CLINICAL MANAGEMENT extra

Scratching the Surface: A Review of Dermatitis

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Acknowledgments: The authors would like to thank the patients for agreeing to have their pictures taken for educational purposes. The author, faculty, staff, and planners, including spouses/partners (if any), in any position to control the content of this CME/CNE activity have disclosed that they have no financial relationships with, or financial interests in, any commercial companies relevant to this educational activity.

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GENERAL PURPOSE:

To present a case-based review illustrating atopic and contact dermatitis, including management of these conditions using topical and systemic therapies.

TARGET AUDIENCE:

This continuing education activity is intended for physicians, physician assistants, nurse practitioners, and nurses with an interest in skin and wound care.

LEARNING OBJECTIVES/OUTCOMES:

After participating in this educational activity, the participant should be better able to:

1. Review the prevalence, etiology, and consequences of the various types of dermatitis.
2. Describe the clinical manifestations and differential diagnosis of the various types of dermatitis.
3. Outline the treatment options for the various types of dermatitis.

ABSTRACT

Eczematous reactions such as atopic dermatitis and contact dermatitis are prevalent worldwide. Despite contrasting pathophysiology, the diagnosis and management of these dermatitides can be challenging for healthcare providers. Differences in the distribution of the affected areas, duration of onset, and associated symptoms may help to distinguish these conditions. Diagnosis of the respective conditions is useful in developing appropriate management plans. Herein, the authors present a case-based review illustrating these different disease entities. Management of these conditions, including the use of topical and systemic therapies, is discussed.

KEYWORDS: atopic dermatitis, contact dermatitis, corticosteroids, dermatitis, eczema, review, systemic therapy, topical therapy

ADV SKIN WOUND CARE 2019;32:542–9.

INTRODUCTION

Cutaneous disease is one of the leading contributors of disease burden worldwide.[1] According to a large cohort study of more than 85 million patients, up to 25% of physician visits in the US were attributable to skin-related conditions in 2013.[2] The corresponding cost was estimated at $75 billion in direct healthcare costs. Of the cutaneous manifestations of disease, atopic dermatitis (AD) and contact dermatitis (which encompasses irritant contact dermatitis [ICD] and allergic contact dermatitis [ACD]) are of considerable interest because of their global prevalence. Indeed, AD is estimated to affect up to 20% of children worldwide.[3] Contact dermatitis is the most common occupation-associated skin condition, with ICD accounting for 80% of cases and ACD constituting the remainder.[4,5]

Similarities in the clinical presentation of these conditions create a challenge for healthcare providers in accurately managing these entities. This case-based review provides a diagnostic approach to manage these common dermatitides.

Atopic Dermatitis

A 36-year old woman presents with an erythematous, pruritic, and papulosquamous rash with secondary lichenification involving the flexural areas of her elbows (Figure 1). Notably, the appearance of the rash coincided with the onset of winter and has persisted since its onset 3 months ago.

Atopic dermatitis, commonly known as eczema, is an inflammatory skin condition of significant morbidity worldwide.[6] The pathophysiology of AD is multifactorial and involves underlying host factors, including epidermal barrier dysfunction, immune dysregulation, and environmental conditions.[7] Atopic dermatitis has a strong genetic component that accounts for approximately 90% of early-onset AD cases.[8] Specifically, genetic mutations associated with impaired skin barrier function, pH, and hydration of the epidermis have been implicated in the pathogenesis of AD.[9] Individuals with AD are also at risk of developing asthma, allergic rhinoconjunctivitis, and food allergies during their lifetime.[10,11] These conditions are referred to as the "atopic march" and may present concurrently or following the development of AD.[12] Once established, AD may persist chronically or relapse throughout an individual's lifetime. In addition, AD may predispose individuals to secondary infections such as *Staphylococcus aureus*, which in turn can exacerbate AD disease severity.

