

## Original Studies

### Common Pediatric and Adolescent Skin Conditions

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**Abstract.** Skin lesions are encountered in all areas of medicine, and it is therefore important for physicians to understand the fundamentals of explaining and diagnosing common skin conditions. This article begins with a discussion of description and documentation of skin lesions based on color, size, morphology, and distribution. Pigmentation disorders such as vitiligo are depicted.

Cutaneous growths that are found in the pediatric and adolescent population include acrochordons, dermatofibromas, keloids, milia, neurofibromas, and pyogenic granulomas. Treatment of these growths usually involves observation or curettage with electrodesiccation.

Psoriasis, atopic dermatitis, poison ivy, and eczema are comprised of scaling patches and plaques; poison ivy and atopic dermatitis may also present with bullous and vesicular changes. Therapy typically consists of topical emollients and corticosteroids; phototherapy is reserved for refractory cases. Acne vulgaris is the most common skin disease of the pediatric and adolescent population. This condition can be psychologically debilitating and, therefore, proper treatment is of paramount importance. Therapeutic options include topical as well as oral antibiotics and retinoids. Extreme caution must be used when prescribing retinoids to post-pubescent females, as these agents are teratogenic.

Vascular anomalies are most commonly exemplified as port wine stains and hemangiomas. Port wine stains may be treated with pulsed dye laser or may be observed if they are not of concern to the patient or physician. Hemangiomas typically spontaneously regress by age ten; however, there has been recent concern that certain cases may need to be treated.

Dermal rashes may be localized or generalized. Treatment of generalized drug eruptions involves elimination of the

inciting agent, topical antipruritics, and systemic corticosteroids for severe reactions.

Infectious etiologic agents of skin disease include bacteria, fungi, and viruses. Many sexually transmitted diseases are bacterial or viral in origin and present as a rash or ulcer. Impetigo is a bacterial infection which may present as a bullous eruption or as an erosion with a honey colored crust. Other bacterial infections include erythema chronicum migrans, folliculitis, and cellulitis. Fungal infections include the various forms of tinea and are usually treated with topical antifungals; if the infection is located in a hair-bearing area, systemic antifungals are necessary. Viral infections include warts, varicella, molluscum contagiosum, and herpes. Treatment varies from observation or antivirals for varicella to cryosurgery and topical imiquimod for warts. Finally, scabies and lice are infectious agents that can be treated with permethrin and pyrethrin solutions.

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**Key Words.** Dermatitis—Dermatology—Eczema—Wart—Nevus—Acne—Tinea—Impetigo—Candida—Pediatric—Adolescent

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#### Introduction

Skin disease within the pediatric and adolescent population is seen in a wide variety of clinical settings. The diagnosis of dermatologic diseases involves history, physical examination, and laboratory testing. Historical information includes the onset of the rash or lesion, the duration, any changes in the presentation, associated signs or symptoms, and the effectiveness of any treatments that have been used.

Physical examination involves both inspection and palpation of the skin. Additionally, hair, nails, and mucous membranes must also be examined and any abnormalities noted. The physical findings must be carefully documented so that changes that occur can be noted upon subsequent examinations.

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**Synopsis:** This article discusses diagnosis, documentation, and treatment of the more common pediatric and adolescent skin diseases and conditions including eczema, psoriasis, acne, atopic dermatitis, and nevi among many others. Cutaneous infectious agents including fungi, bacteria, and viruses are also reviewed.

**Table 1.** Standard Vocabulary for Dermatologic Findings

Primary Lesions	
Macule	Flat circumscribed change in skin color less than 1 cm in diameter
Patch	Flat circumscribed change in skin color greater than 1 cm in diameter
Papule	Solid, elevated, superficial lesion less than 5 mm in diameter
Plaque	Solid, elevated superficial lesion with flat surface greater than 5 mm in diameter (width greater than height)
Nodule	Solid elevated lesion with depth into the underlying tissue, diameter between 5 mm and 2 cm
Tumor	Solid elevated lesion with depth into the underlying tissue, diameter greater than 2 cm
Pustule	Papule or vesicle with free pus
Vesicle	Elevated, superficial, fluid-filled lesion less than 5 mm in diameter
Bulla	Elevated, superficial, fluid-filled lesion greater than 5 mm in diameter
Wheal	Transient flat topped cutaneous papule or plaque due to underlying edema
Abscess	Nodule or tumor with free pus
Secondary Lesions	
Scale	Accumulation of loose superficial fragments of stratum corneum
Lichenification	Thickened skin with accentuated surface markings
Crust	Scab with dried exudates of serum and pus
Erosion	Superficial loss of epidermis
Ulcer	Loss of epidermis and dermis
Sinus	Tunnel within the dermis with opening on the skin surface
Atrophy	Thinning of skin with loss of hair and sweat glands
Excoriation	Linear scratch in the skin surface involving epidermis only
Fissure	Linear crack which involves the dermis
Color	
Erythema	Blanchable redness
Purpura	Non-blanchable deep red to purple color
Hyperpigmentation	Darkening of skin color secondary to excess pigment production
Hypopigmentation	Lightening of skin color secondary to decreased pigment production
Configuration	
Linear	In a line
Herpetiform	Clustered or grouped
Annular	Active border
Nummular	Coin-like
Dermatomal	Following a skin dermatome
Photodistributed	Specific to sun exposed surfaces

When documenting the physical examination, it is essential to have a standardized vocabulary for describing dermatologic findings (Table 1). **Primary** lesions refer to those that are caused by the disease process itself. A **macule** is a flat, circumscribed change in skin color less than 1 cm while a **patch** is a macule that is larger than 1 cm. A **papule** is a solid, elevated, superficial lesion smaller than 5 mm, and a **plaque** describes a solid elevated lesion (width greater than height) larger than 5 mm in diameter. The term **nodule** is used to define a solid elevated lesion with depth into the underlying tissue and a diameter between 5 mm and 2 cm. **Tumor** refers to a nodule with a diameter greater than 2 cm. **Pustule** is used to describe a papule or vesicle containing free pus. A **vesicle** is an elevated, superficial, fluid-filled blister smaller than 5 mm while a **bulla** is an elevated, superficial, fluid-filled lesion larger than 5 mm. **Wheal** refers to a rapidly evolving flat-topped papule or plaque due to transient cutaneous edema, such as the reaction that is seen upon contact with an allergen. An **abscess** is a nodule or tumor containing free pus.

