What Is the Role of Allergic Contact Dermatitis in Patients with Lower Leg Ulcers?

Laurie M. Parsons, MD, FRCPC, Medical Director, Sheldon Chumir Wound Clinic, and Clinical Associate Professor of Dermatology, University of Calgary, Calgary, Canada

CME
1 AMA PRA Category 1 Credit™

NCPD
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GENERAL PURPOSE: To analyze the relationship between contact dermatitis and delayed wound healing, discuss the diagnosis and treatment of lower leg contact dermatitis, and provide an algorithm for the patient with a red leg and delayed wound healing.

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 will:

1. Describe the nature of contact dermatitis.
2. Distinguish between allergic and irritant contact dermatitis and the other major differential diagnoses of delayed wound healing in this clinical scenario.
3. Outline the steps in the diagnosis of allergic contact dermatitis and irritant contact dermatitis and identify common haptens responsible for allergic contact dermatitis in patients with venous leg ulcers.
4. Apply the algorithm for delayed wound healing on a background of lower leg dermatitis.

ABSTRACT

Lower leg ulcers are a common clinical presentation to wound care clinics. They are often associated with the presence of dermatitis on the periwound skin, which can be a factor in delayed wound healing. Correctly diagnosing the underlying etiology is critical to reversing the breakdown in the skin barrier function. The author discusses allergic contact dermatitis as an etiology and describes the most common allergens, fragrances, and preservatives identified from a limited literature review. Patch testing is the criterion standard for the diagnosis of allergic contact dermatitis and is the most appropriate means of identifying causative allergens. An algorithm for the identification and treatment of lower leg dermatitis is provided to simplify the process.

KEYWORDS: algorithm, allergen, contact dermatitis, dermatitis, diagnosis, leg ulcer, patch testing

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INTRODUCTION

Lower leg ulcers (LLUs) are a common presentation to wound clinics throughout the Western world with an estimated prevalence between 1% and 7% of people older than 65 years.[1] Undiagnosed contact dermatitis (CD) and its associated periwound skin changes are a common occurrence with the apparent nature of CD not always easily discernible.[2] These changes include persistent erythema, scaling, and, depending on the acuity of the reaction, may also include edema, weeping, or blisters.

The probability of developing allergic CD (ACD) increases with the duration of venous leg disease and the incidence of sensitization overall; it is more prevalent in patients with venous leg ulcers (VLUs).[3,4] Periwound skin changes are a significant quality-of-life issue for patients,[5] and unrecognized and untreated periwound dermatitis slows wound healing.[6] Correct and timely recognition and treatment of the causes of periwound dermatitis are an essential skill for wound clinicians, requiring an understanding of the normal function of the skin as a barrier and the reaction patterns produced when the skin barrier fails.[7]

In this article, the author outlines the recognition and differential diagnosis of ACD, discusses the role of the skin barrier function, lists the most common sources of allergenic substances associated with ACD, and offers an algorithm for patients who present with dermatitis and a history of LLUs.

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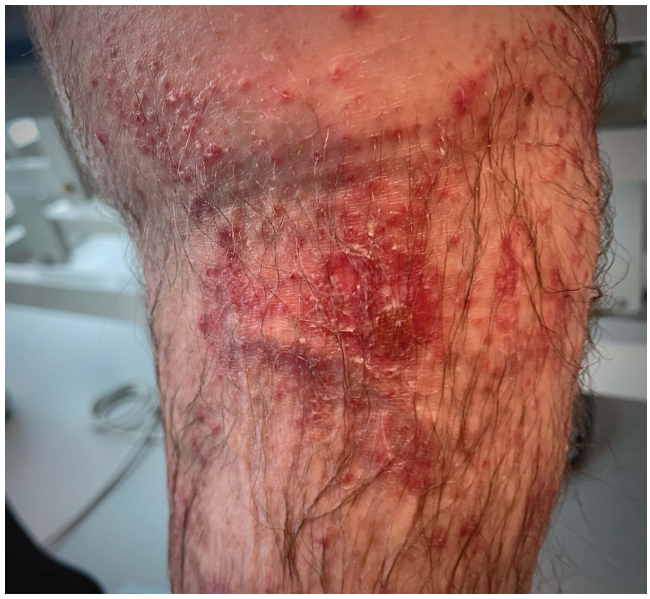
Figure 1. LOCALIZED DERMATITIS REACTION OF THE EPIDERMIS WITH ERYTHEMA AND SCALING



