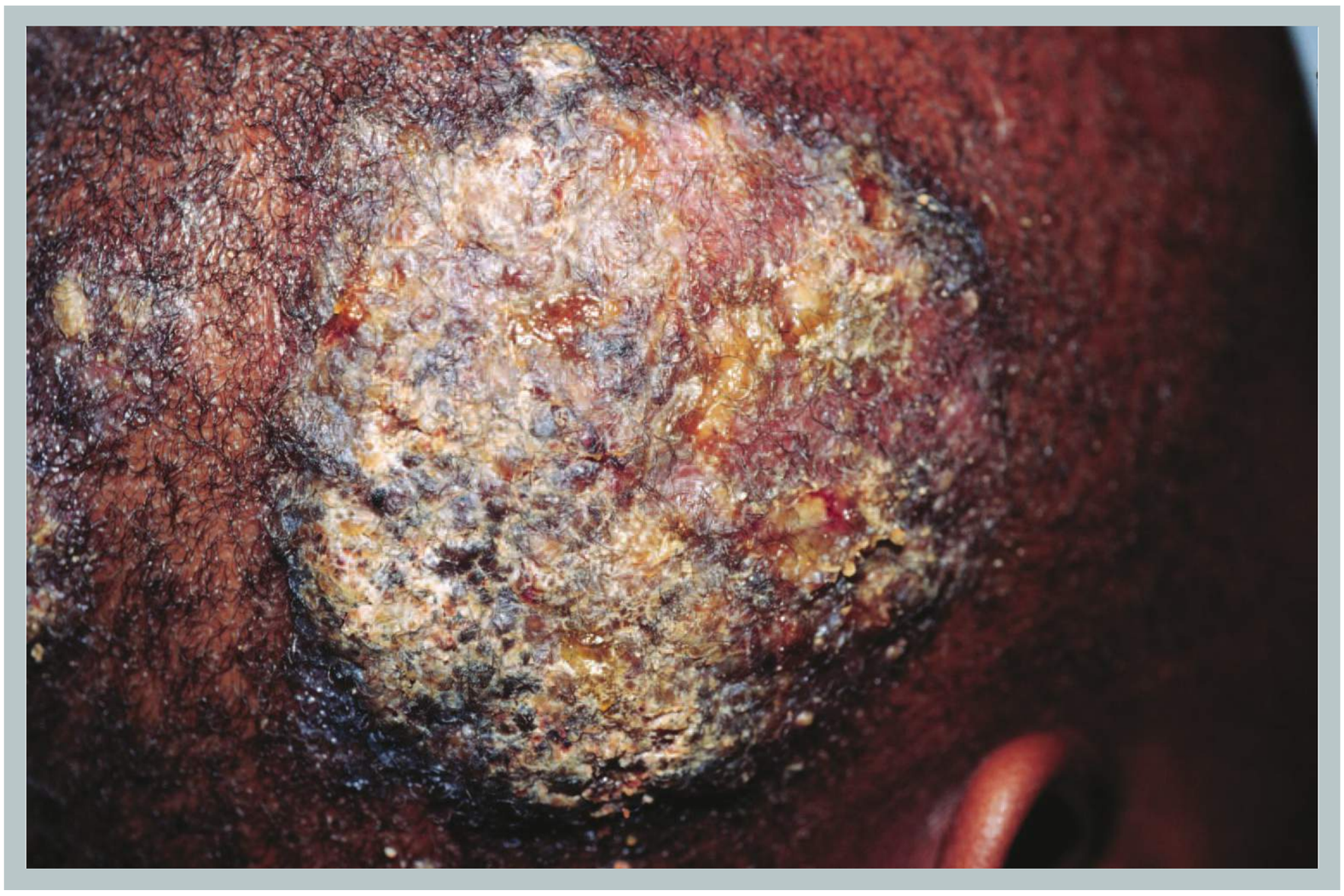
Chapter 13

# CUTANEOUS CONDITIONS

J. Matthew Hardin



**Kerion.** (Photo contributor: Alan B. Storrow, MD.)

The author acknowledges Christopher R. Sartori, Michael B. Brooks, and Sean P. Collins for portions of this chapter written for the first and second editions.

# STEVENS-JOHNSON SYNDROME/TOXIC EPIDERMAL NECROLYSIS

## Clinical Summary

Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) are two ends of a continuum of life-threatening, reactive diseases. Generally, SJS involves epidermal detachment of less than 10% body surface area (BSA) and TEN greater than 30%; TEN and SJS overlap between 10% and 30%. Two or more mucosal sites are usually affected. The overall mortality of SJS is 1% to 5%, while that of TEN approaches 30%.

SJS/TEN begins with a nonspecific prodrome of upper respiratory tract symptoms, fever, fatigue, myalgia, headache, and mucous membrane and skin sensitivities. A rash appears 1 to 3 days later with mucosal lesions first (erythema, erosions, and hemorrhagic crusting) and a simultaneous or lagging, generalized, dusky, erythematous rash. Bullae form, large sheets of epidermis separate from the dermis, and the involved skin is exquisitely tender to palpation. The Nikolsky sign is present when lateral pressure on unblistered skin causes the epidermis to slide off. Progression of involved skin can occur over a single day or slowly evolve over 14 days. In addition to the generalized "skin failure," life-threatening sepsis, respiratory failure, metabolic derangements, and gastrointestinal hemorrhage may occur. These serious complications may be compounded by underlying comorbidities.

With a few exceptions, SJS/TEN results from a drug exposure, generally within 7 to 21 days previous to the prodrome. Sulfonamide antibiotics, aromatic anticonvulsants (phenytoin, phenobarbital, and carbamazepine), penicillins, nonsteroidal anti-inflammatory drugs (NSAIDs), allopurinol, lamotrigine, and antiretrovirals are common causes, although over 200 medications, including over-the-counter (pseudoephedrine) and herbal remedies, have been implicated. Mycoplasma pneumoniae and vaccinations have also been associated with SJS/TEN.

## Management and Disposition

Stop the offending medication (sometimes requiring withholding all). Assess airway status and secure admission to a burn intensive care unit. SJS/TEN patients require careful attention to fluid management; burn protocols may overestimate fluid requirements and contribute to pulmonary edema (resuscitation should maintain urine output at ~0.5-1 mL/kg/h). Dress denuded skin with gauze bandage rolls and moisten with normal saline (transition to antibacterial solution in the burn unit). Emergently consult dermatology and ophthalmology. Meticulous supportive care is the foundation of treatment. With dermatology and burn unit consultation, consider intravenous immune globulins (IVIg) within 72 hours.

## Pearls

1. The only intervention proven to decrease mortality is to stop the offending medication.
2. If a patient notes continued skin pain, even in areas of normal skin, expect additional sloughing.
3. High-risk groups to develop TEN include cancer/hematologic malignancies, HIV/AIDS, immunosuppression, slow acetylator genotypes, anticonvulsant use associated with radiotherapy, and specific human leukocyte antigen alleles (HLA-B*1502 associated with carbamazepine and HLA-B*5801 associated with allopurinol).
4. Infections and pulmonary edema/acute respiratory distress syndrome (ARDS) are the leading causes of death in TEN patients.

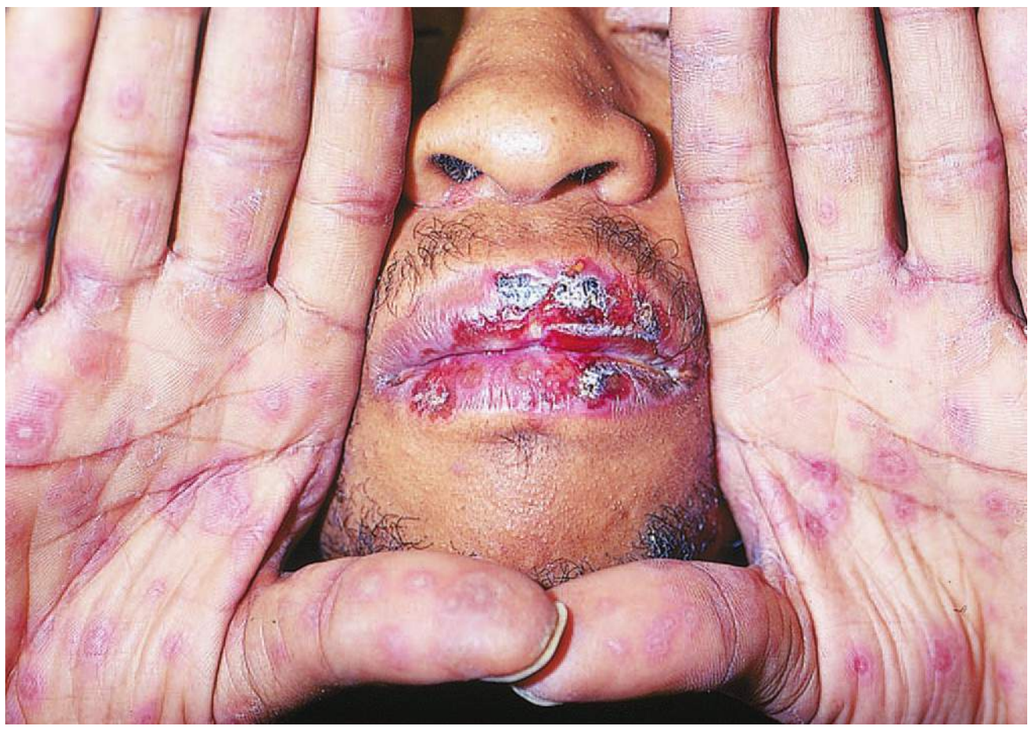


FIGURE 13.1 ■ **Stevens-Johnson Syndrome.** Moderate hemorrhagic crusting on the lips and target-like macules on the palms and fingers—similar to erythema multiforme. (Photo contributor: Alan B. Storrow, MD.)

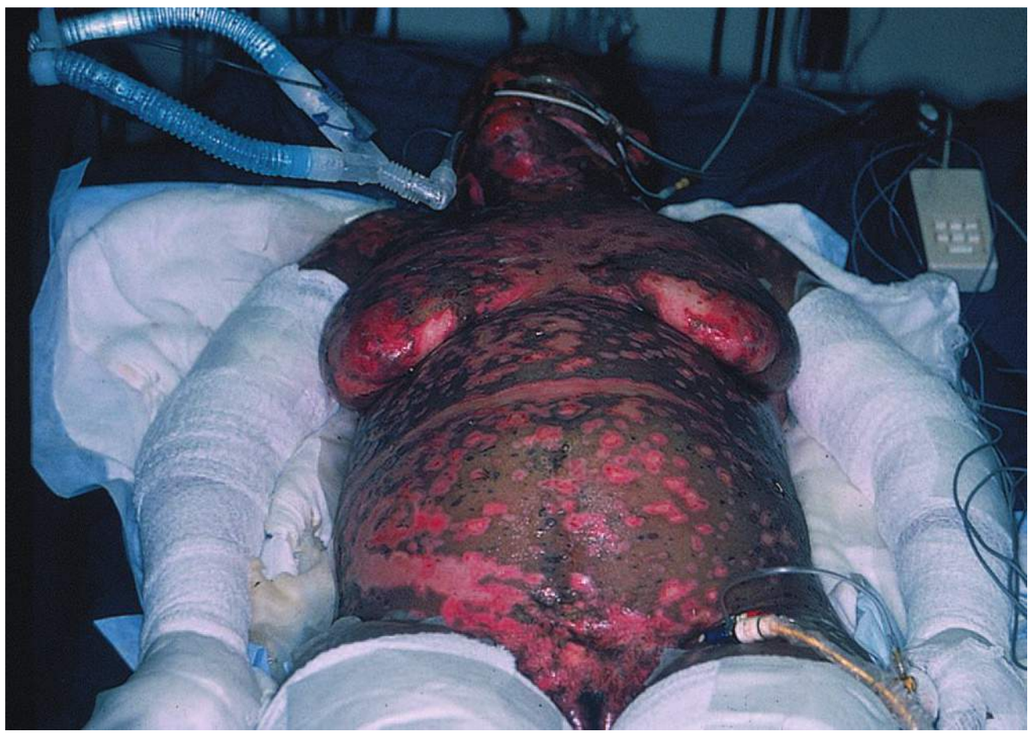


FIGURE 13.2 ■ **Toxic Epidermal Necrolysis.** Widespread desquamation. (Photo contributor: James J. Nordlund, MD.)

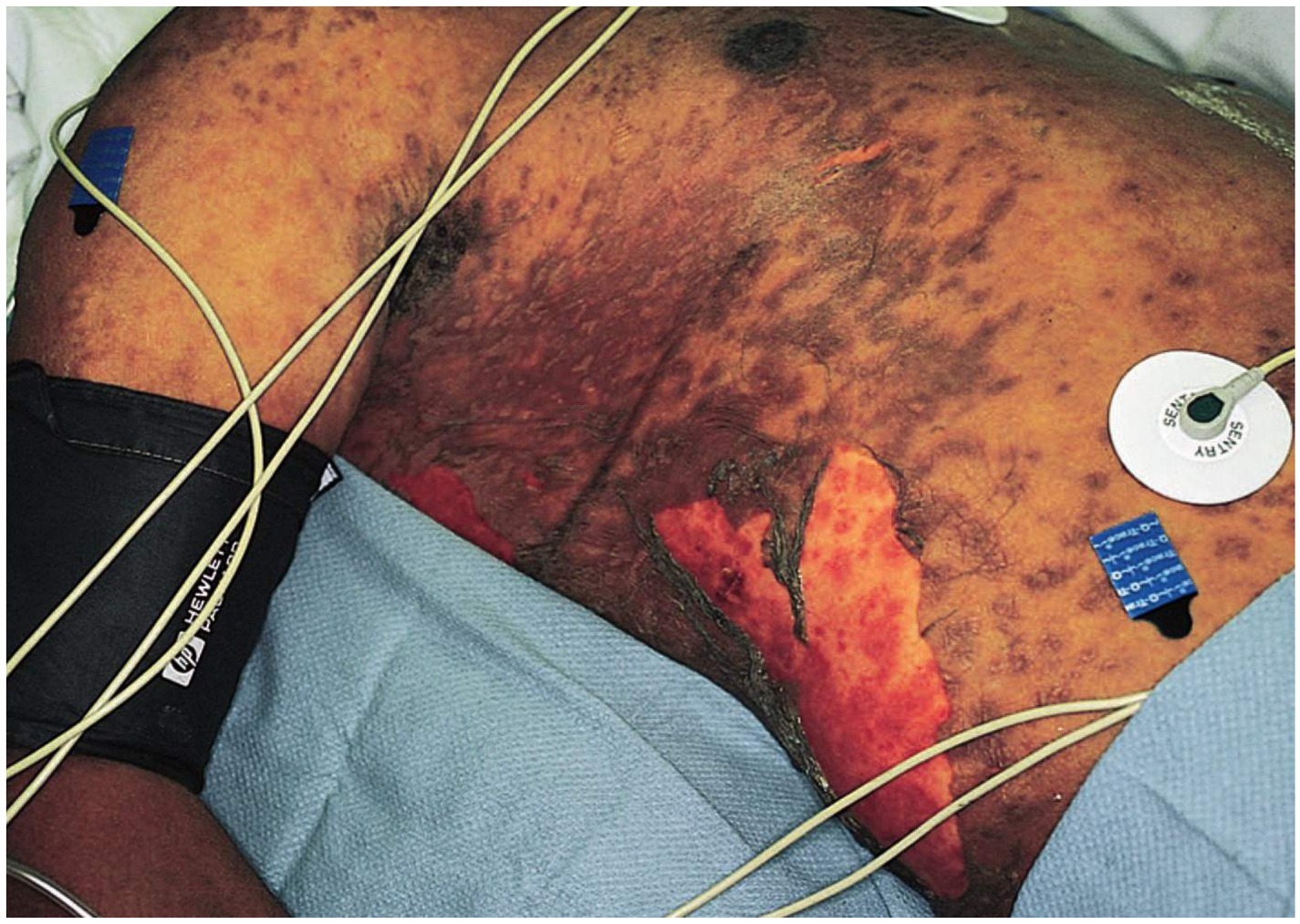


FIGURE 13.3 ■ **Toxic Epidermal Necrolysis.** Extensive epidermal desquamation. In darker skinned patients, the initial macules/patches may be hyperpigmented and erythema difficult to appreciate. (Photo contributor: Keith Batts, MD.)

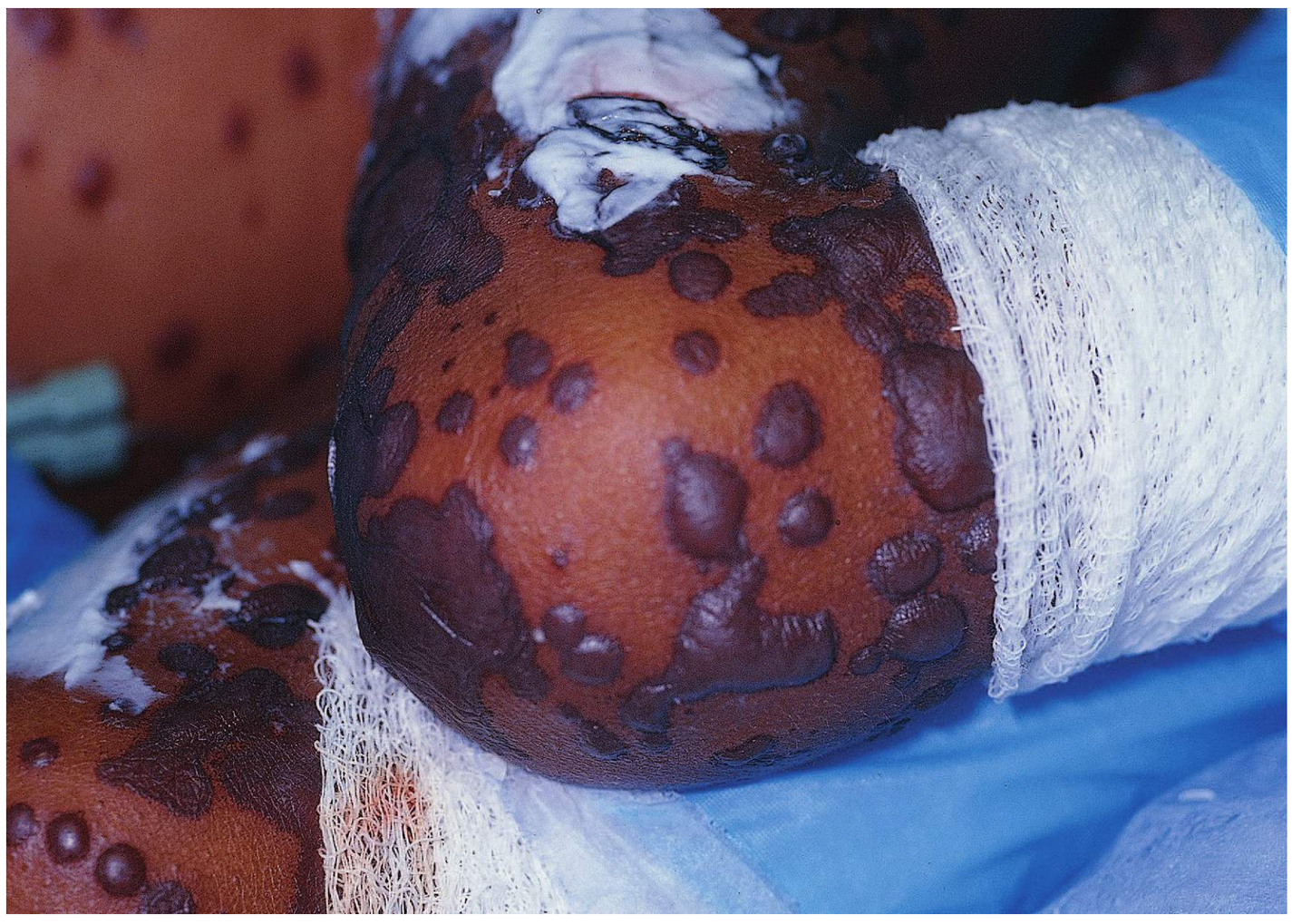


FIGURE 13.4 ■ **Bullous SJS/TEN.** A bullous form of TEN. (Photo contributor: J. Matthew Hardin, MD.)

# ERYTHEMA MULTIFORME

## Clinical Summary

Erythema multiforme (EM) begins with symmetric, erythematous, sharply defined extremity or trunk macules, and evolves into a "targetoid" or "bull's eye" morphology (a flat, dusky, central area with two concentric, erythematous rings). Bullae may appear in the central dusky area (bullous EM). The mucous membranes, typically oral, may become involved and raise concern for SJS. The typical targetoid lesions allow a diagnosis to be made clinically (bullae, purpura, and mucosal involvement should prompt a dermatology consultation). The rash usually persists for 1 to 4 weeks.

Herpes simplex virus (HSV; frequently labialis) is strongly associated but may not be clinically apparent. Other viruses, bacteria (M pneumoniae, Chlamydia, Salmonella, Mycobacterium), and fungi (Histoplasma capsulatum, dermatophytes) are also associated. Medications account for <10%; NSAIDs, sulfonamides, antiepileptics, allopurinol, and antibiotics are responsible for the majority. Physical factors such as trauma, ultraviolet light exposure, and cold have been reported to elicit EM.

## Management and Disposition

Prevention of HSV recurrences is essential. Antivirals administered after lesions present have minimal clinical impact, but patients should be referred for future prophylaxis consideration. Use of facial sunscreens and lip balms may help prevent UVB-induced recurrences. Systemic steroids are discouraged but can be considered in atypical presentations. With the distinctive clinical findings and no systemic symptoms, patients may be discharged home. Systemic symptoms and atypical presentations require admission and dermatologic consultation.

## Pearls

1. EM does not progress to TEN.
2. Reassure patients that the lesions will completely resolve without scarring. Hypopigmentation and hyperpigmentation may occur but should resolve over months.
3. Eye involvement requires a slit-lamp examination and ophthalmologic consultation to exclude active HSV infection.
4. Patients with immunosuppression are more prone to recurrent and prolonged episodes.
5. The dusky centers help differentiate EM from typical exanthematous drug reactions (no dusky centers) and urticaria (normal central zone skin).

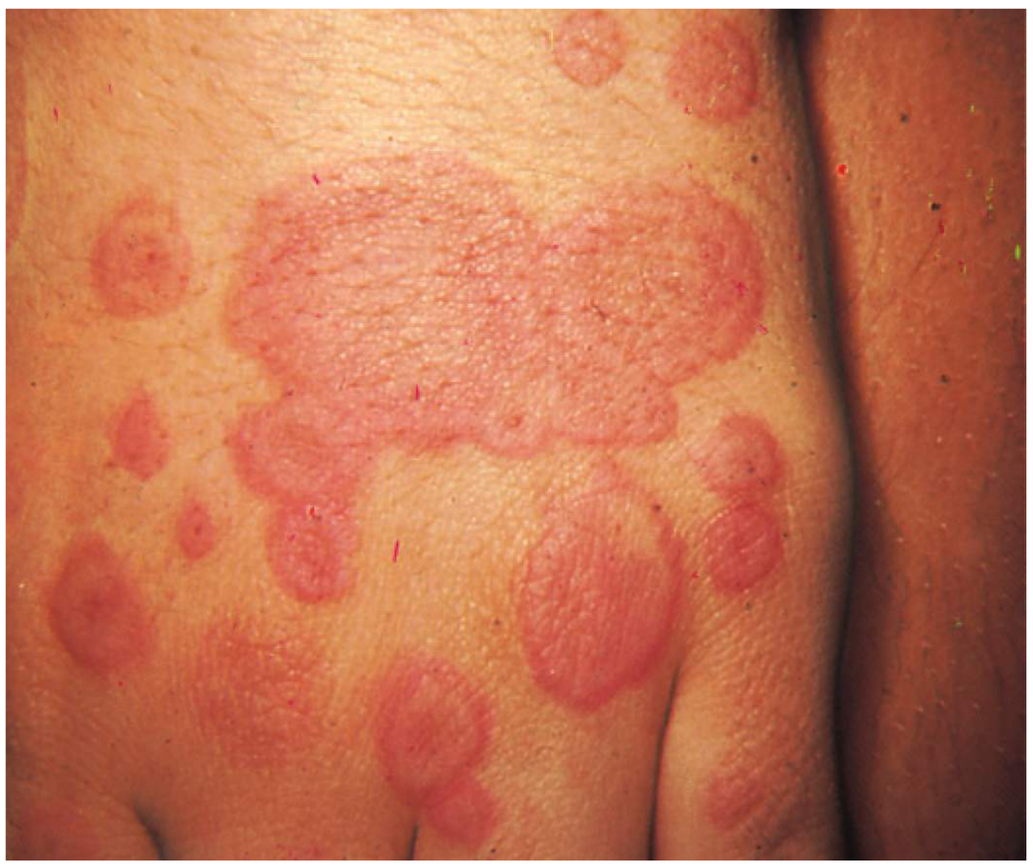


FIGURE 13.5 ■ **Erythema Multiforme.** Wrist, hand, and fingers with typical central dusky centers surrounded by the concentric "bull's eye" rings. (Reproduced with permission from Weinberg S, Prose NS, Kristal L. Color Atlas of Pediatric Dermatology. 4th ed. New York, NY: McGraw-Hill; 2008: Fig. 13-9.)

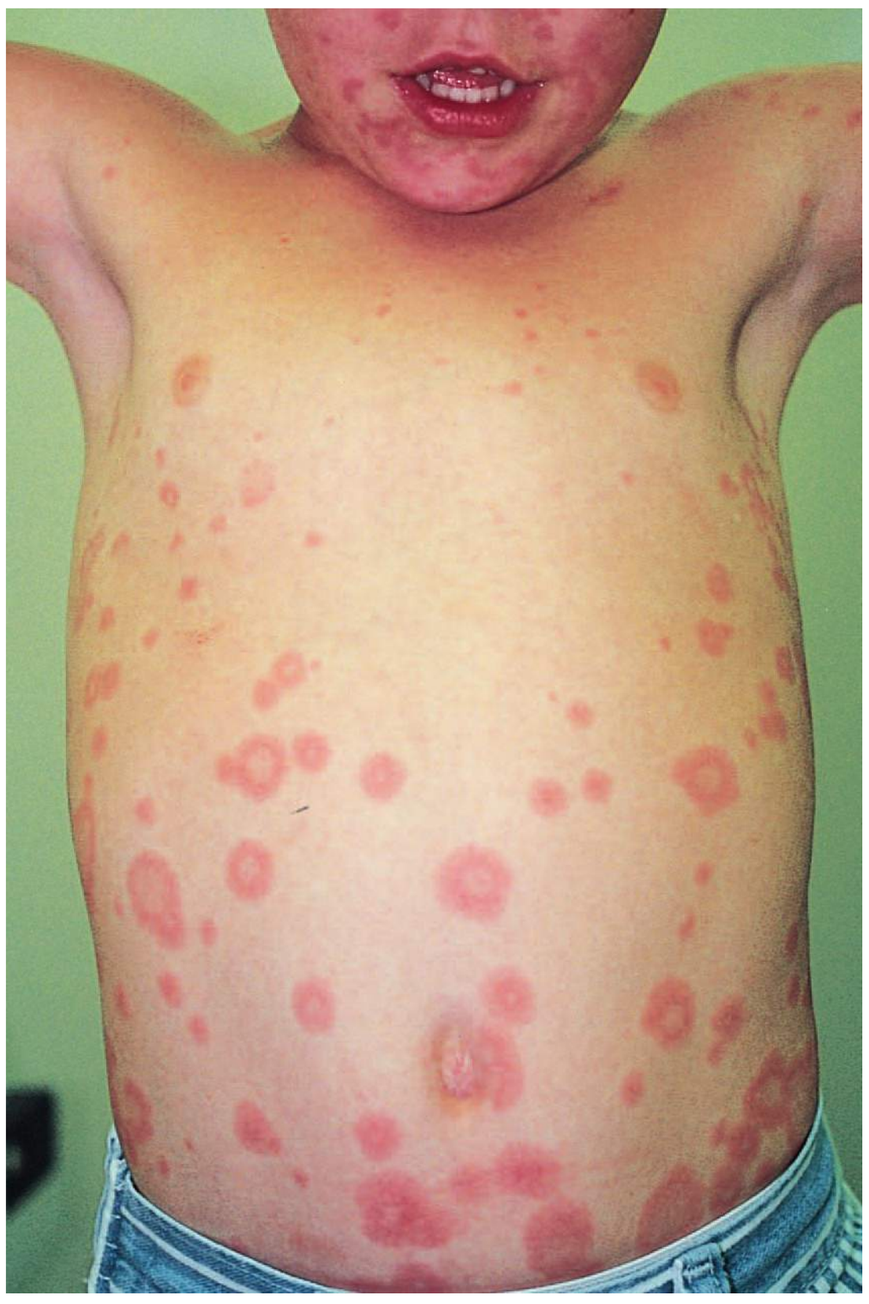


FIGURE 13.6 ■ **Erythema Multiforme.** Symmetric distribution of targetoid macules and plaques. The dusky central zone is more obvious on the left waistline lesions. (Photo contributor: Michael Redman, PA-C.)

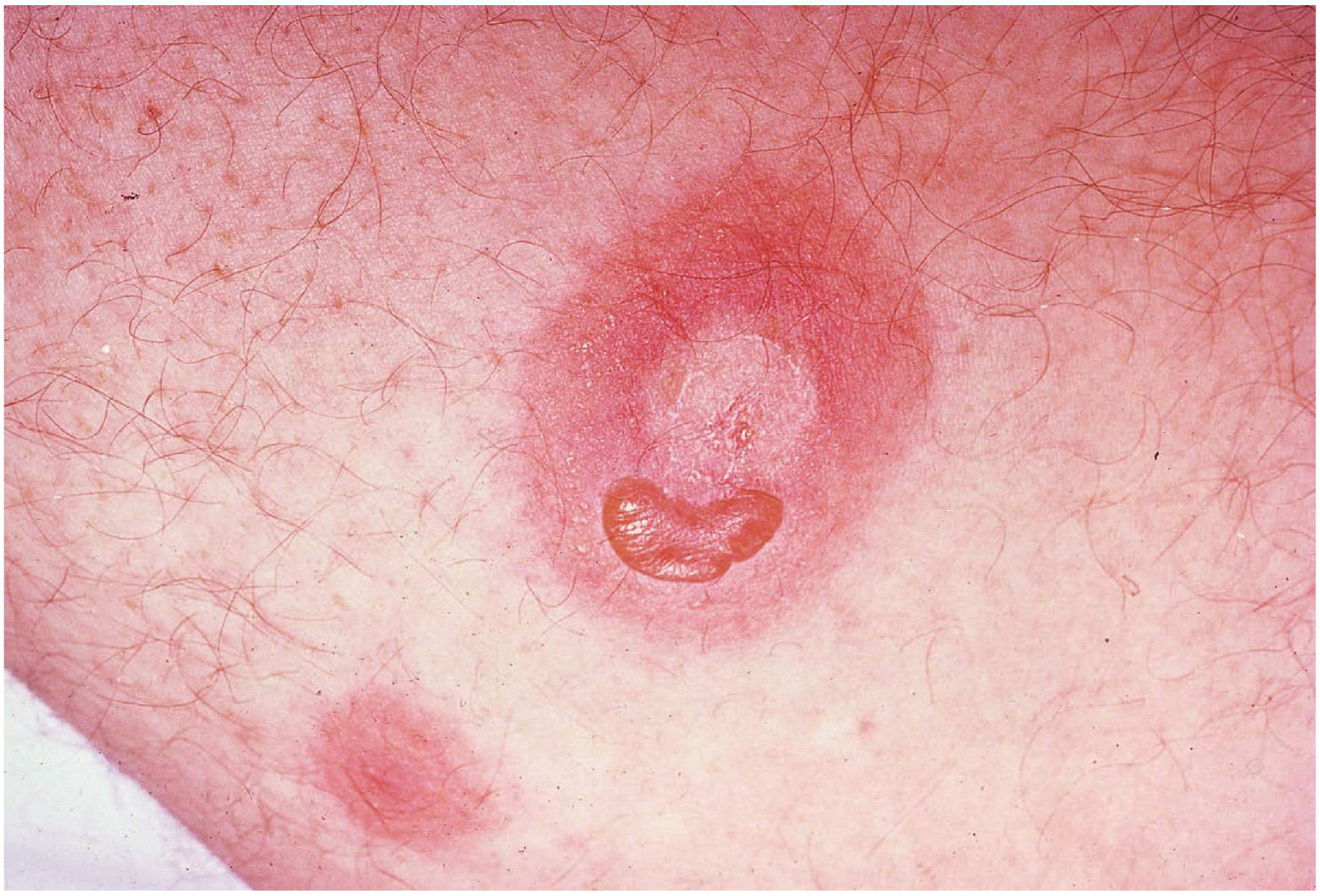


FIGURE 13.7 ■ **Bullous Erythema Multiforme.** Atypical targetoid appearance (only two rings apparent) and bulla formation. (Photo contributor: J. Matthew Hardin, MD.)

# DRUG ERUPTIONS

## Clinical Summary

Exanthematous drug eruptions present 7 to 14 days after a new medication but may appear sooner with rechallenge. A symmetric, erythematous, morbilliform, blanching, eruption is most frequently encountered. Typically, pruritus and low-grade fever are present. The macules and papules usually become confluent and may progress to an exfoliative dermatitis (rarely to erythroderma). The eruption peaks in a few days and, if the culprit medication is stopped, completely resolves over 7 to 14 days.

Acute generalized exanthematous pustulosis (AGEP), a type of drug eruption, presents 1 to 5 days after starting a new medication (typically, a β-lactam or a macrolide antibiotic). A high fever is usually noted with neutrophilia (90% of patients) and eosinophilia (30%). The rash begins on the face and intertriginous areas with edematous erythema studded with nonfollicular, <5 mm pustules. Within hours, it becomes generalized with larger flaccid, flat pustules. Over the next 2 to 4 days, widespread superficial desquamation occurs. Mucosal sites, especially oral mucosa, can be involved. With the cessation of the offending medication, the rash slowly resolves over 2 weeks.

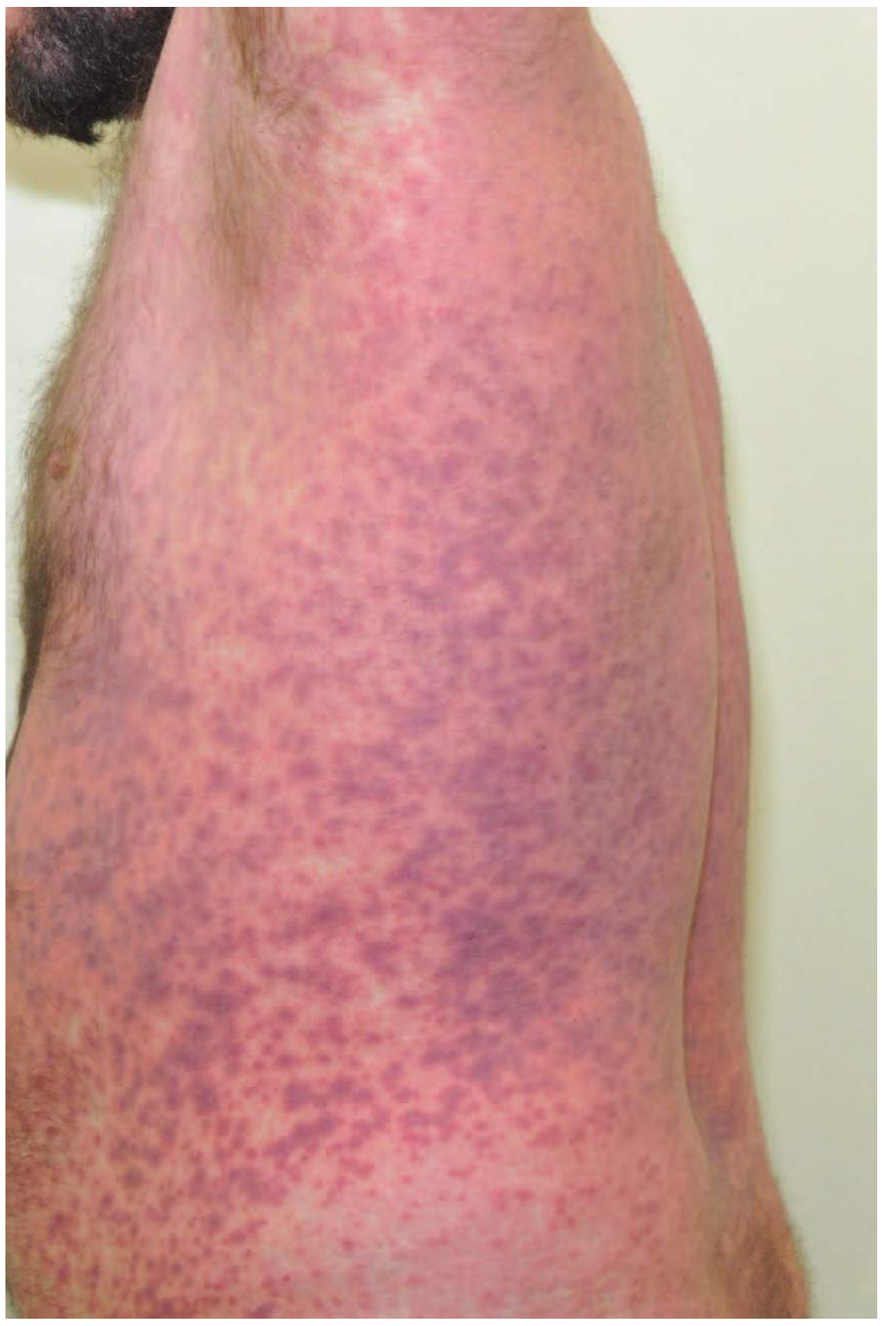


FIGURE 13.8 ■ **Exanthematous Drug Eruption.** Coalescing macules and papules—typically, this is a symmetric exanthem. (Photo contributor: Lawrence B. Stack, MD.)

## Management and Disposition

While exanthematous drug eruptions may resolve despite the medication's continued use, cessation of the causative agent is paramount. Symptomatic management includes antihistamines and topical corticosteroids.

The appearance of AGEP, with pustules, fever, and neutrophilia, is difficult to distinguish from an infectious etiology. Wound and blood cultures should be obtained early. Consult dermatology to help differentiate from pustular psoriasis, cellulitis, EM, bullous diseases, SJS, and TEN. Treatment consists of supportive care. The large surface area of desquamation makes secondary infection a major concern.

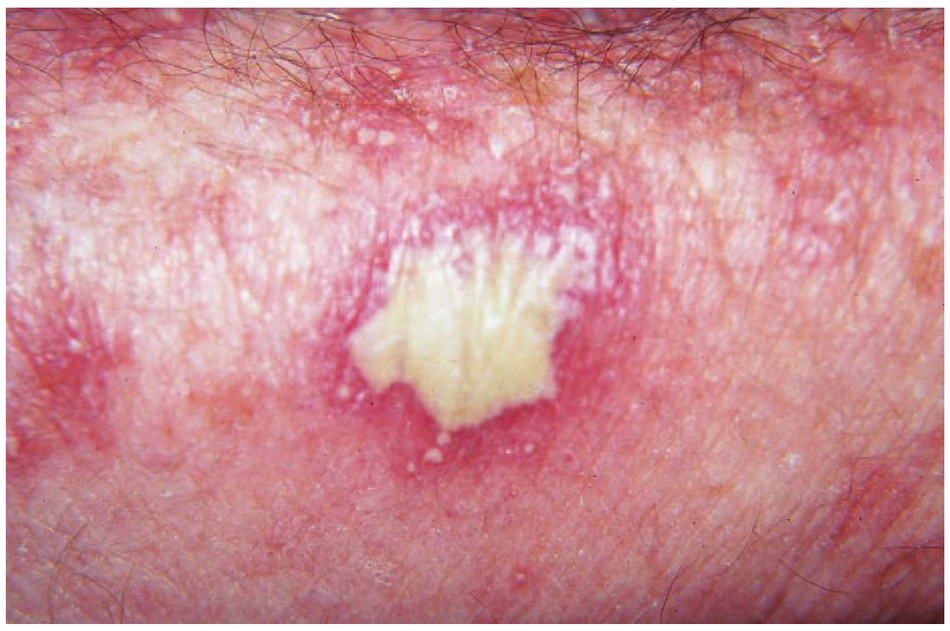


FIGURE 13.9 ■ **Acute Generalized Exanthematous Pustulosis.** Note the large, flaccid pustule (sterile) and surrounding smaller pustules. The smaller pustules are typical of the initial AGEP presentation. These will eventually slough off and leave a superficial, erythematous erosion. (Photo contributor: J. Matthew Hardin, MD.)

## Pearls

1. Exanthematous drug eruptions are usually symmetric and pruritic as opposed to viral eruptions, which are usually asymmetric and asymptomatic.
2. Mononucleosis patients taking amoxicillin or AIDS patients taking sulfa drugs frequently experience this reaction (augmented by viral infections).
3. The desquamation seen in AGEP is much more superficial than the full-thickness desquamation seen in SJS or TEN.

## Clinical Summary

Fixed drug eruptions (FDEs) appear 7 to 14 days after first exposure. The lesions can appear anywhere, including mucous membranes, but are most common on the face, lips, hands, feet, and genitalia. Single or multiple annular, edematous, well-demarcated plaques are typical. A central vesicle, bulla, or erosion may occur. After stopping the offending medication, the lesion(s) fade over several days. Residual hyperpigmentation is common. Within 24 hours of reexposure to the culprit medication, the exact rash reappears. The most common offending medications are sulfonamides, NSAIDs, barbiturates, tetracyclines, and carbamazepine.

## Management and Disposition

Identify all potential medications (prescription, herbal, and over-the-counter) and stop the offending drug. Symptomatic treatment with antihistamines and analgesics is sufficient. Refer to dermatology for further evaluation.

## Pearls

1. Without a thorough history including medication use, FDEs are difficult to identify.
2. Pseudoephedrine, a common over-the-counter medication, is a frequent cause of FDEs.
3. Warn patients that hyperpigmentation is expected and may not completely resolve.

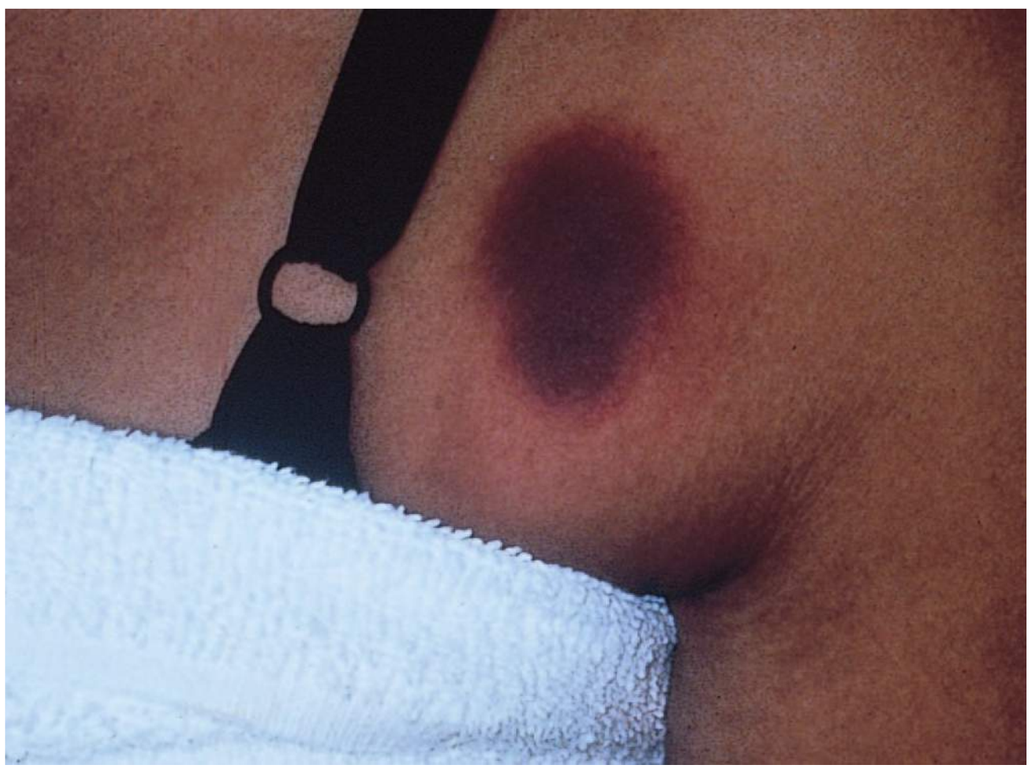


FIGURE 13.10 ■ **Fixed Drug Eruption.** This red to violaceous, pruritic, sharply demarcated patch is a cutaneous reaction to a drug. Repeated exposure will cause a similar reaction in the same location. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)

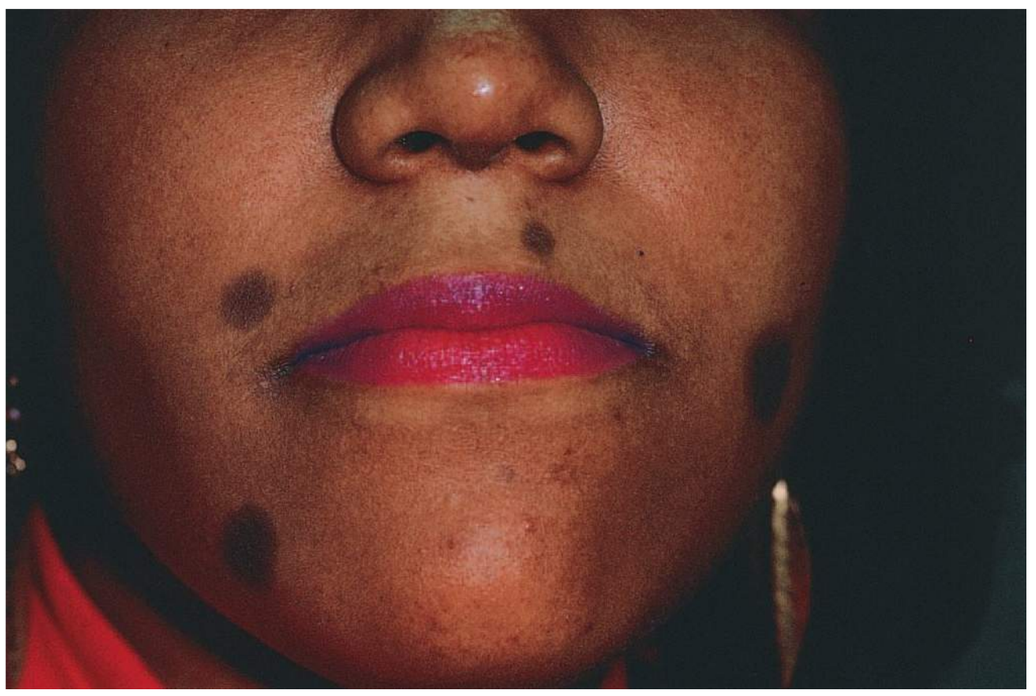


FIGURE 13.11 ■ **Fixed Drug Eruption.** Recurring reaction to acetaminophen. (Photo contributor: J. Matthew Hardin, MD.)

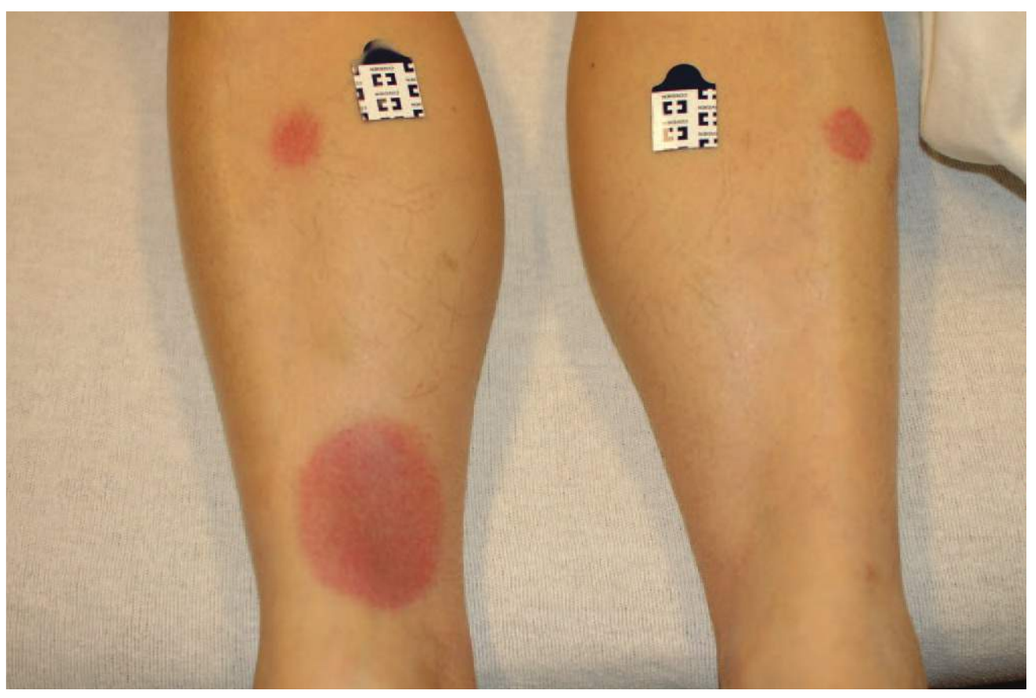


FIGURE 13.12 ■ **Fixed Drug Eruption.** Recurring reaction to sulfamethoxazole and trimethoprim. (Photo contributor: Edmond A. Hooker, MD, DrPH.)

# DISSECTING CELLULITIS OF THE SCALP

## Clinical Summary

Dissecting cellulitis of the scalp occurs predominately in young black males. This condition, along with acne conglobata, hidradenitis suppurativa (HS), and pilonidal cysts, is the "follicular occlusion tetrad." Multiple, fluctuant abscesses on the scalp vertex and occiput are present. Sinus tracts form between the abscesses and purulent; foul-smelling discharge may be present. Over time, extensive scarring alopecia can result.

## Management and Disposition

Exclusion of a secondary infection is paramount. Incision and drainage of new, rapidly forming abscesses may be helpful; obtain cultures if this is attempted. Antibiotics help prevent further abscesses; the tetracyclines help decrease the inflammatory component. Since this is an inflammatory condition, antibiotics alone are not adequate. Refer the patient to a dermatologist for biopsy and further treatment.

## Pearls

1. Consider this diagnosis when presented with recurrent scalp abscesses and draining sinus tracts unresponsive to antibiotics.
2. Early diagnosis and referral can prevent further scarring and alopecia.
3. Most patients are young males; the psychological impact can be significant.

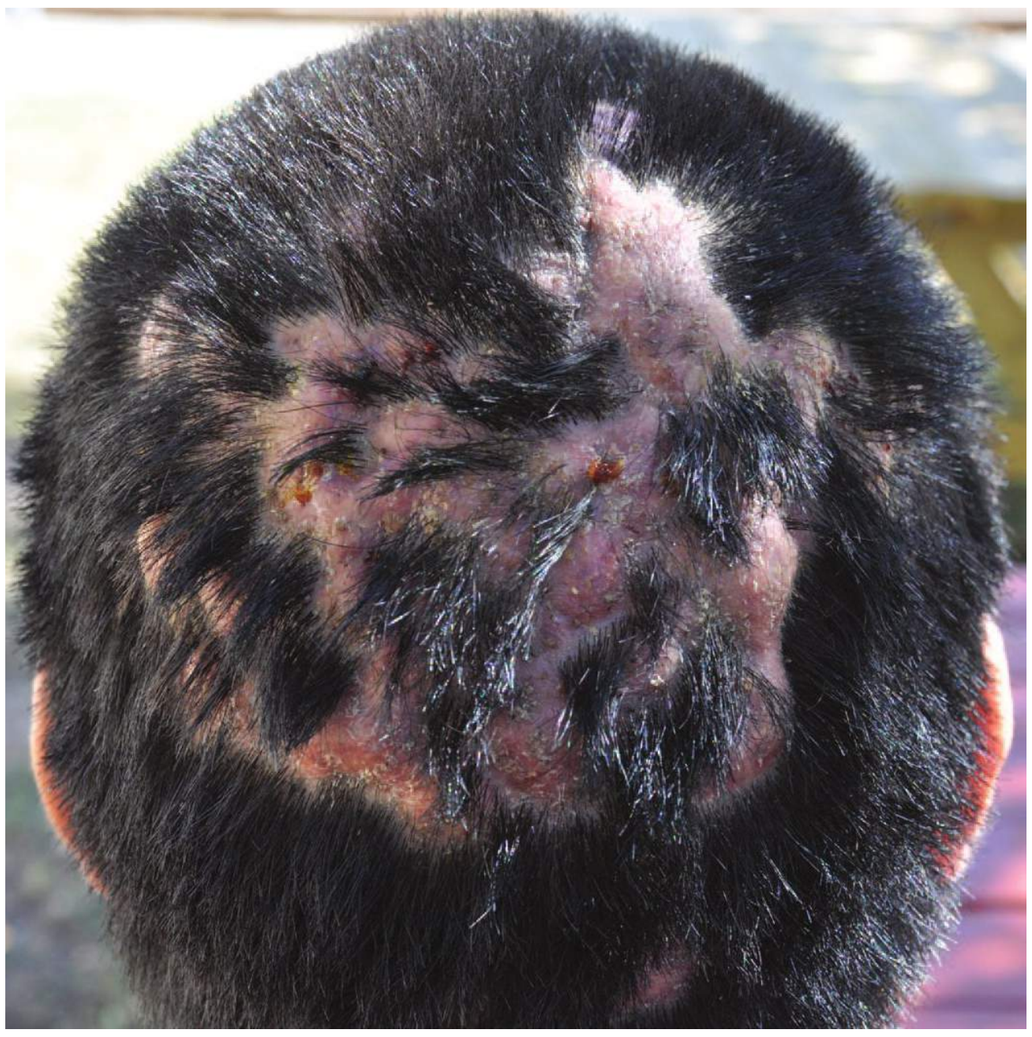


FIGURE 13.13 ■ **Dissecting Cellulitis of the Scalp.** Many active sinus tracts and alopecia in a young Hispanic man. (Photo contributor: Richard P. Usatine, MD. Used with permission. From Usatine RP, Smith MA, Mayeaux EJ, Chumley HS. The Color Atlas of Family Medicine. 2nd ed. McGraw-Hill; 2013: Fig. 189-7.)

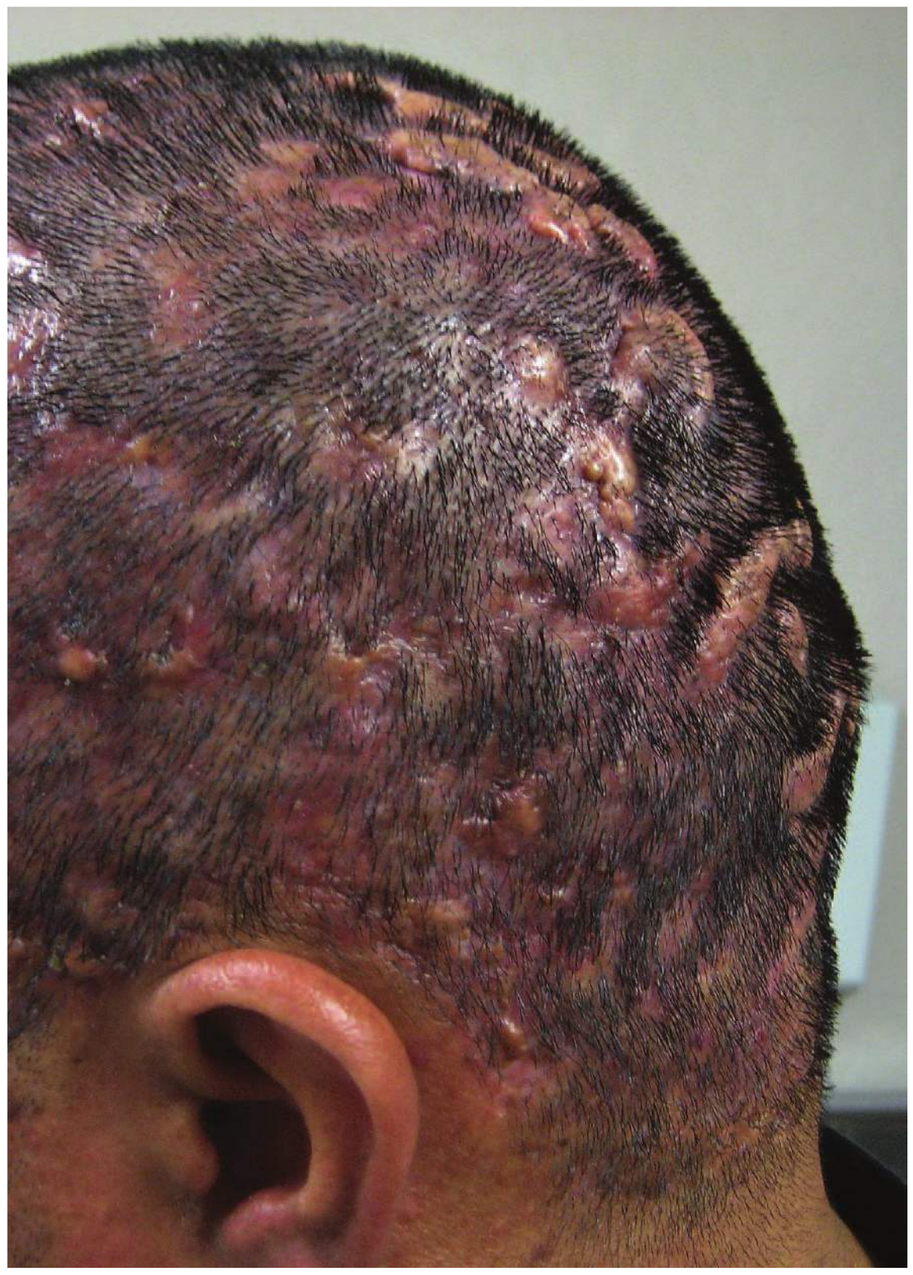


FIGURE 13.14 ■ **Dissecting Cellulitis of the Scalp.** Inactive but with disfiguring scars on the entire scalp. Early referral to a dermatologist may prevent this stage. (Photo contributor: J. Matthew Hardin, MD.)

## Clinical Summary

HS most commonly affects obese, postpubertal females. This unremitting disease involves hair-bearing intertriginous sites such as the axillae, inguinal folds, gluteal fold, perianal area, and inframammary folds. Individual lesions begin with erythematous nodules that become tender and fluctuant. The sterile abscess ruptures with a suppurative discharge and eventual sinus tract formation or fistulae. This is a primary inflammatory response to the follicle and is not infectious; however, secondary infections are common. Severe hypertrophic scar formation may be dramatic and disfiguring. In addition, chronic discharge is difficult to control and may become malodorous. The differential diagnosis includes bacterial furunculosis, granuloma inguinale, mycetoma, tuberculosis, and fistulas associated with inflammatory bowel disease.

## Management and Disposition

HS should be identified and local or systemic infection ruled out. Topical and systemic antibiotics help improve lesions, especially if a secondary infection is suspected. Oral doxycycline or minocycline, with topical clindamycin, is common first-line agents. If possible, incision and drainage should be avoided as this can induce chronic sinus tract formation and worse scarring. Referral to a dermatologist for chronic management and alternative treatments is indicated.

