

Chapter 35

Common Dermatologic Disorders

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In the United States, up to 60% of dermatologic disorders are treated by primary care physicians. Skin complaints account for approximately 5% of internal medicine visits. Rash and pruritis are particularly common complaints and frequently occur together. The presence or absence of pruritis with a primary skin lesion can dramatically change the differential diagnosis. Generalized pruritis can present as the only dermatological complaint signifying an underlying systemic disorder.

Evaluation

Obtain a detailed and targeted history including location of lesions, time of onset and evolution (acute or chronic), and any systemic symptoms such as fever, malaise, gastrointestinal, or upper respiratory symptoms. Rash accompanied by fever is common with disseminated infections (viral or bacterial), drug reactions, collagen vascular diseases, and vasculitis. Investigate external factors, such as new medications, foods, soaps, occupational, environmental, and sun exposures. Patients may self-treat skin eruptions or pruritis with over-the-counter remedies. Eliciting a detailed history of previous treatments may clarify altered natural progression of skin disorders.

It is imperative to perform an inspection of the entire skin and mucosal surfaces. Patients should be entirely undressed, wearing only a hospital gown. Natural light exposure is preferable. Skin palpation can help appreciate texture of the lesions, depth, and tenderness. Learning how to describe skin lesions is critical to

correctly communicate relevant findings. Several attributes of skin lesions are paramount, including location, color, border, type, and arrangement (Table 1).

Dermatitis (Eczematous Rashes)

Eczema denotes epidermal eruptions characterized histologically by intracellular edema. Atopic, contact, seborrheic, and stasis dermatitis are common types of eczema, whereas nonspecific dermatitis is a diagnosis of exclusion when no etiology is identified. Topical corticosteroids and oral antihistamines are usually the treatment of choice for any type of eczema.

Atopic dermatitis is an allergic disorder with genetic and immunologic components characterized by intense itching leading to excoriation, lichenification (epidermal thickening), hyperpigmentation, and papulosquamous eruption (Plate 2). Typical locations are the flexural areas, head, neck, trunk, and hands. Acute eruptions are often accompanied by erythema, vesiculation, and oozing. In addition to the standard treatment of dermatitis, skin moisturizers can be used to decrease dryness and pruritis.

Contact dermatitis is precipitated by local absorption of allergen or irritant through the stratum corneum. Common allergens include metals (e.g., nickel), topical anesthetics, neomycin, poison oak, poison ivy (Plate 3), and strong soaps or personal care products. The location of eruptions may help to identify causative agents (e.g., neck rash caused by a necklace). The mainstay of treatment is avoidance of the allergens and irritants.

Table 1. Dermatologic Lexicon

Description	Definition	Examples
Macule	Flat skin lesion <1 cm in diameter	Freckle
Patch	Flat skin lesion >1 cm in diameter	Tinea versicolor
Papule	Raised skin lesion <0.5 cm in diameter	Acne
Plaque	"Plateau-like" elevated lesion >0.5 cm in diameter	Psoriasis
Nodule	Raised sphere-like lesion >0.5 cm in diameter and depth; called a cyst when filled with liquid or keratin	Erythema nodosum
Vesicle	Blister filled with clear fluid <.5 cm in diameter	Varicella
Bulla	Blister filled with clear fluid >0.5 cm in diameter	Poison ivy
Pustule	Vesicle filled with pus	Folliculitis
Crust (scab)	Dried pus, blood and serum from breakage of vesicles, bullae, or pustules	Herpes zoster (shingles)
Scale	Dry, whitish, and flaky stratum corneum	Seborrheic dermatitis

Other common terms include *induration* (dermal thickening), *lichenification* (epidermal thickening), *atrophy* (loss of epidermal or dermal tissue), *wheel* (dermal edema), *comedones* (lesions of acne), *ulcer* (loss of epidermal tissue and some dermis), *erosion* (superficial loss of epidermis), and *fissure* (linear opening in epidermis).

Seborrheic dermatitis (Plate 4) is characterized by erythematous plaques with a dry or oily scale occurring in hair-bearing parts of the body including scalp (dandruff), eyelashes, eyebrows, beard, chest, the external ear canal and behind the ear, the forehead, and nasolabial folds. The precise etiology is unknown but appears to be related to colonization with the yeast *Malassezia furfur*. Scalp dermatitis is treated with selenium sulfide, coal tar, or ketoconazole shampoos. The face can be cleansed and treated with topical 1% hydrocortisone and 2% ketoconazole cream.

