ORIGINAL ARTICLE

The recurrent aphthous stomatitis frequency in the smoking cessation people

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Abstract This study was aimed to evaluate the frequency of recurrent aphthous stomatitis (RAS) within the 6-week period after quitting smoking. The study group consisted of 90 subjects. Oral, medical findings and tobacco habits were recorded for all subjects. Nicotine replacement therapy (NRT) and behavioral treatment were applied to some of the subjects by a family physician. All subjects were evaluated for their RAS and periodontal measurements on baseline, 1, 3, 6 weeks by a periodontist. While the subjects were in this smoking cessation programme, 64 of the 90 smokers successfully quit smoking within the 6 weeks and 26 smokers dropped out during the third week of the study. Point prevalence of RAS among the subjects on the first day of the quitting period and at the end of the first, third and sixth week after smoking cessation was 3.3% (3/90), 18.9% (17/90), 21.1% (19/90) and 17.1 (11/64), respec-

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İ. Marakoğlu Dental Faculty, Department of Periodontology, Selçuk University, Konya, Turkey tively. In the following weeks, aphthous ulcer point prevalence was significantly higher than the quitting level (p < 0.05). As the time after quitting increased, the incidence of aphthous ulcer decreased. Of 64 patients, 35 (54.6%) completed the 6 weeks using NRT and 29 (45.4%) of them did not use any medication. The aphthous ulcer frequency observed in the patients taking NRT [11.4% (4/35)] was lower when compared with the subjects taking no NRT [24.1% (7/29)] (p > 0.05). The results of this study confirm that RAS is a complication of quitting smoking. Further studies are needed to identify the effects of NRT on RAS.

Keywords Recurrent aphthous stomatitis · Smoking cessation · Smoking · Nicotine replacement therapy · Oral health

Introduction

Recurrent aphthous stomatitis (RAS) is one of the most common oral mucosal diseases. The cause of aphthous ulceration is unclear [24]. There may be a genetic predisposition to aphthous ulceration; about 40.0% of people with RAS have a family history of oral ulceration. In a minority of people, precipitating factors can be identified, including smoking cessation, local trauma (e.g. excessive tooth brushing, chewing sharp or hard foods), stress, food sensitivity (e.g. foods containing some preservatives and flavoring agents such as benzoic acid or cinnamaldehyde), hormone imbalance (there are a few women whose aphthous ulcerations remit during pregnancy) [21, 22].

Tobacco use is a risk factor for oral cancer, oral mucosal lesions and periodontal disease. The incidence of RAS was found to be lower in smokers than in non-smokers and clinical observation suggests that some smokers experience an increase in mouth ulcers upon stopping smoking [2, 16]. Participants in the cessation programmes often allege that the presence of oral ulcers is one of the reasons that they start smoking again [9, 13, 23].

In addition, there are numerous reports showing that smoking affects the periodontal tissue [2, 9, 13, 17, 21–24]. Cigarette smoking is clearly established as one of the most significant risk factors in the development and progression of periodontal disease. Hence, quitting smoking is absolutely essential for improving the healing potential beginning periodontal treatments [17]. In this study, the evaluation of the changes observed in the mouth within the 6 weeks period after quitting smoking was aimed.

Materials and methods

In this study, 90 patients were assessed. The subjects who started to give up smoking sent from Cumhuriyet University Medical Faculty Department of Family Medicine to Periodontology clinic were followed-up regarding aphthous ulcer occurrence evaluated. All participants provided informed consent and ethics approval was obtained.

Design and measures

All participants filled out a questionnaire, which included questions on their age, medical findings, tobacco habits and previous aphthous ulcer frequency. NRT (nicotine patches) was applied to some of the patients by a family physician considering the Fagerstrom nicotine dependency level. Standard questions used in the classification of smoking status in the United States [26] and Fagerstrom test for nicotine dependence (FTND) [6] were used. FTND is a sixitem questionnaire test to determine the level of dependence; most points of a FTND score come from two questions on cigarettes per day and time to the first cigarette of the day. The highest FTND score, 10, indicates the highest level of nicotine dependence.