## Pearls

1. HS is not a primary infectious process but rather inflammatory. This often leads to delayed diagnosis, worse skin involvement, and permanent scarring.
2. One hallmark of hidradenitis is the "double comedones," a blackhead with two or greater surface openings with superficial communication.
3. Perianal involvement should prompt gastroenterology referral to rule out inflammatory bowel disease.

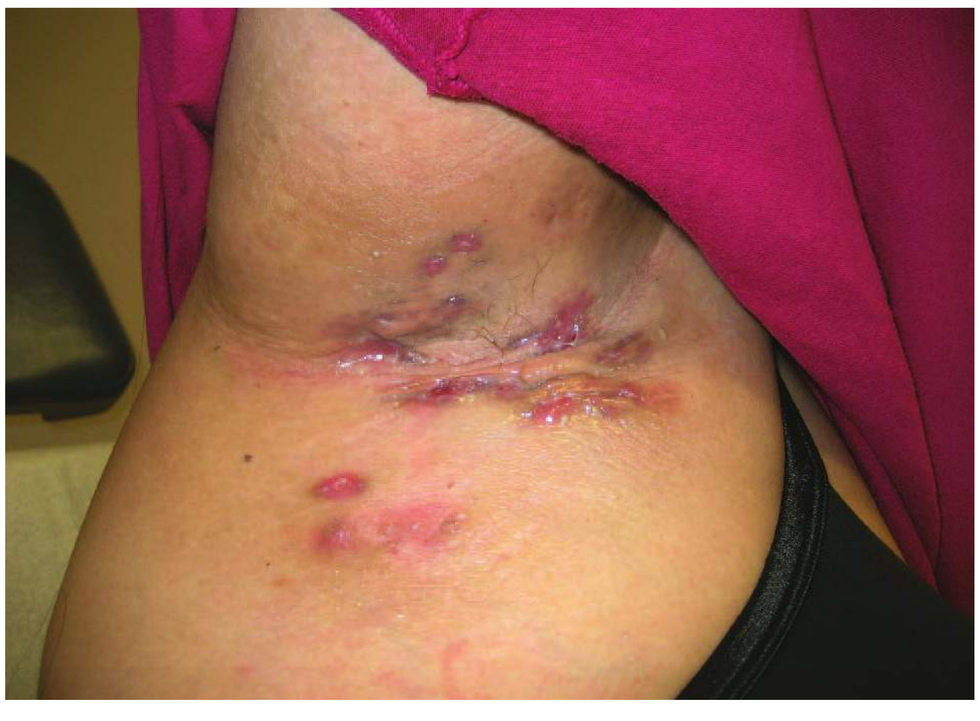


FIGURE 13.15 ■ **Hidradenitis Suppurativa.** Typical lesions present in the axilla. Note longitudinal scarring and multiple ostia. (Photo contributor: J. Matthew Hardin, MD.)

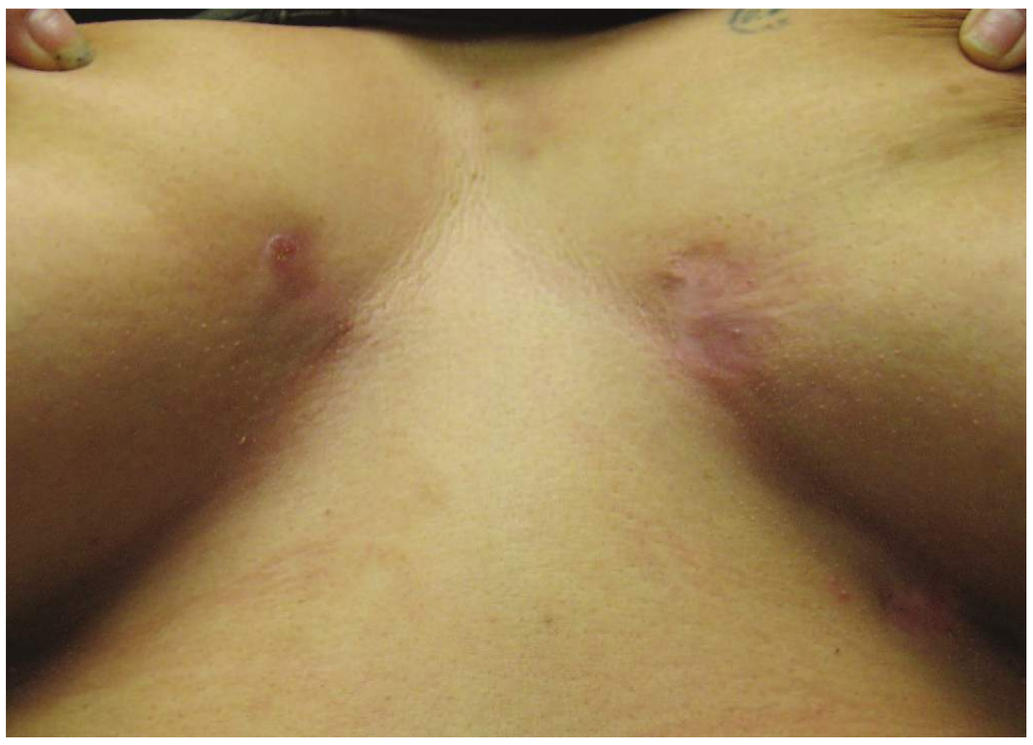


FIGURE 13.16 ■ **Hidiradenitis Suppurativa.** Chronic lesions in the inframammary folds. (Photo contributor: J. Matthew Hardin, MD.)

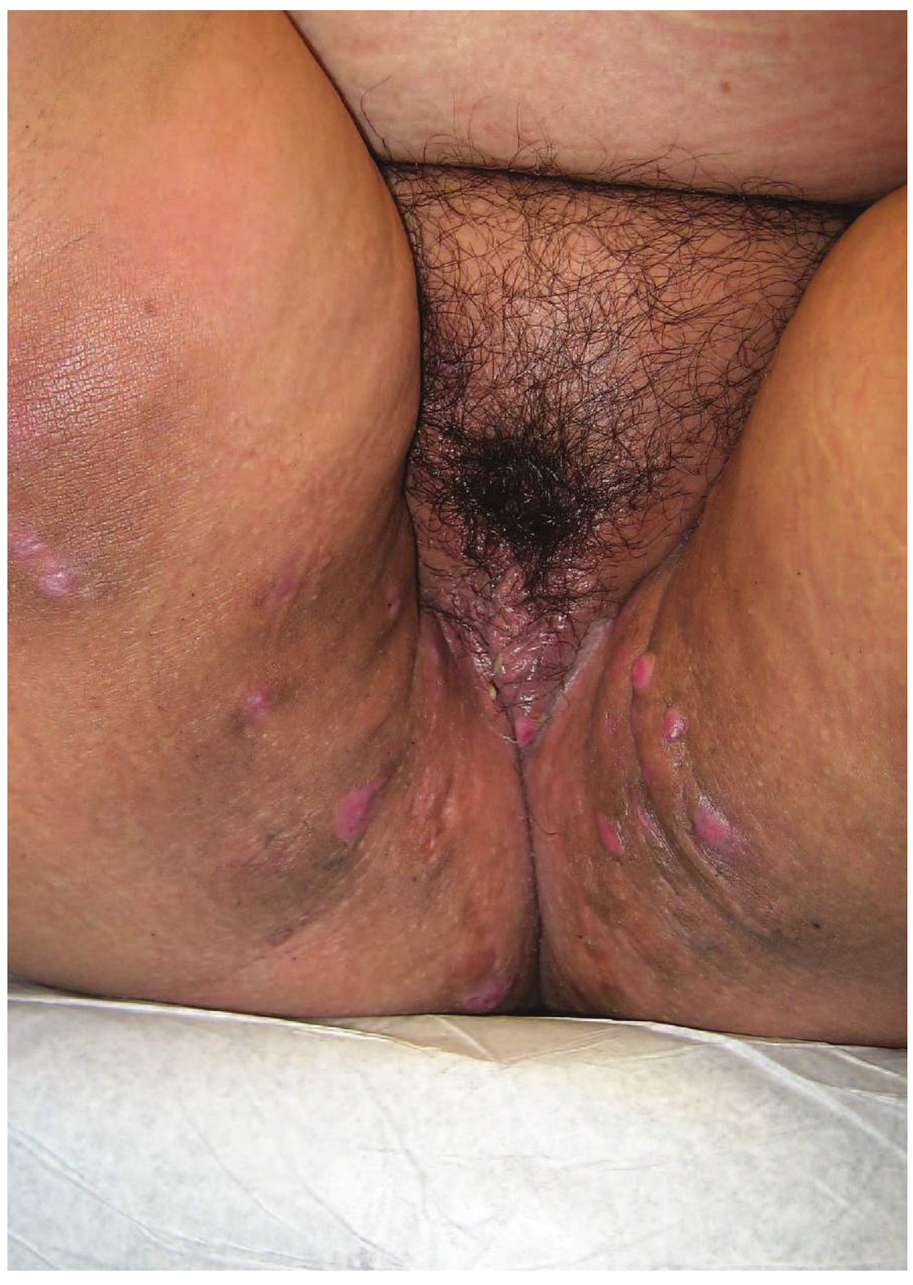


FIGURE 13.17 ■ **Hidradenitis Suppurativa.** Chronic lesions in the inguinal folds. (Photo contributor: J. Matthew Hardin, MD.)

# PYOGENIC GRANULOMA

## Clinical Summary

Pyogenic granuloma presents as an eruptive, friable papule over weeks. They are frequently located on the extremities, face, or at trauma sites. Children are most commonly affected but they can occur at any age. The lesion will bleed with very little trauma. If the papule is not completely removed, it will likely recur at the same site. Pregnant women have a higher incidence of pyogenic granulomas (common on the gingiva).

## Management and Disposition

Most emergency department (ED) presentations of pyogenic granuloma will be due to prolonged, often brisk, bleeding. Silver nitrate applied to the papule base is usually effective (avoid on the face to prevent permanent staining). Refer patients to a dermatologist for possible biopsy and definitive treatment.

## Pearls

1. An association with isotretinoin, indinavir, epidermal growth factor receptor agents, and capecitabine has been described.
2. Approximately one-third of these benign lesions follow some form of minor trauma.
3. Refer patients for biopsy and histology to exclude other vascular tumors.

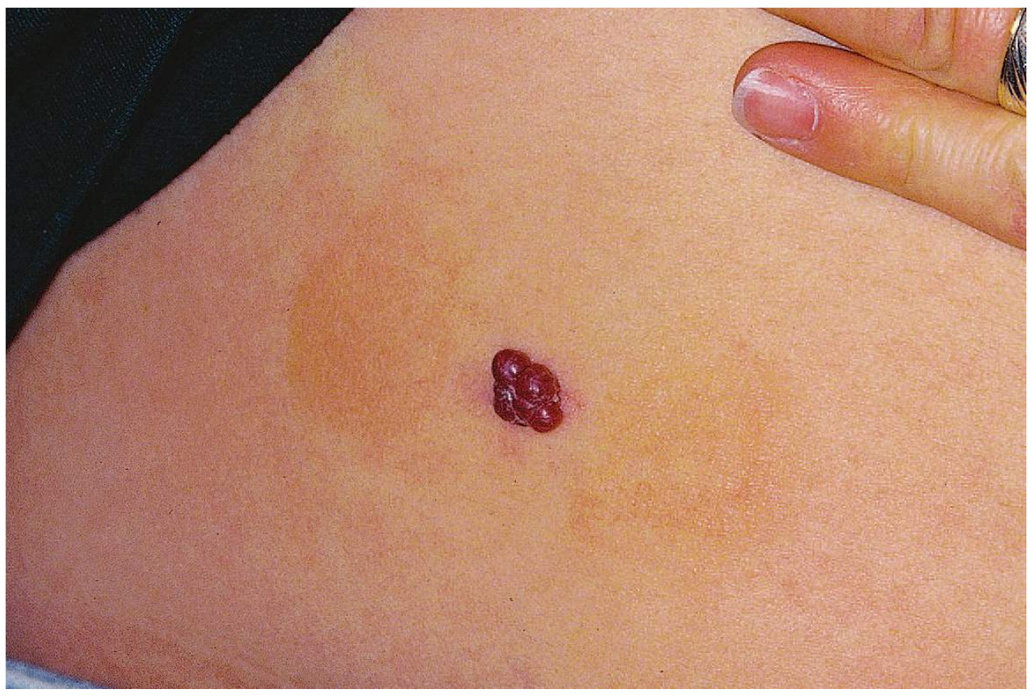


FIGURE 13.18 ■ **Pyogenic Granuloma.** Note the violaceous color and multilobulated nodule. The hyperpigmented patches on either side are secondary to a bandage. (Photo contributor: J. Matthew Hardin, MD.)

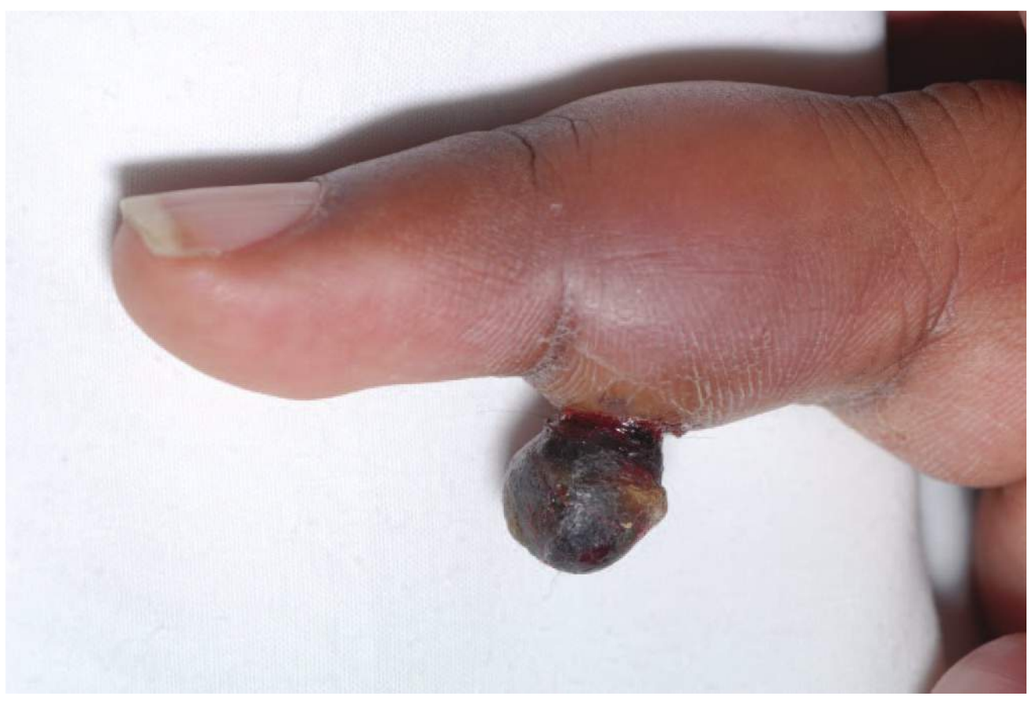


FIGURE 13.20 ■ **Pyogenic Granuloma.** Friable papule with frequent bleeding. (Photo contributor: Lawrence B. Stack, MD.)

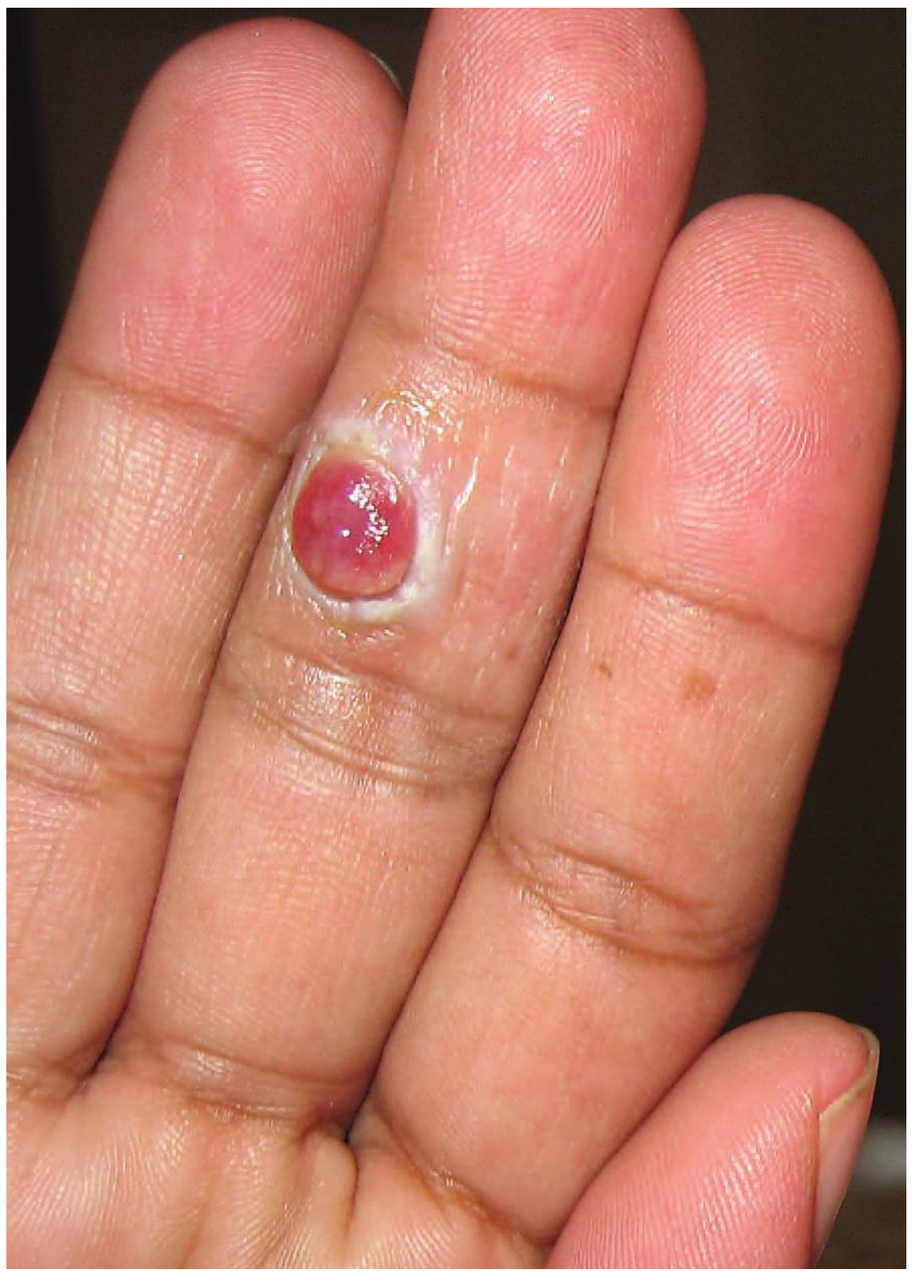


FIGURE 13.19 ■ **Pyogenic Granuloma.** A moist, violaceous, vascular nodule formed at the site of an injury. Note that the nodule is demarcated by a thin rim of friable epidermis. (Photo contributor: J. Matthew Hardin, MD.)

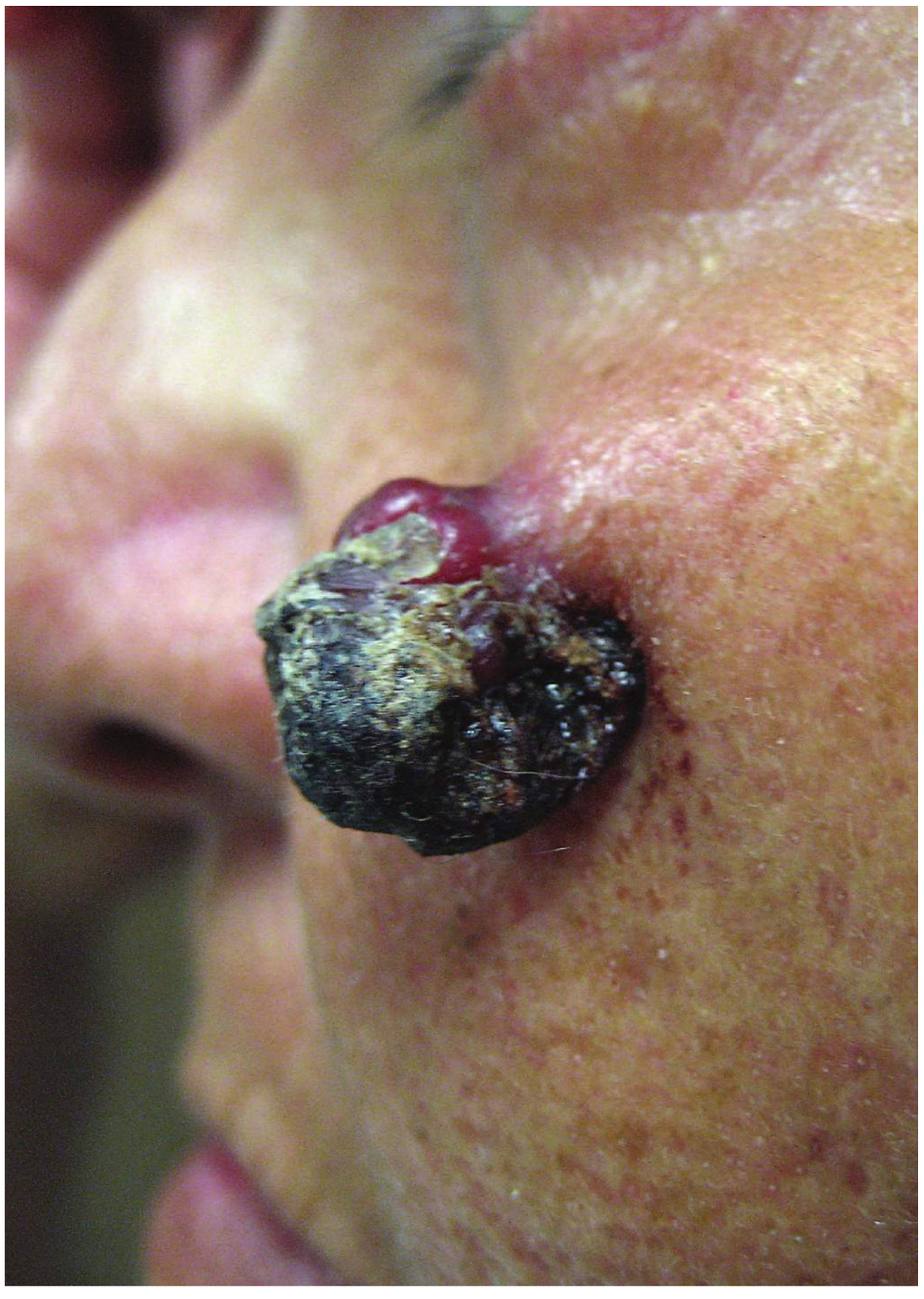


FIGURE 13.21 ■ **Pyogenic Granuloma.** Note the violaceous color and multilobulated nodule. (Photo contributor: J. Matthew Hardin, MD.)

# PYODERMA GANGRENOSUM

## Clinical Summary

Pyoderma gangrenosum (PG) is an inflammatory condition of all ages but is most common among 20- to 50-year-old females. Lesions can be located anywhere (most commonly on the lower extremities) and begin as a papulopustule surrounded by erythema. This pustule erodes to form a necrotic ulcer. Similar satellite pustules and ulcers form around the original lesion and eventually coalesce into a large ulcer. The surrounding border is "rolled," due to the convex elevation, and has a violaceous hue. The ulcers are exquisitely tender to movement and palpation. On the extremities, the ulcers can rapidly involve muscles and tendons. Ostomy sites are a common location and make care very difficult.

Half of cases are idiopathic; the other half are associated with inflammatory bowel disease, hematologic diseases (leukemia, myelodysplasia, monoclonal gammopathy), and the arthritides. Since diagnosis is based on examination, dermatopathology, and exclusion of other causes, it is difficult to confirm and delayed treatment is common. The most concerning diagnosis to exclude is infection due to bacteria, mycobacteria, fungi, syphilis, or amebiasis. Loxosceles spider bites and large vessel vasculitis are also considerations.

## Management and Disposition

Appropriate cultures should be obtained. Broad-spectrum antibiotics are indicated for secondary infections. Consult dermatology for biopsy, tissue culture, and initiation of immunosuppressant therapy.

## Pearls

1. With the number of ulcers seen in the ED, early diagnosis of PG is difficult and requires a high level of suspicion.
2. PG is often not diagnosed until late-stage ulcer formation and after multiple failed skin grafts. Without immunosuppressant therapy, skin grafting is unlikely to succeed.
3. Half of PG cases have associated diseases; each case needs a thorough specialty evaluation.

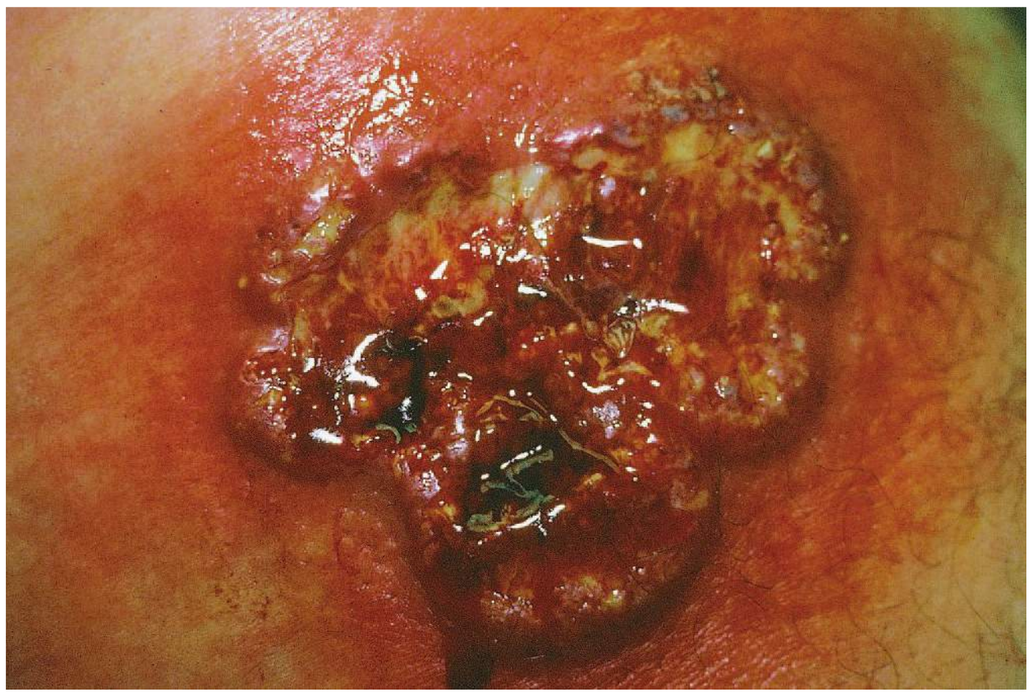


FIGURE 13.22 ■ **Pyoderma Gangrenosum.** Rolled and violaceous borders. This lesion can rapidly enlarge and become secondarily infected. (Photo contributor: J. Matthew Hardin, MD.)

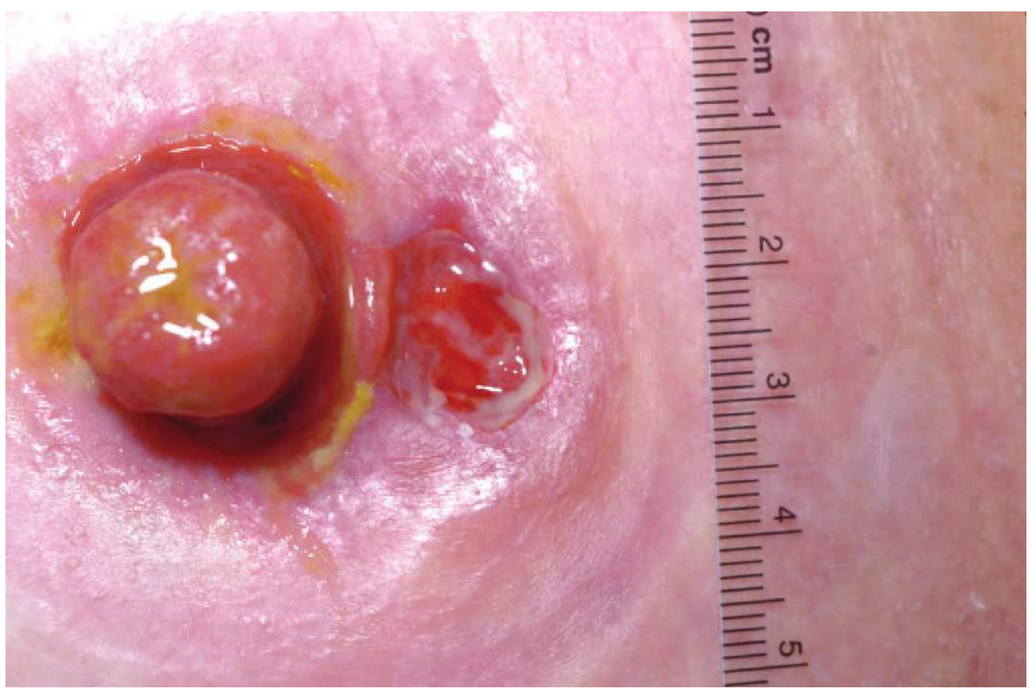



FIGURE 13.23 ■ **Pyoderma Gangrenosum.** Early ulcer formation at the site of an ileostomy dressing. (Photo contributor: Lawrence B. Stack, MD.)

## Clinical Summary

Seborrheic dermatitis represents a spectrum ranging from localized lesions to generalized exfoliative erythroderma. All ages are affected. Lesions have an erythematous base with a yellow, greasy scale. The scalp, external auditory canal, postauricular, eyebrows, eyelids, face (especially the nasolabial folds), axillae, umbilicus, presternal chest, and groin are common locations. Infants can have lesions at the above sites, but focal and confluent lesions are most common on the scalp and called "cradle cap."

## Management and Disposition

Seborrheic dermatitis is a lifelong disease and has no cure; management is directed at control. Localized cases are effectively treated with topical 2% ketoconazole. Scalp involvement can be treated with selenium sulfide, ketoconazole, or zinc pyrithione shampoos. Parents of affected infants should be reassured that infantile seborrheic dermatitis is self-limited. Refer to a dermatologist for confirmation of diagnosis and chronic care.

## Pearls

1. New-onset, severe, or prolonged seborrheic dermatitis may indicate a new HIV infection or immunosuppression.
2. Although there is no cure, reassure adult patients that the rash can be well controlled with topical medications.
3. In infants, seborrheic dermatitis can appear indistinguishable from Langerhans cell histiocytosis. Always have a clear discharge plan to follow up with a pediatrician or dermatologist.

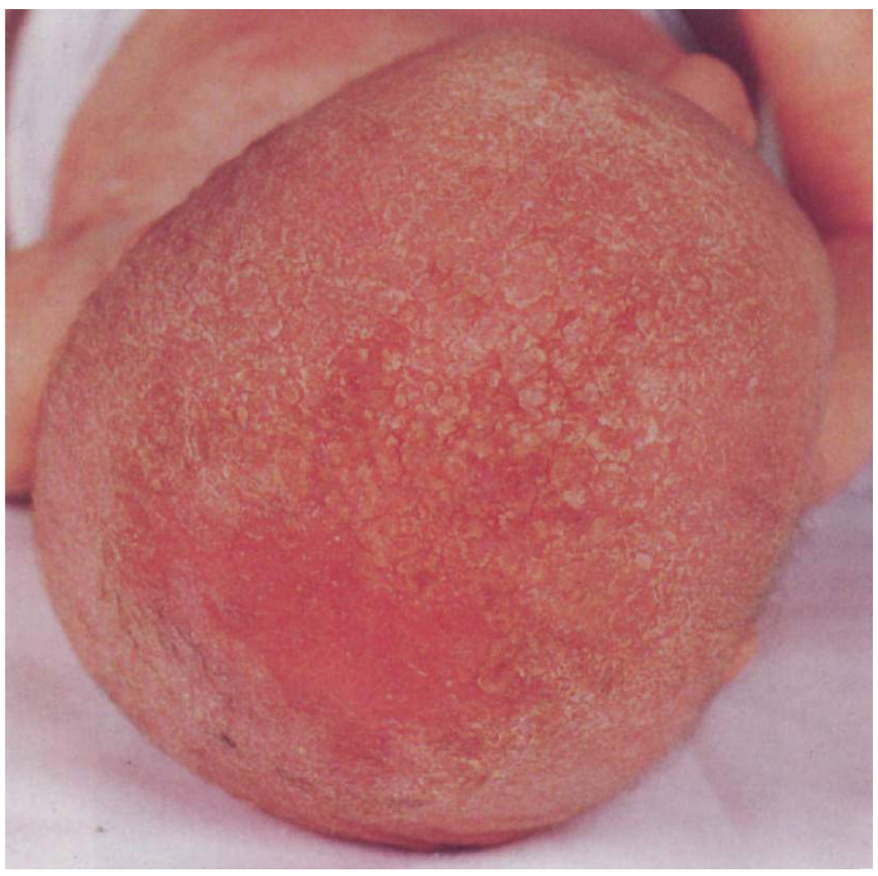


FIGURE 13.24 ■ **Seborrheic Dermatitis.** Erythema and yellow-orange scales and crust on the scalp of an infant ("cradle cap"). Eczematous lesions are also present on the arms and trunk. (Used with permission from Wolff K, Johnson RA, Suurmond D. Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology. 5th ed. New York, NY: McGraw-Hill; 2005: 51.)

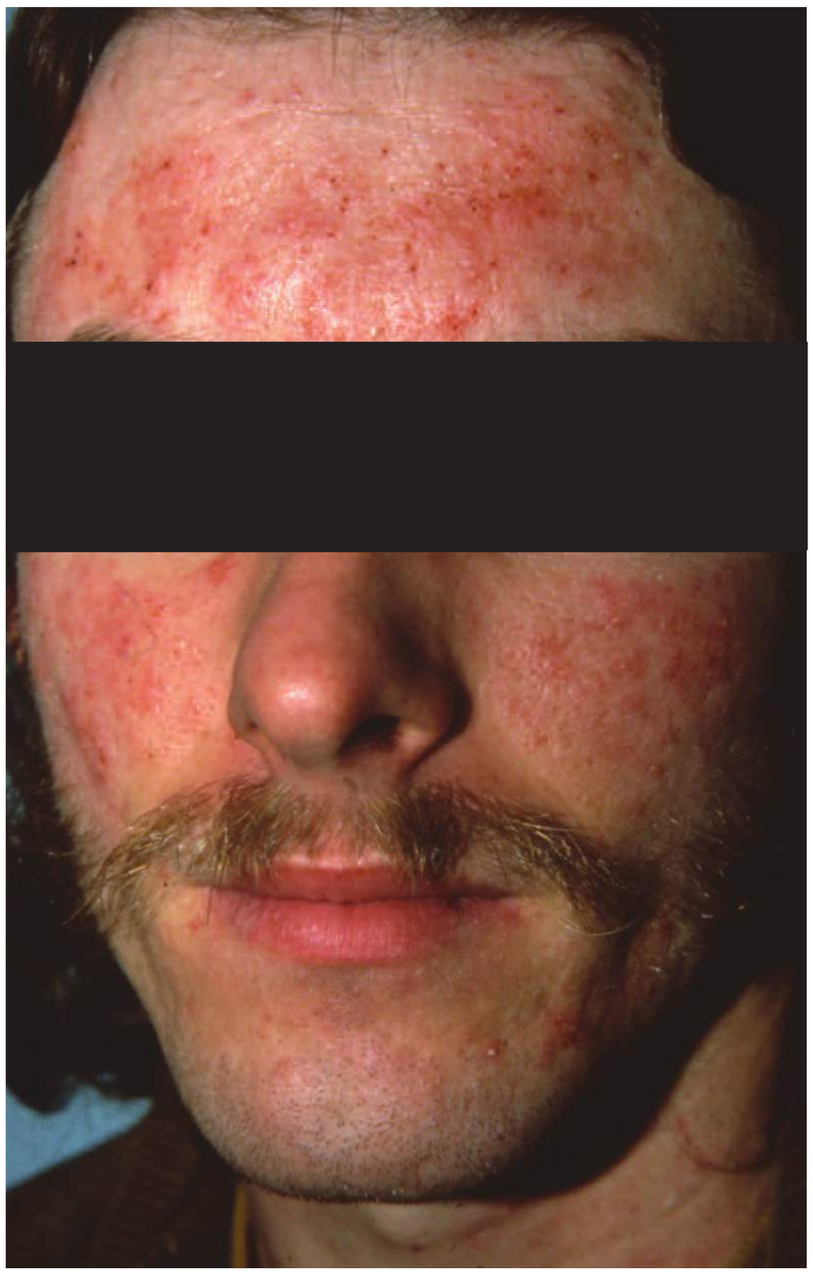


FIGURE 13.25 ■ **Seborrheic Dermatitis.** Intense erythema on the forehead and cheeks. (Photo contributor: David Effron, MD.)

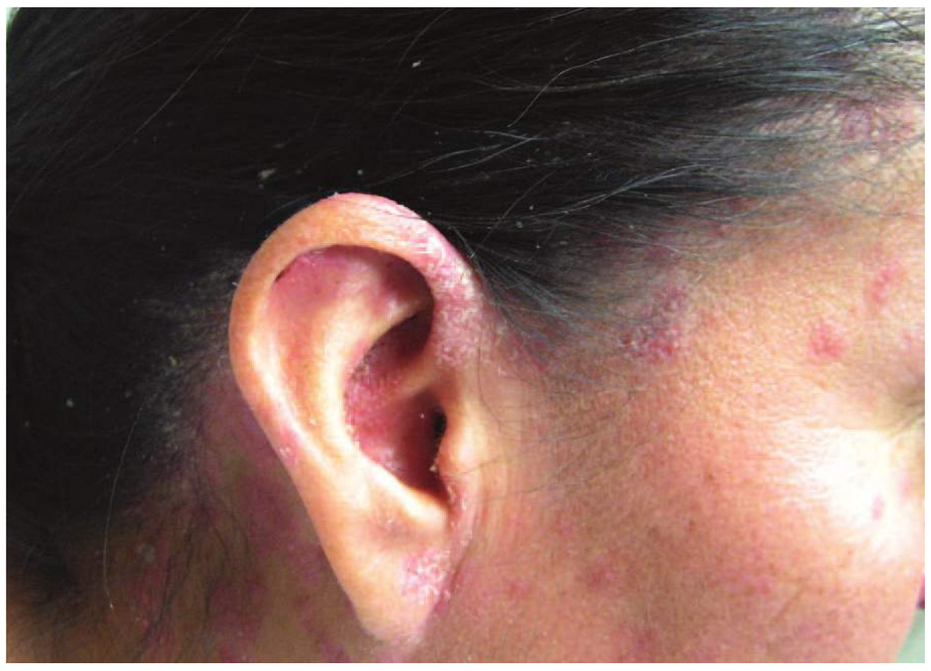


FIGURE 13.26 ■ **Seborrheic Dermatitis.** Typical erythematous, scaling patches on the pinna and conchal bowl. Note also similar lesions on the anterior and posterior hairlines. (Photo contributor: J. Matthew Hardin, MD.)

# PSORIASIS

## Clinical Summary

Psoriasis has many forms. The most common is chronic plaque psoriasis with stable, symmetric lesions on the trunk and extremities, especially the elbows and knees. Lesions are well-defined, erythematous plaques with silvery scales. Inverse psoriasis represents a form that involves the intertriginous areas and, due to the moist environment, the silvery scale is absent. Guttate psoriasis, common in children and young adults, presents with an abrupt eruption of 2- to 5-mm erythematous scaly papules on the trunk and extremities. A preceding respiratory infection, usually streptococcal pharyngitis, can be a precipitant. Pustular forms of psoriasis can present as localized (nail bed, finger, palms, or soles) or generalized. It is characterized by erythema and "lakes of pus." Triggers for pustular psoriasis include steroid withdrawal (as in patients with chronic obstructive pulmonary disease [COPD] and asthma exacerbations), pregnancy, infections, and topical irritants.

## Management and Disposition

ED management should ensure there is no infectious etiology or systemic symptoms. Localized psoriasis typically responds to topical glucocorticoids, although the chronicity and variety of other management options, including phototherapy, should prompt referral to dermatology. Pustular forms may be challenging and require admission. Guttate psoriasis may respond to antistreptococcal antibiotics. Obtain emergent consultation with a dermatologist for patients with generalized presentations and referrals for localized disease.

## Pearls

1. Medication-induced psoriasis is associated with β-blockers, lithium, interferon, and antimalarials.
2. Patients with psoriasis have a higher incidence of coronary artery disease, obesity, tobacco use, and alcoholism.

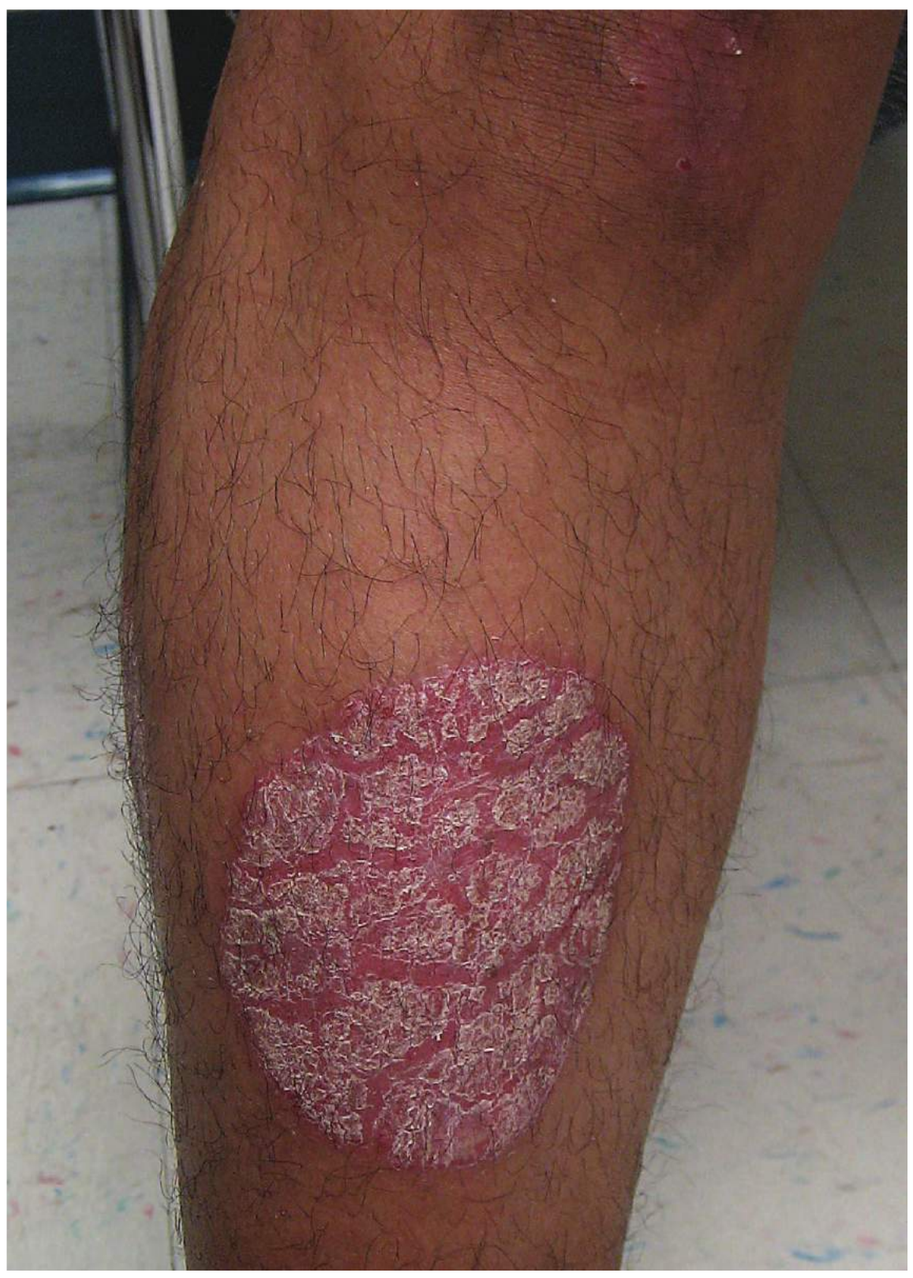


FIGURE 13.27 ■ **Psoriasis.** Annular, well-defined plaque on the shin. (Photo contributor: J. Matthew Hardin, MD.)

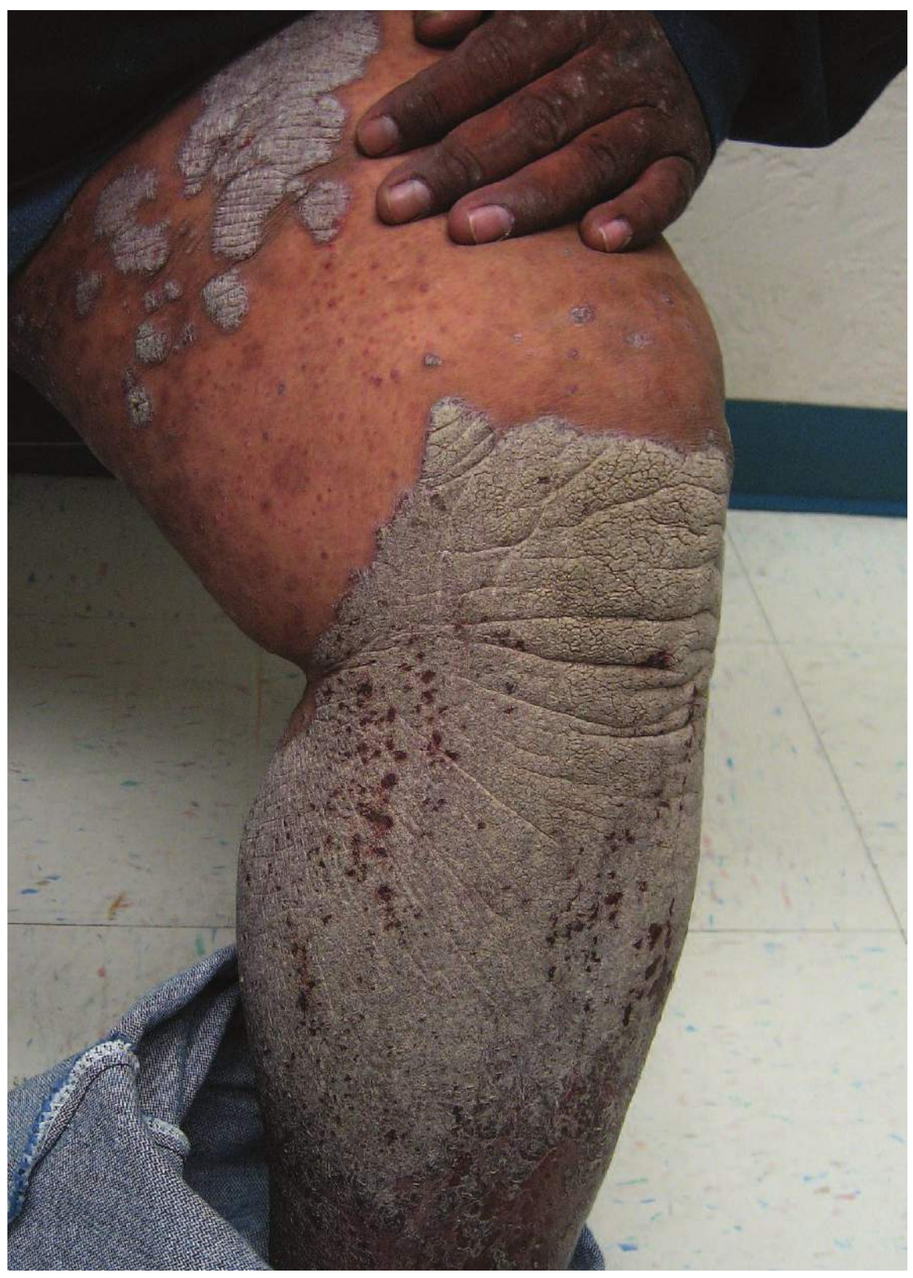


FIGURE 13.28 ■ **Psoriasis.** Classic silvery scale associated with longstanding lesions. (Photo contributor: J. Matthew Hardin, MD.)

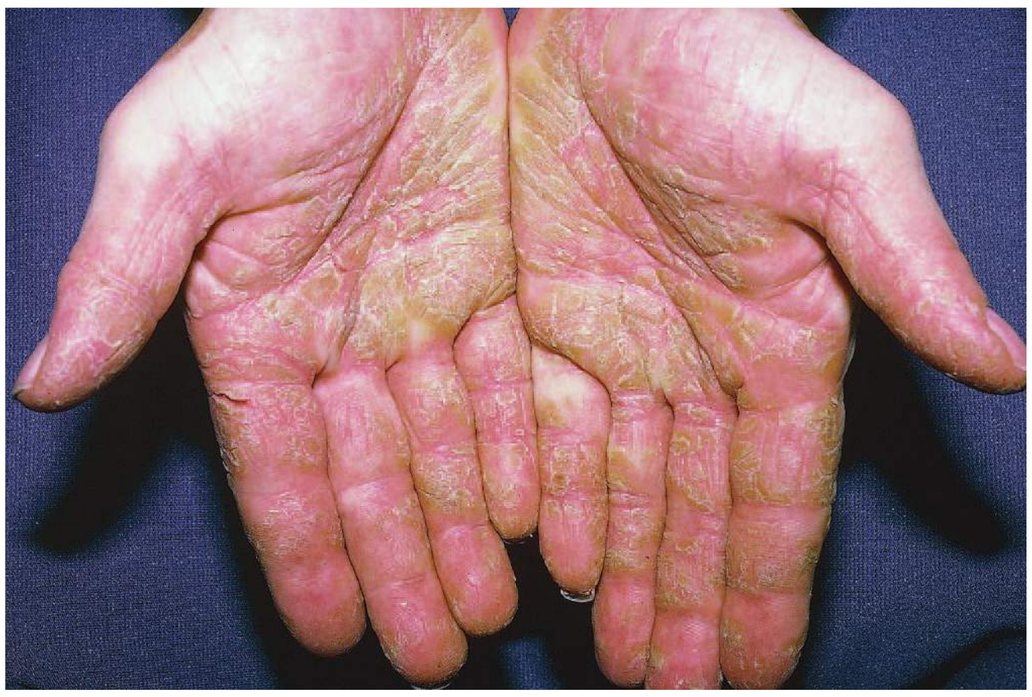


FIGURE 13.29 ■ **Psoriasis.** Note the erythematous plaques with diffuse fissuring in this case of palmar psoriasis. (Photo contributor: J. Matthew Hardin, MD.)

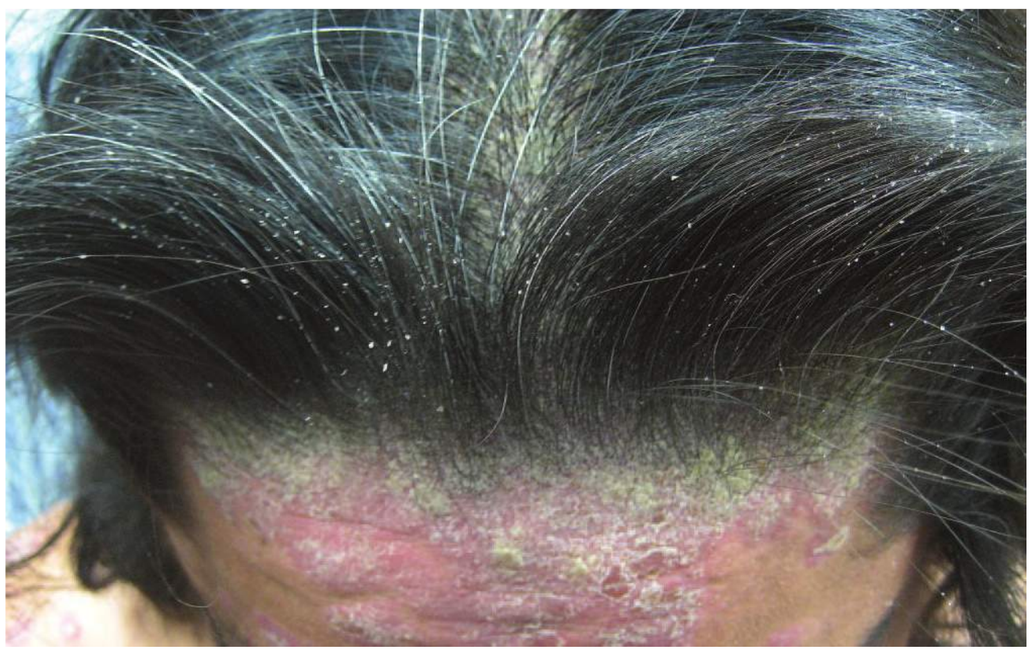


FIGURE 13.31 ■ **Psoriasis.** Erythematous plaques on the forehead and scaling in the scalp. (Photo contributor: J. Matthew Hardin, MD.)

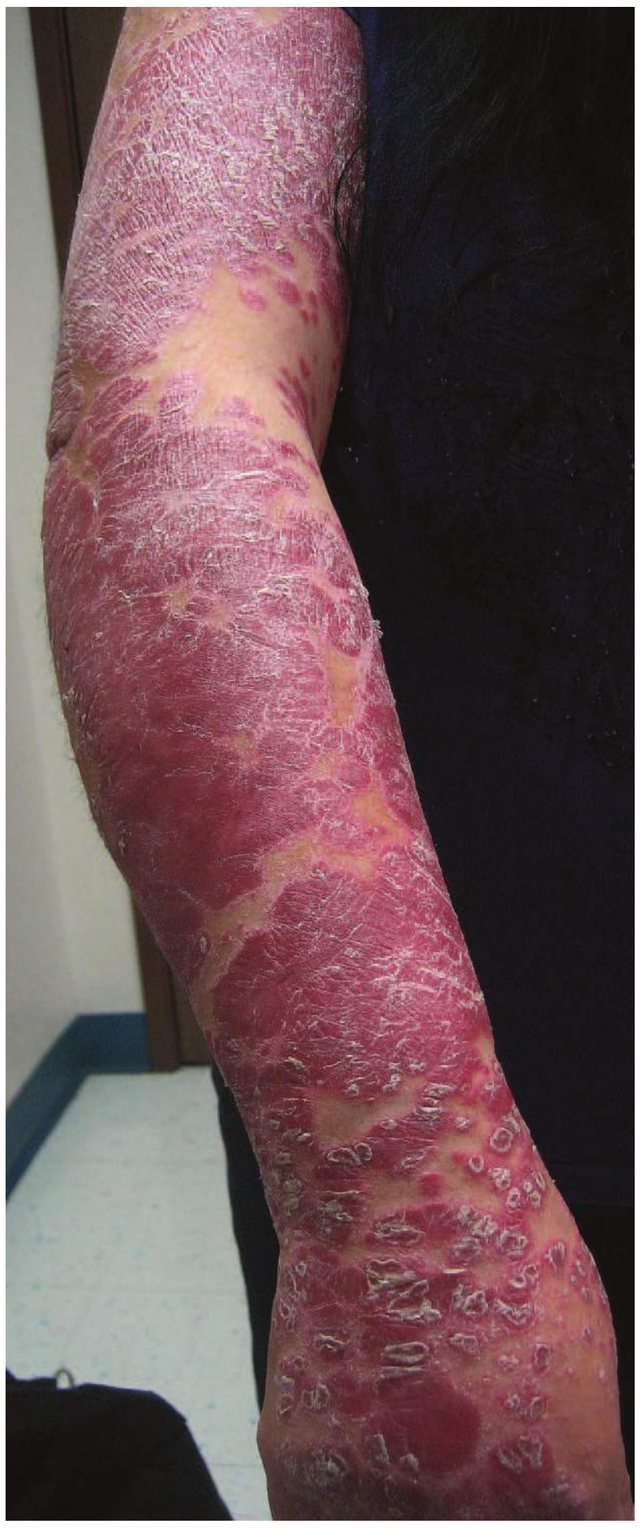


FIGURE 13.30 ■ **Psoriasis.** Erythematous plaques on the upper arm and hand. (Photo contributor: J. Matthew Hardin, MD.)

# PITYRIASIS ROSEA

## Clinical Summary

The first sign of pityriasis rosea (PR) is usually a well-demarcated, salmon-colored macule that evolves into a larger patch (1-4 cm) with peripheral scaling ("herald patch"). Over 1 to 2 weeks, generalized, bilateral, and symmetric macules and plaques appear along cleavage lines. The macules have a peripheral collarette of fine scaling (termed "Christmas tree" pattern). Most patients will have severe itching associated with the generalized eruption. The lesions slowly resolve over 6 to 8 weeks. Atypical presentations in children include inverse PR (presentation on the face, axillae, and/or inguinal areas) and papular PR (peripheral scaling papules with central, hyperpigmented plaques). A viral etiology is postulated due to seasonal variation and case clustering.

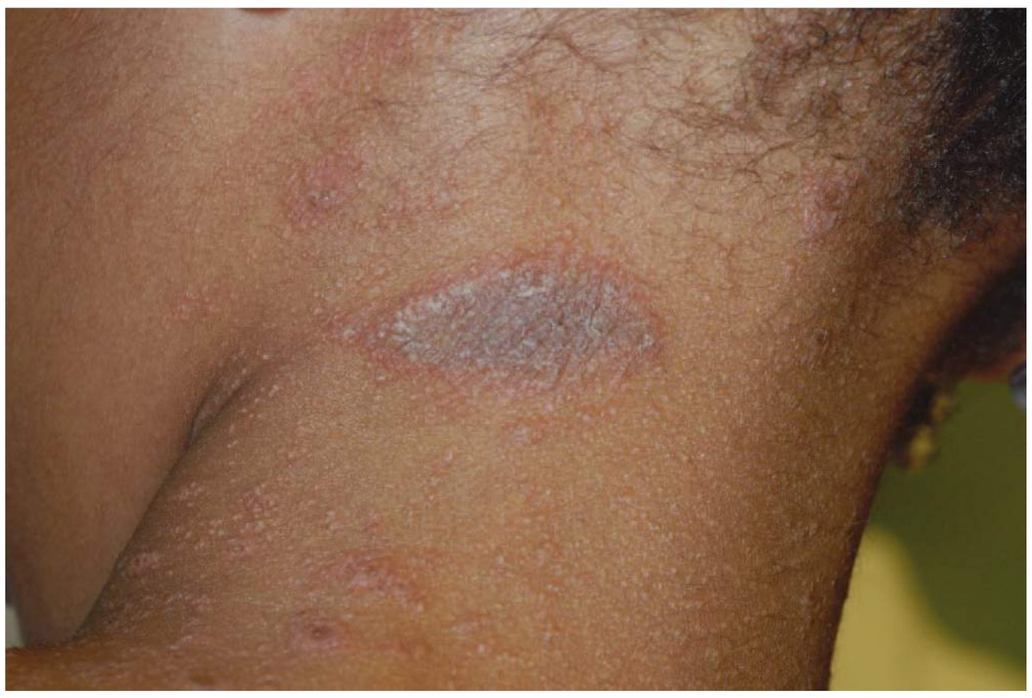


FIGURE 13.32 ■ **Pityriasis Rosea Herald Patch.** The herald patch of PR, a well-demarcated salmon-colored macule with scales, frequently precedes the generalized phase by 1 to 2 weeks. (Photo contributor: Lawrence B. Stack, MD.)

## Management and Disposition

PR is both benign and self-limited. Pruritus can be treated with oral antihistamines, topical steroids, and oatmeal baths.

