Oral Lichen Planus

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CASE SUMMARY

History

A 78-year-old housewife complained of multiple persistent erosions over the lower lip and the left buccal mucosa for one year. The lesions were associated with severe burning pain leading to difficulty in eating and swallowing.

She was a non-smoker and non-drinker. Her past health was good. Her family and drug history were unremarkable. She was wearing a dental prosthesis made of stainless steel for ten years. There was no other metal used in dental restoration.

Physical examination

There were multiple ulcerations over the lower lip (Figure 1). The gingiva was swollen and inflamed (Figure 1). Lace-like whitish streaks and well-defined whitish plaques were found on the lower lip, gingiva and the left buccal mucosa (Figures 1& 2). There was no blister or erosion on the trunk or limbs. There was no perineal, eye or nail involvement.

Differential diagnoses

The clinical differential diagnoses included oral lichen planus, lichenoid drug eruption, immunobullous disease, lupus erythematosus, leukoplakia and malignancy.

Investigations

Complete blood picture, liver and renal function tests were normal. Anti-skin antibody was negative. Anti-nuclear factor was 1:360 and anti-DNA was negative. Syphilitic serology was negative. Wound swab taken from the oral ulceration did not grow any organism. Skin biopsy done on the lower lip showed marked acanthosis and heavy infiltration of predominantly plasma cells mixed with a few lymphocytes in the dermis. A few dyskeratotic cells were present at the dermal epidermal junction. There was no evidence of malignancy, basal cell vacuolation or intraepidermal bulla. Special stains for fungi, bacteria and acid-fast bacilli were negative. The biopsy was suggestive of mucosal lichen planus.

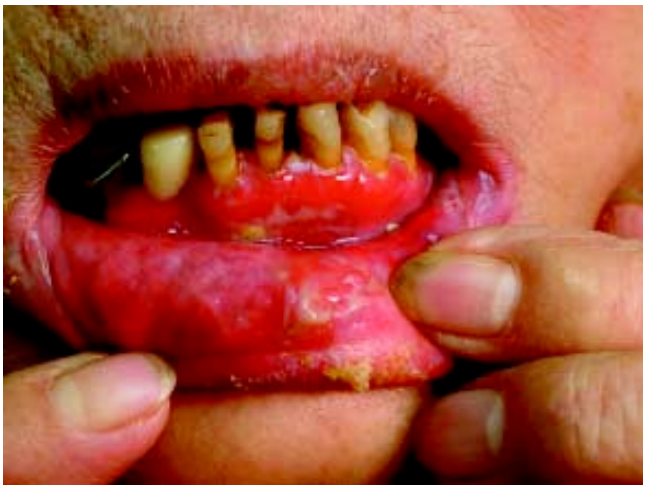

Figure 1: Ulcerations, gingivitis and Wickham's striae on the lower lip and gingiva

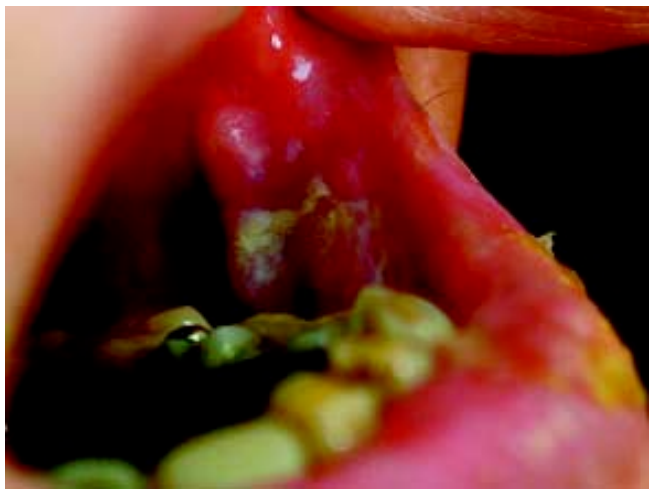

Figure 2: Whitish plaque on the buccal mucosa

Diagnosis

The most likely diagnosis was oral lichen planus.

Management

The patient had dental assessment and no element of contact allergy to any dental material was found. Simple measures including oral analgesic, antiseptic gargles and oral nystatin suspension mouthwash for the treatment of oral candidiasis were initiated. Moderate potent topical steroid was also started but the clinical condition was refractory to the above measures. Systemic prednisolone 20 mg per day was given for nine weeks with partial response only. Intralesional steroid was injected twice into the lower lip erosive area. The size of the erosions and pain reduced subsequently.

REVIEW ON ORAL LICHEN PLANUS

Lichen planus is a common mucocutaneous inflammatory disease. It affects 0.5% to 1% of the world's population.[1] Approximately half of the patients with cutaneous lichen planus have oral involvement.[1] However, mucosal involvement can be the sole manifestation in up to 25% of affected population.[1] Oral lichen planus has a peak incidence in middle age patients and has female predominance of 2:1.[2] It is characteristically associated with persistent clinical course and resistance to most conventional treatments.

