Tobacco Related Lesions of Oral Cavity- A Review

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ABSTRACT

Tobacco in all forms such as cigarettes, pipes, cigars, beedis, paan etc have been implicated in development of premalignancies and malignancies. A *premalignant lesion* is nothing but cellular alteration with a greater than normal risk of malignant transformation. Oral precancers are successfully evaluated and managed as a routine facet of oral health care, despite residual or ongoing controversies of some significance. One of the major advances in this field is the acceptance that a premalignancy does not always transform into malignancy. Cancer is the second leading cause of death in the world; only cardiovascular diseases exact a higher toll. It is believed that tobacco use is responsible for 90% of oral cancers. This article gives a complete review of tobacco related lesions.

Key words: Tobacco, Premalignancy, Oral Cancer.

INTRODUCTION

Tobacco is smoked and chewed in various forms. It is the largest single preventable cause of death and diseases. It is causally related to oral cancers and precancerous lesions. It is also responsible for an array of oral lesions that are not considered precancerous failing to recognize which, can lead to cancer phobia. Tobacco habits in India are practiced in various different forms and are specific to certain areas.¹

Harmful Substances in Tobacco Alkaloid: Nicotine, Irritants, Toxins, Carcinogens

Most potent carcinogens

- Tobacco specific nitrosamines
- Polycyclic aromatic hydrocarbons and others

Tobacco is used in two forms:

Smoke - Preparation of bidis, Cigar / cheroot / chutta, Reverse Chutta Smoking, Chilums, Hookah, Hookli smoker

Smokeless - Pan (betel quid) with tobacco, Pan Masala, Mawa, Mainpuri tobacco, Khaini

Application over the teeth and the gingiva - Gudhaku, Bajjar, Creamy, Snuff.

Smokeless Tobacco induced Keratosis.²

TOBACCO RELATED LESIONS

Tobacco pouch keratosis, precancers, preleukoplakia, Leukoedema, Palatal erythema, Smoker'spalate, pan (betel quid) stain, Pan encrustation, tobacco lime user's lesion, Oral lichen planus like lesion, Central papillary atrophy of the tongue, leukoplakia, erythroplakia¹

TOBACCO POUCH KERATOSIS

Chewing of tobacco leaves or dipping snuff leads to the the development of a white mucosal lesion in the area of tobacco contact, usally called smokeless tobacco keratosis, snuff dipper's keratosis, or tobacco pouch keratosis. While these lesions are accepted as precancerous, they are significantly different from true leukoplakia and have a much lower risk of malignant transformation. The mucosal alterations which are caused by Smokeless tobacco, as it contains several carcinogens like N-nitrosonornicotine (NNN), being one of them, is dependent on duration of exposure.³⁻⁵

LEUKOPLAKIA

Has been reported to develop with the habitual use of snuff for longer than 3 years. All the forms of smokeless tobacco may lead to changes mucosal alterations. Use of a finely powdered tobacco called snuff appeared to be much more likely to cause changes than is chewing tobacco. Prevalence of oral premalignancies and malignancies related to variations in the composition of snuff, in particular, the amount of fermented or cured tobacco in the mixture.

Multiple alterations are seen in habitual users of smokeless tobacco. Mostly changes are associated with the use of smokeless tobacco are present in the area of contact with tobacco. Commonly area involved is the anterior mandibular vestibule, followed by the posterior

vestibule. Mucosal surface appears white and is granular or wrinkled in some cases; a corrugation may be seen. It is frequently associated with gingival recession along with destruction of periodontium particularly in the area of contact. Gingival recession involves the facial aspect of the teeth and depend primarily on the quantity and duration of tobacco being used. The mucosal appearance is gray or gray-white and almost translucent. During examination, the stretched mucosa appears fissured or corrugated, the lesion may become leathery or nodular in chronic tobacco users. Rarely, an erythroplakic component may be seen. Homogenous white lesion is usually asymptomatic and is discovered during routine examination. On Cessation of habit mucosal appearance gets back to normal within 1 to 2 weeks. Biopsy is recommended for the lesion that persists for more than a month. Specfically biopsy indicated for clinically atypical lesions, or having features as surface ulceration, erythroplakia, intense whiteness, or a verrucoid or papillary surface. Rate of malignant transformation is more in chronic smokeless tobacco users.6

PRELEUKOPLAKIA

Pre-leukoplakia is defined as a low grade or very mild reaction of the mucosa, appearing as a grey or grayish-white, but never completely white lesion with a slightly lobular pattern and indistinct borders blending into the adjacent normal mucosa. This lesion is a distinct entity conceived and demonstrated as a precursor stage of Leukoplakia with a low-grade malignant potential. Pre-leukoplakia is strongly associated with smoking.

