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Case Report

Successful Treatment of Refractory Oral Lichen Planus Using Upadacitinib

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Abstract

Oral lichen planus is a chronic, immune-mediated disorder marked by T-cell-driven inflammation targeting basal keratinocytes. Conventional treatments, including topical corticosteroids and calcineurin inhibitors, often provide limited or short-term relief and carry potential side effects. Janus kinase inhibitors, particularly selective Janus kinase 1 inhibitors like upadacitinib, offer a targeted therapeutic alternative. We report a 62-year-old male with biopsy-confirmed, treatment-refractory oral lichen planus who demonstrated marked clinical improvement after initiating upadacitinib 15 mg daily. Previous therapies, including clobetasol, tacrolimus, and hydroxychloroquine, yielded minimal benefit. Within two weeks of treatment, the patient reported reduced oral pain, erythema, edema, and bleeding, significantly improving his ability to eat and speak. No side effects have been observed to date, 11 months into therapy. Upadacitinib inhibits Janus kinase 1-mediated cytokine signaling pathways involved in oral lichen planus pathogenesis, including Interleukin 2, Interleukin-6, Interleukin-21, and Interferon-γ. Its oral administration, rapid onset, and focused immunomodulation make it a promising alternative to traditional immunosuppressants. This case supports further investigation of upadacitinib as a potential addition to the therapeutic armamentarium for refractory oral lichen planus. Larger studies are warranted to confirm its safety and efficacy.

Keywords: Oral lichen planus; Upadacitinib; JAK

Abbreviations

OLP: Oral Lichen Planus; JAK: Janus Kinase; JAK-STAT: Janus Kinase-Signal Transducer and Activator of Transcription; STAT: Signal Transducer and Activator of Transcription; IL: Interleukin; IFN: Interferon

Introduction

Oral Lichen Planus (OLP) is a chronic inflammatory condition that affects the mucous membranes, primarily the buccal mucosa, tongue, and gingiva. Its pathogenesis involves a T-cell-mediated immune response that destroys basal keratinocytes, leading to erythema, white striations, and painful erosions or ulcers. While its exact etiology remains unclear, genetic predisposition, stress, and autoimmune mechanisms are believed to contribute to the disease [1].

Current treatments for OLP are limited and focus on symptomatic relief and immunosuppression, typically using corticosteroids and calcineurin inhibitors [2]. However, these treatments often have side effects and may not provide sustained disease control. As a result, targeted therapies, such as Janus Kinase (JAK) inhibitors, have garnered interest due to their efficacy in autoimmune and inflammatory diseases [3].

Upadacitinib, a selective JAK1 inhibitor, has recently emerged as a promising treatment for inflammatory diseases like rheumatoid arthritis, atopic dermatitis, and ulcerative colitis [4]. However, its role in OLP remains largely unexplored. This case report presents the use of upadacitinib in a patient with refractory OLP, focusing on its safety profile and therapeutic response.

Case Presentation

A 62-year-old male with a history of and biopsy-proven OLP presented to our dermatology clinic seeking treatment for uncontrolled OLP. He was intermittently suffering from painful, swollen, and bleeding oral mucosa for several years. His symptoms were further exacerbated by consuming spicy, hard, or sharp foods and brushing his teeth, which led to oral discomfort and dietary restrictions. He had previously attempted various treatments for OLP, including standard therapies such as clobetasol and tacrolimus for years, and hydroxychloroquine for months, without significant relief.

During his initial dermatologic consultation and examination, mucosal erythema, edema, and Wickham striae were observed (Figure 1 and 2). The patient was started on apremilast 30 mg by mouth daily, which initially improved symptoms; however, treatment was discontinued due to insurance limitations. He was subsequently transitioned back to topical treatments, including clobetasol 0.05%

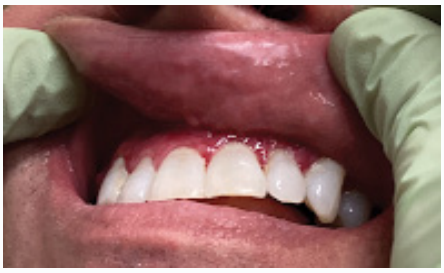

Figure 1: Inflamed, edematous, and friable gingiva of the maxillary alveolar ridge and upper mucosal lip while treating with clobetasol and tacrolimus.

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gel applied twice daily for one week, alternating with tacrolimus 0.1% ointment applied twice daily for one week. Although these topicals alleviated symptoms temporarily, the effect was not sustained, and symptoms returned.

Given the limited success with prior therapies and the patient's ongoing symptoms, treatment with upadacitinib 15 mg by mouth daily was initiated. After two months on upadacitinib, the patient reported significant improvement. He experienced reduced pain, bleeding, redness, and swelling of the oral mucosa (Figure 3a and 3b). These improvements allowed for greater comfort while eating and speaking, markedly enhancing his quality of life. At the three-month follow-up, complete resolution of mucosal inflammation was observed with normalization of oral mucosa and disappearance of erythema, edema and Wickam striae (Figure 4).

