Cutaneous eruptions can be manifestations of primary dermatologic disease or signal underlying systemic illness. In addition to medical and family history, three factors are particularly important: onset and evolution of the skin problem, associated symptoms, and prior treatment.

With adequate lighting available, the physical examination is performed with the patient undressed. In general, the dermatologic evaluation includes a thorough evaluation of the scalp, mouth, and nails. Although the examination depends largely on inspection of the skin, palpation helps assess the texture, consistency, and tenderness of the lesions.

Skin lesions are divided into growths and rashes. Growth is subdivided into epidermal, pigmented, and dermal or subcutaneous proliferative processes. Rashes are divided into two groups, depending on whether the epidermis is involved. Lesions and rashes with epidermal involvement are described as being eczematous, scaling, vesicular, papular, pustular, or hypopigmented. Rashes without epidermal involvement are described as being erythematous, purpuric, or indurated.

The diagnosis is aided by the configuration of the lesions and distribution on the body’s surface. On occasion, a configuration is specific for a disease; however, the morphologic appearance of the primary lesion is usually given more diagnostic weight (Table 120-1). Finally, many skin diseases have preferential areas of involvement, so the location of the eruption may aid in diagnosis. Several serious dermatologic disorders, such as Stevens-Johnson syndrome, may be manifested initially with lesions on mucous membranes only, thus making an examination of the mucous membranes an important part of a dermatologic evaluation.

Emergency department (ED) management focuses on stabilization (e.g., toxic epidermal necrolysis), symptom control (e.g., acute urticaria and pruritus), flare control (e.g., eczema), and treatment (e.g., skin infections or infestations). A variety of treatment options are available for dermatologic problems, including topical, oral, and intravenous preparations. Vehicles for topical preparations include soaks, creams, ointments, foams, gels, and lotions; use of the proper vehicle is a key component of successful treatment.

Aluminum acetate (Burow’s solution), aluminum sulfate—calcium acetate (Domeboro), or oatmeal soaks may provide symptomatic relief of pruritus in patients with conditions with crust or weeping. Saline wet soaks provide a moist environment and may be used to promote epithelialization of denuded areas; wet-to-dry dressings can be used for lesions requiring débridement. When wet occlusive dressings are applied, the normal skin is protected from maceration by the creation of a lipophilic barrier with an ointment. If a wet dressing is allowed to dry, it can stick to a wound and thus negate its benefit when it is removed.

Creams are a mixture of oil, water, and preservatives that penetrate the skin on application. Creams may cause drying of the skin and are best used for acute conditions. Lotions are a mixture of powder and water and may require shaking before application. Lotions may be used on any body surface, especially hair-bearing areas. Ointments are emulsions of water and oil that do not penetrate the skin on application; ointments are not usually applied to hairy areas or in natural folds. Ointments are especially useful for treatment of dry lesions. Occlusive dressings are used to increase potency by promoting absorption. Gels and foams liquefy when they are applied to the skin. They are frequently used for scalp lesions as they penetrate the hair barrier easier than other formulations do. Whenever a potent topical medication is prescribed, patients and caregivers are advised not to use their fingers for application; a cotton-tipped applicator or a plastic spatula is recommended instead.

Topical corticosteroids are commonly used for a variety of inflammatory skin conditions. They induce topical vasoconstriction, depress local immunity, and cause skin thinning with prolonged use. Systemic absorption and side effects are possible. A variety of formulations are available (Table 120-2). The potency of steroids depends on the specific drug, its concentration, and the method of delivery (i.e., whether cream or ointment is used, how often it is applied, whether an occlusive dressing is placed). It is wise for clinicians to familiarize themselves with one or two high-, medium-, and low-potency steroids to limit possible errors. So-called mega potency topical steroids are generally not initiated without consulting a dermatologist. The lowest potency medication available should be used around natural orifices to minimize systemic absorption.

### SCALES, PLAQUES, AND PATCHES

### Fungal Infection

#### Principles of Disease

The dermatophytoses are superficial fungal infections that are limited to the skin. A variety of lesions may occur, but the most common are scaling, erythematous papules, plaques, and patches, which often have a serpiginous or wormlike border. Dermatophytes generally grow best in excessive heat and moisture and grow only in the keratin or outer layer of the skin, nails, and hair. Keratin tends to accumulate in body folds, such as between toes and in the inguinal area, the axilla, and the inframammary areas. With the exception of tinea capitis, dermatophyte infections are not markedly contagious.
Any eruption thought to be a dermatophyte infection can be examined under the microscope in a potassium hydroxide (KOH) preparation. The specimen is examined for the characteristic branching hyphae of the dermatophytes or the short, thick hyphae and clustered spores of tinea versicolor. Affected hair, nail, or scales may be cultured on Sabouraud agar incubated at room temperature for 2 or 3 weeks.

**Tinea Capitis**

**Clinical Features.** Tinea capitis is a fungal infection of the scalp. Although it is primarily regarded as a disease of preschool children, tinea capitis is increasingly recognized in adults, infants, and neonates. It is more common among African Americans, although the reasons for this are unknown. Nascomial transmission of dermatophyte infections, such as *Trichophyton tonsurans*, has also been reported. Recent outbreaks in the United States caused by *T. tonsurans* differ from epidemics of the 1940s and 1950s caused by *Microsporum audouinii* in that many patients have seborrheic dermatitis, bacterial superinfection exists, and scarring alopecia. If intravenous clindamycin is used, the dose is 600 to 1000 mg/day, taken as a single dose with a fat-containing food for a minimum of 6 weeks or 2 weeks after clinical resolution of inflammation. Higher doses may be needed. Griseofulvin can cause sun sensitivity. If treatment is initiated in the ED, refer the patient for a monthly follow-up evaluation. Alternative therapy includes fluconazole 200 mg/day (adults) or 3 to 5 mg/kg/day (children), itraconazole 200 mg daily (adults) or 3 to 5 mg/kg/day (children) for 4 to 6 weeks, oral terbinafine 3 to 6 mg/kg/day (children) or 250 mg/day (adults) for 4 to 6 weeks, and terbinafine cream once a day for 8 weeks. *T. tonsurans* species appear to be more sensitive to newer treatment modalities and require a shorter course of treatment than for *Microsporum* species. Selenium sulfide shampoo, 250 mg twice weekly, decreases shedding of spores. Family members should be evaluated.

**Kerion**

A kerion is a fungal infection affecting hair follicles that is characterized by intense inflammation with erythematous, boggy lesions often with frank pus. The inflammation is generally uniform and does not display satellite lesions. It usually affects the scalp and is more common in children and in African Americans. Local alopecia and scarring can ensue. Accurate differentiation of lesions with and without superinfection can be challenging, and a bacterial culture may be useful when there is doubt. Kerions tend to be less tense and less tender than bacterial abscesses; however, with superinfection or existing drainage, the distinction becomes less pronounced. A toothbrush, Papanicolau smear cytology brush, or moistened cotton swab is helpful for quick, painless sampling of large areas of the scalp.

Kerions are treated the same as tinea capitis, with the addition of prednisone 1 mg/kg/day for 1 or 2 weeks to help decrease the inflammatory reaction and subsequent alopecia and scarring. If bacterial superinfection exists, an antibiotic is added. Antibiotic options include oral cephalixin, 500 mg every 6 hours for adults or 50 mg/kg/day divided in four doses not to exceed 500 mg per dose for children, for 1 week; dicloxacillin 100 mg twice a day; and clindamycin 300 mg orally every 6 hours for adults or 8 to 25 mg/kg/day as palmitate in three or four divided doses for children. If intravenous clindamycin is used, the dose is 600 to 900 mg every 6 to 8 hours for adults or 40 mg/kg/day divided three or four times a day for children not to exceed 600 mg per dose. Clindamycin is recommended when community-acquired methicillin-resistant *Staphylococcus aureus* (CA-MRSA) is a concern, that is, in those cases in which purulent drainage is present. Surgical drainage of kerions is not helpful and should be avoided.

**Tinea Corporis**

**Clinical Features.** Tinea corporis is the classic “ringworm” infection. Its characteristic presentation is a sharply margined, annular lesion with raised or vesicular margins and central clearing (Fig. 120-1). Lesions may be single or multiple; multiple lesions are occasionally concentric. Tinea cruris, which involves the groin, is similar in appearance and may also include the perineum, thighs, and buttocks, but the scrotum is characteristically spared.
Differential Considerations. The differential diagnosis of tinea cruris includes granuloma annulare, psoriasis, intertrigo with secondary candidiasis, and erythrasma.

Management. Infections of the body, groin, and extremities usually respond to topical measures alone. A number of effective topical antifungal agents are available, including clotrimazole (Lotrimin), haloprogin (Halotex), miconazole (Micatin), tolnaftate (Tinactin), terbinafine, naftifine, and griseofulvin 1%. Two or three daily applications of the cream form of any of these preparations result in healing of most superficial lesions in 1 to 3 weeks. A secondary bacterial infection may occur. The vesicular pustular form of tinea pedis should be considered when vesicles and pustules on the instep are noted. The differential diagnosis includes contact dermatitis and dyshidrotic eczema. A KOH preparation is helpful to differentiate between these processes. Interdigital lesions may cause minimal symptoms and serve as a portal of entry for bacterial cellulitis. Treatment options include terbinafine 1% cream twice daily for 2 to 4 weeks; miconazole 2% cream, powder, or spray twice daily for 2 to 4 weeks; and clotrimazole 1% cream, solution, or lotion twice daily for 2 to 4 weeks. For severe disease or if topical treatment has failed, use terbinafine 250 mg orally daily for 2 weeks, fluconazole 150 mg orally weekly for 2 to 4 weeks, or griseofulvin 500 mg orally daily for 2 weeks.

**Table 120-2 Potency of Topical Steroids**

<table>
<thead>
<tr>
<th>BRAND NAME</th>
<th>GENERIC NAME</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elocon cream, 0.1%</td>
<td>Mometasone furoate</td>
</tr>
<tr>
<td>Kenalog cream or spray, 0.1%</td>
<td>Triamcinolone acetonide</td>
</tr>
<tr>
<td>Synalar ointment, 0.03%</td>
<td>Fluocinolone acetonide</td>
</tr>
<tr>
<td>Westcot ointment, 0.2%</td>
<td>Hydrocortisone valerate</td>
</tr>
<tr>
<td>Capex shampoo, 0.01%</td>
<td>Fluocinolone acetonide</td>
</tr>
<tr>
<td>Cordran cream, lotion, or tape, 0.05%</td>
<td>Flurandrenolide</td>
</tr>
<tr>
<td>Cutivate cream or lotion, 0.05%</td>
<td>Fluticasone propionate</td>
</tr>
<tr>
<td>DermAtop cream, 0.1%</td>
<td>Prednicarbate</td>
</tr>
<tr>
<td>DesOwen lotion, 0.05%</td>
<td>Desonide</td>
</tr>
<tr>
<td>Locoid cream, lotion, ointment, or solution, 0.1%</td>
<td>Hydrocortisone</td>
</tr>
<tr>
<td>Pandel cream, 0.1%</td>
<td>Hydrocortisone</td>
</tr>
<tr>
<td>Synalar cream, 0.03%, 0.01%</td>
<td>Fluocinolone acetonide</td>
</tr>
<tr>
<td>Westcot cream, 0.2%</td>
<td>Hydrocortisone valerate</td>
</tr>
<tr>
<td>Aclovate cream or ointment, 0.05%</td>
<td>Alclometasone dipropionate</td>
</tr>
<tr>
<td>Derma-Smoothe/FS oil, 0.01%</td>
<td>Fluocinolone acetonide</td>
</tr>
<tr>
<td>Desonate gel, 0.05%</td>
<td>Desonide</td>
</tr>
<tr>
<td>Synalar cream or solution, 0.01%</td>
<td>Fluocinolone acetonide</td>
</tr>
<tr>
<td>Verdeso foam, 0.05%</td>
<td>Desonide</td>
</tr>
<tr>
<td>Cutivate ointment, 0.005%</td>
<td>Fluticasone propionate</td>
</tr>
<tr>
<td>Lidex-E cream, 0.05%</td>
<td>Flucinonide</td>
</tr>
<tr>
<td>Luxiq foam, 0.12%</td>
<td>Betamethasone valerate</td>
</tr>
<tr>
<td>Topicort LP cream, 0.05%</td>
<td>Desoximetasone</td>
</tr>
<tr>
<td>Class 5—Lower Midstrength</td>
<td></td>
</tr>
<tr>
<td>Class 6—Mild</td>
<td></td>
</tr>
<tr>
<td>Class 7—Least Potent</td>
<td></td>
</tr>
<tr>
<td>Class 4—Midstrength</td>
<td></td>
</tr>
</tbody>
</table>

and recurrence is considered the rule rather than the exception. Monthly prophylaxis with propylene glycol and water, selenium shampoo, or azole creams can help prevent recurrences. Pigmentation may not return to normal for months. Propylene glycol is inexpensive and effective.

Tinea Unguium (Onychomycosis)

Clinical Features. Tinea unguium results in nails that are opaque, thickened, cracked, and crumbled. Subungual debris is present, and the nail may contain yellowish longitudinal streaks. The nail of the great toe is most commonly involved. Involvement of all of the nails of the hands and feet is rare.

