



Contact dermatitis: Allergic and irritant

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Abstract Facial contact dermatitis is frequently encountered in medical practice in both male and female patients. Identifying the underlying cause can be challenging, and the causative agent may be overlooked if it is not considered during the assessment of a patient. The two main types of contact dermatitis are irritant contact dermatitis (ICD) and allergic contact dermatitis (ACD). The mechanisms and common causative agents vary for both ICD and ACD, but the clinical picture is often similar, particularly for chronic disease. Facial contact dermatitis can be successfully treated by avoiding the causative agent. In this review, we focus on the clinical assessment of a patient with facial contact dermatitis and the mechanisms of both ICD and ACD. Common causative agents, including emerging allergens, are discussed in detail, and suggestions are made regarding the management of patients with proven ICD or ACD of the face.

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Introduction

Contact dermatitis is the inflammation of the skin induced by external agents. The two major types are irritant contact dermatitis (ICD) and allergic contact dermatitis (ACD). ICD occurs as a result of direct damage to the stratum corneum by chemicals or physical agents that occurs faster than the skin is able to repair itself. This results in an inflammatory nonimmunologic cutaneous reaction. Prior sensitization is not required. Although susceptibility varies among individuals, given sufficient exposure to an irritant, anyone can develop ICD. ACD is a delayed type IV hypersensitivity reaction to external chemicals (allergens) that only occurs in susceptible individuals who have previously been sensitized.

This review covers the clinical assessment of an individual who is suspected of having a contact dermatitis of the face. It then covers the mechanisms of ICD and the more common agents that may cause it. The important and

emerging allergens responsible for ACD of the face are then addressed in detail. The review finishes with suggestions for the management and treatment of the patient with proven ICD or ACD of the face.

Clinical assessment

History

Although the underlying pathologies are different, the physical and histologic findings are often very similar for both ICD and ACD, particularly when the disease becomes chronic. It can be extremely difficult for the physician to tell the two apart on the basis of the clinical examination alone. A thorough history before and after patch testing is an essential part of formulating the correct diagnosis, planning investigations, and treating and counseling a patient.

A contact dermatitis history should identify potential allergens and irritants and exclude other differential diagnoses. Specific questions should be asked about exposure to common allergens that can cause facial dermatitis; these will be explored further in this review.

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Presenting complaint

The main symptom of contact dermatitis is itching, although this is not always present; burning, stinging, or pain may also be reported. The dermatitis is, in most cases, localized to the site of contact.

The sensitization phase of ACD typically takes 10 to 14 days. The re-exposure of the skin to the allergen after sensitization results in a dermatitis. The reaction is often more severe and rapid in onset with subsequent episodes of re-exposure. Because prior sensitization is not required, ICD can present after a single episode of exposure to a strong irritant (eg, strong acids or alkalis) or repeated exposure to weak irritants. A history of seasonal variation of symptoms throughout the year may indicate a photoallergic contact dermatitis. Patients may report the exacerbation of symptoms after sun exposure, or they may report a rash that appears only after sun exposure.

Medical history

Individuals with a background of atopy (particularly atopic dermatitis) are more susceptible to ICD as a result of the impaired barrier function of the skin. Several studies have reported a high rate of positive patch tests in atopic patients.¹ Atopic dermatitis may present for the first time during adulthood, so a family history of atopy should be noted.

Potential allergens

The use of hair dye and cosmetics of the hair, face and nails is particularly important when dealing with facial contact dermatitis. More than half of the reported cases of ACD related to cosmetics involve the face and the periocular area. Patients may relate the use of certain products with the onset of skin problems, but they may wrongly assume that the long-term use of a product rules it out as a potential allergen.

Cosmetic applicators and tools should also be considered. For example, patients with sensitivity to rubber may react to rubber sponges, eyelash curlers, and adhesives that are used for false eyelashes. Nail varnishes and acrylic nails are common causes of periocular dermatitis as a result of secondary transfer.

Connubial or consort dermatitis

Contact dermatitis in a patient may be caused by the products that his or her partner is using; this can be overlooked if it is not taken into consideration during the taking of the patient history. Connubial dermatitis caused by cosmetics typically presents with a unilateral facial dermatitis, but other patterns have also been reported.²⁻⁵

Medications

Medications are a known cause of contact dermatitis, so a comprehensive list of topical and oral medications, including complementary therapies, is an essential part of the history. ACD can develop in response to either the preservatives or the active ingredients in a medication.

