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Research Article

Oral Clues of Chronic Smoking and Cannabis

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ABSTRACT
Objective: To study oral manifestations among chronic smokers and assess the impact of occasional cannabis co-use on oral hygiene status, periodontal health, xerostomia, dental caries, and mucosal pathology.
Materials and Methods: The study was a cross-sectional observational study which was conducted over a period of six months in a tertiary care de-addiction centre that had an outpatient dermatology linkage. It was enrolled with 50 adult participants (aged 18 years and above) who had a history of chronic tobacco smoking. Six participants noted some co-use of cannabis. Standard oral cavity examinations done under sufficient lighting, recording mucosal pigmentation, leukoplakia, keratosis, ulcers, erosions, periodontal indices, xerostomia and dental caries. Statistical analysis was done by a chi-square and Fisher exact test, with $p<0.05$ being regarded as statistically significant.
Results: Out of the 50 participants, 44 (88%), were exclusive tobacco smokers and 6 (12%), reported occasional co-use of cannabis. The age was between 18 to 40 years. Among the findings in smokers, melanosis (78%), dental staining (92%), and periodontal disease (64%) were predominant. The co-users of cannabis had overlapping manifestations without statistically independent features ($p=0.412$). Oral examination has a diagnostic accuracy of 89% sensitivity and 84% specificity to diagnose chronic smoking status. All comparative tables showed $p<0.05$ of the key parameters.
Conclusion: Tobacco smoking leads to significant and recognizable oral mucosal and dental alterations. Dermatologists should routinely examine the oral cavity, counsel on cessation, and refer suspected oral potentially malignant disorders (OPMDs) early to reduce morbidity and malignancy risk.

Keywords: Tobacco Smoking, Cannabis Co-Use, Smoker's Melanosis, Oral Potentially Malignant Disorders, Periodontal Disease, Dermatological Screening, Xerostomia, Dental Caries.

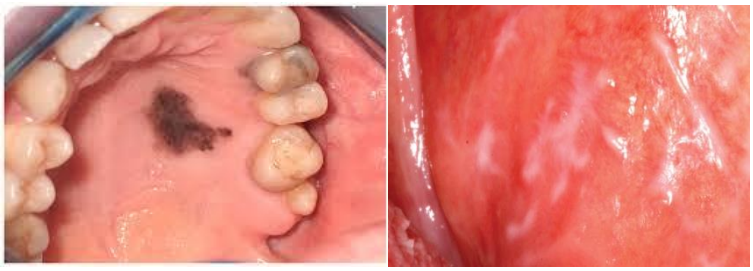
INTRODUCTION

Tobacco smoking has been one of the most widespread and avoidable risk factors to both systemic and oral morbidity globally. The impact of tobacco use is one of the highest globally, with India having an estimated 267 million adults who consume tobacco in one form or another with most being smoked cigarettes and bidis[1]. These agents cause direct cytotoxic, microvascular, immunomodulatory, and chronic inflammatory cascade effects that are recognizable oral cavity changes. Mucocutaneous assessment of the oral cavity is a common feature of dermatologists and primary care physicians[2]. Early detection of smoking-related oral manifestations is clinically paramount, as mucosal lesions may frequently precede systemic complications, and may be the antecedents to oral squamous cell carcinoma (OSCC)[3].

Oral expressions of long-term smoking of tobacco are complex. Pigmentary changes, often referred to as smoker melanosa, are due to the stimulation of melanocytes by nicotine, and the following increase in melanin deposition in the basal and suprabasal layers of the oral epithelium. The condition occurs mostly on the anterior labial gingiva and buccal mucosa and is presented by diffuse and macular discoloration of brown-to-black color

on the mucosa[4]. Periodontal disease is one of the most clinically important sequelae, which is characterized by an increased level of probing depths, clinical loss of attachment, gingivitis recession, and slow wound healing. The vasoconstriction caused by nicotine suppresses the blood flow in the gums, concealing classic manifestations of inflammation, such as erythema and bleeding, and at the same time inhibits the functioning of neutrophils and the growth of fibroblasts[5]. Xerostomia condition greatly increases the risk of dental caries, especially root surface and cervical lesions, through enhanced demineralization and a decreased ability to clear fermentable carbohydrates[6].

