DERMATOLOGY

CHAPTER 6

1. **Cover the two right-hand columns and define the following common terms used in dermatology to describe skin findings:**

Term	*Definition*	*Examples*
Macule	Flat spot <1 cm (nonpalpable, just visible)	Freckles, tattoos
Patch	Same as macule but >1 cm	Port-wine birthmarks
Papule	Solid, elevated lesion <1 cm (palpable)	Wart, acne, lichen planus
Plaque	Same as papule but >1 cm and flat topped	Psoriasis
Nodule	Palpable, solid lesion >1 cm and not flat topped	Small lipoma, erythema nodosum
Vesicle	Elevated, circumscribed lesion <5 mm containing clear fluid (small blister)	Chickenpox, genital herpes
Bulla	Same as vesicle but >5 mm (large blister)	Contact dermatitis, pemphigus
Wheal	Itchy, evanescent, transiently edematous area	Allergic reaction

2. **Define vitiligo. With what diseases is it associated? What is the treatment?**
Vitiligo is characterized by well-demarcated macules or patches of skin depigmentation due to autoimmune destruction of melanocytes of unknown etiology (Fig. 6.1). It is an acquired condition associated with autoimmune diseases such as pernicious anemia, hypothyroidism, Addison disease, and type I diabetes. Patients often have antibodies to melanin, parietal cells, thyroid, or other factors. Vitiligo can be treated with topical corticosteroids, topical calcineurin inhibitors (e.g., tacrolimus, pimecrolimus), and phototherapy.

3. **Name several conditions to think about on the Step 2 exam in patients with pruritus**
Think of serious conditions first, such as obstructive biliary disease, uremia, and polycythemia rubra vera (classically seen after a warm shower or bath due to mast cell degranulation). Pruritus may also be caused by contact or atopic dermatitis, scabies, and lichen planus.

4. **Define contact dermatitis. How do you recognize it? What are the classic culprits?**
Contact dermatitis is usually due to a type IV hypersensitivity reaction, although it may also be due to an irritating or toxic substance. Look for a new exposure to a classic offending agent, such as poison ivy, nickel earrings, or deodorant. Allergic contact dermatitis requires a previous sensitizing event as opposed to irritant contact dermatitis, which does not. The rash is well circumscribed and occurs only in the area of exposure. The skin is red and itchy and often has vesicles or bullae (Fig. 6.2). Avoidance of the agent is required. Patch testing can be done, if needed, to determine the antigen.

5. **Define atopic dermatitis. What history points to this diagnosis? What is the treatment?**
Atopic dermatitis, also known as eczema, is a chronic allergic-type condition that begins in the first year of life with red, itchy, weeping skin on the head, upper extremities on extensor surfaces, and sometimes around the diaper area. In children and adults, it usually develops on the flexor surfaces. The clue to diagnosis is a family and/or personal history of allergies (e.g., hay fever) and asthma. The biggest problem is scratching of affected skin, which leads to skin breaks and possible bacterial infection. Treatment involves avoidance of drying soaps and use of antihistamines, moisturizing creams, topical steroids, and immune modulating agents (topical pimecrolimus or tacrolimus).

6. **Define seborrheic dermatitis. What part of the body does it involve? How is it treated?**
Seborrheic dermatitis causes the common conditions known as cradle cap and dandruff as well as blepharitis (eyelid inflammation). Look for scaling skin with or without erythema on the hairy areas of the head (scalp, eyebrows, eyelashes, mustache, beard) as well as on the forehead, nasolabial folds, external ear canals, and postauricular creases (Fig. 6.3). Treat with dandruff shampoo (e.g., selenium sulfide or tar shampoo), topical corticosteroids, and/or ketoconazole cream.

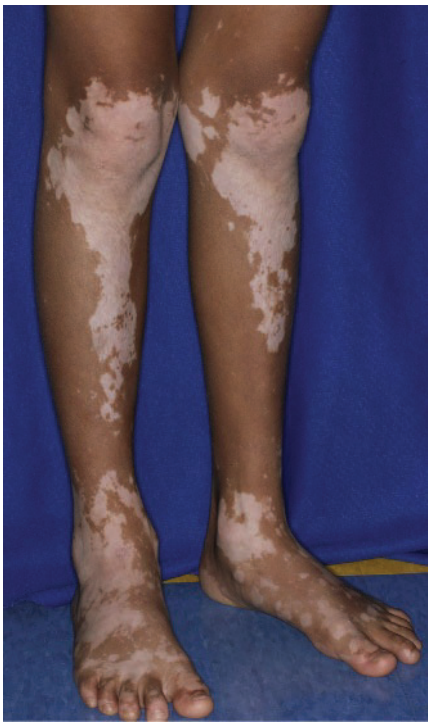

Fig 6.1 Vitiligo. Symmetric depigmentation of the knees and lower extremities. (From Paller AS, Mancini AJ. *Hurwitz Clinical Pediatric Dermatology, A Textbook of Skin Disorders of Childhood and Adolescence.* 5th ed. Philadelphia: Elsevier; 2016.)

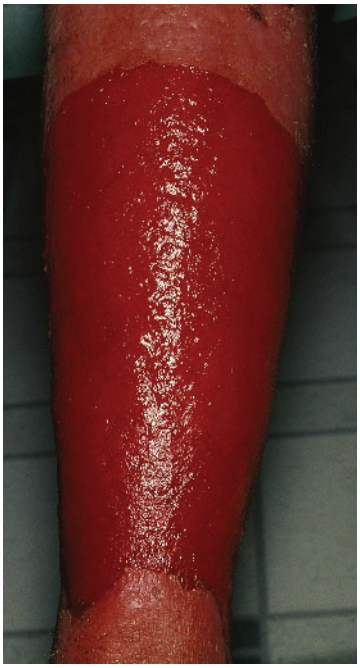

Fig. 6.2 Allergic contact dermatitis of the leg caused by an elastic wrap. Notice the well-marginated distribution that differentiates it from cellulitis. (From Auerbach PS. *Wilderness Medicine.* 6th ed. Philadelphia: Mosby; 2011.)