## Pearls

1. Patients often will not describe the herald patch unless specifically asked.
2. In patients with risk factors for syphilis and HIV, appropriate screening tests should be performed.
3. Patients should be warned of the possible extended course of PR and given appropriate antihistamines and follow-up.
4. Atypical presentations are seen in dark-skinned individuals.

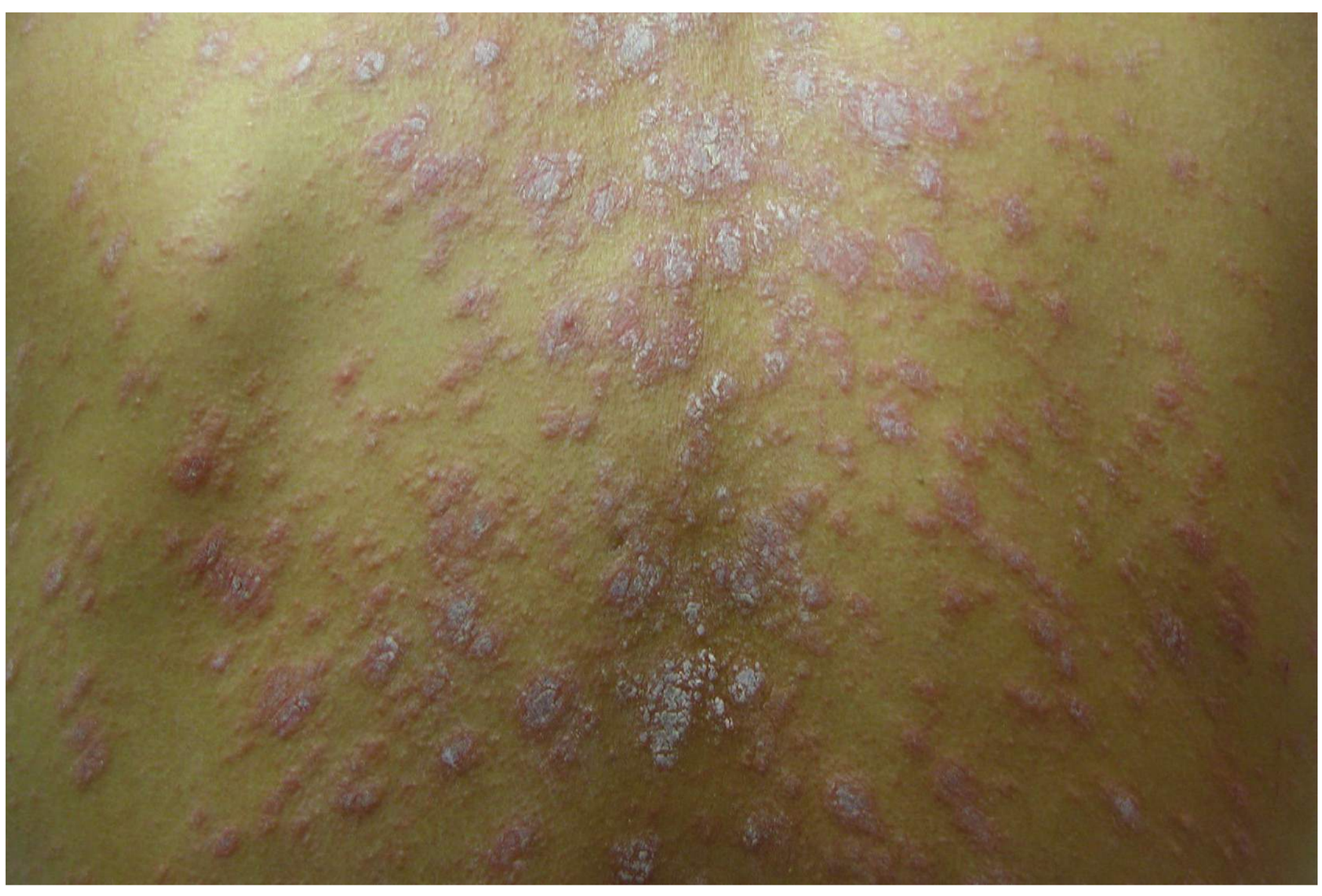


FIGURE 13.33 ■ **Pityriasis Rosea.** An exanthematous, papulosquamous eruption, with the long axis of the oval papules following the lines of cleavage in a Christmas tree–like eruption. (Photo contributor: David Effron, MD.)

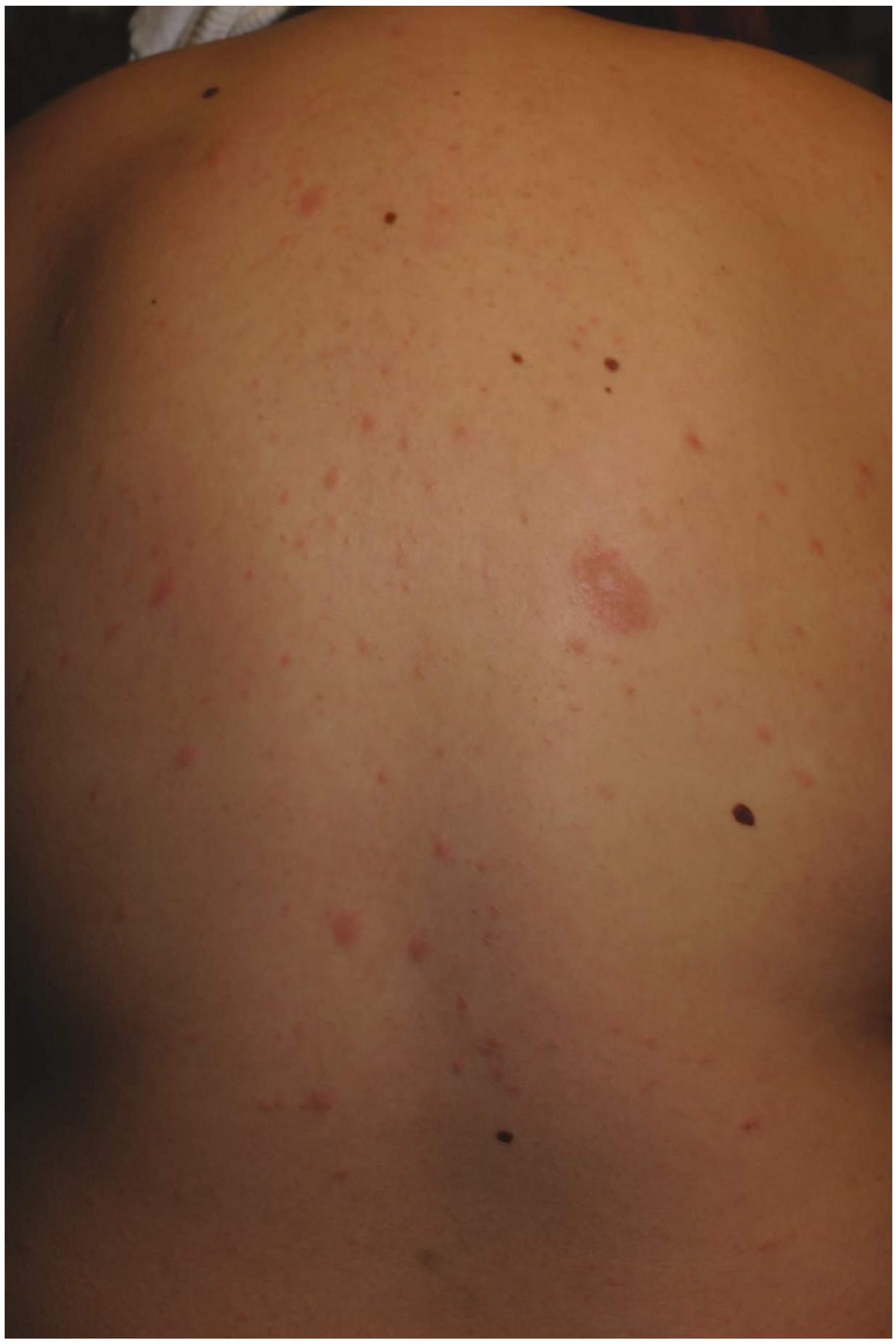


FIGURE 13.34 ■ **Pityriasis Rosea.** A more subtle Christmas tree–like eruption. (Photo contributor: Kevin J. Knoop, MD.)

# ATOPIC DERMATITIS

## Clinical Summary

Atopic dermatitis presents in three overlapping stages: infantile, childhood, and adult. Infantile begins after 2 months of age and is symmetrically distributed on the cheeks, scalp, neck, forehead, and extensor surfaces of the extremities. The lesions begin as erythema or papules, but, with persistent itching and rubbing, they become thin plaques, exudative and crusted. Childhood atopic dermatitis presents with flexural involvement. Other areas frequently involved are the face, neck, and trunk. The scratching induces plaque lichenification and potential for secondary infection. Adult atopic dermatitis is less specific but can present with a childhood-like distribution, papular lesions that coalesce into plaques, and chronic hand dermatitis. Atopic dermatitis can become a generalized exfoliative erythroderma. Differential diagnoses include seborrheic dermatitis, psoriasis, irritant or allergic contact dermatitis, nummular eczema, and scabies.

## Management and Disposition

If a severe flare, suprainfection, punched-out lesions (eczema herpeticum), or a generalized exfoliative erythroderma is present, an ED dermatologic consultation is indicated. Mild cases can be sent home with referral. Patients (caregivers) should avoid soaps, detergents, or any personal products with fragrances. After bathing, pat dry the skin and smear a thin film of petrolatum or mild corticosteroid over the affected areas. Wearing damp pajamas (100% prewashed cotton) after application of emollients/mild corticosteroids can help rapidly heal the skin.

## Pearls

1. Atopic dermatitis is often called the "itch that rashes" since pruritus precedes clinical disease.
2. If dispensing corticosteroids, use appropriate classes for the affected site and patient age. In addition, specific instructions should be written for the patient.
3. Frequent relapses are common and require an astute clinician to differentiate associated complications.

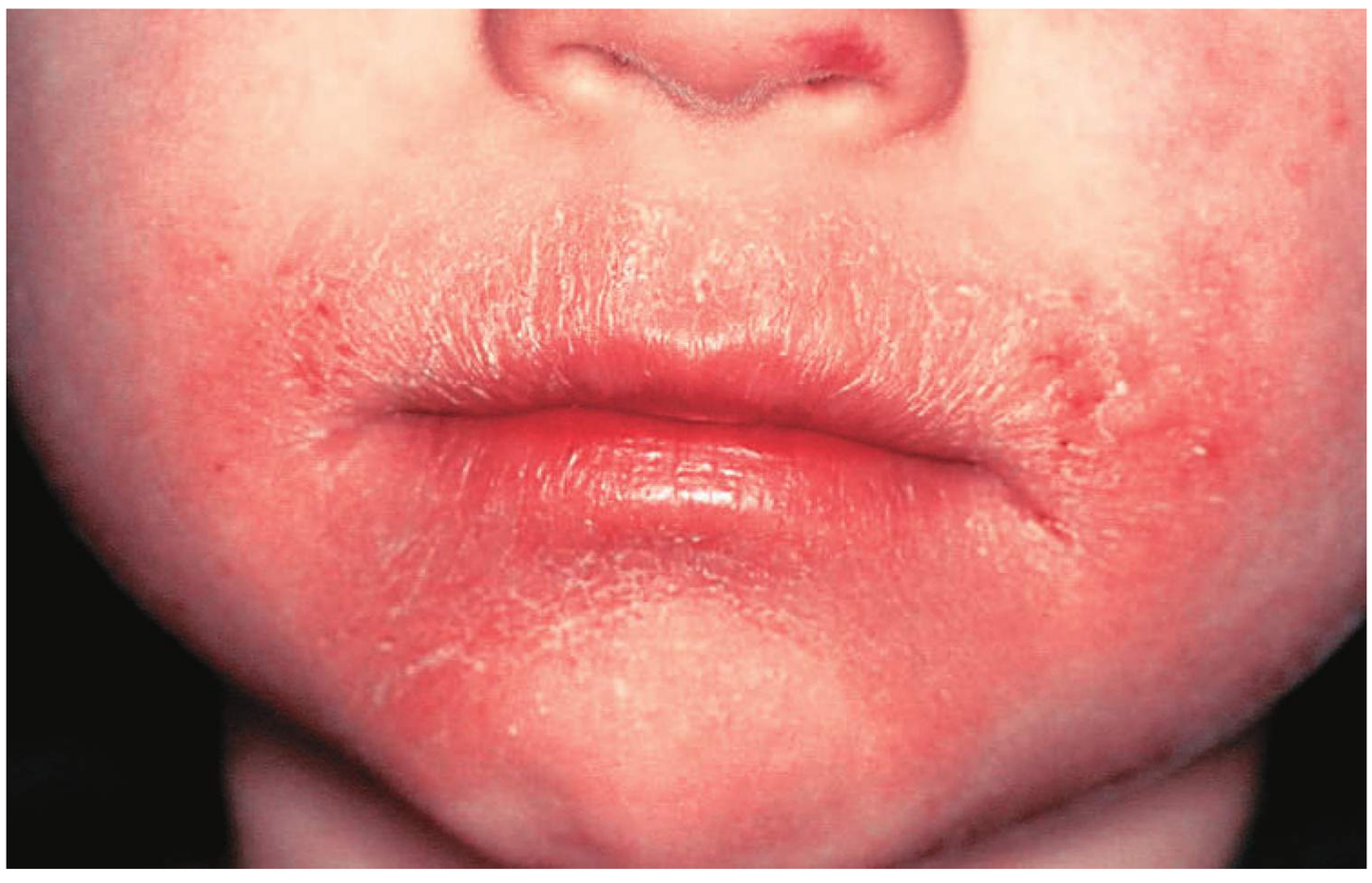


FIGURE 13.35 ■ **Atopic Dermatitis.** A typical localization of atopic dermatitis in children is the region around the mouth, with lichenification and fissuring and crusting. (Used with permission from Wolff K, Johnson RA, Saavedra AP. Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology. 7th ed. McGraw-Hill; 2013: Fig. 2-14.)

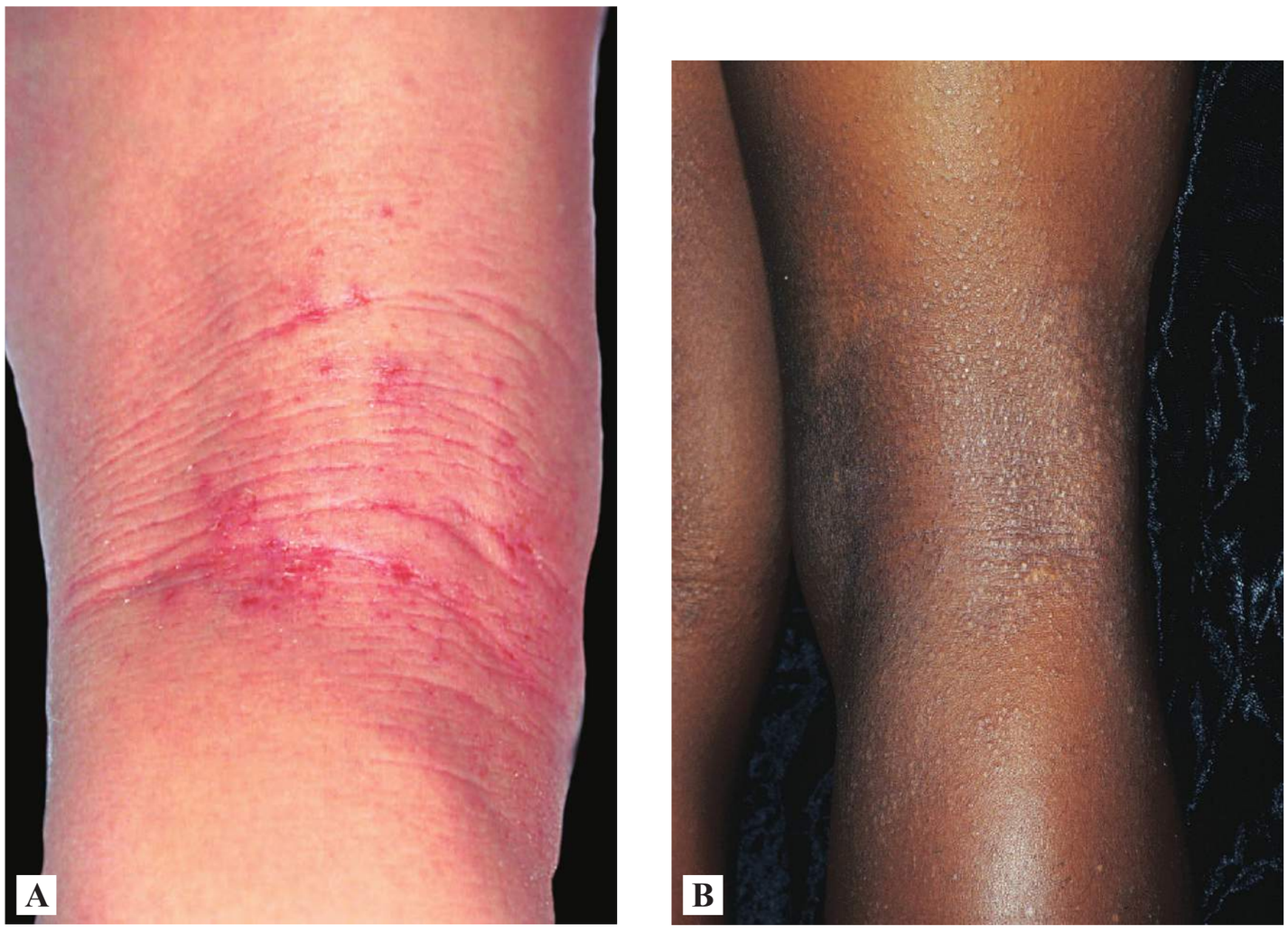



FIGURE 13.36 ■ **Atopic Dermatitis.** (**A**) One of the hallmarks of atopic dermatitis is lichenification in the flexural regions. Note the thickening of the skin with exaggerated skin lines and erosions. (**B**) Atopic dermatitis in black child. Pruritic follicular papules on posterior leg. Follicular eczema pattern is more common in African and Asian children. (Used with permission from Wolff K, Johnson RA, Saavedra AP. Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology. 7th ed. McGraw-Hill; 2013: Fig. 2-15 A and B.)

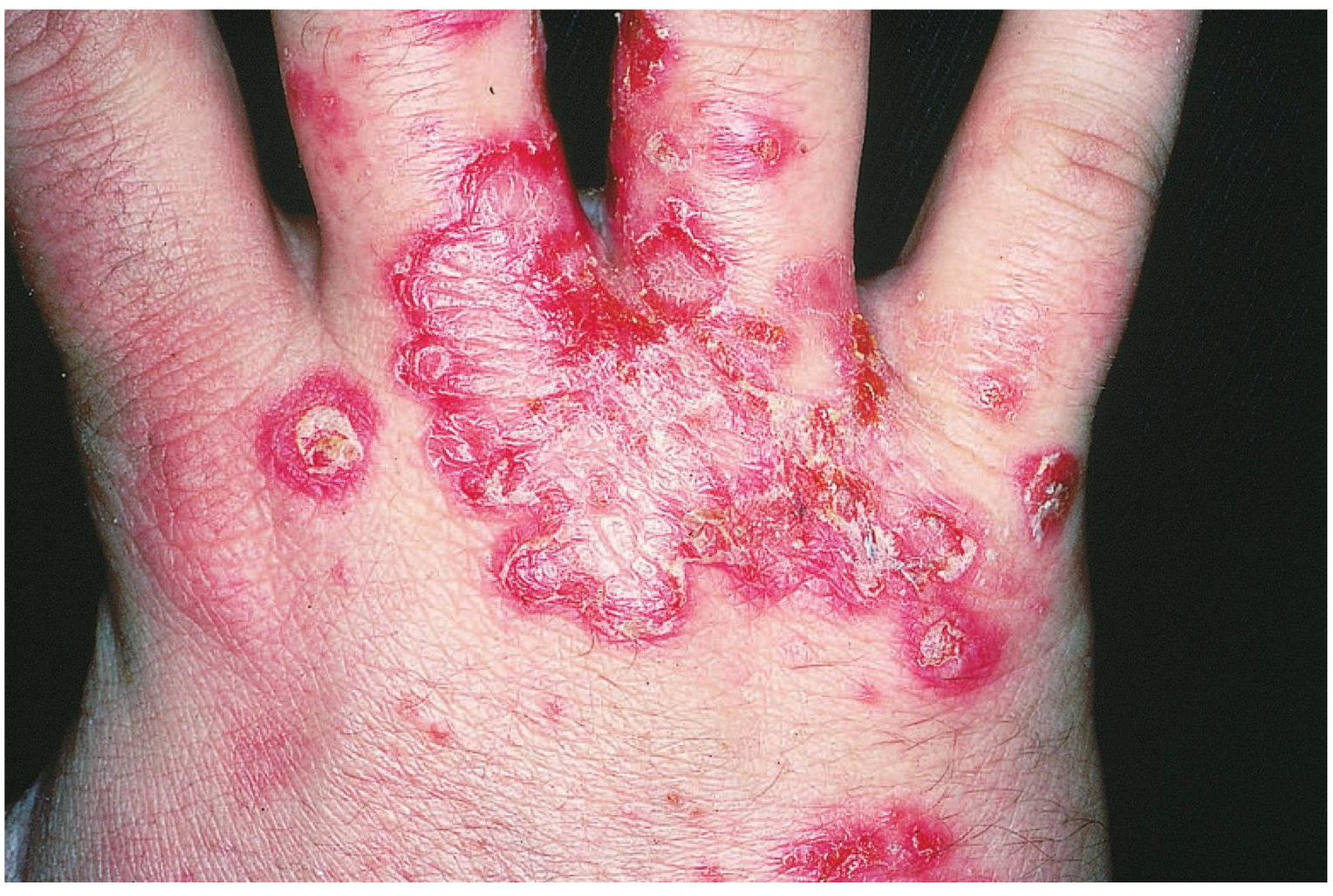


FIGURE 13.37 ■ **Atopic Dermatitis.** Lichenfied plaques, erosions, and fissures are characteristic of adult atopic dermatitis. (Photo contributor: James J. Nordlund, MD.)

# NUMMULAR/XEROTIC ECZEMA

## Clinical Summary

Nummular eczema is characterized by an erythematous, edematous, vesicular, and crusted extremity plaque. The lesions enlarge by forming satellite, peripheral papulovesicles that coalesce with the original lesion. Pruritus is the dominant symptom.

Xerotic eczema (also called winter itch, eczema craquele, and asteatotic eczema) presents on the anterior shins, extensor arms, and flanks. The lesions are erythematous patches with fine, cracked fissures and adherent scaling. The edema and exudate present in nummular eczema is absent. Pruritus can be severe. This is a common finding in the winter and in the elderly.

## Management and Disposition

Treatment of nummular eczema consists of mid- to high-potency topical steroids under occlusion. Prevention of secondary infection is important as patients frequently cannot resist scratching.

Xerotic eczema is treated with topical emollients (petrolatum), three to four applications per day. Topical steroids may be required for areas with inflammation.

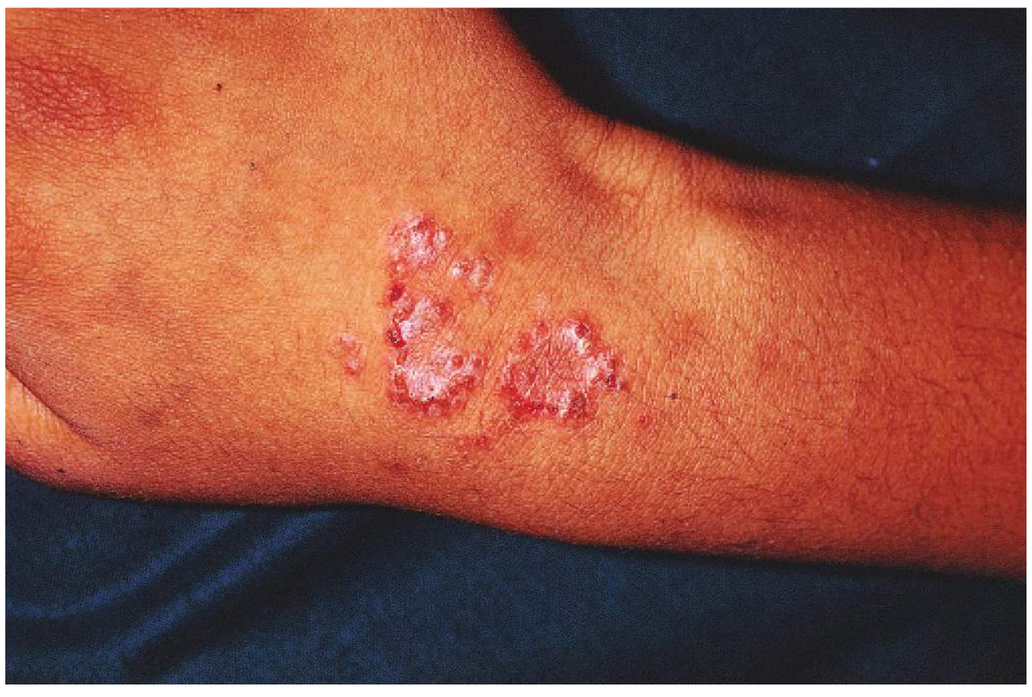


FIGURE 13.38 ■ **Eczema.** Nummular eczema of the wrist. Note the satellite lesions on the periphery. (Photo contributor: J. Matthew Hardin, MD.)

## Pearls

1. Nummular eczema should be considered with lesions unresponsive to antibiotics and pruritus as the dominant feature.
2. Both of these entities are associated with significant pruritus and secondary infections, especially in the young and elderly.

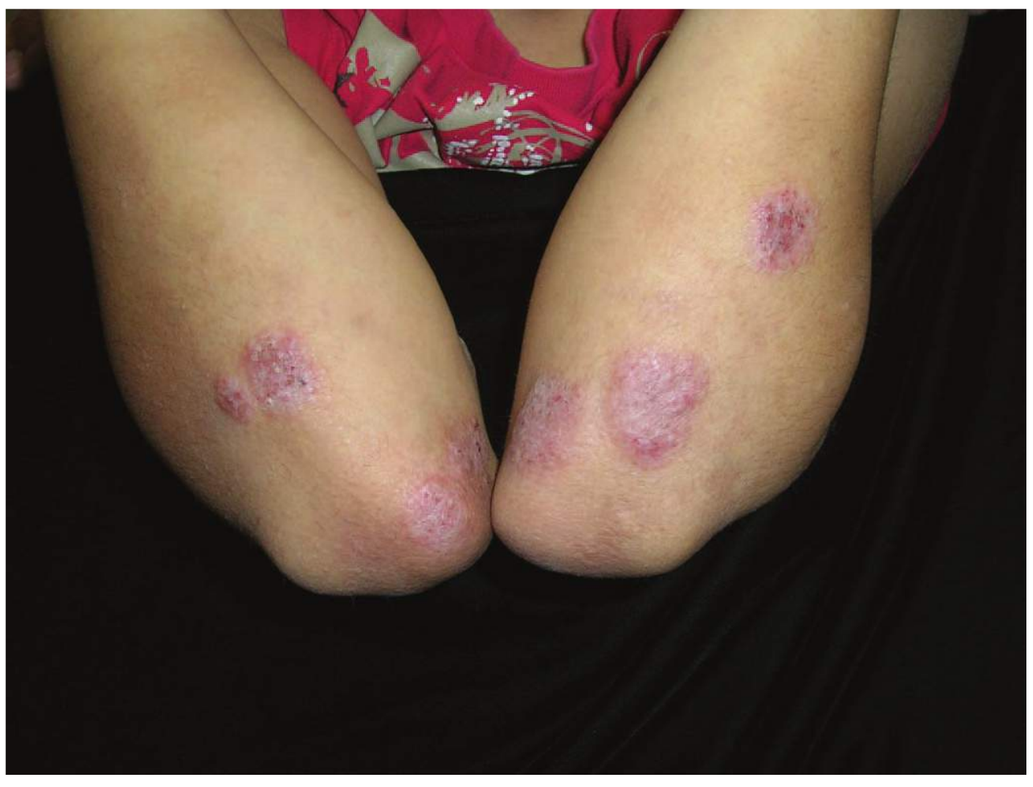


FIGURE 13.39 ■ **Eczema.** Nummular eczema on the extensor surface of the forearms and elbows. Thickened, scaly lesions resemble psoriatic plaques. A biopsy was performed to confirm the diagnosis of nummular eczema. (Photo contributor: Richard P. Usatine, MD. Used with permission. From Usatine RP, Smith MA, Mayeaux EJ, Chumley HS. The Color Atlas of Family Medicine. 2nd ed. McGraw-Hill; 2013: Fig. 148-6.)

## Clinical Summary

Dyshidrotic eczema (also called pompholyx or acute vesiculobullous hand eczema) is the abrupt appearance of deep-seated, 1- to 2-mm vesicles on the sides of the fingers, palms, and soles. The vesicles are extremely pruritic, may coalesce into larger bullae, and may rupture to become dry or fissured. The outbreak usually resolves over a few weeks unless secondary infection develops. The differential includes bullous tinea, id reaction, scabies infestation, or allergic contact dermatitis.

## Management and Disposition

Treatment includes a high-potency topical steroid and prevention of secondary infection. Refer to a dermatologist for long-term treatment; this is often a chronic condition with significant disability.

## Pearls

1. The initial lesions resemble tapioca pudding.
2. These lesions wax and wane and are often exacerbated by stressful events.

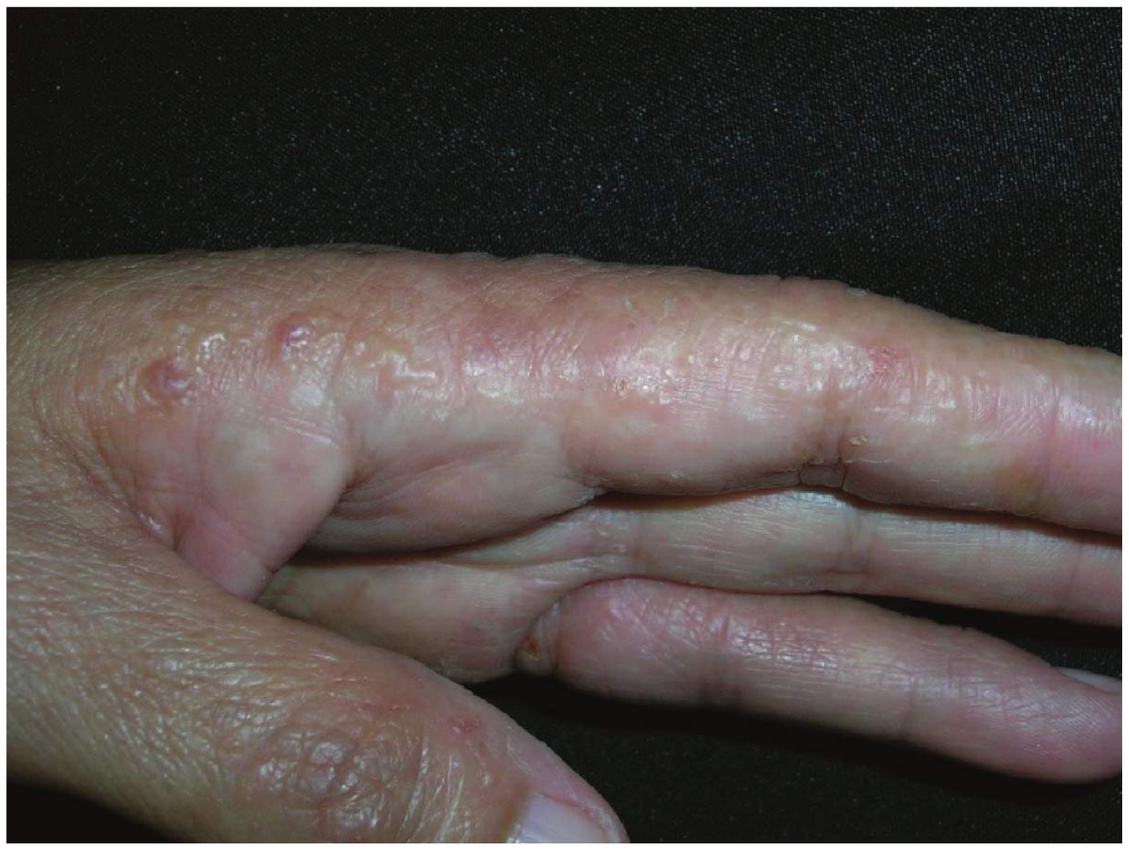


FIGURE 13.40 ■ **Dyshidrotic Eczema.** In most cases dyshidrotic eczematous dermatitis starts with tapioca-like vesicles on the lateral aspects of the fingers. (Photo contributor: Richard P. Usatine, MD. Used with permission. From Usatine RP, Smith MA, Mayeaux EJ, Chumley HS. The Color Atlas of Family Medicine. 2nd ed. McGraw-Hill; 2013: Fig. 147-7.)

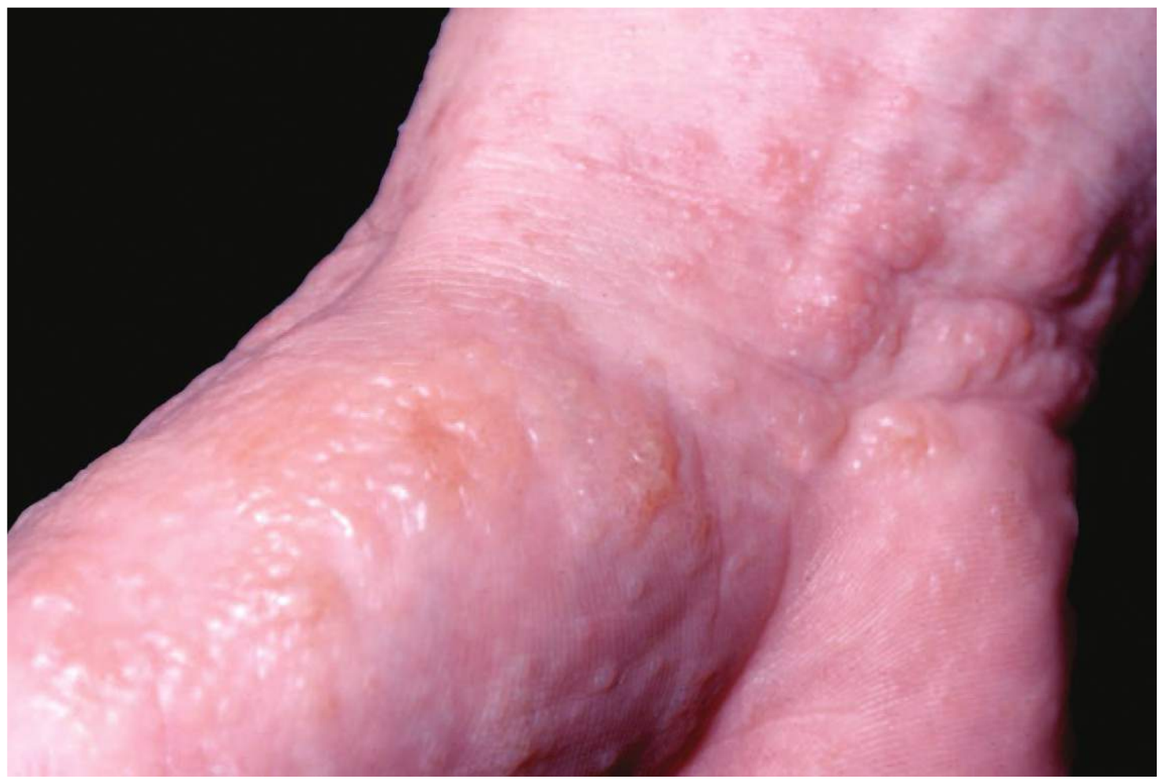


FIGURE 13.41 ■ **Dyshidrotic Eczema.** The vesicles show confluence and spread to the palm but also to the wrist and dorsal aspect of the hand when the eruption progresses. (Used with permission from Wolff K, Johnson RA, Suurmond D. Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology. 5th ed. New York, NY: McGraw-Hill; 2005:45.)

# ID REACTION (DISSEMINATED ECZEMA)

## Clinical Summary

Id reactions are seen in response to a variety of disorders, including fungal infection (tinea capitis and tinea pedis), scabies infestation, pediculosis capitus, molluscum contagiosum, bacterial and mycobacterial infections, and arthropod bites. The rash appears days to weeks after the instigating rash and consists of erythematous papules (sometimes crusted at the apices) as well as eczematous patches and plaques. The rash can be local to the instigating lesions/rash, distant, or generalized. The id reaction usually presents on the extremities, commonly on the sides of fingers, but may occur on the face and trunk. Pruritus is intense. The id reaction will not demonstrate infectious organisms and may not respond to topical steroids.

## Management and Disposition

Recognition and treatment of the initial infection or infestation is curative. Refer to a dermatologist for follow-up to ensure diagnosis and resolution.

## Pearls

1. Repeated ED evaluation for fungal infection or eczematous rash should prompt further investigation for a distant, untreated, or occult fungal infection.
2. Id reactions are intensely pruritic; make sure secondary bacterial infections do not develop from excoriations.
3. Recurrences are common, especially if the primary source is not treated adequately.

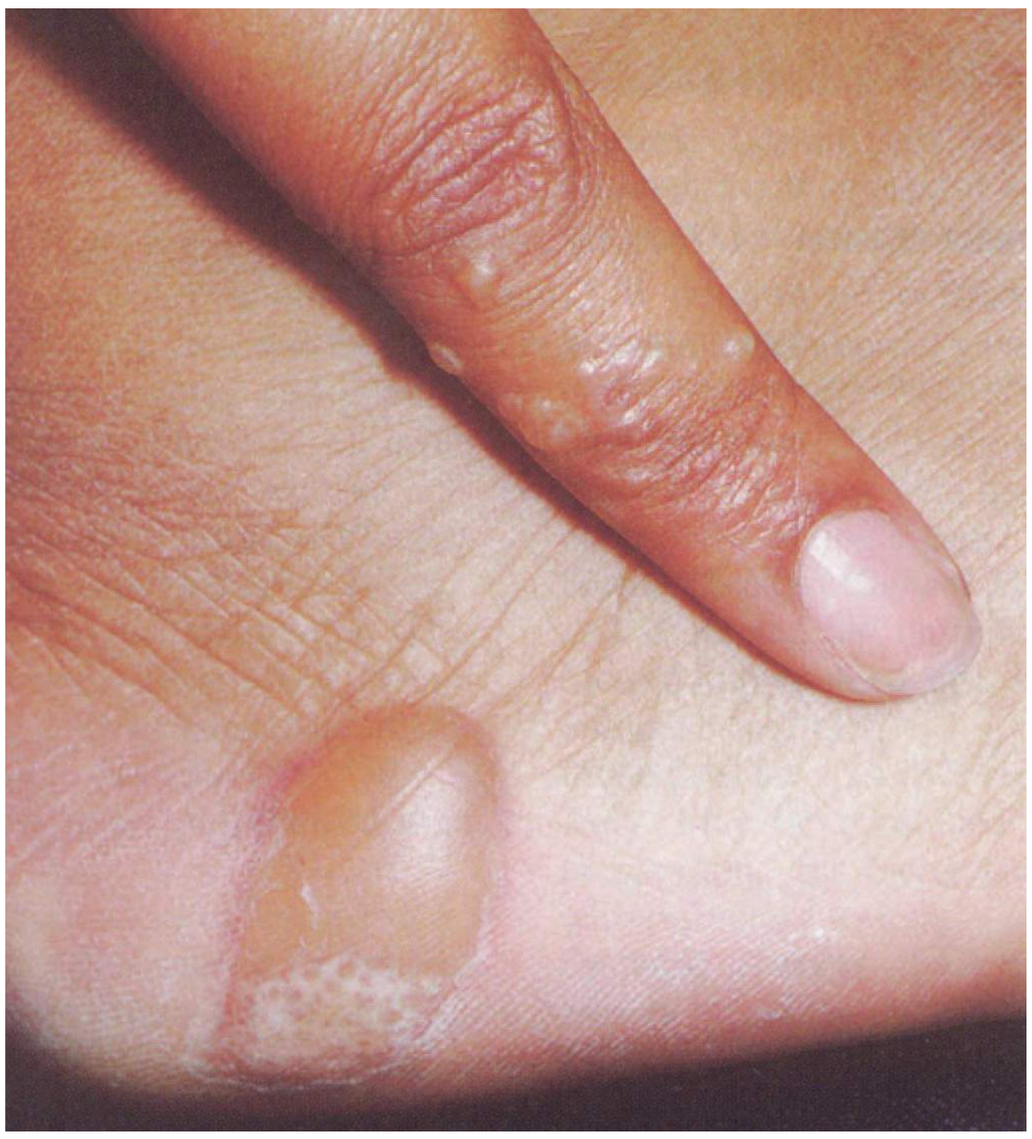


FIGURE 13.42 ■ **Id Reaction.** Bullous tinea pedis causing id reaction on the fingers. (Used with permission from Wolff K, Johnson RA, Suurmond D. Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology. 5th ed. New York, NY: McGraw-Hill; 2005: 48.)

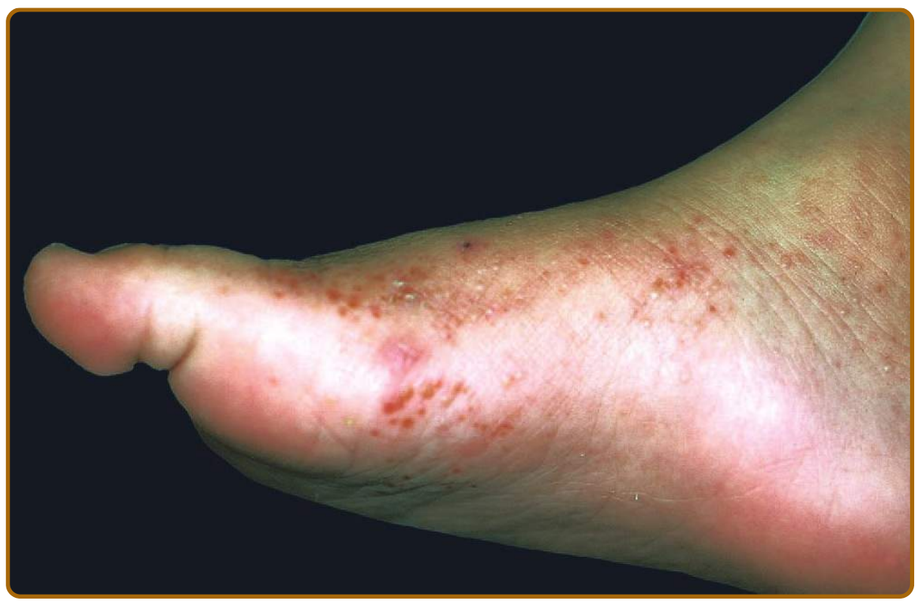


FIGURE 13.43 ■ **Id Reaction.** Id reaction to tinea pedis. Erythematous, partially dried up, pruritic vesicles on the foot. (Used with permission from Goldsmith LA, Katz SI, Gilchrest BA, Paller AS, Leffell DJ, Wolff K. Fitzpatrick's Dermatology in General Medicine. 8th ed. McGraw-Hill; 2012: Fig. 16-7.)

## Clinical Summary

Stasis dermatitis results from venous insufficiency, is characteristically distributed on the distal tibia above the medial malleolus, and appears early with erythematous patches. These can progress to scaling as well as eczematous and weeping plaques. Patients will often have light brown pigmentation distributed on the lower third of the extremity due to microvasculature blood extravasation (hemosiderin deposition secondary to increased superficial capillary pressure). Varicose veins are usually present, although they are often difficult to visualize in obese patients. Patients with heart failure, cirrhosis, and nephrotic syndrome are at increased risk due to a chronic edematous state.

## Management and Disposition

Referral to a primary care physician should be initiated to address the underlying etiology (eg, valvular insufficiency, thromboembolic disease, chronic edematous state). Daily elevation of the extremities and compression hose use should be encouraged. Emollients and mid-potency topical steroids help decrease the pruritus and promote healing.

## Pearls

1. Differentiation of stasis dermatitis and early cellulitis can be extremely difficult. Clinical history and deviation from previous presentations may be helpful. Follow-up for reevaluation should be obtained.
2. Due to the chronic and repetitive nature of stasis dermatitis, patients use many over-the-counter products. These may contribute to allergic and irritant contact dermatitis.
3. An associated contact dermatitis may initiate an "autosensitization" rash—presenting with erythematous patches on the legs and arms. Stop nonessential topical medications or other products.

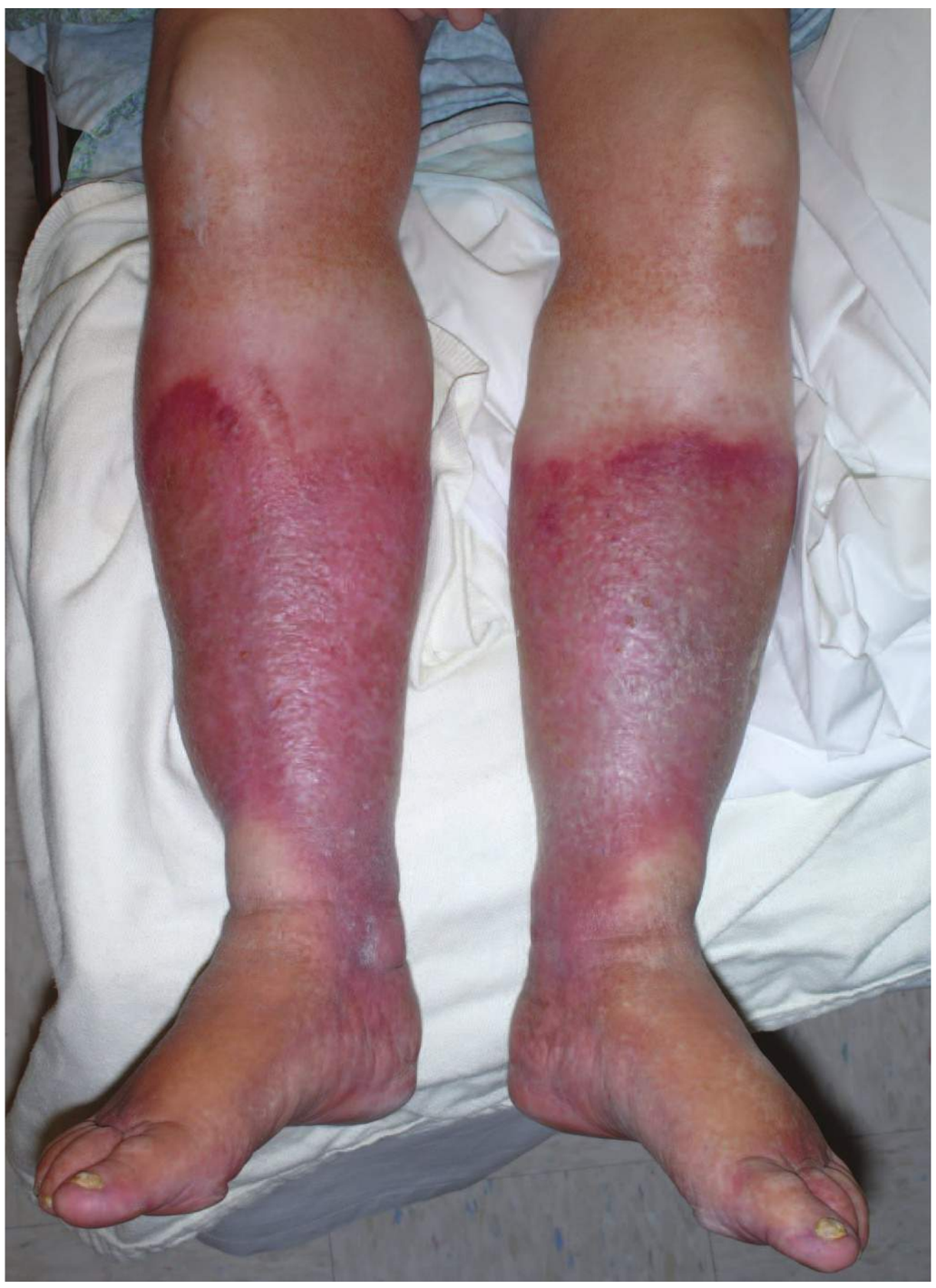


FIGURE 13.44 ■ **Stasis Dermatitis.** Erythematous patches and mild scaling in a patient with chronic venous insufficiency. Note location above the ankles. (Photo contributor: Lawrence B. Stack, MD.)

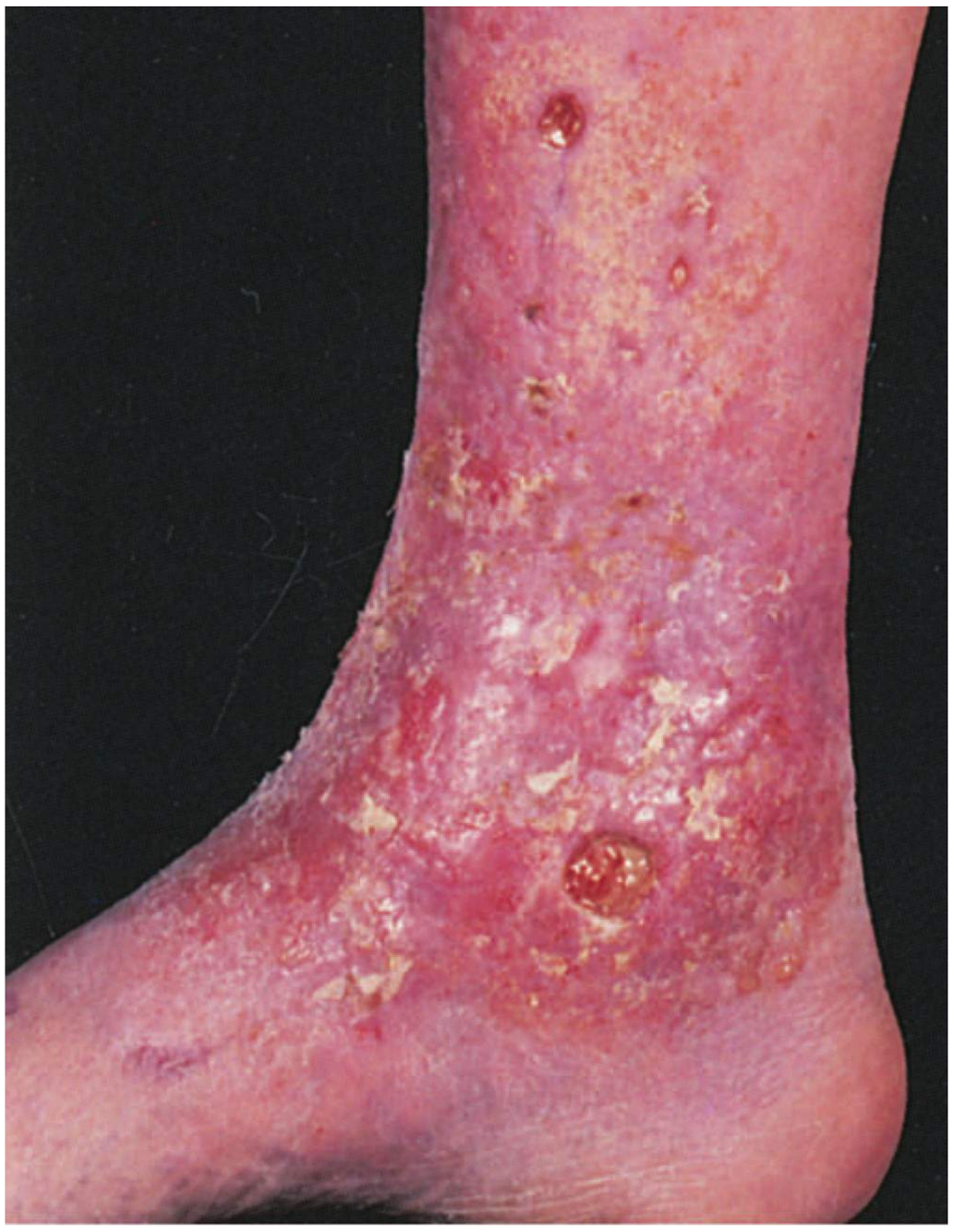


FIGURE 13.45 ■ **Stasis Dermatitis.** An example of stasis dermatitis showing erythematous, scaly, and oozing patches over the lower leg. Several stasis ulcers are also present. (Used with permission from Fauci AS, Braunwald E, Kasper DL, et al. Harrison's Principles of Internal Medicine. 17th ed. New York, NY: McGraw-Hill; 2008: 315.)

# LIVEDO RETICULARIS

## Clinical Summary

Livedo reticularis is a reactive macular, reticulated (lace-like) patch of nonpalpable cutaneous vasodilatation due to vasospasm, vessel wall damage, and intraluminal pathology. It occurs in many systemic diseases including connective tissue disorders, vasculitis, polycythemia vera, cold agglutinins, hypercoagulability diseases, thrombotic thrombocytopenic purpura, embolic disease, decompression sickness, and infections/sepsis. It has also been associated with medications such as amantadine, quinine, and quinidine. Physiologic livedo reticularis is a response to cold temperatures and is a common finding in infants, children, and adults prone to acrocyanosis.

## Management and Disposition

Management is dependent on treating the underlying disorder. Patients without an acute medical condition can be referred to dermatology.

## Pearls

1. Physiologic livedo reticularis improves or disappears with warming, whereas secondary causes usually do not.
2. Patchy, nonsymmetric distribution of livedo reticularis should elicit concern for more serious underlying diseases.

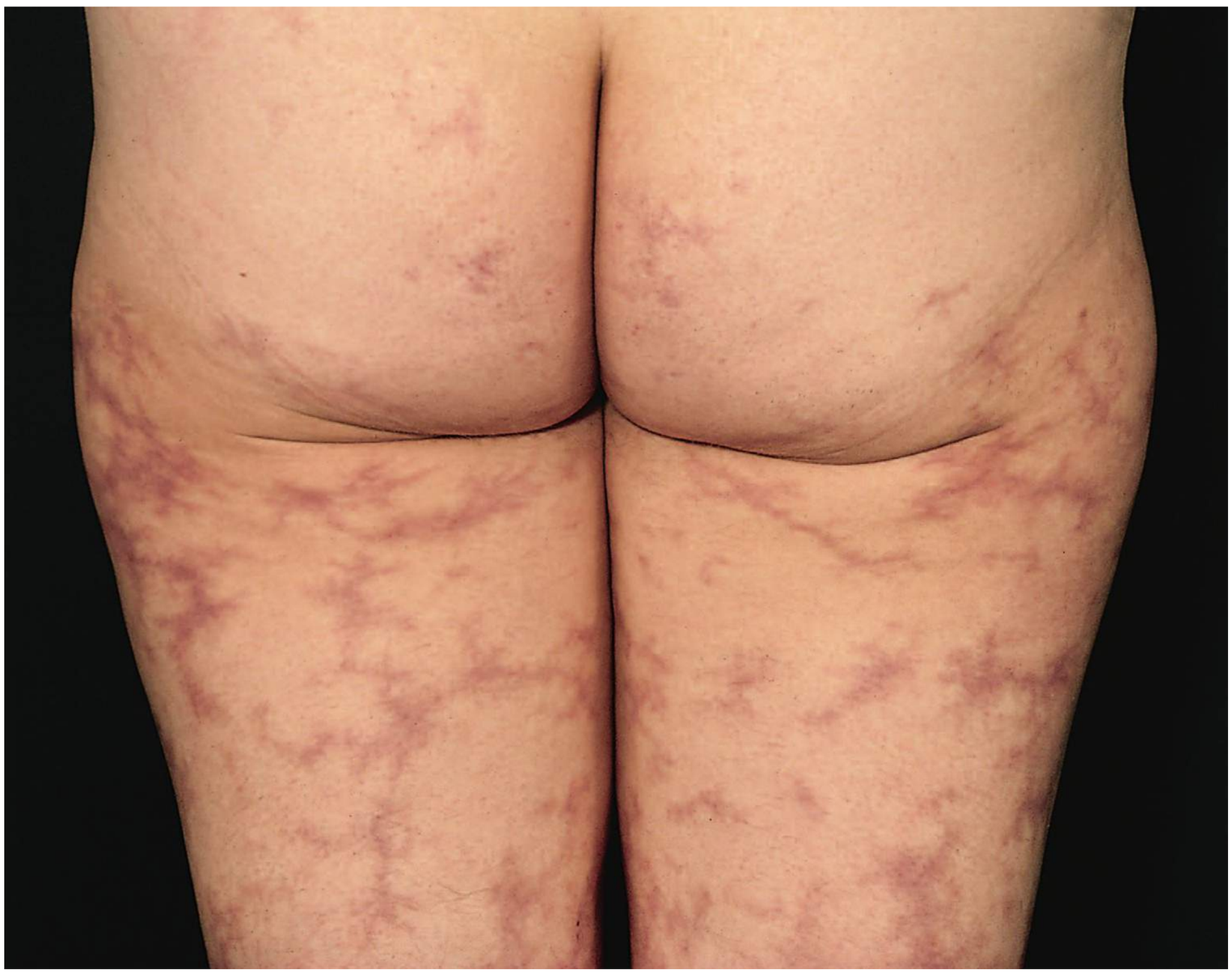


FIGURE 13.46 ■ **Livedo Reticularis.** A netlike, arborizing pattern on the posterior thighs and buttocks defined by violaceous, erythematous streaks resembling lightning. The skin within the erythematous areas is normally pale. (Used with permission from Wolff K, Johnson RA, Saavedra AP. Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology. 7th ed. McGraw-Hill; 2013: Fig. 14-42.)

