



# Oral Cancer and Epithelial Dysplasia: Institutional Prevalence and Clinicopathological Insights into Oral Submucous Fibrosis and Leukoplakia

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## KEYWORDS

Oral Squamous Cell Carcinoma (OSCC), Oral Potentially Malignant Disorders (OPMDs), Leukoplakia, Malignant transformation risk, Survival rate.

## ABSTRACT:

**Introduction:** Oral Squamous Cell Carcinoma (OSCC) is a major cancer in India, linked to tobacco, betel quid and HPV, with a 5-year survival of ~50% due to late diagnosis. Oral epithelial dysplasia (OED), a precursor with ~10% malignant risk, and oral potentially malignant disorders (OPMDs) require early detection and clinicopathological correlation.

**Objectives:** Malignant potential of OPMD's, alongside the high burden of OSCC in India, it is imperative to understand their demographic patterns and site predilections. This study aims to evaluate these parameters in patients diagnosed with OED and OSCC at the Department of Oral and Maxillofacial Pathology in our institution.

**Methods:** A retrospective review was conducted on histopathologically confirmed OED and OSCC cases diagnosed between October 2023 and June 2025. Demographic details, site distribution, and histological grades were recorded. OED was graded using WHO (2005) and OSCC using Broder's system. Clinicopathological correlations with OSMF and Leukoplakia were also assessed.

**Results:** OED prevalence was 15.92%, mainly mild (50.67%), followed by moderate (30.67%), severe (13.33%), and carcinoma in situ (5.33%). OSCC made up 11%, with well-differentiated cases at 61.53%. Both OED and OSCC showed >80% male predominance, occurred mostly at 31–55 years, and predominantly affected the buccal mucosa. Clinicopathological correlation showed that advanced OSMF grades did not consistently correlate with higher dysplasia, whereas Verrucous and Erythro-Leukoplakia subtypes of Leukoplakia exhibited higher malignant potential.

**Conclusions:** OED and OSCC are male-predominant with buccal mucosa predilection. Clinical grading of OSMF and Leukoplakia alone is unreliable; combined clinical and histopathological evaluation is essential for accurate risk assessment.

## 1. Introduction

Oral squamous cell carcinoma (OSCC) arises from the squamous epithelium of the oral cavity and is defined by the WHO as a malignant epithelial neoplasm demonstrating keratin formation and/or intercellular bridges, characteristic of squamous differentiation.<sup>[1]</sup> OSCC remains a major global health concern, with Southeast Asia bearing the highest burden. In India, it is the most common cancer among men and the third most common among women, accounting for approximately 12% of cancers in men and 8% in women.<sup>[2]</sup> Despite therapeutic advances, the five-year survival rate for OSCC has remained stagnant at around 50% for decades, primarily due to late-stage diagnosis.<sup>[3]</sup>

The development of OSCC is often preceded by oral potentially malignant disorders (OPMDs), which represent a heterogeneous group of oral mucosal abnormalities associated with an increased risk of malignant transformation. Clinically, these lesions present with diverse features such as variations in colour (white, red, or mixed), surface texture, and anatomical distribution, which complicates diagnosis and risk assessment. Although not all OPMDs undergo malignant transformation, their clinical importance is highlighted by the fact that nearly 80% of oral cancers arise from such precursor lesions, with an overall malignant transformation rate estimated at 7–8%.<sup>[4]</sup>

Among OPMDs, oral submucous fibrosis (OSMF) deserves particular attention due to its high prevalence in South and Southeast Asia, affecting over 5 million individuals globally.<sup>[5]</sup> This chronic, progressive condition is clinically marked by epithelial atrophy, submucosal fibrosis, and symptoms such as trismus, dysphagia, and a burning sensation of the oral mucosa. The principal etiological factor is areca nut chewing, with its alkaloids and flavonoids implicated in both fibrosis and carcinogenesis; consequently, the nut has been classified as a Group I carcinogen.<sup>[6]</sup> Malignant transformation rates for OSMF have been reported in the range of 2.8–7.6%, with meta-analyses estimating an overall risk of about 4%.<sup>[7]</sup> This underlines the need for early diagnosis, effective counselling on risk habit cessation, and sustained long-term follow-up in affected individuals.

## 2. Objectives

Given the malignant potential of OPMD's, alongside the high burden of OSCC in India, it is imperative to understand their demographic patterns and site predilections. The present study therefore aims to evaluate these parameters in patients diagnosed with OED and OSCC at the Department of Oral and Maxillofacial Pathology in our institution.



