Chapter 17
Pediatric Dermatology

Lubna H. Suaiti, Yasin A. Damji and Margaret S. Lee

A major theory regarding the evolution of skin pigmentation is defense against folate photoproteolysis, as folate is necessary for normal nucleic acid biosynthesis [1]. Thousands of years of migration from our origins in Africa, the distance from the equator, and different environmental parameters are believed to play roles in genetic selection and variations in skin color [2–5]. This chapter will discuss skin conditions that are commonly observed in patients with skin of color or that phenotypically differ compared to presentations in light skin; for example, erythema is not as easily distinguished or might have a violaceous or brown hue in dark-skinned patients. Also, pigmented skin may demonstrate variant responses to skin disease, including pigment lability, follicular vs hyperkeratotic presentations, mesenchymal (granulomatous or keloidal) reactions, and bullous lesions [6, 7]. Normal pigmented skin findings, birthmarks, and pediatric hair disorders will also be discussed. We end with a brief review of the psychosocial impact of congenital and acquired skin conditions, a fundamental aspect of the patient and family experience in pediatric dermatology.

L.H. Suaiti
Department of Dermatology, Boston University Medical Center,
609 Albany St., Boston, MA 02120, USA
e-mail: lsuaiti@gmail.com

Y.A. Damji
Boston University School of Medicine, 72 East Concord St.,
Boston, MA 02118, USA
e-mail: ydamji@bu.edu

M.S. Lee (✉)
Department of Dermatology, Boston University School of Medicine,
609 Albany St., Boston, MA 02118, USA
e-mail: margaret.lee@bmc.org

Birthmarks

Congenital pigmentary lesions vary according to race, sex, and type of birthmark. Birthmarks are either localized and limited to having potential psychosocial impact (discussed later in this chapter) or are extensive and might signify systemic involvement [8–11]. In this chapter, we will review birthmarks that are due to pigment changes, as they are more common among dark-skinned newborns. There is generally no treatment available for pigmented birthmarks, although laser therapy is sometimes helpful.

Dermal Melanocytosis

Between the 11th and 14th weeks of gestation, dermal melanocytes migrate from the neural crest to the epidermis or undergo apoptosis; however, some dermal melanocytes persist at the time of birth and produce melanin within the dermis [12]. Hepatocyte growth factor and other gene mutations, i.e., GNA11 (G protein subunit alpha 11) and GNAQ (G protein subunit alpha G), are believed to play a role in dermal melanocyte survival in head and neck, distal extremities, and sacral skin area resulting in Nevus of Ota, Nevus of Ito, blue nevus, and Mongolian spots. The Tyndall effect, an optical phenomenon, results from reflection of dermal pigment giving the bluish appearance of dermal melanin [12–14].

Mongolian Spots

The most common form of congenital dermal melanocytosis is usually referred to as Mongolian spots, occurring in approximately 90% of infants of color all over the world [15, 16]. They typically are observed at birth as blue-gray or slate gray patches on the lumbosacral region, and fade partially or completely over a period of years. "Aberrant" Mongolian spots can be found in extra-sacral locations. Superimposed Mongolian spots have been reported (a darker macule or patch overlying a lighter colored patch) [13]. Extensive Mongolian spots at birth or persistent Mongolian spots in older children may be associated with lysosomal storage diseases, i.e., Hurler disease, GM1(monosialotetrahexosylganglioside) gangliosidosis, Mucopolysaccharidosis type II, and Niemann–Pick disease [17]. Extensive Mongolian spots are also seen in phakomatosis pigmentovascularis; careful physical exam is crucial to identify coexisting vascular birthmarks, which might be difficult to appreciate in darkly pigmented patients [13, 16, 17]. It is important to note uniformity of color and chronicity, to avoid confusing these birthmarks with ecchymoses due to child abuse or neglect (Fig. 17.1).

