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*Dermatologic complaints commonly are seen in emergency medicine and may pose a diagnostic dilemma for the clinician. Although a detailed understanding of all dermatologic conditions is beyond the scope of practice of emergency physicians, recognition of categories of disease, particularly emergent conditions, is essential. Knowledge of basic disease lesions, patterns, diagnostic tests, and emergent management is crucial to the appropriate treatment of patients with cutaneous disease. This article presents an organized approach to the diagnosis and management of cutaneous conditions, including brief discussions of selected dermatologic complaints.*

—The Editor

## Introduction

Dermatologic complaints in emergency medicine are common, both as the chief complaint or as discovered while performing the history and physical examination. The skin is the largest organ system and, likewise, the number of diseases potentially affecting the integument also is large. Some dermatologic disor-

ders are straightforward, but often it is difficult to sort out the etiology and management of a particular dermatologic complaint. Many dermatologic conditions initially are misdiagnosed.<sup>1</sup> A wide spectrum of dermatologic disorders, from the benign to life-

threatening skin disorders, may be encountered. This article is intended to provide a structured approach to common dermatologic presentations in the emergency department. The authors present a unique question-based approach to dermatologic complaints. (See Table 1.)

Dermatologic findings are particularly common among pediatric emergency patients. More than 50 infectious agents have been identified that may cause exanthems in the pediatric population.<sup>2</sup> One study demon-

strated that 72% of cases of fever and rash in the pediatric population were caused by viruses, and 20% by bacteria.<sup>3</sup> Many exanthems have specific patterns of lesions, distributions, and clinical history, and some of the more common exanthems are detailed in this article. However, many viral exanthems are nonspecific, and establishing a definitive etiology may not be feasible.

## Common Dermatologic Presentations in Emergency Medicine

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A sound understanding of the basic anatomy and physiology of the skin can be helpful when evaluating a dermatologic problem. The skin is comprised of three layers: the epidermis, dermis, and subcutaneous layer. (See Figure 1.)

The epidermis is a thin layer of stratified squamous epithelium mainly consisting of keratinocytes which progress through stages of differentiation as they move from the basal

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to the superficial layer. These layers are respectively named: stratum germinativum, stratum spinosum, stratum granulosum, and stratum corneum. These layers act as a barrier from the environment. The epidermis also includes melanocytes and Langerhans cells. Melanocytes produce the pigment melanin, which functions to absorb harmful ultraviolet radiation. Langerhans cells are a component of the immune system and, as part of their function, these cells ingest and process foreign antigens.

The epidermis is devoid of blood vessels and depends upon the dermis for nutrients and waste disposal by diffusion through the dermal-epidermal junction. This junction consists of two layers, the lamina lucida and the lamina densa. The interface is irregular, with many finger-like projections from both the epidermis and dermis that greatly increase surface area for diffusion. This junction also is the site of immunologic injury resulting in the bulla in bullous pemphigoid and epidermolysis bullosa.

The dermis is a thicker layer consisting of connective tissue, blood vessels, lymphatic vessels, nerve endings, immune cells, and epidermal appendages, all of which are surrounded by mucopolysaccharides. The epidermal appendages are epithelial lined structures with the ability to differentiate and form hair follicles, sweat glands, sebaceous glands, and apocrine glands. The main function of the dermis is to sustain the epidermis and contribute to the protective functions of the skin. Fibroblasts are the primary cell type in the dermis and are responsible for producing procollagen and elastic fibers used to form the connective tissues that give support and elasticity to the skin. Sweat glands and the vast network of blood vessels provide a thermoregulatory function.

The subcutaneous layer is composed of loose connective tissue and adipose tissue and functions to cushion the overlying skin and contains lymph and neurovascular structures.

### History and Physical Examination

As with all emergency department cases, emergency stabilization is the first priority. Airway, breathing, and circulation must be evaluated and addressed immediately. For example, a toxic, lethargic, or confused patient with a rash requires immediate action. For more routine dermatologic complaints, historical information can be valuable, and should include details including time of onset, duration of symptoms, and relation to any new potential allergens, such as foods, medications, soaps, pets, jewelry, etc. Information about evolution of the rash over time should be sought, including whether the rash has progressed, improved, or waxed and waned. Other historical features such as associated pain, pruritus, fever, sexual history, occupation, or hobbies may be relevant. Past medical history should be obtained, including such conditions as general medical conditions, previously diagnosed skin conditions, medications, illicit drug use, allergies, recent travel, sunlight exposure, and family history.

A detailed physical examination is essential. The physical examination of the skin should be performed in a private area

**Table 1. Eleven Questions to Lead Physicians to the Diagnosis and Management**

1. What is the time of onset?
2. Are there any associated historical features?
3. What is the patient's medical history?
4. What is the primary lesion?
5. What are the secondary lesions?
6. What is the distribution of the lesions?
7. Is there evidence of systemic illness?
8. What diagnostic tests would be helpful?
9. How can the rash be categorized?
  - a. Infectious
  - b. Immune
  - c. Vascular
  - d. Allergic
  - e. Malignancy
10. What is the best treatment for this patient?
11. What is the appropriate disposition?

with adequate lighting, and a systemic and thorough visual exam from the head to the soles of the feet should be performed, including skin, mucosa, and genitalia. Lesions may be palpated (wearing gloves) to determine texture, blanching, or sloughing characteristics. A test for Nikolsky's sign may be performed in which gentle rubbing of the skin results in sloughing of the epidermis. Primary and secondary lesions, characteristics, and patterns should be identified.

