CHAPTER 36

Acute Skin Reactions and Bacterial Infections

Ellen Thompson and Andrea Kalus

Acute skin reactions are often symptomatic and account for many of the skin-related complaints of travelers. Sunburn and other ultraviolet (UV) light reactions, bacterial infections, medication-related rashes, dermatitis, and insect bites and stings are all common. These occur with brief exposure to environmental hazards or drugs. In many cases, they can be prevented.

SUNBURN AND OTHER ULTRAVIOLET LIGHT REACTIONS

The intensity of UV radiation from the sun is related to season, latitude, and time of day. The amount of UV light reaching the earth's surface is greater in the tropics. This can lead to *sunburn* or *photosensitive drug eruptions* or unmask or aggravate *photosensitive diseases*, such as solar urticaria, polymorphous light eruption, porphyria, discoid and systemic lupus erythematosus, and dermatomyositis. A number of medications when combined with UV light in vivo can cause photosensitive drug eruptions (see **Table 36.1**). UV light can also trigger recurrent *herpes simplex*. Over the long term, UV exposure promotes premature skin aging and skin cancer.

Etiology

Sunburn is chiefly caused by UVB radiation (290-320 nm), while photoaging and sun-related skin cancers is attributed to UVA radiation (320-400 nm). UVA also penetrates most glass, such as untreated windows of vehicles. Individual tolerance to sun exposure is a function of skin pigmentation, genetic ability to synthesize melanin in response to UV light, and metabolic and pharmacologic factors.

Prevention

Avoidance of the sun is the ultimate protection against UV-induced conditions. The highest intensity of light occurs between 10 a.m. and 4 p.m., so limiting UV exposure during this period is helpful. Brimmed hats, long-sleeved shirts, and long pants can cover the largest areas of exposed skin. Lightweight, UV-protectant clothing designed for hot weather is available at most sporting goods stores. Sunglasses with UV-protectant lenses will limit sun-related effects on the eyes, which can sometimes go unnoticed. Sunglasses without UV protection, meanwhile, can contribute to increased sun damage to the eyes.

When shading exposed areas is not possible, sunscreen can be used to protect the skin. Choose a product with a sun protection factor (SPF) of 30 and look for sunscreens labeled as "broad spectrum." This indicates they provide protection against both UVA and UVB wavelengths. Products may be labeled "water resistant" for either 40 or 80 minutes but then lose effectiveness. Optimal protection requires application 15-20 minutes before sun exposure. The product should be applied liberally; 1 ounce is recommended for a full-body application. The average person uses less than half of the recommended amount to achieve the advertised protection. Reapplication during the day is necessary to maintain protection, particularly after swimming or perspiring heavily. Lip protection against UV light can be provided with specially formulated lip sunscreens.

TABLE 36.1 Drug Eruption Patterns

Reaction Type	Onset	Features	Common Causes
Exanthematous (maculopapular)	About a week after introduction of a new drug	Most common, starts on the trunk with centrifugal spread. Often accompanied by low fever, pruritus. Resolves in 1-2 weeks.	Antibiotics: penicillins, cephalosporins, sulfamethoxazole NSAIDs, some anti-epileptics, among many others
Photosensitive	3-6 h after sun exposure; phototoxic within 1 day of exposure; photoallergic	Sun-exposed distribution with sharp outlines. Naturally shaded areas spared (upper eyelids and below chin and nose). May appear as exaggerated sunburn or vesicular rash.	*Antibiotics*: quinolones, sulfonamides, tetracyclines (esp. doxycycline), trimethoprim Antimalarials: chloroquine, quinine, quinidine. Antifungals: griseofulvin Antiemetics: prochlorperazine Diuretics: furosemide, thiazide NSAIDs Amiodarone Sunscreens: PABA, oxybenzone
Urticaria (hives), angioedema, anaphylaxis	Initial reaction appears in days, but repeated exposure shortens the onset to minutes to hours	Ranges in severity from urticarial to life-threatening angioedema (compromising airway) or anaphylaxis	Antibiotics (especially penicillins), NSAIDs, blood products, opioids/anesthetics, antifungals (fluconazole and ketoconazole); radiocontrast; vaccines containing egg protein; pollen vaccines; ACE inhibitors

ACE, Angiotensin converting enzyme; *NSAIDs*, nonsteroidal anti-inflammatory drugs; *PABA*, para-aminobenzoic acid.

Broad-spectrum coverage can be achieved by including complementary chemicals that absorb UVA and UVB light and/or by physical agents such as zinc oxide or titanium dioxide, which scatter UVA and UVB light. UVA protection is provided by avobenzone and oxybenzone, among other chemicals. UVB protection is provided by aminobenzoic acid, homosalate, octisalate, and octinoxate. Sun blocks often combine these chemicals and/or include titanium dioxide and zinc oxide to provide broad-spectrum coverage. Newer broad-spectrum agents are now available, such as Mexoryl. Some caution should be used with spray sunscreen. Dispersion by wind and failure to rub in sprayed product may lead to less photoprotection than is provided by traditional lotions (**Fig. 36.1**). Accidental inhalation of sprayed products presents another risk.

Self-tanning products (spray or creams) do not impact melanin synthesis and provide no sun protection. Pre-travel tanning is not recommended. It provides only minimal protection and can actually increase overall UV exposure.

Treatment

Sunburn treatment options are directed at symptom control and are minimally effective. The most important first step after a burn is to limit further UV exposure through implementing above-mentioned prevention methods of sun avoidance and sun protection.

