



CONTACT DERMATITIS: UPDATE ON PATHOPHYSIOLOGY, DIAGNOSIS, AND TREATMENT

**Cristina Belén Burgos Ubilla¹, Yoaris Sidney Moreno Maturana²,
Maria Alejandra Betancourt Pantoja³, Natalia Marín Núñez⁴,
Bryam Esteban Coello García⁵**

¹General Practitioner in Independent Practice, Faculty of Medical Sciences,
Universidad Católica Santiago de Guayaquil. Ecuador ORCID <https://orcid.org/0009-0003-5601-9384>

²General Practitioner at "EPS SURA", Faculty of Medical Sciences, Corporación Universitaria Remington.
Colombia ORCID <https://orcid.org/0009-0003-0146-996X>

³General Practitioner at "Hospital General de Medellín", Faculty of Medical Sciences,
Corporación Universitaria Remington. Colombia ORCID <https://orcid.org/0009-0009-5332-4475>

⁴Physician at the Hematopoietic Progenitor Transplant Unit, Instituto Nacional de Cancerología.
Faculty of Medical Sciences, Pontificia Universidad Javeriana Cali. Colombia.
ORCID: <https://orcid.org/0000-0001-9065-8823>

⁵Orthopedic Surgeon and Traumatologist, Fellow in Advanced Hip Surgery, Faculdade Moinhos de Vento.
Porto Alegre – Brasil. ORCID <https://orcid.org/0000-0003-2497-0274>

Corresponding Author : Bryam Esteban Coello García, **Address:** Rua General Neto, 118- Moinhos de Vento. Porto Alegre. Brasil, **Postal Code:** 90560-020

Article DOI: <https://doi.org/10.36713/epra28538>
DOI No: 10.36713/epra28538

ABSTRACT

Introduction: Contact dermatitis is a common inflammatory skin disease resulting from exposure to irritants or environmental allergens. It is mainly classified into irritant contact dermatitis and allergic contact dermatitis, both with distinct pathophysiological mechanisms but similar clinical manifestations. Its high prevalence and impact on quality of life make it a relevant condition in dermatological and occupational practice.

Objective: To conduct an updated literature review on the pathophysiology, classification, diagnosis, and treatment of contact dermatitis, integrating the most recent available scientific evidence.

Methodology: A narrative literature review was conducted through a search of scientific databases such as PubMed, Scopus, and Web of Science. Original articles, systematic reviews, clinical guidelines, and consensus statements published mainly in the last 10 years and related to contact dermatitis were included. The information was analyzed and synthesized descriptively by thematic categories.

Results: Irritant contact dermatitis results from direct damage to the skin barrier with the release of pro-inflammatory cytokines, whereas allergic contact dermatitis is a type IV hypersensitivity reaction mediated by T lymphocytes. The main allergens include nickel, fragrances, preservatives, and metals. Diagnosis is based on clinical history and patch testing, considered the gold standard. Treatment is based on avoidance of the causative agent, use of topical corticosteroids, calcineurin inhibitors, and skin barrier restoration measures, with systemic therapy reserved for severe cases.

Conclusions: Contact dermatitis is a common and potentially recurrent condition that requires accurate diagnosis and comprehensive management. Early identification of the causative agent and patient education are essential to prevent relapses and improve quality of life. Advances in immunological understanding have improved diagnostic and therapeutic strategies, supporting an evidence-based approach.

KEYWORDS: Contact Dermatitis; Allergic Contact Dermatitis; Irritant Contact Dermatitis; Type IV Hypersensitivity; Patch Test; Eczema.

INTRODUCTION

Contact dermatitis is an inflammatory skin disease triggered by direct exposure to external agents capable of disrupting the skin barrier or inducing an immune response. It represents one of the most frequent causes of dermatology consultations and a significant proportion of occupational dermatoses worldwide. It is traditionally classified into irritant

contact dermatitis and allergic contact dermatitis, entities that share similar clinical manifestations but differ in their pathophysiological mechanisms. Early recognition of these variants is essential to establish preventive measures, avoid recurrences, and optimize treatment(1).

From a pathophysiological standpoint, irritant contact dermatitis occurs as a consequence of direct damage to the



stratum corneum and disruption of cutaneous defense mechanisms, whereas allergic contact dermatitis corresponds to a type IV delayed hypersensitivity reaction mediated by T cells. The interaction between individual predisposition, integrity of the epidermal barrier, intensity of exposure, and characteristics of the triggering agent determines the onset and severity of the clinical picture. Advances in immunological understanding have improved knowledge of the inflammatory pathways involved and opened the door to more targeted therapeutic approaches(2).