**Secondary** lesions are those that are resultant to factors such as scratching, infection, or rubbing. A

**scale** is an accumulation of loose superficial fragments of stratum corneum. **Lichenification** is thickened skin with accentuated surface markings. **Crust** refers to a scab containing dried exudates of serum and pus. **Erosion** is a superficial loss of the epidermis, while an **ulcer** is denudation extending into the dermis caused by destruction of superficial tissue. A **sinus** is a tunnel within the dermis culminating in an opening on the skin surface. **Atrophy** refers to the thinning of skin along with a loss of hair and sweat glands secondary to wasting of the dermis. An **excoriation** is a linear scratch in the skin surface, while a **fissure** is a linear crack that extends into the dermis.

Descriptors of color in skin diseases include **erythema** (blanchable redness secondary to dilated blood vessels) and **purpura** and **petechiae** (non-blanchable deep red or purple color due to extravasated blood). **Hyperpigmentation** is a darkening of skin color due to excessive pigment deposition as opposed to **hypopigmentation**, the lightening of skin color due to loss of pigment.

Configurations include **linear** (in a line), **herpetiform** (clustered or grouped lesions), **annular** (active

border), **nummular** (coin-like), **dermatomal** (following skin dermatome), and **photodistributed** (limited to sun-exposed surfaces.)

The laboratory diagnosis of skin disease involves, most frequently, microscopic analysis of skin scrapings and skin biopsy. Additionally, laboratory information that may be useful in diagnosis includes auto-antibody titers, aldolase, cultures, creatine kinase, and serology, including complete blood counts and metabolic panels.

### Disorders of Pigmentation

Normal skin pigmentation is comprised of oxygenated hemoglobin in the arterioles, deoxygenated hemoglobin in the venules, epidermal melanin, and sometimes carotenoids or incompletely metabolized bile. Melanocytic pigmented lesions must be carefully followed over time and any changes or abnormalities noted as these areas may develop into melanomas. Patients should be educated in the ABCD of moles and melanoma: Asymmetry, irregular Border, variation in Color, and Diameter greater than 6 mm. It is important to note, however, that not all melanomas meet these criteria.

A solar **lentigo**, more commonly referred to as a freckle, results from chronic sun exposure over time. This is an area of reticulated pigmentation that appears darker than the surrounding area; it may vary in size from 1 mm to greater than 1 cm. **Café au lait macules** are congenital hyperpigmented patches and can be associated with neurofibromatosis when they number greater than five. To be considered in the diagnosis of neurofibromatosis, café au lait lesions must be at least 1.5 cm in diameter in patients older than 5 years and over 0.5 cm in children younger than 5 years. **Congenital melanocytic nevi** are caused by errors in development of pigment cells and may vary in size from less than 2 mm to greater than 20 cm. Giant and possibly intermediately sized nevi are associated with an increased risk of melanoma. A **blue nevus** is caused by the deep dermal location of melanocytic cells producing a bluish color of the nevus (Tindel effect).

Disorders of hypopigmentation occur with some frequency in the pediatric population. **Vitiligo** (Fig. 1) is a disorder characterized by a complete lack of pigmentation due to loss of epidermal melanocytes, and 50% of patients with vitiligo present before age 20. The affected area is stark white with a well-demarcated border. Vitiligo is associated with immune disorders; therefore a workup including testing for thyroid disease, diabetes, and other autoimmune diseases may be indicated. **Tuberous sclerosis** may present with hypopigmented macules (Ash leaf macules or confetti spots) or a characteristic shagreen patch, a connective tissue nevus that is rough and typically has a peau

d'orange appearance. Other associated findings include facial angiofibromas (adenoma sebaceum) and periungual fibromas (Koenen tumors.)

### Cutaneous Papules and Tumors<sup>1</sup>

#### Achrochordon

**Acrochordons**, also known as skin tags, are soft, pedunculated, flesh-colored to tan papules, usually ranging from 1 to 5 mm in diameter and commonly occurring in areas that are exposed to a high degree of friction. These lesions follow a benign course, although the patient may experience irritation of the affected areas. Treatment includes removal with Gradle scissors, cryosurgery, or electrodesiccation.

#### Dermatofibroma

**Dermatofibromas** (Fig. 2) are fixed, pea-sized dermal papules or nodules with hyperpigmentation usually found on the legs and forearms. They are characterized by the "dimple" sign in which the dermatofibroma dimples into the surrounding skin upon pinching. Treatment is usually unnecessary, although these may be removed for cosmetic reasons. The patient should be warned, however, that the resulting scar may be as cosmetically displeasing as the original lesion. Dermatofibromas should be removed if they are rapidly growing; in these cases, Moh's surgery (microscopic controlled excision) is the treatment of choice.

#### Hypertrophic Scar and Keloid

A **keloid** (Fig. 3) is a fibrous, thickened, reddish plaque that overgrows the boundaries of the original injury. It is often accompanied by symptoms of itching or pain. Keloids are most commonly found on the neck, shoulders, deltoid region, and sternal region. The raised scar is called **hypertrophic** if it remains confined to the site of injury and flattens in time, as opposed to keloid, which does not involute but instead invades the surrounding uninjured skin. Treatment includes intralesional steroid injections such as Kenalog 10 to 40 mg/mL every 4 to 6 weeks, cryosurgery, laser therapy, excision with topical imiquimod status post excision, or topical retinoic acids.

#### Milia

**Milia** are firm white papules 1 to 2 mm in diameter; they are often likened to a grain of sand immediately beneath the epidermis. The lesions can arise spontaneously or in association with diseases such as bullous pemphigoid or porphyria cutanea tarda. They are very common in the newborns and are often of no consequence in this population. Histologic examination reveals miniature epidermal inclusion cysts. Treatment consists of incision with a no. 11 scalpel blade and



**Fig. 1. Vitiligo:** There is a complete lack of pigmentation due to loss of epidermal melanocytes.

expression of the keratin contents of the cyst; topical retinoids may also be used.