### 3. Methods

Among 421 patients, 52 cases & 75 cases of histopathologically diagnosed OSCC & OPMDs between October 2023 to June 2025 were reviewed from the archives of the Department of Oral and Maxillofacial Pathology of our institution. The following variables were recorded: Gender, Age, Grades and most common location of OPMD and OSCC. The data were analysed, and graphs were formulated, respectively. Grading of dysplasia was done according to the WHO 2019 classification [8], and grading of OSCC was done according to Broder et al [9].

The grading was re-evaluated and the case of discrepancies was resolved by common consensus.

#### Inclusion and Exclusion criteria

Patients with histopathologically confirmed cases of OPMDs and OSCCs, which were reported in our institution from the period of October 2023 to June 2025, were included in the study. Patients who had a history of previous malignancy and recurrent cases of dysplasia and OSCCs were excluded from the study.

The overall prevalence in different grades of dysplasia and different grades of OSCCs was tabulated and recorded as mean percentage. Statistical evaluation was done for scale and ordinal variance using IBM Corp. (2017). IBM SPSS Statistics for Windows, Version 25.0. Armonk, NY: IBM Corp. Age distribution, gender distribution and site predilection of different grades of dysplasia and OSCCs were statistically analyzed using the Chi-square test ( $P < 0.05$ )

### 4. Results

Among 421 patients visiting the department between October 2023 to June 2025, 75 patients presented with OPMD's and 52 patients presented with OSCC. A total of 127 clinically diagnosed cases of OPMDs and OSCCs were evaluated for histopathological correlation (Table I). Among them, traumatic fibroma (n=6) and traumatic ulcer (n=9) occasionally demonstrated mild to moderate dysplasia, while a few traumatic and non-healing ulcers (n=8) revealed progression to invasive carcinoma. OSMF constituted the largest group

(n=38), with the majority exhibiting varying grades of epithelial dysplasia and a small subset progressing to well differentiated squamous cell carcinoma (WDSCC). Leukoplakia (n=8) frequently demonstrated dysplastic changes, whereas Erythroplakia (n=5) and Verrucous Leukoplakia (n=4) showed a stronger association with severe dysplasia and carcinoma in situ. Of the clinically suspected malignant cases (n=20), histopathology confirmed a spectrum ranging from epithelial dysplasia to poorly differentiated squamous cell carcinoma (PDSCC). Among cases provisionally diagnosed as squamous cell carcinoma (n=26), the majority were confirmed as WDSCC (n=16), followed by moderately differentiated squamous cell carcinoma (MDSCC) (n=7) and PDSCC types (n=3). Other uncommon clinical presentations, including squamous papilloma, granulation tissue, and chronic inflammatory lesions, were confirmed as benign with dysplastic changes.

Clinical/ Provisional Diagnosis	Total cases	OPMD				OSCC			
		Mild ED	Moderate ED	Severe ED	CA in situ	Early invasive SCC	WD SCC	MD SCC	PD SCC
Traumatic Fibroma	6	-	2	2	-	-	2	-	-
Traumatic Ulcer	9	1	2	2		2	2		
Non-Healing Ulcer	8	1	4				2	1	
OSMF	38	26	7	2	1		2		
Leukoplakia (Speckled & Homogenous)	8	5	3						
Erythroplakia	5	1	2		1	1			
Verrucous Leukoplakia/ Hyperplasia	4	2		2					
Squamous cell carcinoma	26		1	2			16	7	24
Suspected Malignancy	20		1	1	2	3	8	2	3
Squamous Papilloma	1	1							
Granulation Tissue	1	1							
Chronic inflammatory lesion									

**Table I: Correlation of clinically & histological diagnosis of OMPD's & OSCC**

Overall, the findings underscore a lack of consistent correlation between clinical grading/subtyping and histopathological dysplasia in OPMDs, highlighting the indispensability of biopsy and microscopic evaluation for accurate diagnosis and risk stratification.

In the present study, the overall prevalence of oral epithelial dysplasia (OED) was 15.89%. Mild dysplasia was the most frequent finding with a mean prevalence of 50.67%, followed by moderate dysplasia (30.67%), severe dysplasia (13.33%), and carcinoma in situ (5.33%). Overall prevalence of OSCC was 11.04%, with WDSCC being the most common subtype (61.53%), MDSCC (21.15%), early invasive SCC (11.53%) and PDSCC (5.76%) (Table II).