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DIFFERENTIAL DIAGNOSIS

Periwound dermatitis encompasses the most common skin changes seen in patients with LLUs and presents universally as reddened skin with varying degrees of epidermal changes such as scaling, weeping, and swelling (Figure 1). Patient complaints of burning or itching are common, and clinicians are faced with the differential diagnosis of reddened skin around their wounds. Although ACD is a common cause of periwound skin breakdown, it is not the only diagnosis to consider (Table 1).

Understanding the mechanism of epidermal damage in CD is imperative to identifying the underlying etiology. Contact dermatitis is an overarching term describing changes secondary to either the toxic or allergic nature of a substance applied directly to the skin. It is further subdivided into irritant CD (ICD; direct toxic effect) and ACD (type 4 allergy), with ICD being by far the most common.[8]

The occurrence of ICD is a result of direct injury to the skin and may even result in the death of keratinocytes. Patients with ICD most commonly complain of a burning sensation in their skin, whereas patients with ACD experience intense pruritus. The most common causes of ICD are irritants such as water and skin cleansers. In patients with ulcers, wound exudate can be a significant factor.[9] The major differences between ICD and ACD are outlined in Table 2.

In ACD, the reaction is an immune response to a topically applied small molecule (hapten) that penetrates the skin and couples with epidermal proteins, resulting in an antigenic substance.[10] The occurrence of ACD requires previous sensitization. Upon re-exposure, an intense pruritus develops with concomitant skin changes, including erythema, scaling, swelling, and often weeping.[11] Fluid-filled blisters may also develop as a direct result of this immune-mediated process. Clinically, ACD is indistinguishable from other causes of dermatitis, such as atopic dermatitis.[11] However, a subtle clue is the high degree of pruritus associated with ACD, which is significant compared with other etiologies.[12] The most common sources of these haptens are skin cleansers, moisturizers, topically applied antibiotics, dressings, and other topical medicaments such as pain medications and corticosteroids.[10,11,13,14]

Moisture-associated skin damage (MASD) is a form of ICD, with wound exudate playing a significant role in damaging periwound epidermis (Figure 2).[15] The importance of managing wound exudate with appropriate dressings and skin preparation materials has long been the most appropriate corrective measure to reduce MASD and is generally well understood by wound clinicians.

Table 1. DIFFERENTIAL DIAGNOSIS OF LOWER LEG DERMATITIS

Diagnosis	Clinical Features: Signs	Symptoms
Contact dermatitis - Allergic contact dermatitis (ACD) - Irritant contact dermatitis (ICD)	Dermatitis changes secondary to irritants (ICD) or allergens (ACD). Wound exudate as a source of moisture-associated skin damage plays a significant role.	- Redness, scaling, +/- weeping - Itching (ACD) - Burning, pain (ICD)
Cellulitis	Sudden onset of spreading unilateral erythema and swelling. In severe infections, fever and leukocytosis may be present.	- Pain, burning - In severe cases, skin necrosis and bullae
Stasis dermatitis	Hemosiderin staining, accompanying varicosities. Usually worse on the lower third of the leg. Skin changes usually lessen, moving proximally.	- Burning and/or pruritus - Redness, scaling, +/- weeping
Dermatophyte infection	Dermatitis secondary to dermatophyte infection of the skin. Look for concomitant signs of tinea pedis (maceration in fourth web space). Fungal scraping diagnostic.	- Pruritus - Redness, scaling
Trauma	Direct trauma to skin by adhesives and tape stripping/tears.	- Linear patterns of damage - Sudden onset and often witnessed

