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IP International Journal of Medical Microbiology and Tropical Diseases

Journal homepage: <https://www.ijmmttd.org/>

Original Research Article

Oral lichen planus and lichenoid reactions in bronchial Asthma: Prevalence and clinicopathologic correlation

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Abstract

Background: Oral lichen planus (OLP) and oral lichenoid reactions (OLR) are related to systemic diseases and drug exposure. Long-term use of inhaled corticosteroids and β_2 -agonists in bronchial asthma (BA) may lead to the development of such oral lesions.

Aim: To determine the prevalence and clinical correlation of asymptomatic OLP and OLR in individuals with BA.

Materials and Methods: This case-control study included 280 participants. All were adults aged >18 years; those with systemic disorders, pregnancy, lactation, or tobacco habits were excluded. Asthma severity was graded using pulmonary function. Data were analyzed using SPSS 23.0 with t-test, ANOVA, chi-square, and correlation, considering $p < 0.05$ significant.

Results: Asthma patients showed significantly higher prevalence of oral lesions: 26.43% (37/140) had OLP (20%) or OLR (6.43%) versus 3.57% (5/140) in controls ($p < 0.0001$). Buccal mucosa was the most affected site. Lesion frequency correlated with asthma severity ($p = 0.0019$), increasing from mild to severe disease.

Conclusion: Asthma patients exhibited a significantly higher prevalence of OLP and OLR than healthy individuals, independent of disease severity. Regular oral examinations are recommended in asthma management to enable early detection and care of corticosteroid-related or immune-mediated mucosal alterations.

Keywords: Corticosteroid, Drug-induced oral lesions, Lichenoid drug reactions, Mucosal immunopathology, T-Cell mediated immunity.

Received: 14-08-2025; **Accepted:** 15-10-2025; **Available Online:** 19-11-2025

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1. Introduction

Chronic inflammatory diseases of the oral mucosa show overlapping clinical and histopathological patterns. Oral lichen planus (OLP) and oral lichenoid reactions (OLRs) are frequently observed in dental and medical practice.¹⁻⁴ OLP is an idiopathic immune-mediated disorder with a possible premalignant tendency, whereas OLRs occur secondary to known factors such as medications, dental restorative materials, or systemic illness.^{5,6} Clinically, both may appear as white striations, plaques, erosions, or ulcerations on the buccal mucosa, tongue, or gingiva.^{7,8} Histopathological

features include a band-like lymphocytic infiltrate at the epithelial-connective tissue junction and basal cell degeneration.⁹⁻¹¹ Distinguishing OLP from OLRs remains challenging because of their similar clinical and histologic appearance, though this distinction is important for prognosis and management.^{3,8,11}

Systemic disorders characterised by immune dysregulation and chronic inflammation, such as bronchial asthma (BA), may influence the occurrence and course of OLP and OLRs.^{5,7,8} Asthma involves persistent airway inflammation with Th2-mediated cytokines including IL-4, IL-5, and IL-13, which may contribute to mucosal

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inflammatory changes similar to those seen in OLP.^{12,13} Some treatment modules of asthma, including inhaled corticosteroids, long-acting bronchodilators, and biologic agents such as omalizumab, have been reported to trigger or worsen lichenoid tissue reactions.^{14,15} Several studies have described associations between asthma and oral conditions like mucosal lesions, periodontal disease, and dental caries.^{13,16-19} Previous studies have reported that the chances of increase in the prevalence of oral disease in asthma patients,¹⁷ are high with changes in salivary flow, microbial composition, and mucosal integrity owing to the medication used for BA.²⁰

Asymptomatic OLP and OLR in bronchial asthma show overlapping immune pathways involving Th1, Th2, and cytokines such as IFN- γ and IL-21, along with genetic influences like ORMDL3 on chromosome 17q21.^{21,22} Detailed evaluation can help in early recognition and management of mucosal lesions. It is difficult to differentiate OLP from OLR in asthma patients. Chronic inflammation, long-term medication use, and contact of inhaled drugs with the oral mucosal surface may lead to the formation of oral lichenoid lesions (OLL).^{14,15} Previous studies have described the systemic involvement of OLP in general populations,⁷ but asthma-specific data are limited. This study aimed to determine the prevalence and clinicopathologic correlation of OLP and OLR in bronchial asthma (BA).

