Contact dermatitis

INTRODUCTION

Many advanced practice clinicians see a large number of patients with eczematous skin diseases. Although many patients may have atopic eczema, another very common cause of eczema is contact allergy. Allergic contact dermatitis has become a more frequently recognized entity in the United States. It affects 14.5 million Americans each year. It is estimated that allergic contact dermatitis to topical products occurs in 1% to 3% of the general population. However, this type of dermatitis may be overlooked when it is a contributing factor in atopic dermatitis.

Contact dermatitis refers to dermatoses that are related to an external agent. The two main types are irritant contact dermatitis, or ICD, and allergic contact dermatitis, or ACD. Less common are immune contact urticaria, nonimmune contact urticaria and protein contact.

ICD and ACD usually begin as eczematous processes characterized by itching, redness, edema, oozing and crusting. Occasionally they may be noneczematous with urticarial, granulomatous, acniform, lichen planus and hyperkeratotic lesions. Contact allergens may cause erythema multiforme and purpuric eruptions, and can be complicated by lymphangitis and edema. Acniform eruptions also can occur due to contactants. Pigmented contact dermatitis also may occur.

IRRITANT CONTACT DERMATITIS

Irritant contact dermatitis is by far the most common. It occurs when the skin is in contact with a substance that is caustic to the skin. Many substances act as irritants that produce a nonspecific inflammatory reaction of the skin. No previous exposure is necessary. It is not an immunologic reaction, usually occurs within 48 hours and resolves within 96 hours. The only variation in the severity of the dermatitis is related to the condition of the skin at the time of exposure to a given concentration of the irritant. ICD usually is confined to areas of contact exposure with well-demarcated erythematous and sometimes follicular reaction with pain and burning, as opposed to the usual itch of allergic dermatitis that often expands beyond the area of contact, and is marked by induration and/or papulovesicular eruptions. ICD at times precedes or can be a concomitant diagnosis with ACD.

Irritant contact dermatitis often is produced by alkalis, acids, metal salts, fiberglass, dust, capsaicin, tear gas, chlorinated compounds, hydrocarbons and solvents. The following is a short discussion on some of the most common offending agents.

Alkalis

Alkalis include such items as soaps, detergents, bleaches, ammonia and various household cleaners. Alkalis penetrate and destroy deeply because they dissolve keratin. Strong solutions are corrosive, and immediate application of a weak acid — such as vinegar or lemon juice — will lessen their effects. These agents are prominently the cause of hand eczema.

Acids

Powerful acids are corrosive, and weaker acids are astringent. Hydrochloric acid produces burns that are less deep and less likely to form blisters than sulfuric and nitric acids. Hydrochloric acid is encountered in the making of explosives. Sulfuric acid is more likely to form blisters than sulfuric and nitric acids. Hydrofluoric acid is used in rust remover, the semiconductor industry and in germicides, dyes, plastics and glass etching. Nitric acid is used in the making of explosives. Sulfuric acid is more widely used than any other acid and is handled by brass, copper, bronze and iron makers. Treatment of acid burns consists of immediate rinsing with copious amounts of water and alkalinization with sodium bicarbonate or soap solutions.

Metals

As a group, metals are the most common contact allergens. Nickel is the most common cause of metal allergy, followed by gold, chrome, cobalt and mercury. Allergenic sensitivity to a metal usually is highly specific, but cross-sensitivity with other metals is common.

The pure metals usually do not cause hypersensitivity, with the exception of nickel. It is only when they are incorporated into salts that they cause reactions. Most objects containing metal or metal salts are combinations of several metals.
Nickel produces more cases of allergic contact dermatitis than all other metals combined. Erythematous and eczematous eruptions, often with lichenification, appear beneath earrings, bracelets, rings, wristwatches, clasps and blue-jean buttons. Nickel dermatitis frequently is seen on the earlobes. Piercing the earlobes with nickel-plated instruments or wearing nickel-plated jewelry induces nickel sensitivity. Often the clasp in gold jewelry contain nickel. Sweat containing sodium chloride may combine with nickel to form nickel chloride. Therefore, perspiring profusely will affect the degree of nickel dermatitis.

In addition to direct skin contact with metals, dermatitis from metal allergy may manifest itself due to ingestion. The normal daily intake of nickel is 0.3 mg to 0.6 mg. About 1% to 10% of nickel in food is absorbed in the gastrointestinal tract, and the remainder is excreted. The nickel content is determined by the components of the soil in which it is grown, fungicides used on it and the equipment used in handling the food. Nickel in food varies between regions. Foods routinely high in nickel are legumes, nuts, grains, potatoes, chocolate and fish. Refer to Table 1 for additional foods high in nickel. Strict avoidance of foods high in nickel for three to four weeks will rid the body of continued stimulation that could be causing a rash. If the patient’s skin improves with diet, recommend that they add back one food per week to see if the rash returns or flares.

Dermatitis from metal also may manifest itself due to implantation of metals. Dental metal allergy, orthopedic implants and endovascular stents may lead to skin changes.

**ALLERGIC CONTACT DERMATITIS**

The second most common form of contact dermatitis is ACD. This is characterized by a complex immunologic event. It presents as a delayed type IV hypersensitivity reaction. Generally there is a 48- to 96-hour latency period between contact with the substance and reactivity in the skin, resulting in an eczematous reaction at the site of contact. It usually takes weeks to resolve. It occurs in two main stages: The first is sensitization, and the second is elicitation.

Sensitization involves the entry of allergens into the epidermis where they bond to keratinocytes and are recognized by the antigen-presenting cells. These cells process the antigens and present them to naive T cells, resulting in memory T-cell clonal expansion. Patients with atopic dermatitis have an impaired skin barrier, and this can facilitate the entrance of allergens into the skin. Contact allergens are small molecule substances of less than 500 daltons and because of their small size, they penetrate the skin barrier. Yet in order to induce and elicit contact allergy, an antigen needs to have a molecular weight of at least 5,000 daltons.

The second phase of ACD is elicitation, which occurs after repeated exposure to an environmental allergen. When recognition of the allergen occurs, there is cloning of memory T cells and release of inflammatory cytokines. This results in the appearance of dermatitis at the site of contact with the allergen and potentially at prior sites of reactivity.

An important clinical clue to the diagnosis of ACD is that it may take several days after contact with the instigating substance to develop the hypersensitivity response. Because of this, the patient may not be able to identify an association with a source. Furthermore, the allergen may be something that the patient has been in contact with or used repeatedly for years without a problem. This often leads the patient to doubt the association. It is important to explain the concept of threshold to the patient. Threshold is the point at which the skin’s ability to tolerate the chemical has surpassed. Threshold is reached through repeated exposures (elicitation) in a sensitized individual, and thus the dermatitis reaction occurs.