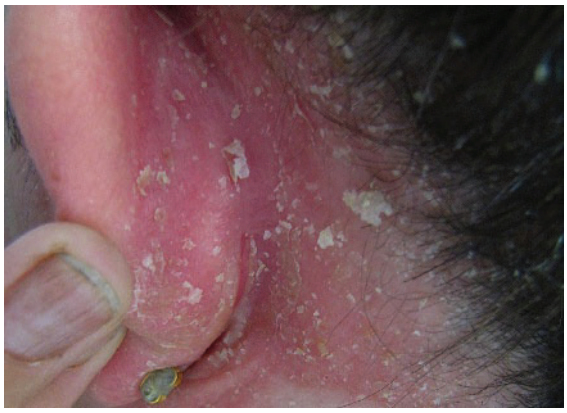

Fig. 6.3 Adult seborrheic dermatitis. Fairly sharply demarcated pink plaque with white and greasy scale. Note the fissure in the retroauricular fold. (From Bolognia J, Schaffer J. *Dermatology Essentials*. Philadelphia: Saunders; 2014:103-108. Photo courtesy of Norbert Reider, MD, and Peter O Fritcsch, MD.)

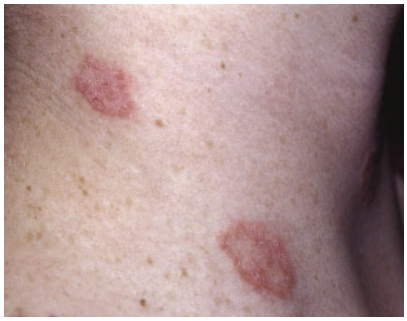

Fig. 6.4 Tinea corporis. Red ring-shaped lesions with scaling and some central clearing. (From Kliegman RM. *Nelson Textbook of Pediatrics*. 19th ed. Philadelphia: Saunders; 2011.)

7. Name the various dermatologic fungal infections

Known as dermatophytosis, tinea, and ringworm. Fungal infections include the following:

Tinea corporis (body/trunk): look for red ring-shaped lesions with raised borders that tend to clear centrally while they expand peripherally (Fig. 6.4).
Tinea pedis (athlete's foot): look for macerated, scaling web spaces between the toes that often itch and may be associated with thickened, distorted toenails (onychomycosis). It may be acquired from using locker rooms or swimming pools. Treatment includes good foot hygiene and disposal of old footwear (or treatment with antifungal powder).
Tinea unguium (onychomycosis): thickened, distorted, and discolored nails with debris under the nail edges.
Tinea capitis (scalp): mainly affects children (highly contagious), who have scaly patches of hair loss with residual "black dots" in the affected area and may have an inflamed, boggy granuloma of the scalp (known as a kerion) that usually resolves on its own.
Tinea cruris (jock itch): more common in obese males; is usually found in the crural folds of the upper, inner thighs. Increased prevalence in patients with diabetes or other immunodeficiency.

8. What organisms cause fungal infections?

Most fungal infections are due to *Trichophyton* species. In tinea capitis, if the hair fluoresces green under the Wood lamp, *Microsporum* species is the cause; if not, it is probably *Trichophyton.*

9. How are fungal infections diagnosed and treated?

Formal diagnosis of any fungal infection can be made by scraping the lesion and doing a potassium hydroxide (KOH) preparation to visualize the fungus via a microscope or by doing a culture. Because they are so common clinically, empiric treatment without a formal diagnosis is common, but for the United States Medical Licensing

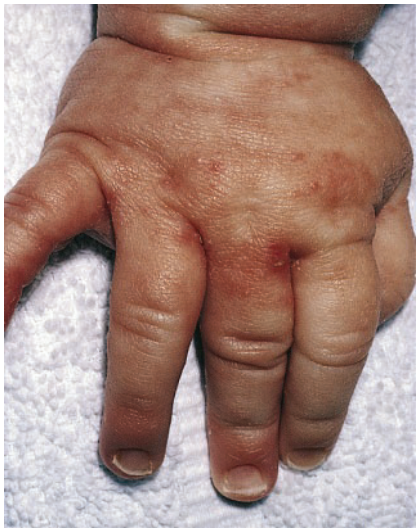

Fig. 6.5 Scabies. Itchy papules and pustules on the web spaces of the hand. (From Paige D, Wakelin S. *Kumar and Clark's Clinical Medicine*. Philadelphia: Elsevier; 2017:1337–1386.)

Examination (USMLE), get a formal diagnosis before treating. Oral antifungals (e.g., griseofulvin) must be used to treat tinea capitis and onychomycosis; the others can be treated with topical antifungals (imidazoles such as miconazole, clotrimazole, and ketoconazole or allylamines such as terbinafine) or oral griseofulvin, which is better for severe or persistent infections.

10. True or false: Candidiasis is often a normal finding in some women and children

True. Oral thrush (creamy white patches on the tongue or buccal mucosa that can be scraped off) is seen in normal children, and *Candida* vulvovaginitis is seen in normal women, especially during pregnancy or after taking antibiotics. However, at other time periods and in different patients, candidal infections may be a sign of diabetes or immunodeficiency; for example, thrush in a man should make you think about the possibility of AIDS, and recurrent vulvovaginal candidiasis should prompt screening for diabetes.

11. How is candidiasis diagnosed and treated?

Diagnose with KOH prep and look for pseudohyphae. Treat with local/topical nystatin or imidazoles (e.g., miconazole, clotrimazole). Oral therapy (nystatin or ketoconazole) is used for extensive or resistant disease.