Clinical features

There are various clinical morphological manifestations of the disease (Table 1). More than one clinical subtype can co-exist in the same patient. The reticular (92%), plaque (36%) and papule (11%) types are usually asymptomatic and require no specific treatment.[3] On the other hand, the atrophic (44%), erosive (9%) and bullous (1%) types usually cause severe burning pain and are refractory to conventional treatments.[3]

The lesions are usually symmetrical. It frequently affects buccal mucosa, tongue, gingiva, lip and palate. Extra-oral mucosal involvements include the anogenital area (vulvovaginal-gingival syndrome), conjunctivae, oesophagus or larynx.

Approximately 1.2% to 5.3% of oral lichen planus lesions will undergo malignant changes.[4-6] Regular follow up of these patients is mandatory. High risk factors for malignant transformation include smoking, excessive alcohol intake, atrophic, ulcerative or erosive clinical types, presence of erythroplakic lesions and sites involving the tongue, gingiva or buccal mucosa.[7]

Histopathology

Basal epidermal keratinocyte damage and lichenoid-interface lymphocytic reaction are the two major pathological findings. There are presence of hyperkeratosis, increased granular layer, irregular acanthosis, liquefactive degeneration of the basal cell layer and band-like lymphocytic infiltrate in the upper dermis. In mucosal lichen planus, plasma cells are more prominent. Degenerated keratinocytes (colloid, Civatte bodies) are present at the dermal-epidermal junction. Direct immunofluorescence shows heavy deposits of fibrin at the dermo-epidermal junction. Deposits of IgM and less frequently IgG, IgA and C3 are also found in the colloid bodies.

Pathogenesis

Lichen planus is a T-cell mediated autoimmune damage to basal keratinocytes.[8] Suspected aetiological factors include drugs, food, oral candidiasis, hepatitis C, cigarette smoking, contact allergic factors such as

Table 1. Clinical features of oral lichen planus

Symptom	Clinical types	Description
Asymptomatic	Reticular	Wickham's striae ± discrete erythematous border
	Plaque-like	Resemble leukoplakia, common in smokers
	Papules	Small white pinpoint papules
Symptomatic	Atrophic	Diffuse red patch, peripheral radiating white striae, chronic desquamative gingivitis
	Erosive	Irregular erosion covered with a pseudomembrane
	Bullous	Small bullae or vesicles that may rupture easily

amalgam or gold dental restorative material and trauma from periodontal prosthesis.[7]

Lichenoid drug eruption

Lichenoid drug eruptions have been reported after ingestion, contact, or inhalation of various types of chemicals.

There were fewer reports of oral medications causing lichenoid drug eruptions with oral predilection. The most likely culprit drugs include allopurinol, angiotensin converting enzyme inhibitor, gold, ketoconazole, methyldopa, non-steroidal anti-inflammatory drugs, penicillamine and sulphonylurea hypoglycaemic drugs.[9] Histology and immunology cannot reliably differentiate oral lichenoid drug eruptions from idiopathic lichen planus or lichen planus-like oral eruptions in graft versus host disease. However, in lichenoid drug eruptions with mucosal involvement, parakeratosis is common and there may be more diffuse and deeper subepithelial eosinophil and plasma cell infiltrate.[9] Clinical features such as unilateral distribution, resolution and recurrence of lesions on withdrawal and re-exposure to the suspected drug, also suggest lichenoid drug eruption.[9]

Oral lesions in relation to metals used in dental restoration

Sensitization to mercury (amalgam) has been proposed as an aetiological factor in oral lichenoid lesions. Koch et al[10] studied the sensitization rate of metal salts in 194 patients with oral lesions, using patch tests consisting of the German standard, dental prosthesis and the metal salt series. The effect of the removal of amalgam was also investigated. The patients suffered from: oral lichenoid lesions adjacent to amalgam fillings (n=19); oral lichen planus without close contact with amalgam or dental gold (n=42); other oral lesions (n=28); oral complaints (n=46) and control (n=59). It was found that significantly more patients with oral lichenoid lesions adjacent to amalgam fillings were sensitized to inorganic mercury (78.9%) as compared to other groups (0% to 12%).[10] One third of the sensitization reactions were observed as late 10 or 17 days after the patch test. Amalgam was then removed in 29 patients with oral lichenoid lesions adjacent to amalgam or oral lichen planus not in close contact with amalgam. In the former group, 72.2% (13/18) in overall or 86.6% (13/15) of those with positive sensitization to inorganic mercury showed clearance or improvement in lesions after the removal of amalgam.[10] In the latter group, only 27.2% (3/11) showed clearance or improvement of the lesions.[10] It was concluded that amalgam fillings should only be removed from patients who were sensitized to inorganic mercury compounds and who had objective oral lesions.[10]

