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A clinicopathological study of oral premalignant and malignant lesions with a special focus on gingivobuccal complex in a tertiary care center

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ABSTRACT

Background: Globally, oral cavity lesions are a serious public health issue. Premalignant lesions of the oral cavity, such as leukoplakia, erythroplakia, and oral submucous fibrosis, are common in India and are strongly associated with tobacco and betel nut use. These lesions have the potential to transform into malignancies, with squamous cell carcinoma (SCC) being the predominant type of oral cancer. Early detection and effective management of early malignant lesions are crucial to improving the prognosis for patients. Histopathological examination is a standard procedure used to identify these lesions. **Aims and Objectives:** The objective of this study is to examine the histopathology of premalignant and malignant lesions of the oral cavity, taking into account age, sex, site distribution, and their association with tobacco consumption. **Materials and Methods:** The study was a prospective, observational study carried out over a period of 3 years on patients with lesions of the oral cavity in a Government Medical College of Northern Odisha. Biopsies were obtained, fixed, and routinely processed. Sections were stained with Hematoxylin and Eosin. The final diagnosis was based on clinical, gross, and microscopic findings. The data was collected and analyzed. **Results:** Out of a total of 197 oral biopsies, 79 cases were malignant lesions, and 35 cases were pre-malignant lesions. The highest incidence of both pre-malignant and malignant lesions was observed in the age group of 51–60 years, with a male predominance. The buccal mucosa was the most affected site. The most frequent type of malignant lesion was well-differentiated SCC, while hyperkeratosis without dysplasia was the most common type of pre-malignant lesion. **Conclusion:** Cancers of the oral cavity are surface malignancies whose signs and symptoms can be recognized early. The most common subsite of cancer is the gingivobuccal complex, and site-specific carcinoma is often due to the use of tobacco and betel quid, as they are typically placed in the gingivobuccal sulcus. The occurrence of carcinoma can be minimized by creating public awareness regarding high-risk habits.

Key words: Histopathology; Oral cavity; Pre-malignant; Malignant

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INTRODUCTION

Oral cavity lesions, both premalignant and malignant, represent a significant public health concern worldwide. The incidence of oral cavity cancer is increasing globally, and it is estimated that there were over 377,713 new cases and 177,757 deaths worldwide in 2020 alone.[1] Premalignant lesions of the oral cavity, such as leukoplakia, erythroplakia, and oral submucous fibrosis, are characterized by histological changes that have the potential to transform into malignancies.[2] 90% of the carcinomas arising from the oral cavity are squamous cell carcinoma (SCC).[3,4] The malignant lesions of the oral cavity, such as SCC, are invasive and can metastasize, leading to poor prognosis.[4] India accounts for

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approximately one-fourth of global incidences with around 77,000 new cases and 52,000 deaths reported annually.[5] The gingivobuccal complex cancer has been rightly described as the Indian oral cancer.[6] Tobacco and areca nuts are the most common cause of gingivobuccal complex cancer where they are placed, typically gingivobuccal sulcus.[7] Smokeless tobacco consumption, Paan (betel nut) chewing and Human Papilloma Virus infections are among the major causes of oral carcinoma in developing countries like India. In addition, poor dental hygiene, lack of self-care, and an unhealthy diet are also contributing factors to oral cancer.[8] Erythroplakia, leukoplakia, sub-mucous fibrosis, and lichen planus are the most common pre-malignant oral conditions in India, which pose a higher risk of transforming into oral cancer.[9] The clinicopathological characteristics of oral cavity lesions are diverse and can vary depending on the lesion's location, size, stage, and grade. The assessment of these lesions requires a multidisciplinary approach, including clinical examination, imaging, and histopathological analysis. The study of clinicopathological features of premalignant and malignant lesions of the oral cavity is critical for accurate diagnosis, appropriate management, and improved patient outcomes.

Aims and objectives

The primary aim and objectives of the research were to study various histopathological features, age and gender distribution, frequency distribution, and the locations of premalignant and malignant lesions occurring in the oral cavity, as well as the addiction habits of the patients.

MATERIALS AND METHODS

This study was a prospective, descriptive, observational study carried out for 3 years in the Department of Pathology, ENT, and Dentistry, PRM Medical College and Hospital, Baripada, Odisha. The Institutional Ethics Committee of PRM Medical College and Hospital, Baripada, approved this research proposal.

A total of 197 oral cavity lesions were included in the present study. Detailed information about each patient was obtained, including patient age, sex, dental hygiene, personal habit, chief complaint, anatomical site of the lesion, clinical diagnosis, and cytological diagnosis if it was done.

Inclusion criteria

1. Oral cavity lesions
2. Specimen that was adequate and representative of the lesion
3. The study uses surgical specimen, such as punch biopsies, incisional biopsies, wedge biopsies, and surgical excision.

Exclusion criteria

1. Inadequately preserved specimens
2. Incomplete clinical record
3. Lesions arising from the oropharynx
4. Odontogenic lesions.

Specimens were received in 10% formalin, and sections were processed and embedded in paraffin after grossing. Multiple serial sections of 4–5-micron thickness were obtained from the paraffin block and stained with Hematoxylin and Eosin. The histopathological findings of oral lesions were studied and classified as per the World Health Organization (WHO) criteria for oral potentially malignant disorders, oral epithelial dysplasia (OED), and malignant lesions. Oral potentially malignant disorders are clinical presentations that carry a risk of cancer development in the oral cavity, for example, leukoplakia, erythroplakia, and submucous fibrosis. According to the WHO, all cases of OED were classified as mild, moderate, and severe.

