PROC (BAYL UNIV MED CENT)
2018;31(4):524–525

https://doi.org/10.1080/08998280.2018.1480246

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Successful treatment of atopic dermatitis with the JAK1 inhibitor oclacitinib

Isabel M. Haugh, MB, BAO, BCh[a], Ian T. Watson,[b] and M. Alan Menter, MD[a]

[a]Department of Dermatology, Baylor University Medical Center, Dallas, Texas; [b]Texas A&M College of Medicine, Bryan, Texas

ABSTRACT

We report the first case of atopic dermatitis successfully treated with the oral Janus kinase-1 (JAK1) inhibitor oclacitinib. A man in his 70s, with a 6-year history of skin disease refractory to topical and biologic therapies, self-prescribed this veterinary medication with rapid remission of symptoms. He has remained in remission for 7 months with no reported adverse side effects or infections. JAK1 plays a central role in expression of proinflammatory cytokines IL-4, IL-5, and IL-13, which play an important role in the pathogenesis of atopic dermatitis. Ruxolitinib and tofacitinib are JAK inhibitors currently approved by the Food and Drug Administration for the treatment of myelofibrosis, rheumatoid arthritis, and psoriatic arthritis in humans. Oclacitinib is not currently indicated for use in humans.

KEYWORDS Atopic dermatitis; eczema; JAK1; oclacitinib

Oclacitinib is a Janus kinase-1 (JAK1) inhibitor approved for the treatment of pruritus secondary to allergic dermatitis and atopic dermatitis in canines. JAK1 plays a role in the expression of interleukin-4 (IL-4), interleukin-5 (IL-5), and interleukin-13 (IL-13) in proinflammatory signaling pathways known to contribute to the pathogenesis of atopic dermatitis.[1] Oclacitinib is not currently approved for use in humans. We report the first case of a man in his 70s who demonstrated significant relief of signs and symptoms of atopic dermatitis following self-prescription of this veterinary medication.

CASE DESCRIPTION

A man in his 70s presented with a 6-year history of atopic dermatitis with refractory pruritus despite the use of topical corticosteroids, topical and oral antihistamines, and a trial of omalizumab. He subsequently self-prescribed the canine medication oclacitinib, which resulted in significant reduction of erythema and itch within 2 hours of the first administration. To date, the patient reports 7 months of continuous therapy, taking an oral dose of 0.12 mg/kg in the morning and 0.32 mg/kg at night. This regimen has maintained excellent control of his symptoms. Immediate return of pruritus was noted by the patient following a single missed dose. The patient denies any adverse side effects or infections.

DISCUSSION

Oclacitinib selectively inhibits JAK1 of the JAK signal transducer and activator of transcription (JAK-STAT) pathway, which plays a central role in cytokine signaling of proinflammatory cytokines in atopic dermatitis, both in dogs and in humans. The pathway is essential for IL-4, IL-5, and IL-13, which are known to play a role in the underlying pathogenesis of atopic dermatitis.[1] Two JAK inhibitors, ruxolitinib and tofacitinib, are currently approved by the Food and Drug Administration for the treatment of myelofibrosis and rheumatoid and psoriatic arthritis, respectively. Tofacitinib inhibits both JAK1 and JAK3 and has been shown to have beneficial effects in the treatment of atopic dermatitis when administered both topically and orally.[2,3] In a phase IIa randomized controlled trial comparing 2% tofacitinib ointment to placebo, following 4 weeks of treatment, the mean percentage change in the Eczema Area and Severity Index score from baseline for the tofacitinib group was −81.7% versus −29.9% for the placebo group.[3] A case series of six patients with moderate to severe atopic dermatitis treated with oral tofacitinib showed an average decrease of 66.6% in the SCORing Atopic Dermatitis (SCORAD) score from 36.5 to 12 during 8 to 29 weeks of treatment with no adverse events reported.[2] Furthermore, the novel potent inhibitor of JAK1, JAK2, and JAK3, JTE-052, has been shown to improve skin

Corresponding author: Ian Talbert Watson, BA, 2008 Woodbury Ct., Southlake, TX 76092 (e-mail: Iwatson@medicine.tamhsc.edu)
Received April 2, 2018; Accepted May 21, 2018.

barrier function through suppressing signal transducer and activator of transcription 3 signaling, which is a key element in the regulation of keratinocyte differentiation.[4] JAK inhibition has excellent potential for the treatment of disrupted barrier function and symptom control in patients with atopic dermatitis, with multiple preparations currently in clinical trials.

To our knowledge, oclacitinib has not been tested in humans to date. This anecdotal evidence for the significant efficacy of JAK1 inhibition in atopic dermatitis provides further evidence to support the utility of targeting the JAK-STAT pathway in the treatment of atopic dermatitis.

FUNDING

No funding was provided by any source for this project. Dr. Menter reports grants and honoraria from AbbVie, Allergan, Amgen, Boehringer Ingelheim, Janssen Biotech, Inc., LEO Pharma, Novartis, Pfizer, and Xenoport; grants from Anacor, Celgene, Dermira, Merck, Neothetics, Regeneron, and Symbio/Maruho; and honoraria from Eli Lilly, Galderma, and Vitae, all outside of the submitted work. The remaining authors do not have funding disclosures, financial support, or industrial affiliations to report regarding any materials or methods used in this study or the findings indicated in this manuscript.

ORCID

Ian T. Watson http://orcid.org/0000-0003-3808-1443

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