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Atopy is associated with sensitization to allergens known to cause systemic contact dermatitis

Jeffrey F. Scott, MD¹, Rosalynn R.Z. Conic, MD¹, InYoung Kim, MD, PhD¹, Douglas Y. Rowland, PhD², and Susan T. Nedorost, MD¹

¹Department of Dermatology, University Hospitals Cleveland Medical Center and Case Western Reserve University, Cleveland OH, USA

²Department of Population and Quantitative Health Sciences, Case Western Reserve University, Cleveland OH, USA

Abstract

Background: Atopic dermatitis (AD) associated with respiratory atopy may represent a form of systemic contact dermatitis (SCD), whereby AD flares after ingestion or inhalation of allergens.

Objective: To compare the prevalence of positive patch tests to allergens known to cause SCD in AD patients with and without respiratory atopy.

Methods: Retrospective study of AD patients patch tested to 23 allergens known to cause SCD. Positive patch tests were compared between AD patients with and without respiratory atopy, stratified by age and wet or dry work occupation.

Conclusions—Children and adolescents, but not adults, with AD and respiratory atopy were more likely than age-matched AD patients without respiratory atopy to have positive patch tests to these allergens (OR 2.33, 95% CI 1.13–4.79). Moreover, AD patients with respiratory atopy and engaging in wet work, but not dry work, occupations were more likely than AD patients without respiratory atopy to have positive patch tests to allergens known to cause SCD (OR 1.47, 95% CI 1.05–2.06). Thus, respiratory atopy and wet work are associated with sensitization to allergens known to cause SCD in patients with AD, and patch testing may be valuable in identifying systemic triggers of dermatitis in these patients.

Keywords

atopic dermatitis; respiratory atopy; asthma; allergic rhinitis; atopy; extrinsic atopic dermatitis; intrinsic atopic dermatitis; patch testing; wet work; barrier dysfunction

Corresponding Author Jeffrey F. Scott, MD, University Hospitals Case Medical Center, Department of Dermatology, Lakeside 3100, 11100 Euclid Avenue, Cleveland OH 44106, USA, Phone: 860-989-8261; Fax: 216-844-8993, jeffrey.scott@uhhospitals.org.

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Introduction

Chronic skin inflammation predisposes to sensitization to weak irritants, including food allergens, and also predisposes to T-helper 2 (T_H2)-skewed immune responses.^{1–2} Both T-helper 1 (T_H1) and T_H2 immune responses can occur to some allergens such as nickel and fragrance, and notably T_H2 responses may be more durable than T_H1 responses in the setting of cutaneous sensitization with chronic inflammation.^{3–7} Thus, cutaneous sensitization in the context of inflamed skin likely plays a key role in the development of systemic allergen sensitization in the atopic march.^{8–15}

Chronic barrier inflammation occurs in childhood-onset dermatitis, or atopic dermatitis (AD), and in wet work.^{14, 15} Allergic contact dermatitis to foods is reported exclusively in patients in these two settings; that is, with AD or wet work and food handling. Moreover, cutaneous sensitization to allergens that can be ingested or inhaled, including peanut, grass pollen, and Compositae, is increasingly recognized in AD.^{16–20} Systemic contact dermatitis (SCD) occurs after cutaneous sensitization and the subsequent generation of skin-homing cutaneous lymphocyte-associated antigen (CLA)+ T cells specific for ingested or inhaled allergens.^{21–24} Thus, AD associated with respiratory atopy may represent a form of SCD, whereby flaring of AD occurs when cutaneously sensitized patients are re-exposed to allergens through the gastrointestinal or upper respiratory tracts.

Our objective was to compare the prevalence of positive patch tests to allergens known to cause SCD among AD patients with and without respiratory atopy patch tested for suspicion of allergic contact dermatitis. This approach was driven by a list of allergens known to cause SCD, rather than starting with patient characteristics of atopy compared to non-atopic patients patch tested to a standard screening series as previously reported.¹⁵

Materials and Methods

Allergen Selection

This study was approved by the Institutional Review Board. Allergens associated with SCD were identified *a priori* and agreed upon by two independent investigators (JFS, STN). We began with a comprehensive set of allergens known to be associated with SCD identified from Fisher's Contact Dermatitis (6th Edition) and a PubMed literature search using the keywords "systemic contact dermatitis."²⁵ We did not consider allergens reported to cause SCD through a primarily non-type IV hypersensitivity mechanism of action, including anaphylaxis (e.g. formaldehyde, paraformaldehyde) and erythema multiforme (e.g. disperse blue 124, epoxy resin).²⁵ Allergens reported to cause SCD through cross-reactions with other ingested or inhaled compounds (e.g. aminoglycosides, sulfonamides, phenothiazines) were also not considered.²⁵ Finally, various allergens reported to cause SCD, including food and aeroallergen proteins, carmine, and sorbic acid, among others, were excluded from the final analysis because these allergens were patch tested in less than 50% of patients. In total, 23 allergens patch tested in greater than 50% of patients were included in the final analysis. These allergens were broadly categorized as plant derivatives (N=9), metals (N=4), steroids (N=6), and miscellaneous (N=4).