The data were collected and entered into a Microsoft Excel spreadsheet in tabulated form. Data on the habit of tobacco use, type of lesions, histopathological diagnosis, age and sex distribution, and site of the lesions were analyzed descriptively using frequencies and ranges.

RESULTS

A total of 197 oral biopsies were received and studied in our department, of which 35 cases were premalignant and 79 were malignant (Table 1).

In age-wise distribution, majority of patients were in the age group of 51–60 years among premalignant and malignant lesions. The male-to-female ratio of malignant lesions was 1.07, with a male predominance, and for premalignant lesions, it was 1.5 (Table 2).

Well-differentiated SCC (Figure 1) was the most prevalent lesion in the malignant group, accounting for 60 cases. This was followed by moderately differentiated carcinoma (7 cases) (Figure 2), mucoepidermoid carcinoma (4 cases) (Figure 3), microinvasive SCC (3 cases), verrucous carcinoma (2 cases) (Figure 4), malignant melanoma (2 cases) (Figure 5), and polymorphous low-grade adenocarcinoma (1 case) (Figure 6) (Table 3).

Table 1: Prevalence of premalignant and malignant lesions

Type of lesion	Number of cases	Percentage
Premalignant	35	17.76
Malignant	79	40.10
Others	83	42.13
Total	197	100

The most common premalignant lesion was hyperkeratosis without dysplasia (14 cases) (Figure 7), followed by mild OED (9 cases), severe OED (7 cases) (Figure 8), moderate OED (2 cases) (Figure 9), oral submucous fibrosis (2 cases), and proliferative verrucous leukoplakia (1 case) (Table 4).

Among nonneoplastic and benign lesions, the most frequent was mucocele (23 cases), followed by squamous papilloma (17 cases) (Table 5).

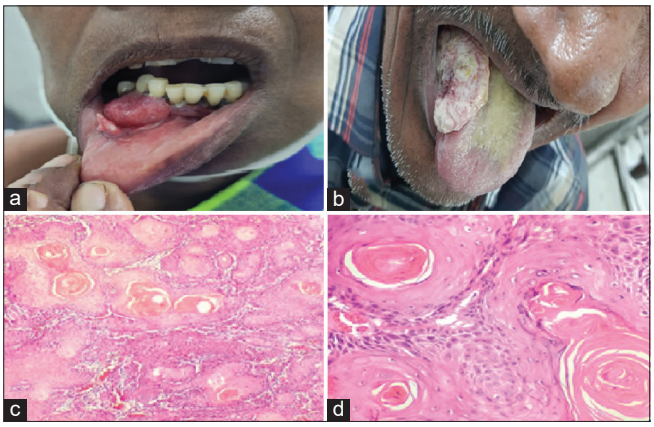

Figure 1: Well-differentiated Squamous Cell Carcinoma. (a) Exophytic growth involving gingiva and buccal mucosa, (b) Exophytic mass over right lateral border of tongue, (c) Photomicrograph showing islands of malignant squamous cells with keratin pearl formation (H&E stain, ×100), (d) Showing dysplastic epithelial cells with keratin pearl (H&E stain, ×400)

Buccal mucosa, including both left and right sites, was the most common site involved in premalignant and malignant lesions, followed by the tongue and gingivobuccal sulcus. The gingivobuccal complex, made up of the gingivobuccal sulcus, alveolus, retromolar trigone, and buccal mucosa, was the most common site of premalignant and malignant lesions of the oral cavity in our study (Table 6).

Lower gingivobuccal sulcus and oral tongue are involved equally in SCC followed by buccal mucosa. The most common site of well-differentiated SCC was the buccal mucosa (18 cases), followed by the lower gingivobuccal sulcus (17 cases), and the oral tongue (16 cases). Moderately differentiated carcinoma affected the oral tongue and lower gingivobuccal sulcus equally, in three cases each. The site of involvement of verrucous carcinoma was buccal mucosa. The most common site involved in mucoepidermoid carcinoma was the hard palate (Table 7).

The most common site for lesions such as hyperkeratosis without dysplasia, mild OED, severe OED, and oral submucous fibrosis was buccal mucosa in this study (Table 8).

With respect to risk factors, patients reported chewing and smoking tobacco, using pan masala, and drinking alcohol. Tobacco chewing was the most common habit

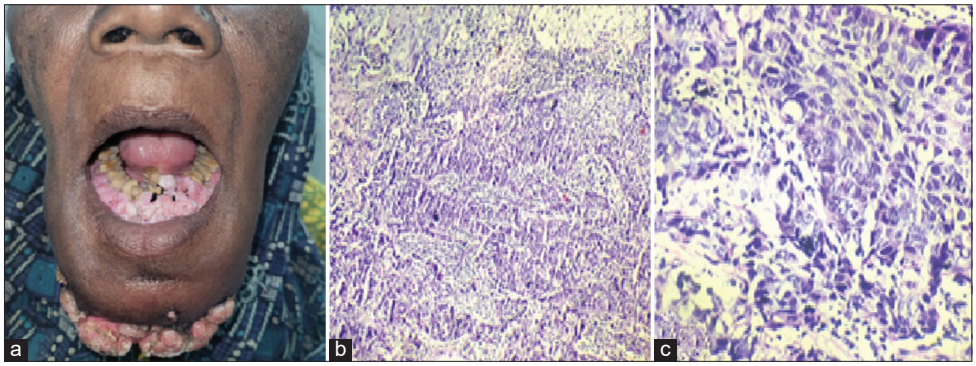

Figure 2: Moderately differentiated Squamous Cell Carcinoma. (a) Exophytic gingivobuccal mucosa lesion with growth over chin, b) Photomicrograph showing malignant cells with no keratinization, but easily recognizable as being squamous epithelial origin (H&E stain, ×100), (c) Showing more pleomorphism, but the squamous nature of tumor is still apparent (H&E stain, ×400)

