

## Case Report

# Interest of periodontal maintenance in improving autoimmune diseases: about an oral lichen planus case report

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**Abstract – Introduction:** Oral lichen planus is an autoimmune inflammatory disease. These clinical manifestations can sometimes be confused and cumulated with clinical signs of periodontal disorders. The aim of this work is to illustrate the interest of a periodontal follow-up in the management of oral lichen planus by a clinical case report. **Observation:** A patient came for a consultation in dermatology for a persistent gingival erythema. The diagnosis of oral lichen planus was made and treatments were successively implemented. After six months of follow-up, an inflammatory gum was still present without showing improvement. A stage 3, grade B periodontitis associated with oral lichen planus was diagnosed and a periodontal treatment was initiated, combined with corticosteroids treatment as mouthwash. Four months after the beginning of this treatment, a marked decrease in gingival erythema was observed. **Comments:** There is often a close relationship between oral lichen planus and periodontal disease therefore a multidisciplinary approach can be very useful for optimal management.

## Introduction

Oral Lichen Planus (OLP) is an autoimmune inflammatory disease affecting 1–2% of the population, the mechanisms of which are still poorly understood. Yet this pathology is clinically and histologically well-known [1]. The Lichen Planus can take many forms: reticulated, flat, patchy, erythematous, erosive, then atrophic. In the oral sphere, one of the pathognomonic manifestations of OLP is the appearance of Wickham's striae [2]. Lichen Planus can also appear on other mucous surfaces and the jugal mucosa, the back of the tongue, and the gums are often affected [3]. On the gum, OLP can take the form of desquamative gingivitis [2]. The erosive manifestations often present a painful clinical picture for the patient [4]. Histologically and molecularly speaking, OLP is defined at the epithelial level by hyperkeratosis and degeneration of basal cells and subepithelial inflammation dominated by lymphocytes and macrophages [5,6]. Simultaneously, periodontal pathologies are inflammatory diseases of bacterial origin. They are defined by the destruction of the collagen structure defining the periodontium with repercussions on the gingival and bone structures [6]. The clinical manifestations of gingival tropism OLP and periodontal pathology are often combined but are

opposed in the differential diagnosis and histology. Indeed, clinically speaking, the lesions due to periodontal diseases are always in contact with the tooth necks, more or less extensive. By contrast, lesions related to OLP can be located at distance from the tooth necks without being related to the dental organ [3]. However, they both involve inflammatory mechanisms at the same sites. It is then possible that they may be both present in the same patient and interact with each other.

The article aims to illustrate, through a clinical case, the interest of periodontal maintenance in the improvement of the management of patients presenting an OLP and to compare with a literary review.

## Observation

A 62-years-old patient was referred by his dental surgeon to the Dermatology Department for a pathology of the oral mucosa showing persistent gingival erythema despite scaling treatment.

Clinical examination showed an erythematous and inflammatory aspect of the gum near and away from the dental collar, but also spontaneous and induced bleeding leading to a decrease in brushing and an increase in residual dental biofilm and tartar (Fig. 1). An OLP was diagnosed, associated with a nail lichen (Fig. 2).

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**Fig. 1.** Erythematous and inflammatory aspects of the gum.



**Fig. 2.** Lichen planus aspect of the hand nails.

The patient reported a history of prostate cancer, an episode of stroke three years ago, and thrombocythemia currently being treated with hydroxycarbamide.

At the first Dermatology consultation and during the follow-up, multiple treatments were implemented:

- Treatment with orodispersible prednisolone 20 mg mouthwash (one tablet morning and evening) was initially prescribed for four months without any significant improvement of the OLP.
- A relay by topical application of clobetasol-based cream was then initiated. This treatment considerably reduced gingival inflammation. However, after two months of application, erythematous gingival hypertrophy was still present. The erythematous and erosive appearance in the left mandibular sector, coupled with the symptomatic OLP, led to the need for a biopsy to rule out any malignant transformation. Histopathological examination revealed a predominantly lymphocytic subepithelial infiltrate without dysplasia or tumour infiltration was found. This is not specific but strongly suggestive of lichen planus.