Table 2. DIFFERENTIATING CHARACTERISTICS BETWEEN ICD AND ACD

Characteristic	ICD	ACD
Onset	Hours to days	48–96 h
Symptom	Burning	Pruritus
Time to resolution	Days	Weeks
Mechanism of injury	Direct damage to keratinocytes	Immune-mediated dermal reaction with secondary epidermal changes
Common causes	Water, cleansers (alkalis), wound exudate	Haptens stimulating the immune response (see Table 3 for a list of the most common haptens)
Frequency	Common	Only 20% in the general population, but incidence between 46% and 82%[15] in patients with VLU

Abbreviations: ACD, allergic contact dermatitis; ICD, irritant contact dermatitis; VLU, venous leg ulcer.

Stasis dermatitis (SD) is commonly associated with VLU (Figure 3).[16] It is believed to be secondary to a low-grade inflammatory response in patients with long-standing lower leg edema.[17] Stasis dermatitis is generally more pronounced at the distal extremity and shows a propensity to lessen (personal observation) as it moves proximally.[18] As with ACD and dermatophyte infections of the skin, pruritus is the most common patient complaint.

Tinea pedis (TP), a dermatophyte infection of the uppermost layers of the epidermis, is common in the general population, with a male predominance and a slightly higher incidence in patients with diabetes.[19] Under favorable conditions, such as is seen with occlusive dressings and moist environments, the dermatophytes responsible for TP can spread onto the intact epidermis, especially under compression wraps, resulting in pruritus, redness, scaling, and weeping: the clinical picture of dermatitis.[18] This occurs more commonly in the presence of corticosteroid creams, which are often used to treat dermatotic skin conditions.[20] Screen recalcitrant LL dermatitis for the presence of dermatophytes with a fungal culture.[21]

Cellulitis of the LL (Figure 4) can occasionally present as LL dermatitis with redness, scaling, and weeping. It is more common, however, to present with sudden onset of unilateral spreading redness and pain.

These etiologies for LL dermatitis do not necessarily exist in isolation. The presence of ACD in the background of SD or a concomitant TP with ICD or any combinations of these conditions can coexist. Thus, the clinical pattern of dermatitis should be the starting point to investigate the cause of a patient's red and scaling rash.

SKIN AS A BARRIER

Intact skin is a significant barrier to pathogens and allergens. The cumulative effect of moisture, occlusion (from dressings and leg wraps), and skin breakdown increases the risk of developing ACD, ICD, SD, and dermatophyte infection.[3] Patients with pre-existing SD, ICD, or a primary skin problem such as atopic dermatitis have an increased risk of developing ACD.[22] Factors such as the allergenic potential of a molecule, the presence of disrupted barrier function, and an increase in exposure time can all increase the potential of developing ACD, even to allergens with a weak potential for sensitization.[23] A disrupted epidermis increases the risk of low-potency allergenic substances penetrating the skin, sensitizing an individual and resulting in ACD.

Occlusion of the skin increases the risk of developing both ICD and ACD. In ICD, skin occlusion by dressings or heavy ointments and medicaments can trap water in the uppermost layers of the epidermis, enhancing skin absorption and increasing the risk of both ACD and

