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Case Report

JAK Inhibitor Baricitinib in the Treatment of Oral Lichen Planus: A Case Report

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Abstract

Baricitinib, a JAK1/2 inhibitor, has been expanding its role as a therapeutic agent with recent FDA approval for dermatologic use with alopecia areata. Clinical trials are underway for further use of baricitinib for severe atopic dermatitis and multiple off-label uses in inflammatory dermatologic conditions such as lichen planus have been suggested. Few reports exist of patients with concomitant alopecia areata and lichen planus treated with baricitinib. We present a case of refractory oral lichen planus with marked improvement within one month following treatment with once daily baricitinib. Our case adds to the growing body of literature regarding the therapeutic benefits of JAK inhibitors in the treatment of lichen planus.

Keywords: Oral Lichen Planus; Alopecia Areata; Baricitinib; JAK/STAT

Abbreviations

LP: Lichen Planus; OLP: Oral Lichen Planus; JAK: Janus Kinase; AA: Alopecia Areata

Introduction

Lichen Planus (LP) is a chronic inflammatory condition characterized by discrete, pruritic, planar, violaceous papules and plaques. Although LP can affect areas including the inner forearms, wrists, ankles and genitals, Oral Lichen Planus (OLP) specifically affects squamous cell oral mucosa and presents with radiating white thread-like lacy papules. LP affects women more commonly than men (1.5:1) with symptom onset reported between the ages 30-60. Approximately 0.2%-1% of the population is affected by cutaneous LP, however, oral LP is more common and found in 1-4% of the population.1 Although the exact etiology is unknown, the current recognized pathogenesis classifies OLP as an autoimmune disease resulting in CD-8 positive cytotoxic T-cell destruction of basement membrane basal cells. Although traditional therapies of OLP include topical and systemic corticosteroids, recent reports have revealed Janus Kinase (JAK) inhibitors as potential novel treatment options.

Baricitinib is a selective reversible JAK1/2 inhibitor disease-modifying antirheumatic drug initially approved for patients with treatment refractory moderate to severe rheumatoid arthritis. However, since its approval in 2018, baricitinib has shown to have therapeutic benefits in a wide array of conditions including COVID-19, atopic dermatitis and recently FDA approved for alopecia areata in 2022. With its expanding therapeutic profile, we add to baricitinib's utility and describe a case of OLP refractory to multiple agents that was successfully treated with baricitinib.

Case Description

A 32-year-old female presented for follow-up of her chronic Alopecia Areata (AA). Her medical history was significant for concurrent OLP and family history was significant for AA. Upon examination, there were several discrete non-scarring patches of hair loss on the left occipital scalp, left inferior occipital scalp and right central parietal scalp. Previous AA treatment attempts included multiple rounds of intralesional triamcinolone ranging from 3-10 mg/cc, 0.05% clobetasol topical gel, 1.5% ruxolitinib topical cream and 5% minoxidil topical foam, all of which provided minimal improvement. After multiple failed treatments, the patient was prescribed 2 mg baricitinib once daily which provided moderate improvement.

Additional physical exam findings were significant for atrophic plaques on the left and right inferior vermillion border consistent with her five-year history of biopsy proven OLP (Fig. 1). Previous interventions with halobetasol propionate 0.05% ointment showed little improvement and worsening severity after two years. 10.0mg/cc intralesional triamcinolone injections and Clobetasol 0.05% topical ointment were administered over the next several years providing intermittent relief, however, symptoms returned to baseline shortly after and the patient experienced increasing pain in the lesional mucosa. After discontinuation of triamcinolone injections and Clobetasol ointment, topical ruxolitnib was prescribed however, no symptom improvement was reported. While the patient was being treated for OLP with ruoxlitinib and clobetasol, she was prescribed baricitinib for her chronic AA. Within one month of initiating baricitinib, the patient had noted significant improvement in her OLP with reduction in lesion size and pain. ruxolitinib therapy was discontinued and 12 weeks later the patient continued to note improvement in her OLP with complete clearance without redness, swelling or pain (Fig. 2). Baricitinib therapy was well tolerated without any adverse effects measured with a CBC, CMP and lipid panel at baseline and follow-up.