Management

In general, non-erosive oral lichen planus is asymptomatic and treatment is often unnecessary. However, patients with erosive type oral lichen planus often present with significant management problems. At present, none of the available treatment is specific or universally effective. An algorithm for the management of oral lichen planus is shown in Figure 3.[2,11,12]

All patients should optimize their oral hygiene. Oral candidiasis should be excluded or treated accordingly. In symptomatic patients with apparent contact dental factor, patch test with replacement of the amalgam or gold restorative material is suggested in those who are sensitized to these metals. In those with no apparent contact factor, topical or intralesional steroid is usually the first line treatment. A short course of systemic steroid may be necessary for more rapid control. In steroid-dependent patients, azathioprine or topical cyclosporin can be added as an adjunctive therapy. In refractory cases, alternative therapies such as topical or systemic retinoids, antimalarial, dapsone, oral PUVA, thalidomide or topical tacrolimus may be considered.[11-15] Surgical treatments such as laser, cryotherapy and excision may exacerbate the conditions due to the Koebner phenomenon.

Learning points:

Oral lichen planus presenting as atrophic, erosive or bullous clinical types are usually symptomatic and refractory to treatments. In patients with oral lichenoid lesions, drug eruption due to oral medications or dental contact factor should be eliminated.

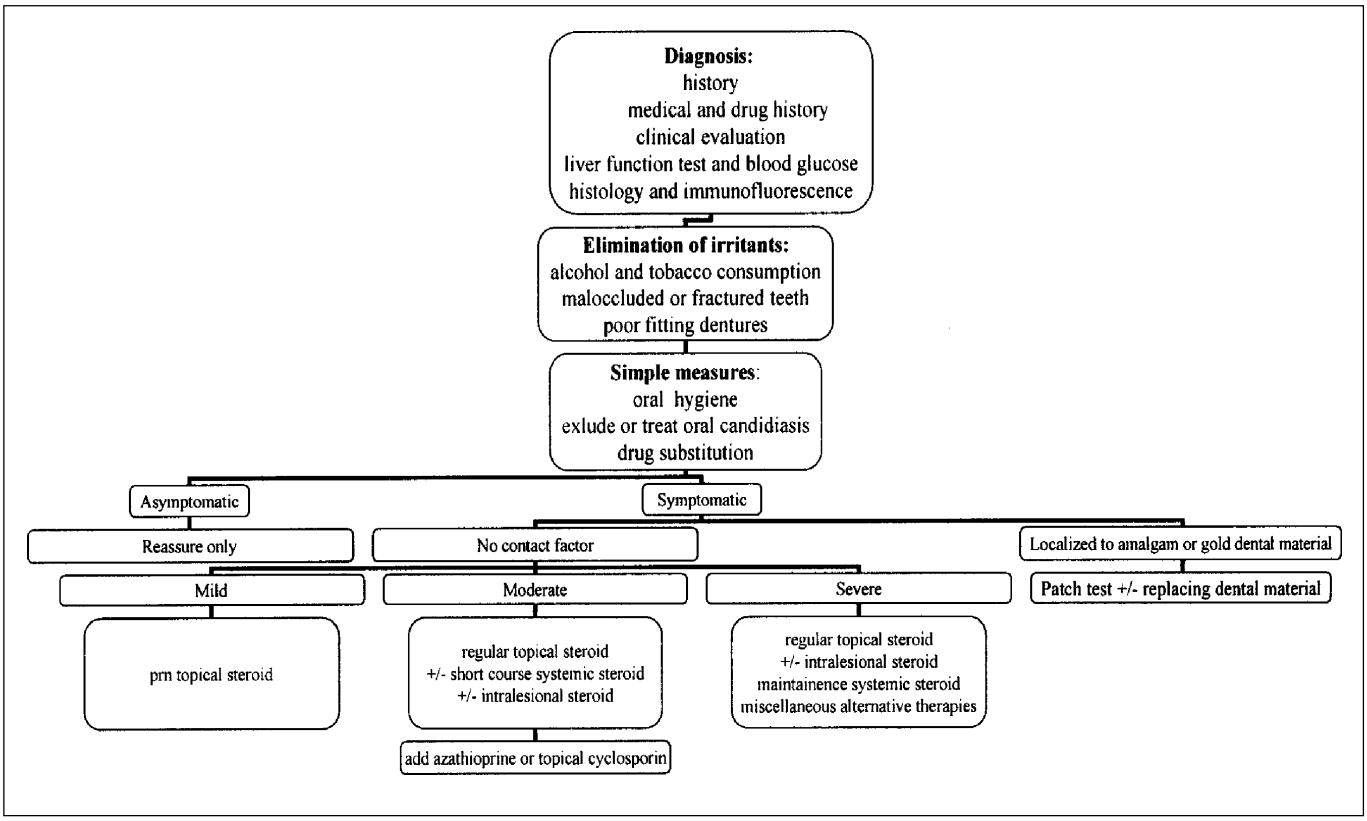

Figure 3: Algorithm for the management of oral lichen planus[2,11,12]

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