CHAPTER 32

CONTACT DERMATITIS

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Contact dermatitis (CD) is an inflammation of the skin caused by exposure to certain substances in the environment.[1] The large amount of chemicals in the workplace, home, soil, and atmosphere are responsible for the high number of people affected by CD. A survey in California demonstrated that contact dermatitis was responsible for 95% of all occupation-related dermatologic problems, and for 30% of all illnesses in the workplace.[2,3] This disorder commonly affects the foot and ankle, and is frequently misdiagnosed due to a high number of similar lesions affecting the lower extremities.[1] Too often, the patient with contact dermatitis is diagnosed with athlete's foot and dispensed antifungal cream for a number of weeks.[1,4] This approach often fails and in some case aggravates the eczematous eruption by causing an allergic response to the chemicals in the medication. Fortunately, a minimal knowledge of dermatology is required to successfully diagnose and treat this disorder.

CLINICAL PRESENTATION

Contact dermatitis is an eczematous reaction caused by direct or indirect exposure to an environmental substance. Many substances found in the environment may cause CD of the feet, however certain materials such as leather, dyes, glues, and rubber have a higher prediction for initiating the reaction.[3,5] Topical medications and skin care products can also initiate CD.

The classic clinical manifestation of acute CD includes bullous formation, weeping vesicles, and severe erythematous lesions resembling cellulitis. Pain and edema are present in many cases. A secondary bacterial infection often aggravates the presentation of acute CD. In contrast, chronic CD is characterized by redness of the skin, xerosis, scaling, crusting, lichenification and pruritus.[6]

The intensity of the skin inflammatory reaction varies depending on the concentration of the substance in contact with the skin, the patient's degree of sensitivity to the substance, and frequency of exposure. Other factors that may alter the degree of reaction include mechanical stress, skin thickness, perspiration and T-cell reaction (Table 1).[1,3,6] Patients with hyperhidrosis tend to have a more severe reaction compared to those with dry feet, since a moist environment increases the likelihood and the severity of CD by increasing the percutaneous penetration of substances that initiate the reaction.[1]

Table 1

EFFECT OF CERTAIN FACTORS ON THE SEVERITY OF THE REACTION

FACTORS	INCREASED SEVERITY OF REACTION	DECREASED SEVERITY OF REACTION
Skin Thickness	Thin skin	Thick skin
Concentration of Substance	High concentration	Low concentration
Duration of Exposure	Increased time of exposure	Decreased time of exposure
Mechanical Factor	Skin mechanically irritated	
Patient's Condition	Pre-existing allergies	Immunosuppressed
Environmental Factors	Hyperhidrosis	Xerosis

IRRITANT VERSUS ALLERGIC CONTACT DERMATITIS

Two varieties of contact dermatitis exist: irritant contact dermatitis (ICD) and allergic contact dermatitis (ACD) depending on whether the substance causing the dermatitis acts as an irritant or an allergen. Clinically, these two reactions appear identical, however they do differ upon close examination (Table 2).[4,7] Therefore it is important to carefully examine the patient and differentiate contact dermatitis resulting from irritation to that caused by an allergen.

Primary ICD is the most common subtype of contact dermatitis. It is responsible for 80% of all cases of CD encountered.[4] Generally, it frequently tends to affect the very young and the very old because of their lower skin irritation threshold, however a strong irritant may affect everyone.[3,6] There are many types of caustic agents responsible for causing ICD (Table 3). When an alkaline substance comes in contact with the skin, the external emulsifying substance extracts the protective lipids from the epidermis.[6] Similarly, if the irritant is acidic, the skin proteins are denatured and the epidermis is disrupted.[3] Once the skin's protective epidermal layer is damaged, or when its function is compromised, exposure to a strong or weak irritant will cause a non-immunologic inflammatory reaction.

Table 3

HIGHLY IRRITATING PRODUCTS

Acids
Alkalis
Oils
Detergents
Amines
Solvents
Petroleum products

Table 2

ALLERGIC CD VS. IRRITANT CD

	IRRITANT CONTACT DERMATITIS	ALLERGIC CONTACT DERMATITIS
People at risk	Everyone in contact with the substance	Genetic predisposition required
Mechanism of response	Non-immunologic	Type IV delayed hypersensitivity
Number of exposures	One	One or more
Nature of substance	Organic solvents, irritants, soaps	Allergen
Concentration of substance	High	Low
Mode of onset	Gradual	Rapid (12-48 hours after re-exposure)
Distribution	Sharply marginated	Spreads beyond the contactant
Severity of the reaction	Reaction fades after removal of irritant	Reaction increases or persists after removal of the allergen
Symptoms	Burns	Itches
Management	Protection of skin against irritant	Complete avoidance
Histology	80% PMN 20% Lymphocytes	20% PMN 80% Lymphocytes