12. What causes scabies? How do you recognize it?

Scabies is caused by the mite *Sarcoptes scabiei,* which tunnels into the skin and leaves visible burrows on the skin, classically in the finger web spaces and flexor surface of the wrists (Fig. 6.5). You should know what these burrows look like. Facial involvement is sometimes seen in infants. Patients also have severe pruritus, and scratching can lead to secondary bacterial infection. Crusted or Norwegian scabies typically occurs in patients who are immunocompromised, elderly, or living in institutions. In this form of scabies, there are hundreds to thousands of mites in the skin (as opposed to 15–20 with regular scabies), leading to a scaly rash or plaque, which may resemble psoriasis.

13. How do you diagnose and treat scabies?

Diagnosis is made by scraping a mite out of a burrow and viewing it under a microscope, though it is often a clinical diagnosis. Treat scabies with 5% permethrin cream applied to the whole body. Remember to treat all contacts (e.g., the whole family). Do *not* use lindane unless permethrin is not an option. Lindane used to be the treatment of choice but can cause neurotoxicity, especially in young children. Oral ivermectin can also be used. Close contacts may also require treatment. The patient's clothing, bedding, and towels must be cleaned or placed in a plastic bag for >3 days (mites can only live for 2–3 days away from human skin) to prevent reinfection.

14. How do you recognize and treat tinea versicolor?

Tinea versicolor (also known as pityriasis versicolor) is usually caused by the *Malassezia globosa* fungus, presenting most commonly with multiple patches of various size and color (brown, tan, and white) on the torso of young adults (Fig. 6.6). It often becomes noticeable in the summer because the affected areas fail to tan and look white. Diagnose from lesion scrapings (KOH preparation yields septated hyphae and yeast in a "spaghetti and meatball" pattern). Treat with selenium sulfide shampoo or topical imidazoles.

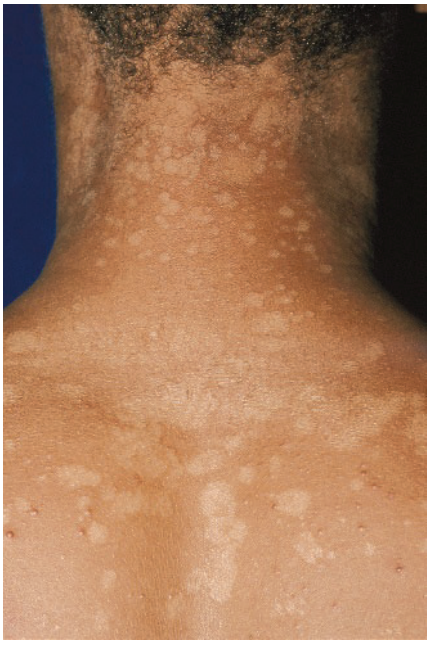

Fig. 6.6 Tinea versicolor. Hypopigmented macules and patches of seborrheic areas of the trunk. (From Paller AS, Mancini AJ. *Hurwitz Clinical Pediatric Dermatology, A Textbook of Skin Disorders of Childhood and Adolescence.* 5th ed. Philadelphia: Elsevier; 2016.)

15. What causes lice? How is lice treated?

Lice (pediculosis) can involve the hair of the head (caused by *Pediculus capitis;* common in school-aged children), body (caused by *Pediculus corporis;* unusual in people with good hygiene), or pubic area (crabs, caused by *Phthirus pubis* and transmitted sexually). Infected areas tend to itch. Diagnosis is made by seeing the lice (live mites or nits) on hair shafts. Treat with permethrin cream (preferred over lindane because of lindane's neurotoxicity) and decontaminate sources of reinfection (wash or sterilize combs, hats, bed sheets, clothing).

16. What causes warts? How are they treated?

Warts are caused by the human papillomavirus (HPV). They are infectious and are most commonly seen in older children, classically on the hands. They are spread by skin-to-skin contact. The most common serotypes are 6 and 11. Multiple treatments are available, including salicylic acid, liquid nitrogen, curettage, cytostatic treatment (5-fluorouracil, trichloroacetic acid), and immune response modifiers (imiquimod and interferon-α). Genital warts are also caused by HPV. Approximately 15 of the HPV serotypes are considered to be high-risk types for the development of cervical cancer; serotypes 16 and 18 are associated with the majority of cases of cervical cancer.

17. Define molluscum contagiosum. How do you recognize it? How is it treated?

Molluscum contagiosum is a poxvirus infection that is common in children but may also be sexually transmitted. Diagnosis is made by the characteristic appearance of the lesions (skin-colored, smooth, waxy, dome-shaped papules with a central depression [umbilicated] that are roughly 0.5 cm) or by looking at contents of the lesion, which include cells with characteristic inclusion bodies (Fig. 6.7). The usual treatment is freezing or curettage. Consider immunodeficiency if the lesions are giant or very diffuse.

18. True or false: A child with genital molluscum is probably a victim of sexual abuse

False. A child who has genital molluscum may or may not have contracted the disease from sexual contact. The more common mechanism is autoinoculation, in which the child has a lesion on the hand that spreads to the genital area from scratching. Do *not* automatically assume child abuse, although it must be ruled out.

19. How is acne described in medical terms? What bacteria may be partially involved in its pathogenesis?

Acne vulgaris can be broken down into various subtypes, including comedonal, inflammatory, and nodular (cystic) acne. Comedonal acne presents with closed (whitehead) or open (blackhead) comedones primarily on the forehead, nose, and chin. Inflammatory acne presents with small (<5 mm), erythematous papules and pustules. Nodular (cystic) acne presents with large (>5 mm) nodules that may merge to form sinus tracts and subsequent scarring. *Propionibacterium acnes* is thought to be partially involved in pathogenesis, as is blockage of pilosebaceous glands.