Table 2: Age and sex distribution of oral premalignant and malignant lesions

Age (in years)	Number of premalignant cases		Total	Percentage	Number of malignant cases		Total	Percentage
	Male	Female			Male	Female		
10–20	0	0	0	0	3	0	3	3.79
21–30	5	0	5	14.28	2	2	4	5.06
31–40	2	2	4	11.42	9	4	13	16.45
41–50	4	6	10	28.57	8	12	20	25.31
51–60	10	3	13	37.14	12	15	27	34.17
61–70	0	3	3	8.57	3	5	8	10.12
71–80	0	0	0	0	3	0	3	3.79
>80	0	0	0	0	1	0	1	1.26
Total	21	14	35	100	41	38	79	100

Table 3: Frequency distribution of malignant lesions

Type of lesion	Number of cases	Percentage
SCC (WD)	60	75.94
SCC (MD)	7	8.86
SCC (PD)	0	0
Microinvasive SCC	3	3.79
Malignant melanoma	2	2.53
Verrucous carcinoma	2	2.53
MEC	4	5.06
PLGA	1	1.26
Total	79	100

SCC: Squamous cell carcinoma, MEC: Mucoepidermoid carcinoma, PLGA: Polymorphous low-grade adenocarcinoma, WD: Well differentiated, MD: Moderately differentiated, PD: Poorly differentiated

Table 4: Frequency distribution of premalignant lesions

Type of lesion	Number of cases	Percentage
Hyperkeratosis without epithelial dysplasia	14	40
Mild oral epithelial dysplasia	9	25.71
Moderate oral epithelial dysplasia	2	5.71
Severe oral epithelial dysplasia	7	20
Oral submucous fibrosis	2	5.71
Proliferative verrucous leukoplakia	1	2.85
Total	35	100

Table 5: Distribution of nonneoplastic and benign lesions of the oral cavity

Type of lesion	Number of cases	Percentage
Granulation tissue	3	3.61
Nonspecific inflammatory lesion	4	4.81
PEH	3	3.61
Mucocele	23	27.71
Squamous papilloma	17	20.48
Fibroepithelial polyp	11	13.25
Capillary hemangioma	8	9.63
Pyogenic granuloma	9	10.84
Fibroma	3	3.61
Lipoma with chondroid metaplasia	1	1.20
Benign spindle cell tumor (leiomyoma)	1	1.20
Total	83	100

PEH: Pseudoepitheliomatous hyperplasia

observed with premalignant and malignant lesions in our study (Table 9).

DISCUSSION

In our study, malignant lesions (79 cases, 40.1%) outnumbered premalignant lesions (35 cases, 17.76%). This was consistent with findings from studies by Tomar and Chouhan (42% malignant, 37% premalignant) and Rathva et al., (44% malignant and 34.28% premalignant).[10,11] Similarly, Kshirsagar et al., and Panda et al., reported a higher prevalence of malignant lesions compared to premalignant lesions.[12,13] In the study by Jagtap et al., out of 173 patients having oral lesions, 21.96% were premalignant lesion and 78.04% were oral cancers.[14] In contrast, Klongnoi et al., found premalignant lesions (57.14%) to be more prevalent than malignant lesions (5.41%).[15]

Majority of the patients were in the age group of 51–60 years among premalignant and malignant lesions in our study. In the study by Tomar and Chouhan majority of patients were in the age range of 46–60 years.[10] Studies by Rathva et al., Kshirsagar et al., and Panda et al., found that the majority of patients were in the age range of 41–50 years.[11-13] Bastakoti et al., found the most common age group was 46–75 years, with the mean age of the cases being 55.9 years in their study.[16]

The male-to-female ratio of malignant lesions was 1.07, with a male predominance, and for premalignant lesions, the ratio was 1.5 in our study. Tomar and Chouhan found a male preponderance with a male-to-female sex ratio of subjects was 2.54 (89:35) in their study.[10] Rathva et al., and Kshirsagar et al., were reported male predominance in their study with a male-to-female ratio of 3.6 and 2.7, respectively.[11,12] Studies by Panda et al., and Pudasaini et al., observed that the incidence of the oral lesion are more among male patients with a male-to-female ratio of 2.93 and 1.33, respectively.[13,17] Senguven et al., found a M/F ratio of 1.02:1 in the study population.[18] Bastakoti et al., in their study, found that three fourth of the cases were male which was the same as the study by Kosam and Kujur.[16,19]

SCC was the most prevalent lesion in the malignant group. On further classifying well-differentiated SCC was the most common followed by moderately differentiated carcinoma (7 cases) in our study. SCC was the predominant malignant lesion observed in studies conducted by Tomar and Chouhan[10] Rathva et al.,[11] Brandizzi et al.,[20] and Dias and Almeida[21] Among SCC cases, moderately differentiated SCC was the most common accounting for 67.31% of subjects, followed by well-differentiated SCC in 28.85% of subjects in the study by Tomar and Chouhan.[10]

The common premalignant lesions observed in various studies included hyperkeratosis without dysplasia, mild, moderate, and severe OED. However, different studies reported varying prevalence rates for these lesions. Tomar and Chouhan observed that keratosis with mild dysplasia was the most common (28.26%), followed by oral submucosal fibrosis (21.83%).[10] Rathva et al., found that keratosis with mild dysplasia (35.41%) was the most common, followed by keratosis without dysplasia (20.83%) and oral submucous fibrosis (20.83%).[11] In the study by Jagtap et al., mild squamous epithelial dysplasia was found