Study Cohort

Patch test results were obtained using a database of 2,416 patients patch tested for suspicion of allergic contact dermatitis at University Hospitals Cleveland Medical Center between January 1, 2003 and November 8, 2017. All patients underwent patch testing using a modified North American Contact Dermatitis Group standard allergen series or specialty allergen series, as indicated for relevant exposures based on patient history. The International Contact Dermatitis Research Group criteria for strength of patch test responses was used to determine patch test positivity.²⁶ Initial patch test readings were performed on days 2, 3, or 4, and final patch test readings were performed on day 7. A positive patch test result was defined as any strength positive reaction on day 7, or greater than 1+ positive reaction on days 2, 3 or 4 in a non-irritant allergen. A late time point for patch test readings was utilized to distinguish a true positive reading from a false positive early irritant reaction, as the majority of irritant reactions resolve more quickly than true allergic reactions.

AD was defined as a physician-assessed history of childhood flexural dermatitis. AD patients were stratified by the presence of respiratory atopy, defined as a self-reported history of asthma and/or allergic rhinitis. AD patients were further stratified by age (<18 versus ≥18 years) and occupation (wet versus dry work). Occupations were classified as wet or dry work by a single investigator (STN) based on job duties elicited at the initial clinic visit, as previously described.¹⁵

Statistical Analysis

Basic demographic characteristics, including age, sex, and race, were extracted for AD patients. The total number of patch tests, and the number of positive and negative patch test results for the 23 allergens known to cause SCD were determined for AD patients with and without respiratory atopy. Positive patch tests were compared between: 1) AD patients with and without respiratory atopy; 2) AD patients with and without respiratory atopy stratified by age (<18 versus ≥18 years); 3) AD patients with and without respiratory atopy stratified by occupation (wet versus dry work). Fisher's exact test was used to compare the relative frequencies of positive reactions. After constructing odds ratios (OR) from the test data comparing each set of groups where there were no zero counts of positive patch tests, the 95% confidence intervals (CI) for each comparison were constructed by the method of Cornfeld.²⁷ Haldane's method was used to construct OR and 95% CI for comparisons involving zero positive patch test results.²⁸ All statistical analyses were performed using R statistical software, Version 3.4.1. *P* values ≤0.05 were considered statistically significant.

Results

A total of 457 (18.9%) patients with AD and 1959 (81.1%) patients without AD were patch tested for suspicion of allergic contact dermatitis between 2003 and 2017. Of these, 278 (60.8%) AD patients with respiratory atopy and 179 (39.2%) AD patients without respiratory atopy were identified (Table 1). More adult AD patients had respiratory atopy (*p*=0.02), but otherwise the groups were similar with regards to sex, race, and occupation type.

Table 2 lists patch test results for each ingested or inhaled allergen known to cause SCD. Supplementary Table ranks the allergens by frequency of positive patch tests from highest to lowest in AD patients with respiratory atopy. Overall, the percentage of positive patch tests to these allergens was 3.7% and 2.9% for AD patients with and without respiratory atopy, respectively (OR 1.28, 95% CI 0.99–1.66; $p=0.07$). Of note, there was no difference in patch test results among non-AD patients with and without respiratory atopy (OR 0.93, 95% CI 0.82–1.05; $p=0.24$). AD patients with respiratory atopy also had a significantly higher percentage of positive patch tests to metals compared to AD patients without respiratory atopy (11.4% versus 7.9%; $p=0.04$). There was no difference in the percentage of positive patch tests to metals in AD patients with respiratory atopy stratified by the presence of dyshidrotic eczema (OR 1.20, 95% CI 0.82–1.74; $p=0.37$).

AD patients less than 18 years of age with respiratory atopy were significantly more likely than those without respiratory atopy to have positive patch tests to the combined 23 allergens (OR 2.33, 95% CI 1.13–4.78; $p=0.02$; Table 3). There was no difference in patch test results among AD patients over age 18 with and without respiratory atopy (OR 1.15, 95% CI 0.87–1.52; $p=0.34$). AD patients with respiratory atopy engaging in wet work, but not dry work, occupations were also more likely to have positive patch tests to the combined 23 allergens compared to AD patients without respiratory atopy (OR 1.47, 95% CI 1.05–2.06; $p=0.03$; Table 3).

