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CASE STUDY ON LICHEN PLANUS: CLINICAL PRESENTATION, DIAGNOSIS, AND MANAGEMENT

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Abstract
Lichen planus (LP) is a chronic inflammatory disorder of unknown etiology, affecting the skin and mucous membranes. It is characterized by pruritic, purple, polygonal papules and is often associated with autoimmune mechanisms and certain triggers such as hepatitis C and medications. This study outlines the clinical features, etiology, pathogenesis, and management strategies, including EMO therapy. It also highlights preventive measures to reduce recurrence and complications, supported by recent literature and evidence-based practices.

***Keywords*:** Lichen Planus, Autoimmune Disorder, Pruritic Papules, Hepatitis C Association, EMO Therapy, Evidence-Based Management.

Introduction
Lichen planus is a mucocutaneous disorder marked by a T-cell-mediated immune response. It occurs in approximately 1% of the general population, commonly between the ages of 30 and 60, with a slight female predominance [1]. Although it may be self-limiting, oral and genital variants can be chronic and painful [2].

Types of Lichen Planus
1. Cutaneous LP-Involves flat-topped, itchy skin papules [3].
2. Oral LP-Affects buccal mucosa with white striae or erosions [4].
3. Genital LP-Leads to pain and erosions in vulva or glans penis [5].
4. Lichen Planopilaris-Causes scarring alopecia on the scalp [6].
5. Nail LP-Nail thinning, ridging, or destruction [7].
6. Hypertrophic LP-Thick, pruritic plaques on lower limbs [8].
7. Actinic LP-Sun-exposed areas, especially in dark-skinned individuals [9].
8. Bullous LP – Rare; involves blister formation [10].
9. Erosive LP-Painful mucosal ulcers with potential malignant transformation [11].
10. Linear LP-Lesions follow lines of Blaschko [12].

Fig 1: Papular Lichen planus coalescing into plaques on the wrist

Etiology
1. Idiopathic (common) [1].
2. Autoimmune basis-CD8+ T-cell attack on basal keratinocytes [13].
3. Drugs-NSAIDs, beta-blockers, antimalarials [2].
4. Infections-Especially hepatitis C virus (HCV) [9, 14].
5. Stress and trauma-May initiate or exacerbate LP [15].
6. Dental materials-Amalgam restorations may trigger oral LP [16].

Fig 2: Flat-topped, shiny Polygonal papules of lichen plauns on the volur wrist

Fig 3: Papular Lichen planus with Koebner phenomenon inskin of colour

Fig 4: Papular Lichen planus with a linear koebner Lesion due to scratch

Symptoms

- Skin: Pruritic, purple papules that are polygonal and flat-topped [1].
- Oral cavity: White lacy lines (Wickham's striae), erosions, burning [4].
- Genitalia: Painful erosions and burning [5].
- Nails: Longitudinal ridging, thinning, anonychia [7].
- Scalp: patches of alopecia in lichen planopilaris [6].
- Pain/Itching: Common due to inflammatory infiltration [3].

Pathophysiology

Autoimmune Reaction

- It is a T-cell-mediated autoimmune disorder.

Triggering Factors

- Certain drugs, infections (like Hepatitis C), or stress may alter keratinocytes, making them appear "foreign" to the immune system.

CD8+ T-Cell Activation

- Cytotoxic CD8+ T-cells are activated and attack basal keratinocytes[13, 17].
- Apoptosis of Keratinocytes:
- The immune attack leads to programmed cell death (apoptosis) of basal layer cells in the skin/mucosa.

Inflammatory Infiltrate

- A dense band-like infiltration of T-lymphocytes occurs at the dermoepidermal junction.
- Cytokine Release:
- Inflammatory cytokines (like IFN-γ, TNF-α) are released, further promoting tissue damage.

Damage to Basal Layer

- Leads to vacuolar degeneration of basal cells and formation of Civatte bodies (apoptotic keratinocytes).

Hyperkeratosis &Hypergranulosis

- The epidermis shows thickening due to increased keratin (hyperkeratosis) and granular layer (hypergranulosis).

Saw-tooth Rete Ridges

- Histologically, the rete ridges appear pointed or "saw-toothed" due to inflammation and basal cell damage [17].

Diagnosis and Clinical Presentation

1. Clinical Presentation

- Classic skin lesions on wrists and ankles [1].
- Oral LP: Reticular or erosive pattern on buccal mucosa [4].
- Nail and scalp involvement depending on subtype [6][7].
- Physical Examination:
- Presence of Wickham's striae (lace-like white lines) [4].
- Koebner phenomenon: lesions on trauma sites [3].