Allergic contact dermatitis is a type IV delayed hypersensitivity reaction caused by an immunologic reaction to a specific allergen that comes in contact with the skin. There are two phases to ACD: the sensitization phase and the elicitation phase.[6] In the first phase the patient is sensitized to the causative allergen and no reaction is developed. Low-molecular-weight allergens, called hapten, penetrate the epidermal barrier and combine with keratinocytes and Langerhans' cells proteins to form an antigen.[6] Langerhans' cells act as an epidermal reticuloendothelial meshing, which combine to the hapten-protein complexes and present the complexes to the T-lymphocytes in the epidermis. Chemotactic factors, produced by the T-lymphocytes, are then directed to the T-cells in the paracortical region of the lymph nodes where the formation of sensitized T-cells occurs.[3,6] This phase generally occurs over a 10 to 30-day period depending on the integrity of the epidermal barrier and the agent's sensitizing index, which is the capacity of the agent to cause an allergic reaction.[4,7]

Once the sensitization phase is completed, the allergic reaction is launched. The elicitation phase is initiated upon further exposure of the skin to the antigen protein complex. These complexes cause the sensitized T-cells to release lymphokines, which recruit other inflammatory cells.[3] These various inflammatory cells are responsible for the rapidly developing inflammatory reaction. Generally, 12 to 48 hours are required for a sensitized individual to develop a cutaneous reaction on re-exposure to an allergen.[6]

Although cross reactivity to a similar antigen is uncommon, some cases have been reported. The inflammatory reaction is initiated by a hapten of similar chemical structure to the original sensitizing hapten.[3] The immune system is unable to differentiate between the two similar antigens and develops an allergic response.[6]

DIAGNOSIS

The patient's history is very crucial when evaluating someone with suspected CD. The patient should be questioned about recent changes in shoes, socks/stockings, laundry detergent, topical medications, lotions, occupations, hobbies, and climate. The time and mode of onset of the lesion is an important factor that may lead to proper diagnosis of the disease. A familial or personal history of atopy, allergy or eruption of similar dermatological lesions should be noted.

The patient's body must be carefully examined for similar lesions, and particular attention should be paid to the involved areas of the lower extremities. It is important to appreciate the pattern of distribution, location, color, morphology and symmetry of the lesions.[1] The morphology and the color of the lesions are helpful to specifically determine if the condition is acute or chronic. The distribution of the lesions most often corresponds to the shape and location of the offending substance. For instance, if a certain chemical involved in the socks sensitizes the skin, then both feet will be involved at the location where the skin is in contact with the socks (Fig. 1).

Any involvement of the nails, plantar aspect of the feet, and interdigital space should be noted. Contact shoe dermatitis is most often bilateral, symmetric, and spares both the interdigital area and the plantar aspect of the feet. Conversely, fungal infection asymmetrically affects the inter-digital spaces, the plantar aspect of the feet and the nails.[1,7] Evaluation of the patient's socks and shoes to determine their condition, and the material they are made of concludes the thorough examination. Old shoes are often responsible

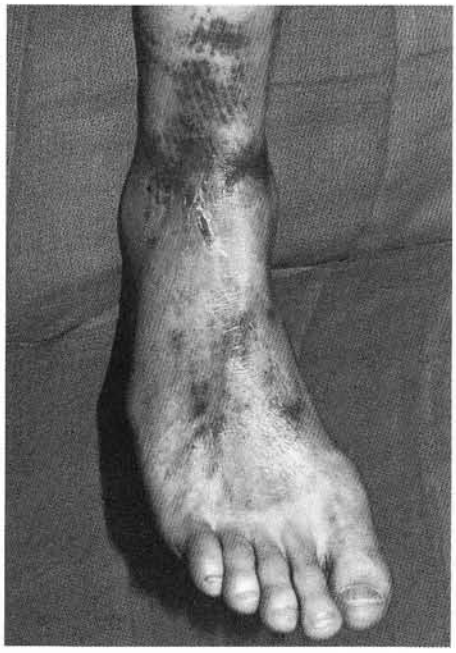

Figure 1. Irritant contact dermatitis caused by a detergent used in the socks. The dermatitis is well-demarcated and localized to the area covered by the socks.

for initiating a reaction since they may contain environmental substances that have soaked into the shoe or sock over a period of time.[8]

When ACD is suspected, patch testing should be performed. The patch test works on the basis that a cutaneous eruption will be produced with the application of the proper antigen to the skin. It is the only reliable test to confirm the diagnosis of ACD and to identify the causative allergen. ICD can be identified according to the appearance of the rash and the presence of a negative patch test.[1]