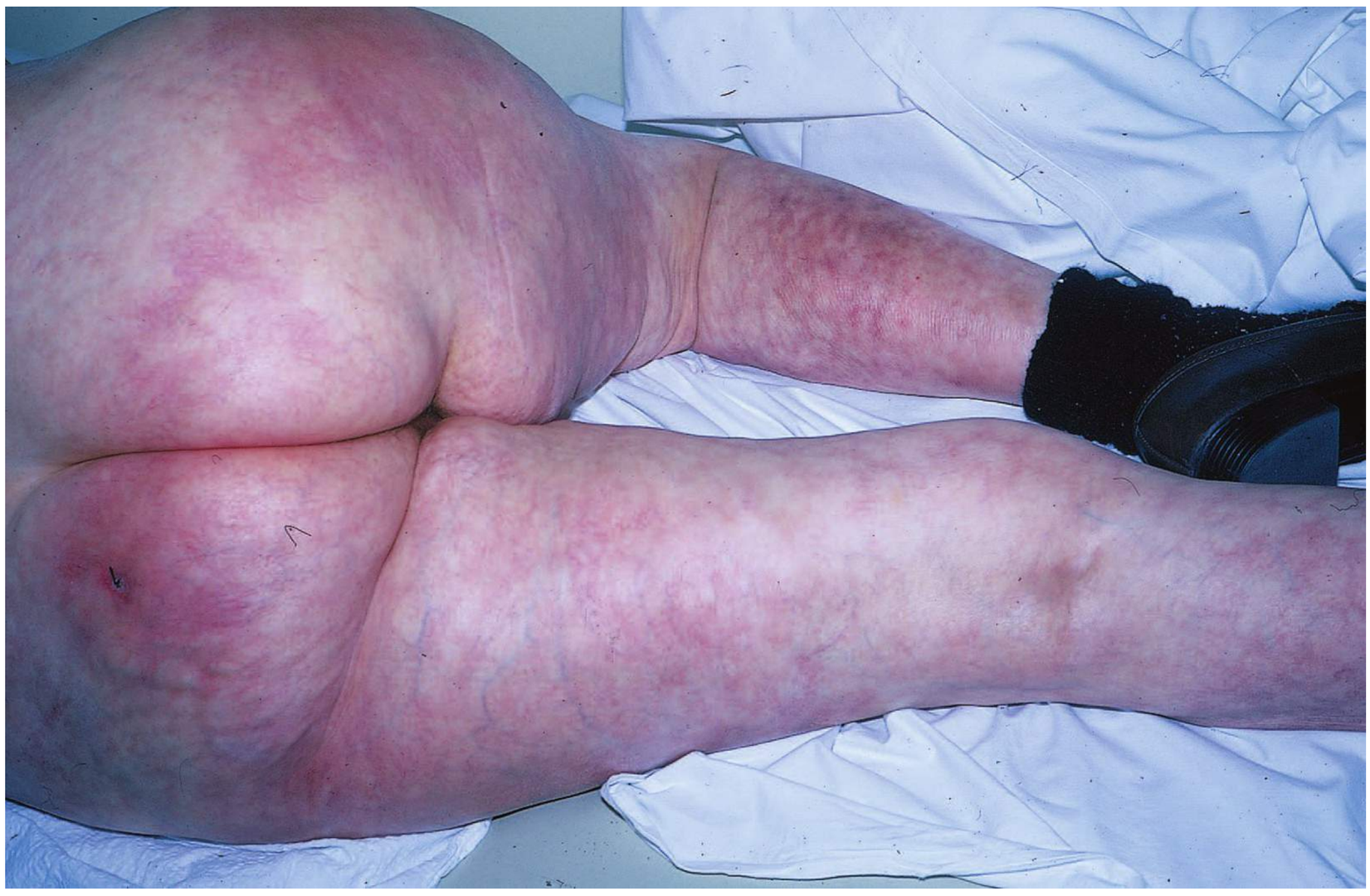


FIGURE 13.47 ■ **Livedo Reticularis.** Reticulated (lacelike) branching erythema symmetrically distributed over the lower extremities (Photo contributor: James J. Nordlund, MD.)

# VASCULITIS (LEUKOCYTOCLASTIC VASCULITIS)

## Clinical Summary

Leukocytoclastic vasculitis (LCV) represents the deposition of immune complexes in small blood vessels with subsequent vessel damage and blood extravasation. Nonblanching, purpuric macules and erythematous papules frequently coalesce into plaques ("palpable purpura"). The lower extremities and dependent areas of the back and buttocks are most frequently involved. Pruritus can be significant. Vesicles, ulcers, and necrosis may be seen within the purpuric lesions. A crop of lesions appear over a few days and usually resolve with hyperpigmentation over 4 to 6 weeks. Associated symptoms may include fever, arthralgias, myalgias, and malaise.

LCV is associated with many chronic diseases (connective tissue diseases, malignancies, and inflammatory bowel disease), medications (cephalosporins, penicillins, sulfonamides, minocycline, thiazides, allopurinol, phenytoin, NSAIDs, oral contraceptives, antithyroid agents), infections (group A β-hemolytic streptococci, mycobacterium leprae, viral hepatitis, HIV), and idiopathic subtypes (Henoch-Schönlein purpura [HSP], acute hemorrhagic edema of childhood, urticarial vasculitis).

HSP is a unique form of LCV with palpable purpura of the lower extremities and buttocks. Occasionally, the lesions may be found on the upper extremities, trunk, and face. A recent respiratory infection, arthralgias, abdominal pain, and hematuria are commonly noted. Renal vasculitis can occur in up to 40%, but chronic renal impairment occurs in only 2%. Adults have a tendency to have necrotic lesions (rare in children), as well as a higher incidence of renal impairment.

Urticarial vasculitis presents with burning and painful urticaria. The lesions are distinguished from common urticaria by their persistence (lasting over 24 hours), associated purpura, and resolution with hyperpigmentation. Urticarial vasculitis can be seen with connective tissue disease, viral infections, medications (NSAIDs, fluoxetine, cocaine, and others), and malignancies.

## Management and Disposition

Recognition that a diverse group of diseases can trigger LCV is the first step. Evaluation for systemic symptoms (fever or other signs of infection, hematuria, gastrointestinal bleeding, and neurologic symptoms) requiring admission and appropriate consultation should be undertaken. Most cases are self-limited and only require supportive care (rest, elevation, antihistamines, and analgesics). Systemic symptoms require admission and consideration of corticosteroids and other immunosuppressants. Dermatologic referral for mild LCV cases is indicated and, if systemic symptoms present, consultation may help expedite the diagnosis. HSP and urticarial vasculitis require dermatologic consultation as well as appropriate specialty consultations.

## Pearls

1. LCV affects all ages and has equal incidence in males and females. The etiologies are often idiopathic, but scrutinize each patient for associated diseases and medicine exposures.
2. Renal involvement associated with HSP is more common in adults and lesions above the waist.

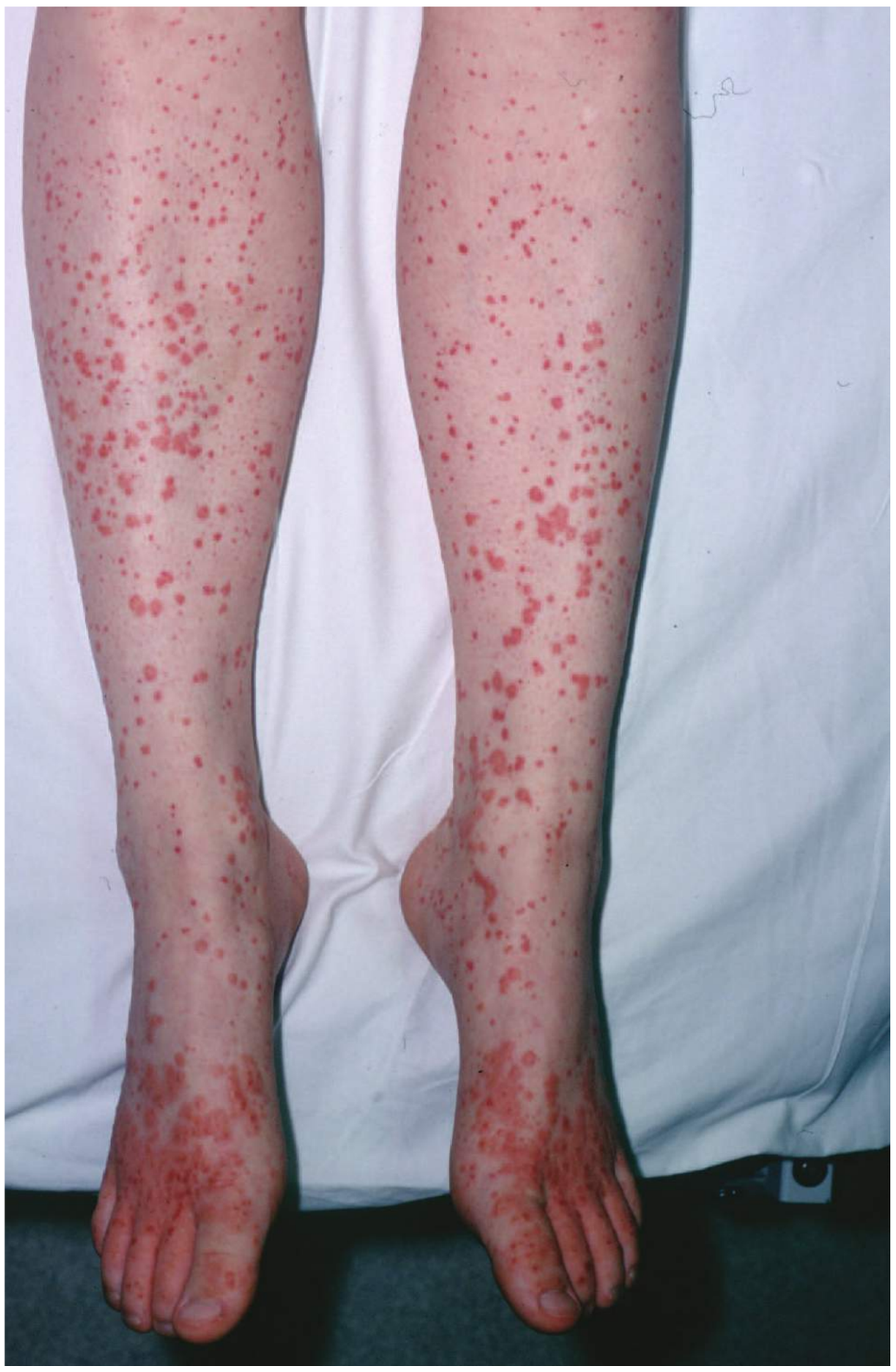


FIGURE 13.48 ■ **Leukocytoclastic Vasculitis.** Lower extremities with erythematous papules and dorsal foot with erythematous plaques. If you were to run your finger across these lesions, they would be raised and not blanch. (Photo contributor: Lawrence B. Stack, MD.)

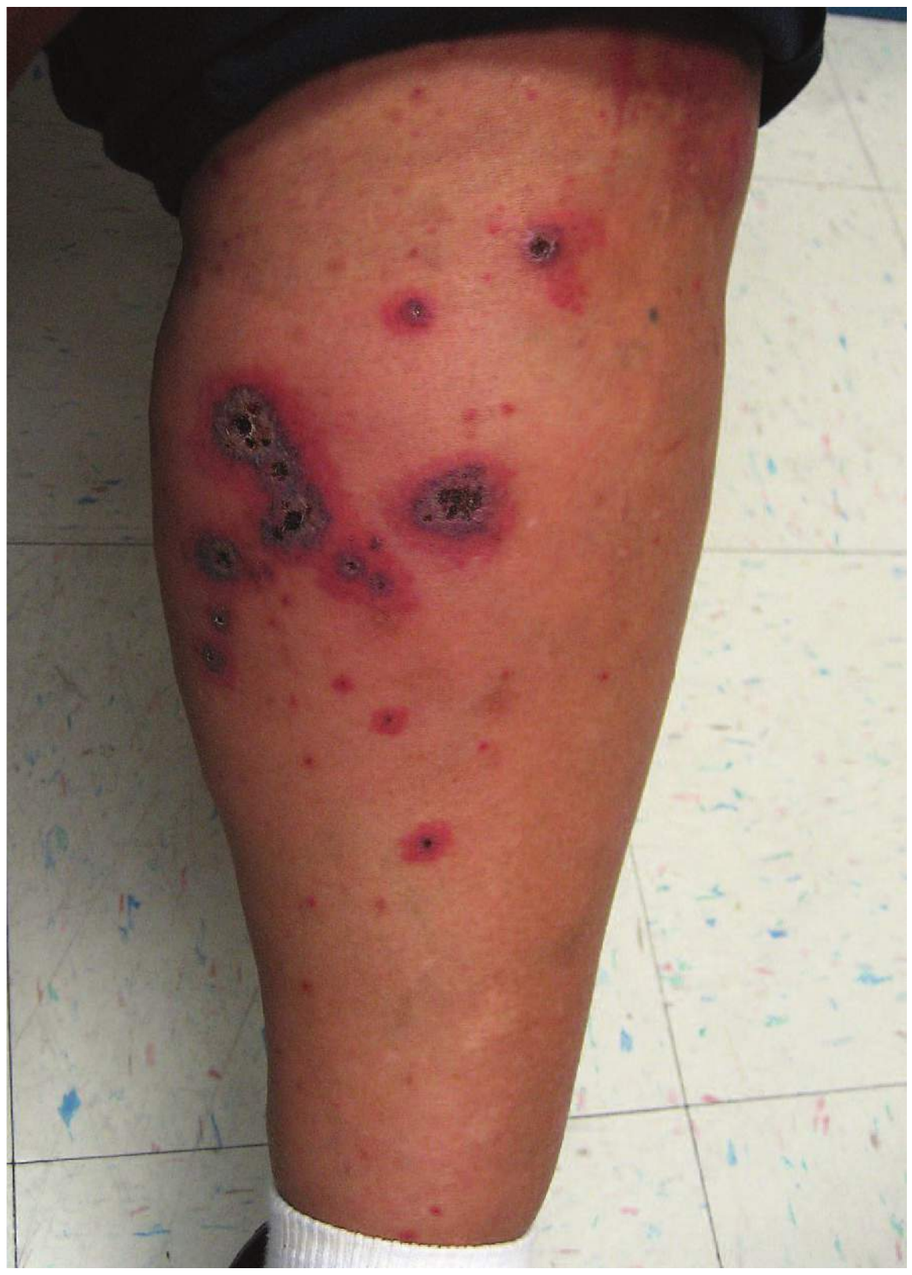


FIGURE 13.49 ■ **Leukocytoclastic Vasculitis.** Acute necrotic LCV. (Photo contributor: J. Matthew Hardin, MD.)

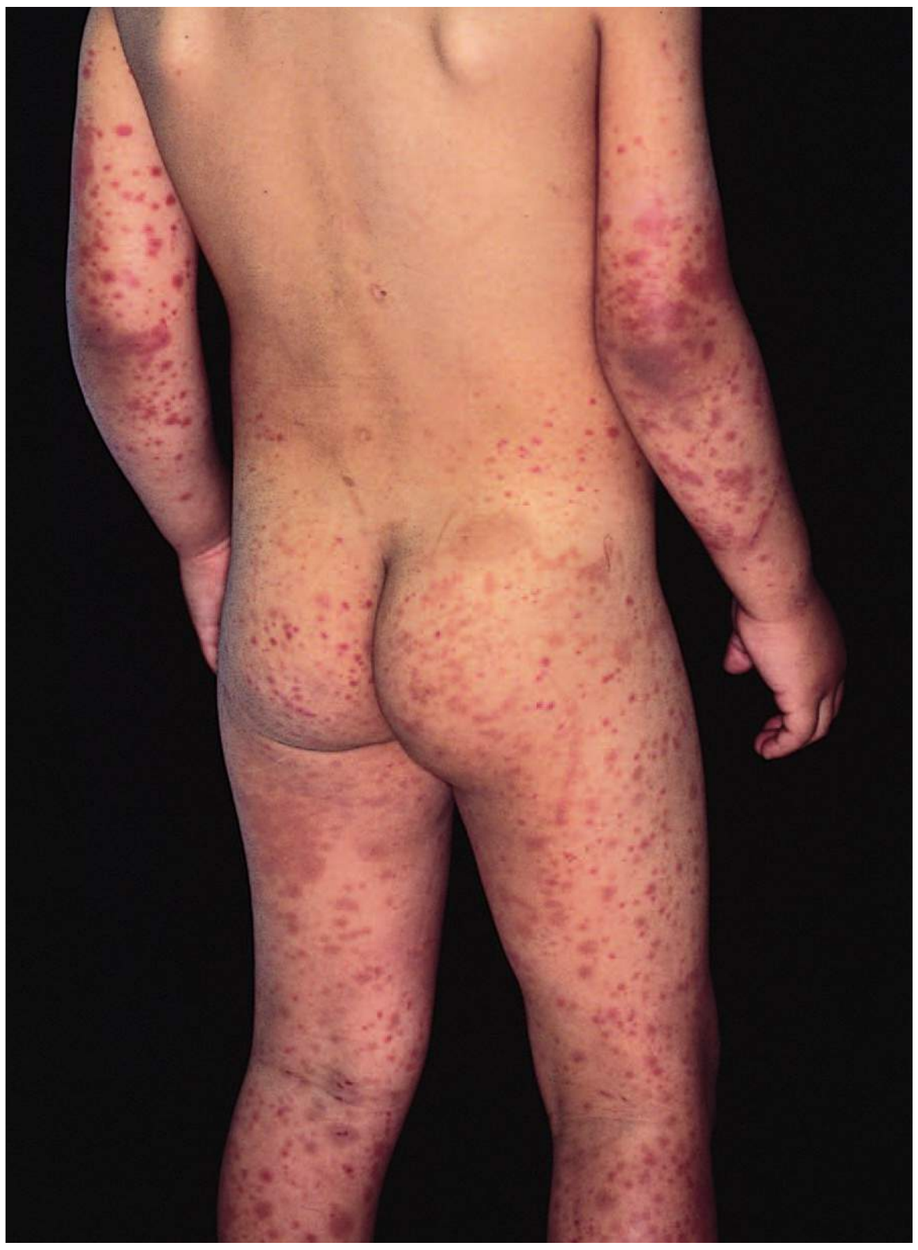


FIGURE 13.51 ■ **Henoch-Schönlein Pupura.** Note the classic acral distribution of HSP (both upper and lower extremities—raising concern for renal involvement). (Photo contributor: Kevin J. Knoop, MD, MS.)

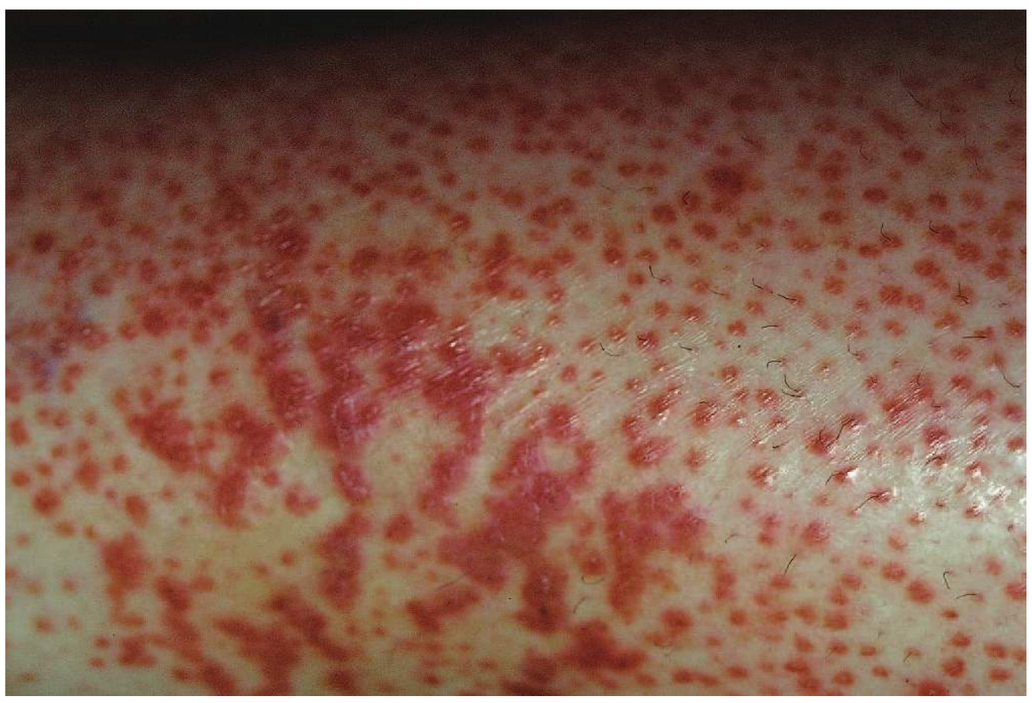


FIGURE 13.50 ■ **Leukocytoclastic Vasculitis.** Note the erythematous papules beginning to coalesce. These would not blanch with pressure. (Photo contributor: J. Matthew Hardin, MD.)

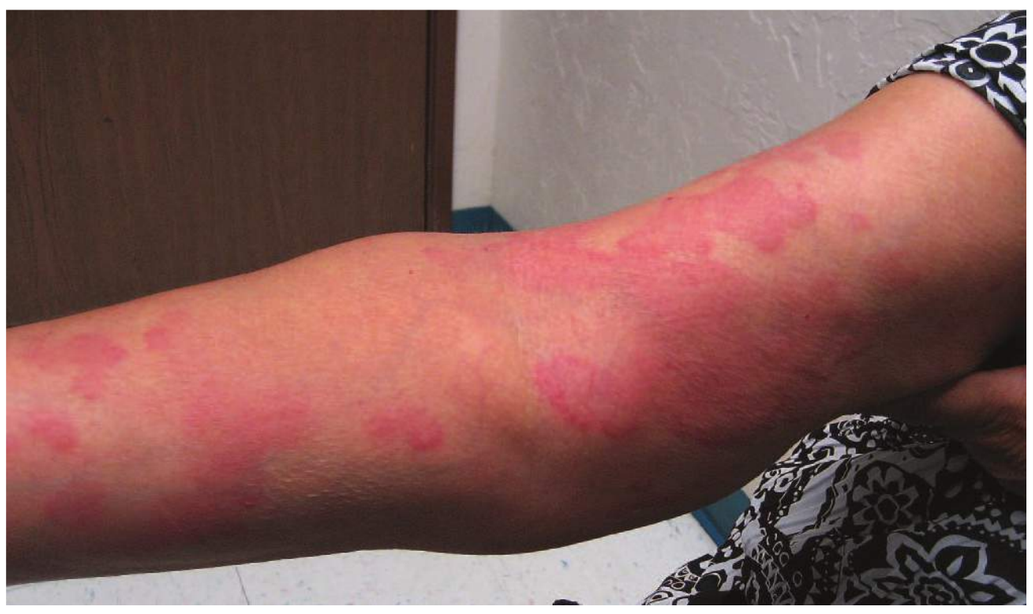


FIGURE 13.52 ■ **Urticarial Vasculitis.** Urticarial plaques present for over 24 hours. (Photo contributor: J. Matthew Hardin, MD.)

# IDIOPATHIC THROMBOCYTOPENIC PURPURA

## Clinical Summary

Idiopathic thrombocytopenic purpura (ITP) occurs as a result of platelet injury and destruction (via IgG autoantibodies to the platelet surface). Pinpoint, red, nonblanching petechiae or nonpalpable purpura and ecchymoses are found on the skin and mucous membranes, either spontaneously (platelets <10,000/mm$^3$) or at the site of minimal trauma (platelets <40,000/mm$^3$). Petechiae are commonly found on dependent areas. Gingival bleeding, melena, hematochezia, menorrhagia, and severe intracranial hemorrhages may also occur in conjunction with the thrombocytopenia. The acute form affects children (peak incidence 2-4-year-olds, equal gender distribution) after a viral illness and completely resolves in 2 months. The chronic form occurs most often in adults, with women outnumbering men 3:1. Other causes of thrombocytopenia include infections (HIV, hepatitis), medications (heparin, quinidine/quinine, sulfonamides), and alcohol.

## Management and Disposition

Consult hematology for medical management guidance. Treatment can include IV corticosteroids, IVIg, or IV Rho (D) immune globulin alone or in combination. If life-threatening hemorrhage is present, IV methylprednisolone and IVIg should be initiated along with platelet transfusions. For adult patients with longstanding, recalcitrant disease, splenectomy may be recommended.

## Pearls

1. Isolated thrombocytopenia is the hallmark finding—WBC, hemoglobin, and coagulation levels should be normal (if no major hemorrhage).
2. Patients with advanced age, comorbidities, and prior history of hemorrhage have a high risk of associated bleeding (eg, central nervous system). Keep a low threshold for imaging and insist on appropriate disposition.
3. IV Rho (D) immune globulin is only used in nonsplenectomized, Rh positive patients. Associated intravascular hemolysis can be seen and should be monitored in the ED if IV Rho (D) immune globulin used.
4. The acute form of ITP has an excellent prognosis (90% spontaneous remission), whereas the course of chronic ITP is one of varying severity with little hope of remission.

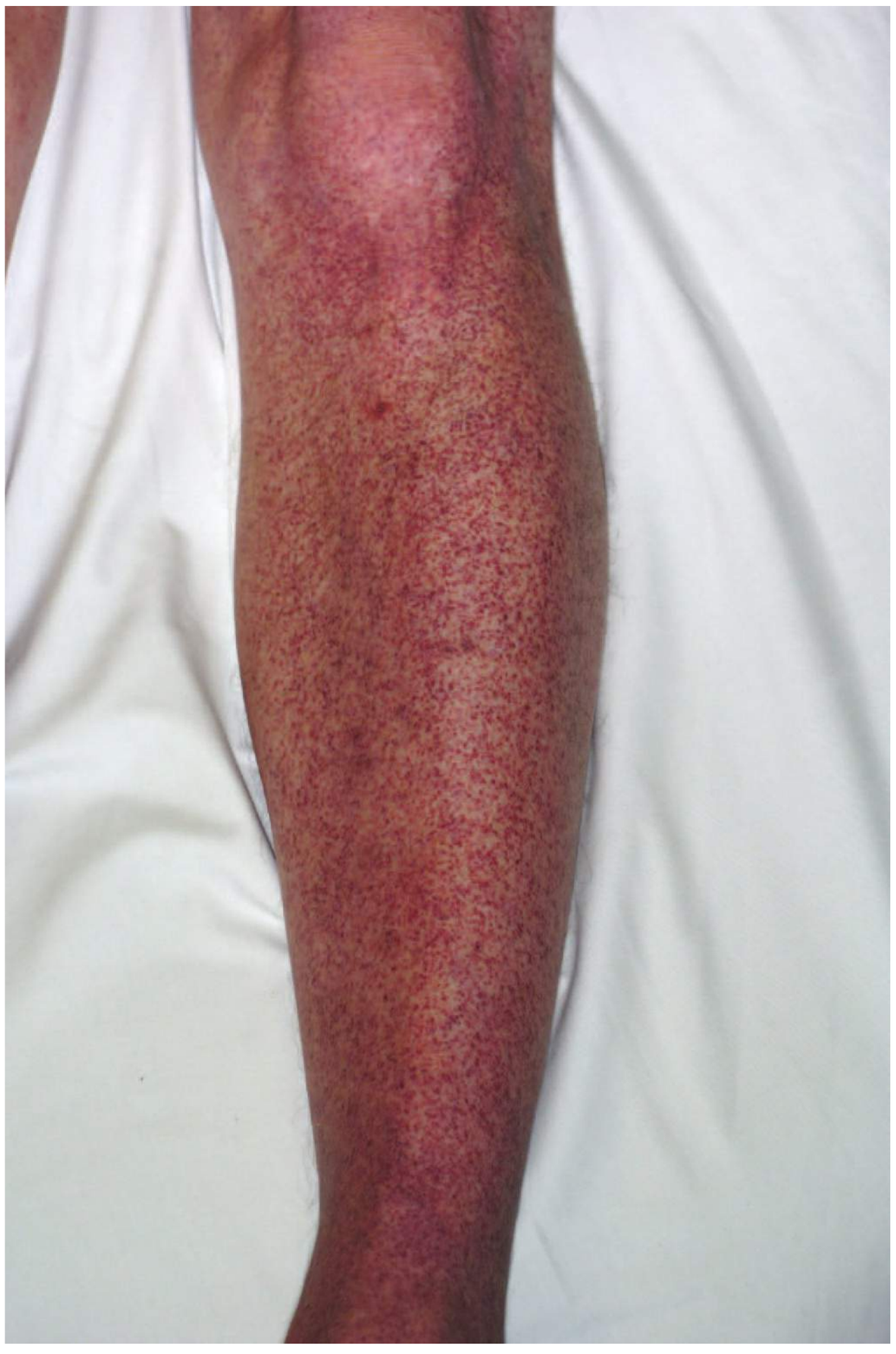


FIGURE 13.53 ■ **Idiopathic Thrombocytopenic Purpura.** This thrombocytopenic patient with splenomegaly has pinpoint, nonblanching, nonpalpable petechiae. (Photo contributor: R. Jason Thurman, MD.)

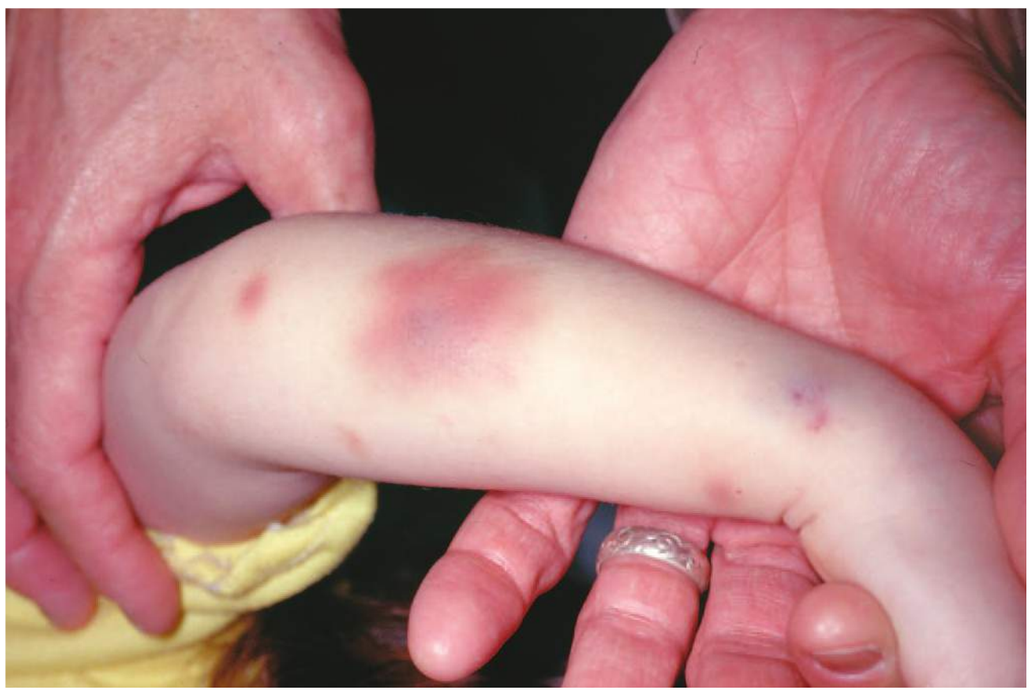


FIGURE 13.54 ■ **Idiopathic Thrombocytopenic Purpura.** Nonpalpable purpura in ITP. (Photo contributor: Lawrence B. Stack, MD.)

## Clinical Summary

Primary (idiopathic) thrombotic thrombocytopenic purpura (TTP) is characterized by the following pentad of symptoms: (1) microangiopathic hemolytic anemia (with characteristic schistocytes on the peripheral blood smear and a reticulocytosis), (2) thrombocytopenia with platelet counts ranging from 5000 to 100,000/μL, (3) renal abnormalities (including insufficiency, azotemia, proteinuria, or hematuria), (4) fever, and (5) neurologic abnormalities (including headache, mental status changes, cranial nerve palsies, seizures, or coma). Clinically, patients will have petechial rash and may have pallor and jaundice secondary to hemolytic anemia and thrombocytopenia.

Secondary TTP-hemolytic uremic syndrome (TTP-HUS) is associated with hemorrhagic colitis (Escherichia coli O157:H7 and Shigella toxin mediated), pregnancy, medications (cyclosporine, tacrolimus, ticlopidine, gemcitabine, mitomycin C, and others), metastatic carcinoma, autoimmune connective tissue disease, and antiphospholipid syndrome.

## Management and Disposition

The cornerstone of primary TTP therapy is total plasma exchange transfusion (plasmapheresis) with fresh frozen plasma. Some patients can be treated with simple plasma infusions alone until transferred to site with total plasma exchange transfusion. Patients recalcitrant to standard therapy may be treated with immunosuppressives (systemic corticosteroids, vincristine, or rituximab) and splenectomy.

Patients with TTP-HUS require supportive care with close attention to fluid balance (especially diarrheal dehydration). Signs of renal failure require emergent nephrology consultation for hemodialysis. Total plasma exchange is reserved for recalcitrant cases in children (majority improve with supportive care alone). Antibiotic use should be avoided due to an associated 17-fold increase in disease progression (presumably by bacterial release of toxin). Avoid antimotility medications with diarrheal disease.

## Pearls

1. Since only 20% to 30% of patients have the typical symptom pentad, diagnosis based on partial findings is challenging.
2. Platelet transfusions should be avoided unless there is life-threatening hemorrhage; platelet transfusions can worsen the thrombotic process leading to myocardial infarction and cerebrovascular infarctions.
3. Disseminated intravascular coagulation and the pregnancy-associated HELLP (hemolysis, elevated liver enzymes, low platelet count) syndrome can present with similar findings to primary TTP and TTP-HUS.

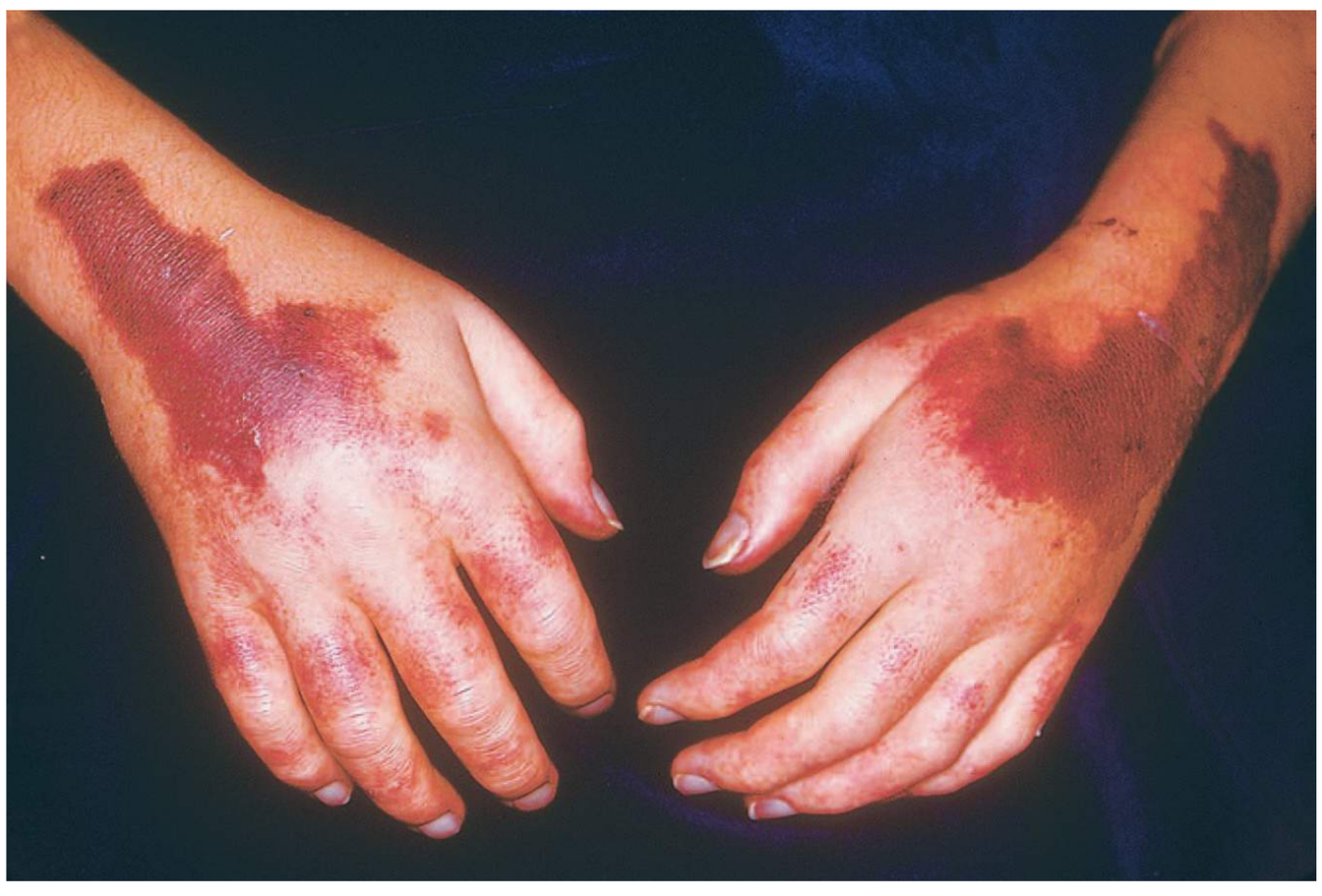


FIGURE 13.55 ■ **Thrombotic Thrombocytopenic Purpura.** Bleeding at initial presentation is seen in about 30% to 40% of patients with TTP. (Photo contributor: James J. Nordlund, MD.)

# URTICARIA AND DERMATOGRAPHISM

## Clinical Summary

Acute urticaria develops over days to weeks and presents with transient wheals. Generally, acute urticaria resolves within six weeks, whereas chronic urticaria lasts longer. Common triggers include medications (penicillin, aspirin, NSAIDs), foods (chocolate, shellfish, nuts, eggs, milk, and others) and infections (streptococcal, hepatitis B and C, mononucleosis, and helminths), and physical factors (exercise, pressure, cold, vibratory and solar-induced urticaria).

Dermatographism is the production of linear urticarial lesions and surrounding erythematous flare after stroking the skin. Simple dermatographism, seen in up to 5% of the population, is considered an exaggerated physiologic response to friction. In contrast, symptomatic dermatographism presents without a previous history. Wheals are typically seen with scratching and friction from tight clothing. Children and young adults are commonly affected and, fortunately, no associated systemic disease, autoimmunity or food allergy exists. Symptomatic dermatographism can persist for several years.

## Management and Disposition

Triggers of urticaria should be investigated and, if present, stopped. H1 and H2 blockers usually help. Systemic steroids and epinephrine are used for severe reactions and anaphylaxis.

## Pearls

1. More than 50% of chronic urticaria is idiopathic.
2. Wheals present for over 24 hours and in the same location are concerning for urticarial vasculitis (see related item) and should prompt dermatology consultation. Draw a circle around the lesions and have the patient monitor changes.

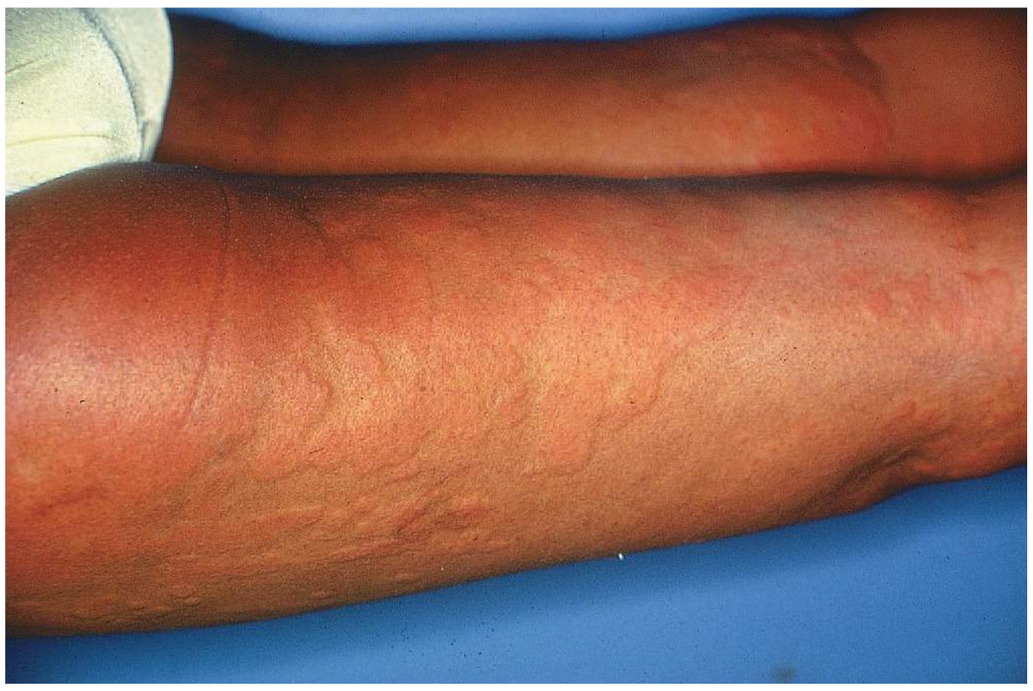


FIGURE 13.56 ■ **Acute Urticaria.** Note the classic raised plaques on the lower extremity of this patient with urticaria. (Photo contributor: James J. Nordlund, MD.)

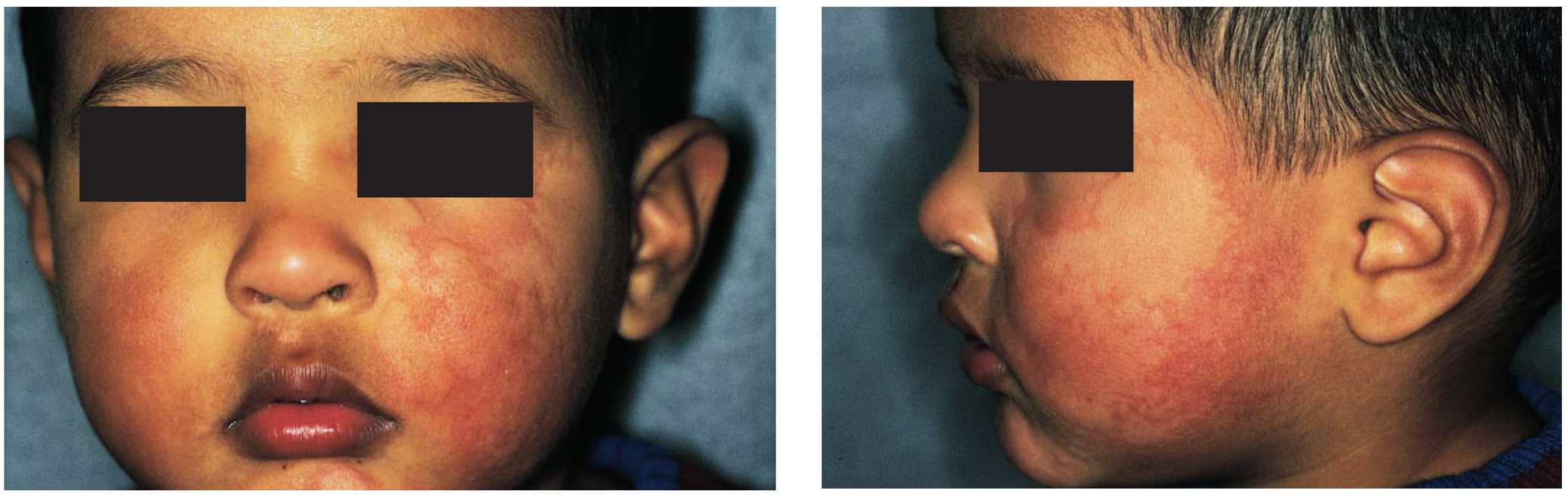


FIGURE 13.57 ■ **Acute Urticaria.** Preschool child with annular, raised pruritic lesions with central clearing and tense edema. The lesions disappeared approximately 5 minutes after presentation. (Photo contributor: Kevin J. Knoop, MD, MS.)

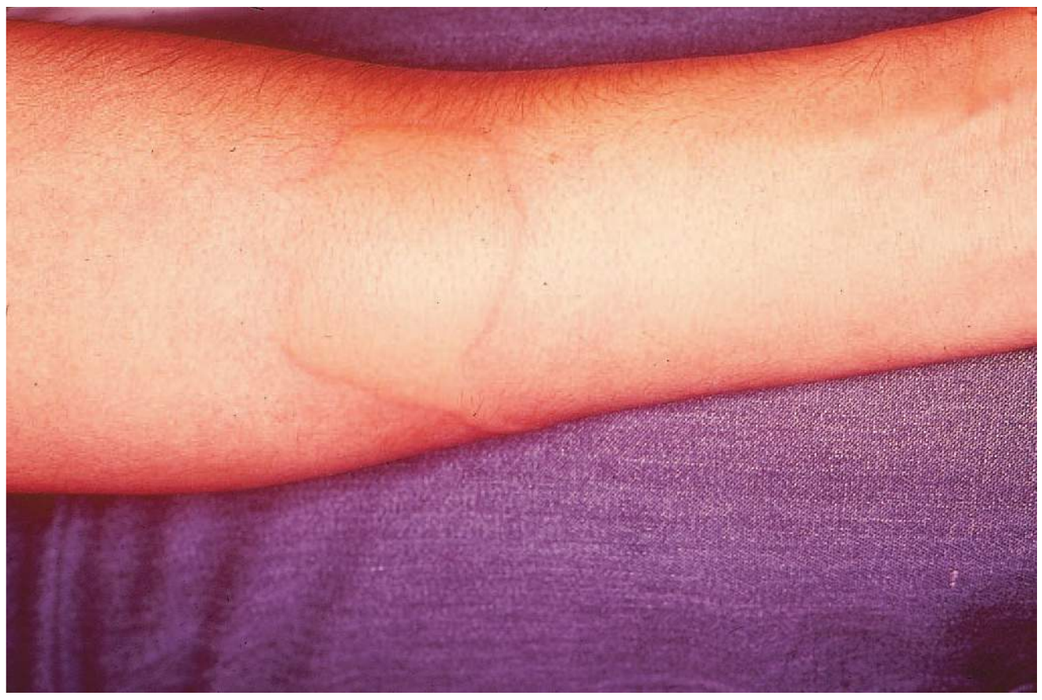


FIGURE 13.58 ■ **Cold Urticaria.** Plaque formation after placement of an ice cube on the skin confirms cold urticaria. (Photo contributor: James J. Nordlund, MD.)

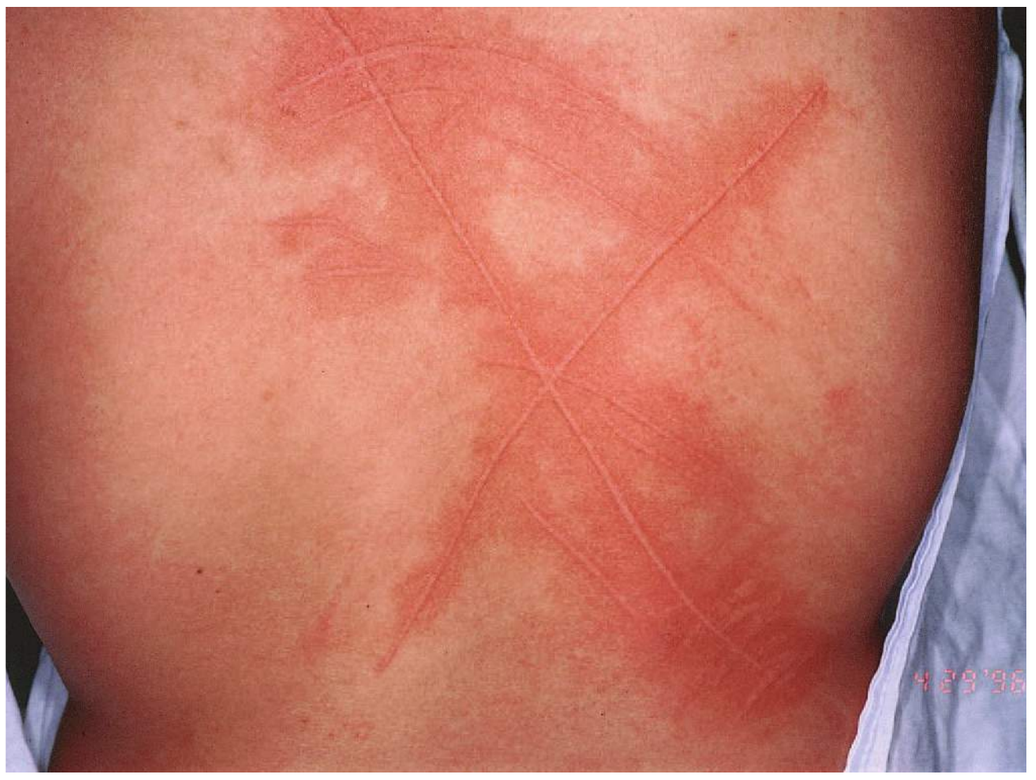


FIGURE 13.59 ■ **Dermatographism.** Note the linear wheal with surrounding erythematous flare after scratching and stroking. (Photo contributor: J. Matthew Hardin, MD.)

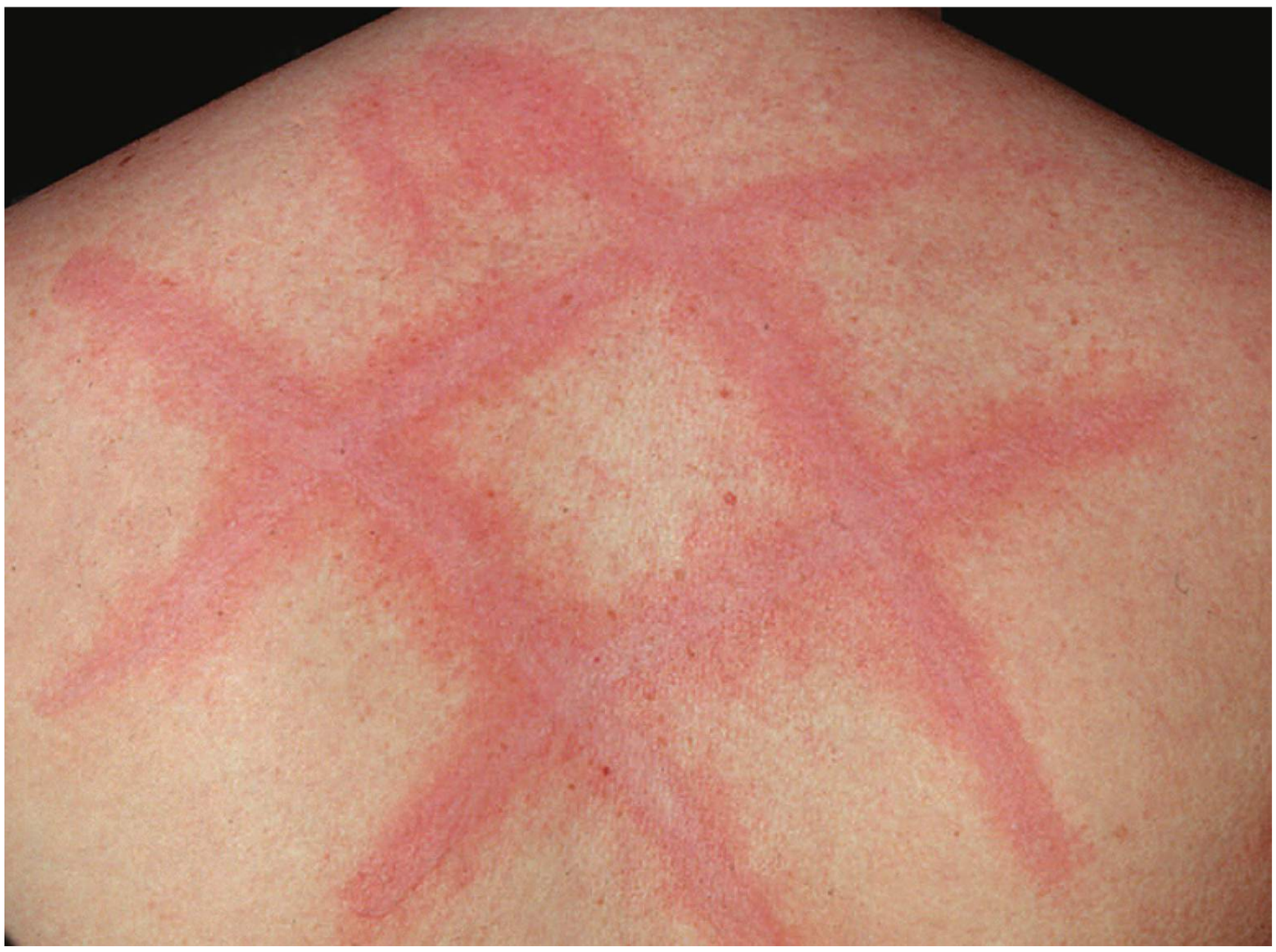


FIGURE 13.60 ■ **Dermatographism.** Urticaria 5 minutes after scratching. (Used with permission from Wolff K, Johnson RA, Saavedra AP. Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology. 7th ed. McGraw-Hill; 2013: Fig. 14-9.)

# ALLERGIC CONTACT DERMATITIS

## Clinical Summary

Allergic contact dermatitis occurs after previously sensitized skin is rechallenged with the same allergen. It represents a delayed-type hypersensitivity reaction. Papules and vesicles first develop; they can become a generalized morbilliform eruption (autosensitization). Pruritus is a dominant feature. The most common causes of allergic contact dermatitis are nickel, toxicodendrons (poison ivy, poison oak, and poison sumac), neomycin, fragrances, balsam of Peru (common in perfumes), formaldehyde, bacitracin, and rubber compounds.

## Management and Disposition

Identification of the causative agent and prevention of further contact is critical. Supportive care is given with antihistamines and topical corticosteroids. Systemic corticosteroids may be needed for generalized eruptions. Refer patients to dermatology for further evaluation and possible patch testing.

## Pearls

1. Obtaining a complete history of any and all exposures is critical. Pay particular attention to personal hygiene products.
2. Toxicodendron allergic contact dermatitis (poison ivy, poison sumac, poison oak) requires a minimum 3-week taper of oral prednisone—a shorter course allows the reappearance of the rash.
3. Bacitracin and neomycin cause 9% and 10% of allergic contact dermatitis cases, respectively.

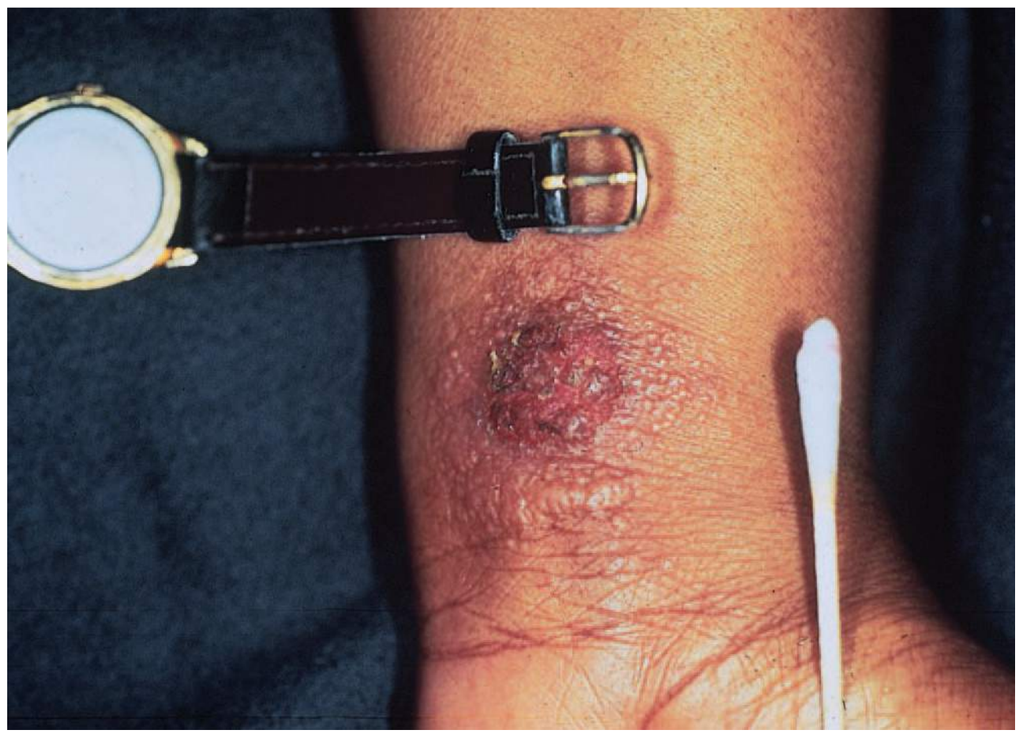


FIGURE 13.62 ■ **Contact Dermatitis.** The erythematous, edematous base of the eruption corresponds to the posterior watch surface. Superimposed on the erythematous base are multiple vesicles with exudate and crust. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)

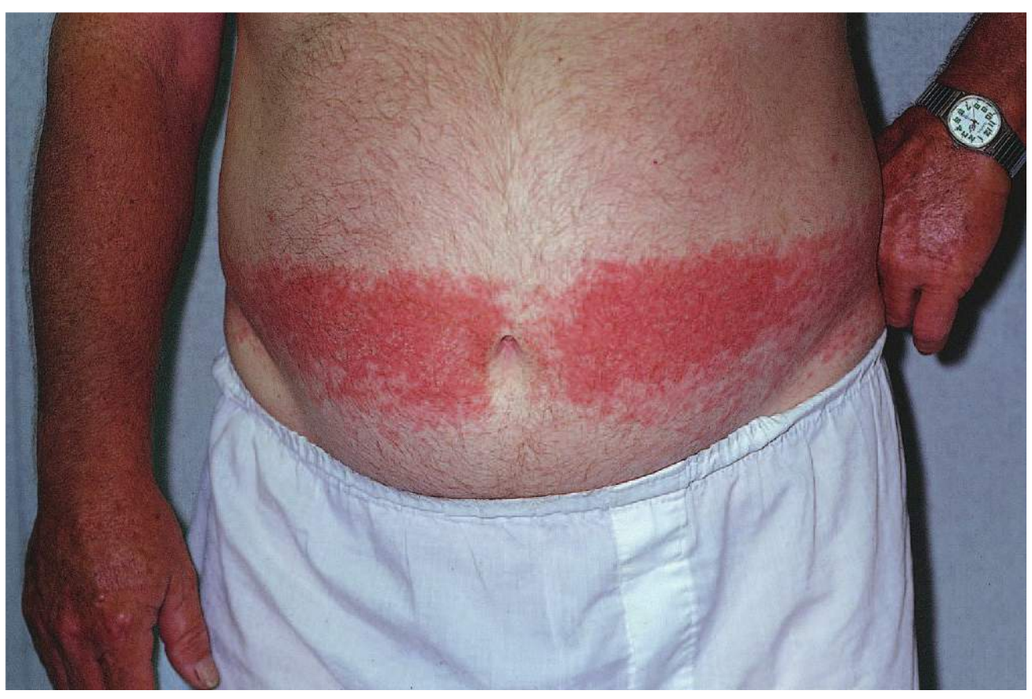


FIGURE 13.61 ■ **Contact Dermatitis.** Erythematous eruption in a waist-band distribution (elastic allergy from underwear). (Photo contributor: J. Matthew Hardin, MD.)

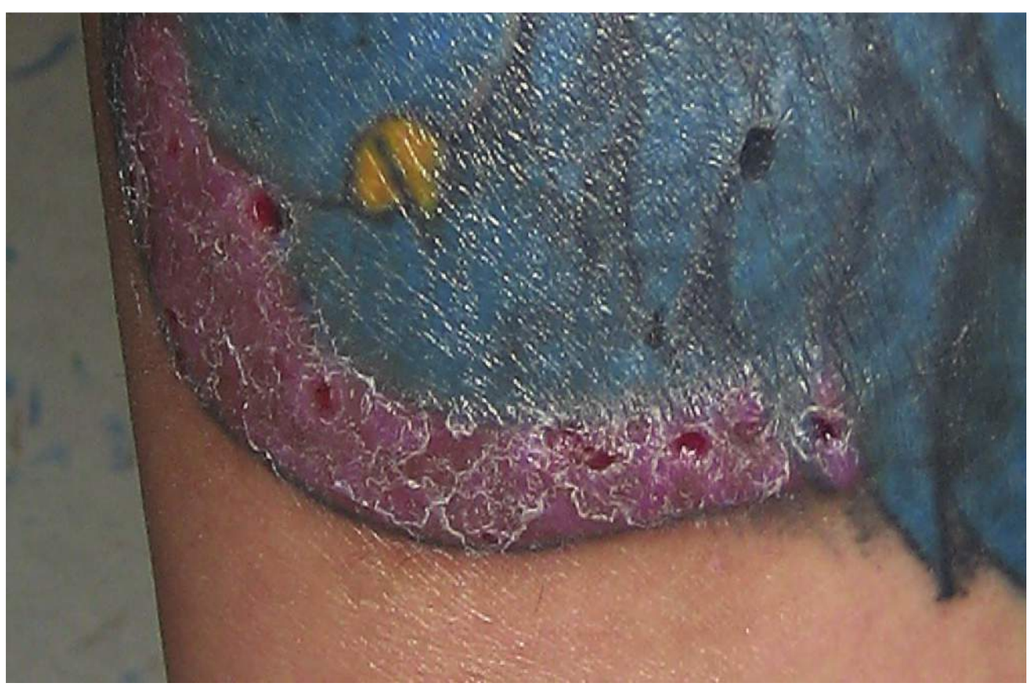


FIGURE 13.63 ■ **Contact Dermatitis.** Allergy to tattoo pigment. Note erythema, scale, and erosions contained within the tattoo image. (Photo contributor: J. Matthew Hardin, MD.)