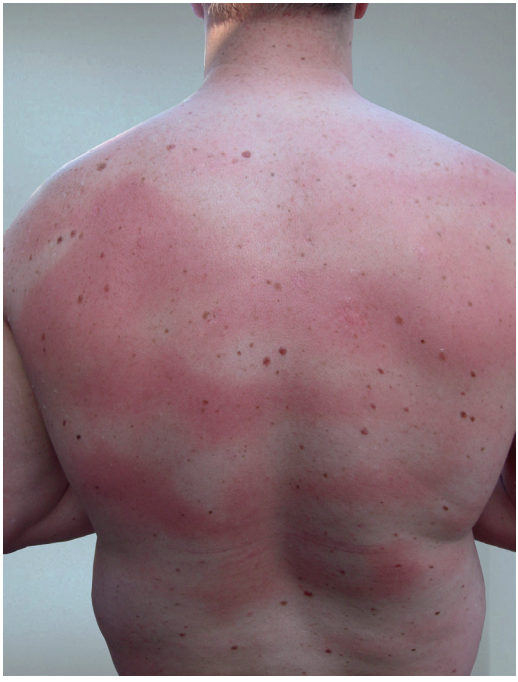

Fig. 36.1 Geographic sunburn after inadequate application of a spray sunscreen. (Courtesy of Corrine Hecht, MD.)

Mild sunburn is treated with nonsteroidal anti-inflammatory drugs and cool compresses, topical calamine, or aloe-based gels. Topical steroids have not demonstrated effectiveness in trials even though they are sometimes recommended.

Severe sunburn often does not respond to the aforementioned measures. Though not commercially available in the United States, topical indomethacin or diclofenac has been reported to be effective in reducing erythema and tenderness when applied soon after exposure. While prednisone seems to be ineffective at lessening the cutaneous sequelae of sunburn, it may improve the systemic symptoms of fever and headache that can occur with severe sunburn. Brief treatment with 40-60 mg/day for 3-4 days may be used.

DERMATITIS IN TRAVELERS

Dermatitis is a general term for superficial inflammation of the skin and can be further classified according to etiology and clinical features. Common types include atopic dermatitis, contact dermatitis, nummular dermatitis, seborrheic dermatitis, and hand or dyshidrotic dermatitis.

Contact dermatitis is responsible for the majority of new onset dermatitis in travelers. Contact dermatitis is further divided into irritant and allergic etiologies. *Irritant contact dermatitis* occurs on contact with an agent capable of causing injury and inflammation in most, if not all, people. *Allergic contact dermatitis* occurs in sensitized individuals (e.g., poison ivy or oak).

Etiology

Irritant contact dermatitis is caused by soaps, solvents, detergents, cleansers, cutting oils, and acid or alkaline solutions. Excessive hand washing is an example of chronic irritant contact dermatitis caused by soaps and frequent wetting of the skin. Individual susceptibility to any irritant varies greatly.

Allergic contact dermatitis can result from exposure to an enormous array of chemical compounds but requires specific sensitization. The most common sensitizers include nickel, fragrances, rubber, formaldehyde, paraphenylenediamine, ethylenediamine, and neomycin. It is important to remember that many products sold for use on the skin contain sensitizers such as neomycin, benzocaine, ethylenediamine, fragrances, and lanolin.

Some topical agents can act in concert with UV light to produce *photocontact dermatitis*. This includes fragrances and, ironically, sunscreen agents, such as PABA and oxybenzone, which can cause photosensitive reactions.

Plant dermatitis (*phytodermatitis*) is a subset of contact dermatitis of particular importance to those traveling to rural areas. Many plants in a number of different families produce sensitizing chemicals. Poison ivy, poison oak, and poison sumac are members of the family Anacardiaceae and produce a resin capable of sensitizing 70% of the population. Related plants containing cross-reacting chemicals include the Japanese lacquer tree (*Rhus verniciflua*), the India marking nut tree, raw cashew shells (*Anacardium occidentale*), mango rind (*Mangifera indica*), and the fruit of the ginkgo (*Ginkgo biloba*). Causes of plant dermatitis depend on the local flora. Mango dermatitis is the most common plant dermatitis in Hawaii. Philodendron is the most common cause in India. Primrose is a frequent sensitizer in Europe.

Phytophotodermatitis is a sun-induced plant dermatitis. Celery, limes, lemons, parsley, figs, and others contain natural psoralens that can incite a phototoxic reaction when contact with these plants is followed by sun exposure. Cases of systemic phototoxicity after ingestion of these plants followed by UV exposure have been reported.

Clinical Features

The primary lesions of dermatitis are erythematous papules and vesicles. In severe cases, bullae form and papules coalesce into plaques. Chronic lesions show scale, secondary changes of lichenification, and, sometimes, bacterial superinfection. Pruritus is a constant feature of dermatitis. Contact dermatitis is distributed in sites of contact. The thicker skin of the palm and soles is more resistant. Allergic reactions may be spread with the hands to other sites of the body, such as the face, eyelids, and genitals. Plant dermatitis classically shows linear blisters where the skin brushed against the causative plant.

Diagnosis

Dermatitis is usually diagnosed clinically. Establishing the cause of contact dermatitis requires a careful history on exposure to plants, soaps, chemicals, topical medications, and the activities associated with the dermatitis. Patch testing is invaluable in establishing the etiology of allergic contact dermatitis.

Differential diagnosis of dermatitis includes scabies, insect bites, drug eruptions, swimmer's itch (cercarial dermatitis), atopic dermatitis, and psoriasis.

Treatment

Dermatitis of any etiology is treated in a similar manner. Known offending agents are avoided and protective clothing or gloves may be helpful when the person must continue activities associated with the dermatitis. Mild cases of dermatitis are treated with mid-potency topical steroids. Weeping or exudative lesions should be compressed with tap water or Burow's solution (see Chapter 35) for 15-30 minutes, four times a day, followed by topical steroids. Severe contact dermatitis is treated with prednisone 40-60 mg/day (adult dose), tapering the dose over 2-3 weeks. In allergic contact dermatitis, stopping prednisone too soon often results in recurrence. If a sensitized person contacts a known allergen, the skin should be promptly washed with soap and water. If secondary bacterial infection is present, it should be treated with antibiotics effective for *Staphylococcus* and *Streptococcus* spp.