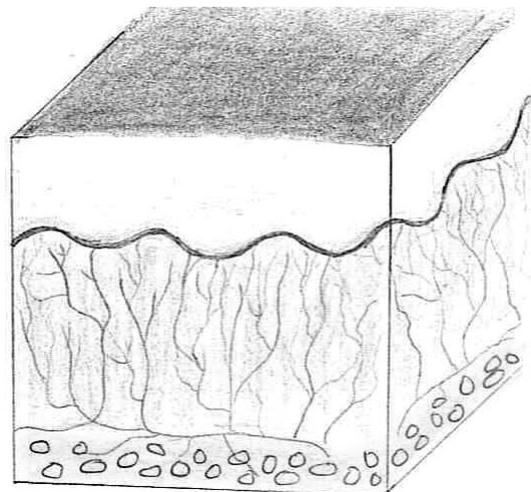
The identification and description of lesions is essential to making the correct diagnosis and determining the appropriate management. Lesions may be categorized as primary or secondary. Primary lesions are those that arise as a result of the disease process and have not been altered. Secondary lesions result from additional forces such as scratching, treatment, healing, or complicating infections. (See Figure 2.) Appropriate terminology allows one to accurately describe cutaneous findings, form a differential diagnosis, and communicate with colleagues or consultants. Primary and secondary lesions are listed with descriptions in Tables 2 and 3.

Following the thorough physical examination, diagnostic tests should be considered. For the nontoxic patient, laboratory tests often are unnecessary. Blood tests for secondary syphilis or heterophile antibodies (mono-spot) and throat swabs for rapid testing and culture of Group A streptococcus may be indicated. For the toxic or unstable patient, complete blood count (CBC), blood cultures, lumbar puncture studies, electrolytes, blood urea nitrogen (BUN), creatinine, glucose, and liver function tests should be considered.

### Categories of Cutaneous Conditions

When formulating a differential diagnosis it is useful to place the disorder into a general category. Dermatologic conditions may be categorized by lesion, distribution, or etiology. For purposes of organization, the authors choose to organize dermatologic conditions by distribution. Table 4 demonstrates this organizational scheme.

**Figure 1. Three Layers of Normal Skin, Including Epidermis, Dermis, and Subcutaneous Layers**



### Selected Dermatologic Presentations

As not all dermatologic presentations can be discussed in a single article, several important cutaneous diseases have been selected for review in this article.

**Erythema multiforme (EM)** is an acute inflammatory disease that is a spectrum of disease and can be further divided into EM minor and major. EM minor manifests as erythematous macular or papular eruptions generally located on the extensor surfaces, palms, soles, and face and affects no more than one mucosal surface. The classically described lesion is an erythematous macule that over 1-2 days develops central clearing and forms what resembles a target. Mucosal involvement may include vesicles and bullae. The lesions may be painful or pruritic. Also, as the name implies, this disease usually is minor and resolves in 2-4 weeks with little if any sequelae. Etiologies of EM minor include bacterial or viral infections, various drugs, and malignancies. Herpes simplex virus often is implicated and frequently is the cause in recurrent EM minor. The cause is undetermined as often as 50% of time. EM major (Stevens-Johnson syndrome) is a systemic disease with greater skin involvement, two or more mucosal surfaces, and often affects internal organs. It typically is preceded by diffuse malaise, myalgias, and fever. Morbidity is much worse, and mortality reaches 5-15%. Treatment depends on the severity of disease and may include treatment of the underlying disease, steroids, skin care, hydration, and, in severe cases, intensive care unit (ICU) or burn unit admission for supportive care.<sup>4,5</sup>

**Secondary syphilis** is a dermatologic condition that mimics many other common rashes and often is misdiagnosed. Syphilis is caused by the spirochete *Treponema pallidum* that penetrates mucous membranes or breaks in the skin. Reported syphilis rates have fallen drastically since reports began in 1941; however, there was a slight increase in the rates in 2001 and 2002 in the South and select urban areas in the specific population of men who have sex with men.<sup>6</sup> Untreated syphilis should be consid-

**Figure 2. Extensive Excoriations Secondary to Toxicodendron Exposure (Poison Ivy)**



In this case, the secondary lesions of excoriations are more prominent than the primary lesions of vesicles and papules.

ered as a diagnostic possibility, particularly in an era in which many sexually transmitted diseases (STDs) are treated with oral agents not providing adequate treatment for incubating syphilis. The first symptom is a painless genital chancre that appears approximately four weeks after inoculation and then remains for 3-6 weeks before resolution. If untreated, in another 3-6 weeks the patient develops generalized malaise, fatigue, fever, lymphadenopathy, and a maculopapular or pustular erythematous rash that usually begins on the trunk and extends peripherally and may include the soles and palms. (See Figure 4.) Lesions of primary and secondary syphilis are contagious and should be examined with gloved hands. *Treponema pallidum* remains sensitive to penicillin in most areas of the United States, and primary syphilis easily is treated with a single dose of benzathine penicillin G (Bicillin-LA) 2.4 million units intramuscularly. For those allergic to penicillin, tetracycline or doxycycline may be used. Secondary syphilis should be treated with three weekly doses of benzathine penicillin G.<sup>7</sup>

**Toxic epidermal necrolysis (TEN)** is a generalized exfoliative skin disease with significant morbidity and mortality. The pathophysiology is thought to be immune-complex mediated and results in a confluent morbiliform rash that may progress to large bullae formation caused by disruption at the dermal-epidermal junction. Although TEN often is idiopathic, there are many potential identifiable causes, with drug reactions being the most common. Sulfonamides, penicillins, cephalosporins, nonsteroidal anti-inflammatory drugs, anticonvulsants, and antituberculosis medications are among those found to have caused TEN. Malignancy, radiation therapy, and viral infections are other identified causes. Symptoms typically begin as vague generalized malaise, fatigue, fever, and upper respiratory infection symptoms, and the generalized rash follows. The rash is tender, erythematous, hot,