Bacterial Skin Infections

The most common skin infections observed in the outpatient setting include cellulitis, folliculitis, and impetigo. Increasingly these infections are caused by community-acquired methicillin-resistant *Staphylococcus aureus* (MRSA), especially in patients who have had close recent contact with persons having a history of a similar infection, including household and athletic team members, prison inmates, and military personnel.

Cellulitis is a deep skin (dermis) infection most frequently caused by *Staphylococcus aureus* or Group A streptococci. A well-demarcated area of warmth, swelling, tenderness and erythema is found on physical exam (Plate 5). Cellulitis can be differentiated from contact dermatitis by absence of vesicles or pruritis. Patients with cellulitis are treated with oral antibiotics and analgesics; intravenous antibiotics may be necessary for failed outpatient treatment (Table 2).

Folliculitis and furunculosis are pustular skin infections arising from hair follicles and caused by *Staphylococcus aureus* and, less frequently, Group A *Streptococcus* (Plate 6). Topical antibiotics

(e.g., mupirocin) and hot compresses may be sufficient for treatment. Furuncles (boils) are pus-filled nodules requiring incision, drainage and systemic antibiotics. Elimination of *S. aureus* nasal carriage using topical or intranasal antibiotics may decrease relapses.

Impetigo is a superficial (epidermis) skin infection that typically presents as a group of crusted pustules caused by beta-hemolytic streptococci and *Staphylococcus aureus* (Plate 7). Cephalexin, erythromycin, or dicloxacillin eradicates the lesion in 90% of cases; so does topical mupirocin ointment. Herpes simplex can be confused with facial impetigo; a history of recurrent vesicles occurring in the same location is consistent with herpes simplex.

Fungal Infections

Dermatophyte organisms are fungi infecting the stratum corneum. Tinea pedis is the most common dermatophyte infection and often presents as chronic maceration, fissuring, and scaling between the toes. Some patients have a chronic moccasin-type, or hyperkeratotic form of infection; the soles are involved with fine silvery scale extending from sole to heel and sides of the feet (Plate 8). Tinea pedis can be associated with recurrent cellulitis. Treatment consists of antifungal creams (e.g., clotrimazole, terbinafine); oral therapy is sometimes necessary with moccasin-type tinea pedis or more extensive disease.

Tinea versicolor is caused by the lipophilic yeast *Malassezia furfur*. The typical clinical features include nonpruritic, light brown or reddish brown macules or hypopigmented macules in darker persons (Plate 9). The characteristic distribution of tinea versicolor is on the chest, back, lower neck, and proximal upper

Table 2. Antibiotic Therapy for Cellulitis

Disease	Notes
Mild, uncomplicated cellulitis at low risk for MRSA (oral treatment)	<ul style="list-style-type: none"> • Dicloxacillin or cloxacillin • First-generation cephalosporin (cephalexin or cefadroxil) • Clindamycin or macrolide (erythromycin, azithromycin, clarithromycin) if allergic to penicillin • An advanced fluoroquinolone (moxifloxacin, levofloxacin) if intolerant to the above drugs
Mild, uncomplicated cellulitis at risk for MRSA infection (oral treatment). In outpatients, consider recent close contact with persons having similar infection including household members, athletic team members, prisoners, and military personnel.	<ul style="list-style-type: none"> • Trimethoprim-sulfamethoxazole* • Clindamycin (if sensitive) • Minocycline or doxycycline
Moderate-to-severe cellulitis with systemic manifestations of infection at low risk for MRSA (parenteral treatment)	<ul style="list-style-type: none"> • Semisynthetic penicillin (nafcillin, oxacillin) • Cephalosporin (cefazolin or cephalothin) • If penicillin-allergic: clindamycin; fluoroquinolone (moxifloxacin, levofloxacin); advanced macrolide (clarithromycin, azithromycin); oxazolidinone (linezolid); vancomycin; daptomycin; tigecycline
Moderate-to-severe cellulitis with systemic manifestations of infection at risk for MRSA (parenteral treatment). Risk categories include recent antibiotic use, recent hospitalization, hemodialysis, illicit IV drug use, diabetes, and previous MRSA infection or colonization.	<ul style="list-style-type: none"> • Clindamycin (if sensitive) • Vancomycin • Linezolid • Daptomycin • Tigecycline

*For non-purulent cellulitis, trimethoprim-sulfamethoxazole is not recommended because the likely cause, Group A streptococci, is in most cases resistant to this antimicrobial agent. MRSA = methicillin-resistant *Staphylococcus aureus*.

extremities. Diagnosis is confirmed by microscopic evaluation of a potassium hydroxide preparation made from scrapings of lesions showing the classic appearance of both spores and hyphae in a “spaghetti and meatball” pattern. Therapy with topical 2.5% selenium sulfide solution or oral, single-dose ketoconazole are equally effective. Infection recurs in 60% to 80% requiring retreatment.

