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Effects of Smokeless Tobacco

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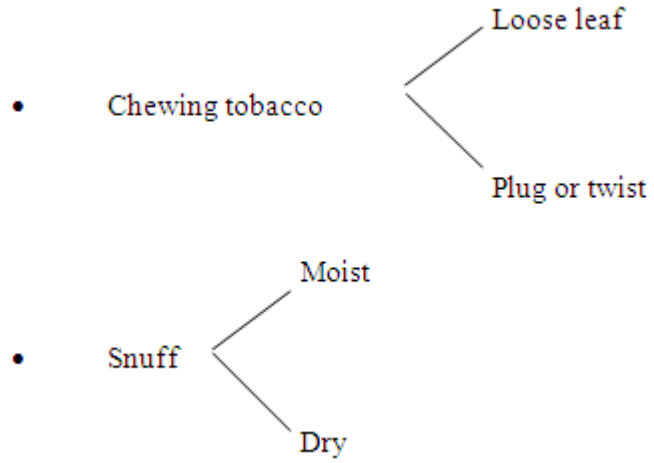
INTRODUCTION

The use of smokeless tobacco is widespread from Sudan and India to Scandinavia and USA.[1,2] Although available in many different forms, smokeless tobacco is mainly used as.



Major effects of smokeless tobacco were gingival recession and associated loss of attachment; oral mucosal lesions that correspond to the placement of the smokeless tobacco quid.

1. **Oral mucosal lesions:** The practice of holding a pinch of finely ground tobacco, a 'wad' of coarse-cut tobacco may increase the risk for oral cancer; induce oral mucosal lesions at the site where tobacco is placed. Large scale studies on athletes who use smokeless tobacco demonstrated a strong relationship to oral leukoplakia. A recent publication demonstrated that 97.5% of these leukoplakia lesions are clinically resolved after abstaining from smokeless tobacco for 6 weeks.

- Oral mucosal lesions appear as white, folded, and striated surfaces that are slightly elevated and diffusely demarcated from the surrounding mucosa. A characteristic white plaque, the smokeless tobacco keratosis, is produced on the mucosa in direct contact with snuff or chewing tobacco.
- Altered mucosa has a soft velvety feel to palpation and stretching of mucosa often reveals distinct pouch (snuff pouch, tobacco pouch) (Fig. 3) caused by flaccidity in the chronically stretched.
- It has been estimated to occur in from 16% to 63% of smokeless tobacco users.
- Smokeless tobacco keratosis usually takes 1-5 years to develop and new lesions seldom arise in persons with a long history of use.

Keratosis typically remains unchanged indefinitely unless the daily tobacco contact time is altered. In some cases, the white lesion gradually becomes thickened to the point of appearing leathery or nodular.

Smokeless tobacco extract is capable of stimulating PGE2 and IL-1 synthesis by human gingival keratinocytes. Luger TA et al (1990) stated that Interleukin 1 (IL-1) is a 17 kD polypeptide produced by numerous cells, including keratinocytes in response to chemical, bacterial or physical insult.[3]Kupper TS (1990) found that the epidermis contains a larger reservoir of biologically active IL-1 which acts in an autocrine manner to stimulate keratinocyte proliferation.[4] Sauder DN et al (1988)

concluded that with in the epithelium, IL-1 stimulates keratinocyte proliferation so that elevated levels may lead to epithelial hyper proliferation which characterizes the snuff induced lesions.[5]

2. **Gingival recession and gingivitis (Fig. 4)** has been reported in smokeless tobacco users by many but not all clinical surveys.

Studies have indicated that subjects free of gingivitis showed neither mucosal pathology nor gingival recession, whereas subjects with co-existing gingivitis showed a marked increase in the prevalence of both conditions.

Robertson PB et al (1990) found the major effects of smokeless tobacco were gingival recession and associated loss of attachment and this loss of periodontal tissues is localized to areas adjacent to mucosal lesions, areas that in turn correspond to the placement of the smokeless tobacco quid.[6]

Recession appears to result from: -

I Injury to gingiva overlying a thin alveolar housing / frank alveolar dehiscence associated with labial eruption in the dental arch are common among lower anterior and premolar teeth, a preferred site for placement of the smokeless tobacco quid.
Injury occurs due to chemical substances like:
Smokeless tobacco products, Nitrosodiethanol amine, Nitrosoproline, tobacco specific nitrosamines.

II Smokeless tobacco induced epithelial proliferation that bridges the narrow lamina propria of sites with an alveolar dehiscence might also result in loss of marginal gingiva.

Robertson PB et al (1990) found prevalence of plaque, gingival bleeding and periodontal pocket formation was the same for users and non users. Occurrence of severe forms of periodontal disease was equally low in both users and nonusers. Extrinsic stain and occlusal attrition were seen more often in users than non users. They concluded that even under conditions of minimal levels of gingival inflammation and regular hygiene care, oral sites where smokeless tobacco is used are at major risk for mucosal lesions, gingival recession, and attachment loss.[6]

Bergstrom J. et al (2006) found no relationship between smokeless tobacco in the form of Swedish moist snuff and the condition of the periodontal bone. Absence of a reaction of the periodontal bone to moist snuff is in distinct contrast to the evident reaction to smoke observed in smokers, thus favouring the assumption that the harmful effect of smoking is caused by toxic products in the inhaled smoke via internal routes rather than being a result of local damage to the periodontal tissues.[7]

Recently a large scale American study reported that smokeless tobacco users were at a slightly increased risk for interproximal attachment loss.[8]

The contradictory results may be due to:

- The American study concerns all types of smokeless tobacco and does not distinguish between snuff and chewing tobacco.
- Differences in composition of American and Swedish snuff which may not be readily comparable.
- Methodological dissimilarities.

In the Fisher et al study assessment of the attachment level included tooth sites adjacent to snuff placement where local tissue recessions frequently occur. Results of Fisher et al lost significance when analysis were restricted to never-smokers, suggesting that unadjusted smoking may be responsible for the association.[8]

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