**Figure 3. Hemorrhagic Reaction to Insect Bites**



and develops into large bullae that slough easily with shearing pressure (Nikolsky sign). Mucous membranes commonly are affected. Complications may include electrolyte disturbances, secondary infections, dehydration, pneumonia with respiratory failure, and gastrointestinal hemorrhage. Mortality rates are 15-40%, with geriatric patients having the worst overall prognosis. Treatment mainly is supportive and includes aggressive fluid management, wound care, and treatment of subsequent complications, all of which may be accomplished effectively in a burn unit or ICU.<sup>3,8</sup>

**Drug reactions** commonly are seen in the emergency department and may present a diagnostic dilemma. (See Figures 5 and 6.) Drug eruptions can present as nearly any form of rash and, therefore, medication history is vital in evaluating all patients with dermatologic complaints. All medications started within the last two months should be identified. Routes of medication administration are important as topical and intramuscular routes have higher rates of cutaneous reactions. Cutaneous drug reactions may occur sporadically after multiple uses of the same drug, and may occur immediately or delayed following administration of the drug. Reactions may be classified into allergic and nonallergic. The allergic reactions further may be described using the Gell-Coombs classification in which there are four types of allergic reactions: type I is immediate hypersensitivity reaction (i.e., anaphylaxis, urticaria, angioedema, etc.); type II is a cytotoxic antibody reaction (i.e., purpura, hemolysis, etc.); type III is immune complex reaction (i.e., vasculitis, angioedema, serum sickness, etc.); type IV is delayed-type hypersensitivity reaction (i.e., contact dermatitis, photoallergic reactions, etc.). Nonallergic reactions are more common and may be the result of drug-drug interactions, toxicity, disruption of normal flora, exacerbation of pre-existing skin conditions, triggering of mast cell activation through nonimmunologic pathways, or systemic effects of bacterial endotoxins released by antibiotic mediated cell destruction. Immunocompromised patients (including those with HIV infection, malignancy, etc.) have a 10-fold greater risk of developing drug eruptions, and women have a 35% higher

**Table 2. Primary Lesions****Macule**

Flat circumscribed change in skin pigmentation < 0.5 cm in diameter

**Patch**

Flat circumscribed change in skin pigmentation > 0.5 cm in diameter

**Papule**

Elevated, solid, palpable lesion < 0.5 cm in diameter with variable color

**Plaque**

Elevated, solid, palpable lesion > 0.5 cm in diameter with variable color

**Nodule**

Solid, palpable, subcutaneous lesion < 0.5 cm in diameter

**Abscess**

Erythematous, fluctuant, tender, fluid filled nodule

**Tumor**

Solid, palpable lesion located in subcutaneous tissue > -0.5 cm in diameter

**Vesicle**

Elevated, thin walled, circumscribed, clear fluid- filled lesion < 0.5 cm in diameter

**Pustule**

Elevated, circumscribed, purulent fluid- filled lesion

**Bulla**

Elevated, thin walled, circumscribed, fluid- filled lesion > 0.5 cm in diameter

**Petechiae**

Flat, erythematous or violaceous non-blanching lesions < 0.5 cm in diameter

**Purpura**

Erythematous or violaceous non-blanching lesions, may be palpable, > 0.5 cm in diameter

incidence than men. These reactions may be mild and short-lived or may be extensive and life-threatening. Severe reactions include but are not limited to toxic epidermal necrolysis, erythema multiforme, Stevens-Johnson syndrome, vasculitis, hypersensitivity syndrome, and anticoagulant skin necrosis erythroderma. Diagnostic tests rarely are useful, although a complete blood count and skin biopsy may be indicated. Treatment includes identification and removal of the offending agent and symptomatic and supportive treatment as indicated by the severity of the condition.<sup>9</sup>

**Meningococcemia** is a serious and potentially life-threatening bacterial infection caused by the encapsulated organism *Neisseria meningitidis*. Infection is preceded by colonization of the mucus membranes in the nasopharynx with subsequent invasion into the blood stream. Some of the population (2-10%) are asymptomatic carriers. The organism resists phagocytosis by virtue of a polysaccharide capsule, is able to shed large amounts of endotoxin, and has an immunoglobulin A1 protease that increases intracellular survival. Transmission occurs via upper respiratory secretions, which include airborne droplets. This

**Table 3. Secondary Lesions****Scale**

Abnormal buildup of keratinized epithelium

**Crust**

Dry plasma proteins, resulting from inflammation

**Fissures**

Deep cracks in dry skin surfaces, extending into dermis

**Erosions**

Disruption of surface epithelium: inflammatory, usually linear, traumatic

**Ulcer**

Erosion extending into dermis

**Scar**

Dense collection of collagen, a result of healing after trauma

**Excoriation**

Linear erosions typically secondary to scratching

**Infections**

Breaks in dermal-epidermal junction allowing bacterial invasion, often erythematous

**Hyperpigmentation**

Increase in melanin containing epidermal cells

**Lichenification**

Abnormally dense layer of keratinized epidermal cells

infection may be manifested by meningitis alone or meningococemia with or without meningitis. Symptoms begin as a vague viral syndrome with sore throat, headache, and cough and rapidly progress to include high sudden fever, severe headache, meningeal signs, altered mental status, and rash. The rash classically is described as petechial or purpuric, typically first appears on the trunk, and may progress to extremities including the palms and soles. These lesions may evolve rapidly into confluent palpable papules. Patients typically appear toxic and may have unstable vital signs at presentation. Because most commercially available vaccines do not provide adequate protection against serogroup B, meningococemia should be considered in the appropriate clinical context, even among immunized individuals. Indicated laboratory studies include: complete blood count, blood cultures, and analysis and culture of cerebral spinal fluid. Prompt treatment is absolutely vital and should not be delayed to obtain the cerebral spinal fluid or other diagnostic tests. Cephalosporins, especially ceftriaxone (Rocephin), penicillin G, and vancomycin (Vancocin), are appropriate antibiotics. Aggressive supportive care and ICU admission are indicated for patients with unstable vital signs. Mortality is high (5-28%) even with optimal treatment. Persons with four hours of close contact or intimate exposure to upper respiratory secretions (including emergency department personnel) should receive antibiotic prophylaxis.<sup>3,10</sup>

