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Oral and Genital Lichen Planus: A Comprehensive Review of Co-Manifestation

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Abstract

Lichen planus (LP) is a chronic immune-mediated inflammatory disease affecting skin, mucosa, nails, and hair. **Oral lichen planus (OLP)** affects 1–3% of adults aged 30–60 years and exhibits a female predominance. **Genital lichen planus (GLP)** often co-occurs with OLP but is underdiagnosed due to asymptomatic or subtle presentation. Studies indicate that 19–57% of OLP patients—with up to 92% asymptomatic genital involvement—have GLP. Erosive variants pose a 1–4% risk of malignant transformation. The disease is driven by $CD8^+$ T-cell–mediated basal keratinocyte apoptosis, involving genetic predisposition (e.g., HLA-DQB1*0201), infections (HCV, HPV), stress, and medications. Diagnosis requires thorough mucosal examination and histopathological confirmation. First-line management includes topical corticosteroids; systemic immunomodulators and emerging therapies such as JAK inhibitors are increasingly used. Multidisciplinary care with biannual-to-annual monitoring is essential given functional morbidity and cancer risk.

Keywords: Lichen planus; oral lichen planus; genital lichen planus; plurimucosal; vulvovaginal-gingival syndrome; HLA-DQB1*0201; malignant potential; topical corticosteroids; JAK inhibitors.

Introduction

Lichen planus (LP) is a chronic, T-cell–mediated autoimmune disease that presents with characteristic mucocutaneous lesions, most commonly affecting

middle-aged adults and particularly women[1,2]. Oral LP (OLP)—prevalence ~1–3% in adults—manifests in several clinical subtypes, and erosive forms carry a 1–4% risk of progression to squamous cell carcinoma[2,3]. Genital LP (GLP), though less visible and often asymptomatic, commonly co-exists with OLP, especially in women. Epidemiological studies report a co-manifestation rate up to 57%, with many cases asymptomatic[4,5]. The shared pathophysiology, potential for progressive scarring, and uncertain malignant risk underscore the importance of early recognition and interdisciplinary management. This review consolidates current evidence on co-manifestation, clinical presentation, pathogenesis, diagnostic challenges, and therapeutic strategies for OLP and GLP.

Discussion

Epidemiology and Prevalence

Up to 2% of the global population may be affected by LP; OLP predominates in women aged 50–60[2,6]. In a Spanish LP cohort of 274 patients, 53.6% had OLP and 13.5% had lesions at multiple mucosal sites[7]. Among 42 women with OLP, 57% had histologically confirmed GLP—92% were asymptomatic[5]. Gender-specific data show genital involvement in 19% of women versus 4.6% of men with OLP[4]. Approximately 60% of generalized LP cases involve mucosa, including genital sites[6,8].

Clinical Manifestations

Oral LP presents in reticular, papular, plaque-like, atrophic, erosive, and bullous forms[2,3]. Erosive variants cause pain, bleeding, dysgeusia, and may show desquamative gingivitis[9].

Genital LP in women manifests as white plaques, erosions, scarring, stenosis, and dyspareunia, while men may develop asymptomatic annular lesions on the glans penis[5,10]. **Vulvovaginal-gingival (VVG) syndrome** is a severe presentation involving multiple mucosal sites and significant morbidity[11,12].

