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Extensive Oral Lichen Planus with Cutaneous Manifestations, A Case Report and Review of the Literature

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I. Case Report

A fifty-two years old female referred to the oral medicine clinic, Faculty of Dentistry, University of the Western Cape complaining ofmouthburning that has been present for more than two years.The patient mentioned that she had been diagnosed with cutaneous lichen planus in 2010. She felt improvement of her skin condition since the diagnosis and discontinued follow up with no treatment.

Her medical history revealed that she was hypertensive and on Pharmapress 10 mg and Radix 12.5 mg daily. She also reported a history of allergy to penicillin and sulphonomide.

Extra-oral examination showed round brown- black pigmented macules and papules that were located in the lower back. There were neither lesions located on the flexor surfaces of the extremities nor associated itching with her back lesions (Fig.1). The intraoral examination revealed diffuse striated white patches that cannot be wiped away with gauze. The lesions (Fig. 2, 3, 4) were located bilaterally on the buccal mucosa, the ventral and dorsal surfaces of the tongue, palate, alveolar mucosa, and gingiva. Erosions were also noted on the upper left premolar and molar area (the patient was edentulous). Pigmented plaques were also noted within the white lesions of the buccal mucosa. The tongue was depapillated and glossy.

The clinical differential diagnosis considered was lichen planus, lichenoid reaction and leukoplakia.Two incisional biopsies were performed from the left buccal mucosa and the palate (Fig. 5). The patient was prescribed Ibuprofen 400 mg three times per day for one week and requested to follow up after one week.

The Biopsy result of the new oral lesions revealed licheanoid interface mucositis with a dense lymphocytic infiltrate in the superficial lamina propria. The previous biopsy from the skin revealed similar changes to the oral lesions in addition to sub-epithelial clefting (Fig. 6).

The biopsy result of oral lesions revealed lichenoid interface mucositis with a dense lymphocytic infiltrate in the superficial lamina propria (Fig. 6). A previous biopsy of her skin revealed similar changes to the oral lesions in addition to sub-epithelial clefting. Based on the history, clinicopathological findings and after excluding other causes of licheanoid reaction, the diagnosis of oral lichen planus was established. The patient was prescribed Chlorohexidine mouth wash 0.12% to use two times per day and was advised to avoid spicy and acidic food. Topical Methylprednisolone Aceponate cream 1mg (15g Tube) was also prescribed three times daily and periodic follow up every three months was recommended.

