

A case of ethanol-induced systemic allergic dermatitis

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We describe a case of systemic contact dermatitis caused by ethanol (eth.). Although this condition was first

identified in 1944 (1), it remains an uncommon diagnosis, and we are aware of only 5 other cases of systemic contact dermatitis caused by eth. (1–5). It has been best studied in the setting of systemic exposure to topical medications, and typically presents as a symmetrical erythematous rash (6). Vesicular dermatitis, a photodistributed rash, erythroderma and baboon syndrome have also been reported (6). Contact dermatitis

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caused by eth. can be induced experimentally (7), as well as from repeated occupational exposures to disinfectants or transdermal patches (4, 8).

Case Report

A 38-year-old woman presented with a history of eth.-induced rash. Her first reaction occurred at 10 years of age, when alcoholic disinfectant was applied to her knee, resulting in local erythema, swelling and pruritus at the site of application. At the age of 18 years, she developed a widespread painful papular rash 8–12 h after drinking liqueur. It was associated with periorbital oedema, but there was no urticaria, pruritus or airway angioedema. Subsequent accidental exposure included a spilled alcoholic beverage, alcoholic swabs after blood collection, and eth.-based perfume. Each of these resulted in an erythematous, non-blanching rash after ~1 h, which subsided within 3 days. Convinced of the association with eth. the patient applied a small quantity of mouthwash containing 27% eth. to her forearm, which resulted in the same rash.

Her medical history includes pollen food allergy syndrome and seasonal rhinoconjunctivitis with sensitization to birch pollen.

Her eth. hypersensitivity was investigated by the use of ~20 µl of 70% eth. with 30% water applied to dry skin. The application was performed with allergEAZE® Patch Test Chambers (SmartPractice®, Calgary, Canada), reinforced at the margins with Hypafix® (BSN Medical®, Luxembourg City, Luxembourg). The skin was occluded for 3 days. No other substances or dilutions were tested in the other chambers. The patient was instructed to keep the area dry to avoid local irritation with added moisture; this irritation is not noted on dry skin, even at eth. concentrations of 100% (9). Her skin was assessed on day 3, and erythema, infiltration and vesicles were observed, compatible with a strong positive reaction (++) to eth. (Fig. 1). As the other chambers were empty, the slight reaction seen at the site of the adjacent chamber was regarded as spill-over. This confirmed the diagnosis of eth.-induced delayed-type hypersensitivity. In our diagnostic pathway, we did not perform an oral challenge, given the convincing history of a systemic reaction and the positive patch test result. The patient is currently managed with eth. avoidance.

Discussion

The role of eth. by-products in delayed-type hypersensitivity is unclear. In the majority of cases in which patch testing was performed with both eth. and acetaldehyde, positive results were obtained with eth. alone (1–3, 10).

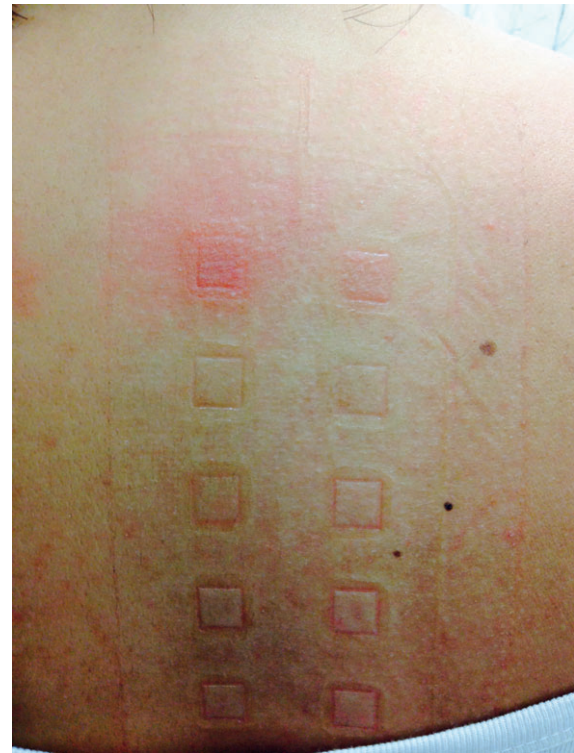


Fig. 1. Evident dermatitis on day 3 after application of 70% ethanol to the upper left patch. Other chambers were empty; the slight reaction seen at the site of the adjacent chamber was regarded as spill-over.

There has been only 1 case of delayed-type hypersensitivity to both eth. and acetaldehyde (11), despite acetaldehyde being more likely to cause irritant dermatitis (9). Similarly, those patch tested with both eth. and acetic acid reacted only to eth. (1, 11).

Acetic acid appears to be more relevant in those with immediate hypersensitivity reactions. This is shown by the fact that some patients react to both eth. and vinegar (12). Patients appear to be sensitized to acetic acid and not to eth. with prick testing (12, 13). This has led some to conclude that immediate hypersensitivity reactions to eth. may actually be caused by its metabolic product acetic acid (14, 15).

The pathogenesis of eth.-induced contact dermatitis has not been identified. Given the small molecular size of eth., immunogenic effects are probably attributable to hapten formation. It has been established by Fourier transform infrared spectroscopy that topical eth. causes structural changes in proteins in the stratum corneum, with greater β -sheet formation (16). It is unclear whether this is attributable to chemical reactions or polar interactions. Such alterations in protein conformation could be

immunogenic. In addition, eth. increases skin permeability by extracting lipids (16), and therefore could facilitate contact with Langerhans cells in the epidermis and antigen recognition.

The pharmacokinetic properties of eth. would permit the development of systemic contact dermatitis after oral administration. First, eth. undergoes variable first-pass metabolism, leading to easily detected blood concentrations. Second, eth. is not bound to any transport protein in blood, and therefore circulates unaltered (17). Third, eth. has the potential to reach the skin, and at least 0.1% of unmetabolized eth. is excreted through sweat (17). Therefore, oral ingestion has the potential to cause re-exposure at an epidermal level, albeit at much lower concentrations. Our patient is of Asian heritage,

and slow-metabolic polymorphisms of alcohol dehydrogenase may impose a greater risk of systemic contact dermatitis. Although these hypotheses are biologically plausible and provide a theoretical framework for understanding eth.-induced contact dermatitis, they still await experimental validation.

This case report contributes to our understanding of eth.-induced systemic contact dermatitis by describing the history and investigative features of this rare condition, and discussing potential mechanisms of pathogenesis.

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