**Erythema nodosum** is a nodular disease caused by inflammation of the subcutaneous adipose tissue. Lesions generally are located on the anterior lower extremities but can be found on the upper extremities or trunk. The lesions are warm, tender, erythematous nodules. Possible etiologies include bacterial, viral, fun-

**Table 4. Common Dermatologic Conditions**

I. LOCAL DISTRIBUTION	II. GENERALIZED DISTRIBUTION
<p><b>A. Erythematous Disorders</b></p> <ol style="list-style-type: none"> <li>1. Erythema multiforme</li> <li>2. Erythema nodosum</li> <li>3. Insect bites</li> <li>4. Sarcoidosis</li> <li>5. Dermatomyositis</li> <li>6. Infections</li> <li>7. Rosacea</li> </ol>	<p><b>A. Erythematous Disorders</b></p> <ol style="list-style-type: none"> <li>1. Erythema multiforme</li> <li>2. Staphylococcal scalded skin syndrome</li> <li>3. Kawasaki syndrome</li> </ol>
	<p><b>B. Papulosquamous Diseases</b></p> <ol style="list-style-type: none"> <li>1. Psoriasis</li> <li>2. Lupus</li> <li>3. Pityriasis rosea</li> <li>4. Lichen planus</li> <li>5. Secondary syphilis</li> <li>6. Drug eruptions</li> <li>7. Scabies</li> </ol>
<p><b>C. Eczematous Diseases</b></p> <ol style="list-style-type: none"> <li>1. Atopic dermatitis</li> <li>2. Seborrheic dermatitis</li> <li>3. Stasis dermatitis</li> <li>4. Nummular eczema</li> </ol>	<p><b>C. Eczematous Diseases</b></p> <ol style="list-style-type: none"> <li>1. Contact dermatitis</li> <li>2. Nummular eczema</li> </ol>
<p><b>D. Vesicular diseases</b></p> <ol style="list-style-type: none"> <li>1. Herpes simplex infection</li> <li>2. Herpes Zoster infection</li> <li>3. Contact dermatitis</li> <li>4. Dyshidrosis</li> <li>5. Insect bites</li> </ol>	<p><b>D. Vesicular diseases</b></p> <ol style="list-style-type: none"> <li>1. Varicella Zoster infection</li> <li>2. Hand, foot, and mouth disease</li> </ol>
<p><b>E. Bullous diseases</b></p> <ol style="list-style-type: none"> <li>1. Erythema multiforme</li> <li>2. Porphyria cutanea tarda</li> <li>3. Burns</li> <li>4. Pressure blisters</li> </ol>	<p><b>E. Bullous diseases</b></p> <ol style="list-style-type: none"> <li>1. Bullous pemphigoid</li> <li>2. Pemphigus vulgaris</li> <li>3. Dermatitis herpetiformis</li> <li>4. Erythema multiforme</li> <li>5. Toxic epidermal necrolysis</li> <li>6. Porphyria cutanea tarda</li> <li>7. Staphylococcal scalded skin syndrome</li> </ol>
<p><b>F. Hemorrhagic/Purpuric diseases</b></p> <ol style="list-style-type: none"> <li>1. Vasculitis</li> <li>2. Necrotizing fasciitis</li> <li>3. Polyarteritis nodosa</li> <li>4. Brown recluse spider bite</li> <li>5. Trauma</li> <li>6. Other bites and stings</li> </ol>	<p><b>F. Hemorrhagic/Purpuric diseases</b></p> <ol style="list-style-type: none"> <li>1. Vasculitis</li> <li>2. Thrombocytopenia</li> <li>3. Coagulopathy</li> <li>4. Necrotizing fasciitis</li> <li>5. Polyarteritis nodosa</li> <li>6. Drugs (NSAIDs, ASA, antibiotics, anticonvulsants, anticoagulants, diuretics, etc.)</li> </ol>
<p><b>G. Pustular diseases</b></p> <ol style="list-style-type: none"> <li>1. Acne vulgaris</li> <li>2. Folliculitis</li> <li>3. Pustular psoriasis</li> <li>4. Fire ant bites</li> </ol>	<p><b>G. Pustular diseases</b></p> <ol style="list-style-type: none"> <li>1. Pustular psoriasis</li> <li>2. Reiter's syndrome</li> </ol>

(Continued on page 43)