Management. Topical therapy of the nails alone rarely results in a cure because penetration into the nail keratin is poor. Fingernails typically respond more rapidly to therapy than toenails do. Oral griseofulvin and ketoconazole require prolonged courses, with high relapse rates and numerous side effects. Newer agents such as itraconazole, fluconazole, and terbinafine are safer and more effective. They also offer shorter treatment periods, thus improving compliance. The infection may be resistant to this regimen as well, however, and surgical removal of the nail is occasionally required. The choice of specific agent may be best left to a consultant or patient's primary physician, given the long course of treatment and frequency of treatment failure. If therapy is started in the ED, reliable follow-up is needed. Griseofulvin ultramicrosize 375 mg twice daily or ketoconazole 400 mg daily is given for 4 to 6 months. Newer agents are preferred by dermatologists: terbinafine 250 mg/day for 6 to 12 weeks (a longer course is given for toenail involvement) is considered first-line therapy. Recurrence is common.

Candidiasis

Perspective

Infection by *Candida albicans* occurs in patients of all ages. Many conditions predispose to infection, including acquired immunodeficiency syndrome (AIDS), pregnancy, obesity, malnutrition, malignant disease, and diabetes and other endocrine imbalances. Patients treated with corticosteroids, immunosuppressive agents, and antibiotics are also prone to cutaneous fungal infections.

Oral Thrush

Clinical Features. Oral thrush is the most common clinical expression of *Candida* infection. Thrush is common in newborns, with one third being affected by the first week of life. It appears as patches of white or gray friable material covering an erythematous base on the buccal mucosa, gingiva, tongue, palate, or tonsils. Fissures or crust at the corners of the mouth may be present. The differential diagnosis of oral thrush includes lichen planus (which unlike *C. albicans* is not easily scraped off). Oral mucous membrane infection with *C. albicans* is an AIDS-defining illness. If the patient does not use dentures and has not taken antibiotics recently, underlying immunosuppression should be considered.

Management. Treatment of oral thrush involves painting of the mouth with 1 mL to each side of the mouth of oral nystatin suspension (100,000 units/mL) four times a day for infants or 4 to 6 mL four times a day swish and swallow for older children and adults. Treatment is continued for 5 to 7 days after the lesions disappear. A preferable treatment option for adults is clotrimazole troches 10 mg dissolved in the mouth two to five times daily. If topical therapy is not effective or in cases of chronic candidiasis, oral fluconazole 3 mg/kg, 100 to 200 mg/day for 1 to 2 weeks, may be prescribed. If that fails, treatment options include posaconazole suspension, 400 mg twice daily for 3 days, then 400 mg daily for preparation reveals short hyphae mixed with spores (“chopped spaghetti and meatballs”).

Management. Tinea versicolor may be treated with 2.5% selenium sulfide shampoo, imidazole creams, ketoconazole cream or foam, or oral ketoconazole as a single 400-mg dose or 200 mg daily for 5 to 10 days. Fluconazole 150 to 300 mg weekly for 2 to 4 weeks may also be used. Recurrence rates vary from 15 to 50%,
Cutaneous Candidiasis

Clinical Features. Cutaneous candidiasis favors the moisture and maceration of the intertriginous areas—the interdigital web spaces, groin, axilla, and intergluteal and inframammary folds. Lesions appear as moist, bright red macules rimmed with a col-larette of scale, which represents the pustule roof with scalloped borders. Small satellite papules or pustules are just peripheral to the main body of the rash. These satellite lesions are the most typical indicators of a Candida infection. Intertriginous lesions are prone to bacterial superinfection.

Candidal onychia and paronychia are conditions that occur in those whose hands are frequently immersed in hot water. These infections also occur with thumb sucking by children who have thrush. The paronychial area becomes red and swollen; the nails are thick and brittle, with transverse ridging. Destruction of the nail plate may occur.

Differential Considerations and Diagnostic Strategies. The differential diagnosis of cutaneous candidiasis includes contact dermatitis, tinea cruris, intertrigo, herpes simplex such as herpetic whitlow, and folliculitis. Candidiasis, however, is less sharply demarcated than tinea cruris and brighter red than intertrigo. A KOH preparation of a specimen taken from a pustule and roof of the lesion will reveal hyphae and pseudohyphae.

Management. Treatment of intertriginous lesions requires the removal of excessive moisture and maceration. Lesions should be exposed to circulating air from a fan several times a day. Inflammatory lesions are either soaked in or covered with compresses of cool water or Burow’s solution. Topical imidazole creams, such as clotrimazole and miconazole, are applied sparingly to affected areas. Prescription creams, such as econazole, ketoconazole, and sulconazole, are also effective.

Protection of the hands from water is an integral part of the treatment of candidal paronychia. Instruct patients to avoid prolonged immersion and to use gloves with cotton liners to prevent contact with water. Nystatin or clotrimazole cream is applied frequently to the nail folds for 6 to 8 weeks.

A search for an underlying immunocompromising condition is recommended in patients with chronic, recurrent candidiasis. Eczematous dermatitis of the nail folds can lead to the development of chronic paronychia.

Diaper Dermatitis

Clinical Features

Diaper dermatitis is a common disorder that is exacerbated by heat, moisture, friction, and the presence of urine and fecal material. It can affect anyone wearing a diaper. Lesions begin as erythematous plaques in the genital, perianal, gluteal, and inguinal areas. More severe involvement results in moist, eroded lesions that may extend beyond the primary areas of appearance.

Lesions infected with Candida are moist, red patches with well-demarcated borders. Papular or pustular satellite lesions are also present.

Diaper dermatitis may reflect the presence of atopic or seborrheic dermatitis in the infant. The presence of lesions elsewhere on the body, particularly on the face in cases of atopic dermatitis or the scalp in cases of seborrhea, alerts the physician to these possibilities. The existence of diaper dermatitis as a true allergic contact dermatitis is rare. Candidal superinfection is common and is manifested with erythematous patches and satellite lesions. Early bacterial cellulitis must also be considered.

Management

Treatment consists primarily of altering the physical environment in which diaper dermatitis thrives. Advise parents to provide continuous air exposure of the area; if this is not possible, diapers should be changed frequently and topical barrier ointments, such as zinc oxide, applied. Treatment includes nystatin or other topical antifungal creams. If exudative lesions are present, treatment with topical, cool, wet compresses of saline or Burow’s solution is indicated for 2 or 3 days. Severe contact or seborrheic dermatitis may require the addition of a short course of a low-potency topical corticosteroid, such as 1% hydrocortisone in a cream base. Particularly severe, recurrent, or not improving diaper rash in infants raises the possibility of an immunodeficiency syndrome, diabetes, or caregiver neglect.

Pityriasis Rosea

Pityriasis rosea is a mild skin eruption predominantly found in children and young adults. The lesions are multiple pink or pigmented oval papules or plaques 1 to 2 cm in diameter on the trunk and proximal extremities. Mild scaling may be present. The lesions are parallel to the ribs, forming a Christmas tree–like distribution on the trunk. Oral lesions are rare. In children, papular or vesicular variants of the disease may occur.

In half the cases, the generalized eruption is preceded by the appearance of a herald patch. This is a larger lesion, 2 to 6 cm in diameter, that resembles the smaller lesions in other respects. The eruption is usually asymptomatic, although pruritus may be present.

Pityriasis rosea is self-limited, resolving in 8 to 12 weeks. Its cause is unknown, although a virus is suspected. The differential diagnosis includes tinea corporis, guttate psoriasis, lichen planus, drug eruption, Lyme disease, and secondary syphilis. Recurrences are rare. Treatment is usually unnecessary, except for symptomatic alleviation of bothersome pruritus. Direct sun exposure or, alternatively, ultraviolet B therapy to severely affected areas has been recommended; the application of moderately potent topical steroids to severely pruritic areas also has its proponents. The benefits of these treatments are questionable, and ED interventions are usually limited to simple reassurance and close follow-up.

Atopic Dermatitis

Principles of Disease

Atopic dermatitis is a common dermatologic condition often referred to as eczema or chronic dermatitis. Atopic dermatitis is the cutaneous manifestation of an atopic state, and although it is not an allergic disorder, it is associated with allergic diseases such as asthma and allergic rhinitis. Patients with atopic dermatitis are known to have abnormalities of both humoral and cell-mediated immunity. The exact mechanism is unclear, but eosinophil, mast cell, and lymphocyte activation triggered by increased production of interleukin-4 by specific T helper cells seems to be involved.
Increased immunoglobulin E levels are found in most but not all patients with atopic dermatitis, but there is a poor correlation between the severity of the dermatitis and the serum immunoglobulin E level. The course of atopic dermatitis involves remissions and exacerbations. More than 90% of patients have the onset of atopic dermatitis before 5 years of age. New-onset atopic dermatitis in older children or adults should suggest other diagnoses.

Clinical Features

Atopic dermatitis has no pathognomonic skin lesions or unique laboratory parameters. Diagnostic criteria include itchy skin plus three or more of the following: history of flexural involvement, generalized dry skin, history of asthma or hay fever, onset of rash before 2 years of age, and flexural dermatitis.13 These criteria are sensitive (85%) and specific (96%).

Skin lesions generally appear as inflammatory thickened, papular, or papulovesicular lichenification and hyperpigmentation. The skin is typically dry and may be scaly, but in the acute phase, it may also be vesicular, weeping, or oozing. The distribution of lesions varies with the age of the patient. In infants, inflammatory exudative plaques are seen on the cheeks, on the extensor surfaces, and in the diaper area. Older children and adults have lesions in the antecubital and popliteal flexion areas, neck, face, and upper chest. Infantile atopic dermatitis usually begins in the fourth to sixth month of life and improves by the third to fifth year of life. The childhood form occurs between 3 and 6 years of age and resolves spontaneously or continues into the adult form.

Intense pruritus is a hallmark of atopic dermatitis. During flares, patients may present with complaints of intense itching and failure of routine treatments to control their symptoms. Patients may also present with secondary infections. The itching may be focal or generalized, is worse during the winter, and is triggered by increased body temperature and emotional stress. It may be particularly annoying at night. Excoriations may be prominent, and secondary bacterial infection of excoriated lesions is common. Repeated scratching and rubbing produce lichenification, a condition of hyperpigmentation, thickening of the skin, and accentuation of skin furrows. Lichenification is a common feature of chronic atopic dermatitis.

Differential Considerations

The differential diagnosis of infantile atopic dermatitis includes histiocytosis X, Wiskott-Aldrich syndrome, chronic seborrheic dermatitis, phenylketonuria, Bruton’s X-linked agammaglobulinemia, psoriasis, and scabies. Fixed drug eruptions and contact dermatitis round out the differential diagnosis regardless of age. Complications of atopic dermatitis include pyogenic skin infections, otitis externa, cataracts, keratoconus, retinal detachment, and cutaneous viral infections.

Management

The optimal protocol for management in children has not been established. Treatment should be aimed at control of inflammation, dryness, and itching. The use of sedating antihistamines at bedtime can be beneficial in patients with atopic dermatitis who have comorbid allergic conditions and sleep disturbances.

Management includes a careful review of daily skin care with patients or caregivers. General recommendations for all patients include avoidance of nonspecific skin irritants, wool, nonessential toiletries, and detergents and use of cotton clothing as much as possible. Patients should take daily warm baths or showers for approximately 10 to 15 minutes to hydrate the skin, followed by gentle pat drying and immediate application of a topical anti-inflammatory medication on the affected areas and a ceramide-based moisturizer such as Cetaphil, EpiCeram, or Cerave on the asymptomatic areas. Medium-potency topical corticosteroids may be sufficient to treat moderate flares. Ointment-based medications are usually better tolerated by most patients during an acute flare.

Skin dryness is treated with lubricating ointments such as Vaseline or 10% urea in Eucerin cream (not lotion). Treatment of exudative areas includes the application of wet dressings, which are useful for their moisturizing, anti-inflammatory, and antipruritic actions. Two or three layers of gauze soaked in Burow’s solution should be applied for 15 to 20 minutes four times a day for exudative lesions. Antihistamines may be helpful in reducing the pruritus and are also useful for their sedative and soporific effects, although there is no convincing evidence that H1 antihistamines decrease itching in patients with atopic eczema.

Topical corticosteroids are the cornerstone of therapy and are prescribed in ointment form. When the dermatitis is severe, the application of a fluorinated corticosteroid ointment such as half-strength betamethasone valerate is recommended to affected areas three times a day. Fluorinated corticosteroids should not be used on the face because they can produce permanent cutaneous atrophy. Milder corticosteroid preparations, such as 0.025% triamcinolone ointment, may be used on the face and intertriginous areas. Patients with extremely severe disease may require systemic steroids. Ultraviolet B treatment is moderately effective, although its mechanism of action is not well understood.