Clinically, contact dermatitis presents a wide variability of manifestations, including erythema, edema, vesiculation, scaling, lichenification, and pruritus, which may complicate differential diagnosis with other eczematous dermatoses. Therefore, a detailed clinical history focused on occupational, domestic, cosmetic, or medical exposures, together with physical examination and complementary studies such as patch testing, remains essential to identify the causative agent and guide management(3).

Contact dermatitis is a common condition in clinical practice. It has been reported that allergic contact dermatitis may affect up to 20% of the pediatric population, while irritant contact dermatitis accounts for up to 80% of occupational dermatitis cases, reflecting its significant public health and occupational impact(4).

Due to the clinical, occupational, and economic burden associated with this disease, there is ongoing interest in updating knowledge regarding its pathophysiology, diagnosis, and treatment. In this context, the present article aims to perform a literature review to synthesize available evidence on the pathophysiological mechanisms of contact dermatitis, currently used diagnostic strategies, and therapeutic alternatives employed in contemporary clinical practice(5).

METHODOLOGY

A narrative literature review was conducted with the aim of synthesizing and updating the available scientific evidence on the pathophysiology, diagnosis, and treatment of contact dermatitis. The methodological structure was developed following quality principles for narrative reviews based on SANRA criteria (Scale for the Assessment of Narrative Review Articles), aimed at improving transparency, reproducibility, and methodological rigor.

The literature search was performed using electronic databases including PubMed, Scopus, Web of Science, and Google Scholar. Publications in English and Spanish available in full text were included. The search strategy used MeSH terms and keywords combined with Boolean operators: ("contact dermatitis" OR "allergic contact dermatitis" OR "irritant contact dermatitis") AND ("pathophysiology" OR "diagnosis" OR "treatment"). Priority was given to articles published between 2016 and 2026, including classical studies when considered relevant.

Inclusion criteria included original articles, narrative reviews, systematic reviews, clinical guidelines, and consensus documents addressing pathophysiology, clinical presentation, diagnosis, and treatment of contact dermatitis. Duplicate publications, articles without full-text access, isolated case reports, and studies focused on unrelated dermatological diseases were excluded. Study selection was performed by title

and abstract screening, followed by full-text review of eligible articles.

The selected information was then organized into thematic categories: pathophysiology, classification, clinical manifestations, diagnosis, and treatment. Findings were synthesized descriptively and comparatively to integrate available evidence and provide an updated overview of contact dermatitis management in clinical practice.

DEVELOPMENT

Pathophysiology of contact dermatitis

Contact dermatitis is a cutaneous inflammatory process triggered by the interaction between environmental factors and host biological mechanisms. Two main forms are recognized: irritant contact dermatitis (ICD) and allergic contact dermatitis (ACD). Although both may present similar clinical features, they differ substantially in cellular and molecular mechanisms. ICD results from direct damage to the epidermal barrier, whereas ACD is an adaptive immune response primarily mediated by T lymphocytes(6).

1. Irritant contact dermatitis

Irritant contact dermatitis is the most frequent form. Its main mechanism involves disruption of the stratum corneum due to repeated or intense exposure to irritants such as detergents, soaps, solvents, alkalis, acids, or prolonged moisture exposure. Initial injury leads to transepidermal water loss, lipid disruption, and keratinocyte damage, triggering innate immune activation through release of proinflammatory cytokines such as IL-1 α , TNF- α , and IL-6, with subsequent cellular recruitment. Severity depends on concentration, duration of exposure, and individual susceptibility(2).

2. Allergic contact dermatitis

Allergic contact dermatitis is a type IV delayed hypersensitivity reaction mediated by T cells. It typically involves two phases: sensitization and elicitation (re-exposure). During sensitization, small molecules known as haptens penetrate the epidermis and bind to self-proteins, forming antigenic complexes. These are captured by antigen-presenting cells—mainly Langerhans cells and dermal dendritic cells—which migrate to regional lymph nodes and activate clonal expansion of specific T lymphocytes. Upon re-exposure, a localized inflammatory response occurs with clinical lesion development(7).

3. Role of the skin barrier and immune response

Epidermal barrier integrity plays a central role in both forms of contact dermatitis. Structural abnormalities, including defects in proteins such as filaggrin or chronic exposure to irritants, increase penetration of external substances and promote immune activation. The inflammatory response is not solely dependent on adaptive immunity; keratinocytes actively contribute by releasing inflammatory mediators and stress signals that amplify and sustain inflammation(8).

4. Modulating factors

Disease onset and severity depend on the interaction between exogenous and endogenous factors. External factors include type of substance, frequency of exposure, humidity,



temperature, and friction. Individual factors include age, genetic susceptibility, pre-existing skin disease, and barrier function status. This interaction explains variability in clinical response among individuals exposed to the same agent(9).

Classification and clinical manifestations of contact dermatitis

Contact dermatitis is primarily classified into irritant contact dermatitis (ICD) and allergic contact dermatitis (ACD). This distinction is essential due to differences in pathophysiology, evolution, and management. Some authors also describe mixed forms or protein contact dermatitis, particularly in occupational settings(6).