**Table 4. Common Dermatologic Conditions (continued)**

I. LOCAL DISTRIBUTION	II. GENERALIZED DISTRIBUTION
<p>H. Hyperpigmented Lesions</p> <ol style="list-style-type: none"> <li>1. Drug reaction</li> <li>2. Café au lait spots</li> <li>3. Freckles</li> <li>4. Pigmented Nevi</li> <li>5. Melasma</li> <li>6. Seborrheic keratoses</li> <li>7. Malignant melanoma</li> </ol>	<p>H. Hyperpigmented Lesions</p> <ol style="list-style-type: none"> <li>1. Drug reaction</li> <li>2. Café au lait spots</li> <li>3. Freckles</li> <li>4. Pigmented nevi</li> <li>5. Seborrheic keratoses</li> </ol>
<p>I. Infections</p> <ol style="list-style-type: none"> <li>1. Viral infections</li> <li>2. Bacterial infections</li> <li>3. Fungal infections</li> </ol>	<p>I. Infections</p> <ol style="list-style-type: none"> <li>1. Viral infections</li> <li>2. Bacterial infections</li> <li>3. Fungal infections</li> </ol>
<p>J. Malignancies</p> <ol style="list-style-type: none"> <li>1. Actinic keratosis</li> <li>2. Basal cell carcinoma</li> <li>3. Squamous cell carcinoma</li> <li>4. Malignant melanoma</li> </ol>	<p>J. Malignancies</p> <ol style="list-style-type: none"> <li>1. Internal malignancy</li> <li>2. Kaposi's sarcoma</li> </ol>
<p>K. Vascular diseases</p> <ol style="list-style-type: none"> <li>1. Erythema multiforme</li> <li>2. Erythema nodosum</li> <li>3. Urticaria</li> <li>5. Vasculitis</li> <li>6. Cutaneous lupus erythematosus</li> <li>7. Dermatomyositis</li> <li>8. Sunburn</li> <li>9. Cellulitis, erysipelas</li> </ol>	<p>K. Vascular diseases</p> <ol style="list-style-type: none"> <li>1. Erythema multiforme</li> <li>2. Erythema nodosum</li> <li>3. Drug eruptions</li> <li>4. Urticaria</li> <li>5. Vasculitis</li> <li>6. Sunburn</li> </ol>

gal, and parasitic infections; pharmacologic agents; sarcoidosis; leukemia; lymphoma; inflammatory bowel disease; and idiopathic cases. Treatment is focused on management of the underlying cause and systemic nonsteroidal anti-inflammatory agents.<sup>11</sup>

**Necrotizing fasciitis** is a life-threatening infection of the soft tissues that does not penetrate the fascial layer deep to the subcutaneous fat. Most cases are polymicrobial, including microbes such as *Streptococcus*, *Peptostreptococcus*, *Staphylococcus aureus*, *Pseudomonas*, *Escherichia coli*, and *Klebsiella*. Often the infection results from local trauma, insect bites, or recent surgical procedures, although comorbid conditions such as diabetes may have a contributory role. The infection typically begins locally and progresses rapidly, with severe pain, discharge, subcutaneous gas formation, bullae, and tissue necrosis. Mortality may be as high as 70-80%. Treatment should be instituted with fluid and/or blood resuscitation as clinically indicated, broad spectrum antibiotics, early surgical debridement, and hyperbaric oxygen therapy.<sup>12,13</sup>

**Insect bites and stings** are encountered often in the emergency department. Most are not serious, causing a local histamine reaction of urticaria or inflammation. (See Figures 3 and 7.) However, more serious complications such as cellulitis, local tissue necrosis, transmission of a disease, or anaphylaxis can

result. Insects of the order of Hymenoptera (which includes wasps, ants, and bees) are responsible for sting injuries and most insect-related anaphylaxis. Brown recluse spider bites may cause local tissue necrosis. These bites often have central clearing at the bite site that looks like a small target and also result in neurological or autonomic system dysfunction. Mosquitoes and ticks of various types may transmit disease through the bite. Lyme disease transmitted by the bite of the *Ixodes* tick and caused by the spirochete *Borrelia burgdorferi* results in the pathognomonic skin lesion erythema migrans. (See Figure 8.) Treatment of insect bites is dependent on the specific etiology and spectrum of symptomatology. Antihistamines may be sufficient to treat local reactions; antibiotics are indicated for Lyme disease or other transmitted diseases; aggressive wound care may be indicated for necrotic lesions; and epinephrine with antihistamines as well as aggressive supportive care are indicated to treat anaphylaxis.<sup>14-17</sup>

**Tinea infections** are fungal skin conditions that are named and treated according to the area of the body affected. *Tinea capitis* refers to the scalp and is characterized by scaly, pruritic patches that may result in alopecia. Treatment requires systemic antifungal therapy for 4-6 weeks. *Tinea corporis* involves the trunk, arms, and legs and is widely inappropriately known as “ringworm” because it is characterized by a pruritic, scaly,

**Figure 4. Maculopapular Lesions Resulting from Secondary Syphilis**



**Figure 5. Vasculitis, Which May Occur as a Drug Reaction**



**Figure 6. Vasculitis Resulting from Sulfonamide Medication**



raised, sharply demarcated annular lesion that clears centrally. *Tinea cruris*, which is similar in appearance, involves the groin. *Tinea versicolor* typically involves the upper trunk and limbs and is manifested as superficial scaly lesions that vary in color and may be tan, pink, or white. These lesions may be pruritic and do not tan with sun exposure. *Tinea corporis*, *cruris*, and *versicolor* all are treated with topical antifungal agents. *Tinea versicolor* should be treated with topical selenium sulfide solution. *Tinea unguium* refers to the nails and results in thickened, opaque, cracked, or crumbled appearance. The great toe most commonly is affected. Treatment with prolonged systemic antifungal therapy may be necessary.<sup>18</sup>

**Herpes zoster** is caused by the reactivation of a previously acquired varicella zoster infection that has been dormant in the dorsal root ganglia. Reactivation usually is caused by a decrease in cellular immunity, and the virus travels along the ganglion's dermatomal distribution to the surface. Symptoms usually begin with pain or paresthesias along the involved dermatome for 2-3 days, followed by eruption of the classic vesicular lesions on an erythematous base that progress to pustules and crusts before resolving. (See Figure 9.) Involvement of the second branch of the trigeminal nerve may implicate ocular disease that may result in blindness if untreated. It is important to perform a slit lamp exam with fluorescein dye to look for dendritic corneal lesions in such cases. Even if ocular involvement is not identified, if the infection involves the face, especially the tip of the nose, urgent ophthalmological consultation is warranted. Ramsay Hunt syn-

drome is involvement of the geniculate ganglion and is characterized by temporary unilateral facial paralysis and lesions on the external ear or tympanic membrane. Herpes zoster is considered disseminated if the rash is found on more than two dermatomes, and also may have visceral involvement. Disseminated zoster may be found in immunocompromised individuals and can be fatal. Pain that persists after resolution of the rash, postherpetic neuralgia, can be quite bothersome and may persist for up to a year. Treatment for herpes zoster should be instituted with oral antiviral medication (such as acyclovir [Avirax, Zovirax], famciclovir [Famvir], or valacyclovir [Valtrex]), analgesics including nonsteroidal anti-inflammatory drugs or narcotics, and appropriate referrals as warranted.<sup>19</sup> Topical capsaicin has demonstrated efficacy for pain relief in some patients. Systemic steroids should be administered for patients within the first 48 hours of symptoms to reduce the incidence of postherpetic neuralgia.

