

her scalp, axilla, and groin. She developed an erythema multiforme (EM) major-like reaction that required 16-day hospital admission to manage her exaggerated response.

Our patient was initially treated unsuccessfully with topical clobetasol, intralesional Kenalog (10 mg/mL) and topical 2% squaric acid dibutylester (sensitization at the left arm and later application to the scalp). This therapy did not cause an atypical hypersensitivity reaction. Subsequently, sensitization with topical 2% DPCP was applied to 2-cm area on her left arm. Two weeks later, bulla formed at the sensitization site. Intramuscular kenalog 40 mg/mL, intralesional kenalog 10 mg/mL, and topical minoxidil 2% solution were administered. Three days later, oral prednisone was started. Her reaction continued to progress to targetoid lesions and severe bullae, and 1 week later, she was treated in the emergency department with intravenous Solu-Medrol and Benadryl infusions. This did not improve her condition.

Six weeks after DPCP sensitization, the patient was admitted with EM major. Her physical examination included extensive targetoid papules and plaques with vesicles on her trunk and extremities (Figs. 1A–C) and erythematous, crusted erosions on her hard palate and lips (Fig. 1D). Erosions on the left chest and bullae on the left upper arm were well healed with no evidence of full-thickness epidermal loss. Skin biopsy specimen showed interface dermatitis consistent with drug eruption (Fig. 1E). Inpatient treatment included intravenous Solu-Medrol pulse therapy (1 g/d for 4 days, followed by oral prednisone 1.0 mg/kg per day), cyclosporine (200 mg by mouth twice a day), oral fluocinonide gel, intravenous immunoglobulin infusions, narcotic analgesics, and Keflex 500 mg by mouth twice a day for 2 weeks. Her symptoms gradually resolved after discharge, and immunosuppressive therapy was weaned without relapse.

Known inducers of EM reactions include viruses (herpes simplex virus), drugs (allopurinol), topical agents (imiquimod), and environmental causes (poison ivy).² Although rare, 7 cases of EM-like reactions after topical DPCP application have been reported.³ All patients treated developed 1- to 4-week EM courses after 0.1% to 2% topical DPCP treatment. Six patients were women, and 3 patients developed reactions after the initial DPCP sensitization. Only 1 patient required hospitalization for systemic corticosteroids. Of interest, 3 (43%) of the reported reactions occurred in patients of Asian descent. This increased proportion may suggest a genetic predisposition to the development of EM secondary to topical DPCP. Asian populations are especially susceptible to known human leukocyte antigens (HLA)-associated drug hypersensitivities.⁴ Interestingly, increased susceptibility to EM regardless of etiology has been made with HLA-B15⁵ and HLA-DQw3² alleles. Further characterization of HLA type and clinical presentation in affected individuals is needed to determine this potential relationship.

Regardless, this case underscores the severity to which adverse effects of standard therapies for AA can impact patients.

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Orthodontic Appliance Intolerance Due to Dental Adhesive Allergy

To the Editor:

A 14-year-old adolescent girl presented for patch testing after experiencing a type IV allergic reaction to her metal braces, which was revealed as a probable dental acrylic adhesive allergy. Contact or type IV allergy to orthodontic braces and other appliances has been widely reported, with nickel most commonly identified as the allergenic culprit.¹ Allergies to dental acrylics are also well known among both dental patients and dental technicians.^{2,3} Invisalign braces and retainers, an alternative to traditional metal braces, are composed of polyurethane from methylene diphenyl diisocyanate and 1,6-hexanediol. Localized and systemic allergic reactions to Invisalign have been reported, particularly among workers in the polyurethane industry and among patients using the orthodontic appliances.⁴

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The adolescent girl was referred for patch testing before placement of a permanent retainer. One year ago, she experienced a severe adverse reaction to the application of standard braces. Two days after placement, she noted a pruritic and burning rash on her forehead that spread down her face and then to her neck and arms. The braces were removed 5 days after placement, leaving the brackets adhered to the teeth. With the rash worsening, the brackets were then removed, and subsequently, the rash resolved completely within a week.

Dental alignment was achieved using Invisalign for 1 year, and the patient experienced no further problems. Having completed alignment, a permanent retainer was now planned for maintenance care. The patient and her mother were concerned about the potential risk of allergic reaction to retainer components. The orthodontist provided details on all materials to be used and included samples of the wires, adhesives, curing agents, and other retainer components for product patch testing.

The patient was patch tested with the North American Core Allergen Series, dental screening patient grouping, and all retainer components intended for direct application to the oral mucosa. At 48 and 96 hours, she had a 1+ reaction with erythema and pruritis to a 3M Transbond Light Cure adhesive sample provided by the orthodontist, which at 96 hours evolved to a 3+ reaction with blistering and epidermal erosion. No reaction was observed to metal wires or other retainer and installation components. The adhesive ingredients include silane-treated quartz, bisphenol A diglycidyl ether dimethacrylate (Bis-GMA), triethylene glycol dimethacrylate, dichlorodimethylsilane, *N,N*-dimethylbenzocaine, and diphenyliodonium hexafluorophosphate. Notably, the same adhesive was used in the original brace brackets, so the patient had a previous exposure, and the timing of both her reaction and recovery implicated the adhesive as the cause of the braces-related rash.

Previous similar case reports of dental adhesive reactions identified Bis-GMA as the responsible allergen based on patch testing results.⁵ However, the dental screening grouping tested on this patient includes Bis-GMA, which was negative on patch testing, suggesting that either a different ingredient was responsible or the concentration differed. One limitation in the current case is that the adhesive sample was applied undiluted and was not light cured, so the more reactive unpolymerized material was in contact with the epidermis, potentially generating a stronger reaction than if light cured.⁶

Based on the patch testing results, a removable Invisalign retainer was chosen for maintenance. With increasing reports of allergies to dental devices, patch testing should be considered in individuals with a history of contact reactions to metals or acrylics. In this case, patch testing was performed after a severe reaction to traditional braces containing metal wires and acrylic bonding materials. Patch testing revealed contact allergies to the dental acrylic adhesive, effectively ruling out installation of a

permanent retainer. This case demonstrates that dental professionals should be aware of the potential risk of allergy not only to the metal components of braces but also to the acrylics and adhesives used in placing the appliances.

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Contact Allergy to Propylene Glycol and Cross-Reactions

To the Editor:

Propylene glycol (PG) and similar derivatives are considered possible cross-reactors. Although PG is on the American Contact Dermatitis Society core series, other related compounds are not commonly tested.¹ Cross-reaction (CR) between PG, its derivatives, and related chemicals is unknown because of a lack of data.

In our center, all patch-positive PG patients are informed to avoid any chemicals containing the exact word “propylene glycol” or the abbreviation “PG” to avoid potential CR. The American Contact Dermatitis Society Contact Allergen Management Program uses the same criteria and currently considers more than 150 derivatives as possible cross-reactors.

Materials were tested with vehicles and concentrations listed in *Patch Testing, Third Edition*.² Propylene glycol acetate (Spectrum Chemical), dipropylene glycol, and PG monostearate (Tokyo Chemical Industry Chemicals), as well as hexylene, butylene, and pentylene glycols (Sigma-Aldrich), were tested.

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