Lichen planus (LP) is a potentially malignant disorder with an immune-mediated etiopathogenesis. The condition frequently affects the skin, oral mucosa, skin appendages, and other mucous membranes. Oral lesions usually precede the onset of skin lesions and in majority of cases may only be presenting symptom. Isolated LP of the lip is rarely encountered in the clinical practice and is usually seen along with skin/other mucous membrane involvement. The clinical appearance poses diagnostic dilemmas and is often misinterpreted. This case report aims to highlight an interesting case of LP of the lower lip in a 50-year-old male patient. The patient presented with a diffuse erosive lesion on the lower lip bordered by white radiating striae on its inner aspect. Histopathological and immunofluorescent studies confirmed LP of the lip. Topical corticosteroids and Vaseline lip therapy were prescribed to the patient. There was considerable healing in the lip lesion during the follow-up period. However, recurrence was noted in the left buccal mucosa.

**Keywords:** Diagnosis, lichen planus, lichen planus lip, malignant lesion

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**Case Report**

A 50-year-old male patient reported to the outpatient department with a chief complaint of ulcerations in the lower lip for the past 6 months. History reveals that the patient was asymptomatic 6 months back when he noticed itching sensation in the lower lip, followed by development of minute ulcerations in the lower lip region. For the past 2 months, he complained of burning sensation and mild dull intermittent pain in the ulcerated areas. His medical history was nonsignificant, except for bidi smoking (two to three packets/day for the past 20 years). The patient has not taken any treatment for ulcerations. There was no associated history of vesicle formation in any area of oral cavity. The patient denied factitial habit of lip biting and excessive sun exposure. Physical examination revealed a diffuse erosive lesion on the lower lip extending till the mucocutaneous junction and inner aspect of lower labial mucosa. The diffuse erosive lesion roughly measured 2 × 3 cm and had irregular exhibit malignant potential, thus necessitating prompt diagnosis and management of such lesions.[1]

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margins. It was surrounded by erythema and covered with areas of blood-tinged crustations. Greyish-white radiating striae were seen bordering the inner aspect of lower labial mucosa. On palpation, the lesion was mildly tender and nonindurated. There was mild diffuse swelling over the lower lip. Gentle manipulation of the normal mucosa did not induce formation of new lesions (negative Nikolsky’s sign) [Figure 1a and b]. The patient’s oral hygiene was poor with generalized grade I mobility in teeth. There were no signs of associated oral, ocular, cutaneous, or genital lesions. History of chronic duration of 6 months, with the clinical evidence of diffuse erosive lesion bordered by characteristic radiating white striae, and a negative Nikolsky’s sign led to a provisional diagnosis of OLP of the lower lip. Oral lichenoid reaction (OLR), discoid lupus erythematosus (DLE), pemphigus vulgaris, erythema multiforme, and actinic cheilitis were considered as the most probable differential diagnosis. OLRs were ruled out as there was no history of drug intake prior to the onset of the lesion. Also, there was no clinical evidence of amalgam restoration in patient’s mandibular anterior teeth. Absence of vesicle/bullae formation, skin lesions, and a negative Nikolsky’s sign ruled out the possibility of pemphigus vulgaris. Erythema multiforme was excluded due to the chronicity of the lesion (6-month duration), absence of vesicle/bullae, and absence of skin lesions (target lesions). DLE was excluded as the erosive lesion remained confined to the vermilion border without blurring the sharp line of the vermilion border, an important feature of LP of the lip. Also, the irradiating white striae are much more delicate in DLE when compared with LP. After informing the patient and obtaining consent, an incisional biopsy was performed from the perilesional region for histopathological and immunofluorescence studies. Histopathological features revealed liquefaction degeneration of epithelial basal layer and inflammatory infiltrates predominated by lymphocytes and saw tooth rete pegs in the connective tissue with no evidence of dysplastic changes [Figure 2]. Absence of atypia and dysplasia histologically excluded the possibility of leukoplakia and carcinoma in situ. OLRs were ruled out as inflammatory infiltrates predominantly comprised lymphocytes (in contrast to OLR where plasma cells, eosinophils, and neutrophils predominate the inflammatory infiltrate). Absence of parakeratosis, keratin plugging, and perivascular infiltrates ruled out lupus erythematosus. Direct immunofluorescence showed a shaggy band of fibrinogen in the basement membrane [Figure 3]. The histopathological and immunofluorescent features were consistent with the clinical diagnosis of OLP. The patient was subjected to thorough oral prophylaxis and instructed to maintain the oral hygiene. Thereafter, the patient was prescribed topical application of low-potency steroids (Kenacort 0.1% paste three to four times daily), along with Vaseline lip therapy and chewable tablet of vitamin C (tab. lymcee two times daily) for a month. The patient was reviewed after 1 month and the lesions had considerably subsided with topical steroid treatment. Lip lesions showed almost complete resolution after 2 months of therapy [Figure 4a and b]. However, recurrence was seen on the left buccal mucosa as an erythematous lesion surrounded by interlacing white striae [Figure 5]. The patient was prescribed the same therapy for recurrent lesions. Unfortunately, the patient did not report for further follow-up visits.