Malignant transformation can occur directly from preleukoplakia, or the lesion may develop first into leukoplakia and then progress to cancer.^{1,7}

LEUKOEDEMA

Oral mucosa has gray opaque appearance as if a grayish film were hanging over it like a veil. When mucosa is stretched, the lesion disappears and reappears when relaxed. This condition is mostly observed among bidi smokers. Unlike leukoplakia, leukoedema does not present a keratinized surface.¹

SMOKER'S PALATE

This lesion consists of a diffused white palate with numerous excrescences having central red dots, corresponding to the orifices of the minor salivary glands. In the early stage, smoker's palate may consist of a grayish palatal mucosa, either with a few or without any excrescences. Protection of the palate against the smoke, or discontinuance of smoking, results in regression of smoker's palate. The intensity of smoker's palate depends on the extent and type of smoking. smoker's palate remains stationary in 66% of the cases, and 34% regress spontaneously.⁴

PALATAL ERYTHEMA

Palatal erythema is a common lesion strongly associated with bidi smoking. It consists of diffused erythema

on the hard palate that occasionally extends to the soft palate. Because of the intense redness in most lesions, palatal erythema can be misdiagnosed as erythroplakia. Microscopically, however, palatal erythema shows an inflammatory reaction with superimposed candidal hyphae, sometimes with minimal epithelial dysplasia. About 10% of individuals with palatal erythema also show papillary hyperplasia that consists of many red, ovoid or spherical, smooth nodules. Palatal erythema may remain stationary, regress spontaneously, or may be transient. Reduction or cessation of the smoking habit results in higher regression rates of palatal erythema.⁴

TOBACCO-LIME USER'S LESION

The habit of using a mixture of tobacco-lime (khaini) often produces a well-defined, thick, yellowish-white plaque. Occasionally, loose tags of tissue may be seen in the lesion. The tobacco lime user's lesion resembles a leukoplakia and can be misdiagnosed as such. Unlike leukoplakia, however, tobacco-lime user's lesion can be scraped off with a piece of gauze. It is hypothesized that the alkaline pH of the tobacco-lime mixture exerts a caustic effect on the oral mucosa, causing an abortive keratin formation and clinically resulting in a yellowish-white plaque. Discontinuation of use results in the clearance of this lesion.²

PAN (BETEL QUID) STAIN

Red coloration of the oral mucosa in betel-quid chewers is called a pan stain. It is transient in occasional chewers, but in habitual and heavy betel-quid chewers, the mucosa is perpetually stained. The bright red color produced during the chewing of pan is due to the formation of o-quinone from the water-soluble polyphenols, notably leucocynidins, at the alkaline pH of 8-9 via secondary reactions. Refraining from the habit or repeated washing generally clears the stain.

PAN ENCRUSTATION

In heavily addicted pan chewers, a thick brownish-black encrustation occurs at the site of placement of pan. This encrustation can be scraped off with a piece of gauze. It seems to be the pan chewer's counterpart of the tobacco-lime user's lesion. The microscopic features of pan encrustation are also akin to the tobacco-lime user's lesion. Discontinuation of the betel-quid chewing leads to nearly complete clearance of the encrustation.

ORAL LICHEN PLANUS LIKE LESION

Oral lichen planus-like lesion consists of white, wavy, parallel, non-elevated striae that do not crisscross as in lichen planus. Betel-quid chewing is strongly associated with this lesion, so much so that it almost occurs particularly at the site where patient is in habit of keeping

of betel quid such as the mandibular groove and the buccal mucosa. Sometimes the striae in oral lichen planus like lesion radiate from a central fibrin covered area corresponding to the site of placement of the betel quid. However, if the betel-quid chewing habit is discontinued, most of the lesions regress.⁶