**COMMON ALLERGENS**

Table 2 outlines the top 10 offending allergens and prevention recommendations. The American Academy of Dermatology also recently announced the 2012 contact dermatitis allergen of the year acrylates. Examples include acrylic dentures, printing plates, orthopedic prostheses, artificial nails and glues. Other common allergens contained in cosmetics and toiletries are outlined below in greater detail.

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**Table 1**

**Foods high in nickel**

<table>
<thead>
<tr>
<th>CATEGORY</th>
<th>TYPE OF FOODS HIGH IN NICKEL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Meat/fish</td>
<td>Herring, mackerel, shellfish (e.g., shrimp and mussels) and tuna</td>
</tr>
<tr>
<td>Vegetables</td>
<td>Beans (i.e., green, brown and white), carrot, kale, leeks, lettuce, onion, peas, spinach, sprouts and tomato</td>
</tr>
<tr>
<td>Grains</td>
<td>Buckwheat, millet, muesli, multigrain breads, oatmeal, rye bran, sesame and sunflower seeds, and wheat bran</td>
</tr>
<tr>
<td>Fruits and berries</td>
<td>Apples, dates, figs, pineapple, prunes and raspberries</td>
</tr>
<tr>
<td>Drinks</td>
<td>Chocolate milk and cocoa drinks</td>
</tr>
<tr>
<td>Nuts</td>
<td>Almonds, hazelnuts and peanuts</td>
</tr>
<tr>
<td>Other</td>
<td>Chocolate and soy</td>
</tr>
</tbody>
</table>

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**Table 2**

**Top 10 offending allergens and steps for prevention**

<table>
<thead>
<tr>
<th>RANK</th>
<th>ALLERGEN</th>
<th>DESCRIPTION</th>
<th>PRESENTATION</th>
<th>PREVENTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Nickel</td>
<td>Found with chromium and cobalt deposits</td>
<td>Local, ectopic, generalized; vesicular hand dermatitis (i.e., pompholyx)</td>
<td>Avoidance of jewelry, jean snaps, buttons, razors, utensils, keys, coins, face paints, cell phones and such foods as chocolate, spinach, oatmeal, nuts and soy</td>
</tr>
<tr>
<td>2</td>
<td>Cobalt chloride</td>
<td>Metal or alloy with nickel, silver, lead, copper and iron</td>
<td>Local, vesicular hand dermatitis (i.e., pompholyx)</td>
<td>Avoidance of paints, dyes, metal-plated objects, bracelets, amalgams and vitamin B12</td>
</tr>
<tr>
<td>3</td>
<td>Thimerosal</td>
<td>Mercuric derivative of thiosalicylic acid (preservative)</td>
<td>Local</td>
<td>Avoidance of cosmetics, ophthalmic medications and inactivated influenza vaccine</td>
</tr>
<tr>
<td>4</td>
<td>Gold</td>
<td>Metal</td>
<td>Local, ectopic (i.e., facial contact dermatitis on eyelid and stomatitis); black dermatographism</td>
<td>Avoidance of jewelry, dentistry and cosmetics with titanium dioxide</td>
</tr>
<tr>
<td>5</td>
<td>Fragrance mix</td>
<td>Combination of common fragrance chemicals</td>
<td>Local, generalized, systemic</td>
<td>Avoidance of fragrances and flavors in cosmetics, lotions, shampoos, candles and perfumes; use fragrance-free products</td>
</tr>
<tr>
<td>6</td>
<td>Neomycin</td>
<td>Topical antibiotic</td>
<td>Local, anaphylaxis</td>
<td>Avoidance of OTC antibiotic ointments and creams; can cross-react with gentamycin, kanamycin and tobramycin</td>
</tr>
<tr>
<td>7</td>
<td>Balsam of Peru</td>
<td>Natural-free resin containing fragrance or flavorant (&gt;400 chemicals)</td>
<td>Local, systemic</td>
<td>Avoidance of fragrances and flavored cosmetics, lotions, cleansers, perfumes and mouthwashes; follow low balsam of Peru diet avoiding citrus, tomato, chocolate, vanilla extract, soda, cloves and cinnamon</td>
</tr>
<tr>
<td>8</td>
<td>Colophony</td>
<td>Resin from pine and spruce trees</td>
<td>Local, systemic</td>
<td>Avoidance of adhesives, eyebrow wax, mascaras, eye shadows, nail polish and diapers</td>
</tr>
<tr>
<td>9</td>
<td>Formaldehyde</td>
<td>Preservative, antimicrobial (e.g., formaldehyde preservative quaternium-15 or nonformaldehyde preservative paraben)</td>
<td>Local, generalized, systemic</td>
<td>Avoidance of personal hygiene products containing formaldehyde-releasing preservatives, wrinkle-resistant, rayon and permanent press clothing, and building materials</td>
</tr>
<tr>
<td>10</td>
<td>Lanolin</td>
<td>Emollient derived from sheep sebum</td>
<td>Local, generalized when associated with lotions</td>
<td>Avoidance of personal hygiene products, medications and industrial products</td>
</tr>
</tbody>
</table>
Cosmetics and toiletries

All topical products require preservation to avoid spoilage and rancidity caused by microbial activity and decomposition of the ingredients. The preservatives are the most common allergens in topical products, closely followed by fragrances. Hair dyes are detergent-based and contain anionic and amphoteric detergents, which are occasional sensitizers. There are five different types of hair dyes that can cause allergy, but the majority is caused by p-phenylenediamine, or PPDA. The severity of reactions ranges from mild erythema at the hairline or ears to swelling of the eyelids and face, and acute vesicular eruption in the scalp. Individuals sensitive to PPDA should be warned about possible cross-reactions with local anesthetics (e.g., procaine and benzocaine), sulfonamides and para-aminobenzoic, or PABA, acid sunscreens. Hair bleaches contain peroxides, persulfates and ammonia, which may act as irritants. Hair sprays contain shellac, gum Arabic, sunscreens and synthetic resins, which are sensitizers.4,5