Discussion

Positive patch tests to allergens known to cause SCD are more common in AD patients with respiratory atopy who are under the age of 18 compared to older patients with a history of AD presenting to a dermatitis clinic, and this is also true for patients doing wet work. These data support prior studies documenting increased rates of positive patch tests to certain allergens in patients with a personal or family history of atopy of any type, as well as increased sensitization to aeroallergens in AD patients.^{16, 29–31}

This study suggests that AD associated with respiratory atopy may signal an immune system capable of developing SCD, in which flaring of AD occurs after systemic exposure to allergens. A T cell response is detected by patch testing. Some patients with a T cell response may exhibit SCD after ingestion or inhalation of an allergen if the T cells are CLA⁺ and home to areas of skin that have previously been in contact with the allergen.²⁴

This work included only commercially available allergens commonly tested on a standard screening series in the majority of our cohort. The logical extension of our conclusions is that SCD to foods and aeroallergens may trigger AD in patients with respiratory atopy (i.e. extrinsic AD) who have antigen-specific CLA⁺ cells. T_H2-skewed immune responses are characteristic of patients with AD and respiratory atopy.³² In contrast, the T_H1-skewed immune responses seen in AD patients without respiratory atopy predispose these patients to have less propensity for developing systemic allergen sensitization.^{32, 33}

Food and pollen allergens are low potency allergens, consistent with prior work showing that barrier disruption, either from AD or wet work, is likely necessary to create the innate

immune signals required for cutaneous sensitization to less potent allergens.^{15, 34} In our study, AD patients with respiratory atopy, and those patients engaging in wet work, were consistently found to have higher rates of positive patch tests to propylene glycol, a weak sensitizer.

This study is limited by its retrospective design, which cannot determine causation and lends itself to misclassification and recall biases. It is also possible that patients with non-atopic etiologies of dermatitis, asthma, and/or rhinitis were incorrectly classified as having AD and respiratory atopy. Moreover, we only considered patch testing as an assay for cutaneous delayed type hypersensitivity. Therefore, no conclusions can be drawn regarding the prevalence of systemic sensitization, SCD, or allergic contact dermatitis in our cohort. In addition, the occupation classifications used in this study only represent current rather than previous jobs, and may not fully reflect all wet exposures. Finally, it is possible that some younger patients in the process of progressing through the atopic march had not yet developed respiratory symptoms at the time of patch testing, and may have been misclassified as not having respiratory atopy.

In conclusion, patients with AD, respiratory atopy, and environmental barrier dysfunction in the form of wet work are characterized by increased positive patch tests to ingested or inhaled allergens known to cause SCD. Patch testing may be of important diagnostic and therapeutic value in AD patients with respiratory atopy, particularly when focusing on specialized allergen series that include weak allergens, food additives, and aeroallergens. The identification of systemic triggers of AD with patch testing, and subsequent systemic and topical avoidance of relevant food additives and aeroallergens, could decrease the frequency of AD flares. Ultimately, patch testing could act as a “steroid-sparing agent,” reducing the use of corticosteroids and other immunosuppressive medications in the management of AD patients.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviations

AD	atopic dermatitis
SCD	systemic contact dermatitis

CLA	cutaneous lymphocyte-associated antigen
T_H2	T helper 2

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Table 1.

Baseline characteristics of AD patients with and without respiratory atopy.

	AD with Respiratory Atopy	AD without Respiratory Atopy	P value
Total patients, N	278 (60.8)	179 (39.2)	
Sex, N (%)			
Male	72 (25.9)	61 (34.0)	0.07
Female	206 (74.1)	118 (65.9)	
Age, N (%)			
< 18 years	79 (28.4)	69 (38.1)	0.02
18 years	199 (71.6)	110 (60.8)	
Race, N (%)			
White	211 (75.9)	129 (71.1)	0.55
Black	54 (19.4)	38 (21.1)	
Other ¹	13 (4.7)	12 (6.7)	
Occupation, N (%)			
Wet work	64 (23.0)	36 (20.1)	0.54
Dry work	196 (70.5)	95 (53.1)	
Unknown	18 (6.5)	48 (26.8)	
Patch Tests², N	5754	2778	

¹ Other includes Asian and Arab² Includes twenty-three allergens known to cause SCD**Abbreviations:** AD, atopic dermatitis; N, number

Table 2.

Patch test results to individual allergens and for allergens in combination in AD patients with and without respiratory atopy patch tested for suspicion of allergic contact dermatitis.