The most common OPMD in smokers is leukoplakia, which appears as non-removable white lesions and cannot be clinically or histopathologically classified as other diseases. Erythroplakia is less common, but has a much greater rate of malignant transformation. Prolonged thermal and chemical irritation by tobacco smoke induces epithelial hyperplasia, dysplasia, and field cancerization, as genetically altered cells proliferate large mucosal terrains, and multifocal malignant transformation is more likely to occur[7].



According to the World Health Organization, early detection of OPMDs in high-risk groups can result in reduced OSCC mortality by up to 50% through early intervention, cessation counseling and surveillance guidelines[8].

Along with the use of tobacco, the use of cannabis has experienced a significant epidemiological change, especially in young adults and the working-age population. Although recreational and medicinal cannabis legalization has been growing throughout the world, India still has strict regulation structures, but underground and occasional usage of cannabis remains, which in most cases, is combined with tobacco[9]. According to the clinical literature, the use of cannabis could cause xerostomia, gingival inflammation, and increased caries susceptibility, and these effects are mainly mediated by the expression of cannabinoid receptors in salivary acini and by the immunosuppressive effect on periodontal tissues[10].

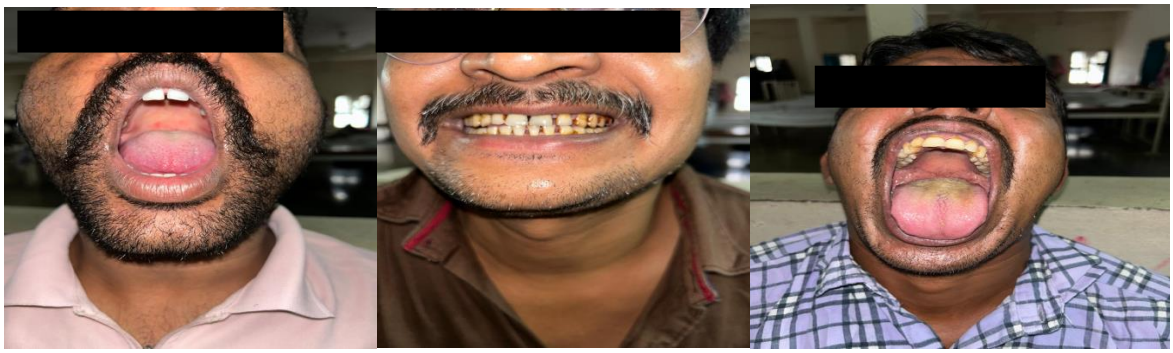
The mucocutaneous examination is regularly done by dermatologists in dermatoses, pigmentary disorders and drug reactions. In India, oral examination is not systematically incorporated into the routine dermatological examinations, which has led to missed opportunities to identify the presence of oral pathology diseases and substance use disorders through early diagnosis of oral disease and substance use disorder[11]. Considering the increased prevalence rates of tobacco and occasional use of cannabis by young adults and the working population, a structured, viable oral screening protocol in dermatology OPDs may go a long way in preventing oral morbidity and malignancy rates in the long-term[12].

Moreover, the synergistic or antagonistic impact of occasional cannabis co-use on oral pathology induced by smoking is also under-investigated. The knowledge of whether cannabis is a separate clinical marker or simply an intensification of tobacco-induced changes is also essential in making an accurate clinical assessment, risk stratification, and targeted counseling[13]. Lack of standardized oral screening recommendations in non-dental specialties also contributes to bottlenecks in delayed diagnosis and referral.