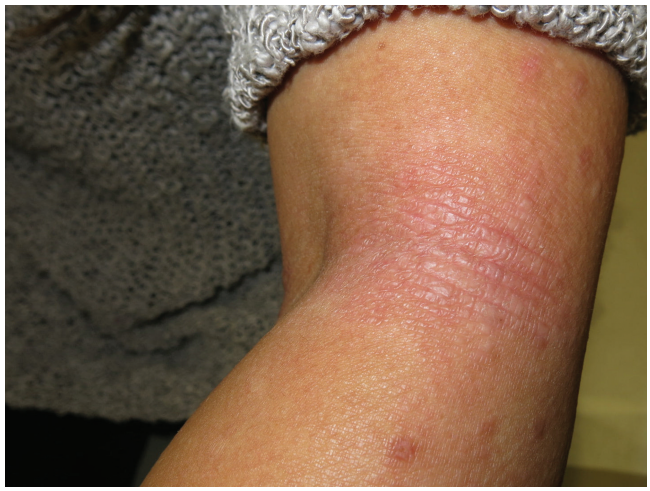
Although seen in individuals of all ages, AD most frequently occurs in childhood. Interestingly, new data suggest that there is a U-shaped prevalence distribution with a high prevalence in childhood that lessens in adulthood and increases again in older age (>75 years). This is likely multifactorial and relates to skin dysfunction in normal aging and/or pathology.[13,14]

Body areas affected by AD can vary by patient age (Figure 2).[15] In infants, the extensor surfaces, forehead, and cheek areas are commonly involved; the trunk is less frequently involved. During childhood, involvement of the flexural folds (ie, front of elbow crease) is most common. In the years of transition to adolescence and adulthood, the flexural sites, wrists, ankles, and neck are most often affected. During adulthood, involvement of the flexural surfaces, face, neck, and anterior chest is more common.[16] Sparing of the groin and axillary regions is seen in all ages.

Although AD is a common entity, chronic dermatoses, infectious processes, and primary immunodeficiency may mimic the presentation.[17] Noninfectious dermatoses include contact dermatitis, seborrheic dermatitis, and psoriasis. Differentiating between AD and contact dermatitis (ICD, ACD) can be challenging for healthcare practitioners and is discussed in a later section. Early-onset AD is often confused with seborrheic dermatitis, which can be distinguished by its involvement of the scalp and absence of pruritus. A diagnosis of psoriasis should be considered in the case of well-demarcated patches with involvement of the groin. Infectious processes such as scabies can be mistaken for AD but may be differentiated by groin involvement and the accompanying vesiculopustules found on the palms and soles.

Atopic dermatitis can have a spectrum of clinical presentations over a lifetime. Acute presentations may accompany early-onset AD and present as edematous, erythematous papules and plaques with or without vesiculation. Subacute dermatitis presents with scaling and/or crusting in addition to erythema. Chronic AD is associated with dry, thickened plaques that may have features of lichenification with a hardened or leathery appearance because of consistent irritation and rubbing. Weeping or impetiginization may be seen in association with *S aureus* infection.[18] Importantly, intense pruritus is associated with all presentations of AD and is essential for

Figure 1.
ATOPIC DERMATITIS



the diagnosis of the disease. Other associated cutaneous manifestations include but are not limited to xerosis, ichthyosis vulgaris, and keratosis pilaris. Postinflammatory hypopigmentation or hyperpigmentation may be seen with AD, especially in patients of color.

