Proposed ICDRG Classification of the Clinical Presentation of Contact Allergy

Korbkarn Pongpairoj, MD, MSc,* Iris Ale, MD,† Klaus Ejner Andersen, MD, DMSc; Magnus Bruze, MD, PhD,§ Thomas L. Diepgen, MD, PhD;¶ Peter U. Elsner, MD,¶ Chee Leok Goh, MD, MRCP(UK), FRCPE, MMed;# An Goossens, Pharm, PhD,** Hemangi Jerajani, MD, DV&D,†† Jean Marie Lachapelle, MD, PhD;‡‡ Jun Young Lee, MD, PhD;‡§ Howard I. Maibach, MD;¶¶ Kayoko Matsunaga, MD, PhD;¶¶¶ Rosemary Nixon, MD;### Pailin Puangpet, MD,*** Denis Sasseville, MD, FRCP;††† Supitchaya Thaiwat, MD,‡‡‡ and John P. McFadden, MD‡‡‡‡

The International Contact Dermatitis Research Group proposes a classification for the clinical presentation of contact allergy. The classification is based primarily on the mode of clinical presentation. The categories are direct exposure/contact dermatitis, mimicking or exacerbation of preexisting eczema, multifactorial dermatitis including allergic contact dermatitis, by proxy, mimicking angioedema, airborne contact dermatitis, photo-induced contact dermatitis, systemic contact dermatitis, noneczematous contact dermatitis, contact urticaria, protein contact dermatitis, respiratory/mucosal symptoms, oral contact dermatitis, erythroderma/exfoliative dermatitis, minor forms of presentation, and extracutaneous manifestations.

PROPOSED INTERNATIONAL CONTACT DERMATITIS RESEARCH GROUP CLASSIFICATION OF THE CLINICAL PRESENTATION OF CONTACT ALLERGY

The single commonest mistake made by clinicians with regard to contact allergy is not considering the diagnosis. Although many cases of contact allergy arise from direct contact of the skin by the allergen producing dermatitis at the exact site, there are many different and subtle other ways in which contact allergy can present. Without an awareness of these different manifestations, many cases of contact allergy will be missed or misdiagnosed. A simple classification is given hereafter, with some examples. This classification is aimed as a practical rather than academic exercise. It is therefore not primarily based on taxonomical or immunological classification. Rather, it is primarily based on the mode of clinical presentation, therein acting as a potential "aide memoire" for the clinical dermatologist. The tables and references are also designed as a reference for the clinical dermatologist. We acknowledge that some of the reported cases/complications are not universally accepted, especially complications such as joint loosening and joint failure from allergy to implanted metals.

Direct Exposure/Contact Dermatitis

A patient with contact allergy usually has a history of direct exposure to the causative allergen(s) and a clinical history of compatible dermatitis, together with a positive reaction on patch testing. The examples are as follows:

- allergic contact dermatitis (ACD) from fragrance applied to the neck/wrist1,2
- ACD to topical agents included in skin care preparations such as moisturizers and eye cosmetics
Six patients complaining of worsening of their seborrheic dermatitis performed in all patients with chronic recalcitrant discoid eczema. Therefore, strongly recommended that patch testing should be performed in all patients with discoid eczema demonstrating positive patch test reactions. It is, however, strongly recommended that patch testing should be performed in all patients with discoid eczema with no apparent causative role or as medicament allergens. Approximately 50% of patients with discoid eczema demonstrate positive patch test reactions. It is, therefore, strongly recommended that patch testing should be performed in all patients with chronic recalcitrant discoid eczema.

The commonest allergens identified are as follows:

- Acne-like dermatitis were triggered/exacerbated by occupational irritant and/or ACD.
- Six cases of atopic eczema–like dermatitis were triggered/exacerbated by occupational irritant and/or ACD.
- Five patients who had history of childhood atopic dermatitis had exacerbation of atopic eczema after repeated chemical exposure, either from their occupations in adulthood or using cosmetics in their teenage years.
- A case of thimerosal allergy and vaccination reaction was described, where thimerosal ACD triggered an exacerbation of atopic dermatitis.
- Medicament allergy commonly presents as a worsening of a treated dermatitis or a dermatosis not responding to treatment.
- Discoid eczema

Allergic contact dermatitis, in particular to chromate from leather or cement exposure, may present as a discoid eczema. Contact allergy may be associated with discoid eczema with no apparent causative role or as medicament allergens. Approximately 50% of patients with discoid eczema demonstrate positive patch test reactions. It is, therefore, strongly recommended that patch testing should be performed in all patients with chronic recalcitrant discoid eczema.

