

I. INTRODUCTION

Cutaneous disorders comprise a small portion (1%) of the examination content. Pictorial identification is especially important. The lesions and rashes you are likely to be asked to identify are described in this chapter. You may also wish to consult a color dermatology atlas, such as *Fitzpatrick's Color Atlas and Synopsis of Clinical Dermatology*, Weston and Lane's *Color Textbook of Pediatric Dermatology*, or Knoop's *The Atlas of Emergency Medicine*. You should be familiar with all the color plates in the most recent edition of Tintinalli's textbook.

II. GENERAL APPROACH TO THE PATIENT PRESENTING WITH A RASH

- A. Inquire about prodromal symptoms, time course, and antecedent events (eg, new medications).
- B. Note patient's age, immune status, past medical history, sexual history, medications, allergies, and presence/absence of toxicity.
- C. Examine the rash and determine its characteristics.
 1. Appearance
 - a. Macular → flat and ≤ 1 cm
 - b. Patchy → flat and > 1 cm
 - c. Papular → raised and ≤ 1 cm
 - d. Plaque → raised and > 1 cm
 - e. Maculopapular, nodular → dermal or subcutaneous solid lesion 1–2 cm
 - f. Tumor → dermal or subcutaneous solid lesion > 2 cm
 - g. Vesicular → blister ≤ 1 cm
 - h. Bullous → blister > 1 cm
 - i. Pustules → small blister containing purulent material
 - j. Scales or keratoses → built up epidermis
 - k. Crusts, erosions → loss of part or all of epidermis
 - l. Ulceration → loss of dermis or deeper
 2. Evolution: determine where it started and how it has spread.
 3. Distribution: note location of the rash, including involvement of mucous membranes, palms, and soles.
 4. Symptoms: determine if pruritic or painful; note any systemic symptoms (fever, odynophagia, malaise).
 5. Treatments: determine what, if anything, the patient has done to treat the rash (eg, applied topical steroids, zinc, or a neomycin-containing antibacterial ointment), because this might have changed the appearance of the rash or caused a secondary contact dermatitis.

III. TOXICODENDRONS (POISON IVY, POISON OAK, POISON SUMAC)

A. Overview

1. Toxicodendron reactions are the most common cause of allergic contact dermatitis in North America.
2. The development of a reaction requires prior sensitization (days or years earlier); these are type IV cell-mediated, delayed hypersensitivity reactions.
3. The antigen responsible for producing the skin reaction is urushiol, which is found in poison ivy, poison oak, poison sumac, and mangoes.

B. Clinical presentation

1. The rash is characterized by erythema (frequently in a linear configuration), papules, and intense pruritus, typically in exposed areas. The rash can progress to vesicles, or bullae with edema. It might weep fluid.



Courtesy of David Efron, MD, FACEP

2. The rash usually appears 5 hours to 15 days after an exposure (most often between 24 and 48 hours).
3. A widely held misconception is that rupture of the vesicles spreads the rash, but the blister fluid does not contain antigen and therefore cannot spread the rash. Of note, different areas of the skin can react at different times, furthering this misconception.

C. Management

1. Mild dermatitis: topical calamine lotion, colloidal oatmeal or baking soda baths, topical steroids (0.1% triamcinolone or 0.1% betamethasone applied bid or tid), cool compresses, and an oral antihistamine
2. Moderate to severe dermatitis
 - a. Wet-to-dry compresses with water or aluminum acetate, eg, Burow solution
 - b. Oral antihistamines (eg, hydroxyzine, diphenhydramine)
 - c. Systemic corticosteroids are indicated for severe reactions. They should be continued for 2–3 weeks, with a gradual taper to avoid rebound. Too short a duration can result in rebound flare. Typical starting dose is prednisone 1–2 mg/kg/day × 1 week.
3. Avoid topical antihistamines and antibiotics.
4. Without treatment, symptoms resolve in 3–6 weeks.

D. Prophylaxis

1. After an exposure, the area should be washed immediately with cold soapy water. Urushiol is absorbed in 10–30 minutes, so the allergen must be removed quickly.
2. This practice can prevent dermatitis from developing, or at least lessen its severity.