Cyclosporine and other immunosuppressant agents are being used with some promising benefit. Further studies are needed to determine ideal dosing and safety profiles for these agents.13

Topical calcineurin inhibitors, including tacrolimus ointment and pimecrolimus cream, are nonsteroidal topical immunosuppressants approved in the United States for use on children 2 years or older; they are useful for treatment of lesions on the thinner skin areas (face, groin, and axillae) where repeated applications of topical corticosteroids may result in skin atrophy or striae.14 A burning sensation at the site of application may occur. Note that the Food and Drug Administration has issued a “black box” warning concerning long-term continuous treatment with topical calcineurin inhibitors and cancer, although there is currently no evidence for a causal link.15

Inpatient admission is a consideration for those patients who have generalized erythema and exfoliation (erythroderma) or intractable itching in that skin breakdown and severe secondary bacterial or viral skin infections may occur.

Skin Infections in Patients with Atopic Dermatitis

Patients with atopic dermatitis are susceptible to infection and colonization by a variety of organisms because of their defective skin barrier functions and local skin immunodeficiency. Widespread disseminated viral infections, such as eczema molluscum, eczema vaccinatum, and eczema herpeticum, and recurrent staphylococcal pustulosis are especially concerning.

Eczema molluscum is self-limited. Eczema vaccinatum results from exposure of patients to vaccinia virus either by intentional inoculation or through contact with someone recently immunized against smallpox. Therapy for eczema vaccinatum requires prompt administration of intravenous immune globulin, which can be obtained from the Centers for Disease Control and Prevention.16

Eczema herpeticum constitutes a medical emergency. Patients present with disseminated eruptions of dome-shaped vesicles that may or may not be superimposed on areas of eczematous rashes, with the head, neck, and trunk commonly affected. Fever, malaise, and local lymphadenopathy are variable, depending on the timing
of presentation and host characteristics. Complications include keratoconjunctivitis, viremia, multorgan involvement with meningitis, and encephalitis. Clinical suspicion of eczema herpeticum mandates initiation of intravenous acyclovir in conjunction with antistaphylococcal antibiotics for possible bacterial superinfection. Lumbar puncture should not be attempted if infected lesions are present over the lumbar area. Ophthalmology consultation is needed for patients with periocular or suspected eye involvement.

**Impetigo**

**Principles of Disease**

Impetigo is a slowly evolving pustular eruption, most common in preschool children. Currently, *Staphylococcus aureus* is the most common pathogen, with group A streptococcus a distant second. Poor health and hygiene, malnutrition, and various antecedent dermatoses, especially atopic dermatitis, predispose individuals to impetigo.

**Clinical Features**

*Streptococcal impetigo* (ecthyma) is found most often on the face and other exposed areas. The eruption often begins as a single pustule but later develops multiple lesions. It begins as 1- to 2-mm vesicles with erythematous margins. When these break, they leave red erosions covered with a golden yellow crust. Lesions may be pruritic but usually are not painful. Regional lymphadenopathy is commonly present. Lesions are contagious among infants and young children and less so in older children and adults. Postpyoderma acute glomerulonephritis is a recognized complication of streptococcal impetigo.

*Staphylococcal impetigo* is differentiated from streptococcal impetigo in that it is more superficial, and there is little surrounding erythema. Other diagnostic considerations are herpes simplex virus (HSV) and inflammatory fungal infections. A Gram stain of the weepy erosion obtained after removal of the crust will reveal gram-positive cocci. MRSA impetigo may be increasingly common. Risk factors include prior infection or colonization with MRSA.

*Bullous impetigo* is caused by phage group 2 staphylococci. It is seen primarily in infants and young children. The initial skin lesions are thin-walled, 1- to 2-cm bullae. When these rupture, they leave a thin serous crust and collarette-like remnant of the blister roof at the rim of the crust. The face, neck, and extremities are most often affected. The differential diagnosis includes contact dermatitis, HSV infection, superficial fungal infections, and pemphigus vulgaris. A Gram stain of the fluid from a bulla reveals gram-positive cocci. Cultures are positive in 95% of cases.

**Management**

Systemic and topical therapies are equally successful in treating impetigo. For more extensive lesions, systemic treatment is recommended. There is no evidence, however, that systemic antibiotics prevent the development of acute glomerulonephritis. The efficacy is similar for topical mupirocin 2% ointment three times a day, oral erythromycin ethylsuccinate 250 mg four times a day for 10 days in adults or 30 mg/kg/day in children, and cephalaxin 50 mg/kg/day not exceed 500 mg per dose three times a day for 7 to 10 days. Mupirocin is avoided when there is concern about methicillin-resistant strains, and care should be taken that it does not get into the patient’s mouth.

**Folliculitis**

**Clinical Features**

Folliculitis is an inflammation in the hair follicle, usually caused by *S. aureus*. It appears as a pustule with a central hair. The lesions are usually on the buttocks and thighs, occasionally in the beard or scalp, and may cause mild discomfort. The differential diagnosis includes acne, keratosis pilaris, and fungal infection. Gram-negative folliculitis with *Pseudomonas aeruginosa* can occur after exposure to infected hot tubs and swimming pools or in individuals taking antibiotics for acne; it can be differentiated from staphylococcal folliculitis by a Gram stain of the lesion.

**Management**

Treatment with an antiseptic cleanser such as povidone-iodine or chlorhexidine every day or every other day for several weeks is usually adequate. For patients with extensive involvement, a 7- to 10-day course of doxycycline 100 mg twice a day or dicloxacillin 500 mg four times a day may be added.

**Hidradenitis Suppurativa**

Hidradenitis suppurativa affects the apocrine sweat glands. Recurrent abscess formation in the axillae and groin resembles localized furunculosis. The condition tends to be recurrent and may be extremely resistant to therapy. Hidradenitis suppurativa may be treated with drainage of abscesses if they are fluctuant, painful, and large. Antistaphylococcal antibiotics are useful if they are administered early and for a prolonged period. Begin treatment for mild disease with topical clindamycin, 10 mg/mL twice daily, for 3 months. In patients with more severe or nonresponsive disease, begin clindamycin, 300 mg twice daily, combined with rifampin, 300 mg twice daily, for 3 to 6 months. Antiandrogen therapy may be considered if antibiotics do not produce improvement. Many cases do not respond, however, and eventually require local excision and skin grafting of the involved area. Sonography will help differentiate abscesses from vascular or lymphoid structures.

**Carbuncle**

A carbuncle is a large abscess that develops in the thick, inelastic skin of the back of the neck, back, or thighs and usually involves hair follicles. Carbuncles may produce severe pain and fever. Septicemia may accompany the lesions. The diagnosis of skin abscess, furuncle, or carbuncle is usually made clinically. Ultrasonography is often helpful in diagnosis of carbuncles or abscesses that may not appear fluctuant on examination.

Local heat should be applied to furuncles and carbuncles, which should be incised and drained when fluctuant. Antibiotics are unnecessary with incision and drainage unless cellulitis or septicemia is present.
Community-Associated Methicillin-Resistant Staphylococcus aureus

Principles of Disease

The incidence of community-associated methicillin-resistant Staphylococcus aureus (CA-MRSA) has soared since the first report in 1993. In many major U.S. cities, CA-MRSA is now the most common pathogen cultured from ED patients presenting with skin and soft tissue infections.20 Concern exists that CA-MRSA may be more virulent than methicillin-sensitive strains, and colonization with CA-MRSA may produce more overt infections.20

Hospital-acquired MRSA isolates can survive on a variety of inanimate surfaces, sometimes for weeks. It is unclear whether this is also true for CA-MRSA isolates; if it is true, their presence on such items as clothing, towels, and athletic equipment might contribute to outbreaks. Pets (including dogs and cats), livestock, and birds have been identified as MRSA carriers; their role in MRSA transmission to humans requires further evaluation.21

Clinical Features

CA-MRSA infections are most often manifested as skin and soft tissue suppuration, such as an abscess, furuncle, or cellulitis. Lesions frequently exhibit central necrosis and are often confused with spider bites by patients.22 Clinical features cannot distinguish with certainty skin and soft tissue infections caused by MRSA from those caused by methicillin-susceptible S. aureus. Although rare, CA-MRSA infection can also be manifested as necrotizing fasciitis.23 Recurrences of CA-MRSA cellulitis are common. Contagion among the close household contacts of patients as well as correctional facility, school, and sports team contacts is well recognized.

Management

Several studies have demonstrated excellent outcomes for abscesses caused by CA-MRSA that are treated with incision and drainage alone.24 If antibiotics are needed, information on local antibiotic resistance patterns can help clinicians assess the likelihood of CA-MRSA infection and guide decisions about empirical treatment. A specimen for culture and susceptibility testing, which was considered to be unnecessary in the pre–CA-MRSA era, may be useful in guiding therapy. Specimens are obtained at the time of incision and drainage of purulent lesions.

In patients with larger abscesses, associated areas of cellulitis, or systemic signs of infection, antimicrobial therapy is needed in addition to incision and drainage. The optimal oral antimicrobial regimen for the treatment of skin and soft tissue infections is not known. The type and route of therapy are guided by the severity of the clinical syndrome.

Clindamycin combines MRSA activity with effectiveness against the majority of other gram-positive organisms. Side effects include diarrhea, Clostridium difficile colitis, and increasing rates of clindamycin resistance.25 Rifamycin has anti-MRSA activity, but resistance readily develops, so it should not be used alone. The advantage of rifamycin is its long half-life, which allows once-a-day administration, and it penetrates well into all tissues and body fluids; its disadvantages include a high potential for drug-drug interactions. Linezolid is active against almost all CA-MRSA isolates and group A streptococci. Disadvantages of its use include high cost, lack of routine availability, hematologic side effects, and potential for resistance among S. aureus strains. Prolonged linezolid administration increases the likelihood of resistance.26

Trimethoprim-sulfamethoxazole or tetracycline is not recommended as sole empirical therapy for a nonpurulent cellulitis of unknown cause because of group A streptococci resistance to these agents. A β-lactam antibiotic may augment treatment. Cephalosporins and macrolides are ineffective against CA-MRSA. Fluoroquinolones should be avoided because S. aureus resistance develops readily.

Patients with large abscesses, abscesses in high-risk locations, fever, signs of systemic infection, young age, or immunodeficiency prompt consideration of hospitalization. Vancomycin is still considered the parenteral drug of choice for patients with invasive S. aureus infection, although clinical failures have been reported. It seems reasonable to combine vancomycin with another effective antistaphylococcal agent because many antibiotics have better bactericidal activity. In severely ill patients, carbapenems such as meropenem, panipenem, and ertapenem are recommended because they are active against CA-MRSA and synergistic with vancomycin.27 Use of parenteral clindamycin (not recommended as monotherapy), trimethoprim-sulfamethoxazole, and linezolid has been described. In addition, daptomycin and tigecycline are now approved for the treatment of skin and soft tissue infections caused by MRSA.28

Recurrent infections are generally treated like initial episodes. Some providers recommend “decolonization” strategies, although neither the indications for their use nor their effectiveness in reducing the risk of recurrences is established. Decolonization strategies include the use of intranasal mupirocin to reduce nasal carriage of MRSA; however, eradication of nasal colonization appears to be transient. The efficacy of attempts to eradicate CA-MRSA among household members has not been studied.

Prevention

Common antiseptics appear to retain reasonable activity against CA-MRSA, although the results of studies are somewhat conflicting. Good personal hygiene, including appropriate handwashing techniques, separation of infected patients from other types of patients, and routine cleaning of shared equipment, are essential to limiting CA-MRSA spread.29

Gonococcal Dermatitis

Clinical Features

The arthritis-dermatitis syndrome is the most common presentation of disseminated gonococcal disease. It occurs in 1 or 2% of patients with gonorrhea, affecting women primarily.26 Fever and migratory polyarthralgias commonly accompany the skin lesions. The lesions are often multiple and have a predilection for periarthritis of the distal extremities. The lesions begin as erythematous or hemorrhagic papules that evolve into pustules and vesicles with an erythematous halo (Fig. 120-3). They closely resemble the lesions of meningococcemia at this stage. They may or may not be tender and may have a gray necrotic or hemorrhagic center. Healing with crust formation usually occurs within 4 or 5 days, although recurrent crops of lesions may appear even after antibiotics have been started.

Diagnostic Strategies

The lesions usually have a negative culture for gonococci, and the Gram stain only occasionally reveals the organisms. A more reliable diagnostic technique is immunofluorescent antibody staining of direct smears from pustules. This method indicates that the lesions may be the result of hematogenous dissemination of nonviable gonococci.

Management

Current treatment of disseminated gonococcal infection is 7 days of ceftriaxone, 1 g intramuscularly (IM) or intravenously (IV)
Cells to the skin tissue denoted by erythema, swelling, and local tenderness (Fig. 120-4). Erysipelas is a streptococcal infection of the skin and subcutaneous tissue. The involved area is red, indurated, and edematous. Soft tissue skin infections are discussed in more depth in Chapter 137.

**RED MACULES**

**Drug Eruption**

**Principles of Disease**

A given drug can produce a skin eruption of a different appearance in different patients or a different appearance in the same patient on different occasions. The most common eruptions are urticaria (hives) (Fig. 120-5) and, more commonly, morbilliform rashes (Fig. 120-6).

Drug reactions tend to appear within a week after the drug is taken, with the exception of reactions to semisynthetic penicillins, which commonly occur later. Skin lesions may appear after a drug has been discontinued and may worsen if the drug or its metabolites persist in the system. Special note should be made of penicillin because it is the most common cause of drug reaction. Serum sickness and urticaria are the most common manifestations of penicillin allergy. Atopic patients and those with a history of hay fever, asthma, or eczema are at special risk.