Onychomycosis is a fungal infection of the nail caused by dermatophytes, yeasts, or molds (Plate 10). It is common, increasing in prevalence with age. Up to half of all patients with thickened toenails have a nonfungal cause for the nail thickening such as psoriasis, subungual warts, lichen planus, and bacterial infections. Fungal infection of the nail should be confirmed with culture of nail debris or clippings before starting therapy. Treatment of onychomycosis is recommended in patients with peripheral vascular disease or diabetes mellitus to prevent development of cellulitis. Oral therapy with itraconazole or terbinafine is effective and preferred; topical therapy is ineffective. Treatment is usually for 2-3 months with close monitoring for adverse reactions (heart failure with itraconazole and hepatotoxicity with terbinafine).

Candidiasis is an inflammatory reaction to *Candida albicans* infection. It usually occurs in moist areas such as skin folds, under the breasts, perianally, and in axillae causing itching and burning (Plate 11). On physical exam, infected areas appear erythematous with scattered satellite papules and pustules. Candidiasis can often be confused with intertrigo, which is an irritant skin fold dermatitis from moisture and rubbing, especially prominent in obese patients. Intertrigo usually lacks the same degree of redness and satellite lesions found in candidiasis and a microscopic examination of a potassium hydroxide preparation will not demonstrate hyphae and pseudohyphae that is diagnostic of candidiasis. Candidiasis is treated with topical nystatin or imidazole creams and attention to hygiene, especially dryness of affected areas.

Acne

Acne vulgaris usually begins at puberty and results from hyperkeratinization of follicles, increased sebum production, proliferation of *Propionibacterium acnes*, and resulting inflammation (Plate 12). Acne is classified by severity and type as comedonal-only acne, mild-to-moderate inflammatory acne, moderate-to-severe inflammatory acne, and severe papulonodular inflammatory acne. Acne is exacerbated by creams and lotions, mechanical trauma (rubbing from clothing, picking, and repetitive scrubbing), medications (e.g., prednisone and progesterone), and sweating. Polycystic ovary syndrome should be considered in women with acne, hirsutism, and acanthosis nigricans (hyperpigmentation of flexural folds). Patients should be advised to use noncomedogenic oil-free make-up and sunscreen; dietary changes do not affect acne. Comedonal acne is treated with topical benzoyl peroxide and a topical antibiotic. Papular and pustular acne are treated with combination topical benzoyl peroxide/topical antibiotic and topical isotretinoin, adapalene, salicylic acid, or azelaic acid. If topical therapy is ineffective, systemic antibiotic therapy is indicated, followed by treatment with 0.05% isotretinoin if necessary. Oral contraceptives and spironolactone are effective for hormonal acne. Metformin may decrease acne in polycystic ovary syndrome. Tretinoin (Accutane) is the only therapy that alters the natural

history but is teratogenic and is given only under the supervision of a dermatologist.

Acne rosacea is a chronic inflammatory skin disorder of unknown etiology affecting the face, typically the cheeks and nose, usually occurring after the age of 30 years (Plate 13). On physical examination there is erythema with telangiectasias, pustules, and papules without comedones. Rosacea can be differentiated from seborrheic dermatitis by the presence of pustules. In early stages, rosacea can present with only facial erythema and resemble the butterfly rash of systemic lupus erythematosus; however, the rash of systemic lupus erythematosus typically spares the nasal labial folds and areas under the nose and lower lip (Plate 14). Rhinophyma (big irregular hyperplastic nose) can develop in some rosacea patients (Plate 15). Treatment consists of metronidazole gel, low-dose oral tetracycline, or erythromycin.