**Discussion**

LP is a chronic mucocutaneous disease of the stratified squamous epithelium. Mucous membranes of the oral and genital region, skin, scalp, and nails are the frequently affected sites. Prevalence rate is 0.1%–4% of the general population, most often in
perimenopausal women. The condition has a predilection for individuals in the age range of 30–60 years, although individuals of any age may be affected. The exact etiology is still not completely elucidated, and multifactorial etiopathogenesis has been suggested. The autoimmune nature of LP has long been proposed, and CD8+ T lymphocytes bring about the apoptosis of keratinocytes and result in damage to the epithelial basal cell layer. Allergic reaction to dental restorative agents (amalgam, gold), chronic irritants such as sharp cusp of tooth and ill-fitting prosthesis (Koebner phenomenon) and hepatitis B or C virus (HCV) infection seems to be the most likely causes of OLP. Genetic factors, lifestyle, and psychological factors (stress and anxiety) may also play a significant role in the pathogenesis of OLP. Lichenoid drug reaction may be caused due to many drugs (nonsteroidal anti-inflammatory drugs and angiotensin-converting enzyme drugs).

Our patient was a 50-year-old male who had a history of smoking (two packets/day) for the past 20 years. However, no other contributory factors could be elicited. According to Andreason, OLP lesions can be classified into six different forms: reticular, papular, plaque-like, atrophic, ulcerative, and bullous. Recent classifications group OLP lesions into reticular (reticular, papular, plaque-like), erythematous (atrophic), and erosive (ulcerative, bullous) forms. Some authors have limited the forms to reticular (reticular, plaque-like) and erosive (atrophic, bullous, and ulcerative).

LP has a site predilection for buccal mucosa, followed by tongue (mainly the dorsum), gingiva, labial mucosa, and vermilion of the lower lip. LP isolated to a single oral site other than the gingiva is also unusual, although occasional cases of isolated lesions on the lips or tongue have been reported.

Our patient showed an isolated diffuse erosive lesion, 2 × 3 cm in diameter on the lower lip region, extending up to the mucocutaneous junction and on its inner aspect with white interlacing striae. Erosive lesion was covered with hemorrhagic crusts and erythema, and gentle manipulation of the normal mucosa elicited a negative Nikolsky's sign.

Only a few case reports and case series of LP of lips have been published so far [Table 1].

The essential clinical characteristics usually comprise symmetrical, bilateral, and reticular lesions. Band-like lymphocytic infiltrates in the connective tissue area, hyperkeratosis with ortho- and/or parakeratosis, liquefaction degeneration of the epithelial basal layer, and absence of epithelial dysplasia constitute the characteristic histopathological features.

In the reported case, the histopathological features were characteristic for LP and aided in histological differential diagnosis with leukoplakia, invasive carcinoma, and other forms of cheilitis.

Immunoflorescence shows a linear pattern of fibrin and shaggy fibrinogen deposits at the epithelial basement membrane or cytid bodies (Russell bodies), or both, in the absence of deposition of fibrinogen. Direct immunofluorescence features in the present case showed a shaggy band of fibrinogen at the basement membrane.