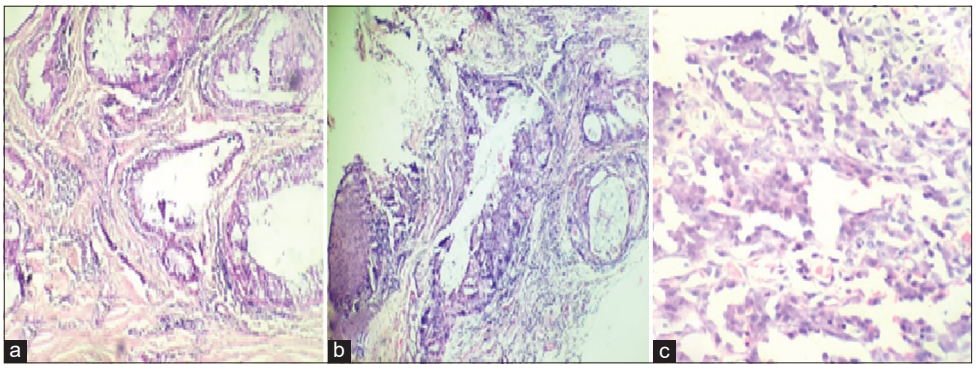

Figure 3: Mucoepidermoid carcinoma. (a) Low grade tumor showing cystic spaces lined by mucous cells (H&E stain, ×100), (b) Intermediate grade tumor showing cyst formation lined by mucous and squamous cells (H&E stain, ×100), (c) High grade tumor consisting of squamous and intermediate cells demonstrating pleomorphism (H&E stain, ×400)

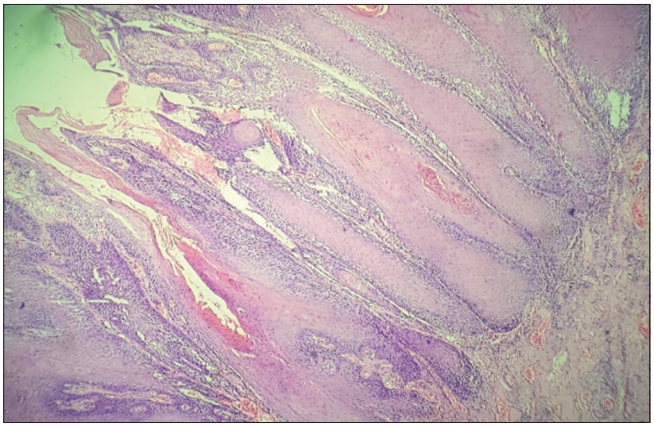

Figure 4: Verrucous carcinoma exhibiting a papillary surface architecture with broad, bulbous, elongated rete pegs reveal pushing encroachment. Prominent keratin clefting is appreciated (H&E stain, scanner view)

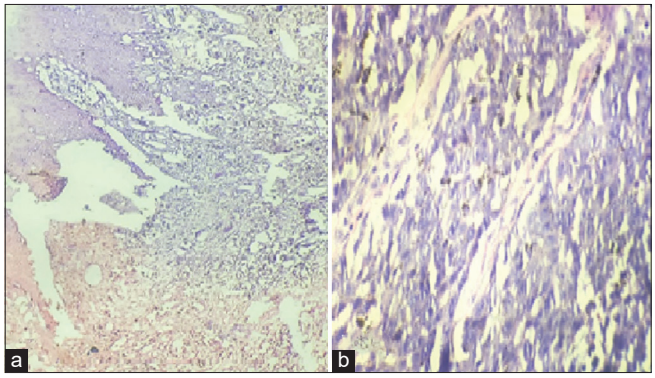

Figure 5: Oral Malignant Melanoma. (a) Photomicrographs showing nests and nodules of tumor cells with melanin pigments (H&E stain, ×100), (b) Showing epithelioid cells with prominent nucleoli (H&E stain, ×400)

in 16 cases (48.48%), followed by severe cases in 12 cases (36.36%).[14] In Bastakoti et al.'s study, 98 cases showed dysplasia, five were lichen planus, and one was proliferative verrucous hyperplasia.[16] Kosam and Kujur reported 14.28% cases of keratosis without dysplasia followed by 5.71% cases of keratosis with mild dysplasia.[19]

In our study, the buccal mucosa was the most affected site in both premalignant and malignant lesions, followed by the tongue and gingivobuccal sulcus. Tomar and Chouhan and Rathva et al., found that buccal mucosa was the most prevalent site involved in oral lesions, followed by the lip and tongue.[10,11] Kshirsagar et al., reported that tobacco-related lesions were most prevalent in the buccal mucosa, followed by the tongue and gingiva.[12] Panda et al., observed that buccal mucosa, followed by alveoli and tongue were the most affected sites.[13] Similarly, Modi et al., found buccal mucosa to be the most common site of involvement, followed by the tongue and gingiva.[22] In Babshet et al.'s study, buccal mucosa was also the most frequently involved site, followed by alveoli and gingiva, the lips, and the tongue.[23]

In our study, SCC was equally involved the lower gingivobuccal sulcus and oral tongue, followed by the buccal mucosa. However, Modi et al., reported that SCC primarily affected the base of the tongue.[22] In our research, well-differentiated SCC predominantly affected the buccal mucosa, while moderately differentiated SCC affected the oral tongue and lower gingivobuccal sulcus equally. Rai and Ahmed found that well and moderately differentiated SCC were most frequently located in the buccal mucosa, followed by the tongue, whereas poorly differentiated SCC affected both sites equally.[24] In Pires et al.'s study, the border of the tongue was the most prevalent site involved by well, moderately, and poorly-differentiated SCC.[25] Mucoepidermoid carcinoma predominantly occurred in the parotid gland, followed by the palate, retromolar area, and buccal mucosa.[26] Among minor salivary glands the hard and soft palate were the preferred sites for mucoepidermoid carcinoma, with the hard palate being the most frequently involved site as in our study.[27]

In our study, the buccal mucosa was the most common site for lesions, including hyperkeratosis without dysplasia, mild OED, severe OED, and oral submucosal fibrosis.