DRUG ERUPTIONS

Cutaneous reactions to drugs are common and can be serious. Travelers frequently take new medications, either as prophylaxis or as therapy for acquired symptoms. Consider an at-home trial of drugs prior to travel. New reactions from long-standing medications may also occur due to intense tropical sunlight. Patients who are told that their medications place them at higher-than-usual risk of photoreactions may be motivated to take extra UV precautions, so providers should review medication lists with this issue in mind before their patients depart.

Clinical Features

Common presentations and etiologies of drug eruptions are listed in **Table 36.1**.

Diagnosis

Take a careful history, inquiring about over-the-counter and herbal/complementary medicines, including the date started. Exanthematous eruptions may be initially confused with viral exanthems. Urticaria may be caused by foods, parasitic and viral infections, or physical agents and often occurs idiopathically. The differential diagnosis of drug-induced photoeruptions includes other photosensitive rashes, such as polymorphous light eruption, lupus erythematosus, and some porphyrias.

Treatment

- Discontinue non-essential drugs.
- Antihistamines
  - Hydroxyzine: 25 mg by mouth every 4-6 hours
  - Diphenhydramine: 25-50 mg by mouth every 4-6 hours
  - Cetirizine: 10 mg/day (a less sedating option).
- Topical steroids for symptomatic relief
- Refer urgently to dermatology if mucous membranes are involved (mouth, vagina, conjunctiva) or if skin blisters.

ARTHROPOD BITES AND STINGS

Arthropod bites and stings are common dermatologic complaints of tropical travelers. Clinical symptoms result from hypersensitivity to arthropod antigens, toxic effects of venoms, or both. Important venom-producing arthropods include some species of spiders, Hymenoptera (bees and wasps), ants, centipedes, and scorpions. Nonvenom-producing, biting arthropods include species of flies, mosquitoes, bedbugs, fleas, mites, lice, and ticks (see also Chapter 20 and Chapter 24 for discussion of tick-borne tropical infections).

Etiology and Clinical Features

There are many biting *spiders*, but several are worth special mention. The black widow spider, *Latrodectus mactans*, and other *Latrodectus* spp. are found worldwide. *L. mactans* is best identified by the red hourglass shape on the ventral abdomen. The bite is often painless, but a neurotoxin, α-lactotoxin, can cause systemic symptoms including muscle spasms, headache, abdominal pain, nausea, and hypotension, which can progress to shock and death.

Loxosceles spp., including the brown recluse spider, are found in North and South America, have a violin shape on the cephalothorax and produce venom containing sphingomyelinase D, causing extensive local skin and soft tissue necrosis. *Brown recluse spider* bites cause a mild urticarial reaction in the majority of cases. Some bites cause severe local reactions characterized by an expanding bulla with surrounding pallor followed by cyanosis and necrosis within 48-72 hours. Systemic involvement is rare but may be fatal and includes disseminated intravascular coagulation, hemolysis, and renal failure.

The wandering spider, *Phoneutria nigriventer*, is a large South American spider measuring 3 cm in body length and produces a potent neurotoxin.

Hymenoptera include bees, wasps, hornets, yellow jackets, and ants and cause painful reactions from venom injected from a posterior stinger. Half of the fatal envenomations in

the United States are due to Hymenoptera. Honeybees produce venom containing histamine, phospholipase A, hyaluronidase, and other constituents. Stings produce a painful wheal that subsides over a few hours. In sensitized individuals, urticaria, laryngeal edema, bronchospasm, and anaphylaxis begin immediately. Less common systemic manifestations include toxic reactions from large numbers of simultaneous stings or a serum sickness-like syndrome that follows the sting by days to weeks.

Solenopsis invicta, the imported fire ant, is common in the southern United States, Argentina, Uruguay, and Brazil. *Fire ant* stings may cause anaphylaxis but usually produce local reactions. Stings cause an immediate wheal, which becomes a vesicle and then a sterile pustule over 12-24 hours.

Many species of *scorpions* are found in arid regions of the tropics and subtropics. Fatal stings are usually seen in children in areas where species produce neurotoxic venom. Scorpion stings cause a painful local reaction, occasionally with some necrosis. Some species produce venom that can induce sympathomimetic, parasympathomimetic, and neurologic symptoms. Infants and children are at highest risk for fatal reactions. Antivenoms are available for some scorpion toxins; however, there are often little data on their benefits, and risks include anaphylactic reaction.

Other arthropods that normally cause more localized responses include the *Scolopendra* centipedes of Hawaii and western United States, biting flies of many varieties found worldwide, fleas (including *Tunga penetrans*; see Chapter 37), bedbugs, and human ectoparasites (Chapter 37). *Bedbug bites* usually occur on the face, arms, ankles, or buttocks and are often arranged in a cluster or line of two or three bites. The appearance of individual bites ranges from small hemorrhagic puncta to papular urticaria that last for several days. The bites themselves occur at night and are typically painless. Bedbugs move from crevices in furniture to the human host for only a few minutes to feed and then retreat.

In addition to bites and stings, simple contact with Lepidoptera (butterflies, moths, and their caterpillars) can cause a pruritic erythematous papular rash within hours of contact. Caterpillar and moth species worldwide have been linked to reactions including conjunctivitis, keratitis, iritis, and pharyngitis.

One particularly dangerous species, the *Lonomia obliqua*, or giant silkworm moth of South America, can cause a fibrinolytic reaction similar to disseminated intravascular coagulation, which can be fatal. Another dangerous species, the *Premolis* caterpillar, lives on rubber trees in the Amazon. Repeated contact with its bristles can cause a periarticular fibrosis with permanent disfiguration.