Figure 2. WOUND EXUDATE CAUSING (A) EROSIONS AND (B) PERIWOUND MACERATION OF THE EPIDERMIS

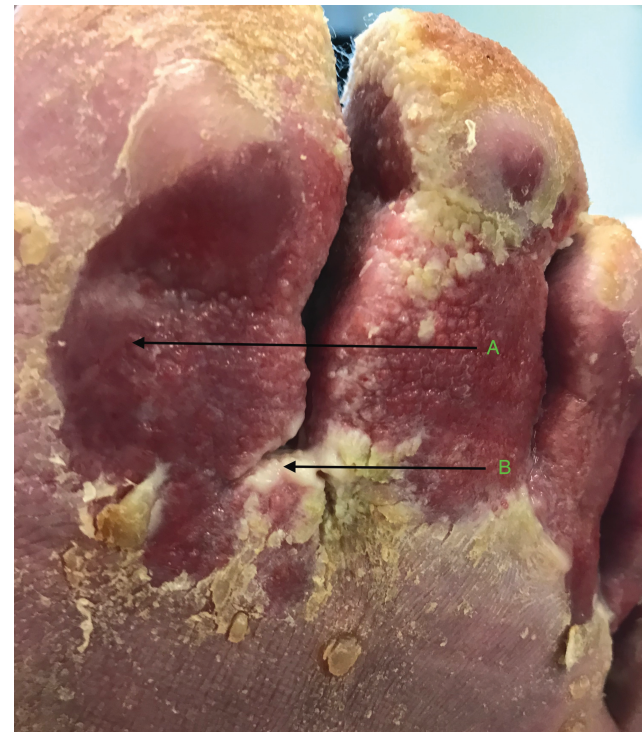

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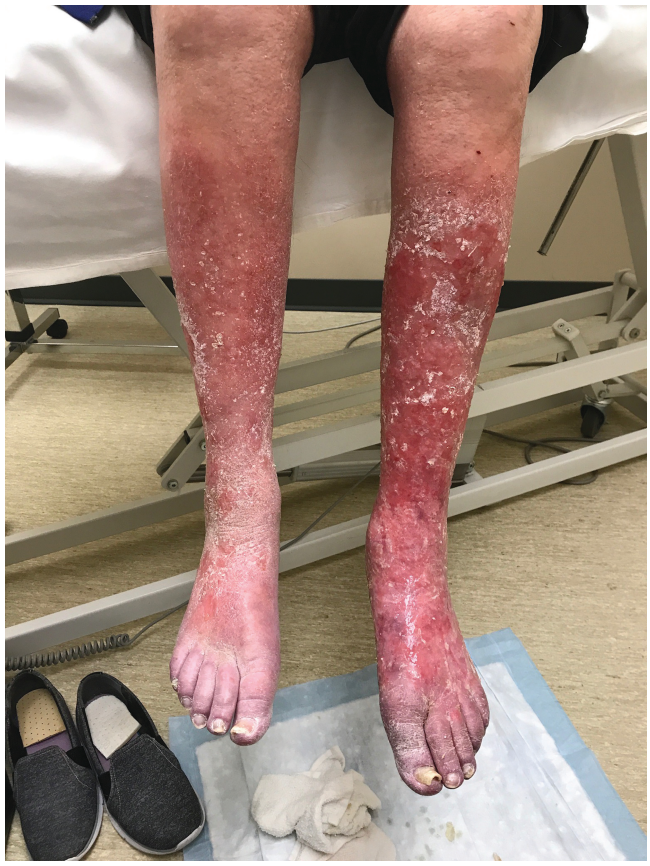
Figure 3. BOTH LOWER LEGS SHOW ERYTHEMA AND SCALING ON A BACKGROUND OF LOWER LEG EDEMA



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MASD. Van der Valk and Maibach[24] demonstrated conclusively that occluding sodium lauryl sulfate significantly increased the risk of ICD on intact human skin. Occlusion also increases the contact time of potential sensitizers, where the risk of penetration of these small molecules into the immune regions of the epidermis is more likely,[25] increasing the risk of developing a contact allergy.[26]

DIAGNOSING ACD

If ACD is suspected in a patient with VLU, the criterion standard of testing for the presence of sensitization is patch testing (PT).[10] This is a method of percutaneous exposure to known sensitizers, with a positive reaction showing the development of dermatitis in the area of exposure. Both standardized haptens and the patient's own products or dressing samples can be tested; however, as a general rule, only products meant to be left on the skin should be tested in a closed format, which involves placing tested substances or standardized haptens in a series of small wells on an adhesive patch. The patch is then adhered to uninvolved skin (usually the upper back) with a hypoallergenic adhesive tape (Figure 5).[27] Because of the delayed nature of positive reactions, results are read at 48 hours and 72 to 96 hours after application. The presence of a reaction in the area defined by the well as indicated by red, edematous, or blistered skin signifies a positive test to that substance.