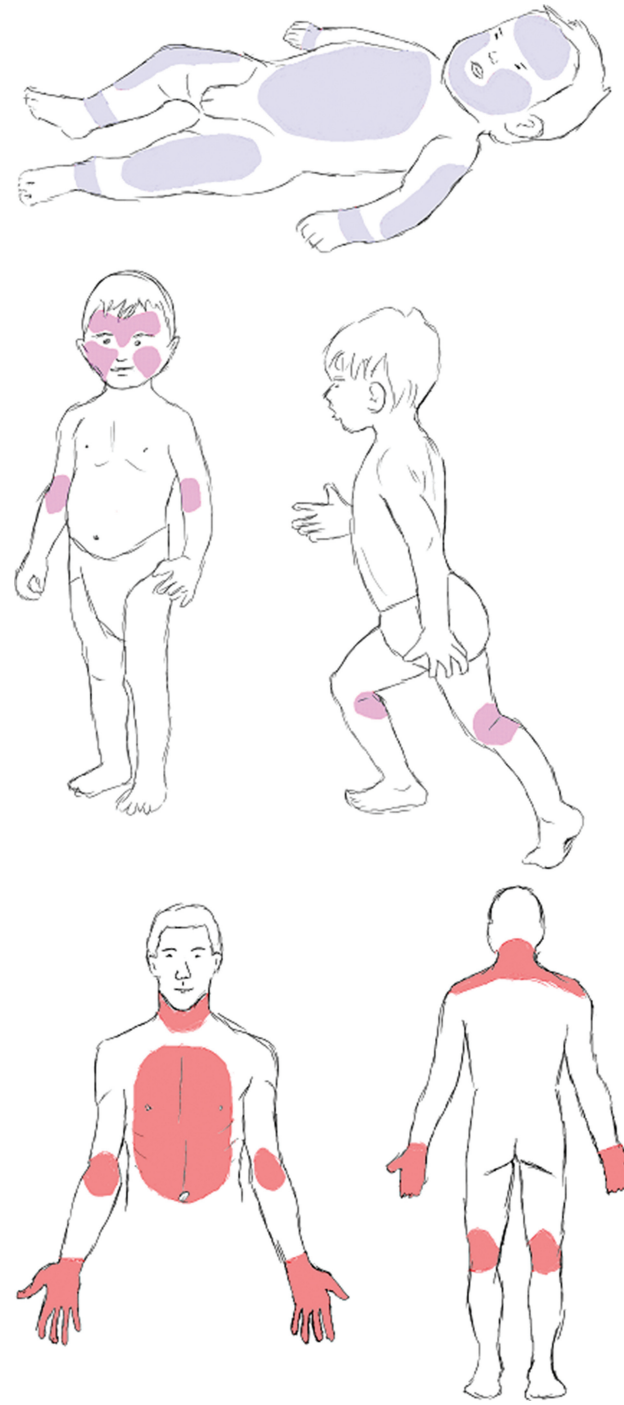
Irritant Contact Dermatitis

A 42-year-old healthcare worker has developed a burning red eruption over his dorsal hands bilaterally (Figure 3). The eruption improves over the weekend but exacerbates during the work week. Just prior to the start of this eruption, his facility instituted a new "safe hand" policy with the use of a daily chlorhexidine scrub.

Irritant contact dermatitis results from direct exposure of the skin to the cytotoxic effects of a chemical or physical agent. The hands and face are the most commonly affected areas.[19] It is associated with occupations in the manufacturing industry, arts, and food services, where exposure to irritating substances is common. Healthcare providers are at considerable risk of ICD because of frequent and repeated hand hygiene practices.[20] Water, detergents, solvents, and oils are common causes, but any substance that disrupts epithelial barrier function may result in ICD.[21] These exposures are influenced by the irritant's concentration and pH levels, as well as duration of exposure. Additional contributors include both host factors (eg, age-related epidermal barrier function) and environmental factors (eg, humidity, temperature).

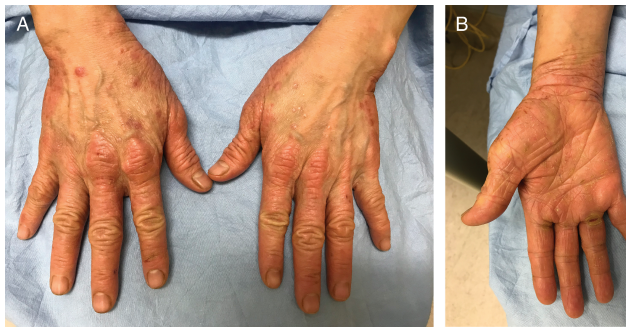
Acute exposure of the skin to the irritant results in the disruption of the stratum corneum. Damage to the keratinocytes and a corresponding inflammatory response give rise to erythema, edema, vesiculation, and skin erosion. Continued and/or repeated exposures to irritants may result in chronic ICD, which presents with lichenification, excoriations, scaling, and fissures,

Figure 2.
THE DISTRIBUTION OF BODY SURFACES AFFECTED BY ATOPIC DERMATITIS CHANGES WITH AGE



Involvement of extensor surfaces, forehead, and cheek areas is common in infants (<2 years). In childhood, involvement of flexural folds is more commonly seen. In adults, involvement of the flexural surface and the face, neck, and anterior chest is seen. In all ages, the groin and axillary area is spared. © R. Somayaji, 2019.

Figure 3.
IRRITANT CONTACT DERMATITIS



A, Dorsal view; B, palmar view.

which are the result of impaired transepidermal barrier recovery. Inflammatory changes appear 8 to 24 hours following initial exposure to the irritant.

Multiple forms of ICD exist; cumulative ICD is the most prevalent form, characterized by repeated, subthreshold exposure to weak irritants impairing barrier function.[21] Essentially, although exposure to weak irritants would not normally result in damage to the skin, the frequency at which the skin is exposed exceeds the body's ability to repair its barrier function. Ultimately, this pattern of irritant exposure results in erythema, dryness, hyperkeratosis, and lichenification. Exposure to a stronger irritant may result in erythematous, edematous lesions and even necrosis of the affected area. This clinical manifestation is often accompanied by a burning sensation, pain, and blistering.

