Clinical Management Review

Contact Dermatitis for the Practicing Allergist

David I. Bernstein, MD *Cincinnati, Ohio*

INFORMATION FOR CATEGORY 1 CME CREDIT

Credit can now be obtained, free for a limited time, by reading the review articles in this issue. Please note the following instructions.

Method of Physician Participation in Learning Process: The core material for these activities can be read in this issue of the Journal or online at the *JACI: In Practice* Web site: www.jaci-inpractice.org/. The accompanying tests may only be submitted online at www.jaci-inpractice.org/. Fax or other copies will not be accepted.

Date of Original Release: September 1, 2015. Credit may be obtained for these courses until August 31, 2016.

Overall Purpose/Goal: To provide excellent reviews on key aspects of allergic disease to those who research, treat, or manage allergic disease.

Target Audience: Physicians and researchers within the field of allergic disease.

Accreditation/Provider Statements and Credit Designation: The American Academy of Allergy, Asthma & Immunology (AAAAI) is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians. The AAAAI designates these educational activities for a maximum of 1 AMA PRA Category 1 Credit. Physicians should only claim credit commensurate with the extent of their participation in the activity.

List of Design Committee Members: David I. Bernstein, MD

Activity Objectives

Learning objectives:

1. To recognize that contact dermatitis should be considered in the differential diagnosis of any patient presenting with a pruritic eczematous rash.

2. To understand that history and physical examination alone may not reliably differentiate irritant contact dermatitis (ICD) from allergic contact dermatitis (ACD).

3. To interpret patch test results in a standardized manner, with the initial reading at 48 hours and the second reading at 3 to 7 days after the application of patch tests.

4. To educate patients regarding the clinical relevance of positive patch test results.

Recognition of Commercial Support: This CME has not received external commercial support.

Disclosure of Significant Relationships with Relevant Commercial Companies/Organizations: D. I. Bernstein is on the Joint Task Force for Practice Parameters and the American Board of Allergy and Immunology; has received consultancy fees from Teva, Novartis, Circassia, and Merck; has received research support from Amgen, Circassia, GlaxoSmithKline, Johnson & Johnson, Merck, Teva, Pfizer, Genentech, Novartis, Boehringer Ingelheim, AstraZeneca, Sanofi, Mylan, Chase, Oriel, Pearl, Cephalon, and Janssen; and has received lecture fees from Merck.

This article provides an overview of important practice recommendations from the recently updated Contact Dermatitis Practice Parameter.[1] This updated parameter provides essential recommendations pertaining to clinical history, physical examination, and patch testing evaluation of patients suspected of allergic contact dermatitis. In addition to providing guidance for performing and interpreting closed patch testing, the updated parameter provides concrete recommendations for assessing metal hypersensitivity in patients receiving prosthetic devices, for evaluating workers with occupational contact dermatitis, and also for addressing allergic contact dermatitis in children. Finally, the document provides practical recommendations useful for educating patients regarding avoidance of exposure to known contact sensitizers in the home and at work. The Contact Dermatitis Parameter is designed as a practical, evidence-based clinical tool to be used by allergists and dermatologists who routinely are called upon to evaluate patients with skin disorders. © **2015 American Academy of Allergy, Asthma & Immunology (J Allergy Clin Immunol Pract 2015;3:652-8)**

Key words: *Allergen; Contact; Dermatitis; Sensitizer; Patch test; Allergy; Allergic*

Patients with cutaneous eruptions are commonly referred to the allergist's office for evaluation of possible allergic contact dermatitis (ACD). For this reason, the practicing allergist must be familiar with common sensitizers that are recognized causes of ACD as well as environmental sources of exposure. A working knowledge of ACD enables the

Division of Immunology, Allergy and Rheumatology, University of Cincinnati College of Medicine, Cincinnati, Ohio

Conflicts of interest: D. I. Bernstein is on the Joint Task Force for Practice Parameters and the American Board of Allergy and Immunology; has received consultancy fees from Teva, Novartis, Circassia, and Merck; has received research support from Amgen, Circassia, GlaxoSmithKline, Johnson & Johnson, Merck, Teva, Pfizer, Genentech, Novartis, Boehringer Ingelheim, AstraZeneca, Sanofi, Mylan, Chase, Oriel, Pearl, Cephalon, and Janssen; and has received lecture fees from Merck.

Received for publication March 31, 2015; revised May 18, 2015; accepted for publication June 4, 2015.

Corresponding author: David I. Bernstein, MD, Division of Immunology, Allergy and Rheumatology, University of Cincinnati College of Medicine, 231 Albert Sabin Way, Cincinnati, OH 45267. E-mail: BERNSTDD@UCMAIL.UC.EDU.

2213-2198

http://dx.doi.org/10.1016/j.jaip.2015.06.006

Abbreviations used
ACD- allergic contact dermatitis
CD- contact dermatitis

identification of appropriate allergens for closed patch testing needed to confirm a diagnosis. An allergy consultant working in this area must understand how to correctly apply patch tests, accurately interpret patch test responses, and define their clinical relevance.

