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Pigmentation in Oral Lichen Planus: A Case Report

Farouq Fatima Ezzahra, Kissi Lamia, Sarfi Dounia, BenYahya Ihssane

Department of Oral Surgery, Université Hassan II, Casablanca, Morocco
Email: Farouqfaty74@gmail.com

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Abstract

Oral lichen planus (OLP) is a chronic inflammatory condition with diverse clinical presentations, the reticular form being the most common. A rare pigmented variant presents as dark brown macules resulting from melanocytic hyperactivity driven by chronic inflammation. Although benign, this post inflammatory pigmentation requires regular monitoring due to the malignant transformation risk associated with OLP. In this article, we report a rare case of pigmented oral lichen planus involving an unusual anatomical location.

Subject Areas

Public Health

Keywords

Oral Lichen Planus, Pigmented Variant, Post-Inflammatory Pigmentation, Case Report

1. Introduction

Lichen planus (LP) is a chronic inflammatory disease, derived from the Greek word "leichen" (tree moss) and the Latin "planus" (flat) [1] [2]. Described by Erasmus Wilson in 1869, LP affects the skin, scalp, nails, and mucous membranes, with rare malignant transformation [1] [2].

The oral variant, oral lichen planus (OLP), is a benign but chronic, non-infectious inflammatory disease that affects the stratified squamous epithelium of the oral mucosa, with characteristic flare-ups and remissions [1] [3].

The prevalence of OLP is estimated to be 2.2%. Oral lesions often precede cutaneous lesions and, in many cases, remain the only manifestation of the disease. OLP occurs in 70% to 77% of patients with cutaneous LP [4].

The female-to-male ratio is 2:1, and the onset age generally ranges between 30 and 60 years. However, cases of OLP have also been reported in children [1] [2].

OLP can affect all mucous membranes of the oral cavity. The most common location is the posterior buccal mucosa, followed by the lingual, gingival, and labial mucosa [5].

The exact and precise etiology of OLP remains unknown. However, the histological features, including basal membrane destruction and a band-like inflammatory infiltrate, strongly suggest an immune-mediated reaction involving T lymphocytes targeting basal membrane keratinocytes [3].

While OLP is typically not associated with melanin pigmentation, chronic inflammatory conditions can lead to melanin deposition in the connective tissue. This results in the appearance of multiple brown-black pigmented areas at the site of the initial lesion, a phenomenon known as post-inflammatory pigmentation [6].

This distinct variant is referred to as pigmented lichen planus and requires differential diagnosis from other pigmented oral conditions [6].

The aim of this article is to discuss the pigmentation of oral lichen planus through the analysis of a clinical case diagnosed in our department.

2. Clinical Observation

A 75-year-old hypertensive woman under treatment consulted the oral surgery department for pain on the lateral border of the tongue, which had appeared a week earlier. The extraoral examination was unremarkable, and the patient had a fair complexion.

The intraoral examination revealed two macules on the right lateral border of the tongue, approximately 1 cm apart. The first macule was rectangular, well-defined, pigmented in a mosaic pattern of white and dark brown, slightly raised, non-painful, and without induration on palpation. The second macule was similar but smaller in size with poorly defined borders.

Anterior to these lesions, small ulcerations on an erythematous base were observed, which were painful and bled on palpation. The patient also had a non-retentive and ill-fitting mandibular complete denture. The patient was scheduled for a biopsy after three days.

On the day of the procedure, she reported a remission of symptoms. Intraorally, the ulcerations had disappeared, while the other lesions persisted (**Figure 1**).

An incisional biopsy was performed, taking a 7 mm-long fragment from the first macule (**Figure 2**). The histopathological examination was consistent with oral lichen planus (OLP) (**Figure 3**).

The patient was advised to stop wearing her mandibular complete denture to establish a differential diagnosis between idiopathic OLP and a lichenoid lesion caused by chronic local trauma induced by the denture.

Follow-up at one week, 15 days, one month, and six months showed persistent absence of pain and stabilization of the observed lesions, confirming the diagnosis

of idiopathic pigmented oral lichen planus.

