Common Dermatologic Rashes—Inflammatory and Infectious Etiologies

Series Editor:
Miriam T. Vincent, MD
Associate Professor, Interim Chair, Department of Family Medicine,
State University of New York, Health Science Center at Brooklyn,
Brooklyn, NY

Contributing Author:
Enitza D. George, MD
Assistant Professor, Department of Family Medicine, Medical Director,
Family Practice Center, State University of New York, Health Science
Center at Brooklyn, Brooklyn, NY

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I. INTRODUCTION

A. Skin disorders are among the most common complaints encountered in primary care practice. In the United States between 1990 and 1994, 316 million visits to physicians were for dermatologic problems, accounting for 9% of all patient visits during this period. Primary care providers manage at least 60% of all dermatologic complaints, whereas dermatologists see approximately 40% of patients with skin-related concerns.1

B. Ten conditions account for nearly 60% of skin diagnoses made by internists: dermatitis, bacterial skin infections, tineas, acne vulgaris, herpes simplex and zoster, epidermoid cysts, exanthems, urticaria, and nonvenomous insect bites.2 Because family physicians care for both children and adults, a list of the most common diseases seen by family practitioners would be much more extensive.

C. Rashes represent an acute or chronic skin eruption. A careful history and physical examination are essential in order to make a diagnosis (Table 1). This issue of the Family Practice Board Review Manual addresses commonly encountered rashes that are inflammatory or infectious in origin.

II. INFLAMMATORY LESIONS

A. Eczemas

1. Diaper dermatitis
   a. Definition and etiology. Acute inflammation in diaper area caused by prolonged exposure to urine or feces owing to infrequent diaper changes
   b. Epidemiology. Most common in infants, but may occur in adults who are incontinent
   c. History. Days or weeks of pain and burning in diaper area, upper thighs, and lower abdomen
   d. Physical examination. Bright red rash, vesicles, erosions that spare body folds
   e. Differential diagnosis
      1) Intertrigo: rash of intertrigo appears erythematous only.
      2) Candidiasis: lesions of candidiasis are characterized by sharp margins, raised scaling edges.
   f. Treatment. Change diapers frequently, air-dry diaper area, avoid plastic occlusive pants, apply zinc oxide paste to prevent recurrences.

2. Contact dermatitis
   a. Definition and etiology. Allergic inflammation of the epidermis and dermis resulting from exposure to external irritating agents
      1) It can result from a primary irritant or a delayed hypersensitivity reaction (type IV) with a latent period of days or weeks from first exposure to re-exposure.
      2) Common irritants include nickel in jewelry, preservatives in cosmetics and soaps, and urushiol in poison ivy.
   b. Epidemiology. Patients of all ages may be affected. Workers may develop dermatitis as a result of occupational exposure to chemicals.
   c. History. Acute, subacute, or chronic itching and burning. History typically includes exposure to an irritant.
   d. Physical examination. Irregular patches of erythema, edema, vesicles, and erosions. With chronicity, there is thickening of the skin (lichenification) and hyperpigmentation. Lesions may be localized to the exposed area or may be generalized, depending on the particular offending agent (Figure 1).
   e. Treatment
      1) Identify and remove the offending agent and apply topical corticosteroids for a short period.
      2) For acute, severe, and generalized cases prednisone may be necessary.
3. **Seborrheic dermatitis**
   a. **Definition.** Chronic inflammatory scaling of areas that have a high concentration of sebaceous glands.
   b. **Etiology.** Unknown. Some authors suggest association with fungal infection. Diet, alcohol, and stress may play a role.
   c. **Epidemiology.** All ages may be affected. Immunocompromised patients are more susceptible.
   d. **History.** Gradual and progressive onset. Chief complaint is pruritus and scaling.
   e. **Physical examination.** White or yellowish greasy scales on an erythematous base. Most commonly affects scalp, eyebrows, beard, nasolabial fold, and body folds (Figure 2).
   f. **Differential diagnosis**
      1) Dandruff: the inflammatory base that is characteristic of seborrheic dermatitis is not present in dandruff.
      2) Psoriasis: scales of psoriasis are dry, rather than greasy.
      3) Contact dermatitis: differentiate by absence or presence of a history of contact with allergen or irritant.
   g. **Treatment.** Imidazole agents may be administered as shampoos (twice weekly) or creams (twice daily). Low-pH shampoos that contain tar or salicylic acid to remove scales are recommended for scalp involvement. Nonfluorinated corticosteroid lotion or ointment may be administered twice daily for skin involvement.
   