Diagnosis

Stings usually present no problem with diagnosis because of the immediate pain. Bites may be more difficult to diagnose if the injury is painless or occurs during sleep. The pruritic papules of typical bites may resemble other hypersensitivity reactions, such as urticaria or dermatitis. Patients presenting with an unwitnessed "spider bite" often have community-acquired methicillin-resistant Staphylococcus aureus (MRSA) skin infection. Bullous or necrotic reactions may mimic other rashes. Definitive diagnosis is possible only if the bite is observed. In unresolved situations a biopsy may be helpful.

Treatment

Most papular and mild bullous reactions are self-limited and can be treated with compresses and topical steroids or oral antihistamines for pruritus. As with all penetrating skin injuries, tetanus prophylaxis should be given if the patient's status is not up-to-date.

Angioedema can be treated with prednisone 30-40 mg/day for several days. More severe generalized urticarial reactions or anaphylaxis are treated promptly with epinephrine 0.3-0.5 mg subcutaneously, repeated every 15-20 minutes. Most patients respond within one or two doses. Intravenous epinephrine may be necessary if hypotension persists despite these measures.

Black widow spider reactions may require hospitalization and treatment with analgesics, muscle relaxants, intravenous calcium, and supportive care. An equine antivenom is available in several countries.

Brown recluse spider bites are usually benign and can be treated symptomatically. Ice and elevation are used for symptomatic relief. Dapsone may prove useful to treat related tissue necrosis. To prevent treatment toxicity, glucose 6-phosphate dehydrogenase status should be evaluated prior to treatment with dapsone. A surgical approach is potentially harmful and should be avoided. Systemic reactions may be life threatening and require hospitalization for supportive care. Antivenom is not available in the United States but is available for South American recluse bites where the reaction is often more severe.

Scorpion stings are treated by local wound care, ice packs, and antihistamines. The Food and Drug Administration recently approved the scorpion antivenom Anascorp, or Centruroides immune F(ab)2, for envenomations resulting in serious clinical symptoms. Antihypertensives or anticonvulsants may be needed for supportive care.

Hymenoptera stingers (from bees, yellow jackets, wasps, or fire ants) should be removed as quickly as possible. While forceps are suggested, they should be applied to the shaft of the stinger immediately proximal to the skin, to avoid squeezing more venom into the patient from the venom sack attached to the stinger. When no instruments are available, an alternative is to scrape off the stinger.

Butterfly and moth (Lepidoptera) bristles, often invisible, can be removed with cellophane tape. Clothing that has been in contact with the moth or caterpillar should be removed and washed, and exposed skin should be washed.

Prevention

People with a history of severe sting reactions at risk of further exposure can undergo venom desensitization. All people with a history of severe reactions, whether or not they have undergone desensitization, should carry kits including antihistamines and a syringe of epinephrine. Epinephrine auto-injectors are commercially available and should be prescribed pre-departure. These devices are sensitive to high temperatures and should be kept within the temperature range on the package insert (e.g., never in a car parked in the sun).

Protective clothing, insect repellant, and even repellant-treated clothing can all help prevent some stings and bites (see Chapter 6). However, insect repellents have no effect on spiders or bees.

Control of bedbugs involves treating crevices in walls and furniture with an insecticide, such as 0.5% lindane or 2% malathion, or spraying bed nets with a permethrin-containing insecticide.

Bacterial Skin Infections

Bacterial infections of the skin are a major problem in the tropics and a common problem among travelers. One study of travelers returning to France found that bacterial infections were the second most common presenting skin condition after cutaneous larva migrans (Chapter 37).

The high prevalence of bacterial infections in tropical climates is attributed to the warm, humid environment and can be worsened by close contact and poor hygiene. Other predisposing factors include insect bites, traumatic lesions, and other dermatoses such as contact dermatitis and scabies. Infections caused by staphylococci and streptococci are far more common in the tropics than are the exotic "tropical diseases."

PYODERMA

Pyoderma refers to superficial bacterial infectious syndromes involving the skin and follicular structures such as impetigo (including bullous or ulcerative forms), folliculitis, furunculosis, paronychia, erysipelas, and cellulitis.

Etiology

Staphylococcus aureus (both MRSA and methicillin-susceptible [MSSA]) and group A streptococci (GAS) are common causes of pyoderma.

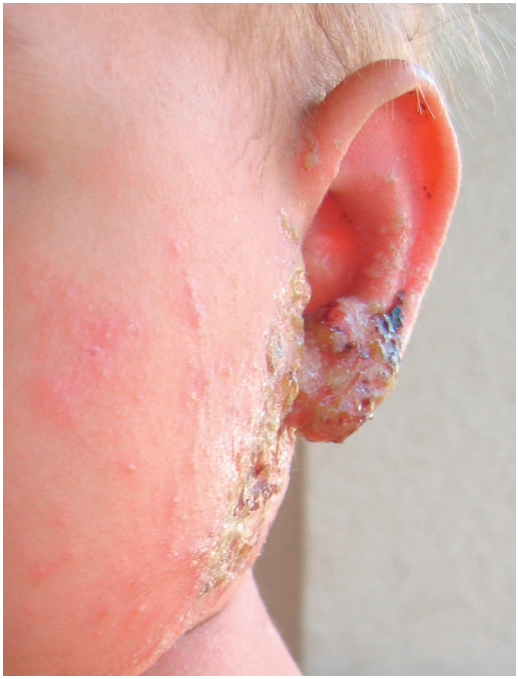

Fig. 36.2 Pustules and honey-colored crusting on the ear of a child with impetigo. (Courtesy of Michi Shinohara, MD.)