IV. DIAPER DERMATITIS

A. Overview

1. This is not a specific entity but rather a nonspecific term referring to several dermatologic conditions that occur in the diaper area. Be able to distinguish irritant, contact dermatitis, and *Candida* diaper rashes.
2. The most important inciting factor is constant moisture. Other factors are diaper detergents, disinfectants, an alkaline pH, fecal material, intestinal enzymes, and friction caused by the diaper.
3. The effects of local irritants are amplified by infrequent diaper changes, inadequate skin cleansing, and occlusive diapers.
4. Incidence is highest in babies 9–12 months old, rarely seen at age <3 weeks. It can also be seen in incontinent and paralyzed adults and may be a red flag for neglect or abuse in this population.

B. Clinical presentation

1. The rash begins as an erythematous eruption over areas of increased friction of skin against the diaper (buttocks, genitals, lower abdomen, and thighs). This progresses to papules, vesicles, erosions, and ulcers. Distinct lines of demarcation may be seen along the diaper margins. Skin folds may be spared.



Courtesy of David Efron, MD, FACEP

2. The involved skin may become hyperpigmented after resolution.
3. Diaper dermatitis can be a manifestation of the following conditions:
 - a. Atopic or seborrheic dermatitis: a clue is the presence of concurrent facial lesions (atopic dermatitis) or scalp lesions (seborrheic dermatitis).
 - b. Primary irritant contact dermatitis: from ammonia and bacterially produced putrefactive enzymes (the odors are characteristic and the skin folds are notably spared) or from a new soap, shampoo, or laundry detergent.
 - c. Secondary infection with *Candida albicans*: a clue is the classic appearance of the rash (moist, beefy-red plaques with wellmarked edges in association with satellite lesions and skin fold involvement) and a duration >3 days.

C. Management

1. General measures
 - a. Discontinue use of plastic or rubber occlusive diaper pants.
 - b. Use super-absorbent disposable diapers or cloth diapers, and change them frequently.
 - c. Avoid the use of harsh cleansing agents, even “baby wipes” and soaps (which remove protective skin oils); use tepid water, and pat dry.
 - d. Leave diapers off for extended periods of time or dry diaper area with cool hairdryer.
 - e. Protect the skin from maceration with a barrier cream such as commercial zinc oxide diaper rash creams or petrolatum ointment. Do not use baby powder with cornstarch, which is metabolized by bacteria.
2. Additional specific measures
 - a. For mild inflammation secondary to atopic, seborrheic, or contact dermatitis, use 1% hydrocortisone cream with a barrier, such as zinc oxide ointment or petrolatum gel (to prevent it from washing off).
 - b. If the rash is more severe or persists >3–4 days, or if the patient has overt signs of *Candida* infection, add nystatin, clotrimazole, or miconazole cream for 2–3 weeks.

V. ECZEMA

A. Overview

1. A pruritic, chronic, relapsing atopic dermatitis: “the itch that rashes”
2. 85% of cases appear by age 5
3. Familial predominance
4. Associated with food allergies and asthma

B. Clinical presentation

1. Children: face, scalp, and torso are most commonly affected
2. Adults: flexure aspects of the extremities
3. Rash is papules and plaques that are intensely pruritic, scaly, and erythematous.
4. Dark-skinned patients could have hypo- or hyperpigmented areas.

C. Management

1. Topical corticosteroids should be applied to the affected areas.
2. Minimize showers; avoid bathing to help maintain skin hydration.
3. Use antihistamines for pruritus.
4. Topical emollients or petroleum jelly can be applied to damp skin to maintain moisture.

VI. PSORIASIS**A. Overview**

1. An intermittent, chronic inflammatory disease that can develop at any age
2. Affects approximately 2% of the world's population
3. Chronic plaque psoriasis can affect 1%–90% of the body surface area.
4. Inverse psoriasis is a variant that affects intertriginous or thin-skinned areas (eg, genitals, eyelids).
5. Pustular psoriasis is a rare variant characterized by diffuse erythematous papules.
6. Guttate psoriasis is seen in children/young adults with sores that are shaped like a water drop and are commonly triggered by a bacterial infection like a streptococcal pharyngitis. Scales tend to be less thick, and resolution is expectant but relapses can occur.

