

# Allergic Contact Dermatitis: Therapeutic Management

*I. Poveda-Montoyo, MD*

*P. J. Álvarez-Chinchilla, MD*

*J. F. Silvestre, MD*

## Address

Dermatology Department, Instituto de Investigación Sanitaria y Biomédica de Alicante (ISABIAL), Hospital General Universitario de Alicante, Alicante, Spain  
Email: inespovedamontoyo@gmail.com

Published online: 25 January 2018

© Springer International Publishing AG, part of Springer Nature 2018

This article is part of the Topical Collection on *Contact Dermatitis*

**Keywords** Allergen avoidance · Allergic contact dermatitis · Allergic dermatitis · Therapeutic management · Treatment · Therapy · Management

## Abstract

**Purpose of Review** Allergic contact dermatitis (ACD) is a common condition and may entail negative impacts on people's quality of life along with a substantial economic burden. Appropriately managing it through patient education, allergen avoidance, and symptomatic treatment is crucial for its resolution. Our review aims to establish a practical approach to ACD management, based on a comprehensive discussion of available therapeutic options for ACD, a review of the latest findings in this field, and personal insights from our clinical practice experience.

**Recent Findings** Topical treatments constitute the first-line therapy for ACD. Randomized clinical trials have shown topical corticosteroids to improve clinical and nonclinical outcomes, while tacrolimus has proven effective and safe. Evidence is lacking for oral corticosteroids in ACD sufferers, so recommendations for its use are primarily based on results obtained in dermatitis of other etiologies. In contrast, some studies have assessed azathioprine and phototherapy for ACD.

**Summary** Patient education and allergen avoidance are paramount for managing ACD. However, individualized symptomatic treatment is warranted in several clinical situations. Although these treatments are widely used in clinical practice, few studies support their use for ACD. Further research is needed.

## Introduction

Allergic contact dermatitis (ACD) is a delayed hypersensitivity reaction to one or more chemical substances found in the patient's personal or work environment.

This commonly diagnosed entity accounts for approximately 20% of cases of contact dermatitis [1••], and is estimated to affect between 10 and 27% of the European

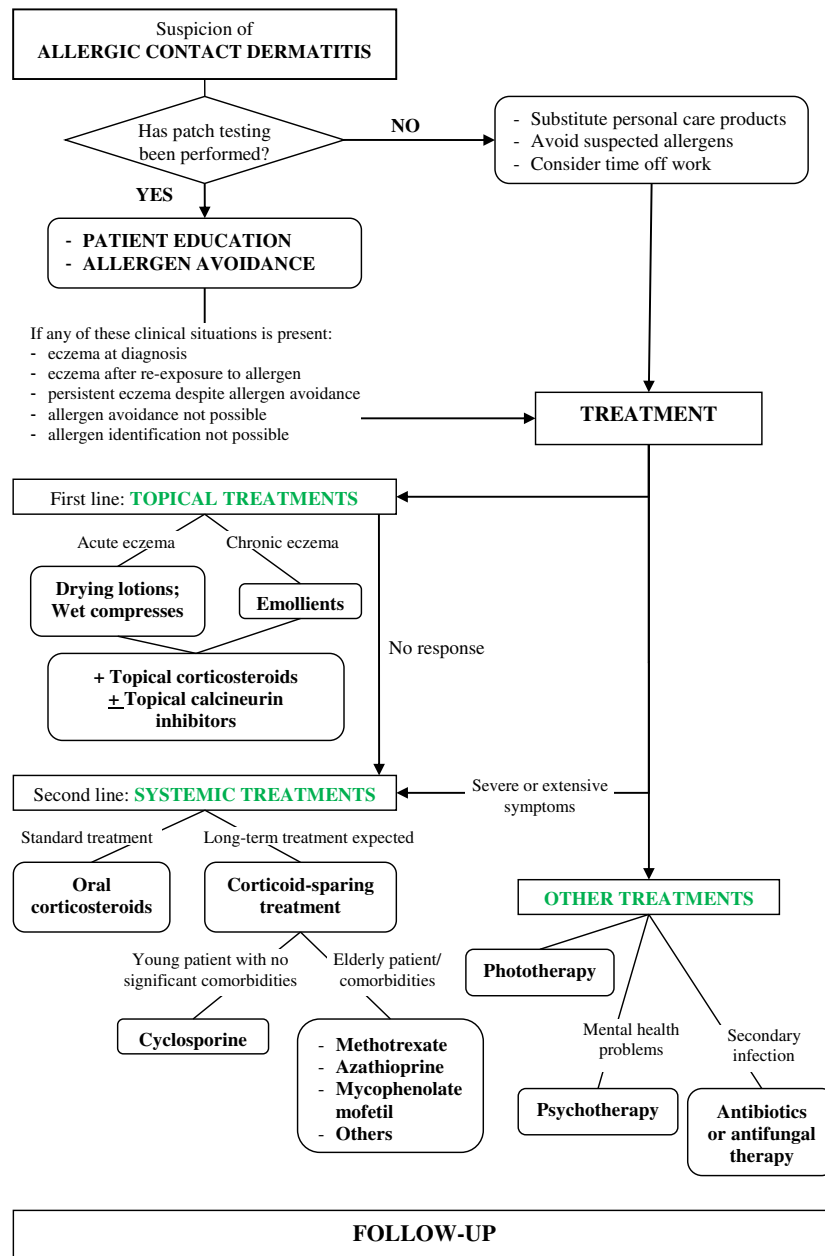


**Fig. 1.** Flowchart of therapeutic management of allergic contact dermatitis.

population [2]. Current incidence and prevalence rates are difficult to measure for two main reasons: firstly, sufferers sometimes self-diagnose and do not seek medical advice, and secondly, patients who access the health service do so through various points of care (e.g. emergency department, primary care service), without establishing a proper registry [3]. Nonetheless, studies have

shown that ACD is the most common occupational skin condition and the second most common occupational disease after musculoskeletal disorders [4, 5]. ACD can have a significant impact on sufferers' quality of life. Moreover, occupation ACD entails a substantial cost to mutual societies, insurance companies, and the national healthcare system.