4. **Atopic dermatitis**
   a. **Definition and etiology.** This is a chronic pruritic inflammation of the epidermis and dermis.
      1) It is caused by a type 1 (immunoglobulin E-mediated) hypersensitivity reaction characterized by the release of vasoactive substances from mast cells.
      2) It is frequently hereditary, a personal or family history of allergic rhinitis, atopy, or asthma is often present.
   b. **Epidemiology.** It may occur in patients of any age, but is more common in children, especially boys.
   c. **History.** Pruritus, with a vicious cycle of itch-scratch-rash-itch. Commonly associated with stress, wool, and changes in temperature.

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**Table 1.** Diagnostic Approach to Skin Rashes

<table>
<thead>
<tr>
<th>Category</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>History</strong></td>
<td>Onset, progression, distribution Symptoms related to the skin (eg, pruritus, bruising, pain) Accompanying systemic signs or symptoms Aggravating or soothing factors Treatments used before the patient presented for consultation</td>
</tr>
<tr>
<td><strong>Physical examination</strong></td>
<td>Assess patient stability: define general appearance, assess vital signs Describe the lesions: color, number, pattern, distribution, size, spread</td>
</tr>
<tr>
<td><strong>Ancillary tests</strong></td>
<td>Exclude or confirm specific diagnostic entities Collect skin scrapings for microscopic examination and cultures</td>
</tr>
<tr>
<td><strong>Therapeutic trial</strong></td>
<td>Often useful in defining etiologic process as well as therapeutic options</td>
</tr>
</tbody>
</table>
d. Physical examination. There is a characteristic pattern of distribution (Figure 3), which is usually symmetrical. Excoriations and erythema later develop lichenification. Secondary infection, especially herpes simplex infection, may develop. Facial examination may reveal infraorbital folds (Dennie-Morgan sign) and infraorbital darkening (“allergic shiners”).

e. Differential diagnosis
1) Seborrheic dermatitis: seborrheic lesions are yellowish and greasy, and do not itch.
2) Contact dermatitis: differentiate by absence or presence of history of allergic contact and by distribution of lesions (generally asymmetrical in contact dermatitis).

f. Treatment
1) Avoid rubbing and scratching. Use topical corticosteroids, oiled water, and emollients (to prevent dryness).
2) A systemic antihistamine may be necessary to treat pruritus.

3) A topical or systemic antibiotic may be necessary to treat secondary infection.

5. Dyshidrotic eczema (pompholyx eczema)
a. Etiology. Idiopathic. May be related to stress or atopy.
b. Epidemiology. Most patients are younger than 40 years.
c. History. Sudden appearance of blisters on the palms and soles associated with severe pruritus and burning. Patient often has a history of atopy. Hyperhidrosis may or may not be present.
d. Physical examination. Small vesicles that resemble tapioca are present in clusters on the hands or feet (Figure 4). Distribution is generally symmetrical.

e. Differential diagnosis
1) Contact dermatitis: differentiate by history.
2) Fungal infection: differentiate by mycologic examination.
3) Secondary syphilis: differentiate by serology.

f. Treatment. Limit hand washing, and apply moisturizer regularly. Apply topical
corticosteroids with occlusive cotton gloves and socks. Soak hands and feet with Burow’s solution. PUVA (psoralen plus ultraviolet A) photochemotherapy may be effective for severe resistant cases.