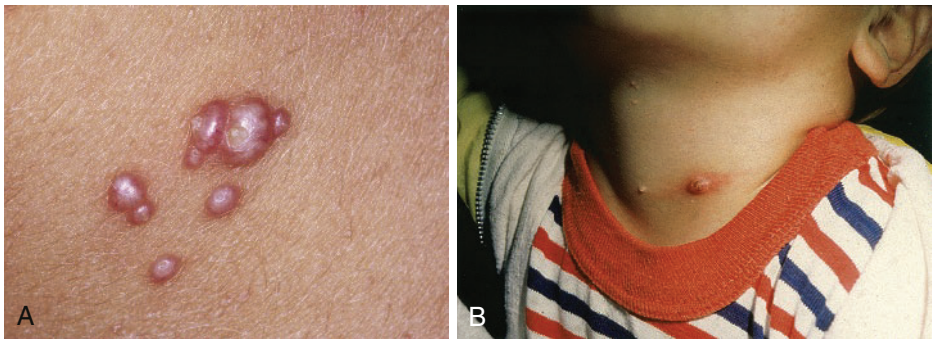

Fig. 6.7 Molluscum contagiosum. (A) Multiple papules of molluscum contagiosum demonstrating a characteristic central keratotic core. (B) Inflammatory molluscum contagiosum in a young child demonstrating both small, waxy, umbilicated papules and an inflammatory lesion simulating a furuncle. (From Nguyn N, Reed B. *Dermatology Secrets Plus.* Philadelphia: Elsevier; 2016:229-234. A, Courtesy James E. Fitzpatrick MD; B, courtesy Fitzsimons Army Medical Center teaching files.)

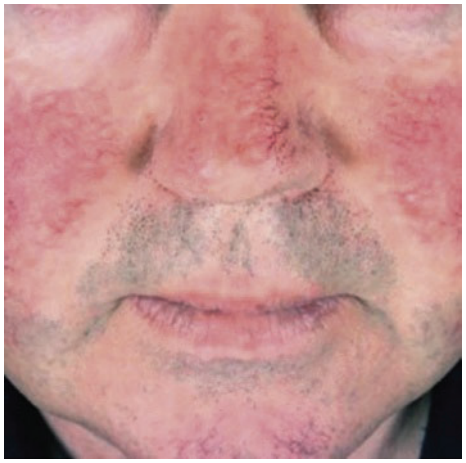

Fig. 6.8 Rosacea. Telangiectasias and erythema due to chronic actinic damage. (From Bolognia J, Schaffer J. *Dermatology Essentials.* Phildelphia: Saunders; 2014:261-267.)

20. True or false: Acne is not related to food, exercise, or sex

True. Acne has *not* been proven to be related to food, exercise, or sex (including masturbation). However, if the patient relates acne to a food, you can try discontinuing it. Cosmetics may aggravate acne.

21. What are the treatment options for acne?

Treatment options are multiple. Start with topical retinoids and salicylic or azelaic acids; then try topical benzoyl peroxide, topical clindamycin or erythromycin, either with or without an oral antibiotic (typically a tetracycline or erythromycin for *Propionibacterium acnes* eradication). Oral isotretinoin is the *last resort.* Although highly effective, isotretinoin is teratogenic; pregnancy testing in women before and during therapy as well as contraceptive use is mandatory. Women of childbearing age must be on two forms of contraception. In addition, it may cause dry skin and mucosae, muscle and joint pain, and liver function test abnormalities.

22. Define rosacea. In what age group is it seen? How do you treat it?

Rosacea often looks like acne but begins in middle age. There are several different subtypes (papulopustular, erythematotelangiectasia, ocular) of rosacea. The most common is the papulopustular subtype. Typically, patients present with facial erythema and flushing (Fig. 6.8). There are numerous triggers for rosacea, including sun exposure, emotional stress, alcohol consumption, spicy foods, and hot weather. Also look for **rhinophyma** (bulbous red nose) and coexisting blepharitis. Treat the papulopustular subtype with topical metronidazole or oral tetracycline. Treat the erythematotelangiectasia subtype with topical brimonidine. The pathogenesis is incompletely understood.

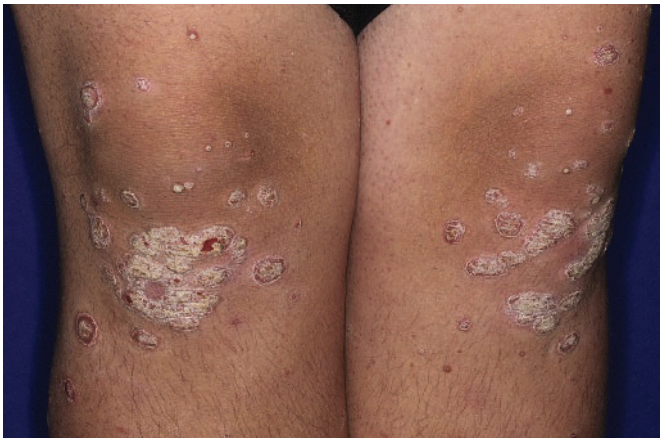

Fig. 6.9 Typical plaques of psoriasis with thick scale overlying erythema. (From Paller AS, Mancini AJ. *Hurwitz Clinical Pediatric Dermatology, A Textbook of Skin Disorders of Childhood and Adolescence.* 5th ed. Philadelphia: Elsevier; 2016.)

23. What should you think about if hirsutism is described on the Step 2 exam?

Hirsutism is most commonly idiopathic, but other signs of virilization (e.g., deepening voice, clitoromegaly, frontal balding) suggest an androgen-secreting ovarian tumor (check serum androgen levels). The most common cause of hirsutism in a female is polycystic ovary syndrome. Also consider Cushing syndrome and drugs (minoxidil, corticosteroids, and phenytoin).

24. What are the common pathologic causes of baldness?

Watch out for trichotillomania (a psychiatric disorder in which patients pull out their hair; baldness is patchy and irregular), alopecia areata (idiopathic but associated with antimicrosomal and other autoantibodies), and telogen effluvium (caused by stress). Baldness may also be seen in patients with lupus erythematosus or syphilis and after cancer chemotherapy.

25. What causes ordinary male pattern baldness?

Although the exact pathophysiology is still not clear, male pattern baldness is considered a genetic disorder that requires androgens for expression.