PALATAL CHANGES AMONG REVERSE SMOKERS

The term "palatal changes" describes the reaction of the palatal mucosa to reverse chutta smoking. This form of smoking evokes diverse alterations in the palatal mucosa, which can be categorized into several interrelated components such as: Palatal keratosis, Excrescences, Patches, Red areas, Ulcerations, and Pigmentation changes. Therefore, although red areas, patches, and excrescences resemble erythroplakia, leukoplakia, and smoker's palate, respectively, they are not classified in these terms.⁴

PALATAL KERATOSIS

Palatal keratosis denotes the diffuse whitening of the palatal mucosa. It may be mild, moderate, or severe in intensity. Palatal keratosis may occur independently or coexist with other components.

EXCRESCENCE

Comprises 1 cm elevated areas, often with central red dots marking the orifices of the palatal mucosal glands. Some 46% of the palatal changes consist of excrescences. Excrescences represent the initial palatal reaction and they are generally transient. The milder form of excrescences resembles the smoker's palate seen in conventional smokers.

REVERSE DHUMTI SMOKER'S LESION

Dhumti is a kind of cigar used in Goa. It is smoked reverse by a small section of people. This form of smoking produces a palatal reaction akin to, but less severe than the palatal changes induced by reverse chutta smoking.

PATCHES

Patches are well-defined, elevated plaques, which could qualify for the clinical term leukoplakia. Palatal patches show characteristic histologic features that differ from the features of leukoplakia. Patches can be small or large. These account up to 12% of the palatal components.

RED AREAS

Red areas are well-defined reddening of the palatal mucosa. Clinically, they are indistinguishable from erythroplakias. Red areas form only 2% of the palatal components. Nevertheless, they are the most serious, showing epithelial dysplasia in 52% of the cases. Longterm studies demonstrate a high rate of malignant transformation.

ULCERATED AREAS

Ulcerated areas are characterized by crater-like ulcerations with deposits of fibrin often surrounded by keratinization. Ulcerations form only 2% of the palatal components. They represent a "burn" type reaction of the palatal mucosa from the intense heat of the lighted end of chutta.

HYPERPIGMENTATION

Pigmentation changes that include hyperpigmentation and loss of pigmentation occur in almost all reverse chutta smokers. Hyperpigmentation manifests in various forms, such as the spotted, linear, patchy, diffuse, and reticular types. Palatal pigmentation in reverse smokers is perhaps a protective reaction to the heat and smoke.

NON PIGMENTED AREAS

Non pigmented areas indicate areas of palatal mucosa, which are clinically devoid of melanin pigmentation. Nonpigmented areas result following the regression of red areas. Loss of pigmentation may render the palatal mucosa more vulnerable to the action of carcinogens in tobacco. Epithelial dysplasia was observed in 19% of nonpigmented areas. The palatal red area is the most dangerous component of palatal changes. Indicate all palatal patches and red areas must be biopsied. If they show moderate or severe epithelial dysplasia, they must be treated accordingly. All patients must be educated to discontinue their tobacco use, as it is known that discontinuation will lead to higher regression rates of palatal changes.⁶

ORAL CANCER

Cancer is a multifactorial disease, with the etiological agents being clinical carcinogens, physical factors and possibly, viral agents. Tobacco use leads to cancer as a result of alterations in cellular growth control processes, together with changes in the interactions between cells and their surroundings which give rise to invasion and metastasis. Many signs and symptoms of oral cancer can be divided into early and late presentations. Early are persistent red or white patch, non healing ulcer, progressive swelling, sudden tooth mobility, unusual oral bleeding or epistaxis, prolonged hoarseness of voice. Late are indurated area, parasthesia, dysthesia of tongue or lips, airway obstruction, chronic earache, trismus and dysphagia, cervical lymphadenopathy, persistent pain referred pain. Patient should be treated according to stage of cancer.8

CONCLUSION

Tobacco in various forms is the largest single preventable cause of death and diseases. It is causally related to oral cancers and precancerous lesions. It is also responsible for an array of oral lesions that are not considered precancerous failing to recognize which, can lead to cancer phobia. All oral mucosal lesions encountered in a tobacco user should

be carefully examined and diagnosed. They should be correlated with the habits and considered for long term follow up. Thus a thorough knowledge and understanding of these tobacco related lesions is essential for differential diagnosis and patient management.

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