

Review

Contact dermatitis and patch testing for the allergist

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Key Messages

- Contact dermatitis should be suspected in patients presenting with acute, subacute, or chronic dermatitis characterized by pruritus, erythema, vesicles, and scale.
- The gold standard for diagnosing allergic contact dermatitis (ACD) is the patch test (PT). Immunosuppressant medications such as systemic corticosteroids, cyclosporine, and/or mycophenolate or topical corticosteroids, topical calcineurin inhibitors, or radiation to the PT site may reduce allergic PT responses.
- Nickel and Balsam of Peru are the 2 most common causes of systemic contact dermatitis when ingested in food whereas ACD from cosmetics and personal care products are commonly due to fragrances and preservatives.
- Present relevance of a positive PT is the combination of definite, probable, and possible relevance and should be correlated with the patient's history and physical examination.
- Treatment of ACD includes patient education about their allergens, avoidance, and alternative products they can use.

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ABSTRACT

Objective: To review of contact dermatitis (CD) and its key allergens and provide updates and recommendations for the practicing allergist.

Data Sources: Through the use of various scientific search engines (eg, PubMed and MEDLINE), we reviewed literature on CD, patch tests (PTs), key allergens, occupational dermatitis, and treatment.

Study Selections: Studies on CD, important allergens, and PTs were considered.

Results: Contact-induced dermatitis may be due to allergic CD, irritant CD, systemic CD, contact urticaria, and protein CD. Key allergens include metals (nickel, gold), topical medicaments (topical corticosteroids), and cosmetics and personal care products (fragrances and preservatives such as methyl- and methylchloroisothiazolinone). Present relevance of a positive PT result is the combination of definite, probable, and possible relevance and should be correlated with the patient's history and physical examination. Treatment of allergic CD includes identification of relevant allergens, patient education, avoidance, and provision of alternative products the patient can use.

Conclusion: CD is a common inflammatory skin disease and should be suspected in patients presenting with acute, subacute, or chronic dermatitis. The gold standard for diagnosing allergic CD is a PT. This article provides practical recommendations for the diagnosis and management of CD commonly seen by the allergist in their practice.

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Introduction

Contact dermatitis (CD) is an inflammatory skin disease characterized by pruritus, erythema, vesicles, and scale. It can present as acute, subacute, or chronic dermatitis. A total of 80% of CD cases are irritant contact dermatitis (ICD), and 20% of CD cases are allergic contact dermatitis (ACD). ICD is defined as a localized, nonimmunologically driven, inflammatory reaction. ACD is a type 4 mediated hypersensitivity to a specific allergen that also results in a subsequent inflammatory reaction.¹

ACD or ICD should be suspected in patients with chronic eczematous or noneczematous dermatitis. ACD or ICD usually begins as an eczematous process characterized by itching, redness, edema, and a tendency toward oozing and crusting; however, it may be noneczematous with urticarial, granulomatous, acneiform, lichen planus–like or dry, hyperkeratotic lesions. Erythema multiforme, purpuric eruptions, and pigmented contact dermatitis may result from exposure to contact allergens.² It is important to review the home and workplace for sources of contact allergens in addition to personal products used by a patient. ACD should be suspected and evaluated in the patient with generalized and anatomically localized skin eruptions (such as the hands, face, eyelids), which may indicate contact with substances in the environment.³

CD is the most common form of occupational skin disorder, with estimated annual costs exceeding \$1 billion.⁴ Occupational CD has a significant effect on worker health and quality of life, workplace function, and health service use.⁵ Occupational dermatoses are based on the unique exposure and environment the patient is exposed to. Male and female rates vary based on profession; however, as a whole, men tend to have an age-related increasing rate of occupational CD, whereas women see a peak between the ages of 16 and 29 years. Occupational CD is seen at significantly higher rates in individuals with a manufacturing or mining job, with the most common cause arising from soaps and cleaners. In a UK report, the most common cause of ACD in an occupational setting was found to be rubber (23.4%), then nickel (18.2%), epoxy and other resins (15.6%), fragrances and cosmetics (8.0%), and lastly preservatives (7.3%).⁶