Viral Skin Infections

Herpes zoster (shingles) is caused by reactivation of varicella zoster virus in older or immunosuppressed patients. Patients may report localized sensations ranging from mild itching or tingling to severe pain that precedes the development of the skin lesions by 1-5 days. Skin changes begin with an erythematous maculopapular rash followed by the appearance of clear vesicles in a dermatomal distribution; the vesicles become pustular and eventually crust with healing (Plate 16). Bacterial superinfection of cutaneous lesions occasionally occurs. Patients are treated with an oral antiviral (i.e., acyclovir, valacyclovir, or famciclovir); treatment is most effective if begun within 48-72 hours of the onset of rash. The addition of corticosteroids to antiviral therapy reduces the duration of acute neuritis but not the incidence of postherpetic neuralgia. Postherpetic neuralgia is a painful sequela of shingles treated with amitriptyline, gabapentin, or long-acting opioids. There is a 10% to 20% lifetime incidence of herpes zoster in those who have had chickenpox, and the incidence increases with age. Immunization with VZV (varicella zoster virus) live vaccine of immunocompetent adults older than age 60 years is currently recommended in order to reduce the incidence and severity of zoster and postherpetic neuralgia.

Herpes labialis is caused by reactivation of herpes simplex virus type I (HSV-1). Primary infection with HSV-1 is asymptomatic in up to 90% patients but may present as acute, painful gingivostomatitis (Plate 17). Recurrent infections are characterized by grouped vesicles on an erythematous base located on the lips (“cold sores”) and usually recur in the same location, lasting 5-10 days. Treatment with oral or topical antiviral drugs does not shorten the duration. In patients with clearly identifiable prodromal symptoms of localized itching or burning, early treatment before the outbreak of vesicles reduces the duration of the rash by 1-2 days but does not prevent recurrences.

Benign Growths

Seborrheic keratosis is a painless, non-malignant growth appearing as a waxy, brownish patch or plaque (Plate 18). These lesions are more common in elderly patients and do not require treatment except for cosmetic reasons.

Warts (*verruca vulgaris*) are caused by infection of the epithelial tissues with human papillomavirus (HPV) (Plate 19). Diagnosis is based on the typical verrucous appearance of a papule or plaque on hands or feet. Nongenital warts in immunocompetent patients are harmless and two-thirds resolve spontaneously by 2 years. Topical salicylic or lactic acid is available over-the-counter and hastens the resolution of 60% to 80% of warts. Cryotherapy combined with topical therapy is used for warts that do not respond to initial topical management.

Miscellaneous Skin Conditions

Psoriasis is a chronic, relapsing skin disorder characterized by discrete and well-demarcated raised plaques or papules covered by a silvery white scale on the scalp, extensor surface of the extremities, low back, and intergluteal cleft, and behind the ears (Plate 20). Symptoms are primarily cosmetic, although itching and pain occur. Nail pitting, onycholysis, subungual hyperkeratosis, or discoloration of the nail surface occurs in up to 50% of patients. Limited chronic plaque psoriasis can be managed with sunlight, topical corticosteroids, and tar shampoo or lotion if the scalp is involved. Topical corticosteroids should be discontinued slowly to avoid rebound of psoriasis. Chronic plaque psoriasis involving more than 10% of the body surface, and more severe forms of psoriasis should be referred to a dermatologist. Infection, particularly streptococcal infections of the upper respiratory tract; injury to the skin; and medication (e.g., lithium and β -blockers) can exacerbate psoriasis. An inflammatory, seronegative spondyloarthropathy, psoriatic arthritis, can occur in up to 25% of patients with psoriatic skin lesions (Plate 21). Inflammatory bowel disease—including ulcerative colitis and Crohn's disease—occurs more commonly in patients with psoriasis.

Urticaria (hives) are raised, intensely pruritic red lesions with sharp borders, typically 2–4 mm in diameter (Plate 22). In acute urticaria, individual lesions may last 30 minutes to 2 hours, and an episode usually resolves within a few days or weeks. In chronic urticaria, individual lesions may last 4 to 36 hours. An attack usually continues for several days or weeks, but may persist beyond 6 weeks in one third of the patients. Common causes of urticaria are medications, foods, viral or bacterial infection, latex or other physical contacts, and stinging insects. Indiscriminate laboratory testing is rarely helpful. Urticaria, in rare cases, can precede anaphylaxis or angioedema in complement-induced allergic reactions. The standard therapy for urticaria is antihistamines. If histamine-1 blockade is not sufficient, histamine-2 receptor or leukotriene antagonists can be added. Corticosteroids are reserved for severe symptoms unresponsive to maximal doses of antihistamines.