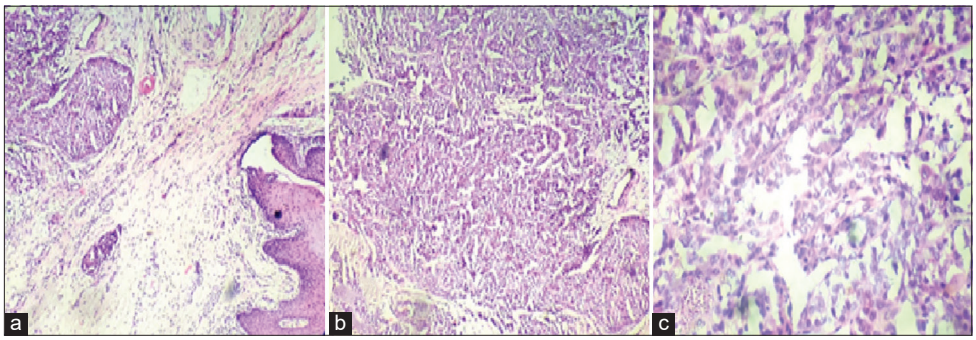

Figure 6: Polymorphous Low-Grade Adenocarcinoma. (a) Photomicrographs showing normal surface epithelium with lamina propria separating the tumor tissue comprising of glandular tissue (H&E stain, ×100), (b) Tumor cells arranged in a ductal pattern (H&E stain, ×100), (c) Uniform round to polygonal tumor cells with indistinct cell borders and vesicular nucleus (H&E stain, ×400)

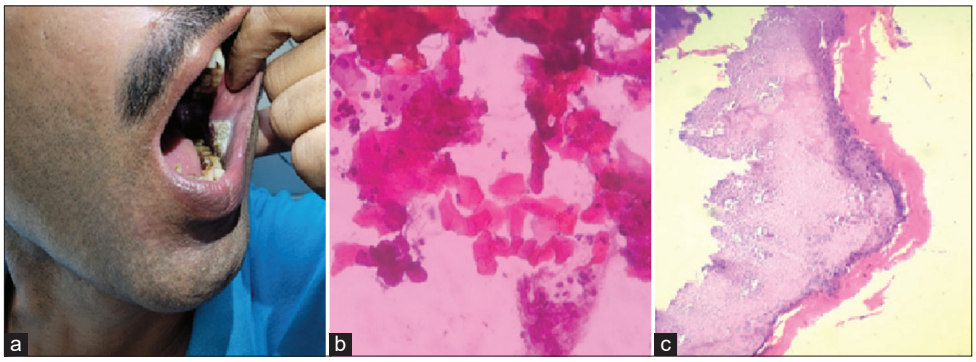

Figure 7: Leukoplakia. (a) Whitish patch over buccal mucosa, (b) Scrape cytology reveals benign mature squamous cells and anucleated squames (H&E stain, ×400), (c) Photomicrograph of leukoplakia exhibits hyperkeratosis with hypergranulosis and no cytological atypia (H&E stain, ×400)

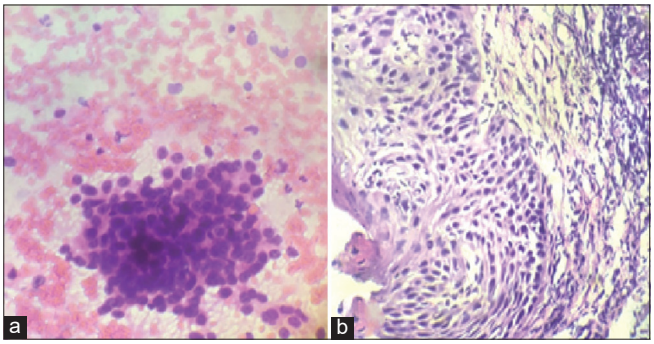

Figure 8: Severe Epithelial Dysplasia. (a) Scrape cytosmear showing cells with marked pleomorphism and hyperchromasia (H&E stain, ×400), (b) Photomicrograph demonstrates atypical cells involving most of the epithelial thickness (H&E stain, ×400)

Kiran et al., also reported the buccal mucosa as the most involved site of epithelial dysplasia (66.7%), followed by the tongue (33.3%), retromolar area (13.3%), labial mucosa (6.67%), and palate (6.67%).[28] Cai et al., found that the buccal mucosa (88.87%) was the most prevalent site of oral submucous fibrosis, followed by the tongue (7.57%), lip (1.93%), palate (1.34%), and gingiva (0.30%).[29]

