A Pediatric Case of Bullous Tinea Pedis Caused by *Trichophyton violaceum* in the United States

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**Abstract**

*Trichophyton violaceum* is an uncommon cause of tinea pedis. We report a case of bullous tinea pedis caused *T. violaceum* in a 7-year-old otherwise healthy child living in the United States. She achieved clinical recovery with a 1-month course of oral terbinafine.

**Keywords:** *Trichophyton violaceum*, Bullous tinea pedis

**Introduction**

Tinea pedis is a superficial dermatophyte infection of the plantar and interdigital webspaces of the feet. There are four clinical presentations of tinea pedis with the interdigital type being the most common variant: 1) Moccasin type presenting as diffuse scaling and hyperkeratotic plaques on plantar surfaces of feet; 2) Interdigital type presenting as scaling and maceration between toe webspaces; 3) Bullous type presenting as vesicles and bullae on the medial foot; and 4) Ulcerative type presenting as ulcerations or erosions as an exacerbation of the interdigital variant [1]. Bullous tinea pedis is caused by *Trichophyton mentagrophytes*, *T. mentagrophytes* var. *interdigitale*, and *T. mentagrophytes* var. *rubrum* [2]. This type of tinea pedis is uncommon but has been described in both adults and children [3]. Another less common causes of tinea pedis include *Trichophyton violaceum*, a species that is endemic to Africa [4] and Europe [5]. We report a case of bullous tinea pedis caused by *T. violaceum* in a child living in the United States.

**Case Report**

A 7 year old Caucasian girl was referred to our dermatology clinic with a complaint of pruritic scaly plaques on her left foot for three months. The area subsequently developed vesicles (Figure 1), and within a couple of months, a rash of a different morphology of papules and erythematous plaques spread up to her buttocks, trunk, and elbows. She denied fever, chills, malaise, sore throat, oral lesions, nausea, or diarrhea. There was no history of travel outside the United States or contact with anyone who had recently traveled outside the U.S. On physical exam, she was a well-appearing, well-developed, playful child. There was no cervical lymphadenopathy. Skin exam finding was consistent with a large scaly, crusted thin plaque with an ill-defined border on the plantar aspect of the left foot. Laboratory results showed normal CBC, chemistry panel, ESR, and ANA. Anti-streptolysin O titer was positive at 734 with negative strep throat swab suggestive of prior streptococcal infection. She was given a course of oral prednisone and trimethoprim-sulfamethoxazole for 7 days for presumed impetiginized eczema with resolution of the ID reaction on her body and arms. However, despite the use of tacrolimus 0.03% ointment, topical triamcinolone cream, and clobetasol ointment, the pruritic rash on her left foot continued to spread (Figure 2). A fungal culture was obtained at that time from the foot lesion and the result was consistent with *T. violaceum*. Per family’s request, an x-ray of the foot was obtained to rule out foreign body, and the result of the imaging study was normal. She was then instructed to stop all topical steroid creams and to begin oral terbinafine 125 mg daily along with topical application of ciclopirox cream to the area for 30 days. At the 4-week follow up appointment, clinical recovery was confirmed without evidence of active infection (Figure 3).

**Figure 1:** Vesicles grouped into bulla on the left sole of a 7 year old Caucasian girl.
Discussion

*T. violaceum* is an anthropophilic dermatophyte which can cause endoethrix type of tinea capitis infection in children and adolescents [6]. It is endemic to parts of Europe [5], Asia [7], and North Africa [4] with Ethiopia having the highest prevalence [8,9]. However, this organism is emerging as a frequent cause of tinea capitis in Milan, Italy secondary to the immigrant population from Africa [10]. A recent epidemiologic study reviewing cases of tinea capitis among children in Columbus, Ohio from 2001-2006 revealed eight out of 189 (4.2%) cases were positive for *T. violaceum* [11]. This finding is also confirms the trend towards increasing number of *T. violaceum* in non-African cities. *T. violaceum* can also cause tinea corporis, tinea pedis, tinea manuum, and onychomycosis [10]. However, compared to tinea capitis, tinea pedis caused by *T. violaceum* remains rare with only one case report of a Southeast Asian immigrant with tinea pedis caused by *T. violaceum* in the U.S. [12]. Disease activity tends to be indolent as reported by a case of a Chinese woman suffering from tinea capitis, tinea corporis, and onychomycosis for over 40 years caused by *T. violaceum* [13]. The source of our patient’s infection is unclear. She is a healthy, immunocompetent child who may have acquired the infectious organism from a carrier. A study looking at asymptomatic scalp carriers of dermatophytes in Greece revealed that *T. violaceum* was the most common organism found [14].

Regarding treatment, a recent study examined *in vitro* antifungal susceptibility of *T. violaceum* and showed that posaconazole, terbinafine, and voriconazole are the most potent antifungal agents [15]. Our patient responded well to the oral terbinafine and she obtained clinical cure with the treatment. When approaching patients with an inflammatory and vesicular cutaneous eruptions on the feet, we should consider differential diagnoses including dyshidrostatic eczema, palmar plantar psoriasis, secondary syphilis, juvenile plantar dermatosis, bacterial infections, and erythrasma. Aside from a thorough clinical evaluation, mycological examination with KOH and fungal culture should be considered in patients with inflammatory vesicular eruption of the feet. To our best knowledge, this is the second reported case of tinea pedis caused by *T. violaceum* in the United States.

References