ACD is sometimes clinically indistinguishable from other forms of dermatitis, such as irritant eczema, and correct diagnosis therefore requires patch testing [6]. Once the causative allergen is identified, correct ACD management can pose a real challenge for both patient and doctor. For the condition to resolve, the allergen must be removed from the patient's work and personal environment. Protective measures and treatments are also available for controlling symptoms, but cannot substitute avoidance measures [6]. Moreover, irritant contact dermatitis and atopic dermatitis often coexist with ACD, complicating the diagnostic and therapeutic management of the latter [7, 8].

Symptomatic treatment plays a key role in several clinical situations: for relieving symptoms in the period

between the initial patient assessment and patch testing; in concomitant use with avoidance measures to accelerate the healing process once the allergen has been identified; and, occasionally, to treat dermatitis that persists despite allergen avoidance, a clinical situation that has been reported in some patients with allergies to chromium, epoxy resins and *Compositae* [9••]. Treatment is also required when the allergen cannot be identified or completely avoided, or in the event of intermittent re-exposure resulting in cutaneous manifestations.

Comprehensive management of ACD should include patient education, allergen avoidance, and suitable treatment (Fig. 1). In this review, we aim to establish a practical approach to ACD management using the most recent published evidence.

## Patient education

Educating patients on the identified allergens is the first step in ACD management. Most allergen names are difficult for patients to remember: in one study, 50% of allergens identified through patch testing could not be recalled by sufferers [10]. Health professionals must therefore provide information orally and in writing, tailoring the language to patients' educational level. This information should include synonyms of the allergens, a brief description of their use, the places they can be found and the substances they may cross-react with and that should therefore be avoided [11•, 12•], as well as a list of products that are free of the causative allergen.

Follow-up visits are crucial and allow patients to become familiar with the allergens and put any questions they may have to the health professionals. Nursing staff could play a key role in this regard. Through follow-up, moreover, healthcare professionals can ensure patients are complying with avoidance measures and introduce or modify treatment as required [11•].

## Treatment of ACD

The most common clinical presentation of ACD is eczema, and symptomatic treatment is no different from that of other eczema types. The main goal of ACD management, however, is to isolate the patient from the causative allergen, and all other therapeutic efforts are usually insufficient if this is not achieved [13].

Dermatitis treatment should be specific to each patient. The type of drug used will depend primarily on eczema severity and extension and the response to previous treatment. Impact on quality of life should also be taken into account and there are scales available for this purpose, including the Dermatology Life Quality Index and the Hand Eczema Severity Index [9••]. These scales can be helpful in the initial patient assessment and during follow-up.

There are few double-blind randomized controlled trials on ACD treatment in the literature. Nonetheless, some therapeutic measures have proven effective

in eczema of various etiologies. Below we will describe the treatments available for people with allergic contact dermatitis.

## Topical treatments

Topical treatments constitute the first-line therapy for ACD. Therapeutic options should be distinguished according to the stage of the eczema. Acute lesions benefit from wet compresses or soaks with diluted drying agents such as saline, potassium permanganate, copper sulfate, aluminum sulfate or zinc sulfate. Calamine lotions may also help [13]. One study reported comparably good results with both saline and distilled water compresses [14]. These treatments involve applying compresses soaked in water-diluted drying agents to the affected areas for 10 to 30 min per day. The number of daily applications can be increased if necessary. However, to prevent excessive drying, compresses should not be used for more than recommend that patients then use emollient creams as a second phase of acute eczema treatment. Emollient creams help to restore the stratum corneum of the skin and thus prevent recurrence [15,16]. They also assist in the treatment of chronic eczema, as they prevent the appearance of cracks and fissures [13]. What makes these creams effective are their occlusive and humectant properties. Experts recommend using emollients that are free from fragrances and from the preservatives most commonly associated with skin sensitization [17,18]. One valid and inexpensive option is pure petroleum jelly.

Barrier creams are preparations formulated to protect the skin against irritants and allergens [13]. They create a protective layer on the skin and are therefore normally considered an allergen avoidance measure. However, due to their active ingredients (astringents, ultraviolet absorbers, and complex agents), they may also have some therapeutic efficacy. A more appropriate description for these preparations would therefore be protective creams [19••]. Results on the efficacy of barrier creams are controversial. In a study conducted in people with nickel-induced ACD, the authors analyzed the efficacy of a topical corticosteroid combined with a barrier cream versus three controls (no treatment, corticosteroid alone, and barrier cream alone). The participants who received the combination and those treated with barrier cream alone had better clinical and nonclinical outcomes than the other two control groups [20]. In other clinical trials, barrier cream efficacy was greatly influenced by patients' application technique [21]. People using barrier creams must be made aware that they are not a substitute for gloves to prevent any false sense of security.

Topical corticosteroids are the symptomatic treatment of choice for allergic eczema [1••, 22••], and according to the updated guidelines for the management of contact dermatitis, the use of topical corticosteroids in combination with emollients is the accepted treatment for established contact dermatitis [23]. The anti-inflammatory effect of corticosteroids in allergic dermatitis arises from their capacity to inhibit T cell activation and leukocyte migration [24]. They are also thought to have an important effect on macrophages and neutrophils [25]. In a systematic review of ACD treatment, Saary et al. found five randomized clinical trials evaluating the efficacy of corticosteroids. None of these trials included quality of life or return to work as an indicator of outcome [26]. Hachem et al. found that fluticasone propionate 0.05% improves clinical

and nonclinical outcomes compared to control treatment [27]. The same authors analyzed fluticasone propionate 0.05% in combination with a barrier cream versus three controls (no treatment, corticosteroid alone and barrier cream alone), finding that the combination therapy was associated with statistically significant improvement in various outcomes [20]. In another study, the use of clobetasone butyrate 0.05% resulted in significantly improved nonclinical outcomes compared to hydrocortisone 1% and no treatment [28]. Clinically favorable results have also been reported for clobetasol propionate 0.05% versus hydrocortisone 1% and topical anti-inflammatories (indomethacin 2.5% and bufexamac 5%) [29]. These four trials were conducted in patients with nickel-induced ACD. In the last of the studies included in the systematic review, clobetasol propionate 0.05% was shown to be more effective than petrolatum for treating dermatitis experimentally induced by urushiol [30].

