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# Oral Medicine and Pathology Quiz – Case 6

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A 54-year-old female was referred by her dentist for evaluation of gingival and tongue lesions, causing tenderness and dysphagia. The lesions were first noticed before 5 months and considered to be due to local periodontal inflammation. The patient underwent periodontal treatment and used an over-the-counter oral coating agent without any improvement. The patient was in good health and was not a smoker.

On clinical examination, erythematous, atrophic and erosive areas were noticed on the anterior labial gingiva of both maxilla and mandible. The gingival lesions were bordered by fine, white radiating striae and were painful on palpation. Nikolsky sign was negative. The clinical pattern could be described as desquamative gingivitis (fig. 1). Furthermore, interlacing white lines were noticed on the buccal mucosa bilaterally. On the dorsal surface of the tongue, multiple ulcerations along with non-removable white plaques were seen (fig. 2). No other oral or skin lesions were noticed. Partial biopsy of the tongue lesions was performed and the splitted specimen was submitted for histopathologic examination (in formalin solution) and for direct immunofluorescence (in normal saline solution). Microscopic examination revealed degeneration of the basal epithelial cell layer and intense band-like lymphocytic infiltrate of the underlying connective tissue (fig. 3). The direct immunofluorescence demonstrated only the presence of fibrinogen in the basement membrane zone. Based on the diagnosis rendered, initial administration of systemic corticosteroids resulted in significant improvement. Intralesional corticosteroid injection provided complete healing of the persistent tongue lesions. Application of topical corticosteroid cream within fabricated splints, along with meticulous oral hygiene, allowed good control of the gingival lesions. The patient is on periodic follow-up, occasionally using topical corticosteroids to control disease exacerbations, and remains free of symptoms.

## Comment

*Lichen planus (LP) is a relatively common, chronic mucocutaneous disease that often affects the oral mucosa. It is believed to be an immunologically mediated disorder; relationship with stress or anxiety has been suggested but remains unproven. The disease is most often noticed in middle-aged adults, with a 3:2 female predominance.*

*It is estimated that the prevalence of cutaneous LP is around 1%. These skin lesions have the clinical pattern of purple polygonal papules, along with a lacelike network of fine, white lines (Wickham's striae). The oral counterpart is called oral lichen planus (OLP) and affects 0.1–2.2% of the population. OLP most commonly manifests with the typical asymptomatic reticular form of interlacing white lines bilaterally distributed usually on the buccal mucosa. Nevertheless, any oral mucosal site may be affected. On the other hand, the erosive/ulcerative form of OLP is frequently associated with pain, tenderness and dysphagia and may occur anywhere on the oral mucosa. Involvement of the gingiva by erosive/ulcerative*

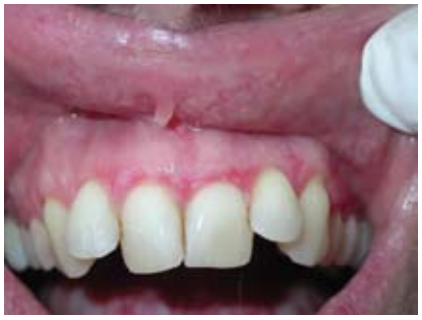

**Figure 1**

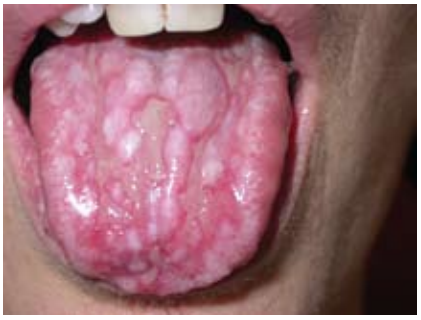

**Figure 2**

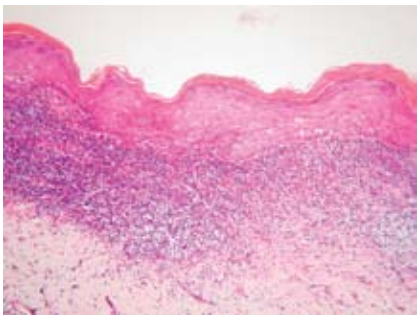

**Figure 3**

*or atrophic OLP may present as desquamative gingivitis (a clinical appearance also seen in other immunologically-mediated diseases, such as pemphigoid and pemphigus). Other forms of OLP include the hypertrophic –characterized by white plaques that cannot be removed– and the rare bullous and pigmented variants.*

*OLP diagnosis is usually based on the medical history of the patient, the clinical appearance and often requires histologic examination of perilesional tissue and direct immunofluorescence. The diagnosis of the typical bilateral reticular form of OLP is usually based on clinical signs alone. Chronic ulcerative stomatitis, lupus erythematosus, graft-versus-host disease and lichenoid allergic reactions may mimic OLP lesions. The hypertrophic form is often misdiagnosed clinically as leukoplakia, traumatic hyperkeratosis or other conditions presenting as non-wipeable white plaques. The differential diagnosis of erosive/ulcerative and atrophic OLP forms may also include mucous membrane pemphigoid, pemphigus, and other ulcerative diseases.*

*Topical (e.g. amalgam) and systemic (e.g. medication-induced) allergic reactions may present as lichenoid lesions. A complete history that will reveal a temporal and spatial relationship between the potential cause and the onset of the oral lesions will point to the correct diagnosis. Further, discontinuation of the suspected causative agent should result in rapid disappearance of the lesions.*

*Histopathologic examination of OLP lesions reveals hyperkeratosis, degeneration of the basal epithelial layer, intense band-like lymphocytic infiltrate of the connective tissue and saw-toothed rete ridges. Perivascular lymphocytic infiltration is not typically present, as opposed to lupus erythematosus and lichenoid reactions. Direct immunofluorescence demonstrates the presence of fibrinogen in the basement membrane zone in 90–100% of the cases.*

*Asymptomatic reticular OLP requires no treatment. Symptomatic erosive/ulcerative forms are often managed by topical application of potent corticosteroids several times per day. Extensive or persistent lesions require systemic corticosteroid treatment and/or intralesional corticosteroid injections. The aim of the clinician is to achieve long-term control of disease activity and symptomatology by using the minimum necessary dosage of topical corticosteroids. In case of superimposed or iatrogenic candidiasis, antifungal drugs should be used. The efficacy of immunotropic agents is contradictory and their use is limited to severe cases, non-responsive to steroid therapy.*

*It is believed that OLP, especially the erosive form, has a limited malignant potential. Of course, both OLP and oral squamous cell carcinoma are not rare and independent development cannot be easily ruled out. Moreover, there are no strict clinical and histopathologic criteria for the definition of OLP malignant transformation. It may be assumed that malignant transformation of OLP is quite rare. Nevertheless, these patients should be monitored on a tactic base for early detection of malignant changes as well as for maintenance of the state of remission with appropriate adjustments in therapy.*

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