B. Clinical presentation

1. Sharply demarcated thick, silver, scaly, mildly pruritic plaques generally affecting the scalp, elbow, and knees
2. Can also affect the hands, feet, trunk, or nails
3. Removing plaques causes pinpoint bleeding (Auspitz sign).

C. Management

1. High-potency topical corticosteroids (eg, fluocinonide applied every 12 hours) if disease is localized. Do not use on areas of thin skin or near the eyes.
2. Tar-based shampoos for scalp lesions
3. The use of systemic steroids can lead to severe exacerbations of the disease when therapy is discontinued and should therefore be avoided.
4. Requires long-term management by a dermatologist or primary care physician.

VII. EXFOLIATIVE DERMATITIS/EXFOLIATIVE ERYTHRODERMA SYNDROME**A. Overview**

1. Widespread erythematous pruritic dermatitis
2. Etiology
 - a. Idiopathic is most common form.
 - b. Drug-induced (>50 drugs have been implicated)
 - c. Underlying malignancy (cutaneous lymphoma, leukemia, or other lymphoreticular malignancy) or immunosuppression (HIV)
 - d. Preexisting dermatoses (eg, psoriasis, eczema, seborrhea)
 - e. Allergic contact dermatitis

B. Clinical presentation

1. Shiny, erythematous, pruritic rash with scaling; begins localized then spreads and generalizes; spares the palms and soles
2. Classic clinical scenario: A 57-year-old man who has a past medical history of seizures and who is taking a new medication appears ill and complains of itching, a chilly sensation, and "tightness" of the skin. He has a low-grade fever and is hypotensive with tachycardia. On examination, a scaly, warm, erythematous rash is found to be covering >50% of his body surface area. There is no oral involvement, and the rash is not tender to the touch. Nikolsky sign is negative.



Courtesy of David Efron, MD, FACEP

3. Other findings can include fever or hypothermia, dehydration, lymphadenopathy, hepatosplenomegaly, lower extremity edema, or gynecomastia.
4. Scratching can result in lichenification and erosions.
5. Because of increased blood flow to the skin, the patient might have high output heart failure.

C. Diagnostic evaluation

1. Consider CBC, serum chemistries, liver function tests, erythrocyte sedimentation rate, urinalysis, and HIV testing in the search for systemic causes.
2. Because this disorder is usually the result of an underlying cutaneous disease, a systemic disease, or a response to a drug or chemical, patients should be admitted for a diagnostic evaluation. The mortality rate is as high as 30%.
3. Obtain skin biopsy (lymph node biopsy if significant lymphadenopathy is present)

D. Differential diagnosis

1. Erythema multiforme
2. Toxic epidermal necrolysis
3. Toxic shock syndrome
4. Staphylococcal scalded skin syndrome
5. Kawasaki disease (children)

E. Management

1. Goal is to correct/eliminate the underlying cause while providing symptomatic relief and maintaining skin moisture.
2. Stop new medications, if at all possible.
3. Antihistamines
4. Topical steroids covered with an occlusive dressing and continued for weeks or months.
5. Warm water baths with bath oils and skin emollients are also helpful.
6. Severe or resistant cases are treated with systemic corticosteroids.

VIII. ERYTHEMA MULTIFORME (EM)

A. Overview

1. There has been controversy regarding the nomenclature of EM through the years, but it is now generally agreed that there are two forms: erythema multiforme minor and erythema multiforme major (or bullous EM)
2. Occurs as a response to infection
 - a. Most commonly HSV-1 or HSV-2
 - b. Varicella, parpoxvirus, adenovirus, coxsackievirus, influenza, HIV, hepatitis, *M pneumoniae*, *Salmonella*, streptococci