Corticosteroid potency and length of treatment should depend on factors such as eczema severity and location [31]. High or very high potency corticosteroids should not be used on areas of thin skin such as the face or folds. On the palms and soles, however, a very high strength corticosteroid is required and can even be applied occlusively. Due to the side effects of corticosteroids, most notably skin atrophy, short courses are recommended. The choice of corticosteroid vehicle will depend on the morphology and stage of the eczema. Creams, lotions and gels are recommended for acute eczema, while ointments and salves are recommended for chronic eczema (except on the face, where creams are always preferable) [13, 19••]. With these considerations in mind, we recommend one or two daily applications of corticosteroids over 2 to 4 weeks.

Topical calcineurin inhibitors, only approved for atopic dermatitis, have been proven effective and safe in the treatment of ACD. Their mechanism of action is based on blocking T cells and inhibiting cytokine production [32]. They have very few side effects and can therefore be used for longer periods than corticosteroids, and in various locations, including areas of thin skin such as the face, folds, and genitals [22••]. For this reason, topical calcineurin inhibitors are commonly used in chronic or recurrent eczema to spare corticosteroids. Randomized clinical trials have demonstrated the efficacy and safety of tacrolimus 0.1% in people with nickel-induced ACD [33, 34]. Other studies have shown that the efficacy of this calcineurin inhibitor in ACD patients is similar to that of mometasone furoate 0.1% [35, 36]. In addition, tacrolimus 0.1% has been proven to be an effective and safe alternative therapy in eyelid ACD [37]. The results obtained with pimecrolimus, meanwhile, are controversial: while one study found an efficacy similar to that of betamethasone valerate 0.1% in nickel-induced ACD [38], another study found no statistically significant improvement with pimecrolimus 1% compared to the control treatment in poison ivy-induced ACD [39]. For long-term treatment, we recommend using topical calcineurin inhibitors, especially after topical corticosteroids in chronic eczema, to spare corticosteroids and prevent recurrence.

## Systemic treatments

If topical treatment is insufficient, systemic drugs may be required. The correct prescription of these drugs requires that the age, comorbidities and concomitant medication of the patient, as well as the possible adverse effects of treatment, should be taken into account [22••].

Systemic corticosteroids have a known effect on patch testing. Oral administration of prednisone in doses greater than 20 mg may prevent allergic reactions, resulting in false negatives [40]. These results have also been reported with 2 mg doses of betamethasone [41]. However, few studies have examined the efficacy of systemic corticosteroids in ACD sufferers. The recommendation of this treatment is primarily based on results obtained in dermatitis of various etiologies [42]. If ACD symptoms are very severe or very extensive, as with other types of eczema, experts recommend using oral corticosteroids such as prednisone at 0.5 to 1 mg/kg/day or equivalent. Oral corticosteroids can be taken in short courses of 5 to 7 days. If a longer duration is anticipated, tapering the administration can prevent recurrence and reduce the risk of adrenal insufficiency.

Corticosteroid-sparing immunosuppressants such as azathioprine, methotrexate, cyclosporin, and mycophenolate mofetil are other second-line options. The choice of drug will depend on the patient's clinical presentation and comorbidities [1••]. These treatments may also improve recalcitrant allergic hand eczema. Some studies have assessed the efficacy of azathioprine in ACD to parthenium: 150-mg daily doses improved lesions in almost all patients after 1 year of treatment [43], and 300-mg weekly pulse doses cured symptoms in 80 to 100% of patients after 6 months of treatment [44]. Studies with methotrexate, mycophenolate mofetil, and 5-fluorouracil showed inhibited hypersensitivity during the sensitization phase but no decrease in the inflammatory reaction during allergen challenge [45]. Although cyclosporine has proven useful in chronic hand eczema and atopic dermatitis, there are limited data in people with ACD [1••, 9••].

When patients have needed multiple short courses of oral corticosteroids, we recommend starting oral cyclosporine in young people with no contraindications and methotrexate in older people or people with contraindications, although the response to this drug is slower.

Retinoids are also effective in chronic hand eczema of various etiologies. Alitretinoin, which exerts an immunomodulating and anti-inflammatory effect, has proven safe and effective for chronic hand eczema resistant to topical treatment [46, 47]. Acitretin has also been effective for hyperkeratotic hand eczema [48, 49].

Anti-TNF biological drugs may be useful since their target, an inflammatory cytokine, is involved in ACD [50]. However, the results of studies on the effect of anti-TNF in this condition are inconsistent.

There are limited data on the efficacy of oral antihistamines in ACD. Some authors recommend using sedative antihistamines such as hydroxyzine to control pruritus in ACD sufferers. Another accepted regimen is the combination of selective and non-selective antihistamines, using the latter at night [13]. In our opinion, antihistamines do not provide adequate control of pruritus in people with ACD.

## Other treatments

Phototherapy with UVA, PUVA or UVB may be useful owing to the immunosuppressive effects it exerts through various mechanisms. Several studies have assessed the efficacy of PUVA and UVB therapy in chronic ACD. In one series, PUVA treatment resolved allergic hand eczema in all participants and UVB therapy produced a significant clinical improvement in most participants [51].