Fig. 17.1 Dermal melanocytosis on an infant's back mimicking ecchymoses

Nevus of Ota (Oculodermal Melanocytosis/Nevus Fuscoceruleus Ophthalmomaxillaris)

Nevus of Ota is common among Asians and other females with skin of color [18]. It is usually a unilateral blue-gray or brown mottled patch, located on skin and mucous membranes innervated by the ophthalmic and maxillary branches of the trigeminal nerve. Approximately 5% are bilateral [19]. Up to 70% of patients have ipsilateral scleral involvement. It presents shortly after birth and darkens around puberty or may not be obvious until puberty, when it is thought that hormones stimulate pigmentation [18, 20–23]. Although rare, cutaneous, choroidal or leptomeningeal melanoma has been reported within nevus of Ota [24, 25].

Nevus of Ito (Nevus Fuscoceruleus Acromiodeltoideus)

Clinically and histologically similar to nevus of Ito, this form of dermal melanocytosis is located on the posterior supraclavicular, scapular, and deltoid areas (lateral cutaneous nerve distribution) [19, 26]. Malignant melanoma transformation within nevus of Ito has been reported [27]. Coexistence of nevus of Ito with nevus spilus [26] or nevus of Ito with nevus of Ota [19] have been described.

Blue Nevus (Dermal Melanocytoma)

Blue nevi are congenital in 25% of cases, and 75% are acquired during childhood or later in life. They are usually solitary, blue or blue-black papules on the scalp, sacral, and extensor distal extremities [23, 28]. Giant congenital blue nevi are rare, commonly seen on the scalp, with risk of malignant transformation during childhood or later in life [28, 29]. Blue nevi are categorized as common (1 cm or less, often located on the dorsal hand), cellular (1–3 cm, most often on the buttock and sacral area), or epithelioid (sporadic or in association with Carney complex, usually on trunk and extremities) [23, 28–30]. It is challenging to differentiate atypical cellular blue nevi from malignant blue nevi histologically.

Café-Au-Lait Macules

Café-au-lait macules (CALMs) are a very common finding in the general population; prevalence of CALMs in large cohort studies range from 0.3 to 2.7% of newborns [11, 31, 32]. In one large cohort study, 18.3% of black infants versus 0.3% of white infants were found to have CALMs at birth [11]. These are solitary or multiple, well-defined, evenly pigmented brown macules or patches. Smooth borders are described as "coast of California" while serrated borders are described as "coast of Maine." Average size is 0.2–4 cm in newborns and 1.5–30 cm in older children [32]. Histopathologic examination reveals increased melanin content in the basal layer as well as in melanocytes, with no evidence of increased melanocytic infiltration. Multiple CALMs could be a normal finding with no other systemic disease association (1.9% of normal children with skin of color reportedly have more than 2 CALMs) or might signify systemic involvement in the context of several genetic disorders. Neurofibromatosis 1, neurofibromatosis 2, ring chromosome syndrome, and Watson syndrome are most closely associated with multiple CALMs, and McCune Albright syndrome with large/segmental CALMs [32]. Other systemic disorders with questionable association with CALMs include tuberous sclerosis, ataxia-telangiectasia, Bloom, and Silver–Russell Syndromes [31, 32]. Full discussion of associated genodermatoses is beyond the scope of this chapter.

Patterned Pigmentation (Pigmentary Mosaicism)

Patterned pigmentation refers to hyper- or hypopigmentation that results from two different cell clones (chromosomal mosaicism) [33–35]. A wide range of clinically distinct presentations include linear Blaschkoid, broad checkerboard (flag-like), and phylloid (leaf-like) hyper- and hypopigmentation. Patterned pigmentation is rarely associated with cutaneous, skeletal, and neurological abnormalities. Cases are

sporadic; however, a few familial cases due to functional (epigenetic) modification of autosomal genes have been described [34, 35].