**Lice infestation** is caused by one of three types of ectopara-

**Figure 7. Vesicular and Papular Lesions Secondary to Insect Bites**



sites. *Pediculus humanus capitis*, *Pediculus humanus corporis*, and *Pthirus pubis* are, respectively, head, body, and pubic lice. Head lice primarily infest the scalp with predilection to the occipital and postauricular areas. The eggs of head lice are called nits and are attached to the base of the hair shaft 3-4 mm from the scalp. Body lice, as named, primarily infest the body but can include the scalp margins. Nits from the body louse may be attached to the patient's clothing. Pubic lice primarily infest the pubic and anogenital regions. Pediculosis typically is acquired from close contact with an infested person or sharing of items such as hair brushes, clothing, and headgear. All age groups and races are affected, with a slightly higher prevalence in females. Hair length and hygiene do not affect infestation rates. Symptoms include pruritis, which leads to excoriation and potentially secondary skin infections with regional lymphadenopathy. The treatment of choice is topical permethrin.<sup>20</sup> There are a number of topical treatments arranged in kits that can be purchased over-the-counter or by prescription. Most treatments should be repeated in 7-10 days to kill recently hatched nits. Treatment also should include washing clothing and linens in water greater than 55° C for 5 minutes or longer. Unwashable small items such as stuffed animals may be sealed in a plastic bag for 12-14 days.<sup>21</sup>

### Conclusions

Dermatologic presentations are common in emergency medicine. An understanding of common dermatologic definitions and a systematic approach to the diagnosis and therapy can be useful in the appropriate management of dermatologic complaints.

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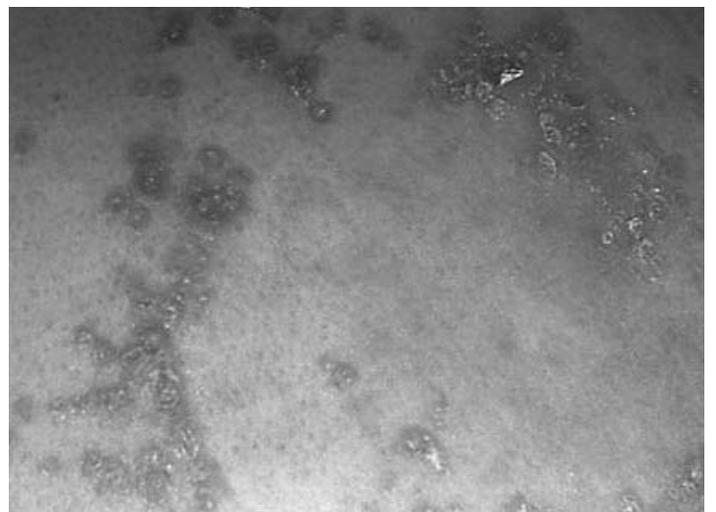
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**Figure 8. Erythema Migrans Associated with Lyme Disease**



Note nonscaling annular erythema.  
Photograph courtesy of: David Brancati, DO

**Figure 9. Herpes Zoster**



Note vesicles, crusting, and excoriations on an erythematous base, in a dermatomal pattern.

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### Physician CME Questions

31. Which of the following lesions is considered secondary?
  - A. Excoriation
  - B. Papule
  - C. Pustule
  - D. Bulla
32. Stevens-Johnson syndrome represents a severe variant of which cutaneous disease?
  - A. Toxic epidermal necrolysis
  - B. Erythema multiforme
  - C. Erythema migrans
  - D. Pemphigus vulgaris
  - E. Necrotizing fasciitis
33. A patient presents with urticaria of immediate onset after receiving an intramuscular penicillin shot. What type of allergic reaction does this represent?
  - A. Type I
  - B. Type II
  - C. Type III
  - D. Type IV
34. Which of the following statements best describes the management of suspected meningococemia?
  - A. A lumbar puncture should be performed immediately.

### CME Instructions

Physicians participate in this continuing medical education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to evaluate their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. *After completing this activity, you must complete the evaluation form that will be provided at the end of the semester and return it in the reply envelope provided to receive a certificate of completion.* When your evaluation is received, a certificate will be mailed to you.

### Emergency Medicine Reports

#### CME Objectives

*To help physicians:*

- quickly recognize or increase index of suspicion for specific conditions;
- understand the epidemiology, etiology, pathophysiology, and clinical features of the entity discussed;
- apply state-of-the-art diagnostic and therapeutic techniques (including the implications of pharmaceutical therapy discussed) to patients with the particular medical problems discussed;
- understand the differential diagnosis of the entity discussed;
- understand both likely and rare complications that may occur.

