Not just thinning: A case of alopecia universalis after mild COVID-19

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INTRODUCTION

SARS-CoV-2 infection and its inflammatory sequelae have been reported to affect hair, with the most common association being telogen effluvium (TE).[1] Onset of TE noted as diffuse thinning following SARS-CoV-2 infection is reported to occur after 1 to 3 months on average, with trichoscopy findings of empty hair follicles, thin terminal hairs, and 1-hair follicular units.[2] Implicated mechanisms include stress of the disease, proinflammatory cytokine release, or direct viral damage to the hair follicles.[2] COVID-19 has also been associated with exacerbation of autoimmune conditions. Although 1 study showed that patients with preexisting alopecia areata (AA) did not have worsened hair loss after mild-to-moderate COVID-19,[3] reports on new-onset or recurrent AA associated with COVID-19 are scarce.[4-7]

The majority of SARS-CoV-2 infections are considered being of mild severity (81%),[8] defined by the National Institutes of Health as having signs and symptoms such as fever, cough, loss of taste or smell, and diarrhea, but without dyspnea or abnormal findings on chest imaging. Although we do not usually expect mild cases to involve a severe impact on hair, we report a case of a patient with rapid-onset *de-novo* AA that presented in a diffuse pattern and that in the early stages was clinically indistinguishable from TE.

Abbreviations used:

AA: alopecia areata
SALT: severity of alopecia tool
TE: telogen effluvium

CASE REPORT

A 28-year-old woman with a history of allergic rhinitis initially presented to an outside dermatologist with a new-onset rapid diffuse hair loss 1 month after a mild SARS-CoV-2 infection. A scalp biopsy taken by the outside dermatologist was consistent with AA, showing a decrease in the number of anagen follicles and an increase in the number of catagen and telogen ones. Surrounding inferior portions of numerous follicles showed aggregates of lymphoid cells. Periodic acid—Schiff staining was negative for hyphae. Despite timely treatment with 1 to 2 cc intralesional triamcinolone injections (5.0 mg/cc) every 2 to 6 weeks, methylprednisolone dose pack, and platelet-rich plasma injections to the scalp, she had near-complete hair loss, when she presented to our dermatology clinic 3 months after onset. She was previously healthy, with no previous personal or family history or AA, no recent surgeries, allergy exacerbations, or new medications. She had not received any COVID vaccinations, as they were not yet available to the general public at this time. She had acne that was well controlled on spironolactone 50 mg daily and norethindrone/ethinyl estradiol. Her family history was notable for psoriasis and psoriatic arthritis in her father. Physical examination revealed a 90% hair loss on the scalp with a Severity of Alopecia Tool (SALT) score of 90 (Fig 1, *A*) and complete loss of her eyelashes and eyebrows. Trichoscopy of the scalp revealed yellow dots, short vellus hairs, black dots, and exclamation mark hairs. General examination revealed no other cutaneous or

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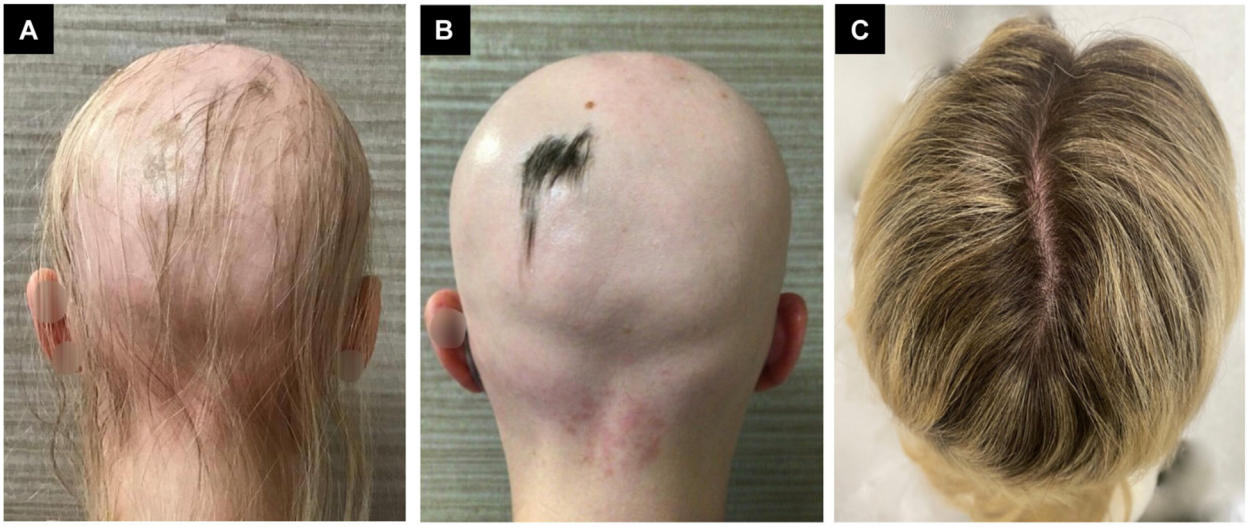