6. **Lichen simplex**
   a. **Definition.** Chronic inflammation caused by repeated rubbing and scratching
   b. **Etiology.** Idiopathic
   c. **Epidemiology.** Occurs primarily in patients older than 20 years and is more common in women
   d. **History.** Chronic pruritus; scratching becomes an inevitable and even pleasurable activity.
   e. **Physical examination.** Plaques of lichenification with minimal scaling. Generally hyperpigmented. Can occur as a single isolated lesion or as multiple scattered lesions. Most commonly occurs in the nuchal area, legs, and anogenital area (Figure 5).
   f. **Differential diagnosis**
      1) Contact dermatitis: differentiate by history.
      2) Psoriasis: scaling typical of psoriasis is absent in lichen simplex.
      3) Atopic dermatitis: atopic dermatitis is more chronic (it generally appears in childhood) and the area of distribution is different.
   g. **Treatment.** Treatment can be difficult. An antipruritic and/or anti-inflammatory agent should be prescribed to suppress scratching and rubbing, especially at nighttime. Because of its sedative properties, a good choice at bedtime is diphenhydramine. A topical corticosteroid agent should be applied and covered with an occlusive dressing. Intralesional corticosteroids are highly effective for small lesions.

B. **Acne vulgaris**
   1. **Definition.** Chronic inflammation of the pilosebaceous unit
   2. **Etiology.** The inflammatory process results from complex effects of androgens and Propionibacterium acne bacteria in the pilosebaceous unit. Some medications (eg, lithium, hydantoin, systemic corticosteroids) may worsen the condition. Acne is not caused by foods, although this is a common misconception.
   3. **Pathophysiology.** Androgens stimulate the sebaceous glands to increase sebum production. P. acne contains lipases that convert lipids into fatty acids. Sebum and fatty acids cause sterile inflammation of the pilosebaceous unit, resulting in hyperkeratinization and enlargement of the follicular lumen (ie, a whitehead). A black plug (a blackhead) is formed when tyrosine oxidizes to melanin. When the distended follicle wall breaks and the contents enter the dermis, a foreign-body response is provoked, resulting in formation of papules, pustules, and nodules.
   4. **Epidemiology.** Occurs mostly in adolescents. Although it is more severe in men than in women, acne may persist longer in women than in men.
5. **History.** Chronic recurring lesions that worsen in fall and winter and with stress. Lesions may be painful at times. In women, acne may be associated with a history of polycystic ovarian syndrome.

6. **Physical examination.** Lesions may be isolated or scattered. They occur mainly on the face, neck, upper arms, and trunk.
   a. Typical lesions of acne
      1) Comedones: open (blackheads) or closed (whiteheads)
      2) Papules with or without inflammation
      3) Nodules and cysts
      4) Atrophic and hypertrophic scars
   b. Seborrhea of the face may be present.

7. **Differential diagnosis**
   a. Acne: differentiate by age at onset and by absence of telangiectasia and flushing in acne. Comedones are not present in rosacea.
   b. Systemic lupus erythematosus (SLE): papules are not characteristic of SLE.

8. **Treatment**
   a. Mild cases may be treated with topical clindamycin or erythromycin and with benzoyl peroxide gels (5% to 10%).
   b. Severe cases may require treatment with oral antibiotics (eg, erythromycin, tetracyclines are good choices).
   c. Cystic lesions may be treated with oral 13-cis-retinoic acid (Accutane).

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**C. Rosacea**

1. **Definition.** A vascular condition characterized by chronic acneiform inflammation.
2. **Etiology.** Unknown. Capillaries dilate and pimples form in response to heat, certain foods, alcohol, medications, hormones, emotional stress, and chronic sun exposure. Reactivity of capillaries increases and the sebaceous glands enlarge, giving rise to the acne-like lesions.
3. **Epidemiology.** Onset is between 30 to 60 years of age. Occurs predominantly in women and in people with fair skin.
4. **History.** Chronic and frequent flushing. Lesions may be tender at times.
5. **Physical examination.** Face appears red with symmetrical telangiectasia, papules, and nodules. Cheeks, chin, forehead, and nose are most frequently affected (Figure 6). Rhinophyma is a characteristic manifestation.
6. **Differential diagnosis**
   a. Acne: differentiate by age at onset and by absence of telangiectasia and flushing in acne. Comedones are not present in rosacea.
   b. Systemic lupus erythematosus (SLE): papules are not characteristic of SLE.