### Neurofibroma

**Neurofibromas** are soft, fleshy, pedunculated polyps that are often confused with acrochordons. Histologically, these are comprised of wavy collagen fibers. The presence of a neurofibroma may be an isolated finding or a marker of neurofibromatosis. Treatment consists of tangential surgical excision.

### Pyogenic Granuloma

**Pyogenic granulomas** are friable vascular growths that may be sessile or pedunculated. Often found at sites of injury, and sometimes referred to as “proud flesh tumors,” their typical course begins with a small erythematous papule that rapidly enlarges and becomes pedunculated. Treatment consists of curettage followed by electrodesiccation of the base.



**Fig. 2. Dermatofibroma:** A fixed dermal papule with hyperpigmentation is characterized by the “dimple sign” in which the dermatofibroma “dimples” into the surrounding skin upon pinching.



**Fig. 3. Keloid:** This fibrous and thickened scar has overgrown the boundaries of the original injury.

### Scaling Patches and Plaques/Papulosquamous Disorders

#### Psoriasis

**Psoriasis** (Fig. 4) is a disease that may manifest itself in a variety of different manners. The presentation depends on several factors including genetics, environment, associated disease, medications, and immunologic status.<sup>2</sup> Although the mean age of onset of this disease is within the third decade, it affects many children and adolescents.

The most classic presentation of psoriasis is a symmetric, inflammatory distribution of thick, erythematous patches with silvery scales with a predilection for the scalp and extensor extremities. The patient often complains of pruritus. Several variants of classic psoriasis have been described, including **guttate psoriasis** (droplet-sized, diffuse, erythematous lesions which predominate on the trunk and proximal extremities), **pustular psoriasis** (yellowish pustules on a background of redness and swelling), and **erythrodermic**



**Fig. 4. Psoriasis:** The thickened erythematous plaque typically presents with silvery scales.

**psoriasis** (generalized scaling, pruritic inflammatory plaques). Additionally, up to 50% of patients will have involvement of the nail with pitting of the nail plate, onycholysis (lifting of the distal nail plate from the underlying nail bed), and/or yellow-brown spots (oil spots) in the nail bed. The diagnosis of psoriasis is usually based on the history and physical exam; however, microscopic analysis to rule out fungal infection, serologic testing to rule out syphilis, and skin biopsy may be helpful in confirming the diagnosis.

The cause of psoriasis remains unknown, but several theories have been proposed. There may be a genetic component, primarily involving the genes that control the immune system; the HLA-C Cw6 variant and the HLA-A A13 variant (both within the HLA region of chromosome 6) have both been linked with an increased prevalence of the disease. Other genes within or near the HLA region such as *psors1* and *cdsn* have also been suggested to play a role in development of the disease. There is still a great deal of research in progress regarding the role of genetics in psoriasis. The pathophysiology may involve an aberration of interleukin-2 regulation. Psoriasis may also develop in an area of previous trauma, a process known as the Koebner phenomenon. Certain drugs such as lithium, beta-blockers, NSAIDs (non-steroidal anti-inflammatory drugs), and anti-malarials may also induce psoriasis or aggravate pre-existing disease.<sup>2</sup>

Treatments can be divided into topical, photo-, systemic, and biologic therapies. Topical therapies include corticosteroids in a cream, gel, solution, foam, or ointment base applied directly to the plaques. Caution must be used when treating with high potency corticosteroids, which can cause systemic side effects, such as hypertension and hyperglycemia, as well as topical complications, including skin atrophy and telangiectasias. Other topical treatments include calcipotriene (vitamin D3 analog), anthralin, tazarotene (retinoid), coal tar preparations, and keratolytics, all of which thin the hypertrophied scaling plaques of psoriasis. Natural sunlight and phototherapy have also been demonstrated to improve the clinical appearance of psoriasis; however, these treatments do increase the patient's risk of skin cancer and photoaging. Systemic treatments include sulfasalazine, methotrexate, cyclosporin, and oral retinoids.<sup>3</sup> Recently, biologic therapies have been used for cases resistant to the classic treatments. These work by interfering with the disease at the level of the immune system. Options include Amevive (administered intravenously or intramuscularly), Enbrel (currently approved for rheumatoid arthritis), and Remicade (currently approved for rheumatoid arthritis and Crohn's disease.) Each treatment has its own risks and requires familiarity with these possibilities so that proper monitoring may take place.

## Eczema

Eczema (Fig. 5) is not a disease itself but rather a variety of diseases, including the different subclasses of dermatitis (atopic, contact, seborrheic, stasis, and photocontact), lichen simplex chronicus, and niacin or riboflavin deficiencies. Eczema typically presents as erythematous papules and plaques with microvesicles and macrovesicles; scaling is sometimes present. The patient typically complains of intense pruritus. The diagnosis can be supported with biopsy that reveals spongiosis due to epidermal intracellular edema and vesiculation. There is a perivascular T-cell infiltrate within the dermis, illustrating the role that T-cells have in creating eczematous eruptions. The more common specific subclasses of eczematous dermatitis as well as the treatments will be discussed in the following sections.

## Contact Dermatitis

Contact dermatitis can be subdivided into two primary types: allergic and irritant. Allergic contact dermatitis



**Fig. 5. Eczema (atopic dermatitis):** Erythematous papules and plaques may present with scaling and are typically intensely pruritic.

(ACD) results from direct skin contact with a substance that the body recognizes as foreign. The resulting skin inflammation is a delayed-hypersensitivity immunologic reaction. This is contrasted with irritant contact dermatitis, which is non-immunologic and is caused by a direct insult to the tissues.

Allergic contact dermatitis occurs more commonly on thinner skin surfaces such as eyelids and genital skin, and there is often a period of latency before the dermatitis presents clinically. The manifestation of **poison ivy allergic contact dermatitis**, the most commonly encountered ACD (Fig. 6), serves as a good example of the typical clinical course.

