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Contact Dermatitis (Inclusive of irritant contact dermatitis and allergic contact dermatitis)

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ABSTRACT
Contact dermatitis is a common skin disorder that is classified into two categories: irritant contact dermatitis and allergic contact dermatitis. Each differs from one another by the immune response elicited by the body; ICD is predominately innate, while ACD is classified as a T cell-mediated process type IV delayed hypersensitivity reaction. CD is commonly induced by occupations (due to excessive hand washing or detergents), nickel, or perfume allergies. With a proper history, physical examination, and patch testing, CD can be diagnosed. Treatment with corticosteroids and barrier cream can significantly improve quality of life. Preventative measures should be encouraged, such as avoidance of triggers and allergens.

Keywords: Dermatitis, irritant contact dermatitis, allergic contact dermatitis, management

INTRODUCTION

Contact dermatitis (CD) is an inflammatory disease caused by exogenous substances, such as chemicals or metal ions. This results in pruritic and erythematous eczematous patches on the skin. Contact dermatitis can be classified into two categories based on the type of response to exogenous substances. The two major categories are irritant contact dermatitis (ICD) and allergic contact dermatitis (ACD). The first type, ICD, is a nonspecific inflammatory response in which a toxic effect is induced without a T-cell response. The second type is ACD, which is a delayed type IV hypersensitivity reaction where there is a modification in proteins that induce innate and adaptive immune responses. The two can present simultaneously with a similar phenotype, whereas ICD is more common [1] [2] [5]. Besides CD being difficult to differentiate due to similarities in clinical presentation and histology, it is usually self-limiting. In chronic cases, it can affect quality of life and result in major socio-economic impacts due to extended absence from work [1] [5.]

ETIOLOGY

Contact dermatitis can be classified into two subcategories each with different etiologies.

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Irritant contact dermatitis (ICD):

This is the result of chronic or repeated exposure to exogenous substances, leading to skin damage and lesions. This is usually a non-immune-modulating skin irritation. Irritation could be physical, such as friction, abrasions, or chemicals such as detergents such as sodium lauryl sulfate [1] [2] [3] [4]. Significant damage must be done to the stratum corneum. Factors such as damage to the skin barrier, epidermal cells, dry skin, or previous atopic dermatitis can contribute to the release of proinflammatory cytokines by keratinocytes and employ the innate immune system. All these factors contribute to the pathophysiology of ICD. Proinflammatory cytokines released are interleukin (IL)-1alpha, IL-1beta, IL-6, and tumor necrosis factor (TNF)-alpha from keratinocytes. This results in the migration of monocular and polymorphonuclear cells to the site of damage [1] [2].

Allergic contact dermatitis (ACD):

ACD is induced by an adaptive immune response, as the body is sensitized by previous exposure to an allergen. This process is a T cell-mediated process, classified as a type IV delayed hypersensitivity reaction. Sensitization occurs up to 10-14 days in two phases: the afferent(sensitization) and efferent (elicitation) phases and is highly influenced by the integrity of the epidermal barrier [2] [3]. The most common triggers are nickel, fragrances, and poison ivy in the United States [1].

The development of ACD occurs when there is a second exposure to the site by the initial allergen. This triggers antigen-specific T-cells to prime T cells in the lymph nodes and direct them to the site of exposure. The inflammatory response is supported by proinflammatory cytokines, such as TNF-α and IL1-beta. The MHC molecules were upregulated [2] [5]. Cutaneous lymphocyte antigen (CLA) positive T cells remain in the skin for a longer period. T cells produce IFN-γ, IL-2, and IL-17 and have an apoptotic effect on keratocytes, resulting in ACD. [5] Constant exposure of the site to the allergen increases the severity of the ACD reaction and the speed of onset of symptoms [2]. Although ICD and ACD may present similarly, ICD inflammation is dependent on the irritant, while ACD is dependent on the dose of the irritant itself [5].

EPIDEMIOLOGY

Contact dermatitis makes up 80-95% of occupational skin disorders [5] [6] [7]. ICD accounts for 80% of all contact dermatitis while ACD is 20% of cases [2]. Contact dermatitis is very common in the population, affecting almost 20% of children. Elderly individuals are also affected by alterations in the epidermal barriers and immune reactivity [1] [5]. In the COVID-19 era, it has been observed that increased handwashing has increased the frequency of ICD among children and health workers. In Denmark, it was observed that 42.4% of children experienced ICD due to increased hand washing with school children having a 1.5 increased risk of developing ICD [8]. Pre Covid-19 occupational skin disease among healthcare workers was 20 %–50%.[9] Doctors and nurses have increased hand disinfecting practices since the beginning of COVID-19, resulting in a 97.0% increase in skin damage with a baseline of 80.2% in a study in Kiev city, Ukraine [10].