2. Materials and Methods

This cross-sectional case-control study was done at a tertiary care centre with respiratory, dermatology, and oral health departments after approval from the Institutional Review Board (SRMDC/IRB/2022/PhD/No.159). Written consent was obtained from all participants. The study enrolled 140 adults (≥ 18 years) with clinically and spirometrically confirmed bronchial asthma and 140 age- and sex-matched healthy controls. Exclusion criteria removed participants with systemic illness, pregnancy, lactation, or tobacco habits to reduce confounding. Each case was assessed for demographic profile, asthma history, disease duration, medication pattern, and severity using a structured form. Pulmonary function tests were applied to grade asthma as mild, moderate, or severe. Oral assessment under clinical light identified OLP and OLR, with lesion site, type, and appearance recorded. Examinations were performed and verified by an oral medicine specialist. Statistical analysis in SPSS version 23.0 used descriptive data summaries, t-tests, chi-square, and one-way ANOVA for comparison.

3. Results

280 individuals comprising of 140 bronchial asthma patients (Group A) and 140 healthy controls (Group B) were included in this study. Both groups showed similar age (55.61 ± 8.79 vs. 56.96 ± 9.53 years; $p = 0.46$) and sex distribution ($p = 0.118$). The asthma group included slightly more females

(85) than males (55) (**Table 1**). Based on pulmonary function tests, asthma severity was classified as mild in 38 cases, moderate in 57, and severe in 45. Oral mucosal lesions occurred more often in asthma patients, with 37 cases (26.43%) presenting OLP (28 cases, 20.0%) or OLR (9 cases, 6.43%), while 5 controls (3.57%) showed only OLP ($p < 0.0001$) (**Table 2; Figure 1**). Lesion distribution is presented in **Table 3** and **Figure 2**. The buccal mucosa was most affected (24 of 37 lesions: 19 OLP, 5 OLR), followed by the tongue (7 lesions: 6 OLP, 1 OLR) and gingiva (3 OLP). Palatal lesions occurred only in OLR ($n = 3$), and none were noted on the labial mucosa. Site differences were significant ($p = 0.045$).

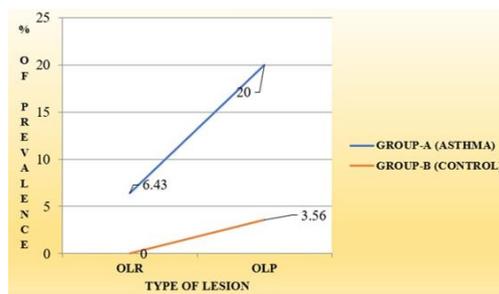


Figure 1: Comparison of prevalence of OLP/OLR in groups A & B

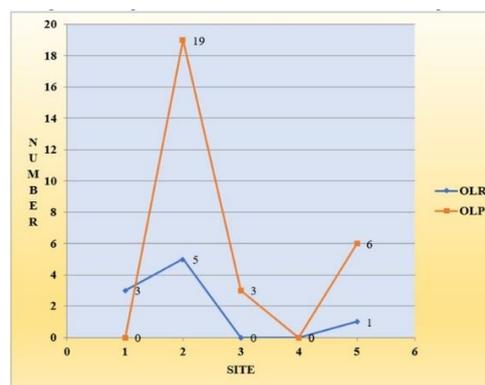


Figure 2: Clinical pattern and site distribution of OLP and OLR in Bronchial Asthma patients

Table 4 shows a clear relation between asthma severity and lesion pattern ($p = 0.0019$). Mild asthma was mainly associated with OLP (15 cases vs. 1 OLR), while severe asthma had equal OLP and OLR (6 each), indicating that OLR rises with disease severity and prolonged therapy. **Table 5** compares demographic features of OLP and OLR. OLP patients were older (55.33 vs. 48 years; $p = 0.031$) and mostly female ($25/28$ vs. $5/9$; $p = 0.045$). OLR occurred more often in severe asthma ($6/9$), whereas OLP was frequent in mild cases ($15/28$) ($p = 0.026$). **Table 6** outlines drug associations. Inhaled corticosteroids correlated with OLR (19.35% ; $p = 0.02$). OLP showed stronger relation with leukotriene modifiers (31.03%), long-acting β_2 -agonists (21.88%), and combination therapy (28%) ($p = 0.01-0.04$). Oral corticosteroids showed no significant relation ($p = 0.19$).