Linear Nevoid Hypomelanosis Versus Linear and Whorled Nevoid Hypermelanosis

Linear nevoid hypomelanosis, also known as hypomelanosis of Ito (HI), describes hypopigmented Blaschkoid linear swirls and streaks [36, 37]. It presents at birth or during childhood as a result of somatic mosaicism with two clones of cells, where one clone produces less melanin. It affects males and females with no racial predilection; however, it is easier to appreciate in darker skinned patients [36, 38]. Reported association with neurologic, ocular, and musculoskeletal abnormalities varies widely, between 30 and 94% of patients; it is now believed that the lower estimates are more likely [37].

Conversely, linear and whorled nevoid hypermelanosis (LWNH) involves hyperpigmented linear swirls that follow Blaschko's lines due to pigmentary mosaicism causing increased melanin in one clone. Similar to HI, it has been associated with ocular, musculoskeletal, and neurological abnormalities, but in both conditions the risk of extracutaneous abnormalities should not be overemphasized to patients and parents. In one case review of 16 cases with LWNH, only one out of six patients with extensive LWNH had extracutaneous abnormalities [39]. Dermoscopic exam shows linear and circular streak-like pigmentation organized in a parallel linear manner [40]. Differential diagnoses include incontinentia pigmenti and macular/early epidermal nevus. Histopathology reveals increased basal layer hyperpigmentation with no melanophages [33].

Pigmentary Demarcation Lines

Pigmentary demarcation lines, also known as Futcher's lines or Voight's lines, are normal in skin of color and persist through adulthood [41]. They represent an abrupt transition of pigmentation established during fetal development [42–44]. The most commonly observed lines are found on the anterolateral upper arms and postero-medial legs (Fig. 17.2a, b).

Transient Infantile Patterned Hyperpigmentation

An often-overlooked diagnosis, transient infantile patterned hyperpigmentation is a benign condition that fades over a few months [45]. Skin examination reveals multiple transverse, parallel pigmented thin lines on the extremities and/or torso. In contrast to hormonal hyperpigmentation of the genitals and areolar skin, transient infantile patterned hyperpigmentation is believed to be due to intrauterine fetal position and friction [45, 46].
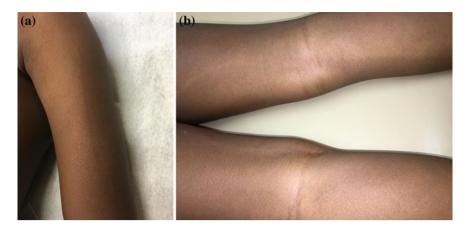
Fig. 17.2 **a** Futcher's line on the upper arm. **b** Futcher's line on the posterior leg

Pigmented Nevi

Many pigmented lesions are benign and more frequently observed in children of color. Mucosal hyperpigmentation has been reported in 72–100% of darker skinned populations, with 25% onset before the age of 10. Positive family history is usually elicited. Scleral hyperpigmentation is reported in 78%, 8% before the age of 10 [6]. Hyperpigmented palms, melanonychia (pigmented nail plate), and congenital melanocytic nevi are also common.

Melanonychia and Nevi of the Nail Matrix

Longitudinal melanonychia, also known as melanonychia striata, represents melanin deposition within the nail plate. While 5–10% of longitudinal melanonychia in adults are due to malignant melanoma, no cases of invasive melanoma have been reported in association with pediatric longitudinal melanonychia [47]. Melanonychia presents as a longitudinal hyperpigmented band with variable widths (up to complete involvement of the nail plate) affecting fingernails, especially thumbs, more often than toenails [47, 48]. Color can vary from bluish-gray, brown, to black. Melanonychia of childhood is predominantly caused by benign lentigines and melanocytic nevi, much less commonly due to atypical melanocytic nevi or malignant melanoma in situ. Biopsy or surgical excision is recommended only for lesions with concerning signs: irregularity of pigmentation or borders, width of the pigmentation exceeding 3 mm, hazy border, Hutchinson's sign (pigmented proximal or lateral nail bed), irregular pigmented lines on dermoscopy, and rapid evolution [48].

