A case of rapid progression of central centrifugal cicatricial alopecia after COVID-19 infection

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Key words: Central centrifugal cicatricial alopecia after COVID-19 infection; COVID-19; hair loss; scarring alopecia.

INTRODUCTION
Hair loss has been reported in roughly 25% of patients following infection with COVID-19.1 Nonscarring alopecia is the most common type of hair loss seen after COVID-19 infection, with multiple studies reporting increased incidence of telogen effluvium (TE), alopecia areata (AA), and androgenetic alopecia (AGA).1,2 TE has been described in patients without a prior history of hair loss, while most cases of AA and AGA after COVID-19 infection occur as an exacerbation of pre-existing disease.2,3 AGA exacerbation has also been associated with increased COVID-19 severity.1 In contrast, there are few reports of scarring alopecia in association with COVID-19 infection, with 1 report of worsening hair loss in a patient with frontal fibrosing alopecia.5 We highlight a patient with a known history of central centrifugal cicatricial alopecia (CCCA), a primary form of scarring alopecia commonly seen in women of African descent, who experienced an acute exacerbation of hair loss 2 weeks after COVID-19 infection.

CASE REPORT
A 33-year-old Black female with a history of biopsy-proven CCCA presented with a new, large patch of alopecia on the crown extending toward the frontal scalp, associated with scalp pain and tenderness. She noted that alopecia developed 2 weeks following a mild infection with COVID-19. The patient was fully vaccinated with 2 doses of the Pfizer-BioNTech COVID-19 vaccine 9 months prior to the onset of COVID-19 infection, and she did not note worsening alopecia after vaccination.

The patient had a 4-year history of CCCA. At initial presentation, she was noted to have a large patch of alopecia on the crown of her scalp. She was treated over the following 2 years with oral doxycycline 100 mg twice a day, clobetasol 0.05% solution, minoxidil 5% foam, ketoconazole 2% shampoo, and multiple rounds of intralesional triamcinolone acetonide injections at doses ranging from 2.5 mg/ml to 10 mg/ml with near-complete regrowth of hair. In the year prior, her CCCA was well-controlled with minoxidil 5% foam daily and clobetasol 0.05% solution 5 days per week.

Physical examination of the scalp revealed a 16.5 × 8 cm area of decreased hair density with notable perifollicular scale compared to 2 months prior (Fig 1). The distribution of decreased hair density was the same as her initial presentation of CCCA, and her clinical presentation was consistent with progression

Abbreviations used:
AA: alopecia areata
AGA: androgenetic alopecia
CCCA: central centrifugal cicatricial alopecia
TE: telogen effluvium
of her CCCA. She was re-started on oral doxycycline 100 mg twice daily, and the affected area of the scalp was treated with 2 cc of 10 mg/cc intralesional Kenalog, with plans to repeat every 6 weeks until stability was achieved. The patient was directed to increase clobetasol 0.05% solution to daily and continue minoxidil 5% solution daily. After 2 months of topical treatment and 3 rounds of intralesional triamcinolone acetonide injections, the patient noted an improvement in her scalp symptoms and moderate hair regrowth (Fig 2).

**DISCUSSION**

CCCA is a chronic progressive, inflammatory scarring alopecia that can lead to permanent hair loss. Hair loss begins at the vertex or crown of the scalp and spreads centrifugally over time. Patients often report associated symptoms of scalp burning, itching, tenderness, and scaling. Dermoscopy may be used to identify specific features of CCCA, including a peripilar gray/white halo around the emergence of the hair follicle, honeycomb pigmented rete ridges and hypomelanotic dermal papillae, perifollicular erythema, and hair breakage appearing as black dots on the scalp. Biopsy of the scalp may be used to confirm the diagnosis of CCCA; histologic findings include premature desquamation of the inner root sheath, eccentric atrophy of the outer root sheath, and perifollicular concentric lamellar fibroplasia.

Our patient experienced acute hair loss at the crown of the scalp consistent with the pattern of distribution of hair loss associated with CCCA. While TE may be considered, this patient experienced hair loss on the crown of her scalp only, instead of the diffuse hair loss seen in TE. Additionally, TE characteristically presents 2 to
3 months after an inciting stressor, such as viral infection, while our patient developed hair loss 2 weeks after infection with COVID-19. However, due to the increased cases of TE seen after COVID-19 infection, it is possible that our patient had concomitant TE and CCCA, with more pronounced hair loss at the site of prior alopecia.

Various factors, including gene expression, infection, autoimmune disease, and hair grooming techniques, have been implicated in the pathogenesis of this condition. Proposed mechanisms of TE, AA, and AGA include the upregulation of proinflammatory cytokines, the loss of hair follicle immune privilege, and androgen-mediated upregulation of the transmembrane serine protease 2 (TMPRSS2) that enables entry of SARS-coronavirus 2 through the angiotensin-converting enzyme 2 receptor, respectively. Underlying processes that may have contributed to a proinflammatory environment exacerbating our patient's CCCA include the loss of immune privilege of hair follicles, direct viral damage to the hair follicle, and damage to the hair matrix by cytokines.

Our case highlights the rapid onset of hair loss 2 weeks after COVID-19 infection and suggests that COVID-19 may contribute to exacerbation of CCCA. In our patient, hair regrowth was noted after 2 months of treatment. Recognition of relapsing CCCA following COVID-19 infection and early treatment is important in halting scarring and establishing hair regrowth in areas of the scalp that are not permanently scarred.

Conflicts of interest
None disclosed.

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