The commonest allergens identified are as follows:

- Nickel sulfate
- Potassium dichromate
- Cobalt chloride
- Colophony
- Neomycin sulfate
- Rubber chemicals
- Formaldehyde
- Potassium peroxymonosulfate in hot tubs, which has been reported to cause a nummular dermatitis

**Mimicking or Exacerbation of Pre-existing Eczema**

Allergic contact dermatitis can mimic and/or exacerbate pre-existing eczema (sometimes, it can be hard to distinguish between these). These patients usually present with difficult-to-treat or worsening rashes. The examples are the following:

- ACD can sometimes present with a seborrheic pattern or worsening seborrheic dermatitis, for example, ACD to methylisothiazolinone (MI) (Pongpairoj, unpublished data, 2015), isothiazoline derivatives, methylidibromo glutaronitrile, Parthenium, and ACD to nail varnish allergy, which can present with seborrheic dermatitis–like lesions. A case of propylene glycol and dodecyl gallate contact allergy and a case of ACD to propyl gallate and pentylene glycol in emollient cream presented with exacerbation of seborrheic dermatitis.
- Six patients complaining of worsening of their seborrheic dermatitis were found to be allergic to their steroid medications.
- A child who had been sensitized with MI from wet wipes presented with ACD to airborne MI exposure from freshly painted walls in a new apartment with an apparent worsening of his flexural atopic dermatitis. A similar case of MI allergy from airborne exposure mimicking atopic dermatitis has also been described.
- Six cases of atopic eczema–like dermatitis were triggered/exacerbated by occupational irritant and/or ACD.
- Five patients who had history of childhood atopic dermatitis had exacerbation of atopic eczema after repeated chemical exposure, either from their occupations in adulthood or using cosmetics in their teenage years.
- A case of thimerosal allergy and vaccination reaction was described, where thimerosal ACD triggered an exacerbation of atopic dermatitis.
- Medicament allergy commonly presents as a worsening of a treated dermatitis or a dermatosis not responding to treatment.
- Discoid eczema

**Multifactorial Dermatitis Including ACD**

Hand dermatitis may be a result of the following multiple factors including a component of ACD:

- Hand dermatitis in hairdressers will often be multifactorial, that is, a combination of ACD (eg, from hair dyes, perming agents) with irritant dermatitis (eg, from surfactants in shampoos) on an atopic background.
- The loss of function filaggrin gene predisposes not only to atopic dermatitis but also to both irritant and some causes of ACD, making a combination of irritant, ACD, and atopic hand eczema feasible.
- Multiple relevant contact allergies can become “multiple ACD.” For example, leg ulcer patients may have concomitant allergies to several medicament allergens (such as antimicrobials, topical steroids), bases (such as cetostearyl alcohol), rubber chemicals in elasticated dressings, and ingredients of dressings.

**By Proxy**

Allergic contact dermatitis “by proxy,” also known as “consort or connubial dermatitis,” represents a contact dermatitis to an allergen via exposure through another individual. The routes of exposure can be via direct contact with other people, airborne, or even from contaminated clothes and beds. Occupational sources (eg, work clothes soiled with epoxy resin) are occasionally reported. The patients with ACD by proxy are usually highly sensitive to the causative agent(s). Clinical presentation can be either classical or atypical. Eczema, nummular eczema, pseudolymphomatoid rash, and plaques have been reported. The lesions may present with a bizarre pattern and can be either generalized or unilateral.

Offending allergens include the following:

- Fragrance
- Cosmetic preservatives
- Plant products as ingredients in cosmetics
- Hair dyes
- Medicaments
- Chemical products applied to the genitalia

**Mimicking Angioedema**

Severe facial ACD can mimic angioedema. Both conditions may have a short onset from time of exposure, and both may have facial edema as their dominant initial characteristic. However, severe...
facial ACD will usually take longer (several days) to resolve, usually with the appearance of epidermal changes such as desquamation. Positive patch tests, together with negative skin prick test and normal serum IgE level will indicate that the angioedema-like lesions are caused by ACD rather than immediate hypersensitivity reaction (type 1 hypersensitivity). The patients may be exposed through direct exposure, airborne, or inhaled exposure. Reported causative agents include the following:

- para-phenylenediamine (PPD)
- MI
- inhaled budesonide
- castor oil
- benzoyl peroxide
- diphenylcycloprenone
- cinnamon
- pine caterpillar

