

ALLERGIC CONTACT DERMATITIS AS A CLINICAL AND OCCUPATIONAL PROBLEM DIAGNOSIS PATCH TESTING AND PREVENTION

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Abstract

Allergic contact dermatitis is a common T-cell-mediated inflammatory skin disease caused by repeated exposure to contact allergens in sensitized individuals. Its clinical appearance often overlaps with irritant contact dermatitis, atopic dermatitis, psoriasis, fungal infection, and other eczematous disorders, which contributes to delayed recognition and persistent exposure. This review thesis summarizes current evidence on pathogenesis, clinical patterns, diagnostic patch testing, major allergens, treatment, and occupational prevention. Hapten penetration through an impaired epidermal barrier activates keratinocytes and innate immune pathways, followed by antigen presentation and allergen-specific T-cell sensitization. Re-exposure produces an elicitation phase characterized by pruritus, erythema, edema, papules, vesicles, and, in chronic disease, scaling, fissuring, and lichenification. Patch testing remains the reference diagnostic method, but a positive reaction proves contact allergy rather than clinical relevance; diagnosis requires correlation with the distribution of dermatitis and verified exposure. Successful management depends primarily on identification and avoidance of the responsible allergen, supported by barrier repair, appropriate anti-inflammatory therapy, patient education, and workplace modification. A structured exposure history and timely patch testing can reduce chronicity, improve quality of life, and prevent unnecessary treatment escalation.

Keywords: allergic contact dermatitis; contact allergy; patch testing; haptens; occupational dermatitis; allergen avoidance; skin barrier

Background

Allergic contact dermatitis (ACD) is an acquired delayed hypersensitivity reaction that develops after skin contact with a substance capable of inducing specific immune sensitization. Metals, fragrances, preservatives, rubber accelerators, hair-dye chemicals, topical medicines, adhesives, and acrylates are among the most important allergen groups. Exposure occurs through cosmetics, jewelry, clothing, household products, medical devices, topical preparations, and occupational materials. Because the same eczematous pattern can result from irritation or from endogenous inflammatory skin disease, the etiologic diagnosis cannot be made reliably from morphology alone [1-3].

The burden of ACD extends beyond visible inflammation. Pruritus, pain, fissuring, sleep disturbance, embarrassment, impaired hand function, and repeated medical visits can reduce quality of life. Occupational disease may cause presenteeism, absence from work, job modification, or loss of employment. Recent evidence also indicates clinically relevant psychosocial and financial consequences, particularly when dermatitis is chronic, affects the hands or face, or continues because the allergen remains unidentified [4,5]. These outcomes make causal investigation as important as short-term suppression of inflammation.

Aim

The aim of this thesis was to summarize current evidence on the immunopathogenesis, clinical recognition, differential diagnosis, patch-test evaluation, treatment, and prevention of allergic contact dermatitis, with particular attention to occupational exposure and the practical interpretation of allergen relevance.

Materials and Methods

A targeted narrative search of PubMed, guideline repositories, and the reference lists of recent reviews was conducted for English-language publications. Search terms included allergic contact dermatitis, contact allergy, patch testing, delayed hypersensitivity, occupational dermatitis, major allergens, avoidance, treatment, prevention, and quality of life. Priority was given to the 2026 European Society of Contact Dermatitis patch-testing guideline, international clinical reviews, systematic reviews, meta-analyses, surveillance studies, and publications with direct implications for clinical practice. Evidence was synthesized narratively because definitions, populations, allergen series, exposure patterns, and outcomes differed across studies.

Immunopathogenesis and Skin Barrier Dysfunction

Most contact allergens are low-molecular-weight chemicals called haptens. They become immunogenic after penetrating the stratum corneum and binding to cutaneous proteins. Barrier disruption caused by wet work, solvents, detergents, friction, inflammation, or atopic skin facilitates penetration. Keratinocyte stress and innate immune activation lead to the release of danger signals, cytokines, and chemokines. Dendritic cells take up hapten-protein complexes, migrate to regional lymph nodes, and present antigen to naive T lymphocytes. Clonal expansion then establishes allergen-specific effector and memory T cells. This clinically silent sensitization phase may follow repeated low-dose exposure or a shorter period of intense exposure [6,7]. During the elicitation phase, renewed contact with the allergen activates resident memory and circulating T cells. Cytotoxic and inflammatory pathways recruit additional immune cells and produce spongiosis and epidermal injury. The classical classification as a type IV delayed reaction remains useful, but current models

recognize substantial heterogeneity involving CD8-positive and CD4-positive T cells, Th1, Th2, Th17, and Th22-associated responses, depending on the allergen and host. Innate pathways, neuroimmune signaling, and the condition of the epidermal barrier influence the intensity and persistence of inflammation [6,8].