The addictions identified in our study included pan masala use, chewing and smoking tobacco, and alcohol consumption. Chewing tobacco was found to be the most frequent habit associated with premalignant and malignant lesions. In the study by Kshirsagar et al., common forms of tobacco addiction included chewing tobacco with lime (31%), pan with betel nut and tobacco (23%), gutkha or mawa (8%), and mishri application over gum and teeth (5%). Additionally, 33% of patients had combined addictions of habitual smoking and alcohol use.[12] Panda et al.'s research also highlighted a high prevalence of tobacco use, with males being the most commonly involved.[13] The most common habit observed in the study by Jagtap et al., was tobacco chewing.[14] In a study by Babshet et al., it was found that 38.3% of individuals had a habit of tobacco chewing, 28.3% had a smoking habit, and 25% had a combination of quid, tobacco, or gutkha chewing habits along with alcohol consumption.[23] In another study by Pires et al., on 346 patients with oral SCC, information regarding tobacco use was available for 281 patients. Among them, 225 patients (80%) were either current or past tobacco users. Similarly, information on alcohol use was available for 208 patients, and 146 patients (70%) were either current or past alcohol users.[25] According to Bagate et al., oral submucous fibrosis was most prevalent among individuals using tobacco with lime, followed by those with mixed habits. The study also found that the incidence of leukoplakia and malignancy was higher among

Table 6: Site-wise distribution of oral premalignant lesions and malignant lesions

Site	Number of premalignant lesions	Percentage	Number of malignant lesions	Percentage	Total (%)
Left BM	9	25.71	13	16.45	22 (19.29)
Right BM	5	14.28	10	12.65	15 (13.15)
Floor of mouth	2	5.71	1	1.26	3 (2.63)
Oral tongue	11	31.42	21	26.58	32 (28.07)
Left RMT	0	0	2	2.53	2 (1.75)
Right RMT	1	2.85	1	1.26	2 (1.75)
Left upper alveolus	0	0	0	0	0
Left lower alveolus	0	0	1	1.26	1 (0.87)
Right upper alveolus	0	0	0	0	0
Right lower alveolus	1	2.85	2	2.53	3 (2.63)
Left upper GBS	1	2.85	0	0	1 (0.87)
Left lower GBS	5	14.28	12	15.18	17 (14.91)
Right upper GBS	0	0	0	0	0
Right lower GBS	0	0	8	10.12	8 (7.01)
Hard palate	0	0	5	6.32	5 (4.38)
Lip	0	0	3	3.79	3 (2.63)
Total	35	100	79	100	114 (100)

BM: Buccal mucosa, RMT: Retromolar trigone, GBS: Gingivobuccal sulcus

Table 7: Site distribution of malignant lesions according to histopathological features

Site	SCC (WD) (%)	SCC (MD) (%)	Microinvasive SCC (%)	MM (%)	VC (%)	MEC (%)	PLGA
BM (left and right)	18 (30)	0	1 (33.33)	1 (50)	2 (100)	1 (25)	0
Floor of mouth	0	0	1 (33.33)	0	0	0	0
Oral tongue	16 (26.66)	3 (42.85)	1 (33.33)	1 (50)	0	0	0
RMT (left and right)	3 (5)	0	0	0	0	0	0
Upper alveolus (left and right)	0	0	0	0	0	0	0
Lower alveolus (left and right)	3 (5)	0	0	0	0	0	0
Upper GBS (left and right)	0	0	0	0	0	0	0
Lower GBS (left and right)	17 (28.33)	3 (42.85)	0	0	0	0	0
Hard palate	1 (1.66)	0	0	0	0	3 (75)	1 (100)
Lips	2 (3.33)	1 (14.28)	0	0	0	0	0
Total	60 (100)	7 (100)	3 (100)	2 (100)	2 (100)	4 (100)	1 (100)

WD: Well differentiated, MD: Moderately differentiated, MM: Malignant melanoma, VC: Verrucous Carcinoma, MEC: Mucoepidermoid carcinoma, BM: Buccal mucosa, RMT: Retromolar trigone, GBS: Gingivobuccal sulcus

Table 8: Site distribution of premalignant lesions according to histopathological features

Site	Hyperkeratosis without dysplasia (%)	Mild OED (%)	Moderate OED (%)	Severe OED (%)	OSMF (%)	PVL
Buccal mucosa (left and right)	5 (35.71)	4 (44.44)	0	3 (42.85)	2 (100)	0
Floor of mouth	1 (7.14)	1 (11.11)	0	0	0	0
Oral tongue	5 (35.71)	3 (33.33)	1 (50)	2 (28.57)	0	0
RMT (left and right)	0	0	0	1	0	0
Upper alveolus (left and right)	0	0	0	0	0	0
Lower alveolus (left and right)	0	0	1 (50)	0	0	0
Upper GBS (left and right)	0	0	0	1 (14.28)	0	0
Lower GBS (left and right)	3 (21.42)	1 (11.11)	0	0	0	1 (100)
Hard palate	0	0	0	0	0	0
Lips	0	0	0	0	0	0
Total	14 (100)	9 (100)	2 (100)	7 (100)	2 (100)	1 (100)

OED: Oral epithelial dysplasia, OSMF: Oral submucous fibrosis, PVL: Proliferative verrucous leukoplakia

individuals using tobacco with lime.[30] Another study by Abdelaziz and Osman revealed that the consumption of alcohol and cigarette smoking can increase the risk of developing atypical cellular changes in the oral cavity.[31] In the study by Klongnoi et al., a higher proportion of male patients were either smokers (42.3%) or ex-smokers (16.2%), alcohol drinkers (38.7%). Conversely, a greater percentage of female patients had a past habit of chewing betel quid (56.0%) or using smokeless tobacco (18.5%).[15] In observations made by Khandekar et al., 71.3% of

