

Itchy Vesicular Rash

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A 38-year-old woman presented with a rash of 5 days' duration that initially appeared on the wrists after playing with her kitten, with subsequent involvement of the chest, back, abdomen, and upper and lower extremities. Physical examination revealed multiple annular plaques with raised erythematous borders, rare peripheral vesicles, and superficial central scaling. Extreme pruritus accompanied the plaques, both of which developed after playing with her kitten. The patient noted that all lesions on the upper extremities evolved in areas subject to deep puncture while more superficially excoriated areas were unaffected. She denied any other prior skin conditions and had received a 5-day course of azithromycin without improvement prior to presentation; triamcinolone ointment 0.1% had provided only temporary relief. Primary care providers prescribed a short course of oral prednisone; however, she did not start it prior to presentation.

WHAT'S YOUR DIAGNOSIS?

- benign inoculation lymphoreticulosis (cat scratch disease)
- bullous fixed drug reaction
- dermatitis herpetiformis
- linear IgA bullous dermatosis
- tinea corporis bullosa

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THE DIAGNOSIS: Tinea Corporis Bullosa

At the time of presentation, a potassium hydroxide (KOH) preparation, fungal culture, and punch biopsy of the right ventral wrist was performed. The KOH preparation was positive for fungal hyphae characteristic of dermatophyte infections. Histologically, the biopsy showed intraepidermal and subepidermal blisters with neutrophil- and lymphocyte-rich contents (Figure 1). Fungal hyphae and spores were present within the stratum corneum and superficial epidermis (Figure 2), and fungal cultures grew *Microsporum canis*. The extent of the rash (upper and lower extremities, chest, and back), positive fungal culture, and KOH preparation all supported the diagnosis of tinea corporis bullosa, which was confirmed with biopsy. Oral prednisone use was discouraged and triamcinolone ointment was discontinued given that inappropriate treatment with steroids in the setting of fungal infection suppresses an inflammatory response and alters clinical appearance, obviating the persistent underlying infection.

Tinea corporis bullosa is a rare superficial dermatophyte fungal infection that often is acquired by close person-to-person contact or contact with domestic animals. The infection begins as a circular pruritic plaque, generally with raised borders, which may be erythematous or hyperpigmented. By definition, tinea corporis occurs in sites other than the face, feet, hands, or groin area. Bullae formation is thought to be secondary to a delayed hypersensitivity reaction provoked by the presence of a dermatophyte antigen.¹

Linear IgA bullous dermatosis is an immune-mediated disease characterized by IgA deposition at the dermoepidermal junction. Linear IgA bullous dermatosis classically presents as widespread tense vesicles in an arciform or annular pattern. Mucosal involvement is

common and typically presents with erosions, ulcerations, and scarring.² Given the absence of mucosal involvement in our patient and a positive KOH preparation, linear IgA bullous dermatosis was an unlikely diagnosis.

Benign inoculation lymphoreticulosis, more commonly known as cat scratch disease (CSD), is a *Bartonella henselae* infection that results from a cat scratch or bite. Cat scratch disease can present as localized cutaneous and nodal involvement (lymphadenopathy) near the site of inoculation, or it may present as disseminated disease. Cutaneous lesions generally progress through vesicular, erythematous, and papular phases. Regional lymphadenopathy proximal to the inoculation site is the hallmark of CSD.³ Given the absence of lymphadenopathy in our patient as well as the sporadic distribution of lesions, CSD was an unlikely diagnosis.

Dermatitis herpetiformis (DH) is an autoimmune disorder with cutaneous manifestations of gluten sensitivity. Dermatitis herpetiformis presents as extremely pruritic papules and vesicles arranged in groups on areas such as the elbows, dorsal aspects of the forearms, knees, scalp, back, and buttocks. Most patients with DH have celiac disease or small bowel disease related to gluten sensitivity.⁴ Given our patient's acute presentation in adulthood and lack of gluten sensitivity, DH was an unlikely diagnosis.

Bullous fixed drug reaction is a cutaneous eruption that typically presents in the setting of exposure to an offending drug/agent. Drug reactions can have various cutaneous presentations, with the most common being pigmented macules that progress into plaques.⁵ Given the isolated nature of our patient's episode and apparent lack of association with medication, bullous fixed drug reaction was an unlikely diagnosis.