Alopecia consists of generalized or patchy hair loss, usually from the scalp, but occurring in other sites as well. Alopecia areata is a self-limiting condition associated with atopy and other autoimmune processes. Patients with alopecia areata have a well-demarcated, completely bald area with no signs of inflammation, desquamation, or scarring (Plate 23). Intralesional corticosteroids can help stimulate growth but are not routinely used. Androgen-dependent hair loss or male-pattern baldness is common, and its incidence increases with age. There is usually a genetic predisposition to the age of onset and severity of the baldness in these

patients. A small proportion of postmenopausal women may also have this type of baldness. Conventional medical treatments for male-pattern baldness include oral finasteride or topical preparations of 2% or 5% minoxidil. Other causes of alopecia include drug-induced alopecia, lichen planus, trichotillomania (neurotic hair pulling) or traction alopecia (e.g., tight braiding of hair), fungal or bacterial folliculitis, and discoid lupus erythematosus.

Scabies is caused by *Sarcoptes scabiei* var. *hominis*, an obligate human parasite preferentially affecting impoverished, immobilized or immunosuppressed persons. Spread is direct, personal contact, especially during sex. Acquisition from bedding or clothes is rare. Scabies infestation causes intense itching and a papular or vesicular rash (Plate 24). Burrows are visible as short, wavy lines. Location in the interdigital webs, flexure surface of the wrists, penis, axillae, nipples, umbilicus, scrotum, and buttocks is diagnostic.

Pediculosis (head lice) and scabies are closely related conditions caused by arthropods. Pruritus is the primary symptom of this disorder; excoriations and pyoderma may also occur. The diagnosis of head lice is established by identifying crawling lice in the scalp or hair. Lice egg cases are called *nits* and are found sticking to the hair shaft in patients with lice. Nits are generally easier to see than lice because they are often found in the occipital or retroauricular portions of the scalp. Permethrin is the treatment of choice for both scabies and head lice.

Dermatologic Signs of Systemic Disease

Erythema multiforme presenting as circular erythematous plaques with a raised, darker central circle (“target lesions”) is pathognomonic for this disorder (Plate 25). Palms and soles are frequently involved. Wide-spread blisters and mucosal lesions with systemic symptoms are seen in Stevens-Johnson syndrome, a severe form of erythema multiforme. Immunologic reaction to drugs or infections involving immune complexes in the skin is a possible etiology of the disease. No specific therapy is indicated for minor forms of erythema multiforme other than treating the underlying condition. Systemic corticosteroids and supportive measures are used to treat the Stevens-Johnson syndrome.

Erythema nodosum is characterized by tender, deep, erythematous nodules frequently limited to lower legs and often accompanied by fever and joint pain (Plate 26). Erythema nodosum is the result of a hypersensitivity immune reaction to infection or inflammation (e.g., streptococcal pharyngitis, sarcoidosis, inflammatory bowel disease) involving subcutaneous fat. The mainstay of management is treatment of underlying disease. Lesions usually resolve in several weeks with symptomatic relief from non-steroidal anti-inflammatory medications. Corticosteroids can be used if infectious etiology is ruled out.

Generalized pruritus without rash usually has local or systemic causes. In elderly patients, the most common cause of pruritus is xerosis (dry skin) (Plate 27). Frequent bathing, poor chronic hydration, and dry winter weather exacerbate dry skin and the accompanying itch. The treatment of xerosis should include advice about using a humidifier in dry weather conditions, avoiding excess bathing and scrubbing of skin, use of moisturizing soaps, and routine use of moisturizers and occlusives. Pruritus may be

caused by fleas or mites from pets, or by using new soap (contact dermatitis) or medication (allergic dermatitis). When more than one household member has pruritus, empiric treatment for scabies may be appropriate. Persistent pruritus in patients with no skin lesions warrants further evaluation for systemic causes, including hyperthyroidism, cholestasis (e.g., primary biliary cirrhosis), chronic renal failure, infection (e.g., HIV, hepatitis C), hematologic disease (e.g., polycythemia vera, lymphoma), and malignancy. Doxepin, a sedating antidepressant and antihistamine, may alleviate nocturnal pruritus. Nonsedating antihistamines are rarely beneficial for isolated pruritus.

Book Enhancement

Go to www.acponline.org/essentials/general-internal-medicine-section.html to view additional clinical images of common

dermatologic conditions and review tables on the differential diagnosis for psoriasis and herpes zoster, drug treatment for acne and herpes zoster, and the evaluation of pruritus. In *MKSAP for Students 4*, assess yourself with items 44-57 in the **General Internal Medicine** section.

Bibliography

American College of Physicians. Medical Knowledge Self-Assessment Program 14. Philadelphia: American College of Physicians; 2006.