Clinical Features

Impetigo is an exceedingly common condition seen primarily in children. Invasion of the skin by pathogenic *S. aureus* or GAS often follows minor trauma. An isolated, erythematous papule or pustule accounts for the initial lesion. The primary lesion rapidly gives rise to a distinctive amber- or "honey"-crusted erosion with or without an erythematous border (**Fig. 36.2**). Pruritus may accompany the lesions, and regional lymphadenopathy may be present. The condition usually affects the central face around the nares and lips but can occur anywhere.

Bullous impetigo is a superficial blistering condition caused by the elaboration of a toxin *S. aureus*. Children are most frequently affected. Bullous impetigo presents as flaccid, well-demarcated bullae without surrounding erythema that arise rapidly from vesicles, often in intertriginous areas. The bullae rupture spontaneously in 1 or 2 days, leaving shallow erosions covered by a light brown, varnish-like crust.

Impetiginization describes when *S. aureus* or GAS secondarily infects skin that has been compromised by a pre-existing dermatosis. Atopic dermatitis, contact dermatitis, insect bites, dermatophyte infection, and infestations with mites or lice are frequent precursors. Lesions present as focal or widespread papules or plaques with honey-colored crusts. Close observation at the periphery may reveal the primary lesions of the underlying dermatosis.

Folliculitis is an infection of the hair follicle most often caused by *S. aureus*. Superficial folliculitis presents as follicle-based 1- to 2-mm pustules on an erythematous base, often with a central protruding hair shaft. The most frequent areas involved are the scalp, thighs,

buttocks, axillae, and, in men, the beard, where it is called folliculitis barbae or sycosis barbae.

Furunculosis refers to isolated or multiple cutaneous infections centered on hair follicles with pus extending through the dermis, forming an abscess in the subcutaneous tissue. They occur most often in areas of friction and/or perspiration such as the axillae and buttocks. They present as painful, erythematous papules or nodules, with or without an obvious central follicular ostium. After several days, the lesion may come to a point and drain purulent material. A carbuncle refers to several communicating furuncles. Both furuncles and carbuncles are commonly referred to as "boils."

Ecthyma is often a deeper extension of untreated impetigo or folliculitis that presents as 5- to 15-mm punched-out erosions with elevated, erythematous borders. A densely adherent, thick serum crust overlies each lesion, giving a characteristic appearance. Regional lymphadenopathy is common. Lesions are most common on the buttocks and legs but can occur anywhere.

Acute paronychia is a suppurative infection of the proximal and lateral nail folds. It often follows a break in the skin resulting from minor trauma. *S. aureus* or *S. pyogenes* are the most common pathogens in acute paronychia. The presentation is that of an exquisitely tender, hot, erythematous nail fold, with or without frank abscess formation.

Erysipelas is an infection of the skin and superficial lymphatic channels usually due to GAS. It classically presents as a well-demarcated, brightly erythematous, hot, tender indurated plaque on the face or lower extremities. The pathogenic organisms gain entry into the skin via minor trauma, including pre-existing dermatitis.

Cellulitis may result from untreated erysipelas, but usually arises de novo, and is an infection of the deeper cutaneous and subcutaneous tissues. Cellulitis differs from erysipelas clinically by having indistinct borders and less pronounced brawny edema. Either GAS or *S. aureus* can cause cellulitis, although in the absence of a drainable focus of pus, GAS is the leading cause.

Diagnosis

Diagnosis of the various pyodermas is usually made primarily on clinical findings, and cultures are often unnecessary. Impetigo contagiosa may be difficult to differentiate from an exudative dermatitis, which may also have a crust. Bullous impetigo should be distinguished from other blistering disorders, such as bullous arthropod bites, pemphigus vulgaris, bullous pemphigoid, acute vesicular dermatitis, erythema multiforme, and bullous drug reactions.

Folliculitis, although most often caused by Gram-positive cocci, has many causes including chemical irritants, yeasts (*Candida* and *Malassezia*), Gram-negative bacteria (*Pseudomonas* spp., so-called hot tub folliculitis), dermatophytes, herpes simplex virus, pseudofolliculitis barbae, and various drug eruptions.

Hidradenitis suppurativa should be considered if furuncular lesions are localized to the axillae, groin, or intergluteal cleft. Furuncular myiasis (see Chapter 37) must be considered in anyone presenting with furunculosis after travel to an endemic area.

Treatment

See **Figure 36.3.**

- Superficial infections: topical antimicrobials such as mupirocin applied several times a day is recommended, as well as warm compresses that promote drainage. This treats both GAS and *S. aureus* infections.
- Nonpurulent skin infections (e.g., cellulitis): GAS should be covered. A first-generation cephalosporin or a penicillinase-resistant penicillin is first-line therapy. Clindamycin may be substituted for patients with a true beta-lactam allergy (although these are actually rare).
- Purulent skin infections: *S. aureus* (both MRSA and MSSA) is most likely. Incision and drainage may be necessary—and sufficient—for infections involving furuncles or abscess. Incision and drainage alone is almost always appropriate for staphylococcal abscesses or boils, and patients with these very rarely require antibiotics at all. However, patients