B. Clinical presentation

1. EM minor: target lesions or raised papules with acral distribution; no mucous membrane involvement



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2. EM major (or bullous EM): <10% body surface area (BSA) blistering (vesiculobullous lesions) and epidermal detachment plus typical target lesions or raised atypical target lesions (urticarial-like plaques); possible oral or ocular mucous membrane involvement
3. Target lesions are erythematous plaques with dusky centers and bright red borders resembling a bull's-eye or target.
4. Both: rash evolves over 72 hours then remains fixed until it fades after 2 weeks.
5. Distribution is generally symmetric and involves the upper extremities and face but can involve the trunk and legs.
6. May be recurrent

C. Management

1. If the patient has herpes simplex infection, antiviral therapy is indicated.
2. This is a self-limited disease, and treatment focuses on symptomatic relief (topical steroids, antihistamines, NSAIDs, and cool compresses).
3. Patients with EM can be discharged.



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IX. STEVEN-JOHNSONS SYNDROME AND TOXIC EPIDERMAL NECROLYSIS

A. Overview

1. A drug reaction that causes sloughing of skin and mucous membranes
2. Previously thought to be a variant of erythema multiforme
3. Now the spectrum of Stevens-Johnson syndrome (SJS) → SJS/toxic epidermal necrolysis (SJS/TEN) overlap → TEN is recognized as its own entity.
4. Death rate: SJS, 1%–5%; SJS/TEN, 6%; TEN, 25%–35%
5. Worse outcome with increased BSA involvement, advanced age, malignancy, tachycardia, BUN >10 mEq/L, acidosis, and hyperglycemia.
6. Common inciting agents are NSAIDs, antibiotics (sulfonamides, penicillins, cephalosporins), anticonvulsants, and allopurinol.

B. Clinical presentation

1. **SJS: <10% BSA blistering and epidermal detachment plus widespread erythematous or purpuric macules or flat atypical target lesions; primarily on the trunk and face; mucous membrane involvement**
2. **SJS/TEN overlap: 10%–30% BSA blistering and epidermal detachment plus widespread purpuric macules or flat atypical target lesions; mucous membrane involvement**
3. **TEN: ≥30% BSA blistering and epidermal detachment with widespread purpuric macules or flat atypical target lesions; mucous membrane involvement**
4. **All are characterized by widespread bullous lesions, severe mucous membrane involvement, and multisystem pathology.**
5. **Nikolsky sign is positive.**
6. **TEN is characterized by extensive cutaneous and mucosal blistering as well as desquamation; epidermis becomes necrotic and sloughs in large sheets, leaving behind exposed dermis.**
7. **A flu-like prodrome often precedes development of the mucocutaneous lesions by 1–14 days.**
8. **Denuded skin and mucous membranes result in fluid loss and susceptibility to secondary bacterial infection.**
9. **Significant ocular sequelae (corneal ulceration, blindness) are possible.**
10. **Renal involvement (hematuria, renal tubular necrosis, renal failure) can occur but is rare.**
11. **Death, when it occurs, is most often due to fulminant sepsis.**
12. **Classic clinical scenario:** The patient appears very ill and gives a history of fever, malaise, myalgias, and arthralgias, followed by the abrupt onset of bullous mucocutaneous lesions that subsequently eroded. Multiple mucosal surfaces (eyes, mouth, lips, urogenital area, and anus) are involved. Patients often cannot eat because of painful stomatitis. Conjunctivitis is common, and vesicles on the conjunctiva are sometimes seen; the eyelids may be red, swollen, and crusted. An overall skin examination is likely to reveal widespread erythematous (or purpuric) macules or flat atypical “targets.”

C. Management

1. Consult with a dermatologist.
2. Identify the precipitant cause (if possible) and treat accordingly. Discontinue any suspicious drug.
3. Hospitalization, IV fluid resuscitation to correct hypovolemia, and correction of electrolyte abnormalities.
4. Systemic steroids are controversial. There is no hard evidence that they are of benefit, and there is some concern that they might increase complications.
5. IVIG for TENS
6. Obtain ophthalmology consult for patients with ocular lesions because of the risk of long-term morbidity from scarring.
7. Close monitoring for infections (avoid prophylactic antibiotics).
8. Consider burn unit admission.