Formaldehydes are a very small chemical and thus penetrates the stratum corneum easily. It is a very common allergen and is found in a variety of products most frequently in skin, cosmetic and hair products. Often it is not listed on the ingredient label. It’s also found in paper, smoke (i.e., component of smoke from burning wood, coal, charcoal, cigarettes and cigars), cleaning products and disinfectants. Quaternium-15 is the most widely used topical preservative. Propylparaben and methylparaben esters are popular preservatives in many cosmetics and topical products to prevent bacterial and fungal contamination. Other preservatives may include benzalkonium chloride, benzyal alcohol, thimerosal, triclosan, sorbates, sodium laurel sulfate, propylene glycol and lanolin. Almost all cosmetic preparations and skin care products contain fragrance. Even those labeled unscented often contain a masking fragrance that may be a sensitizer. The most common individual allergens identified are cinnamon alcohol, oak moss, cinnamon aldehyde, hydroxyl citronellal, musk ambrette, isoeugenol, geraniol, coumarin and eugenol. Frequently, combinations of different fragrances, such as balsam of Peru, will cause allergic dermatitis.4,5

Baby products

Diaper area is a frequent site of irritant contact dermatitis. Baby products often are fragranced. Baby oil, talc and cornstarch have simple compositions with little sensitizing potential, except for the fragrances. Baby lotions or creams may contain fragrance, preservatives, lanolin or propylene glycol that are sensitizers.4,5

Bath preparations

Adverse reactions to bubble bath are due to the detergent content of the product. Cocamidopropyl betaine is the surfactant that is allergenic. The detergent content of these products may aggravate dryness and inflammation of the skin, especially in atopic individuals.4,5

Hair preparations

Permanent waving solutions contain agents that can cause irritant reactions ranging from erythema to bullous dermatitis. Hair breakage and loss may result when permanent waves are improperly used. Currently, the majority of salon perms use glyceryl thioglycolate, or GTG. GTG has become a major allergen in hairdressers and less so in their clients. Chemical straighteners can cause hair breakage and chemical burns. Shampoos are detergent-based and contain anionic and soaps and detergents. Reactions to aftershaves causing contact dermatitis usually are due to fragrance. Such anti-aging products as retinoids and alpha hydroxy acids are agents that frequently cause irritant reactions. Irritant dermatitis from the keratolytic action of depilatories on the skin is common. Epilating waxes contain beeswax, rosin, and fragrance, which are potential sensitizers. However, these epilating cosmetics usually cause mechanical irritation. Dentifrices and mouthwashes contain sensitizers, such as the essential oils used as flavoring agents, preservatives, formalin and antiseptics.4,5

Dermatitis resulting from plants

A large number of plants — including trees, flowers, grasses, vegetables, fruits and weeds — are potential causes of dermatitis. The best-known plant allergens are the Toxicodendrons, which include poison ivy, poison oak and poison sumac — and all contain identical allergens. Cross-reactions occur between these plant allergens and Japanese lacquer, cashew nut trees, mango and the Indian marking nut tree. Sesquiterpene lactones are another plant allergen found in ragweed, sagebrush, burdock, chamomile and chrysanthemum species, including tansy, feverfew and pyrethrum.

Plant dermatitis may be classified as allergic sensitization, mechanical irritation, chemical irritation, phytophotodermatitis and pseudopyodermatitis. In some plants, the sensitizers may be in the leaves and stem, and others are found in the flowers, pollens and roots. For example, the water-soluble protein fraction of ragweed causes respiratory allergy, and the oleoresin found in the pollen, stem and leaf causes dermatitis.

Eruptions vary considerably in appearance but usually are vesicular and edematous. After previous exposure and sensitization to the active substance in the plant, the typical dermatitis results from re-exposure. The onset is usually a few hours or days after contact.4,5

Toxicodendron plants

Poison ivy and poison oak are the principal causes of Toxicodendron dermatitis in the United States. Poison oak (i.e., T. Toxicodendron, T. diversiloba) is more prominent on the West Coast, but poison ivy (T. radicans) occurs throughout the United States and is most abundant along the coast of New Jersey, New York, through the Carolinas and along the coast of New England. A form of poison ivy is found in Arkansas, Louisiana and Mississippi. Another subspecies is found in Oklahoma. A certain species is found in Mexico, Arizona and Baja California. Poison sumac, or poison dogwood (i.e., T.vernix), is only found in wooded, swampy areas. Cross sensitivity between the Toxicodendron plants exist, and the antigen of all is the same. Dark-skinned individuals are less susceptible than others to Toxicodendron dermatitis.4,5

The eruption produced by poison ivy is an allergic eczematous contact dermatitis charac-
The term “urushiol” often is used for the oleoresins of the plants. An individual can acquire ivy dermatitis from oleoresin-contaminated animals, clothing, tools, golf clubs, fishing rods and baseball bats. Fomites contaminated with the oleoresin in a dry atmosphere may remain antigenic for a long time, whereas a warm, moist climate favors loss of potency.4,8

After first exposure to the oleoresin, sensitization and dermatitis occur after seven to 10 days. After a previously sensitized person contacts the oleoresin, the eruption appears within two days, and delay of onset rarely exceeds 10 days. After exposure, it takes just minutes for urushiol to penetrate the outer layer of skin and bond with cell membranes. Sixty minutes after exposure, urushiol is completely bonded to the skin and can no longer be washed off with regular soap and water.4

Dermatitis usually begins with itching and redness; then streaks of erythema or papules in linear arrangement soon appear. In severe cases, extensive diffuse redness and swelling can occur, along with large blisters, open weeping lesions and disabling itching or pain. If there is no itching, the dermatitis is probably not caused by urushiol. The severity of the dermatitis depends on the person’s level of sensitization, the skin thickness at the site of exposure and the amount of urushiol that binds with the skin.4,5

Urushiol can be spread by the hands during the post-exposure phase before it’s been absorbed into the skin. Cutaneous contact with urushiol can lead to auto-eczematization (aka “id reaction”) with dozens of red papules and vesicles on the trunk and extremities. This is referred to as a systemic reaction because the sensitized white blood cells are thought to travel to other areas of skin, causing an itchy rash in places where there was no skin contact with urushiol. Individuals should be informed that the rash is not contagious, and the fluid in the blisters will not spread the rash.4,8

Prevention

The best prophylaxis for any type of allergic dermatitis is complete avoidance of the allergen. All individuals exposed to these plants should thoroughly wash the entire body with soap and water — strong soap not needed. Complete change of clothing is advisable, and contaminating clothing should be washed. The poison ivy antigen enters the skin so rapidly that the oil must be totally removed within 10 minutes of exposure. When exposure is unavoidable, individuals should wear long pants, long sleeves and gloves. An OTC skin barrier cream containing bentoquatam can help prevent urushiol from penetrating the skin. If there is a need to handle plants, vinyl gloves are resistant to urushiol.4,8

The oleoresin component of poison ivy contains the pentadecylcatechols that produce dermatitis. Urushiol retains its antigenic potential in the dry state indefinitely. The reaction of urushiol with the sensitized skin is almost immediate; a complex protein is formed. No amount of washing will remove the already reacted complex of catechol protein. After the dermatitis appears, washing will have no effect. Washing may remove excess un-united urushiol from dry areas of the skin. Urushiol dries quickly on clothing, shoes and tools, retaining its potency for months or even years on such fomites. Cleaning fluid solvents can effectively remove urushiol from fomites, and ordinary laundering is effective for clothing.4,8

Treatment

Cool compresses may help to reduce itching, redness and blisters. Burow’s solution may be used as a drying agent. Bathing in warm water with colloidal oatmeal or cornstarch is soothing and antipruritic. Calamine lotion can dry moist skin and cool the skin.

