Alopecia areata (AA) is a nonscarring form of hair loss characterized by well-defined patches of alopecia, typically involving the scalp, and less commonly by near-complete or complete scalp and body hair loss. In a murine model of AA, up-regulation of interleukin-15 in hair follicles leads to recruitment and activation of natural killer gene 2D-expressing CD8 T cells, which, in turn, produce interferon-γ, further activating the hair follicle epithelial cells. Cell signaling via interferon-γ and interleukin-15 occurs via the Janus kinase (JAK) family of enzymes, and JAK inhibitors have been found to reverse disease. In particular, the JAK1/3 inhibitor, tofacitinib, and the JAK1/2 inhibitor, ruxolitinib, have been found in larger series of patients to be effective for severe disease. There is a report of a patient with AA and chronic atypical neutrophilic dermatosis with lipodystrophy and elevated temperature (CANDLE) syndrome, whose AA was successfully treated during treatment of CANDLE syndrome with high-dose baricitinib, 7 to 11 mg daily, in addition to prednisone. Baricitinib is a relatively new JAK1/2 inhibitor that was recently approved for the treatment of rheumatoid arthritis in Europe and Japan at doses of 2 and 4 mg daily and in the United States at 2 mg daily. Here we describe a case of a woman with severe AA, with complete scalp and near-complete body hair loss, who experienced complete hair regrowth with baricitinib 4 mg daily.
DISCUSSION

To our knowledge, this is the first report of a patient with AA successfully treated with baricitinib monotherapy. While the potential risks of JAK inhibition must be discussed with every patient, they are especially important to consider in a patient with a history of cancer. Like the tumor necrosis factor-α inhibitors (eg, adalimumab), tofacitinib and baricitinib carry a black box warning regarding infection and malignancy risk. In phase 2, phase 3, and long-term extension studies of baricitinib and tofacitinib treatment of rheumatoid arthritis, the rate of all malignancies (excluding nonmelanoma skin cancer) was 0.8 per 100 patient years with baricitinib (95% confidence interval, 0.6 to 1.0) and 0.85 per 100 patient years with tofacitinib (95% confidence interval, 0.7 to 1.02), similar to what is observed for rheumatoid arthritis, in general. It is interesting that during the controlled clinical trials of tofacitinib in ulcerative colitis (1220 patients, up to 52 weeks of treatment), there were no cases of solid organ malignancy or lymphoma. It is interesting that during the controlled clinical trials of tofacitinib in ulcerative colitis (1220 patients, up to 52 weeks of treatment), there were no cases of solid organ malignancy or lymphoma. It is interesting that during the controlled clinical trials of tofacitinib in ulcerative colitis (1220 patients, up to 52 weeks of treatment), there were no cases of solid organ malignancy or lymphoma.

AA is a common disorder that is often associated with poor quality of life, depression, and anxiety. As with atopic dermatitis and psoriasis, the treatment of AA with systemic medications is merited, and it has been shown that quality of life in AA can be restored with successful treatment. Although effective therapy for patients with severe disease has historically been elusive, the emergence of JAK inhibitors is changing this. This report of successful treatment of a patient with severe AA using the new JAK inhibitor, baricitinib, adds to the growing body of evidence showing efficacy not only of this class of medication as a whole, but also of JAK inhibitors of different specificities (eg, JAK1/2 vs JAK1/3) for AA. We eagerly await the results of ongoing clinical trials using baricitinib (NCT#03570749) and other JAK inhibitors (NCT#03732807, 03594227, 03811912).

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