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**D. Psoriasis**

1. **Etiology**
   a. Psoriasis is an immune system–modulated disease. Mitosis of keratinocytes is increased and inflammation in the dermis and epidermis is present.
   b. The appearance of lesions is multifactorial. Triggers include minor trauma (Koebner’s phenomenon), certain drugs (lithium, antimalarial agents, clonidine, indomethacin, angiotensin-converting enzyme inhibitors, and β-blockers), alcohol consumption, infection with β-hemolytic streptococcus, and exposure to sunlight.
2. **History.** Psoriasis may appear abruptly but generally has a chronic course. Pruritus may be generalized or localized. Psoriasis may be associated with arthritis (psoriatic arthritis) or with an acute illness syndrome.
3. **Physical examination.** Plaques and/or papules
are sharply demarcated and have white-silvery scaling. There may be erythroderma (ie, diffuse involvement of the skin without identifiable borders). It is generally bilateral, favoring exposed areas and areas of pressure such as the elbows and knees (Figure 7). Nails may be affected with pitting.

4. **Differential diagnosis**
   a. Seborrheic dermatitis: differentiate by distribution patterns and by the lack of the greasy scales that are characteristic of seborrheic dermatitis.
   b. Lichen simplex: this can complicate psoriasis as a result of chronic pruritus.
   c. Drug eruption: indicated by a history of taking a new medication.
   d. Syphilis: differentiate by serologic evaluation.
   e. Tinea: differentiate by mycologic evaluation.

5. **Treatment**
   a. All patients can benefit from lubricating the skin and avoiding rubbing and scratching of the lesions. Application of keratolytic agents containing salicylic acid can soften scales.
   b. Mild cases may be treated with topical agents, including coal tar, anthralin, topical corticosteroids of varying potency (depending on severity and extent of lesions), calcipotriene (a vitamin D derivative), and tazarotene (a receptor-selective retinoid).
   c. Treatment of severe cases may include the following:
      1) Phototherapy with ultraviolet B (UVB) light for extensive or resistant cases.
      2) PUVA phototherapy using methoxsalen, a photosensitizing psoralen agent given orally or as a bath or soaks. Possible side effects include nausea and increased risk of cataracts and some skin cancers.
      3) Systemic agents (eg, methotrexate, oral retinoids, cyclosporine) for severe or resistant cases.

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E. **Pityriasis rosea**

1. **Definition.** An acute, self-limited exanthem heralded by the appearance of a solitary lesion.
2. **Etiology.** Unknown; most likely viral.
3. **Epidemiology.** Occurs primarily in patients between the ages of 10 and 35 years.
4. **History.** A herald patch precedes the exanthem. Pruritus may or may not be present.

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5. **Physical examination.** Pityriasis rosea is characterized by fine, scaling, ovoid macules and papules of a dull pink color. Lesions are scattered discretely, following the lines of cleavage of the skin (Figure 8), forming a pattern resembling a Christmas tree. The rash is confined to the trunk and proximal areas of the extremities.

6. **Differential diagnosis**
   a. Drug eruption: differentiate by history.
   b. Syphilis: differentiate by serology.
   c. Guttate psoriasis: the marginal collarette typical of pityriasis rosea is not present in guttate psoriasis.

7. **Treatment.** Spontaneous remission occurs in 4 to 6 weeks. UVB therapy may relieve severe pruritus.

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### III. INFECTIOUS LESIONS

A. **Bacterial infections**

1. **Impetigo**
   a. **Definition and etiology.** Superficial infection of the skin caused by streptococcus (impetigo vulgaris) or staphylococcus (impetigo bullosa). Associated with crowding, poor hygiene, eczema, and scabies.
b. **Epidemiology.** Affects primarily children and young adults

c. **History.** Acute or recurrent. Several days of pruritus.

d. **Physical examination.** Thin-roofed vesicles later become crusted, giving way to erosions (*Figure 9*). Lesions are golden-yellow with central clearing. Occurs on face, arms, and legs. Autoinoculation causes satellite lesions.

e. **Differential diagnosis**
   1) Herpes simplex and varicella infection during the vesicular stage: microscopic examination of impetigo vesicles shows gram-positive cocci in chains and clusters.
   2) Tinea during the erosion stage: distinguish by mycologic evaluation.
   3) Infected eczema: distinguish by lack of pruritus in impetigo.

f. **Treatment.** Oral penicillin (erythromycin if the patient is allergic to penicillin). Topical bacitracin should be applied to the lesions, including lesions in the nostrils. Change sheets and towels daily to avoid autoinoculation.

