

Proposed ICDRG Classification of the Clinical Presentation of Contact Allergy

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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.

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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 practical "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/wrist^{1,2}
- ACD to topical agents included in skin care preparations such as moisturizers and eye cosmetics

- ACD to earrings, necklaces, and rings caused by nickel/cobalt
- ACD to rubber chemicals and chromate in leather causing glove dermatitis
- ACD to rubber, chromate, and para-tertiary-butylphenol-formaldehyde resin causing shoe dermatitis
- ACD to colophonium causing adhesive plaster reaction
- ACD to hair dye
- ACD to lanolin in lipstick causing cheilitis
- ACD to topical agents in medicaments
- cobalt chloride
- colophony
- neomycin sulfate
- rubber chemicals
- formaldehyde
- potassium peroxymonosulfate in hot tubs, which has been reported to cause a nummular dermatitis¹⁸

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) (Pongpairaj, unpublished data, 2015), isothiazolinone derivatives,³ methyldibromo glutaronitrile,⁴ *Parthenium*,⁵ and ACD to nail varnish allergy, which can present with seborrheic dermatitis-like lesions.⁶ A case of propylene glycol and dodecyl gallate contact allergy⁷ and a case of ACD to propyl gallate and pentyleneglycol in emollient cream⁸ presented with exacerbation of seborrheic dermatitis.
- Six patients complaining of worsening of their seborrheic dermatitis were found to be allergic to their steroid medicaments.⁹
- 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.¹⁰ A similar case of MI allergy from airborne exposure mimicking atopic dermatitis has also been described.¹¹
- Six cases of atopic eczema-like dermatitis were triggered/exacerbated by occupational irritant and/or ACD.¹²
- 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.¹³
- A case of thimerosal allergy and vaccination reaction was described, where thimerosal ACD triggered an exacerbation of atopic dermatitis.¹⁴
- Medicament allergy commonly presents as a worsening of a treated dermatitis or a dermatosis not responding to treatment.
- Discoid eczema^{15–17}

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

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.¹⁹
- 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.²⁰
- 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.^{21,22}

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.²³ Occupational sources (eg, work clothes soiled with epoxy resin²⁴) 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,²⁵ nummular eczema, pseudolymphomatoid rash,^{26,27} and plaques²⁸ have been reported. The lesions may present with a bizarre pattern and can be either generalized or unilateral.^{29–31}

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)^{32,33}
- MI³⁴
- inhaled budesonide³⁵
- castor oil³⁶
- benzoyl peroxide³⁷
- diphenylcyclopropanone³⁸
- cinnamon³⁹
- pine caterpillar⁴⁰

Airborne Contact Dermatitis

Airborne contact dermatitis^{41,42} 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.⁴³

Photoinduced Contact Dermatitis

A. Photoallergic Contact Dermatitis^{44,45}

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, cinnamates, benzophenones, dibenzoylmethanes, camphor derivatives, and phenylbenzimidazole sulfonic acid
- nonsteroidal anti-inflammatory drugs: ketoprofen, ibuprofen, suprofen, tiaprofenic acid, diclofenac, etofenamate, piroxicam, indomethacin, fepradinol, flufenamic acid, etofenamate, and benzydamine
- pesticides: maneb, fenitrothion, mancozeb, and tetrachloroisophthalonitrile
- 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)/MI⁴⁶
- *Rosmarinus officinalis*⁴⁷
- thiabendazole⁴⁸
- ketoprofen⁴⁹
- epoxy resin^{50,51}

In addition, individuals with chronic actinic dermatitis are at risk of developing multiple contact and photocontact (principally sunscreen) allergies.⁵²

Systemic Contact Dermatitis

Systemic exposure to a specific allergen in a sensitized individual can cause reactions, known as "systemic contact dermatitis."⁵³ 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 metabisulfite allergy⁵⁴
- 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 patient⁵⁵
- reappearing or flare of the previous sites of ACD and/or previous patch test reactions (eg, sesquiterpene lactone, budesonide inhalation,⁵⁶ nickel, and gold⁵⁷)
- widespread eczema (eg, rosin in a dental product⁵⁸)
- erythroderma (eg, urushiol/lacquer in health foods⁵⁹)

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^{133,134} present with wheals and flares occurring within 30 minutes after exposure to an external substance. Some agents, for example, cinnamal, 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 persulfate¹³⁵
- fragrance

- metals
- latex¹³⁶
- disinfectants
- preservatives
- emulsifiers
- flavorings
- coloring agents
- nickel^{137,138}
- aluminum¹³⁷
- epoxy resins¹³⁹
- isocyanate¹⁴⁰
- hydrolyzed wheat proteins^{141–143}
- parvalbumin of fish¹⁴⁴

