involvement in PG is rare but has been reported to affect the eye, orbit, and ocular adnexa with presentations ranging from orbital destruction to sclerokeratitis.2-4 Similar reports of PG causing ectropion have been reported3,5 and highlight the concern for formation of PG lesions at sites of surgical incisions, such as graft sites, coined *postoperative PG*.6 Pathergy has been described in roughly 20% to 30% of patients with PG,1 and given this risk, preoperative and postoperative immunosuppression with systemic corticosteroids has been recommended. Use of oral prednisone at the time of surgery might have prevented the development of the new ulceration in this patient.

Caren Campbell, MD
Jeffrey P. Callen, MD

**Author Affiliations:** Division of Dermatology, University of Louisville,Louisville, Kentucky (Campbell, Callen); Associate Editor, *JAMA Dermatology* (Callen).

**Corresponding Author:** Caren Campbell, MD, Division of Dermatology, University of Louisville, 3810 Springhurst Blvd, Ste 200, Louisville, KY 40241 (cfcamp02@louisville.edu).

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**Tofacitinib Citrate for the Treatment of Nail Dystrophy Associated With Alopecia Universalis**

Nail dystrophy associated with alopecia areata (AA) and variants alopecia totalis and alopecia universalis (AU) may cause discomfort or pain and limitation of function. Currently, no reliably effective treatment options are available.1 The successful treatment of AA and variants with the oral Janus kinase (JAK) inhibitors tofacitinib, ruxolitinib, and baricitinib2-4 suggests the possibility that this class of agents may be a therapeutic option for nail dystrophy associated with AA.

**Report of Cases** | We describe 3 patients with AU with severe nail dystrophy who experienced normalization of nail growth during treatment with tofacitinib, a JAK-1/3 inhibitor.

**Case 1.** Patient 1 was a man in his 20s who presented with AU and trachyonychia causing substantial discomfort (Figure, A). He was treated with tofacitinib, 5 mg, twice daily for 2 months and then, because he was not experiencing hair growth, 10 mg in the morning and 5 mg at night for the next 4 months.

**Case 2.** Patient 2 was a woman in her 40s who presented with AU and frequent episodes of onycholysis for which she needed to keep 1 or 2 fingertips wrapped with tape daily. She was treated with tofacitinib, 5 mg, twice daily.

**Case 3.** Patient 3 was a man in his 20s who presented with AU and severe nail pitting and nail fragility that was the cause of frequent painful injury. He was treated with tofacitinib, 5 mg, twice daily.

The results of baseline laboratory evaluation, including complete blood cell count, comprehensive metabolic panel, lipid panel, QuantiFERON-TB Gold (Quest Diagnostics), and human immunodeficiency virus, hepatitis B, and hepatitis C blood tests, were normal in all 3 patients.

All 3 patients experienced remission of nail changes and associated discomfort or pain during treatment with tofacitinib over a period of 5 to 6 months (Figure, B).
Interestingly, patients 2 and 3 also experienced hair growth, but patient 1 (Figure) did not. Ten weeks after discontinuation of tofacitinib treatment, patient 1 did not exhibit recurrence of nail dystrophy. Patients 2 and 3 continued to take tofacitinib. Tofacitinib was well tolerated in all cases, and infections, cytopenias, transaminisits, decreased renal function, or increased lipid levels were not observed.

**Discussion** | Nail dystrophy in AA and variants is common, affecting 7% to 66% of patients, and includes nail pitting, trachyonychia, onychorrhexis, red spotting of the lunulae, onycholyis, onychomadesis, and Beau lines. Little is known about the pathogenesis of these changes. Recently, it has been shown that hair follicle gene expression of interleukin-15, NKG2D ligands, and major histocompatibility complex molecules leads to recruitment and activation of interferon γ-producing, NKG2D-expressing CD8 T cells that target the hair follicle for attack. Because JAK family protein kinases are downstream effectors of the interferon γ receptor in keratinocytes, JAK signaling mediates interleukin 15 activation of T cells, explaining the effectiveness of JAK inhibitors for the treatment of AA. The nail matrix, like the hair follicle, is an epithelial keratin-producing structure, and it may be that nail matrix keratinocytes exhibit gene expression similar to hair follicle keratinocytes in AA. In this case, JAK inhibitors would be expected to reverse nail dystrophy associated with AA.

The outcomes in the 3 patients described here suggest that tofacitinib and other JAK inhibitors may be effective in the treatment of severe nail dystrophy associated with AA and variants. Additional studies will be needed to confirm their efficacy and further explore their safety.

**Arjun Dhayalan, BS**  
Brett A. King, MD, PhD

**Author Affiliations:** Albany Medical College, Albany, New York (Dhayalan); Department of Dermatology, Yale University School of Medicine, New Haven, Connecticut (King).

**Corresponding Author:** Brett A. King, MD, PhD, Department of Dermatology, Yale University School of Medicine, PO Box 208059, New Haven, CT 06520 (brett.king@yale.edu).

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