26. Describe the classic psoriatic lesion

Psoriatic lesions are classically described as dry, well-circumscribed, erythematous, silvery, scaling papules and plaques that are *not* pruritic (Fig. 6.9). Classic lesions are found on the scalp, lumbosacral region, intergluteal clefts, and extensor surfaces of the elbows and knees. Look for Auspitz sign, which is a small amount of bleeding when a psoriatic scale is scraped away. It is caused by abnormal proliferation of keratinocytes.

27. What other historical points and physical findings may be seen with psoriasis? How is it diagnosed and treated?

A family history of psoriasis is often present, and the disease mostly occurs in Whites with onset in early adulthood. Affected patients may have pitting of the nails and an arthritis that resembles rheumatoid arthritis but is rheumatoid factor negative. Diagnosis of psoriasis can often be made by appearance alone, but a biopsy can be used in doubtful cases. Certain types of psoriasis are associated with infectious causes. For example, diffuse, sudden-onset psoriasis is associated with human immunodeficiency virus (HIV) infection. Guttate psoriasis (scaly, droplike plaques/papules) typically occurs after a streptococcal infection. Treatment is complex but involves exposure to ultraviolet light, lubricants, topical corticosteroids, calcipotriene, and keratolytics (e.g., coal tar, salicylic acid, anthralin). Oral therapies may include immunosuppressive and immunomodulating drugs such as methotrexate, cyclosporine, and biologic agents.

28. Give the classic description and natural course of pityriasis rosea

Pityriasis rosea is typically seen in young adults. Look for a herald patch (slightly erythematous, scaly, ring-shaped or oval patch classically seen on the trunk), followed 1 week later by many similar lesions that tend to itch (Fig. 6.10). Look for lesions on the back with a long axis that parallels the Langerhans skin cleavage lines, typically in a Christmas tree pattern. The condition usually remits spontaneously in about 1 month. The etiology is unknown, but some think it is related to human herpesvirus 6 (HHV-6) and HHV-7. Think about syphilis (which presents with a maculopapular rash in the secondary form) in the differential diagnosis. Treat with reassurance.

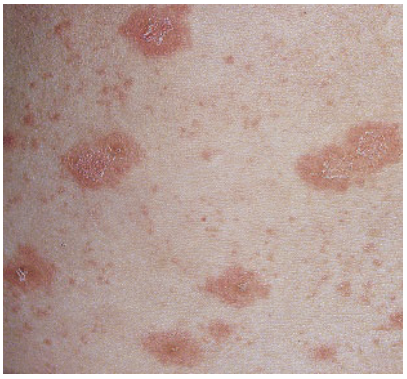

Fig. 6.10 Pityriasis rosea. Both small, oval plaques and multiple, small papules are present. (From Habif TP. *Clinical Dermatology*. 5th ed. St. Louis: Mosby; 2009.)

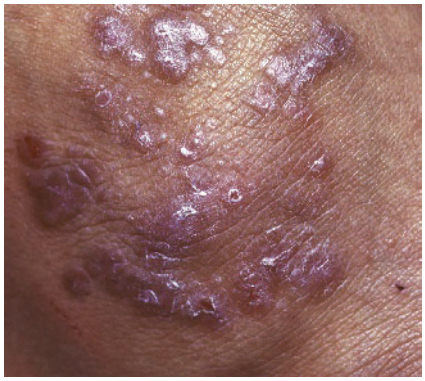

Fig. 6.11 Lichen planus. Flat-topped, purple polygonal papules of lichen planus. (From Kliegman RM. *Nelson Textbook of Pediatrics*. 19th ed. Philadelphia: Saunders; 2011.)

29. What are the four Ps that clinch a diagnosis of lichen planus?

Pruritic, **p**urple, **p**olygonal **p**apules (or plaques) classically on the wrists, lower legs, or genitalia, usually of adults (Fig. 6.11). Oral mucosal lesions with a whitish, lacelike pattern (Wickham striae) may also be present. These oral lesions must be monitored as they may increase the risk for oral cancer. It is associated with hepatitis C virus (HCV) infection.

30. List the classic drugs that cause photosensitivity of the skin

Tetracyclines, phenothiazines, and birth control pills. Other drugs include furosemide, hydrochlorothiazide, antipsychotics (chlorpromazine, prochlorperazine), fluoroquinolones, amiodarone, and promethazine.

31. Describe the classic lesion of erythema multiforme. What drugs classically cause it?

Look for the classic target (iris) lesions (Fig. 6.12). The classic cause is sulfa drugs or penicillins, but herpes infections may also cause erythema multiforme, and some cases are idiopathic. Erythema multiforme exists on a spectrum. As it becomes more severe and widespread, it is known as **Stevens-Johnson syndrome** (**SJS**), which is often fatal. SJS encompasses <10% of skin. Patients with SJS are treated supportively with therapy similar to what a burn victim would receive (wound care, fluid and electrolyte management, pain control, nutritional support, and monitoring for and treatment of superinfections). If >30% of skin is involved, it is classified as toxic epidermal necrolysis.

32. Describe the classic lesion of erythema nodosum. With what diseases is it commonly associated? What should the workup include?

Erythema nodosum (Fig. 6.13) is an inflammation of the subcutaneous tissue and skin, classically over the shins (pretibial). Look for tender, red, elevated nodules. Sarcoidosis, coccidioidomycosis, or ulcerative colitis classically accompany this condition on the USMLE, though multiple other infections (e.g., streptococcal, tuberculosis [TB]) and drugs (e.g., sulfonamides) can also result in this finding. The most common vasculitis associated

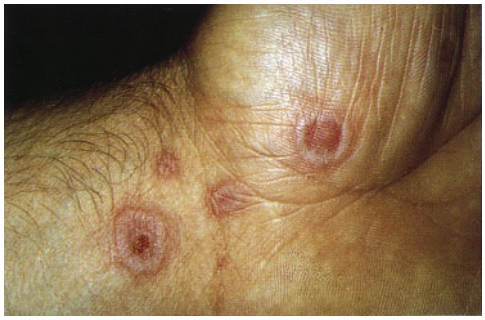

Fig. 6.12 Erythema multiforme. Bull's-eye annular lesions with central vesicles and bullae. (From Goldman L. *Goldman's Cecil Medicine.* 24th ed. Philadelphia: Saunders; 2011.)

