

Baricitinib-Induced Trichilemmal Cyst Reactivation in a Woman With Alopecia Areata

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PRACTICE POINTS

- The rapid growth of trichilemmal cysts may serve as an indicator of a quick-responder phenotype to baricitinib in cases of alopecia areata (AA), although more evidence is needed.
- It is imperative to consider personal and family history of trichilemmal cysts prior to initiating baricitinib treatment for AA.

To the Editor:

Alopecia areata (AA), an autoimmune disease characterized by inflammatory and nonscarring hair loss, can have a considerable impact on quality of life.¹ Baricitinib is a Janus kinase inhibitor that recently was approved by the US Food and Drug Administration for treatment of severe AA in adult patients, becoming the only on-label treatment available.² So far, the most common adverse effects reported in phase 3 trials have been acne, upper respiratory tract infections, headaches, urinary tract infections, and elevated creatine kinase levels.³

At our trichology unit in the dermatology department of a Spanish tertiary-care hospital in Seville, we have successfully used baricitinib to treat 18 patients with severe, therapy-resistant AA. Herein, we present a case of trichilemmal cyst reactivation in one of our patients following successful treatment with baricitinib.

A 53-year-old woman with a history of trichilemmal cysts presented to the dermatology department with total body hair loss of 5 years' duration that was diagnosed as AA universalis (Figure, A). The patient reported that

the trichilemmal cysts had shrunk drastically 1 month after complete loss of body hair (Severity of Alopecia Tool [SALT] score, 100)(Figure, B). The largest cyst was



FIGURE. A, A 53-year-old woman with alopecia areata prior to treatment with oral baricitinib. B, By week 8 of treatment, total hair regrowth was achieved with reactivation of a trichilemmal cyst on the frontal scalp.

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surgically removed, and the diagnosis was histologically confirmed by a pathologist. Her mother and sister also had a history of multiple trichilemmal cysts.

The patient previously had failed treatment with oral prednisone 50 mg/d, oral cyclosporine 4 mg/kg/d, oral dexamethasone 4 mg twice weekly, and oral azathioprine 300 mg/wk. Due to the new indication of baricitinib for AA, we opted to start the patient on oral baricitinib 4 mg/d. By week 8 of treatment, she had achieved total hair regrowth (SALT score, 0). This rapid response might indicate a quick-responder phenotype, referring to a subset of patients who exhibit a fast and robust response to treatment (SALT90), generally before week 16, although more evidence is needed.

Notably, we observed the reactivation of 4 trichilemmal cysts on the scalp 6 weeks after starting baricitinib. To our knowledge, this side effect has not previously been reported. We hypothesize that reactivation of the cysts may have been due to the inhibition of the Janus kinase/signal transducer and activator of transcription pathway, which reduces the effects of cytokines and leads to reactivation of hair follicles that were

inactive because of inflammation.⁴ As a result, the outer root sheath of the hair follicle can once again be filled with keratin, thereby reactivating the trichilemmal cysts. Based on our experience with this case, it may be relevant to consider personal and family history of trichilemmal cysts before starting treatment with baricitinib for AA and advise the patient about the possibility of this adverse effect.

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