**Airborne Contact Dermatitis**

Airborne contact dermatitis is a common type of contact dermatitis, which is caused by contact allergens and/or irritants in the air. These agents may be in the forms of fibers, dust particles, sprays, vapors, or gases. Plant allergens, for example, Compositae and *Parthenium*, are also commonly present with airborne ACD. Skin reactions that have been reported include eczema, acne, exfoliative dermatitis, fixed drug eruption, hyperpigmentation and hypopigmentation, lichenoid dermatitis, lymphomatoid contact dermatitis, pellagra-like lesions, purpura, pustular reactions, telangiectasia, erythema multiforme–like eruptions, erythroderma, photocontact urticaria, and phototoxic and photoallergic contact dermatitis. The distribution of rashes is usually symmetrical. The periorbital areas, face and neck, or all exposed sites are usually involved. Eyelids are particularly susceptible and may be the only affected site. However, allergens may be trapped with sweat and cause reactions under clothing; thus, cutaneous lesions can rarely be found on both exposed and covered areas.

The principle allergens described in airborne contact dermatitis are as follows:

- fragrance
- preservatives (eg, isothiazolinones)
- plants (eg, sesquiterpene lactones)
- woods
- epoxy resins
- drugs (eg, corticosteroids)

A potential diagnostic pitfall is airborne irritant contact dermatitis, which can be mistaken for or difficult to differentiate from airborne ACD.

**Photoinduced Contact Dermatitis**

**A. Photoallergic Contact Dermatitis**

Patients with photoallergic contact dermatitis usually present with sharply demarcated, symmetrical eczematous lesions on sun-exposed areas. The sparing of shadowed regions, such as eyelids, retroauricular, and submandibular areas, is an important clue to differentiate from airborne contact dermatitis. Unilateral rash is possible if a photoallergen is applied only on 1 side, asymmetrical exposure to the light, or by proxy. Other clinical features, including erythema multiforme–like reaction, leukomelanoderma, lichenoid photosensitive dermatitis, urticaria, and purpura, have been reported. Fever, rigors, diarrhea, and abnormal liver function may be found. Some patients with photoallergic contact dermatitis may follow with persistent light reaction. Reported contact photoallergens include the following:

- sunscreen: para-aminobenzoic acid, salicylates, cinnamic acids, benzophenones, dibenzoylmethanes, camphor derivatives, and phenylbenzimidazole sulfonate
- nonsteroidal anti-inflammatory drugs: ketoprofen, ibuprofen, suprofen, tiaprofenic acid, diclofenac, etofenamate, piroxicam, indomethacin, fepradinol, flufenamic acid, etofenamate, and benzylamine
- pesticides: maneb, fenitrothion, mancozeb, and tetrachloroisophtalonitrile
- cadmium (tattoo)
- cinchocaine
- halogenated salicylanilides
- fragrances: musk ambrette and 6-methylcoumarin

**B. Photoaggravated Contact Dermatitis**

Photoaggravated ACD is evident with a history of dermatitis exacerbated by UV light in conjunction with positive results in both patch and photopatch tests and a stronger reaction at the irradiated site. The important causative agents, which have been reported, are as follows:

- methylchloroisothiazolinone (MCI)/MI
- *Rosmarinus officinalis*
- thiabendazole
- ketoprofen
- epoxy resin

In addition, individuals with chronic actinic dermatitis are at risk of developing multiple contact and photocontact (principally sunscreen) allergies.

**Systemic Contact Dermatitis**

Systemic exposure to a specific allergen in a sensitized individual can cause reactions, known as “systemic contact dermatitis.” A variety of clinical manifestations with the responsible allergens include the following:

- refractory vesicular hand dermatitis (eg, nickel, cobalt, chromate, formaldehyde, garlic, balsam of Peru, food preservatives)
- “baboon syndrome” (eg, *Myroxylon pereirae*)
- pruritic papules on the extensor regions of the elbows and/or knees (eg, dietary metals, propylene glycol)
- acral and anogenital erythemas (eg, drugs, *Myroxylon pereirae* foods)
- groin dermatitis from systemic sodium metabisulphite allergy
- itchy papules, macules, and vesicles on the inner forearms, upper eyelids, and perianal areas with malaise and a subfebrile temperature
after receiving a homeopathic therapy containing gold in a gold-allergic patient\textsuperscript{55}

• reappearing or flare of the previous sites of ACD and/or previous patch test reactions (eg, sesquiterpene lactone, budesonide inhalation,\textsuperscript{56} nickel, and gold\textsuperscript{57})
• widespread eczema (eg, resin in a dental product\textsuperscript{58})
• erythroderma (eg, urushiol/lacquer in health foods\textsuperscript{59})

**Noneczematous Contact Dermatitis**

Allergic contact dermatitis can present with several clinical patterns other than eczematous lesions as shown in Table 1.