Common allergens associated with ACD include nickel, cobalt, chromium, formaldehyde, fragrances, neomycin, and Peru balsam. In regions such as North America, urushiol exposure from Toxicodendron plants is a frequent cause. Severity depends on concentration, exposure time, and host susceptibility(4).

1. Irritant contact dermatitis

ICD is the most common form and can affect any individual, as it does not require prior sensitization. It presents with erythema, burning, xerosis, scaling, and in severe cases fissures, vesicles, or erosions. It typically affects areas of direct exposure such as hands, forearms, or moisture/friction-prone regions. It may be acute after intense exposure or chronic due to repeated low-grade exposure, as seen in healthcare or cleaning workers(2).

2. Allergic contact dermatitis

ACD requires prior sensitization and is mediated by T lymphocytes. It typically presents with intense erythema, edema, vesiculation, and marked pruritus. Lesions may extend beyond the initial contact site due to immune dissemination. Chronic stages show lichenification, hyperkeratosis, and fissuring. Common allergens include nickel, fragrances, preservatives, latex, and topical medications(7).

3. Distribution and clinical patterns

Lesion distribution depends on exposure type. Hands are most commonly affected due to repeated contact. The face and eyelids are frequently involved due to cosmetics or volatile agents, while trunk and extremities may be affected by clothing, metals, or medical adhesives. In ACD, distribution may not strictly correspond to the initial contact area due to localized immune spread(10).

4. Clinical evolution

Evolution may be acute, subacute, or chronic. Acute forms present with erythema and vesiculation after recent exposure; subacute forms show scaling and mild persistent inflammation; and chronic forms are characterized by skin thickening, lichenification, and fissuring. Persistent exposure or failure to identify the causative agent contributes to chronicity and recurrence, significantly impacting quality of life(1).

Diagnosis of contact dermatitis

Diagnosis is primarily clinical, based on detailed exposure history, lesion morphology, and anatomical

distribution. However, due to similarity with other eczematous dermatoses, complementary tests are often required to confirm diagnosis and identify the causative agent(6).

1. Clinical history and physical examination

History taking is fundamental and includes occupational, domestic, cosmetic, and medication exposures. Establishing temporal correlation between exposure and lesion onset is essential, as is improvement after avoidance. Physical examination characterizes lesion type and distribution, suggesting typical patterns such as hands, face, eyelids, or direct contact areas(7).

2. Patch testing

Patch testing is the gold standard for diagnosing allergic contact dermatitis. Standardized allergens are applied to the patient's back and readings are performed at 48 and 96 hours. A positive reaction indicates prior sensitization. Interpretation must correlate clinically to distinguish relevant allergy from incidental sensitization. Reactions are classified as negative, irritant, doubtful, weak positive (+), moderate (++), or strong (+++). Additional tests such as dimethylglyoxime testing may be used for nickel allergy(4,10).

3. Additional tests and differential diagnosis

Additional tests may include prick testing (for immediate hypersensitivity), skin biopsy, or repeated open application tests. Differential diagnosis includes atopic dermatitis, psoriasis, tinea corporis, nummular eczema, and drug reactions. Differentiation relies on clinical pattern, evolution, distribution, and test results(1).

4. Occupational dermatitis evaluation

In suspected occupational cases, systematic workplace assessment is required, including chemical safety data sheets, protective measures, and exposure duration. Identifying the causative agent is essential for prevention and reducing chronic disease development(11).

Treatment of contact dermatitis

Treatment is based on three main pillars: identification and avoidance of the causative agent, restoration of the skin barrier, and control of inflammation. Strategy varies according to severity, extent, and whether ICD or ACD is present(6).

1. Avoidance of causative agent

The most important measure is elimination of the irritant or allergen. In ACD, this includes avoidance of nickel, fragrances, preservatives, or latex; in ICD, reduction of exposure to detergents, solvents, and moisture is recommended. Patient education and behavioral modification are essential for long-term control(7).

In urushiol-induced dermatitis, early washing with soap and water within two hours is critical. High-potency topical corticosteroids are effective early, especially within 12 hours, but less effective once vesicles are established. Treatment must be individualized, avoiding potent steroids in thin skin areas. Moderate to severe cases may require systemic corticosteroids with tapering. Calcineurin inhibitors (tacrolimus, pimecrolimus) and emollients are useful adjuncts(4).



2. Topical treatment

Topical corticosteroids are first-line therapy, reducing inflammation, erythema, and pruritus. Potency depends on lesion location and severity. In sensitive areas, low-potency steroids or calcineurin inhibitors are preferred. Emollients are essential for barrier restoration and recurrence prevention(10).