Congenital Melanocytic Nevi (CMN)

Congenital melanocytic nevi are melanocytic neural crest-derived hamartomas, common in skin of color. They occur as a result of unregulated melanoblast growth between the 5th and 24th weeks of gestation. Approximately 1% of newborns are affected, with slightly higher female prevalence [49]. Giant CMN have different histological elements because they originate from pluripotent stem cells and are difficult to evaluate using dermoscopy, as the cells extend deeper into the dermis and subcutaneous tissue [49, 50]. Hypertrichosis, perifollicular hypo/hyperpigmentation, pseudomilia, and vascular structures are common dermoscopic findings of CMN [50]. Neurocutaneous melanosis (NCM) is a melanocytic proliferation of the leptomeninges (brain and/or spinal cord) that occurs in association with larger and axial CMN [50]. Diagnosis and management is the same for all skin phototypes.

Disorders of Late-Onset and Secondary Hyperpigmentation

Hyperpigmented lesions occurring beyond infancy include entities of congenital origin. Dermal melanocytosis can manifest later in life, i.e., Nevus of Ito, Ota-like (Hori nevus), and acquired dermal melanocytosis of the hands [51, 52]. Also, late-onset linear and whorled nevoid hypermelanosis in a zosteriform distribution has been reported [53].

Becker's Nevus (Pigmented Hairy Epidermal Nevus)

Becker's nevus is thought to be a hamartomatous congenital lesion of ectodermal and mesodermal origin where the pigmentation usually presents years later, although congenital cases have been described [54]. A pigmented patch may appear during childhood then darken during puberty along with coarse hair development. Males are more often affected. Androgen is believed to play a major role in its development. It is usually unilateral, on chest, shoulder, lateral arm, and scapular regions. Hypertrichosis and acneiform lesions may be seen within Becker's nevus. Rare cases have been reported involving hands, feet, and segmental face with mucosa [55]. Becker's nevus syndrome is characterized by association with musculoskeletal abnormalities such as ipsilateral mammary hypoplasia or odontomaxillary dysplasia [56, 57]. Treatment is mainly reassurance and camouflage. Laser treatment of the hypertrichosis may be attempted with low-fluence high-repetition-rate diode lasers (808–810 nm) [58], and the pigmentation has been treated with long-pulsed ruby laser [59] and fractional resurfacing [60], although greater risk of dyspigmentation exists for patients of color.

Postinflammatory Hyperpigmentation

Many dermatologic diseases heal with hyperpigmentation or hypopigmentation, especially in darker skinned patients [61, 62]. The exact mechanism is not fully understood; however, many inflammatory mediators have been found to increase melanogenesis, such as interleukin 1 and 6 [62]. Other studies have suggested that epithelial–mesenchymal interactions via growth factors such as keratinocyte growth factor and fibroblast-derived growth factor modulate melanogenesis [63].

Friction Hypermelanosis

Friction hypermelanosis is an acquired hyperpigmentation resulting from continuous rubbing of the skin [64, 65]. Friction hypermelanosis is common in darker skin but it is not commonly observed in infants or young children. Unique curvilinear hyperpigmented patches were described in infants wearing tight socks and mittens for a prolonged length of time, also known as sock-line and mitten-line hyperpigmentation. The differential diagnosis includes child abuse and amniotic band syndrome [66]. Vertically oriented, round, discrete, hyperpigmented patches overlying the lumbosacral area have been described in an institutionalized orphan as a result of repetitive backward and forward movement, inspiring the name "orphan rocker tracks" [67]. A similar hyperpigmentation, Davener's dermatosis, has been described among young orthodox Jewish males who perform repetitive rocking motions during religious rituals [65].