Once a reaction has begun, the goal of topical therapy is to decrease itching, redness and heat. The use of OTC topical products containing an-
tihistamines, anesthetic with benzocaine, neomycin or bacitracin should be avoided because of their potential to cause their own allergic contact dermatitis when absorbed through large breaks in the skin. Topical antihistamines do not work to reduce itching. An OTC product called Zanfel may help to reduce redness and blistering. Potent topical corticosteroids can reduce itching when used before presentation of blisters. Oral antihistamines can provide some relief. Oral and intramuscular steroids are effective with significant skin surface exposure or marked swelling, and those with a history of severe dermatitis.4

Flowers and houseplants
Many varieties of wild and cultivated flowers may cause dermatitis. Sensitivity occurring in gardeners and florists is confined to the hands, forearms, face and neck. Usually episodic acute vesicular reactions occur. Chrysanthemum, asters and daisies all belong to the same family of plants. Most of these flowers can produce dermatitis. Philodendron is a plant widely used for interior and patio decorations. Contact dermatitis to the hands and arms can occur when the patient washes, oils or plucks the leaves.

Other plants that may cause dermatitis include Algerian ivy, English ivy, oleander, castor bean, daffodil/narcissus, Chinese rice paper plant, dendropanax, bougainvillea, hydrangea, dahlia, narcissus and hyacinths, marigold and chrysanthemum. Various plant insecticides, including arsenical and malathion sprays, can produce contact dermatitis.4

Vegetables
The essential oils of the edible umbellifers — carrots, parsnips, squash, asparagus and celery — may cause allergic eczematous dermatitis in sensitized individuals. Pine, terpineol and cineole are the sensitizers. Onions and garlic may cause a contact dermatitis that can cross react with tulips and hyacinths.5

Trees
In the United States, the native woods that are cutaneous sensitizers include acacia, alder, ash, beech, birch, chestnut, cedar, elm, maple, mesquite, oak, pine, poplar, prune and spruce. The major components of wood, such as cellulose and lignin, are not sensitizers; but the minor components — such as resins, terpenes, oils, phenols, formic acid and nitrogen-containing substances — may cause allergic contact dermatitis. Freshly cut woods are more apt to cause dermatitis than are older woods.4

Pollens and seeds
The pollens in ragweed can cause respiratory symptoms from the protein fractions or contact dermatitis from the oil-soluble portion. Ragweed oil dermatitis is seasonal mainly during spring and fall. Contact with the plant or wind-blown fragments of dried plants produces the dermatitis. The oil causes swelling and redness of the eyelids and face, and a red blotty eruption on the forearms resembling atopic dermatitis.4

Spices
In the United States, the five spices that most commonly produce allergic dermatitis are capiscum, cinnamon, cloves, nutmeg and vanilla. All these spices can produce flares at the healed sites of allergic contact dermatitis when they are ingested or inhaled. Spices also may produce urticaria when ingested. Spices are used in perfumes, cosmetics and topical medications. All these spices contain essential oils that can irritate the skin. Mustard and capiscum are spices that can irritate the mucous membranes of the eyes and respiratory passages.4

Foods
Additional foods capable of producing contact dermatitis include garlic, lettuce, kiwi and mushrooms. The oleoresin of the mango skin can cause an allergenic chelitis to individuals sensitized to poison ivy. Mango and poison ivy/oak are commonly thought to cross-react. Pineapple juice contains bromelain, which can cause a primary irritant dermatitis.5

Regional contact dermatitis
Allergic contact dermatitis occurs in the area of contact with an allergen. However, there are instances when the location of the dermatitis is not directly related to the site of contact exposure with the allergen. This is called ectopic ACD and refers to the transfer of allergen into the area in which it would not usually be found. Outlined below are special considerations and potential offending agents by body area or region

Eyelid
The eyelid skin is one of the most sensitive areas. This is because of the thinness of the eyelid (0.55 mm compared to 2.0 mm thickness of the face). Dermatitis can be caused by rubbing the eyelid with the hands and fingers, which are exposed to many substances. Any substance used on the scalp, face or hands can produce allergic eczematous contact dermatitis of the eyelids. Airborne pollen and dust can affect the eyelids. Contamination of the fingers with small amounts of allergen can result in transfer of material to the eyelids to produce dermatitis, and no visible signs can be found elsewhere on the body.

Household sprays, insecticides and animal hairs also can produce eyelid dermatitis. Poison ivy, poison oak and poison sumac may produce marked swelling of the eyelids with minimum dermatitis of the face. Nickel-sensitive individuals may acquire eyelid dermatitis from nickel-plated eyelash curlers and tweezers. If this is determined to be the cause, advise patients to replace with stainless-steel type.4

Hair dyes and bleaching agents containing ammonium persulfate and perfumed hair sprays, hair setting lotions and shampoos containing formaldehyde, may affect the eyelids without producing scalp or forehead dermatitis. Sometimes eyelid dermatitis is the only manifestation of contact dermatitis to ingredients in shampoo.4

Eyelid dermatitis due to cosmetics
Contact dermatitis of the lids and periocular area is caused by cosmetics applied to the hair, face or fingernails more often than by cosmetics applied directly to the eye area. This is especially true for hair dye and nail polish. Allergic and irritant dermatitis reactions caused by face creams, makeup and blush may be also be limited to the eyelids.