Pathogenesis and Pathology

ACD is a type IV mediated hypersensitivity reaction that occurs in response to a specific allergen. These allergens cause an activation of T_H1 cells in a previously sensitized individual. The T_H1 release of associated inflammatory cytokines includes interleukin (IL) 1 α , IL-1 β , IL-8, IL-13, tumor necrosis factor α , and granulocyte-macrophage colony-stimulating factor. Dermal dendritic cells are critical in the process of activating naive T cells in the lymph node to become antigen-specific effector cells during sensitization.¹ Several studies have found that antigen application on tape-stripped murine skin induced T_H2 polarization via increased expression of IL-4 and IL-13, increased levels of serum IgE and IgG1, and reduced expression of T_H1-related cytokines and serum antibodies.⁷

ICD has a different process regarding cytokine stimulation, which begins with the penetration of the allergen through the permeability barrier via disruption of keratinocytes. This results in T-cell activation and subsequent cytokine release, which also includes intracellular adhesion molecule 1, an important feature of ICD.⁷

Histologic analysis for ACD and ICD may be helpful in the acute phase of the condition because the late phase can be nondiagnostic. Acute stages of ACD exhibit marked spongiosis, with occasional intraepidermal vesicles. Mixed dermal inflammatory infiltrate can be seen with a variable number of eosinophils. ICD varies from ACD in that necrotic keratinocytes can be seen in the epidermis without the marked spongiosis seen in ACD.¹

Contact-Induced Dermatitis

ACD

ACD is often a well-demarcated, pruritic, eczematous eruption, which may be acute (blistering, weeping, and/or edema) or chronic (lichenified or scaly plaques). It is typically localized to the area of skin that comes in contact with the allergen, but depending on the nature of the causative allergen, patchy or diffuse distributions may be seen. Transferred allergens spread initially from the hands to distant sites, including the head and feet. This subset of ACD is referred to as ectopic CD.

Systemic Contact Dermatitis

Although most cases of ACD are from direct exposure and a localized response, other routes should be considered. Inhalation, ingestion, or infusion can result in a widespread, systemic dermatitis in patients contact sensitized to the allergen. Systemic contact dermatitis (SCD) to a known contact sensitizer should be considered in a patient who presents with generalized dermatitis, intertriginous and flexural exanthema (Baboon syndrome), and/or a flare at previous cutaneous sites of exposure.³ It can also manifest as a rash at the site of a previous positive patch test (PT) result, vesicular hand dermatitis, pruritic papules on elbows and knees, erythroderma, and vasculitis-like lesions.⁸ Occasionally, it is accompanied by nausea, vomiting, diarrhea, fever, malaise, headaches, and arthralgia.⁹ This reaction is believed to be attributable to a delayed T-cell-mediated immune response. SCD is caused by allergens, including metals (mercury, nickel), medications and foods (Balsam of Peru), plants, and herbal products, and usually occurs within 1 to 2 days of exposure (Table 1). Once sensitization has occurred, only small amounts of the allergen are required to incite the dermatitis, which can make it difficult to avoid an allergen that is prevalent throughout the home or workplace. However, removal of the inciting agent may improve the dermatitis. Postinflammatory hyperpigmentation has not been reported in Baboon syndrome.^{9,10}

ICD

Most occupational dermatoses are a result of repeated weak chemical exposure, causing cumulative direct cell damage. Individual susceptibility differs widely; however, skin breakdown in the setting of hand eczema increases the risk substantially. Occupational CD caused by ICD most commonly involves the hands. The most frequent causes of ICD in the workplace include soaps (22.0%), wet work (19.8%), petroleum products (8.7%), solvents (8.0%), and cutting oils and coolants (7.8%).^{5,6}

