The two major forms of contact dermatitis are irritant contact dermatitis and allergic contact dermatitis. Irritant contact dermatitis is seen in roughly 80% of contact dermatitis cases. Irritant contact dermatitis is an inflammatory, but nonimmunologic dose- and time-dependent skin response to direct cellular injury caused by a skin irritant, and it does not require prior sensitization. Common irritants in the pediatric age group include excessive bathing, urine and feces in the diaper area, bubble baths, cleansers, or wipes.

Allergic contact dermatitis (ACD) is a delayed-type, T-cell mediated hypersensitivity reaction (type IV immune response) wherein the allergens are small haptens that bind to epidermal carrier proteins to form complete antigens. Dermal dendritic cells in the skin process these antigens and present them to naive T cells in the lymph nodes, stimulating an immune response that results in T-cell activation and memory T-cell clonal expansion. Upon re-exposure to the same allergen, the sensitized T cells, primarily T helper 1 (Th1) type, hone to the skin and release inflammatory mediators, producing intercellular edema of the epidermis and the clinical manifestations of ACD. Susceptibility to ACD varies according to age, frequency, type, and duration of allergen exposure.

The pathogenesis of allergic dermatitis (AD) is complex and multifactorial. Traditionally, AD has been categorized as an immune-mediated condition driven primarily by Th2 cells, yet many now believe that the primary defect in AD is a disrupted skin barrier. AD patients often have insufficient amounts of filaggrin, which is critical for the skin to absorb atmospheric water and, thus, maintain moisturization. In addition, decreased amounts of ceramides, fatty molecules that promote water retention within the epidermis and prevent the ingress of foreign harmful substances, have also been demonstrated in the skin of atopics. The resulting compromised epidermal barrier facilitates the passage of pathogens, allergens, and irritants through the skin, leading to a cascade of events that initiates a Th2 response and the clinical manifestations of AD.
Over time, this defective skin barrier, by allowing ingress of allergens and irritants, can predispose the patient to both ACD and irritant contact dermatitis (ICD). Furthermore, recent evidence suggests that the severity and chronicity of AD may be influenced by Th1-type cell immunity. This transition to Th1 immunity could also effectively promote the development of ACD in these individuals.

**Epidemiology of ACD**

The exact incidence and prevalence of ACD in children is unknown and difficult to quantify because the majority of pediatric patients are not referred for patch testing (the gold standard diagnostic method to confirm ACD). Also, pediatric ACD was previously overlooked, based on the belief that children had fewer exposures and less susceptible immune systems.

Referral bias is a frequent confounder in estimating the prevalence of ACD in children. The overwhelming majority of published studies on pediatric ACD examine populations with recalcitrant dermatitis specifically consulted for patch testing on high clinical suspicion of ACD. Uncommon reports of patch testing in unselected (random) populations yield positive patch test rates ranging from 13.3% to 24.5%, which are much lower than the 14.5% to 70.7% prevalence rates noted in one review of the literature of published studies in symptomatic patients.

Yet, it is imperative to understand that a positive patch test does not equate to the clinical diagnosis of ACD. It is imperative to understand that a positive patch test does not equate to the clinical diagnosis of ACD.

Nonetheless, there is increasing evidence to suggest that childhood ACD is not as rare as previously believed, and is, in fact, a growing concern in this population. It has been established that contact sensitization can occur at a young age. In 2008, two US studies demonstrated that both adults and children referred for patch testing had comparable rates of ACD. The studies also emphasize that differences in the age at exposure and the frequency, type, and length of exposure required to induce sensitization to specific chemicals are significant in the development of ACD. These exposure patterns likely account for the common, but slightly different contact allergens in adults vs. children referred for patch testing as well as the variability among geographic locales.

**Clinical Presentation**

As with any skin condition, a thorough history and physical examination is key. Particularly important is obtaining a detailed history of the patient’s as well as siblings’ and parents’ exposures to hygiene products, clothing and footwear, jewelry, topical medications or herbal supplements, hobbies and extracurricular activities (athletic gear and equipment), recent travel, and environments. As the reactions of ACD do not occur until 24 to 48 hours after the re-exposure to the sensitized allergen, it is vital to obtain a detailed account of all events in the preceding week.