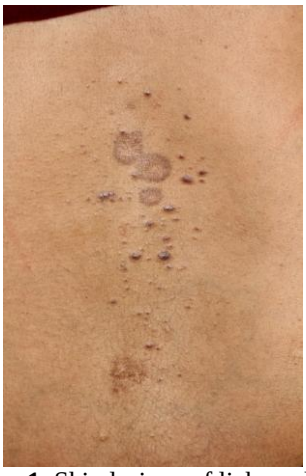

Figure 1. Skin lesions of lichen planus

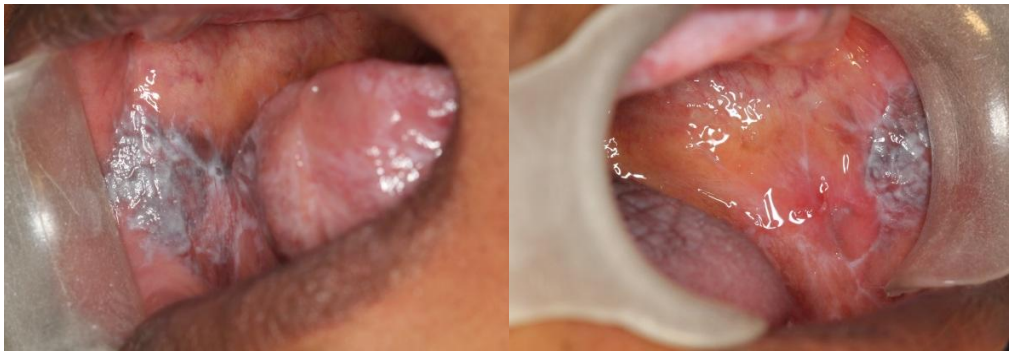

Figure 2. Lichen planus in the buccal mucosa with post-inflammatory pigmentation

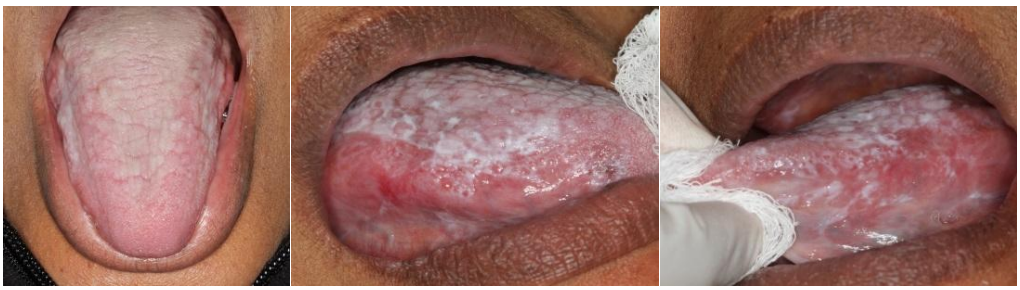

Figure 3. Lichen planus of the Tongue

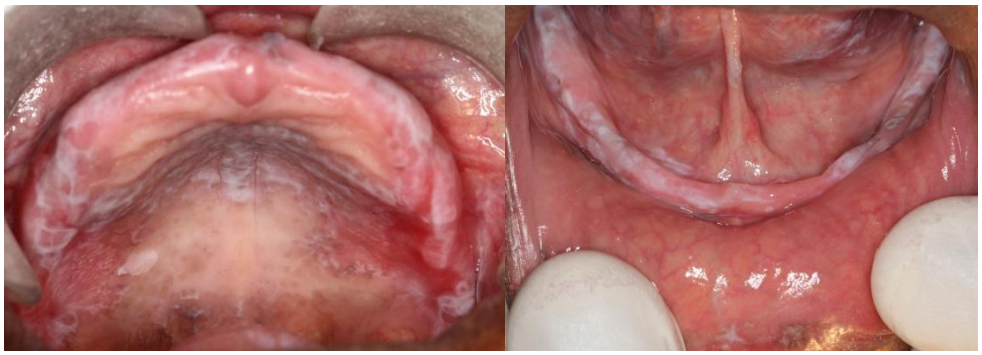

Figure 4. Lichen planus involving the palate, alveolar mucosa, ventral surface of the tongue and floor of the mouth

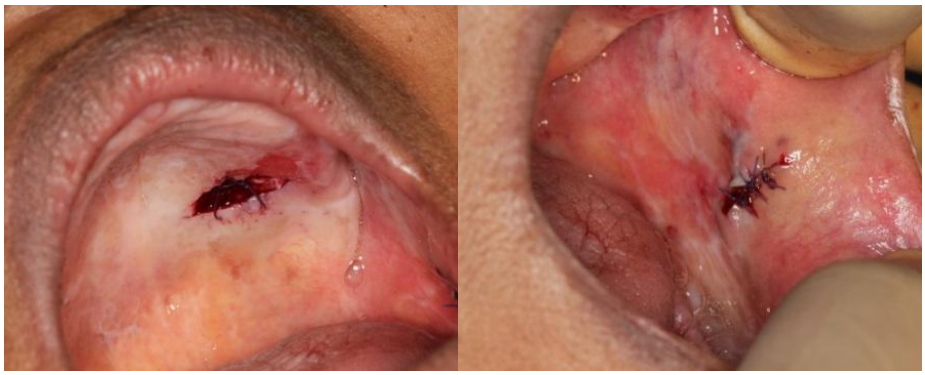

Figure 5. Two incisional biopsies were performed from the palate and buccal mucosa

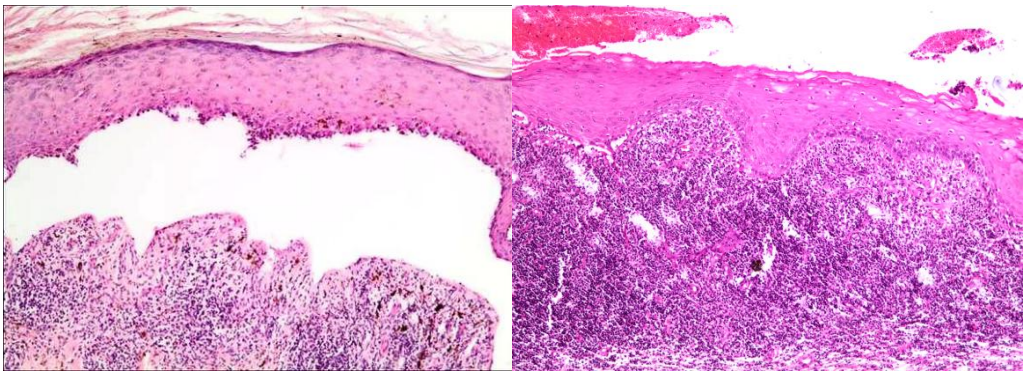

Figure 6. The histological features of the cutaneous lesions showing subepithelial cleft and dense lymphocytic infiltrate in the superficial connective tissue which is characteristic of lichen planus (left). Oral lichen planus showing licheanoid interface mucositis with dense infiltration of lymphocytes in the superficial connective tissue (right)

II. Discussion

OralLichen planus (OLP) is a chronic mucosal disease of middle aged or older females. The exact cause is unknown but an immune-mediated etiopathogenesis has been suggested [1,2].OLP can cause significant morbidity and in some instances, can transform into oral squamous cell carcinoma[3]. The diagnosis requires clinico-pathological correlation and management usually includes topical or systemic corticosteroids [4].

