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Olgu Sunumu

Oral Lichen Planus After Treatment with Ixekizumab for Plaque Psoriasis: A Case Report

Plak Psöriasis için İxekizumab Tedavisi Sonrası Oral Liken Planus: Olgu Sunumu

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ABSTRACT

Recently, the use of biological agent treatments for psoriasis has increased. Ixekizumab (anti-IL 17A) is a biological agent used for the treatment of moderate to severe plaque psoriasis. Although ixekizumab is generally well tolerated, various side effects may occur after the use of this medication. Herein, we present a case of oral lichen planus possibly induced by ixekizumab in a 46-year-old woman with psoriasis. Clinicians who use ixekizumab in their patients should be careful about this side effect and follow up patients in terms of mucocutaneous liken planus.

Keywords: Biologic Agents, IL-17 İnhibitors, Lichen Planus, Psoriasis, Ixekizumab.

ÖZET

Son yıllarda psoriaziste biyolojik ajan tedavilerinin kullanımı artmaktadır. İxekizumab (anti-IL 17A), orta ila şiddetli plak psoriazisinin tedavisinde kullanılan bir biyolojik ajandır. İxekizumab genellikle iyi tolere edilse de, bu ilacın kullanımından sonra çeşitli yan etkiler görülebilir. Burada, 46 yaşında psoriazisli bir kadında ixekizumab ile indüklenen oral liken planus vakasını sunuyoruz. Hastalarında ixekizumab kullanan klinisyenler bu yan etki konusunda dikkatli olmalı ve hastaları mukokutanöz liken planus açısından takip etmelidir.

Anahtar kelimeler: Biyolojik Ajanlar, IL-17 İnhibitörleri, Liken Planus, Psöriasis, İxekizumab.

Ixekizumab is an IgG4 type humanized monoclonal antibody that specifically binds and inhibits interleukin 17A in inflammatory diseases such as psoriasis and psoriatic arthritis (1). As the use of biological agent treatments for psöriasis becomes more widespread, reports of side effects associated with these drugs are also increasing. Here in, we report the case of a 46-year-old woman who obtained complete skin clearance of psoriasis plaques after 8 months of ixekizumab treatment together with the appearance of lichen planus (LP) lesions localized in the oral mucosa.

CASE REPORT

A 46-year-old female patient, who was receiving ixekizumab treatment for psoriasis, admitted to our clinic due to newly developed lesions on her oral mucosa in the 8th month of ixekizumab treatment. When physical examination revealed a white plaque with an ulcerated center on the tongue and reticular structures on the buccal mucosa (Figur 1 and 2), it was decided to take a biopsy from the patient with the preliminary diagnosis of oral LP. The biopsy result was consistent with oral LP (Figur 3).

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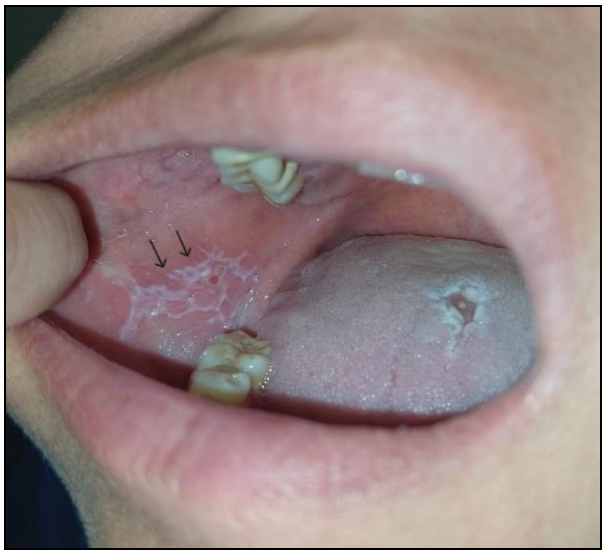
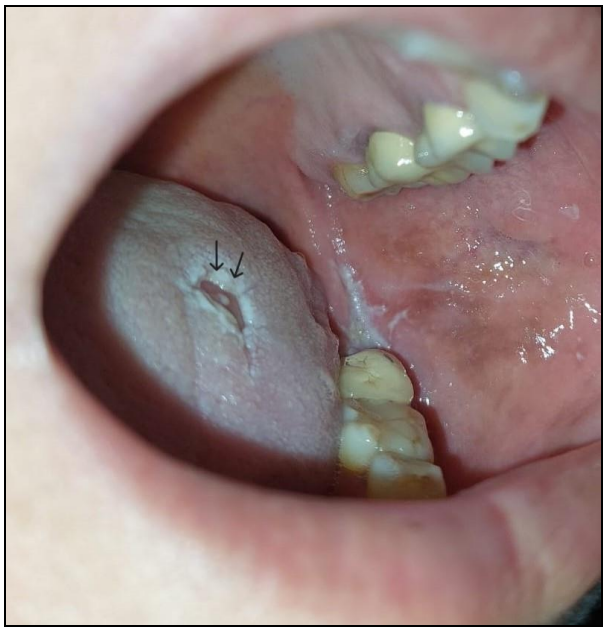
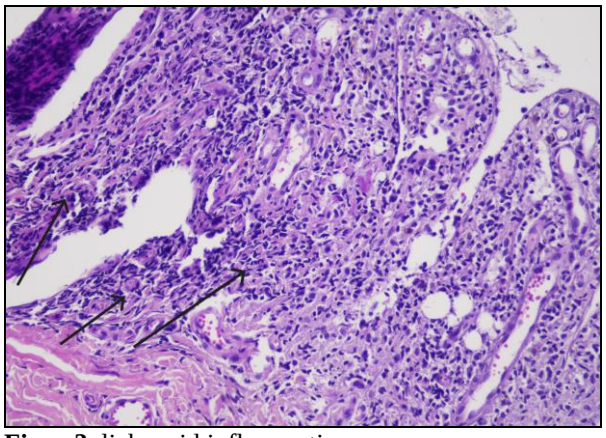


Figur 1. Reticular structures on the buccal mucosa.