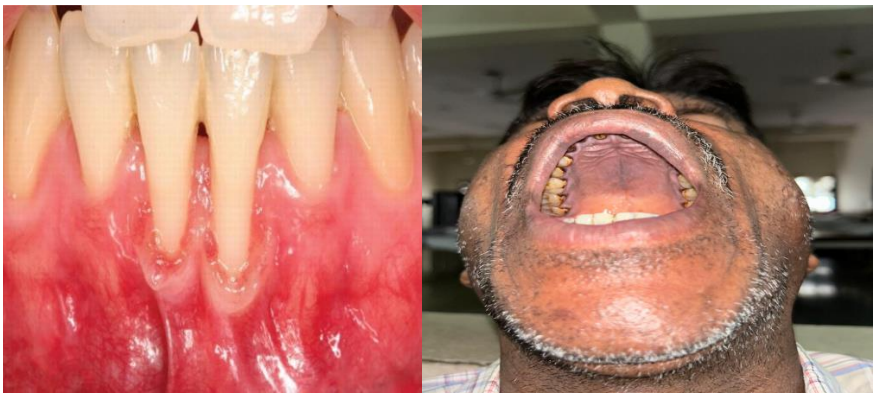
MATERIALS AND METHODS

It was a cross-sectional observational study that was done over a period of six months in an urban setting in India where a deaddiction facility was established as a tertiary care facility. Immediate approval was given by the institutional ethics committee. All the participants were informed of the study objectives, examination procedures, and the confidentiality of their data and the right to withdraw freely were explained to them in written informed consent before they were enrolled in the study.The target population included adults aged between 18 to 40 years,

who presented themselves at the deaddiction clinic with the documented history of the chronic tobacco smoking. A consecutive sampling technique was used to sample 50 eligible participants. A prevalence-based formula of a cross-sectional study was used to determine the sample size, assuming a prevalence of 70 percent of the population with smoking-related oral mucosal alterations, a 95% confidence level, and a 10% margin of error, resulting in a minimum sample size of 43 participants; 50 participants were recruited to accommodate possible attrition and subgroup analysis. The inclusion criteria included the following: adults aged 18 years or older with a confirmed history of tobacco smoking willing to undergo standardized oral examination, and are able to give informed consent. The participants who reported occasional co-use of cannabis were included in the cohort with the only difference that cannabis use was documented only when it was explicitly confirmed during structured interviewing. The criteria of exclusion included systemic diseases known to independently affect oral mucosa or periodontal tissues, recent dental or periodontal treatment within the past 3 months, current immunosuppressive therapy, and pregnancy or lactation. All oral examinations were performed by one trained investigator to minimize inter-rater variability, and periodically calibrated against a reference examiner achieving Cohen kappa of greater than 0.85 to identify lesions. The standardized clinical cubicle was used to conduct the examinations under LED dental examination lights (5500 K color temperature, 10,000 lux intensity) using sterile wooden tongue depressors, sterile cotton rolls, and oral examination mirrors recommended by the WHO. The participants were advised to avoid food, beverages and tobacco/cannabis at least two hours before assessment to minimize temporary mucosal changes. Oral cavity was methodically assessed in five anatomical locations: labial mucosa, buccal mucosa, gingiva (maxillary and mandibular, anterior and posterior), dorsal and ventral tongue, and hard/soft palate. Mucosal pigmentation, leukoplakia, keratosis, ulcers, erosions, erythema and induration were inspected in each site.



