To the Editor:
A 17-year-old adolescent girl presented to the dermatology clinic with a tender pruritic rash on the left wrist that was spreading to the bilateral arms and legs of several years’ duration. An area of a prior biopsy on the left wrist was healing well with use of petroleum jelly and halcinonide cream. The patient denied any constitutional symptoms.

Physical examination revealed numerous erythematous papules coalescing into plaques on the bilateral anterior and posterior arms and legs, including some erythematous macules and papules on the palms and soles. The original area of involvement on the left dorsal medial wrist demonstrated a background of erythema with overlying peripheral scaling and resolving violaceous to erythematous papules with signs of serosanguineous crusting (Figure 1). Scattered perifollicular erythema was present on the posterior aspects of the bilateral thighs and arms (Figure 2). Baseline complete blood cell count and complete metabolic panel were within reference range.

Clinical histopathology showed evidence of a pustular superficial dermatophyte infection, and Grocott-Gomori methenamine-silver stain demonstrated numerous fungal hyphae within subcorneal pustules, indicating pustular tinea. Based on the clinicopathologic correlation, the initial presentation was diagnosed as pustular tinea of the entire left wrist, followed by a generalized id reaction 1 week later.

The patient was prescribed oral terbinafine 250 mg once daily to treat the diffuse involvement of the pustular tinea as well as once-daily oral cetirizine, once-daily oral diphenhydramine, a topical emollient, and a topical non-steroidal antipruritic gel.

PRACTICE POINTS
• Id reactions, or autoeczematization, can occur secondary to dermatophyte infections, possibly due to a hypersensitivity reaction to the fungus. These eruptions can occur in many forms of tinea and in a variety of clinical presentations.
• Treatment is based on clearance of the original dermatophyte infection.

FIGURE 1. A, Left dorsal medial wrist with erythematous papules coalescing into plaques. B, Papules and plaques displaying overlying peripheral scale were noted.
Tinea is a superficial fungal infection commonly caused by the dermatophytes *Epidermophyton*, *Trichophyton*, and *Microsporum*. It has a variety of clinical presentations based on the anatomic location, including tinea capitis (hair/scalp), tinea pedis (feet), tinea corporis (face/trunk/extremities), tinea cruris (groin), and tinea unguium (nails). Tinea infections occur in the stratum corneum, hair, and nails, thriving on dead keratin in these areas. Tinea corporis usually appears as an erythematous ring-shaped lesion with a scaly border, but atypical cases presenting with vesicles, pustules, and bullae also have been reported. Additionally, secondary eruptions called id reactions, or autoeczematization, can present in the setting of dermatophyte infections. Such outbreaks may be due to a delayed hypersensitivity reaction to the fungal antigens. Id reactions can manifest in many forms of tinea with patients generally exhibiting pruritic papulovesicular lesions that can present far from the site of origin.

Patients with id reactions can have atypical and varied presentations. In a case of id reaction due to tinea corporis, a patient presented with vesicles and pustules that grew in number and coalesced to form annular lesions. A case of an id reaction caused by tinea pedis also noted the presence of pustules, which are atypical in this form of tinea. In another case of tinea pedis, a generalized id reaction was noted, illustrating that such eruptions do not necessarily appear at the original site of infection. Additionally, in a rare presentation of tinea invading the nares, a patient developed an erythema multiforme id reaction. Id reactions also were noted in 14 patients with refractory otitis externa, illustrating the ability of this fungal infection to persist and infect distant locations.

Because the differential diagnoses for tinea infection are extensive, pathology or laboratory confirmation is necessary for diagnosis, and potassium hydroxide preparation often is used to diagnose dermatophyte infections. Additionally, the possibility of a hypersensitivity drug rash should remain in the differential if the patient received allergy-inducing medications prior to the outbreak, which may in turn complicate the diagnosis.

Tinea infections typically can be treated with topical antifungals such as terbinafine, butenafine, and luliconazole; however, more involved cases may require oral antifungal treatment. Systemic treatment of tinea corporis includes itraconazole, terbinafine, and fluconazole, all of which exhibit fewer side effects and greater efficacy when compared to griseofulvin.

Treatment of id reactions centers on the proper clearance of the dermatophyte infection, and treatment with oral antifungals generally is sufficient. In the cases of id reaction in patients with refractory otitis, some success was achieved with treatment involving immunotherapy with dermatophyte and dust mite allergen extracts coupled with a yeast elimination diet. In acute id reactions, topical corticosteroids and antipruritic agents can be applied. Rarely, systemic glucocorticoids are required, such as in cases in which the id reaction persists despite proper treatment of the primary infection.

**REFERENCES**