Topical preparations (eg, Neomycin, corticosteroids) are an important cause of facial ACD. An allergy to topical corticosteroids should be considered if a patient's signs and symptoms are exacerbated by or unresponsive to their use.

Photodrug reactions can mimic contact dermatitis; therefore, a full list of medications, including recent antibiotic use, should be documented.⁴

Occupation

Airborne contact dermatitis can result in ACD or ICD that commonly affects the face and other exposed sites. Most cases occur as a result of occupational exposure to chemicals, so specific questions should be asked about the workplace, including the availability of personal protective equipment. The improvement of symptoms during time away from work may suggest an occupational cause.

The most common causes of an airborne contact dermatitis are plants, particularly those of the family *Compositae*. Woods, plastics, rubbers, glues, natural resins, pharmaceutical chemicals, insecticides, and pesticides have also been implicated.^{6,7}

Hobbies and pastimes

Hobbies may be the source of facial contact dermatitis. Gardening involves the risk of an airborne contact dermatitis in response to being near plants. Rubber allergy may be present in patients who wear facemasks or goggles during scuba diving or swimming.⁴

Clinical presentation

The type of reaction seen in contact dermatitis varies and depends on the offending chemical, the duration of contact, and the host's susceptibility. In general, the clinical findings include erythema, scaling, vesiculation, and bullae during the acute phase and lichenification and fissuring with chronic disease. In most cases, the dermatitis is localized to the site of contact; however, patchy or diffuse disease can also occur, depending on the nature of the allergen or the development of autosensitization dermatitis, which is a reaction of the skin to contact dermatitis elsewhere on the body. Autosensitization often affects the face, particularly the periocular region.

Examples of allergens that can cause patchy or diffuse disease as a result of secondary transfer include nail varnishes and the fragrances or preservatives in shampoos or shower gels.

The examination findings of airborne contact dermatitis and a photodistributed dermatitis may look almost identical, because they both affect exposed sites. Clinical clues include the sparing of Wilkinson's triangle (ie, the area behind the ears), the nasolabial folds, and the area under the chin with photodermatitis.

Certain allergens are known to produce clinically atypical reactions. For example, paraphenylenediamine (PPD) is a black dye that is found in hair coloring and some temporary henna tattoos; it can result in acute and dramatic facial swelling that may be mistaken for a type I reaction.⁸

Pigmented contact dermatitis

Pigmented contact dermatitis (PCD) is a variant of contact dermatitis that is characterized by reticulate brown or grey hyperpigmentation with little or no signs of dermatitis. Allergens that have been implicated in the past have included the optical whiteners found in washing powder and the fragrances and chemicals in cosmetics (eg, aniline dyes). In 1917, Riehl observed a distinctive facial hyperpigmentation in 17 of his patients. Patients with identical signs were patch tested in another series and were found to react to the aniline dye found in face powder, which indicated that this type of melanosis was most probably a variant of contact dermatitis.⁴ The term *Riehl melanosis* is now synonymous with PCD. PCD caused by cosmetics was noted to be a particular problem in Japan after World War II, when an unusual pattern of facial hyperpigmentation was noted in a number of women. It was later discovered that this was caused by allergies to ingredients in certain cosmetics. After this discovery, cosmetic companies in Japan eliminated a number of allergens from their products, and the incidence of PCD declined significantly.^{4,9}

Differential diagnosis

The differential diagnosis of facial contact dermatitis includes other forms of dermatitis, such as atopic dermatitis and seborrheic dermatitis, as well as a broad range of other inflammatory skin disorders that can produce facial erythema, including rosacea, psoriasis, and connective tissue disorders.

Irritant contact dermatitis

Definition

ICD is a nonimmunologic, nonspecific localized skin reaction to a wide range of causes that may be physical, mechanical, or chemical. It results from direct contact with irritants and damage to the skin, and it is a multifactorial disorder that involves a combination of endogenous and exogenous factors.^{10,11} ICD is known to be the most common type of contact dermatitis. It represents approximately 80% of occupational contact dermatitis cases, and it is considered the most common cause of hand eczema.^{12,13}

Mechanisms

Multiple interlinked pathways are now considered to be involved in ICD, including pathophysiologic changes of skin barrier disruption; epidermal cellular damage; proinflammatory mediators released from keratinocytes; and the activation of innate immunity.^{14–16}