A thorough clinical examination of the mouth, radiographic periodontal status (Fig. 3) and periodontal charting (Fig. 4) led to the diagnosis of stage 3 grade B periodontitis associated with OLP [7], due to the progression of bone lysis and the age of the patient. It was then decided to associate a periodontal treatment with the multidisciplinary follow-up in dermatology department. Periodontal management consisted in an initial motivation for oral hygiene which reduced gingival inflammation and spontaneous bleeding (Fig. 5). Then scaling followed by root planning in the full mouth was undertaken with antibiotic therapy, Amoxicilline 1g morning and evening for seven days.

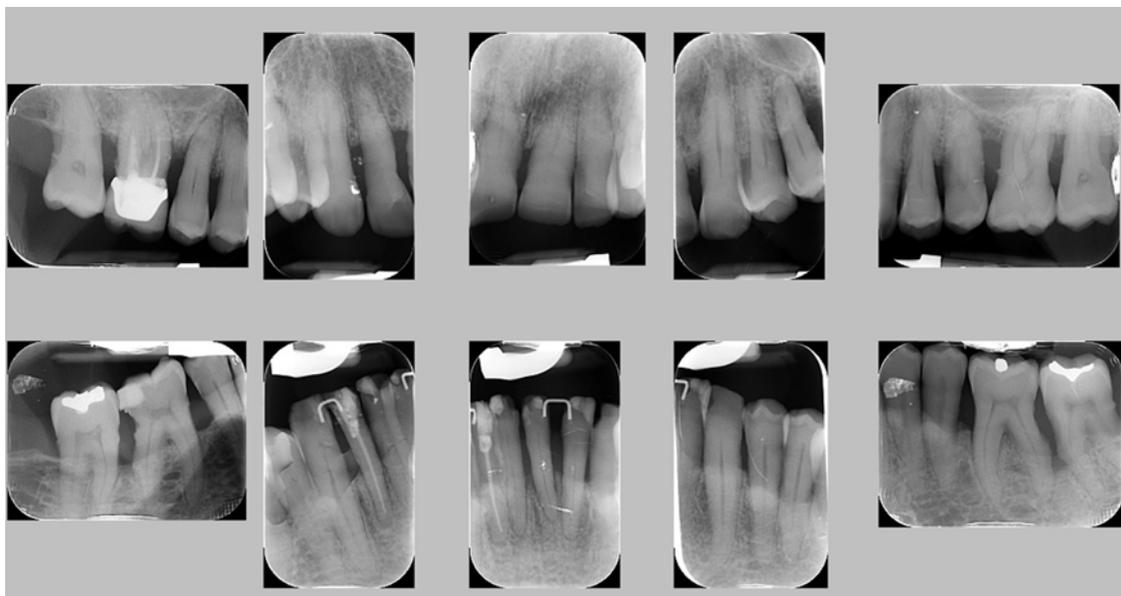
Persistence of gingival erythematous area away from the tooth collar revealed lesions due to OLP. Treatment with betamethasone 2 mg, two tablets morning and evening daily mouthwash was initiated with controls at one (Fig. 6) and four months (Fig. 7).

At this date, a marked decrease in the inflammatory zones away from the tooth collar was observed. However, these did not completely disappear, possibly in relation with the persistence of tartar on the mandibular front teeth (Fig. 7).

A bi-annual check-up was considered as necessary to monitor the patient's motivation in oral hygiene, to follow up the evolution of OLP, and to interfere early if any change in OLP.

## Discussion

Recently, a new classification of periodontal disease has emerged in which inflammatory and autoimmune diseases (including OLP) are recognized as responsible for gum disease not induced by dental biofilm [7]. A study showed that good control of dental biofilm helps to reduce the OLP symptoms and improves the overall quality of patient's life [8]. More recently, Gracia-Pola *et al.* showed through a systematic review of the literature that rigorous oral hygiene education at home and regular periodontal maintenance check-ups significantly reduced the clinical signs and discomfort caused by OLP [9]. This conclusion was also reported by Giovanni *et al.* in a recent randomised case control study [10]. In this clinical case, the patient's oral hygiene is greatly improved even if still perfectible and improvement in clinical signs of OLP are evident. OLP has an autoimmune component, leading to an inflammatory phenomenon. Although Katz *et al.* suggested through a clinical case, that the appearance of OLP lesions was secondary to local aggression [11], its aetiology remains unknown. OLP has often been described and studied in the literature in association with other infectious diseases. Heinz Herpenstein *et al.* described in a case report the reduction of inflammation, stabilization of periodontal disease and the complete interruption of evolution of OLP without specific therapy [12], permitted by a periodontal treatment allowing a decrease of dental biofilm and its constant control.