Figur 2. a white plaque with an ulcerated center on the tongue.



Figur 3. lichenoid inflammation.

There was no abnormality in the patient's laboratory parameters. She had no other known disease and no history of drug use other than ixekizumab.

Considering all these, it was thought that developing oral LP may be a side effect of ixekizumab.

The patient had a PASI100 response. It was decided to stop ixekizumab treatment and continue treatment with acitretin. In addition, topical corticosteroid treatment was given for oral lichen planus. The patient was followed for approximately 1 year with acitretin. At the end of 1 year, as the patient's psoriasis lesions increased again, acitretin was stopped and methotrexate treatment was started. No progression was observed in the patient's oral LP lesions during this period.

Since there was no sufficient regression in the patient's psoriasis lesions after 2 months of methotrexate use, guselkumab was added to the treatment and then methotrexate was slowly stopped and treatment with guselkumab was continued.

In the 3rd month of guselkumab treatment, the patient stated that the feeling of pain and soreness, which occured from time to time in the oral mucosa, had recently increased and was making her very uncomfortable, especially while eating. During the physical examination, it was observed that the lesions in the buccal mucosa had progressed to some extent.

According to the anamnesis, it was learned that the patient had not received any treatment for lichen planus for a long time. Intralesional corticosteroid treatment was applied to the lesions and follow-up planned 1 month later. During the control, it was observed that the patient's complaints had subsided and her lesions had regressed. One more dose of intralesional corticosteroid was administered and follow-up planned 1 month later.

DISCUSSION

Ixekizumab is a humanized monoclonal immunoglobulin G (IgG) 4 antibody generally tolerated well. Common side effects observed in patients using ixekizumab include: neutropenia, injection site reaction, hypersensitivity reactions. Also, fungal infections, nausea, thrombocytopenia are seen, and rarely Crohn's disease and ulcerative colitis are observed (2-3). In our case, the patient had no previous symptoms, no other medication use other than ixekizumab or no known disease, so we conclude that she developed LP potentially caused by ixekizumab. There are cases of oral lichen planus in the literature that occurred as a result of the use of secukinumab, another IL-17 inhibitor, but to our knowledge, this is the first case of oral lichen planus that developed with the use of ixekizumab (4-5).

We think that the exacerbation of LP lesions during the patient's use of guselkumab, an IL-23 inhibitor, may be due to the patient's lack of treatment at that time rather than a possible side effect of guselkumab. Because, a significant regression was observed in the lesions after

intralesional steroid treatment administered without discontinuing the patient's guselkumab treatment.

Lichen planus (LP) is a chronic inflammatory disease characterized by violaceous papules and plaques. LP, which affetcs both skin and oral mucousa, is more commonly seen in women between the ages of 30 and 60. The prevalence of oral lesions varies between 0.1 and 2.2%. While lichen lesions on the skin respond well to treatment and tend to be self-limiting, oral mucosal lesions are chronic and often exhibit multiple remissions and exacebrations. Clinical types of oral LP include plaque-like, atrophic, papular, erosive and bullous type. Whickam's lines are a fine, asymptomatic, interwoven, lace- like pattern seen in the reticular type. The erosive type is painful due to erosions and rarely carries the risk of developing SCC. The etiology of oral LP has not been fully explained, and existing studies in the literature highlight its immunopathogenic basis with enhanced cytokine regulation (6-7).

It is known that biological treatments reduce the formation of pro-inflammatory cytokines by inhibiting the expression and differentiation of cytokines such as Th1 and Th17, which are involved in the pathogenesis of psoriasis (8).

However, IL-17 plays a critical role in mucocutaneous immunity by inducing antimicrobial peptides and chemokines that protect against fungal and bacterial infections. Inhibition of IL-17 may lead to reactivation of latent pathogens or new infections, triggering a T-cell-mediated immune response, particularly involving CD8+ T cells and dendritic cells. In patients with psoriasis, regulatory T cell (Treg) dysfunction is common; these dysfunctional Tregs, especially those producing IL-17, fail to suppress immune responses adequately, contributing to autoimmunity. This dysregulation, combined with IL-17 inhibition, may result in excessive activation of cytotoxic T cells and dendritic cells, provoking a lichenoid inflammatory response (9-10). Based on these pathogenic mechanisms, we believe oral lichen planus in our case may have developed through a similar immunological process. Future studies are warranted to help deepen this issue.

Nowadays, biological agents are being used with increasing frequency in psöriasis. Although the selectivity of biological agents has increased, new adverse reactions may still occur. Clinicians who use ixekizumab in their patients should be careful about this side effect and follow up patients in terms of cutaneous-mucosal LP.

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