Idiopathic Eruptive Macular Pigmentation

This condition presents in children and adolescents with sudden onset of multiple brown nonconfluent macules on the trunk, neck, face, and proximal extremities with no preceding inflammatory skin disease or medication administration [68, 69]. The pathogenesis in unknown, although hormonal etiology, has been suggested [68]. Histopathologic exam reveals basal cell layer hyperpigmentation and occasional dermal melanophages without structural changes in the basal layer or lichenoid inflammatory infiltrate. It is important to differentiate it from ashy dermatosis in which the macules are gray, sometimes with erythema, and basal layer vacuolization. Other differential diagnoses include pityriasis versicolor, urticaria pigmentosa, café-au-lait macules, and postinflammatory hyperpigmentation [68, 70]. It resolves over the course of a few months to years [68].

Reticulate Hyperpigmentation

Skin disorders with reticulate hyperpigmentation (net-like pattern) are not very common, and they can be idiopathic, genetic, or drug-induced. Family history, age of onset, race, sex, and distribution as well as full hair and skin exam are important when approaching the patient [71]. Confluent and reticulated papillomatosis (CARP) usually affects peripubertal females, presenting with reticulated hyperpigmented patches, papules, and plaques on the chest and upper back, sometimes axillae [72]. Prurigo pigmentosa, which has a similar distribution to CARP but affects young Japanese females, presents with pruritic inflammatory papules that heal with reticulate hyperpigmentation [73]. Both disorders have shown response to oral minocycline [74] and other tetracyclines.

Genetic disorders with reticulate hyperpigmentation are rare [75]. They can be generalized, flexural, and acral. Genital reticulate hyperpigmentation in association with vitiligo on the same skin area has been described [71, 75].

Disorders with Later Onset Hypopigmentation and Depigmentation

Postinflammatory Hypopigmentation and Depigmentation

Although acquired partial or total pigment loss affects all skin types, it is more noticeable and troublesome for darker skinned patients. Certain skin diseases have a tendency to resolve with postinflammatory hypopigmentation, including lichen striatus and pityriasis lichenoides chronica; infections such as impetigo, pityriasis versicolor, and chicken pox; and skin injuries such as burns and cryotherapy [76]. Leukotriene C4 released during inflammation was found to reduce melanogenesis [62].

Vitiligo

Vitiligo is a common disorder of acquired depigmentation, affecting 1–2% of the world population [77]. About half of the cases occur before the age of 20 and about one-fourth of total cases occur before the age of 8. No gender or skin phototype predilection is known [77, 78]. The underlying pathogenesis includes autoimmunity, genetic, neurologic, and biochemical events resulting in destruction of melanocytes [77–79]. Hypopigmented and depigmented macules or patches with focal, generalized, or segmental distribution is observed [77]. Trichrome vitiligo represents the combination of hypopigmentation, depigmentation, and normal skin pigment in the same patient [80]. Treatment of childhood vitiligo remains challenging; about one-third of patients do not respond to treatment [77–79]. Topical treatment is

preferred in childhood vitiligo if less than 20% of body surface area (BSA) is involved, and includes topical steroids, topical calcineurin inhibitors, and calcipotriol. A combination of these may yield a better response in patients who have failed monotherapy [78]. Camouflage is a good option for small lesions or when side effects are a concern. Other treatment options include narrow-band UVB, excimer laser, surgical approaches including epidermal autotransplantation, intralesional steroid injection, and total depigmentation for the most extensive involvement [78, 79]. Many of these are difficult to perform on children without sedation.

Pityriasis Alba

Pityriasis alba is a common skin disorder affecting children 3–16 years of age, with higher incidence reported in patients of color and the atopic population [81–83]. It may be a mild form of atopic dermatitis, with spontaneous relapse and remission. Clinically, it can present with subtly erythematous and scaly, poorly defined small patches, typically on the cheeks, then hypopigmented patches with minimal to no scale [83]. Most patients present with poorly defined hypopigmentation and deny preceding inflammation. Differential diagnosis includes postinflammatory hypopigmentation and pityriasis versicolor. Photoprotection, emollients, topical tacrolimus [84], and low potency topical steroids may improve pityriasis alba [81].

