Phototoxic reaction to oral terbinafine due to Tinea capitis in a child

Dear Editor,

We report the case of an 18-month-old boy who developed a phototoxic skin reaction to terbinafine on his scalp, ears, and face in the form of disseminated erythematous plaques, which resembled subacute lupus erythematosus (SCLE) in their clinical presentation. Skin changes appeared a short time after the boy was exposed to sunlight during the period of time when he was treated with oral terbinafine due to *Microsporum canis* fungal scalp infection.

Tinea capitis is a common dermatophyte infection primarily affecting prepubertal children (1). *Microsporum canis* remains the predominant causative organism in many countries of the Mediterranean basin, the most important dermatophyte carriers being stray cats and dogs. Systemic therapy is required for treatment because topical antifungal agents do not penetrate down to the deepest part of the hair follicle (2). Terbinafine is commonly used in the treatment of microsporosis, as its fungicidal action permits short periods of treatment (3,4).

The first skin changes occurred in the parietal scalp region in the form of round scaly alopecia, with the presence of unevenly broken hairs and enlarged regional lymph nodes (Figure 1). Diagnosis of fungal infection included clinical assessment and Wood's light examination, which revealed green-yellow fluorescence on the lesional scalp region. Fungal culture identification was performed according to conventional methods, revealing fungal culture positive for dermatophytes from the genus *Microsporum canis*. The boy had a history of contact with a cat. Systemic therapy with the oral antifungal drug terbinafine was administered at a dose of 62.5 mg per day (5 mg/kg), with topical application of antifungal cream (miconazole), 10% Ichthyol cream in the evening, and antifungal shampoo (ketoconazole) twice a week. After two weeks of therapy, we observed initial regression of scalp lesions. Oral terbinafine was well-tolerated, and the patient did not experience any side-effects. Laboratory findings included liver function tests and were within normal ranges. At this point, the oral dose of terbinafine was increased to 125 mg per day (10 mg/kg) at a revised schedule according to body weight: 10-25 kg, 125 mg/day (5). Approximately five weeks after starting the treatment with oral terbinafine, after the boy was exposed to the sun, acute disseminated erythematosus lesions appeared on the face and scalp.

Clinical presentation of the lesions and acute onset during exposure to sunlight raised the suspicion of a phototoxic reaction to terbinafine (Figure 2). The patient was not taking any other medication at that time, had no history of drug or food allergies, and had

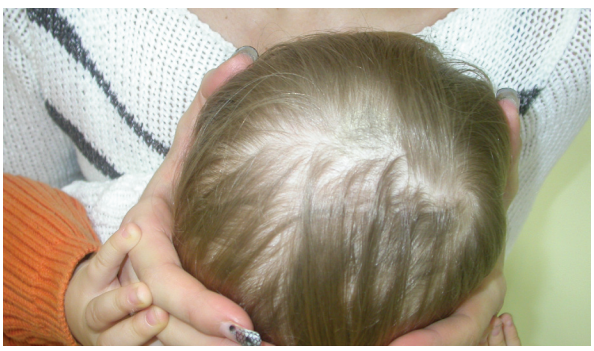

Figure 1. The first skin changes occurred in the parietal scalp region in the form of round scaly alopecia, with the presence of unevenly broken hairs and enlarged regional lymph nodes.

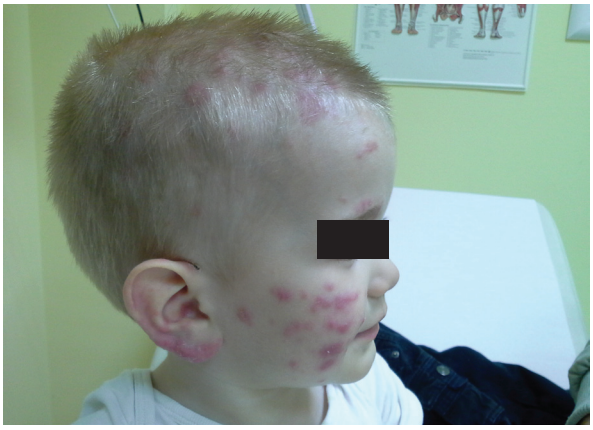

Figure 2. Clinical presentation of lesions and acute onset during exposure to sunlight raised the suspicion of a phototoxic reaction to terbinafine.

not previously experienced photosensitive skin reactions. Due to the inflamed skin changes resembling subacute lupus and photosensitivity, an immunological assay tests were also performed. Due to the young age of the patient, no skin biopsy or photo-patch test was performed. Despite the recent skin changes and suspicions of phototoxicity secondary to medication, oral terbinafine treatment was continued due to persistently positive mycological results (Wood's light and fungal culture). The parents were advised to stricly prevent the child from being exposed to sunlight. Systemic treatment with terbinafine was completed after three months of therapy, once a second negative fungal culture was obtained and clinical regression of the lesions was achieved. The acute disseminated inflamed phototoxic skin lesions were treated with topical application of corticosteroid cream (mometazone furoate), strict avoidance of sun exposure, and use of sun block cream. Complete regression of inflammatory skin changes occurred within a few days after introducing treatment with corticosteroid cream. Immunological assay results were completely negative (ANA, ClC-IgG, C3, anti-ds-DNA, antiRo/SS-A, antiLa/SS-B, antiSm1, antiU1RNP, antihistone antibodies, anti PmScl, anti PCNA).

When faced with a rash that appeared after sun exposure, exogenous photosensitization is the most likely cause, especially when clinical signs appear suddenly after the introduction of terbinafine, and when there is no history of previous solar allergy.

Photosensitive drug eruptions, including both phototoxicity and photoallergy, have been reported for a number of systemic medications. Photosensitivity due to terbinafine appears to be very rare. Until now, only isolated cases of terbinafine-induced lupus erythematosus (LE), subacute cutaneous LE, or terbinafine-exacerbated LE have been reported (6).

Phototoxic reactions have been very rarely reported as a side-effect of terbinafine (e.g. photodermatitis, allergic photosensitivity and polymorphic light eruptions) (7). In the literature, we found only one patient who had a photoallergic reaction to oral terbinafine, and which was confirmed by the author by carrying out a photo-patch test (8). Cases of terbinafine-induced subacute cutaneous lupus erythematosus (SCLE) and exacerbation of systemic lupus erythematosus by terbinafine have been reported, with positive immunoassays and positive anti-histone antibodies (9).

Further monitoring of the present patient revealed no recurrence of inflammatory skin changes, while two small residual alopecic scars remained on the scalp as a result of a deep mycoses skin infection. The outbreak of inflamed skin changes during exposure to sunlight, at the time the patient was being given terbinafine, with rapid regression of skin inflammation after local corticosteroid treatment and negative immunoassay results, enabled a diagnosis of a phototoxic reaction to terbinafine to be established. Terbinafine is rarely mentioned in the literature as being photosensitizing, but this clinical case provides an example and highlights the importance of pharmaceutical advice about this side-effect, especially in the summer season (7).

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