## Clinical Summary

Erythema nodosum (EN) can present at any age but is most common in young, adult females. The most typical presentation is bilateral, erythematous, subcutaneous, tender nodules on the pretibial and lateral lower extremities (usually spares the posterior calves). Rarely, the nodules can be found on the thighs, upper extremities, and face. Concomitant symptoms often include lower extremity edema and arthralgias. Systemic symptoms can occur and include fever, headache, and gastrointestinal complaints. Generally, the nodules resolve over days to weeks with flattening and a change in color to a blue-green (like a deep bruise). There is no ulceration and the skin slowly returns to normal. Recurrence occurs in up to one-third of cases.

Multiple etiologies can present with EN—although over one-third of cases are idiopathic. Infectious causes include streptococcal, tuberculosis, Yersinia, Salmonella, Shigella, coccidioidomycosis, histoplasmosis, sporotrichosis, blastomycosis, and toxoplasmosis. EN has also been associated with pregnancy, sarcoidosis, and inflammatory bowel disease. Oral contraceptives, sulfonamides, bromides, and iodides are known to be common causative agents, among many others.

## Management and Disposition

With the many etiologies of EN, it is critical to exclude and treat an infectious, systemic, or medication cause. Supportive care with elevation of the extremity, rest, and NSAIDs is prudent. Recurrences do occur and should prompt a further workup for occult infection or persistent medication. Referral to a dermatologist for a biopsy may help confirm the diagnosis.

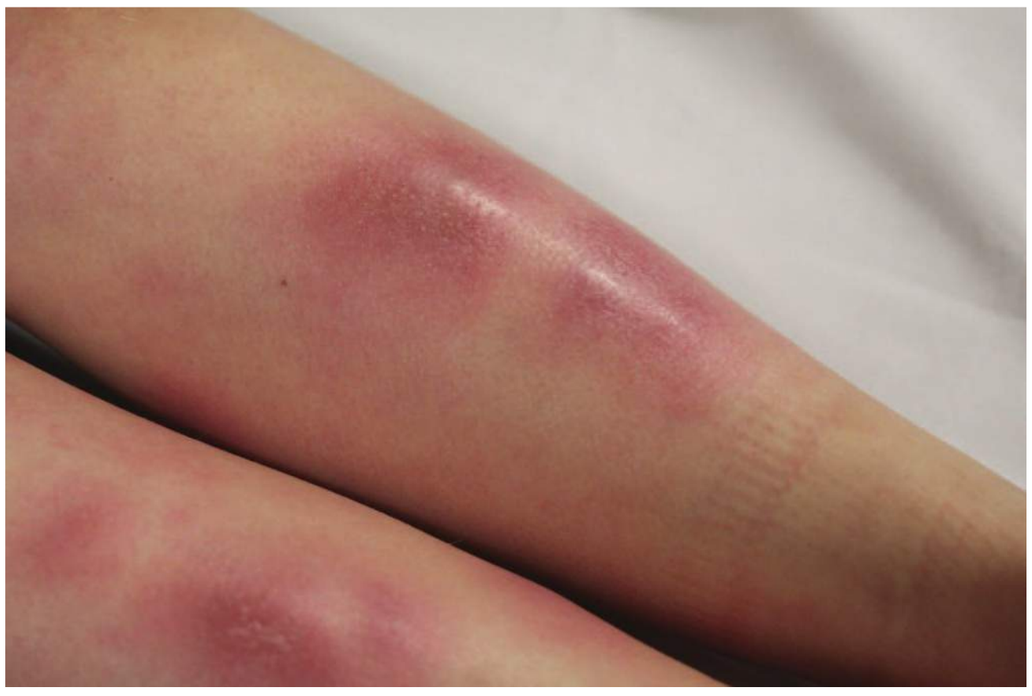


FIGURE 13.64 ■ **Erythema Nodosum, Acute.** Note the pretibial, erythematous, and subcutaneous nodules. (Photo contributor: Gianina Best, MD.)

## Pearls

1. The patient's history is very helpful in determining possible etiologies. A complete medication, travel, and past medical history must be performed.
2. Systemic glucocorticoids can be considered, but only when the etiology is clearly known and infectious agents are excluded.
3. EN is considered a good prognostic sign in sarcoidosis and pregnancy-associated coccidioidomycosis.

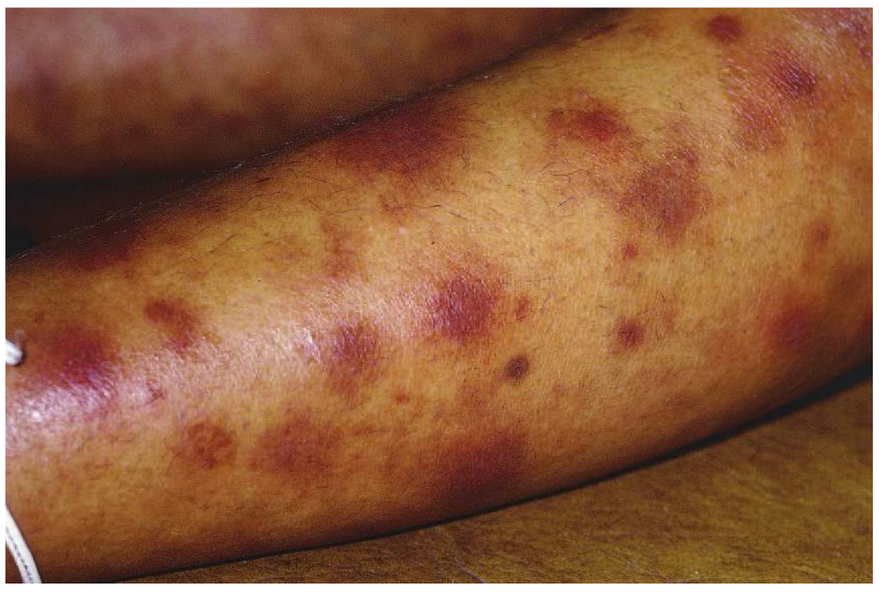


FIGURE 13.65 ■ **Erythema Nodosum, Resolving.** Note the bruise-like appearance of the resolving phase. (Photo contributor: J. Matthew Hardin, MD.)

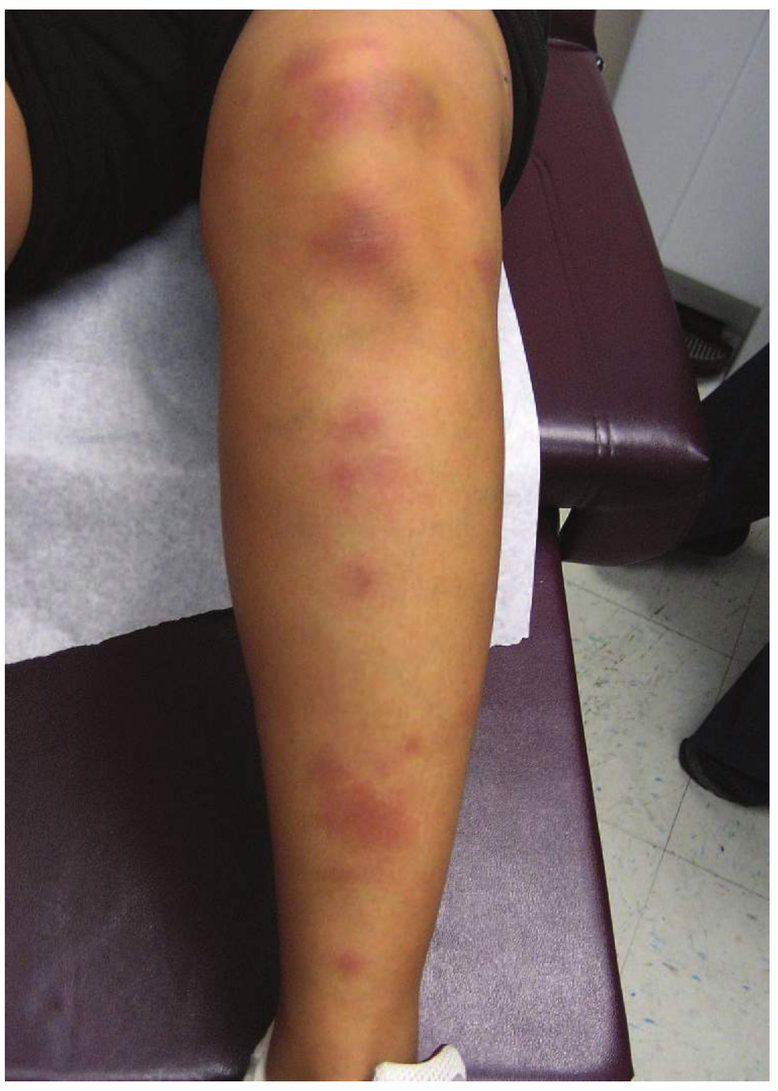


FIGURE 13.66 ■ **Erythema Nodosum.** Tender subcutaneous nodules with overlying erythematous and hyperpigmented macules—indicating a new crop of lesions. (Photo contributor: J. Matthew Hardin, MD.)

# ERYSIPELAS

## Clinical Summary

Erysipelas is a group A streptococcal cellulitis (groups G, B, C, and D can be implicated) involving the skin. The most commonly affected sites are the face and the lower extremities. Sudden onset of fever, chills, and malaise is followed by the appearance of an erythematous, edematous, and painful plaque. The characteristic border is sharp and elevated. This nonpitting edematous plaque is different from typical cellulitis due to dilation of the superficial lymphatics. Regional lymphadenopathy and lymphangitis may be present. Patients with chronic lymphedema are prone to repeated infections.

## Management and Disposition

All infections require rest, elevation, and antibiotics. Mild presentations may be treated on an outpatient basis with oral penicillins. More severe illness or toxicity requires hospitalization and IV antibiotics.

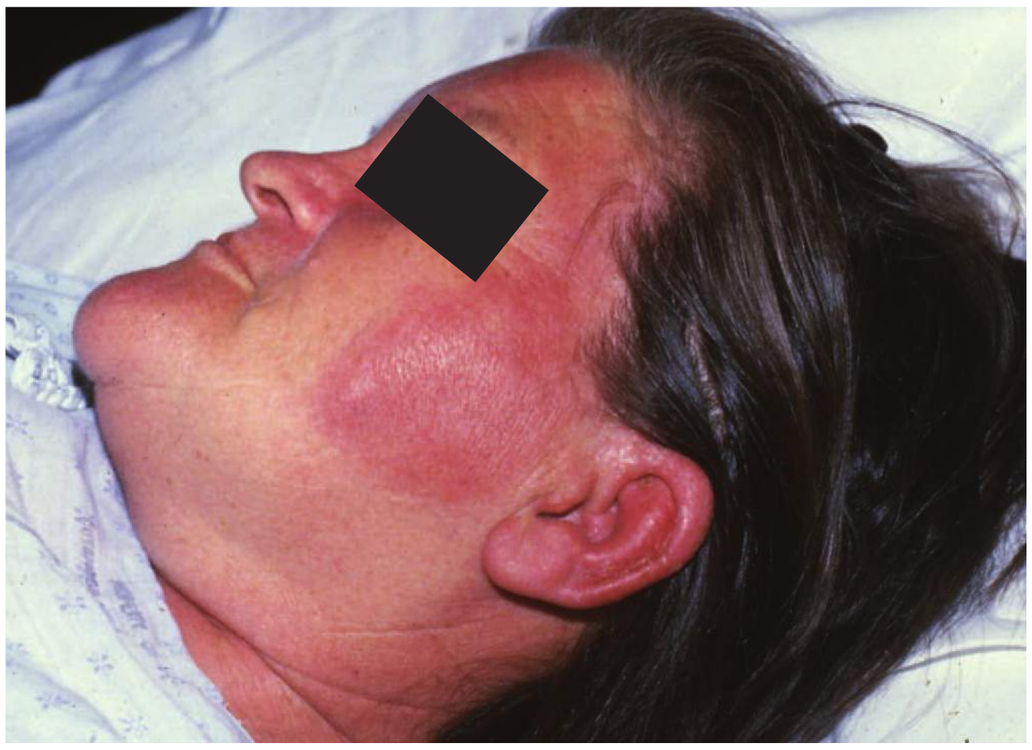


FIGURE 13.67 ■ **Erysipelas.** Sharply demarcated and elevated erythema. (Photo contributor: David Effron, MD.)

## Pearls

1. The sharp elevated border is diagnostic of erysipelas.
2. A similar shiny, erythematous (although more violaceous) plaque on the face of a febrile child may be caused by Haemophilus influenza type B in nonimmunized or immunosuppressed individuals.

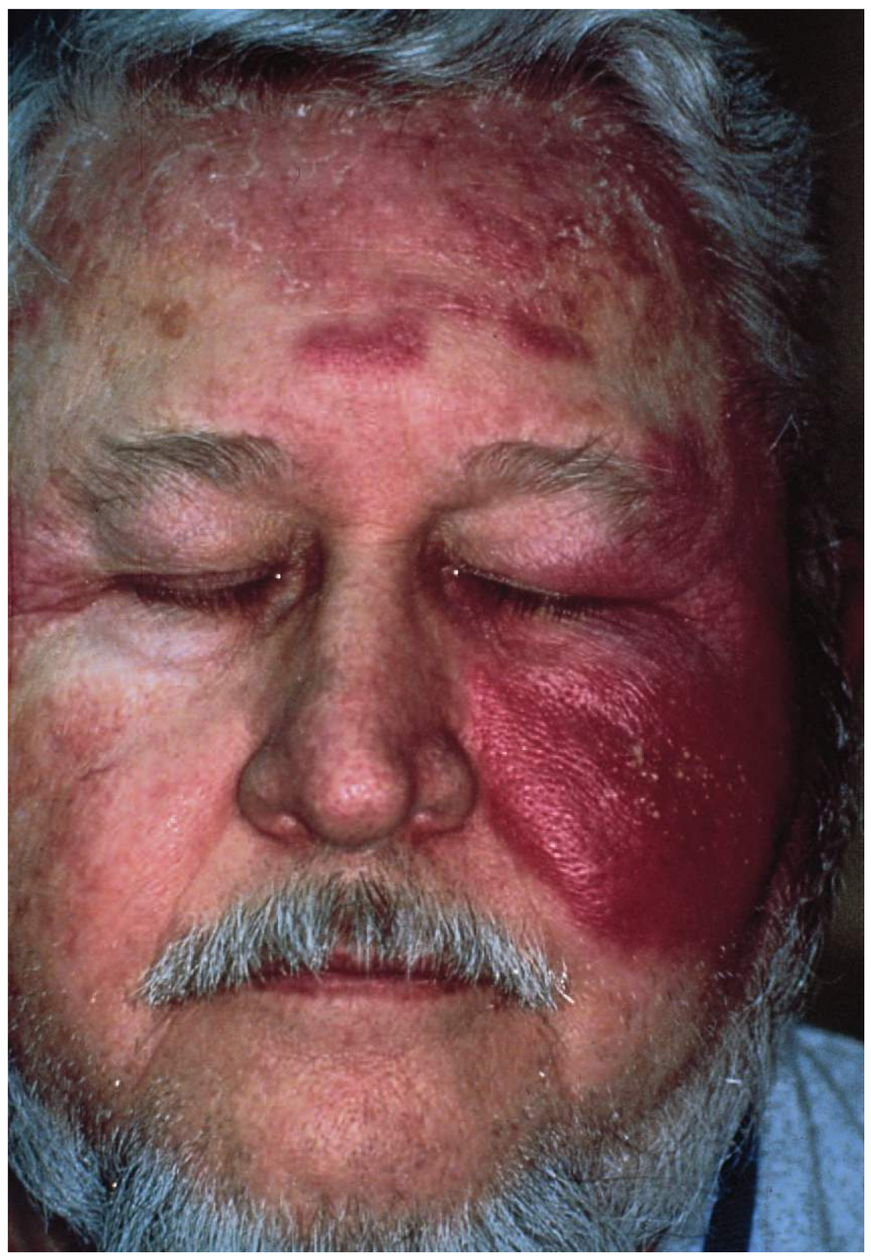


FIGURE 13.68 ■ **Erysipelas.** Note the well-demarcated, edematous, erythematous, shiny plaque. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)

## Clinical Summary

Infective endocarditis (IE) is characterized by infection of the endocardium (including the valves, mural endocardium, or septal defect) manifested by a number of cutaneous findings. Causes of IE are diverse and can be broadly categorized as (1) acute and subacute native valve IE, (2) early and late prosthetic valve IE, (3) IV drug abuse IE, and (4) iatrogenic IE. The latter encompasses IE associated with recent hospital admission or a healthcare procedure causing bacteremia or endocardial damage. Overall, Staphylococcus aureus is the most common causative organism but many other bacteria and fungi are implicated. Janeway lesions (septic emboli forming microabscesses) are nontender, small, erythematous (occasionally with central hemorrhage) macules on the palms or soles. Osler nodes (immune complex deposition resulting in small vessel vasculitis) consist of transient, tender, purplish nodules on the pulp of the fingers and toes. Subungual splinter hemorrhages are black, linear discolorations beneath the conjunctiva and nail plate. Murmurs, retinal hemorrhages, septic arthritis, and significant embolic episodes such as pulmonary embolism or cerebral vascular embolism may also be present.

## Management and Disposition

Empiric IV antibiotic therapy is the mainstay of treatment. Obtain blood cultures prior to antibiotic administration. Consult cardiology for emergent, echocardiographic assessment of the cardiac valves. Consult infectious disease for empiric antibiotic recommendations. Emergent surgical intervention may be required for valvular dysfunction or associated abscess.

## Pearls

1. Acute bacterial endocarditis presents with fever (over 90% of patients) and a toxic patient—this contrasts with a more insidious presentation of subacute bacterial endocarditis.
2. Previous episode of IE, congenital heart disease, IV drug abuse, prosthetic heart valves, recent medical procedures, and cardiac transplants with valvulopathies are risk factors.
3. Although splinter hemorrhages in the nail bed have long been associated with IE, the most common causes are trauma, psoriasis, and onychomycosis. Proximal nail bed splinter hemorrhages have a higher likelihood of systemic disease (including IE, drug reactions, vasculitis, antiphospholipid antibody syndrome, and trichinosis).

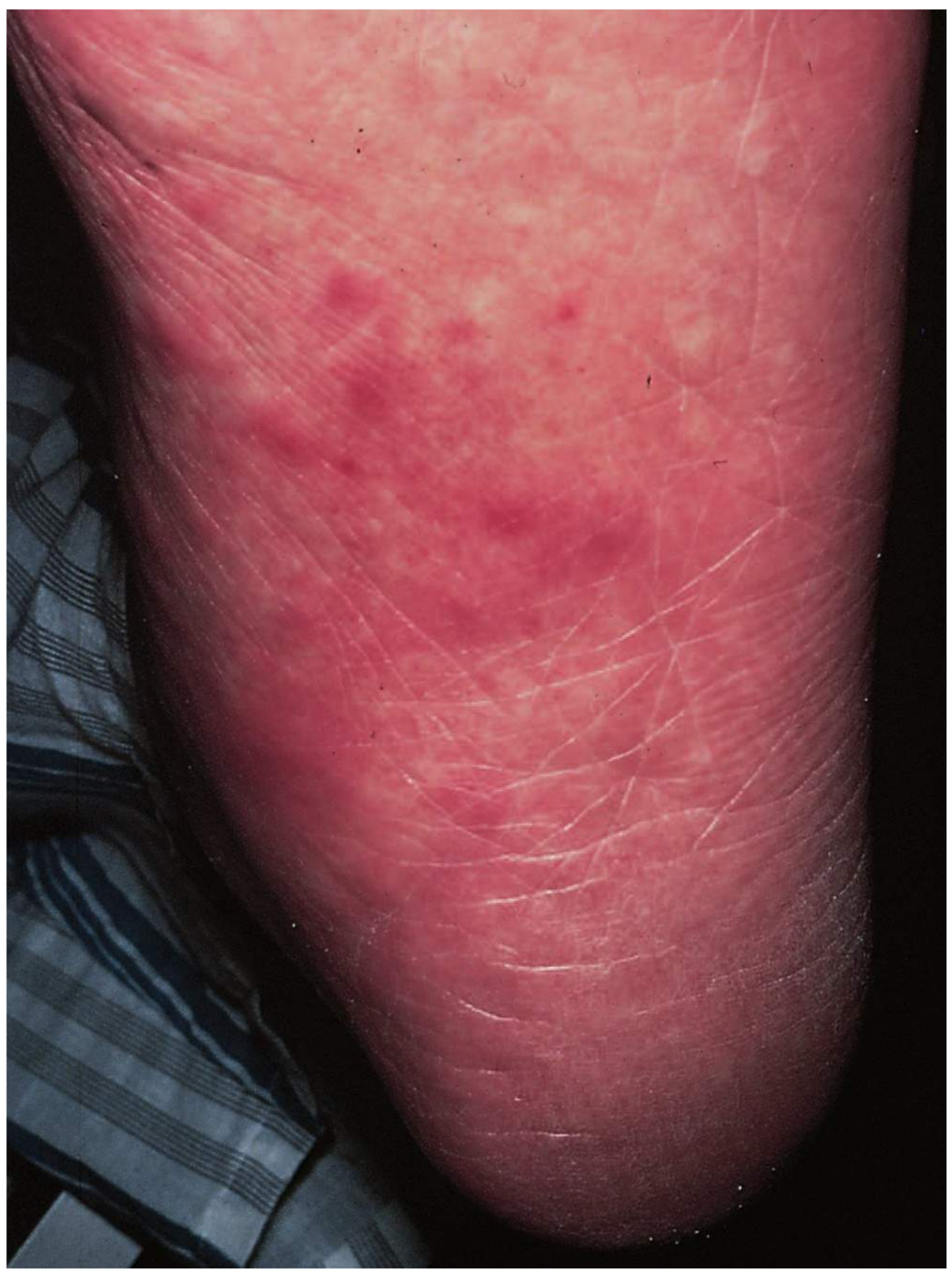


FIGURE 13.69 ■ **Janeway Lesions.** Peripheral embolization to the sole, resulting in a cluster of nontender, erythematous macules known as Janeway lesions. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)

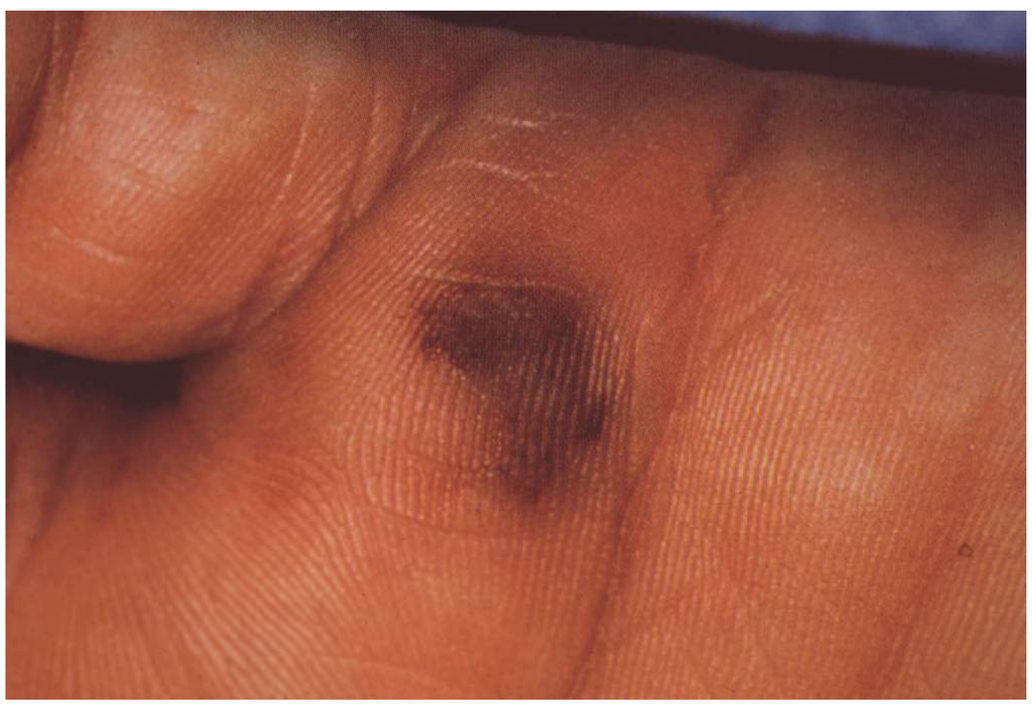


FIGURE 13.70 ■ **Janeway Lesion.** Embolization to the hand in a patient with infectious endocarditis. (Photo contributor: Alan B. Storrow, MD.)

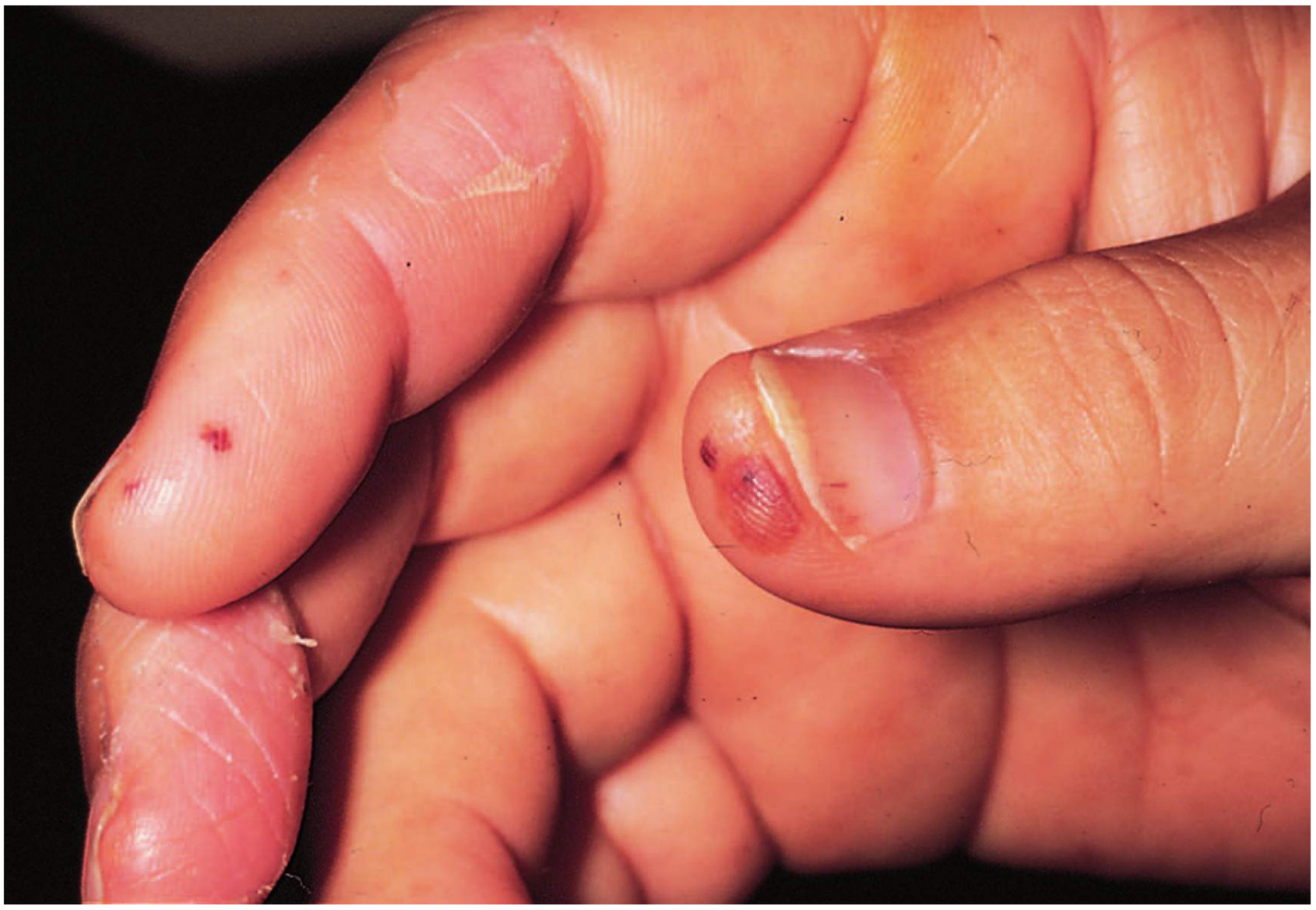


FIGURE 13.71 ■ **Osler Nodes.** Tender, subcutaneous, violaceous nodules in the pulp of the fingers known as Osler nodes. (Photo contributor: Armed Forces Institute of Pathology, Bethesda, MD.)

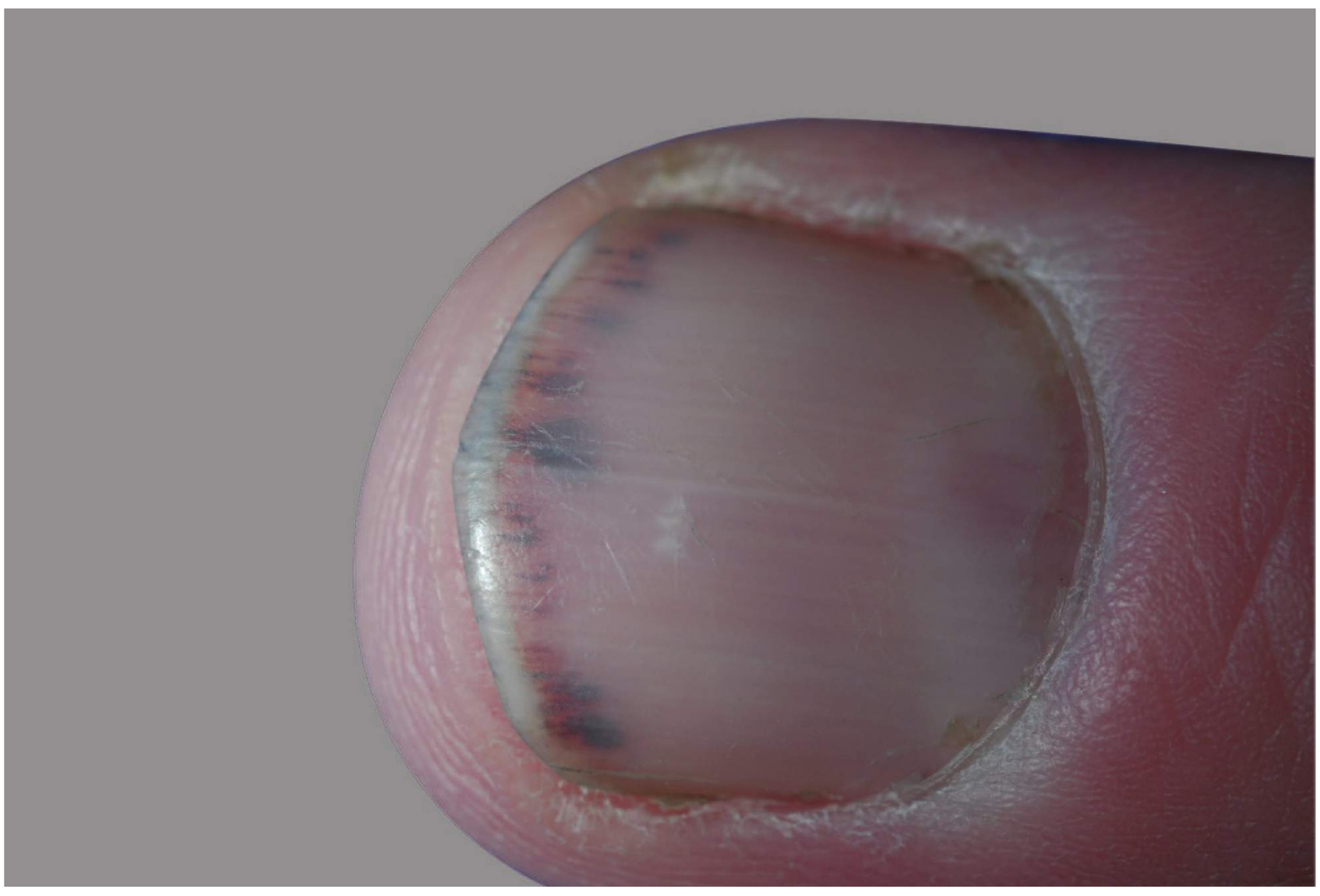


FIGURE 13.72 ■ **Splinter Hemorrhages.** Note the splinter hemorrhages along the proximal and distal aspect of the nail plate—emboli from subacute bacterial endocarditis. (Photo contributor: R. Jason Thurman, MD.)

## Clinical Summary

Borrelia burgdorferi is the tick-borne spirochete responsible for Lyme disease, and erythema chronicum migrans (ECM) is the pathognomonic rash. ECM typically presents 1 to 2 weeks after the bite. The initial prodromal symptoms of fever, myalgias, arthralgias, and headache are followed by a macule or papule progressing to a plaque at the bite site. This plaque expands (usually to 5 cm or larger) its red, raised border as it clears centrally, leading to an annular appearance ("bull's eye"). The plaque may burn and is rarely pruritic. Less frequently, secondary ECM-like lesions can appear due to multiple bites or spirochetemia. ECM is seen in 60% to 90% of patients and represents the early localized stage.

## Management and Disposition

The duration and choice of antibiotic depends on the clinical features (presence of ECM, early disseminated disease with mild or severe symptoms, cranial nerve palsies, heart block, meningitis, and radiculopathy). Doxycycline is the drug of choice for adults and children over 8 years of age. Pregnant or lactating females and children younger than 8 years of age should be treated with amoxicillin. Patients with minimal symptoms may be treated and followed up on an outpatient basis. Those patients with significant toxicity (severe disseminated rash, systemic symptoms, meningitis, radiculopathy, or third-degree heart block) require admission, supportive care, and IV antibiotics.

## Pearls

1. Over 50% of untreated ECM can progress to an asymmetric, episodic, oligoarticular arthritis weeks to months after the initial infection.
2. Facial nerve palsies are the most common neurologic manifestation of untreated Lyme disease.
3. Early serology may not demonstrate elevated anti-Borrelia antibodies and, if not followed up, one may miss the diagnosis.

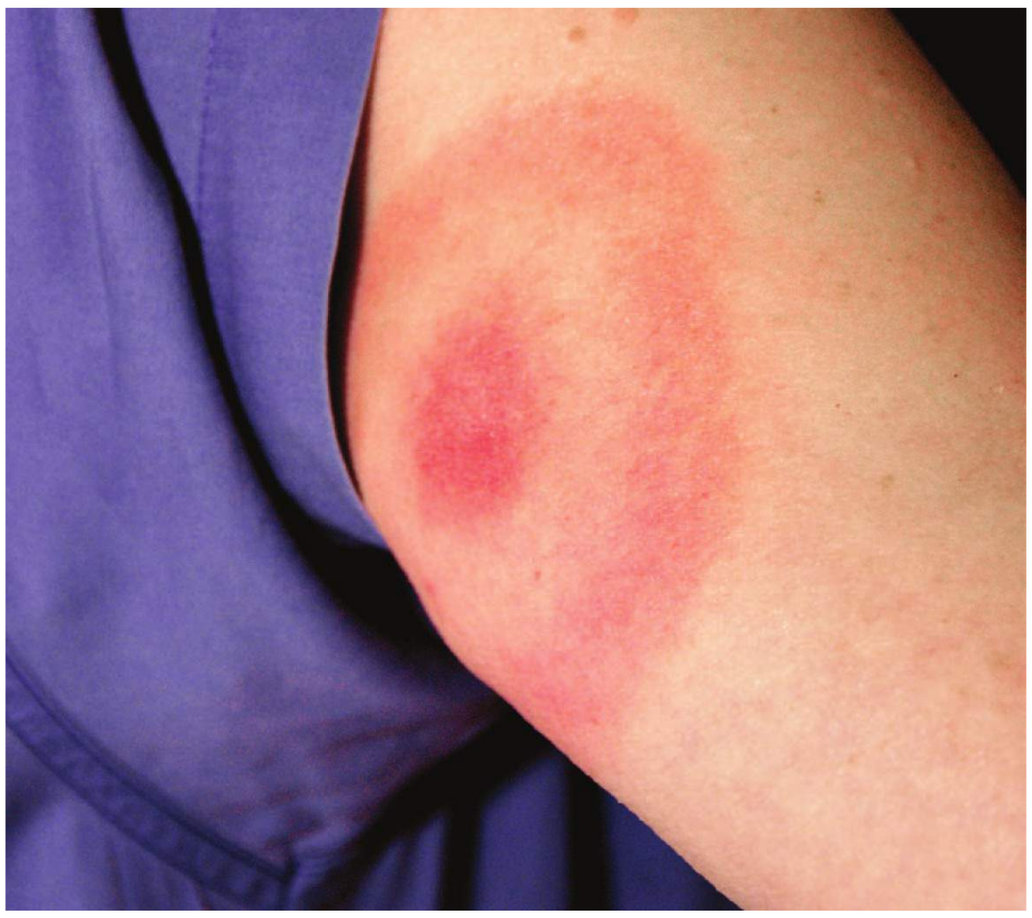


FIGURE 13.73 ■ **Erythema Chronicum.** The eruption of Lyme disease forms at the site of the tick bite. The initial papule forms into an expanding oval of erythema. There is central "bull's eye" clearing as the erythema progresses. (Photo contributor: James Gathany, Public Health Image Library, US Centers for Disease Control and Prevention.)

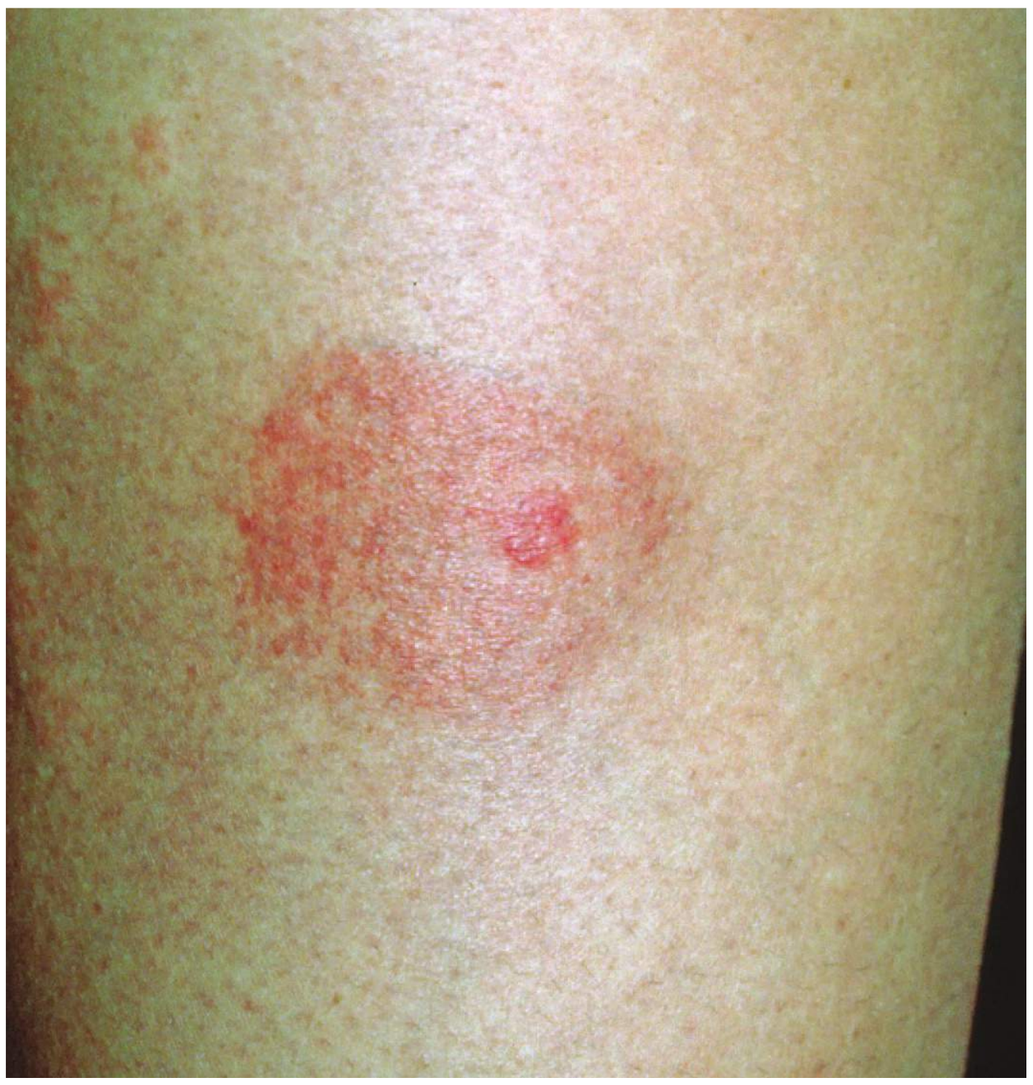


FIGURE 13.74 ■ **Erythema Chronicum.** An atypical erythematous patch of erythema chronicum—always have a high level of suspicion. (Photo contributor: David Effron, MD.)

# ROCKY MOUNTAIN SPOTTED FEVER

## Clinical Summary

Rickettsia rickettsia, the causative organism of Rocky Mountain spotted fever (RMSF), is transmitted by the bite of an infected tick. Fever, headache, rigors, abdominal pain, myalgias, and malaise occur 2 to 14 days after inoculation. Three to five days after the onset of symptoms, the rash begins with erythematous, blanching macules on the distal extremities (wrists and ankles). This is followed by centripetal spread to the trunk and to the palms and soles. The lesions evolve into papules and petechia. Without treatment, RMSF has a 25% mortality; delayed diagnosis and delayed antimicrobial treatment results in 3% to 4% mortality.

## Management and Disposition

Prompt initiation of doxycycline is recommended for adults and children. Consultation with an infectious disease specialist should be initiated to help guide treatment—this is especially critical for pregnant females. Mildly ill patients may be treated with oral antibiotics on an outpatient basis as long as close follow-up can be confirmed. More severely ill patients require admission.

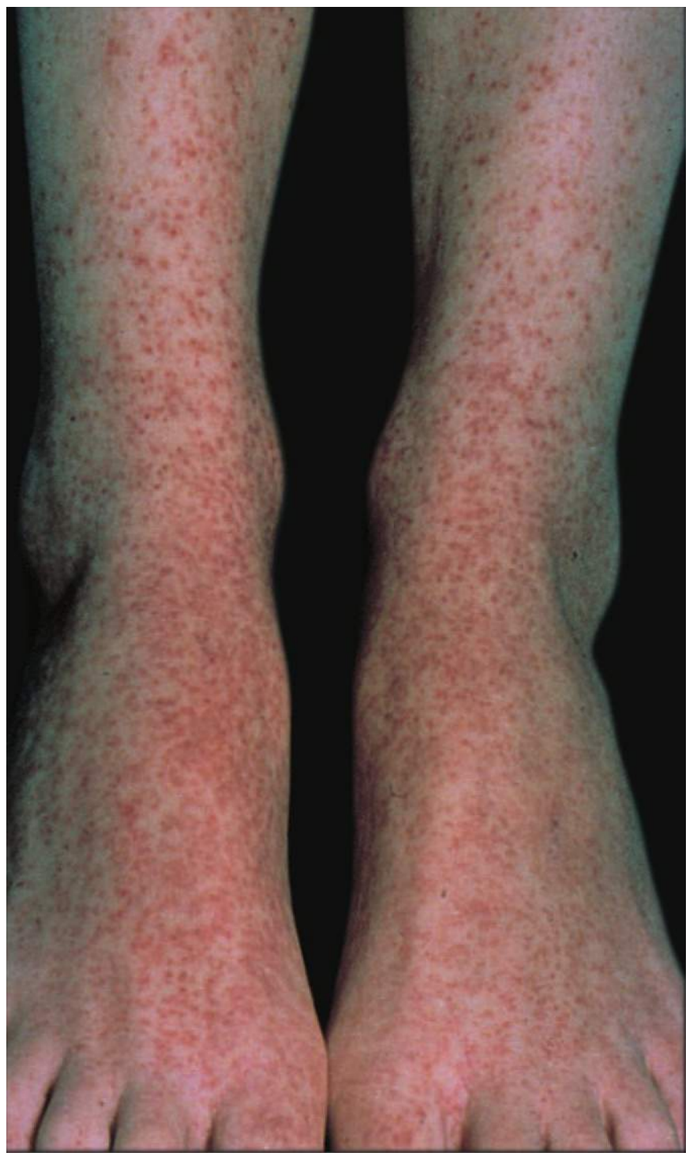


FIGURE 13.76 ■ **Rocky Mountain Spotted Fever.** Early erythematous and hemorrhagic macules and papules appeared initially on the ankles of an adolescent. (Used with permission from Wolff K, Johnson RA, Saavedra AP. Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology. 7th ed. McGraw-Hill; 2013: Fig. 25-49.)

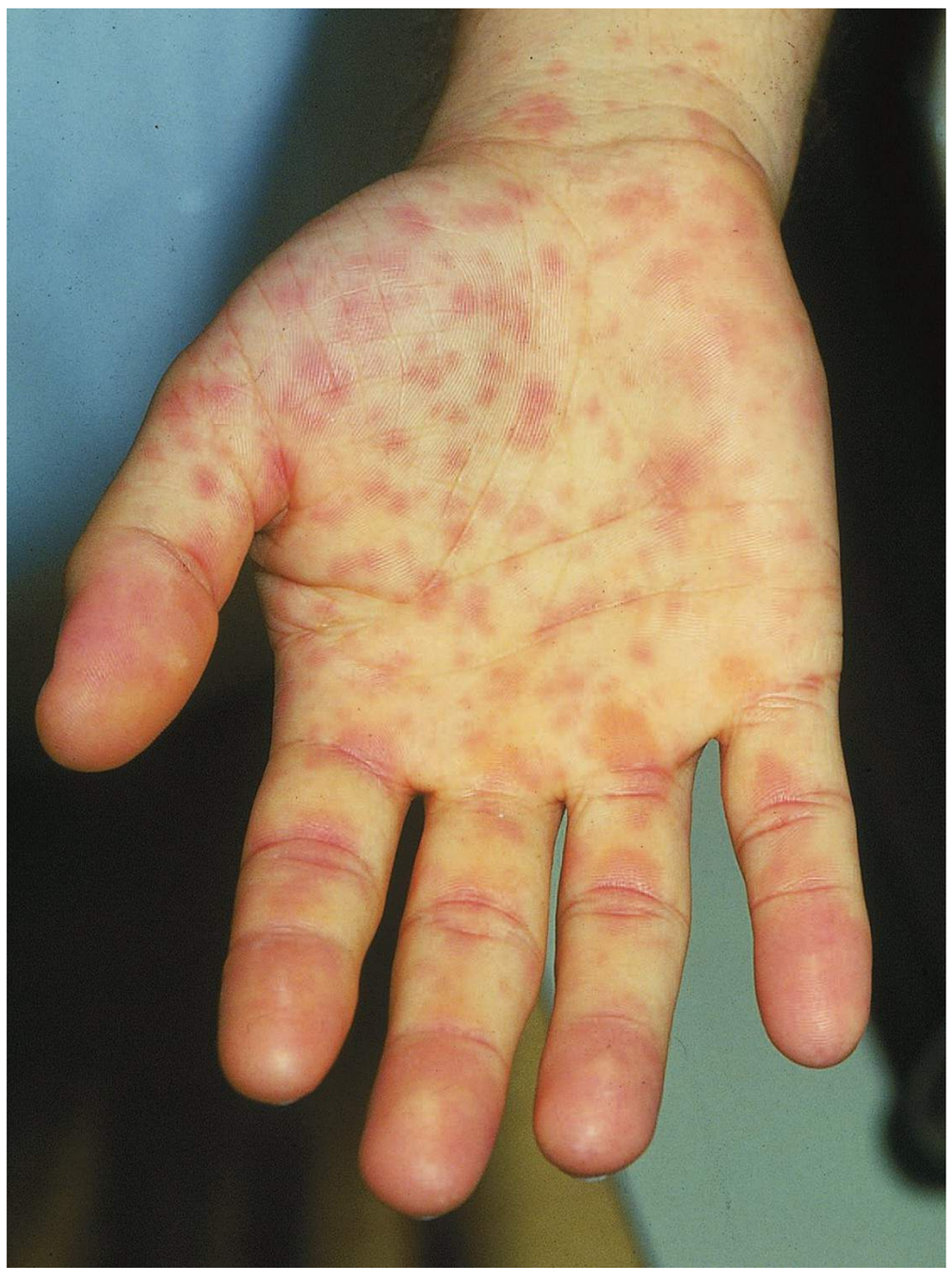


FIGURE 13.75 ■ **Rocky Mountain Spotted Fever.** These erythematous macular lesions will evolve into a petechial rash that will spread centrally. (Photo contributor: Daniel Noltkamper, MD.)

## Pearls

1. Typical extremity lesions in a severely ill patient should be treated as RMSF until proven otherwise. Treatment should not be delayed for laboratory confirmation.
2. Most cases occur between April and October, with the highest US incidence occurring in the Southeast and South-Central states (not Rocky Mountain states).
3. Forty percent of patients do not recall the inciting tick bite.
4. Up to 15% of cases present without any cutaneous manifestations. Patients with darker skin types may have less obvious cutaneous findings.
5. In pediatric patients less than 9 years old, the recommended course of doxycycline has a negligible effect on permanent tooth discoloration. Previously recommended chloramphenicol has a higher mortality rate than doxycycline.

# DISSEMINATED GONOCOCCUS

## Clinical Summary

Disseminated gonococcus (gonococcemia) is a systemic infection that presents in 1% to 3% of untreated mucosal (urethral, endocervical, pharyngeal, and rectal) gonorrhea infections. The hematogenous dissemination of Neisseria gonorrhea results in fever, arthralgias, and scattered pustules. The initial lesion is an erythematous macule that evolves into a papule and hemorrhagic pustule. Petechial or purpuric macules can be seen as well. These lesions are few in number, asymmetric, painful, and predominantly located on the distal extremities. The spectrum of disease varies from skin lesions alone to associated tenosynovitis, septic arthritis, endocarditis, and meningitis.

## Management and Disposition

Therapy consists of IV or intramuscular ceftriaxone until symptoms either improve or resolve, followed by an additional 7 days of orally administered cefixime. Hospitalization is recommended for initial treatment. Dermatology consultation may help establish the diagnosis.

## Pearls

1. Pregnant or menstruating females have a higher risk of disseminated gonococcemia.
2. Disseminated gonococcemia is the most common cause of septic arthritis in young, sexually active adults.

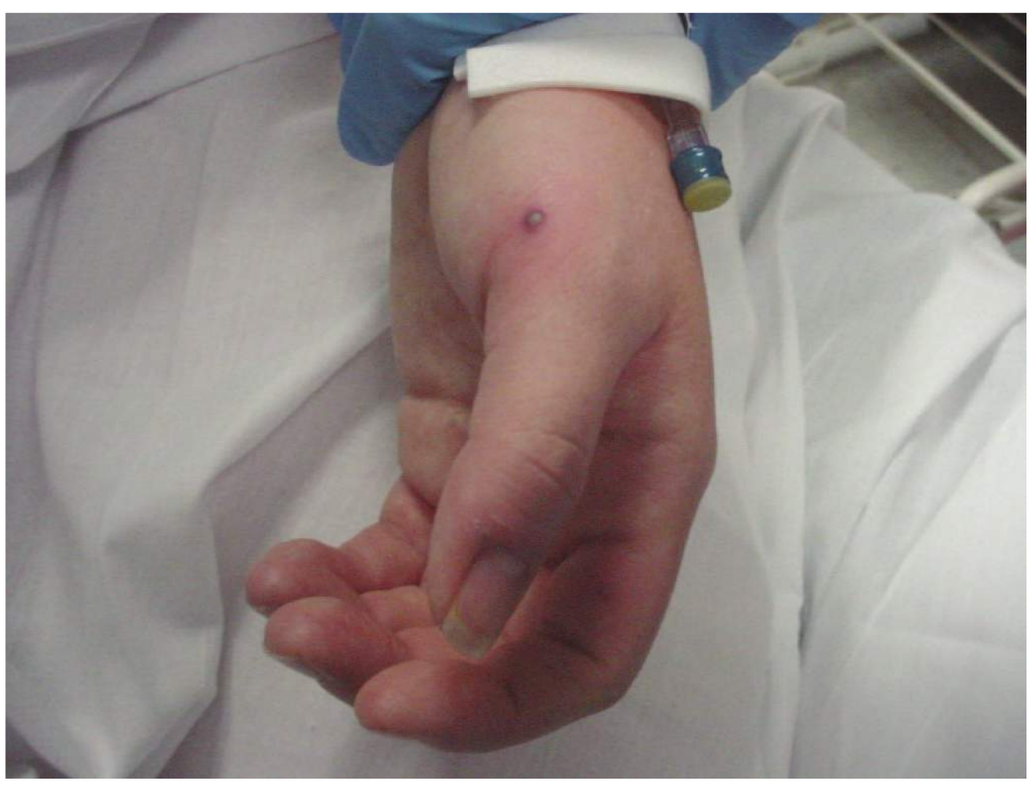


FIGURE 13.77 ■ **Disseminated Gonococcus.** Vesiculopustule on the hand of a patient with disseminated gonococcus. (Photo contributor: Stephan E. Russ, MD.)

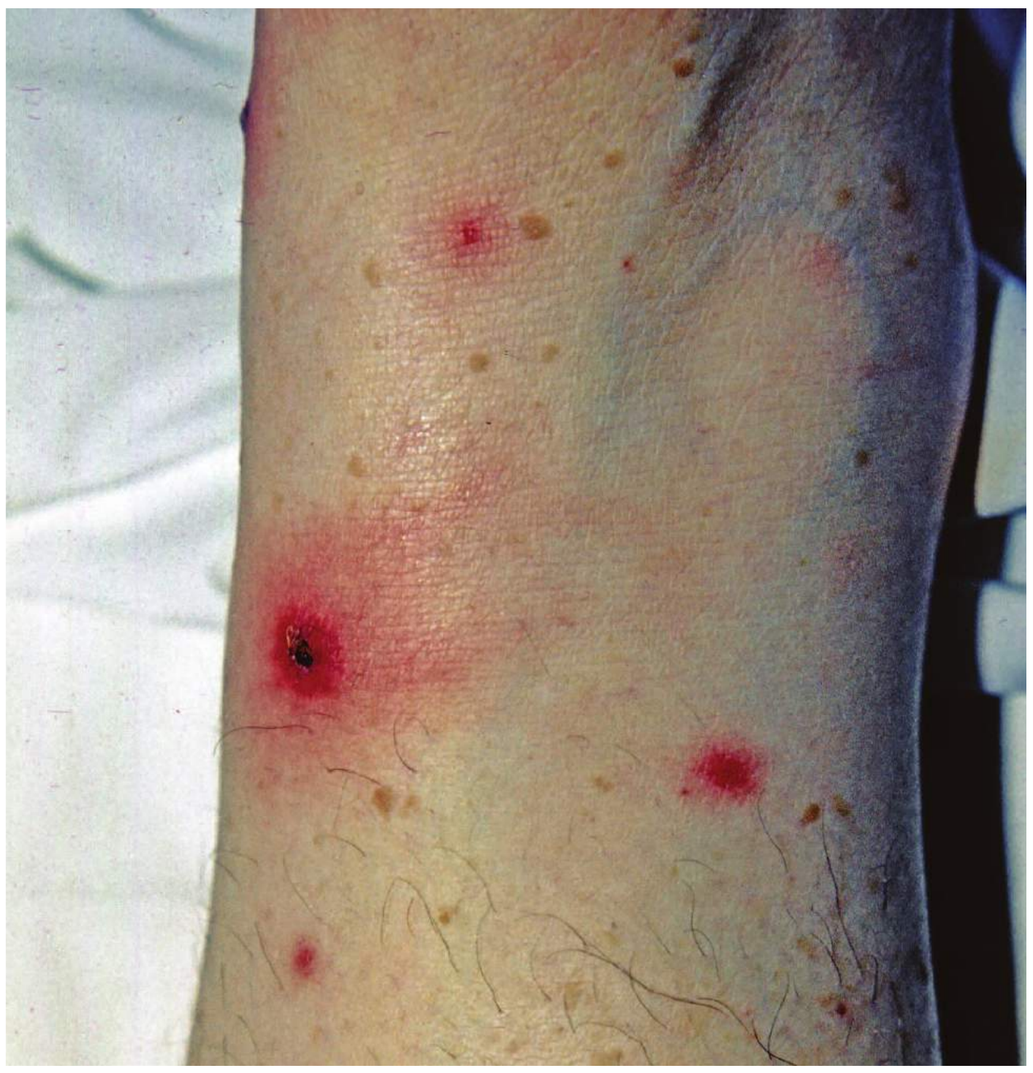


FIGURE 13.78 ■ **Disseminated Gonococcus.** Erythematous macules of disseminated gonococcus—these will evolve into hemorrhagic pustules. (Photo contributor: David Effron, MD.)

# HOT TUB FOLLICULITIS/PSEUDOMONAS HOT-FOOT SYNDROME

## Clinical Summary

Hot tub folliculitis is a pruritic, pustular eruption confined to the hair follicle and is secondary to a cutaneous infection with Pseudomonas aeruginosa. Patients have a history of hot tub, whirlpool, or swimming pool exposure within 24 to 72 hours of the eruption. The lesions have a predilection for areas covered with bathing suits. Headache, sore throat, earache, and fever may accompany the pustules (but do not necessarily indicate systemic disease).

Pseudomonas hot-foot syndrome is related to hot tub folliculitis; patients report wading in a swimming pool (later discovered to have elevated P aeruginosa concentrations). The weight-bearing aspects of the soles have multiple 1 to 2 cm erythematous nodules. Unlike hot tub folliculitis, these lesions are exquisitely painful and not associated with other symptoms. Like hot tub folliculitis, the lesions resolve spontaneously without treatment.

