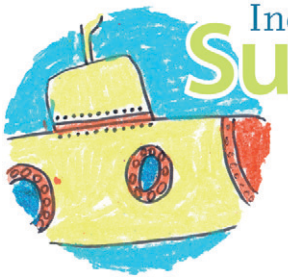

Index of Suspicion

4 Prolonged and Intensely Pruritic Rash on the Hand of a 6-year-old Girl

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AUTHOR DISCLOSURE Drs Ward and Cox have disclosed no financial relationships relevant to this article. This commentary does not contain a discussion of an unapproved/investigative use of a commercial product/device.

PRESENTATION

A 6-year-old girl presents with a 9-month history of a nontender but intensely pruritic rash on her left hand and right index finger. She has been evaluated by numerous physicians; diagnosed as having tinea manuum, eczema, and cellulitis; and treated with antifungal agents, antiyeast agents, topical corticosteroids of varying strengths, and 3 courses of oral antibiotic drugs. The rash improves with application of corticosteroids but reappears as corticosteroids are withdrawn. She has normal growth and development with no systemic symptoms. Her medical history includes eczema, asthma, and seasonal allergies. Besides topical corticosteroids, her current medications include cetirizine and montelukast.

Physical examination reveals a well-appearing, well-developed white girl. Inspection of her left hand reveals a well-defined geometric erythematous plaque spanning the lateral one-third of the palm on the left hand, including the first through third digits (Fig 1). A similar rash is present on the right index finger. There is no tenderness, fissuring, or signs of secondary infection. There are no nail changes. Her face, scalp, neck, ears, flexural creases, including antecubital and popliteal fossae, wrists, and ankles are free of lesions.

After physical examination, the patient requests to use her mother's cell phone. She places the cell phone, which has a plastic cover, in her left hand and supports the phone with her distal palm and second digit. The patient's mother confirms that the patient always holds the cell phone in this fashion.

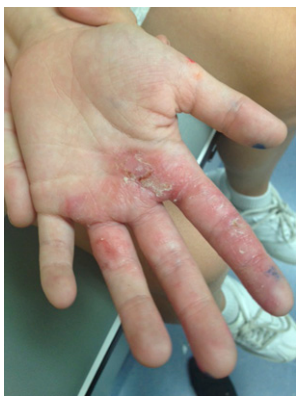

Figure 1. Left hand before treatment and irritant avoidance.

The Case Discussion and Suggested Readings appear with the online version of this article at http://pedsinreview.aappublications.org/content/38/2/97.

DISCUSSION

This patient presented with an asymmetrical, well-demarcated, erythematous plaque that was more pruritic than painful; it was most consistent with irritant contact dermatitis (ICD) or allergic contact dermatitis (ACD). The hallmark of ICD/ACD is resolution on avoidance of exposure to the causative agent with recurrence on subsequent exposure. Eczema is generally symmetrical, with a predilection for the face, hands, wrists, and flexural creases. The patient avoided contact with the cell phone for 30 days and experienced significant improvement of skin lesions (Fig 2) and symptoms. She was then permitted access to the cell phone for up to 30 minutes daily, with reappearance of erythema and dryness in areas that came in contact with the cell phone.

Differential Diagnosis

The differential diagnosis for pruritic eczematous hand lesions includes eczema, fungal infections, ACD, ICD, and psoriasis. Eczema usually presents before age 5 years and is associated with a history of atopic disease as well as dry skin. It has a predilection for flexural surfaces and, as opposed to ACD/ICD, is usually symmetrical. Psoriatic lesions are well-demarcated, salmon-colored plaques with coarse silver scales that can involve the gluteal region, axilla, groin, and often scalp in children. Patients frequently report a family history of the disease. Interestingly, psoriasis causes accumulation of the antimicrobial peptides cathelicidins and β-defensins, leaving those with the disease less susceptible to secondary infection with *Staphylococcus aureas* relative to patients with eczema. Suspected superficial fungal infections appear similar to eczematous lesions and should be evaluated for fungal elements in scrapings in potassium hydroxide (KOH) preparation. History is key in differentiating ACD from ICD. Reaction typically occurs within hours of exposure for ICD, with pain or a burning sensation being more commonly reported; ACD evolves over days, and pruritus is the predominant symptom. Allergic contact dermatitis is typically the better marginated of the 2 conditions. Appearance of rash/itch can take several days for first exposure in ACD, but on reexposure the signs and symptoms typically return rapidly. Irritant contact dermatitis does not classically produce pustules.