2. **Erysipelas**
   a. **Definition.** Superficial cellulitis with involvement of lymphatics
   b. **Etiology.** Group A streptococcal infection
   c. **Epidemiology.** All ages
   d. **History.** Abrupt onset of tender, painful

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skin rash associated with an “acute illness” syndrome

e. **Physical examination.** The patient typically appears toxic and has fever. Bright red plaques have advancing elevated sharp margins, are warm to the touch, and are indurated. The cellulitis involves large areas in a diffuse arrangement and occurs predominantly on the face, abdomen, and legs.

g. **Differential diagnosis**
   1) Herpes zoster: erysipelas has no vesicles and no pain prior to onset.
   2) Contact dermatitis: fever or tenderness is not characteristic of contact dermatitis.
   3) Cellulitis: erysipelas lesions have sharp, raised margins.

h. **Treatment.** Penicillin V or erythromycin for 2 weeks
B. **Fungal infections**

1. **Tineas**
   a. **Definition and etiology.** These are superficial fungal infections caused by dermatophytes, which are fungi that thrive only on nonviable skin tissue (e.g., stratum corneum, nails, hair). The 4 main genera are *Microsporum*, *Trichophyton*, *Epidermophyton*, and *Pityrosporum*.

   b. **Epidemiology.** May occur at all ages. Immunosuppressed patients are more susceptible and more difficult to treat.

   c. **History.** Variable severity of pruritus.

   d. **Physical examination.** May affect any body part: scalp (tinea capitis), body (tinea corporis), hands (tinea manus), groin (tinea cruris), feet (tinea pedis). Erythema, scaling, hyperkeratosis, and vesicles are present to various degrees. Margins are sharp with central clearing and advancing crescentic edges (Figure 10).

   e. **Laboratory examination.** To confirm the diagnosis the lesions must be scraped, the skin placed on a glass slide, and a 10% to 20% potassium hydroxide (KOH) solution applied. Microscopic examination will reveal hyphae, arthrospores, or budding cells.

   f. **Differential diagnosis**
      1) Contact dermatitis: distinguish by history.
      2) Seborrheic dermatitis: distinguish by microscopic examination of skin scraping.

   g. **Treatment.** Options for treatment include application of topical imidazoles for 4 to 8 weeks, systemic antifungal treatment with imidazoles or griseofulvin, and topical application of 5% salicylic acid or 5% benzoic acid creams.

2. **Candidiasis**
   a. **Etiology.** Candida species, most commonly *C. albicans*

   b. **Epidemiology.** May occur at all ages. Pre-disposing factors include diabetes, frequent immersion of the hands in water, obesity, and immunosuppression.

   c. **History.** Acute or chronic, pruritic, and sometimes painful lesions.

   d. **Physical examination.** Candidiasis of the skin is characterized by a macerated appearance, erythema, and easily detachable cigarette paper-like collar scaling surrounding lesions. Commonly affected regions include the genital area and skin folds.

   e. **Laboratory evaluation.** Direct examination of skin scraping in KOH preparation reveals pseudomycelia and clusters of grapelike yeast cells.

   f. **Differential diagnosis**
      1) Eczema: differentiate by laboratory evaluation.
      2) Seborrheic dermatitis: differentiate by laboratory evaluation. Also, patterns of distribution differ.

   g. **Treatment.** Application of nystatin topical ointment, powder, or cream. The affected area should be kept dry by avoiding plastic or rubber pants, air-drying frequently, and wearing loose clothing. Oral antifungal agents may be necessary for resistant cases.