Allergic Contact Dermatitis

A 35-year-old hairdresser has begun to develop an intensely pruritic rash involving her hands and distal forearms (Figures 4 and 5). The eruption is on both the dorsal and palmar aspects of her hands with more pronounced involvement on the palmar aspect. Small vesicles weeping clear fluid are often preceded by intense pruritus of the affected skin.

Compared with ICD, ACD accounts for a minority of occupational contact dermatitis cases caused by exposure to external agents.[22] It has no age, race, or sex predilection and is seen most commonly on the hands and face but may vary depending on the allergen. For example, allergic reactions to nickel may be seen on the umbilicus, ears, or neck from jewelry or belt buckles. After nickel, the most common allergens that cause ACD in North America include sulfate, fragrances, sanitary wipes (methylisothiazolinone), neomycin, bacitracin, cobalt chloride, formaldehyde, balsam of Peru, temporary tattoos (*p*-phenylenediamine), and sunscreen products (benzophenone-3).[23] When the associated allergens are identified using patch testing, a reaction to topical corticosteroids has been seen in up to 5.7% of cases.[24] Establishing a clinical diagnosis in these cases may be difficult because of the overlap between the underlying disease process and the reaction to the corticosteroid. As such, topical steroid allergy should be considered for cases in which topical corticosteroids fail to control a presumed ACD case, or when a rash worsens despite adequate steroid therapy.

Allergic contact dermatitis is a delayed-type hypersensitivity immune response.[25] The process starts with an initial exposure and penetration of the allergen into the epidermis, followed by a chain of physiologic processes that results in the sensitization

Figure 4.
ALLERGIC CONTACT DERMATITIS, HANDS

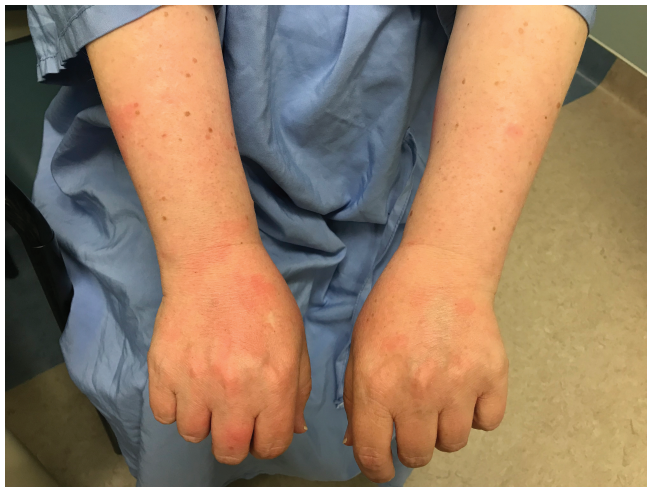
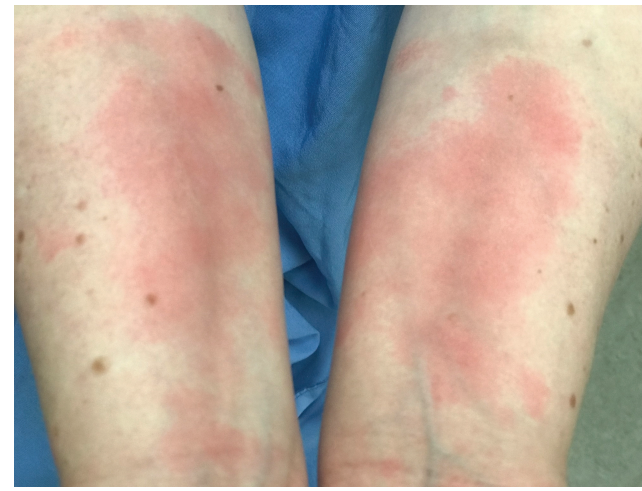


Figure 5.
ALLERGIC CONTACT DERMATITIS, FOREARMS



of the body to the allergen. Upon re-exposure of the allergen, the allergen-specific inflammatory cells activate and result in the clinical manifestation of eczema. Importantly, the manifestation is dependent on the characteristics and exposure pattern of the allergen. Acute ACD commonly presents as a well-demarcated, erythematous, pruritic patch or plaque with edema, blistering, or weeping. If chronic, the eruption will often have signs of lichenification or scale.