Preservatives in eye cosmetics also are a potential cause. Parabens are common to all eye-area products. Parahydroxybenzoic acid is frequently combined with an antimicrobial — such as phenyl mercuric acetate, imidazolidinyl urea or parterium-15 — to protect against yeasts, molds and pseudomonads.4

Two forms of contact dermatitis attributable to eye-area cosmetics are allergic contact and irritant contact dermatitis. The features of these two forms are not always distinguishable. The degree of inflammation and the interval may be the same between the initial exposure and the onset of the dermatitis. Plus, potential irritants in the eye-area cosmetics usually are weak and, repeated exposures often are required to induce a reaction. It is often difficult to identify the cause of contact dermatitis of the eyelid.

The following approach is recommended:4

1. Take a detailed history of exposure, which should include agents other than eye-area cosmetics that are known to cause localized contact dermatitis of the eyelids, introduction of a new product and renewal of a previously used product. Modification or revision of formulations without a change in packaging is common practice in the cosmetic industry. The method used to remove eye makeup needs to be considered. Use of eyelash curlers may lead to contact dermatitis from the rubber edges or nickel plating.

2. When the history does not reveal the cosmetic involved, the use test is often helpful in pinpointing the causative agent. The product is applied to the back of the ear or the antecubital fossa two or three times a day for four to five days to try to reproduce the dermatitis.

3. Patch test with the components of the products incriminated by the history and use test are carried out in an attempt to identify causative allergens.

For advanced practice clinicians practicing in the retail clinic setting, referral to a dermatology specialist for patch testing is recommended.

Irritant eyelid and conjunctival reactions
Stinging and burning of the eyes and lids on application are the most common complaints. These subjective symptoms usually are transitory and unaccompanied by objective signs of irritation. Some common causes are propylene glycol,
sunscreen and soap emulsifiers. Conjunctivitis may be caused by such physical irritants as mascara flakes, eye shadow dust, particles of eyeliner, eyelash extenders or chemical irritants, such as solvents, soap emulsifiers, preservatives and fragrances. Some preservatives in ophthalmic medications produce not only conjunctivitis, but also eyelid dermatitis. Benzalkonium chloride and thimerosal are the most common.4

Facial dermatitis

Allergens can be transferred to the face not only by direct exposure but also indirectly from airborne or hand–to–face exposure. The face also is the most common site of photodermatitis and Toxicodendron dermatitis (from plants). Facial tissues containing perfume, formaldehyde or benzalkonium chloride may produce dermatitis. Newsprint and carbon paper also can produce dermatitis. The following rubber articles have produced facial dermatitis: scuba diver face mask, bathing cap, rubber cosmetic sponges, rubber-edged eyelash curlers, balloons and children’s toys. Nickel-plated objects used on the hair, such as bobby pins and curlers, may produce facial dermatitis in nickel-sensitive individuals. Nickel-plated objects during surgery or dentistry that have contact with the face can produce dermatitis.

Pomades are hair straighteners that contain paraffin and petrolatum mixed with gums and perfumes. They are the least damaging of all hair products, but they often cause acneiform facial lesions localized to forehead and temples.4

Status cosmeticus due to stinging compounds in cosmetics

Status cosmeticus is nonspecific irritation from cosmetics in which every cosmetic or soap applied to the face produces itching, burning or stinging sensations. Patients who have status cosmeticus have an unremarkable clinical picture with mild erythema and slight edema of the eyelids compared with their subjective symptoms of discomfort. This condition is an irritant and not an allergic reaction. Underlying conditions — such as seborrheic dermatitis, rosacea or atopic dermatitis — may complicate the assessment.4

Perioral

Perioral dermatitis and cheilitis may be caused by flavoring agents in dentifrices and gum, as well as fragrances, shellac, medicaments and sunscreens in lipstick and lip balms.4

Scalp

The scalp is particularly resistant to contact dermatitis. Allergens applied to this area often produce dermatitis of the eyelids, ears, neck and hands. Permanent hair dyes use p-phenylene-diamine, or PPDA, a potent sensitizer that may cross-react with many chemicals. Sensitivity is manifested by itching, redness and puffiness of the upper eyelids, tops of the ears, temples and back of the neck without affecting the scalp. Hair bleaching products use peroxides, persulfates and ammonia, which act as primary irritants. These may produce a local urticarial or generalized histamine reaction. Permanent wave solutions that contain ammonium thioglycolate when applied improperly may produce severe irritant reactions, hair breakage and burns of the scalp. Hair sprays are sensitizers and allergic reactions occur infrequently.4

Cosmetic damage to the scalp in black patients

Most hair products for black people are used to change the hair from curly to straight. The most potent hair relaxers are those containing alkali sodium hydroxide. When used improperly, it can cause devastating damage to the scalp and hair. The most common problem is hair breakage either immediately or remotely four to six months after application.4

Ears

Dermatitis of the external ear, particularly the helix, may be due to hair sprays, shampoos and dyes. Earlobe dermatitis is a cardinal sign of nickel sensitivity to costume jewelry earrings. Otitis externa may be due to sensitizing medications, such as neomycin. Insertion of metallic objects — such as hairpins, pens and pencils — into the canal may produce dermatitis in nickel-sensitive individuals. The entire auricle may react in sensitized individuals to plastic helmets or bathing caps. Piercing of earlobes is a common precipitating cause of nickel sensitivity that can persist indefinitely. It is advisable to insert stainless-steel earrings for the first three weeks. Postauricular dermatitis may occur due to metal or plastic eyeglass frames, plastic hearing aids, headsets or perfume.4

Neck

The neck is a highly reactive site. Nickel-sensitive individuals may acquire dermatitis from necklaces, zippers and stethoscopes. Other causes — such as leotards, starched collars and cosmetics applied to the face and hair — can cause dermatitis.4

Chest

Medallions, crosses, zippers and nickel-plated buttons can produce dermatitis, as well as perfume dermatitis of the upper chest and breast area. In women, brass cause dermatitis from the material, elastic, metal snaps or underwear.4

Periaxillary, axillae and trunk region

Symmetrical dermatitis in these areas may be due to finishes or dyes in clothing, deodorants, antiperspirants or perfume dermatitis. Antiperspirants contain aluminum salts and zinc salts, which are primary irritants. Deodorants that contain cinnamic aldehyde can induce irritation.4

Hands

Hand dermatitis is experienced by 10% of women and 5% of men over periods of time. It is often impossible to distinguish between irritant and allergic contact dermatitis of the hands without performing patch testing. Patch testing is the only proof that a contactant is a dermatitis-producing allergen. It is valuable to refer patients with chronic hand dermatitis for patch testing.4

ICD due to a nonspecific reaction can occur in all individuals, provided the irritants are in contact with the skin for a sufficient length of time and sufficient high concentrations. This dermatitis usually is characterized by dryness, some fissuring and thickening of the skin. It differs from ACD in that vesicles are rare. Detergents and solvents are the main cause. Irritants damage the skin by direct cytotoxic action. Individuals who frequently wash their hands or are exposed to cleaners — like housewives, dental workers, janitors, housekeepers and food preparers — tend to develop this type of irritant contact dermatitis.