For these reasons, the Joint Task Force for Practice Parameters commissioned a workgroup of experts to update the Contact Dermatitis Practice Parameter initially published in 2006. The updated Contact Dermatitis Practice Parameter, recently published in the *Journal of Allergy and Clinical Immunology In Practice*, is written as a practical clinical guide for the practicing allergist.[1] This update provides clinically useful, evidence-based recommendations pertaining to medical history, patch testing, and overall management. An outline of its content is listed in Table I. The final document was peer-reviewed by members of the Joint Taskforce as well as external reviewers including allergists and dermatologists with recognized expertise in the field. Novel contemporary issues in the updated parameter include preoperative patch test screening for metal allergy; evaluation and management of occupational contact dermatitis (CD); potential role and limitations of drug patch testing; and comprehensive aspects of disease management including avoidance and prevention.[1]

This review will highlight key points from the updated Contact Dermatitis Practice Parameter, emphasizing important "clinical pearls" to assist the allergist in recognizing, diagnosing, and managing challenging patients.[1] Specifically, the following questions are addressed:

1. What are the clinically useful clues in identifying potential causes of ACD?
2. Which dermatologic conditions should be considered in the differential diagnosis?
3. In which patients should patch testing be performed?
4. How should patch testing be optimally performed and interpreted?
5. What are the common causes of occupational CD?
6. What are the most common sources of contact allergens?
7. When should presurgical patch testing for metals be considered?

CLINICAL CLUES

CD should be considered in the differential diagnosis of any patient presenting with a pruritic skin rash with erythematous papules, vesicles, or an eczematous rash with crusted lesions. Chronic CD is associated with secondary skin changes such as lichenification, fissuring, thickening, and scaling. It is well known that the clinical history and physical appearance of skin are often not reliable in differentiating ACD from irritant CD, and patch testing is often the only way to distinguish the 2 conditions. There are also a number of other dermatologic conditions that can be confused with ACD and should be considered in the differential diagnosis. Table II appears in the updated parameter and lists the clinical features of various skin conditions useful in differentiating ACD from irritant CD, atopic dermatitis, seborrheic dermatitis, dyshidrotic eczema, psoriasis, dermatitis herpetiformis, and mycosis fungoides.[1] In some cases, the skin biopsy may be used for differentiating CD from some of the aforementioned skin disorders. ACD can coexist with any of these conditions when patients develop allergy to topically applied products or medications; secondary ACD, for example, is commonly recognized in patients with atopic dermatitis.

TABLE I. Outline of content of the Updated Contact Dermatitis Practice Parameter[1]

1. Evaluation (Summary Statements 1-15)
• Medical history and examination
• Differential diagnosis
• Geographical location providing clues to causation
2. Patch testing recommendations (Summary Statements 16-27)
• Choosing appropriate test antigens
• Test devices
• When and how to interpret patch tests
• Recognizing and managing possible false-negative and false-positive results
• Testing for photoallergic dermatitis
3. Sources of exposure to relevant contact allergens (Summary Statements 28-33)
• Airborne exposure
• Personal care products
• Hair products
• Ectopic transfer of allergen to other areas of the skin
• Causes and sources of photo ACD
4. Iatrogenic causes (Summary Statements 34-37)
• Topical medications
• Preoperative testing for metal allergy
• Drug patch testing
5. Special patient populations (Summary Statements 38-41)
• Children
• Workers
6. Treatment and prevention (Summary Statements 42-45)

Geographical location of the cutaneous eruption provides clues

The North American Contact Dermatitis Group identified the face, hands, and generalized distribution over the entire body as the 3 most commonly involved geographical areas for involvement with ACD.[2] Rashes located in specific sites including the face, eyelids, lips, neck and scalp, hands, axilla, anogenital region, feet, and legs provide specific clues as to causation. Table III lists different geographical locations of eruptions, potential sources of exposure, and specific causative allergens.

Women are at a greater risk for facial ACD caused by chemical and natural botanical sensitizers contained in personal care or cosmetic products applied to the face. Airborne facial exposure from plant sources (eg, seasonal pollens) as well as inadvertent ectopic transfer of contact allergens by hands from other parts of the body should be considered.[3] Nickel, natural botanical ingredients, and nail product chemicals (acrylates, tosylamide/formaldehyde resin) are often ectopically transferred from other sites, causing eyelid dermatitis.[4]

TABLE II. Clinical characteristics of dermatologic disorders considered in the differential diagnosis of ACD

