Dermatology Clinic

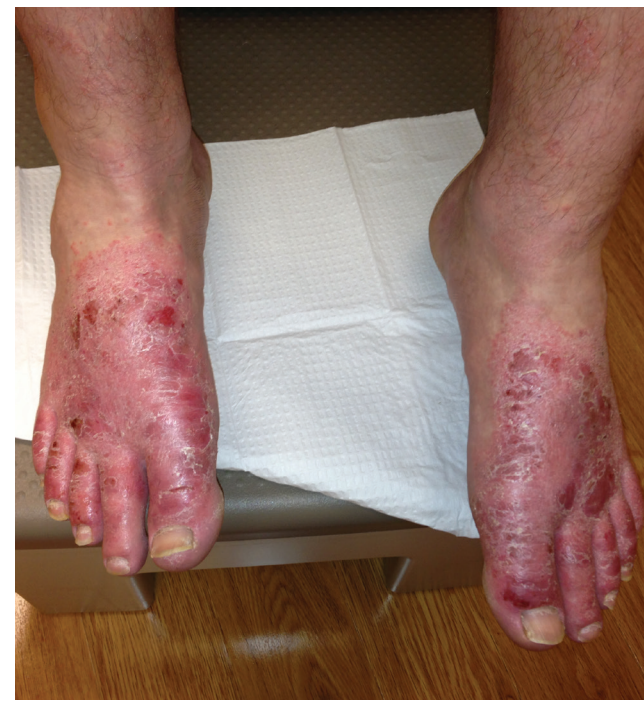
CASE #1

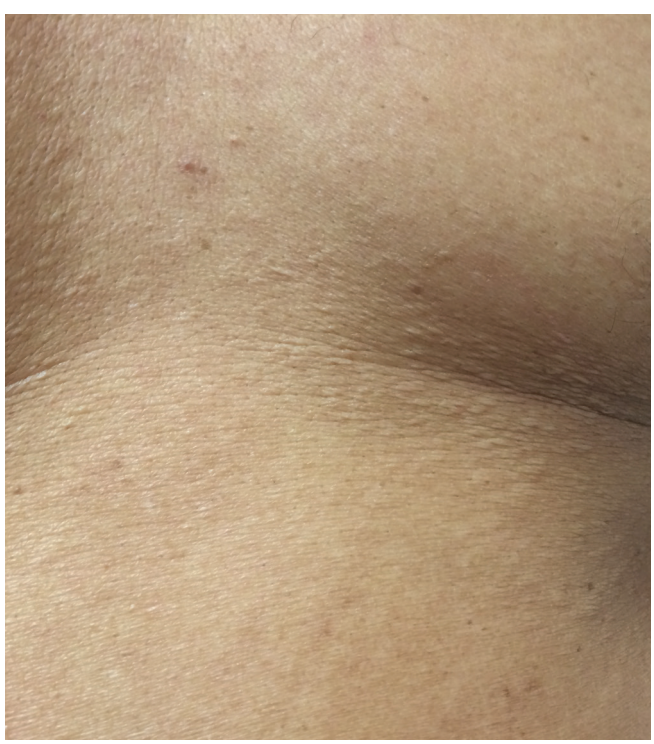
An itchy rash on the feet

ANNA GREGOIRE, BS, AND TIFFANY SHIH, MD

A 25-year-old man with no remarkable past medical history presents with an itchy rash on the feet. Symptoms began 2 months ago, starting over the dorsal toes with redness, scaling, and itching. The eruption spread over the dorsum of the feet and progressed to weeping and crusting. He reports recently buying new shoes for work as a chef, which requires 15 hours daily of standing. Due to profuse sweating, his socks become soaked daily. Examination reveals symmetric erythematous plaques over the bilateral dorsum of both feet with fissures, scaling, and lichenification. No blisters or vesicles are present, but there are scattered crusted erosions.

What is your diagnosis? Turn to page 76





CASE #2

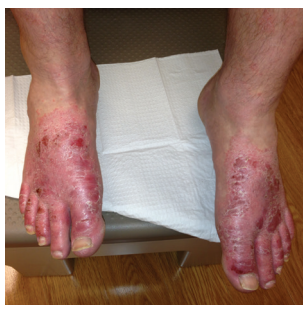
Fine wrinkles on the neck, chest, axilla, and back

JULIE NGUYEN, BS, AND MAURA HOLCOMB, MD

A 55-year-old black man presents for evaluation of fine wrinkles that had developed over the past year. Physical examination reveals diffuse areas of fine wrinkling that run parallel to lines of cleavage on his neck, chest, axilla, trunk, back, and upper arms bilaterally. The affected skin has normal pigmentation and lacks erythema, induration, and atrophy. Lateral tension obliterates the wrinkling. The patient never notes any inflammation, itching, or pain. There are no signs or symptoms of systemic involvement. He denies sunbathing and denies any history of skin disorders or other significant health problems.