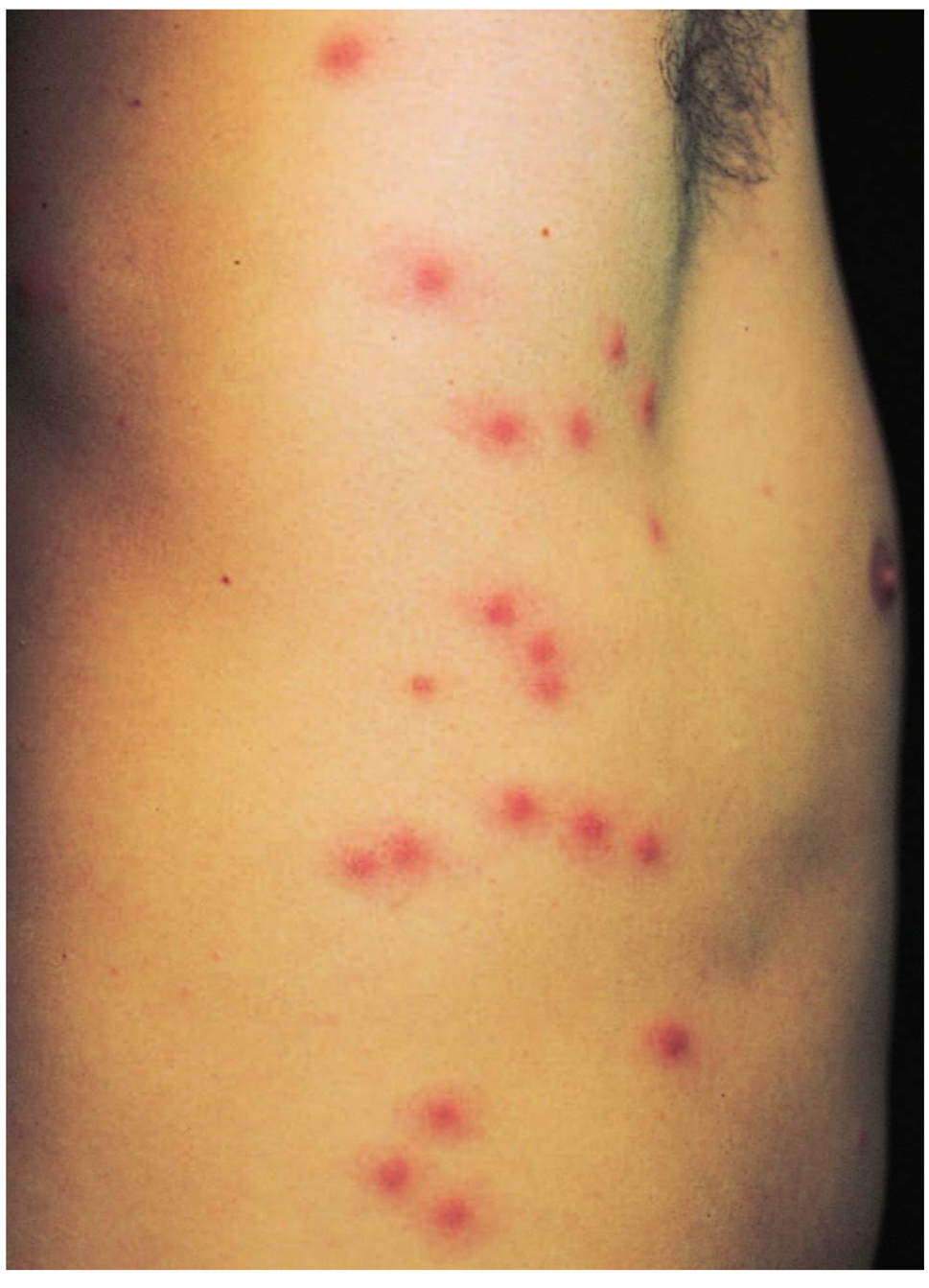


FIGURE 13.79 ■ **Hot Tub Folliculitis.** Note the pustules localized to the hair follicles of the trunk and proximal extremity. (Photo contributor: Jeffrey S. Gibson, MD.)

## Management and Disposition

The folliculitis usually involutes in 1 to 2 weeks without treatment; however, oral ciprofloxacin or topical gentamicin may decrease recovery time. In addition, the implicated source of exposure must be decontaminated to avoid reexposure. Patients with immunosuppression, widespread lesions, or concern for systemic involvement should be treated with oral ciprofloxacin.

## Pearls

1. Ensure patient is aware of the cause and insist on decontamination of the water source.
2. Hot tub folliculitis may also result from contact with depilatory agents.

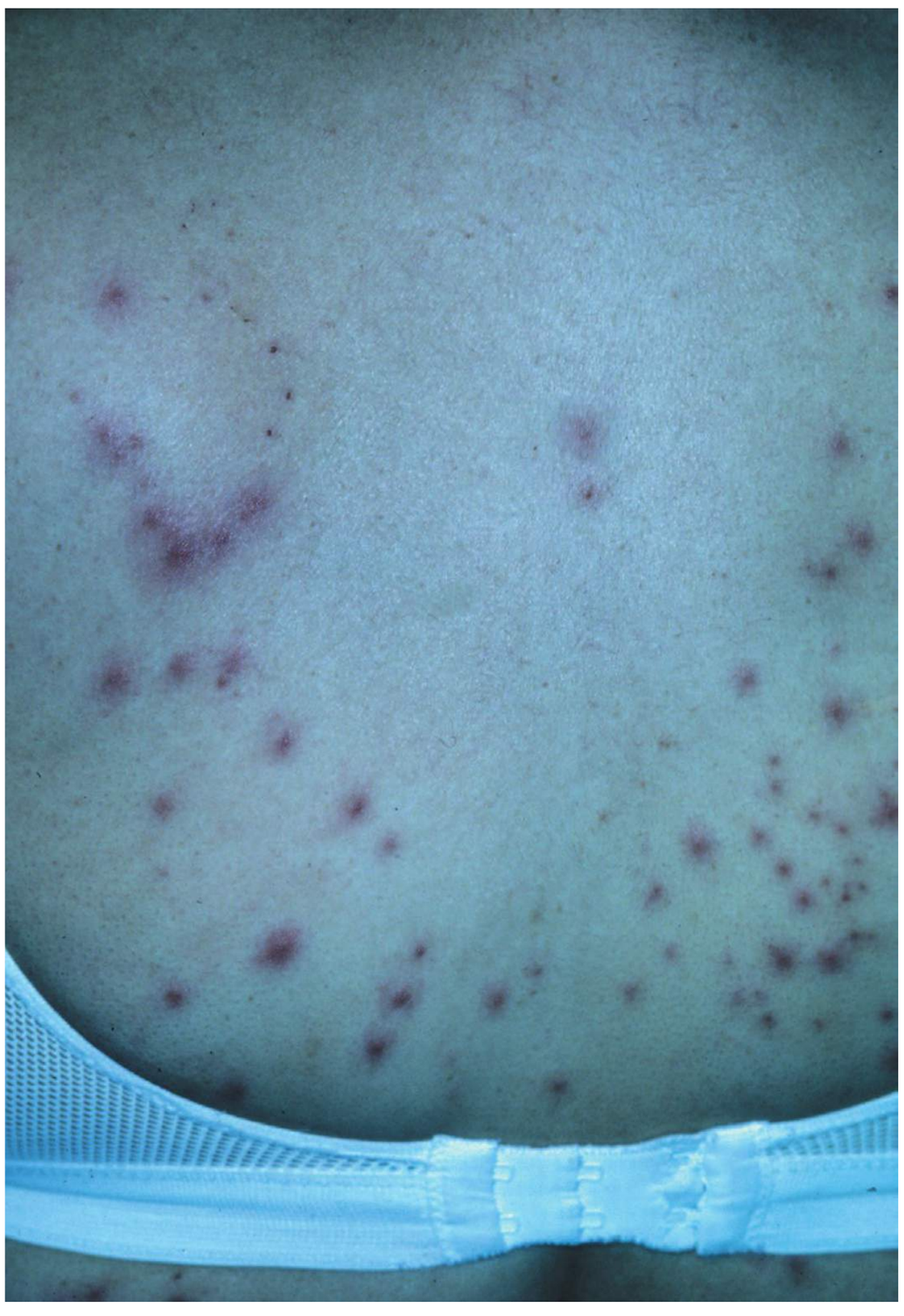


FIGURE 13.80 ■ **Hot Tub Folliculitis.** Folliculitis from Pseudomonas aeruginosa in a hot tub. (Photo contributor: Richard P. Usatine, MD. Used with permission. From Usatine RP, Smith MA, Mayeaux EJ, Chumley HS. The Color Atlas of Family Medicine. 2nd ed. McGraw-Hill; 2013: Fig. 117-1.)

# ECTHYMA GANGRENOSUM

## Clinical Summary

Ecthyma gangrenosum is a cutaneous manifestation of P aeruginosa septicemia occurring in an immunocompromised or neutropenic patient. Patients have a toxic appearance with fever, abnormal vital signs, and altered consciousness. The initially erythematous macules develop bullae or pustules surrounded by violaceous halos. Bullae become hemorrhagic and rupture (pustules breakdown), becoming necrotic ulcers with central black or gray eschars. The time course from macule to eschar can occur within 12 to 24 hours. The lesions primarily affect the anogenital areas (over 50%) but may appear anywhere on the body. Ecthyma gangrenosum is seen in up to 13% of patients with P aeruginosa septicemia and rarely in nonbacteremic patients.

## Management and Disposition

Ecthyma gangrenosum is associated with life-threatening P aeruginosa sepsis and a high mortality. Appropriate supportive care measures and rapid initiation of antipseudomonal antibiotics are required. Dermatology consultation for diagnosis confirmation and infectious disease consultation for antibiotic guidance are helpful. Patients with oncologists should consult the oncology service and follow oncology protocols.

## Pearls

1. Patients with ecthyma gangrenosum are severely immunosuppressed and many other bacteria and fungi can cause similar lesions. Discuss additional antibiotics and antifungal medications with the oncology and infectious disease services.
2. Without examining the entire patient, these lesions can be completely missed.
3. Multiple lesions, delayed diagnosis, and delayed institution of appropriate antibiotics portend a poor prognosis.

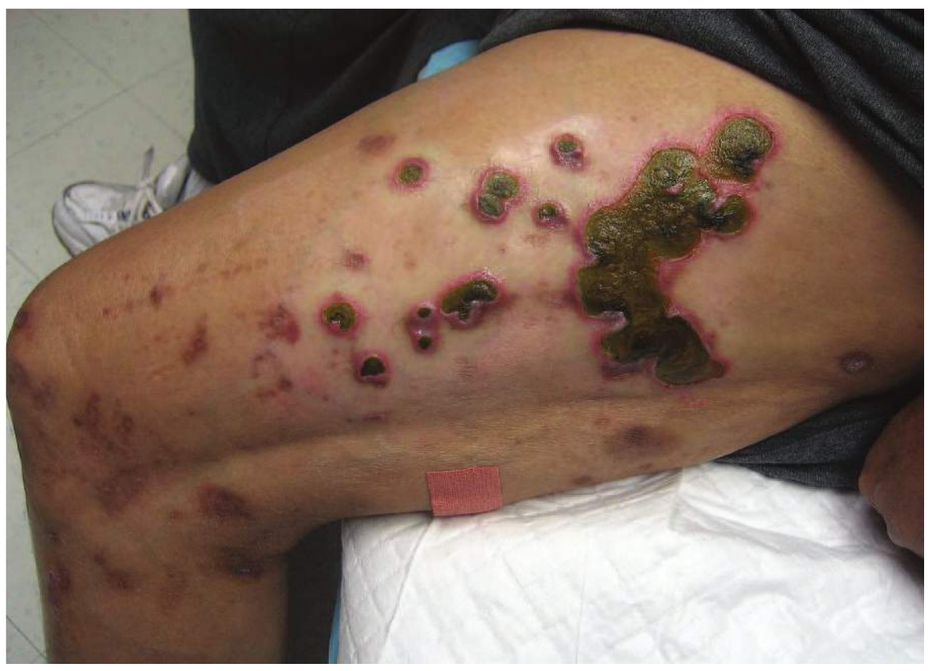


FIGURE 13.82 ■ Pseudomonas aeruginosa **Infected Ulcerations.** Note the green colored crusting. (Photo contributor: J. Matthew Hardin, MD.)

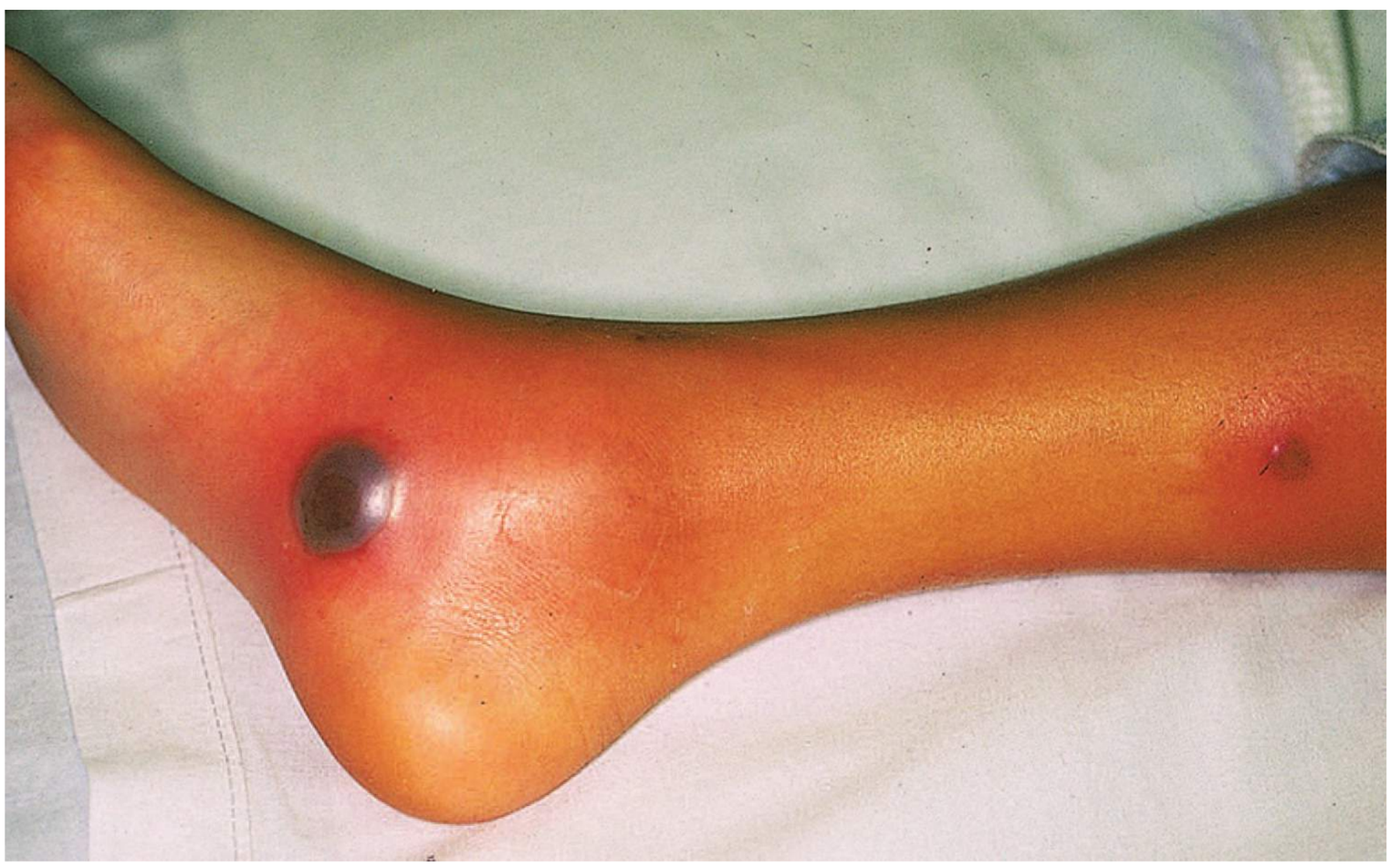


FIGURE 13.81 ■ **Ecthyma Gangrenosum.** A typical hemorrhagic bulla of ecthyma gangrenosum secondary to pseudomonal sepsis. (Photo contributor: James Mensching, DO.)

## Clinical Summary

Primary or recurrent HSV infection presents as grouped vesicles on an erythematous base. Recurrent HSV is typically less severe than the primary infection. A prodrome is frequently noted with fever, malaise, anorexia, and regional lymphadenopathy. The vesicles progress to pustules and crusted erosions. They heal over 2 to 3 weeks. Mucocutaneous involvement of the mouth and lips are the most common sites. Herpetic whitlow is a painful HSV infection of a distal finger seen primarily in children and adolescents. Herpes gladiatorum spreads via direct skin-to-skin contact in sports such as rugby and wrestling.

## Management and Disposition

Oral antivirals in addition to analgesics and antipyretics are useful. To be most effective, antivirals should be started within 72 hours of the eruption or during the typical prodrome. Immunocompromised patients may require admission and IV antivirals. Dermatology consultation is recommended for complicated presentations.

## Pearls

1. Wear protective gloves; herpetic infections are an occupational hazard in the medical and dental professions.
2. Wrestlers (or any skin-to-skin contact sport) with vesicles and ulcers may not participate in organized sports until completely healed.
3. Recurrent, same-site HSV infections on the fingers should alert the clinician to consider herpetic whitlow. These infections are often misdiagnosed as cellulitis, blistering dactylitis, and paronychia.

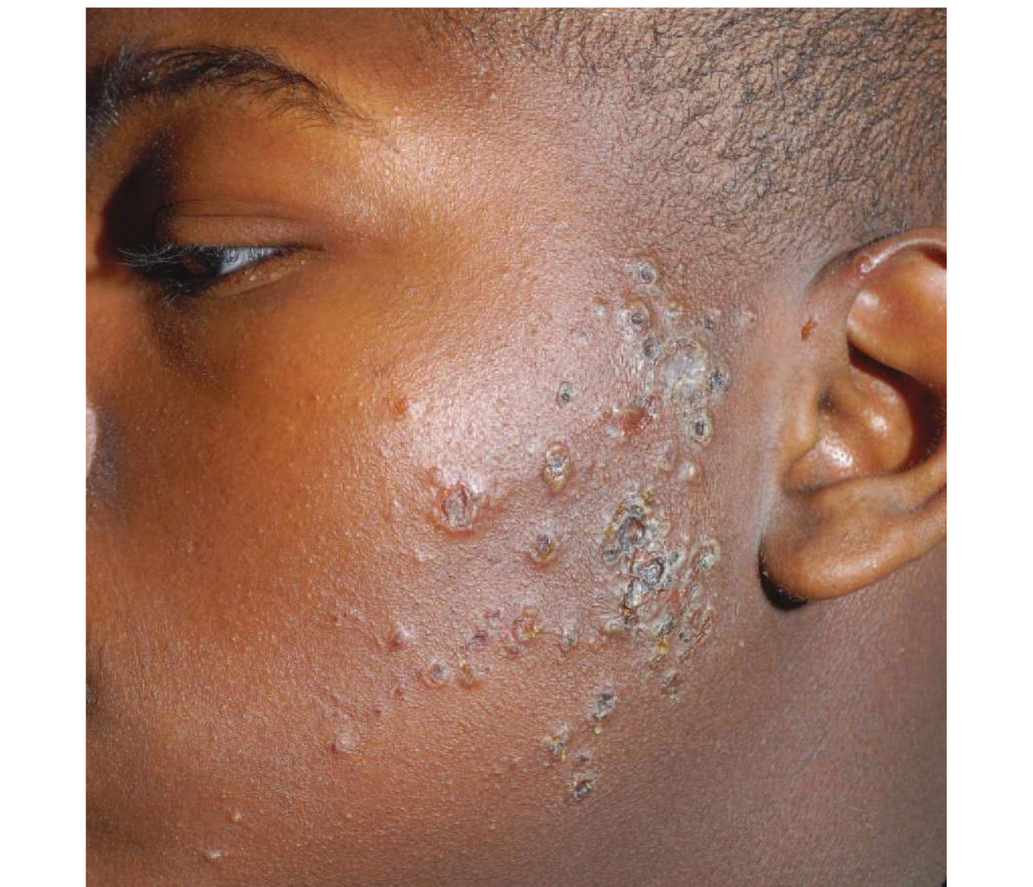


FIGURE 13.84 ■ **Herpetic Whitlow.** Vesicles on an erythematous base. (Photo contributor: Lawrence B. Stack, MD.)

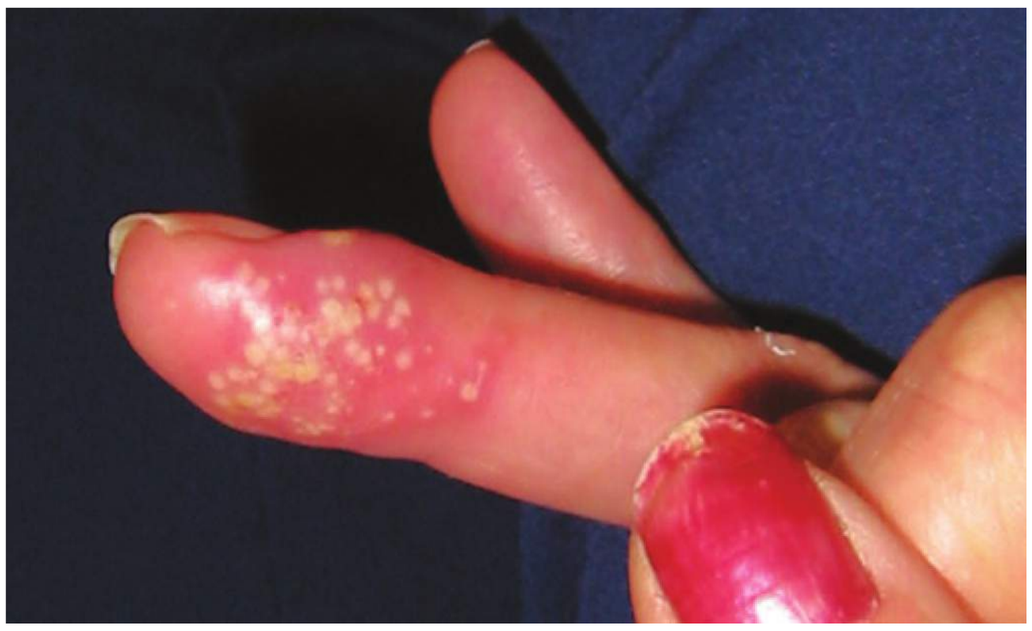


FIGURE 13.83 ■ **Herpetic Whitlow.** Painful, grouped, confluent vesicles and an erythematous base on the distal finger. (Photo contributor: Selim Suner, MD, MS.)



FIGURE 13.85 ■ **Herpes Gladiatorum.** Primary herpes gladiatorum in a wrestler. Note the varied stages of lesions, from vesicular to dried crusts. (Photo contributor: Lawrence B. Stack, MD.)

## Clinical Summary

Herpes zoster is a dermatomal, unilateral reactivation of the varicella zoster virus. All ages, including infants, can be affected. The eruption may occur anywhere on the body but most commonly on the face and trunk. Pruritus, pain, tenderness, and dysesthesias may present 4 to 5 days prior to an eruption composed of umbilicated, grouped vesicles on an erythematous, edematous base. The vesicles may become purulent or hemorrhagic. Occasionally, nerve involvement may occur without cutaneous involvement. Rare presentations involve multiple dermatomes or cross midline. Ophthalmic zoster (see related item) involves the nasociliary branch of the fifth cranial nerve and presents with vesicles on the nose and cornea (Hutchinson sign). Ramsay Hunt syndrome (see related item) is a herpes zoster infection of the geniculate ganglion with tinnitus, decreased hearing, facial palsy, and vesicles on the tympanic membrane, pinna, and ear canal.

## Management and Disposition

Uncomplicated cases of herpes zoster can be managed with supportive care, especially pain control. However, acyclovir, famciclovir, or valacyclovir hastens the healing, decreases the pain, and decreases postherpetic neuralgia incidence and symptoms, if started within 72 hours of vesicle appearance (still helpful even after 7 days). Gabapentin decreases postherpetic neuralgia pain.

Admission to the hospital for IV acyclovir is usually reserved for complicated cases involving multiple dermatomal distributions, involvement of the ophthalmic branch of the trigeminal nerve, disseminated disease, or immunocompromised patients. Herpes zoster keratitis requires emergent ophthalmologic consultation to avoid any potential scarring or vision loss.

## Pearls

1. The nonimmune or immunocompromised should avoid lesional contact from the prodrome until reepithelialization, since the crusts can contain the varicella zoster virus.
2. Herpes zoster during pregnancy confers no risk to the mother or unborn baby (as opposed to primary varicella virus infection).
3. Postherpetic neuralgia affects 10% to 20% of patients and is more common with advancing age, family history, and immunosuppression.
4. Immunocompromised patients can have unusual presentations with diffuse cutaneous and visceral involvement.

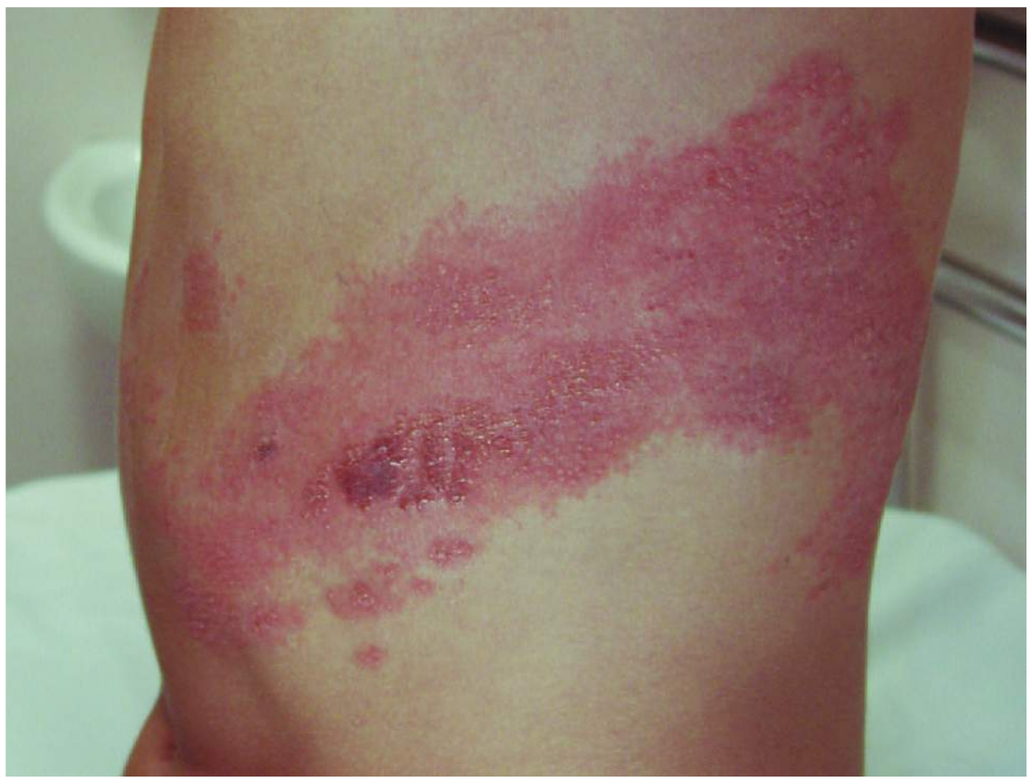


FIGURE 13.86 ■ **Herpes Zoster.** Dermatomal crusted erosions within an erythematous patch. (Photo contributor: Selim Suner, MD, MS.)

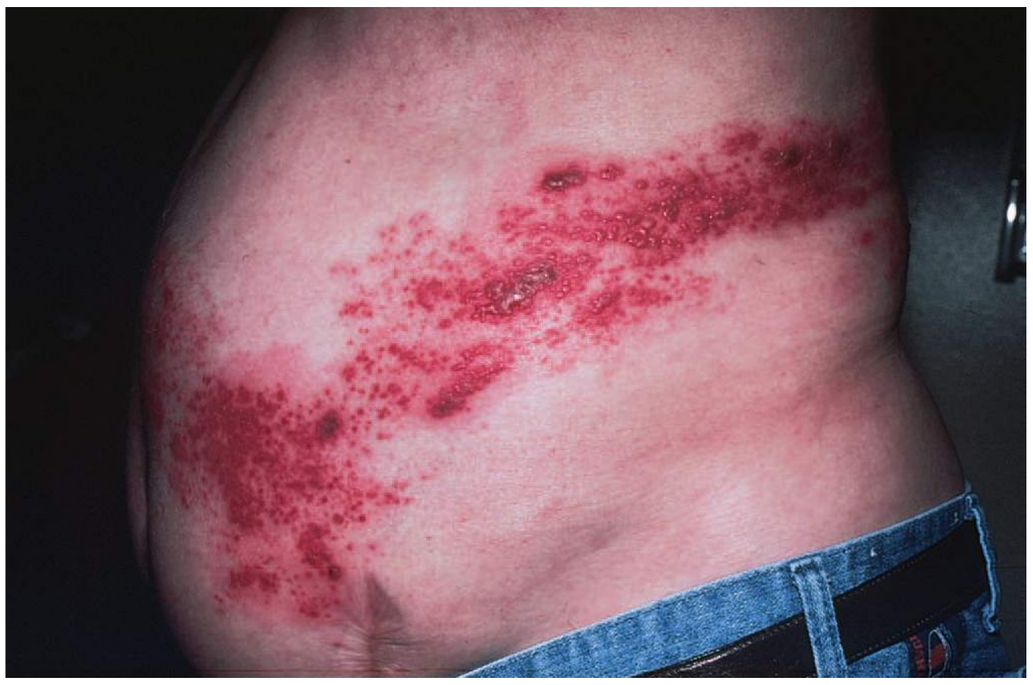


FIGURE 13.87 ■ **Herpes Zoster.** Dermatomal distribution of vesicles and crusted erosions. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)

# DERMATOPHYTE INFECTIONS

## Clinical Summary

Tinea corporis includes all dermatophyte infections excluding the scalp, face, hands, feet, and groin. The dermatophytosis is pruritic and consists of a well-circumscribed scaly plaque with a slightly elevated border and central clearing. This annular configuration is most commonly found on the trunk and neck. Skin scrapings examined with a KOH preparation demonstrate hyphae.

Tinea faciale (dermatophyte infection of the facial skin) commonly appears as a well-circumscribed scaling and erythematous patch. Tinea manuum (hands) presents with long-term scaling of the palms. Tinea cruris, or "jock itch," is a pruritic dermatophytosis of the intertriginous areas, usually, but not always, sparing the penis and scrotum. The scaly, erythematous plaque spreads peripherally with well-defined borders.

Tinea pedis, or "athlete's foot," consists of erythema and scaling of the sole and interdigital spaces, frequently with maceration, vesiculation, and fissure formation. The toenails may also be affected (tinea unguium).

Tinea capitis (scalp) presents as a pruritic, erythematous, scaly plaque. This may develop into a delayed-type hypersensitivity reaction, where the initial erythematous, scaly plaque becomes boggy with inflamed, purulent nodules and plaques (kerion). The hair follicle is frequently destroyed by the inflammatory process in a kerion, leading to a scarring alopecia.

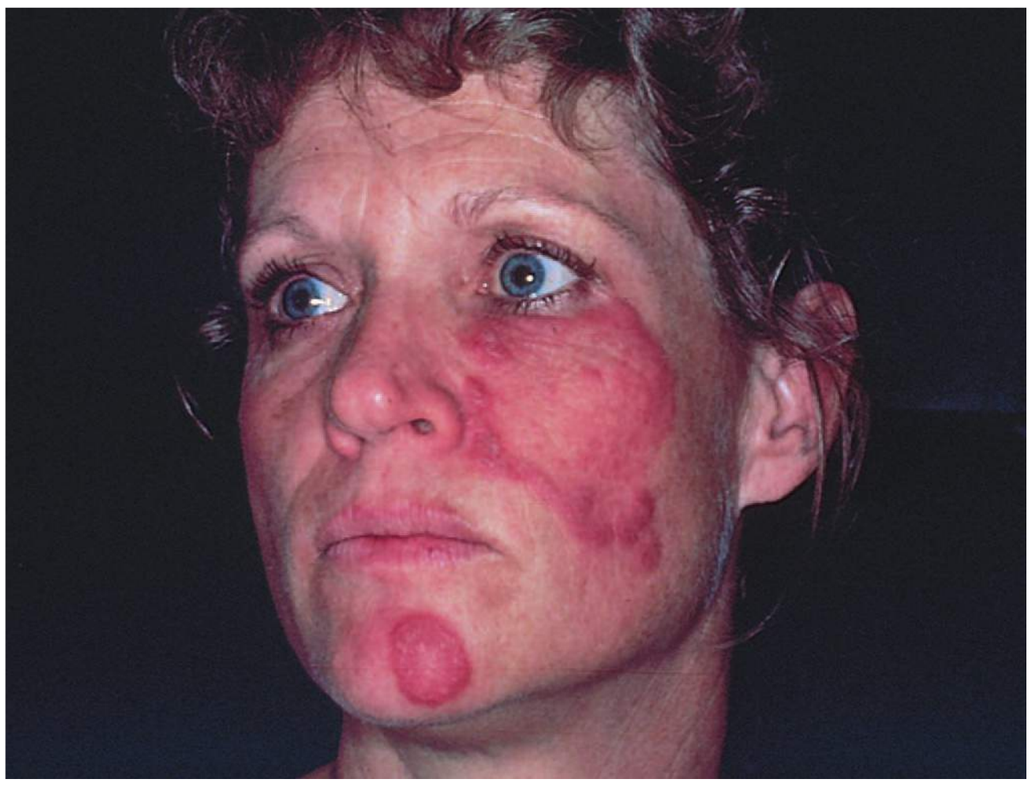


FIGURE 13.89 ■ **Tinea Faciale.** Note the sharply marginated, annular plaques with central clearing. KOH preparation is positive. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)

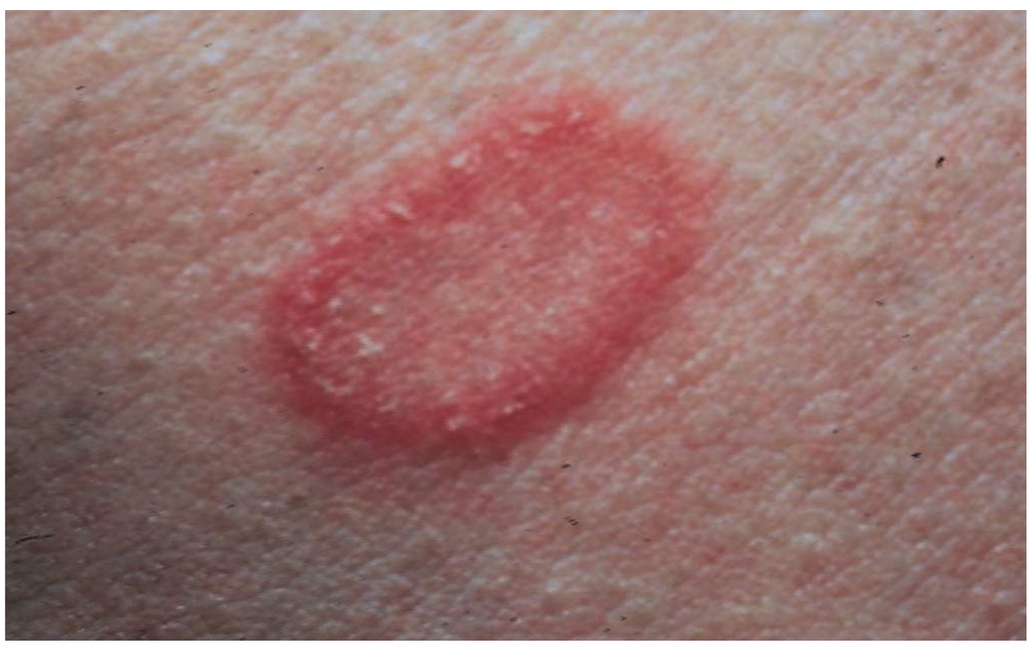


FIGURE 13.88 ■ **Tinea Corporis, Ringworm.** A well-defined, annular, pruritic plaque with a raised, scaly border and central clearing. KOH preparation is positive. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)

## Management and Disposition

Systemic antifungals are required to treat tinea capitis and tinea unguium infections. Due to the long-term treatment requirement and associated side effects, referral to a dermatologist is recommended. Topical antifungal medications are used for tinea corporis, tinea faciale, tinea cruris, and tinea pedis infections. Rarely, systemic antifungals may be required.

## Pearls

1. The scale is usually located at the leading edge of erythema and provides the best yield for positive scrapings seen on KOH examination (see related item for slide preparation).
2. Macerated areas may become secondarily infected by bacteria. Discuss the importance of good wound care.
3. When using topical antifungals, it is important to treat for one to two weeks beyond the point of clinical cure to ensure success.

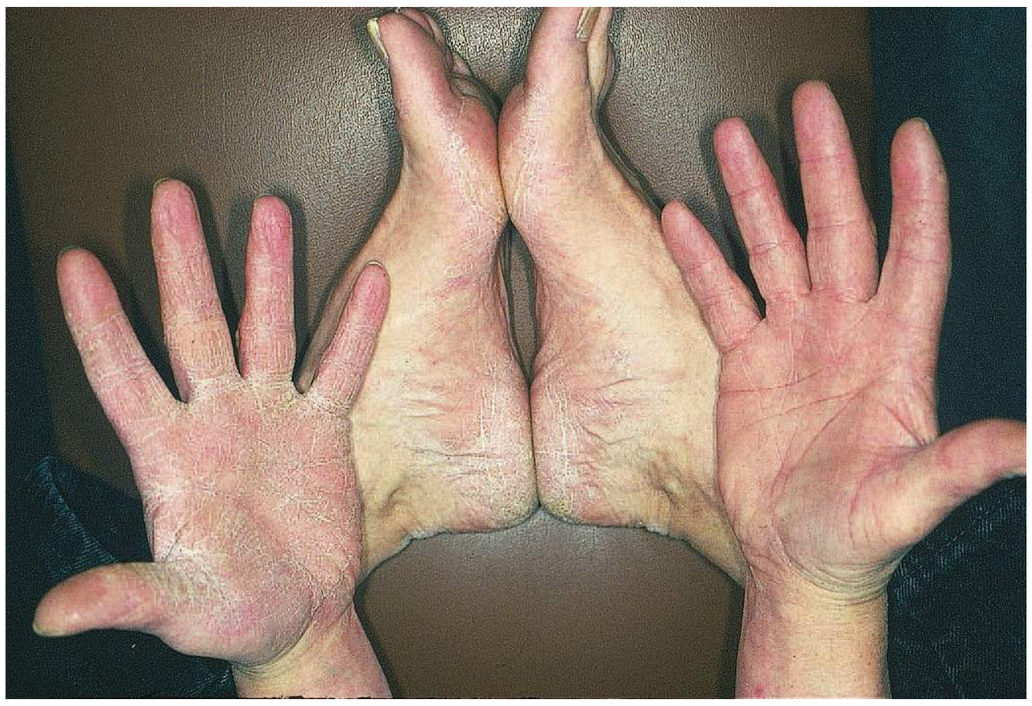


FIGURE 13.90 ■ **Tinea Manuum and Tinea Pedis.** The involvement of both the hands and feet are common. (Photo contributor: James J. Nordlund, MD.)

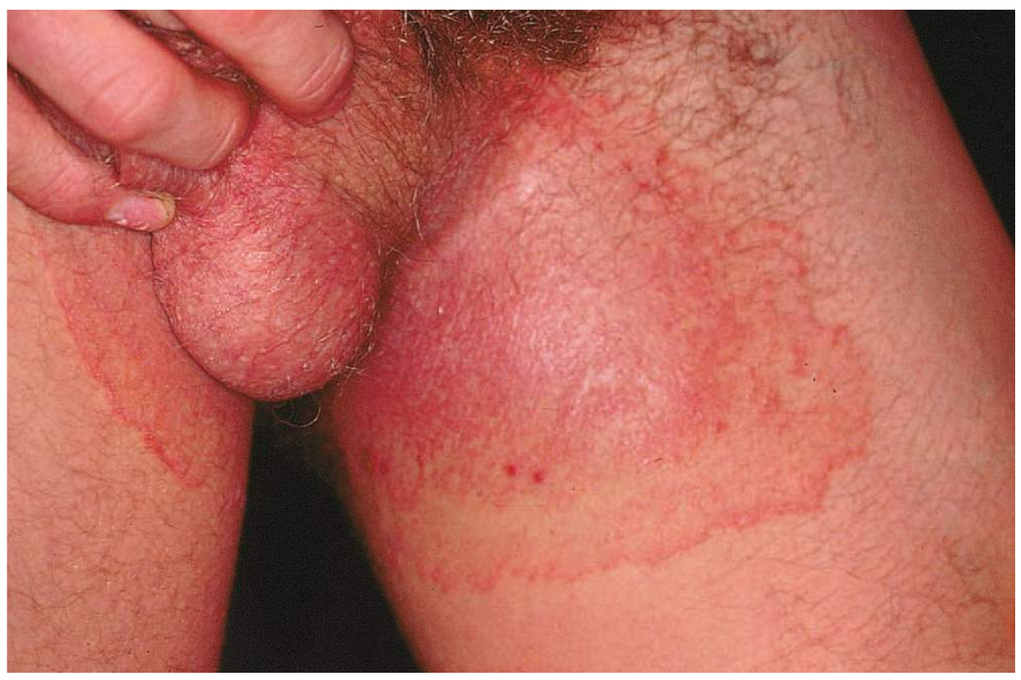


FIGURE 13.91 ■ **Tinea Cruris.** Erythematous plaque with accentuated and well-defined border. The scale may not be appreciated in this anatomic site. (Photo contributor: James J. Nordlund, MD.)

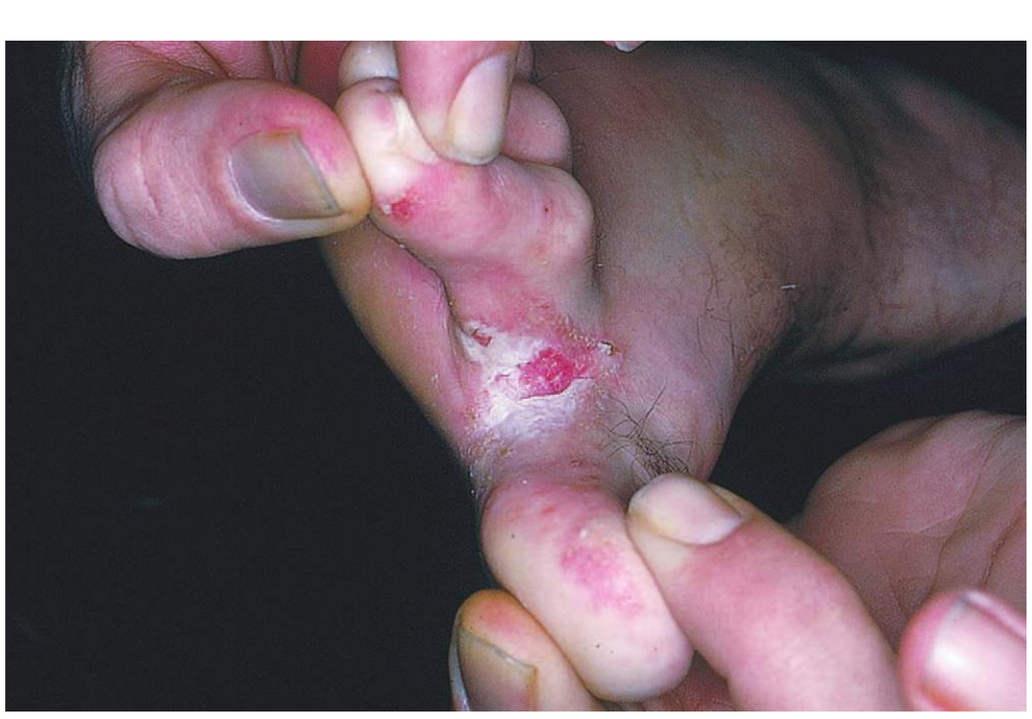


FIGURE 13.92 ■ **Tinea Pedis.** The interdigital spaces are characteristically involved. (Photo contributor: James J. Nordlund, MD.)

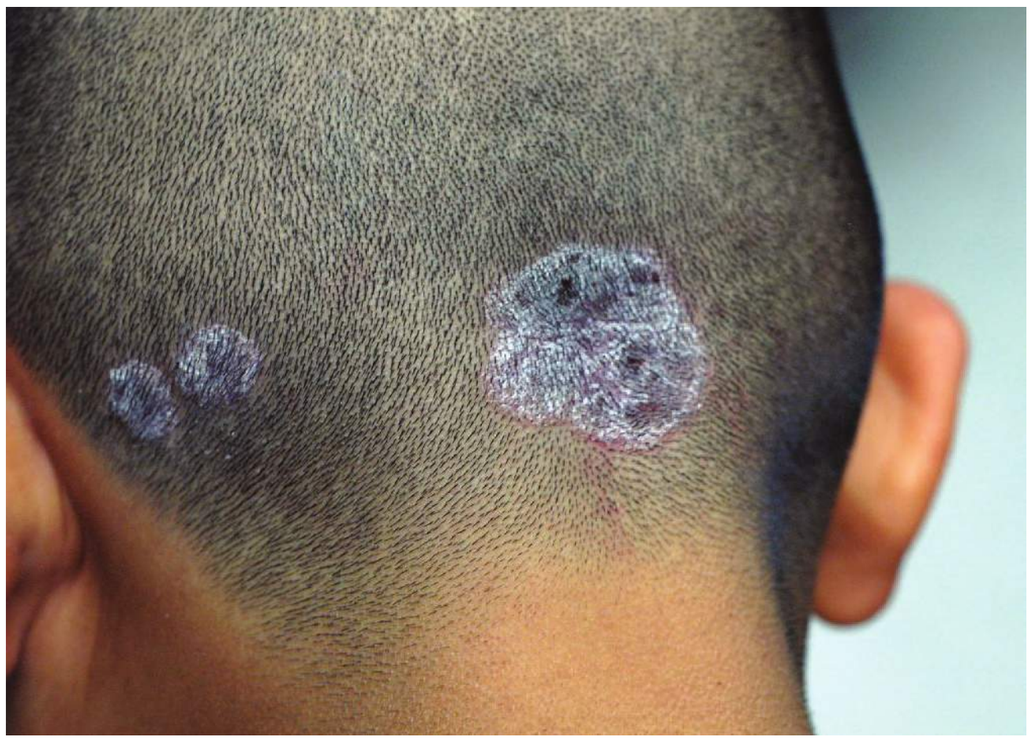


FIGURE 13.93 ■ **Tinea Capitis.** Multiple, well-defined, scaly plaques on the occiput. (Photo contributor: J. Matthew Hardin, MD.)

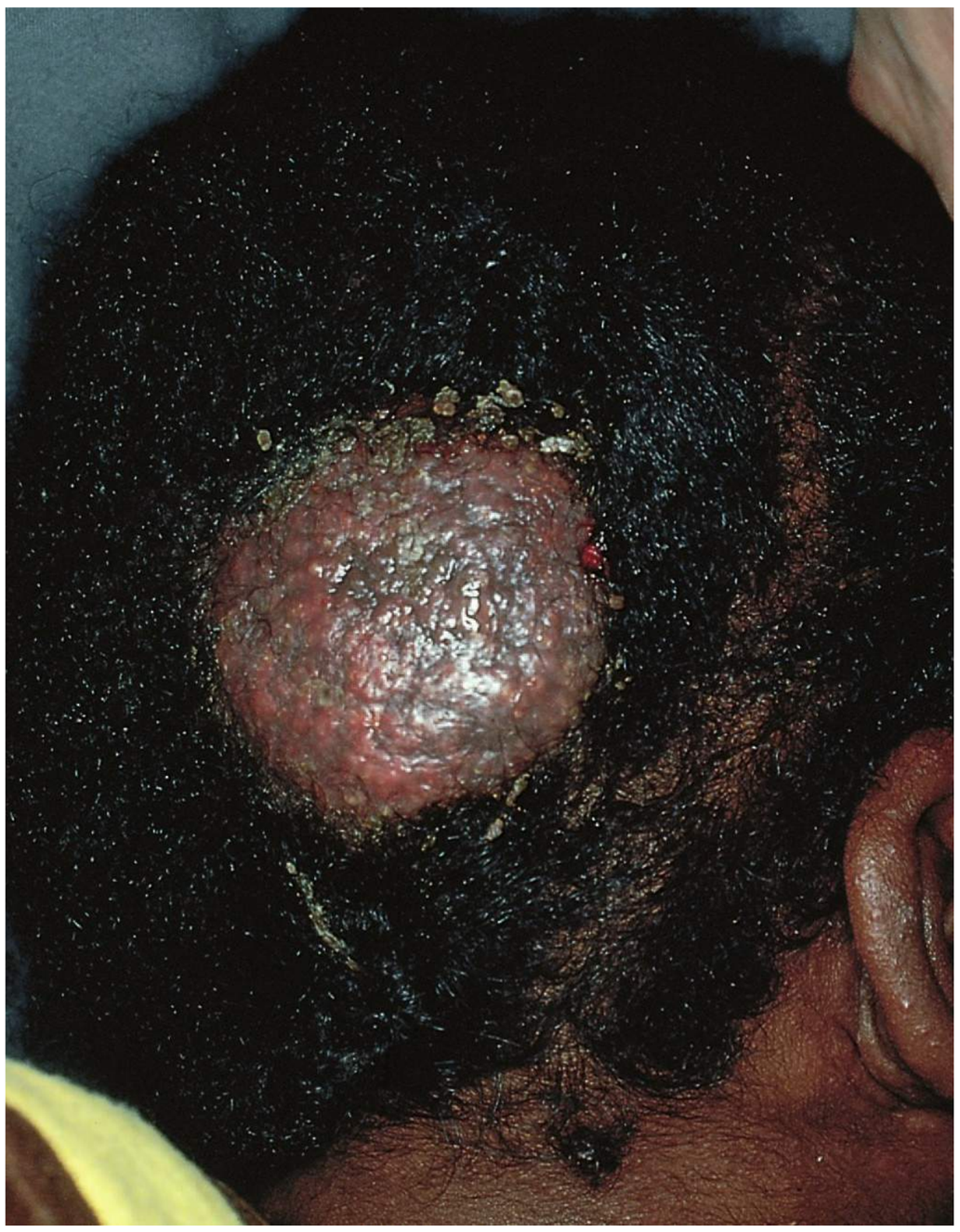


FIGURE 13.94 ■ **Kerion.** Occipital boggy swelling with hair loss consistent with kerion. (Photo contributor: Anne W. Lucky, MD.)

# TINEA (PITYRIASIS) VERSICOLOR

## Clinical Summary

Tinea versicolor, or pityriasis versicolor, is a chronic, superficial fungal infection that involves the trunk and extremities with rare facial involvement. The fungus is part of normal skin flora. Finely scaling brown macules are present in fair-skinned patients, whereas scaly hypopigmented macules are often noted in dark-skinned patients.

## Management and Disposition

Treatment consists of short applications of selenium sulfide lotion, topical antifungal creams, or topical ketoconazole. Resistant cases require referral and consideration of oral antifungal.

## Pearls

1. Tinea versicolor is more common in adolescents and young adults.
2. Clinically active areas or areas colonized with the fungus may be identified by orange fluorescence noted on the Wood lamp examination.
3. Normal pigmentation may take months to return.

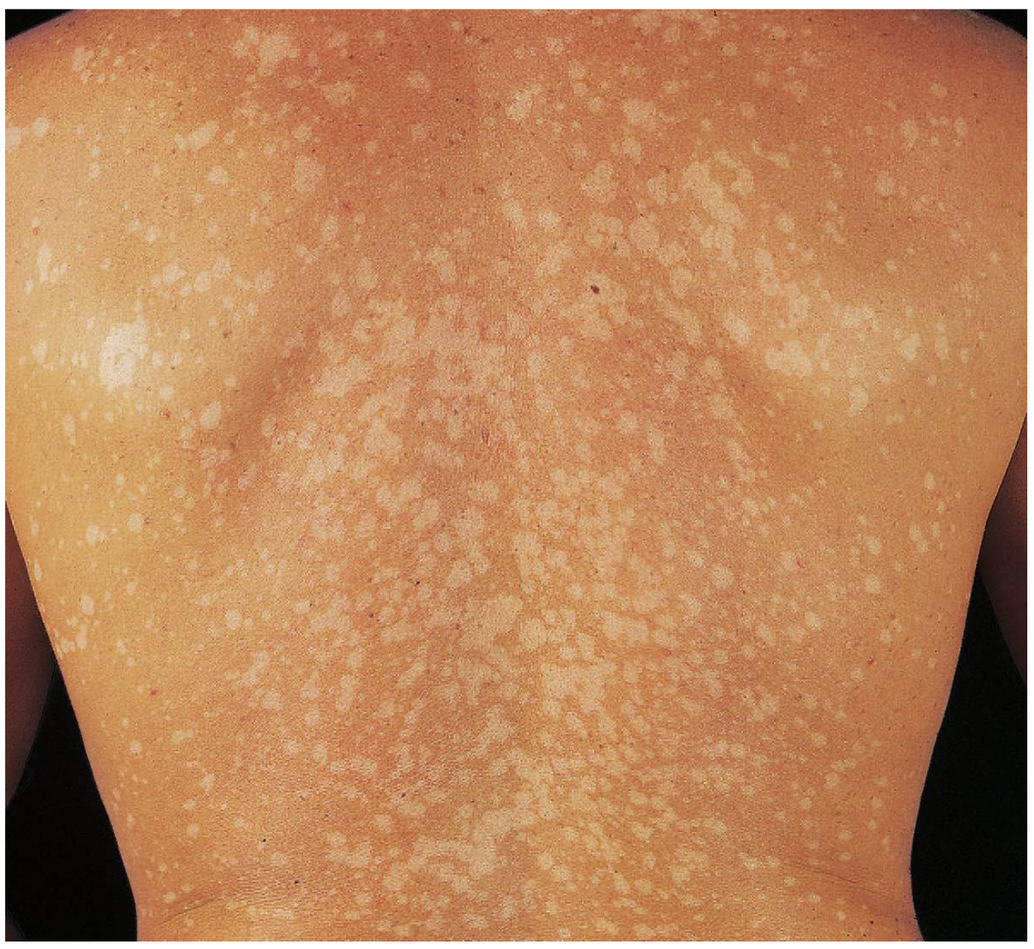


FIGURE 13.95 ■ **Tinea Versicolor.** Multiple, small-to-medium-sized, well-demarcated hypopigmented macules on the back of a tanned individual with white skin. (Used with permission from Wolff K, Johnson RA, Saavedra AP. Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology. 7th ed. McGraw-Hill; 2013: Fig. 26-19.)

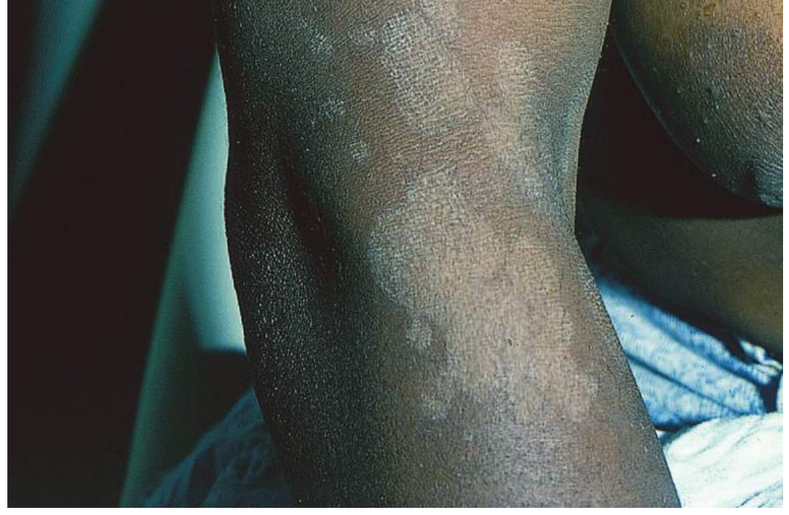


FIGURE 13.96 ■ **Tinea Versicolor.** An example of hypopigmented areas on dark skin. (Photo contributor: James J. Nordlund, MD.)

## Clinical Summary

Onychomycosis is an invasion of the nails by any fungus. Four clinical subtypes are described. Distal subungual presents as discolorations of the free edge of the nail with hyperkeratosis leading to subungual accumulation of friable keratinaceous debris. White superficial consists of sharply outlined white areas on the nail plate which leave the surface friable. Proximal subungual presents as discolorations which start proximally at the nail fold. Candidal onychomycosis encompasses the entire nail plate, leaving the surface rough and friable.

## Management and Disposition

Oral antifungals are required to eradicate the fungus. Refer to a dermatologist or primary care physician.

## Pearls

1. All that causes the nail plate to separate from the nail bed is not necessarily fungus. Psoriasis, lichen planus, and various other nail dystrophies, such as distal onycholysis (caused by excessive water exposure or drugs) must be differentiated from this fungal infection.
2. Treatment requires proper monitoring and long-term follow-up.

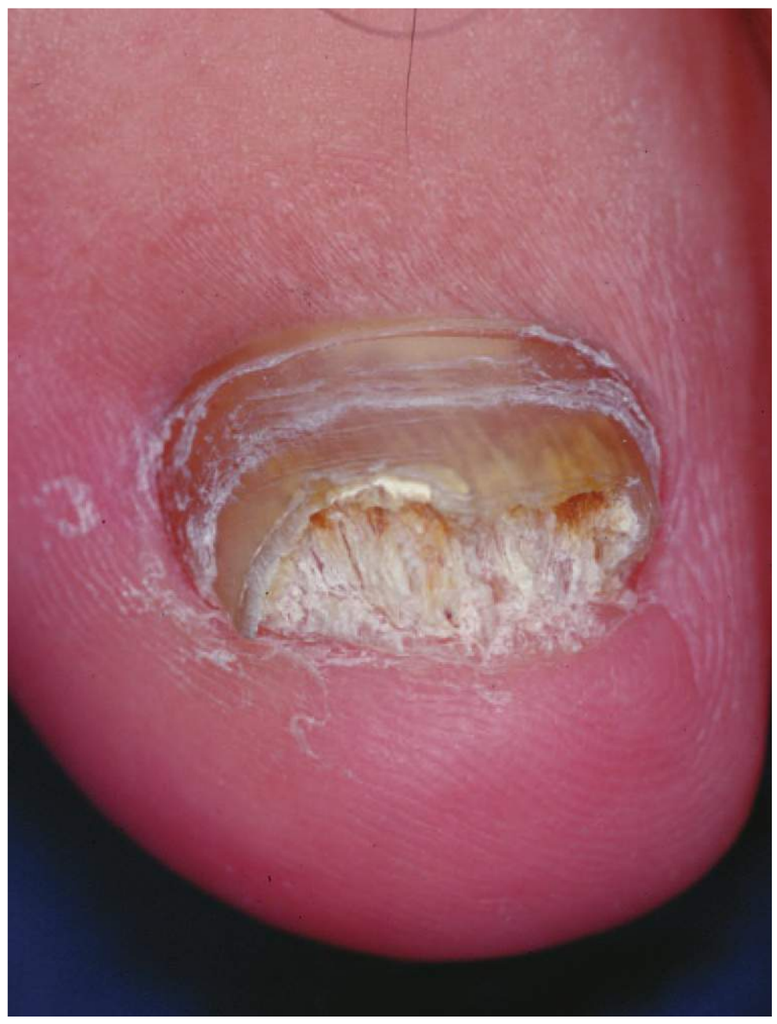


FIGURE 13.97 ■ **Onychomycosis.** Invasion of the nail bed by fungus. (Photo contributor: Lawrence B. Stack, MD.)