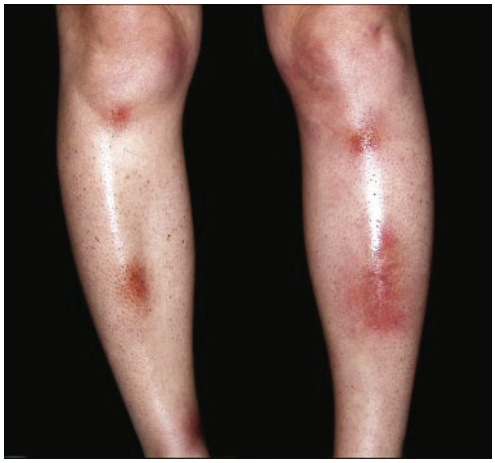

Fig. 6.13 Erythema nodosum on the legs of a young woman. (From Hochberg MC. *Rheumatology.* 5th ed. Philadelphia: Mosby; 2010.)

with it is Behcet disease. Treat the underlying disease and provide symptomatic therapies such as nonsteroidal antiinflammatory drugs (NSAIDs), leg elevation, and compressive bandages. The workup should include basic laboratory testing, TB skin testing, antistreptolysin-O antibodies, and a chest x-ray to look for sarcoidosis and TB.

33. Define and describe pemphigus vulgaris. How is it different from bullous pemphigoid?

Pemphigus vulgaris is a potentially life-threatening autoimmune disease of middle-aged and elderly patients. It presents with multiple flaccid bullae, starting in the oral mucosa and spreading to the skin of the rest of the body. These bullae rupture easily. Look for Nikolsky sign, which is sloughing and ulcerations that occur when minor pressure is applied to the skin. Biopsy can be stained for antibody (an IgG antibody to desmoglein III, which is associated with desmosomes) and shows a lacelike or fishnet immunofluorescence pattern. Tombstone cells are seen on histology. Treat with oral corticosteroids.

Bullous pemphigoid is a similar but milder condition that often presents as multiple tense bullae all over the body. Biopsy reveals a linear immunofluorescence pattern (different antibody), and this condition is also treated with oral corticosteroids (Fig. 6.14). Nikolsky sign is not present with bullous pemphigoid. Bullous pemphigoid is due to antibodies directed against the hemidesmosome. It is associated with certain neurologic diseases (Parkinson disease, multiple sclerosis) and malignancy. Treat with topical corticosteroids.

34. What skin disease is associated with celiac disease (gluten intolerance or sensitivity)? How is it treated?

Dermatitis herpetiformis is associated with celiac disease. Patients have intensely pruritic vesicles, papules, and wheals on the extensor aspects of the elbows and knees and possibly on the face or neck (Fig. 6.15). Look for diarrhea and weight loss (due to gluten sensitivity). On biopsy, the skin has IgA deposits even in unaffected areas. Test for celiac disease, and treat both conditions with a gluten-free diet. Additionally, dapsone can be used for acute treatment.

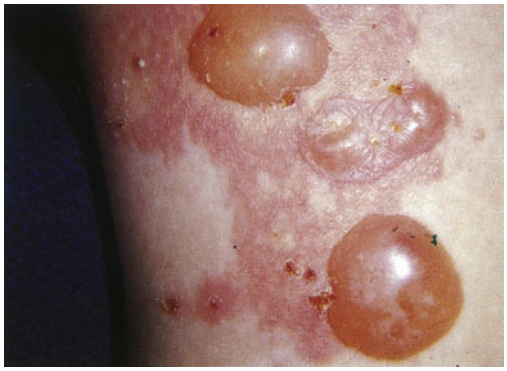

Fig. 6.14 Bullous pemphigoid. Tense subepidermal bullae on an erythematous base. (From Goldman L. *Goldman's Cecil Medicine*. 24th ed. Philadelphia: Saunders; 2011.)

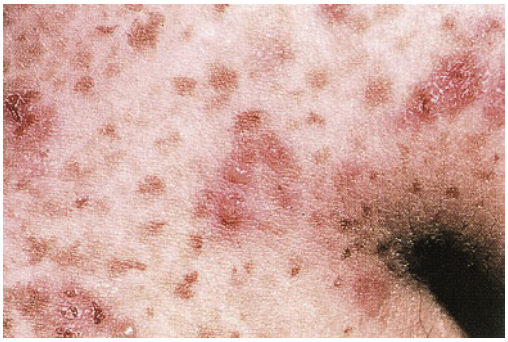

Fig. 6.15 Dermatitis herpetiformis is characterized by pruritus, urticarial papules, and small vesicles. (From Feldman M. *Sleisenger and Fordtran's Gastrointestinal and Liver Disease*. 9th ed. Philadelphia: Saunders; 2010. Courtesy of Dr. Timothy Berger, San Francisco, CA.)

35. What are decubitus ulcers? What is the best method of prevention?

Decubitus ulcers (bedsores or pressure sores) are skin ulcers caused by prolonged pressure against the skin. The best treatment is prophylaxis. Periodic turning of paralyzed, bedridden, or debilitated patients (the populations in which they are most common) and use of special air mattresses prevents bedsores. Cleanliness and dryness also help to prevent decubitus ulcers. Periodic skin inspection ensures that the problem is recognized early. When missed, the lesions can ulcerate down to the bone and become infected, possibly leading to sepsis and death. Treat major skin breaks with aggressive surgical debridement; if signs of infection are present, administer antibiotics.