On the other hand, a number of drugs in common use rarely produce eruptions. Among these are acetaminophen, aluminum hydroxide (Maalox), codeine, digoxin, erythromycin, ferrous sulfate, meperidine (Demerol), morphine, and prednisone.
Fixed drug eruptions appear and recur at the same anatomic site after repeated exposure to the same drug. The lesions are usually sharply margined and round or oval. They may be pigmented, erythematous, or violaceous. Pruritus may be prominent.

Differential Considerations
The differential diagnosis of drug eruptions includes viral exanthem, chronic exfoliative erythroderma caused by psoriasis or atopic dermatitis, malignant disease, scarlet fever, staphylococcal scarlatiniform eruptions, and Kawasaki disease.

Management
Treatment of drug eruptions begins with discontinuation of the inciting agent. Warn patients that drug eruptions often clear slowly after discontinuation of the offending agent. Itching may be treated with the application of a drying antipruritic lotion such as calamine. Cool compresses, tepid water baths with colloidal oatmeal (Aveeno) emollient or cornstarch, and diphenhydramine, 50 mg (5 mg/kg/24 hr in children) every 6 hours, are likely to be beneficial.

Staphylococcal Scalded Skin

Clinical Features
Staphylococcal scalded skin syndrome generally occurs in children 6 years of age or younger. It is caused by an infection with phage group 2 exotoxin-producing staphylococci. The illness begins with erythema and crusting around the mouth. The erythema then spreads down the body, followed by bulla formation and desquamation. Mucous membranes are usually not involved, but minimal involvement is occasionally seen. After desquamation occurs, the lesions dry up quickly, with clinical resolution in 3 to 7 days.

Management
Most group 2 toxin-producing organisms are penicillin resistant. Although most patients will recover without antibiotic treatment, intravenous therapy with 50 to 100 mg/kg of nafcillin daily or cephalixin 50 mg/kg/day or dicloxacillin is recommended. Clindamycin, vancomycin, or linezolid may be considered in cases of suspected MRSA.

Toxic Epidermal Necrolysis

Principles of Disease
Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) are often considered a continuous spectrum of the same disease. Both are true dermatologic emergencies. The main feature of non–staphylococcal-induced TEN, or Lyell’s disease, is the separation of large sheets of epidermis from underlying dermis. Drugs that can cause TEN include the long-acting sulfa drugs, penicillin, aspirin, barbiturates, phenytoin, carbamazepine, allopurinol, and nonsteroidal anti-inflammatory drugs. TEN has occurred after vaccination and immunization against poliomyelitis, measles, smallpox, diphtheria, and tetanus. It has also been found in association with lymphoma.

Clinical Features
TEN commonly begins with prodromal symptoms, such as malaise, rhinitis, sore throat, body aches, and fever. These are followed by the abrupt development of a macular rash that may or may not appear as target lesions. Mucous membrane

Clinical Features
A variety of skin reactions are associated with commonly used drugs, such as aspirin, penicillin, sulfonamides, and phenytoin. Exanthematous drug eruptions resemble the skin manifestations of various viral or bacterial infections and are usually widespread symmetric maculopapular eruptions. Severe cases may progress to exfoliative dermatitis.

Eczematous drug rashes resemble those of contact dermatitis but are generally more extensive. They begin as erythematous or papular eruptions that may become vesicular. Prior sensitization to a topical medication is common in cases of this type of eruption.

Vasculitic lesions begin as erythematous papules or nodules but may ulcerate and become gangrenous. Urticarial vasculitis is characterized by persistent urticarial lesions with histologic evidence of leukocytoclastic vasculitis. Wheel-and-flare–like lesions that hurt or burn more than itch, lesions lasting more than 24 hours, and urticarial lesions that leave prolonged pigmented changes or inflammatory lesions should suggest urticarial vasculitis. Purpuric drug eruptions may be the result of bone marrow suppression, platelet destruction, or vasculitis (Fig. 120-7). Ultimately, a skin biopsy is needed to confirm the diagnosis of vasculitis.

Photosensitive drug reactions require the presence of sunlight and are seen most commonly on sun-exposed areas of skin. This class of reactions is commonly divided into phototoxic and photoallergic. Phototoxic reactions are more common. Sulfonamides, sulfonylureas, thiazide diuretics, and tetracyclines are common causes (see Fig. 120-7). This type of reaction does not primarily involve immunologic mechanisms and occurs in any person taking an adequate quantity of the drug and exposed to sunlight. The lesions usually have the appearance of a severe sunburn but may be bullous or papular. Pruritus is typically minimal or absent.

Photoallergic reactions are the result of antigen formation that results in the formation of sensitized lymphocytes. These reactions therefore represent a delayed immunologic response. A photoallergic reaction occurs only in sensitized individuals, usually 2 weeks or longer after exposure to the drug and sunlight. Its occurrence is not dose related, and the eruption usually appears eczematous and intensely pruritic. Chlorpromazine, promethazine, and chlor Diazepoxide are common sensitizers of photoallergic reactions.

Patients who have photoallergic reactions should be withdrawn from the inciting drugs. Patients who are subject to photosensitive drug eruptions may be required to avoid prolonged sunlight exposure; use of a sunscreen containing 5% aminobenzoic acid is recommended for any sun exposure.

Figure 120-7. Purpuric lesions. (Courtesy David Effron, MD.)
Toxic Shock Syndrome

Principles of Disease

Toxic shock syndrome (TSS) is an acute febrile illness characterized by a diffuse desquamating erythroderma. Classically composed of high fever, hypotension, constitutional symptoms, multiorgan involvement, and rash, the syndrome gained notoriety in the early 1980s because of association with tampon use. However, it is also well known in men and children. Its appearance has often been linked to exotoxin-producing S. aureus. Most cases of nonmenstrual TSS occur in the postoperative setting. TSS has also been associated with various staphylococcal and streptococcal infections, including empyema, osteomyelitis, fasciitis, septic abortion, peritonsillar abscess, sinusitis, burns, and subcutaneous abscess. TSS is associated with severe group A beta-hemolytic streptococcal infections. It has been reported in previously healthy patients, immunocompromised patients, and elders. Fatigue, localized pain, and nonspecific symptoms herald the onset of this disease, followed by septic shock and multisystem organ failure.

Clinical Features

Diagnosis of TSS requires the presence of (1) temperature of at least 38.9° C; (2) hypotension, with a systolic blood pressure of 90 mm Hg or less; (3) rash; and (4) involvement of at least three organ systems. Systemic involvement may include the gastrointestinal tract, muscular system, or central nervous system (CNS) and laboratory evidence of renal, hepatic, or hematologic dysfunction. Headache, myalgias, arthralgia, alteration of consciousness, nausea, vomiting, and diarrhea may be present. The rash is typically a diffuse, blanching, macular erythroderma. Accompanying nonexudative mucous membrane inflammation is common. Pharyngitis, sometimes accompanied by a “strawberry tongue,” conjunctivitis, or vaginitis, may be seen. As a rule, the rash fades within 3 days of its appearance. This is followed by a full-thickness desquamation, most commonly involving the hands and feet.

Management

The treatment of TSS consists of intravenous fluid replacement, ventilatory support, pressor agents, antibiotics covering S. aureus (including MRSA) and Streptococcus pyogenes with addition of clindamycin, and drainage of infected sites. Initial empirical antibiotic regimens include clindamycin 900 mg every 8 hours plus imipenem 500 mg every 6 hours or meropenem 1 g every 8 hours or ticarcillin-clavulanate 3.1 g every 4 hours or piperacillin-tazobactam 4.5 g every 6 hours.
Urticaria

Principles of Disease

Urticaria may occur in isolation or as part of a systemic anaphylactic reaction. The following discussion pertains to urticaria occurring in the absence of systemic symptoms. Anaphylactic reactions and angioedema are discussed in Chapter 119. Approximately 15 to 20% of the population experiences urticaria during their lifetime. Acute urticaria is seen in both sexes and is more likely to have an allergic cause. Chronic urticaria is more common in women in their 40s and 50s. Half of all patients with chronic urticaria have the disease for 5 years and one fourth for 20 years.

Various mediators, including histamine, bradykinin, kallikrein, and acetylcholine, are thought to play a role in urticaria production. Urticaria may be initiated by immunologic or nonimmunologic mechanisms. Hives found in anaphylaxis and serum sickness represent an immunologic reaction. Nonimmunologic urticaria may be produced by degranulation of mast cells, which may be caused by a number of foods and drugs, including aspirin and narcotics.

Substances that can cause urticaria by contact with the skin include foods, textiles, animal dander and saliva, plants, topical medications, chemicals, and cosmetics. The role of drugs in the production of urticaria is discussed in the section on drug eruption. Almost any drug may produce urticaria, although penicillin and aspirin are the most common. Traces of penicillin may be present in dairy products as well as in medications. The mechanism of production of urticaria by aspirin is unknown but is probably nonimmunologic, and the effects of aspirin may persist for a number of weeks after ingestion.

A variety of food allergies, such as fish, eggs, and nuts, may result in urticaria. In addition, foods such as lobster and strawberries can release histamine through a nonimmunologic mechanism. Hereditary forms of urticaria include familial cold urticaria and hereditary angioneurotic edema.

Infections are an uncommon cause of urticaria, except in children, in whom viral infections often cause hives. Occult infections with Candida, the dermatophytes, bacteria, viruses, and parasites may trigger hives. Viral infections that produce urticaria include hepatitis, mononucleosis, and Coxsackievirus infections.

The inhalation of pollens, mold, animal dander, dust, plant products, and aerosols may produce urticaria. Respiratory symptoms may accompany the dermatosis, and a seasonal pattern of occurrence may be present. Stings and bites of insects, arthropods, and various marine animals may also produce a urticarial eruption.

On occasion, patients with systemic lupus erythematosus, lymphoma, carcinoma, hyperthyroidism, rheumatic fever, and juvenile rheumatoid arthritis develop a urticarial eruption. The association is uncommon enough that it is not necessary for a urticaria workup to include a search for malignant disease in most cases.

A number of physical agents produce urticaria. Dermatographism is present when firm stroking of the skin produces a urticarial wheal within 30 minutes (Fig. 120-10) and is the most common form of physical urticaria. Pressure urticaria is distinct from dermatographism in that the onset of urticaria is delayed by 4 to 8 hours after the application of physical pressure. There is no other particular significance to this form of urticaria.

Cold urticaria may be either familial or, more commonly, acquired. Cold urticaria may also be associated with underlying illness, such as cryoglobulinemia, cryofibrinogenemia, syphilis, and connective tissue disease. Nonsedating antihistamines, such as rupatadine 10 to 20 mg daily, help suppress primary cold urticaria. Antihistamines taken 30 to 60 minutes before cold exposure may be helpful. Cholinergic urticaria is induced by exercise, heat, or emotional stress. It may be associated with pruritus, nausea, abdominal pain, and headache. The lesions of cholinergic urticaria are wheals 1 to 3 mm in diameter surrounded by extensive erythematous flares and, occasionally, satellite wheals. Nonsedating antihistamines are generally used to treat cholinergic urticaria.

Heat is a rare cause of hives. Solar urticaria, also uncommon, is confined to sun-exposed areas of skin and clears rapidly when the light stimulus is removed. Extensive sun exposure may cause wheezing, dizziness, and syncope in a susceptible individual. Sunscreens have not been proved to be effective for the prevention of solar urticaria. Phototherapy may be used to induce tolerance.

Clinical Features

Urticaria appears as edematous plaques with pale centers and red borders and is easily recognizable (see Fig. 120-5). Individual hives are transient, lasting less than 24 hours, although new hives may continuously develop, which represents localized dermal edema produced by transvascular fluid extravasation.

Differential Considerations

The differential diagnosis of urticaria includes erythema multiforme, erythema marginatum, and juvenile rheumatoid arthritis.

Management

Treatment of urticaria involves the removal of the inciting factor, when applicable, and the administration of antihistamines or other antipruritics. Hydroxyzine (Atarax and Vistaril) in a dose of 25 to 50 mg (children’s dose not to exceed 50 mg/day for children younger than 6 years and 100 mg/day for children older than 6 years) is usually effective in providing symptomatic relief. Alternatives are nonsedating antihistamines, such as loratadine 10 mg to 20 mg once a day or fexofenadine 60 mg twice a day. Prednisone is also effective, but the urticaria can rebound, making cessation of prednisone sometimes difficult. For chronic urticaria, long-term therapy with antihistamines may be needed. Nonsedating antihistamines are preferred, especially during the daytime. Cetirizine 10 to 20 mg/day, fexofenadine 180 mg/day, or loratadine 10 to 20 mg/day can be used. A single dose of an H2 blocker may be added.