C. **Viral infections**

1. **Herpes simplex virus**
   a. **Etiology.** Herpes simplex virus (HSV) infections are caused by two different virus types. HSV-1 is generally associated with oral infections and rashes above the waist. HSV-2 generally causes genital lesions. However, because of oral-genital sexual contact, HSV-1 may cause genital lesions and HSV-2 may cause lesions above the waist.
b. Epidemiology
1) HSV-1. All age groups can be affected by HSV-1. HSV-1 may be spread by respiratory droplets, by direct contact with an active lesion, or by fluid containing virus (eg, saliva).
2) HSV-2. HSV-2 is a sexually transmitted disease, and hence most commonly affects sexually active adults. If HSV-2 is isolated in children, a history of sexual abuse should be considered.

c. History. HSV infections have 2 phases, primary infection and secondary phase.
1) Primary infection. Symptoms appear 3 to 7 days after contact. Local tenderness, pain, mild paresthesias, or burning occur before the vesicles appear. Other systemic symptoms (eg, fever, headaches, generalized malaise) may be present.
2) Secondary phase. During the primary phase, the virus enters the nerve endings in the skin and ascends through peripheral nerves to the dorsal root ganglia, where it remains in a latent stage. Any type of stress (eg, sunlight, illness, trauma, fatigue, menses, fever) may reactivate the virus, which then travels down the peripheral nerves to cause focal recurrent infections.

d. Physical examination. Grouped vesicles on an erythematous base appear and subsequently erode. Vesicles are uniform in size. Mucous membrane lesions accumulate exudate, whereas skin lesions form a crust. Lesions last 2 to 6 weeks, unless secondarily infected, then heal without scarring.

e. Diagnosis. Diagnosis is made clinically, with laboratory confirmation via Tzanck smear or viral cell culture. Best results are obtained from intact vesicles. Material for the Tzanck smear is obtained by deroofing vesicles with a surgical blade. Cells are obtained from the margins of the lesions, smeared on a glass slide, fixed for 1 minute with absolute alcohol, and then stained with Giemsa or Wright’s stain. Positive smears show giant cells with up to 15 nuclei.

f. Differential diagnosis. Herpes zoster should be considered in the differential diagnosis. In herpes zoster, vesicles are not uniform and may vary in size. Also, viral cultures will be negative for HSV and positive for varicella-zoster virus.

g. Treatment
1) Local: cold compresses, silver nitrate, and acyclovir ointment (applied every 3 hours for 7 days) may be prescribed.
2) Systemic: available antiviral agents include:
   a) Acyclovir: 800 mg every 4 hours for 7 to 10 days
   b) Famciclovir: 500 mg every 8 hours for 7 days
   c) Valacyclovir: 1 g every 8 hours for 7 days

2. Herpes zoster
a. Definition and etiology. Herpes zoster is an infection of a ganglion and the corresponding sensory nerve. It results from reactivation of varicella-zoster virus that is latent in the sensory ganglia.

b. Epidemiology. Typically affects adults 50 years and older and immunocompromised patients.

c. History. Pain and paresthesia in the involved dermatome precede the skin rash by several days. Fever, generalized malaise, and headaches may occur.

d. Physical examination. Manifestations include erythema, papules, vesicles, pustules, and crusts (Figure 11). Lesions are arranged in a dermatomal pattern.
Distribution is unilateral. Thoracic, cervical, trigeminal, and lumbosacral areas are most commonly affected. Painful lymphadenopathy is present.

d. **Differential diagnosis.** Several maculopapular exanthems occur in childhood. History and physical examination are crucial in the differential diagnosis (Table 2).

e. **Treatment.** Treatment is supportive unless complications occur.