Several allergens have been found to play a prominent role in allergic contact dermatitis of the hands, including nickel, potassium dichromate, ethylenediamine, rubber, paraphenylenediamine and preservatives. Hand eczema, whether it begins as ICD or ACD, may get worse because of an allergy to preservatives or other agents in topical medicaments and skin care products. The formaldehyde releasers — including quaternium-15, imidazolidinyl urea and diazolidinyl urea — are the common causes of ACD. Food handlers may acquire contact dermatitis of the hands. Table 3 outlines foods with potential to cause various hand dermatitis symptoms.4

Allergic reactions to nickel-plated rings may occur in nickel-sensitive individuals. Gold and palladium occasionally cause allergy. Platinum and silver almost never cause ACD. The dermatitis under a ring usually is produced by soaps, detergents, waxes, polishes or cosmetic creams that accumulate under the ring and cause a primary irritant type of dermatitis. Rings should be removed when washing the hands and regularly cleaned to remove accumulated material. Hand eczema frequently begins under a ring. If dermatitis owing to rings is not properly treated, the
eruption may spread from the finger onto the hand and to other fingers. In addition, ACD and hand pompholyx (i.e., dyshidrotic eczema) may flare following ingestion of nickel in patients sensitized to this metal.

Excessive exposure to water, soap and detergents leads to changes in the skin. The amount of damage depends on traumatic and environmental influences, such as cold weather, low humidity, emotional stress, hyperhidrosis, exposure to other contactants (e.g., vegetables, fruits, meat, juices and other foods) and contact with cleansing products containing bleaches, abrasives, alkalies and solvents. Irritation by soaps and detergents is increased by the concentration of the detergent solution on the skin (e.g., cleansers trapped under rings during dishwashing). All laundry soaps contain builders used to build a product — including such additives as sodium carbonate, sodium phosphate, ash, borax and sodium silicate — which may irritate skin but not sensitize. Other additives to soaps may be allergens and include the following: lanolin, perfumes, resin, antiseptics, deodorants, vitamin E, antiperspirants, turpentine, dyes, surfactants and emulsifiers. Studies have shown that alcohol-based disinfectants cause less visible skin irritation and less skin barrier disruption; therefore, alternating detergent with alcohol-based disinfectant is less irritating than use of detergent alone.6

Nails

Nail enamels contain various chemicals that can cause allergic reaction. Often the dermatitis occurs at sites distant from the fingers — commonly the eyelids, around the mouth and chin, sides of neck and genitalia. Tosylamide formaldehyde resin, or TSFR, is responsible for almost all of the allergic reactions. Yellow discoloration of the nail plate commonly occurs in women who wear colored nail polish due to staining from various dyes. This discoloration will fade over time only if use of the nail polish is discontinued. Artificial nails can cause an irritant or allergic reaction to the liquid monomers, as well as the ethyl cyanoacrylate glue used to attach the prosthetic nail. Paronychia, onycholysis, onychia and dermatitis of the finger also may occur.7

Below the belt

The waistline may be the site of rubber dermatitis from the elastic in pants and undergarments. The metallic rivets in blue jeans may lead to dermatitis in nickel-sensitive patients. Nickel-sensitive individuals can develop periumbilical dermatitis from metallic buttons of blue jeans, belt buckles or piercings of the umbilicus. Infants develop an allergic dermatitis after the cord is tied due to antibiotics with neomycin or antiseptic with thimerosal. Coins and keys in pockets may cause thigh dermatitis.

The use of such sensitizers as benzocaine, neomycin and balsam of Peru should be considered in patients with pruritus ani. Patients with hemmorhoids may use anal hygiene products — such as tucks, rectal wipes or Balneol — to which the pruritus ani is sensitized. Perfumes or colored toilet paper may have dyes or essences that are sensitizers. Ingestion of spices, food with seeds, antibiotics or laxatives may cause leakage of oil or paraffin products that may cause itching. Foreign bodies inserted into the rectum and rectal intercourse may produce irritation made worse with use of anesthetics containing benzocaine.4

Vulvitis

Douches containing acids (e.g., alum, citric acid and lactic acid) or alkalis (e.g., sodium bicarbonate or sodium borate) may produce an irritant vulvitis. Feminine hygiene sprays consist of perfume, emollients, preservatives and antibiotic agents that may produce allergic reactions. Vaginal spermicides may produce allergic vulvitis. They contain foaming agents and emulsifiers that may be irritating. Rubber condoms may produce an acute dermatitis of the vulva and inner aspect of the thighs in rubber-sensitive women.

There are other potential causes of vulvitis, including perfume or perfumed toilet tissue, medicated soaps containing sensitizing antiseptics, such medications as benzocaine and bubble baths with prolonged immersion, particularly in children. Nickel-plated objects on sanitary napkins may cause vulvitis in nickel-sensitive persons. Such cosmetics as nail polish that is not yet dry and contact with vulvar or anal area can cause vulvitis and may cause an allergic reaction. Urine in incontinent patients may cause vulvitis from ammonia in urine. Also, wearing apparel that includes dyes and synthetic resins in underclothing, such close-fitting undergarments as pantyhose and girdles, and sanitary napkins may cause vulvitis.4

Penis

The glans penis and prepuce may acquire contact dermatitis from douches, contraceptive jellies, feminine hygiene sprays and other medicaments used by a sexual partner. Some men cleanse after intercourse with a strong detergent that may produce irritant dermatitis. Rubber condom dermatitis may be due to sensitivity to the rubber latex, lubricant or a preservative powder dusted onto the condom.4

Legs

The most common cause of allergic dermatitis of the legs is application of sensitizing medications to eczema and ulcers. The shins may be the site of rubber dermatitis from elastic stockings. Feet are sites for shoe dermatitis most often attributable to rubber sensitivity.4

SYSTEMIC CONTACT-TYPE DERMATITIS

Allergic eczematous contact dermatitis usually is produced by external exposures of the skin to an allergen. But occasionally in sensitized individuals, a systematically administered allergen may reach the skin through the circulatory system and produce contact-type dermatitis. Although the eczematous condition is produced by systemic administration, the first sensitizing exposure to the allergen may have been by topical application.4