The rhus oleoresin from the poison ivy leaves acts as an antigen to produce the associated dermatitis that is characterized by papulovesicular linear streaks and plaques. A common misconception is that the rash spreads by contact with infected skin; however, the rash is not contagious. Rather, the rash erupts in various locations at different time intervals, sometimes giving the appearance of spread from one part of the body to another.

Prevention involves avoidance of the poison ivy plant. The skin and any clothes that may have come in contact with the rhus oleoresin must be washed, as remaining resin will cause further eruption upon contact with the skin.

Treatment for poison ivy dermatitis, as with most types of allergic contact dermatitis, is primarily symptomatic. Patients may experience relief with the use of topical corticosteroids, cold compresses, and calamine lotion. For severe cases, a tapering dose of oral corticosteroids, such as prednisone, may be used; however, available “dosepaks” of corticosteroids are of little use as they deliver too little corticosteroid for too short a period of time and often result in a rebound reaction. Rather, prednisone, 1–2 mg/kg, may be used with a taper upon response; a 2-week taper is fairly standard.



**Fig. 6. Poison ivy:** Erythematous blisters with pruritus appear where the rhus oleoresin has come in contact with the skin.

Other frequent causes of allergic contact dermatitis include nickel and rubber. Prevention includes avoidance of these substances in allergic individuals and treatment consists of corticosteroids.

Irritant contact dermatitis (ICD) is the second subclass of contact dermatitis. The clinical manifestation of irritant contact dermatitis depends on the type of irritant, the duration of exposure, and the atopic predisposition of the individual. Irritant contact dermatitis usually presents as pruritus, pain, and erythematous papules and vesicles in the affected area. Many patients with chronic ICD also experience cracking and fissuring of the affected skin surface. ICD is commonly seen in individuals exposed to wet work such as dishwashers, hairdressers, and bartenders.<sup>4</sup>

Prevention involves avoidance of the inciting agent; barrier creams have inconsistent effectiveness.<sup>4</sup> Topical corticosteroid creams, lotions, and ointments are useful for chronic lesions, and the choice of preparation is dependent on the severity of the reaction and the site on the body.

### Atopic Dermatitis

**Atopic dermatitis (AD)** is a chronic inflammatory dermatosis that affects 10–20% of children, most commonly infants and young children. Studies have demonstrated that AD significantly impacts quality of life and, therefore, new treatments should offer increased efficacy, less frequent application, and fewer side effects.<sup>5</sup> The incidence of AD is rapidly increasing, probably secondary to the interaction of pre-existing genetic factors with the dynamic environment.<sup>6</sup> The pathogenesis involves cutaneous immune dysfunction, which has a genetic component.<sup>7</sup> Patients generally exhibit increased levels of IgE as well as proliferation of both Th1 and Th2 cells causing an overproduction of cytokines.<sup>8</sup>

Diagnosis is based on a personal and family history of atopy, pruritus, and physical eczema. Patients commonly exhibit additional symptoms of allergic rhinitis or asthma. Erythematous, excoriated, scaling papules and plaques with intense pruritus characterize AD. Repeated excoriations and trauma lead to chronic lesions, which are exemplified by thick plaques with lichenification and fibrotic papules. In patients aged 2 and older, common locations of lesions include the antecubital and popliteal flexural areas, while infants may display lesions on the scalp, face, cheeks, and extensor surfaces of the extremities.

The conventional approach to treatment of AD has included skin hydration, topical corticosteroids, and elimination of precipitating irritants. Although intermittent topical corticosteroid use is a common therapy, maintenance topical corticosteroid use is avoided due

to concerns about potential side effects such as skin atrophy and immunosuppression. Short-term (2-week) courses of systemic corticosteroids may be used for acute crises but should be avoided on a chronic basis. Recent evidence suggests that pimecrolimus 1% cream or tacrolimus 0.03% or 0.1% ointment twice daily, immunomodulators that block T-cell activation, may be advantageous compared to corticosteroids in that they do not appear to induce systemic immunosuppression<sup>8</sup> or cutaneous atrophy. In patients refractory to the above-mentioned treatments, phototherapy, gamma interferon, or intravenous immunoglobulin may be used.<sup>9</sup> Oral antihistamines may offer symptomatic relief of the associated pruritus, and the related asthma may be controlled with inhaled corticosteroids and  $\beta$ 2 agonists as needed.

## Acne

**Acne vulgaris** (Fig. 7) is the most prevalent skin disease in the pediatric population and is nearly universal in that most individuals will be affected by it at some point in their lives. Acne may become clinically evident as early as 8 or 9 years of age and usually resolves by the early twenties; however, it may persist until the fifth decade.<sup>10</sup> The clinical manifestation of the disease is a combination of intrinsic and extrinsic factors.<sup>11</sup> Intrinsic factors are related to lipase, protease, and chemotactic factor production by *Propionibacterium acnes*, all of which contribute to the immunologic response mounted by the host, which involves the humoral, cell-mediated, and complement pathways. The microenvironment, such as pH and oxygen tension, within the pilosebaceous unit likely plays a role in the amount of exoenzymes produced by *P. acnes*, as does the sebum production and amount of hyperkeratinization.<sup>12</sup> Extrinsic factors that have been implicated in the aggravation of acne potential include friction, sunlight, chemicals, and certain cosmetics.<sup>11</sup>



**Fig. 7. Acne:** Erythematous pustules and comedones are present in this patient.

The lesions of acne vulgaris can be classified into inflammatory (papules, pustules, nodules, and cysts) or non-inflammatory (open and closed comedones), and these two classifications should be approached separately. Closed comedones, commonly called whiteheads, are caused by the filling of sebaceous follicles with keratin, sebum, and bacteria. The appearance of an open comedone, or blackhead, is due to accumulation of oxidized lipids and pigment. Inflammatory involvement of the dermis can cause scarring which may appear as post-inflammatory hyperpigmentation, depressions in the skin, or keloid formation.