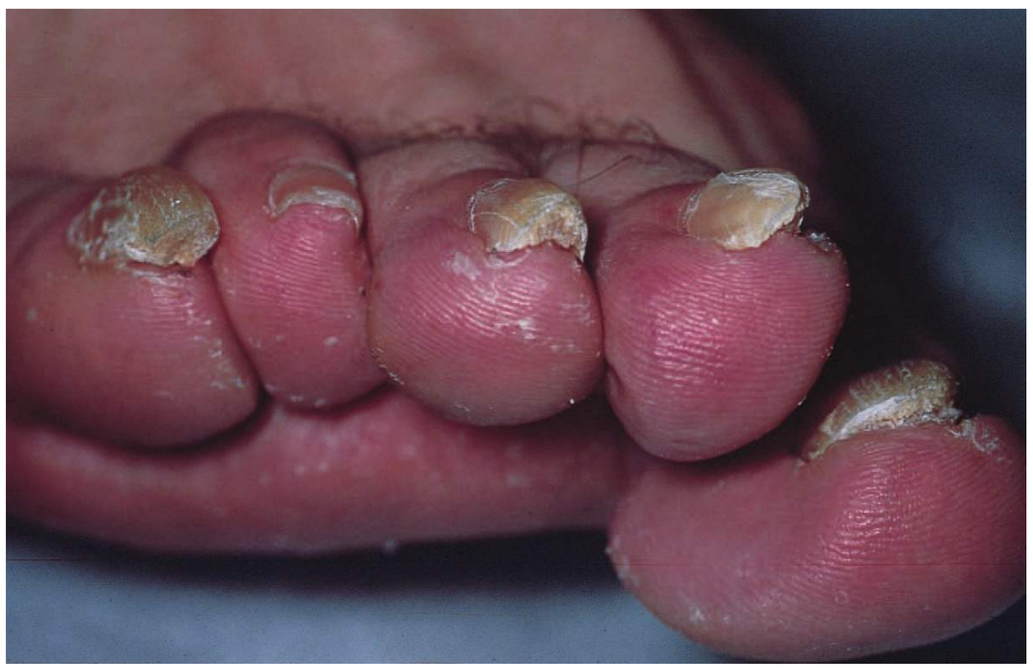


FIGURE 13.98 ■ **Onychomycosis.** Note that multiple nail beds have been invaded by the fungus, leading to chronic hyperkeratosis and subungual accumulation of friable keratinaceous debris. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)

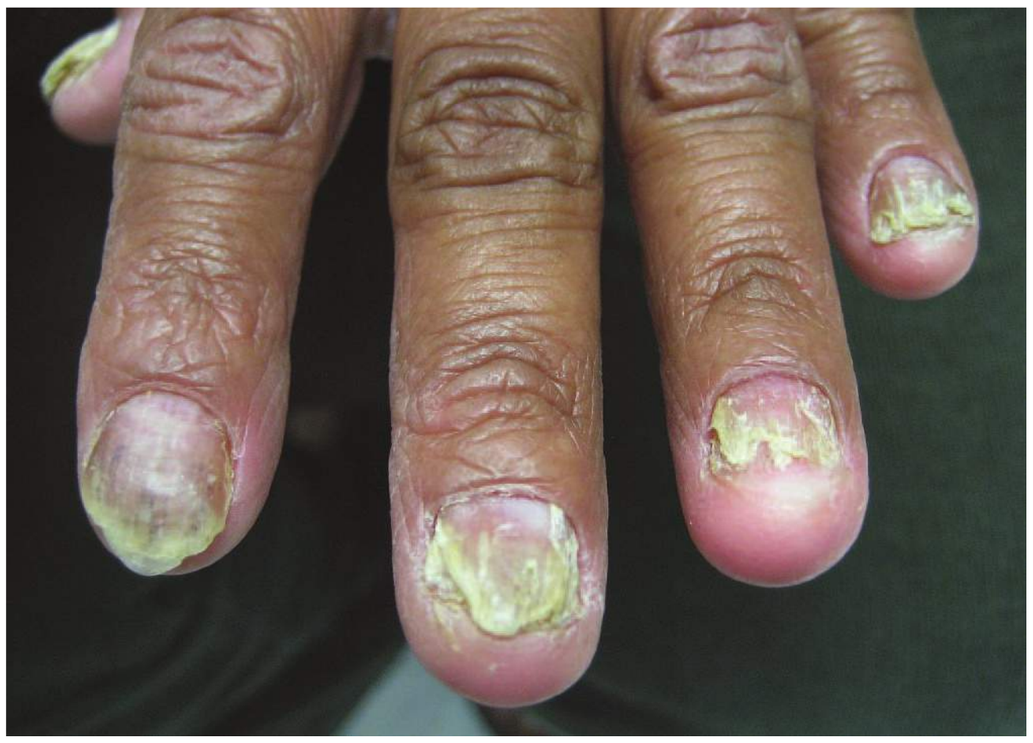


FIGURE 13.99 ■ **Onychomycosis.** Multiple nail beds with dystrophy and yellow subungual debris. (Photo contributor: J. Matthew Hardin, MD.)

# INTERTRIGO

## Clinical Summary

Intertrigo is a dermatitis occurring on opposed surfaces of skin, such as the creases of the neck, folds of the groin and armpit, or a panniculus. It is characterized by a tender, red patch or plaque with a moist, macerated surface. The juxtaposed skin surfaces create a chronic friction and this can easily become suprainfected with candida, fungal, or bacterial infections.

## Management and Disposition

Local care, empiric topical antifungal treatment, and good personal hygiene are recommended.

## Pearls

1. The nature of the intertriginous areas make them a high risk for secondary infection.
2. Potent topical corticosteroids should be avoided because of the high atrophy risk.

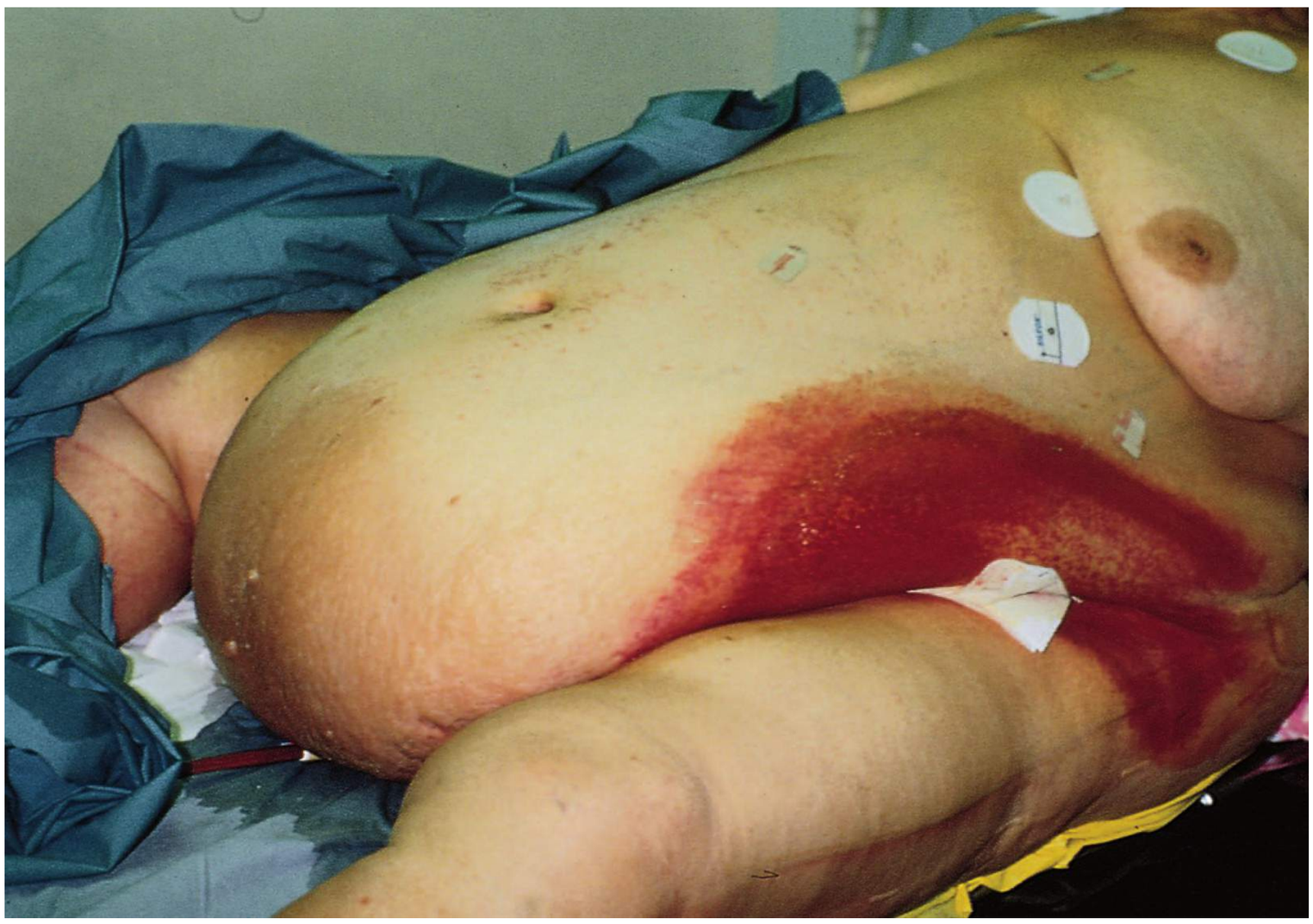


FIGURE 13.100 ■ **Intertrigo of the Panniculus.** In this dramatic case, the weight of the pannus creates constant friction on the abdominal wall. This leads to erythema and tissue breakdown. This patient also had fever, suggesting secondary infection. (Photo contributor: Lawrence B. Stack, MD.)

## Clinical Summary

Human scabies is caused by Sarcoptes scabiei var. hominis, a mite that lives within the layers of the epidermis. Transmission of scabies occurs after direct skin contact with an infected individual or possibly from infested clothing and bedding. The female mite burrows into the individual's skin and deposits two to three eggs daily. Fecal pellets (scybala) are also deposited in the burrow and may be responsible for the localized pruritus. Nocturnal pruritus is characteristic of scabies. The pink-white, slightly elevated burrows are typically found in the web spaces of the hands and feet, penis, buttocks, scrotum, and extensor surfaces of the elbows and knees. Crusted, or Norwegian, scabies seen in immunosuppressed or debilitated patients (see HIV chapter) present with asymptomatic acral crusting, but can occur anywhere.

## Management and Disposition

Topical 5% permethrin cream is commonly used to treat scabies. Apply from the neck to the toes overnight (8-12 hours) and then wash off. Repeat treatment in 7 days. Bedding, towels, and clothing should be washed in hot water and dried on high heat (an alternative is to place items into a plastic bag for 10 days).

## Pearls

1. Intimate contacts and all family members in the same household should be treated.
2. Patients often experience "postscabietic itch" which can last for 2 to 4 weeks. This is not a new infestation but rather an immunologic reaction to the dead mite. Confirm completion of an appropriate treatment.

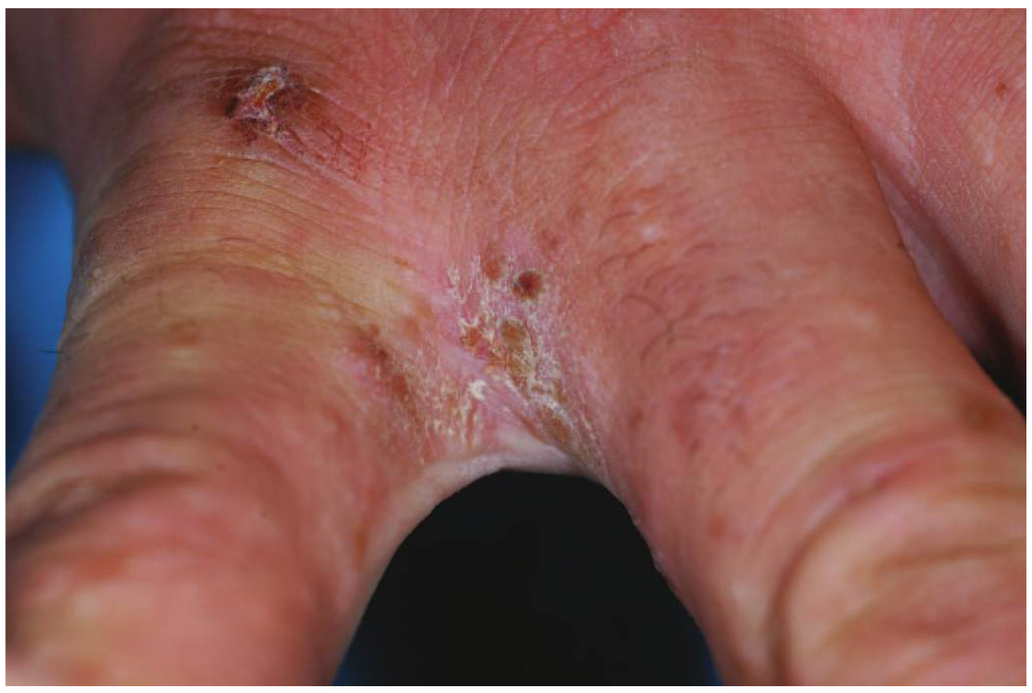


FIGURE 13.102 ■ **Scabies.** Burrows and erosions in the web space of the hand. (Photo contributor: David Effron, MD.)

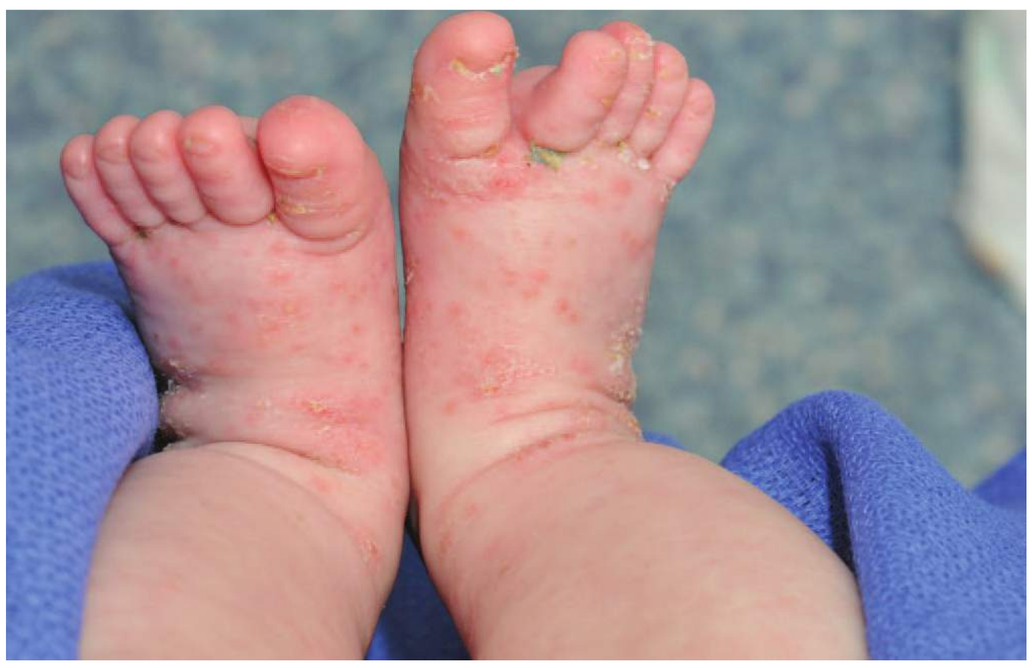


FIGURE 13.101 ■ **Scabies.** Burrows and erosions from itching on the feet of a patient with scabies. (Photo contributor: Lawrence B. Stack, MD.)

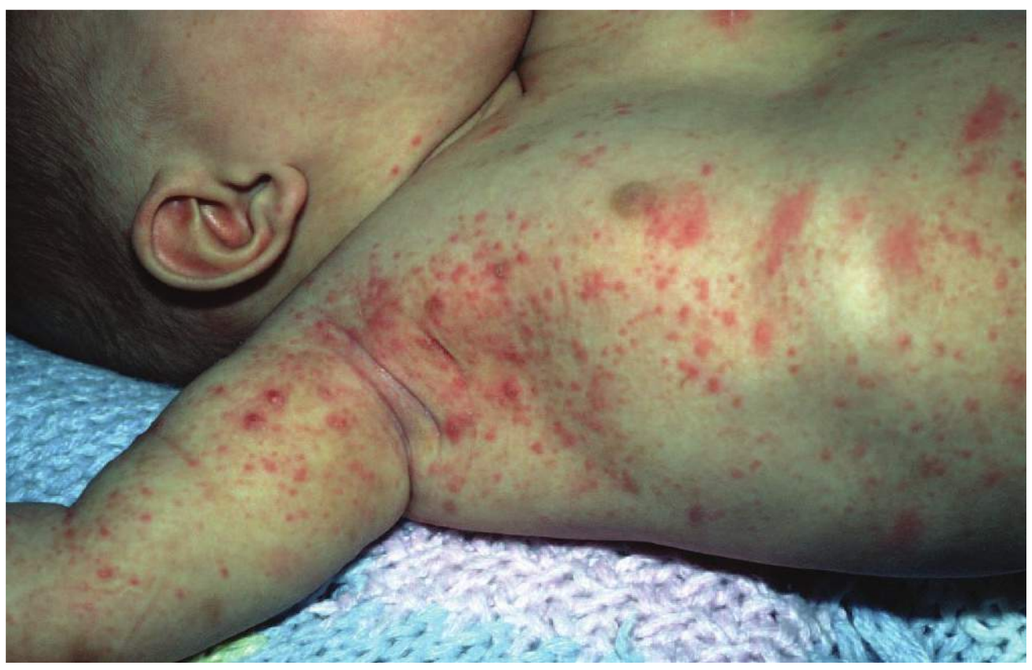


FIGURE 13.103 ■ **Scabies.** Extensive burrows and erosions in an infant. (Photo contributor: David Effron, MD.)

# JAUNDICE

## Clinical Summary

Jaundice is a light yellowing of the skin, mucous membranes, and sclera; it is generally detectable when bilirubin levels are about 3.0 mg/dL. Many patients may not be aware of the faint yellowing and present with seemingly unrelated symptoms. Be aware that up to 50% of patients with jaundice will have pruritus. The most important diagnoses to rule out are: hemolytic anemias, viral hepatitis, chronic alcohol abuse, autoimmune hepatitis, medications, primary biliary cirrhosis, primary sclerosing cholangitis, cholelithiasis, surgical strictures, and obstructive malignancies. Acetaminophen, penicillins, and oral contraceptives are some of the more common medications associated with jaundice.

## Management and Disposition

As the etiology of jaundice is broad, a thorough history focusing on associated symptoms (fever, pruritus, vomiting, hematochezia, melena, and abdominal pain), previous surgical procedures, and medication history (including over-the-counter medications) is essential. Physical findings of fever, abdominal tenderness, and hepatomegaly should be sought. Workup of jaundiced patients should include white blood cell count and differential, liver function tests including bilirubin levels, hepatitis viral screening, and imaging studies.

## Pearls

1. Patients who consume large amounts of β-carotene (found in squash and carrots) may have mild yellowing of their skin (especially palms and soles) but will lack scleral icterus or elevations in bilirubin.
2. Women starting oral contraceptives may experience cholestasis in the first few months that may cause jaundice.

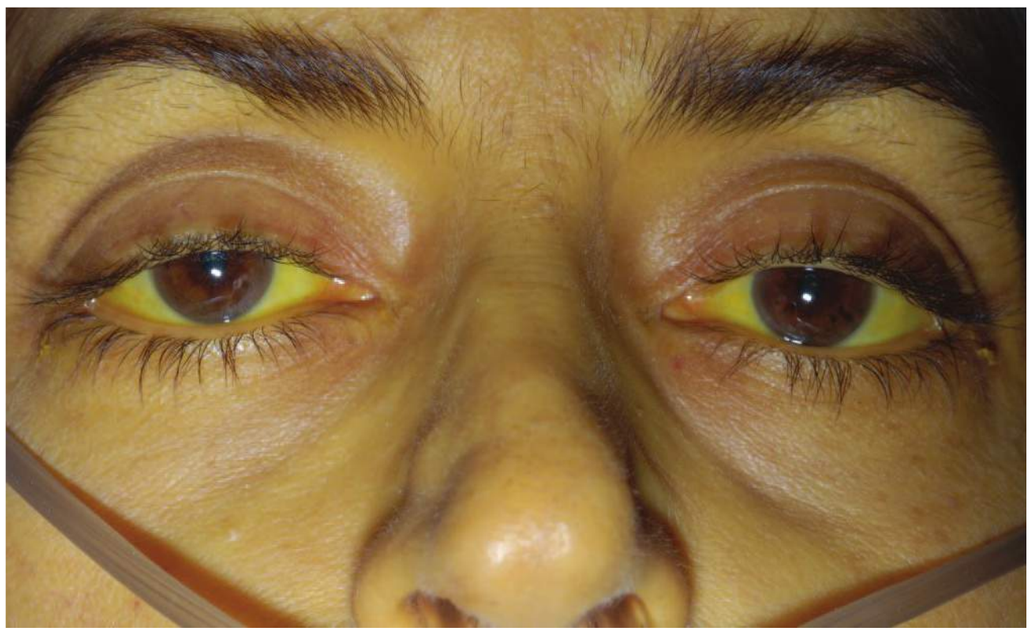


FIGURE 13.105 ■ **Jaundice, Scleral Icterus.** Yellowing of the sclera in a patient with liver disease. (Photo contributor: R. Jason Thurman, MD.)

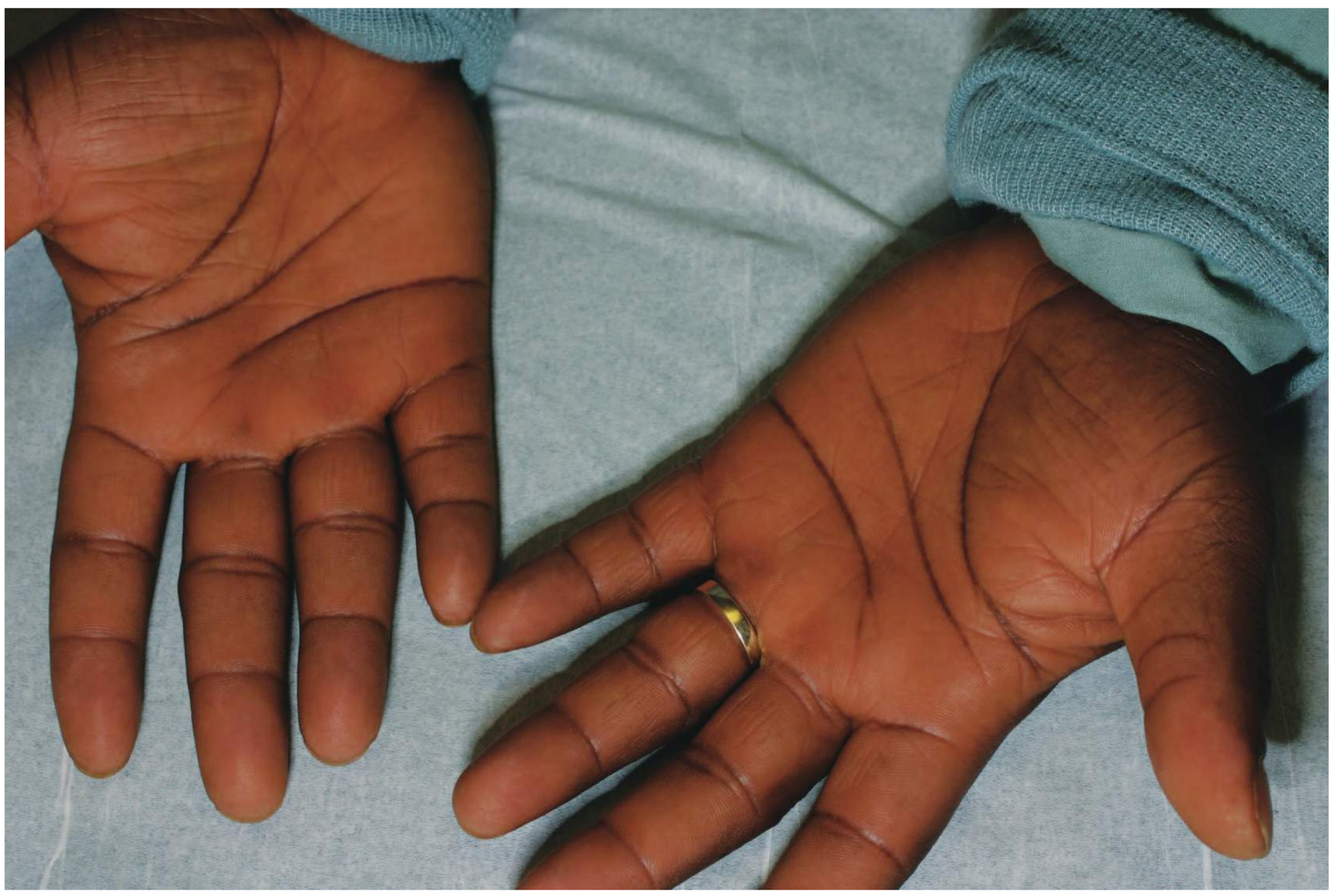


FIGURE 13.104 ■ **Jaundice.** Mild palmar jaundice in a dark-skinned patient. (Photo contributor: Kevin J. Knoop, MD, MS.)

## Clinical Summary

Melasma is commonly seen on the face of young adult females. It consists of symmetric, well-defined, light to dark brown patches. The most common sites are on the malar cheek, lateral forehead, upper cutaneous lip, and mandible. Factors associated with accentuation of melasma include sunlight exposure, pregnancy (often called "the mask of pregnancy"), and oral contraceptives.

## Management and Disposition

Most patients will be concerned about accentuation of their previously imperceptible melasma. Rule out pregnancy in new onset or worsening melasma. Essential to any treatment of melasma is strict sun avoidance. Refer patients to a dermatologist.

## Pearls

1. Melasma is common in young females with darker skin types but all races can be afflicted.
2. Sun exposure on other parts of the body can cause accentuation of facial melasma.
3. Contrasted with the hyperpigmentation of melasma, Addison disease is diffuse hyperpigmentation with accentuation in sun-exposed areas.

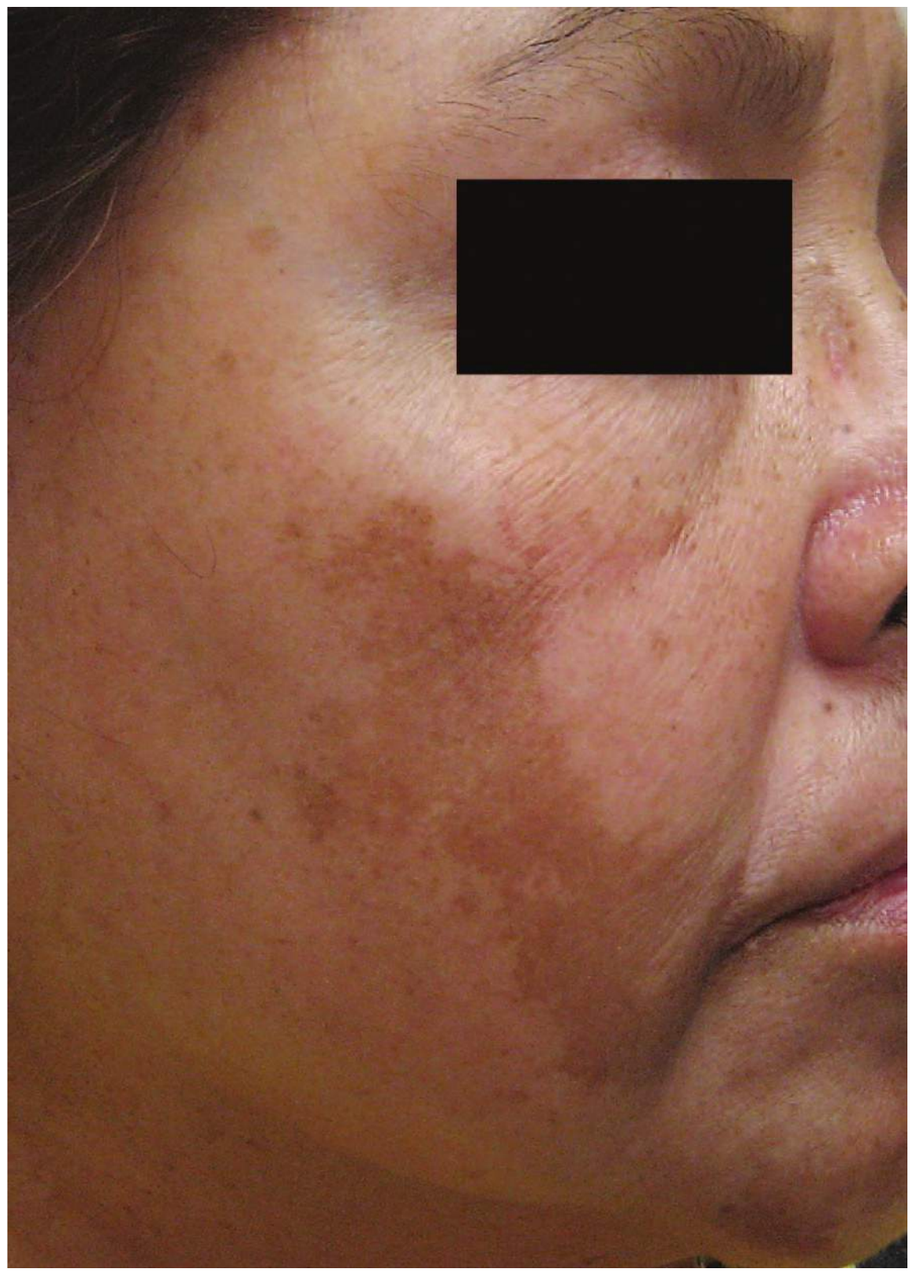


FIGURE 13.106 ■ **Melasma.** Well-demarcated, hyperpigmented patch seen on the cheek. (Photo contributor: J. Matthew Hardin, MD.)

# ABDOMINAL STRIAE (STRIAE ATROPHICAE)

## Clinical Summary

Abdominal striae are linear, depressed, pink or bluish scar-like lesions that may later become silver or white. They are caused by weakening of the elastic cutaneous tissues from chronic stretching. They most commonly occur on the abdomen but are also seen on the buttocks, breasts, axilla, and thighs. Striae are commonly seen in obesity, pregnancy, rapid growth associated with puberty, Cushing syndrome, and chronic topical corticosteroid treatment.

## Management and Disposition

This finding seldom presents as a condition requiring acute treatment; thus, attention is directed to determining and treating the underlying cause.

## Pearls

1. Recent striae with moon facies, hypertension, renal calculi, osteoporosis, and psychiatric disorders are suggestive of Cushing syndrome.
2. The striae color (red/purple) caused by pregnancy typically fades with time, unlike striae associated with Cushing syndrome.
3. Inappropriate use of topical corticosteroids can result in permanent striae.

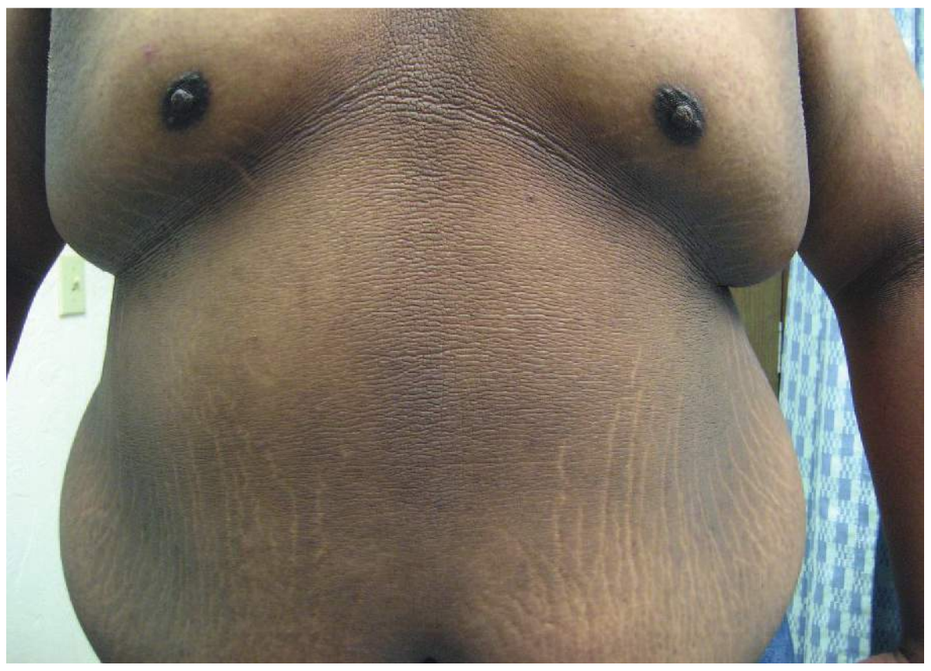


FIGURE 13.108 ■ **Abdominal Striae.** Obese patient with lower lateral wall striae. Also noted is thickened, hyperpigmented, abdominal skin, typical of acanthosis nigricans (associated with diabetes). (Photo contributor: J. Matthew Hardin, MD.)

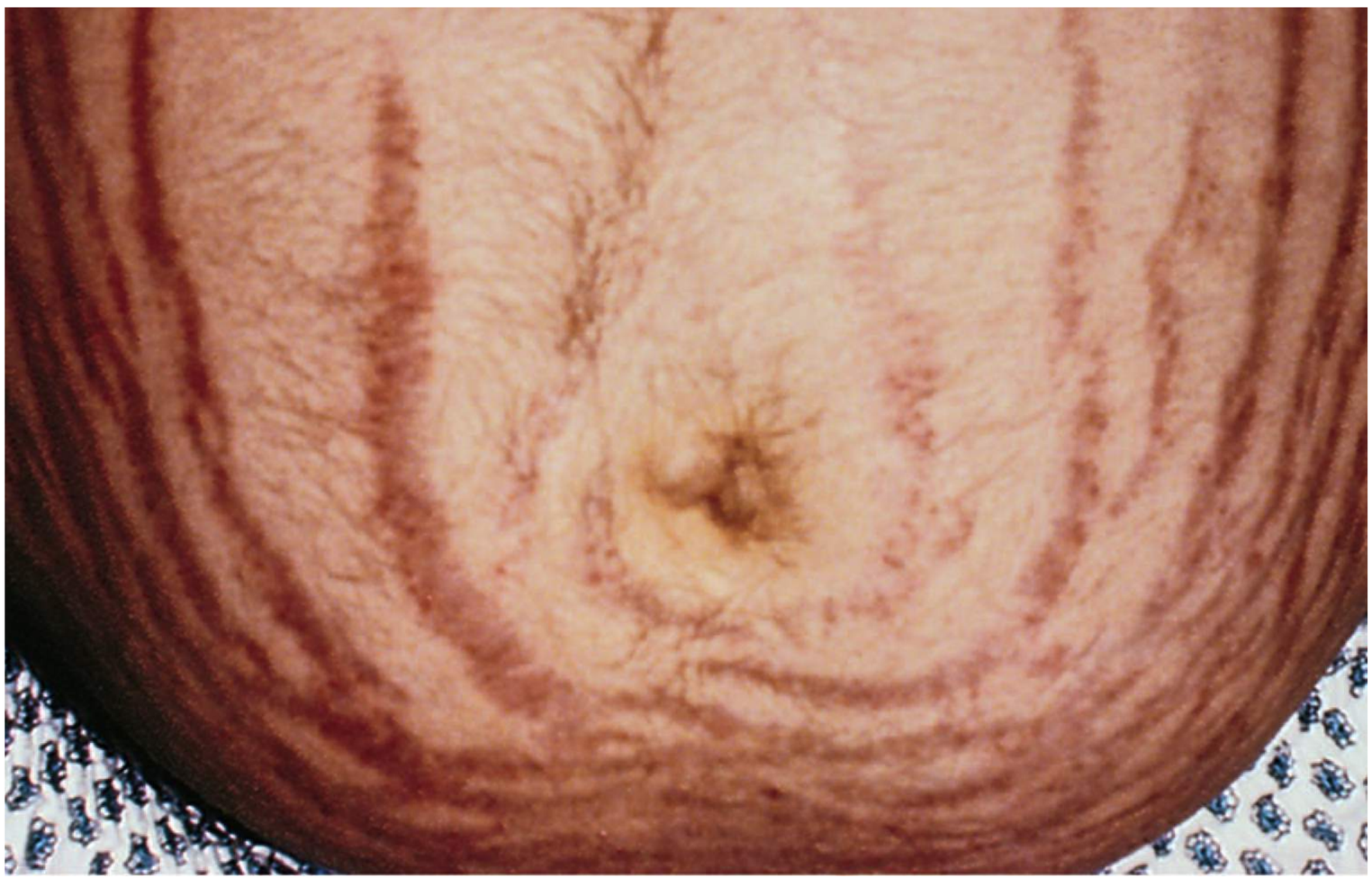


FIGURE 13.107 ■ **Abdominal Striae.** These striae are seen in a patient with recent weight gain, moon facies, and altered mental status. The patient was diagnosed with Cushing syndrome.

## Clinical Summary

Vitiligo is an acquired loss of pigmentation that commonly involves the backs of the hands, face, and body folds. There is a positive family history in 30% of the cases. Initially the disease is limited, but it then slowly progresses over years. Vitiligo is secondary to absence of epidermal melanocytes, which may be due to an autoimmune phenomenon against melanocytes. Approximately half of these cases begin in patients less than 20 years of age.

## Management and Disposition

Refer patients to a dermatologist for further workup and long-term treatment.

## Pearls

1. Vitiligo can occur at sites of trauma (Koebner phenomenon).
2. Wood lamp examination helps identify hypopigmented areas in patients with light complexions.
3. Tinea versicolor, in contrast, has a scale and positive KOH preparation.

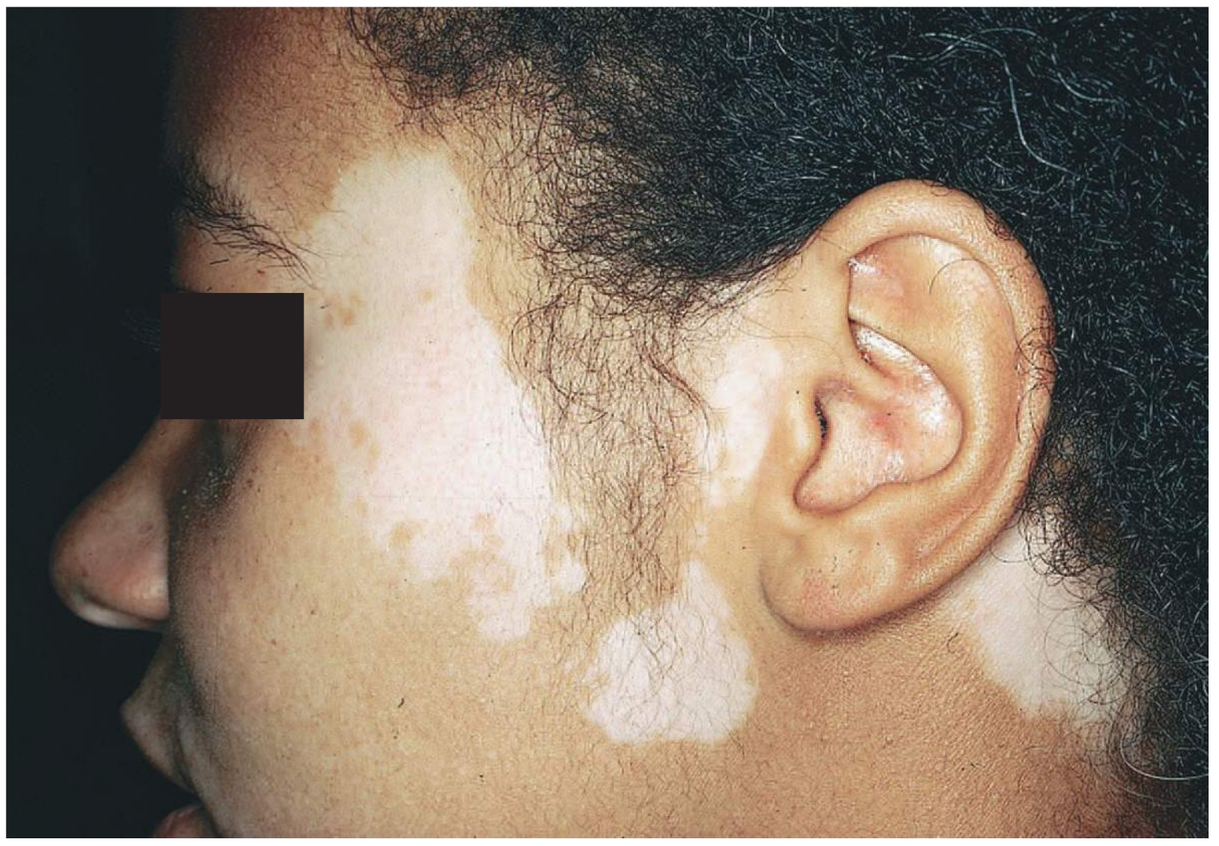


FIGURE 13.109 ■ **Vitiligo.** Well-defined, hypopigmented areas characteristic of vitiligo. (Photo contributor: James J. Nordlund, MD.)

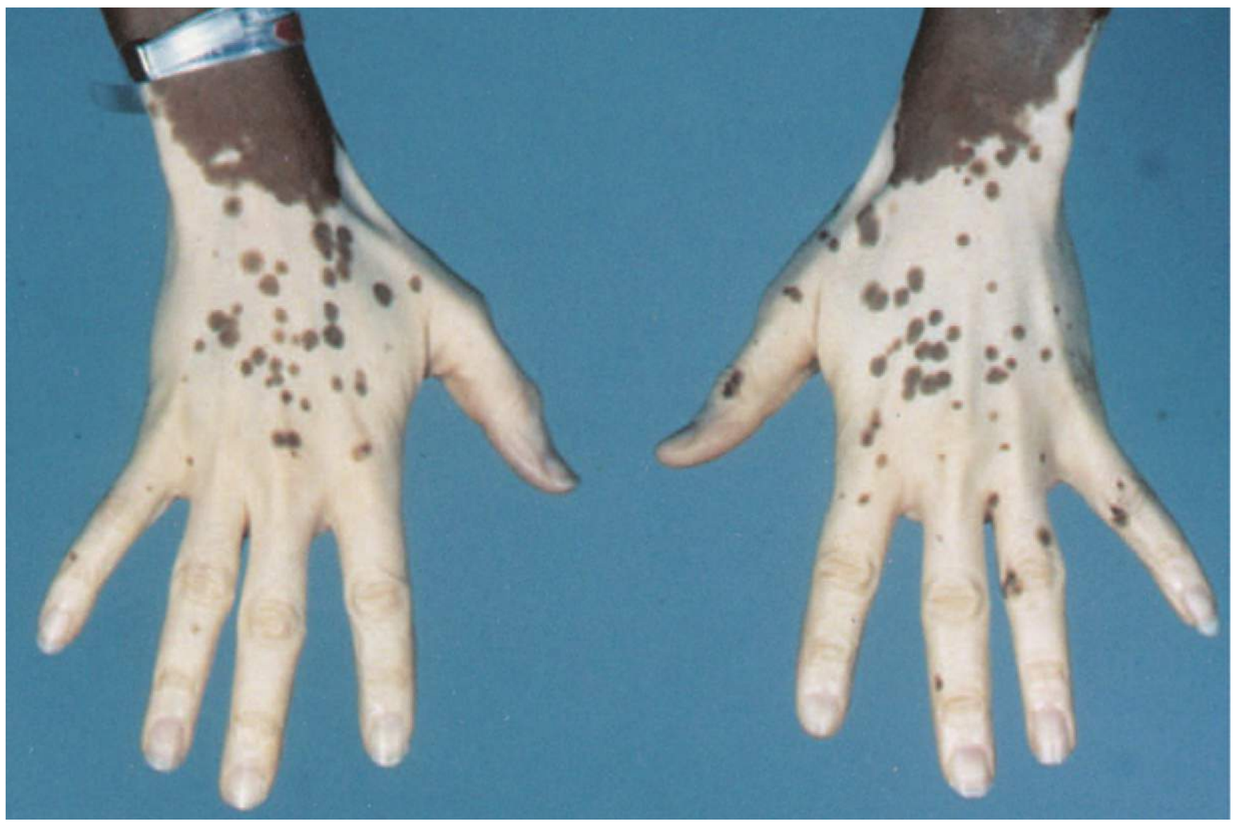


FIGURE 13.110 ■ **Vitiligo.** Almost complete depigmentation of the hands. (Used with permission from Fauci AS, Braunwald E, Kasper DL, et al. Harrison's Principles of Internal Medicine. 17th ed. New York, NY: McGraw-Hill; 2008: 312.)

# UREMIC FROST

## Clinical Summary

Uremic frost is a classic manifestation of chronic renal failure; it is rarely seen today. It develops as a result of accumulation of urea in sweat. In advanced uremia, the accumulation of urea in sweat may reach such a critical level that, upon its evaporation, a fine white powder is left on the skin surface. Associated hyperkalemia may also be present owing to concurrent renal failure.

## Management and Disposition

Treatment of the underlying condition that resulted in the patient's uremia may prevent further accumulation of uremic frost. Typically, urgent dialysis is indicated.

## Pearls

1. Although rare today, this condition may be seen in noncompliant patients or environmental stressors, including inadequate air conditioning.
2. For patients presenting with altered mental status, attention to the airway, oxygenation, and rapid assessment and treatment of associated metabolic disorders, such as hyperkalemia, is paramount.

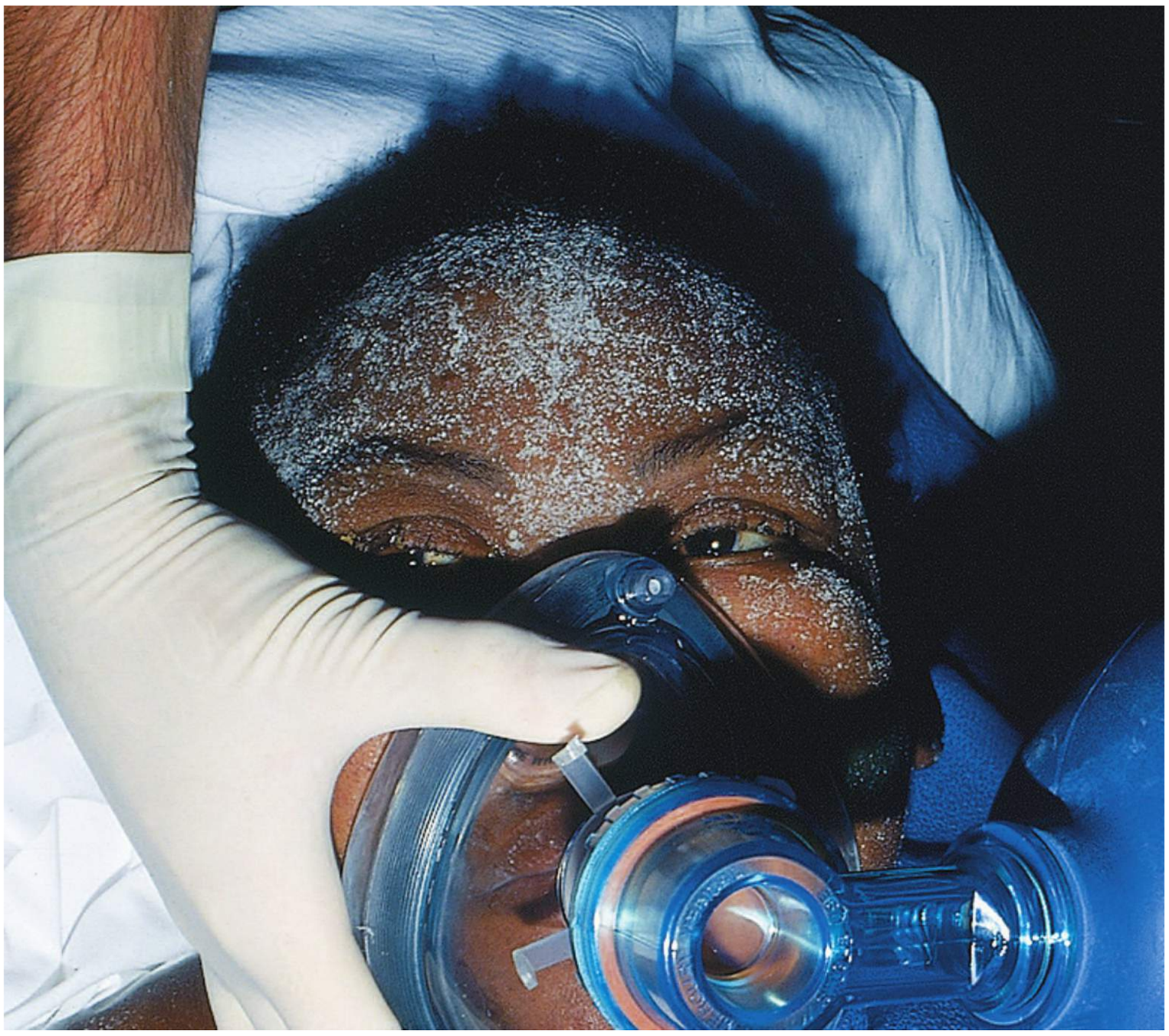


FIGURE 13.111 ■ **Uremic Frost.** Note the fine white powder on the skin of this patient with end-stage renal disease. (Photo contributor: Alan B. Storrow, MD.)

## Clinical Summary

Autoimmune bullous diseases are uncommon but have dramatic presentations. Bullous pemphigoid (BP) results from autoantibodies to the epidermal basement membrane and creates tense bullae, frequently located on the proximal extremities. In pemphigus vulgaris (PV), the autoantibodies are directed against the epidermal keratinocytes. This results in flaccid bullae (more superficial bullae that easily slough off to form erosions). PV delicate bullae and subsequent erosions commonly present in the pharynx, scalp, and trunk. Paraneoplastic pemphigus presents with severe oral ulcerations (similar to SJS/TEN; see related item) and resolves with treatment of the associated malignancy.

## Management and Disposition

Considering an autoimmune bullous disease in the differential is the first step. Admission with early dermatologic consultation for histologic and immunofluorescent studies should be considered. Systemic corticosteroids and immunosuppressant therapy are required for control. Patients with significant BSA involvement should be treated in a burn unit.

## Pearls

1. Oral erosions and ulcerations should always raise the suspicion of autoimmune bullous diseases.
2. The high morbidity and mortality of this disease is now significantly lower due to modern steroid-sparing immunosuppressants and wound care.
3. Always consider medication history and signs of underlying malignancies when evaluating a patient with extensive bullae or erosions.

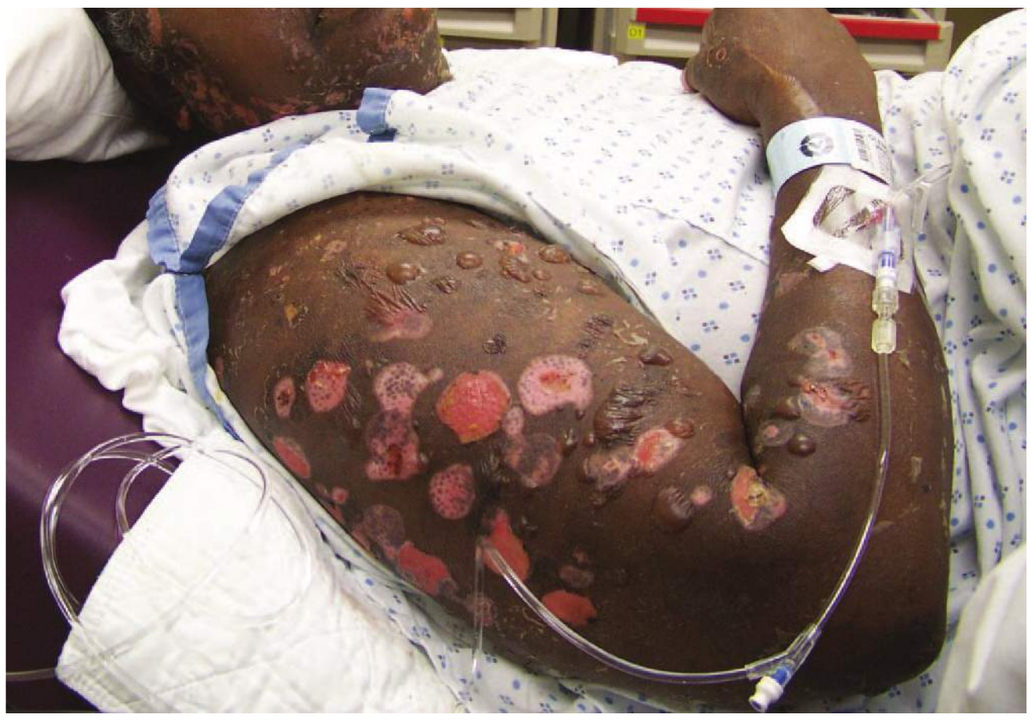


FIGURE 13.112 ■ **Bullous Pemphigoid.** Tense blister formation among confluent erosions and plaques. (Photo contributor: Lawrence B. Stack, MD.)

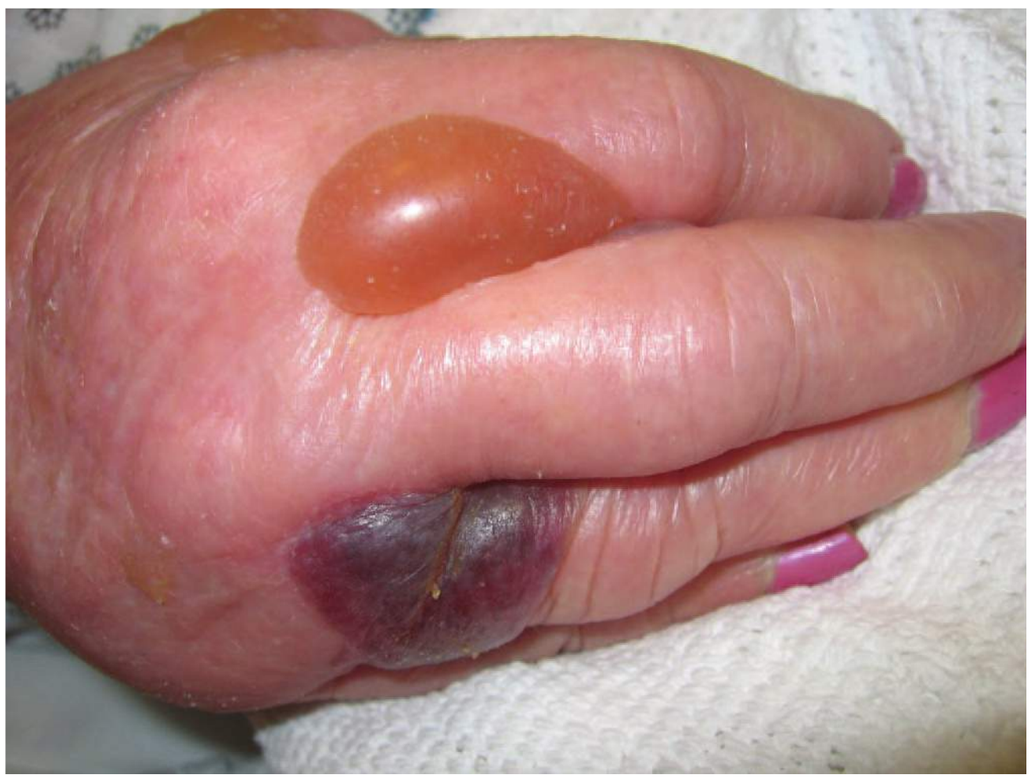


FIGURE 13.113 ■ **Bullous Pemphigoid.** Tense bullae on in the distal extremities are a common presentation of BP. (Photo contributor: Selim Suner, MD, MS.)

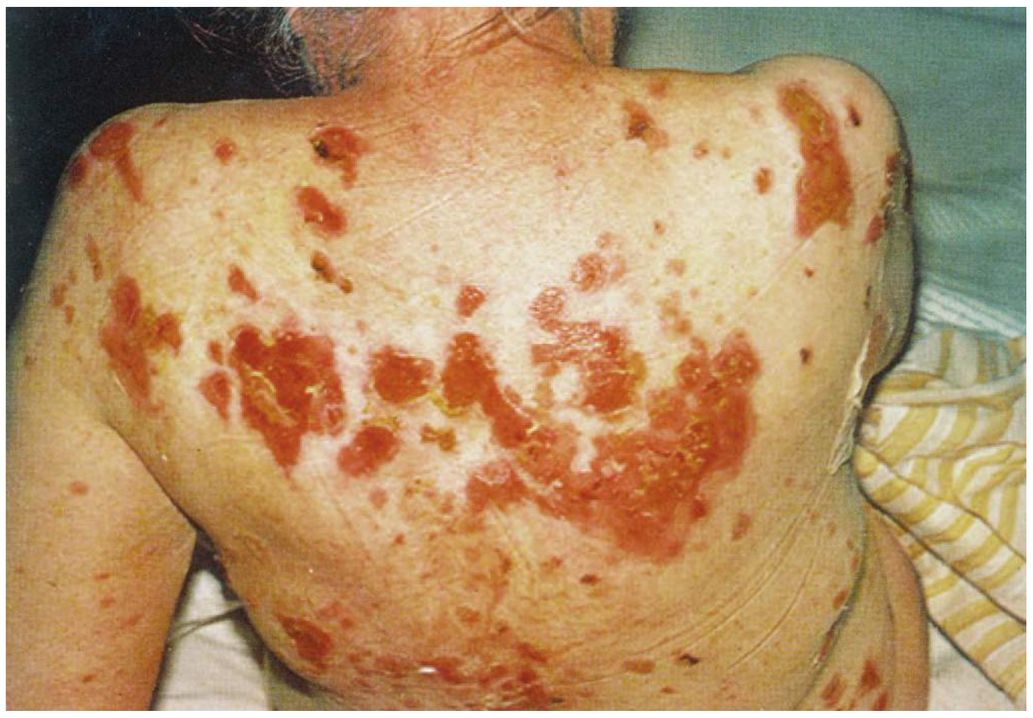


FIGURE 13.114 ■ **Pemphigus Vulgaris.** Multiple flaccid bullae now de-roofed and at risk for secondary infection. (Photo contributor: J. Matthew Hardin, MD.)

# SYSTEMIC LUPUS ERYTHEMATOSUS

## Clinical Summary

Systemic lupus erythematosus (SLE) has four cutaneous manifestations: malar rash, discoid rash, photosensitivity, and oral ulcers. Eighty percent of patients have skin findings at some point of the disease. The malar "butterfly" rash presents with erythema and mild edema over the bridge of the nose and malar cheeks. Similar erythematous patches may be seen on the ears, neck, and chest. Discoid lesions can appear at any site but are usually found above the neck, including the scalp. They are characterized as annular, erythematous macules, or plaques that eventually become atrophic and scarred.

## Management and Disposition

Urgent referral to a dermatologist or rheumatologist is essential for early diagnosis and prevention of other systemic manifestations.

## Pearls

1. Consider any suspicious rash on the face a manifestation of SLE and refer to a dermatologist early.
2. The malar rash resolves without scarring, whereas discoid lesions (primarily on the face, ears, and scalp) result in permanent scarring.
3. Not all photoexacerbated rashes are sunburns.