The initiating event of ICD is considered to be the disruption of the epidermal barrier (ie, the stratum corneum) by irritants. This results in increased skin permeability and

transepidermal water loss. In experimental studies, this acute disruption from exposure to surfactants (eg, sodium lauryl sulfate) induces the release of cytokines (eg, interleukin-1 alpha, interleukin-1 beta, interleukin-6) and tumor necrosis factor alpha from keratinocytes.^{17–20} These cytokines then act as signals for the release of further proinflammatory chemokines, which attract mononuclear and polymorphonuclear cells at the site of injury.^{18,21} Anti-inflammatory cytokines are also released in response to irritant exposure and may be involved in the resolution of the inflammatory process.²²

Some individuals develop a tolerance to repeated exposure to irritants. The adaptation of the skin to this is known as the *hardening phenomenon*. The actual mechanisms that underlie the hardening phenomenon are unclear. Contributory factors consist of irritant-induced changes in skin morphology (eg, acanthosis, hyperkeratosis), the lipid composition of the stratum corneum, the permeability of the skin barrier, and the expression of inflammatory mediators.^{23,24}

Predisposing factors

The predisposing factors for the development of ICD are influenced by a combination of host-related and environmental factors.

- Host-related factors include the following:
- Age: Skin reactivity to irritants tends to decrease with age.²⁵
- Sex: ICD is seen more frequently among women than men, likely as a result of increased exposure rather than genuine susceptibility.¹²
- Body site: The face, the dorsa of the hands, and the finger webs are more prone to chemical irritants than the palms, the soles, and the back.^{13,26}
- Atopy: Individuals with atopic dermatitis have increased susceptibility to irritants as a result of a chronically impaired barrier function.^{27,28}
- Genetic factors: Twin studies indicate that genetic factors other than atopy may influence susceptibility to ICD.²⁹

Environmental factors such as temperature, airflow, humidity, and occlusion affect the skin's response to irritants.³⁰ Cold temperatures and low ambient humidity increase transepidermal water loss and subsequently skin susceptibility to irritants,^{31,32} whereas increased humidity can disrupt the skin barrier and enhance the inflammatory response to irritants.³³

Irritants

Irritants are agents that are capable of producing cellular alteration if they are applied to the skin for a sufficient amount of time at a sufficient concentration.

Common or important irritants that have been implicated in ICD of the face are outlined in the following sections.

Eye cosmetics

Eye cosmetics are used by consumers to emphasize the appearance of the eyes. The eyelid skin is the thinnest of the whole integument, so careful selection must be made, especially by patients with preexisting skin disease or who wear contact lenses. It is important to remember that eye cosmetics can cause either ICD or ACD, both of which can cause the upper eyelid dermatitis syndrome.³⁴

Mascara

The composition of mascara influences its irritability to the skin. Loden and Wessman applied mascaras to the skin in aluminum chambers and evaluated the skin reaction with the use of both visual assessments of erythema and noninvasive measurements.³⁵ Seven mascaras were tested on 15 healthy individuals in a randomized, blinded fashion. Two of the seven tested mascaras induced pronounced skin inflammation when applied to normal skin that had been placed under occlusion. These two mascaras were based on volatile petroleum distillate; this was in contrast with the other five mascaras, which were conventional emulsions with stearate as the main emulsifier. These findings suggest that solvent-based mascaras are more likely to induce ICD.

Detergents

In health care, the prevention of infection has led to a significant increase in the use of disinfectants, detergents, and antiseptics in clinical activities. Most of these products are skin irritants, which can cause skin disorders in exposed workers. A retrospective study conducted on occupational dermatitis diagnosed in an occupational health service targeted exposed workers in the health, food, and cleaning industries. The most frequently reported dermatoses were ICD in 42% of cases and ACD in 26.3% of cases.³⁶

Shampoo

Shampoos contain the irritating chemical sodium lauryl sulfate and related detergents. Medicated shampoos may also contain benzalkonium chloride, a surfactant and irritant that is widely used in cosmetics, skin disinfectants, and ophthalmic preparations. Although there are reports of ACD in the literature, benzalkonium chloride is generally considered to be an irritant rather than allergen.³⁷