Topical therapy for non-inflammatory acne (comedones) typically involves retinoids in a cream, gel, or solution form. This therapy works by keratinolysis as well as by reducing sebum production thereby reducing skin surface lipids.<sup>13</sup> The patient must be cautioned about increased sensitivity to ultraviolet light and sunburn when using these treatments. Additionally, there is often an initial worsening of a papular flare, and the effect of comedone reduction may take up to 3 months to become evident. Inflammatory acne may be treated with topical antibiotics such as benzoyl peroxide, clindamycin, and erythromycin. If pustular acne is refractory to topical antibiotics, oral antibiotics (tetracycline, minocycline, doxycycline, and erythromycin), or oral isotretinoin may be used. The patient must be warned about the possible teratogenic side effects of these oral medications, and birth control is essential in females using these treatments. Oral isotretinoin carries additional possible side effects of hepatotoxicity, elevation of triglyceride and cholesterol levels, pseudotumor cerebri, bone pain, hair loss, and depression. Routine laboratory testing of liver function tests, fasting lipids, and beta-hCG is necessary in patients receiving oral isotretinoin.

Hormonal treatment is often effective in females, due to an underlying hormonal imbalance resulting in hyperandrogenemia, and includes oral contraceptives such as those containing norgestimate (Ortho TriCyclen<sup>®</sup>) or drospirenone (Yasmin<sup>®</sup>) and ethinyl estradiol. The combination of norgestimate and ethinyl estradiol (Ortho TriCyclen<sup>®</sup>) was the first oral contraceptive with a specific indication for treating acne vulgaris.<sup>14</sup> Ortho TriCyclen<sup>®</sup> offers the additional benefit of increasing high-density lipoprotein cholesterol (HDL-C) significantly while lowering the ratio of low-density lipoprotein cholesterol to HDL-C. These results are consistent with those of previous studies and indicate that the triphasic preparation of norgestimate and ethinyl estradiol is a selective and minimally androgenic oral contraceptive agent. Long-term therapeutic benefit may accrue from the favorable influences on the lipid profile.<sup>15</sup> Yasmin has a similarly favorable side effect profile and causes minimal weight gain and possibly weight loss due to decreased water retention.<sup>16</sup>

Low dose corticosteroids (prednisone 2.5 to 5 mg at bedtime) and androgen inhibitors (spironolactone 50 to 200 mg daily)<sup>10</sup> may also be used.

## Vascular Malformations and Tumors

### Port-wine Stain

A **port-wine stain** (Fig. 8) is good example of a localized vascular tumor. These are congenital vascular malformations, affecting approximately 1% of the population, with the majority occurring on the face. They typically present as purpuric plaques involving the ophthalmic and maxillary divisions of the trigeminal nerve, but they may appear on the extremities or trunk in addition to the face in 40% of cases. Sturge-Weber Syndrome is associated with facial port-wine stains (nevus flammeus), usually within the V1 division of the trigeminal nerve (V2 and V3 may also be involved); excessive blood vessel growths (leptomeningeal angiomas), accumulations of calcium inside the brain, congenital glaucoma, and seizures are sometimes associated with this syndrome. Port-wine stains are preferentially treated with laser therapy using the pulsed-dye laser. The discomfort of the procedure can be alleviated with a local anesthetic cream applied prior to the treatment with the laser.<sup>17</sup> Another option is the use of cryogen spray cooling or cold-air cooling during the laser therapy to help ameliorate the associated pain. These methods result in similar clearance rate as traditional laser therapy.<sup>18</sup> The use of cryogen spray cooling may also allow use of the pulsed dye laser at high fluences, although this has not been shown



**Fig. 8. Port wine stain (nevus flammeus):** The purpuric plaque involves the distribution of the trigeminal nerve.

to be advantageous in treatment.<sup>19</sup> Approximately 40% of patients with port-wine stains will achieve 75% lightening after laser treatment, and more than 80% will lighten by at least 50%.<sup>20</sup>

### Hemangioma

Capillary **hemangiomas** (Fig. 9) are benign tumors of infancy. They typically appear between the first and fourth week of life, proliferate for 6–9 months, then undergo an involution that is usually complete by age 10. Most hemangiomas are of cosmetic concern only, but occasionally they may affect other systems; for example, periocular hemangiomas can interfere with vision and subglottic hemangiomas may affect swallowing and respiration. When considering treatment for these lesions, it is important to consider the natural history of slow spontaneous regression. These vascular malformations may be treated with laser therapy, systemic corticosteroids, and interferon alpha. Pulsed-dye laser treatment may slow or arrest proliferation of early hemangiomas; however, this treatment may cause scarring and can penetrate a depth of only 1 mm. Treatment with interferon alpha may cause malaise, fatigue, and fever, as well as elevation of liver enzymes; additionally, severe neurologic sequelae may result. In conclusion, the risks and benefits of treatments must be carefully weighed before embarking on treatment of the capillary hemangioma as many will spontaneously regress by age 10.<sup>21</sup>

## Generalized Dermal Rashes

### Drug Eruption

Generalized dermal rashes are usually attributed to viral exanthems, which will be discussed in the following section, and drug eruptions. Exanthematous **drug eruptions** are the most common rashes seen by physicians. Simple drug eruptions usually begin within 1



**Fig. 9. Hemangioma:** This vascular malformation involves the upper arm and will likely spontaneously regress.

to 2 weeks of beginning a medication, are not associated with fever, and resolve within 1 to 2 weeks of discontinuing the offending agent. Urticarial eruptions are characterized by pruritic erythematous wheals; when involvement of the deep dermal and subcutaneous tissues is also present, the condition is known as angioedema. IgE dependent urticaria occurs within hours of drug exposure in the immunized host and is related to mast cell activation and resulting vasodilation. The most serious drug eruptions may present as blisters or bullae as seen in erythema multiforme, Stevens-Johnson syndrome (SJS), and toxic epidermal necrolysis (TEN). The distribution of a rash can give a clue to the diagnosis, and it is therefore very important to note and document the distribution and pattern. Photosensitive eruptions are the result of an interaction of the drug or its metabolites with light energy.

The treatment of drug eruptions involves identification and removal of the inciting agent, antihistamines, and topical antipruritics. In the more severe hypersensitivity reactions such as SJS and TEN, systemic corticosteroids or intravenous immunoglobulin (IVIG) therapy might be required,<sup>22</sup> although the use of systemic corticosteroids remains controversial.