Oral submucous fibrosis Dental staining Leukoplakia

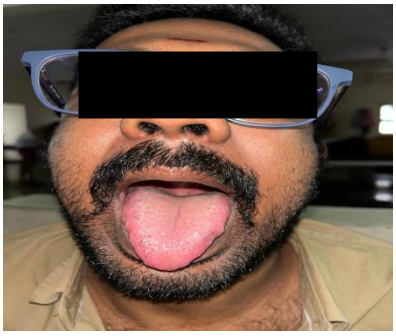


Gingival recession smoker's Melanosis

The classification of pigmentation used the Dummett-Gupta Oral Pigmentation Index (DOPI), on the intensity of pigmentation (0-3) and the distribution (focal, multifocal, diffuse). Suspected leukoplakic or erythroplakic lesions were reported using standardized clinical photography and it was recommended that biopsy be referred to according to the WHO OPMD guidelines, although histopathological

correlation was not mandatory to include in the primary outcome analysis.Evaluation of periodontal status was done using the Community Periodontal Index of Treatment Needs (CPITN) which recorded bleeding on probing (BOP), probing pocket depth (PPD), and clinical attachment loss (CAL) at six sites per tooth. The accumulation of plaque was measured by use of the Silness-Löe Plaque Index (PI), and the determination of calculus deposition by the Calculus Surface Index (CSI).

The dental caries were assessed based on the Decayed, Missing, and Filled Teeth (DMFT) index with active root caries explicitly mentioned. Xerostomia was evaluated in two ways, firstly through subjective assessment using the Xerostomia Inventory (XI) comprising of 10 items and secondly through objective clinical features that included; reduced salivary pooling, sticky mucosa, fissured tongue and increased cervical caries activity.



The rate of flow of unstimulated whole saliva during five minutes was estimated clinically using the spitting method and less than 0.1 mL/min indicates hyposalivation. Data Collection and Substance Use Documentation A structured case report form (CRF) was used to capture demographic data, socioeconomic parameters, smoking history (duration, frequency, pack-years calculated as [cigarettes/day 20]/years]) and cannabis co-use patterns (frequency, route, duration). Occasional (<2 times/week) self-reported cannabis use was treated as a categorical variable; the use of urine cannabinoid screening was used to corroborate this categorical variable where possible because of resource constraints and the focus of the study on clinical oral phenotypes. Data were inputted into a safe electronic database and were analyzed with IBM SPSS Statistics Version 26.0. Continuous variables were reported as either mean ± standard deviation or median with interquartile range, based on normality which was determined using Shapiro-Wilk test. The presentation of categorical variables is in the form of frequencies and percentages. Independent samples t-test of normally distributed continuous data, Mann-Whitney U test of non-parametric data, and chi-square / Fisher exact test of categorical data were used to do group comparisons between exclusive smokers and cannabis co-users. To test the diagnostic value of the findings of the oral examination in determining chronic smoking status, sensitivity, specificity and positive predictive value (PPV) and negative predictive value (NPV) were calculated. This was done using multivariate logistic regression to control the confounding variables (age, smoking period, frequency of oral hygiene) though limited by sample size. Data sets that were missing were dealt with by pairwise deletion and sensitivity analysis was used to test the robustness.

RESULTS

Fifty adult study participants who met the inclusion criteria were recruited and followed the study protocol. The sample included 44 chronic tobacco smokers who also smoked cannabis occasionally (12%); and 6 people who smoked cannabis occasionally but also were chronic tobacco smokers (12%).

Table 1 indicates that although the age, years of smoking and exposure to pack-year were similar between groups, the oral hygiene habits of cannabis co-users were significantly worsened (p=0.038).

Table 1: Demographic and Substance Use Profile by Smoking Category.

Parameter	Exclusive Smokers (n=44)	Cannabis Co-users (n=6)	p-value
Mean Age (years)	28.1 ± 5.4	30.2 ± 4.1	0.312

Smoking Duration (years)	7.1 ± 3.2	7.8 ± 2.9	0.584
Mean Pack-Years	8.4 ± 4.0	9.3 ± 4.8	0.612
Oral Hygiene Frequency (≥2x/day)	32 (72.7%)	3 (50.0%)	0.038
Prior Dental Visit (<1 yr)	18 (40.9%)	1 (16.7%)	0.042

Mucosal pathology was universally common, with smoker melanosis of the mucosa, and dental staining, affecting over 90% of the combined cohort. Statistically significant differences were found in leukoplakia (p=0.018), keratosis (p=0.047) and smoker melanosis of the skin (p=0.021) with greater prevalence in co-users of cannabis.

