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Successful Terbinafine Treatment for a Girl with Kerion Caused by a *Microsporum Canis* Infection

Chien-Ming Lin[1], Chih-Chien Wang[1], Wei-Hsuan Huang[2], Chi-Chieh Lai[1], Fu-Ming Wang[1], and Shin-Nan Cheng[1*]

[1]Department of Pediatrics; [2]Department of Dermatology, Tri-Service General Hospital, National Defense Medical Center, Taipei, Taiwan, Republic of China

Oral griseofulvin is considered the 'gold standard' therapy for patients with kerion because it can interfere with the synthesis of proteins, cell walls and nucleic acids in growing dermatophytes including *Trichophyton*, *Microsporum* and *Epidermophyton*. Although cure rates of griseofulvin for *Microsporum canis* infections are significantly better than for terbinafine, the longer therapeutic course of griseofulvin causes poor compliance among infected children. We report a 2-year-old Chinese girl with kerion caused by *M canis*. She exhibited good clinical responses to 7 weeks of treatment with oral terbinafine (4 mg/kg per day) without any obvious adverse events. During 6 months of follow-up, her scalp lesion cleared completely with new hair regrowth and the laboratory evaluation of hematology and biochemistry study including liver transaminases was normal.

Key words: griseofulvin, kerion, Trichophyton, Microsporum, Epidermophyton, terbinafine

INTRODUCTION

Tinea capitis is a fungal infectious process involving the scalp and associated hair, which is caused by 4 main genera of dermatophytes: *Trichophyton (T)*, *Microsporum (M)*, *Epidermophyton (E)* and *Arthroderma (A)*.[1] These fungi can be classified by their host preference and natural habitat as anthropophilic (humans), geophilic (soil) or zoophilic (animals). Six major clinical patterns of tinea capitis have been outlined including: (1) a seborrheic form that is scaling, often without noticeable hair loss; (2) a pustular, crusted pattern, either localized or more diffuse; (3) a black dot variety characterized by small black dots within areas of alopecia; (4) a kerion presenting with a boggy, oozing inflammation of the scalp; (5) scaly annular patches and (6) gray patch alopecia. However, kerion, an infrequent type of tinea capitis, is the result of a severe inflammatory response to dermatophytes and occurs almost in children. Because kerion with its pus-filled swellings can look like bacterial abscesses, the gold standard examination—fungal culture—plays a crucial role in early recognizing the potential infection of dermatophytes.

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*Corresponding author: Shin-Nan Cheng, Department of Pediatrics, Tri-Service General Hospital, National Defense Medical Center, No. 325, Sec. 2, Cheng-gong Rd, Taipei 114, Taiwan, Republic of China. Tel: +886-02-87927025; Fax: +886-02-87927293; E-mail: Pedcsn@yahoo.com.tw

CASE REPORT

A 2-year-old healthy Chinese girl presented with a 2-week history of an erythematous, boggy, painful mass with abscesses over the left parietal and temporal regions of scalp. Initially, she received a 1-week course of oral and topical antibiotics, but the cutaneous manifestations worsened. About 7 days after the development of the scalp lesion, multiple scattered erythematous pruritic papules over her posterior neck, postauricular region and extremities were prominent (Figure 1). These papules were sterile, as verified by bacterial culture. A history of contact with a dog about 3 weeks previously was also noted.

On admission, physical examination revealed a solitary painful, elevated, boggy, granulomatous mass measuring 6.5×4 cm, studded with several pustules and exudates (Figure 2). Hairs on the infected area were brittle and broken. Bilateral cervical lymphadenopathy with tenderness was also noted. The remainder of the physical examination was unremarkable.

Complete blood cell counts, C-reactive protein and hepatic enzyme levels were normal. Bacterial culture of

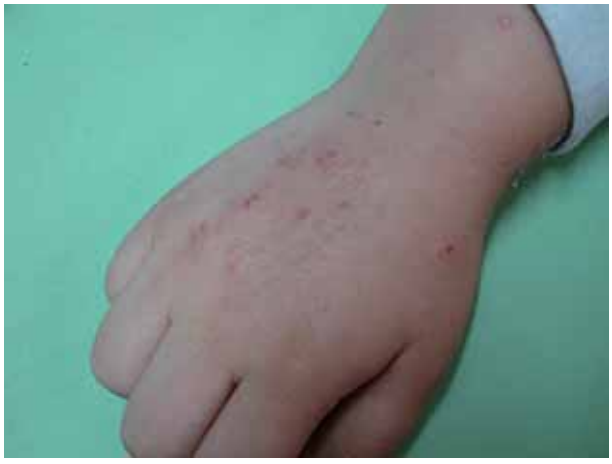

Fig. 1 The ID reaction showed multiple scattered erythematous pruritic papules on the dorsal part of right hand.