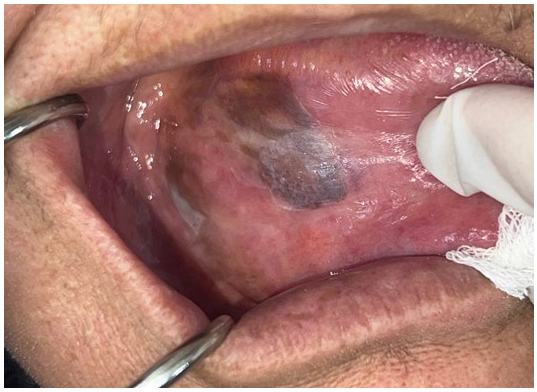

Figure 1. Intraoral view—Two macules on the right lateral tongue border, one rectangular, well-defined with a mosaic pattern of white and dark brown, slightly elevated and non-painful; the other smaller, with less defined borders.

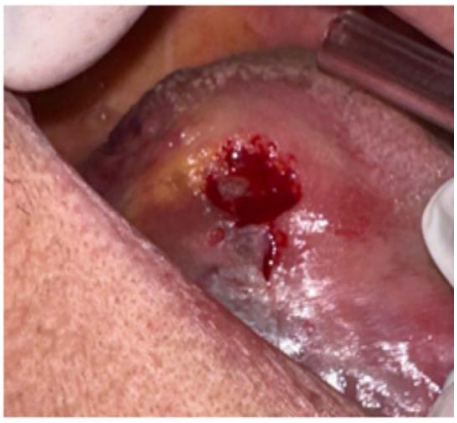

Figure 2. Incisional biopsy.

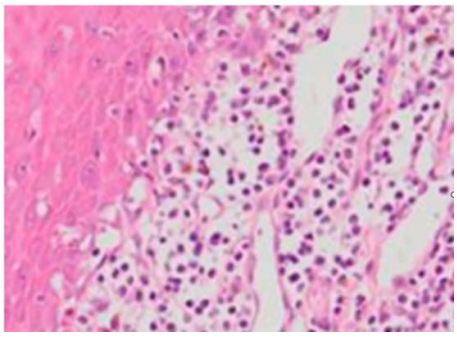

Figure 3. Scattered melaninin continence observed within the inflammatory cells.

3. Discussion

LP is well known as a chronic complex mucocutaneous disease of unknown etiology, uncertain pathogenesis, and varied histopathological features [2] [4].

Clinically, there are six clinical subtypes of OLP that can be seen individually or in combination: reticular, plaquelike, atrophic, erosive/ulcerative, papular and bullous [2] [4] [7].

Reticular form is the most common presentation, characterized by multiple whitish-gray papules forming a lace-like network known as Wickham's striae. This form is often asymptomatic and requires histological confirmation when the classic reticular pattern is absent.

Erosive/ulcerative forms present with inflammation-induced erythema, epithelial thinning, ulcerations, and pseudomembrane formation. The periphery of the lesions is often bordered by reticular keratotic striae. If the erosive subtype of OLP only affects the gingival tissue, the descriptive clinical term desquamative gingivitis is often used.

Plaque Form is Resembling leukoplakia, this form appears as homogeneous, slightly elevated, multifocal white lesions. It most commonly affects the buccal mucosa and tongue. Bullous and papular forms are rare in the oral mucosa. The bullous form is characterized by transient blisters, whereas the papular form presents as small, white, raised papules [2] [4] [7].

These subtypes may vary in severity and evolve over time, reflecting the chronic and relapsing nature of the disease. OLP may present with varying degrees of severity, ranging from asymptomatic or mild forms to moderate and severe manifestations. Periods of exacerbation often coincide with factors such as stress, anxiety, trauma, or exposure to chronic low-grade irritants, including dental plaque, tobacco use, dental restorative materials, or dietary triggers [8].

In our case, an unstable prosthesis was considered a potential source of chronic trauma to the lingual mucosa.

Among the various forms of OLP, there is a distinct pigmented variant known as lichen planus pigmented (LPP). First described in 1974 in India by Bhutani *et al.*, this clinical entity is characterized by a gradual onset of dark brown to black macules. Over time, these macules may merge to form large hyperpigmented patches. While LPP primarily affects the face, trunk, and upper limbs, its involvement of the oral mucosa remains rare [9]. When it does occur intraorally, this pigmented presentation is most frequently associated with the reticular form of OLP, as observed in our case [10].

The particular histological feature of this type is the increased production of melanin by the melanocytes and accumulation of melanin-laden macrophages in the subepithelial zone, associated with the typical characteristics of OLP, including a dense sub-epithelial band of inflammatory T cells, along with intra-epithelial lymphocytic cell infiltration and liquefaction degeneration/apoptosis in the basal cell layer to form colloid bodies [4] [11] [12].