Patients may also test known substances using the repeat open application test. A topical cream or other medicament is applied directly to a patch of skin (eg, the antecubital fossa) twice daily for 5 to 7 days. The development of redness, swelling, and pruritus signifies a positive reaction.[28] By this method, whole creams are tested, and a positive test cannot be interpreted as a reaction to a specific component of the tested substance.

For low-potency haptens or a suspected false-negative test, tape stripping of the test area on the upper back may be performed. Tape stripping is a means of reproducing the effect of dermatotic LL skin in the PT protocol and is indicated if LL dermatitis is suspected to be ACD to a low-potency hapten.[29]

When determining potential allergen sources for dermatitis in patients with VLU, consider all products that encounter the periwound skin. Moisturizers, skin preparations and cleansers, medicaments, topical corticosteroids, topical antibiotics, anesthetics, tapes, dressings, sutures, and instruments can all be sources of the allergy. Patients may also react to their own personal care products used at home, which, when occluded by dressings, can result in ACD. Patients may also be using herbal remedies, which can be a significant source of hidden sensitivity. Gilissen et al[13] reported on 15,980 patients who underwent PT between 1990 and 2016. They found that 1.4% of patients diagnosed with ACD tested positive to a herbal

Figure 4. CELLULITIS WITH (A) SKIN NECROSIS AND (B) EROSIONS

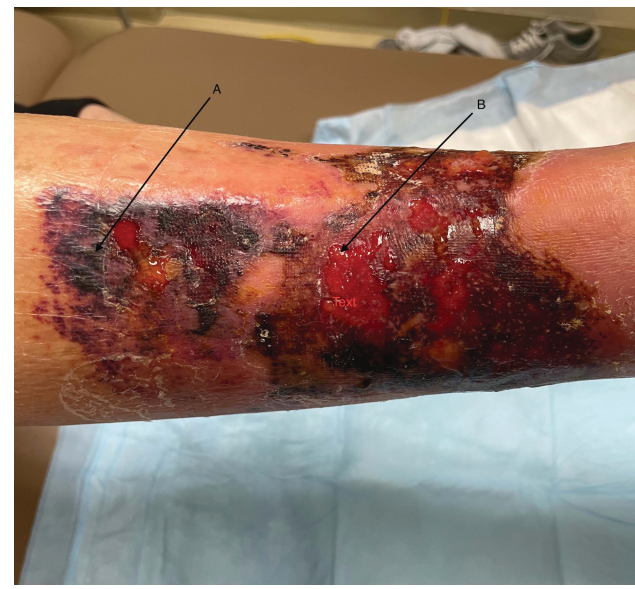

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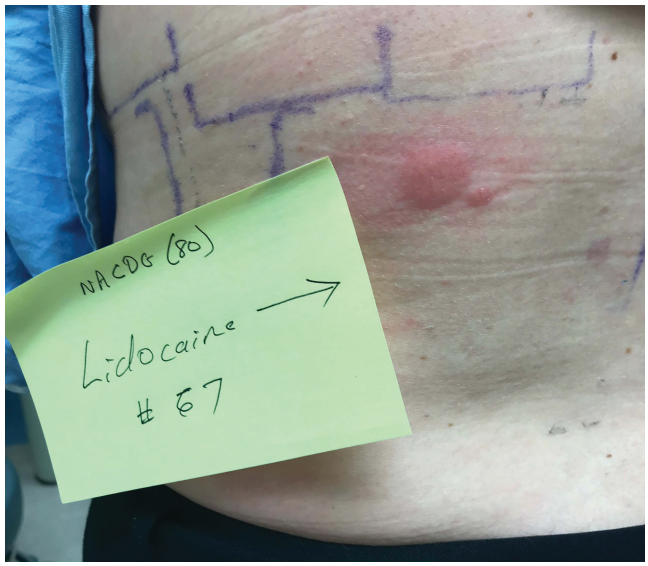
Figure 5. A POSITIVE PATCH TEST REACTION TO LIDOCAINE



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remedy, with fragrance allergens being the most common sources of the hapten.[13]