Airborne agents

Chlorothalonil is a pesticide that is used in agriculture, horticulture, and floriculture as well as in wood preservatives and in paint. An outbreak of airborne ICD, conjunctivitis, and upper airway complaints among seamstresses in a Portuguese trailer tent factory was attributed to chlorothalonil. All exposed workers had work-related skin symptoms; a delayed irritation response to chlorothalonil and to the textile extracts that contained high concentrations of chlorothalonil was noted after 72 hours.³⁸

Physical agents and stingers

Physical agents that may cause skin irritation include metal tools, wood, fiberglass, plant parts (eg, thorns, spines, sharp-edged leaves), paper, dust, and soil.^{39–41} It is also important to consider other natural stingers (eg, jellyfish) as potential sources of ICD.⁴²

Complementary medicine

Complementary medicine is increasingly used by patients who present with facial dermatitis. A structured questionnaire study of adults who were referred to a contact dermatitis clinic in a city-center teaching hospital in Leicester, UK, reported that 30% had used or intended to use complementary medicine to treat their skin conditions; this usage was associated with belonging to minority ethnic groups.⁴³

Allergic contact dermatitis

Definition

ACD is a T-cell-mediated delayed-type hypersensitivity reaction that occurs after skin exposure to a specific hapten in previously sensitized individuals. The inflammatory response in classical ACD first requires a sensitization phase, when the hapten first penetrates the epidermal skin barrier. An elicitation phase, which is responsible for the recruitment and activation of specific T cells at the site of hapten skin challenge, occurs with subsequent exposure to the same hapten.⁴⁴

The importance of the stratum corneum's barrier function and the signaling pathways that allow for keratinocyte proliferation and the generation of proinflammatory factors are increasingly recognised.⁴⁵ The association between filaggrin null mutations in ichthyosis vulgaris and atopic dermatitis,⁴⁶ together with protease and lipid defects, highlights the role of barrier disruption that allows for the increased access of environmental agents, microbes, irritants, and allergens.

Human skin is now exposed to a huge variety of cosmetic allergens. The majority of reactions occur after exposure to fragrances, preservatives, and hair dyes. Such reactions can often be occult. As a result, a high index of suspicion is needed when assessing a patient with facial or cosmetic dermatitis.⁴⁷ A recent review of patients who were patch tested in the United States reported that, of women with a positive patch test reaction, 24% of cases were the result of a documented cosmetic source.⁴⁸

To improve usability for the consumer, cosmetics often have a high water content. This leaves the preparation at a risk of being contaminated by pathogenic microorganisms, such as *Staphylococcus aureus* and *Pseudomonas aeruginosa*. Bacterial contamination may alter the composition of

the product or pose a health risk to the consumer. To prevent this, preservatives, including biocides, are added to cosmetics. Varying concentrations of the same preservative are found in related products, which results in the question of whether some of these preparations are overpreserved. Because the development and elicitation of ACD are dose dependent,⁴⁹ the overpreservation of cosmetics potentially leads to an increased rate of contact allergy.⁵⁰

Important and emerging allergens

Cosmetics

Methylchloroisothiazolinone and methylisothiazolinone.

Methylchloroisothiazolinone (MCI) and methylisothiazolinone (MI) have become among the most important allergens found in cosmetics during the last few years. Optimal testing concentrations have been the subject of debate. After the changing of the concentration of MCI/MI from 0.01% to 0.02% in the British Society for Cutaneous Allergy baseline series in September 2009, detection rates have increased, with a quarter of positive patients having used moist cleansing wipes, which are a well-known source of MCI/MI. Other sources of exposure include shampoo, dishwashing liquid, and cosmetics. It is important to also test for MI on its own. This is not as potent an allergen as MCI, but, with its increased use—especially as one of the ingredients in many “gentle” cosmetics and wet wipes—allergic reactions are becoming increasingly significant.⁵¹

Other isothiazolinones have been developed specifically for use in paints, adhesives, and metalworking fluids. In contrast with MCI and MI, these chemicals are rare sensitizers. There is little cross-reactivity between the allergens used in cosmetics and those used in industry.⁵²

Iodopropynyl butylcarbamate. Iodopropynyl butylcarbamate (IPBC) is a biocide that was originally developed for use in an industrial context, first in metalworking and later as a wood preservative, where concentrations of up to 4% are permitted.⁵³ Contact allergies in these industrial settings are well described.⁵⁴ Due to its usefulness as a biocide, IPBC has more recently been incorporated into cosmetic products and cleansing wipes,⁵⁵ where the maximum permitted concentration is 0.1%. Reports of ACD related to IPBC appeared only a few years after this substance’s introduction as a cosmetic ingredient.⁵⁶

Although IPBC is only thought to be weakly allergenic, it is a small lipophilic molecule that may readily penetrate the skin.⁵⁷ As reports of contact allergy from IPBC exposure in cosmetics have increased, it is now incorporated into the cosmetic series of patch test allergens in many countries.