The patient's condition must be evaluated before the test can be performed. Testing should be deferred on a patient with an intense contact dermatitis since contact with the offending allergen may exacerbate the cutaneous reaction. Office-made tests are performed by cutting a two-centimeter square of the suspected material. If the material is composed of multiple layers, each layer must be separated from each other, moistened and applied to the skin using a hypo-allergenic tape.[1] Preferable areas include non-hairy areas such as the upper back and the upper arm. Patches are removed 48 hours following the application, and the results are read 72 to 96 hours after the application of the test. The most difficult part of this test is to maintain the patient's cooperation since many materials may have to be applied to the patient's skin and time is required on the part of the patient.[9] Interpretation of the results may be difficult and subjective. Contrarily, the standard patch test series is easier to use and requires minimal experience to interpret the results.[1]

The T.R.U.E. (Thin Layer Rapid Use Epicutaneous, Glaxo-Wellcome, Inc. Research Triangle Park, NC) test is a commercially available standardized allergy panel that identifies allergy-causing substances (Fig. 2). It is a safe, simple and objective diagnostic test that verifies the patient's reactivity for 80% of the chemicals responsible for ACD.[1,10] Each test consists of twelve polyester patches on two hypoallergenic tapes with a dehydrated hydrophilic gel attached to a water-proof outer layer. Twenty-three of the polyester patches are coated with a film containing the specific allergen and the remaining polyester patch is a negative control patch used to interpret questionable reactions.[11,12]

T.R.U.E. test requires no preparation; it is applied directly to healthy skin on the patient's back making sure that each allergen makes firm contact with the skin. Each panel is applied in a similar fashion on each side of the vertebral column (Fig. 3). The skin should not be cleansed with anything but normal sterile saline, if necessary.[6] The test is left in place and kept dry for the following 48 hours. On the following visit, the panels are removed and the skin is kept free from contact with any chemical until it is interpreted 72 hours following the application. The 24-hour period elapsed between the removal of the panels and the reading of the test, allows the body to develop an allergic reaction.[13]

Finally, an identification template is used to provide a quick and accurate reading of the causing allergen. Two reactions reflect a true positive reaction: the extreme positive reaction and the strong positive reaction both eliciting a papular, vesicular erythema with infiltration. Coalescing vesicles and bullous formation may be seen with an extreme positive reaction. A weak reaction probably does not reflect a true reaction.[14] False

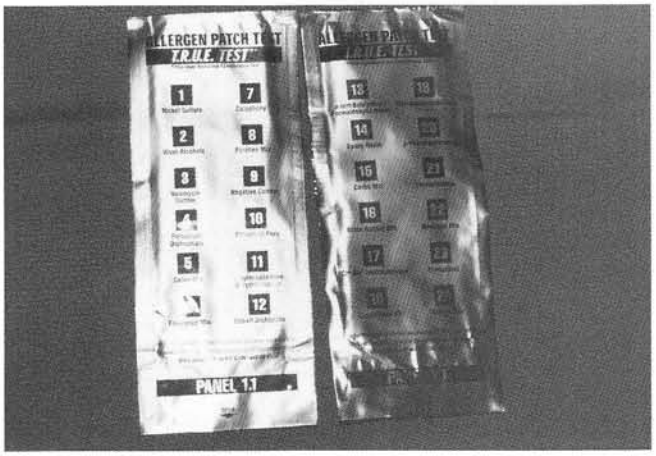

Figure 2. T.R.U.E. test's two panels of twelve polyester patches.

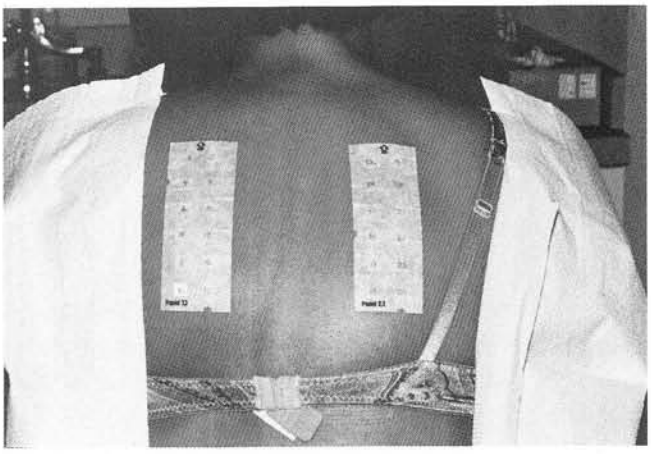

Figure 3. The two panels applied to the patient's back on each side of the vertebral column.

positive results present themselves through a faint macular homogeneous erythematous reaction without inflammation. They are most often caused by strong reaction to the adjacent allergen, irritable skin syndrome, or poor storage and aging of the material.[15] Should there be any doubt regarding false positive results, the test should be repeated at a new site after a 3 week waiting period.[13] A study by Flori et al. suggested administration of 5mg/kg/day of cyclosporin A to enable hyper-reactive patients with false positive reaction to be patch tested reliably.[16]

True negative results are obtained in 25% to 60% of all cases.[10] It indicates that the patient is not sensitive to 80% of all allergen causing ACD. Such information minimizes the need to restrict the patient from using certain products. If the symptoms persist, other pathologies including ICD, urticaria or various other types of eczema should be considered, along with the need to refer the patient to an allergen expert for further testing.