Etiopathogenesis

OLPis suggested to result from an autoimmune response to unknown antigen [5]. The disease is mediated by T-cell lymphocytic response that causes apoptosis and degeneration of the basal epithelial keratinocytes[6]. The death of basal keratinocytes is followed by detachment of the epithelium from the basement membrane, causing intercellular spaces to appear [6]. The basement membrane shows Immune deposits (mainly fibrin and seldomly IgM), while the remaining epithelium undergoes acanthosis and hyperparakeratosis leading to characteristic white lesions that are seen clinically. The symptomatic red lesions usually seen are the result of epithelial atrophy and erosion due to the chronicity of the inflammatory process[6,7].

Lichenoid lesions is a term used to describe lesions that are clinically and histologically similar to oral lichen planus but with a known aetiology[8]. This group includes heterogeneous causes such as graft-versus-host disease, liver diseases and infections, certain medications (e.g.Non-steroidal anti-inflammatory drugs, antihypertensives and antidiabetics) and some dental restorations[8,9].

Clinical features

OLP may occur in isolation or in association with other cutaneous lesions[10]. The clinical presentation is typically of white lesions that can adopt many forms such as reticular lines or striae, papules, plaques, atrophic and erosive lesions, erosion with ulcers and rarely bullous forms[9,11].The reticular form is the most commonly encountered variant and presents with minimal clinical symptoms while the bullous form is rarely seen in the oral cavity and is more associated with skin lesions[11,12]. The erosive form is usually ulcerative, painful and has been linked to the malignant change of the disease[13].Locations that are commonly involved include the buccal mucosa (usually symmetrical and bilateral), gingiva (mainly in a form of desquamative gingivitis) and the dorsum of the tongue. The palate, lips and floor of the mouth are less frequently affected[13].The course of the disease is often asymptomatic or mildly discomfort unless the lesions become atrophic or erosive. Symptoms are

particularly felt with eating or drinking acidic or spicy food. In some cases, the discomfort can be severe and may require management with systemic anti-inflammatory agents[13,14].

Unlike the skin lesions which frequently clear within 18 months, oral lesions in LP are often recalcitrant and can persist for many years[15]. The variant of lichen planus known as lichen planus pigmentosus; which is characterised by pigment incontinence and associates with long-standing lichen planus lesions, was demonstrated clearly in the skin and oral lesions of this case[16].

Extra-oral lesions when present, are usually purple, polygonal, pruritic and papular with a fine lacy white network of striae (Wickham's striae). Flexor surfaces of the wrists, lower back, ankles and shins are commonly affected [10,17]. Involvement of skin appendages may result in alopecia and nail changes[7,17]. Genital and ocular mucosa can be also affected resulting in ulceration and scarring. In most cases, genital involvement can be associated with oral lesions, particularly in the gingiva (vulvo-vaginal-gingival and peno-gingival syndrome)[17].

Diagnosis

Oral lichen planus can be clinically confused with systemic lupus erythematosus, chronic ulcerative stomatitis, keratosis, oral cancer and lichenoid lesions.The diagnosis of lichen planus requires clinicopathological correlation to exclude lesions with similar histological features, such as systemic lupus erythematosus, chronic ulcerative stomatitis, lichenoid drug reaction, lichenoid contact reaction and graft versus host disease. The immunofluorescence can also aid in the diagnosis by excluding some of these conditions[10,18].

Management

The management of OLP is mainly aimed towards relieving symptoms[7,19]. Predisposing factors such as dental fillings, medications or liver disorders should be always checked and corrected whenever possible. A referral to the relevant specialist is indicated when extraoral organs such as skin or genitalia are suspected[19]. Topical pain medication such as 2% Lidocaine gel and oral hygiene measures help alleviate the symptoms. Topical corticosteroids are usually the treatment of choice for controlling OLP. Management can include the use of medium potency agents such as Betamethasone valerate 0.1% cream for moderate involvement or higher potency agents such as Clobetasol propionate 0.05% cream for recalcitrant conditions[10,19]. Topical corticosteroids are usually applied in small amounts three times daily. Care must be taken to ensure maximum contact with the medication for at least half an hour. Systemic corticosteroids (e.g. Prednisolone 5 mg) are indicated in severe symptomatic cases with multiple site involvement. Therapies such as topical Cyclosporines, Vitamin A analogues are also available and regular follow up is usually recommended[19,20].

Malignant potential

OLP carries a small potential (3-10%) for malignant transformation[20]. The true frequency of this potential however, remains controversial. A part of this confusion is due to absence of prospective follow-up studies that are performed among OLP patients with a control group of patients who are free of the disease[21]. In addition to that, a consensus on the inclusion and exclusion criteria regarding malignant transformation of the disease is required. This may include the presence of certain risk factors for developing oral cancer (e.g. tobacco smoking) and the influence of immunosuppressive medication on the development of oral cancer[22].

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