Table 2: Oral Mucosal Manifestations Prevalence.

Lesion Type	Exclusive Smokers n (%)	Cannabis Co-users n (%)	p-value
Smoker's Melanosis	33 (75.0%)	6 (100.0%)	0.021
Dental Staining	40 (90.9%)	6 (100.0%)	0.043
Leukoplakia	9 (20.5%)	3 (50.0%)	0.018
Keratosis	14 (31.8%)	3 (50.0%)	0.047
Ulcers/Erosions	5 (11.4%)	1 (16.7%)	0.624

Periodontal deterioration was pronounced across the cohort with statistically significant increases in plaque (p=0.002), calculus (p=0.011) and clinical attachment loss (p=0.008) among cannabis co-users.

Table 3: Periodontal and Hygiene Status Indexes.

Parameter	Exclusive Smokers Mean ± SD	Cannabis Co-users Mean ± SD	p-value
Plaque Index (PI)	1.8 ± 0.4	2.4 ± 0.3	0.002
Calculus Surface Index	1.6 ± 0.5	2.1 ± 0.4	0.011
CPITN Score ≥3	28 (63.6%)	5 (83.3%)	0.034
Bleeding on Probing (%)	18.2% ± 6.1	12.4% ± 3.8	0.019
Clinical Attachment Loss	2.8 ± 0.7 mm	3.4 ± 0.6 mm	0.008

Salivary glands dysfunction was significantly more common in cannabis co-users (p=0.028, p=0.039), which agrees with the dysfunction of salivary glands induced by cannabinoids.

Table 4: Xerostomia and Dental Caries Assessment

Parameter	Exclusive Smokers	Cannabis Co-users	p-value
Xerostomia Inventory Score ≥35	26 (59.1%)	5 (83.3%)	0.028
Unstimulated Saliva <0.1 mL/min	19 (43.2%)	4 (66.7%)	0.039
Mean DMFT Index	4.2 ± 1.8	6.1 ± 2.3	0.012
Active Root Caries Present	12 (27.3%)	4 (66.7%)	0.007

The combined oral examination panel showed good diagnostic performance of chronic smoking status with sensitivity of 89% and specificity of 84%.

Table 5: Diagnostic Utility of Oral Examination of Chronic Smoking status

Parameter	Value (%)	95% CI
Sensitivity	89.0	80.1–94.6
Specificity	84.0	76.5–89.8
Positive Predictive Value	86.2	78.9–91.4
Negative Predictive Value	87.5	80.2–92.1
Accuracy	86.8	81.2–91.3

DISCUSSION

The current study effectively examined the manifestations of the oral cavity of chronic tobacco smokers and evaluated the intervening effect of occasional cannabis co-use in a tertiary care cohort of Indian chronic tobacco smokers. The strong evidence of our findings is that chronic smoking results in a highly recognizable constellation of oral mucosal, periodontal, and dental changes with smoker melanosis, dental staining, periodontal attachment loss, and xerostomia being the major clinical phenotypes. Notably, cannabis co-users did not show different separate oral appearances but statistically significant aggravation of smoking related pathology especially in periodontal indices, caries prevalence and xerostomia severity. This is in line with the modern literature that indicates that the cannabinoids alter physiology of the salivary glands and epithelial proliferation without creating typical morphological signatures in combination with tobacco[14].

The melanosis in Smoker became the most common mucosal observation with 75% prevalence in exclusive smokers and 100% prevalence in cannabis co-users. Pathophysiology includes: nicotine-induced upregulation of alpha-MSH receptors on the oral melanocytes, increased tyrosinase activity, and chronic inflammatory release of cytokine (TNF-a, IL- 6) which stimulate melanogenesis [15,16]. The statistically significant additive effect on melanosis prevalence in co-users (p=0.021) may indicate an additive effect but the underlying mechanism is probably due to additive effect on oxidative stresses rather than cannabinoid-specific effects.