**Contact Urticaria**

Patients with contact urticaria\textsuperscript{133,134} present with wheals and flares occurring within 30 minutes after exposure to an external substance. Some agents, for example, cinnamon, can cause both nonimmunological and immunological contact urticaria. Then, the lesions are totally clear within hours without residual signs. Systemic symptoms can be found in “contact urticaria syndrome,” including rhinoconjunctivitis, asthmatic attack, and orolaryngeal and gastrointestinal symptoms. Anaphylaxis and anaphylactoid reactions are rarely observed in severe cases. The following allergens are reported as causing contact urticaria:

• ammonium persulfate\textsuperscript{135}
• fragrance

**Protein Contact Dermatitis**

Clinical manifestations of protein contact dermatitis\textsuperscript{133,145} include itchy wheals and flares appearing within minutes after contacting with high molecular weight protein allergens, mainly protein in animals or plants. In contrast to contact urticaria, some patients may present with eczematous lesions with or without urticarial rashes. Vesicular and dyshidrotic eczema may be clinical manifestations. Patch test is often negative, whereas scratch and prick test are positive. The association between protein contact dermatitis and atopy is demonstrated in approximately 50% of the cases. Occupational protein contact dermatitis has been reported among food handlers.

<table>
<thead>
<tr>
<th>Table 1: Various Clinical Manifestations of Noneczematous Contact Dermatitis and the Reported Causative Allergens</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Clinical Manifestations</strong></td>
</tr>
<tr>
<td>Azo dyes, naphthol AS,60 fragrance, insoluble cutting oils,61 PPD,62 and hydroquinone63</td>
</tr>
<tr>
<td>Hair dye (PPD,\textsuperscript{64-66} 4-phenylenediamine base and 4-aminophenol\textsuperscript{67}), propyl gallate,\textsuperscript{68} dimethylfumarate,69 pramin,70 azo dye,71 epoxy resin,72 paraterythral butylphenol,73 and thiram and mercaptobenzothiazole74</td>
</tr>
<tr>
<td><strong>Depigmented</strong></td>
</tr>
<tr>
<td>Dyes,75 epoxy resin,76 chromium,77 PPD,78 and medicaments79-82</td>
</tr>
<tr>
<td><strong>Purpuric</strong></td>
</tr>
<tr>
<td>Dyes,75 epoxy resin,76 chromium,77 PPD,78 and medicaments79-82</td>
</tr>
<tr>
<td><strong>Lichenoid</strong></td>
</tr>
<tr>
<td>Skin: PPD in hair dye,83 color developers (chemicals derived from PPD),84 primula, epoxy resins,85 nickel,86 aminoglycoside,87 and methacrylic acid esters88</td>
</tr>
<tr>
<td>Oral mucosa89:</td>
</tr>
<tr>
<td>Dental metals: copper,90 zinc,91 mercury,92 gold,93-95 beryllium, cobalt, chromium, nickel, palladium, silver, and tin</td>
</tr>
<tr>
<td>Dental adhesives: acrylic compounds and eugenol</td>
</tr>
<tr>
<td>Other dental restoration materials: composite, glass ionomer, and porcelain</td>
</tr>
<tr>
<td>Flavorings: balsam of Peru, cinnamon, cinnamic aldehyde, eugenol, menthol, peppermint, and vanillin</td>
</tr>
<tr>
<td>Oral, cutaneous, and genital lichenoid reactions: metals96</td>
</tr>
<tr>
<td><strong>Erythema multiforme</strong></td>
</tr>
<tr>
<td>Nickel,97 cobalt,98 ethylenediamine,99,100 PPD (local and distal),66,101-103 woods, plants,104 urushiol,17 topical medicaments (neomycin,100 pyrrolnitrin, sulfonamide,105 promethazin,106 mafenide acetate,107 mephenesin\textsuperscript{107}), nonsteroidal anti-inflammatory drugs (phenylbutazone,108 bufexamac,109 mofubutazone\textsuperscript{110}), corticosteroids (budesonide,111 triamcinolone acetone\textsuperscript{112}), and epoxy resin113</td>
</tr>
<tr>
<td><strong>Pseudolymphomatous dermatitis</strong></td>
</tr>
<tr>
<td>PPD, para-tertiary-butylphenol-formaldehyde resin, gold, ethylenediamine, nickel,104 and methylisothiazolinone\textsuperscript{114}</td>
</tr>
<tr>
<td><strong>Granulomatous</strong></td>
</tr>
<tr>
<td>Metals\textsuperscript{115}: palladium, beryllium, zirconium, titanium, nickel, mercury, chromium, cobalt, and aluminum\textsuperscript{116}</td>
</tr>
<tr>
<td><strong>Pustular</strong></td>
</tr>
<tr>
<td>Nitrofurazone,117 black rubber,118 minoxidil,119 and fragrances120</td>
</tr>
<tr>
<td><strong>Neutrophilic and eosinophilic dermatitis</strong></td>
</tr>
<tr>
<td>PPD\textsuperscript{121}</td>
</tr>
<tr>
<td><strong>Bullous</strong></td>
</tr>
<tr>
<td>PPD,122 Betadine,123 acrylicates,124 colophonium,125 and Critonia aromatisans (Compositae family)126</td>
</tr>
<tr>
<td><strong>Sclerodermoid</strong></td>
</tr>
<tr>
<td>Epoxy resin\textsuperscript{127} and vitamin K\textsubscript{1}\textsuperscript{128}</td>
</tr>
<tr>
<td><strong>Granulomatous cheilitis</strong></td>
</tr>
<tr>
<td>Betel quid,129 benzoates, cinnamon,130 octyl gallate, dodecyl gallate\textsuperscript{131}, and sodium metabisulfite\textsuperscript{132}</td>
</tr>
</tbody>
</table>
The patients presented with chronic, recurrent hand and/or forearm eczema. Respiratory/Mucosal Symptoms

