Enlarging alopecic patch in an African American woman with central centrifugal cicatricial alopecia: A case of concomitant tinea incognito

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Key words: alopecia; tinea capitis; tinea incognito; *Trichophyton tonsurans*.

INTRODUCTION

Central centrifugal cicatricial alopecia (CCCA) is a scarring alopecia that typically presents with an enlarging alopecic patch on the vertex of the scalp, most commonly in African American females.[1] Tinea incognito is a dermatophyte infection of skin and hair exacerbated by the erroneous application of immunosuppressive therapies. Its presentation is variable and may mimic other dermatologic conditions, resulting in delays in accurate diagnosis and management.[2]

Tinea capitis, a cutaneous fungal infection of the scalp or hair, is a curable cause of hair loss. Though common in the pediatric population, tinea capitis may be misdiagnosed due to its relative rarity in adults and clinical resemblance to other primary scarring alopecias.[3] We present a case of an African American woman with biopsy-proven CCCA, who stopped responding to treatment of topical and intralesional steroids, found to have a concomitant diagnosis of tinea incognito.

Abbreviation used:

CCCA: central centrifugal cicatricial alopecia

CASE REPORT

A 49-year-old African American woman with a history of atopic dermatitis and alopecia areata presented for the evaluation of a pustule at the scalp vertex, first noticed 2 weeks previously. She reported localized pain and generalized hair thinning, but no itching or drainage. She denied a history of braids, weaves, or other high-tension hairstyles.

On examination, the patient was found to have 2, 4-6−mm inflammatory papules surrounded by decreased hair density. Trichoscopy revealed perifollicular scale, but no erythema, exclamation point hairs, or follicular dropout. The patient was treated with mupirocin ointment. Given her history of alopecia areata and non-specific trichoscopic findings, she also received intralesional triamcinolone acetonide injections.

At a 2-month follow-up visit, the patient was found to have substantial enlargement of the alopecic patch. A biopsy confirmed a diagnosis of CCCA, showing perifollicular fibrosis, polytrichia, and loss of sebaceous gland lobules. The patient was initiated on doxycycline 100 mg twice daily, and daily topical 5% minoxidil solution, clobetasol 0.05% solution, and tacrolimus 0.1% ointment. She was also administered a second round of intralesional triamcinolone acetonide (10 mg/mL).

Despite therapy, the patient's condition worsened with expansion of the alopecic patch to 7 × 4 cm (Fig 1, *A*). She also reported new-onset, tender,

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Authors Klein and Karim contributed equally as co-first authors, and Lo Sicco and Shapiro contributed equally as co-senior authors.

Funding sources: None.

IRB approval status: Not applicable.

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JAAD Case Reports 2022;23:67-9.
2352-5126

https://doi.org/10.1016/j.jdcr.2022.02.028

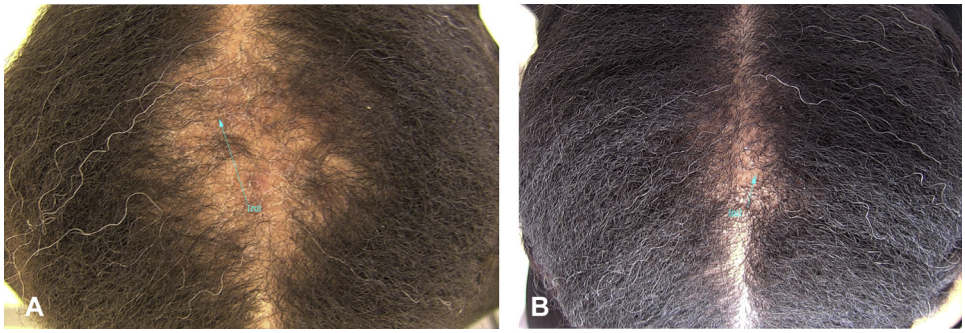

Fig 1. A, Photograph following treatment with immunosuppressive therapy. **B,** Photograph following treatment with terbinafine.

Table I. Characteristics of central centrifugal cicatricial alopecia (CCCA) and tinea capitis

	Epidemiology/ clinical history	Physical examination	Trichoscopy	Pathologic features
CCCA	Most common in African American women May have a history of traumatic hair care practices (eg, hot combs, chemical relaxers)	Distribution: most commonly affects the vertex or crown Features: may be associated with dysesthesia, pruritus, or tenderness	Peripilar white gray halo Perifollicular scale Follicular dropout[6]	Follicular dropout Perifollicular concentric fibrosis and compound follicular dropout Naked hair shafts in giant cells Premature degeneration of the inner root sheath Migration of the hair shaft through the outer root sheath[6,7]
Tinea capitis	Most common in children Contagious: may be acquired through direct contact with scalp or through spores that persist on fomites[3]	Distribution: any area of the scalp Features: with or without scales or pruritus Additional findings: lymphadenopathy[3]	Black dots Comma hairs Corkscrew hairs Bar code-like hairs Zigzag hairs[6,8]	Fungal hyphae and spores within affected hair shafts with periodic Acid—Schiff stain[6]

CCCA, Central centrifugal cicatricial alopecia.

cervical lymphadenopathy. After a third follow-up visit, a repeat biopsy was performed, this time revealing refractile fungal hyphae, with subsequent culture growing out *Trichophyton tonsurans.* The patient was advised to discontinue her use of corticosteroids and began 4 months of oral terbinafine (250 mg/day). Over the next several months she exhibited marked improvement (Fig 1, *B*).

DISCUSSION

Though tinea capitis primarily affects children, there has been a recent increase in its documented incidence in adults, particularly among African American women. In a retrospective analysis of patients with tinea capitis at an urban hospital, 11.4% of the individuals were adult African Americans, of whom 77% were women.[4] In adults, tinea capitis usually presents as indolent disease, with up to 30% of patients demonstrating no symptoms other than mild flaking.[3] Mild disease may be mistaken for seborrheic dermatitis, especially in African American patients, in whom the incidence of seborrheic dermatitis (6.5%) is higher than that of the general population.[3,5]

Inflammatory tinea capitis, which has clinical features that can be indistinct from other scarring alopecias, such as CCCA, may also be misdiagnosed, as was the case with our patient. Both tinea capitis and CCCA may present with perifollicular erythema, expanded hair shafts, and follicular plugging.[3] Though black dots, comma hairs, and corkscrew hairs are more specific to tinea capitis, diagnosing tinea when there is concern for a secondary scarring process may ultimately require pathologic evaluation. Histopathology with periodic Acid—Schiff stain would reveal fungal hyphae and spores within affected hair shafts (Table I).[6-8]

The relative rarity of tinea capitis outside the pediatric population is thought to be due to adults' increased production of triglyceride-rich sebum,

which inhibits dermatophyte growth.[3] However, this immunologic defense is less effective against certain endothrix infections including those caused by *T. tonsurans*, which is believed to establish a symbiotic relationship with its human host.[3] *T. tonsurans* is the most common etiologic agent in US urban areas and is the fungus that infected our patient. Additionally, the relative risk of tinea capitis in African Americans is greater than that in the general US population, in part due to hypothesized differences in host defenses.[9,10] A history of atopy, as was present in our patient, may also be a risk factor due to impaired skin barrier function.[10]

This case was complicated due to the presence of multiple etiologies of hair loss in a single patient. Though our patient had a history of alopecia areata and CCCA, an acceptable treatment for these conditions, corticosteroids, was ultimately an exacerbator of her secondary dermatophyte infection. The case highlights the importance of keeping a broad differential diagnosis and maintaining high clinical suspicion when patients do not predictably respond to appropriate treatment.

Conflicts of interest

None disclosed.

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