ACD, like other skin disorders, can provoke anxiety, depression and problems in social and work relationships. Psychotherapy may help to improve the quality of life of people suffering these effects [1••].

Various studies have attempted to induce tolerance to allergens through immunotherapy or gradual exposure to the allergen. These attempts have failed, and this approach is therefore not recommended in ACD patients [13, 22••].

### Treatment for complications of eczema

A relatively frequent complication of eczema is superinfection. The causative microorganisms can be bacteria, such as *Staphylococcus aureus* or *Streptococcus pyogenes*, or fungi, namely dermatophytes and yeasts. Bacterial superinfection may respond to topical combinations of corticosteroids and antibiotics [13]. If impetigo is extensive or does not respond to topical treatment, oral antibiotics are required: most commonly amoxicillin/clavulanic acid or cloxacillin. A topical antifungal agent can be applied to control localized mycological infections. People with severe infections or tinea incognito require systemic treatment with drugs such as azoles or terbinafine [13]. The recommended steps in this case, as in other infections, is taking a culture, starting empirical treatment and adjusting the antibiotic subsequently according to the antibiogram results. Neomycin and azole powders should be avoided due to their high sensitizing potential.

Another situation that may complicate ACD patient management is sensitization to the active ingredients or excipients (mainly preservatives and fragrances) of the topical preparations used. Topical administration of antihistamines, anti-inflammatories, analgesics and antibiotics such as neomycin should be avoided due to their known sensitizing capacity [6, 12•]. There has also been an increase in delayed hypersensitivity reactions to corticosteroids. The estimated prevalence of corticosteroid-induced ACD is 2.6% in Europe and 4.6% in the USA [52, 53]. Sensitization to corticosteroids should be suspected in patients who do not improve or who worsen despite correct treatment and allergen avoidance. In this case, patch testing should be repeated with the standard series and, if required, a corticosteroid series or product provided by the patient [54•]. According to Baeck et al., corticosteroids can be divided into three groups according to their structure and cross-reactivity, and delayed hypersensitivity reactions are most commonly to group 1. These authors also classify patients into two profiles depending on whether they are sensitized to one or more corticosteroid groups. Profile 1 includes people who suffer allergic reactions to a single group while profile 2 patients react to the whole spectrum of corticosteroids. It has been proposed that profile 1 patients avoid only the corticosteroids from the group to which they are sensitized, although the prescribed drug should always be tested beforehand. Profile 2 patients, meanwhile, should avoid corticosteroids altogether and seek other therapeutic alternatives [55–57]. We recommend managing ACD to corticosteroids with calcineurin inhibitors or with a topical corticosteroid that has been previously tested on the patient without provoking any allergic reaction.

## Work-related ACD

Occupational ACD is an entity that should be reviewed on a case-by-case basis due to the particular characteristics of its management strategy.

The latest data published by the North American Contact Dermatitis Patch Group show that 8.9% of patients with positive patch test results had occupational ACD [58•]. Moreover, work-related ACD accounts for up to 34% of occupational diseases registered in Europe [59•].

Due to the prevalence and considerable morbidity of occupational ACD, prevention is crucial. Prevention falls into three categories: primary prevention, which involves taking measures to prevent the condition from developing in healthy people; secondary prevention, which involves trying to prevent further flare-ups or new sensitizations in people already affected; and tertiary prevention, which focuses on reintegrating patients into their work and daily life [19••].

Patients with severe allergic eczema are highly recommended to adjust their tasks or take temporary sick leave to speed up recovery [60]. We recommend that patients with occupational eczema stay away from the workplace and receive treatment until they have fully recovered. The topical and systemic treatments used to relieve the symptoms of occupational eczema are similar to those used in other types of allergic contact dermatitis. Some patients may benefit from hospital admission until symptom resolution [61].

People with occupational ACD should resume work in the best possible conditions. This means the allergen must be identified and removed from the workplace. It may be necessary to contact the suppliers of the materials used by the worker to determine whether the allergen is present and to identify suitable replacement products [3]. If allergen avoidance is not possible, individual and collective protection measures can be taken.

The main individual preventive measures are goggles, masks, suitable clothing, gloves, and barrier creams [12•]. The correct type of gloves should be selected according to the allergen. It may also be helpful to wear cotton gloves below rubber gloves. Latex gloves offer good protection against aqueous substances but not against solvents, fats, and chemicals, and are therefore disadvised. Nitrile gloves protect against fats and solvents, while vinyl gloves offer better protection against most chemicals [13, 19••]. Overall, 4H laminated gloves, which are impermeable to more than 90% of organic chemicals when tested over 4 h at 35 °C [6], appear to offer the best chemical resistance.

Because workers sometimes report difficulties in performing their tasks while wearing gloves, barrier creams have been assessed for their protection against allergens. As mentioned above, barrier creams are not a substitute for gloves but may act as a complementary protective measure as well as providing some therapeutic effects.

Individual protection measures also apply to the home environment of both occupational and non-occupational ACD sufferers. The employer must also introduce collective protection measures. This is especially important if employees suffer from airborne contact dermatitis. Although individual and collective protection measures are applicable at all three levels of prevention, they are of greatest use at the primary level.

Patients with suspected occupational ACD may require assessment by an occupational medicine specialist, who can complement the avoidance measures recommended by other specialists and suggest changes in the patient's work station [60].

Nonetheless, various studies report that dermatitis symptoms persist in some people even when they avoid the allergen or change their work station.

Up to 70% of the patients included in one series had dermatitis for years despite avoiding chromium, and the authors reported higher chronicity where diagnosis was delayed [6, 62]. People who suffer recalcitrant eczema despite allergen avoidance require suitable symptomatic treatment.