Contact Urticaria

Contact urticaria is characterized by a pruritus with a wheal-and-flare–like response classically seen with urticaria. Plant- and animal-derived proteins have been seen as the most common causes of contact urticaria in the workplace.¹¹ Latex protein has become an important, widely recognized contact allergen, especially in health care workers. Natural latex contains polyisoprene (30%–40%) together with other plant chemicals, including proteins.¹ Bakers and agricultural workers have the highest and second-highest risk of developing contact urticaria, respectively. Flour, grains, food stuffs, and baking-associated enzymes are common causes of contact urticaria in bakers, whereas cow dander is recognized as the highest frequency occupational contact urticant in agricultural workers.¹¹ Systemic symptoms, including gastrointestinal and nasopharyngeal symptoms, can be seen in individuals with immunologically mediated contact urticaria. Certain foods have also been implicated

Table 1
Reported Causes of Systemic Contact Dermatitis (SCDm)^a

	Topical non-drug-induced SCD	Topical drug-induced SCD	Systemic drug-induced SCD
Foods	Aspartame Balsam of Peru Compositae plants (chamomile, <i>Echinacea</i>) Garlic Propylene glycol Propolis		
Metals	Chromate Cobalt Gold Mercury Nickel Zinc		Mercury Gold
Medications		Ampicillin Anesthetics (Benzocaine, Cinchocaine) Aminosalicylic acid Buprenorphine Diclofenac Hydroxyquinoline	Amoxicillin Cephalosporins Corticosteroids Erythromycin Estrogen Ethylenediamine Gentamycin Neomycin Nystatin

^aAdapted with permission from Dr Marcella Aquino and Dr Greg Rosner.

in occupational contact urticaria as hand dermatitis in food handlers (those working in catering industry) and oropharyngeal symptoms secondary to ingestion.¹¹ These foods include meats, fish, eggs, fruits, vegetables, and flour as well as associated enzymes, such as α -amylase.¹ After a detailed history and clinical examination, skin testing may be undertaken to confirm the diagnosis of contact urticaria.¹ The diagnosis of contact urticaria includes open testing or percutaneous testing. Open testing involves the application of a small amount of the presumed offending agent to normal skin on the volar aspect of the forearm. The site is observed for 20 to 60 minutes for the characteristic wheal-and-flare reaction.¹²

Protein CD

Protein contact dermatitis (PCD) is a dermatosis that presents as a chronic eczema with episodic acute exacerbations of itching and/or stinging a few minutes after contact with the offending allergen. PCD is considered to be a combination of immediate type I and delayed type IV allergic responses. The 4 principal groups responsible for PCD are (1) fruits, vegetables, spices, and plants; (2) animal proteins; (3) grains; and (4) enzymes (α -amylase). Most patients who have PCD are food handlers, such as butchers and slaughterhouse workers, bakers, and chefs.¹³

The results of PTs with the responsible allergen are usually negative, and the diagnosis can only be made by means of scratch or prick tests with the allergen. Sometimes, specific IgE antibodies can be detected in the blood.¹³

Differential Diagnosis

OCD can be an elusive diagnosis, requiring a thorough history and detailed history with clinical examination. Skin testing, including PTs, can be performed to further elucidate the offending allergens. Atopic dermatitis was associated with an increased incidence of CD, specifically, with increased frequency of reported reactions to cocamidopropyl betaine, wool alcohol and lanolin, tixocortol pivalate, and parthenolide.¹⁴ Underlying atopic dermatitis and other

dermatoses, including mycosis fungoides, should always be considered when evaluating an eczematous dermatitis.

PT Tips

Whom to PT

CD can negatively affect the quality of life and impose substantial costs to the health care system. The gold standard for diagnosis of ACD is the PT. Patients with acute, subacute, and chronic eczematous dermatitis, such as erythema, edema, weeping, crusting, scaling, hyperkeratosis, and lichenification, are likely to have the highest yield for PTs.¹⁵ Although empiric avoidance of suspected allergen may improve the dermatitis, identification of the specific allergen by PTs increases patients' options, increases adherence, and improves outcomes.