Because AD, ACD, and ICD may all present as eczematous dermatitis, it may be difficult to distinguish between them based on clinical findings alone. Classically, ACD presents as a pruritic, eczematous dermatitis typified by edematous vesicles and papules coalescing into plaques. These lesions are highly geometric or linear, and correspond to the sites of greatest allergen exposure and often spread beyond. Sometimes, the eruption can favor certain locations based on skin sensitivity. For example, tosylamide/formaldehyde in nail polish tends to cause ACD symptoms on the thinner skin of the eyelids and neck (see Figure, page 422), but spares the initial sites of contact on the thicker skin of the hands and fingers. “Id” reactions, wherein the lesions are distant from the initial contact site of the culpable allergen or in a generalized distribution over the body, can also occur in ACD. Such
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Spot the Rash
Lawrence F. Eichenfield, MD

The Usual Suspects
Lyme Disease: Fact and Fiction
Margaret C. Fisher, MD, FAAP
Vaccines 2013: An Update on Uptake
Mark H. Sawyer, MD

Keynote Address: Bacterial Meningitis – A New Look at an Old Foe
George H. McCracken Jr., MD

What’s Your Diagnosis?
James H. Brien, DO

Lunch Symposium
Outlook on RSV: Emerging Science
This portion of the meeting is supported by an educational grant from MedImmune. Symposium Director: Robert Belhne, MD

The New Developments
Kawasaki Disease: Typical, Incomplete and the Newer Immunomodulatory Treatments
Stanford T. Shulman, MD

The Resurgence of Measles in Our Neighborhood
Mark H. Sawyer, MD

Red Book 2012 Update
Sarah S. Long, MD

Diagnosis and Treatment of Pneumonia in Children: National Guideline Recommendations
Samir S. Shah, MD, MScE

Neonatal Viral Infections
David W. Kimberlin, MD

Challenging Cases from Private Practice
Stan L. Block, MD, FAAP

SUNDAY, NOVEMBER 18
Breakfast Symposium
HPV Disease Update 2012
This portion of the meeting is supported by an educational grant from Merck & Co, Inc. Symposium Director: Kenneth A. Alexander, MD, PhD

The Usual Suspects
Antibiotics: What’s New, and How Do We Best Use What We Already Have?
George H. McCracken Jr., MD
Sinusitis: A Diagnostic and Management Update
Stan L. Block, MD, FAAP
Lymphadenopathy: The Big, the Bad and the Ugly
C. Buddy Creech, MD, MPH

Keynote Address: Staph Aureus: Continuing Developments in the Epidemic
Sheldon L. Kaplan, MD

What’s Your Diagnosis?
James H. Brien, DO

Lunch Symposium
Infection Protection: New Insights and Ideas for Assessing and Treating Bacterial Conjunctivitis
This portion of the meeting is supported by an educational grant from Alcon Laboratories, Inc. Symposium Director: Rudolph S. Wagner, MD

The New Developments
HPV Disease Manifestations and Prevention
Joseph A. Boschin Jr., MD

Once Bitten, Twice Shy – Preventing Mosquito and Tick Bites
Gordon E. Schutze, MD

Clostridiun difficile – New Diagnostics and Therapeutics
C. Buddy Creech, MD, MPH

PFAPA: Reassuring Families When Periodic Fevers Affect Children
Kathryn M. Edwards, MD

Top 10 Infectious Disease Articles of 2012 for the Pediatrician
Sarah S. Long, MD, Gordon E. Schutze, MD

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reactions may occur with allergens such as nickel, which is one of the top causes of systemic contact dermatitis.\(^1\)

In contrast, ICD is typically less edematous and usually presents as discrete, linear, or geometric erythematous patches matching the contact sites of the offending irritant. Whereas ACD is usually pruritic, ICD is more often characterized by burning.

The location of the body sites involved can also be helpful in the diagnosis. For example, one might suspect clothing allergens when the dermatitis is focused on areas of the body such as the flexures of the extremities or subaxillary bands where garments may chronically rub against the skin. Facial, neck, or periorbital dermatitis may suggest allergy to preservatives or fragrances in cosmetic products.\(^19\) A periumbilical, neck and wrist, or earlobe dermatitis might indicate exposure to nickel in the clasps of jeans, necklace, bracelets, or earrings.\(^1\)

**DIAGNOSIS**

**Patch Testing**

Epicutaneous patch testing is the gold standard for the diagnosis of ACD. The process involves placing defined, preselected concentrations of suspected chemical allergens under occlusion on unaffected areas on the patient’s back and/or inner arms. Intramuscularly injected and oral corticosteroids should not be administered within 4 and 2 weeks, respectively, of the patch test or during testing as they extinguish the patients’ response to the chemicals.

In addition, topical steroids should be discontinued to the back and upper arms (the sites of patch testing) 7 days before and during the entire week of the patch test. However, patients are encouraged to use topical corticosteroids and emollients to treat skin eruptions that develop on sites other than where the patches are placed. Additionally, patients may take oral antihistamines to control pruritus.

Within 48 hours, the patches are removed and the first reading of any reactions is performed. Irritant reactions, which may be follicular or erythematous in nature, may be noted at this first reading, but typically resolve by the time of the delayed read, which is performed between 3 and 7 days.\(^1,18\) As previously mentioned, positive patch tests must be clinically correlated with the patient’s active dermatitis. Avoidance of the suspected allergen with resolution of the dermatitis definitively confirms the diagnosis of ACD.