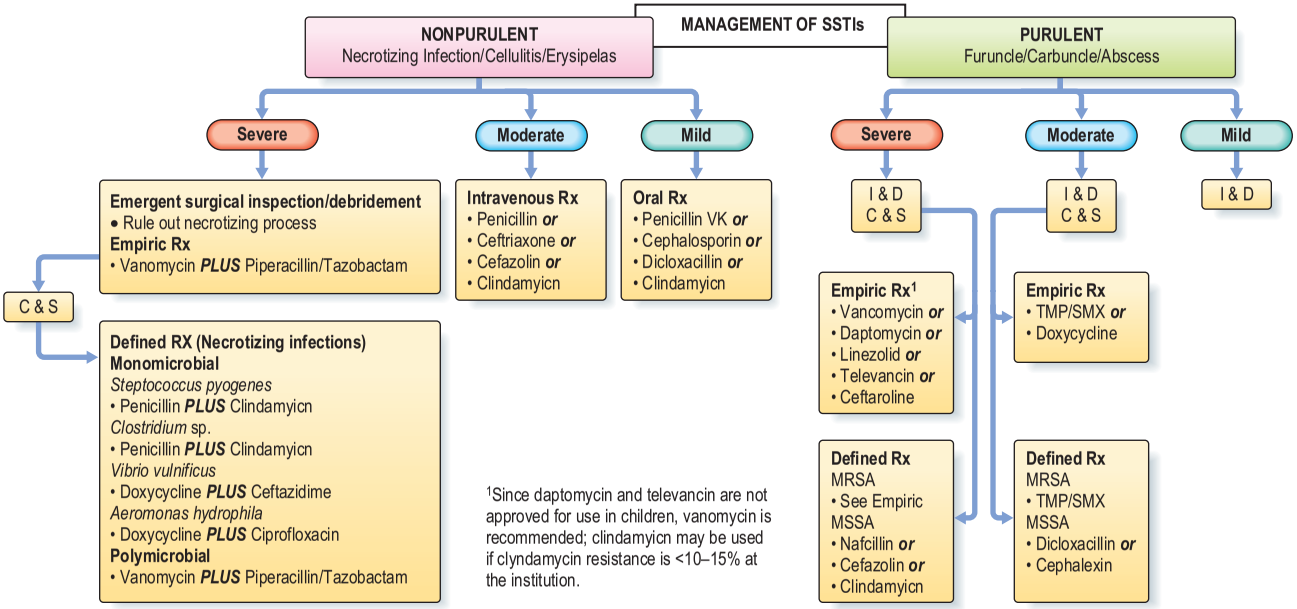

Fig. 36.3 **Infectious Diseases Society of America algorithm guidelines. *C & S*, Culture and sensitivity; *I & D*, incision and drainage; *MRSA*, methicillin-resistant *Staphylococcus aureus*; *MSSA*, methicillin-susceptible *S. aureus*; *SSTIs*, skin and soft tissue infections; *TMP/SMX*, trimethoprim-sulfamethoxazole.** (From: Stevens, D.L., Bisno, A.L., Chambers, H.F., et al., 2014. Practice guidelines for the diagnosis and management of skin and soft tissue infections: 2014 update by the Infectious Diseases Society of America. Clin. Infect. Dis. 59 (2), 147–159.)

presenting with multiple lesions, large areas of cellulitis, immune suppression, and any systemic symptoms of disease should be treated with systemic antibiotic therapy, such as empiric trimethoprim-sulfamethoxazole, or a tetracycline, plus a first-generation cephalosporin. Clindamycin alone provides less reliable MRSA coverage. Pus should be sent for culture and sensitivity, and antibiotics focused based on results. Parenteral antibiotics may be necessary in people showing systemic disease (fever and chills).

PYOMYOSITIS

Etiology

Pyomyositis, or purulent infection of skeletal muscle, is caused predominately by *S. aureus* but may also be due to streptococci along with other more rare causes.

Epidemiology

Pyomyositis occurs in tropical areas and is a rare but increasing problem in temperate climates. Most cases occur in children or young adults; males are affected more often than females. Up to 50% of cases are associated with a history of trauma to the affected muscles. Other predisposing factors include toxocariasis, immunodeficiency (specifically human immunodeficiency virus), malnutrition, and injection drug use.

Clinical Features

The characteristic features of pyomyositis are pain and tenderness of the involved muscle, fever, and leukocytosis. The most frequently affected muscles include those of the thigh, calf, deltoid, buttocks, iliopsoas, pectoral, and latissimus dorsi. The first week or two of infection is characterized by localized pain, with "woody" muscle texture on palpation. Two to three weeks after initial symptoms present, fever and extreme muscle tenderness is common. Many patients seek care at this point. A deep abscess can often be detected, and pus can be drained from it. Without treatment patients can worsen to a toxic stage with many life-threatening complications including septic shock, septic emboli, endocarditis, pneumonia, and renal failure.

Diagnosis

Differential diagnosis includes muscle hematoma, deep venous thrombosis, thrombophlebitis, neoplasm, and sickle cell crisis. Iliopsoas pyomyositis may mimic appendicitis. Diagnosis is made by recovery of pus from the affected muscle by needle aspiration or surgical exploration. Magnetic resonance imaging, computed tomography, and ultrasound, especially during the purulent stage, aid in diagnosis.

Treatment

In the earliest stage of disease systemic antibiotics can be curative, but most patients present in later stages when treatment requires surgical drainage and parenteral antibiotics. Antibiotic therapy should be guided by cultures. Pending results, empirical therapy should be directed to cover *S. aureus*, including MRSA. In immunocompromised patients, broader coverage for Gram-negative and anaerobic organisms should be provided.

Skin Ulcers

Skin ulcers in the tropics may be the result of bacterial infections not often encountered in temperate climates. Six bacterial causes are discussed in the following section.

Buruli Ulcer

Buruli ulcer (also known as Bairnsdale ulcer) is a painless skin ulcer caused by a slow growing acid-fast bacillus, *Mycobacterium ulcerans*. It is the third most common mycobacterial disease and may soon overtake leprosy for overall worldwide disease burden.

Epidemiology

Buruli ulcer occurs in tropical, humid environments in areas with stagnant swampy water and often in rural and remote areas. It is most common in West Africa but also found in

Mexico, South America, Australia, Papua New Guinea, and Japan. Cases acquired by travelers have been reported. Disease rates in some endemic populations range as high as 16-22%. While the mode of transmission has not been established, it is thought to involve direct contact with contaminated soil or water. Young people are most often affected, with the usual age of infected persons ranging from 4 to 22 years.