Dermatologic condition	Differentiating features and clues to diagnosis
Irritant CD	• Glazed, parched, or scalded appearance
	• Sharply circumscribed dermatitis
	• Healing begins promptly on withdrawal of offending agent
	• Patch testing negative
Atopic dermatitis	• Personal or family history of atopy
	• Early age of onset
	• Chronic and recurrent
	• Dry, scaly, very pruritic
	• Typical distribution
	Facial in infancy
	Extensors in early childhood
	Flexural areas in adolescents and adults
Seborrheic dermatitis	• Distribution: areas with sebaceous glands
	Scalp, periauricular, face (medial eyebrows, glabella, nasolabial folds), presternal trunk, interscapular
	• Blepharitis common
	• Dandruff appears to be a precursor
	• Distinctive morphology: dull, yellowish-red, sharply demarcated lesions covered with greasy-looking scales
Dyshidrotic eczema	• Small (1-2 mm) vesicles, deep seated on nonerythematous base "tapioca" or "sago grain" like
	• Palms, soles, and/or lateral aspects of fingers, often symmetrical
	• Intensely pruritic and itching prodrome
	• Persists for 2 to 3 wk, then resolves by involution and desquamation
Psoriasis	• Plaques typically have dry, thin, silvery-white or micaceous scale
	• Auspitz sign: removing scale reveals a smooth, red, glossy membrane with tiny punctate bleeding
Dermatitis herpetiformis	• Genetic predisposition for gluten sensitivity
	• Intensely pruritic
	• Symmetrically grouped (herpetiform) papules and vesicles
	Elbows, knees, buttocks, scapulae, scalp
	Direct immunofluorescence of the skin shows granular IgA at dermal papillae and occasionally along the dermoepidermal border
Mycoses fungoides and cutaneous T-cell lymphoma	• Patches with thin, wrinkled quality, often with reticulated pigmentation
	• Pruritus varies from minimal or absent to common in premycotic phase and may precede mycoses fungoides by years
	• Often on lower trunk and buttocks
	• Cutaneous biopsy required for confirmation

Adapted from Fonacier et al.[1]

Acute or chronic lip inflammation (cheilitis) is usually caused by chemical or physical irritation. Fragrances and flavoring chemicals contained in lip and oral hygiene products (eg, lip balm and toothpaste) are sensitizers responsible for approximately one third of the cases of acute contact cheilitis.[5,6] Because the oral mucosa is considered an immune privileged site and unlikely to be involved with ACD, other diagnoses should be considered (eg, lichenoid tissue reactions and recurrent aphthous stomatitis).

When dermatitis is localized to the scalp and neck, metal sensitizers in jewelry or chemicals in hair products or cosmetics should be considered. Females are twice as likely to be diagnosed with ACD by virtue of greater exposure to cosmetics, hair products, and jewelry.[7] Common allergens found in hair products are listed in Table III. When the scalp is rarely affected, medicinal products (eg, neomycin) or hair products may be responsible.[8]

Although hand dermatitis is usually caused by irritants (ie, irritant CD), ACD is more commonly diagnosed in occupations such as hairdressing and health care.[9] Common sensitizers associated with hand dermatitis are listed in Table III. Patch testing is highly recommended in all patients presenting with chronic hand eczema.

Dermatitis localized to the axilla can be due to allergic reactions to fragrance chemicals in deodorants (Table III).[10] Antiperspirants rarely contain sensitizers and more often cause irritant dermatitis.[11] Disperse blue dyes in clothing can cause ACD involving the outer axillary skin and should be suspected with involvement of other skin areas coming in direct contact with clothing.[12-14] Anogenital dermatitis can be caused by irritants or sensitizers contained in topical medications (topical corticosteroids, fragrances, neomycin).[15] A new preservative, methylisothiazolinone, used in wet baby wipes may cause ACD involving the perianal areas of children.[16]

ACD should be suspected and evaluated in patients presenting with chronic dermatitis of the feet or soles. The sources of these sensitizers almost always are chemicals used to manufacture shoes including adhesives, chromates, and rubber chemicals

TABLE III. Geographical location of cutaneous eruptions with sources and causative allergens[1]

Geographical location	Sources of sensitizers	Causative allergens
Face	Cosmetics, plant sources, topical medicines, ectopic transfer resulting in eyelid and periorbital dermatitis (nickel, nail enamel)	Botanical ingredients, airborne pollen (Compositae), fragrances, Balsam of Peru, neomycin, methyl methacrylate (artificial nails), tosylamide/ formaldehyde (nail polish)
Lip inflammation (cheilitis)	Lip and oral hygiene products (eg, lip balm and toothpaste)	
Scalp and neck	Cosmetics, hair products, and jewelry	*Hair products:* Paraphenylenediamine, glycerol thioglycolate (permanent wave products); cocoamidopropyl betaine (shampoo surfactant) *Cosmetics:* Fragrances, Balsam of Peru, and Quarternium-15
Hands	Cosmetics, rubber gloves	Quarternium-15 (a preservative), Balsam of Peru, nickel, fragrance mix, topical antibiotics (eg, neomycin), rubber chemicals (thiurams, carbamates, mercaptobenzothiazole)
Axilla	Deodorants, clothing dyes	Fragrance chemicals: hydroxyisohexyl-3-cyclohexene, cinnamic aldehyde; disperse blue dyes
Anogenital	Topical medications, diaper products	Topical corticosteroids, fragrances, neomycin; methylisothiazolinone preservative in baby wipes
Feet or soles	Shoe materials or chemicals including adhesives, chromates, and rubber chemicals	Dialkyl thioureas, carbamates, thiurams, chromates
Legs	Topical preparations often to treat leg ulcers	Fragrances, Balsam of Peru, antibiotics, topical corticosteroids, and lanolin
Sun-exposed areas	Photoallergens in sunscreens	Para-aminobenzoic acid (PABA)