**Fig. 3.** Periodontal radiographic status revealing alveolar bone loss.

Hepatic and herpes infections have been shown to cause a local immune deficiency that potentiates lichen lesions [13,14]. Yildirim and Nagarro go even further in explaining that these infections may play a role in the OLP aetiology [13,14]. In the context of periodontal pathologies, the bacterial origin is well-known [12]. Ertugrul *et al.* showed through a clinical study, that periodontopathogen bacteria were found in greater quantities in patients with OLP associated with periodontal disease than in patients with periodontal disease only. They conclude that there is a real correlation between these two pathologies. They also theorized that periodontopathogen bacteria play a role in the aetiology of OLP itself [15]. It is clear from these various studies that there is an interaction between autoimmune disease and periodontal microbiota. Some authors even assert the hypothesis that the OLP appearance is secondary to a first pathological manifestation, which implies that initial management of the infectious pathology should be a prerequisite for any treatment of OLP.

This reported clinical case is in concordance with literature. Indeed, multidisciplinary management (periodontology/oral dermatology) was essential and the order in which the treatments were carried out seemed to be important. The patient initially received treatment only focusing on oral lichen planus without prior treatment or maintenance check-ups of periodontal disease (during the treatment). Despite the use of multiple medical treatments, no improvement could be observed. This may be firstly explained by the use of local corticosteroids to treat the OLP, but resulting in a decrease in local immune defences that may favour opportunistic infections [1]. Secondly, this patient's periodontal disease probably played an aggravating role in the aetiology of this

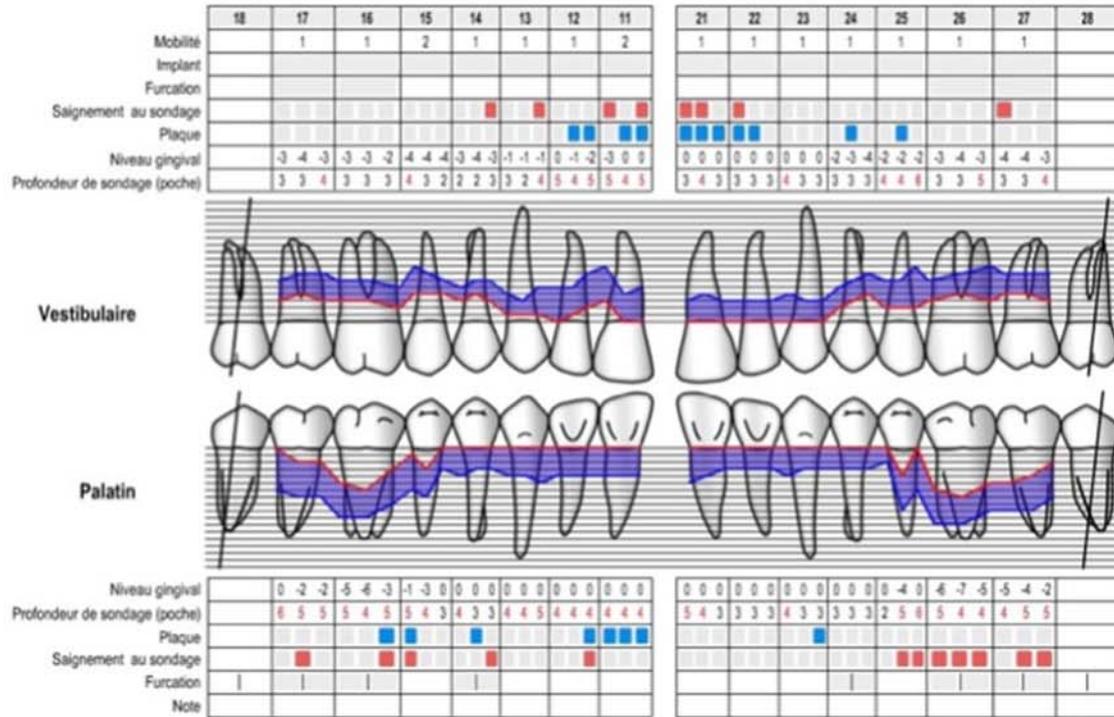
OLP. This hypothesis is supported by clinical observations: when periodontal disease management preceded the management of OLP, the patient responded positively to treatment. One study focused on the interaction between OLP and periodontal disease at the molecular level. Indeed, Ertugrul *et al.* showed that active forms of OLP lead to an increase in MMP1 and MMP9 and a decrease in TIMP (protease inhibitor), causing destruction of the extracellular matrix, weakening the protective effect of the surface epithelium and thus promoting bacterial colonization and the development or maintenance of periodontal disease [16]. More recently Romano *et al.* has shown that plaque control leads to a decrease in MMP1 and MMP9, however, patients with active OLP seem to maintain a higher concentration of MMP than the general population even when hygiene control is rigorous [17].