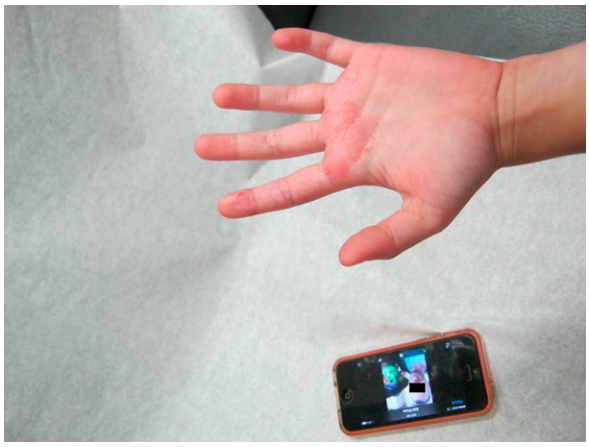

Figure 2. Left hand after treatment and irritant avoidance, with the cell phone in the background.

The Condition

Allergic contact dermatitis is a type IV, T-cell–mediated, delayed hypersensitivity reaction. After sensitization, the rash requires days to develop and, in the absence of the allergen, resolves within weeks. Acute episodes begin with a pruritic papulovesicular rash, progressing to erosions, crusts, and scaling in severe cases. Long-term exposure results in well-developed lichenified plaques with excoriations, fissuring, and possibly secondary infection. Topical corticosteroid therapy blunts the disease progression, as was the case here; thus, a thorough history of the present illness is crucial.

Allergic contact dermatitis requires sensitization by previous exposure and involves 2 phases. The afferent phase (sensitization phase) follows initial exposure. It is characterized by binding of haptens to skin proteins. Haptens are low-molecular-weight, electrophilic substances that penetrate the skin and induce an immune response. The hapten-protein complex is then transported to regional lymph nodes by antigen-presenting cells (typically Langerhans cells and dendritic cells), and T cells specific for the hapten are generated. During the efferent phase (elicitation phase) not only does the hapten bind to skin proteins but endogenous glycolipids also are released, triggering natural killer T cells (NKT cells) to interact with B cells, resulting in the production of hapten-specific immunoglobulin M. This immunoglobulin M ultimately causes movement of effector cells ($CD8^+$ T, NK, and NKT cells) to the challenge site. The skin changes that accompany early ACD are principally due to $CD8^+$ T-cell–directed apoptosis of keratinocytes and to edema and vesiculation due to massive inflammatory infiltrate.

Cell phone dermatitis due to the presence of nickel and chromium is well documented. The cover in question, however, is fully plastic, with no listing of chemicals or compounds on the product insert or company website. Allergic contact dermatitis to cell phone covers has been previously described with reactions to "adhesives, plasticizers and accelerants used during production."

Evaluation and Treatment

Tissue biopsy can be diagnostic, but it is unnecessary when symptoms respond to topical corticosteroids and wax and

wane based on exposure. When ACD/ICD is suspected but not readily apparent by history, patch testing can be useful for diagnosis. The cost and inconvenience to the patient should be weighed against pretest probability. As in our case, avoidance and rechallenge confirmed the diagnosis without biopsy or patch testing.

Management of ACD/ICD is 3-pronged: avoidance, reduction of inflammation, and barrier restoration. Hapten avoidance is paramount to resolution. Decreasing inflammation with topical corticosteroid use reduces symptoms and secondary excoriations. Triamcinolone is a midpotency corticosteroid with excellent efficacy and a reasonable adverse effect profile. Treatment with fluocinonide, a high-potency corticosteroid, is reserved for short-term use (2 weeks) for severe cases. Topical mupirocin use is highly recommended over oral antibiotic drug therapy for secondary bacterial infection. Coupled with avoidance, gentle skin cleansing practices and regular application of petrolatum-based emollients is sufficient. Physical barriers, such as dimethicone, offer modest protection but are not a substitute for avoidance. Treatment with systemic antihistamines and corticosteroids should be reserved for severe cases.

The patient's skin lesions completely resolved with avoidance, emollient use, and 1 week of twice-daily triamcinolone administration. Her symptoms relapsed when she resumed regular use of the cell phone.

Cell phones permeate the lives of the children for whom we care. These devices capture functions previously provided by multiple platforms, and their handheld nature dictates intimate contact. Two hours of smartphone time implies not only screen time but also prolonged exposure to potential allergens. Although nickel is the most common allergen, a nonmetallic cell phone cover does not exclude the diagnosis of ACD or ICD. The ubiquity of smartphone covers together with the growing touch time of our youth requires a high index of suspicion to diagnose cell phone ACD.

Lessons for the Clinician

- Pruritus is the hallmark feature of an eczematous process, in contrast to pain with skin infection.
- In contrast to eczema, psoriasis has increased antimicrobial peptides and marked reduction in secondary infection. Therefore, if the lesion is secondarily infected it is eczema or ACD/ICD, not psoriasis.
- Treatment requires irritant avoidance, inflammation reduction, and skin barrier replacement by using skin moisturizers.

Disclaimer: The views expressed herein are those of the author(s) and do not necessarily reflect the official policy or position of the Department of the Navy, Department of Defense, or United States Government.

Suggested Readings

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Thyssen JP, Linneberg A, Menné T, Johansen JD. The epidemiology of contact allergy in the general population: prevalence and main findings. *Contact Dermat*. 2007;57(5):287–299

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