(Table III). Topical preparations applied to treat leg ulcers may cause ACD and contain many possible sensitizers and are listed in Table III.[17,18] ACD caused by allergy to components of topical preparations could prolong the healing of leg ulcers, and products containing sensitizers should be avoided in such patients.

The "Baboon syndrome" or multiple eruptions localized to axillary, intertriginous, and flexural areas may be an expression of systemic CD. In this case, dermatitis is elicited by systemic exposure via ingestion or infusion to known contact sensitizers. Although uncommon, systemic CD has been associated with ingestion of metal in patients with metal allergy (eg, nickel, mercury, or gold); intravenous aminoglycosides in patients sensitized to neomycin; ingestion of foods that contain chemicals in Balsam of Peru fragrance (citrus, cinnamon chutney); or systemic administration of corticosteroids.[19,20]

Photoallergic dermatitis should be suspected in patients presenting with eczema affecting light-exposed areas of the skin such as the anterior neck, dorsal aspects of the hands, and forearms. The upper eyelids, upper lips, and postauricular areas are often spared. Chemicals in sunscreens are frequently identified as photoallergens. Titanium dioxide and zinc oxide are very commonly used nonallergenic ultraviolet blockers and have not been reported to cause ACD or photoallergic reactions.

CD due to sensitization to pollen allergens should be considered in patients presenting with seasonal history of eczematous dermatitis affecting air-exposed skin during a pollen season. This can often be attributed to weed allergens from species of the Compositae family. Although not standardized, "atopy patch tests" can be carefully performed with pollen extracts and read at 24 and 72 hours.[21,22]

PATCH TESTING: INDICATIONS

If allergy is suspected, patch testing should be considered in any patient with acute or chronic dermatitis regardless of age. Although the exposure and medical history is very useful, studies have shown that the history and physical examination have moderate sensitivity and specificity for diagnosing ACD.[23] In 1 study, in 50% of the patients with nonspecific generalized dermatitis, contact sensitization was demonstrated to clinically relevant sensitizers.[24] Although atopic dermatitis is associated with an abnormal skin barrier, it is uncertain whether patients with atopic dermatitis are at a greater risk for ACD than are nonatopic individuals.[25] Nevertheless, patch testing should strongly be considered in patients presenting with uncontrolled atopic dermatitis to avoid missing a secondary diagnosis of ACD. Severe recalcitrant cases of atopic dermatitis have been ascribed to sensitizers contained in topical medications (eg, topical corticosteroids).[26]

PATCH TESTING: PROCEDURES

The clinician should be familiar with medications that can modify or reduce patch test responses in sensitized individuals. More details about patch testing are included in an accompanying article in this issue.[27] Diminished patch test reactivity occurs among patients receiving more than the equivalent of 20 mg of oral prednisone daily. If necessary, patch testing can still be performed in patients receiving low-dose prednisone or cyclosporin without compromising interpretation.[28] Patch tests can be affected by ultraviolet irradiation or topical application of high- and medium-potency corticosteroids and should be withdrawn for at least 5 to 7 days before applying patch tests.[29-31]

TABLE IV. Cosmetic preservatives[1]

Formaldehyde releaser	Nonformaldehyde releaser
Formaldehyde	Iodopropynylbutylcarbamate
Quarternium 15	Methychloroisothiazolinone/ methylisothiazolinone (MCI/MI)
Diazolidinyl urea	Parabens
Imidazolidinyl urea	Methyldibromoglutaronitrile
Bromonitropropane	Chloroxylenol
DMDM hydantoin	Benzalkorium chloride
	Thimerosal
	Phenoxyethanol

Concurrent oral antihistamine treatment has no significant impact on patch test results.[32]

Selection of relevant allergens based on exposure history is very important. Although many dermatologists and allergists use a standard panel (eg, North American Contact Dermatitis Group panel of 70 antigens or TRUE Test containing 35 antigens), as many as 25% of the patients are sensitized to clinically relevant allergens not included in a standard antigen panel.[2] Such patients often react to antigens from extended standard panels or specialized supplementary panels prepared for specific occupations (eg, cosmetologists and machinists) or exposure sources (eg, reactive dye panels in clothing and topical medications panels).