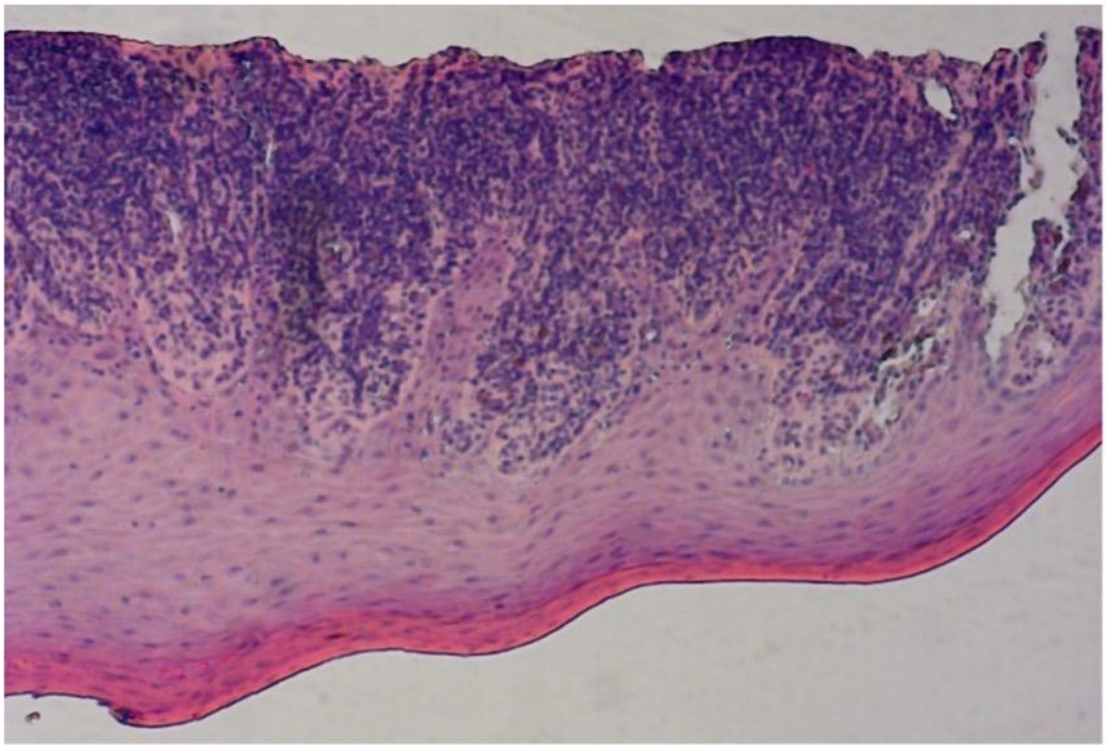

Figure 1: Histopathologic examination of lower vermillion shave biopsy with findings consistent with OLP. Slide stained with hematoxylin and eosin demonstrating epidermal hyperplasia with overlying hyperkeratosis and subjacent band like lymphocytic infiltrate. Dyskeratosis and disruption is noted at the dermal-epidermal junction.

