**Contact Allergy to Corticosteroid Often Masked**

**BY BRUCE JANCIN**

**WAILEA, HAWAII**—Contact allergy to a corticosteroid molecule is considerably underdiagnosed—and it’s no wonder why. Even when suspicion runs high enough that patch testing is performed, the anti-inflammatory action of the corticosteroid often masks the allergic contact reaction, at least early on, Dr. Joseph F. Fowler Jr. said at the annual Hawaii dermatology seminar sponsored by Skin Disease Education Foundation.

Contact allergy to the corticoidal molecule itself—not to some component of the medication’s vehicle—is by no means rare. The incidence in various studies is 0.5%-5%. “Just because it’s an anti-inflammatory molecule doesn’t mean your body can’t make an allergen to it,” observed Dr. Fowler of the University of Louisville (Ky.).

Contact allergy to a corticosteroid should be suspected when a patient has a long-standing skin disorder that isn’t responding to appropriately prescribed topical steroid therapy, or when a dermatitis is getting bigger and bigger as the patient applies more medication, he said. Contact dermatitis experts divide corticosteroids into the following five groups for allergy purposes, based on their molecular structure:

- **Group A**, known as the hydrocortisone type.
- **Group B**, the triamcinolone acetonide type.
- **Group C**, the betamethasone type.
- **Group D1**, the betamethasone dipropionate type.
- **Group D2**, the hydrocortisone-17-butyrate type.

In the United States, because of usage patterns, at least 90% of cases of corticoid allergy involve Group A, and most of those involve hydrocortisone. Allergy to Group B steroids is the next most common, accounting for 5%-7% of cases. Most of the rest involve Group D. Group C steroids are almost never allergenic, according to Dr. Fowler, who is the current president of the North American Contact Dermatitis Group.

Tixocortol pivalate is the standard agent that represents Group A in patch testing. The others in Group A are fluocortisone aceate, hydrocortisone acetate, and—importantly—methylprednisolone and prednisone. Cross-reactivity can occur within steroid groups, so a patient with contact allergy to a Group A steroid that’s used in topical medications could be at risk for a serious reaction to oral or injectable prednisone, he noted. Group B is composed of all the steroids ending in “-ide.” Budesonide is the one most commonly represented in patch testing. Group D steroids all end in “-ate.” Clobetasol propionate is employed as the representative of Group D1 in patch testing; hydrocortisone-17-butyrate is the test material for Group D2.

Group C, which almost never causes contact allergy, is a select group that comprises clorotolone pivalate, desoximetasone, and dexamethasone. When allergy to corticosteroids is known or suspected, a switch to a Group C steroid is the safest bet. Clorotolone cream is the hypoallergenic midpotency steroid of choice, whereas desoximetasone cream or ointment is the safest mid- to high-potency option, Dr. Fowler said.

With regard to contact allergy to the matrices used in topical corticosteroid medications, Dr. Fowler said the big offenders are the various preservatives and propylene glycol.

**Short Course of 3.75% Imiquimod Reduces AKs**

**BY HEIDI SPLETE**

**ORLANDO**—Daily application of 3.75% imiquimod cream with a 2-week dosing cycle was well tolerated and effective for treating actinic keratoses in adults, based on data from two studies.

In the first study, 160 patients were randomized to 3.75% imiquimod cream (Al dara, Graceway Pharmaceuticals), 160 patients to 2.5% imiquimod cream, and 159 patients to a placebo cream. The patients, aged 18 years and older, had 5-20 clinically diagnosed actinic keratoses in adults, based on data from two studies. The others in Group A are fluocortisone acetate, hydrocortisone acetate, and importantly—methylprednisolone and prednisone. Cross-reactivity can occur within steroid groups, so a patient with contact allergy to a Group A steroid that’s used in topical medications could be at risk for a serious reaction to oral or injectable prednisone, he noted. Group B is composed of all the steroids ending in “-ide.” Budesonide is the one most commonly represented in patch testing. Group D steroids all end in “-ate.” Clobetasol propionate is employed as the representative of Group D1 in patch testing; hydrocortisone-17-butyrate is the test material for Group D2.

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**Colchicine Cut Steroid Use in Chronic Urticaria Patients**

**BY HEIDI SPLETE**

**NEW ORLEANS**—Colchicine is an effective steroid-sparing agent that can be used to treat refractory chronic idiopathic urticaria, based on data from a review of adults who received colchicine for CIU from 2003 to 2008.

Colchicine has been shown to decrease mast cell degranulation, suppress leukotriene generation, and decrease leukocyte adhesiveness and migration, said Dr. Mary S. Georgy of Northwestern University, Chicago, and her associates.

To assess the agent’s effectiveness in this setting, the investigators reviewed charts from 55 patients with CIU who were treated with colchicine for at least 7 days, focusing on the type of urticaria, type of response, and use of oral steroids before and after colchicine treatment.

Overall, 24 patients responded to colchicine, 2 partially responded, and 29 did not respond (44%, 4%, and 53%, respectively). The average number of steroid courses in the responders dropped significantly between the 6 months prior to and the 6 months after colchicine use (2.44 vs. 0.33). Information on the average number of steroid courses was available only for the responders. The findings were presented at the annual meeting of the American Academy of Allergy, Asthma, and Immunology.

Major Finding: Patients with chronic urticaria who responded to colchicine used significantly fewer steroids after starting colchicine than before starting it.

Data Source: A review of 55 patients who were treated with colchicine for chronic urticaria.

Disclosures: Dr. Georgy had no financial conflicts to disclose.