### Table 2. Characteristics of Maculopapular Exanthems in Children

<table>
<thead>
<tr>
<th>Disease</th>
<th>Prodrome</th>
<th>Exanthem</th>
<th>Other Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Measles (Rubeola)</td>
<td>Fever, cough, conjunctivitis, coryza, ill appearance</td>
<td>Reddish-brown; begins on face and becomes confluent</td>
<td>Koplik’s spots, otitis media, encephalitis, pneumonia</td>
</tr>
<tr>
<td>Rubella</td>
<td>Minimal; lymphadenopathy</td>
<td>Pink, discrete; begins on face</td>
<td>Arthritis, encephalitis</td>
</tr>
<tr>
<td>Roseola infantum (exanthema subitum)</td>
<td>3–4 days of high fever preceding rash</td>
<td>Rose-colored, discrete; appears after defervescence</td>
<td>Febrile seizure</td>
</tr>
<tr>
<td>Erythema infectiosum (fifth disease)</td>
<td>None</td>
<td>Red-flushed cheeks; eruption of extremity may recur</td>
<td>Arthritis</td>
</tr>
<tr>
<td>Scarletina</td>
<td>Fever, vomiting, sore throat</td>
<td>Erythematous, punctate, sandpaper-like texture; favors flexor surface, skin folds</td>
<td>Cultures positive for group A Streptococcus, rheumatic fever, acute glomerulonephritis</td>
</tr>
<tr>
<td>Varicella (chickenpox)</td>
<td>Fever, malaise</td>
<td>Erythematous macules progressing to papules and vesicles; pruritic; central, distal sparing</td>
<td>Mucosal ulcers, dehydration, Reye’s syndrome, pneumonia, secondary bacterial infection, encephalitis</td>
</tr>
</tbody>
</table>

### BOARD REVIEW QUESTIONS

Choose the single best answer for each question.

**Questions 1 and 2 refer to the following case study.**

A 34-year-old woman presents with a pruritic rash on her trunk and extremities that began yesterday (**Figure Q1**). Three days ago she developed a sore throat and took some “tablets” that were left over from a prescription for a sore throat she had approximately 1 year ago. Her past medical history is positive for psoriasis affecting the elbows and knees only, which has been effectively controlled with triamcinolone ointment and sunbathing. Physical examination reveals salmon-pink lesions that are sharply demarcated and slightly indurated with white scales. The face, palms, soles, and nails are spared.

1. All of the following are included in the differential diagnosis of this rash EXCEPT:
   - A) Syphilis
   - B) Herpes zoster
   - C) Drug eruption
   - D) Pityriasis rosea
   - E) Guttate psoriasis
2. The patient is treated with penicillin V and betamethasone ointment but shows only mild improvement. You refer her to a dermatologist, who prescribes PUVA (psoralen plus ultraviolet A) photochemotherapy. Before accepting PUVA treatment, the patient consults you regarding the possible side effects of PUVA therapy. All of the following are possible side effects EXCEPT:

A) Cataracts
B) Nausea and malaise
C) Squamous cell carcinoma
D) Melanoma
E) Hair loss

3. A 3-year-old girl is brought into your office by her parents because of a bright red rash on her cheeks. Her mother says that the child has been well except for the rash. The child has received all of her immunizations, has had no fever, and has been playful. The family hired a new babysitter recently, and they are concerned that the new sitter may have slapped the child on the cheeks. What is the most likely diagnosis?

ANSWERS

1. (B) Figure Q1 depicts a characteristic appearance of guttate psoriasis (guttate: Latin, spots that resemble drops), an exanthem that often appears after a streptococcal pharyngitis. The differential diagnosis includes syphilis and pityriasis rosea. Because the patient refers to taking “tablets” for a sore throat, it is important to obtain a complete history of past drug allergies. This patient’s throat culture is positive for streptococcus, and guttate psoriasis is diagnosed.

2. (E) The most significant limiting side effects of PUVA therapy are nausea and malaise. Squamous cell carcinoma occurs in 1 to 4 of 1000 patients receiving PUVA. The risk of cataracts is increased, and the risk of melanoma is increased even after treatment ceases.3

3. (C) Erythema infectiosum, also known as slapped-cheek disease or fifth disease, is caused by parvovirus infection. It is characterized by sudden onset of coalescing red papules on the face followed 2 to 3 days later by a net-like rash of red maculopapules on the arms and legs.

REFERENCES