2. Laboratory Investigations:

- Skin biopsy: Gold standard; shows band-like lymphocytic infiltrate and saw-tooth rete ridges [17].
- Direct immunofluorescence (DIF): Fibrinogen deposits along basement membrane [19].
- HCV serology: Especially in oral LP patients [9][14].
- Liver function tests: If systemic symptoms present[14].

Treatment

EMO Therapy (Erythema, Mucosal, Other lesions)

- Topical corticosteroids (e.g., clobetasol): For skin and mucosal lesions [2, 15].
- Oral corticosteroids: In severe or generalized LP [2].
- Calcineurin inhibitors (e.g.,tacrolimus): For resistant oral/genital LP [19].
- Antihistamines: For pruritus relief [3].
- Phototherapy: Narrowband UVB or PUVA for generalized LP [20].
- Systemic immunosuppressants: Methotrexate, cyclosporine, or acitretin in severe or erosive LP [15, 20].

Preventive Measures

- Avoid known triggering medications [2].
- Maintain good oral hygiene [4].
- Avoid smoking, spicy foods, and alcohol in oral LP [4].
- Manage stress with relaxation techniques [15].
- Routine monitoring of oral erosive LP due to risk of malignant transformation [11, 18].

Case Study

A 45-year-old female patient was admitted to the dermatology department with chief complaints of itchy, violaceous, flat-topped papules distributed over the wrists, forearms, lower back, and ankles, persisting for the past three weeks. Additionally, she reported the presence of white, lacy patches on the buccal mucosa, which caused a burning sensation, particularly when consuming spicy foods. The patient had no prior history of autoimmune disease but reported recent psychological stress due to a personal loss and had initiated a new antihypertensive medication, Hydrochlorothiazide, one month earlier. She has been a known hypertensive for five years with stable control on medication. Laboratory investigations revealed hemoglobin at 12.5 g/dL, platelet count of 3.2 lakh/mm^3, white blood cell count of 8700 cells/mm^3, SGPT of 32 IU/L, and SGOT of 28 IU/L. Both Hepatitis B and C serologies and ANA profile were negative. A skin biopsy confirmed the diagnosis of Lichen Planus, revealing histopathological features including hyperkeratosis, saw-tooth rete ridges, and a band-like lymphocytic infiltrate at the dermoepidermal junction. During hospitalization, the patient was treated with intramuscular dexamethasone (2 cc), oral prednisolone 25 mg once daily in the morning, topical 0.1% betamethasone applied twice daily, oral cetirizine 10 mg at night, chlorpheniramine 4 mg in the morning, topical liquid paraffin applied twice daily, and pantoprazole 40 mg once daily before breakfast. Upon discharge, she was prescribed the continuation of ointment 0.1% betamethasone (1-0-1), tablet cetirizine 10 mg at night (0-0-1), tablet chlorpheniramine 4 mg in the morning (1-0-0), topical liquid paraffin (1-0-1), tablet pantoprazole 40 mg before breakfast (1-0-0), and a tapering dose of tablet prednisolone after breakfast.

Discussion

Lichen Planus (LP) is a chronic, immune-mediated inflammatory disorder that affects both the skin and mucous membranes, though its exact cause remains unknown. It most commonly occurs in middle-aged adults and can involve various sites, including the skin, oral and genital mucosa, nails, and scalp. Diagnosis is confirmed through skin biopsy, which typically reveals hallmark histopathological features such as “saw-tooth” rete ridges and a dense band-like lymphocytic infiltrate at the dermoepidermal junction. Early recognition and appropriate treatment are essential for improving patient outcomes and minimizing complications such as post-inflammatory hyperpigmentation or erosive forms of LP. Given the rare but potential risk of malignant transformation to oral squamous cell carcinoma, long-term follow-up is recommended.

Conclusion

Lichen Planus is a chronic inflammatory condition that can significantly affect a patient's quality of life due to its persistent symptoms and frequent recurrences. Early diagnosis and appropriate management using a combination of systemic and topical corticosteroids, antihistamines, and emollients are crucial in relieving symptoms and preventing complications. In this case, the patient responded well to the treatment regimen, showing significant clinical improvement within 10 days. Continuous monitoring and patient education on adherence and follow-up are essential for long-term disease control and relapse prevention.

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Author Contribution

Both authors are contributed equally.

Inform Consent and Ethical Permission

Prior informed consent was obtained from the patient for documenting this case. Ethical approval was taken from the Department Head and Medical Superintendent of the Government General Hospital, Guntur.

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Conflict of Interest

No Conflict of Interest.

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