## Infectious Rashes

### Bacterial Infections

**Impetigo. Nonbullous impetigo** (Fig. 10) may be caused by group A beta-hemolytic streptococci or by *Staphylococcus aureus*. Recent evidence suggests typical initial infection with group A strep followed by rapid colonization with *S. aureus*.<sup>23</sup> The infection is classically preceded by minor skin trauma such as insect bites and usually presents as small vesicles or pustules that soon rupture causing a characteristic honey-colored crust. New lesions typically appear in rapid succession, and particular attention should be paid to skin disease in the diaper area that can be complicated with fungal infections or gram-negative bacteria.

**Bullous impetigo**, usually caused by *S. aureus*, affects children primarily between ages 2 and 5 years. The disease typically presents as flaccid bullae and blisters less than 3 cm in diameter that result from the epidermolytic toxin produced by *S. aureus*. These bullae and blisters may rupture leaving a crust on the surface of the lesion.<sup>24</sup>

Complications of impetigo are uncommon but may include cellulitis, poststreptococcal acute glomerulonephritis, and superinfection of the initial lesion.

Treatment includes topical mupirocin, three times daily for 7 days. This treatment should not be used in patients with extensive fever or lymphadenopathy or



**Fig. 10. Bullous impetigo:** Note the flaccid bullae and blisters as well as the characteristic honey colored crust on the ruptured lesions.

patients in whom compliance may be a problem. Systemic treatments include penicillinase-resistant penicillins (dicloxacillin), erythromycin, cephalosporin, or clindamycin.<sup>25</sup>

**Cellulitis and Erysipelas.** Cellulitis refers to infection of the dermis and subcutaneous fat and typically presents as a warm, tender, erythematous poorly defined patch or plaque. There is often an associated portal of entry such as a wound or macerated area. It should be differentiated from erysipelas, which presents as an indurated, elevated, erythematous plaque with well defined borders usually found on the face or lower extremities; the site of infection in erysipelas is the more superficial dermis. Both conditions may be accompanied by findings of fever, leukocytosis, lymphangitis, and lymphadenopathy. Group A beta-hemolytic strep (GABS) is the predominant cause of both conditions.

Oral penicillin, or erythromycin in penicillin-allergic patients, for 10 days is usually sufficient treatment. Oral dicloxacillin is often the best choice since it provides better staphylococcal coverage for the rare causes that may involve *S. aureus* infection. More

severe infections may require IV antibiotics. A rare but very serious complication of cellulitis is necrotizing fasciitis, a rapidly progressive infection of the fascia and superficial fat. Management involves aggressive surgical debridement as well as intravenous antibiotics effective against GABS.<sup>25</sup>

**Erythema chronicum migrans.** **Erythema chronicum migrans** is the skin manifestation of Lyme disease that results from infection with *Borrelia burgdorferi*. This rash appears a few days to a few weeks after the tick bite and begins as an erythematous papule at the site of the bite and progresses to an annular erythematous rash that may reach up to 20 cm in size and typically has an area of central clearing. Other symptoms of Lyme disease include fever, malaise, headache, sore throat, stiff neck, muscle aches, and fatigue. Some people develop the flu-like illness without developing a rash. Treatment for Lyme disease includes antibiotics such as amoxicillin and doxycycline.

**Folliculitis.** *S. aureus* is a common cause of folliculitis and presents as an eruption of 1–5 mm pustules with a halo of erythema; these pustules are follicular in origin and often a hair can be seen emerging through the center of the pustule. The distribution may be focal or diffuse. Predisposing factors include obesity, friction, diabetes, and hyperhidrosis. Treatment options include topical antibiotics; if this fails, systemic antibiotics may be used. *Pseudomonas* folliculitis is usually associated with hot tub use and typically presents as a diffuse eruption on the trunk. The rash consists of tender follicular-based erythematous papules and nodules with follicular pustules often confined to the area covered by a swimsuit.

Most cases do not require treatment as they are self-limited, but severe or recurrent cases may be treated with oral ciprofloxacin. Prevention involves regular drainage of hot tubs to remove the buildup of skin cells, which serve as the prime nutrient source for the *Pseudomonas*.<sup>25</sup>

## Fungal Infection

Fungi have the ability to colonize almost any environment, and although fungal infections of the feet, nails, and groin are uncommon in the pediatric age group, fungal infections of the scalp are very common and must be diagnosed early or they can lead to permanent hair loss.<sup>26,27</sup> Some of the more common types of fungal infections, such as yeasts and dermatophytes, will be discussed in this section. Yeast infections include *Candida* and *Malassezia*. Dermatophyte infections are also referred to as tinea.

**Tinea corporis.** (Fig. 11) may manifest in several different manners, the most common of which is a classic ringworm appearance, which presents as a flat scaly area with a raised and sometimes erythematous border that advances circumferentially. Pruritus is a commonly associated symptom and vesicles or pustules may form as the lesion becomes inflamed. Tinea capitis can vary in presentation from a few pustules to kerions which are boggy, pustular, inflammatory reactions to the fungus.<sup>28</sup> Tinea pedis is a fungal infection of the feet that usually presents with peeling, fissures, pruritus, and maceration. The affected area may be pink and covered with silvery white scales.

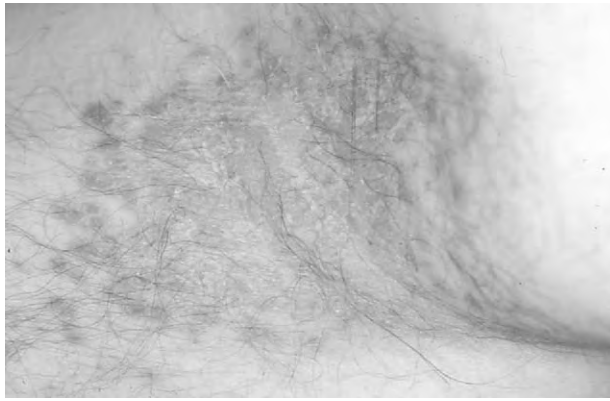
*Candida* species may infect the skin or mucous membranes and typically produce an erythematous, moist, desquamating rash. The diagnosis of *Candida* infection (Fig. 12 and 13) should be considered in presentations of diaper dermatitis in infants, erythematous rashes in the oral commissures, erythematous rashes of the genital region, and cottage cheese-like discharge from the vagina in females. *Malassezia furfur* infection, known as **tinea versicolor**, presents as slightly scaly hypopigmented or hyperpigmented macules and patches usually involving the trunk and commonly occurring during the summer months.