Sensitization is generally long-lasting. Therefore, therapy cannot be expected to eliminate immunologic memory. Clinical control depends on reducing exposure below the individual elicitation threshold and restoring the barrier. This explains why apparently minor, intermittent, or indirect exposure may maintain disease and why treatment alone often fails when the causative substance remains present.

Clinical Patterns and Differential Diagnosis

Acute ACD commonly presents with intense pruritus, erythema, edema, papules, vesicles, and sometimes bullae or exudation. Chronic exposure produces dryness, scaling, hyperkeratosis, fissures, and lichenification. The distribution provides an exposure map. Eyelid dermatitis may reflect cosmetics, nail products transferred by the fingers, ophthalmic preparations, or airborne allergens. Facial and neck dermatitis can follow fragrance, preservative, hair-dye, or plant exposure. Earlobe, wrist, and periumbilical eczema suggest metal contact. Hand disease may be associated with wet work, gloves, disinfectants, resins, cutting fluids, plants, foods, or repeated use of personal-care products [2,3].

A diagnostic distinction must be made between allergic and irritant contact dermatitis. Irritant disease results from direct toxic injury and can occur in any person when exposure is sufficient; ACD requires previous sensitization and can be elicited by a much smaller dose. In practice, both mechanisms frequently coexist. Atopic dermatitis, seborrheic dermatitis, psoriasis, tinea, scabies, photodermatitis, cutaneous drug reactions, and contact urticaria must also be considered. The EADV practical review emphasizes integrating morphology, anatomical distribution, time course, exposure history, treatment response, and targeted testing rather than relying on a single clinical feature [9].

Warning signs for ACD include dermatitis that is localized to a contact area, sharply worsens after a new product, improves away from work, recurs despite appropriate therapy, begins on the eyelids, face, hands, feet, or perianal region, or develops after a medical device or topical medication is introduced. Systemic contact dermatitis is less common but may occur in sensitized individuals after systemic exposure to a relevant allergen.

Patch Testing and Assessment of Clinical Relevance

Patch testing is the reference method for diagnosing delayed contact allergy. Standardized allergens are applied under occlusion to uninvolved skin, usually on the

upper back. The chambers are removed according to the protocol, and reactions are read on more than one occasion because allergic responses may evolve after removal. The 2026 European Society of Contact Dermatitis update reinforces standardized allergen preparation, careful application, trained reading, documentation, and interpretation in the context of exposure [1]. Patch testing should not be confused with skin-prick testing, which evaluates immediate IgE-mediated allergy and does not diagnose ACD.

Patient selection is critical. Testing is appropriate for persistent, recurrent, severe, occupational, hand, facial, eyelid, anogenital, or treatment-resistant dermatitis and when allergy to topical medicines, cosmetics, devices, or protective equipment is suspected. A baseline series detects common allergens, but additional occupation-specific, cosmetic, dental, hairdressing, rubber, adhesive, acrylate, or medication series may be required. Testing the patient's own products can be useful only when concentration and vehicle are safe and validated; inappropriate testing of irritant products can produce false-positive reactions.

A positive patch test demonstrates sensitization, not automatically the cause of the current dermatitis. Clinical relevance is established by confirming actual exposure, compatibility between the allergen and product ingredients, anatomical distribution, timing, and improvement after avoidance. Relevance may be current, past, or unknown. False-negative results may occur when the responsible allergen is not included, the concentration is inadequate, immunosuppressive treatment suppresses reactivity, ultraviolet exposure alters the test site, or readings are incomplete. Irritant reactions, active dermatitis, and excessive concentrations can lead to false-positive interpretation [1,10].