Sodium metabisulfite. The sulfites are a relatively ubiquitous group of chemicals. Sodium metabisulfite is present in food and drink as a preservative and an antioxidant, where it is labeled as “E223.” It is a component of photographic chemicals; it is used in rubber manufacture, leather tanning, and mineral extraction; and it serves as a bleaching agent in fabric treatments.⁵⁸ It has more recently been noted that sodium metabisulfite is present in some

ketoconazole-containing products as well as in Trimovate® and Timodine creams,⁵⁹ and it accounts for relevant positives in patients who have developed ACD while using these products.⁶⁰

Sodium metabisulfite has now been identified as a component of cosmetic creams,⁵⁹ hair coloring, and skin bleaching and false tanning products.⁵⁸

Although contact allergy with patch testing is common, the relevance of these reactions remains unclear. Up to one third of patch-test–positive patients have been reported to have dermatitis of the face and neck, a positive reaction to sodium metabisulfite may not always be clinically relevant.⁵¹

Propolis. Propolis is a resinous substance that is collected from poplars and processed by honeybees and that is used as a sealant to maintain the structure of the hive. It is therefore a well-recognized cause of occupational contact dermatitis among apiarists.⁶¹ It also has a variable chemical composition, and it is regarded as a potent skin sensitizer.⁶²

As a result of its purported antibacterial and anti-inflammatory properties, propolis has been increasingly used in a number of “natural” over-the-counter products, such as cough syrups, lozenges, shampoos, conditioners, lipsticks, lip balms, lotions, toothpastes, and cosmetics.^{63,64} Although this was accompanied by an associated early increase in reported allergic reactions, rates of ACD do now appear to be diminishing. Propolis was dropped from the British Society for Cutaneous Allergy Standard Series in 2012.

Dicaprylyl maleate. Dicaprylyl maleate (DCM), which is also known as dioctyl maleate, is an emollient and solvent. It is also a good example of a case in which the original chemical testing indicated that the compound did not cause ACD.⁶⁵

Ten years after its introduction into cosmetic products, DCM has been found to cause contact allergy in selected patients. It has been used as an ingredient in false tanning lotions, moisturizers, foundations, and sunscreens.⁶⁶ A series of 22 patients who were suspected of having contact allergic reaction to DCM were recently tested to freshly manufactured DCM and deliberately aged DCM. Only eight patients did not have a positive DCM patch test; of these eight patients, six developed a positive reaction to the aged DCM.⁶⁵

This phenomenon also occurs with other contact allergens (see the information about limonene later in this paper) and is thought to be a response to chemical degradation (autoxidation), which may produce allergenic byproducts.⁶⁷ This is ironic in that it may be seen as supporting the cosmetic industry’s argument for the increased use of newer preservatives in cosmetic products.

Hair dyes

Hair dyeing both at home and in the salon—and, therefore, exposure to PPD and related allergens, including aminophenol and diaminotoluene sulfate—should always be considered in the patient with facial dermatitis.

Patients with strong reactions to PPD on patch testing are significantly more likely to have given a clear history of reacting to normal consumer hair dye. Conversely, those with weak reactions on patch testing can and do continue to dye their hair, and this must be taken into account when taking a history from these patients. Consumers and hairdressers should be made aware that the 24-hour test application of hair dye, which has been proposed as a self-screen, does not reliably identify all individuals who are allergic to PPD. A significant rise in the frequency of PPD allergy has been observed in many centers, but active sensitization from standard PPD patch testing is uncommon.⁶⁸

Sunscreens

Sunscreens contain ultraviolet (UV) filters that help to protect the user from the acute and chronic damaging effects of UV radiation (UVR), including sunburn, skin pigmentation, skin carcinogenesis, and photoaging. UV filters are important for the protection of patients with photosensitive dermatoses, and they are increasingly used to protect skin care products themselves from photodegradation. As the public has become increasingly aware of both the dangers of excessive sun exposure and the prevention of photoaging, the use of sunscreen in the formulation of personal care products has also increased.⁶⁹