Whom Not to PT

Patients with acute generalized dermatitis or extensive eczema on the back need control of the underlying dermatitis before PTs. Ideally, patients should not be taking immunosuppressant medications, such as systemic corticosteroids, cyclosporine, and/or mycophenolate, before PTs. However, some studies have found that PTs in patients taking low doses of prednisone (<20 mg/da) and cyclosporine may still yield clinically relevant results.^{2,16} Topical corticosteroids (TCSs) and topical calcineurin inhibitors (TCIs) should not be applied on the test site for 5 to 7 days before PTs.^{17,18} Patients should not get a suntan or use a sunbed 2 to 4 weeks before the PT. Although there are no current recommendations on PT patients who are pregnant or breastfeeding, it is prudent not to PT at that time. Systemic antihistamines are generally not believed to interfere with the PT readings.

Test Site

The concentration of standard allergens has been determined for the skin of the back only. The area must be free of hair, moisturizers, and other oily substances before application of the patches.¹⁹

Loading the Chambers

Ideally, tests should be prepared at the time they are performed because early preparation of some allergens may result in false-negative results. The general rule is that acrylates, fragrances, and volatile allergens in aqueous vehicle should be loaded in the chambers and placed on the patient right away. However, some allergens on petrolatum base may be prepared 24 to 48 hours before application.

Reading of the PT Results

The patches are removed at approximately 48 hours after their application, and the initial reading is performed after approximately 30 minutes. A second reading should be performed between 3 and 7 days after application, with a 96-hour reading as likely the optimal time for a second reading. Certain allergens are associated with late peak reactions, and a late reading 7 to 10 days after PT application should be considered if there is a negative early reaction, when the clinical history strongly supports sensitization. These allergens include metals (nickel sulfate, gold sodium thiosulfate, palladium chloride, potassium dichromate, cobalt chloride), some antibiotics (neomycin), TCSs, and dyes (paraphenylenediamine). On the other hand, allergens reported to peak early include thiuram mix, carba mix, and balsam of Peru.

Some irritant reactions appear within the first 48 hours then disappear (decrescendo effect) by 96 hours. Allergic reactions tend to increase (crescendo effect). Thus, the third visit is important to help determine whether the reaction is an irritant or an allergic one.

What to Do with a Positive PT Result: Determining Relevance

A positive PT result may be clinically relevant, depending on current or past exposures. Careful history and physical examination must be correlated with the PT result to establish clinical relevance. Present relevance is the combination of definite, probable, and possible relevance. A definite relevance is whether the PT or use test result with the suspected material is positive (repeat open application test). For example, fragrance is in the patient's foundation, the dermatitis corresponds to the area of contact (face), and the dermatitis improves with avoidance or recurs with rechallenge. Relevance is probable if the antigen is present in known skin contactant and the clinical presentation is consistent with that exposure. For example, fragrance is found in the patient's environment and the dermatitis corresponds to the area of contact, but follow-up information on improvement with avoidance or rechallenge is not available. Relevance is possible if skin contact with materials known to contain the allergen was likely. Past relevance is considered if the PT result is positive but the exposure is not current but was in the past. For example, a patient has hand dermatitis and has a positive PT result to thimerosal. Sensitization likely occurred during childhood vaccination and the patient's products do not contain thimerosal. Relevance has been shown to vary with allergens. For example, cocamidopropyl betaine had 89.2% present relevance, whereas thimerosal has a present relevance in only 7.3% of cases.³

Selected Allergen Pearls

Metals

Nickel is the most common allergen, with 19% of PT clinic patients reacting to it. The relevance of these reactions has been estimated to be 50%. Nickel is widely used in jewelry, buckles, snaps, coins, tools, utensils, nickel-plated objects (telephones and laptops), and even clothing and food. An exposure to nickel that may be relevant in causing SCD is its high content in canned foods, cocoa,

chocolate, soy, legumes, oats, almonds, cashews, shellfish and seafood, herring, salmon, and mackerel.