Table 9: Tobacco habits in oral premalignant and malignant lesions

Habits	Premalignant lesions no of cases		Total	Percentage	Malignant lesions no of cases		Total	Percentage
	Males	Females			Males	Females		
Tobacco chewing	10	8	18	51.42	10	15	25	31.64
Smoking+tobacco chewing	2	0	2	5.71	7	1	8	10.12
Alcohol+tobacco chewing	3	2	5	14.28	8	5	15	18.98
Pan masala	2	3	5	14.28	3	8	11	13.92
Pan masala+tobacco chewing	1	0	1	2.85	6	5	11	13.92
Mixed	1	0	1	2.85	2	0	2	2.53
No addiction	2	1	3	8.57	5	4	9	11.39
Total	21	14	35	100	41	38	79	100

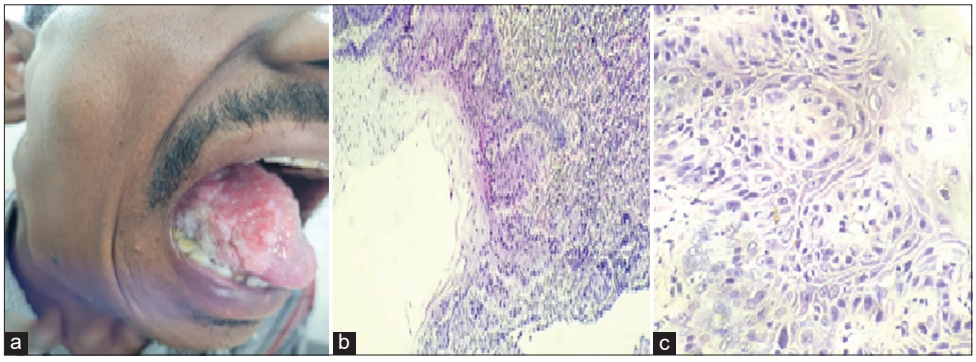

Figure 9: Moderate Epithelial dysplasia. (a) Whitish patch over lower and right lateral border of tongue, (b) Photomicrograph showing dysplastic changes extend to the midpoint of epithelium (H&E stain, ×100), (c) Showing nuclear hyperchromasia, pleomorphism and cellular crowding (H&E stain, ×400)

patients had a habit of chewing tobacco, 63.3% of male were tobacco smokers, and 22.5% of the patients used tobacco in both chewing and smoking form.[32] According to Johnson et al., oral submucous fibrosis was primarily associated with tobacco and alcohol consumption, both in the past and present, making them the most significant risk factors for the disease.[33]

Limitations of the study

Since this study was carried out in a tertiary care center and used convenient sampling, the findings cannot be generalized to the broader population. Moreover, immunohistochemistry was not done due to lack of resources.

CONCLUSION

Despite the increasing burden of oral cavity lesions in India, there are significant challenges in the diagnosis and management of these lesions. These challenges include limited access to healthcare services, lack of awareness, and cultural practices such as tobacco and betel nut chewing, which are deeply ingrained in Indian society. This study will provide a valuable resource for clinicians, researchers, and healthcare providers, highlighting the current understanding of the clinicopathological characteristics of premalignant and malignant lesions of the oral cavity. The findings from this study can potentially aid in the development of effective preventive and management strategies to reduce the burden of oral cavity lesions in the Indian population.

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REFERENCES

1. Sung H, Ferlay J, Siegel RL, Laversanne M, Soerjomataram I, Jemal A, et al. Global Cancer Statistics 2020: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. CA Cancer J Clin. 2021;71(3):209-249. https://doi.org/10.3322/caac.21660
2. Warnakulasuriya S. Global epidemiology of oral and oropharyngeal cancer. Oral Oncol. 2009;45(4-5):309-316. https://doi.org/10.1016/j.oraloncology.2008.06.002
3. Conway DI, Petticrew M, Marlborough H, Berthiller J, Hashibe M and Macpherson LM. Socioeconomic inequalities and oral cancer risk: A systematic review and meta-analysis of case-control studies. Int J Cancer. 2008;122(12):2811-2819. https://doi.org/10.1002/ijc.23430
4. Neville BW, Damm DD, Allen CM and Chi AC. Oral and Maxillofacial Pathology. 4th ed. Philadelphia, PA: Saunders/ Elsevier; 2016.
5. Laprise C, Shahul HP, Madathil SA, Thekkepurakkal AS, Castonguay G, Varghese I, et al. Periodontal diseases and risk

of oral cancer in Southern India: Results from the HeNCe Life study. Int J Cancer. 2016;139(7):1512-1519.
https://doi.org/10.1002/ijc.30201