Contact sensitization to a particular allergen may lead to airway hyperresponsiveness.

- Many cases of allergic asthma were reported in association with allergy to acrylates in beauticians. In addition, there are reported cases related to fragrance mix I, mint, nickel, and to isocyanate.
- ACD to black henna hair dye and ACD to MI can cause severe reactions with respiratory compromise.
- Airborne ACD to ammonium persulfate in a hairdresser can present with rhinitis and bronchial asthma.
- Respiratory allergy associated with hypersensitivity to polyfunctional aziridine hardener in water-based paints and inks has been reported.

- Asthma of a 3-year-old boy was treated with inhaled budesonide to which he developed airborne ACD. On exposure to budesonide, he developed both conjunctivitis and facial dermatitis.

Oral Contact Dermatitis

The clinical manifestations of oral contact allergy vary, including mucosal dryness, burning mouth syndrome, perioral dermatitis, cheilitis, stomatitis, lichenoid reaction, and orofacial granulomatosis. Furthermore, urticaria, rhinorrhea, and perianal dermatitis have been reported. Life-threatening conditions, such as laryngeal swelling, anaphylaxis, and cardiac arrhythmias, may occur. The causative allergens and their clinical presentations are shown in Table 2.

Erythroderma/Exfoliative Dermatitis

Erythroderma/exfoliative dermatitis can be a clinical manifestation of direct exposure/contact dermatitis, airborne contact dermatitis, and systemic contact dermatitis as shown in Table 3.

---

**TABLE 2. Reported Allergens of Oral Contact Allergy and Their Clinical Features**

<table>
<thead>
<tr>
<th>Allergens</th>
<th>Clinical Presentations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Composite</td>
<td>Oral lichenoid reactions</td>
</tr>
<tr>
<td>Acrylates</td>
<td>Stomatitis, oral, cutaneous, and genital lichenoid reactions</td>
</tr>
<tr>
<td>Fissure sealant, sealant contains bisphenol A-glycidylmethacrylate</td>
<td></td>
</tr>
<tr>
<td>Metals</td>
<td>Asthma and urticaria</td>
</tr>
<tr>
<td>Mercury (amalgam)</td>
<td>Oral, cutaneous, and genital lichenoid reactions</td>
</tr>
<tr>
<td>Nickel</td>
<td>Itchy, red lesions on the face, neck and oral mucosa, and</td>
</tr>
<tr>
<td></td>
<td>oral lichenoid reactions</td>
</tr>
<tr>
<td>Titanium*</td>
<td>Urticaria, eczema, erythema of the mucosa, facial eczema,</td>
</tr>
<tr>
<td></td>
<td>and drug rash with eosinophilia and systemic symptoms</td>
</tr>
<tr>
<td>Formaldehyde</td>
<td>Hand and facial eczema</td>
</tr>
<tr>
<td>Eugenol</td>
<td>Gingivitis and stomatitis</td>
</tr>
<tr>
<td>Sodium hypochlorite*</td>
<td>Immediate mucosal swelling, burning sensation, airway</td>
</tr>
<tr>
<td></td>
<td>compromise, type 1 allergy</td>
</tr>
<tr>
<td>Latex (gloves are the source of latex exposure.)</td>
<td>Stomatitis, airway compromise, angioneurotic edema</td>
</tr>
<tr>
<td>Palladium</td>
<td>Contact stomatitis and linear lichen planus in the region</td>
</tr>
<tr>
<td>Hydroquinone</td>
<td>of the mandibular nerve</td>
</tr>
<tr>
<td>*Titanium and sodium hypochlorite disputed by some authors as true contact allergens.</td>
<td></td>
</tr>
</tbody>
</table>