The systemic reaction to ingested agents can be mediated by both a type 3 and a type 4 immunologic reaction. For example, it is not unusual for the nickel-sensitive patient who is provoked with nickel to develop a systemic eruption within hours and after 12 to 48 hours to exhibit a flare in hand dermatitis.4

Ingestion of an allergen by a person previously sensitized by contact may result in different types of systemic contact dermatitis. Pompholyx and dyshidrotic hand eczema result in recurring itchy eruptions with deeply seated vesicles localized to palms, volar aspects and sides of fingers. Exacerbations come at weekly to monthly intervals without obvious external reasons. Unusual routes of exposure — including ophthalmologic solution, oral ingestion and vapors — have been associated with erythema multiforme, such as purpura and vasculitis. Generalized maculopapular vesicular rash consists of symmetric eruption localized to the elbow flexures, axillae, eyelids, side of the neck and genital area. Baboon syndrome eruption that is prominent on buttocks and bright red can be due to such agents as penicillin, aminophylline, nickel, cimetidine, erythromycin, ampicillin, mercuric and amoxicillin. Eczematous systemic contact dermatitis may be accompanied by urticaria and rarely leads to anaphylaxis.4

Systemic contact dermatitis resulting from medicaments occurs when sensitization has taken place by topical application of the drug, and later the patient has a systemic reaction when the drug or immunologically related allergen is taken orally or parenterally. The opposite situation also can occur, when the patient has an exanthema due to an antibiotic and later develops a localized dermatitis when the drug is topically applied.4

High sensitization medicaments include such antibiotics as penicillin, streptomycin, neomycin, bacitracin, tetracyclines, sulfonamides, nystatin, aspirin, corticosteroids and phenothiazines. Other high-sensitization medicaments include preservatives (e.g., parabens); sorbic acid (e.g., red fruits, such as plums, prunes, strawberries, currants, cranberries and chestnuts); thimerosal in vaccines; flavoring agents (e.g., cinnamon oil); toothpaste; gum; tobacco; vermouth; cola; and balsam of Peru used in such spices as cloves, cinnamon, Jamaica pepper, vanilla, quinine, hydroxyzine, 5FU, dibucaine, fluorides and metals (e.g., nickel, cobalt, chrome, oral balsams and dental metals). Also of note: Garlic has been associated with fingertip dermatitis, but ingested garlic extract can cause pompholyx dermatitis.4

DIAGNOSIS

Allergic contact dermatitis and irritant contact dermatitis can be extremely difficult to distinguish from each other. This is where the patch test becomes a highly useful tool. Patients who have uncontrollable or worsening dermatitis and those who have failed standard
treatments are good candidates for patch testing. Advanced practice clinicians who practice in the retail clinic setting should refer patients who require patch testing to a dermatology specialist.4

The patch test is used to detect hypersensitivity to a substance that is in contact with the skin so that the allergen may be determined and corrective measures taken. So many allergens can cause ACD that it is impossible to test a person for all of them. In addition, a good history and observation of the pattern of the dermatitis, its localization on the body and its state of activity

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### Table 4: Treatment algorithm for ACD3

<table>
<thead>
<tr>
<th>TREATMENT</th>
<th>EXAMPLES</th>
<th>USE</th>
<th>RISKS</th>
<th>SIDE EFFECTS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>TOPICAL AGENTS</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Emollients or barrier creams</td>
<td>Aquaphor®, petrolatum, CeraVe cream, Cetaphil cream, Epiderm®, Ecolip®, Hylatopic®</td>
<td>Prophylaxis</td>
<td>Possible reactivity to ingredients</td>
<td></td>
</tr>
<tr>
<td>Topical corticosteroids: Strength depends on body location</td>
<td>Face, groin and axilla (Class VII-V): Examples include hydrocortisone 1% to 2.5%, desonide and hydrocortisone butyrate</td>
<td>Treatment</td>
<td>Limit duration to two to three weeks, location and surface area of use</td>
<td>Atrophy, hirsutism, acne, striiae, telangiectasia, changes in pigmentation, systemic and endocrine side effects, glaucoma and cataracts</td>
</tr>
<tr>
<td>Topical immune modulators (calcineurin inhibitors)</td>
<td>Tacrolimus (protopic) and pimecrolimus (elixide)</td>
<td>Management — good for use on thin skin areas, such as face and eyelids</td>
<td>Good safety profile for short- and long-term use</td>
<td>Mild to moderate irritation, burning, erythema and pruritus; potential for sensitization</td>
</tr>
</tbody>
</table>

**SYSTEMIC AGENTS AND OTHER TREATMENT OPTIONS**

- **Oral antihistamine**
  - Diphenhydramine, hydroxyzine, cetirizine, loratadine and fexofenadine
  - Management — pruritus
  - Potential for sensitization
  - Drowsiness
- **Systemic corticosteroids**
  - Prednisone, kenalog IM and betamethasone
  - Management — acute, severe or widespread reactions
  - Limit duration of use; taper dose after symptoms are controlled
  - Osteoporosis, hyperglycemia, hypertension, immunosuppression and hypothalamic-pituitary-adrenal axis suppression
- **Phototherapy**
  - Shortwave UV light (UVB)
  - Management — difficult chronic cases
  - Potential for cutaneous carcinoma and sunburn
- **Steroid-sparing systemic immune modulators**
  - Azathioprine, methotrexate, mycophenolate mofetil and cyclosporine
  - Management — severe recurrent cases
  - Caution if infection; may be associated with an increased risk of lymphoproliferative disorders; need monthly labs
- **Biologics**
  - Adalimumab and etanercept
  - Management — severe recurrent cases
  - Caution if infection; may cause increased risk of malignancy, including nonmelanoma skin cancer and hematologic malignancies

**PATIENT SCENARIO 2**

**Case history:** A 16-year-old female presents to the clinic with a three-week history of pruritic eruption. She has a long-standing history of eczema since the age of 2 years. The last episode was two years ago with the exception of intermittent earlobe rashes that started three years ago. Past treatments have included topical corticosteroids and moisturizers. She had recent exposure to keys, jean snaps, spinach and chocolate, and recently pierced ears, which all contain one common ingredient: nickel.

**Presentation:** Eruption refractory to treatment with hydrocortisone 1% cream, a pramoxine-containing lotion (Sarna) and an over-the-counter anti-itch cream containing diphenhydramine. Oral hydroxyzine 25 mg at bedtime helped to relieve pruritus and assist with sleeping, but had no effect on the eruption.

**Physical exam:** Areas involved include periumbilical, antecubital and popliteal fossa, posterior neck and bilateral earlobes. Symptoms appeared as lichenified, excoriated eczematous dermatitis. Patch testing confirmed nickel contact allergy.

