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A Hospital Based Survey on Tobacco Associated Lesions

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I. INTRODUCTION

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

Tobacco is one of the most important cause of both addiction and development of oral cancer. Christopher Columbus first discovered tobacco and was widely used in Europe at that time. Later Spanish and Portuguese sailors carried this tobacco to other parts of world. Initially tobacco is known as a plant with medicinal values and was used in various forms like tobacco ointments, pastes, mouth rinses etc., to treat various maladies.[2]

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.[1]

The aim of our hospital based study was to determine the prevalence of tobacco induced lesions in Central India in the city of Bhopal.

INCLUSION CRITERIA –

1.Habbit history of tobacco and associated products
2.Above 18 years of age

II. MATERIALS AND METHOD

A total number of 40600 patients from September 2021 till September 2022 with different oral and dental symptoms were screened out of which 10500 patients satisfying inclusion and exclusion criteria were included in the present study. Before the commencement of the study informed consent was taken. The patients were interviewed for their tobacco associated habits and examination was done by trained dental professionals for the presence of any tobacco associated lesion (TAL). Along with this patients demographic details were also taken and detailed information regarding history was also recorded. Using software STAT 13.1 percentage analysis of various tobacco associated lesions was done and the following results were obtained.

III. RESULTS

In our study the most prevalent tobacco induced lesion was found to be Smoker's palate followed by oral submucous fibrosis and leukoplakia. We found more than one lesion in a single person also.

TYPE OF LESION	**PERCENTAGE (%)**
SMOKER'S PALATE	75
ORAL SUBMUCOUS FIBROSIS	72
LEUKOPLAKIA	69
TOBACCO POUCH KERATOSIS	56
CHEMICAL BURN	5

PAN ENCRUSTATION	2
ORAL CANCER	1.5
SNUFF DIPPERS LESIONS	0
PALATAL CHANGES IN REVERSE SMOKERS	0

IV. DISCUSSION

Tobacco Induced Lesions

Several lesions are caused by tobacco. They are :-

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. Nicotine stomatitis presents as a red circular lesion around the orifice of the minor salivary glands on hard palate mucous. Its prevalence rate is 2%. In the early stage, Depending on the smoking habit and duration, the heat released attacks the palatal mucosa and minor salivary glands and produce the characteristic clinical picture. Hard palate turns gray to white coloured depending on the amount of smoke. Scattered raised areas are ususally present with red coloured centers. Raised areas are formed by the clumps of minor salivary glands and their duct openings are seen as red dots because they do not keratinize instead the entire palate undergo keratinization and appears white. 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. It is not a potential Pre malignant condition.[1] In our study 75% prevalence of smoker's palate was found.

ORAL SUBMUCOUS FIBROSIS

Oral submucous fibrosis (OSMF) is a chronic, potentially malignant disorder of oral mucosa which was first described by Scwartz in 1952 .Oral submucous fibrosis is a chronic disease affecting the oral mucosa, as well as the pharynx and the upper two-thirds of the esophagus. The major effect of this disease lies in its inability to open the mouth and possessing the highest malignant transformation rate which accounts to about 7-13%. The Etiopathogenesis of OSMF is much more complex and involves multiple factors such as consumption of chillies, nutritional deficiencies, betel quid chewing, genetic susceptibility, altered salivary constituents, autoimmunity and collagen disorders. Clinically characterized by burning sensation in mouth, reduced mouth opening, blanching of mucosa, ulceration and palpable fibrous bands. We found OSMF in 72% patients.

LEUKOPLAKIA

In 1978, WHO defined leukoplakia as a white patch or plaque that cannot be characterized clinically or pathologically as any other disease. Later in 1984 at International seminar at Malmo, leukoplakia was defined as "A white patch or plaque that cannot be characterized clinically or pathologically as any other disease and which is not associated with any physical or chemical agent except the use of tobacco. The clinical types of leukoplakia are (1) homogeneous (2) ulcerated (3) ulcerated with pigmentation (3) nodular (4) speckled and (5) verruciform.[3]

It has a prevalence rate of 2.65% and malignancy conversion rate ranging from 0.12% to 17.51%.[4.] Our study found 69% prevalence of leukoplakia.