What is your diagnosis? Turn to page 77

CASE #1 Shoe dermatitis



Shoe dermatitis represents a form of allergic contact dermatitis (ACD) that varies in frequency and presentation based on environmental factors, such as climate conditions, cultural traditions regarding footwear, and diverse manufacturing techniques.[1] As a form of ACD, shoe dermatitis is classified as a delayed type IV hypersensitivity reaction (cell-mediated). The pathogenesis of ACD requires an initial sensitization phase, with exposure to a chemical, which penetrates the skin and elicits a cascade of events. Following sensitization, subsequent exposure leads to antigen presentation to "primed" T cells, causing a release of cytokines and chemotactic factors, triggering the clinical picture of contact dermatitis.[2]

Shoe dermatitis often starts on the dorsal toes. This initial involvement with the dorsum of the toes and then feet is attributed to greater surface area, thin stratum corneum, and sustained contact with the upper portion of the shoe.[3] Often, the eruption presents as erythema, lichenification, and weeping and crusting in severe cases. With extended contact, areas of exposure may become xerotic with more prominent scaling. After resolution, postinflammatory hypo- and hyperpigmentation may occur.[2] Pruritis, burning, and pain present as the most commonly reported symptoms.[3] Secondary bacterial or fungal infections frequently occur. In severe cases, an id reaction may occur, resulting in an eruption on the hands. The distribution is important, often with symmetric involvement and sparing of the toe webs, which have no contact with the offending agent. The distribution shown in this case is classic. Another pattern of shoe dermatitis involves the sole of the foot, with sparing of the instep and flexural creases of the toes. Patients with hyperhidrosis and atopy are at increased risk for development of shoe dermatitis.[4]

Common offending allergens include rubber accelerators, 2-mercaptobenzothiazole, carbamates, tetramethylthiuram disulfide, potassium dichromate in leather, and adhesives in synthetic materials.[1,2] One report also described a case of shoe dermatitis resulting from the allergen colophonium.[5] Foam rubber padding in athletic shoes contain diisocyanates, which are thought to be a causative agent. Other possible offenders include cork liners, felt, formaldehyde, dyes, asphalt, dimethyl fumarate, and tar.[2] The allergen, in conjunction with the environment within shoes, leads to susceptibility to shoe dermatitis. Moist environment, chronic venous insufficiency, friction, and heat can exacerbate foot dermatitis. It is more common in patients who wear occlusive footwear, including factory workers and military personnel, to have a higher incidence of shoe dermatitis.[3]

Patch testing is the gold standard for diagnosis of shoe dermatitis. Patch testing with pieces of various shoe parts may be done by soaking them for 15 minutes in water with application to the back for 72 to 96 hours. It is important to apply patches on disease-free skin, test all footwear, and use materials of at least 1 cm and less than 2 mm in thickness. Gas chromatography, mass spectroscopy, and high-performance liquid chromatography have also been used in cases of shoe dermatitis to identify the offending agent.[3] Once the allergen is identified, selection of shoes without the offending substance leads to resolution. This is a difficult process, as most shoes are made in areas without mandatory labeling requirements. Plastic, wooden, or fabric shoes, which contain fewer allergens, are often impractical.[3]

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Alternative diagnoses to consider with ACD that clinically present similarly include irritant dermatitis, dyshidrosis, psoriasis, insect bite reaction, tinea pedis, lichen planus, juvenile plantar dermatosis, Sézary syndrome, and mycosis fungoides.[2,3] Irritant contact dermatitis occurs with rubbing of footwear against the feet or from contact with irritants, including detergents, soaps, cement, or topical medications. The main difference is that irritant contact dermatitis has no immunologic mechanism involved in the response.[3] Dyshidrosis, or dyshidrotic eczema, can also present as scaly pink patches, but often is isolated to the palms and occasionally the soles of the feet. Patients often develop blisters or vesicles in ACD and dyshidrosis, which can also be seen in bullous tinea pedis and some exuberant insect bite reactions. Thinner plaques of psoriasis and mycosis fungoides can also look similar but are not limited to the acral surfaces. Therefore, history of new contacts to allergens is essential for diagnosis. If there is concern about an alternative diagnoses, a biopsy should be performed.[2,5]