Sunscreen products usually contain one or more UV filters that can be classified as organic or physical agents according to their mechanism of action. Organic or chemical UV filters absorb UVR, whereas physical UV filters act as barriers and reflect UVR. The physical UV filters include titanium dioxide and zinc oxide, which are chemically and biologically inert and thus unlikely to cause irritation or skin sensitization. As a result of the opaque appearance of physical agents on the skin, chemical UV filters are generally considered more cosmetically acceptable and are therefore more commonly used in personal care products. Chemical UV filters can be further subdivided into UVA protectors, which operate in the 320-nm to 400-nm range of wavelengths, and UVB protectors, which operate in the 290-nm to 320-nm range of wavelengths. These agents can potentially cause ACD or photoallergic contact dermatitis.⁶⁹

The sun-exposed sites are predominantly involved during the initial stages of dermatitis; however, as a result of the presence of circulating activated T lymphocytes, the condition may subsequently affect sites that had been protected from the sun.⁶⁹

At present, sunscreen chemicals are the most common cause of photoallergic contact dermatitis in the United Kingdom. The most common photoallergens currently include benzophenone 3, butylmethoxydibenzoylmethane, and the newly emerging benzophenone-4 and octyl triazone (octocrylene).^{70,71} Benzophenone-3 and -4 do not appear to cross react.⁷² Newer sunscreens contain copolymers that have been recently reported to cause ACD.⁷³ In an interesting illustration of the increasing use

of allergenic sunscreens in cosmetics (and vice versa), the same copolymer product has now been reported to cause ACD in response to cosmetics,⁷⁴ whereas benzophenone-4 has been reported in facial moisturizers, makeup, and hair care products.⁷²

Consumers should be reminded that even so-called "organic" and "safe" sunscreens contain allergenic UV filters.⁷⁵

Fragrances

Contact dermatoses caused by perfumes include ACD, ICD, contact urticaria, and photoallergic and phototoxic contact dermatitis.⁷⁶ Considering the ubiquitous occurrence of fragrance materials, the risk of allergy is small. In absolute numbers, however, fragrance allergy is common.⁷⁷ The prevalence of ACD as a result of fragrance in the general population has been estimated at 1.8% to 4.2%.⁷⁸

Fragrance is the second most common cause of ACD, after nickel. The distribution of the skin's response classically involves the axillae, the face (including the eyelids), and the neck. Well-circumscribed patches in areas where perfumes have been "dabbed on" (eg, the wrists, behind the ears) and the aggravation of hand eczema have also been described. The degree of sensitivity and therefore the severity of the dermatitis vary from mild to severe. In the worst cases, fragrance contact allergy can cause disseminated eczema and even erythroderma. Patients with profound sensitivity are affected by airborne or connubial fragrance usage. The products with the highest concentrations of allergens have been shown to be prestige perfumes intended for women.⁷⁹

Patch testing with the use of the Fragrance Mix I combination was introduced in Europe during the late 1970s in response to the important work of Larsen,⁸⁰ and it is estimated to still detect 70% to 80% of all cases of fragrance sensitivity.⁸¹

Between 2002 and 2003, Fragrance Mix II was subjected to trials in an attempt to pick up the remaining 15% to 20% of cases.⁸² This mix consisted of Lyrall[®], citral, farnesol, citronellol, α -hexyl-cinnamic aldehyde (AHCA), and coumarin; it entered the British Society for Cutaneous Allergy Standard Series in 2007.

Lyrall[®] (hydroxyisohexyl 3-cyclohexene carboxaldehyde) has a lily-like scent. It is commonly used in deodorant, shampoo, soap, and fine fragrances.⁸³ European contact allergy rates to Lyrall[®] vary between 1.5% and 3%.⁸⁴ This rate is much higher than the US rate of 0.4%,⁸⁵ possibly as a result of the higher concentrations of Lyrall[®] in European deodorants.⁸⁴