Clinical Features

Lesions of Buruli ulcer present as firm, nontender, mobile subcutaneous nodules, usually on an extremity after local trauma. Pruritus often accompanies this early lesion. In 1 or 2 months, toxin production causes necrosis of the nodules and underlying tissue and an indolent, generally painless ulcer with indurated, undermined borders and substantial fat necrosis without involving deeper tissue. Ulcers heal spontaneously after months or years with significant scarring, contractures, and limb deformities. Infection is not accompanied by systemic illness or regional lymphadenopathy, and fatalities are rare.

Diagnosis

The diagnosis is often made clinically. The acid-fast bacilli may be demonstrated by Ziehl–Nielsen staining of swabs or biopsies from the undermined ulcer border. Cultures may take as long as 8 weeks to turn positive.

Differential diagnosis includes other infections such as tropical ulcer (see below), cutaneous tuberculosis, leprosy (Chapter 40), leishmaniasis (Chapter 39), and fungal infections (Chapter 38). Other non-infectious ulcerating conditions, such as pyoderma gangrenosum and venous stasis, should be considered.

Treatment

Size at presentation will determine treatment. Prolonged courses of antibiotics can be helpful and may be combined with surgery. Combination therapy with streptomycin, clarithromycin, fluoroquinolones, and rifampin has been effective. Early lesions may be treated by excision and closed primarily or with grafting.

Tropical Ulcer

Tropical ulcer (also called tropical phagedenic ulcer or Malabar ulcer) is a condition in which a large, painful, ulcer forms rapidly on areas of skin prone to trauma. The majority of tropical ulcers appear on the leg. The etiology of this condition is believed to be polymicrobial infection with anaerobes (especially *Fusobacterium* spp.) in early disease and spirochetes in later infection. Ulcer formation is associated with malnourishment and underlying chronic disease.

Epidemiology

Tropical ulcer occurs in most hot, humid tropical regions of the world, including Africa, India, and the Western Pacific region.

Clinical Features

Lesions are most common on the lower extremities, and usually solitary. Ulcers begin as an erythematous papule or hemorrhagic bulla that breaks down within 7-10 days to form a large, well-demarcated, cup-shaped ulcer with an indurated, undermined border. The ulcer is painful and foul-smelling, and its granulating base is often covered by a yellowish membrane. Left untreated, ulcers may be deep enough to involve periosteum of underlying bone. Patients may be febrile and systemically ill, but regional lymphadenopathy is rare. With time, the ulcer may heal with significant scarring and functional disability. In some cases, ulcers have been known to persist for 10 years or more.

Diagnosis

This is a diagnosis of exclusion of other skin infections such as those included in this section (especially Buruli ulcer) and conditions such as cutaneous tuberculous, leishmaniasis, venous stasis, and pyoderma gangrenosum.

Treatment

Underlying nutritional deficiencies or chronic disease should be addressed. Local care for the ulcer includes rest, elevation, and local wound care with appropriate bandages or

compresses. Antibiotics, most frequently penicillin, metronidazole, or a tetracycline, are administered until healing occurs. Reconstructive surgery and grafting may be required.

CUTANEOUS DIPHTHERIA

Diphtheria (veldt sore), while not commonly seen in temperate regions, is still endemic in many tropical countries.

Epidemiology

Cutaneous diphtheria often occurs in unvaccinated children and has been associated with skin trauma, often associated with infected insect bites. In some instances, the lesions act as reservoirs of the infectious agent and may cause respiratory and cutaneous infections in contacts. The bacteria may also be found in dust and fomites.

Clinical Features

Cutaneous diphtheria usually begins as a small papule or vesicle, often at the site of a minor skin wound. In 2-5 days the vesicle breaks down to form a well-demarcated shallow, painful, punched-out ulcer with elevated borders and surrounded by a rim of erythema. The base of the ulcer is covered by an adherent gray membrane. The ulcer may enlarge gradually and become anesthetic. The ulcer rarely exceeds 3 cm.

Diagnosis

Diagnosis can be confirmed by culture of swabs taken from the ulcer base. In chronic cases, a mixed infection is often present, and *Staphylococci* are frequently cultured from the same lesions.

Treatment

Treatment of cutaneous diphtheria consists of diphtheria antitoxin (if toxigenic) and a 2-week course of systemic antibiotics (erythromycin or penicillin). This is a reportable condition, and close contacts should be screened and offered prophylaxis with antibiotics and a diphtheria immunization booster if the index strain is found to be toxigenic.

CUTANEOUS TULAREMIA

Cutaneous tularemia (ulceroglandular fever) is the most common presentation of *Francisella tularensis*, a highly virulent, pleomorphic, Gram-negative coccobacillus. In the cutaneous form, the bacteria are transferred from infected animal reservoirs (often rabbits or hares), by the bite of an arthropod, or by direct contact with the blood or tissue of an infected animal.

Epidemiology

Tularemia is endemic in many areas of the northern hemisphere, including the United States, Canada, the Nordic countries, and Japan; it is also seen in Mexico and Central America. Many cases are reported in hunters, who become infected while skinning infected animals. The eyes may become involved if bacteria inoculate that area. Pneumonic tularemia is a serious but separate clinical entity, arising when bacteria are directly inhaled (e.g., infected ground rodent is aerosolized by a lawn mower).

Clinical Features

The incubation period of cutaneous tularemia is 3-5 days. The initial lesion, a red, painful nodule, usually appears 1 or 2 days after the onset of a high fever, headache, chills, myalgia, and prostration. Within a few days, the nodule rapidly becomes pustular and then breaks down to form a well-demarcated ulcer. The ulcer often heals spontaneously, leaving a small scar. Within a few days of the onset of the disease regional lymph node enlargement and tenderness develops (ulceroglandular tularemia), often prompting the patient to seek medical attention.