**Differential diagnosis:** Atopic dermatitis, allergic contact dermatitis

**Treatment:**
1. Start skin care regimen with gentle skin cleanser and diffuse moisturizer application.
2. Discontinue topical hydrocortisone cream, topical pramoxine lotion and topical diphenhydramine cream.
3. Add mid- to high-potency topical corticosteroid cream twice daily to affected areas for a maximum of two weeks, followed by the application of a calcineurin inhibitor until clearance is achieved.
4. Continue oral hydroxyzine at bedtime.
5. Patient instructed about avoidance recommendations. Each nickel exposure is additive, thus making avoidance necessary in order to get beneath the threshold and into the rash-free zone.
6. Recommend dimethylglyoxime spot testing to evaluate metal products for presence of nickel.

**Commentary:** Allergic contact dermatitis may complicate atopic eczema. Nickel has been identified as the most common contact allergen among children and adolescents.13
are all helpful in determining the cause. Also, examining and reviewing the products used by patients and their habits can help determine the allergens. The patch test is confirmatory and diagnostic only within the context of the history and physical findings.2,4

The patch test is artificial and does not necessarily duplicate clinical exposure, in which sweating, maceration and multiple applications may play roles in producing dermatitis. The real life exposure of skin to allergen will have variable contact time for the transfer of allergen to skin, whereas the patch test is applied for a fixed, standardized time. The concentration of allergen in a real life exposure is seldom known, whereas the patch test exposure is defined and adjusted to minimize the chance for an irritant reaction.

The patch test consists of application to intact uninfamed skin, in a nonirritating concentration, of substances suspected to be causes of the contact dermatitis. Patch testing may be administered by the thin-layer rapid-use epicutaneous, or TRUE, test or by an individually prepared aluminum (e.g., Finn) chamber mounted on Scanpor tape.2,4

Test substances are applied to the upper back. The patches are removed after 48 hours and read. The patch sites need to be evaluated again after four or five days because positive reactions may not appear earlier. Some allergens may take up to seven days to show a reaction. Erythematous papules and vesicles with edema are indicative of allergy. The reading also helps to distinguish between allergic and irritant contact reactions, because ICD reactions are typically seen early and usually are in the healing resolution phase after 72 to 96 hours. ACD reactions, on the other hand, continue to worsen over the duration of the patch test procedure.2,4

**TREATMENT AND MANAGEMENT**

An individual may be exposed and sensitized to an allergen for days to years before actually developing an allergic contact dermatitis. Exposures can be additive, causing one's immune system to cross a threshold, at which time a cutaneous response is elicited. Just as repeated contact over time leads to an immune response, repeated avoidance of the exposure over time is required to induce remission. The goal of therapy is avoidance for both ACD and ICD, and the patient must be treated until the dermatitis subsides. Avoidance of specific allergens can be a tedious task for patients. There are programs available to aid patients, such as the Contact Allergen Management Program, a service offered through the American Contact Dermatitis Society.2,4

There are a large number of patients suffering from potentially debilitating skin dermatitis who could live dermatitis-free given the appropriate testing, counseling and allergen-free alternatives. In order for these patients to obtain optimal success, it is necessary for them to receive reinforced education regarding avoidance of the substances to which they are allergic.10,11 Avoidance of the relevant chemicals to which the patient has reacted on patch testing is the mainstay of treatment.12 There are instances when pharmacotherapy needs to be used, as an allergen may not be identified or avoidance may not be possible; and in this case, utilization of a treatment algorithm is advised (Table 4). Medical management of allergic reactions can involve topical corticosteroids, barrier devices, systemic antihistamines, calcineurin inhibitors, UVB, UVA and systemic immunosuppressants.3

**CONCLUSION**

There are millions of people who suffer from ACD and ICD. Advanced practice clinicians are on the front line encountering a large number of patients every year. Many of these patients will respond to standardized treatments, but there will be others that demonstrate recalcitrant dermatitis. Primary care providers play a large role in the identification and successful treatment of patients experiencing ACD and ICD. The intent of this lesson was to help familiarize advanced practice clinicians with various allergens and their significance, recognition of ACD and ICD, and when to refer patients for comprehensive patch testing.

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6 MilitelloG, Nijhawen RI, Matiz C and Jacob SE. Contact Dermatitis: From basics to allergodromes.
Successful completion of “Contact dermatitis” is accredited for 1.3 hours of continuing education credit, of which 0.3 hour is considered pharmacology credit. To obtain credit, answer the following questions and complete the evaluation online at DSNCollaborativeCare.com.

1. What typically presents as a delayed type IV hypersensitivity reaction where generally there is a 48- to 96-hour latency period between contact with the substance and reactivity in the skin resulting in an eczematous reaction at the site of contact?
   a. Irritant contact dermatitis
   b. Allergic contact dermatitis
   c. A and B
   d. None of the above

2. Which of the following is the most common cause of metal allergy?
   a. Chrome
   b. Gold
   c. Mercury
   d. Nickel

3. Which of the following are the main causes of irritant contact dermatitis on the hands?
   a. Detergents and solvents
   b. Nickel and potassium dichromate
   c. Rubber and preservatives
   d. All of the above

4. Patients who have uncontrollable or worsening dermatitis and those who have failed standard treatments are good candidates for patch testing.
   a. True
   b. False

5. Which of the following foods have the potential to cause immediate contact urticaria of the hands?
   a. Lobster
   b. Beef
   c. Carrots
   d. A and B
   e. All of the above

6. The mainstay of treatment for both irritant contact dermatitis and allergic contact dermatitis is:
   a. Avoidance of the relevant chemicals to which the patient has reacted on patch testing.
   b. Topical corticosteroids
   c. Systemic immunosuppressants
   d. B and C

7. Which of the following are the most common allergens in topical cosmetic products?
   a. Excipients
   b. Fragrances
   c. P-phenylenediamine
   d. Preservatives

8. Which of the following categories accounts for the largest number of cosmetic-related contact dermatitis?
   a. Deodorants
   b. Facial makeup
   c. Moisturizers
   d. Nail polish

9. The best-known plant allergens are the Toxicodendrons, which include which of the following?
   a. Poison ivy
   b. Poison oak
   c. Chrysanthemum
   d. A and B
   e. All of the above

10. In addition to direct skin contact with metals, dermatitis from nickel allergy may manifest itself due to ingestion of which of the following?
    a. Chocolate
    b. Peanuts
    c. Shellfish
    d. B and C
    e. All of the above