Treatment of nickel allergy includes topical avoidance. Legislative change in the United States to decrease nickel content of products coming into contact with the skin has been advocated. Patients who do not respond to topical avoidance alone may benefit from a low-nickel diet. Low-nickel diet and topical avoidance are generally advised because risks are negligible. Chelation therapy with disulfiram or tetraethyl thiuram disulfide, which results in excretion of nickel from the body tissue via urine, bile, and sweat, has been reported.²⁰

Gold has been removed from the standard test allergen from the North American Contact Dermatitis Group Standard Series but is currently part of the standard T.R.U.E. Test series. Although a PT to gold has a high rate of positive reactions (8.7%),²¹ questions about relevance is a concern and so is the potential for persistent PT reactions.²² However, gold is still an important allergen to consider in the proper clinical context, such as when evaluating eyelid dermatitis or in patients with gold dental restorations.²¹

Medications

Topical medicaments are used frequently and for prolonged periods for chronic skin disease, such as atopic dermatitis and chronic stasis ulcers.^{23,24} PTs should be considered in patients with worsening of previously well-controlled dermatitis or poorly responding to current prolonged treatment. There is a higher rate of sensitivity in older individuals to the topical antibiotics (neomycin and bacitracin) and preservatives in topical medications and personal care products (methylidibromoglutaronitrile, quaternium-15, formaldehyde, diazolidinyl urea, and imidazolidinyl urea).²⁵

Corticosteroid ACD is rarely suspected likely because the history and morphologic features of the dermatitis may be modified by its anti-inflammatory action. Any patient with a long-standing nonhealing dermatitis (such as stasis dermatitis, atopic eczema, or chronic hand eczema) who have been treated with TCSs should be investigated for ACD to corticosteroids if there is poor response to treatment or deterioration of a previous dermatitis.²⁶ Reactions reported are varied from acute edema with erythema and vesicles, angioedema and oozing eczematous reaction,²⁷ erythema multiforme-like CD,²⁸ and generalized rashes.^{29,30}

Pratt et al³¹ recommended a screening series of TCSs to include tixocortol-21-pivalate 1% petroleum (group A), budesonide 0.1% petroleum (group B), desoximetasone 1% petroleum (group C), clobetasol-17-propionate 1% petroleum (group D1), and HC-17-B 1% petroleum (group D2).³¹ These steroids serve as markers within the cross-reacting groups (groups A-D2). The ACD can also manifest as reactions to corticosteroids in the patient's medications to asthma and rhinitis and even SCD to systemic corticosteroids.^{32,33}

There are also some issues to address about PTs for corticosteroids. A high PT concentration may result in a negative test result, especially on the earlier reading, because of the anti-inflammatory action of the corticosteroid itself, such as budesonide 0.10%.^{34,35} Another phenomenon is the edge effect, which is a positive test reaction just outside the PT site and a negative reaction inside the patch chamber area. For most allergens, the edge effect is interpreted as a negative test result. However, in TCSs, the test result will usually be positive on a later day (day 6 or 7), emphasizing the importance of late readings for TCSs (Fig 1). The allergic reaction may be suppressed at early readings and may become positive once the anti-inflammatory effect decreases. Thirty percent of the cases of contact allergy to corticosteroids would have been missed had late readings not been performed.^{36,37}

Drugs that have been reported to cause SCD from topical absorption include corticosteroids, nonsteroidal anti-inflammatory drugs

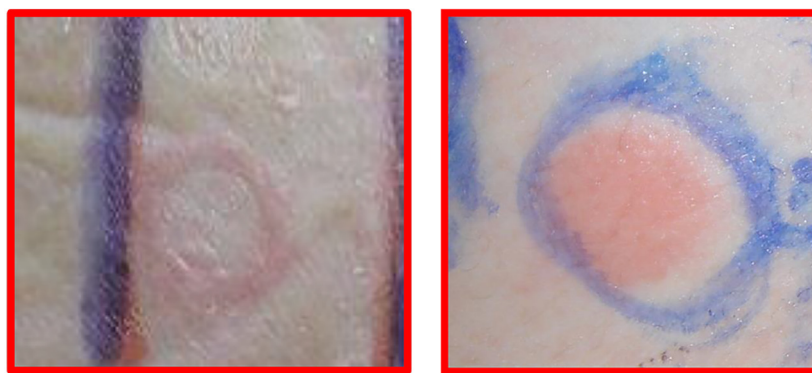


Figure 1. Budesonide 0.1% in petrolatum at 72 hours showing the edge effect and 7 days later showing a delayed positive test result.