S.N.	Diagnosis	Prevalence N (%)	Mean %
1.	Mild Epithelial Dysplasia	38 (8.06)	50.67
2.	Moderate Epithelial Dysplasia	23 (4.88)	30.67
3.	Severe Epithelial Dysplasia	10 (2.1)	13.33
4.	Carcinoma In-situ	04 (0.85)	5.33
5.	Early Invasive SCC	06 (1.27)	11.53
6.	Well differentiated SCC	32 (6.8)	61.53
7.	Moderately differentiated SCC	11 (2.33)	21.15
8.	Poorly differentiated SCC	03 (0.64)	5.76

**Table II: Overall Prevalence of OED and OSCC**

The age-wise distribution of OPMD and OSCC is shown in (Table III). All OPMD's & OSCC cases were mostly seen in the age group of 31-55 years, followed by <30 years and the least number of cases were seen in >56 years of age.

Gender-wise distribution revealed a marked male predominance for both OPMD's and OSCC (Table IV). In OPMD's, mild dysplasia was observed in 37.33% of males and 13.33% of females, while moderate dysplasia occurred in 28% of males and 2.7% of females. Severe dysplasia was found in 10.7% of males and 2.7% of females, carcinoma in situ was restricted to 5.33% of males. Overall, 81.3% were males and 18.7% were females, with no significant difference on chi-square testing ( $\chi^2=2.8089$ ,  $p=0.422041$ ).

S.N.	Diagnosis	Age group N (%)		
		<30 years	31-55 years	>56 years
1.	Mild Epithelial Dysplasia	12 (16)	20 (28)	05 (6.7)
2.	Moderate Epithelial Dysplasia	08 (10.7)	14 (18.7)	02 (2.7)
3.	Severe Epithelial Dysplasia	01 (1.33)	08 (10.7)	01 (1.33)
4.	Carcinoma In-situ	00 (00)	02 (2.7)	02 (2.7)
5.	Early Invasive SCC	00 (00)	03 (5.77)	03 (5.77)
6.	Well differentiated SCC	02 (3.84)	25 (48.07)	05 (9.61)
7.	Moderately differentiated SCC	00 (00)	07 (13.5)	04 (7.69)
8.	Poorly differentiated SCC	00 (00)	02 (3.84)	01 (1.92)

**Table III: Distribution of OED & OSCC according to age among diagnosed cases**

A similar pattern was noted in OSCC, where early invasive SCC was recorded in 7.69% of males and 3.8% of females. WDSCC was the most common subtype, observed in 53.8% of males and 7.69% of females. MDSCC affected 15.4% of males and 5.7% of females, while PDSCC occurred in 5.7% of males with no female cases. In total, 82.7% cases were male and 17.3% cases were female, with chi-square analysis showing no significant difference ( $\chi^2=2.2737$ ,  $p=0.517579$ ).

The anatomical distribution of OPMD's and OSCC is summarized in (Table V). Buccal mucosa was the most



common site for all grades of OED, including 48% of mild dysplasia, 22.7% of moderate, 10.7% of severe and 4% of carcinoma in situ. The buccal vestibule recorded 2.7% of moderate dysplasia, with no other grades observed. Labial mucosa showed 2.7% each of mild and moderate dysplasia, and 1.33% each of severe dysplasia and carcinoma in situ. Retromolar trigone had 2.7% of moderate dysplasia and 1.33% of severe dysplasia, while the floor of the mouth had no reported cases. Overall, 50.7% were mild, 30.7% moderate, 13.33% severe, and 5.33% carcinoma in situ, with no significant association between OED grade and site ( $\chi^2=10.482$ ,  $p=0.313$ ). For OSCC, buccal mucosa was also the most frequently affected site, with 5.77% cases of early invasive SCC, 42.3% of WDSCC, 11.5% of MDSCC, and 1.92% of PDSCC. The buccal vestibule had 3.84% cases of early invasive SCC, 13.5% of WDSCC, 7.69% of MDSCC, and 1.92% of PDSCC. Retromolar trigone presented 1.92% of early invasive SCC, 3.84% of WDSCC and 1.92% of PDSCC. The floor of the mouth had 1.92% case of MDSCC, and labial mucosa had 1.92% case of WDSCC. Overall, 11.5% were early invasive, 61.5% were well-differentiated, 21.15% were moderately differentiated, and 5.77% were poorly differentiated, with no significant correlation between SCC grade and site ( $\chi^2=10.157$ ,  $p=0.602$ ).