## Avoidance measures for the most prevalent allergens

The North American Contact Dermatitis Group (NACDG) and the European Surveillance System of Contact Allergies (ESSCA) recently presented the results of patch testing in patients in North America and Europe, respectively. The five most prevalent allergens, in descending order, were nickel sulfate, fragrance mix I, methylisothiazolinone (MI) and methylchloroisothiazolinone/methylisothiazolinone (MCI/MI), cobalt chloride and *Myroxylon pereirae* (balsam of Peru). Other common allergens identified in these series were paraphenylenediamine (PPD), fragrance mix II, neomycin sulfate, bacitracin, potassium dichromate, formaldehyde, and colophony [58, 63•].

The metals that most frequently cause ACD are nickel and cobalt. Patients should avoid metal utensils, nail varnish and eyelid cosmetics that contain these allergens. It may be helpful to perform the dimethylglyoxime test, available in pharmacies, to identify products that contain nickel. Chromium is another metal that provokes ACD relatively frequently. It may be present in metal utensils, leather goods, cement and paints. However, the main challenge posed by metal allergies is related to joint prostheses and dental amalgams and prostheses. Sensitized patients should not have medical devices containing these metals implanted [11•]. Problems with an implanted device may be resolved by its removal, provided the clinical features are compatible and patch testing is positive.

Fragrances are common allergens often found in cosmetic, hygiene or cleaning products; pharmaceutical preparations; perfumes; and air fresheners. Balsam of Peru is a fragrance that can cause multiple cross-reactions, all of which should be taken into account [64,65]. The labeling of these products may be confusing or misleading and the small print size is often difficult to read. The terms “fragrances,” “perfume,” “colognes,” or “essential oils” are sometimes used without specification of the components. Furthermore, some products containing these allergens are wrongly labeled “perfume free,” “fragrance free,” or “hypoallergenic” [66]. We recommend completing the study with the specific series of fragrances so the patient can try to avoid only the fragrance to which they are allergic, although recommending fragrance-free products is also a valid option.

Preservatives such as methylchloroisothiazolinone (MCI), methylisothiazolinone (MI), methylchloroisothiazolinone/methylisothiazolinone (MCI/MI), formaldehyde, and formaldehyde releasers can be found in hygiene, cosmetic and cleaning products. MCI/MI is also known in practice as Kathon. Although this name is easier to remember, patients should be warned that the preservative will be listed on labels as methylchloroisothiazolinone/methylisothiazolinone. People allergic to MCI/MI should also avoid MI and vice versa [67]. Formaldehyde and formaldehyde releasers are also used as anti-crease substances in clothes, which means new clothes can cause problems in sufferers. Many manufacturers have replaced formaldehyde with formaldehyde releasers. The main formaldehyde releasers are quaternium-15,

diazolidinyl urea, DMDM hydantoin, imidazolidinyl urea and 2-bromo-2-nitropropane-1,3-diol. In addition, people with positive patch tests for formaldehyde should avoid formaldehyde releasers and vice versa [66, 68].

We recommend that people with suspected ACD awaiting patch testing for allergen identification replace their usual hygiene products with products that are free from fragrances and common preservatives. If the patient has eczema at the time of the consultation, a suitable treatment should also be prescribed to relieve symptoms.

Topical antibiotics such as neomycin and bacitracin can be found in ointments, creams and eye, nose, and ear drops. Sensitized people should avoid these allergens. Neomycin may also cross-react with other aminoglycosides [6, 12•].

Paraphenylenediamine (PPD) is present in permanent hair dyes, temporary black henna tattoos, and in some textile and shoe dyes. Vegetable or semipermanent dyes (4-amino-paraphenylenediamine) are derivatives of PPD and can also cause problems in sensitized people. Metallic dyes (Grecian formula) or henna dyes are safe alternatives. Patients should be made aware that PPD can cross-react with PABA sunscreens, ester local anesthetics, black rubber and drugs such as sulfonamides, thiazide diuretics and sulfonylureas [68].

Topical NSAIDs and the ultraviolet filters contained in sunscreens are the agents most commonly associated with photoallergic contact dermatitis [69•, 70]. This is a delayed hypersensitivity reaction caused by contact between the skin and an exogenous agent in the presence of ultraviolet or visible light. Avoiding the sun is a crucial factor in the management of this condition. For this reason, suitable clothing and sun cream are necessary protective measures, provided they are not the cause of the photoallergic contact dermatitis.

## Conclusions

The prevalence of ACD in the general population and in the workplace is high, although current rates are difficult to estimate. Its clinical manifestations can be indistinguishable from those of other types of dermatitis, which is why patch testing is essential for a correct diagnosis. Management of ACD is based on three key areas: patient education, allergen avoidance and treatment of symptoms.

Health professionals must participate proactively in patient education, transmitting information in a clear way that is tailored to each patient. Follow-up is essential to patient management.

For symptoms to improve, patients must avoid the allergen in their personal and working environment.

Various treatments are available to control symptoms in allergic eczema. Although these treatments are widely used in clinical practice, few studies have been conducted in ACD sufferers. The topical treatments that have shown the greatest efficacy are corticosteroids and calcineurin inhibitors. Barrier creams may be helpful although they are not a substitute for gloves. Systemic treatments, due to their potential side effects, are reserved for patients with severe or extensive symptoms that do not respond to topical treatment. As in all types of eczema, complications may occur and should be managed appropriately. Topical or oral antibiotics are recommended for treating infections. Allergic patients may develop new sensitizations, which should be confirmed with

further patch tests. Health professionals should bear in mind the increased incidence of ACD to topical corticosteroids.

In occupational ACD, primary, secondary, and tertiary prevention are essential, and individual and collective prevention measures must be taken.

Adequately controlling ACD is a challenge for patients and health professionals, and a comprehensive management approach is therefore crucial.

## Compliance with Ethical Standards

### Conflict of Interest

Inés Poveda-Montoyo declares that he has no conflict of interest. P. J. Álvarez-Chinchilla declares that he has no conflict of interest. J. F. Silvestre declares that he has no conflict of interest.