Currently, patch testing is limited by the fact that the commercially available Thin-Layer Rapid-Use Epicutaneous (T.R.U.E.) test is only approved for individuals 18 years of age and older. Furthermore, allergens may need to be selected specifically for the patient based on exposures because patient size may limit the number of patches that can potentially be placed. Thus, it becomes imperative to screen children with individualized allergens based on personal exposure history and clinical distribution of the dermatitis.

**TREATMENT**

Topical therapies with corticosteroids, emollients, and calcineurin inhibitors, and — if indicated — oral corticosteroids may provide initial symptomatic relief while an avoidance regimen is instituted. Complete avoidance of the offending allergen or irritant is the ultimate treatment for sustained relief of ACD or ICD. Education of the patient, parents, primary health care providers, and teachers is of utmost importance to ensure adequate avoidance of these allergens.\(^1\)

**Special Considerations for ACD and AD**

It is commonly accepted that AD patients may be more susceptible to ICD because the compromised skin barriers of AD patients may allow easier penetration of topical substances on the skin with subsequent irritation.\(^20\) The coexistence of AD with ACD has also been demonstrated.\(^9\) In the past, patients with AD were considered to be less susceptible to developing ACD because of decreased Th1-mediated cellular immunity.\(^5\)

Yet, more recent evidence suggests that although AD is initiated by Th2, it shifts to a predominantly Th1-immunity in the chronic phase.\(^5\) In addition, it has been noted that atopic, but not nonatopic, patients can have positive patch test reactions within 24 hours and that this is a Th2 (type I immediate hypersensitivity) response.\(^5,21,22\) In addition, both nonatopic and atopic patients who have ACD will manifest positive patch test reactions at 48 to 96 hours, the time frame required to elicit a type IV hypersensitivity reaction.\(^2\) Indeed, it has been demonstrated that Th1 and Th2 reactions are not mutually exclusive and can occur concurrently in atopic patients.\(^23\) Thus, it is notable that nickel, which consistently tops the list of clinically relevant allergens in multiple studies,\(^9,15,16\) is not only capable of inducing both type I and type IV hypersensitivity reactions,\(^24\) but has also been shown to induce higher rates of contact allergy in those with loss-of-function filaggrin gene mutations, commonly seen in atopic patients.\(^25\)

More than one study has demonstrated that the rate of contact
sensitization may be proportional to the duration and/or severity of the AD.8,26 Belhadjali and colleagues8 proposed that the much higher prevalence of contact sensitization in atopic patients (60.9%) compared with those with moderate (37.5%) or mild (30%) AD related to the more frequent exposures to topical and oral medications, emollients, and cleansers that atopic children experience.

For example, Mailhol and colleagues26 showed that although contact allergy to topical treatments for AD was not high, it was also not negligible (6.2%), with the most common allergens being emollients and antiseptics (chlorhexidine). Similarly, Nedorost and colleagues27 suggested that AD patients might have more frequent positive patch testing to components of topical medicaments than non-AD patients.

Along these lines, the North American Contact Dermatitis Group (NACDG) demonstrated a higher prevalence of concurrent AD in pediatric patients (compared with adults) who had a relevant positive patch test, which might suggest that AD is a higher risk factor for ACD in children than in adults;16 however, they also noted that AD is more common in children. A study on children referred for patch testing at the University of Miami demonstrated that there was a higher prevalence of certain allergens in their atopic population, specifically nickel, neomycin, disperse dyes, and myroxylon pereirae.9,16,29

In a study by Giordano-Labadie and colleagues28 of 137 atopic children, the most frequent contact sensitizations were not to topical corticosteroids, but rather to metals (19.3%), of which nickel was the primary culprit (14.9%); fragrance (4.4%); lanolin (4.4%); balsam of Peru (2.6%); neomycin (2.6%); and emollients (2.6%). These allergens are not entirely dissimilar to those reported in children with or without AD who were patch tested for suspected ACD in other study centers.9,16

CONCLUSION

Quantifying the prevalence of ACD in the atopic populations (the majority of whom are children) is a difficult task because, as previously described, the lesions of AD and ACD can be similar in morphology. A high index of suspicion is required when diagnosing ACD in atopic patients. The general consensus is that ACD should be considered when atopic patients have worsening disease with an increase in the body surface area affected; disease unresponsive to traditional therapies; present with lesions in specific locations (eyelids, face, hands, neckfolds); have new-onset dermatitis; or present with dyshidrosis.1,6,16,20

Studies have shown that identification of ACD as a possible exacerbating factor to an underlying atopic predisposition or as a possible alternative diagnosis can avoid the need for systemic therapies with significant adverse effects, particularly in a pediatric population that is still undergoing significant active growth and development.29 Thus, patch testing should be undertaken if there is suspicion that ACD may be contributing to recalcitrant dermatitis in the atopic patient. Patch testing may need to be more individualized in the pediatric atopic population and consideration should be given to emollients, topical antibiotics, and topical corticosteroids if clinically indicated.

REFERENCES

18. Jacob SE, Steele T, Brod B, Crawford GH.


