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Inflammatory tinea capitis mimicking dissecting cellulitis in a postpubertal male: a case report and review of the literature

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Summary

Tinea capitis in postpubertal patients is unusual and may be misdiagnosed as dissecting cellulitis. We report a case of a healthy 19-year-old Hispanic male presenting with a 2-month history of a large, painful subcutaneous boggy plaque on the scalp with patchy alopecia, erythematous papules, cysts and pustules. Although initially diagnosed as dissecting cellulitis, potassium hydroxide evaluation (KOH preparation) of the hair from the affected region was positive. A punch biopsy of the scalp demonstrated endothrix consistent with tinea capitis, but with a brisk, deep mixed inflammatory infiltrate as can be seen with chronic dissecting cellulitis. Fungal culture revealed *Trichophyton tonsurans*, and a diagnosis of inflammatory tinea capitis was made. The patient was treated over the course of 17 months with multiple systemic and topical antifungal medications, with slow, but demonstrable clinical and histopathological improvement. A rare diagnosis in adults, clinicians should have a high index of suspicion for this condition in an adult with an inflammatory scalp disorder not classic for dissecting cellulitis or with a *recalcitrant* dissecting cellulitis. Prompt, appropriate diagnosis and treatment is necessary to prevent the long-term complications of scarring alopecia.

Keywords

Tinea capitis; dissecting cellulitis; kerion; *Trichophyton tonsurans*; scarring alopecia; cicatricial alopecia

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Conflict of interest and disclosures

The views expressed in this article are those of the authors and do not necessarily reflect the official policy or position of the Department of the Navy, Army, Department of Defense, nor the United States Government. We certify that all individuals who qualify as authors have been listed; each has participated in the conception and design of this work, the analysis of data (when applicable), the writing of the document and the approval of the submission of this version; that the document represents valid work; that if we used information derived from another source, we obtained all necessary approvals to use it and made appropriate acknowledgements in the document; and that each takes public responsibility for it.

Introduction

Tinea capitis is a common scalp condition, seen primarily in the paediatric population. Its occurrence is recognised in postpubertal individuals, but is far less common and can present bizarrely, often leading to misdiagnosis.^{1–5} Inflammatory tinea capitis results from a hypersensitivity reaction to a dermatophytic infection.⁵ If correct diagnosis and treatment are delayed, it can result in scarring alopecia.⁶ Herein, we describe a case of inflammatory tinea capitis in a postpubertal male mimicking dissecting cellulitis. To date, we are aware of only three reported similar cases of inflammatory tinea capitis presenting in this manner.^{7,8} This case further adds to the literature regarding the condition, illustrating the importance of early diagnosis and prompt treatment to prevent the longterm complications of scarring alopecia.

Case history

A 19-year-old, healthy active duty Hispanic male presented to our Dermatology Clinic with a 2-month history of painful, patchy alopecia of the scalp associated with erythematous papules, cysts, pustules and a large subcutaneous boggy plaque. Referred to our clinic by primary care, the patient was initially diagnosed with dissecting cellulitis and completed 1 month of treatment with benzoyl peroxide, minocycline 50 mg twice daily and fluocinonide gel without improvement. He was otherwise healthy and denied constitutional symptoms and was not immunosuppressed.

On physical examination, there was a 15 × 9 × 20 cm well defined plaque of patchy alopecia and peripheral pustules on the right temporo-parietal scalp. Within the plaque there were black dots, broken hairs, follicular plugs and doll's hairs and an approximately 10 cm subcutaneous, boggy plaque that exuded pus with pressure (Fig. 1). There was no warmth or erythema and scant scale. On the right arm was an 18 cm annular plaque with scattered and peripheral 2–3 mm erythematous papules and an advancing, scaly border. Within the plaque were brown, hyperpigmented macules and patches representing postinflammatory hyperpigmentation (Fig. 2). There was no associated cervical, occipital or supraclavicular lymphadenopathy. White scale was present on the soles and sides of his feet bilaterally. There were no additional lesions seen on complete examination of the skin and nails.