Table 1: Demographics and clinical profile of study population

Variable	Group A (Asthma Patients)	Group B (Control)	P-Value
Number of participants	140	140	-
Age (mean ± SD)	55.61 ± 8.79	56.96 ± 9.53	0.46
Gender (M/F)	Male – 55 Female – 85	Male – 68 Female – 72	0.118
Severity of asthma (years)	Mild – 38 Moderate – 57 Severe – 45	Non asthma	0.00*
Medication (Steroid use, etc.)	Beta agonists – 32 Inhaled steroids – 31 Oral corticosteroids – 23 Leukotriene Modifiers – 29 Combination Therapy - 25	No medication	

Table 2: Prevalence of OLP and OLR in study and control groups (unpaired t-test)

Oral Lesion Type	Group A (Asthma) (n=140)	Group B (Control) (n=140)	Percentage (Asthma)	Percentage (Control)	p-value
Asymptomatic OLR	9	0	6.43%	0.00%	-
OLP (symptomatic or asymptomatic)	28	5	20.0%	3.56%	0.0001*
Total Oral Lesions	37	5	26.43%	3.57 %	0.0001*

Table 3: Clinical pattern and site distribution of OLP/OLR in asthma patients

Site Distribution	Frequency	OLR	OLP	P value
Palate	3	3	0	*0.045
Buccal mucosa	24	5	19	
Gingiva	3	0	3	
Labial mucosa	0	0	0	
Tongue	7	1	6	
None	103	---	--	
Total	140	9	28	

Table 4: Association between asthma severity and presence of OLP/OLR

	Type of Oral Lesion			Total
	OLR	OLP	None	
Mild	1	15	22	38
Moderate	2	7	48	57
Severe	6	6	33	45
Total	9	28	103	140
P value	0.001937 (sig)			

Table 5: Demographic profile of OLP and OLR in asthma patients

Variable	OLR (N = 9)	OLP (N = 28)	P-Value
Age (mean ± SD)	48 ± 2.65	55.33 ± 9.58	0.031*
Gender (M/F)	Male – 4 Female – 5	Male – 3 Female – 25	0.045*
Asthma Severity (mild/moderate/severe)	Mild =1 Moderate =2 Severe =6	Mild =15 Moderate =7 Severe =6	0.026*

Table 6: Association of asthma medications with oral lesions (One-way ANOVA)

Medication Type	Number of Patients on Medication	Oral Lichenoid Reaction (%)	Oral Lichen Planus (%)	P-Value
Inhaled Corticosteroids	31	6 (19.35)	0	0.02*
Long-acting β ₂ -agonists	32	0	7 (21.88)	0.01*
Oral Corticosteroids	23	1 (4.35)	5 (21.74)	0.19
Leukotriene Modifiers	29	1 (3.45)	9 (31.03)	0.01*
Combination therapy	25	1 (4)	7 (28)	0.04*