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Nickle allergic contact dermatitis (Ni-ACD) in 1996-2006 accounted for 22.1% of adults aged 20-40 years. It currently affects 20% of Americans [11]. Second, fragrance ACD is the most common allergen in patients with ACD. Fragrances such as limonene and linalool hydroperoxides have a high prevalence of sensitization. In the general adult population, 4.5% of the population is allergic to fragrance [28]. Overall, risk factors such as age, genetic predispositions, occupation, and environmental exposure determine an individual's risk of developing contact dermatitis.

HISTOPATHOLOGY

Irritant contact dermatitis (ICD):

ICD presents histological confocal features such as parakeratosis, spongiosis, perivascular neutrophilic inflammatory infiltrate of the epidermis, and macrovesicle formation. Follicular spongiosis is more commonly observed in patients with ACD [1] [12] [14].

Allergic contact dermatitis (ACD):

ACD is diagnosed by a combination of clinical presentation, history, physical examination, and a positive patch test for the suspected allergen. Sometimes, a biopsy may be required. Spongiosis is typically observed; mononuclear infiltrates comprise T cells in the dermis and epidermis with intercellular epidermal edema [1] [13] [14].

IMMUNOPATHOGENESIS

Irritant contact dermatitis (ICD):

ICD occurs when an exogenous irritant agent is exposed. Inflammation arises from proinflammatory cytokines (IL-1alpha) released from keratinocytes and corneocytes in response to stimuli such as chemicals (acid solutions) and physical agents (UV radiation and X-rays). This results in skin barrier disruption and epidermal cellular changes, specifically to the protective lipid film on the surface of the horny [1] [3] [29].

Allergic contact dermatitis (ACD):

ACD includes two phases: sensitization and elicitation. During the sensitization phase, the allergen penetrates the epidermis, causing tissue stress and damage. It manifests as broken skin, vesicles, or redness. Once this occurs, stressed skin cells release signals such as reactive oxygen species (ROS), ATP, and damage-associated molecular patterns (DAMPs). The innate immune system is triggered by stress via mast cells and neutrophils, which activate Toll-like receptors (TLRs) and NOD-like receptor pyrin containing (NLRP3). Skin inflammation occurs, and dendritic cells (DCs) are activated, which migrate to the skin-draining lymph nodes. DCs present an allergen to naïve T cells, resulting in their activation and differentiation. The elicitation phase occurs when there is repeated contact with the allergen. Inflammation is induced, and T cells are recruited to the area, resulting in the clinical symptoms of ACD. [15] [29]

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SIGNS AND SYMTOMPS

TABLE 1: Showing the signs and symptoms associated with two types of contact dermatitis (Irritant contact dermatitis and allergic contact dermatitis).

CONTACT DERMATITIS	SIGNS	SYMPTOMS
IRRITANT CONTACT DERMTAITIS (ICD)	Dry, fissured skin, erythema, blisters, pustules, hemorrhage, crusts, scales and erosions, less distinct boarders	Burning, pruritus, pain,
ALLERGIC CONTACT DERMTAITIS (ACD)	Vesicles and bullae, erythema, crusts, distinct angles, lines and boarders.	Pruritus is dominant
[4] [3]		

INVESTIGATIONS

For contact dermatitis to be diagnosed, the following steps must be performed:
1. A comprehensive history related to the development of dermatitis and allergen triggers in professions where contact dermatitis is common, profession-specific questionnaires are available to individuals.
2. A clinical examination of the affected sites should be done to observe the integrity of the dermis and the extension of the damage.
3. A patch test should be performed if ACD is suspected. [16]

Patch Test:
Path testing is an in vivo test that is the gold standard for the clinical management of ACD [18]. The most common test used was the thin-layer rapid-use epicutaneous (T. R. U. E.) test. This included 35 allergens in hydrophilic gels. The panel is outdated, as 50% of participants usually have a positive reaction to an allergen not listed on the T.R.U.E. test. Other panels that consist of 65 allergens are available and are commonly used by the North American Contact Dermatitis Group. [17]

The allergens were placed on intact skin on the back. After 48 h, the patches were removed for initial reading. After five days, the patient returned for a final reading. During this time, the patient must avoid water, detergents, sweating, sunlight exposure, and use of external medications such as steroids. A true allergic reaction would remain after 5 days. [17]
The positive reactions are scored on the following scale:

?/+ : doubtful (macular erythema)
+ : weak (papular erythema)
++ : strong (edematous or vesicular)
+++ : extreme (bullous or ulcerative).

