

Correspondence

A holistic view on dermatitis: Patch testing should be considered in patients with atopic dermatitis

To the Editor:

With interest we read "Atopic dermatitis: a practice parameter update 2012."¹ Although the authors acknowledge that allergic contact dermatitis (ACD) should be considered in the differential diagnosis of atopic dermatitis (AD) and state that contact allergy to topical corticosteroids should sometimes be ruled out, patch testing for delayed-type hypersensitivity was excluded from the flowchart of the diagnosis and management of AD.

We believe that a more holistic view of dermatitis, taking the critical role of ACD in AD morbidity into account, would optimize patient care and that the 2 disorders are likely to be much more closely related clinically and pathomechanistically than previously thought. The former general "consensus" on the inverse relationship between ACD and AD is explained, at least in part, by a well-conducted study that clearly showed that experimental induction of contact allergy to irritating chemical allergens was compromised in patients with AD in a dose-dependent manner.² Furthermore, because clinical epidemiologic studies have often rejected an association between AD and ACD, the logical conclusion drawn by many physicians has been to omit patch testing from the routine diagnostic work-up of patients with AD.

Here we provide arguments to support that patch testing of patients with AD is indeed clinically valuable and thereby challenge the consensus. First, the treatment of xerotic and inflamed skin in patients with AD results in continued exposure of irritated skin to allergens found in topical products. Therefore it is not surprising that fragrance and other contact allergies occur significantly more often in both juvenile and adult patients with AD than control subjects³ and that contact allergy to topical drugs and emollients is common in children with severe AD and early onset.⁴

Second, filaggrin gene (*FLG*) mutations, which are strongly associated with AD, are positively associated with nickel allergy and nickel ACD, perhaps because of increased penetration of nickel ions through a histidine-depleted stratum corneum.

Third, in animal models repeated skin application of a contact allergen results in acute ACD, followed by subacute ACD and finally a phenotype of AD. Similarly, patients with nickel allergy without a predisposition to atopy who are continuously exposed to nickel on the skin in high concentrations have generalized dermatitis, which is clinically indistinguishable from AD ("chemical atopic dermatitis").⁵

Fourth, a recent study showed that allergic reactions to weaker irritants seem to occur more often in patients with AD than in control subjects and that reactions to inherently irritant contact allergens occur somewhat less often.⁶

Fifth, intrinsic AD, which is characterized by the absence of specific IgE to aeroallergens and food allergens, might be more associated with contact allergy to nonprotein antigens,⁷ whereas extrinsic AD might be a form of systemic contact dermatitis to food and airborne protein allergens. Because of the relatively wide overlap of AD and ACD, we consider it important to include patch testing in the diagnostic battery of physicians caring for juvenile and adult patients with AD. This might ultimately prevent unnecessary morbidity, improve quality of life, and save health care costs.

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