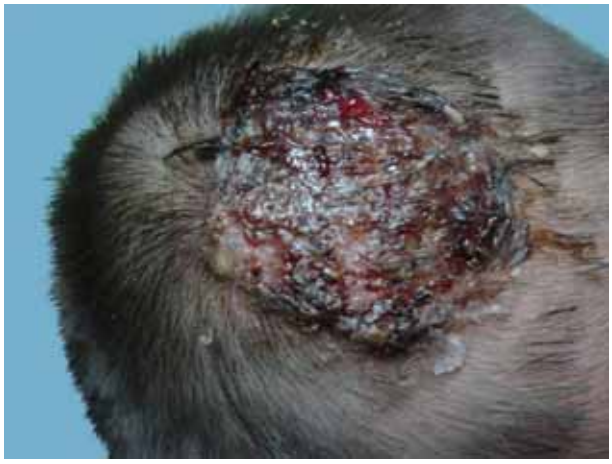

Fig. 2 At admission, suppurative discharge from a crusty matted mass with hairless desquamating areas around left parietal and temporal regions.

skin swabs disclosed no microorganism growth. Microscopic examination of the patient's broken hairs after treating them with 10% potassium hydroxide plus Parker's blue-black permanent ink solution showed profuse spores on the hair with an ectothrix pattern. Mycological culture with Sabouraud dextrose agar showed a profuse *M canis* infestation. Oral terbinafine (4 mg/kg per day) was immediately used for this patient after a definitive diagnosis of kerion with immunodiffusion (id) reaction. In the second week of treatment, there was significant improvement in the scalp lesions, as well as the secondary maculopapular eruption of her neck and extremities. After completing 7 weeks of therapy, fungal culture of the previously infected scalp region was repeated and this revealed no microorganism growth. Both the kerion and the id reaction disappeared. However, several patches of alopecia remained on the previous lesion sites (Figure 3). During 6 months of follow-up, repeated complete blood cell counts and hepatic enzymes were all within normal range and hair regrowth was noted.

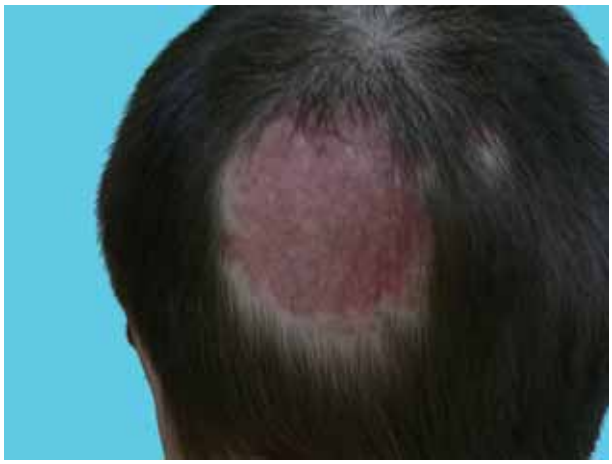

Fig. 3 After 7 weeks therapy of terbinafine, patchy alopecia on the previous lesional site was noted.

DISCUSSION

The clinical diagnosis of tinea capitis can be challenging, as symptoms can vary from minimal pruritus with no hair loss to severe tenderness, purulence and permanent scarring in inflammatory kerion lesions. That might explain why so many clinicians treated tinea capitis as cellulitis. The id reaction, a delayed hypersensitivity response resulting from circulating fungal antigens, is characterized by a distant skin manifestation of an established fungal infection. This secondary eruption is a papular or papulovesicular rash that is erythematous or skin colored, typically pronounced on the face, neck and upper chest. Furthermore, the id reaction is often associated with tinea pedis, but is rare in cases of tinea capitis except for severe kerion caused by zoophilic dermatophytes such as *Trichophyton verrucosum*.[2] Because the main transmission pathway of *M canis* is also through direct contact with animals, the delayed hypersensitivity reaction in this patient was presumably related to kerion caused by *M canis*.[3-5]