### Human and Animal Rights

This article does not contain any studies with human or animal subjects performed by any of the authors.

## References and Recommended Reading

Papers of particular interest, published recently, have been highlighted as:

- Of importance
- Of major importance

1. •• Jacob SE, Castanedo-Tardan MP. Pharmacotherapy for allergic contact dermatitis. *Expert Opin Pharmacother*. 2007;8:2757–74.
- A comprehensive review of the symptomatic and immunosuppressive therapy available in allergic contact dermatitis.
2. Diepgen TL, Ofenloch RF, Bruze M, Bertuccio P, Cazzaniga S, Coenraads PJ, et al. Prevalence of contact allergy in the general population in different European regions. *Br J Dermatol*. 2016;174:319–29.
3. Mowad CM, Anderson B, Scheinman P, Pootongkam S, Nedorost S, Brod B. Allergic contact dermatitis. *J Am Acad Dermatol*. 2016;74:1043–54.
4. Mark BJ, Slavin RG. Allergic contact dermatitis. *Med Clin N Am*. 2006;90:169–85.
5. Behrens V, Seligman P, Cameron L, Mathias CGT, Fine L. The prevalence of back pain, hand discomfort, and dermatitis in the US working population. *Am J Public Health*. 1998;84:1780–5.
6. Belsito DV. The diagnostic evaluation, treatment, and prevention of allergic contact dermatitis in the new millennium. *J Allergy Clin Immunol*. 2000;105:409–20.
7. Rundle CW, Bergman D, Goldenberg A, Jacob SE. Contact dermatitis considerations in atopic dermatitis. *Clin Dermatol*. 2017;35:367–74.
8. Nosbaum A, Vocanson M, Rozieres A, Hennino A, Nicolas JF. Allergic and irritant contact dermatitis. *Eur J Dermatol*. 2009;19:325–32.
9. •• Johnston GA, Exton LS, Mohd Mustapa MF, Slack JA, Coulson IH, English JS, et al. British Association of Dermatologists' guidelines for the management of contact dermatitis. *Br J Dermatol*. 2017;176:317–29.
- Guidelines providing up-to-date and evidence-based recommendations for the management of contact dermatitis.
10. Scalf LA, Genebriera J, Davis MDP, Farmer SA, Yiannias JA. Patients's perceptions of the usefulness and outcome of patch testing. *J Am Acad Dermatol*. 2007;56:928–32.
11. • Mowad CM. Contact dermatitis. Practice gaps and challenges. *Dermatol Clin*. 2016;34:263–7.
- Elaborate manuscript discussing the most frequent gaps and challenges in contact dermatitis.
12. • Bernstein DI. Contact dermatitis for the practicing allergist. *J Allergy Clin Immunol Pract*. 2015;3:652–8.
- Current overview of the diagnosis and treatment of contact dermatitis.
13. González de Domingo MA, Conde-Salazar Gomez L: Tratamiento de las dermatosis laborales. In: Conde-Salazar L, Ancona-Alayón A editors. *Dermatología profesional*. Aula Médica;2004.
14. Levin CY, Maibach HI. Do cool water or physiologic saline compresses enhance resolution of experimentally-induced irritant contact dermatitis? *Contact Dermatitis*. 2001;45:146–50.
15. Brown T. Strategies for prevention: Occupational contact dermatitis. *Occup Med Oxf Engl*. 2004;54:450–7.