Haptens can be grouped into categories based on their properties and reason for inclusion in a dressing or a topical medication or lotion. Fragrances are a diverse group of haptens, with balsam of Peru or fragrance mix used most often as screening allergens with PT. Fragrances are added often as a masking agent for an otherwise unappealing odor in a product. Table 3 provides an overview of the most common groups of allergenic substances and their potential sources of exposure for patients with VLU.[2]

Overall, the most common haptens responsible for ACD are fragrances, lanolin, antimicrobials, and glues. Most of these haptens are found on the standard series of allergens for a particular region. A standard series is a grouping of common allergens that each country's or region's ACD society identifies as being the most relevant. This is important because components of topical therapies will differ from region to region. A standardized series should also be supplemented by whatever dressings and topical agents the patient uses for VLU therapy and skin care. Cleansers and other topical agents that are meant to be rinsed off and not used in direct contact with skin under occlusion should be patch tested using serial dilutions or in an open (uncovered) patch; irritant reactions are common and may be difficult to distinguish from ACD.[9]

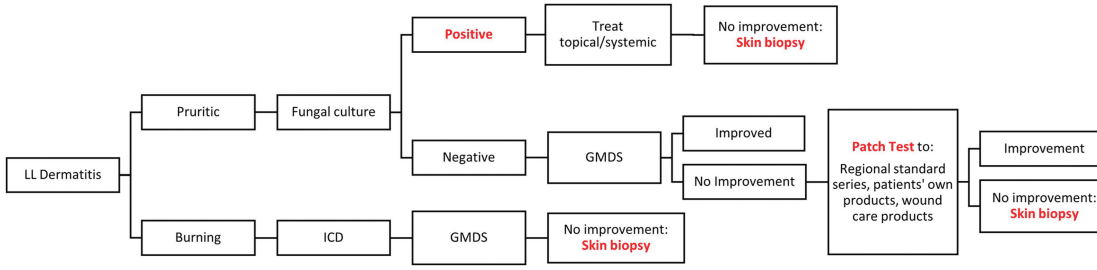
Multiple authors have published on the most common allergens responsible for ACD in patients with VLU, with variations in rates of sensitization based in part on local prescribing patterns.[30,31] The most common by far is fragrance; however, adhesives, rubber allergens, lanolin, topical antimicrobials, anesthetics, and topical corticosteroids, are close contenders.[2,32-35] Fragrances are well-known sensitizers, and because they are often a hidden component of skin cleansers, wound-dressing products, moisturizers, and topical antimicrobials, it is not surprising that they top the list.[34] The most common haptens found in topical antimicrobials are neomycin and bacitracin, which are components of many over-the-counter antibiotic creams.[2] Numbing agents for pain and itch are also hidden allergens; inspect a patient's topical medicaments for these components when a recalcitrant LL dermatitis has been identified.[36] Self-adhering dressings need adhesives, and colophony and acrylates are the most likely to cause ACD.[37] Although most of these haptens can be tested using regional standard series, it is always prudent to test using the patient's own products whenever possible.[31]

Table 3. OVERVIEW OF COMMON HAPTENS IN VLU: SCREENING ALLERGENS AND ALLERGEN SOURCES

Hapten	Sources	Screening Allergens
Fragrance	Masking fragrance: Wound cleansers, moisturizers, topical medicaments, cleansers	Balsam of Peru, fragrance
Antimicrobials	Fixative and bactericide: Wound cleansers, moisturizers, topical medicaments, pharmaceuticals, some preservatives, topical dressings, topical medications	Iodine, bacitracin, fucidin
Glues	Dressings, tape, moisture-trapping materials	Acrylates, colophony
Propylene glycol	Preservative and vehicle: Wound cleansers, hydrogels, moisturizers, topical medicaments, hydrogels	Propylene glycol, benzylkonium chloride, parabens
Rubber accelerators	Dressings, gloves, bandages	Thiuram, diacyl-thiourea, mercapto mix
Lanolin	Medicated ointments, moisturizers, waxes	Amercol, wool alcohol
Corticosteroids	Anti-inflammatory	Tixacortol-21-pivalate, budesonide
Anesthetics	Numbing agent	Benzocaine, lidocaine
Emulsifier	Emulsifies water and oil: Wound cleansers, moisturizers, topical medicaments	Cetyl stearyl alcohol
Dyes	Textile and fur dyes	P-phenylene diamine
Chromates	Various industrial uses: Fabrics, glues, detergents	Potassium dichromate

Abbreviation: VLU, venous leg ulcer.