36. How are decubitus ulcers staged?

Stage 1 is intact skin with nonblanchable redness of a localized area. Stage 2 is partial-thickness loss of the dermis presenting as a shallow open ulcer. These may also present as an intact or ruptured blister. Stage 3 is full-thickness tissue loss. Subcutaneous fat may be visible, but bone, tendon, or muscle is not exposed. Stage 4 is full-thickness skin loss with exposed bone, tendon, or muscle. An unstageable ulcer has full-thickness loss in which the base of the ulcer is covered with slough or eschar. The true depth of the ulcer cannot be determined until the slough or eschar is removed.

37. What conditions should excessive perspiration suggest on the USMLE?

We all know people who sweat too much for no apparent reason. On the Step 2 exam, however, look for a serious cause, such as a myocardial infarction, tuberculosis or infection, hyperthyroidism, or pheochromocytoma.

38. True or false: Most melanomas start out as simple moles

True. Moles are common and benign, but malignant transformation is possible (Fig. 6.16). **ABCDE characteristics of a mole** that should make you suspicious of malignant transformation: **a**symmetry, **b**orders (irregular), **c**olor (change in color or multiple colors), **di**ameter (the bigger the lesion, the more likely that it is malignant), and **e**volution over time. Excise any mole (or do a biopsy if the lesion is very large) if it enlarges suddenly, develops irregular borders, darkens or becomes inflamed, changes color (even if only one small area of the mole changes color), begins to bleed, begins to itch, or becomes painful.

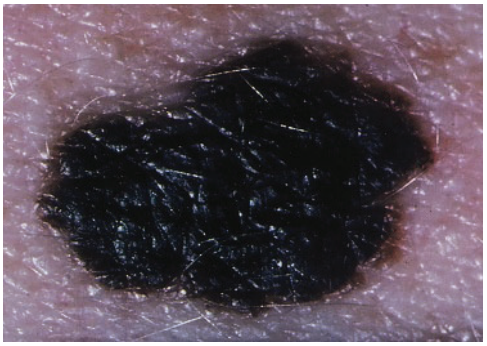

Fig. 6.16 Melanoma (superficial spreading type). (From Goldman L, Schafer AL. *Goldman's Cecil Medicine*. 24th ed. Philadelphia: Saunders; 2011.)

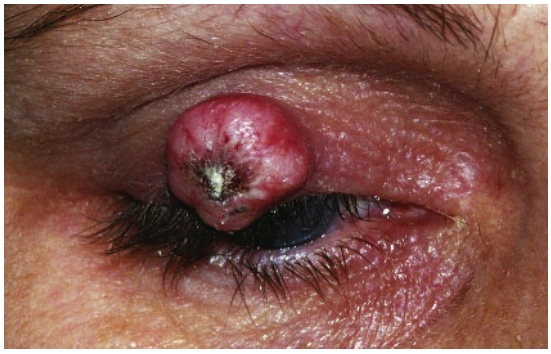

Fig. 6.17 Keratoacanthoma on the right upper lid. Lesions are solitary, smooth, dome-shaped red papules or nodules with a central keratin plug. (From Albert DM. Albert & Jakobiec's Principles and Practice of Ophthalmology. 3rd ed. Philadelphia: Saunders; 2008.)

39. Define dysplastic nevi syndrome. How is it managed?

Dysplastic nevus syndrome is a genetic condition with multiple dysplastic-appearing nevi (usually >100 moles). Also look for a family history of melanoma. Treat with careful and regular follow-up, excision or biopsy of any suspicious lesions, avoidance of sun exposure, and sunscreen use.

40. Why is keratoacanthoma of note?

Keratoacanthoma can mimic skin cancer (especially squamous cell cancer). Look for a flesh-colored lesion with a central crater that contains keratinous material, classically on the face (Fig. 6.17). Keratoacanthoma has a very rapid onset and grows to its full size in 1–2 months (which almost never happens with squamous cell cancer). The lesion involutes spontaneously in a few months and requires no treatment. If unsure, the best step is a biopsy, but choose observation as the answer in patients with a classic history of keratoacanthoma.

41. When and where are keloids seen?

Keloids are overgrowths of scar tissue after an injury and extend beyond the margins of the original wound. They are seen most frequently in Blacks. They are usually slightly pink and classically appear on the upper back, chest, and deltoid area. Also look for keloids to develop after ear piercing (Fig. 6.18). Do not excise these lesions because it may worsen scarring.

42. Describe the classic lesion of basal cell cancer. What should you do if you suspect it?

Basal cell cancer classically begins as a shiny papule on a skin-exposed area (the head is classic) and slowly enlarges and develops an umbilicated center with pearly borders (and later may ulcerate and bleed easily) with peripheral telangiectasias (Fig. 6.19). Like all skin cancers, sunlight exposure increases the risk. It is more common in elderly, light-skinned people. Treat with excision. Biopsy any suspicious skin lesions in the elderly. It is the most common type of skin cancer.

43. True or false: Basal cell skin cancer almost never develops metastases

True. However, it may be locally invasive and destructive.

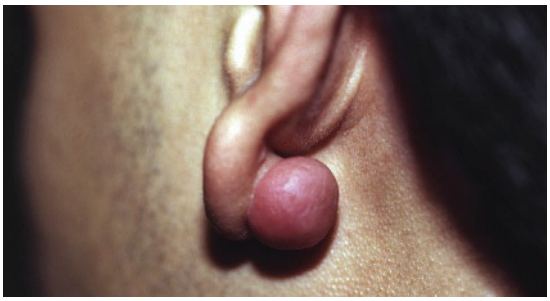

Fig. 6.18 Keloid of the ear lobe after piercing. (From Kliegman RM, Stanton BF, St. Geme JW, et al. *Nelson Textbook of Pediatrics*. 19th ed. Philadelphia: Saunders; 2011.)

