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MINI REVIEW

Relation between Smoking and Recurrent Aphthous Stomatitis

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ABSTRACT

Introduction: Recurrent aphthous stomatitis (RAS) is a common oral mucosal disease characterized by the presence of multiple round or ovoid ulcers covered by a pseudomembranous slough and surrounded by an erythematous halo. The ulcer usually exists in the lining or nonkeratinized mucosa. There exists a relationship between smoking and occurrence of RAS due to the effects of nicotine which increases the keratinization of oral mucosa.

Aims: Some researchers have hypothesized that smoking has a protective role in preventing the occurrence of RAS. They have also stated that nicotine decreases the secretion of pro-inflammatory cytokines which are known to produce RAS. This communication aims to give a perspective regarding the effects of nicotine in oral mucosa.

Conclusion: The protective effect of smoking on RAS was only noticed when persons were heavy smokers or smoked for longer periods of time, according to the available literature. Lower prevalence of RAS in heavy smokers should not encourage smokers who suffer from RAS to increase their consumption.

Keywords: Nicotine, Recurrent aphthous stomatitis, Smoking.

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INTRODUCTION

Recurrent aphthous stomatitis (RAS) is a common oral mucosal lesion which is usually seen on the buccal mucosa, labial mucosa,[1] floor of the mouth, tongue, and palate (Fig. 1). It is characterized by the presence of yellowish-gray base with raised margins and surrounded by an erythematous halo. The lesions are extremely painful, and it gets aggravated on eating, speaking, and swallowing.[2-4]

In RAS, mostly a cell-mediated immune response mechanism is involved and causes generation of T-cells and tumor necrosis factor alpha (TNF-α) by these other leucocytes (macrophages and mast cells). The TNF-α cytokine, a major inflammatory mediator, induces initiation of the inflammatory process by its effect on endothelial cell adhesion and a chemotactic effect on neutrophils.[5]

DISCUSSION

Some investigators[1] believe that smoking has a protective role in the occurrence of RAS. This protective role is attributed to the presence of increased keratinization of oral mucosa in smokers. The keratin layer acts as a mechanical and chemical barrier against the invasion of microbes or trauma. The increased keratinization in oral mucosa makes it less prone to injury and irritation.

As some authors[1] believe that RAS is known to be less common in smokers, there have been previous reports of nicotine being used as successful treatment for aphthous stomatitis and Behçet's disease. Some[1] have hypothesized that nicotine may be the responsible agent for the reduction in prevalence rates of RAS in smokers. Nicotine is known to affect the immune response in inflammatory conditions by inducing the production of adrenal steroids through the hypothalamus–pituitary–adrenal axis and reducing the production of TNF-α and interleukins 1 and 6 through its direct effect on macrophages. Nicotine also decreases the secretion of proinflammatory cytokines (TNF-α and interleukins 1 and 6) and increases the secretion of anti-inflammatory cytokines.[1,3]

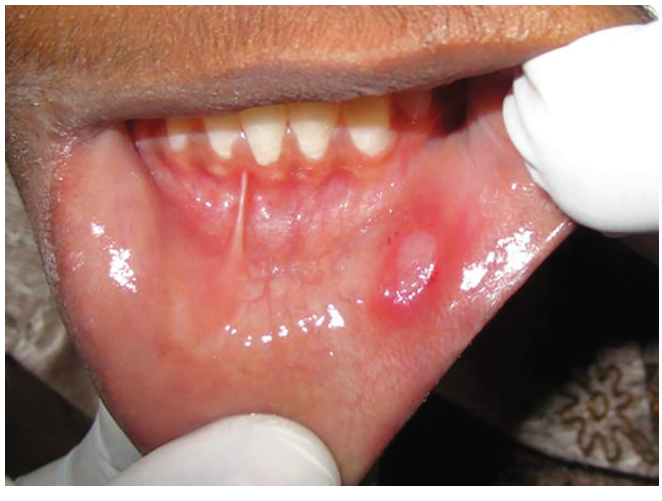

Fig. 1: Recurrent aphthous stomatitis in labial mucosa

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Some investigators[1] support the belief that nicotine may act as protector of the oral mucosa in the patients with RAS, while subjects who quit smoking often complain of RAS and resumption of smoking results in the faster resolution of RAS. Few investigators suggested that smokers may be less psychologically stressed than non-smokers and that some psychological trigger might affect RAS development. In addition, those who quit smoking are less likely to develop RAS if they use nicotine replacement therapy.[1]

CONCLUSION

Significant differences exist in the prevalence of RAS among cigarette smokers, which could be related to dose and duration of the habit. According to literature, the "protective effect" on RAS was noticed only when persons were heavy smokers or smoked for longer periods of time. Of course, this lower prevalence of RAS in the heavy smokers should not encourage smokers who suffer from RAS to increase their consumption. Data upon the "protective effect" of smoking on RAS are controversial, particularly with respect to a possible underlying mechanism.[6] More studies have to be carried out to find the underlying possible mechanism of fewer incidences of RAS in smokers.

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