

Allergic contact dermatitis caused by VEA® lipogel: compound allergy?

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The term compound allergy is used to describe a condition in patients who are patch test-positive to formulated products, but are patch test-negative to all the ingredients when these are tested individually. We report 8 cases of contact dermatitis to VEA® lipogel (Ulka, Rovigo, Italy), a cosmetic that has previously been reported as the cause of compound allergy (1). All cases were observed at the Allergological and Occupational Dermatology Unit in Florence from October to December 2015.

Patients and Methods

This observational study involved 10 patients (2 males and 8 females) with suspected contact dermatitis caused

by VEA® lipogel. The patients' age ranged from 8 to 62 years (mean 36.5 years; median 34 years). All patients had applied the cosmetic cream to treat a pre-existing dermatosis: 7 patients had a history of atopy, 2 patients suffered from genital lichen sclerosus, and 1 patient suffered from Sjögren syndrome. All patients used VEA® lipogel for relief of skin and mucosal dryness.

Patch tests were performed with the baseline patch test series suggested by the Italian Society of Allergological, Occupational and Environmental Dermatology (SIDAPA), according to the recommendation of the ICDRG. Allergens provided by FIRMA (Florence, Italy) and Chemotechnique Diagnostics (Vellinge Sweden), and patients' own products and VEA® lipogel tested 'as is', were applied on the patients' backs with van der Bend® Square Chambers (van der Bend, Brielle, The Netherlands) and occluded with Medipore™ tape (3M Company, Minneapolis MN, USA); readings were performed on day (D) 2, D4, and D7.

At D2 and D4, ++ reactions to VEA® lipogel (tested 'as is') were observed in 8 patients. Of these 8 patients, 7 had facial involvement and 1 had genital involvement.

The manufacturer provided their separate ingredients 'as is': cyclopentasiloxane, tocopheryl acetate,

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hydrogenated castor oil, ethylhexyl palmitate, and dimethiconol. A second session of patch testing took place 1 month later with the separate ingredients tested 'as is', and tocopheryl acetate (10% pet.; Chemotechnique Diagnostics); however, negative patch test results were observed in all 10 patients tested. However, clinical relevance of the positive patch test reactions to VEA® lipogel 'as is' had, in the meantime, been confirmed by the complete resolution of the dermatitis after avoidance of VEA® lipogel. Testing of 10 controls with VEA® lipogel and its constituents did not cause irritant reactions. In the 2 patients with negative patch test results for VEA® lipogel, a repeated open application test with VEA® lipogel yielded no reaction, and a final diagnosis of irritant contact dermatitis on a pre-existing dermatosis was made in these 2 patients.

The manufacturer suggested the possibility that storage and production at higher temperature may have modified single ingredients contained in VEA® lipogel, and acknowledged that batches could contain tocopheryl acetate from two different suppliers. Two patients consented to undergo further patch tests with tocopheryl acetate 'type A' (25% pet.) and tocopheryl acetate 'type B' (25% pet.). Furthermore, hydrogenated castor oil (25% pet.), dimethiconol (25% pet.) and cyclopentasiloxane (25% pet.) were heated to 90°C and left to cool before being patch tested. Our 2 patients showed positive reactions to tocopheryl acetate 'type A' on D2 and D4, and negative results for the other ingredients, confirming a final diagnosis of allergic contact dermatitis caused by tocopheryl acetate 'type A'.

Conclusion

In the literature, we found 1 case of compound allergy to VEA® lipogel (1) and 2 cases of allergic contact dermatitis caused by vitamin E contained in VEA® oil and VEA® lipogel (2). In another case, it was suggested that compound allergy to VEA® lipogel was attributable to the interaction between cyclopentasiloxane and tocopheryl acetate (3).

A recent review reported 931 cases of vitamin E-induced allergic contact dermatitis since the first observation in 1965, and concluded that the incidence is low despite the widespread use of vitamin E in skincare products (4). The allergenic potential of some substances may be increased by oxidation (2), and compounds may change when used in production, when stored, or when handled by the consumer (5). Furthermore, ingredients delivered by the manufacturer may not always be reliable and complete, thereby causing false-negative reactions and leading to the diagnosis of 'pseudo'-compound allergy.

In the present case series, we have shown that tocopheryl acetate 'type A' may well be the actual cause of allergic contact dermatitis caused by VEA® lipogel in some patients, perhaps because of impurities contained in this specific cosmetic ingredient. This could also explain the unexpected negative results observed by Corazza et al. when they repeated tests 1 month later with different VEA® lipogel samples (2), and the tolerance that our 8 patients reported when they used different brand products containing vitamin E. The high prevalence of contact dermatitis caused by VEA® lipogel in our patients suggests an epidemic outbreak; therefore, we expect to see more cases in the future.

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