Protein Contact Dermatitis

Clinical manifestations of protein contact dermatitis^{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 1. Various Clinical Manifestations of Noneczematous Contact Dermatitis and the Reported Causative Allergens

Clinical Manifestations	Allergens
Pigmented	Azo dyes, naphthol AS, ⁶⁰ fragrance, insoluble cutting oils, ⁶¹ PPD, ⁶² and hydroquinone ⁶³
Depigmented	Hair dye (PPD, ^{64–66} 4-phenylenediamine base and 4-aminophenol ⁶⁷), propyl gallate, ⁶⁸ dimethylfumarate, ⁶⁹ primin, ⁷⁰ azo dye, ⁷¹ epoxy resin, ⁷² paratertiary butylphenol, ⁷³ and thiuram and mercaptobenzothiazole ⁷⁴
Purpuric	Dyes, ⁷⁵ epoxy resin, ⁷⁶ chromium, ⁷⁷ PPD, ⁷⁸ and medicaments ^{79–82}
Lichenoid	Skin: PPD in hair dye, ⁸³ color developers (chemicals derived from PPD), ⁸⁴ primula, epoxy resins, ⁸⁵ nickel, ⁸⁶ aminoglycoside, ⁸⁷ and methacrylic acid esters ⁸⁸ Oral mucosa ⁸⁹ : Dental metals: copper, ⁹⁰ zinc, ⁹¹ mercury, ⁹² gold, ^{93–95} beryllium, cobalt, chromium, nickel, palladium, silver, and tin Dental adhesives: acrylate compounds and eugenol Other dental restoration materials: composite, glass ionomer, and porcelain Flavorings: balsam of Peru, cinnamon, cinnamic aldehyde, eugenol, menthol, peppermint, and vanillin
Erythema multiforme	Oral, cutaneous, and genital lichenoid reactions: metals ⁹⁶ 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 promethazine, ¹⁰⁰ mafenide acetate, ¹⁰⁶ mephenesin ¹⁰⁷), nonsteroidal anti-inflammatory drugs (phenylbutazone, ¹⁰⁸ bufexamac, ¹⁰⁹ mofebutazone ¹¹⁰), corticosteroids (budesonide, ¹¹¹ triamcinolone acetonide ¹¹²), and epoxy resin ¹¹³
Pseudolymphomatous dermatitis	PPD, para-tertiary-butylphenol-formaldehyde resin, gold, ethylenediamine, nickel, ¹⁰⁴ and methylisothiazolinone ¹¹⁴
Granulomatous	Metals ¹¹⁵ : palladium, beryllium, zirconium, titanium, nickel, mercury, chromium, cobalt, and aluminum ¹¹⁶
Pustular	Nitrofurazone, ¹¹⁷ black rubber, ¹¹⁸ minoxidil, ¹¹⁹ and fragrances ¹²⁰
Neutrophilic and eosinophilic dermatitis	PPD ¹²¹
Bullous	PPD, ¹²² Betadine, ¹²³ acrylates, ¹²⁴ colophonium, ¹²⁵ and <i>Critonia aromatisans</i> (Compositae family) ¹²⁶
Sclerodermoid	Epoxy resin ¹²⁷ and vitamin K ₁ ¹²⁸
Granulomatous cheilitis	Betel quid, ¹²⁹ benzoates, cinnamon, ¹³⁰ octyl gallate, dodecyl gallate ¹³¹ , and sodium metabisulfite ¹³²

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

Allergens	Clinical Presentations
Composite	Oral lichenoid reactions ^{163,164}
Acrylates	Stomatitis ^{161,165}
Fissure sealant, sealant contains bisphenol A-glycidylmethacrylate ¹⁶⁶	Asthma and urticaria ¹⁶⁷
Metals	Oral, cutaneous, and genital lichenoid reactions ⁹⁶
Mercury (amalgam)	Itchy, red lesions on the face, neck and oral mucosa, and oral lichenoid reactions ¹⁶⁸
Nickel	Burning sensation, gum hyperplasia, numbness on sides of tongue, ¹⁶⁹ and systemic allergic contact dermatitis ¹⁷⁰
Titanium*	Urticaria, eczema, erythema of the mucosa, ^{171,172} facial eczema, ¹⁷³ and drug rash with eosinophilia and systemic symptoms syndrome ^{174,175}
Formaldehyde	Hand and facial eczema, ^{176,177} generalized urticaria, ^{178,179} anaphylactic reactions ^{180/} shock ¹⁸¹
Eugenol	Gingivitis ¹⁸² and stomatitis
Sodium hypochlorite*	Immediate mucosal swelling, burning sensation, airway compromise, ¹⁸³ and skin test suggests type 1 allergy
Latex (gloves are the source of latex exposure.)	Stomatitis, airway compromise, ¹⁸⁴ and angioneurotic edema ¹⁶²
Palladium	Contact stomatitis ^{185,186} and linear lichen planus in the region of the mandibular nerve ¹⁸⁷
Hydroquinone	Cheilitis and stomatitis ¹⁸⁸

*Titanium and sodium hypochlorite disputed by some authors as true contact allergens.