S.N.	Diagnosis	Age group N (%)	
		Male	Female
1.	Mild Epithelial Dysplasia	28 (37.33)	10 (13.33)
2.	Moderate Epithelial Dysplasia	21 (28)	02 (2.7)
3.	Severe Epithelial Dysplasia	08 (10.7)	02 (2.7)
4.	Carcinoma In-situ	04 (5.33)	00 (00)
	Total	61 (81.3)	14 (18.7)
5.	Early Invasive SCC	04 (7.69)	02 (3.8)
6.	Well differentiated SCC	28 (53.8)	04 (7.69)
7.	Moderately differentiated SCC	08 (15.4)	03 (5.7)
8.	Poorly differentiated SCC	03 (5.7)	00 (00)
	Total	43(82.7)	09 (17.3)

**Table IV: Comparison of grades of Oral Epithelial Dysplasia according to Gender among diagnosed cases of Oral Epithelial Dysplasia & Oral Squamous Cell Carcinoma**

Location	Mild ED N (%)	Moderate ED N (%)	Severe ED N (%)	CA In-situ N (%)	Early Invasive SCC N (%)	WD SCC N (%)	MD SCC N (%)	PD SCC N (%)
Buccal Vestibule	00 (00)	02 (2.7)	00 (00)	00 (00)	02 (3.84)	07 (13.5)	04 (7.69)	01 (1.92)
Buccal Mucosa	36 (48)	17 (22.7)	08 (10.7)	03 (4.00)	03 (5.77)	22 (42.3)	06 (11.5)	01 (1.92)
Floor of the Mouth	00 (00)	00 (00)	00 (00)	00 (00)	00 (00)	00 (00)	01 (1.92)	00 (00)
Labial Mucosa	02 (2.7)	02 (2.7)	01 (1.33)	01 (1.33)	00 (00)	01 (1.92)	00 (00)	00 (00)
Retromolar Trigone	00 (00)	02 (2.7)	01 (1.33)	00 (00)	01 (1.92)	02 (3.84)	00 (00)	01 (1.92)
Total	38 (50.7)	23 (30.7)	10 (13.33)	04 (5.33)	06 (11.5)	32 (61.5)	11 (21.15)	03 (5.77)

**Table V: Presentation of grades of OED & OSCC according to location among total diagnosed cases**

## 5. Discussion

The present study evaluated the prevalence, demographic distribution, site predilection, and clinicopathological

correlations of OPMD's and OSCC in an institutional setting, reinforcing established epidemiological trends while highlighting variations between clinical grading and histopathology in OPMD's. The overall prevalence of OED



(15.89%) and OSCC (11.04%) aligns with Gupta B et al. [10], reflecting the persistently high burden of oral cancer in India. Mild dysplasia predominated among OED cases (50.7%), consistent with Warnakulasuriya S et al. [11], while WDSCC (61.5%) represented the typical histological spectrum.

A marked male predominance (>80%) and peak incidence in the 31–55-year age group were observed for both OED and OSCC, consistent with Gupta B et al. [12], reflecting higher exposure to tobacco, smoking, and betel quid. Rising cases in individuals <30 years may indicate earlier initiation of deleterious habits, sociocultural acceptance, or genetic susceptibility, underscoring the need for targeted awareness programs. Buccal mucosa was the most commonly affected site, followed by the alveolo-buccal complex, mirroring habit-related exposure patterns in India (Gupta PC et al., [13]), whereas high-risk sites in Western cohorts, such as tongue and floor of the mouth, were less frequent. [14]

In Leukoplakia, high-risk subtypes, including verrucous and Erythro-Leukoplakia, exhibited moderate to severe dysplasia, while homogenous lesions also demonstrated moderate dysplasia in some cases, reflecting biological heterogeneity and limitations of clinical assessment. [15]

No statistically significant associations were found between age, gender, and histopathological grades in OED or OSCC, likely due to sample size and multifactorial influences. Overall, these findings emphasize that clinical evaluation alone is insufficient for risk assessment. Integrated clinical and histopathological assessment, regular surveillance, multiple-site biopsies when indicated, and adjunctive diagnostic tools are essential for early detection and accurate risk stratification. Public health interventions targeting tobacco cessation, lifestyle modification, and early screening remain critical to reducing the burden of OSCC in high-prevalence regions. This study demonstrates a significant prevalence of OPMD's and OSCC, with strong male predominance and buccal mucosa predilection. Clinical staging of OPMD's did not consistently correlate with histopathological dysplasia, emphasizing the need for mandatory biopsy and combined clinicopathological evaluation for accurate risk assessment. The predominance of mild dysplasia and WDSCC suggests potential for earlier detection, while the rising occurrence in younger age groups highlights the importance of preventive strategies targeting high-risk habits.

Because the study is retrospective, involves a limited number of cases, and is conducted at a single center, its findings may be less generalizable. Additionally, relying only on biopsy samples may fail to reflect the true heterogeneity of the lesions. Future multicentric, prospective studies with larger cohorts, habit documentation, and molecular profiling are needed to improve risk prediction. Integrating clinicopathological assessment with biomarker-based approaches will enhance early detection, guide personalized management, and ultimately reduce the burden of oral cancer in high-prevalence regions.

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