The diagnostic process continues after the test. Ingredient labels, safety data sheets, workplace materials, hobbies, medical devices, and indirect exposure from family members or contaminated objects should be reviewed. Clear written information should include allergen synonyms, cross-reactors, typical sources, and a practical list of safe alternatives. Without this translation from a test result to daily behavior, even technically correct patch testing may have limited clinical value.

Major and Emerging Contact Allergens

Nickel remains an important cause of contact allergy and is encountered in jewelry, clothing fasteners, tools, mobile devices, and occupational objects. Cobalt and chromium may accompany metal or cement exposure. Fragrance mixtures and individual fragrance substances occur in perfumes, cosmetics, cleansers, topical medicines, and products marketed as natural. Preservatives such as isothiazolinones and formaldehyde-releasing compounds can be present in personal-care products and

industrial materials. Rubber accelerators in gloves and footwear, paraphenylenediamine in oxidative hair dyes, and topical antibiotics are other established causes [2,3,10].

Changing technology creates new exposure patterns. Methacrylates and acrylates are relevant in artificial nails, dental materials, adhesives, hearing aids, wound closure systems, and glucose-monitoring or insulin-delivery devices. Epoxy resins remain important in construction, painting, manufacturing, and recreational crafts. Oxidized limonene and linalool hydroperoxides may sensitize users of fragranced products. Surveillance data show that allergen frequency varies by geography, regulation, occupation, age, and product use; therefore, the test series must be adapted to the patient's environment rather than treated as universal [10,11].

Treatment and Prevention

Complete or maximal feasible allergen avoidance is the central intervention. Patients should receive specific counseling rather than a general instruction to avoid chemicals. Product labels must be checked for exact ingredient names, and replacement products should be selected using reliable allergen databases or verified manufacturer information. In occupational disease, substitution of the allergen, enclosure of the process, improved ventilation, reduction of wet work, task rotation, correct glove selection, and safe work practices are preferred to relying only on personal protective equipment [4,5].

Barrier recovery requires gentle cleansing, lukewarm water, avoidance of unnecessary solvents and fragranced products, and frequent use of a bland emollient. During active localized disease, a topical corticosteroid of suitable potency, vehicle, anatomical location, and duration is commonly used. Topical calcineurin inhibitors may be useful on sensitive sites or when a steroid-sparing approach is needed. Secondary infection, extensive blistering, severe facial involvement, erythroderma, or occupational disability requires medical reassessment. Systemic corticosteroids may be used briefly for severe widespread episodes, but rebound and adverse effects limit routine use. Chronic refractory disease warrants specialist review and reconsideration of diagnosis, exposure, adherence, infection, and coexisting atopic or irritant disease [3,5].

Antihistamines may reduce associated sleep disturbance in some patients but do not block the T-cell mechanism of ACD. Newer immunomodulatory treatments are being investigated for difficult chronic disease, yet evidence remains less established than for atopic dermatitis. Escalating anti-inflammatory treatment without controlling exposure is unlikely to produce durable remission. Follow-up should assess skin clearance, pruritus, sleep, hand function, work capacity, and the patient's ability to apply avoidance instructions.

Primary prevention is especially important in high-risk occupations. Education should begin before repeated exposure occurs and include correct glove use, avoidance of allergen contamination inside gloves, regular glove replacement, skin-care routines, and early reporting of symptoms. Secondary prevention includes prompt clinical evaluation and patch testing when indicated. Tertiary prevention combines allergen avoidance, optimized treatment, rehabilitation, and workplace adaptation to prevent chronicity and loss of employment.

Conclusion

Allergic contact dermatitis is a clinically important and potentially preventable cause of recurrent eczema. It develops through hapten-induced, T-cell-mediated delayed hypersensitivity, but its expression is strongly influenced by epidermal barrier damage, exposure intensity, anatomical site, occupation, and coexisting skin disease. Patch testing is the reference diagnostic method; however, its value depends on appropriate allergen selection, standardized readings, and careful assessment of current clinical relevance. The most effective long-term strategy combines identification and avoidance of the responsible allergen with barrier repair, targeted anti-inflammatory treatment, patient education, and workplace modification. Routine attention to occupational history and quality-of-life effects can shorten the diagnostic pathway and reduce chronic disease burden.

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