Limonene is widely used as a fragrance in cosmetics. It is present as a component of certain essential oils (eg, rosemary, peppermint, lemongrass, lavender, eucalyptus, caraway) and of tea tree oil.⁸⁶ As a result of its solvent capacity, it is used in domestic cleaning products and also in industry as a metal degreaser, where it can occur in concentrations up to 95%.⁸⁷ It first appeared for industrial

use during the late 1980s, and it then became widely used because it was regarded as more environmentally friendly than its organic solvent equivalents. With exposure to air, however, limonene easily oxidizes into products with considerable sensitizing capacity, such as limonene oxide, *l*-carvone, and the limonene hydroperoxides.⁸⁸ A Swedish group patch tested 2800 patients with varying concentrations of fresh and aged *d*-limonene. The highest rate of contact allergy (5.1% of patients) was found when 5% *d*-limonene was exposed to air for 10 weeks.⁸⁸

Individuals with perfume contact allergy or hand eczema have more frequent and severe eye or airway symptoms after exposure to volatile fragrance products. Because new and occult fragrance chemicals can be used without restriction until emerging contact allergy is detected by dermatologists and the relevant regulators then informed, it appears that fragrance allergy will continue to be a problem for many years to come.^{89,90}

Ophthalmic preparations

As with the application of other topical drugs, the use of ophthalmic medications may induce local adverse effects. One of the undesirable—although relatively rare—reactions to topically applied ophthalmic medications is contact inflammation of the skin around the eye (periocular dermatitis) and the conjunctiva (conjunctivitis).⁹¹

ACD has been reported in response to preservatives such as benzalkonium chloride, thimerosal, phenylmercuric salts, metabisulfites, and chlorobutanol in addition to antiglaucoma medications, and nonsteroidal anti-inflammatory drugs. Mild to life-threatening allergic reactions can result from the application of topical antibiotics. Aminoglycosides and sulfonamides are among the most allergenic classes of topically applied antibiotics, although ACD in response to other classes is rare. Topical anesthetics are a large group of potentially allergenic medications that are widely used in ophthalmology for diagnostic and treatment procedures. The most frequently applied local anesthetics (ie, tetracaine, oxybuprocaine [benoxinate], and proparacaine) have been reported causes of both contact dermatitis and conjunctivitis.⁹¹

Management

Counseling

The definitive treatment of contact dermatitis is the identification and avoidance of the underlying cause.

Patients should be made aware of the substances that they are allergic to and advised about how to avoid further exposure to those substances. An informational leaflet is a useful tool for patient education; it should provide the name of the chemical, its synonyms, its common uses, and examples of the types of products in which it may be found. Patients should be advised to check the lists of

ingredients of all of their personal care products and topical medications before applying them. It is also important to inform patients about the risk of cross-reactivity to other related chemicals. If a patient is unsure about a product, a repeated open application test can be performed at home. To perform this test the product is applied to a dermatitis-free area of the skin that can be clearly visualized; common sites that are used include the antecubital fossa and the forearm. The product should be applied to the area twice a day for 1 to 2 weeks. If a reaction develops, then the product in question is best avoided.

Patients with ICD should be given information about how best to avoid irritants both at home and in the workplace. After an irritant has been identified, measures should be taken (eg, the use of personal protective equipment in the workplace) to reduce the risk of future exposure.

It is important for the dermatologist to remember that patients who are using cosmetics on their faces may not welcome this news and may not always want to stop using the products that have been identified as problematic. Patients who have been proven to have ACD related to PPD may continue to color their hair after patch testing. This practice can result in chronic problems and requires skillful discussion during the counseling and ongoing management of these patients.⁶⁸

It has been demonstrated that many patients attempt dietary manipulation in the belief that their dermatitis is caused by ingested foodstuffs. The role of counseling about the causes of ACD and the importance of interactions between allopathic and complementary therapies will again be an important one.⁹²

Topical treatment

The regular use of emollients enhances the barrier function of the skin and is an important part of the management of contact dermatitis.

Topical corticosteroids have been shown to be effective for the treatment of contact dermatitis if the underlying allergen or irritant is avoided. Topical tacrolimus or pimecrolimus has also been shown to be effective for the management of facial dermatitis.⁹³

Systemic corticosteroids and immunomodulators

Systemic corticosteroids may be required for the short term during an acute phase of an extensive or severe contact dermatitis. If left untreated, contact dermatitis can develop into chronic dermatitis. Psoralen and UVA treatment, narrow-band UVB treatment, or systemic treatment with immunomodulators (eg, methotrexate, cyclosporine) and targeted biologic therapy may be considered for patients with chronic dermatitis that is unresponsive to other measures. The treatment of any underlying skin conditions (eg, atopic dermatitis, psoriasis) should also be optimized.⁹⁴

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