Rapid and sensitive diagnosis involves potassium hydroxide (KOH) preparation, in which a sample of scale, nail, or hair is obtained with a scalpel, placed on a glass slide, and 10% to 20% KOH is added. The slide is then gently heated and then examined under a microscope for fungal hyphae. In general, samples are best obtained from the active border or edge of a suspicious lesion. When a diagnosis is still in question, a fungal culture may be used for definitive diagnosis.

Topical treatments for tinea corporis include miconazole 2% cream, clotrimazole 1% cream, imidazole, ciclopiroxamine, or benzylamine. Application is



**Fig. 11. Tinea:** This patient shows annular lesions with the characteristic erythematous circumferentially advancing border. Pruritus is often associated with these infections.



**Fig. 12. Candidiasis (axillary):** An erythematous, moist, desquamating rash is present in the axilla of this patient.

twice daily for 3 to 4 weeks. Oral therapy such as ketoconazole, itraconazole, terbinafine, or griseofulvin should be used for widespread tinea infections, those involving the scalp, or those that do not respond to topical drugs. Tinea pedis may be treated with terbinafine hydrochloride 1% cream, which has been tolerated very well in the pediatric population,<sup>29</sup> or econazole 1% cream once daily; resistant cases may be treated with oral griseofulvin for 6 to 8 weeks. The infection may be prevented by wearing open shoes, avoiding communal showers, and keeping the feet dry. Candidiasis is treated with topical ketoconazole or econazole twice daily for 2 to 4 weeks; other options include oral fluconazole 150 mg for one dose<sup>30</sup> or oral ketoconazole 200 to 400 mg every day for 7 days. These same therapies may be used for the treatment of tinea versicolor.

### Viral Infection

This section will discuss some of the more frequently encountered viral exanthems as well as viral eruptions.



**Fig. 13. Tinea cruris:** An erythematous rash of the genital region.

Viral infections vary greatly in their presentation from focal infections with papilloma viruses, which cause localized warts, to the generalized skin eruption of chicken pox caused by the varicella virus. The common diagnostic finding will be discussed as well as treatment modalities.

### Warts

Warts are common and usually benign. They can be classified into four characteristic clinical types: common wart (*verruca vulgaris*; Fig. 14), plantar wart (*verruca plantaris*), flat wart (*verruca plana*), and genital wart (*condyloma accuminata*). One half of all common warts in children will spontaneously regress without therapy.<sup>31</sup>

Diagnosis is usually made by history and recognition of these familiar growths. Common warts usually present as one or more verrucous papules on the hands or extremities, while plantar warts occur on the soles of the feet, interrupting the skin lines and containing dark dots within them, representing thrombosed blood vessels. Flat warts are usually asymptomatic flat papules or plaques that may be pink, flesh-colored, or slightly hyperpigmented and range in size from 1 mm to 1 cm; these are commonly found on the face. Genital warts are also asymptomatic flesh-colored or pink verrucous papules. The differential diagnosis of genital warts includes molluscum contagiosum, folliculitis, nevi, and skin tags. White vinegar (5% acetic acid) will give rise to a white appearance of genital warts when applied for 3 to 5 min; however, many false-positive and false-negative results may occur with this method. The most sensitive technique of detecting human papilloma virus (HPV) involves use of a DNA probe to detect HPV DNA.<sup>32</sup>

There is no uniformly effective treatment for warts, and many resolve spontaneously. Treatments for common warts include keratolytic acid paintings with



**Fig. 14. Wart (*verruca vulgaris*):** Note the verrucous flesh colored papule; closer examination would reveal thrombosed blood vessels within the wart.

17% salicylic acid twice daily or liquid nitrogen cryosurgery. Plantar warts are particularly difficult to eradicate and may be treated with the same modalities as for common warts. CO<sub>2</sub> laser vaporization may be used when the aforementioned therapies fail and has a 90% success rate. Additionally, case reports suggest that imiquimod 5% cream, which has been proven effective for genital warts, may be more effective on the thickly keratinized skin of the sole, especially when used in combination with cryosurgery which can weaken the stratum corneum to allow deeper penetration of the imiquimod.<sup>33</sup> Although the exact mechanism of action of imiquimod is unknown, it is proposed to work by activation of immune mediators. Imiquimod may be used 3 days a week for 16 weeks. Flat warts may be treated with topical tretinoin cream (Retin-A) applied twice daily.<sup>31</sup> For the treatment of genital warts, podophyllin is the initial therapy of choice and cure rates of up to 98% have been reported using this treatment.<sup>34</sup> Liquid nitrogen cryosurgery, imiquimod, and CO<sub>2</sub> laser vaporization may also be used and produce a cure rate up to 97%.<sup>34</sup>

### Molluscum Contagiosum

Molluscum infection is caused by a member of the poxvirus family. The most common sites of infection include the trunk and face of children and the genitals and inner thighs of sexually active individuals. The infection presents as 2- to 6-mm flesh colored or pink papules with central umbilication. The disease is often self-limited with spontaneous resolution within 18 months of onset. Immunocompromised patients may present with more aggressive disease that requires therapy. Treatment options include curettage of the papules, liquid nitrogen cryosurgery, and papule incision with a scalpel blade and expression of the contents. In patients with lesions in the genital region, testing for syphilis, HIV, and hepatitis B is recommended.