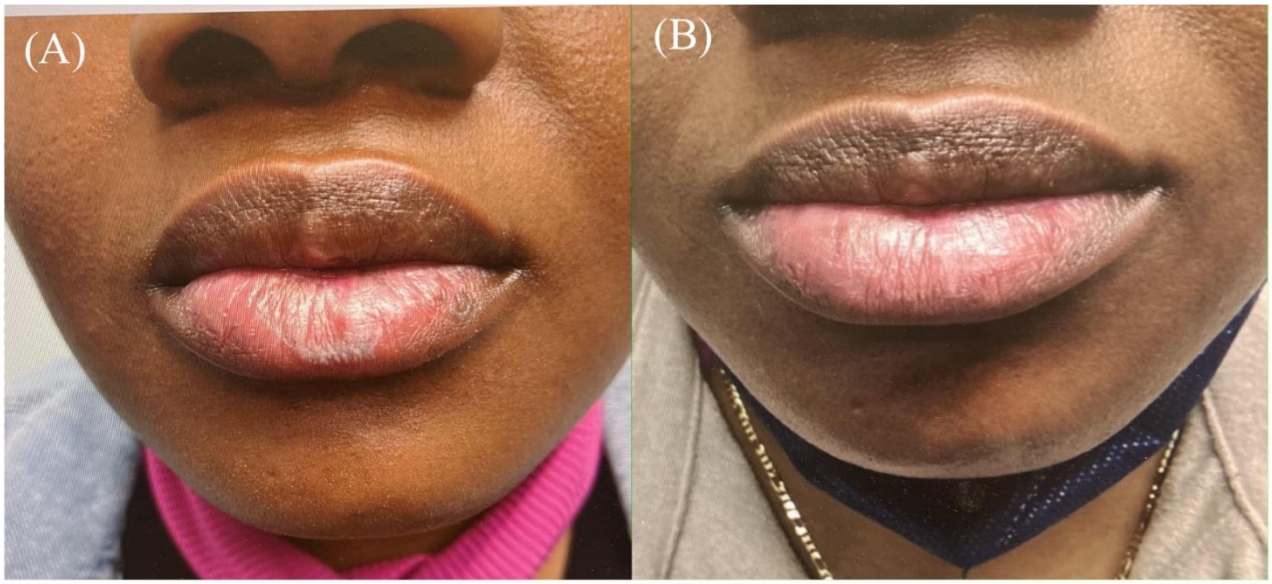

Figure 2: a) OLP presented on lower vermilion border pre-Baricitinib treatment (left); b) Resolved OLP at clinical visit 12 weeks post-Baricitinib treatment (right).

Discussion

We present a case of OLP refractory to multiple interventions that was successfully treated with baricitinib, a JAK1/2 inhibitor. OLP is a T-cell mediated autoimmune disease localized to the oral mucosa. Topical corticosteroids are typically first line agents in OLP treatment, followed by oral corticosteroids or injections if oral agents fail. OLP can be refractory to multiple treatments in a subset of patients rendering treatment and management difficult [1,2].

A recent review of OLP pathogenesis noted that disease process may start with expression of an unmarked antigen on keratinocyte surface. The antigen presentation leads to the induction of MHC-1 and CD4+ lymphocytes ultimately activating CD8+ T cells. An increased expression of MHC-2 on Langerhans cells releases interleukin-2 and IFN- γ in keratinocytes further activating T-cell mediated apoptosis of basal keratinocytes via the JAK-STAT pathway. Baricitinib, a JAK1/2 inhibitor, is FDA approved disease-modifying antirheumatic drug approved for AA. Recent trials have also demonstrated efficacy of baricitinib in severe AD [3]. Shoa, et al., used baricitinib to further assess the therapeutic benefit of a JAK inhibitors in LP and discovered that keratinocytes treated with Baricitinib were completely resistant to cell-mediated cytotoxicity [4]. In one recent systematic review authored by Abduelmula A, et al., baricitinib demonstrated efficacy for overall all lichen planus subtypes in 16 patients. Of these patients 9/16 (56%) had achieved either complete or partial resolution with an average time to outcome observed at 38 [1]. days. Of note, in the included studies, there were reports of 8 total adverse events including hypercholesterolemia, neutropenia, transaminitis or fatigue, with one patient requiring discontinuation of the medication [5].

To date, 4 case studies, consisting of 6 patients (5 female and 1 male) have reported the therapeutic benefit of JAK inhibitors in the treatment of OLP specifically [6]. Three patients reported effective improvement via tofacitinib, a JAK1/3 inhibitor and 2 patients via upadacitinib, a JAK1 inhibitor. Although few case studies have recently reported treatment of various type of LP with baricitinib, only one case report has reported its treatment potential in OLP [7]. A recent review reported the therapeutic potential of JAK inhibitors in the treatment of various LP subtypes.

Although existing literature regarding the treatment of OLP with baricitinib is limited to case reports, an ongoing clinical trial (NCT05188521) is actively investigating the safety and efficacy of baricitinib in addressing treatment-refractory cutaneous LP. Should this trial yield positive results, these results, in conjunction with the findings from this case report and previously cited reports, could open avenues to assess barcitinib's efficacy in treating OLP.

Conclusion

Our case adds to the growing body of literature regarding the therapeutic benefits of JAK inhibitors in the treatment of LP, specifically OLP. Although typical OLP therapy includes topical and systemic corticosteroids, this regimen may not be effective in all patient profiles. Therefore, patients with treatment refractory OLP may benefit from JAK inhibitors, such as baricitinib. This further supports the clinical evidence published in recent case reports/series and emphasizes the rationale regarding the need for larger clinical studies to examine the impact of JAK inhibitors in the treatment of LP and its subtypes.

Informed Consent

We have obtained explicit written informed consent from the patient to publish this anonymized case report.

Ethics Statement

Our institution does not require ethics approval for reporting case studies.

Conflict of Interest

The authors have no conflict of interest to declare.

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