ACD, including shoe dermatitis, is usually self-limited after removal of the causative agent; most cases resolve in days to weeks. However, due to a lack of product information, identifying and minimizing contact with the causative agent presents a challenge to treatment. Treatment for specific allergens includes

chromium-free leather for patients allergic to chromium or switching to new leather shoes every few months. Patients with allergies to components of work boots are advised to wear two pairs of socks or alternate different pairs of boots daily. Those allergic to rubber can replace the insoles with cork composite or felt using a nonrubber cement adhesive. Custom-made shoes represent another option, although additional expense is associated with this option.[3] A newer treatment option is the use of barrier socks developed to prevent skin contact with shoe allergens. Hyperhidrosis should also be treated, because a moist environment may exacerbate ACD. Medical treatment options include occasional use of topical and oral corticosteroids, as well as immunosuppressive treatments such as cyclosporine or mycophenolate mofetil.[3,4] Secondary bacterial and fungal infections may also require treatment.[5]

For the patient in our vignette, we asked him to avoid wearing his new shoes, as they were highly suspected to be the cause of the dermatitis. Because severe hyperhidrosis presented a problem at work, we asked him to change into dry socks whenever he soaked through the socks he was wearing and to use aluminum chloride, an antiperspirant, to help with his perfuse sweating. The patient did not have significant superimposed infection such as *Staphylococcus* colonization or tinea pedis; however, such infections must frequently be treated with vinegar soaks, bleach baths, mupirocin ointment, or antifungal creams. Finally, we prescribed fluocinonide ointment twice daily to quickly reduce the inflammation. Because the skin is thick and often hyperkeratotic in this condition, it requires a strong class I or II topical corticosteroid. The patient improved quickly over the next few weeks and returned with complete resolution.

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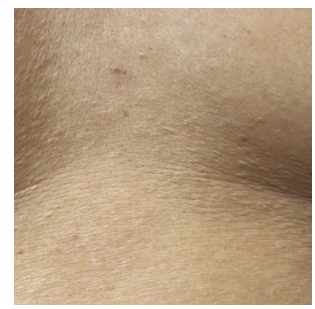
CASE #2 Mid-dermal elastolysis



Mid-dermal elastolysis (MDE) is a rare acquired elastic tissue disorder that is characterized clinically by well-circumscribed patches and plaques of fine wrinkles and histopathologically by focal loss of elastic fibers in the mid-dermis.[1,2] MDE frequently manifests on the trunk and proximal extremities. The clinical appearance of MDE is variable and may include perifollicular papular protrusions and inflammatory skin changes. There is a significant sex predominance observed in MDE; most cases are in white women of reproductive age, and the median age of onset is 34 years.[2] MDE is exceedingly rare; fewer than 100 cases have been reported in the literature since its first description in 1977.

MDE is a chronic condition in which the only clinical manifestation is fine wrinkling that gives the skin a prematurely aged appearance. MDE is categorized into types according to the observable skin changes: well-circumscribed patches of fine wrinkles arranged parallel to skin cleavage lines (type I), small soft papular lesions composed of perifollicular protrusions (type II), or persistent reticular erythema with wrinkling (type III).[2,3] The most frequently affected sites are the trunk, neck, and proximal aspects of the extremities; the face and hands are notably spared of wrinkling.[2,3] Most patients with MDE present with asymptomatic, well-demarcated, symmetrically arranged lesions of fine wrinkling that follow Blaschko lines, a normally invisible pattern that represents pathways of epidermal cell migration during development.[1,2]

The size of the lesions can vary from a few centimeters in diameter to much larger, diffuse areas that involve the entire back. The affected skin generally lacks pigmentary changes, erythema, scaling, induration, atrophy, and telangiectasia.[3,4] Patients typically do not experience any itching, burning, or pain, although some report mild erythema. MDE appears to be a localized cutaneous disorder that has no associated systemic involvement. Once the lesions appear, they remain relatively stable in morphology for life.

Sporadic cases have been reported in which the onset of MDE is preceded or accompanied by an inflammatory dermatosis; for most patients, however, there is no history of skin disorders. Reported associations include urticaria, atopic dermatitis, granuloma annulare, pityriasis rosea, Sweet

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