There are 2 categories of testing devices available to perform patch testing, either the preloaded device (the preloaded thin-layer rapid-use epicutaneous testing kit such as TRUE Test) or individually loaded chamber systems such as the aluminum Finn chamber. Surprisingly, there is only moderate concordance (62%-63%) in comparative studies of the same antigens tested with preloaded devices versus individually loaded chamber systems.[33,34]

PATCH TEST INTERPRETATION

The International Contact Dermatitis Research Group scoring system is most widely used for interpreting patch test results.[35] All patch tests must be removed and read 48 hours after application. Nonspecific redness from pressure caused by the patch device and tape should be allowed to dissipate for 30 minutes before reading. Because as many as 30% of relevant sensitizers may yield a false-negative result at 48 hours, second readings are almost always performed between 3 and 7 days after application.[36,37] It is not entirely clear which time interval is optimal for a second reading, although a report suggests 96 hours.[37] Delayed late reactions at 7 or more days are more commonly observed with metal antigens, topical antibiotics such as neomycin, topical corticosteroids, glues, and preservatives. Positive tests that are negative at 48 hours but increase at 96 hours and 7 days (ie, crescendo) are likely allergic, whereas positive or questionable reactions that appear at 48 hours and then disappear at later readings (decrescendo) are more consistent with irritant reactions.[38]

SOURCES OF EXPOSURE TO CONTACT ALLERGENS

In the initial evaluation of a patient suspected of ACD, it is important to review all personal care products as well as potential sources of allergens encountered in the home or workplace. The clinician should be attuned to any change in the work environment or job that could result in new exposures to chemicals. Nearly all health care workers use gloves, resulting in rubber chemical exposure, and experience irritation from frequent hand washing. Material safety data sheets should be requested and may reveal hidden sensitizers. Certain hobbies (eg, photography and gardening) may result in frequent exposure to sensitizing materials.

Personal care products are major sources of exposure to allergens, with the most common being fragrances, topical antibiotics, preservatives, excipients, nickel, and sunscreens. As already mentioned, cosmetics usually cause dermatitis at the site of the application, but sources of allergens can also be inadvertently ectopically transferred from distal sites.

Fragrances are common ingredients of cosmetics, household products, and topical medications. Products advertised as "unscented" can contain masking fragrances; there are indeed fragrance-free products that can be tolerated by fragrance-sensitized individuals. Botanicals (eg, tree oils), even those contained in fragrance-free personal care products, can be highly allergenic.[39,40] Patch testing with Balsam of Peru, Fragrance Mix I, and Fragrance Mix II will detect 73% of individuals allergic to fragrances.[41] Because manufacturers are not required to list specific fragrance ingredients, it is often useful to perform a usage test to determine whether a given product can be tolerated.

Preservatives are commonly used in personal care products and cosmetics and classified either as formaldehyde releasers or as nonformaldehyde releasers. The most common sensitizers in each category are listed in Table IV and are included in most standard patch test panels. Twenty percent of personal care products sold in the United States contain a formaldehyde releaser preservative.[42] Obviously, patients allergic to formaldehyde should avoid products containing these preservatives. In recent years, methyldibromoglutaronitrile (contained in Euxyl K 400) is being recognized as a common nonformaldehyde-releasing agent sensitizer. Not only is this commonly used in body lotions and hair products but is also present in solvents and oils, representing an important cause of occupational hand dermatitis. Methylisothiazolinone and methylchloroisothiazolinone are preservatives and important sensitizers used in various personal care products including shampoos, hair conditioners, baby soaps, and baby wipes as well as in industrial products such as metal working fluids.

It is important to emphasize again that many types of botanicals or plant extracts are commonly added to many skin care products. These are important causes of ACD and should be suspected as potential causative sensitizers along with preservatives and fragrances. Examples of these are tea tree oil, used in skin products, and propolis found in lip balm, toothpastes, and many other personal care products. Because standard patch tests may not be available for plant extracts and essential oils, patients should be made aware of their sensitizing potential. When strongly suspected, avoidance of such products containing plant materials can be curative.

Hair products including dyes, shampoos, and permanent wave solutions are key sources of potential sensitizers. The most important chemical sensitizer in hair dye products is paraphenylenediamine, causing ACD in consumers and hairdressers. However, there are many other sources of exposure to paraphenylenediamine, which is used to manufacture leather, rubber

products, and textiles. It is also used as an ingredient in body painting and application of black henna tattoos.[43] Glycerol thioglycolate is an essential ingredient in permanent wave products and can be problematic in sensitized patients because it is not easily washed out of hair. Shampoo and other personal cleaning products often contain cocoamidopropyl betaine, a surfactant that can cause ACD appearing on the eyelids, face, scalp, and neck.

ACD from nail products is usually due to tosylamide/formaldehyde resin, a chemical sensitizer in nail polish enamel. Interestingly, only a minority of cutaneous reactions in affected individuals appear around the nails, with 80% of the reactions affecting the neck, face, lips, and eyelids from ectopic transfer.[44] Acrylic nail products can cause allergic skin reactions, and specialized acrylic test panels are available for confirming allergy.