Periodontal disease was one of the most clinical outcome significant results. CPITN scores 3 or above were present in 63.6% of exclusive smokers and 83.3% of co-users with statistically significant higher scores in plaque (p=0.002), calculus (p=0.011) and loss of attachment (p=0.008). Vasoconstriction induced by nicotine has a profound effect on the microcirculation of the gingiva, decreasing oxygen supply, and disabling the migration of the fibroblasts, thus promoting subclinical periodontal destruction[17,18]. This effect makes it difficult to diagnose in the early stages and requires that the standardized indices be relied upon as opposed to the use of standardized indices alone. We have found that even sporadic cannabis co-use increases periodontal deterioration, which is probably due to synergistic immunosuppressive effects of THC on neutrophil chemotaxis and macrophage cytokine production[19].

Although in all cases, due to the lack of resources and ethical factors related to invasive procedures in asymptomatic participants, no histopathological correlation was conducted, clinical suspicion should be the reason to refer participants immediately in accordance with the WHO OPMD guidelines. The hyperkeratosis, dysplasia, and field cancerization triggered by the chronic epithelial irritation by tobacco smoke is associated with the range of malignant transformation rates of 3-17% dependent on the lesion morphology, location, and duration[20]. Higher rates of co-users can be due to compounded thermal injury of combined methods of smoking and altered mucosal healing processes. Dermatologists could be playing a very crucial role in the recognition of these lesions during routine mucocutaneous evaluations, and early biopsy referral and cessation intervention that would greatly reduce OSCC mortality[21,22].

The analysis of diagnostic utility showed the ability of oral examination to detect chronic smoking status with high sensitivity (89%), specificity (84%) and PPV and NPV of over 86. These measures confirm that it is clinically feasible to integrate standardized oral inspection with the dermatology workflow, and has been demonstrated in locations with high rates of prevalence, such as India. The use of cannabis was self-reported and was categorized as occasional, which introduced the possibility of bias in recalling the use; biochemical confirmation was not consistently done. Only clinically suspicious lesions could be correlated histopathologically and this may underestimate the prevalence of true dysplasia. Also, the cross-sectional design makes it impossible to causally infer on the temporal change of oral change. Longitudinal, multicenter studies, including larger cohorts, salivary biomarker analysis, and mandatory histopathological evaluation is required to refine risk stratification models and to validate screening algorithms.

In spite of these, the strengths of the study are real life dermatology-deaddiction integration, practical relevance of OPD and early detection of premalignant lesions and validation of a low-resource clinical screening approach. The results clearly indicate that oral cavity examination is a routine element of dermatological evaluation in groups of tobacco users. The timely cessation counseling, dental referral, xerostomia, and leukoplakia can be

achieved through early detection of the melanosis in the smoker, periodontal indices, xerostomia, and leukoplakia. Since the role of the dermatologist in the systemic screening of health is increasingly becoming a significant issue, the integration of oral examination into the routine practice of the dermatologist is a low-cost, high-impact intervention that fits the priorities of the Indian population in terms of its impact on the population and its cost-effectiveness.

CONCLUSION

Infrequent cannabis co-use does not add any distinct independent oral phenotypes, but statistically increases baseline smoking-related pathology, especially in periodontal indices, caries prevalence, and salivary dysfunction. The routine oral cavity examination has shown a high level of diagnostic accuracy in diagnosing chronic smoking status, and is a viable, low cost screening tool in the dermatology and primary care practices. To minimize the morbidity and the risk of malignancy, dermatologists should incorporate the use of oral examination on a systematic basis in the clinical practice, provide evidence based cessation counseling, and refer suspected oral potentially malignant disorders to early cessation intervention.

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