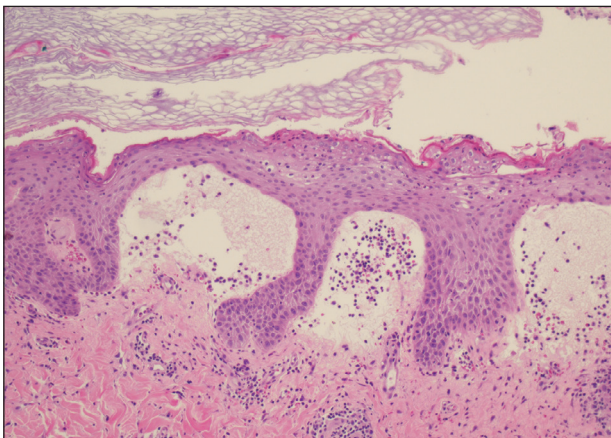


FIGURE 1. Subepidermal blister with neutrophil- and lymphocyte-rich inflammatory infiltrates (H&E, original magnification $\times 10$).

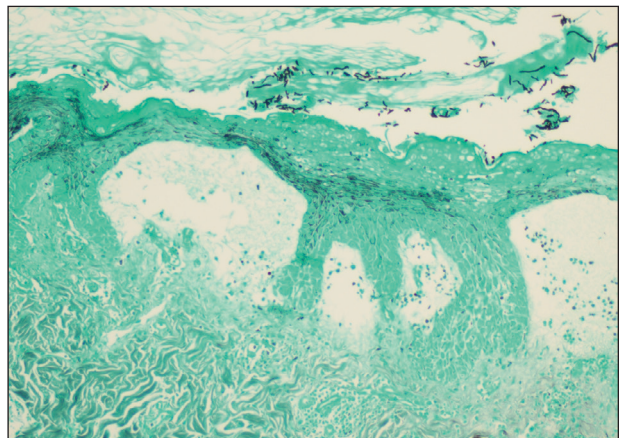


FIGURE 2. Grocott-Gomori methenamine-silver staining showed fungal hyphae invading the stratum corneum (original magnification $\times 10$).

Tinea corporis bullosa is a rare clinical variant of tinea corporis that has only been reported in patients with a history of contact with different animals. There are many causative organisms related to tinea corporis; *Trichophyton rubrum* is the most common etiology of tinea corporis, while tinea corporis due to close contact with domesticated animals often is caused by *M canis*.⁶ The immunoinhibitory properties of the mannans in the fungal cell wall allow the organisms to adhere to the skin prior to invasion. Cutaneous invasion into dead cornified layers of the skin is credited to the proteases, subtilisin-like proteases (subtilases), and keratinases produced by the fungus.¹ There are many different clinical presentations of tinea corporis due to the variability of causative organisms. An annular (ring-shaped) lesion with a central plaque and advancing border is the most typical presentation. Tinea corporis bullosa is characterized by the presence of bullae or vesicles in the borders of the scaly plaque. Rupture of the bullae subsequently leads to erosions and crusts over the plaque.

The diagnosis of tinea corporis bullosa often is clinical if the lesion is typical and can be confirmed using KOH preparation and fungal culture. Once the diagnosis is

confirmed, topical antifungals are the standard treatment approach for localized superficial tinea corporis. Systemic antifungal treatment can be initiated if the lesion is extensive, recurrent, chronic, or unresponsive to topical treatment.¹ Given our patient's characteristic presentation, she was managed with an over-the-counter topical antifungal (terbinafine). The patient's lesions dramatically improved, rendering oral therapy unnecessary. At 1-month follow-up, the rash had nearly resolved.

REFERENCES

1. Leung AK, Lam JM, Leong KF, et al. Tinea corporis: an updated review [published online July 20, 2020]. *Drugs Context*. doi:10.7573/dic.2020-5-6
2. Guide SV, Marinkovich MP. Linear IgA bullous dermatosis. *Clin Dermatol*. 2001;19:719-727.
3. Lamps LW, Scott MA. Cat-scratch disease: historic, clinical, and pathologic perspectives. *Pathology Patterns Reviews*. 2004;121(suppl):S71-S80.
4. Caproni M, Antiga E, Melani L, et al. Guidelines for the diagnosis and treatment of dermatitis herpetiformis. *J Eur Acad Dermatol Venereol*. 2009;23:633-638.
5. Patel S, John AM, Handler MZ, et al. Fixed drug eruptions: an update, emphasizing the potentially lethal generalized bullous fixed drug eruption. *Am J Clin Dermatol*. 2020;21:393-399.
6. Ziemer M, Seyfarth F, Elsner P, et al. Atypical manifestations of tinea corporis. *Mycoses*. 2007;50:31-35.