3. Systemic treatment

Severe or extensive cases may require oral corticosteroids, especially in acute ACD. Chronic or refractory cases may be treated with immunomodulators such as cyclosporine, methotrexate, or azathioprine. Antihistamines may be used as adjuncts for pruritus control, although they do not target the main inflammatory pathway(1).

4. Integrated management and prevention

Optimal management requires a multidisciplinary approach involving dermatology, primary care, nursing, and clinical pharmacy, with emphasis on patient education and avoidance strategies. Prevention includes protective equipment in occupational settings, proper skin hydration, avoidance of irritants, and early recognition of relapse. Workplace interventions can significantly reduce incidence and improve functional outcomes(4,10–12).

CONCLUSION

Contact dermatitis is a common and potentially recurrent inflammatory skin disease that represents a significant clinical and occupational problem. Its appropriate management depends on an accurate diagnosis based on clinical history and complementary tests such as patch testing. Correct classification into irritant and allergic forms improves the understanding of its pathophysiological mechanisms and guides more effective treatment, primarily focused on avoidance of the causative agent, restoration of the skin barrier, and control of inflammation. Patient education and early identification of triggering factors are essential to prevent relapses and improve quality of life, while advances in immunological knowledge have optimized diagnostic and therapeutic strategies, supporting an increasingly evidence-based approach.

REFERENCES

1. Martin SF, Rustemeyer T, Thyssen JP. Recent advances in understanding and managing contact dermatitis. *F1000Research*. 2018 Jun 20;7:810. doi:10.12688/f1000research.13499.1
2. Patel K, Nixon R. Irritant Contact Dermatitis – a Review. *Curr Dermatol Rep*. 2022 Apr 7;11(2):41–51. doi:10.1007/s13671-021-00351-4
3. Pesqué D, Aerts O, Bizjak M, Gonçalo M, Dugonik A, Simon D, et al. Differential diagnosis of contact dermatitis: A practical-

approach review by the EADV Task Force on contact dermatitis. *J Eur Acad Dermatol Venereol*. 2024 Sep;38(9):1704–22. doi:10.1111/jdv.20052

4. Litchman G, Nair PA, Atwater AR, Bhutta BS. Contact Dermatitis. In: *StatPearls* [Internet]. Treasure Island (FL): StatPearls Publishing; 2026 [cited 2026 Jun 26]. Available from: <http://www.ncbi.nlm.nih.gov/books/NBK459230/> PubMed PMID: 29083649.
5. Brar KK. A review of contact dermatitis. *Ann Allergy Asthma Immunol*. 2021 Jan;126(1):32–9. doi:10.1016/j.anai.2020.10.003
6. Scheinman PL, Vocanson M, Thyssen JP, Johansen JD, Nixon RL, Dear K, et al. Contact dermatitis. *Nat Rev Dis Primer*. 2021 May 27;7(1):38. doi:10.1038/s41572-021-00271-4
7. Tramontana M, Hansel K, Bianchi L, Sensini C, Malatesta N, Stingeni L. Advancing the understanding of allergic contact dermatitis: from pathophysiology to novel therapeutic approaches. *Front Med*. 2023;10:1184289. doi:10.3389/fmed.2023.1184289 PubMed PMID: 37283623; PubMed Central PMCID: PMC10239928.
9. Martin SF, Rühl-Muth AC, Esser PR. Orchestration of inflammation in contact allergy by innate immune and cellular stress responses. *Allergo J Int*. 2023 Nov 9;33(2):41–8. doi:10.1007/s40629-023-00275-4
10. Yu J, Milam EC. Comorbid Scenarios in Contact Dermatitis: Atopic Dermatitis, Irritant Dermatitis, and Extremes of Age. *J Allergy Clin Immunol Pract*. 2024 Sep;12(9):2243–50. doi:10.1016/j.jaip.2024.04.052
11. Van Zuuren EJ, Arents BWM, Vermeulen S, Schoones JW, Fedorowicz Z. Global Guidelines in Dermatology Mapping Project (GUIDEMAP) – A systematic review of the methodological quality of contact dermatitis clinical practice guidelines. *Contact Dermatitis*. 2024 Jun;90(6):543–55. doi:10.1111/cod.14530
12. Lau MYZ, Burgess JA, Nixon R, Dharmage SC, Matheson MC. A review of the impact of occupational contact dermatitis on quality of life. *J Allergy*. 2011;2011:964509. doi:10.1155/2011/964509 PubMed PMID:21603173; PubMed Central PMCID: PMC3095907.
13. Strauss RM, Gawkrödger DJ. Occupational contact dermatitis in nurses with hand eczema. *Contact Dermatitis*. 2001 May;44(5):293–6. doi:10.1034/j.1600-0536.2001.440508.x

Conflict of Interest Statement

The authors report no conflicts of interest.

Funding

The authors report no funding from any organization or company.