## Conclusion

In conclusion, the link between OLP and periodontal disease no longer needs to be proven today; it is obvious. This clinical observation supported by data from the literature highlights interactions between OLP and periodontal disease. It is therefore not possible to properly treat OLP without managing an existing periodontal disease. A multidisciplinary approach between oral mucosa pathologists and periodontologists appears essential for the patients well-being. Furthermore, it is essential for patients to become aware of the fact that treatment success relies on their adherence to the rules of oral hygiene and that this success can be reversible if the rules are not followed. These patients will need to be followed throughout their lives to limit recurrences, bearing in mind that the main risk of OLP is the evolution of a malignant lesion.

# État initial

## Maxillaire



## Mandibulaire

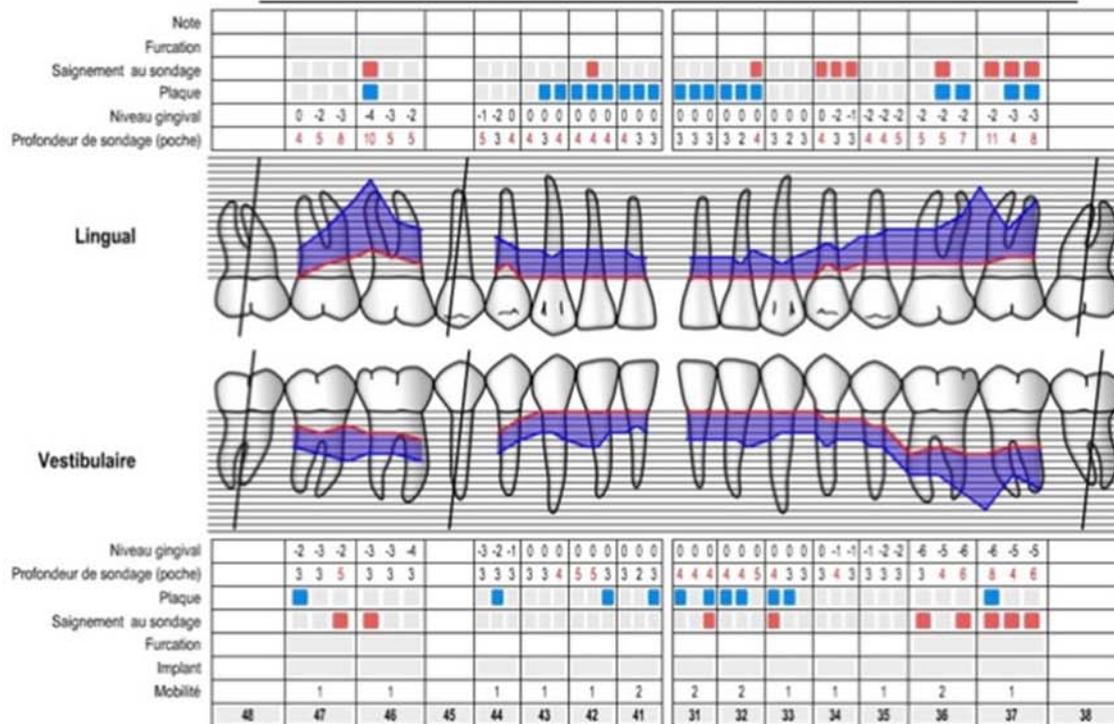


Fig. 4. Initial periodontal charting..



**Fig. 5.** Gingival aspect after the initial periodontal management.



**Fig. 6.** Gingival aspect one month after betamethasone treatment.



**Fig. 7.** Gingival aspect four months after betamethasone treatment.

### Conflict of interest

The authors declare that there is no conflict of interest.

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### Ethical approval

Ethical approval was not required.

### Informed consent

This article does not contain any studies involving human subjects.

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