(bufexamac, diclofenac), acetylsalicylic acid, anesthetics (cinchocaine), antibiotics (ampicillin, neomycin), and ethylenediamines³⁷ (Table 1).

Cosmetics

The term *cosmetic* includes personal care products, hair and nail care products, and sunscreens. The most common allergens in cosmetics are fragrances and preservatives. Fragrances account for up to 30% to 40% of allergic reactions to cosmetics.^{38,39} There are several hundred different chemicals that give fragrances a unique odor. The fragrance mix (FM) I in standard series PTs contain 8 perfume components⁴⁰ and can detect 70% to 80% of all perfume allergies. However, testing with additional markers, such as in FM II, may be necessary to increase the sensitivity. Thirty-two percent of patients who tested positive to FM II had negative reactions to FM I.⁴¹

Products labeled fragrance free may actually contain fragrance that is classified or acts as a preservative, emollient, fixative, or disinfectant. Unfortunately, there is no US federal standard definition that governs the use of the term *hypoallergenic* by manufacturers.⁴²

Myroxylon pereira (balsam of Peru) is a naturally occurring fragrance material that is the second most common allergen identified by the North American Contact Dermatitis Group.⁴³ In the proper clinical setting, PT results demonstrating sensitivity to balsam of Peru or FM can support the diagnosis of a SCD attributable to balsam-related foods or spices, such as tomatoes, citrus, cinnamon, and chocolate. A diet that avoids such foods may result in an improvement of the dermatitis.^{44,45}

Many PT reactions to FM I and II are often weak, perhaps irritant, and hard to reproduce. Thus, determining present clinical relevance for fragrance PT reactions, although important, may be difficult to ascertain.⁴⁶ Many persons with positive PT reactions to fragrance may tolerate certain fragrances. Cited present relevance to fragrance ranges from 64% to 87%, depending on the data set reviewed.⁴⁷ Fragrances left on fabrics by laundering that causes dermatitis is very low, and their threshold concentrations may be below induction levels.⁴⁸

Preservatives

Preservatives (including biocides) are added to cosmetics to prevent contamination. They are classified as formaldehyde and formaldehyde releasers (including imidazolidinyl urea, diazolidinyl urea, quaternium 15, and dimethylol dimethyl hydantoin) and nonformaldehyde releasers.^{38,49} The formaldehyde-releasing action can induce sensitization by repeat exposure, thereby causing ACD.⁴⁸

Methyl- and methylchloro-isothiazolinone mixture, a non-formaldehyde-releasing preservative, is one of the most common causes of preservative CD. Methylisothiazolinone can be found as a preservative in baby products (lotion, oils, powders, creams, wipes),

bath products (soaps, detergents, bubble baths), makeup (eyeliners, makeup remover, blushes, face powders), hair care products (shampoo, conditioners, sprays, straighteners, rinses, wave sets), hair-coloring products (dyes and colors, tints, bleaches), nail care products, deodorants, shaving products (after shaves, shaving creams), skin care products (cleansers, creams, lotions, moisturizers), suntan products, and sunscreens among others.⁵⁰ Methylisothiazolinone should be considered as a potential suspect allergen among patients with suspected cosmetic dermatitis, facial dermatitis, and sunscreen allergy. Testing with the methyl- and methylchloro-isothiazolinone mix misses approximately 40% of allergy to methylisothiazolinone, likely because of the low concentration of methylisothiazolinone in the methyl- and methylchloro-isothiazolinone mixture. In Europe, several groups have documented frequency of allergy to methylisothiazolinone of approximately 1.5%.⁴⁹ A positive patch test result to the methyl- and methylchloro-isothiazolinone mix has a high relevance.⁵¹

Paraben (non-formaldehyde-releasing preservative) is a weak cosmetic sensitizer and is found in 99% of leave-on products and 77% of rinse-off products.⁴⁹ Para-phenylenediamine remains an important cause of ACD to hair dye, skin paintings, and temporary tattoos. It serves as the primary coloring agents in permanent and semipermanent hair dyes and is also the main allergen used as an additive in henna. Henna without an additive gives a red coloring and is generally safe in para-phenylenediamine allergic individuals.