Along with the questionnaire, diagnosis of RAS, periodontal measurements were taken from all patients by a periodontist with a Williams's periodontal probe and the melanin pigmentation presence was evaluated for all oral mucosa.

The periodontal examination included measurements of plaque accumulation, gingival inflammation, probing depth and radiographic bone loss. Patients were classified into 3 groups, according to the clinical and radiographic criteria: the healthy group, the gingivitis group with gingival inflammation but no evidence of bone loss and the periodontitis group showing radiographic evidence of bone loss and attachment loss of more than 5 mm in at least 8 sites.

The clinical criteria for RAS were: well-demarcated, rounded ulcers covered by gray, white or yellowish

fibrinous exudates and surrounded by a red, inflammatory zone (Fig. 1). All participants were assessed on the day of smoking cessation (baseline) and then on the first, third and sixth weeks of smoking cessation by a periodontist.

Analysis of data

Statistical analyses were carried out using the Chi-square and Mac Nemar tests. *P* values lower than 0.05 were considered statistically significant.

Results

As participants in this smoking cessation programme, 64 of the 90 smokers (53 men and 37 women, mean ages $31.96\pm$ 11.06 years) successfully quit smoking within the sixth week and 26 smokers dropped out during the third week of the study. These patients started smoking again and consequently 64 individuals completed the study.

The range of cigarette consumption was 5–70 cigarettes a day with the median consumption of the group being 22.85 cigarettes a day and the mean smoking period was 15.1 ± 10.98 years. The percentage of the patients who had the previous quitting smoking experience was 63.3%. Using the Fagerstrom scoring, the level of nicotine addiction score was found among all smokers as 15.6% scoring 0–2 (*n*=14), 21.1% scoring 3–4 (*n*=19), 14.4% scoring 5 (*n*=13), 27.8% scoring 6–7 (*n*=25) and 21.1% scoring 8–10 (*n*=19). The statistically significant relations were not determined between the aphthous ulcer frequency with the amount of smoked cigarette and the FTND scores (*p*>0.05).

The frequency of the RAS on the first day of the quitting period and at the end of the first, third and sixth week are shown in Table 1. Only 15.6% (14/90) of the patients had previous aphthous lesion histories.

Aphthous ulcer was established in 3 (3.3%) out of 90 subjects on the first day of quitting smoking. After 1 week,



Fig. 1 Intra-oral view of the apthous ulcer

 Table 1 The aphthous ulcer frequency (point prevalence) in the quitting smoking subjects

Time	Number	Percentage
The first day of quitting period	3/90	3.3
At the end of the first week after quitting	17/90	18.9 ^a
At the end of the third week after quitting	19/90	21.1 ^b
At the end of the sixth week after quitting	11/64	17.1 ^c

^a It is significantly higher than that on the first day of quitting; p=0.001 with the Mac Nemar test.

^b It is significantly higher than that on the first day of quitting; p=0.000 with the Mac Nemar test.

^c It is significantly higher than that on the first day of quitting;

p=0.02 with the Mac Nemar test.

the number out of 90 cases with aphthous ulcer was 17. Of these 17 subjects, 15 had been found healthy in their first examination. At the end of first week, the point prevalence was 17/90 (18.9%) (Table 1) and the rate of incidence per week was $15/(72 \times 1 + 15 \times 0.5) = (18.9\%)$ (Table 2). The number of the cases with aphthous ulcer was 19 among these 90 people in the examination carried out at the end of the third week and the point prevalence of aphthous ulcer was determined as 19/90 (21.1%) (Table 1). In this examination, aphthous ulcer was found in 12 subjects out of 72 who had not been diagnosed with aphthous ulcer before. Aphthous ulcer incidence rate per week calculated according to this data was: $[12/(60 \times 2 + 12 \times 1)] = 9\%$ (Table 2). At the end of the sixth week, of the 64 cases that came to the examination, 11 (17.1%) cases were diagnosed with aphthous ulcer (Table 1). Of the 43 cases that had been diagnosed as healthy in their previous examinations, aphthous ulcer was established in 6 of these cases during examination at the end of the sixth week. Aphthous ulcer incidence rate per week for the fourth to sixth week was found as $[6/(37 \times 3 + 6 \times 1.5)] = 5\%$ (Table 2).