6. Misra S, Chaturvedi A and Misra NC. Management of gingivobuccal complex cancer. Ann R Coll Surg Engl. 2008;90(7):546-553.
https://doi.org/10.1308/003588408X301136
7. Auluck A, Hislop G, Poh C, Zhang L and Rosin MP. Areca nut and betel quid chewing among South Asian immigrants to Western countries and its implications for oral cancer screening. Rural Remote Health. 2009;9(2):1118.
8. Varshitha A. Prevalence of oral cancer in India. J Pharm Sci Res. 2015;7(10):845-848.
9. Nair DR, Pruthy R, Pawar U and Chaturvedi P. Oral cancer: Premalignant conditions and screening--an update. J Cancer Res Ther. 2012;8 Suppl 1:S57-S66.
https://doi.org/10.4103/0973-1482.92217
10. Tomar A and Chouhan RP. Histopathological study of pre-malignant and malignant Lesions of oral cavity: A prospective study from SAMS medical college. IP J Diagn Pathol Oncol. 2021;6(1):6-8.
11. Rathva H, Shah A and Goswami H. Histopathological study of Pre-malignant and malignant lesions of oral cavity at tertiary care center. Int J Clin Diagn Pathol. 2020;3(3):6-9.
https://doi.org/10.33545/pathol.2020.v3.i3a.251
12. Kshirsagar GR, Kumavat PV, Chaudhari CS, Shedge RT and Kshirsagar M. Tobacco related oral cavity lesions, scrape cytology with histopathological correlation: A 5 year study at tertiary level hospital in Mumbai. Indian J Pathol Oncol. 2020;7(1):123-130.
13. Panda S, Kar A, Dash S, Behera PK, Satpathy BB, Samanataray S, et al. Differential diagnosis of oral malignant and premalignant lesions by exfoliative cytology with emphasis on demographic factors. Int J Med Res Prof. 2017;3(4):156-159.
https://doi.org/10.21276/ijmrp.2017.3.4.032
14. Jagtap SV, Warhate P, Saini N, Jagtap SS and Chougule PG. Oral premalignant lesions: A clinicopathological study. Int Surg J. 2017;4(10):3477-3481.
15. Klongnoi B, Sresumatchai V, Clypuing H, Wisutthajaree A, Pankam J, Srimaneekarn N, et al. Histopathological and risk factor analyses of oral potentially malignant disorders and oral cancer in a proactive screening in northeastern Thailand. BMC Oral Health. 2022;22(1):613.
https://doi.org/10.1186/s12903-022-02646-9
16. Bastakoti S, Shrestha G, Gautam DK, Dhungana I, Jha N, Pandey G, et al. Clinico-pathological spectrum of oral cavity lesions at a tertiary care center in central Nepal: A descriptive cross-sectional study. JNMA J Nepal Med Assoc. 2021;59(234):124-127.
https://doi.org/10.31729/jnma.5539
17. Pudasaini S and Baral R. Oral cavity lesions: A study of 21 cases. J Pathol Nepal. 2011;1(1):49-51.
https://doi.org/10.3126/jpn.v1i1.4452
18. Senguven B, Baris E, Yildirim B, Shanibat A, Yuzel OO, Museyibov F, et al. Oral mucosal lessions: A retrospective review of one institution's 13-year experience. Turk J Med Sci. 2015;45(1):241-245.
https://doi.org/10.3906/sag-1312-9
19. Kosam S and Kujur P. Pattern of oral cavity lesion: A retrospective study of 350 cases. Int J Sci Stud. 2016;4(3):65-69.
20. Brandizzi D, Gandolfo M, Velazco ML, Cabrini RL and Lanfranchi HE. Clinical features and evolution of oral cancer: A study of 274 cases in Buenos Aires, Argentina. Med Oral Patol Oral Cir Bucal. 2008;13(9):E544-E548.
21. Dias GS and Almeida AP. A histological and clinical study on oral cancer: Descriptive analyses of 365 cases. Med Oral Patol Oral Cir Bucal. 2007;12(7):E474-E478.
22. Modi D, Laishram RS, Sharma LD and Debnath K. Pattern of oral cavity lesions in a tertiary care hospital in Manipur, India. J Med Soc. 2013;27:199-202.
23. Babshet M, Nandimath K, Pervatikar S and Naikmasur V. Efficacy of oral brush cytology in the evaluation of the oral premalignant and malignant lesions. J Cytol. 2011;28(4):165-172.
https://doi.org/10.4103/0970-9371.86342
24. Rai HC and Ahmed J. Clinicopathological correlation study of oral squamous cell carcinoma in a local Indian population. Asian Pac J Cancer Prev. 2016;17(3):1251-1254.
https://doi.org/10.7314/apjcp.2016.17.3.1251
25. Pires FR, Ramos AB, Oliveira JB, Tavares AS, Luz PS and Santos TC. Oral squamous cell carcinoma: Clinicopathological features from 346 cases from a single oral pathology service during an 8-year period. J Appl Oral Sci. 2013;21(5):460-467.
https://doi.org/10.1590/1679-775720130317
26. Pires FR, Pringle GA, de Almeida OP and Chen SY. Intra-oral minor salivary gland tumors: A clinicopathological study of 546 cases. Oral Oncol. 2007;43(5):463-470.
https://doi.org/10.1016/j.oraloncology.2006.04.008
27. Munhoz Ede A, Cardoso CL, Tjioe KC, Sant'ana E, Consolaro A, Damante JH, et al. Atypical clinical manifestation of mucoepidermoid carcinoma in the palate. Gen Dent. 2009;57(6):e51-e53.
28. Kiran K, Agarwal P, Kumar S and Jain K. Micronuclei as a predictor for oral carcinogenesis. J Cytol. 2018;35(4):233-236.
https://doi.org/10.4103/JOC.JOC_141_17
29. Cai X, Yao Z, Liu G, Cui L, Li H and Huang J. Oral submucous fibrosis: A clinicopathological study of 674cases in China. J Oral Pathol Med. 2019;48:321-325.
https://doi.org/10.1111/jop.12836
30. Bagate A, Dukare S, Gawai K and Grace D. Oral cytomorphological study of chronic tobacco chewers in rural area: A prospective study. Pharma Innov J. 2015;4(6):78-82.
31. Abdelaziz MS and Osman TE. Detection of cytomorphological changes in oral mucosa among alcoholics and cigarette smokers. Oman Med J. 2011;26(5):349-352.
https://doi.org/10.5001/omj.2011.85
32. Khandekar SP, Bagdey PS and Tiwari RR. Oral cancer and some epidemiological factors: A hospital-based study. Indian J Community Med. 2006;31:157-159.
33. Johnson NW, Jayasekara P and Amarasinghe AA. Squamous cell carcinoma and precursor lesions of the oral cavity: Epidemiology and aetiology. Periodontol 2000. 2011;57(1):19-37.
https://doi.org/10.1111/j.1600-0757.2011.00401.x

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