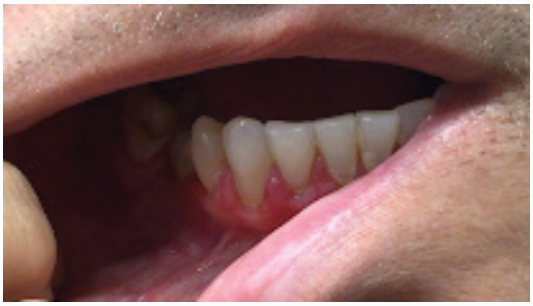

Figure 2: Wickham striae of the buccal mucosa.

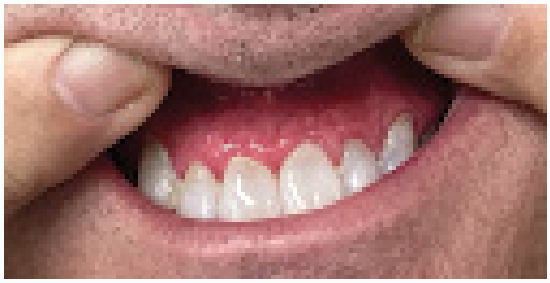

Figure 3a: Two months after initiating treatment, clinical improvement in maxillary alveolar ridge gingival inflammation.

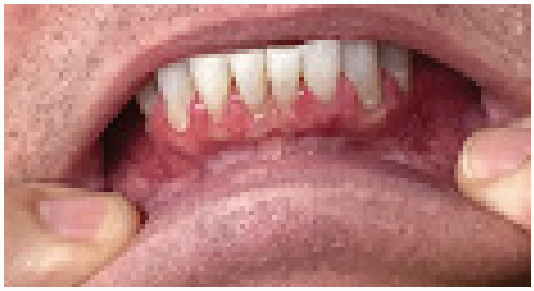

Figure 3b: Evident reduction in gingival inflammation of the mandibular alveolar ridge two months after starting treatment.

Discussion

Upadacitinib selectively targets JAK1 in the Janus Kinase-Signal Transducer and Activator of Transcription pathway (JAK-STAT), inhibiting the production of key pro-inflammatory cytokines (IL-2, IL-4, IL-6, IL-21, and IFN-γ), which drive T-cell-mediated inflammation in OLP [3]. Elevated levels of IFN-γ, IL-21, and STAT1 in lichen planus lesions further support this mechanism [5]. This targeted approach makes upadacitinib an attractive option for conditions like OLP, where immune dysregulation and chronic inflammation are central to the disease pathogenesis.

Unlike traditional treatments, upadacitinib's oral formulation and selective JAK1 inhibition may reduce systemic side effects commonly associated with chronic topical or systemic corticosteroids and other immunosuppressive therapies [6]. Additionally, upadacitinib's rapid onset of action, often yielding improvements within weeks, offers timely symptom relief for patients experiencing the pain and discomfort associated with OLP lesions [7].

However, the use of upadacitinib in OLP is not without risks. Although generally well-tolerated, JAK inhibitors carry potential risks for serious adverse events, including infections, thrombosis, and malignancies. These risks must be carefully considered when initiating treatment [3]. Since starting treatment 11 months ago, the patient has not experienced side effects associated with upadacitinib. However, long-term safety and efficacy of this drug in OLP have not been fully established, as most data comes from studies on other immune-mediated diseases. Further controlled trials and long-term follow-up studies are needed to assess the safety profile and optimal dosing regimen of upadacitinib in OLP. Currently, upadacitinib is also being studied off label for treatment of lichen planus, hidradenitis suppurativa, palmoplantar pustulosis, and granuloma annulare, among others [8]. To the author's knowledge, there is only one clinical trial assessing the efficacy of upadacitinib in lichen planus. Compared to other immunosuppressive therapies, upadacitinib's rapid action and favorable side effect profile suggest it could be a valuable addition to the therapeutic armamentarium for this challenging condition.

Conclusion

In this case, upadacitinib effectively managed refractory OLP, resulting in marked improvement in the patient's oral mucosal health and quality of life. Although reports on its use in OLP remain limited, these findings align with upadacitinib's demonstrated efficacy in other inflammatory conditions, where it significantly reduces disease activity and improves clinical outcomes [8]. As a selective JAK1 inhibitor, upadacitinib targets key cytokine pathways central to OLP pathogenesis, offering the potential to shift management from conventional symptomatic relief toward true disease control. The observed clinical and symptomatic improvement underscores its promise in treating this chronic and debilitating condition. Further research, including randomized controlled trials, is warranted to establish its long-term safety, efficacy, and optimal role within the therapeutic spectrum of OLP management.

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