



Figure 1. Blue hives were observed on arms, breasts, chest, abdomen, pelvis, and thighs. It is interesting that its distribution was bilateral and symmetric.

In summary, we report a patient who developed generalized blue urticaria secondary to patent BD for sentinel node mapping. Skin tests were positive and suggested an immunoglobulin E-mediated mechanism. Physicians must be aware of this sort of contact urticaria, and immediate recognition of early symptoms is crucial because anaphylaxis may occur.

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REFERENCES

1. Cohen S, Ad-El D, Erez E, et al. Beware of the blue hives. *Dermatol Surg* 2008;34:400–402.
2. Keller B, Yawalkar N, Pichler C, et al. Hypersensitivity reaction against patent blue during sentinel lymph node removal in three melanoma patients. *Am J Surg* 2007;193:122–124.
3. Montgomery LL, Thorne AC, Van Zee KJ, et al. Isosulfan blue dye reactions during sentinel lymph node mapping for breast cancer. *Anesth Analg* 2002;95:385–388, table of contents.
4. Sadiq TS, Burns WW 3rd, Taber DJ, et al. Blue urticaria: a previously unreported adverse event associated with isosulfan blue. *Arch Surg* 2001;136:1433–1435.
5. Forschner K, Kleine-Tebbe A, Zuberbier T, et al. Type I sensitization towards patent blue as a cause of anaphylaxis. *Allergy* 2003;58:457–458.
6. Maibach HI, Johnson HL. Contact urticaria syndrome. Contact urticaria to diethyltoluamide (immediate-type hypersensitivity). *Arch Dermatol* 1975;111:726–730.

Recall Dermatitis at Patch Test Sites in Alopecia Areata Treated With Diphencyprone

To the Editor:

Topical immunotherapy using synthetic allergens to elicit a delayed-type (type IV) hypersensitivity reaction is an effective, albeit tedious, modality for treating alopecia areata. We present a 62-year-old white woman with widespread alopecia areata of the scalp sensitized with 2% diphencyprone (DCP; Spectrum Chemicals, Gardena, Calif) by epicutaneous application to bald areas, which reacted with erythematous macules on the application site starting within hours of the application. To identify the maintenance therapeutic dose (minimum dose producing visible inflammation), she was patch tested 3 weeks later to a range of DCP doses (0.0001%–1%) on the left side of the back with single interpretation at 48 hours. Showing no reactivity at patch test reading, she was resensitized with 2% DCP to the same bald areas of the scalp, and she again reported similar erythematous macules as before. After 4 weeks, a second set of patch tests using the same series of DCP dilutions was performed on the right side of the back. At 48 hours, not only did she react with erythema, induration, papules, and some discrete vesicles (++) on the right side of the back to DCP doses 0.01% and higher, but also she reacted to original patch tests on the left side of the back corresponding to DCP doses 0.05% and higher with erythema and induration (+) (Fig. 1).

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Figure 1. Patch testing to DCP after repeat sensitization. The right side represents the second round of patch testing from 0.0001% (marked as 1) to 1% (marked as 7) DCP. The left side represents previously applied patch testing of the same dilution series that demonstrated recall reaction with remote application.

Recurrent dermatitis to nickel, gold,¹ or chromate² at previously sensitized sites following re-exposure to new (nonadjacent) sites has been reported. Recurrent dermatitis to neomycin³ and nickel⁴ at previously sensitized areas after patch testing at distant sites has also been described. Although “recall reactions” with synthetic allergens may be encountered on primary sensitized areas, our case is the first to report a recall reaction at a remote applied patch test site. The mechanism for these reactions is unclear. We hypothesize that effector memory T cells persist at the original site of sensitization, and these cells are reactivated by resensitization at a remote site.⁵ Conversely, it could be the allergen that persists at the original site of sensitization, with allergic dermatitis becoming manifest; thereafter sufficient numbers of effector memory T cells populate it following re-exposure to allergen at a distant site.

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REFERENCES

1. Möller H, Ohlsson K, Linder C, et al. The flare-up reactions after systemic provocation in contact allergy to nickel and gold. *Contact Dermatitis* 1999;40(4):200–204.
2. el Sayed F, Bazex J. Airborne contact dermatitis from chromate in cement with recall dermatitis on patch testing. *Contact Dermatitis* 1994;30(1):58.
3. Jacob SE, Barland C, ElSaie ML. Patch-test-induced “flare-up” reactions to neomycin at prior biopsy sites. *Dermatitis* 2008;19(6):E46–E48.

4. Theler B, Bucher C, French LE, et al. Clinical expression of nickel contact dermatitis primed by diagnostic patch test [published online ahead of print April 7, 2009]. *Dermatology* 2009;219(1):73–76.
5. Moed H, Boorsma DM, Tensen CP, et al. Increased CCL27-CCR10 expression in allergic contact dermatitis: implications for local skin memory. *J Pathol* 2004;204(1):39–46.

Acute Generalized Exanthematous Pustulosis Associated With Acyclovir, Confirmed by Patch Testing

To the Editor:

Acyclovir, a commonly prescribed antiviral drug, has rarely been implicated in severe cutaneous adverse drug reactions.^{1,2}

A 53-year-old man with a herpetic retinitis was being treated with systemic acyclovir and methylprednisolone. Previously, he had also received antibiotics with no adverse effects. Three weeks after starting acyclovir (intravenously during 2 weeks and orally for 7 days), he developed a severe febrile eruption consisting of generalized erythema with numerous small pustules that initially appeared on major intertriginous areas (Fig. 1). His medical history was unremarkable, and he denied any personal or family history of psoriasis or previous use of topical acyclovir. Laboratory evaluation disclosed leukocytosis (24,400/ μ L), neutrophilia, and an elevated C-reactive protein. A skin biopsy revealed multiple subcorneal spongiform pustules filled with neutrophils and a perivascular, lymphomononuclear inflammatory infiltrate in the upper dermis. A diagnosis of acute generalized exanthematous pustulosis (AGEP) was established. Careful revision of prescription charts allowed identification of acyclovir as the probable cause, and it was replaced by foscarnet, while methylprednisolone was maintained. Complete resolution, after a desquamative phase, was achieved in less than 2 weeks.

Two months later, patch tests were performed with an extended European Baseline Series, systemic antibiotics, excipients for topical drugs, acyclovir 10% pet (Chemotechnique Diagnostics, Vellinge, Sweden), commercial creams containing acyclovir at 2% or 5%, and a commercial cream containing penciclovir at 1% (Fenivir). Also, pure acyclovir supplied by the pharmaceutical industry was

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Histopathologic specimens were reviewed by Prof Oscar Tellechea, from the Dermatopathology Unit, Hospitais da Universidade de Coimbra. Prof Artur Paiva, from the Centro de Histocompatibilidade do Centro, Coimbra performed the lymphocyte stimulation test.

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