The patients presented with chronic, recurrent hand^{146–149} and/or forearm eczema.¹⁵⁰

- 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.¹⁵⁷

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.¹⁵¹ In addition, there are reported cases related to fragrance mix I,¹⁵² to mint,¹⁵³ to nickel,¹³⁸ and to isocyanate.¹⁴⁰
- ACD to black henna hair dye¹⁵⁴ and ACD to MI can cause severe reactions with respiratory compromise.³⁴
- Airborne ACD to ammonium persulfate in a hairdresser can present with rhinitis and bronchial asthma.¹⁵⁵
- Respiratory allergy associated with hypersensitivity to polyfunctional aziridine hardener in water-based paints and inks has been reported.¹⁵⁶

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.^{158–161} Furthermore, urticaria, rhinorrhea, and perianal dermatitis have been reported. Life-threatening conditions, such as laryngeal swelling, anaphylaxis, and cardiac arrhythmias, may occur.¹⁶² 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 3. Three Categories of Clinical Manifestations of Allergic Contact Dermatitis That Can Present With Erythroderma/Exfoliative Dermatitis and the Responsible Allergens

Categories	Allergens
Direct exposure/contact dermatitis	A generalized erythrodermic reaction after pseudoephedrine exposure in a patient with contact sensitization to phenylephrine ¹⁸⁹ Antioxidant, nordihydroguaiaretic acid ¹⁹⁰ Cocobolo wood ¹⁹¹ Clothing disperse dyes, ¹⁹² nb, can also cause perianal dermatitis Dimethyl fumarate ¹⁹³ Exfoliative dermatitis from ethylenediamine ¹⁹⁴ Exfoliative dermatitis from <i>valiya narayana thailam</i> (an ayurvedic oil used in aromatherapy) ¹⁹⁵
Airborne contact dermatitis	Airborne Compositae dermatitis from ragweed ¹⁹⁶ and <i>Parthenium</i> dermatitis ^{197,198}
Systemic contact dermatitis	Urushiol/lacquer in health foods ⁵⁹

TABLE 4. Extracutaneous Presentations of Contact Allergy and Their Reported Allergens

Extracutaneous Manifestations	Procedures	Allergens
Aseptic lymphocytic vasculitis-associated lesions ^{219–221}	Hip arthroplasty	Metals
Pseudotumors ^{222–226}	Hip or knee arthroplasty	Metals
Aseptic implant loosening ²²⁷	Total hip replacement	N,N-dimethylparatoluidine in bone cement
Implant failure ^{228,229}	Arthroplastic implantation	Metals
In-stent restenosis ²³⁰	Vascular and cardiac implants	Stainless steel stents: nickel, chromate, manganese, and molybdenum ²³¹ Gold-coated stents: gold ^{232–236}
Generalized ache, especially in the left cheek*	Systemic gold provocation test with intramuscular injection of gold sodium thiomalate ⁵⁷	Gold
Abdominal pain and diarrhea*		
Body temperature change*		
Generalized malaise with subfebrile temperature*	Oral homeopathic drug	

Some relating to joint complications are controversial.

*The reported extracutaneous manifestations were present with skin rash.

Minor Forms of Presentation

These include hair loss,^{199,200} increased hair growth,²⁰¹ xanthelasma palpebrarum,²⁰² and tattoo reactions.²⁰³ In addition, ACD mimicking other skin diseases, such as basal cell carcinoma,²⁰⁴ prurigo nodularis,^{205,206} actinic prurigo,²⁰⁷ psoriasis,^{208–210} lichen nitidus,²¹¹ 1-hand 2-feet syndrome,²¹² folliculitis decalvans,²¹³ lupus erythematosus,³ drug eruption,²¹⁴ and cutaneous infection (recurrent carbuncles and abscesses)²¹⁵ have been reported.

Some strong sensitizers, proposed term “super contact allergens,” can cause sensitization after a single exposure.²¹⁶ 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.^{217,218}

Nondermatological/Nonmucosal Manifestations

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

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