Patients with chronic urticaria may be discharged with a prescription for a combination of an H1 and H2 antihistamine. Strong evidence supporting addition of an H2 blocker is lacking. The use of steroids in management of urticaria is controversial and strong evidence for their use does not exist. Steroids are not indicated for mild cases. Patients with moderate or severe urticaria
may benefit from prednisone 60 mg orally initially, followed by 40 mg daily for 5 days or dexamethasone (Decadron) 6 mg orally initially, followed by another 6 mg orally in 48 hours. Patients with recurrent urticaria may benefit from longer courses of oral steroids (14 to 21 days with a taper). Chronic administration of steroids is not recommended.

**EXANTHEMS**

**Principles of Disease**

An exanthem is defined as a skin eruption that occurs as a symptom of a general disease. Approximately 30 enteroviruses, predominantly the Coxsackievirus and echovirus groups, and four types of adenoviruses are known to produce exanthemata. Other viruses may also do so as well. The exanthemata of the Coxsackievirus and echovirus are most thoroughly documented. Most viral exanthemata are maculopapular, although scarlatiniform, erythematous, vesicular, and petechial rashes are occasionally seen. The eruptions are variable in their extent, are nonpruritic, and do not desquamate. Oropharyngeal lesions may be present.

Infection with echovirus type 9 may be accompanied by meningitis and a petechial exanthem resembling meningococcemia, although the exanthem also occurs without meningeval involvement. Infections caused by echovirus type 16 (Boston exanthem) and Coxsackievirus group B, type 5, may resemble roseola infantum but are more likely to occur in adults.

Infections caused by Coxsackievirus group A, type 16, cause a distinctive syndrome of vesicular stomatitis and 1- to 4-mm oral vesicles involving the dorsa of the hands and lateral borders of the feet. Disease caused by Coxsackievirus group A, type 9, has been the most extensively studied. It may be associated with meningoencephalitis or interstitial pneumonia. The rash is usually maculopapular, begins on the face or trunk, and spreads to the extremities. A vesicular eruption resembling varicella may occur.

The classic viral exanthemata are rubeola (measles), rubella (German measles), herpesvirus 6 (roseola), parvovirus B19 (erythema infectiosum or fifth disease), and the enteroviruses (echovirus and coxsackievirus). Widespread immunization programs have reduced the incidence of rubeola and rubella.

**Measles**

**Clinical Features**

Measles is a highly contagious viral illness spread by contact with infectious droplets, with an incubation period of 10 to 14 days. Patients are contagious from 1 or 2 days before onset of symptoms up to 4 days after the appearance of the rash. Symptoms begin with fever and malaise. The fever usually increases daily in a stepwise manner until the temperature reaches approximately 40.5° C on the fifth or sixth day of the illness. Cough, coryza, and conjunctivitis begin within 24 hours of the onset of symptoms.

On the second day of the illness, Koplik’s spots, which are pathognomonic of the disease, appear on the buccal mucosa as small, irregular, bright red spots with bluish white centers. Beginning opposite the molars, Koplik’s spots spread to involve a variable extent of the oropharynx.

The cutaneous eruption of measles begins on the third to fifth day of the illness. Maculopapular erythematous lesions involve the forehead and upper neck and spread to involve the face, trunk, arms, and finally the legs and feet. Koplik’s spots begin to disappear coincident with the appearance of the rash. By the third day of its presence, the rash begins to fade, doing so in the order of its appearance, and the fever subsides.

Complications include otitis media, encephalitis, and pneumonitis. Otitis media is the most common complication. Encephalitis occurs in approximately 1 in 1000 cases of measles and carries a 15% mortality. Measles pneumonia may also be life-threatening.

**Management**

If bacterial invasion occurs with otitis or pneumonia, the use of antibiotics is indicated. Otherwise, treatment is supportive. Isolation of infected children is of limited value because exposure usually occurs before the appearance of the rash, and the presence of Koplik’s spots render the disease diagnosable. Measles is not contagious after the fifth day of the presence of the rash. Infection confers lifelong immunity.

The illness can be modified or prevented by the administration of human immune serum globulin (ISG) in a susceptible person within 6 days of exposure. The recommended dose of ISG is 0.25 mL/kg IM in children. Live measles virus vaccine given within 72 hours of exposure may be effective in preventing measles.39 The incidence of measles has decreased since the resurgence seen in 1989 to 1991. The patterns observed during outbreaks include a shift from preschool-aged children to older adults and among groups who do not routinely obtain vaccination, such as immigrants.

**Rocky Mountain Spotted Fever**

**Principles of Disease**

Rocky Mountain spotted fever is caused by *Rickettsia rickettsii*, an organism harbored by a variety of ticks. The organism is transmitted to humans through tick saliva at the time of a tick bite or when the tick is crushed while in contact with the host. Many patients do not report tick exposure. Although originally described in the Rocky Mountain region, this disease occurs in other areas of North, South, and Central America. Most reported cases are from the southeastern United States.

**Clinical Features**

The onset of the illness is usually abrupt, with headache, nausea and vomiting, myalgias, chills, and a fever spiking to 40° C. On occasion, the onset is more gradual, with progressive anorexia, malaise, and fever. The disease may last 3 weeks and may be severe with prominent involvement of the CNS and other organ systems (cardiac, pulmonary, gastrointestinal, renal), disseminated intravascular coagulation, or shock.

The rash develops on the second to fourth day or, occasionally, as late as the sixth day of the illness. It begins with erythematous macules that blanch on pressure, appearing first on the wrists and ankles. These macules spread up the extremities and to the trunk and face in a matter of hours. They may become petechial or hemorrhagic. Lesions on the palms and soles are particularly characteristic. Increased capillary fragility and splenomegaly may be present.

**Diagnostic Strategies**

The Weil-Felix reaction is the best known serologic diagnostic test, but the development of Weil-Felix agglutinins in cases of Rocky Mountain spotted fever is not constant, and more specific immunofluorescent procedures have been developed. Treatment should not await the result of such tests, however, but should begin as soon as the disease is suspected on clinical grounds.

**Management**

Doxycycline at 2 to 4 mg/kg/day divided twice daily IV or orally is the antibiotic of choice. Doxycycline should not be withheld...
Established.

Doxycycline efficacy in this disease is not resistant to penicillins, cephalosporins, aminoglycosides, and erythromycin. Ehrlichiosis may be difficult to differentiate from Rocky Mountain spotted fever clinically and is reliably treated because they can exacerbate the illness. Rickettsiae are routinely resistant to penicillins, cephalosporins, aminoglycosides, and erythromycin. Ehrlichiosis may be difficult to differentiate from Rocky Mountain spotted fever clinically and is reliably treated with doxycycline. Chloramphenicol efficacy in this disease is not established.

Roseola Infantum

Roseola infantum, otherwise known as exanthem subitum or sixth disease, is a benign illness caused by human herpesvirus 6 and characterized by fever and a skin eruption. A roseola-like illness has occasionally been associated with other illnesses. Ninety-five percent of cases are seen in children 6 months to 3 years of age, and most of these are in infants younger than 2 years. A febrile seizure may occur. The fever typically has an abrupt onset, with temperature rising rapidly to 39° to 41° C, and is present consistently or intermittently for 3 or 4 days, at which time the temperature drops precipitously to normal.

The rash appears with defervescence. The lesions are discrete pink or rose-colored macules or maculopapules 2 or 3 mm in diameter that blanch on pressure and rarely coalesce. The trunk is involved initially, with the eruption typically spreading to the neck and extremities. The eruptions are occasionally limited to the trunk. The rash clears during 1 or 2 days without desquamation.

Despite the presence of a high fever, the infant usually appears well. Encephalitis is a very rare complication. The prognosis is excellent, and no treatment is necessary.

Rubella

Rubella, or German measles, is a viral illness characterized by fever, skin eruption, and generalized lymphadenopathy. It is spread by droplet contact, and peak incidence is in the winter and early spring. The incubation period is typically 14 to 21 days, and the rash heralds the onset of the illness in children. The maximum time of communicability is in the few days before and 5 to 7 days after the onset of the rash. Infants with congenital rubella can shed virus for more than 1 year. In adults, a 1- to 6-day prodrome of headache, malaise, sore throat, coryza, and low-grade fever precedes the rash. These symptoms generally disappear within 24 hours after the appearance of the skin eruption.

The rash of pink to red maculopapules appears first on the face and spreads rapidly to the neck, trunk, and extremities. Those on the trunk may coalesce, but lesions on the extremities do not. The rash begins on the chest and spreads rapidly, usually within 24 hours. The trunk is intensely red on the face and gives a “slapped-cheek” appearance with circumoral pallor. A maculopapular macular rash, which may be noted on the arms, moves caudally to the trunk, buttocks, and thighs. The rash may recur with changes in temperature and exposure to sunlight. The incubation period is usually between 4 and 14 days.

Parvovirus B19 infection may also result in asymptomatic infection, upper respiratory infection, atypical rash, and arthritis without rash. Rarely, it has been reported to cause hepatitis. Infected immunodeficient patients may experience chronic anemia as a result of this disease. Patients with sickle cell disease or other hemolytic anemias may develop an aplastic crisis lasting 7 to 10 days. Parvovirus B19 infection during pregnancy can cause fetal hydrops and death. No congenital anomalies have been reported. No treatment is required.

Scarlet Fever

Clinical Features

The incidence of scarlet fever has declined in recent years. The illness has an abrupt onset with fever, chills, malaise, and sore throat, followed within 12 to 48 hours by a distinctive rash that begins on the chest and spreads rapidly, usually within 24 hours. Circumoral pallor may be noted. The skin has a rough sandpaper-like texture because of the multitude of pinhead-sized lesions. The pharynx is injected, and there may be erythematous lesions or petechiae on the palate. After the resolution of symptoms, desquamation of the involved areas occurs and is characteristic of the disease.

Complications include the development of a streptococcal infection of lymph nodes, tonsils, middle ear, and respiratory tract. Late complications include rheumatic fever and acute glomerulonephritis (Fig. 120-11).

Roseola Infantum

Figure 120-11. Erythema marginatum associated with rheumatic fever. (Courtesy David Effron, MD.)
Management

Treatment is 10 days of oral penicillin VK 50 mg/kg/day (40,000-80,000 units) in four divided doses in children or 250 mg four times a day in adults. IM benzathine penicillin (given as Bicillin C-R) is another option; dosing is 300,000 units in patients weighing less than 30 pounds, 600,000 units in patients weighing 31 to 60 pounds, 900,000 units in patients weighing 61 to 90 pounds, and 1.2 million units in patients weighing more than 90 pounds. In patients allergic to penicillin, the treatment is 10 days of erythromycin, 250 mg four times a day in adults or 40 mg/kg/day in children. Other macrolides and certain other cephalosporins may also be used.

**PAPULAR LESIONS**

**Contact Dermatitis**

**Principles of Disease**

Contact dermatitis is an inflammatory reaction of the skin to a chemical, physical, or biologic agent. The inducing agent acts as an irritant or allergic sensitizer. Allergic contact dermatitis is a form of delayed hypersensitivity mediated by lymphocytes sensitized by the contact of the allergen to the skin. It is less common than irritant contact dermatitis. Caustics, industrial solvents, and detergents are common causes of irritant dermatitis. Dermatitis may result from brief contact with a potent caustic or from repeated or prolonged contact with milder irritants. Latex allergy causing contact dermatitis is of specific interest.

Clothing, jewelry, soaps, cosmetics, plants, and medications contain allergens that commonly cause allergic contact dermatitis. The most common allergens include rubber compounds; plants of the *Toxicodendron* species, including poison ivy, oak, and sumac; nickel, often used in jewelry alloys; paraphenylenediamine, an ingredient in hair dyes and industrial chemicals; and ethylenediamine, a stabilizer in topical medications.

**Clinical Features**

The primary lesions of contact dermatitis are papules, vesicles, and bullae on an erythematous bed. Streaky, linear, intensely pruritic lesions are characteristic. Atypical or diffuse rash develops with exposure to smoke from burning plants or when power tools are used to cut and mulch brush. Once the plant is touched, urushiol can be transferred to various anatomic locations (face, genitalia) or to other people. Domestic animals can serve as vectors if they are petted shortly after exposure to plants.

Eruptions associated with contact dermatitis can appear as soon as several hours after the exposure or may be delayed for days. Allergy to topical steroid preparations used to treat contact dermatitis can occur.

**Management**

Treatment of contact dermatitis includes avoidance of the irritant or allergen and treatment of secondary bacterial infection. Oozing or vesiculated lesions should be treated with cool wet compresses of Domeboro or Burow’s solutions (aluminum acetate) applied for 15 minutes three or four times a day. Topical baths, available over the counter, may also be comforting. Systemic antihistamines, such as hydroxyzine and diphenhydramine, may help control pruritus; nonsedating antihistamines are preferred for use during the day. Low-potency topical steroid creams may be applied to erythematosus areas around natural orifices and medium-potency creams can be used elsewhere. Topical steroids are ineffectual on blistered areas.

**Poison Ivy**

**Clinical Features**

Of the allergens, *Toxicodendron* species are the most likely to cause bullous eruptions. Oozing, crusting, scaling, and fissuring may be found along with lichenification in chronic lesions. The distribution of the eruption depends on the specific contact and may be localized, asymmetric linear, or unilateral (Figs. 120-12 and 120-13). Mucous membranes are usually spared unless they are directly exposed to the inciting agent. A history of exposure is the most significant factor favoring the diagnosis. If doubt exists about the diagnosis, the patient should be referred for allergic patch testing. Sensitization to poison ivy results in sensitization to other plants in this family, such as cashew, mango, lacquer, and ginkgo trees.