Melanocytes in the oral mucosa were first described in 1927. Of neural crest origin, these cells reside in the basal layer of the stratified squamous epithelium. Their primary function is to synthesize melanin within specialized organelles called melanosomes and transfer it to adjacent keratinocytes, thereby contributing

to the normal pigmentation of the oral mucosa [11] [13] [14]. Under physiological conditions, especially in light-skinned individuals, melanocyte activity is relatively low and often insufficient to produce clinically visible pigmentation [11]. However, in chronic inflammatory conditions of the oral mucosa, such as OLP, melanocyte activity may become dysregulated or excessively stimulated, leading to increased pigmentation [13] [14].

Although the exact mechanisms underlying hyperpigmentation in OLP are not fully elucidated, two interdependent processes have been proposed. The first involves the direct stimulation of melanocytes by pro-inflammatory cytokines such as G-CSF, IL-12, IL-6, IL-1α, and TNF-α, which leads to an increased synthesis of melanin.

The second mechanism is pigmentary incontinence, resulting from damage to the basal epithelial layer. This allows melanin to leak into the underlying connective tissue, where it is subsequently phagocytosed by macrophages, forming melanophages—an established marker of postinflammatory pigmentation. Under normal conditions, melanocyte activity in the oral mucosa is minimal. However, chronic inflammation alters this balance, and when the clearance of melanin through lymphatic pathways is reduced, particularly in the context of therapeutic suppression of the inflammatory response, melanophages tend to persist in the tissue, contributing to long-lasting pigmentation [6] [11] [15]-[17].

This correlation is further supported by the findings of A. Patsakas *et al.*, who demonstrated that the number of melanophages in the gingival epithelium is directly associated with the severity of inflammation in the underlying connective tissue [18].

The differential diagnosis of oral LPP should be established with a group of hyperpigmentation disorders, such as ethnic pigmentation, considered a physiological variation in individuals with darker skin. Other conditions to consider, exogenous pigmentations like amalgam tattoos and smoker's melanosis, drug-induced pigmentations observed notably with treatments like minocycline or synthetic antimalarials**.** Genetic conditions, systemic diseases like Addison's disease, and tumor-related pigmentation, especially oral mucosal melanoma, should also be considered [8] [11] [12] [19] [20].

The treatment of oral LPP follows the same therapeutic principles as classic oral OLP, with the primary goals of reducing inflammation and relieving symptoms. The choice of treatment depends on the presence or absence of symptoms. Asymptomatic lesions, as was the case for our patient, generally do not require active treatment but should be subject to regular clinical monitoring. Follow-up is recommended every two months initially and can be extended to once a year, with additional biopsies performed if any suspicious clinical changes are observed [1] [2] [5].

However, for symptomatic lesions, local or systemic pharmacological treatment is indicated. Corticosteroid therapy remains the first-line treatment: topical corticosteroids, available as creams, gels, mouthwashes, or sublesional injections, show

good efficacy, although they may promote the development of oral candidiasis. For gingival lesions, which are often resistant to topical treatments, the use of a custom-made tray can help increase the contact time between the drug and the mucosa. Furthermore, other therapeutic options such as topical calcineurin inhibitors (tacrolimus, cyclosporine), oral retinoids, or laser biomodulation have also been explored for the management of oral lichen planus [1] [2] [5].

The incidence of malignant transformation of OLP is estimated to range between 0.4% and 5.3% according to various studies [2]. Pigmented OLP may reflect more intense or prolonged inflammatory activity, which could promote basal layer damage and increased cellular turnover, two mechanisms implicated in carcinogenesis [21] [22]. However, current data do not support the conclusion that pigmentation alone constitutes an independent risk factor for malignancy in OLP. Further studies are needed to determine whether pigmented forms of OLP carry a higher potential for malignant transformation compared to non-pigmented forms [10] [17].

4. Conclusions

Pigmented oral lichen planus is a rare variant of OLP, with pigmentation resulting from chronic inflammation and complex immune mechanisms.

The reported case highlights the clinical diversity of this entity and the importance of regular follow-up, even in asymptomatic patients.

A better understanding of its pathophysiology could help improve the management of these atypical presentations.

Conflicts of Interest

The authors declare no conflicts of interest.

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