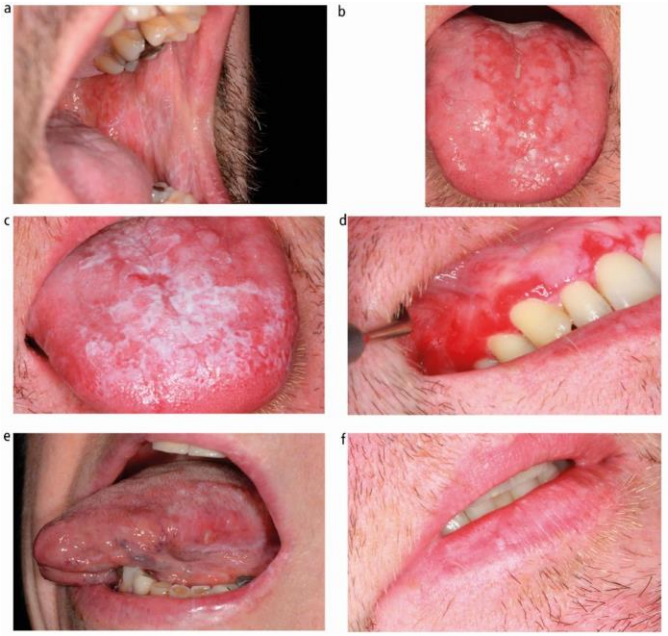

Fig. 1: Clinical manifestations of oral mucosal lesions.
(a) Reticular white striations on the buccal mucosa.
(b) Erythematous and atrophic patches on the dorsal surface of the tongue.
(c) Extensive white plaques with erythematous areas on the tongue.
(d) Ulcerative and erythematous lesion on the gingival mucosa.
(e) Erythematous and atrophic changes on the lateral border of the tongue.
(f) Erythematous lesion on the lower lip mucosa.
These images illustrate the characteristic clinical features of oral mucosal disorders such as oral lichen planus, highlighting the varied presentation from reticular, erosive to erythematous forms.

Pathophysiology

LP results from $CD8^+$ T-cell–mediated basal keratinocyte destruction, potentially triggered by HLA types (notably HLA-DQB1*0201 present in 80% of VVG-LP cases), viral infections (HCV/HPV), stress, or

medications[1,13,14]. Autoantibodies against epithelial antigens, including stratified epithelium-specific antinuclear antibodies, are present in extensive disease and associated hair/scalp involvement (lichen planopilaris) in roughly 75% of cases[15].

Diagnosis

Diagnosis entails thorough oral and genital examination (colposcopy/vulvoscopy for women, penile inspection for men)[4,10], with biopsy essential to identify:

- Interface dermatitis
- Band-like lymphocytic infiltrate
- Basal cell liquefaction
- Saw-tooth rete pattern
- Presence of Civatte bodies

  This aids in differentiating LP from lichenoid drug eruption, lichen sclerosus, pemphigoid, or early malignancy[9,16,17].

Treatment

The cornerstone of therapy is **topical corticosteroids** (e.g., clobetasol, triamcinolone)[9,13]. Refractory cases may require **topical calcineurin inhibitors** (tacrolimus), or **systemic therapies** like hydroxychloroquine, methotrexate, systemic steroids, and JAK inhibitors [3,14,18,19]. **Photodynamic therapy** shows promise in resistant VVG-LP cases[12], and supportive measures—such as oral/genital hygiene, tobacco/alcohol avoidance, and dietary changes—play critical roles. Patient-reported benefits from JAK inhibitors, hydroxychloroquine, and dietary interventions are noted, though evidence remains anecdotal[20].

Surveillance & Prognosis

A follow-up interval of 6–12 months with repeat biopsies for suspicious or evolving lesions is recommended[2,9,13]. Erosive OLP carries a 1–4% risk of malignant transformation[3,6], while VVG syndrome complications include strictures, stenosis (vaginal, esophageal, auditory), and functional deficits; early identification and intervention are crucial[11,14].

Conclusion

OLP and GLP frequently co-occur—especially among women—and often without overt genital symptoms. The VVG variant represents the most severe erosive phenotype, with multi-site involvement and a risk for scarring and stenosis. Immunological underpinning includes shared T-cell mechanisms, HLA predisposition, and possible viral triggers. Diagnosis requires comprehensive mucosal assessment and histological confirmation. Management involves topical corticosteroids, systemic immunomodulation (when needed), and emerging therapies such as JAK inhibitors or photodynamic therapy. Considering cancer risk and structural consequences, multidisciplinary teams, regular monitoring, and patient education are essential for optimal outcomes.

Future Directions

1. Large-scale, multicenter prospective studies to better define GLP prevalence, disease trajectory, and cancer risk.
2. Establish standardized genital screening protocols for OLP patients at diagnosis.
3. Conduct immunogenetic and autoantibody profiling (HLA, ANA) to stratify high-risk individuals.
4. Investigate efficacy of novel treatments—photodynamic therapy, JAK inhibitors, biologics—in multicenter randomized trials.
5. Assess patient quality-of-life, psychological effects, sexual health, and long-term functional outcomes.

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