Courtesy of David Efron, MD, FACEP

X. ERYTHEMA NODOSUM

A. Overview

1. An inflammatory/immunologic reaction of the panniculus (fat) that most commonly affects women 15–30 years old
2. Streptococcal infection is the most common precipitant in children.
3. Sarcoidosis, inflammatory bowel disease, malignancy, and drug reaction (particularly to birth control pills and sulfonamides) are the most common triggers in adults.

B. Clinical presentation

1. Characterized by painful, deep-seated nodules (2–5 cm) on the lower extremities (bilateral but not symmetrical)
2. Nodules are initially bright red and slightly elevated; then, over 1–2 weeks, they become violet or maroon and less raised.
3. Nodules are typically located in the pre-tibial region but can also be seen on the arms and (rarely) the face.
4. Arthralgias (particularly of the ankles) often precede the cutaneous eruption.
5. Single lesions generally last about 2 weeks, but new lesions may continue to recur up to 3–6 weeks.



Courtesy of David Efron, MD, FACEP

C. Management

1. Identify and treat the underlying cause; this condition is self-limited (3–6 weeks) if the cause can be eliminated.
2. Symptomatic measures include bed rest and leg elevation, compressive dressings, NSAIDs, and colchicine.

XI. PITYRIASIS ROSEA**A. Overview**

1. A self-limited rash, typically lasting 4–8 weeks
2. Unclear cause, presumed to be viral
3. May be preceded by a flu-like prodrome
4. Risk of premature birth if it occurs during pregnancy

B. Clinical presentation

1. A single salmon-colored oval patch or plaque (the herald patch) appears first.
2. Days to weeks later, salmon-colored circular or oval patches and plaque with scaling appear in a Christmas tree pattern.
3. Rash may be asymptomatic or pruritic.



Courtesy of Laura J. Bontempo, MD, MEd, FACEP

C. Management

1. Self-limited; no treatment required.
2. Antihistamines, if needed, for pruritus.

XII. CELLULITIS/ERYSIPELAS**A. Overview**

1. Cellulitis is a local soft-tissue inflammatory reaction secondary to bacterial invasion of the skin.
2. Although cellulitis is classically associated with comorbidities such as diabetes or peripheral vascular disease, <5% of all patients have such coexisting conditions.
3. In immunocompetent adults, the cause is most commonly *Staphylococcus aureus* (including MRSA) and Group A streptococci. In immunocompromised adults, the cause is most commonly gram-positive cocci and gram-negative aerobes.
4. Most infections involve the extremities.
5. Anything that compromises skin integrity (eg, trauma, IV drug abuse) or blood flow to the extremity increases the risk of cellulitis.

B. Clinical presentation

1. The classic symptoms are the result of a localized inflammatory reaction characterized by pain, warmth, erythema, and induration.



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2. Systemic involvement such as fever and bacteremia can occur and is most common in immunosuppressed patients.
3. Erysipelas
 - a. A specific type of cellulitis caused by group A streptococci, usually *Streptococcus pyogenes*
 - b. Characterized by a bright red color, sharp margins, and tender plaques
 - c. Commonly affects the face and lower extremities (especially in patients with venous insufficiency)
 - d. Generally affects patients >60 years old and is associated with fever, chills, and malaise



Courtesy of David Efron, MD, FACEP

C. Diagnostic evaluation

1. Blood cultures, tissue biopsies, and swabs are not routinely recommended.
2. Blood cultures are recommended for patients with cellulitis who are immunocompromised, who suffered an immersion injury, and who were bitten by an animal.

D. Management

1. Elevate the affected area as much as possible.
2. Cellulitis associated with penetrating trauma, MRSA infection, a history of MRSA, IV drug abuse, or systemic infection should be treated with vancomycin.
3. Cellulitis in an immunocompromised host, in a patient with neutropenia, after an animal bite, or after an immersion injury should be treated with vancomycin plus either piperacillin/tazobactam, imipenem, or meropenem.
4. Duration of treatment is usually 5 days but may be extended if infection is not improving.
5. Outpatient treatment is acceptable for immunocompetent patients without systemic infection.