- B. A skin biopsy should be performed immediately.
  - C. Blood cultures should be obtained immediately.
  - D. ICU admission should be arranged immediately.
  - E. Antibiotics should be administered immediately.
35. Which of the following therapies for herpes zoster reduces the incidence of postherpetic neuralgia?
- A. Acyclovir
  - B. Capsaicin
  - C. Prednisone
  - D. Ibuprofen
36. What is the treatment of choice for pediculosis capitis?
- A. Lindane
  - B. Permethrin
  - C. Penicillin
  - D. Prednisone
  - E. Selenium sulfide
37. What condition should be suspected in a patient with disseminated Herpes zoster?
- A. Drug reaction
  - B. Erythema multiforme
  - C. Co-existing pediculosis
  - D. Noncompliance
  - E. Immunocompromised state
38. What type of insect reaction is commonly associated with tissue necrosis?
- A. Flea bite
  - B. Tick bite
  - C. Hymenoptera sting
  - D. Brown recluse spider bite
  - E. Pediculosis infestation
39. All of the following statements are true regarding toxic epidermal necrolysis (TEN) *except*:
- A. It is most commonly associated with a drug reaction.
  - B. It may be associated with a malignancy.
  - C. Mucous membranes never are involved.
  - D. Treatment is mainly supportive and includes aggressive fluid

- management, wound care, etc.
- E. Mortality rates are 15-40%.

40. Local pustular diseases include all the following *except*:
- A. Acne vulgaris
  - B. Folliculitis
  - C. Pustular psoriasis
  - D. Fire ant bites
  - E. Reiter's syndrome

### In Future Issues:

### Food Allergy

### CME Answer Key

- |       |       |
|-------|-------|
| 31. A | 36. B |
| 32. B | 37. E |
| 33. A | 38. D |
| 34. E | 39. C |
| 35. C | 40. E |

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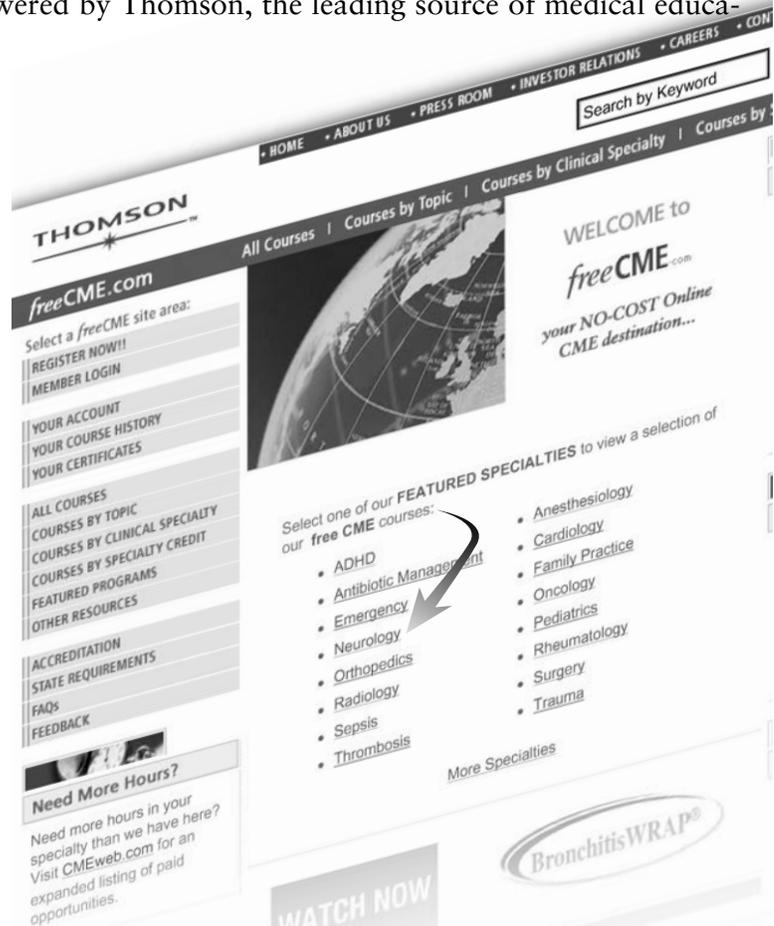
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## Common Dermatologic Presentations

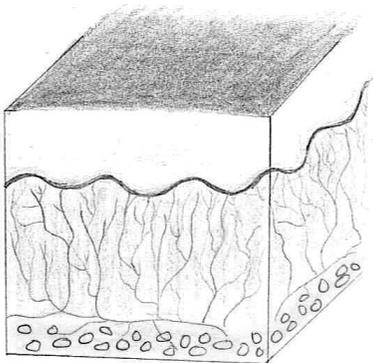
### Eleven Questions to Lead Physicians to Diagnosis and Management

1. What is the time of onset?
2. Are there any associated historical features?
3. What is the patient's medical history?
4. What is the primary lesion?
5. What are the secondary lesions?
6. What is the distribution of the lesions?
7. Is there evidence of systemic illness?
8. What diagnostic tests would be helpful?
9. How can the rash be categorized?
  - a. Infectious
  - b. Immune
  - c. Vascular
  - d. Allergic
  - e. Malignancy
10. What is the best treatment for this patient?
11. What is the appropriate disposition?

### Secondary Lesions

- Scale**  
Abnormal buildup of keratinized epithelium
- Crust**  
Dry plasma proteins, resulting from inflammation
- Fissures**  
Deep cracks in dry skin surfaces, extending into dermis
- Erosions**  
Disruption of surface epithelium: inflammatory, usually linear, traumatic
- Ulcer**  
Erosion extending into dermis
- Scar**  
Dense collection of collagen, a result of healing after trauma
- Excoriation**  
Linear erosions typically secondary to scratching
- Infections**  
Breaks in dermal-epidermal junction allowing bacterial invasion, often erythematous
- Hyperpigmentation**  
Increase in melanin containing epidermal cells
- Lichenification**  
Abnormally dense layer of keratinized epidermal cells

### Three Layers of Normal Skin, Including Epidermis, Dermis, and Subcutaneous Layers



### Extensive Excoriations Secondary to Toxicodendron Exposure (Poison Ivy)



In this case, the secondary lesions of excoriations are more prominent than the primary lesions of vesicles and papules.