**Management**

In addition to the aforementioned treatment regimens for contact dermatitis, a course of systemic corticosteroids is often necessary to treat *Toxicodendron*-associated dermatitis. Prednisone in a dosage of 30 to 80 mg/day (depending on the severity of involvement) can be prescribed initially and then tapered during at least 10 to 14 days and 21 days for poison ivy. The long, slow taper is needed to prevent rebound of the disease. The treatment may be discontinued when a daily dose of 5 mg is reached. In cases of *Toxicodendron* exposure, counsel the patient to wash all clothes.
that might have contacted the plant because the irritant plant oil can persist. Once the offending agent is reliably removed from the skin and clothes, ongoing outbreak is attributable to the initial contact, not spread from the serous fluid from the bullae. The patient is not contagious to others unless there is direct contact with the plant oil in people who are sensitized.

Erythema Multiforme

Principles of Disease

The most common precipitating factors in erythema multiforme are exposure to drugs and HSV infection. Additional causes include other viral infections, especially hepatitis and influenza A. Less common causes include fungal diseases, such as dermatophytosis, histoplasmosis, and coccidioidomycosis, and bacterial infections, especially streptococcal infections and tuberculosis. Various collagen vascular disorders have been known to precipitate erythema multiforme, particularly rheumatoid arthritis, systemic lupus erythematosus, dermatomyositis, and periarteritis nodosa. Pregnancy and various malignant neoplasms have also been associated with erythema multiforme. No provocative factor can be identified in approximately half of all cases. Differential diagnosis includes urticaria, scalded skin syndrome, pemphigus, and pemphigoid and viral exanthems.

Clinical Features

Erythema multiforme is an acute, usually self-limited disease precipitated by a variety of factors. It is characterized by the sudden appearance of skin lesions that are erythematous or violaceous macules, papules, vesicles, or bullae. Their distribution is often symmetric, most commonly involving the soles and palms, the backs of the hands or feet, and the extensor surfaces of the extremities. The presence of lesions on the palms and soles is particularly characteristic.

The target lesion with three zones of color is the hallmark of erythema multiforme. It is a central, dark papule or vesicle that is surrounded by a pale zone, a halo of erythema (Fig. 120-14), and is commonly found on the hands or wrists.

Management

Treatment begins with a search for the underlying cause. Mild forms with no systemic symptoms, lesions limited to extremities, and no mucous membrane involvement resolve spontaneously in 2 or 3 weeks. Patients with lesions on the trunk and patients who are immunocompromised, especially those with multiple lesions, require a course of systemic steroids for 14 to 21 days with a taper and urgent dermatology referral. Patients with mucous membrane involvement, systemic symptoms, or vesicle formation raise concern for SJS.

Pediculosis

Clinical Features

The diagnosis is made by identification of nits or adult lice on microscopic examination of plucked hairs from the symptomatic area. Nits are relatively more common than the adult louse form. Nits attach to the bases of hair shafts and appear as white dots (Fig. 120-15). Adult forms look like blue or black grains. The patient complains of intense itching and scratching. A secondary infection may result from the scratching.

The organisms causing pediculosis corporis reside in the seams of clothing and bedding materials while they feed on the human host. Except for heavily infested individuals, the parasites are absent from the body. Erythematous macules or wheals may be present along with intense pruritus. The treatment consists of laundering or boiling of clothing and bed linen. If nits are found in the body hair, a treatment with lindane lotion may be instituted, but this is not necessary in most cases (Figs. 120-16 and 120-17).

Pediculosis capitis is seen more commonly in small children than in adults. Pruritus is the major symptom and may be confined to the occipital or postauricular scalp. Excoriations commonly result in secondary bacterial infections and regional lymphadenopathy.

Diagnostic Strategies

The diagnosis is made by the identification of nits cemented to hairs at the hair-scalp junction (see Fig. 120-15).

Management

Permethrin (Nix) is the recommended treatment. It remains active for 2 weeks. Lindane (Kwell) lotion or cream is no longer the preferred prescription topical treatment. Cream rinses and conditioning shampoos should not be used during this period because they coat the hairs and protect the lice from the insecticide. Permethrin is applied to the scalp after the hair is shampooed and dried. It is rinsed out with water after 10 minutes. It must be applied when the hair is dry because lice can close down their respiratory airways for up to 30 minutes when immersed in water.
partners should also be treated. Other uninfested household members need not undergo a course of therapy. Underclothing, pajamas, and sheets and pillowcases should be machine washed (hot water) and dried, laundered and ironed, or boiled. Pruritus that persists after the course of therapy may result from an irritation of the skin by the pediculicide, sensitization, or anxiety of the patient.

Scabies

Clinical Features

Scabies is a mite infestation characterized by severe itching, which usually worsens at night. The areas of the body most commonly involved are the interdigital web spaces, flexion areas of the wrists, axillae, buttocks, lower back, penis, scrotum, and breasts (Fig. 120-18). The infestation tends to be more generalized in infants and children than in adults. The typical lesions are reddish papules or vesicles surrounded by an erythematous border and scratch marks. Scabies in infants and young children often has generalized involvement of the skin, including the face, scalp, palms, and soles. In infants, the most common presenting lesions are papules and vesiculopustules.

Nodular scabies is a clinical variant in which extremely pruritic nodules are present on the male genitalia, buttocks, groin, and axillary regions. The nodules are reddish to brown, do not contain mites, and are thought to represent hypersensitivity reactions. They can persist for weeks despite adequate scabicidal treatment.

Immunosuppressed patients may develop Norwegian scabies, which is manifested by extensive hyperkeratosis and crusting of the hands, feet, and scalp. It is highly contagious because of excessive mite proliferation. Secondary infections of these lesions are common.

Close personal contact is involved in transmission of scabies. Multiple family members are likely to become infested. The infestation is also transmitted with sexual contact.

Management

Treatment options include crotamiton (Eurax) lotion and cream, permethrin 5% cream (Elimite), and ivermectin (oral or topical). Lindane is not recommended as a first-line therapy; however, it does have a role in treating those cases that fail to respond to first-line therapies. Permethrin 5% cream (Elimite) applied overnight once weekly for 2 weeks over the entire body is the treatment of choice for infants and small children. It is more effective than crotamiton (Eurax) in eliminating the mite, in reducing secondary bacterial infection, and in reducing pruritus. Postscabetic nodules and pruritus may persist for months, even after successful
treatment. Treatment of Norwegian scabies may require repeated treatment with scabicides and sometimes sequential use of several agents.

A single dose of oral ivermectin, 200 µg/kg, may also be used. A second dose given 1 week later has been demonstrated to substantially improve the cure rate. Patients with crusted scabies may require repeated doses of ivermectin (200 µg/kg) along with topical scabicides (full-body application, repeated initially every few days) and keratolytics.

The full benefit of ivermectin becomes evident when eradication of scabies in epidemic or endemic situations is needed because ivermectin leads to reliable disease control. The safety of ivermectin has been documented in millions of people with microfilarial diseases. Although ivermectin does not normally penetrate the blood-brain barrier and there should be no risk of seizures, neurotoxicity has been reported in the elderly. Because of limited safety data, ivermectin should not be used in children younger than 5 years or during pregnancy or lactation.

All family members and sexual contacts should also be treated. Intimate articles of clothing and sheets and pillowcases should be washed and dried by machine (hot water), laundered and ironed, or boiled.

It may take several weeks after therapy for the signs and symptoms to abate. A hypersensitivity state or anxiety may prolong symptoms long after the mites have been destroyed.

### Syphilis

#### Clinical Features

Syphilis is transmitted only by direct contact with an infectious lesion. The causative organism is the spirochete *Treponema pallidum*. After an incubation period of 10 to 90 days, the primary lesion appears, which lasts 3 to 12 weeks and heals spontaneously. In 6 weeks to 6 months after exposure, the disease enters the secondary stage, which involves a variety of mucocutaneous lesions. These lesions also heal spontaneously in 2 to 6 weeks as the disease enters the latent phase. Either a prolonged latent phase or tertiary syphilis follows. Of untreated patients, 25% display at least one relapse of mucocutaneous lesions of the oral cavity or anogenital region.

The chancre is the dermatologic manifestation of primary syphilis. Chancres usually appear as single lesions but may be multiple. They appear at the site of spirochete inoculation, usually the mucous membranes of the mouth or genitalia. The chancre begins as a papule and characteristically develops into an ulcer approximately 1 cm in diameter with a clean base and raised borders. The chancre is painless unless it is secondarily infected, and it may be accompanied by painless lymphadenopathy.

The secondary stage usually follows the primary stage by 6 weeks or more but rarely overlaps primary syphilis. There are a number of cutaneous manifestations of secondary syphilis. Lesions may be erythematous or pink macules or papules, usually with a generalized symmetric distribution (Fig. 120-19). Pigmented macules and papules classically appear on the palms and soles (Figs. 120-20 and 120-21). The lesions may be scaly but are rarely pruritic. Papular, annular, and circinate lesions are more common in people of color. Generalized lymphadenopathy and malaise accompany the skin lesions. Irregular, patchy alopecia may be seen. Moist, flat, verrucous condyloma latum may appear in the genital area. These lesions are highly contagious.

#### Diagnostic Strategies

The diagnosis of primary syphilis is made primarily by the identification of spirochetes with darkfield microscopy. Because a darkfield microscope is often not available to the emergency physician, the diagnosis of primary syphilis must be suspected on clinical grounds and the patient referred to a dermatologist or appropriate public agency for diagnosis and treatment. The result of the Venereal Disease Research Laboratory (VDRL) test, the most commonly used diagnostic serologic test, is positive in approximately three fourths of patients with primary syphilis, but the test result tends to be negative early in the course of the disease.
The VDRL test result is invariably positive in cases of secondary syphilis, usually in titers of 1:16 or greater. The findings on dark-field examination of moist lesions may also be positive, but the diagnosis in this stage is based on a positive serologic test result. The most specific and sensitive serologic test is the fluorescent treponemal antibody absorption (FTA-ABS) test.

A biologic false-positive serologic test response for syphilis is defined as a positive VDRL test result with a negative FTA-ABS test result. This situation is seen acutely after vaccination or infections, especially mycoplasmal pneumonia, mononucleosis, hepatitis, measles, varicella, and malaria, and in pregnancy. Chronic biologic false-positive reactions (i.e., those lasting longer than 6 months) may occur with systemic lupus erythematosus, thyroiditis, lymphoma, and narcotic addiction or in elderly patients. Most false-positive reactions are in low titer ranges of 1:1 to 1:4.

Management

Guidelines for syphilis treatment, including in penicillin allergic individuals, are available through www.cdc.gov. Benzathine penicillin (Bicillin L-A) is the preferred agent and is recommended whenever possible. Protocols are available for skin testing and inpatient desensitization if allergy to penicillin is evident. Pregnant patients must be treated with penicillin to prevent transplacental transmission. In penicillin allergic individuals, doxycycline 100 mg twice daily or tetracycline 500 mg four times a day for 14 days can be used. Whereas azithromycin 2 g single dose is usually effective, treatment failures have been reported. Primary and secondary syphilis is treated with benzathine penicillin G in a dose of 2.4 million units IM. HIV-infected patients require more intensive therapy. Patients with latent syphilis are treated the same as patients with primary disease; latent syphilis and tertiary syphilis are treated with benzathine penicillin G, three doses of 2.4 million units IM at weekly intervals for a total of 7.2 million units. Treatment of neurosyphilis requires infusion of aqueous crystalline penicillin, 3 to 4 million units IV every 4 hours for 10 to 14 days.

Treatment may be administered in the ED if the diagnosis can be made on clinical, microscopic, or serologic grounds. If this cannot be done, a serologic sample should be drawn and the patient referred for treatment. The VDRL test response may be expected to return to nonreactive 6 to 12 months after the treatment of primary disease or 1 to 1½ years after the treatment of secondary disease. Patients with tertiary syphilis who are adequately treated may nevertheless retain a positive serologic result. Within 12 hours of receiving therapy, patients may experience a febrile reaction and diffuse rash called the Jarisch-Herxheimer reaction; thus it is best to warn patients of this possibility. The reaction resolves spontaneously, usually within 24 hours.

A number of diseases are associated with erythema nodosum; these include tuberculosis, sarcoidosis, coccidioidomycosis, histoplasmosis, ulcerative colitis, regional enteritis, pregnancy, and infections with streptococci, Yersinia enterocolitica, and Chlamydia. As with erythema multiforme, many cases of erythema nodosum are idiopathic. The relationship of drugs to erythema nodosum was noted in the section on drug eruption. Oral contraceptive agents are a leading cause of drug-induced cases. The differential diagnosis includes traumatic bruises and subcutaneous fat necrosis.