Figure 6. DIAGNOSIS AND TREATMENT ALGORITHM FOR VLU-ASSOCIATED DERMATITIS



Abbreviations: GMDS, general measures for dermatitis skin avoidance of irritants (soaps, fragrances, excessive moisture) and use of moisturizers and barrier creams; ICD, irritant contact dermatitis; LL, lower leg; VLU, venous leg ulcer.

There are some restrictions to PT. The testing field requires a large expanse of normal skin that is free of eczema and tattoos. Moreover, the presence of immunomodulating medications or recent sun exposure of the testing field may result in false-negative reactions.[10] When testing a patient's own dressings, leave-on products may be tested directly as per the standardized PT protocol. Give careful consideration to testing rinse-off products such as cleansers because they can result in epidermal damage when placed under occlusion. With care, these can sometimes be tested as serial dilutions.[38,39] It is generally inadvisable to apply unknown substances for PT.[39]

Multiple Reactions

In a patient sensitized to one hapten, it is common to find multiple positive reactions. Up to 57% of patients will have two or more significant positive reactions.[2] Further, multiple sensitizers are often found concomitantly in specific patient products such as wound dressings, cleansers, and tapes.[2,30,32]

ALGORITHMIC APPROACH TO PERSISTENT VLU-ASSOCIATED DERMATITIS

When faced with a persistent LL dermatitis, a reasonable approach is to combine the clinical presentation of signs and symptoms with knowledge of the most likely diagnoses for this scenario. Figure 6 provides a framework for working through this differential diagnosis. Failure of the dermatitis to resolve within a reasonable timeframe, however, should alert the clinician to the possibility of an alternative diagnosis.

FUTURE CONSIDERATIONS

Currently, there is no standardization for labeling the composition of wound dressings, tapes, devices, or topically applied moisturizers or medicaments. Mestach et al[37] reported on two cases of ACD from acrylate-based adhesives in a catheter used to deliver chemotherapeutic agents intravenously. In 2019, Herman et al[33] reported on the difficulty of getting information about the composition of a continuous glucose monitoring device from the parent manufacturer when an ultrasonic bath preparation using components of the device tested positive with PT on suspect individuals. These patients had unfortunately tested negative using standardized series. Not knowing the composition of the products we use to heal can lead to significant morbidity in patients experiencing ACD, one aspect of which is diminished wound healing trajectories.[10,12,26,33] This major deficiency in product labeling is easily rectified and should be a primary concern of patients and healthcare systems. Unfortunately, material safety data sheets have a very limited role in identifying potential allergens. They are used to convey the presence of hazardous material and, as such, are not complete ingredients lists. A number of authors have also shown them to be inaccurate and misleading.[40,41]

CONCLUSIONS

Lower leg dermatitis is a common finding when treating patients with venous disease. It is important to exclude other common etiologies for these skin reactions and provide appropriate and timely treatments to minimize morbidities such as delayed wound healing. Understanding the clinical presentations of the most common etiologies for LL dermatitis, its diagnosis, and its treatments is important for standardized patient care and best practice outcomes. ●

PRACTICE PEARLS

- Dermatitis is not a diagnosis but a common clinical pattern seen as an epidermal response to injury resulting in inflammation. The clinical picture can include redness, scaling, and weeping.
- The etiology of an LL dermatitis may be multifactorial.
- Any recalcitrant LL dermatitis should be cultured and screened for the possibility of a dermatophyte infection.
- Occlusion (eg, dressings, heavy ointments) and preexisting broken skin both increase the risk of developing ACD.
- There are an average of 1.88 allergens in each wound care product, and 57% of patients have more than one identified sensitivity.[2,32]

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