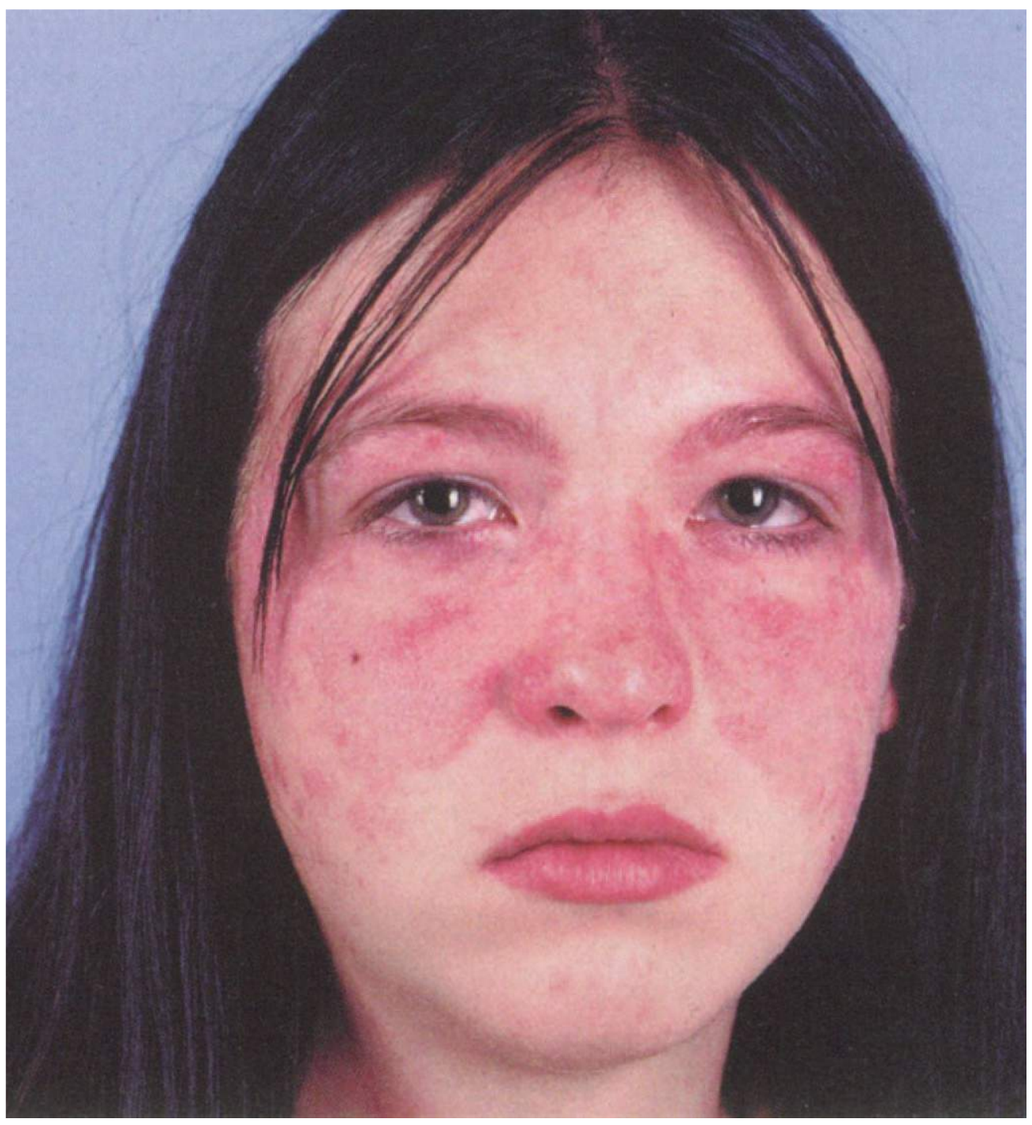


FIGURE 13.115 ■ **Butterfly Rash of SLE.** Erythematous malar rash with slight edema and minimal scaling ("butterfly pattern"). (Used with permission from Wolff K, Johnson RA, Suurmond D. Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology. 5th ed. New York, NY: McGraw-Hill; 2005: 385.)

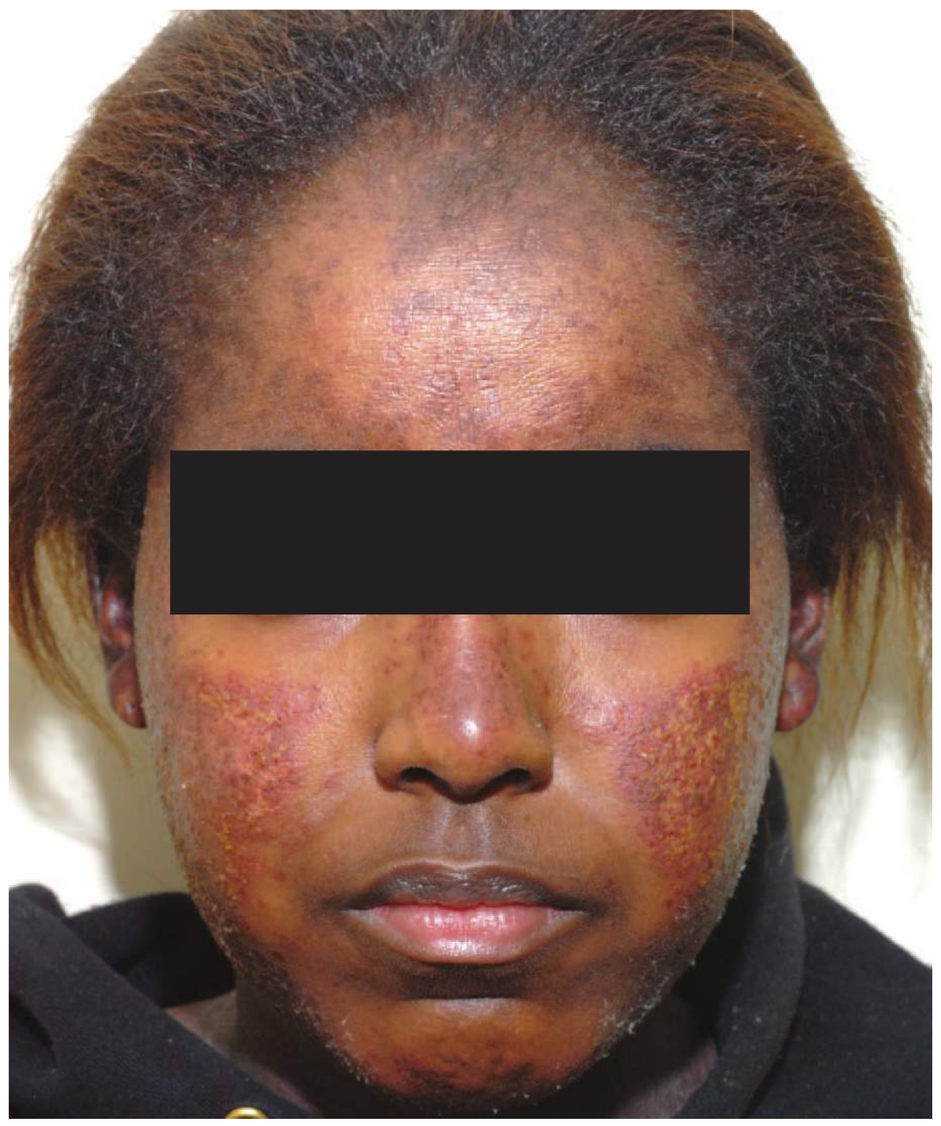


FIGURE 13.116 ■ **Butterfly Rash of SLE.** The appearance of the malar "butterfly" rash in a dark-skinned individual. Note mild scale on the cheeks and frontal thinning hairline. (Photo contributor: Lawrence B. Stack, MD.)

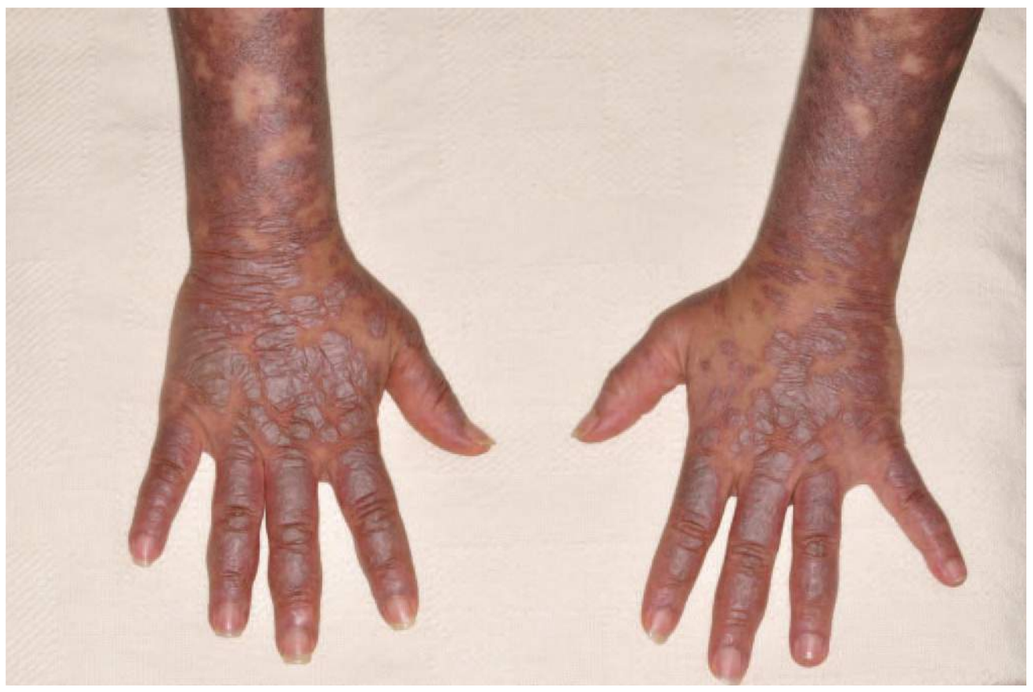


FIGURE 13.117 ■ **Discoid Lupus.** Well-defined scaly patches with atrophy. (Photo contributor: Kristin L. Stevens, MD.)

## Clinical Summary

Sweet syndrome, also known as acute febrile neutrophilic dermatosis, is characterized by fever, peripheral neutrophilia, and a nonvasculitic neutrophilic cutaneous eruption. Sweet syndrome can occur at any age but most commonly from 30 to 60 years old. Typically, the lesions are tender, well-demarcated erythematous plaques with an edematous periphery (pseudovesiculation). Some progress to ulcerations and hemorrhagic crusting. Lesions can occur anywhere on the body, but most frequently on the upper extremities, neck, and face. Variants have been isolated to the face (erysipelas-like) and the dorsal hands. The plaques generally cause a burning pain and are nonpruritic.

Sweet syndrome is associated with preceding upper respiratory infections, vaccinations, medications, malignancies, inflammatory bowel disease, autoimmune connective tissue diseases, and pregnancy.

## Management and Disposition

The associated diseases need to be considered and appropriate consultation is warranted. A dermatologist should be consulted for diagnosis confirmation and further evaluation. Management includes prednisone tapered over 4 to 6 weeks.

## Pearls

1. Unless a specific infection is identified (Streptococcus, Yersinia, or Staphylococcus), antibiotics do not improve Sweet syndrome.
2. Malignancies associated with Sweet syndrome account for 20%; acute myeloid leukemia is the most common hematologic malignancy.
3. Although preceding infections can cause this rash, the lesions are not infectious.

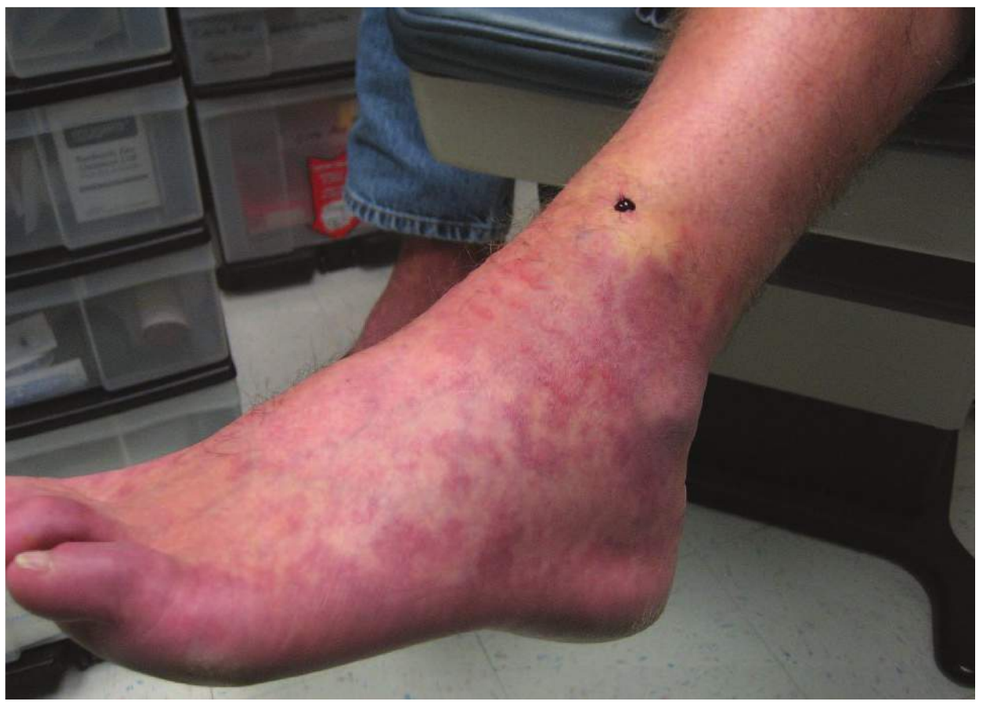


FIGURE 13.119 ■ **Sweet Syndrome.** Less obvious erythematous plaques on the foot. (Photo contributor: J. Matthew Hardin, MD.)

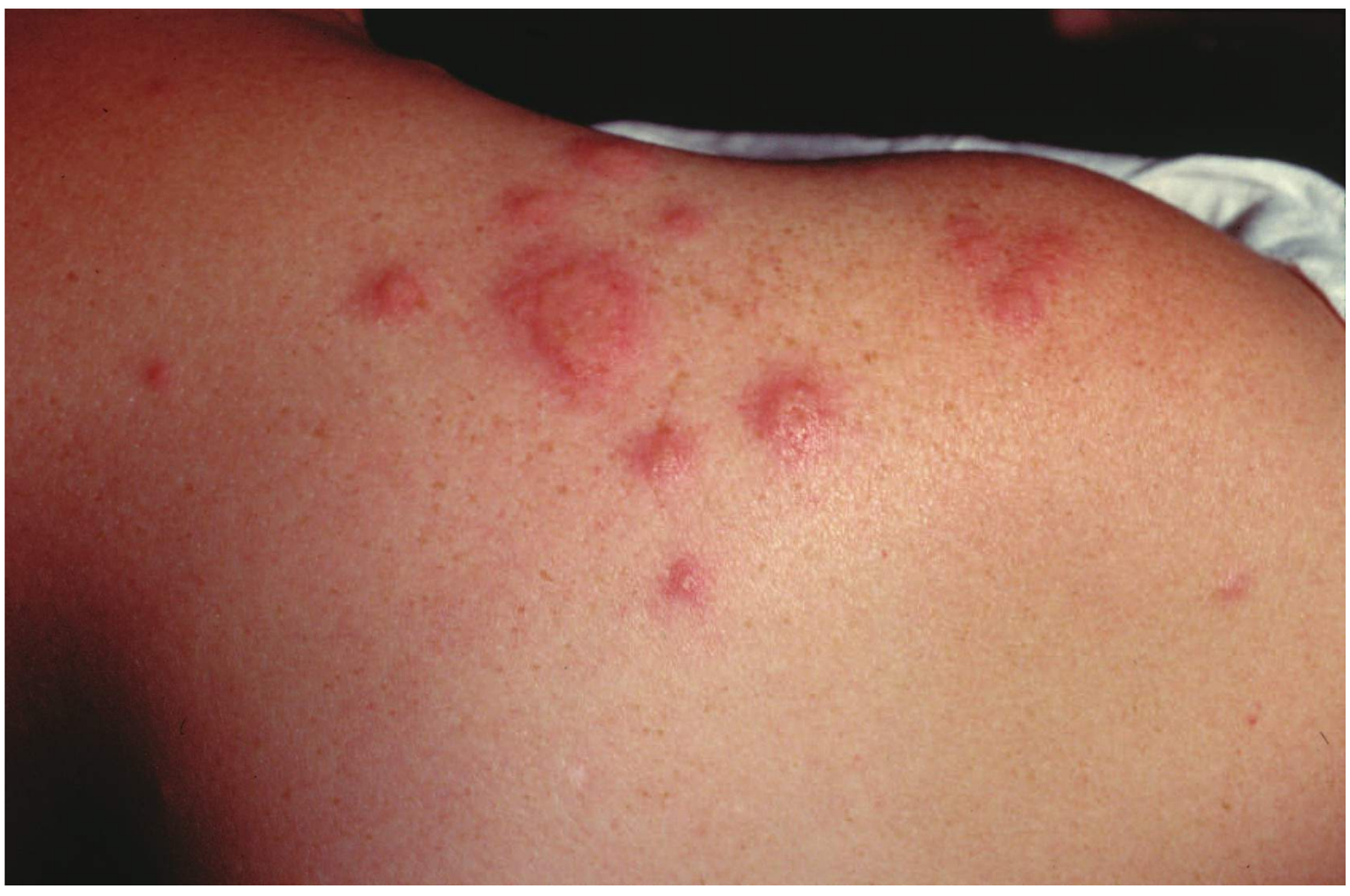


FIGURE 13.118 ■ **Sweet Syndrome.** This young woman complained of sudden onset of fever and painful skin lesions. Her WBC was 22,000 with neutrophilia. This constellation of symptoms suggests Sweet syndrome, and was confirmed by histology. There is a hint of peripheral vesiculation on some lesions. (Photo contributor: Lawrence B. Stack, MD.)

# PORPHYRIA CUTANEA TARDA

## Clinical Summary

Porphyrias are associated with enzymatic defects in heme biosynthesis. Porphyria cutanea tarda (PCT) is the most common type. Patients have photosensitivity, skin fragility, and characteristic lesions on sun-exposed sites (most common on the dorsal hands and forearms). Typically, the skin is easily traumatized with blisters, erosions, superficial scars, milia, and hypertrichosis. PCT does not cause the life-threatening neurologic attacks associated with the acute porphyrias.

PCT may be induced by ethanol, estrogens, oral contraceptives, infections (Hepatitis C and HIV), polychlorinated hydrocarbons, dialysis, and iron overload. Pseudoporphyria, clinically indistinguishable from PCT, is associated with dialysis and certain medications (NSAIDs, furosemide, hydrochlorothiazides, and amiodarone, among others).

## Management and Disposition

Laboratory examination may begin in the ED with blood chemistries, porphyrin studies (24-hour urinary porphyrins can be ordered), and referral to a dermatologist. Long-term treatment includes phlebotomy and antimalarials (which can induce fatal hepatotoxicity in PCT patients). Discontinue any medications that might initiate PCT or pseudoporphyria.

## Pearls

1. Consider PCT in a patient with fragile skin with erosions and scarring, but only on sun-exposed skin.
2. PCT does not have acute attacks of abdominal pain, neurologic deficits, psychosis, or autonomic dysfunction. Variegate porphyria, common in South Africans with Dutch ancestry, does present with the typical skin lesions and acute attacks.
3. Examination of the urine with a Wood lamp may reveal orange-red fluorescence.
4. Always consider medications (both prescribed and over-the-counter medications) when considering PCT.

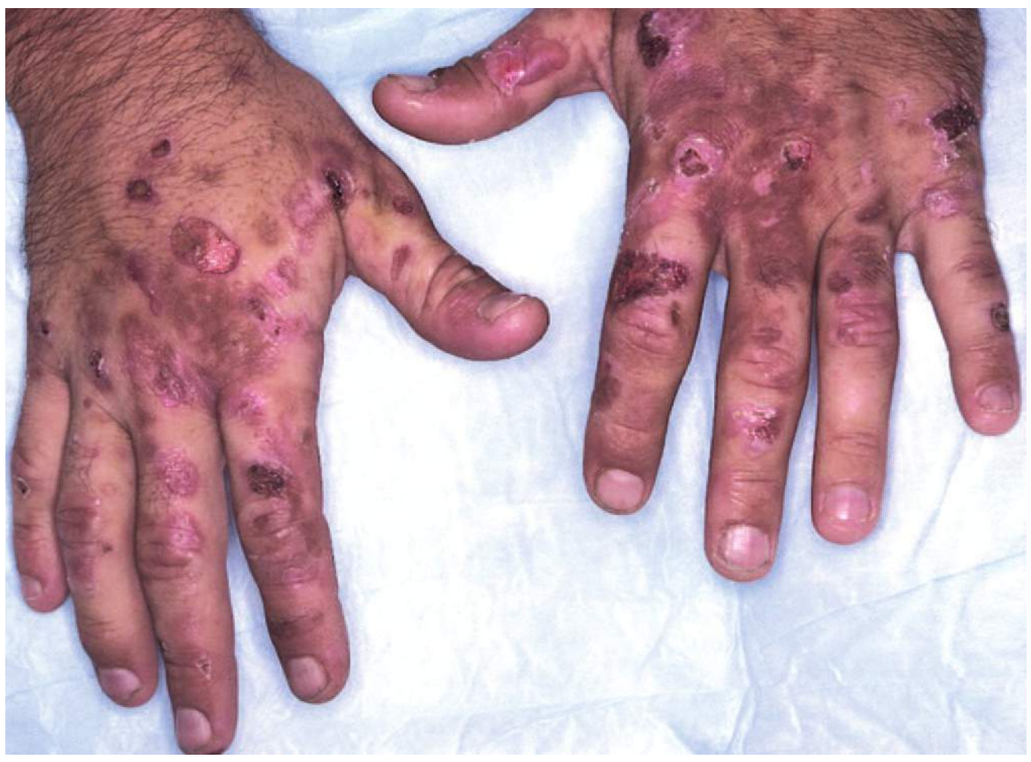


FIGURE 13.120 ■ **Porphyria Cutanea Tarda.** Blisters and erosions of PCT. (Photo contributor: Selim Suner, MD, MS.)

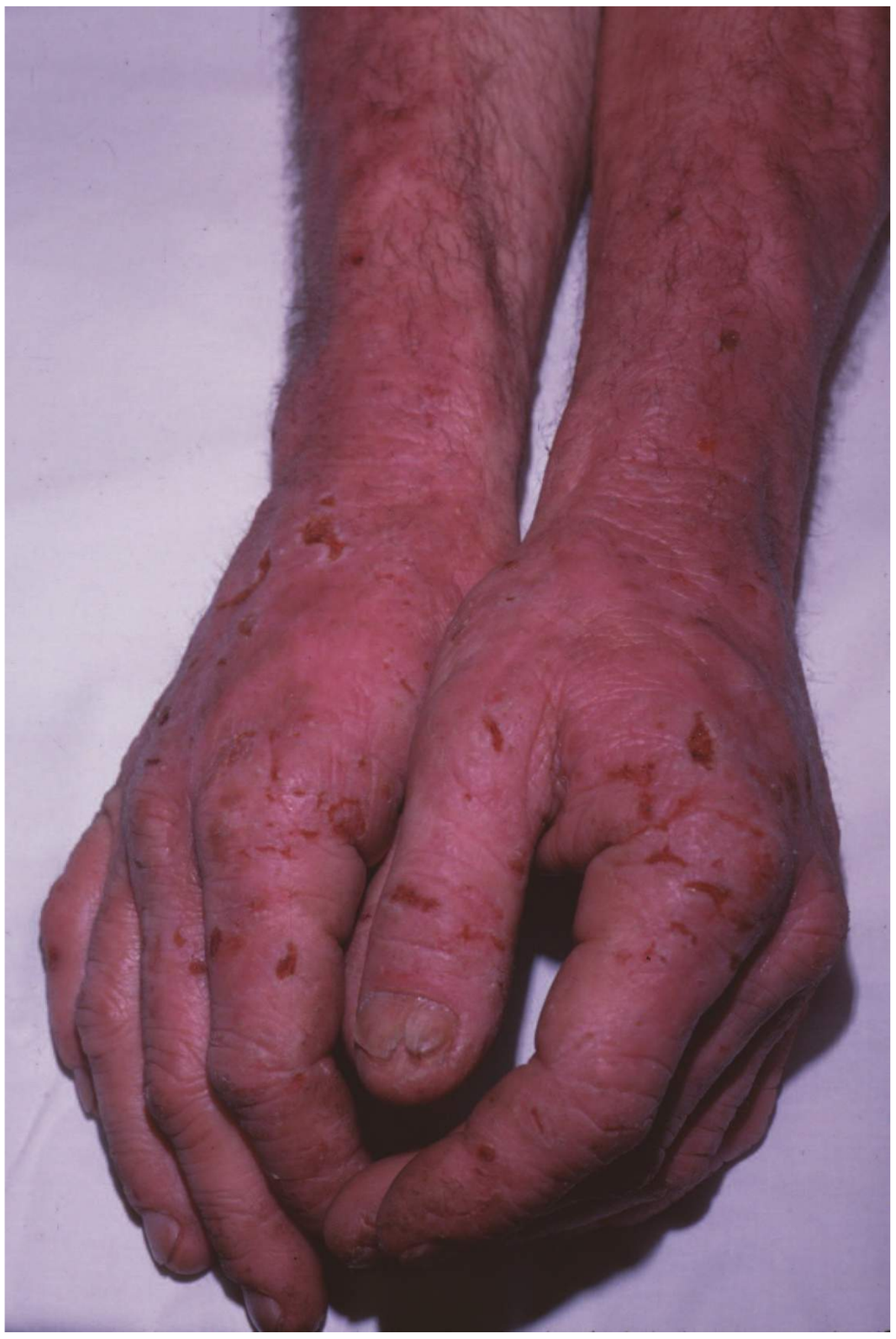


FIGURE 13.121 ■ **Porphyria Cutanea Tarda.** The easily traumatized skin and erosions of PCT. (Photo contributor: Alan B. Storrow, MD.)

# SQUAMOUS CELL CARCINOMA

## Clinical Summary

Squamous cell carcinoma (SCC) is the second most common skin cancer. It is associated with a higher incidence in males, increased age, chronic sun exposure, immunosuppressive treatment, and chronic burns or scars. Initially, SCC presents with erythematous macules that develop into firm papules and plaques. Most are located on the sun-exposed sites of the head, neck, and upper extremities.

## Management and Disposition

After ensuring a secondary infection is not present, prompt outpatient dermatologic referral is indicated.

## Pearls

1. There is a higher risk of metastasis with SCC versus basal cell carcinoma (although still very low). As with basal cell carcinoma, metastatic potential is higher on the ears, periocular area, nose, and lips—do not miss the opportunity to refer patients with questionable lesions!
2. Any persistent nodule, plaque, or ulcer should be examined for SCC.

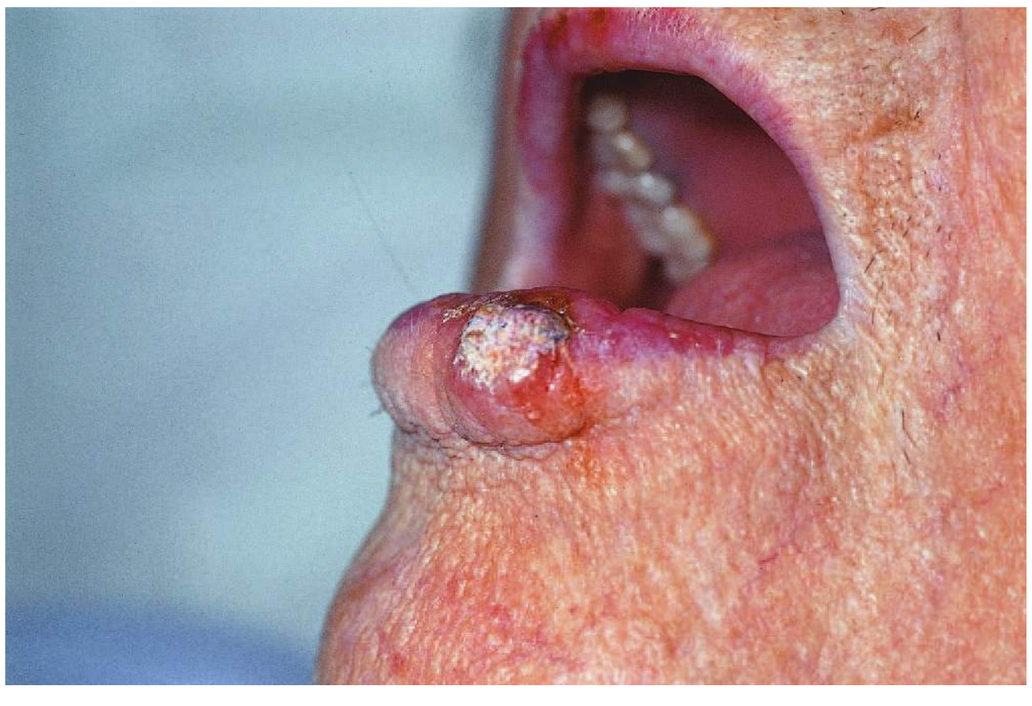


FIGURE 13.122 ■ **Squamous Cell Carcinoma.** The lower lip is exposed to more sunlight and is therefore involved more frequently. (Photo contributor: J. Matthew Hardin, MD.)

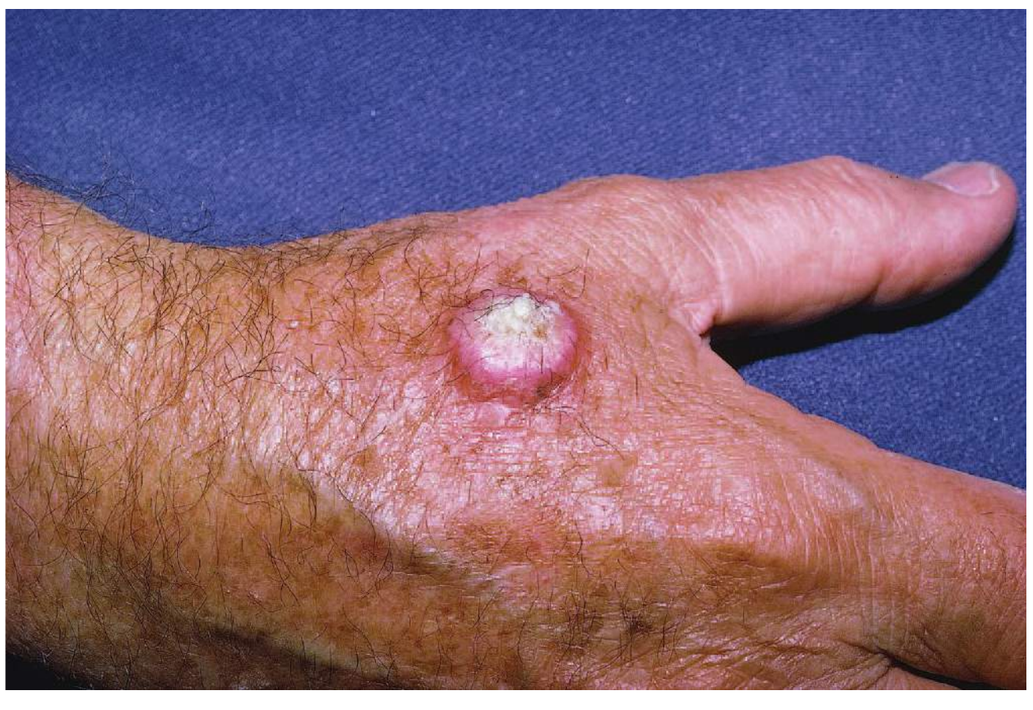


FIGURE 13.124 ■ **Squamous Cell Carcinoma.** This nodule has a central keratogenous core. (Photo contributor: J. Matthew Hardin, MD.)

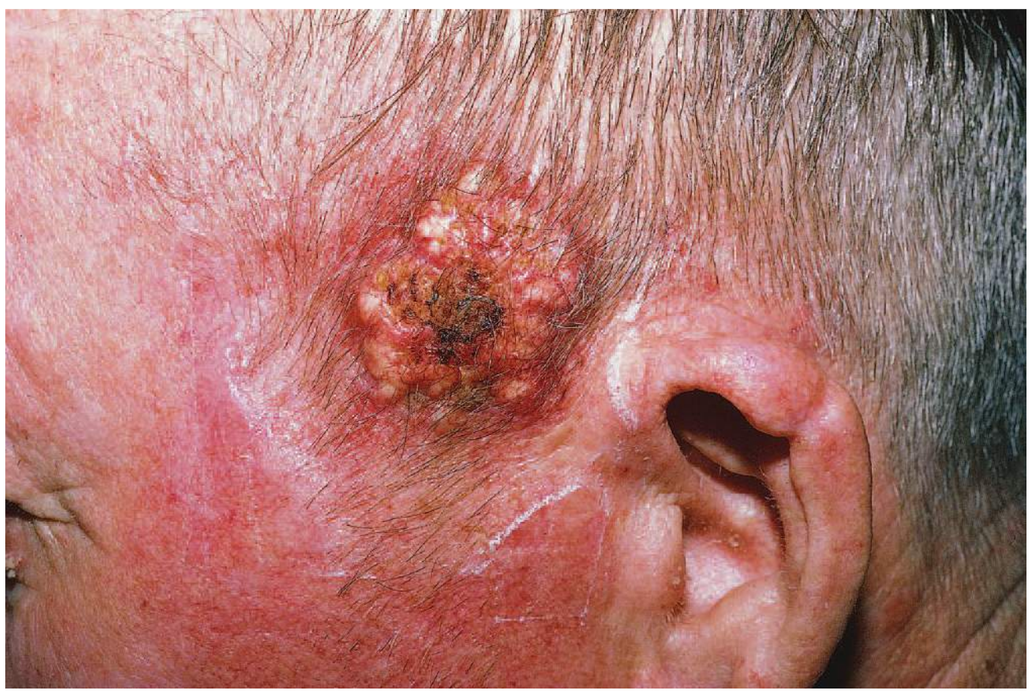


FIGURE 13.123 ■ **Squamous Cell Carcinoma.** This nodule with central ulceration slowly developed over 1 year. (Photo contributor: J. Matthew Hardin, MD.)

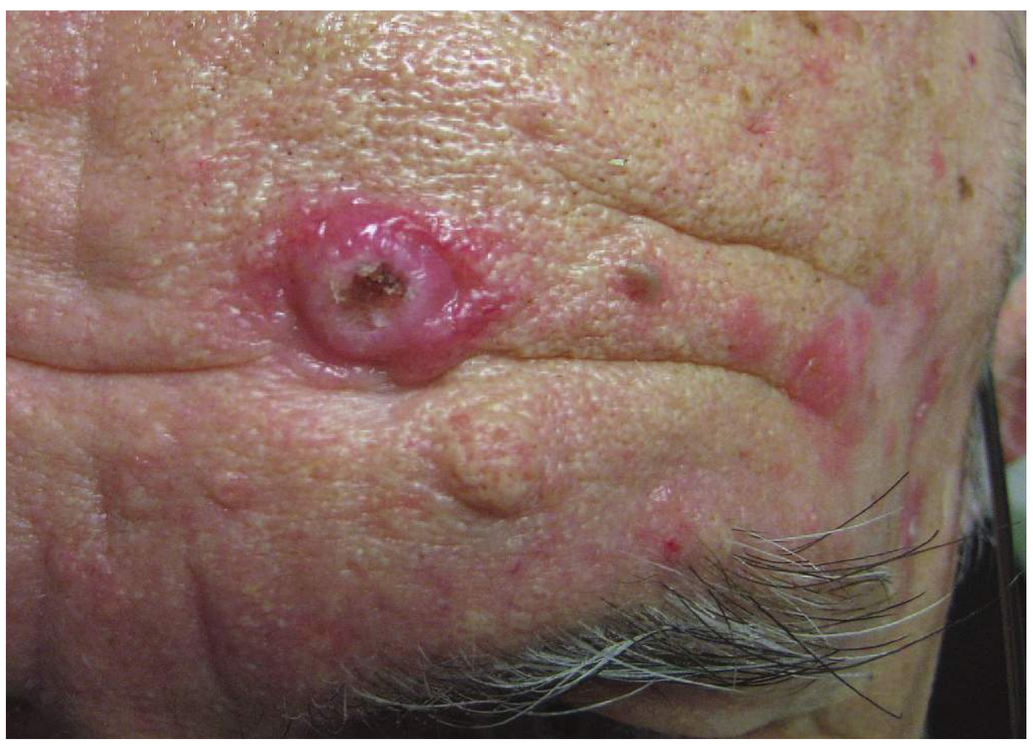


FIGURE 13.125 ■ **Squamous Cell Carcinoma.** Ulcerated nodule on the forehead. (Photo contributor: J. Matthew Hardin, MD.)

# BASAL CELL CARCINOMA

## Clinical Summary

Basal cell carcinoma (BCC) is the most common nonmelanoma skin cancer. BCC can present anywhere but is most common on sun-exposed areas. The typical lesion begins as a pearly papule with telangiectasias (nodular BCC). The lesion may ulcerate and bleed. Other forms of BCC include superficial BCC (pink, scaly plaque with pearly border), pigmented BCC (appears as a nodular or superficial BCC with dark brown to black center), and morpheaform BCC (appears as a rapidly expanding scar).

## Management and Disposition

After ensuring a secondary infection is not present, prompt outpatient dermatologic referral is indicated.

## Pearls

1. The metastatic potential of BCC is very low (0.1%) but higher on the ears, periocular area, nose and lips. Do not miss the opportunity to refer patients with questionable lesions.
2. BCC is not rare in darker skinned persons (a common misperception).

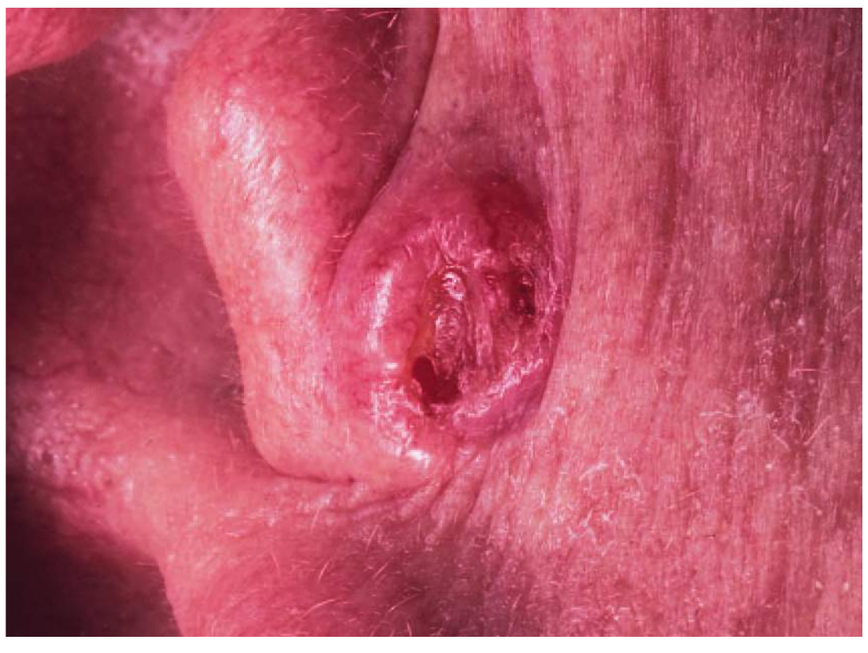


FIGURE 13.126 ■ **Basal Cell Carcinoma.** Nodular basal cell carcinoma consists of a firm, centrally ulcerated (rodent ulcer) nodule with a raised, pearly, telangiectatic border. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)

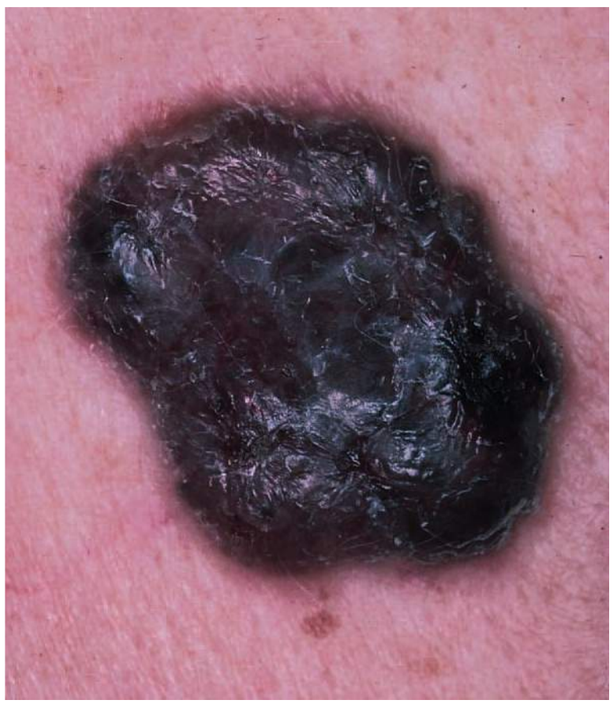


FIGURE 13.127 ■ **Pigmented Basal Cell Carcinoma.** Translucent, brownish-black, flat papule, easily confused with melanoma. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)

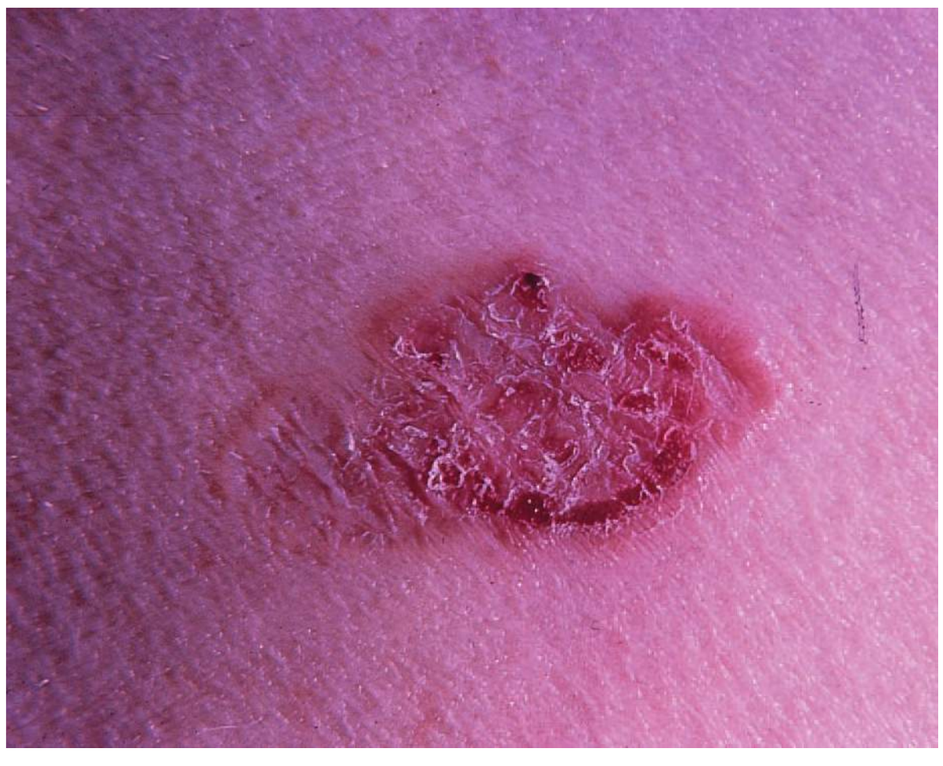


FIGURE 13.128 ■ **Basal Cell Carcinoma.** A superficial basal cell carcinoma is frequently disregarded. Note the flat, erythematous, scaly plaque with its elevated, irregular border. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)

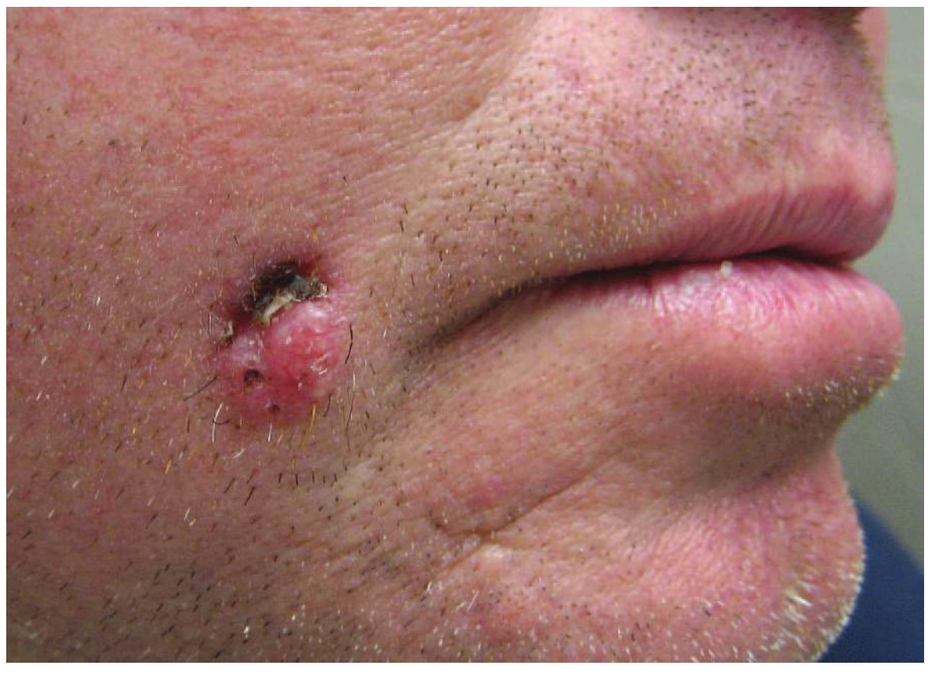


FIGURE 13.129 ■ **Basal Cell Carcinoma.** This chronic, erythematous papule and crusted ulcer appeared 2 years prior to presentation. (Photo contributor: J. Matthew Hardin, MD.)

## Clinical Summary

Melanoma is a potentially fatal cutaneous tumor derived from epidermal melanocytes. Any age can be affected, but the peak incidence is 20- to 45-year-old patients (much younger than basal cell or SCCs). The most significant risk factor is a primary relative with melanoma. Evaluation of any pigmented lesion should include the ABCDE rule (A for asymmetry, B for irregular borders, C for color variegation, D for diameter greater than 6 mm, and E for elevation and evolving). Any lesion with these characteristics is considered suspicious for melanoma.

## Management and Disposition

Prompt outpatient dermatologic referral is indicated. Simply acknowledging suspicious lesions seen during emergency care may encourage earlier follow-up.

## Pearls

1. The palms, soles, and subungual sites are most common with dark-skinned individuals.
2. Melanoma can occur in sites not exposed to the sun (genitalia/buttocks). In the course of an exam, note worrisome lesions and refer.
3. Any growing pigmented or nonpigmented lesion should be referred to dermatology.
4. Most patients will not have new moles after 35 years old. A new mole in this setting should be evaluated by dermatology.

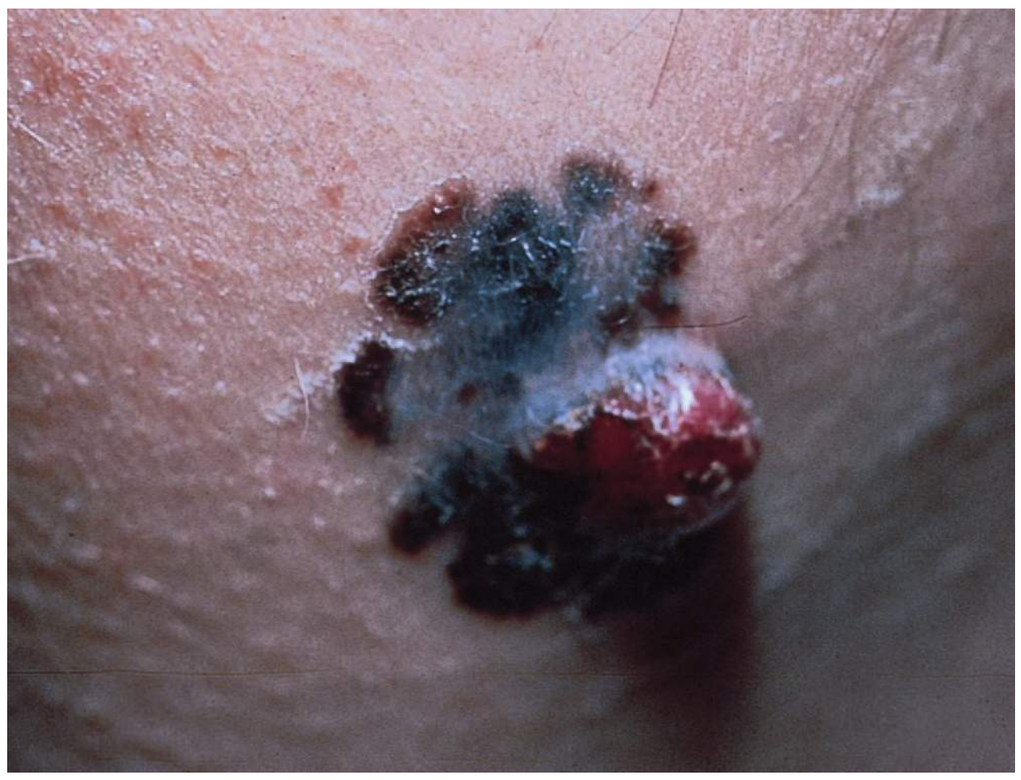


FIGURE 13.131 ■ **Nodular Melanoma.** This melanoma has progressed to an exophytic tumor, which was deeply invasive histopathologically. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)

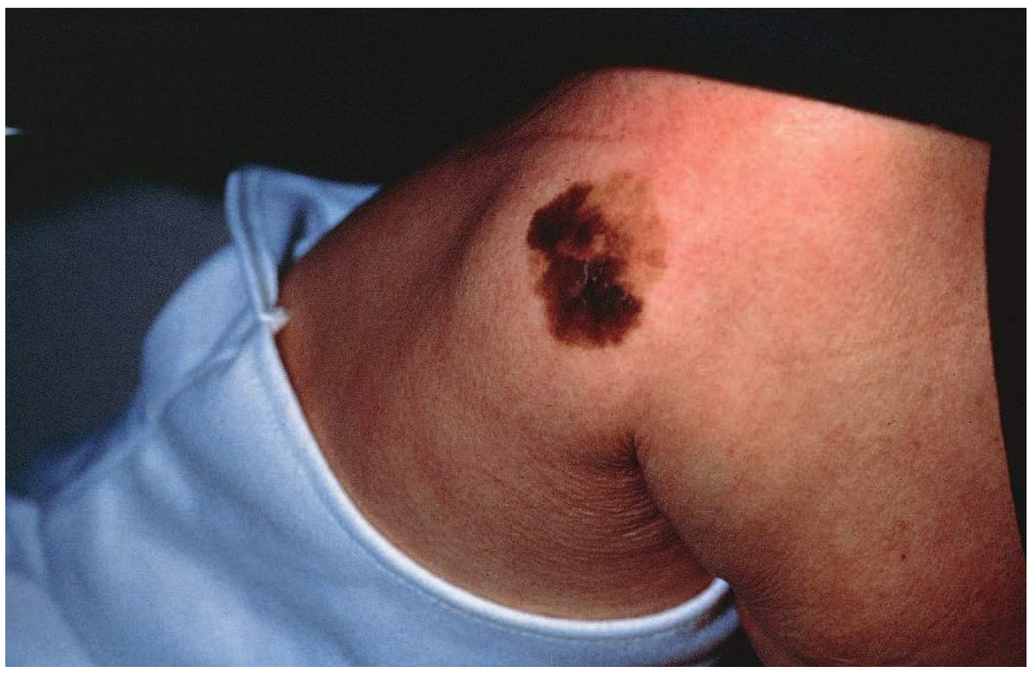


FIGURE 13.130 ■ **Melanoma.** This lesion demonstrates asymmetry, color variegation, and a diameter greater than 6 mm. (Photo contributor: J. Matthew Hardin, MD.)

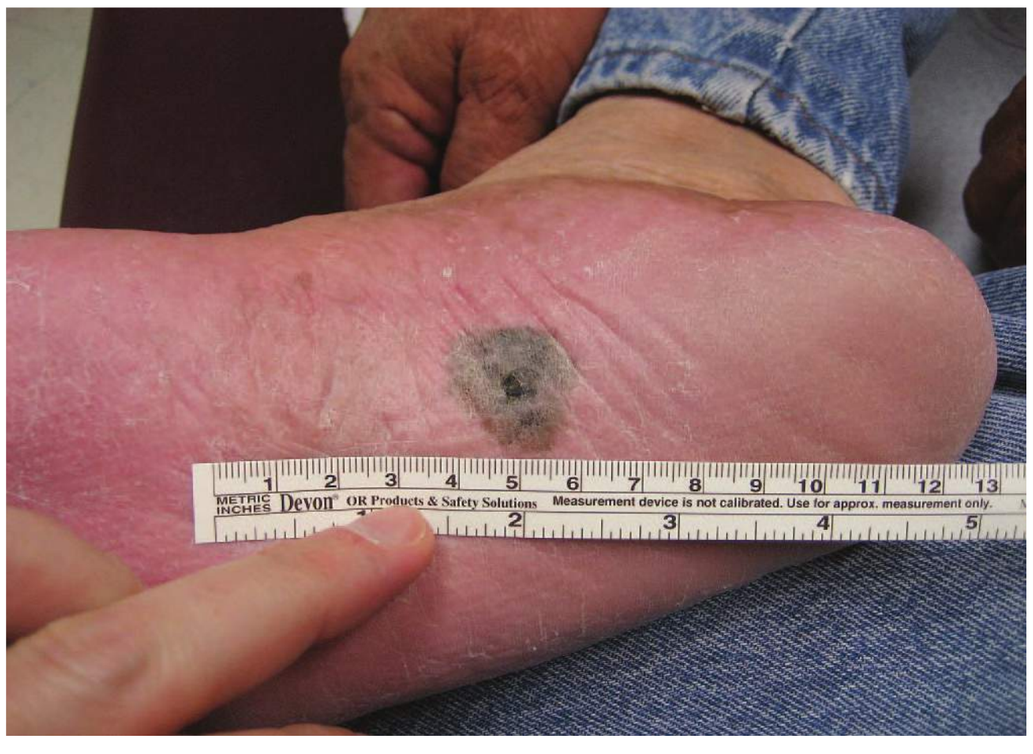



FIGURE 13.132 ■ **Melanoma.** Slow-growing, pigmented plaque on the arch of the foot. (Photo contributor: J. Matthew Hardin, MD.)

# LIP LICKER'S DERMATITIS

## Clinical Summary

Lip licker's dermatitis is caused by repeated exposure of the vermillion border and the cutaneous lips to saliva. Children and young adults are more commonly affected but older adults, including the elderly, can be afflicted. Irritants in topical lip preparations can initiate this process or can worsen a presentation. Winter is the most common time to see lip licker's dermatitis due to the constant exposure to cold air and low humidity but presentations occur throughout the year.

Lip licker's dermatitis typically starts with red papules that slowly progress into an erythematous, thin plaque. As the cycle of licking continues, the area becomes confluent circumferentially around the vermillion border and the cutaneous lips. The skin begins to break down with fissuring and scaling and the area can become very painful. The rash does not extend beyond the tongue's reach on the cutaneous lips.

## Management and Disposition

Treatment is focused on identifying and preventing repetitive lip licking. Many children are not aware that they are contributing to this rash and care should be taken to not blame them for this predicament (especially younger children). Use of 100% petrolatum (Vaseline) applied all day (any time the area becomes dry or the patient desires to lick) will help. Stop the use of any other topical products. The addition of 1% hydrocortisone ointment may hasten the resolution.

## Pearls

1. Never approach a young child blaming them for their condition; it is best to use indirect questioning to make them aware of the cause ("Do you think you ever lick your lips?").
2. The use of over-the-counter lip preparations or flavored lip products may cause or exacerbate the condition.
3. Lip licker's dermatitis never ends until the lip licking behavior ends!

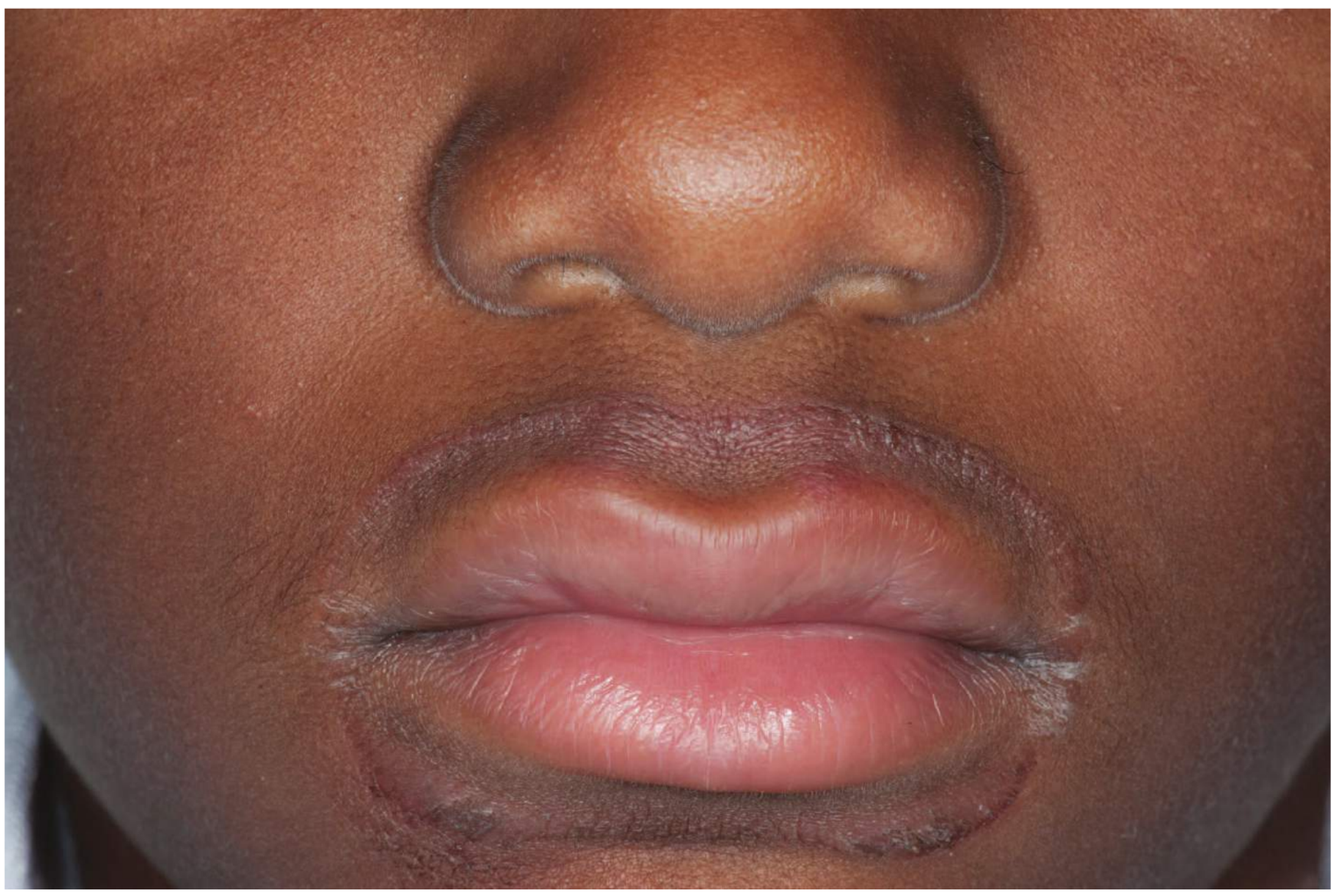


FIGURE 13.133 ■ **Lip Licker's Dermatitis.** Circumoral dryness and pain. (Photo contributor Aubrey Mowery, MPH, CHES.)

PART 2

# Specialty Areas