Management

Management begins with treatment of identified underlying conditions. Chest radiography may be considered to rule out sarcoidosis, tuberculosis, or deep fungal infection. Bed rest, elevation of the legs, and wearing of elastic stockings reduce pain and edema. Aspirin in a dosage of 650 mg every 4 hours or other nonsteroidal anti-inflammatory agents may also afford some relief. Erythema nodosum is a self-limited process that usually resolves in 3 to 8 weeks. Patients with severe pain may be treated with 360 to 900 divided three times daily of potassium iodide daily for 3 or 4 weeks. Stopping of therapy before this time may result in a relapse. Potassium iodide may act through an immunosuppressive mechanism mediated by heparin release from mast cells.

VESICULAR LESIONS

Perspective

Vesicles are elevated lesions that contain clear fluid. Vesicles larger than 1 cm are known as bullae. Vesicles may sometimes be associated with red papular lesions, as in contact dermatitis or erythema multiforme.

Pemphigus Vulgaris

Clinical Features

Pemphigus vulgaris is an uncommon but important dermatologic disorder. The mortality rate before the use of steroids was approximately 95%. The current mortality rate is 10 to 15%, related more to steroid-induced complications than to the disease. Pemphigus is a bullous disease, affecting both sexes equally, and is most common in patients 40 to 60 years old. The disease is mostly prevalent in people of Jewish, Mediterranean, or south Asian descent.
Herpes Simplex

Perspective

Two known variants of HSV cause human infection: HSV-1 and HSV-2. HSV-1 primarily affects nongenital sites, whereas lesions caused by HSV-2 are found predominantly in the genital area and are transmitted primarily by venereal contact.

Clinical Features

The hallmark of skin infection with HSV is painful, grouped vesicles on an erythematous base (Fig. 120-24). Those above the waist are usually caused by HSV-1, whereas those below the waist generally result from HSV-2. The lesions are usually localized in a nondermatomal distribution. The skin distribution may become more generalized in patients with atopic eczema and other dermatoses. Adults with HSV infection should avoid contact with children with atopic dermatitis, especially in the first 3 to 5 days of infection.

The mouth is the most common site of HSV-1 infections. Children are affected more commonly than adults are. Small clusters of vesicles appear but are soon broken, leaving irregularly shaped, crusted erosions (Fig. 120-25). The severity of gingivostomatitis varies from the presence of small ulcers to extensive ulceration of the mouth, tongue, and gums accompanied by fever and cervical lymphadenopathy. The infection may be so severe that oral fluid intake is difficult, and dehydration may result. Healing typically

Management

Pain control and local wound care are essential components of therapy. Once the diagnosis is made, treatment with oral glucocorticoids in initial doses of 100 to 300 mg of prednisone, or an equivalent drug, should be instituted in conjunction with a dermatologist. Other immunosuppressant drugs may also be used. Despite the condition’s localization to the skin and mucous membranes, death was the rule before treatment with steroids, and the mortality rate continues to be substantial. Deaths are related to an uncontrolled spread of the disease, secondary infection, dehydration, and thromboembolism. Other medical illnesses as well as the side effects of high-dosage corticosteroids also contribute to mortality.
occurs in 7 to 14 days unless a secondary infection with streptococci or staphylococci occurs.

HSV-2 infections in men are seen with either single or multiple vesicles on the shaft or glans penis. Fever, malaise, and regional adenopathy may be present. A prodrome of local pain and hyperesthesia may precede the appearance of the cutaneous lesions. The vesicles erode after several days, become crusted, and heal in 10 to 14 days. Infections in women involve the introitus, cervix, or vagina. Vesicles may be grouped or confluent. Herpetic cervicitis or vaginitis may be the cause of severe pelvic pain, dysuria, or vaginal discharge. Recurrence is common, but recurrent episodes tend to be less severe. A correlation based on serologic and epidemiologic data has been discovered between HSV-2 reproductive tract infections and carcinoma of the cervix.

Management

Recommended treatment for a first clinical episode of genital herpes is with acyclovir (Zovirax) 200 mg orally five times a day for 7 to 10 days, famciclovir 250 mg three times a day, or valacyclovir 1000 mg orally twice a day for 7 to 10 days or until clinical resolution occurs. These agents reduce the duration of viral shedding, accelerate healing, and shorten the duration of symptoms, but they have not succeeded in preventing recurrent episodes. Prophylactic administration of acyclovir may be effective in ameliorating the severity of recurrent genital herpes, but the effects of long-term administration are unknown. Although many episodes of recurrent herpes infection do not benefit from acyclovir therapy, 200 mg five times a day may be given orally for recurrences at the beginning of the prodrome. Famiciclovir, 125 mg twice a day for 5 days, and valacyclovir, 500 mg three times a day for 3 days, are equally effective.

Severe initial attacks of genital herpes have been successfully treated with the intravenous infusion of acyclovir. Admission to the hospital is required, however, because such treatment is necessary for several days, especially for the immunocompromised patient. A mucocutaneous herpes infection in such patients is potentially fatal because it has a propensity for generalization and dissemination to the internal organs.

Any vesicular eruption on skin or mucous membranes in a neonate should prompt concern for HSV infection in that there is a high likelihood of dissemination in this group. Unless an alternative diagnosis is established, urgent testing of the vesicle’s fluid for HSV along with acyclovir therapy is needed.

Supportive care is important and pain control is a major concern. Systemic analgesics and topical anesthetic agents may be useful. Education of the patient about the prevention or spread of the disease during sexual contact and the birth process is imperative.

Varicella

Clinical Features

Varicella, or chickenpox, is an infection caused by the varicella-zoster virus. After an incubation period of 14 to 21 days, the illness begins with a low-grade fever, headache, and malaise. The exanthem coincides with these symptoms in children and follows them by 1 or 2 days in adults.

The skin lesions rapidly progress from macules to papules to vesicles to crusting, sometimes within 6 to 8 hours. The vesicle of varicella is 2 or 3 mm in diameter and surrounded by an erythematous border (Fig. 120-26). An unusual form of varicella has larger bullae (Fig. 120-27). The drying of the vesicle begins centrally, producing umbilication. The dried scabs fall off in 5 to 20 days.

Lesions appear in crops on the trunk, where they are seen in the highest concentration, and on the scalp, face, and extremities. The hallmark of varicella is the appearance of lesions in all stages of development in one region of the body. Extensive eruptions are often associated with a high and prolonged fever.

Complications of chickenpox include encephalitis or meningitis, pneumonia, staphylococcal or streptococcal cellulitis, thrombocytopenia, arthritis, hepatitis, and glomerulonephritis. Varicella pneumonia occurs more commonly in adults than in children.

Management

The illness is self-limited, and treatment is symptomatic only. Salicylates should be avoided in patients with chickenpox to minimize the risk of subsequent Reye’s syndrome. Oral acyclovir may be effective if it can be started within 24 hours of development of rash for patients with chronic respiratory or skin disease. Some studies report a diminution in duration and magnitude of fever and number and duration of lesions with the early use of acyclovir.45

Isolation of infected patients is often futile because the disease may be transmitted before the diagnosis is clinically evident. Because the disease has the potential to be contagious until all vesicles are crusted and dried, infected persons should be kept at home until this stage is reached.

Varicella-zoster and varicella titers should be checked in pregnant women and immunocompromised patients who are exposed to chickenpox, and if the response is negative, varicella-zoster immune globulin is recommended within 96 hours of exposure.
Fetal infection after maternal varicella in the first or early second trimester of pregnancy may result in varicella embryopathy, a condition characterized by limb atrophy, scarring on extremities, and CNS and ocular manifestations. Maternal varicella that occurs between 5 days before delivery and 2 days after delivery may result in disseminated herpes in the newborn.

The varicella vaccine is a live attenuated virus; it is highly efficacious and very safe. A single dose is effective in children between the ages of 1 and 13 years. For older children, two doses separated by 4 to 8 weeks are recommended. In addition, the incidence of zoster occurring after vaccination appears to be lower than that of naturally acquired disease.

**Herpes Zoster**

**Clinical Features**

Herpes zoster, or shingles, is an infection caused by the varicella-zoster virus. It occurs exclusively in individuals who have previously had chickenpox. Before the rash appears, the patient typically develops pain in a dermatomal distribution. This pain precedes the eruption by 1 to 10 days and is variable in intensity; it may be described as sharp, dull, or burning in quality. The rash consists of grouped vesicles on an erythematous base involving one or several dermatomes. The thorax is involved in most cases, and the trigeminal distribution is the next most commonly involved region.

The vesicles initially appear clear and then become cloudy and progress to scab and crust formation. This process takes 10 to 12 days, and the crusts fall off in 2 or 3 weeks (Figs. 120-28 and 120-29). Herpes zoster has a peak incidence in patients 50 to 70 years old and is unusual in children. Although the association with leukemia, Hodgkin’s lymphoma, and other malignant neoplasms is well known, rarely does the appearance antedate the diagnosis of such diseases. Most cases of herpes zoster occur in healthy individuals.

Herpes zoster may be transmitted from patients with chickenpox to susceptible individuals. Chickenpox may also be acquired by contact with shingles, although this is less common. It is generally believed, however, that herpes zoster is caused by a reactivation of latent varicella-zoster virus present since the initial infection with chickenpox. During the latent period between the two illnesses, the virus is thought to reside in dorsal root ganglion cells.

Herpes zoster has a very low mortality rate and is rarely life-threatening, except when dissemination to the visceral organs occurs. Complications include CNS involvement, ocular infection, and neuralgia. Meningoencephalitis, myelitis, and peripheral neuropathy have been reported.

Ocular complications occur in 20 to 70% of cases involving the ophthalmic division of the trigeminal nerve. The severity varies from mild conjunctivitis to panophthalmitis, which threatens the eye. Eye involvement may produce anterior uveitis, secondary glaucoma, and corneal scarring. There is a close correlation between eye involvement and vesicles located at the tip of the nose.

Postherpetic neuralgia (pain that persists after the lesions have healed) occurs more commonly in elders and immunosuppressed patients. It may last a number of months and is often resistant to treatment with standard analgesic medications.

Herpes zoster generally tends to be more severe in immunosuppressed patients, especially those with AIDS, Hodgkin’s disease, or other lymphomas. Cutaneous dissemination occurs more commonly in these patients than in the general population. Visceral and CNS dissemination is also more likely to occur in these patients; therefore, they should be considered for hospitalization.

**Management**

Treatment other than analgesia is rarely necessary. Burow’s solution compresses diluted 1:20 to 1:40 in water may be applied to hasten drying. Early systemic corticosteroid therapy may shorten the duration of postherpetic neuralgia but does not lessen the severity of pain or the rate of the healing of the lesions. Antiviral chemotherapy, with acyclovir, famciclovir, vidarabine, foscarnet, valacyclovir, and interferon alfa, has been shown to be effective for immunocompromised patients. Postherpetic neuralgia is a complicated problem with few satisfactory solutions. Some success has been achieved with capsaicin cream, but this cannot be applied to inflamed or eroded skin.

Intravenous administration of acyclovir may be of some benefit in the treatment of severe ocular herpes zoster. Treatment includes mydriasis and the application of topical corticosteroids. Unlike the situation with herpes simplex conjunctivitis, eye involvement caused by herpes zoster does not appear to be exacerbated by corticosteroids. Immunization against herpes zoster is recommended in older adults.

**Smallpox**

The last naturally occurring case of smallpox was in Somalia in 1977. Subsequently, the routine vaccination of the general public was stopped. Except for laboratory stockpiles, the variola virus had been eliminated. Because of recent concerns about biologic agents...
ILLNESS INCLUDE AIDS, SARCOIDOSIS, DIABETES MELLITUS, CONNECTIVE TISSUE DISEASES, AND ENDOCRINE DISORDERS.

**CLINICAL FEATURES OF LESIONS ASSOCIATED WITH INTERNAL MALIGNANT DISEASE**

Cutaneous lesions most directly indicative of an internal malignant disease arise from the extension of the tumor to the skin or by hematogenous or lymphatic metastasis. The neoplasms that most commonly produce such a cutaneous extension are lymphomas, leukemias, and carcinomas of the breast, gastrointestinal tract, lung, ovary, prostate, uterus, and bladder. Skin metastases generally signify a poor prognosis.

**Acanthosis Nigricans**

Acanthosis nigricans is associated with internal malignant disease, despite the fact that most patients do not have tumors. Benign cases may be familial or related to endocrine disease or obesity. The term malignant acanthosis nigricans is used to designate the form associated with neoplastic disease. This phrasing is misleading because acanthosis nigricans is only a marker of the underlying disease and is never infiltrated with malignant cells.

The lesion appears as a hyperpigmented verrucous, velvet-like hyperplasia and hypertrophy of the skin accompanied with accentuation of the skin markings. The chief sites of involvement are the body folds, especially the axillae, antecubital fossae, neck, and groin.

More than 90% of cases of malignant acanthosis nigricans are associated with intra-abdominal malignant neoplasms. Regardless of the tumor type, acanthosis nigricans is associated with tumors that are usually highly malignant and metastasize early. The mechanism of this dermatosis in cases of internal malignant disease is postulated to be a result of tumor products that bind to and stimulate insulin-like growth factors in the skin.

**Dermatomyositis**

The incidence of dermatomyositis with malignant disease ranges from 6 to 55% and is generally higher in older patients. In younger individuals, the appearance of dermatomyositis does not necessarily call for a tumor workup. Tumors commonly associated with dermatomyositis are carcinomas of the breast, ovary, and gastrointestinal and female genital tracts. Polymyositis occurring alone as weapons, it is important that smallpox be differentiated from chickenpox (Table 120-3; Fig. 120-30).