Cosmetics have been an important part of our daily lives for thousands of years. New products continue to be marketed, and individual ingredients used to make them continue to change. Thus, new contact allergies are emerging.

Treatment

Once the allergen or irritant has been identified, the patient should be counseled on avoidance of contact with the offending agent and informed of any cross-reactivity concerns.³ Allergen avoidance is an optimal treatment plan but may not be possible in certain situations. In addition to informing patients of what allergen they reacted to, a list of products that would be safe to use will increase adherence.³ In addition to avoidance of exposure, the physician could prescribe appropriate adjunct medical treatment.³ TCSs are moderately effective in certain individuals. A short course of oral corticosteroids may be needed in ACD, such as in poison ivy. The use of appropriate skin protection, such as barrier creams and emollients as adjunct therapy, may be helpful in some cases.³ TCIs are not approved for use in the treatment of ACD. However, they have been used with some efficacy and offer a welcome alternative to TCSs because the long-term use of TCSs has been associated with numerous adverse effects, such as skin atrophy, rebound dermatitis,

telangiectasia, striae, and dyspigmentation.⁵² Phosphodiesterase 4 inhibitors have proved to be useful with relief of pruritus and eczema in atopic dermatitis but have not been studied for ACD.⁵³

Education of workers with ACD or ICD should include prognosis and information that their disease may persist and need long-term management even after treatment and workplace modifications.³ During preemployment screening, a history of severe childhood atopic dermatitis, particularly with hand involvement, indicates an individual at risk of developing dermatitis from exposure to irritants. Employment in a dry job should be recommended. In the workplace, the use of protective equipment and skin care preparations should be encouraged.⁵⁴

Recommendations to prevent CD include the following: (1) not wearing rings so that chemicals and water may not accumulate underneath; (2) hand washing with lukewarm water and drying thoroughly because skin damage from detergent is reduced at a low temperature and dermatitis often begins at sites that are missed, such as finger webs; (3) wear dry, clean, and intact gloves for wet work, a short period, and with a cotton inner liner if prolonged exposure because wet work is the major risk factor for CD; (4) use moisturizer for barrier repair and prevention of sensitization and development of ACD to common allergens; and (5) use alcohol gel for antiseptics.⁵⁴

The outlook for occupational CD is poor. In a follow-up study of 954 patients with occupational dermatitis seen during 8 years, in which at least 60% were assessed more than 2 years after diagnosis, 55% still had disease. Of those who were still in the same job or occupation, 68% were still symptomatic compared with 37% of those who had moved to a different work environment. Notably, when changing jobs, 25% of individuals had inappropriately moved to a workplace with the same or similar chemical exposures. Of the total, 11.5% had persistent disease in the absence of any obvious cause.⁸

Conclusion

CD is a common inflammatory skin disease and should be suspected in patients presenting with acute, subacute, or chronic dermatitis characterized by pruritus, erythema, vesicles, and scale. Contact-induced dermatitis may be caused by ACD, ICD, SCD, contact urticaria, or PCD. The gold standard for diagnosing ACD is PTs.

Nickel is the most common contact allergen and has the potential to cause SCD when ingested in food. ACD to topical medicaments used frequently and for prolonged periods should be considered. Present relevance of a positive PT result is the combination of definite, probable, and possible relevance and should be correlated with the patient's history and physical examination. Treatment of ACD includes patient education about their allergens, avoidance, and alternative products they can use.

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