Leukoplakia 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.

Clinically, smoking induced leukoplakias are characterised by fine white striae that imitate a fingerprint pattern in the mucosa. So, the lesions are referred to as fingerprint lesions or a pumice stone type of lesion which possibly disappear upon tobacco cessation and are generally highly potential pre-malignant disorder of the oral cavity.

There is a site and tobacco habit relationship in leukoplakia

1. **Leukoplakia on buccal mucosa**: It is seen in individuals who smoke until only the small "butt" of cigarette remains.
2. **Hookli associated leukoplakia**: The stem of hookli becomes hot when smoked causing leukoplakic lesions on lower and upper labial mucosa. These lesions usually have a delicate keratinized appearance.

3. **Mishri associated leukoplakia**: Mishri is a roasted powdered tobacco popularly used for application over teeth and gingiva. These lesions occur most often on labial mucosa and gingiva. The associated lesions are thick and extensive, faint and small.
4. **Homogeneous leukoplakia on labial commissure**: It is most common in beedi smokers
5. **Ulcerated leuoplakia**: It is most common in commissure areas
6. **Khaini associated leukoplakia**: These lesions are usually thin and white with cracked mud appearance occurring most commonly in premolar region of the buccal mucosa.
7. **Nodular leukoplakia** shows higher risk for malignant transformation.[3]

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.Typically it occurs in the buccal sulcus or the labial sulcus and corresponds to the site where the tobacco is held in the mouth and is usually painless .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.[1] In our study we found tobacco pouch keratosis in 56% of patients.

CHEMICAL BURN

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.[1] Only 5% prevalence of chemical burn was in our study.

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.[1] We found only 2% prevalence of pan encrustation.

ORAL CANCER

Cancer is a multifactorial disease, It is traditionally defined as Oral Squamous cell carcinoma (OSCC) because in the dental area 90% of cancers are histologically originated in squamous cells, 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 signs and symptoms are persistent red or white patch, non healing ulcer, progressive swelling, sudden tooth mobility, unusual oral bleeding or epistaxis, prolonged hoarseness of voice whereas 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.[1] 1.5% of patients had oral cancer in our study.

SNUFF DIPPERS LESIONS

According to Hirsch et al, in snuff users there will be higher incidence of keratinized lesions, sialadenitis and slight dysplasia. The oral mucosa reacts to snuff by inducing hyperplasia in the basal cell layers and lethal damage in the surface layers. Juxta-epithelial band of sulphated mucopolysaccharides (GAGs) is usually seen as a stromal reaction to external damage by the snuff. The salivary glands and excretory ducts exhibits degenerative changes. Sialadenitis is more common compared to pathological changes in mucosal epithelium. The loss of salivary gland functions leads to a decreased production of saliva and hence decreased protection of the epithelium against snuff and other exogenous factors.[3]

PALATAL CHANGES IN REVERSE SMOKERS

Reverse smoking evokes diverse alterations in the palatal mucosa which can be of various patterns such as palatal keratosis, excrescences, patches, red areas, ulcerations and pigmentation changes

(1) Palatal keratosis presents as diffuse whitening of palatal mucosa.
(2) Excrescences are 1-3 mm elevated reas, with central red dots representing the orifices of palatal minor salivary glands.
(3) Patches are well defined, elevated plaques.
(4) Red areas are well defined reddening of palatal mucosa.
(5) Ulcerated areas are crater like ulcerations with deposits of fibrin surrounded by keratinization.
(6) Hyperpigmentation is a protective reaction to heat and smoke from tobacco.
(7) Nonpigmented areas denote areas devoid of melanin pigmentation. Loss of pigmentation renders oral mucosa more vulnerable to action of carcinogens in tobacco.[3]

No patients of snuff dippers lesions and palatal changes associated with reverse smoking were seen in our study. We will further continue our study and expand our results.

V. 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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