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4. If the patch test is inconclusive, a repeat open application test (ROAT) is performed. This usually tests cosmetics and topical drugs and is performed for up to 2 weeks [18] [19].
5. Histological analysis via skin biopsy should be performed if the eczema symptoms are atypical. This is typically not performed, as it does not help differentiate between allergic and non-allergic reactions [17] [18].

COMPLICATIONS

When there is extensive damage to the epidermis and dermis from contact dermatitis, there is a significant barrier dysfunction. This dysfunction results in excessive allergy responses from foreign antigens and can lead to chronic skin inflammation, like atopic dermatitis. [20] In cases of contact dermatitis, there is damage to the stratum corneum, which is an important barrier against microorganisms [21]. This damage makes individuals more vulnerable to infection. A patient with chronic dermatitis can present with secondary infections such as staphylococci (weeping dermatitis), dermatophytes (hands and feet), or Candida infections in folds. These secondary infections can make the diagnosis more difficult [16]. ACD has a major effect on the quality of life of patients and has major socioeconomic consequences. Quality of life is highly associated with presenteeism and loss of work productivity. The quality of life decreases when sick leaves are prolonged. [30]

LIST OF DIFFERENTIAL DIAGNOSES

TABLE 2: Showing the most common differential diagnosis of contact dermatitis with locations.

DIFFERENTIAL DIAGNOSIS	LOCATIONS
Atopic dermatitis	Trunk, head, hands, feet,
Psoriasis	Scalp, elbows, trunk
Erythrasma	Axillae and groin
Fungal infection (Mycoses, tinea versicolor Pityriasis rosea)	Extremities, trunk
Scabies (eczematized)	Hands (interdigital), mamilla
[19] [22]	

TREATMENT/MANAGEMENT

Supportive measures such as cool compresses and fragrance-free emollients/moisturizers are recommended for soothing. If the lesions are weeping, aluminum subacetate (Borrow's solution) can be applied, calamine, or an oatmeal bath is also suggested. [25]

Irritant contact dermatitis (ICD):
Lipid-rich moisturizers are effective for short-term treatment of experimentally induced ICD. In a previous study, canola oil, the sterol-enriched fraction of canola oil, and hydrocortisone helped with the degree of irritation. The petrolatum-based moisturizers showed improvements. [23]

Management includes avoiding soaps and detergents. At work, it is important to follow the three

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tips of avoidance, protection, and substitution [27]. When handwashing, abrasive maneuvers are avoided to avoid skin irritation. A combination of humectant and occlusive emollients is recommended to attract and seal the water in the corium layer. Gloves should be applied to dry hands and hands remoisturized when removed [26].

Allergic Contact Dermatitis (ACD):

Topical steroids are a first-line treatment. Therefore, a combination of topical corticosteroids and antibiotics is recommended [27]. The use of fluticasone propinoate .05% (coupled with/without a barrier cream), clobetasone butyrate .05%, and clobetasol propionate .05% all showed effectiveness in the treatment of ACD. The outcomes also increased with the barrier cream alone [23]. In instances of poison ivy, topical corticosteroids are recommended as first-line treatment. If lesions cover >20% of body surface area, systemic corticosteroids such as prednisone 0.5-1 mg/mg/kg/day) for 5-7 days and then half the dose for another 5-7 days is recommended. A calamine lotion can be applied if there is a weepy lesion [23]. General management includes avoiding triggers. These include coverings of clothing facets in cases of nickel allergies. Reducing handwashing and moisturizing after washing [25].

CONCLUSION

Contact dermatitis is a commonly overlooked skin disorder that, when developed into a chronic condition, can significantly impact the quality of life of patients. Prevention methods should be encouraged to prevent the development of ICD, which is predominantly an occupational-induced disorder. In a COVID-19 world where handwashing is encouraged, preventive measures should be highlighted to prevent progression to ACD. CD can be controlled by proper treatment and avoidance of triggers.

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