Fig 1. A 28-year-old woman with new-onset alopecia universalis after mild COVID-19 infection. **A**, 3 months after infection. **B**, 4 months after infection. **C**, At 16-month follow-up, with diffuse hair regrowth after 13 months of treatment with tofacitinib 5 mg twice daily.

nail findings. Her complete blood cell count, complete metabolic panel, and lipid panel were unremarkable. She could not tolerate oral minoxidil 1.25 mg daily because of the development of significant leg edema. Treatment with oral tofacitinib 5 mg twice daily was initiated, which initially did not halt the progression of the SALT score to 98 after 1 month (Fig 1, *B*), but which at the 10-month follow-up resulted in fine, diffuse regrowth, with a SALT score <5%. At her 16-month follow-up, she had no hair loss, and the SALT score was 0 (Fig 1, *C*).

DISCUSSION

Although the clinical presentations of hair loss following COVID-19 may vary, most reports are of TE, with very infrequent reports of AA.[4-7] A retrospective cohort study of 32 patients with preexisting AA showed no significant worsening of hair loss after mild-to-moderate COVID-19.[3] In contrast, we have noted an increased trend of hair shedding in several patients with AA in our clinic (unpublished data). Our search of the literature revealed only one research letter reporting a case of alopecia universalis following a SARS-CoV-2 infection[5] but did not provide detailed information from examination findings, trichoscopy, biopsy, clinical photos, or follow-up details. What was striking in our patient was not only the rapid progression of hair loss, but also the early clinical presentation of a diffuse pattern very reminiscent of TE. This *de-novo* case of alopecia universalis kept progressing and eventually declared itself.

This case raises the following question: is the elusive TE after COVID-19 truly just that? Or, in addition to many things that we learned from COVID-19, we can further implicate the role of inflammation, be it systemic or local in diffuse hair loss. The triggers for AA in genetically predisposed patients, whether new onset or exacerbations, fall along the lines of those speculated for TE, including recent health changes, medications, infections, hormonal alterations, and patient-reported stress.[9] Infections with SARS-CoV-2, cytomegalovirus and Epstein-Barr virus, as well as hepatitis A or B virus vaccination, have been shown to be associated with AA.[9] The purported mechanisms are speculated to be primarily driven by the proinflammatory cytokine response to SARS-CoV-2 rather than direct viral or associated psychogenic stress related to the infection. Viruses may trigger AA via an antiviral interferon mediated response, a T-helper 1—promoting cytokine, which induces major histocompatibility complex class I expression in the proximal outer root sheath of the hair follicle, resulting in its loss of immune privilege.[4] Also, viral infections may cause a large release of proinflammatory cytokines, including interleukin 6, a T-helper 2—promoting cytokine, which plays a significant role in COVID-19 and the hair follicle cycle.[2] Although AA conventionally belongs to the T-helper 1—mediated adaptive immune system regulation, the contributing T-helper 2 component is becoming more evident. Other potential mechanisms for how viral infections may trigger AA include downregulation of immunosuppressive cytokines,[10] molecular mimicry, super

antigens, and epitope spreading.[9] Future studies are needed to further elucidate the relationship between AA exacerbations and COVID-19 and the role of the T-helper 1/T-helper 2 response.

Conflicts of interest

None disclosed.

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