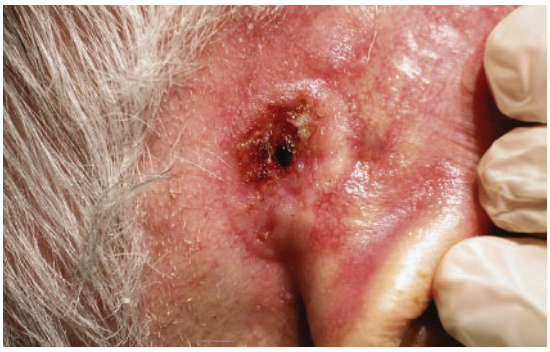

Fig. 6.19 An ulcerated basal cell carcinoma with rolled borders on the posterior ear. (From Abeloff DA, Armitage JO, Nienderhuber JE. *Abeloff's Clinical Oncology*. 4th ed. Philadelphia: Churchill Livingstone; 2008.)

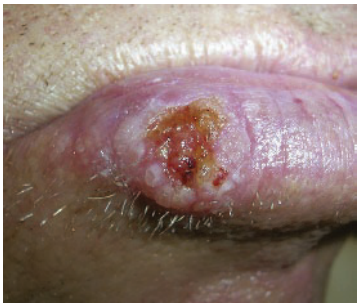

Fig. 6.20 Squamous cell carcinoma on the lower lip. (From Rakel RE. *Textbook of Family Medicine*. 8th ed. Philadelphia: Saunders; 2011. © Richard P. Usatine.)

44. From what lesion does squamous cell cancer classically develop? What is Bowen disease?
Squamous cell cancer (Fig. 6.20) often develops in areas with preexisting actinic keratoses (hard, sharp, red, often scaly lesions in sun-exposed areas; Fig. 6.21) or burn scars. The lesions become nodular, warty, or ulcerated; do a biopsy if such transformation occurs. Squamous cell cancer in situ is known as Bowen disease, and lesions are typically well demarcated. Although metastases are rare in squamous cell cancer, they occur more frequently than in basal cell cancer. They can also cause numbness/paresthesias due to early perineural invasion.

45. To what parameter is the prognosis of a malignant melanoma most closely related?
The Breslow thickness (or depth) of the tumor. The 10-year survival rate decreases as the thickness of the tumor increases. Tumors <1.0 mm thick have the best prognosis.

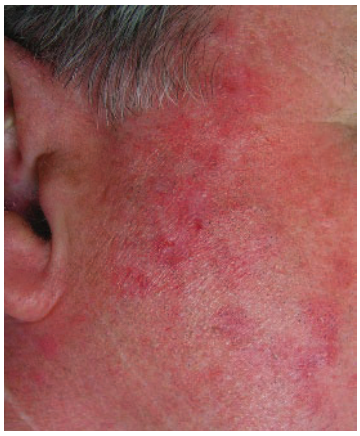

Fig. 6.21 Multiple actinic keratoses visible as thin, red, scaly lesions. (From Goldberg DJ. *Procedures in Cosmetic Dermatology: Lasers and Lights* [vol. 1]. 2nd ed. Philadelphia: Saunders; 2008.)

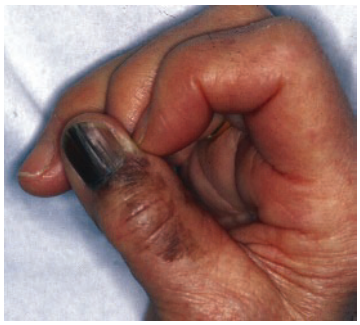

Fig. 6.22 Nailbed melanoma. (From Goldman L, Schafer AL. *Goldman's Cecil Medicine*. 24th ed. Philadelphia: Saunders; 2011.)

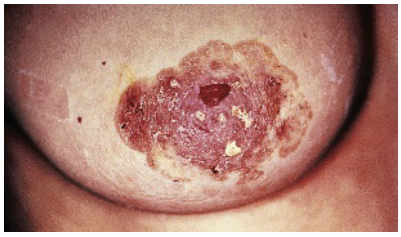

Fig. 6.23 Paget disease of the breast. Note the erythematous plaques around the nipple. (From Lentz GM, Lobo RA, Gershenson DM, et al. *Comprehensive Gynecology*. 6th ed. Philadelphia: Mosby; 2011. Originally from Callen JP. Dermatologic signs of systemic sisease. In: Bolognia JL, Jorizzo JL, Rapini RP, eds. *Dermatology*. Edinburgh: Mosby; 2003:714.)

46. What type of melanoma do Black patients tend to develop? How do you recognize it?

Although uncommon in Blacks, melanoma tends to be of the acrolentiginous type. Look for black dots on the palms or soles or under the fingernail (Fig. 6.22) that start to change in appearance or cause symptoms.

47. Describe Paget disease of the breast. What is its significance?

Paget disease of the breast presents as a unilateral, red, oozing or crusting nipple in an adult woman that fails to respond to typical dermatology treatments (Fig. 6.23). Though rare (roughly 1%–2% of breast cancers), it signifies an underlying breast cancer (usually invasive ductal carcinoma or ductal carcinoma in situ) with extension to the skin.

48. Define stomatitis. What does it suggest?

Stomatitis is an inflammation of the mucous membranes of the mouth. The classic finding is fissuring of the corners of the mouth (angular stomatitis). Watch for deficiencies of B-complex vitamins (riboflavin, niacin, pyridoxine) or vitamin C. Additional causes include drugs such as methotrexate and sulfasalazine.

49. What should you think of in an elderly patient with painful and pruritic lesions that resolve only to recur at another site a month later?

Necrolytic migratory erythema is a feature of glucagonoma along with weight loss, hyperglycemia, and diarrhea. Diagnosis is made with measurement of glucagon level (>500 pg/mL confirms the diagnosis) and abdominal imaging (magnetic resonance imaging [MRI] or computed tomography [CT] scan).

50. What dermatologic findings are classically associated with nutritional deficiencies?

Koilonychia and diffuse hair loss: iron deficiency
Alopecia and pustular rash of extremities and perioral region: zinc deficiency
Brittle hair and skin depigmentation: copper deficiency
Bitot spots: vitamin A deficiency