16. Ramsing DW, Agner T. Preventive and therapeutic effects of a moisturizer. An experimental study of human skin. *Acta Derm Venereol.* 1997;77:335–7.
  17. Menné T, Johansen JD, Sommerlund M, Veien NK, Danish Contact Dermatitis Group. Hand eczema guidelines based on the Danish guidelines for the diagnosis and treatment of hand eczema. *Contact Dermatitis.* 2011;65:3–12.
  18. Diepgen TL, Elsner P, Schliemann S, Fartasch M, Köllner A, Skudlik C, et al. Guideline on the management of hand eczema ICD-10 Code: L20. L23. L24. L25. L30. *J Dtsch Dermatol Ges J Ger Soc Dermatol JDDG.* 2009;7(Suppl 3):S1–16.
  - 19.●● De León FJ, Berbegal L, Silvestre JF. Abordaje terapéutico en el eczema crónico de manos. *Actas Dermosifilogr.* 2015;106:533–44.
- A thorough review of the management of chronic hand eczema, including the allergic etiology. Practical recommendations about prevention and treatment are provided in this manuscript.
20. Hachem JP, De Paepe K, Vanpee E, Kaufman L, Rogiers V, Roseeuw D. Combination therapy improves the recovery of the skin barrier function: an experimental model using a contact allergy patch test combined with TEWL measurements. *Dermatology.* 2001;202:314–9.
  21. Wigger-Alberti W, Maraffio B, Wernli M, Elsner P. Self applications of a protective cream. Pitfalls of occupational skin protection. *Arch Dermatol.* 1997;133:861–4.
  - 22.●● Brasch J, Becker D, Aberer W, Bircher A, Kränke B, Jung K, et al. Guideline contact dermatitis. S1-Guideline of the German Contact Allergy Group (DKG) of the German Dermatology Society (DDG), the Information Network of Dermatological Clinics (IVDK), the German Society for Allergology and Clinical Immunology (DGAKI), the Working Group for Occupational and Environmental Dermatology (ABD) of the DDG, the Medical Association of German Allergologists (AeDA), the Professional Association of German Dermatologists (BVDD) and the DDG. *Allergo J Int.* 2014;23:126–38.
- Guidelines describing the diagnosis and therapeutic and interventional approaches of contact dermatitis based on the latest scientific evidence.
23. Bourke J, Coulson I, English J, British Association of Dermatologists Therapy Guidelines and Audit Subcommittee. Guidelines for the management of contact dermatitis: an update. *Br J Dermatol.* 2009;160:946–54.
  24. Cohen DE, Heidary N. Treatment of irritant and allergic contact dermatitis. *Dermatol Ther.* 2004;17:334–40.
  25. Tuckermann JP, Kleiman A, Moriggl R, Spanbroek R, Neumann A, Illing A, et al. Macrophages and neutrophils are the targets for immune suppression by glucocorticoids in contact allergy. *J Clin Invest.* 2007;117:1381–90.
  26. Saary J, Qureshi R, Palda V, DeKoven J, Pratt M, Skotnicki-Grant S, et al. A systematic review of contact dermatitis treatment and prevention. *J Am Acad Dermatol.* 2005;53:845–55.
  27. Hachem JP, De Paepe K, Vanpee E, Bogaerts M, Kaufman L, Rogiers V, et al. Efficacy of topical corticosteroids in nickel-induced contact allergy. *Clin Exp Dermatol.* 2002;27:47–50.
  28. Parneix-Spake A, Goustas P, Green R. Eumovate (clobetasone butyrate) 0.05% cream with its moisturizing emollient base has better healing properties than hydrocortisone 1% cream: a study in nickel-induced contact dermatitis. *J Dermatol Treat.* 2001;12:191–7.
  29. Quielle-Roussel C, Duteil L, Padilla JM, Poncet M, Czernielewski J. Objective assessment of topical anti-inflammatory drug activity on experimentally induced nickel contact dermatitis: comparison between visual scoring, colorimetry, laser Doppler velocimetry and transepidermal water loss. *Skin Pharmacol.* 1990;3:248–55.
  30. Vernon HJ, Olsen EA. A controlled trial of clobetasol propionate ointment 0.05% in the treatment of experimentally induced Rhus dermatitis. *J Am Acad Dermatol.* 1990;23:829–32.
  31. Drake LA, Dinehart SM, Farmer ER, Goltz RW, Graham GF, Hordinsky MK, et al. American Academy of Dermatology. Guidelines of care for the use of topical glucocorticosteroids. *J Am Acad Dermatol.* 1996;35:615–9.
  32. Bornhøvd E, Burgdorf WH, Wollenberg A. Macrolactam immunomodulators for topical treatment of inflammatory skin diseases. *J Am Acad Dermatol.* 2001;45:736–43.
  33. Belsito DV, Wilson DC, Warshaw E, Fowler J, Ehrlich A, Anderson B, et al. A prospective randomized clinical trial of 0.1% tacrolimus ointment in a model of chronic allergic contact dermatitis. *J Am Acad Dermatol.* 2006;55:40–6.
  34. Saripalli YV, Gadzia JE, Belsito DV. Tacrolimus ointment 0.1% in the treatment of nickel-induced allergic contact dermatitis. *J Am Acad Dermatol.* 2003;49:477–82.
  35. Alomar A, Puig L, Gallardo CM, Valenzuela N. Topical tacrolimus 0.1% ointment (protopic) reverses nickel contact dermatitis elicited by allergen challenge to a similar degree to mometasone furoate 0.1% with greater suppression of late erythema. *Contact Dermatitis.* 2003;49:185–8.
  36. Katsarou A, Makris M, Papagiannaki K, Lagogianni E, Tagka A, Kalogeromitros D. Tacrolimus 0.1% vs mometasone furoate topical treatment in allergic contact hand eczema: a prospective randomized clinical study. *Eur J Dermatol.* 2012;22:192–6. <https://doi.org/10.1684/ejd.2011.1615>.
  37. Saripalli YV, Gadzia JE, Belsito DV. Tacrolimus ointment 0.1% in the treatment of allergic contact eyelid dermatitis. *J Eur Acad Dermatol Venerol.* 2009;23:382–7. <https://doi.org/10.1111/j.1468-3083.2008.03056>.
  38. Gupta AK, Chow M. Pimecrolimus: a review. *J Eur Acad Dermatol Venereol.* 2003;17:500.