In some cases, a diffuse or widespread rash may be seen in response to systemic exposure to an allergen to which the patient was previously sensitized with a topical agent. This is referred to as autoeczematization or an "id" reaction. This secondary dermatitis presents distally to the original dermatitis within 1 or more weeks following the initial onset. There is a range of morphologic presentations, although it is usually symmetrical with areas of erythematous papules and vesicles. Although the pathogenesis remains unclear, id reactions are seen in the context of contact dermatitis and underlying infections.[26–28]

Differentiating AD, ICD, and ACD

Despite similar manifestations as an eczematous rash, there are notable differences among these disease processes (Table 1). Conceptualizing the physiologic processes of these entities may be useful in understanding the differences. Briefly, the mechanism driving the development of AD primarily involves an endogenous problem of epithelial cell dysfunction.[15] Conversely, ICD and ACD are more consistent with exogenous exposure to an irritant. Although allergens are highly diverse, fragrances and preservatives are most common.[29,30]

Whereas the direct effects of the irritant primarily drive ICD, ACD is a delayed-type hypersensitivity reaction caused by prior sensitization and exposure to the allergen of interest. A thorough history and physical examination can provide further insight into these conditions. For instance, individuals with AD may describe its onset during childhood with recurrent episodes following an age-dependent pattern throughout their lifetime. In contrast, patients with contact dermatitis may describe its onset following an

Table 1.
KEY FEATURES OF DERMATITIS BY TYPE

	Endogenous	Exogenous	
	Atopic Dermatitis	**Irritant Contact Dermatitis**	**Allergic Contact Dermatitis**
Relevant features	Erythematous, edematous rash which follows an age-dependent distribution Chronically relapsing course Positive family history	Well-demarcated erythematous, edematous rash with vesiculation Improves with rest and worsens with repeated irritant exposure	Typically a well-demarcated erythematous, edematous rash with vesiculation Systemic exposure may produce a diffuse rash on distal surfaces
Skin distribution	Infants: facial and extensor involvement Children: flexural surfaces Adult: flexural surfaces, neck, face, anterior chest	Most commonly hands and feet	
Diagnosis	Clinical	Clinical	Clinical and patch testing Patch testing is the standard for diagnosis; however, a negative result does not rule out ACD
Symptoms	Intense pruritus	Burning/irritation, soreness	Pruritus
Duration of onset	May appear anytime in life Preferentially affects areas of dryness	May appear acutely within 8–24 h of exposure No sensitizing exposure is required	Lesions appear 24–72 h after exposure Requires sensitizing exposure
Treatment	Emollients Topical corticosteroids Topical calcineurin inhibitors Crisaborole	Irritant avoidance Emollients Topical corticosteroids	Allergen avoidance Emollients Topical corticosteroids Topical calcineurin inhibitors
Systemic therapies	Systemic corticosteroids Phototherapy/psoralen and UV-A Dupilumab	Not typically used	Systemic corticosteroids Phototherapy/psoralen and UV-A

acute incident, such as exposure to strong irritants or after employment at a new job where certain hygiene practices are common.

Relying on the clinical morphology alone may not be enough to distinguish ICD from ACD. As such, understanding the area of distribution, duration of onset, and associated symptoms may aid in diagnosis. Both ICD and ACD present as sharply demarcated areas, but diffuse involvement of the distal skin can be seen in ACD. The timeline from exposure to the symptom onset is another distinguishing characteristic. In cases of ICD, symptoms present in minutes to hours. In ACD, onset is delayed and may occur up to 72 hours following repeated exposure. Patients with ICD typically manifest with a burning sensation, whereas pruritus is observed in ACD. When ICD and ACD cannot be distinguished clinically, patch testing is necessary (the criterion standard for the diagnosis of ACD).[31]

MANAGEMENT

The goals of treatment are to resolve the current skin eruption, relieve symptoms, treat the underlying epithelial dysfunction, and engage in maintenance therapy to reduce the risk of future symptoms. In all cases, education is the cornerstone of management to identify and avoid known exacerbating factors. In the case of ICD and ACD, protective equipment such as gloves should be used when working with known irritants.[32] General care includes a regular skin care routine and the use of moisturizers to protect against dry skin.[33–35] Using moisturizers reduces xerosis, pruritus, erythema, fissuring, and lichenification, thereby lessening the severity of disease.[36] In AD, moisturizers are associated with extension to the time of flare, a reduction in the number of flares, and less corticosteroid required to achieve control.[37,38] Although various formulations exist, including ointments and creams, no particular preparation has been shown to be significantly more effective.