Topical corticosteroids and topical antibiotics are used to treat many inflammatory and infectious skin conditions. However, when a rash worsens rather than improves, ACD due to the topical medications or ingredients should be considered. Sensitizers found in topical medications include lanolin, para-aminobenzoic acid (sunscreens), antibiotics, antihistamines, nonsteroidal anti-inflammatory agents, and corticosteroids. Most standard patch test panels contain corticosteroid antigens. Ninety percent of cases due to corticosteroids can be identified by patch testing with tixocortol, budesonide, triamcinolone, and the patient's commercial steroid product. The updated parameter document lists potential corticosteroid sensitizers, and these are categorized into 4 major groups (Group A, B, C, and D) on the basis of known intragroup patterns of clinical cross-reactivity.[1] Upon Identification of a specific corticosteroid sensitizer, patients with ACD should be advised to avoid all other products in that group.

PREOPERATIVE PATCH TESTING FOR METAL ALLERGY

Allergy to surgical implants is discussed in detail in another article in this issue.[45] Patch testing to metal should be considered for patients with a relevant history of metal allergy but not for patients lacking a history. There is inadequate evidence to support broad recommendations or guidelines for replacing implants in patients with metal hypersensitivity. Replacing a joint or prosthesis is a difficult decision that must take into account the clinical circumstances of each case combined with the desires of the patient and the clinical judgment of the treating physician.

ACD IN CHILDREN

ACD is a common problem in children and may be occur as frequently in teenagers as in adults. ACD in children is the subject of an accompanying article in this issue.[46] Common sources of allergens can be elicited by taking an age-appropriate history, which may identify potential sensitizers in diaper and hygiene products, cosmetics, sun blocks, textiles and dyes, medications, and sporting accessories (eg, dialky thioruea or p-tert butyl formaldehyde in shin guards or protective pads).

OCCUPATIONAL CD

CD is one of the most common occupational illnesses affecting up to 10% of the US work force and incurring annual costs exceeding $1 billion. Occupational dermatitis is categorized as being allergic or irritant in nature, with irritant CD representing 80% of all cases. Common causative irritants include wet work, solvents, alcohols, cutting oils, coolants, degreasers, soaps, and detergents. The most important occupational sources of sensitizers causing ACD include metals, rubber compounds, epoxy resins, acrylics, organic dyes, plant materials, foods, medications, and biocides germicides. The most common jobs or occupations associated with occupational CD are food services, cosmetology, health care, agriculture, cleaning, painting, mechanical work, electronic work, printing, and construction. Outdoor workers and those exposed to plants are at risk for ACD caused by Toxicodendron plant−derived allergens including poison ivy, poison oak, and poison sumac. The allergen uroshiol binds to skin but is water soluble, and the skin area impacted should be washed immediately. The diagnosis is based on history, and patch testing with Toxicodendron is contraindicated because of severe local reactions and the possibility of sensitizing a nonsensitized patient.

The hands are most commonly involved with occupational CD. It is essential for the treating physician to confirm the diagnosis of occupational CD by demonstrating aggravation or causation in the work environment. There are 7 accepted criteria that establish work-related causation or aggravation: (1) clinical appearance consistent with CD; (2) suspected causes present in the work environment; (3) anatomic distribution consistent with occupational exposure; (4) the temporal relationship between occupational exposure and appearance of dermatitis; (5) exclusion of nonoccupational causes; (6) dermatitis that improves with avoidance and recurs upon reexposure; and (7) demonstration of relevant positive patch test results to offending allergens.[47]

TREATMENT AND MANAGEMENT OF CD

Once causative irritants or allergens are identified, patients should be advised about avoidance measures and recognize substances that are cross-reactive with relevant sensitizers. It is very challenging for the patient to recall the chemical names of sensitizers and to comply with avoidance recommendations. Two online databases provide a list of products to patients that are free of specific sensitizers. The Contact Allergen Management Program is accessible to physicians who belong to the American Contact Dermatitis Society (www.contactderm.org), and the Mayo Clinic has made available the SkinSAFE database (www.SkinSAFEapp.com) to physicians and consumers. In some cases, failure to comply with irritant or sensitizer avoidance recommendations can result in chronic, severe, and disabling dermatitis.

Medical treatment may be indicated for acute or chronic dermatitis. Topical corticosteroids may be sufficient, but systemic steroids may be necessary for more severe generalized dermatitis. Prolonged use of systemic steroids should be avoided. Use of potent fluorinated topical corticosteroids should be avoided in areas with thin skin such as the face and eyelids. As mentioned, failure to respond or worsening dermatitis may result from contact sensitization to the topical steroid or its other product ingredients.[48]

CD can be prevented by avoiding exposure to known irritants and allergens by use of appropriate skin protection. In the workplace, if possible, this can be accomplished by substituting the use of an alternative material that is nonsensitizing. Use of personal protective equipment including gloves, goggles, face shields, and full-body uniforms is useful in preventing skin

exposure. Barrier creams applied to the skin may or may not be useful in a work environment.[49] It is important to educate workers that the dermatitis can persist for months or even years despite the institution of appropriate job exposure modification or complete avoidance.