There is no definitive treatment protocol for OLP. The therapy mainly aims at improvement of symptoms and regular observation of dysplastic changes. Topical glucocorticoids usually form the mainstay of treatment for erosive OLP, although systemic and intralesional steroids are also in use. Use of orabase should be exclusively on moist intraoral sites, and topical steroids in orabase vehicle should be avoided for lip lesions.

Topical and systemic immunosuppressive agents, such as retinoids, cyclosporine, and tacrolimus, may also be used. Thalidomide and psoralen–UV-A are reserved for the recalcitrant disease. Yu et al. reported the role of oral traditional Chinese medications and topical wet dressing in management of isolated lip LP. Surgical management, including cryosurgery and carbon dioxide (CO2) laser, has been performed on OLP lesions. As LP is an inflammatory condition prone for recurrence, and therefore, surgical excision is not considered as the first-choice treatment of LP.

![Figure 4: (a and b) Considerable resolution of the lesion after 1 month. Almost completely healed lesion after 2 months](image)

![Figure 5: Recurrence of the lesion in the left buccal mucosa](image)
| Author           | Year | No. of patients | Age/sex | Site                | Features                      | Clinical type | Cutaneous/other mucosal/skin appendage involvement | Systemic pathologies | Diagnosis | Management                  | Results                      |
|------------------|------|-----------------|---------|---------------------|-------------------------------|---------------|-------------------------------------------------|--------------------|-----------|-----------------------------|-----------------------------|
| Whittle CH       | 1937 | 01 69/M         | Lower lip | Irritation | Plaque (appeared as leukoplakic patch) | Genital mucosa | NA                      | Mercury, arsenic, X-rays | LPL?  | Stable                      |                             |
| Altman J         | 1961 | NA NA           | NA NA NA  | NA N/A             | NA                            | NA            | NA                               | NA                | LPL       | NA                          | NA                          |
| Piamphongsant T. | 1978 | 02 NA           | Lower lip | NA NA             | Nodular                       | Skin involvement | H/O smoking 10 years back | OLP                | Topical steroid     | Recurrence as SCC, vermilionectomy and radiotherapy for SCC |
| Harland CC       | 1992 | 01 23/M         | Lower lip + buccal mucosa | NA | Nodular | Skin involvement | H/O smoking 10 years back | OLP                | Topical steroid     | Recurrence as SCC, vermilionectomy and radiotherapy for SCC |
| Itin PH          | 1995 | 01 44/M         | Lower lip | Edematous lip with erosions and crusting | Erosive | No | LPL | Acitretin + steroids + sunscreen | Resolved lesions |
| Allan SJ         | 1996 | 01 51/M         | Lower lip | Itching and scales | Reticular | No | LPL | Steroids | Resolved lesions |
| Isogai Z         | 1997 | 01 54/M         | Lower lip | Pain | Erythematous | Skin+nails | NA | LPL | Steroid | Resolved lesions in 4 weeks |
| De Argila D      | 1997 | 01 51/F         | Lower lip | NA | NA | No | LPL | Chloroquine phosphate | Resolved lesions |
| Melato M         | 2000 | 01 NA           | Upper lip | NA | NA | Morphea on upper lip | Vitiligo | LPL + morphea | NA | Resolved lesions in 4 weeks |
| Demitsu T        | 2000 | 01 62/F         | Lower lip | NA | erosive | NA | LPL | Steroid-resistant case treated with cyclosporine | Resolved lesion in 4 weeks |
| Cecchi R         | 2002 | 01 45/M         | Lower lip | Swollen lip with burning | Erythematous/ulcerated | No | LPL | Steroid | Resolved lesion, lichenoid papule after 4 months |
| Chiang CT        | 2002 | 01 36/F         | Lower lip | Painful ulcers | Erythematous | No | LPL + superficial mycosis | Unsatisfactory results from ketoconazole | Griesofulvin + steroids | Resolved lesions in 3 weeks, recurred 1 week after therapy |
| Yu TC            | 2003 | 01 44/M         | Lower lip | Lip edema with burning pain | Erosive | No | LPL | Topical steroids | Resolved lesions in 6 weeks |
| Donovan JC       | 2005 | 01 51/M         | Lip | Pain | Erosive | NA | LPL | Steroid refractory LP treated with 0.1% tacrolimus | Resolved lesions within 2 weeks of therapy, no recurrence after a year's follow-up |