4. Discussion

The study showed that OLP and OLR were more frequent in patients with BA than in healthy individuals, with a clear relation to disease severity. Among those with BA, OLP was present in 20% and OLR in 6.43%, while controls showed 3.56% and none, respectively ($p < 0.0001$), indicating BA as a strong risk factor for lichenoid changes. The link is biologically plausible since BA involves Th2 cytokines (IL-4, IL-5, IL-13) and OLP is driven by Th1 cytokines (IFN- γ , IL-21).^{12,13,21} In chronic or severe BA, overlap of Th1 and Th2 responses may promote lesion formation. Medication exposure also contributes to the occurrence of OLR in asthma patients. Inhaled corticosteroids and long-acting β_2 -agonists, commonly used in BA management, can irritate the mucosa and induce lichenoid changes through local drug deposition.^{14,15} In this study, 19.35% of patients using inhaled corticosteroids developed OLR ($p = 0.02$). Teoh et al. reported similar results, showing that several respiratory drugs may trigger lichenoid reactions through cytotoxic or hypersensitivity responses.¹⁵ Leukotriene modifiers and combination therapy were also associated with increased OLP occurrence ($p = 0.01$ and $p = 0.04$). Lesion site patterns support these mechanisms. Most lesions were found on the buccal mucosa (24 of 37 cases), consistent with typical OLP distribution^{1,3,8} and the known settling of inhaled drug particles in the buccal vestibule.^{15,20} The lack of lip involvement and few palatal lesions point to a local drug-deposition effect, while the bilateral buccal and tongue lesions seen in OLP indicate systemic immune activity. A clear dose-response trend was noted: severe BA showed more OLR cases (6 of 9), whereas mild BA had more OLP (15 of 28). This implies that OLP stems largely from immune imbalance within BA, and OLR from extended or high-dose medication exposure^{14,15} which was consistent with a study done by Lemmetyinen et al.¹⁷

Females showed more OLP cases (25 of 28), suggesting hormonal influence on immune function.^{1,6,8} OLR occurred in both sexes almost equally (4 males, 5 females), pointing to medication-related causes rather than hormonal effects.¹⁵ Asthma patients often show oral changes linked to long-standing inflammation and medication use.^{13,16,19} Inhaled corticosteroids can lead to oral dryness, thinning of mucosa, and secondary irritation, which may trigger lichenoid changes [20]. Immune dysregulation and fragile mucosal barriers in asthma²³ may contribute to OLP even when the disease is mild. Viral factors, including hepatitis C, can trigger similar lesions through cross-reactive immune responses.²⁴ Although hepatitis C was not examined here, ongoing inflammation in BA may raise this risk. Genetic factors also play a part. The ORMDL3 gene on chromosome 17q21, a known BA susceptibility site, influences sphingolipid balance and stress responses in the endoplasmic reticulum, affecting airway and oral immunity.^{22,25} ORMDL3 and GSDMB also modify T-cell regulation and epithelial

integrity, linking asthma with mucosal immune reactions.²⁵ A higher number of lesions in females and in severe asthma cases supports a genetic connection between BA and mucosal disease. Similar findings have been reported in other studies on asthma and allergic conditions.^{17,26} The oral-lung axis explains how microbial and immune processes link the mouth and airways, allowing inflammation to persist in both.²⁷ Inhaled medication, low saliva levels, and microbial imbalance in asthma can irritate the oral lining, while oral inflammation may worsen breathing symptoms.^{20,27} The absence of OLR in controls and its clear link to medication underline the need for careful clinical and histologic distinction between OLP and OLR.^{3,15} BA increases the occurrence of OLP and OLR through overlapping immune, genetic, and medication-related factors that act both locally and systemically. Routine oral examination is recommended, especially for patients on long-term inhaled therapy or with severe asthma, with collaborative care among pulmonologists, allergists, and oral physicians. This matched case-control study evaluated asthma severity, medication history, and lesion patterns. Study drawbacks included incomplete histologic verification, absence of salivary or genetic analysis, possible recall bias, and limited sample size. More multicenter studies are required to better define underlying mechanisms and confirm this association.

5. Conclusion

A clear association between bronchial asthma and oral lichenoid lesions was established in this study. OLP and OLR occurred more often in asthma patients than in healthy individuals. Most lesions were mild and located on the buccal mucosa. Females were affected more often, and lesion frequency rose with asthma severity. Use of leukotriene modifiers and β_2 -agonists was linked with OLP, while inhaled corticosteroids showed minimal relation to OLR. Regular oral checks can help detect these lesions early and guide proper treatment in patients on long-term asthma medication.

6. Source of Funding

None.

7. Conflict of Interest

None.

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Cite this article: Deshpande A, Rajkumar K, Goswami P, Byatnal A. Oral lichen planus and lichenoid reactions in bronchial Asthma: Prevalence and clinicopathologic correlation. *IP Int J Med Microbiol Trop Dis*. 2025;11(4):430-434.