**TABLE 3. Three Categories of Clinical Manifestations of Allergic Contact Dermatitis That Can Present With Erythroderma/Exfoliative Dermatitis and the Responsible Allergens**

<table>
<thead>
<tr>
<th>Categories</th>
<th>Allergens</th>
</tr>
</thead>
<tbody>
<tr>
<td>Direct exposure/contact dermatitis</td>
<td>A generalized erythrodermic reaction after</td>
</tr>
<tr>
<td></td>
<td>pseudoephedrine exposure in a patient with contact</td>
</tr>
<tr>
<td></td>
<td>sensitization to phenylephrine</td>
</tr>
<tr>
<td></td>
<td>Antioxidant, nordihydroguaiaretic acid</td>
</tr>
<tr>
<td></td>
<td>Cocobolo wood</td>
</tr>
<tr>
<td></td>
<td>Clothing disperse dyes, nb, can also cause perianal</td>
</tr>
<tr>
<td></td>
<td>Dermatitis</td>
</tr>
<tr>
<td></td>
<td>Dimethyl fumarate</td>
</tr>
<tr>
<td></td>
<td>Exfoliative dermatitis from ethylenediamine</td>
</tr>
<tr>
<td></td>
<td>Exfoliative dermatitis from valiya narayana thailam</td>
</tr>
<tr>
<td></td>
<td>(an ayurvedic oil used in aromatherapy)</td>
</tr>
<tr>
<td>Airborne contact dermatitis</td>
<td>Airborne Composites dermatitis from ragweed and</td>
</tr>
<tr>
<td>Systemic contact dermatitis</td>
<td>Parthenium dermatitis</td>
</tr>
<tr>
<td></td>
<td>Urushiol/lacquer in health foods</td>
</tr>
</tbody>
</table>

Copyright © 2016 American Contact Dermatitis Society. Unauthorized reproduction of this article is prohibited.
Minor Forms of Presentation

These include hair loss, increased hair growth, xanthelasma palpebrarum, and tattoo reactions. In addition, ACD mimicking other skin diseases, such as basal cell carcinoma, prurigo nodularis, actinic prurigo, psoriasis, and lichen nitidus, 1-hand 2-feet syndrome, folliculitis decalvans, lupus erythematosus, drug eruption, and cutaneous infection (recurrent carbuncles and abscesses) have been reported.

Some strong sensitizers, proposed term “super contact allergens,” can cause sensitization after a single exposure. The patients present with delayed onset of reactions up to 14 to 28 days. The reported “super allergens” include the following:

- MCI/MI
- epoxy resin
- acrylates
- Some patients with hair dye allergy have been sensitized with PPD in previous temporary black “henna” tattoos.

Nondermatological/Nonmucosal Manifestations

Allergic contact dermatitis can sometimes present with signs and symptoms other than skin and mucosa as shown in Table 4.

REFERENCES

42. Lachapelle JM. Industrial airborne irritant or allergic contact dermatitis. Contact Dermatitis 1986;14(3):137–145.


Chiu CS, Tsai YL. Cheilitis granulomatosa associated with allergic contact urticaria and asthma: simultaneous immediate and delayed allergy to diphenylmethane-4,4'-diisocyanate. *Contact Dermatitis* 2008;58(2):112–113.


Szema AM, Barnett T. Allergic reaction to mint leads to asthma. *Allergy Rhinol (Providence)* 2011;2(1):43–45.


Copyright © 2016 American Contact Dermatitis Society. Unauthorized reproduction of this article is prohibited.