### Vasculitis Resulting from Sulfonamide Medication



### Primary Lesions

- Macule**  
Flat circumscribed change in skin pigmentation < 0.5 cm in diameter
- Patch**  
Flat circumscribed change in skin pigmentation > 0.5 cm in diameter
- Papule**  
Elevated, solid, palpable lesion < 0.5 cm in diameter with variable color
- Plaque**  
Elevated, solid, palpable lesion > 0.5 cm in diameter with variable color
- Nodule**  
Solid, palpable, subcutaneous lesion < 0.5 cm in diameter
- Abscess**  
Erythematous, fluctuant, tender, fluid filled nodule
- Tumor**  
Solid, palpable lesion located in subcutaneous tissue > 0.5 cm in diameter
- Vesicle**  
Elevated, thin walled, circumscribed, clear fluid-filled lesion < 0.5 cm in diameter
- Pustule**  
Elevated, circumscribed, purulent fluid-filled lesion
- Bulla**  
Elevated, thin walled, circumscribed, fluid-filled lesion > 0.5 cm in diameter
- Petechiae**  
Flat, erythematous or violaceous non-blanching lesions < 0.5 cm in diameter
- Purpura**  
Erythematous or violaceous non-blanching lesions, may be palpable, > 0.5 cm in diameter

### Vesicular and Papular Lesions Secondary to Insect Bites



### Vasculitis, Which May Occur as a Drug Reaction



### Hemorrhagic Reaction to Insect Bites



### Erythema Migrans Associated with Lyme Disease



Note nonscaling annular erythema. Photograph courtesy of: David Brancati, DO

## Common Dermatologic Conditions

### I. LOCAL DISTRIBUTION

#### A. Erythematous Disorders

1. Erythema multiforme
2. Erythema nodosum
3. Insect bites
4. Sarcoidosis
5. Dermatomyositis
6. Infections
7. Rosacea

### II. GENERALIZED DISTRIBUTION

#### A. Erythematous Disorders

1. Erythema multiforme
2. Staphylococcal scalded skin syndrome
3. Kawasaki syndrome

#### B. Papulosquamous Diseases

1. Psoriasis
2. Lupus
3. Pityriasis rosea
4. Lichen planus
5. Secondary syphilis
6. Drug eruptions
7. Scabies

#### C. Eczematous Diseases

1. Atopic dermatitis
2. Seborrheic dermatitis
3. Stasis dermatitis
4. Nummular eczema

#### C. Eczematous Diseases

1. Contact dermatitis
2. Nummular eczema

#### D. Vesicular diseases

1. Herpes simplex infection
2. Herpes Zoster infection
3. Contact dermatitis
4. Dishydrosis
5. Insect bites

#### D. Vesicular diseases

1. Varicella Zoster infection
2. Hand, foot, and mouth disease

#### E. Bullous diseases

1. Erythema multiforme
2. Porphyria cutanea tarda
3. Burns
4. Pressure blisters

#### E. Bullous diseases

1. Bullous pemphigoid
2. Pemphigus vulgaris
3. Dermatitis herpetiformis
4. Erythema multiforme
5. Toxic epidermal necrolysis
6. Porphyria cutanea tarda
7. Staphylococcal scalded skin syndrome

#### F. Hemorrhagic/Purpuric diseases

1. Vasculitis
2. Necrotizing fasciitis
3. Polyarteritis nodosa
4. Brown recluse spider bite
5. Trauma
6. Other bites and stings

#### F. Hemorrhagic/Purpuric diseases

1. Vasculitis
2. Thrombocytopenia
3. Coagulopathy
4. Necrotizing fasciitis
5. Polyarteritis nodosa
6. Drugs (NSAIDs, ASA, antibiotics, anticonvulsants, anticoagulants, diuretics, etc.)

#### G. Pustular diseases

1. Acne vulgaris
2. Folliculitis
3. Pustular psoriasis
4. Fire ant bites

#### G. Pustular diseases

1. Pustular psoriasis
2. Reiter's syndrome

## Common Dermatologic Conditions

### I. LOCAL DISTRIBUTION

#### H. Hyperpigmented Lesions

1. Drug reaction
2. Café au lait spots
3. Freckles
4. Pigmented Nevi
5. Melasma
6. Seborrheic keratoses
7. Malignant melanoma

### II. GENERALIZED DISTRIBUTION

#### H. Hyperpigmented Lesions

1. Drug reaction
2. Café au lait spots
3. Freckles
4. Pigmented nevi
5. Seborrheic keratoses

#### I. Infections

1. Viral infections
2. Bacterial infections
3. Fungal infections

#### I. Infections

1. Viral infections
2. Bacterial infections
3. Fungal infections

#### J. Malignancies

1. Actinic keratosis
2. Basal cell carcinoma
3. Squamous cell carcinoma
4. Malignant melanoma

#### J. Malignancies

1. Internal malignancy
2. Kaposi's sarcoma

#### K. Vascular diseases

1. Erythema multiforme
2. Erythema nodosum
3. Urticaria
5. Vasculitis
6. Cutaneous lupus erythematosus
7. Dermatomyositis
8. Sunburn
9. Cellulitis, erysipelas

#### K. Vascular diseases

1. Erythema multiforme
2. Erythema nodosum
3. Drug eruptions
4. Urticaria
5. Vasculitis
6. Sunburn

## Herpes Zoster



Note vesicles, crusting, and excoriations on an erythematous base, in a dermatomal pattern.

## Maculopapular Lesions Resulting from Secondary Syphilis



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