Topical corticosteroids and calcineurin inhibitors are used in the first-line medical management of AD, ICD, and ACD. Although topical corticosteroids are observed to be efficacious in ACD, however, the evidence surrounding their use in ICD remains unclear.[39,40] Topical corticosteroids are used in localized dermatitis involving less than 10% of the body surface area.[41] Steroids typically come in cream, lotion, or ointment formulations. The choice of steroid vehicle is influenced by the severity and location of the lesion, as well as the extent of xerosis (Table 2). High-potency corticosteroids should be avoided in intertriginous areas and areas of thin skin, including the face, flexural surfaces, eyelids, and the anogenital region because of the risk of cutaneous atrophy.[36] In addition, striae (stretch marks) and telangiectasia may result from prolonged use. Options for mild to moderate cases of dermatitis on the body include a triamcinolone acetonide 0.1% or a clobetasol propionate 0.05% cream or ointment.[36,42,43] Providers should expect resolution of the rash within several days following a once- or twice-daily application of topical corticosteroid.[44]

Steroid-sparing therapies include topical calcineurin inhibitors such as pimecrolimus 1% cream for mild or moderate disease and tacrolimus 0.03% to 0.1% ointment for moderate to severe disease.[43,45,46] Common adverse reactions associated with topical calcineurin inhibitors include local stinging and/or burning. In addition, topical calcineurin inhibitors contain a black box warning for increased lymphoma risk,[47] but postmarketing surveillance since the early 2000s has not substantiated this risk. Recent developments in topical therapies for the treatment of mild to moderate AD include crisaborole 2% ointment.[36,48] Intermittent use of these topical agents can be continued after the initial rash has been resolved, once or twice per week. Proactive use of mild-potency topical corticosteroids or topical calcineurin inhibitors once or twice per week is recommended to reduce the recurrence of flares.[36]

Systemic therapies (such as systemic steroids) are occasionally used in AD and ACD but not typically in ICD. Systemic corticosteroids are reserved for cases that involve greater than 10% of the body surface area.[41] In ACD, systemic corticosteroids are used in cases of severe poison ivy, disseminated ACD, or failure of topical therapies.[49] In AD, systemic steroids are reserved for acute, severe exacerbations as bridge therapy to topical corticosteroids. However, systemic corticosteroids are not recommended as a long-term treatment for dermatitis.

Narrowband UV-B phototherapy or psoralen and UV-A may be used in cases of AD and ACD. In particular, the use of narrowband UV-B phototherapy in patients with moderate to severe AD has resulted in significant clinical improvement.[50] However, its utility is limited by patient adherence, owing to the frequency of treatments and sparse location of treatment facilities.

Table 2.
EXAMPLES OF TOPICAL CORTICOSTEROIDS[44]

Mild Potency (Class 6/7)	Moderate Potency (Class 4/5)	High Potency (Class 1)
Desonide 0.05% Hydrocortisone 2.5%	Mometasone furoate 0.1% Betamethasone valerate 0.1% Triamcinolone acetonide	Betamethasone dipropionate 0.05% Clobetasol propionate 0.05%

Recently, a new systemic therapy for the treatment of AD has emerged. Dupilumab is an injectable systemic therapy used for patients recalcitrant to topical treatments.[51,52] However, there is currently no evidence for the use of these novel therapies in ACD or ICD.

CONCLUSION

Given the importance and prevalence of atopic and contact dermatitis within the modern context, establishing an accurate diagnosis is critical in guiding management. Managing these conditions involves education, avoidance of the provoking agent, and treatment of flares using topical agents. However, the efficacy of newly developed immunologic therapies in contact dermatitis is not well understood. Future research investigating the potential use of these treatments could provide new avenues for managing dermatitis.

PRACTICE PEARLS

- Atopic dermatitis, ACD, and ICD are common dermatologic conditions encountered in clinical practice.
- Atopic dermatitis most commonly presents in childhood and patients will often have a family history of the condition.
- Identifying and avoiding triggers are the cornerstone of treatment for ACD and ICD.
- Education, maintenance therapies, and a regular skin care routine with moisturizers are key to managing and decreasing episodes for all forms of dermatitis.
- Patients should avoid the use of high-potency corticosteroids in intertriginous areas and areas of thin skin because of the risk of cutaneous atrophy.

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