Contd...
| Author          | Year | No. of patients | Age/sex | Site | Features | Clinical type | Diagnosis | Management | Results                  |
|-----------------|------|-----------------|---------|------|----------|---------------|-----------|------------|--------------------------|
| Schindah E R    | 2006 | 02              | 64/M    | Lower lip + buccal mucosa | Pain | Erosive-| OLP | Tacrolimus | Resolved lesion, No recurrences reported |
| Van Tuyll SAM   | 2007 | 01              | 74/F    | Lower lip + buccal mucosa | Burning + bleeding | Erosive- | OLP | Tacrolimus | Resolved lesion, No recurrences reported |
| Petruzzi M      | 2007 | 10              | NA      | Lower + upper lip | Erosion with hemorrhagic crusts | Erosive | LPL | Topical + intralesional steroids | Resolved lesion after 3 years |
| Johnson H       | 2008 | 01              | 42/F    | Lower lip | Dryness + peeling | Erosive | LPL | Tacrolimus | Stable |
| De Morais PC    | 2011 | 01              | 50/M    | Upper lip | NA | NA | LPL | Topical + intralesional steroids | Resolved lesion after 3 years |
| Gencoglan G     | 2011 | 04              | 50/M, 61/M, 65/M, 22/M | Lower lip | Painful erosion/erythematous plaques | Erosive/erythematous | LPL | Imiquimod cream | Resolved lesion after 3 years |
| Sugashima Y     | 2012 | 01              | 32/F    | Lower + upper lip | Asymptomatic Amnular erythematous plaques | Erosive | LPL | Topical + intralesional steroids | Resolved lesion after 3 years |
| Holmboe S       | 2015 | 01              | 50/M    | Lower lip | Precipitated by sun exposure | Erosive | LPL | Topical + intralesional steroids | No recurrences after 2 year follow up |
| Choi E          | 2017 | 01              | 62/F    | Lower lip | Recurrent ulcerations | Amnular | LPL | Topical tacrolimus + corticosteroids | Resolved lesion after 3 years |
| Hasan S         | 2017 | 01              | 60/M    | Lower lip | Burning in the ulcerated region | Amnular | LPL | Topical tacrolimus | Resolved lesion after 3 years |

OLP: Oral lichen planus; SCC: Squamous cell carcinoma; LPL: Lichen planus Lip; LES: Lupus erythematosus; H/O: History of; NA: Not applicable.
Our patient was prescribed topical application of low-potency steroid (kennacort 0.1% paste three to four times daily), along with Vaseline lip therapy and chewable tablet of vitamin C (tab. lymcee two times daily) for a month. Lip lesions had considerably subsided with topical steroid treatment; however, recurrence on the left buccal mucosa was noted after 2 months of follow-up. The patient was lost for subsequent follow-up visits.

LP is associated with various comorbidities including diabetes mellitus, metabolic syndrome, dyslipidemia (predisposing factor for cardiovascular diseases), thyroid dysfunction (hypothyroidism), and HCV infection. Hence, patients with OLP require special attention from health professionals and should be thoroughly screened for early detection of these ailments, thus preventing complications and prolonged disabilities.[21]

The predisposition to oral squamous cell carcinoma (OSCC) is the most dreaded complication of OLP.[22] The first case of OLP with malignant changes was reported by Hallopeau in 1910.[23] The predisposition of malignant transformation ranges between 0.4% and 5%.[24] and about 1.1% of patients with OLP with a high frequency of smoking, alcohol abuse, and HCV infection have a propensity to develop OSCC.[25] Our patient did not show any signs of malignant changes during the 2-month follow-up period.

Conclusion

Lip involvement is an uncommon and unusual site of presentation of OLP. Early and correct diagnosis and effective management together with periodic follow-up is mandatory to alleviate the pain and symptoms. This might also be a significant preventive protocol for development of squamous cell carcinoma in lip LP lesions.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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