39. Amrol D, Keitel D, Hagaman D, Murray J. Topical pimecrolimus in the treatment of human allergic contact dermatitis. *Ann Allergy Asthma Immunol*. 2003;91:563–6.
  40. Anveden I, Lindberg M, Andersen KE, Bruze M, Issakson M, Liden C, et al. Oral prednisone suppresses allergic but not irritant patch test reactions in individuals hypersensitive to nickel. *Contact Dermatitis*. 2004;50:298–303.
  41. Verma KK, Mahesh R, Bhari N, Pandey RM. Effect of betamethasone on patch test reactivity in patients with parthenium dermatitis. *Contact Dermatitis*. 2016;75:193–4.
  42. Rietschel RL, Fowler JF. Fisher's contact dermatitis. Lippincott Williams Wilkins. Philadelphia. 2001:715–21.
  43. Sharma VK, Chakrabarti A, Mahajan V. Azathioprine in the treatment of parthenium dermatitis. *Int J Dermatol*. 1998;37:299.
  44. Verma KK, Bansal A, Sethuraman G. Parthenium dermatitis treated with azathioprine weekly pulse doses. *Indian J Dermatol Venereol Leprol*. 2006;72:24–7.
  45. Quéméneur L, Michallet MC, Ferraro-Peyret C, Saint-Mézard P, Benetière J, Ducluzeau MT, et al. Immunosuppressive antimetabolites inhibit induction of contact hypersensitivity while lymphoablative drugs also prevent its expression. *Eur J Dermatol*. 2003;13:540–7.
  46. Schindler M, Drozdenko G, Kühl AA, Worm M. Immunomodulation in patients with chronic hand eczema treated with oral alitretinoin. *Int Arch Allergy Immunol*. 2014;165:18–26.
  47. Ruzicka T, Lynde CW, Jemec GBE, Diepgen T, Berth-Jones J, Coenraads PJ, et al. Efficacy and safety of oral alitretinoin (9-cis retinoic acid) in patients with severe chronic hand eczema refractory to topical corticosteroids: Results of a randomized, double-blind, placebo-controlled, multicentre trial. *Br J Dermatol*. 2008;158:808–17.
  48. Thestrup-Pedersen K, Andersen KE, Menné T, Veien NK. Treatment of hyperkeratotic dermatitis of the palms (eczema keratoticum) with oral acitretin. A single-blind placebo-controlled study. *Acta Derm Venereol*. 2001;81:353–5.
  49. Capella GL, Fracchiolla C, Frigerio E, Altomare G. A controlled study of comparative efficacy of oral retinoids and topical betamethasone/salicylic acid for chronic hyperkeratotic palmoplantar dermatitis. *J Dermatol Treat*. 2004;15:88–93.
  50. Piguet PF, Grau GE, Hauser C, Vassalli P. TNF is a critical mediator in hapten-induced irritant and contact hypersensitivity reactions. *J Exp Med*. 1991;173:673–9.
  51. Rosen K, Mobacken H, Swanbeck G. Chronic eczematous dermatitis of the hands. A comparison of PUVA and UVB treatment. *Acta Derm Venereol*. 1987;67:48–54.
  52. Dooms-Goossens A, Andersen KE, Brandao FM, Bruynzeel D, Burrows D, Camarasa J, et al. Corticosteroid contact allergy: An EECDRG multicentre study. *Contact Dermatitis*. 1996;35:40–4.
  53. Jacob SE, Steele T. Corticosteroid classes: A quick reference guide including patch test substances and cross-reactivity. *J Am Acad Dermatol*. 2006;54:723–7.
  54. Berbegal L, DeLeón FJ, Silvestre JF. Hypersensitivity reactions to corticosteroids. *Actas Dermosifiliogr*. 2015;106:816–22.
- Useful manuscript detailing patients' profiles and management of corticosteroids allergy, a potential complication present in allergic contact dermatitis.
55. Baeck M, Chamelle JA, Goossens A, Nicolas JF, Terreux R. Corticosteroid cross-reactivity: Clinical and molecular modelling tools. *Allergy*. 2011;66:1367–74.
  56. Baeck M, Goossens A. Immediate, delayed allergic hypersensitivity to corticosteroids: Practical guidelines. *Contact Dermatitis*. 2011;66:38–45.
  57. Baeck M, Chemelle JA, Rasse C, Terreux R, Goossens A. C(16)-methyl corticosteroids are far less allergenic than the non-methylated molecules. *Contact Dermatitis*. 2011;64:305–12.
  58. DeKoven JG, Warshaw EM, Belsito DV, Sasseville D, Maibach HI, Taylor JS, et al. North American Contact Dermatitis Group Patch Test Results 2013–2014. *Dermatitis*. 2017;28:33–46.
- Study showing the latest patch testing results in North America. This manuscript identifies the most prevalent allergens and also presents the rate of occupational contact dermatitis.
59. Pesonen M, Jolanki R, Larese Filon F, Wilkinson M, Kręcis B, Kieć-Swierczyńska M, et al. Patch test results of the European baseline series among patients with occupational contact dermatitis across Europe - analyses of the European Surveillance System on Contact Allergy network, 2002–2010. *Contact Dermatitis*. 2015;72:154–63.
- Study presenting the latest results of prevalence and most frequent allergens in occupational allergic contact dermatitis in European population.
60. Smedley J, on behalf of the OHCEU and BOHRF Dermatitis guideline development groups. Concise guidance: diagnosis, management and prevention of occupational contact dermatitis. *Clin Med*. 2010;10:487–90.
  61. Weisshaar E, Skudlik C, Scheidt R, Mattered U, Wulfhorst B, Schönfeld M, et al. Multicentre study "rehabilitation of occupational skin diseases - optimization and quality assurance of inpatient management (ROQ)" - Results from 12-month follow-up. *Contact Dermatitis*. 2013;68:169–74.
  62. Pryce DW, Irvine D, English JS, Rycroft RJ. Soluble oil dermatitis: a follow-up study. *Contact Dermatitis*. 1989;21:28–38.
  63. Uter W, Amario-Hita JC, Balato A, Ballmer-Weber B, Bauer A, Belloni-Fortina A, et al. European Surveillance System on Contact Allergies (ESSCA): results with the European baseline series, 2013/14. *J Eur Acad Dermatol Venereol*. 2017;31:1516–1525.
- A recent study presenting the most prevalent allergens after patch testing in European population.

64. Arribas MP, Soro P, Silvestre JF. Allergic Contact Dermatitis to Fragrances. Part 1. *Actas Dermosifiliogr.* 2012;103:874–9.
65. Arribas MP, Soro P, Silvestre JF. Allergic contact dermatitis to fragrances: part 2. *Actas Dermosifiliogr.* 2013;104:29–37.
66. Nelson SA, Yiannias JA. Relevance and avoidance of skin-care product allergens: pearls and pitfalls. *Dermatol Clin.* 2009;27:329–36.
67. Leiva-Salinas M, Francés L, Silvestre JF. Update on allergic contact dermatitis due to methylchloroisothiazolinone/methylisothiazolinone and methylisohiazolinone. *Actas Dermosifiliogr.* 2014;105:840–6.
68. Castanedo-Tardan MP, Zug KA. Patterns of cosmetic contact allergy. *Dermatol Clin.* 2009;27:265–80.
69. • Consuegra-Romero G, Castro-Gutiérrez B, González-López MA. Photoallergic contact dermatitis. *Eur J Intern Med.* 2017;38:e4–5.
- Interesting case report and review of photoallergic contact dermatitis, a special type of allergic contact dermatitis that should be taken into account
70. Kerr A, Ferguson J. Photoallergic contact dermatitis. *Photodermatol Photoimmunol Photomed.* 2010;26:56–65.