The chance of having a false negative reaction is relatively low when a standardized test is used. There are several causes, other than an incorrect use of the test, for a false negative result. Several positive reactions may be delayed for more than 72 hours following the application of patch test. The patients should be aware that if a delayed reaction occurs, they must contact the physician.[13] False negative results may also be caused by certain medications. Oral cortisone must be discontinued for two months or longer before patch testing can be performed.[6] Oral antihistamine may also cause a false negative reaction for up to four weeks following the last dose.[1] Topical steroids should not be applied to the area where the patch test is to be placed on the skin, since it may cover a positive reaction. Therefore it is of utmost importance to ascertain that the patient has not been taking any cortisone or antihistamine for a sufficient period of time in order to maximize the accuracy of the test.

TREATMENT

The therapeutic plan is first directed at eliminating the causative agent. Then, carefully chosen topical or systemic medications are used to treat the affected patient depending on the stage of the rash. With an acute inflammation, the goal is to reduce the inflammation and dry the blistered skin. Cold, wet compresses applied to the bulla are highly effective. These compresses are used for 15 to 30 minutes every two hours for the ensuing one to three days.[3,6] Complete immersion of the affected areas in Burrow's solution (1:10 concentration) or permanganate soaks (1:5000 concentration) for 15 minutes, three to six times daily, is highly soothing.[10,17] Drying creams, calamine lotions and fluorinated steroid creams can be used for a short time but should be discontinued after 2 to 3 days since they may cause excessive drying of the skin.[3] Ointments and other greasy agents should not be used in this stage since they do not penetrate through blisters.

Pressure caused by the fluid-filled blisters may be painful. Some authorities advocate relieving the pain by puncturing the bulla to release the enclosed fluid. It is advised not to completely remove the entire top of the blister as it acts as a protection for the inflamed underlying skin.[2]

In the chronic stage of contact dermatitis, treatment is aimed at decreasing the scales, the inflammation and the pruritus. After vesiculation and drainage have ceased, fatty topical medications such as ointments are the hallmark of therapy.[7]

A short course of oral systemic anti-inflammatory and antipruritic medications may be used for severe widespread inflammation. Antihistamines such as hydroxyzine and diphenhydramine control pruritus and facilitate sleep. The newer agents such as loratadine, cetirizine hydrochloride and astemizole have less sedative effect but are not as efficacious.[3] Systemic corticosteroids are indicated in patients with severe contact dermatitis.[6] Prednisone, administered in a dosage of 20 mg to 30 mg twice a day for one complete week, may be used if no relative contraindications such as diabetes, peptic ulcer disease, hypertension, or pregnancy are present.[4] To prevent adrenal suppression, corticosteroids should be delivered in a decreasing dose even when administered for a short period of time.[3]

In the case of CD caused by shoe material, the entire shoe may not need to be discarded. Removal of one part of the shoe may be sufficient to terminate the symptoms. When this method fails to work then shoes made with materials to which the patient is not allergic may be purchased.[8]

Control of hyperhidrosis for patients with shoe dermatitis is important. The importance of a daily change of socks cannot be overemphasized. In the sensitized patient, controlling perspiration

may decrease the severity of the pruritic rash. Absorbent powders such as Z-Sorb or Drysol are examples of highly effective antiperspirants that can be used.[1]

Antibiotics for gram-positive organism may be useful if an infection is suspected in conjunction with CD. Topical steroids should be avoided in patients with infected lesions.

CONCLUSION

CD is a cutaneous reaction that can be debilitating. Identification of the causative agent through a complete history and a thorough examination is important. Frequently, the use of a standardized patch test is needed to properly diagnose the cutaneous rash. Depending on the distribution, location and severity of the dermatitis, other conditions to consider in the differential diagnosis of CD are atopic dermatitis, pustular eruption, psoriasis, herpes simplex, infections, insect bites, parasitic infestations, fungal infections, nummular eczema, erythema multiforme, and cutaneous drug reaction.[4,10] The prognosis of CD is very good. In a child, the allergic reaction to a certain substance may disappear or become less intense with time. Unfortunately, in adults, desensitization to an allergen rarely occurs and they must avoid contact with the offending substance.

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