Treatment of Steroid-Resistant Beard-Restricted Alopecia Areata with Baricitinib

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ABSTRACT
Alopecia areata is an autoimmune disorder causing localized hair loss due to T-cell attack on hair follicles. Beard involvement holds psychological and cultural significance, and treatment guidance for steroid-resistant cases is limited. We present a case of the successful treatment of steroid-resistant beard alopecia areata (BAA) with baricitinib, a JAK-1/2 inhibitor, in a 37-year-old male. Prior therapies, including topical ruxolitinib and intralesional steroids, had failed. Over 9 months, baricitinib (4 mg daily) and oral minoxidil led to reduced alopecic patches, increased hair density, and resolved inflammation. This case underscores the potential of JAK inhibitors for BAA, emphasizing the importance of establishing clear guidelines to direct treatment of in order to optimize patient outcomes.

INTRODUCTION
Alopecia areata (AA) is an inflammatory autoimmune disorder in which T-cell attack of the hair follicles results in well-circumscribed patches of non-scarring hair loss.¹ While it can affect any hair-baring region of the body, alopecia areata of the beard (BAA) can be particularly distressing for men given its psychosocial and cultural significance.² Topical corticosteroids are most commonly used as initial treatment, followed by intralesional steroids, but there is limited data on the appropriate management of steroid-resistant cases.¹² Baricitinib, an oral Janus kinase (JAK)-1/2 inhibitor, is recently the first FDA-approved treatment for adults with severe AA. Yet, its efficacy in BAA is not well established, and there are no clear guidelines for its use in AA limited to the beard. Herein, we present a case report of a male with steroid-resistant BAA who demonstrated a significant treatment response to baricitinib.

CASE REPORT
A 37-year-old male presented with 9 months of clinically diagnosed alopecia areata of the beard with no additional past medical history. Failed therapies included 6 sessions of intralesional triamcinolone injections at 2.5 mg/cc and 5 months of topical ruxolitinib. Skin exam showed inflamed, eczematous, erythematous patches of non-scarring, coin-shaped hair loss on the chin and bilateral cheeks (Figure 1A-C). No other areas of hair loss were noted. Baricitinib was started at a dose of 2 mg by mouth once daily, and all other therapies were discontinued. He returned to care 3 months later tolerating the medicine well with no reported side effects. Hair loss was stable on examination, with neither new patches nor regrowth. His dose
was increased to 4 mg daily, and minoxidil 2.5 mg tablet was added 2 months later to optimize treatment. On follow-up, 9 months after the initiation of baricitinib and 6 months after the dosage increase, the patient demonstrated significant improvement. Alopecic patches were noticeably reduced in size with 50% of the affected area showing evidence of regrowth. There was an increase in hair density in the affected regions, and the erythematous inflammation had resolved (Figure 1D-F). The patient was still tolerating the medicine well with no reported side effects, and he was continued on the current treatment regimen. He will be seen regularly for further follow up.

DISCUSSION

Janus kinase (JAK)-inhibitors disrupt the intracellular signaling pathways involved in inflammation and immune function and have demonstrated promising efficacy in the treatment of various inflammatory dermatoses. Specifically, baricitinib is an oral selective JAK-1/2 inhibitor that has revolutionized the treatment of alopecia areata of the scalp. However, though beard involvement affects 28% of men with AA, there is limited discussion on its targeted treatment. The beard holds considerable psychosocial significance across a wide array of cultural, social and religious groups.
Nevertheless, BAA is often overlooked in research, with clinical and observational studies primarily emphasizing AA of the scalp, eyebrows, and eyelashes. Classically, the first line treatment of BAA consists of topical and intralesional steroids, yet there is a paucity of research directing clear guideline for the effective management of steroid-resistant cases. 

According to a multicenter review of 55 patients with BAA, 45.5% of patients went on to develop scalp AA within 12 months. Thus, it may be prudent to approach BAA treatment aggressively, particularly in patients who have failed steroids and are suffering from significant psychological distress. A case presentation of a motivated male patient with total BAA reported complete regrowth of the beard after 4 months of 20 mg twice daily oral ruxolitinib, another selective JAK-1/2 inhibitor. A retrospective review of 45 patients with BAA on 3 months of tofacitinib, a JAK-1/2/3 inhibitor, found that 29 patients (64%) experienced partial to complete beard regrowth. There was a strong direct correlation between the extent of beard and scalp regrowth, yet, in contrast to scalp AA, there was an inverse association between disease duration and BAA treatment response.

A similar retrospective review of 60 patients with BAA on 3 months of baricitinib with concomitant low-dose oral minoxidil found even more favorable results. Fifty-one patients (85%) experienced partial to complete beard hair regrowth, and there was a strong direct correlation between beard and scalp regrowth. The mean dose of baricitinib was 3.9 ± 1.5 mg with a mean treatment duration of 12.1 ± 5.0 months. The average duration of BAA exceeded 4 years, and the time to onset of hair regrowth approximated 3 months. Notably, patients with total beard hair loss experienced poorer outcomes with limited regrowth. However, only 7 out of the 60 patients with BAA had hair loss restricted to the beard area, as appeared in our patient.

It is important to note that oral JAK inhibitors have been associated with adverse effects, such as infections, viral reactivations, and thromboembolic events. Due to the better safety profiles of topical JAK inhibitors, there has been investigation into the efficacy of topical tofacitinib for the treatment of beard alopecia. A systematic review of 9 patients with BAA receiving 2% tofacitinib gel twice daily, 7 patients experienced at least partial regrowth. Of note, our patient failed therapy with topical ruxolitinib, which has a mechanism of action that is more selective than that of tofacitinib.

Our case presentation highlights the treatment response of steroid-resistant, beard-restricted AA to baricitinib. Of note, 4 mg daily was necessary to achieve clinical regrowth. This is particularly important to consider given the dose-response nature of baricitinib’s adverse effect profile. Moreover, it is notable that our patient, like those in the retrospective review of baricitinib, received concomitant low-dose oral minoxidil, as there is evidence suggesting that the combination of JAK inhibitors with minoxidil can produce synergistic therapeutic effects. Unlike the patients in the retrospective review of baricitinib, our patient had been experiencing BAA for months rather than years prior to baricitinib initiation. Nevertheless, we selected baricitinib as therapy given his steroid-resistance and motivation for regrowth.

Our case adds to the growing body of information on the use of JAK inhibitors in the treatment of BAA. The favorable outcome may serve as supporting evidence for the pursuit of a formalized prospective study on the efficacy of baricitinib in BAA, and the
establishment of clear guidelines to direct the
treatment of steroid-resistant BAA remains
necessary to optimize patient outcomes.

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