**Cutaneous Anthrax**

Cutaneous anthrax begins as a pruritic pustule or vesicle that enlarges and erodes during 1 or 2 days. Subsequently, a necrotic ulcer with central black eschar is formed. The lesion may be painless and may be surrounded by significant edema (Fig. 120-31).

### Table 120-3 Differentiation of Chickenpox from Smallpox

<table>
<thead>
<tr>
<th></th>
<th>CHICKENPOX</th>
<th>SMALLPOX</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prodromal signs and symptoms</td>
<td>Prodromal signs and symptoms absent or mild</td>
<td>1 to 4 days of systemic signs and symptoms before onset of rash</td>
</tr>
<tr>
<td>Illness severity</td>
<td>Illness usually not severe unless complications or immunosuppressed</td>
<td>Very ill from onset, may be toxic</td>
</tr>
<tr>
<td>Lesion development</td>
<td>Superficial vesicles developing rapidly (1 day) and in multiple stages in each affected area</td>
<td>Hard, circumscribed pustules developing slowly (during days); lesions in same stage in every affected area</td>
</tr>
<tr>
<td>Lesion locations</td>
<td>Commonly on face and trunk, <em>not</em> palms and soles</td>
<td>Commonly on face and extremities, including palms and soles</td>
</tr>
<tr>
<td>Contagiousness</td>
<td>Contagious until all lesions crusted over</td>
<td>Contagious until <em>all</em> scabs have fallen off</td>
</tr>
</tbody>
</table>

Numerous systemic illnesses have cutaneous manifestations (Table 120-4; Figs. 120-32 to 120-39). Some of the most common illnesses include AIDS, sarcoidosis, diabetes mellitus, connective tissue diseases, and endocrine disorders.
<table>
<thead>
<tr>
<th>DISEASE</th>
<th>LESIONS</th>
<th>COMMENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>AIDS</td>
<td>Chronic ulcerative herpes simplex</td>
<td>Diagnostic for AIDS</td>
</tr>
<tr>
<td></td>
<td>Kaposi’s sarcoma (Figs. 120-32 and 120-33)</td>
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<td></td>
<td>Severe herpes zoster</td>
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<td></td>
<td>Oral hairy leukoplakia</td>
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<td></td>
<td>Genital warts</td>
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<td></td>
<td>Molluscum contagiosum (Fig. 120-34)</td>
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<td></td>
<td>Seborrheic dermatitis, <em>Pityrosporum</em></td>
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<td></td>
<td>Recurrent staphylococcal abscesses</td>
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<td></td>
<td>Mycobacterial papules, nodules, abscesses</td>
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<td></td>
<td>Oral and rectal squamous cell carcinoma</td>
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<td></td>
<td>Lymphoma</td>
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<td></td>
<td>Severe psoriasis</td>
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<td>Acquired ichthyosis</td>
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<td></td>
<td>Folliculitis</td>
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<td>Human papillomavirus infection</td>
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<td></td>
<td>Lichenoid photoeruptions</td>
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<td></td>
<td>Diabetic dermopathy</td>
<td>Most common</td>
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<td></td>
<td>Necrobiosis lipoidica diabetorum</td>
<td>Most characteristic</td>
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<td></td>
<td>Cellulitis (Fig. 120-35)</td>
<td>Control of diabetes does not affect presence</td>
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<td></td>
<td>Vascular ulceration (Fig. 120-36)</td>
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<tr>
<td></td>
<td>Acanthosis nigricans</td>
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<td></td>
<td>Bullous diabetorum</td>
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<td></td>
<td>Diabetic thick skin</td>
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<tr>
<td>Dermatomyositis</td>
<td>Heliotrope discoloration and edema of eyelids</td>
<td>Skin lesions may precede muscle disease</td>
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<td>Scaly erythema of malar prominences</td>
<td>Symmetric proximal weakness, remissions, exacerbations</td>
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<tr>
<td></td>
<td>Erythematous dermatitis over joint extensor surfaces, especially hands (Fig. 120-37)</td>
<td>Increased creatine kinase and aldolase with active disease</td>
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<td></td>
<td>Raynaud’s phenomenon</td>
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<tr>
<td>Systemic lupus erythematosus</td>
<td>Discoid lesions</td>
<td>Patients with cutaneous discoid lupus generally have benign diseases</td>
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<td></td>
<td>Malar erythema (Fig. 120-38)</td>
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<td></td>
<td>Hypertrophic or verrucous palm and sole lesions</td>
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<td>Lupus panniculitis</td>
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<td>Oral ulcers</td>
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<td>Raynaud’s phenomenon</td>
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<td>Rheumatoid arthritis</td>
<td>Rheumatoid nodules and necrobiosis</td>
<td>Still’s disease</td>
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<td>Vasculitic lesions</td>
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<td></td>
<td>Pyoderma gangrenosum</td>
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<td></td>
<td>Urticaria</td>
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<tr>
<td>Hyperthyroidism</td>
<td>Fine, velvety, smooth skin</td>
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<td></td>
<td>Increased sweating</td>
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<td>Hyperpigmentation or hypopigmentation</td>
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<td>Pretibial edema</td>
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<td>Alopecia</td>
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<td>Onychosis</td>
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<td></td>
<td>Urticaria</td>
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<td>Hypothyroidism</td>
<td>Dry, coarse skin</td>
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<td></td>
<td>Myxedema (Fig. 120-39)</td>
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<td>Carotene color</td>
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<td>Pruritus</td>
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<td>Atopic dermatitis</td>
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<td></td>
<td>Ichthyosis</td>
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<td>Erythema nodosum</td>
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<td>Easy bruising</td>
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<td></td>
<td>Alopecia (lateral third of eyebrows)</td>
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<td>Ulcerative colitis</td>
<td>Pyoderma gangrenosum</td>
<td>Associated with state of disease</td>
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<td></td>
<td>Erythema nodosum</td>
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<td></td>
<td>Aphthous stomatitis</td>
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</tbody>
</table>

**Table 120-4 Skin Lesions Associated with Systemic Disease**
Figure 120-32. Kaposi's sarcoma associated with AIDS. (Courtesy David Effron, MD.)

Figure 120-33. Kaposi's sarcoma in an AIDS patient. (Courtesy David Effron, MD.)

Figure 120-34. Molluscum contagiosum caused by a virus is more prevalent with AIDS. (Courtesy David Effron, MD.)

Figure 120-35. Gangrene of the toe with cellulitis in a diabetic patient. (Courtesy David Effron, MD.)

Figure 120-36. Vascular ulceration secondary to diabetes. (Courtesy David Effron, MD.)

Figure 120-37. Erythematous dermatitis over the joint extensor surfaces, dermatomyositis. (Courtesy David Effron, MD.)
hyperkeratosis of the palms and soles. Hodgkin’s disease is the most common malignant disease associated with the nonfamilial form of ichthyosis. Non-Hodgkin’s lymphoma and carcinomas of the breast, lung, colon, and cervix have also been associated with acquired ichthyosis.

**Pruritus**

Itching may be an important indicator of Hodgkin’s disease, leukemia, adenocarcinoma or squamous cell carcinoma of various organs, carcinoid syndrome, multiple myeloma, and polycythemia vera. It may appear years before the underlying malignant disease is identified. In cases of Hodgkin’s disease, the itching is usually continuous and may be accompanied by a severe burning sensation. Although it is usually generalized, pruritus commonly begins in the feet and may be limited to the lower extremities. It may be intractable and associated with urticaria, erythroderma, excoriation, or lichenification.

The pruritus of leukemia and systemic carcinoma is generally less severe than that found with Hodgkin’s disease. Nevertheless, itching associated with internal malignant disease may be difficult to control. Conventional anti-H1 antihistamines, cimetidine, and cholestyramine have each been used with variable results. On occasion, only the suppression of the tumor is beneficial.

**Purpura**

Purpura is the most common manifestation of acute granulocytic and monocytic leukemia. It may also be associated with myeloma, lymphoma, and polycythemia vera. Although the most common cause of purpura in these conditions is thrombocytopenia secondary to bone marrow infiltration, in some instances the platelet count is normal and the causative mechanism obscure. Purpura is caused by vascular abnormalities, thrombocytopenia, or other coagulation defects. A variety of diseases and conditions may be the underlying cause, and the treatment should be directed toward this cause whenever possible (Boxes 120-1 and 120-2). Thrombocytopenic and nonthrombocytopenic forms are differentiated by the results of the patient’s platelet count. Serious bleeding seldom occurs if the platelet count is higher than 50,000/mm³. If the platelet count is less than 10,000/mm³ or serious bleeding is encountered, platelet transfusion should be initiated. Because of the short circulating half-life of infused platelets, transfusion should be used as a short-term measure only.

**Erythema Multiforme**

Erythema multiforme may be associated with acute forms of leukemia. It should be differentiated from other skin manifestations of leukemia, such as necrotizing vasculitis.

**Erythema Nodosum**

Erythema nodosum is another reaction found in association with leukemia and Hodgkin’s lymphoma as well as with metastatic carcinoma and inflammatory bowel disease.

**Erythroderma**

Generalized erythroderma is seen in Hodgkin’s disease; it is also a skin manifestation of lymphocytic leukemia. Although less common, it is also seen with other forms of leukemia, carcinoma, and mycosis fungoides. The appearance of erythroderma may precede the diagnosis of internal malignant disease by many years. The skin eruption is invariably accompanied by intractable pruritus.

**Acquired Ichthyosis**

Acquired ichthyosis is a skin condition manifested as generalized dryness of the skin, scaling, and superficial cracking or as without the accompanying skin findings is rarely associated with malignant neoplasms.46

**Box 120-1 Causes of Purpura**

**Thrombocytopenic**
- Aplastic anemia
- Drug induced
- Idiopathic
- Malignant disease
- Sarcoïdosis
- Splenomegaly
- Systemic lupus erythematosus
- Thrombotic
- Tuberculosis

**Nonthrombocytopenic**
- Drugs
- Infection (meningococcemia, Rocky Mountain spotted fever)
- Qualitative platelet defect
- Vasculitis

**Figure 120-38.** Malar erythema in a patient with systemic lupus erythematosus. (Courtesy David Effron, MD.)

**Figure 120-39.** Severe myxedema in a hypothyroid patient. (Courtesy David Effron, MD.)

**Erythema Multiforme**

Erythema multiforme may be associated with acute forms of leukemia. It should be differentiated from other skin manifestations of leukemia, such as necrotizing vasculitis.
Urticaria

Urticaria is occasionally found in Hodgkin’s disease and more rarely in leukemia and internal carcinoma. Cold urticaria may occur with multiple myeloma (Table 120-5).

CLINICAL FEATURES OF LESIONS ASSOCIATED WITH NARCOTIC ADDICTION

Individuals who inject opiates and other drugs parenterally develop characteristic skin lesions secondary to such use. Skin lesions have been most extensively described in heroin addicts. Skin tracks, or indurated linear hyperpigmented streaks, are produced by repeated intravenous injection (Fig. 120-40). They follow the course of the superficial veins used in the injection, most commonly in the antecubital fossae and the dorsa of the hands.

Subcutaneous injection results in round or oval hyperpigmented atrophic depressed scars 1 to 3 cm in diameter (Fig. 120-41). Abscesses, which often require drainage, commonly precede the development of such scars. Hypertrophic scarring and keloid formation may also occur. Increased pigmentation may occur in sun-exposed areas and at the site of tourniquet applications.

In addition to the characteristic skin lesions associated with drug injection, people who inject intravenous drugs are prone to sharp foreign body retention, pseudoaneurysm, gram-negative local and systemic infections, wound botulism (associated with the use of black tar heroin), and numerous other illnesses.

Physicians should become familiar with one or two topical steroid preparations of low, medium, and high potency to avoid prescribing errors.

Fungal infections involving the scalp and toenails require long-term systemic treatment. They do not respond to topical therapy.

It is essential to consider kerion in the differential diagnosis of inflammatory or purulent scalp lesions as incision is not indicated except in cases complicated with bacterial superinfection.

Newer nonsedating antihistamines are a useful alternative to older sedating ones to control pruritus and histamine-mediated rashes while allowing the patient to remain active.

Infection with *C. albicans* can occur normally during infancy and pregnancy and in obese people and elders. In other patients, the following underlying problems should be considered: AIDS and other immunodeficiency states, diabetes and other endocrine imbalances, malignant disease, malnutrition, and other debilitating illnesses.

Rashes that are associated with mucosal lesions, blisters, or desquamating skin are often caused by significant soft tissue infections, drug eruptions, or immune disorders.

Purpura results from leaking of blood from vessels into the skin and does not blanch when pressure is applied. Purpuric lesions of less than 3 mm in diameter are called petechiae. Nonpalpable purpuric lesions are often caused by coagulation defects (usually platelet abnormalities), whereas palpable purpuric lesions are usually a sign of vasculitis.

Diffuse pruritus in the absence of a rash may be a sign of underlying malignant disease or liver disease.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.
References