Tinea capitis is a common superficial fungal infection of the scalp and hair shaft in prepubescent children, particular in those of African descent.[3] It often associates with positive family history, contact with pets, crowded

living conditions, poor socioeconomic groups and is endemic in many developing countries.[6,7] In North America and the United Kingdom, tinea capitis is caused by *Trichophyton tonsurans* in over 90% of cases, whereas *M canis*, commonly found in pets, is a significant pathogen in Europe, especially in Central Europe and the Mediterranean.[8] With respect to kerion, causative species of dermatophytes not only vary from country to country but from region to region. This distribution is influenced by climate, temperature, relative humidity, geographic location and the genetic susceptibility of the host, as well by as the virulence of the strain.[2,9] Of interest, kerion with a severe id reaction caused by *M canis* was first reported in Chinese children.

Kerion is considered the most exaggerated cell-mediated response to the fungus. It is boggy, tender and pustular, and is often confused with bacterial abscesses.[10] From the aspect of treatment, surgical intervention for kerion is the wrong therapy, as it can provide no therapeutic cure in the absence of adequate antimicrobial chemotherapy.[11] Even though griseofulvin (a fungistatic agent) remains the mainstay of therapy for children with tinea capitis, there has been a substantial increase in the therapeutic dosage required as treatment failure has become more common.[3,8] On the other hand, terbinafine (a fungicidal agent) achieves high concentrations in the hair and stratum corneum that persist for several weeks following drug administration.[12] Terbinafine has been studied to determine whether its shorter therapeutic duration is safe and efficacious for children with kerion. Consequently, 2 to 4 weeks of terbinafine treatment have been shown to be at least as effective as 6 to 8 weeks of griseofulvin treatment for the treatment of *Trichophyton* infections.[13] Although griseofulvin is likely to be superior to terbinafine for treating the rare infections caused by *Microsporum* species,[13] Silm et al. reported that a 4-week treatment of terbinafine for tinea capitis caused by *M canis* may reach an effective cure rate of 65.4%.[14] In support of all these findings, this Chinese girl with kerion and an id reaction caused by *M canis* exhibited a good therapeutic response to systemic terbinafine. Furthermore, another study reported that approximately 50% of affected children treated with griseofulvin and terbinafine show adverse reactions such as nasopharyngitis, headache, pyrexia, gastrointestinal disturbances and rash.[15] Nevertheless, our patient did not manifest any deleterious responses or deterioration in hepatic enzyme profile after a complete 7-week course of systemic terbinafine. Although this girl with kerion caused by *M canis* was treated successfully with terbinafine, a larger-scale prospective study is still needed to confirm whether the shorter treatment duration of terbinafine is an effective and well-tolerated alternative in such cases.[16]

Asymptomatic subjects can carry the fungus for months as demonstrated by repeated cultures, and represent a natural reservoir of the infection. These unrecognized carriers are important for the diffusion of tinea capitis and their existence has largely been documented for *M canis*.[17,18] Robert and Friedlander have also substantiated that up to 50% of children with tinea capitis have at least 1 family member with a positive fungal culture.[8] Herein, the treatment of tinea capitis consists of curing the infected patients and controlling any spread of the infection to non-infected children in the same household or school. Because *M canis* is quite difficult to treat due to its ectothrix properties, the shorter therapeutic course of terbinafine rather than that of griseofulvin seems to be a convenient and effective alternative for children with refractory *M canis* infection. Moreover, the combination of systemic terbinafine and adjunctive therapy such as 2% ketoconazole shampoo or products with 1% selenium sulfide should be considered to reduce the fungal burden and transmission.[19-21]

In conclusion, this Chinese girl diagnosed with kerion and severe id reaction showed a good response to 7 weeks of therapy with systemic terbinafine. This adds to the evidence that systemic terbinafine may be efficacious for kerion caused by *M canis*, particularly in poorly compliant children. Any abscess-like lesions on the scalp should be investigated carefully by dermatologists and pediatricians to evaluate whether they are the result of a dermatophytic infection so that the appropriate treatment can be initiated.

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