REFERENCES

1. Fonacier L, Bernstein DI, Pacheco K, Holness DL, Blessing-Moore J, Khan D, et al. Contact dermatitis: a practice parameter-update 2015. J Allergy Clin Immunol Pract 2015;3:S1-39.
2. Warshaw EM, Belsito DV, Taylor JS, et al. North American Contact Dermatitis Group patch test results: 2009 to 2010. Dermatitis 2013;24:50-9.
3. Feser A, Plaza T, Vogelgsang L, Mahler V. Periorbital dermatitis—a recalcitrant disease: causes and differential diagnoses. Br J Dermatol 2008;159:858-63.
4. Guin JD. Eyelid dermatitis: a report of 215 patients. Contact Dermatitis 2004; 50:87-90.
5. Zug KA, Kornik R, Belsito DV, DeLeo VA, Fowler JF Jr, Maibach HI, et al. North American Contact Dermatitis Group. Patch-testing North American lip dermatitis patients: data from the North American Contact Dermatitis Group, 2001 to 2004. Dermatitis 2008;19:202-8.
6. Lim SW, Goh CL. Epidemiology of eczematous cheilitis at a tertiary dermatological referral centre in Singapore. Contact Dermatitis 2000;43:322-6.
7. Warshaw EM, Buchholz HJ, Belsito DV, Maibach HI, Fowler JF Jr, Rietschel RL, et al. Allergic patch test reactions associated with cosmetics: retrospective analysis of cross-sectional data from the North American Contact Dermatitis Group, 2001-2004. J Am Acad Dermatol 2009;60:23-38.
8. Hillen U, Grabbe S, Uter W. Patch test results in patients with scalp dermatitis: analysis of data of the Information Network of Departments of Dermatology. Contact Dermatitis 2007;56:87-93.
9. Carlsen BC, Andersen KE, Menne T, Johansen JD. Sites of dermatitis in a patch test population: hand dermatitis is associated with polysensitization. Br J Dermatol 2009;161:808-13.
10. Bruze M, Johansen JD, Andersen KE, et al. Deodorants: an experimental provocation study with cinnamic aldehyde. J Am Acad Dermatol 2003;48:194-200.
11. Gall H, Kempf E. [Contact allergy due to the topical antiperspirant propantheline bromide (author's transl)] [in German]. Derm Beruf Umwelt 1982;30:55-6. 57.
12. Giusti F, Massone F, Bertoni L, Pellacani G, Seidenari S. Contact sensitization to disperse dyes in children. Pediatr Dermatol 2003;20:393-7.
13. Ryberg K, Isaksson M, Gruvberger B, Hindsen M, Zimerson E, Bruze M. Contact allergy to textile dyes in southern Sweden. Contact Dermatitis 2006;54: 313-21.
14. Wentworth AB, Richardson DM, Davis MD. Patch testing with textile allergens: the Mayo Clinic experience. Dermatitis 2012;23:269-74.
15. Warshaw EM, Furda LM, Maibach HI, Rietschel RL, Fowler JF Jr, Belsito DV, et al. Anogenital dermatitis in patients referred for patch testing: retrospective analysis of cross-sectional data from the North American Contact Dermatitis Group, 1994-2004. Arch Dermatol 2008;144:749-55.
16. Chang MW, Nakrani R. Six children with allergic contact dermatitis to methylisothiazolinone in wet wipes (baby wipes). Pediatrics 2014;133:e434-8.
17. Smart V, Alavi A, Coutts P, Fierheller M, Coelho S, Linn Holness D, et al. Contact allergens in persons with leg ulcers: a Canadian study in contact sensitization. Int J Low Extrem Wounds 2008;7:120-5.
18. Barbaud A, Collet E, Le Coz CJ, Meaume S, Gillois P. Contact allergy in chronic leg ulcers: results of a multicentre study carried out in 423 patients and proposal for an updated series of patch tests. Contact Dermatitis 2009;60:279-87.
19. Theler B, Bucher C, French LE, Ballmer Weber B, Hofbauer GF. Clinical expression of nickel contact dermatitis primed by diagnostic patch test. Dermatology 2009;219:73-6.
20. Jacob SE, Zapolanski T. Systemic contact dermatitis. Dermatitis 2008;19:9-15.
21. Kerschenlohr K, Darsow U, Burgdorf WH, Ring J, Wollenberg A. Lessons from atopy patch testing in atopic dermatitis. Curr Allergy Asthma Rep 2004;4:285-9.
22. Darsow U, Abeck D, Ring J. [Allergy and atopic eczema: on the value of the "atopy patch test"] [in German]. Hautarzt 1997;48:528-35.
23. Josefson A, Farm G, Meding B. Validity of self-reported nickel allergy. Contact Dermatitis 2010;62:289-93.