Diagnosis

The diagnosis of tularemia is suggested by the history of exposure to wild rabbits, rodents, or ticks and the associated finding of a small skin ulcer with central eschar and significant, tender lymphadenopathy.

Diagnosis can be confirmed by a serum microagglutination test or through identification (using fluorescent antibodies) of *F. tularensis* in smears from the base of the ulcer. Bacteria are highly virulent. Laboratory workers should be notified if cultures are to be attempted.

Treatment

- Mild disease: doxycycline 100 mg, twice daily for 14 days
- Moderate or severe disease: streptomycin 10 mg/kg intramuscularly every 12 hours for 7-10 days (not to exceed 2 mg/daily), or gentamicin 5 mg/kg daily in three divided doses intramuscularly or parenterally for 7-10 days.

CUTANEOUS ANTHRAX

Anthrax is a zoonotic illness caused by *Bacillus anthracis*, an aerobic spore-forming Gram-positive bacillus. There are three major clinical forms of the disease (cutaneous, pulmonary, and gastrointestinal), with the cutaneous form being the most common. Cutaneous anthrax is usually acquired from inoculation through a minor skin wound or abrasion, often during skinning sheep or other livestock. Although systemic forms of anthrax are often fatal, when treated appropriately the cutaneous type of the disease causes death in <1% of cases.

Epidemiology

Cutaneous anthrax has been found in tropical and subtropical regions of Africa, South and Central America, the Caribbean Islands, and the Philippines. Most cases are found in people with direct contact with animal hides.

Clinical Features

Cutaneous anthrax begins as a small painless skin nodule within 1 week after bacterial or spore invasion, usually through a minor wound. Lesions are found most commonly on the head or neck and the upper extremities. Within 2-3 days, the nodule becomes a blister, and then breaks down to form a shallow, painless ulcer with an edematous border. The ulcer base becomes covered by a characteristic black eschar. Systemic symptoms are not common, although regional lymph node enlargement is noted. In untreated cases, the eschar loosens and falls off after 2-3 weeks, and the ulcer then heals, usually without scar formation.

Diagnosis

The diagnosis is suggested by the clinical appearance of the lesion, especially the presence of a typical black eschar covering the ulcer. It can be confirmed by the demonstration of the Gram-positive bacilli in a smear taken from the ulcer under the eschar or in tissue removed by biopsy. *B. anthracis* may also be cultured; immunohistochemical stains are available for its identification in tissue specimens.

Treatment

Skin infection caused by *B. anthracis* is susceptible to most antibiotics. A 7- to 10-day course of ciprofloxacin (500 mg twice daily) or doxycycline (100 mg twice daily) is recommended. Infection cannot spread from person to person, thus no special precautions are indicated when caring for patients with any form of anthrax. However, laboratory personnel should be notified when this condition is suspected, because it can be spread when grown in culture.

CUTANEOUS MELIOIDOSIS

Skin lesions may be a prominent feature of melioidosis (also known as Whitmore disease), an infection caused by the pleomorphic Gram-negative bacillus *Burkholderia pseudomallei*. The

disease may be divided into three clinically distinct patterns: localized, septicemic, and pulmonary. Skin lesions are a feature of both the septicemic and acute localized forms.

B. pseudomallei have been isolated from soil, mud, and surface water and abound in rice paddies and marshes. Infection in humans is thought to arise mainly from bacteria in the environment that enter the skin through small wounds and abrasions.

Epidemiology

Melioidosis is endemic in several countries in Southeast Asia and is distributed widely elsewhere in the tropics, including Central and South America and the Caribbean Islands, Australia, Africa, and the Middle East. The disease has been increasingly recognized as a hazard to those who travel "off road" as backpackers or eco-tourists. Diabetes mellitus is a risk factor for the disease.

Clinical Features

The skin signs of melioidosis range from localized cutaneous ulcers thought to occur at the site of percutaneous inoculation to multiple pustules or caseous nodules that appear widely over the skin surface as an expression of the septicemic form of infection. The incubation period appears to be influenced by the amount of inoculum and may vary widely, although it generally falls within 2-21 days from the date of the initial inoculation.

The skin lesions in localized melioidosis usually take the form of an ulcerated indurated plaque on an exposed area of the body. These lesions tend to be chronic and often drain a serosanguineous fluid. Cutaneous melioidosis may also appear as widespread miliary pustules in the septicemic form of the disease. Rarely, skin lesions of melioidosis may present as deep subcutaneous abscesses.

Disease severity can range from subclinical and localized skin infection described above to systemic disease including high fever, rigors, and sometimes confusion, stupor, jaundice, and diarrhea. It can also reactivate body-wide at times of stress, as has occurred in American veterans of the Vietnam War years after their initial infection.

Diagnosis

The skin signs of cutaneous melioidosis are not specific, and several other infectious processes should be considered, including tularemia, nocardiosis, and anthrax. The diagnosis may be suspected, however, in patients who show chronic indurated draining skin ulcers, with relevant travel history.

Smears from ulcers or abscesses yield Gram-negative bacilli, which show a characteristic bipolar "safety pin" pattern with Wright's stain. *B. pseudomallei* also can be grown in culture, and an indirect hemagglutination assay may be available in the countries where the disease is endemic. Because it can be spread to laboratory personnel, they should always be notified when this diagnosis is considered.

Treatment

Although *B. pseudomallei* is usually susceptible to most antibiotics, it is recommended that specific susceptibility tests be done on each isolate. A treatment regimen of ceftazidime or imipenem (during an initial hospital stay) followed by an outpatient course of chloramphenicol, trimethoprim-sulfamethoxazole, and doxycycline, for up to 20 weeks, has been shown to reduce mortality by 50%. Healthcare workers should use standard precautions when caring for these patients.

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