Microscopic 10% potassium hydroxide examinations (KOH preparation) of scale from both his scalp and right arm plaques were positive. A 4 mm scalp biopsy revealed numerous fungal elements located inside the hair shaft representing an endothrix tinea capitis (Fig. 3) as well as an intense, chronic, mixed inflammatory infiltrate of neutrophils, lymphocytes, plasma cells, eosinophils and histiocytes located perifollicularly and in the deep dermis (Fig. 4). Special stains for fungus were positive. A bacterial culture of the scalp revealed no growth, and a fungal culture grew *Trichophyton tonsurans*.

The patient was prescribed oral terbinafine 250 mg daily, prednisone 40 mg each morning for 5 days, ketoconazole 2% shampoo daily to scalp and body and topical naftifine 1% cream daily for his right arm. He was followed clinically and continued the antifungal course for 7 months, regrowing 95% of his scalp hair, but continuing to have mild symptoms of scalp pruritus and erythematous papules with scale and follicular plugging (Fig. 5).

Microscopic KOH preparation of the scalp after 7 months of therapy was positive. The patient was switched to itraconazole 200 mg daily, naftifine 1% cream daily and ketoconazole 2% shampoo alternating with salicylic acid 2%/sulphur 2% shampoo. He completed 2 months of therapy with maintenance of hair regrowth and resolution of symptoms and scale. He had a negative KOH preparation of hair from the affected scalp, but his scalp examination still demonstrated scattered papules and follicular plugs.

The patient was then deployed and lost to follow-up for 7 months during which time he was solely using topicals. He represented complaining of persistent pruritus of the scalp. KOH preparation was negative, so he was placed on a regimen of itraconazole 200 mg daily for 1 week each month for 3 months while awaiting fungal culture which was also negative. He was again lost to follow-up for 10 months, but represented due to scalp pruritus. His hair regrowth had been maintained for 1 year and 5 months. Physical examination demonstrated small subcentimeter patchy areas of alopecia without erythema, scale or pustules. Two erythematous papules remained in the patches of alopecia (Fig. 6). A KOH preparation was negative, and a 4 mm punch biopsy of one of the persistent papules on the vertex of his scalp revealed dermal fibrosis with an entrapped hair shaft which was negative for fungal stains.

Discussion

Tinea capitis is a common scalp dermatosis in the prepubertal population, affecting ages 3–7 years.¹ Tinea capitis is rarely seen in postpubertal individuals, but has been described in this population^{1–5} and is reported to be more common in women, African–Americans, large families, crowded living environments and persons of lower socioeconomic status. Transmission occurs via infected individuals and their shed hair, animal vectors and fomites.⁶

During the last 50–60 years, the predominant etiologic agent of tinea capitis has shifted away from *Microsporum audouinii* and *M. canis* towards *T. tonsurans*, now the preponderant agent of tinea capitis in North and Central America. This shift is thought to be due in part to the sensitivity of *Microsporum* spp. to griseofulvin treatment, vigilant school surveillance and to the importation of *T. tonsurans* by people emigrating from geographic areas where it had been the prominent cause of tinea capitis.^{1,4}

The clinical manifestations of infection with *T. tonsurans* can be variable. Traditionally, it causes the classically described ‘black dot’ tinea capitis where patches of alopecia are found with weakened hairs that break at or below the surface of the scalp, leading to the appearance of black dots. In the seborrheic distribution tinea capitis, there may be only minimal hair loss with or without mild scalp erythema, scale and pruritus. Very rarely, *T. tonsurans* can lead to an inflammatory tinea capitis with unpredictable signs and bizarre presentations, often leading to misdiagnoses of impetigo, folliculitis, recurrent furunculosis, pyoderma, folliculitis decalvans, tufted hair folliculitis or dissecting cellulitis of the scalp.^{1,3,7–14} The acute, pustular and suppurative appearance of the scalp is also known as kerion.⁴ Diagnosis of tinea capitis due to *T. tonsurans* is sometimes complicated because its clinical presentation is often on a spectrum of severity and infected hairs do not fluoresce yellow-green under Wood’s light examination.³ Diagnosis must be confirmed by KOH preparation

of infected hairs or by fungal culture. Many hair stubs should be submitted to be inoculated on appropriate selective medium. Because *T. tonsurans* is also a common causative agent of tinea corporis, a thorough physical examination is critical to gain clues in the diagnosis of scalp disease. In addition, *T. tonsurans* causes an endothrix type of hair invasion which can often be readily appreciated if a biopsy of affected scalp is submitted for histopathological evaluation which can often confirm the diagnosis before fungal culture results are obtained.