While the point prevalence of aphthous ulcer on the quitting day was 3.3%, it increased considerably after the first week and reached 18.8%. In the following weeks, the aphthous ulcer point prevalence was significantly higher than the baseline level (Table 1). As the time after

Table 2 The weekly aphthous ulcer frequency (incidence) in thesubjects after quitting smoking

Periods after quitting smoking	Number of new cases/number of person-weeks at risk	Incidence rate per week (%)
First week	15/(72×1+15×0.5)	18.8
Second to third weeks	$12/(60 \times 2 + 12 \times 1)$	9.0
Fourth to sixth weeks	6/(37×3+6×1.5)	5.0

quitting increased, the incidence of aphthous ulcer decreased (Table 2).

The cumulative aphthous ulcer occurrence percentage was 32.2% (29/90) in the first 3 weeks. Cumulative aphthous ulcer frequency was found as 39.1% (25/64) in 64 patients who completed 6 weeks.

When oral health was examined, the melanin pigmentation presence was determined as 56.7% (51/90). Of the patients, 85.5% (77/90) had got periodontal disease (gingivitis or periodontitis). Of the patients quitting smoking, 33.3% (30/90) had periodontitis and 52.2% (47/90) had gingivitis. The rate of periodontal disease, among patients who smoked 11 and more cigarettes per day was found significantly higher than those smoked who 10 and less cigarettes per day (p<0.05). The percentage of aphthous ulcers in the patients with periodontal disease; 40.3% vs 30.8%. But a statistically significant relation was not found between aphthous ulcer frequency and periodontal disease (p>0.05).

Of the various aids used in assisting the subjects on the quit smoking programme, out of 90 patients, 52 (57.8%) had used NRT and 38 (42.2%) did not use anything. Of the 64 patients, 35 (54.6%) completed the 6 weeks using NRT. The aphthous ulcer frequency observed in the patients who completed 6 weeks taking NRT [11.4% (4/35)] was lower when compared with the patients taking no NRT [24.1% (7/29)]. However, this difference was not statistically significant (p>0.05).

Discussion

The prevalence of aphthous ulcer was found as 2.7% in a study carried out among 11,360 people in Turkey [4]. Other population studies have found RAS in about 1.9% of Spanish [8] and 0.5% of Malaysian [29] examined adults, while 17.7% of Swedish [2] and 9.7% of adults in Ljubljana (Slovenia) [12] have a history of possible RAS. RAS seems to be infrequent in Kuwaiti Bedouins (5%) [7] and it was found in only 0.1% of Indians in Malaysia [29]. However, RAS may be especially common in North America [5]. While the 3% ulcer incidence prevalence we obtained from our study is similar to the results of these studies, the prevalence of aphthous rate can vary owing to the racial differences.

Ussher et al. [27] evaluated the oral ulcers and cold symptoms within 7 weeks period after quitting smoking. They reported a statistically significant increase of the RAS 1–2 weeks after quitting smoking. Moreover, McRobbie et al. [14] evaluated oral ulceration occurrences within 4 weeks period after stopping smoking and reported frequent RAS occurrences in 40% of patients who quit smoking in the first 2 weeks. Moreover, another study showed negative correlation between the prevalence of RAS and tobacco habits [2]. Similar to the results of these studies, we found that the prevalence of RAS was significantly higher in the following weeks than on the first day of quitting smoking.