### Viral Exanthems

In discussing viral exanthems, it is helpful to first understand how viruses produce the associated rash. The virus responsible for generating the exanthem disseminates to the skin through the blood during the viremic phase of the viral illness. The observed exanthem is the result of the cutaneous host response to the virus. The most commonly observed viral exanthems are called morbilliform eruptions and have closely set macules and very slightly raised papules that are distributed diffusely over appreciable portions of the body.<sup>35</sup> Blistering eruptions appear as discrete blisters on a red base, “dewdrops on a rose petal.” Lacy red eruptions characterize infection with Parvovirus B19 (erythema infectiosum.)

Morbiliiform eruptions can be caused by measles, rubella, human herpesvirus 6 (roseola), and coxsackieviruses. These rashes are often confused with drug eruptions, which may have a similar presentation. It is, therefore, very important to obtain a detailed history including associated symptoms and medications. Many viral exanthems will be photoexaggerated (more lesions in sun-exposed areas).

**Erythema infectiosum**, or fifth disease, is linked to human parvovirus B19 and is distinctive in its presentation as a “slapped-cheek” rash with lacy erythema on the extremities and trunk. The contagious period of this infection precedes the development of cutaneous manifestations. Treatment is symptomatic. This disease has been identified as an increased risk factor for spontaneous abortion; however, the risk is small: 2–9% after infection during the first 16 to 28 weeks of pregnancy.<sup>36</sup>

Vesiculo-bullous viral eruptions most commonly include **herpes simplex virus types I and II** (HSV I and HSV II; Fig. 15, 16, and 17) and **varicella zoster virus** (VZV). Primary infection with HSV involves access gained through the host epithelial surface, replication, and infection of the associated neurons. The infection may present with painful cutaneous vesicles and systemic symptoms; however, many primary infections are asymptomatic. HSV infections may be definitively diagnosed with a viral culture. Tzanck smear may show the presence of multi-nucleated giant cells but is unable to distinguish between HSV and VZV. Varicella infection, or chickenpox, is characterized by the appearance of two or three successive crops of erythematous diffuse, pruritic vesicles and papules which then develop into pustules and crusted erosions. The polymorphous skin eruption typically begins on the torso and spreads centrifugally. The infection is highly contagious. **Herpes zoster**, also known as shingles, is due to reactivation of latent VZV. The cutaneous eruption involves painful pruritic vesicles that are distributed in a dermatomal pattern.

There is no cure for HSV, and superinfection is the most common complication. Systemic antiviral agents including acyclovir, valacyclovir, and famciclovir can be used for treatment and inhibit viral DNA synthesis, shortening the duration of recurrent infections. VZV is usually treated symptomatically with antihistamines and calamine lotion. Systemic antivirals may be used in immunocompromised individuals and adults, as they are at greater risk for complications. Routine childhood vaccination against VZV is currently recommended. Herpes zoster is generally self-limited, but again antiviral therapy is recommended for immunocompromised individuals, adults over age 50, or patients with ophthalmic zoster.



**Fig. 15. Herpes simplex virus (HSV):** Erythematous cutaneous vesicles are present periorally. A Tzank smear can help confirm the diagnosis.

### Other Infectious Rashes

We have discussed viral infections caused by bacteria, fungi, and viruses. This section will focus on other causes of rashes such as those produced by infestation with the scabies mite and with lice.

### Scabies

**Scabies** (Fig. 18) is a highly contagious infestation caused by the mite *Sarcoptes scabiei*. The mite and its feces typically cause severe pruritus. The female burrows into the epidermis, lays her eggs and dies within a month. Scabies can be diagnosed by the presence of red pruritic, papulovesicles with diagnostic linear burrows typically found in the interdigital webs of the hands, the volar wrist, axillae, genitalia, and anterior thighs. Definitive diagnoses can be made by scraping the burrow and finding the mite, eggs, or fecal matter under the microscope.

Treatment includes 5% permethrin cream, which is applied from the neck down; application is repeated after 7 days. In children under age 1, permethrin



**Fig. 16. Recurrent herpes simplex virus:** The erythematous vesicles surround the oral region.



**Fig. 17. Herpes (genital):** Painful erythematous vesicles are clustered on the penis of this patient.

should be applied from head to toe. Lindane 1% lotion may be applied in a similar manner; however, there is some concern of neurotoxic effects of lindane, especially in young children. For pregnant or lactating women and infants under 2 months of age, 5–10%



**Fig. 18. Scabies:** Erythematous, pruritic papulovesicles are present with the characteristic linear burrows.

sulfur ointment applied each day may be used. Additionally, it is important to ensure that all close contacts are treated, as the infestation is highly contagious.<sup>37</sup> Symptomatic treatment includes antihistamines as well as low dose topical corticosteroids for relief of the associated pruritus.

### Lice

Lice infection is ubiquitous throughout the United States and is caused by an infestation of *Pediculus humanus capitis* (head lice), *Pediculus humanus* (body louse), or *Phthirus pubis* (pubic louse). Intense pruritus is the most common symptom of pediculosis and arises from the host's reaction to the saliva and anticoagulant that is injected into the dermis by the feeding louse. Head lice is more common in the pediatric population, and the incidence is much lower in African Americans. The condition is most commonly diagnosed by school nurses, public health personnel, and family physicians.<sup>38</sup> Pediculosis pubis, or pubic lice, is transmitted sexually, shows no racial difference in distribution and is usually diagnosed in venereal disease clinics and student health services.<sup>39</sup>

Diagnosis is made by history of intense itching. Erythema and scaling as well as pruritic papules may be present, and the adult louse may be visible to the naked eye. Diagnosis is confirmed by plucking the hairs and microscopically examining nits, live adult lice, immature nymphs, or eggs.

Several agents are available for treatment of lice infestation. Permethrin 1% cream rinse is applied to towel-dried hair after shampooing with regular shampoo, left on for 10 min and rinsed off with water. A single application is usually sufficient although some dermatologists recommend a second treatment 7 to 10 days later. RID is an over-the-counter synergized pyrethrin which is applied undiluted to the scalp and left on for 10 min. Hair is then washed thoroughly and nits are removed with a fine-toothed comb. Finally, lindane shampoo, 30–40 mL can be lathered onto the scalp for 4 min then rinsed thoroughly.

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