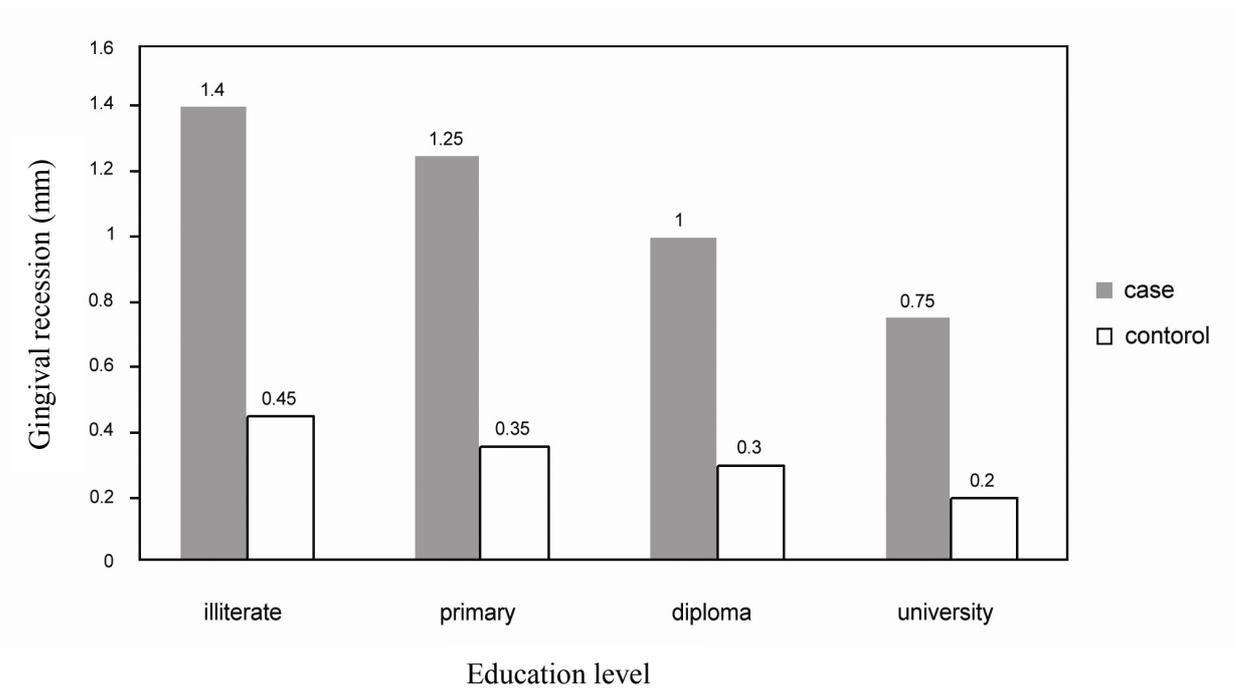
The data revealed that gingival recession was greater in smokers (1.12 mm) compared to non-smokers (0.36 mm), which was significant ( $P < 0.001$ ). There was a significant relationship between gingival recession and age in case and control groups ( $r = 0.398$ ,  $P = 0.017$ ; Figure 1). There was a significant relationship between gingival recession with plaque index ( $r = 0.672$ ; Figure 2,  $P < 0.05$ ), duration of smoking ( $r = 0.369$ ) and number of cigarettes ( $r = 0.392$ ). There was a significant reverse relationship between gingival recession and level of education in smokers (Figure 3,  $r = -0.407$ ,  $P < 0.05$ ). That is, more educated cases had less gingival recession. Also, there was a significant relationship between gingival recession and plaque control methods ( $r = -0.445$ ,  $P < 0.05$ ), which implies that use of both tooth brush and dental floss decreased gingival recession more effectively than the use of just one method or no method.



**Figure 1.** The relationship between gingival recession and age ( $P = 0.017$ ).



**Figure 2.** The relationship between gingival recession and plaque index ( $P < 0.001$ ).



**Figure 3.** The relationship between gingival recession and education level ( $P = 0.022$ ).

**Discussion**

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 consi-

tent with the study done by Muller et al.<sup>7</sup> and Bokor-Bratic.<sup>8</sup> In a study done by Checchi et al.<sup>9</sup> comparing Norway academicians and tea labourers, gingival recession was more frequent in labourers, which implies that it is less frequent in educated people such as those included in our study. This higher rate could be due to a decrease 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 PGE2 by monocytes in response to LPS is also a contributing factor. The exact changes in the immunologic mechanisms involved in the rapid tissue destruction seen in smokers are currently unclear.<sup>1</sup> Further studies are needed to define the effects of tobacco use on the immune response and tissue destruction in periodontitis. However, the effects of smoking on periodontal disease progression are reversible with smoking cessation. Thus, smoking cessation programs should be an integral component of periodontal education and therapy. Performing studies with greater sample sizes among current smokers, former smokers and non-smokers is recommended.

### Acknowledgement

We would like to acknowledge the financial support of Vice Chancellery for Research of Mash-

had University of Medical Sciences(Grant No: 1875).

### References

1. Newman MG, Takei H, Carranza FA, Klokkevold PR. Carranza's clinical periodontology. 10<sup>th</sup> ed. St. Louis: Mosby - Saunders; 2006.
2. Kassab MM, Cohen RE. Treatment of gingival recession. *J Am Dent Assoc* 2002; 133(11): 1499-506.
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(10): 1377-86.
4. Persson L, Bergstrom J. Smoking and vascular density of healthy marginal gingiva. *Eur J Oral Sci* 1998; 106(5): 953-7.
5. Bergstrom J, Persson L, Preber H. Influence of cigarette smoking on vascular reaction during experimental gingivitis. *Scand J Dent Res* 1988; 96(1): 34-9.
6. Hanioka T, Tanaka M, Ojima M, Takaya K, Matsu-mori Y, Shizukuishi S. Oxygen sufficiency in the gingiva of smokers and non-smokers with periodontal disease. *J Periodontol* 2000; 71(12): 1846-51.
7. Muller HP, Stadermann S, Heinecke A. Gingival recession in smokers and non-smokers with minimal periodontal disease. *J Clin Periodontol* 2002; 29(2): 129-36.
8. Bokor-Bratic M. [Effects of smoking on the periodontium]. *Med Pregl* 2002; 55(5-6):229-32.
9. 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(5): 276-80.