24. Zug KA, Rietschel RL, Warshaw EM, et al. The value of patch testing patients with a scattered generalized distribution of dermatitis: retrospective cross-sectional analyses of North American Contact Dermatitis Group data, 2001 to 2004. J Am Acad Dermatol 2008;59:426-31.
25. Thyssen JP, Linneberg A, Engkilde K, Menne T, Johansen JD. Contact sensitization to common haptens is associated with atopic dermatitis: new insight. Br J Dermatol 2012;166:1255-61.
26. Jacob SE, Brod B, Crawford GH. Clinically relevant patch test reactions in children—a United States based study. Pediatr Dermatol 2008;25:520-7.
27. Fonacier L. Patch testing. J Allergy Clin Immunol Pract 2015;3:669-75.
28. Rosmarin D, Gottlieb AB, Asarch A, Scheinman PL. Patch-testing while on systemic immunosuppressants. Dermatitis 2009;20:265-70.
29. Green C. The effect of topically applied corticosteroid on irritant and allergic patch test reactions. Contact Dermatitis 1996;35:331-3.
30. Green C. Effect of previous topical corticosteroid on patch testing. Contact Dermatitis 2007;56:300.
31. Prens EP, Benne K, Geursen-Reitsma AM, van Dijk G, Benner R, van Joost T. Effects of topically applied glucocorticosteroids on patch test responses and recruitment of inflammatory cells in allergic contact dermatitis. Agents Actions 1989;26:125-7.
32. Chen XJ, Chen LL, Shi X, Xie LX, Leng H, Ji J, et al. The effect of desloratadine on patch test reactions in Chinese patients. Asian Pac J Allergy Immunol 2012;30:209-13.
33. Goh CL. Comparative study of TRUE Test and Finn Chamber patch test techniques in Singapore. Contact Dermatitis 1992;27:84-9.
34. Wilkinson JD, Bruynzeel DP, Ducombs G, et al. European multicenter study of TRUE Test, Panel 2. Contact Dermatitis 1990;22:218-25.
35. Wilkinson DS, Fregert S, Magnusson B, Bandmann HJ, Calnan CD, Cronin E, et al. Terminology of contact dermatitis. Acta Derm Venereol 1970;50:287-92.
36. Davis MD, Bhate K, Rohlinger AL, Farmer SA, Richardson DM, Weaver AL. Delayed patch test reading after 5 days: the Mayo Clinic experience. J Am Acad Dermatol 2008;59:225-33.
37. Geier J, Gefeller O, Wiechmann K, Fuchs T. Patch test reactions at D4, D5 and D6. Contact Dermatitis 1999;40:119-26.
38. Flannigan SA, Smolensky MH, Harrist R, Machinski G, McGovern JP. Time considerations in scoring contact irritant patch test sites. Contact Dermatitis 1983;9:519-20.
39. Johansen JD. Fragrance contact allergy: a clinical review. Am J Clin Dermatol 2003;4:789-98.
40. Ortiz KJ, Yiannias JA. Contact dermatitis to cosmetics, fragrances, and botanicals. Dermatol Ther 2004;17:264-71.
41. Wenk KS, Ehrlich A. Fragrance series testing in eyelid dermatitis. Dermatitis 2012;23:22-6.
42. de Groot A, White IR, Flyvholm MA, Lensen G, Coenraads PJ. Formaldehyde-releasers in cosmetics: relationship to formaldehyde contact allergy, part 2: patch test relationship to formaldehyde contact allergy, experimental provocation tests, amount of formaldehyde released, and assessment of risk to consumers allergic to formaldehyde. Contact Dermatitis 2010;62:18-31.
43. Schnuch A, Lessmann H, Frosch PJ, Uter W. para-Phenylenediamine: the profile of an important allergen. Results of the IVDK. Br J Dermatol 2008;159: 379-86.
44. Lazzarini R, Duarte I, de Farias DC, Santos CA, Tsai AI. Frequency and main sites of allergic contact dermatitis caused by nail varnish. Dermatitis 2008;19: 319-22.
45. Pacheco KA. Allergy to surgical implants. J Allergy Clin Immunol Pract 2015; 3:683-95.
46. Goldenberg A, Silverberg N, Silverberg JI, Treat J, Jacob SE. Pediatric allergic contact dermatitis: lessons for better care. J Allergy Clin Immunol Pract 2015;3: 661-7.
47. Mathias CG. Contact dermatitis and workers' compensation: criteria for establishing occupational causation and aggravation. J Am Acad Dermatol 1989;20: 842-8.
48. Davis MD, el-Azhary RA, Farmer SA. Results of patch testing to a corticosteroid series: a retrospective review of 1188 patients during 6 years at Mayo Clinic. J Am Acad Dermatol 2007;56:921-7.
49. Saary J, Qureshi R, Palda V, et al. A systematic review of contact dermatitis treatment and prevention. J Am Acad Dermatol 2005;53:845.