We observed in our study that the incidence of aphthous ulcer decreased as the time after quitting went by. The fact that the observed decrease in the incidences in time is not reflected in the point prevalence in this study is related to the duration of aphthous ulcer. If following-up was maintained, the decrease in point prevalence might have been observed.

Smoker's melanosis may be due to the effects of nicotine on melanocytes located along the basal cells of the lining epithelium of the oral mucosa. Studies confirmed that smoker's melanosis was found in 25–31% of the smoking population [15, 25]. Our study showed a smoker's melanosis ratio in smokers as 56.7%. Smoker's melanosis ratio among ever smokers, in the Swedish population was found to be 27% [10], in Germans 18% [19], in Japanese 22% [1], in Indians 12.0% [11] and in Turkish 27% [19]. In our study, the smoker's melanosis ratio was found to be higher than the findings of the abovementioned studies. This can be attributed to the fact that the individuals in this study were heavy smokers. In addition, these results support that the main cause of smoker's melanosis in the smokers group may strongly be attributed to smoking.

Periodontal diseases are caused by bacteria. Smoking was strongly implicated as a risk factor for the initiation and propagation of periodontal diseases. The prevalence of periodontal disease is higher in smokers than non-smokers and the alveolar bone is more susceptible to resorption in smokers [16]. However, Muller and Stadermann's study did not reveal evidence for the attenuation of the plaque/gingival bleeding relationship in heavy smokers [18]. In our study, 85.5% of the patients had got periodontal disease. The rate of periodontal disease, among patients who smoked 11 and more cigarettes per day was found higher than who smoked 10 and less cigarettes per day. These results imply the relation between smoking and periodontal disease.

Patients who stop smoking often complain of RAS (mouth). A feature of interest is that RAS are infrequently seen in patients who smoke tobacco. The main explanation is that tobacco may increase keratinization of the oral mucosa, which in turn may render the mucosa less susceptible to ulceration [14]. The protective effect of oral NRT was reported in various studies [14, 23, 28]. In addition, in a patient with Behçet's syndrome, the aphthous lesions improved when nicotine patches were reported [20]. The selection of the type of nicotine replacement should be individualized based on the patient's smoking habits and preferences. Case studies suggest that smokers quitting with

NRT may be less likely to develop mouth ulcers than those quitting without NRT [3]. In our study, the aphthous ulcer frequency was determined to be lower in the patients using NRT than in the patients using nothing [11.4% (4/35) vs 24.1% (7/29)], but this difference was not statistically significant (p>0.05).

Our results confirm that RAS is a complication of quitting smoking. Further studies are needed to identify the effects of NRT on RAS.