	AD with Respiratory Atopy (N=278 patients)			AD without Respiratory Atopy (N=179 patients)			OR (95% CI)	P value
	# Tests	# (+)	% (+)	# Tests	# (+)	% (+)		
All Allergens (N=23)	5754	213	3.7	2778	81	2.9	1.28 (0.99–1.66)	0.07
Plant Derivatives (N=9)	2363	59	2.5	1121	29	2.6	0.96 (0.60–1.57)	0.91
Balsam of peru	252	3	1.2	123	3	2.4	0.48 (0.10–2.42)	0.40
Benzyl alcohol	264	2	0.8	134	0	0.0	2.56 (0.12–53.75) *	0.55
Cinnamic aldehyde	259	3	1.2	127	1	0.8	1.48 (0.15–14.34)	>0.99
Compositae	243	10	4.1	106	2	1.9	2.23 (0.48–10.37)	0.36
Eugenol	209	2	1.0	81	0	0.0	1.96 (0.09–41.35) *	>0.99
Fragrance mix I & II	480	31	6.5	232	17	7.3	0.87 (0.47–1.61)	0.75
Propolis	215	3	1.4	100	6	6.0	0.22 (0.05–0.91)	0.03
SQL	259	3	1.2	131	0	0.0	3.59 (0.18–70.00) *	0.55
Tea tree oil	182	2	1.1	87	0	0.0	2.42 (0.12–51.03) *	>0.99
Metals (N=4)	990	113	11.4	481	38	7.9	1.50 (1.02–2.21)	0.04
Cobalt	247	31	12.6	120	6	5.0	2.73 (1.11–6.73)	0.03
Gold	245	38	15.5	120	13	10.8	1.51 (0.77–2.96)	0.26
Nickel	251	36	14.3	123	15	12.2	1.21 (0.63–2.30)	0.63
Potassium dichromateY	247	8	3.2	118	4	3.4	0.95 (0.28–3.23)	>0.99
Steroids (N=6)	1448	19	1.3	702	6	0.9	1.54 (0.61–3.88)	0.40
Budesonide	262	5	1.9	138	2	1.4	1.32 (0.25–6.91)	>0.99
Clobetasol	207	5	2.4	96	1	1.0	2.35 (0.27–20.41)	0.67
Desoximetasone	197	0	0.0	85	0	0.0	0.43 (0.01–22.00) *	>0.99
Hydrocortisone	281	1	0.4	121	0	0.0	1.30 (0.05–32.13) *	>0.99
Tixocortal pivate	261	6	2.3	143	3	2.1	1.10 (0.27–4.46)	>0.99
TAC acetamide	240	2	0.8	119	0	0.0	2.51 (0.12–52.60) *	>0.99
Miscellaneous (N=4)	953	22	2.3	474	8	1.7	1.38 (0.61–3.12)	0.56
Dibucaine	195	2	1.0	83	1	1.2	0.85 (0.08–9.50)	>0.99
Ethylenediamine	250	4	1.6	119	2	1.7	0.95 (0.17–5.27)	>0.99
Parabens	246	0	0.0	122	2	1.6	0.10 (0.01–2.05) *	0.11
Propylene glycol	262	16	6.1	150	3	2.0	3.19 (0.91–11.12)	0.09

Abbreviations: AD, atopic dermatitis; OR, odds ratio; CI, confidence interval; SQL, sesquiterpene lactones; TAC, triamcinolone

* Haldane's method was used to construct OR and 95% CI for comparisons involving zero positive patch test results, which adds 0.5 to all cells to accommodate possible zero counts.

Cumulative patch test results for the combined 23 ingested or inhaled allergens known to cause SCD in AD patients with and without respiratory atopy, stratified by age <18 years and age 18 years, and wet or dry work occupation.

Table 3.

	AD with Respiratory Atopy and < age 18 (N=110 patients)			AD without Respiratory Atopy and < age 18 (N=69 patients)			OR (95% CI)	AD with Respiratory Atopy and age 18 (N=199 patients)			AD without Respiratory Atopy and age 18 (N=79 patients)			OR (95% CI)
	#	%	Tests	#	%	Tests		#	%	Tests	#	%	Tests	
All Allergens (N=23)	1326	46	3.5	592	9	1.5	2.33 (1.13–4.78)	4547	171	3.8	2287	75	3.3	1.15 (0.87–1.52)
	AD with Respiratory Atopy and Wet Work (N=64 patients)			AD without Respiratory Atopy (N=179 patients)			OR (95% CI)	AD with Respiratory Atopy and Dry Work (N=196 patients)			AD without Respiratory Atopy (N=179 patients)			OR (95% CI)
	#	%	Tests	#	%	Tests		#	%	Tests	#	%	Tests	
All Allergens (N=23)	1464	62	4.2	2778	81	2.9	1.47 (1.05–2.06)	4057	137	3.4	2778	81	2.9	1.16 (0.87–1.56)