Inflammatory tinea capitis is thought to be due to a hypersensitivity reaction to the dermatophytic infection.^{1,15} Four varieties of inflammatory tinea capitis have been characterised: favus, kerion, dermatophytic or Majocchi's granuloma and mycetoma.¹⁶ The type of inflammatory tinea capitis seen in this case, kerion, is characterised by a suppurative and painful carbunclelike boggy plaque of the scalp and is often associated with purulent drainage and regional lymphadenopathy.¹⁶ Histopathologically, kerion is distinguished by a perifollicular inflammatory response which varies in degree of intensity based on the patient's own immune response as well as the causative fungal species.¹⁵ Arenas *et al.* described five histopathological categories of inflammatory patterns of kerions. These patterns vary from an initial mild lymphocytic perifollicular infiltrate to involvement of the follicle itself with a mixed infiltrate and fungal elements. More intense inflammatory patterns demonstrate a mixed infiltrate of lymphocytes, neutrophils and plasma cells extending into the surrounding dermis leading to suppuration and follicular destruction. Ultimately, histiocytes and multinucleated giant cells can be found filling the dermis with a granulomatous inflammation. A very inflammatory kerion may likely result in fibrosis, or increased collagen in the dermis, with destruction of follicles; thus leading to a scarring alopecia.^{15,16}

The mainstay of treatment for tinea capitis caused by *T. tonsurans* is oral antifungals. Griseofulvin, terbinafine, fluconazole and itraconazole have all been used successfully to treat tinea capitis due to *T. tonsurans*.^{1,3,6,16} It is imperative to treat for at least 6–8 weeks and ideally to confirm cure with clinical examination, KOH preparation and scalp fungal cultures to ensure the condition has resolved and testing is negative for persistent fungal infection. Inflammatory tinea capitis due to *T. tonsurans* should additionally be cultured to rule out bacterial superinfection, and the scalp should be kept clean to prevent this complication. Adjunct therapies can include topical agents, such as antifungal shampoos. In addition, strong consideration should be given to using a short course of systemic corticosteroids if the inflammatory tinea capitis is particularly severe to prevent permanent alopecia and scarring.²

Our patient's scalp eruption, initially misdiagnosed and treated as dissecting cellulitis, was later appropriately diagnosed as inflammatory tinea capitis (kerion) using KOH preparation, histopathological evaluation and a positive fungal culture. Appropriate treatment with both an oral and topical antifungal regimen led to clearance of his scalp eruption with regrowth of 95% of his hair in the affected region. His clinical outcome was complicated by lack of follow-up as well as the rigorous deployment schedule of this active duty male.

In conclusion, inflammatory tinea capitis in postpubertal individuals is uncommon, often presents peculiarly, and therefore is commonly misdiagnosed. This case adds to the literature

of the few known cases of inflammatory tinea capitis mimicking dissecting cellulitis. It demonstrates the need for a high index of suspicion in any adult with inflammatory alopecia to identify the condition early and begin therapy promptly to prevent the potentially devastating longterm complication of scarring alopecia.

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Figure 1.
Large plaque of patchy alopecia on the right occipital scalp extending to the right temporo-parietal scalp with black dots, broken hairs, follicular plugs, doll's hairs and peripherally located pustules and cysts.

Figure 2. Large annular plaque on the right arm with scattered and peripheral 2–3 mm erythematous papules, an advancing, scaly border and postinflammatory hyperpigmentation.

Figure 3.
Photomicrograph demonstrating endothrix tinea capitis (4009).

Figure 4.
Photomicrograph of scalp biopsy showing a brisk, chronic, mixed inflammatory infiltrate perifollicularly and in the deep dermis (409).

Figure 5.
Regrowth of approximately 95% of hair after 7 months of therapy.

Figure 6.
Small patchy areas of alopecia.