References

- Araki S, Murata K, Ushio K, Sakai R (1983) Dose–response relationship between tobacco consumption and melanin pigmentation in the attached gingiva. Arch Environ Health 38:375–378
- Axell T, Henricsson V (1985) Association between recurrent aphthous ulcers and tobacco habits. Scand J Dent Res 93:239–242
- Bittoun R (1991) Recurrent aphthous ulcers and nicotine. Med J Aust 154:471–472
- Cicek Y, Canakci V, Ozgoz M, Ertas U, Canakci E (2004) Prevalence and handedness correlates of recurrent aphthous stomatitis in the Turkish population. J Public Health Dent 64:151–156
- Embil JA, Stephens RG, Manuel FR (1975) Prevalence of recurrent herpes labialis and aphthous ulcers among young adults on six continents. Can Med Assoc J 113:627–630
- Fagerstrom KO, Heatherton TE, Kozlowski LT (1992) Nicotine addiction and its assessment. Ear Nose Throat J 69:763–768
- Fahmy MS (1976) Recurrent aphthous ulcerations in a mixed Arab community. Community Dent Oral Epidemiol 4:160–164
- Garcia-Pola Vallejo MJ, Martinez Diaz-Canel AI, Garcia Martin JM, Gonzalez Garcia M (2002) Risk factors for oral soft tissue lesions in an adult Spanish population. Community Dent Oral Epidemiol 30:277–285
- Griesel AG, Germishuys PJ (1999) Salivary immunoglobulin A levels of persons who have stopped smoking. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 87:170–173
- Hedin CA (1977) Smokers' melanosis. Occurrence and localization in the attached gingiva. Arch Dermatol 113:1533–1538
- Hedin CA, Axell T (1991) Oral melanin pigmentation in 467 Thai and Malaysian people with special emphasis on smoker's melanosis. J Oral Pathol Med 20:8–12
- Kovac-Kovacic M, Skaleric U (2000) The prevalence of oral mucosal lesions in a population in Ljubljana, Slovenia. J Oral Pathol Med 29:331–335
- Mallın R (2002) Smoking cessation: integration of behavioral and drug therapies. Am Fam Physician 65:1107–1114
- McRobbie H, Hajek P, Gilison F (2004) The relationship between smoking cessation and mouth ulcers. Nicotine Tob Res 6:655–659
- Messadi DV, Waibel JS, Mirowski GW (2003) White lesions of the oral cavity. Dermatol Clin 21:63–78
- Mirbod SM, Ahing SI (2000) Tobacco-associated lesions of the oral cavity: part I. Nonmalignant lesions. J Can Dent Assoc 66:252–256
- Morozumi T, Kubota T, Sato T, Okuda K, Yoshie H (2004) Smoking cessation increases gingival blood flow and gingival crevicular fluid. J Clin Periodontol 31:267–272
- Muller HP, Stadermann S (2006) Multivariate multilevel models for repeated measures in the study of smoking effects on the association between plaque and gingival bleeding. Clin Oral Investig 10:311–316

- 19. Özbayrak S (1983) Zur Fage der Rauchermelanose der Mundschleimhaut. Thesis, Berlin Freinen Universität, Berlin
- Scheid P, Bohadana A, Martinet Y (2000) Nicotine patches for aphthous ulcers due to Behçet's syndrome. New Engl J Med 343:1816–1817
- Scully C, Gorsky M, Lozada-Nur F (2003) The diagnosis and management of recurrent aphthous stomatitis: a consensus approach. J Am Dent Assoc 134:200–207
- 22. Scully C, Shotts R (2000) ABC of oral health. Mouth ulcers and other causes of orofacial soreness and pain. BMJ 321:162–165
- Shiffman S, Rolf CN, Hellebusch SJ, Gorsline J, Gorodetzky CW, Chiang YK et al (2002) Real-world efficacy of prescription and overthe-counter nicotine replacement therapy. Addiction 97:505–516
- 24. Sklavunou-Andrikopoulou A, Maragou P (2000) Recurrent aphthous ulcers. Current status on the etiology and management. Balk J Stomatol 4:129–134

- 25. Ünsal E, Paksoy C, Soykan E, Elhan AH, Şahin M (2001) Oral melanin pigmentation related to smoking in a Turkish population. Community Dent Oral Epidemiol 29:272–277
- 26. US Department of Health and Human Services (1990) The health benefits of smoking cessation. A report of the Surgeon General. Public Health Service, Centers for Disease Control, Office on Smoking and Health, Rockville, Maryland
- 27. Ussher M, West R, Steptoe A, McEwen A (2003) Increase in common cold symptoms and mouth ulcers following smoking cessation. Tob Control 12:86–88
- Wallström M, Nilsson F, Hırsch JM (2000) A randomized, doubleblind placebo-controlled clinical evaluation of a nicotine sublingual tablet in smoking cessation. Addiction 95:1161–1171
- 29. Zain RB (2000) Oral recurrent aphthous ulcers/stomatitis: prevalence in Malaysia and an epidemiological update. J Oral Sci 42:15–19

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