Progressive Macular Hypomelanosis

Progressive macular hypomelanosis manifests as hypopigmented, non-scaly macules coalescing over the trunk. It usually affects adolescents, with no gender predilection [85]. Pathology shows only reduced melanin content compared to normal skin. The etiology remains unknown, although a *Propionibacterium* species is thought to play a role, causing red fluorescence under Wood's lamp and in some cases improving after treatment with benzoyl peroxide 5% gel with or without clindamycin 1% lotion [86–88]. Other treatment options include psoralen and Ultraviolet A (PUVA), narrow-band Ultraviolet B, and oral tetracycline derivatives [85, 87]. It is important to counsel the patient regarding the course and the outcome of the disease, as recurrence after successful treatment is often reported, and it may take up to 5 years before spontaneous regression, if any, is observed [85].

Clear Cell Papulosis

Clear cell papulosis is a rare entity predominantly affecting Asian children, between 4 months and 5 years of age. It presents with multiple asymptomatic non-scaly

hypopigmented macules or thin papules on the lower abdomen and tends to follow mammary lines [89–92]. Pathology shows normal basal keratinocytes admixing with larger keratinocytes characterized by pale cytoplasm that stain positive for cytokeratin 7, EMA, CEA, and AE; therefore, some authors consider it a benign form of extramammary Paget disease [93]. Not all cases display reduced basal pigmentation, so the exact etiology of hypopigmentation remains unknown. Spontaneous regression has been reported [93].

Dermatitis

Atopic Dermatitis

Atopic dermatitis (AD) is the most common pediatric skin disorder; more than two-thirds of patients present before the age of 5 years [94, 95]. Genetic predisposition, barrier dysfunction, and cytokine signaling along with immunoglobulin switching are thought to contribute to etiology, and interestingly, recent studies have concluded that pediatric AD differs from adult AD in their pathophysiology [96, 97]. For example, pediatric AD patients display increased expression of CD69 on T cells soon after CD4 and CD8 are activated, but lower levels of IL-22, which increases susceptibility to infection [97]. IL-22 is believed to induce epidermal hyperplasia, suggesting that it plays an important role in chronic lesions, seen more in adults than in children with AD. About two-thirds of pediatric patients outgrow their AD within 10 years, likely due to upregulation of T_H1 and T_H17, resulting in decreased T_H2 and T_H22 levels [97].

African American (AA) patients reportedly have a higher incidence rate of AD, and of greater severity, compared to Asians and Caucasians [96]. Although studies on the structure and function of ethnic skin are limited and somewhat controversial, with conflicting results, some have shown that AA patients have the lowest ceramide-to-cholesterol ratio in the stratum corneum, higher transepidermal water loss, lower response to capsaicin, and decreased itch signaling due to lower pH levels in their skin [95, 98]. Genetic susceptibility also seems to differ in AA patients. Fewer AA patients have filaggrin mutations than their Caucasian counterparts, where filaggrin mutation is linked to AD susceptibility and severity [95]. Caucasian AD patients tend to develop eczematous plaques on extremity flexor surfaces while AA patients tend to alternatively or additionally develop discrete follicular papules, prurigo nodularis and lichen simplex chronicus or lichen planus-like lesions on the extensor extremities (Fig. 17.3). The infraorbital crease (commonly referred to as Dennie–Morgan lines) is a minor feature of AD often seen in AA and Asian patients, but is common among darker skinned children regardless of atopy, so its presence should not be overemphasized in diagnosing AD in these patients [99].

Fig. 17.3 Atopic dermatitis in a child with thick, lichenified plaques

Clinicians should tailor management toward individual symptoms and severity of skin findings (including degree of lichenification or presence of prurigo nodules), as well as comorbidities such as food and environmental allergies or superinfections. First-line therapy always involves optimization of baseline skin care using bland, hypoallergenic emollients, and cleansing regimens, and the safe but effective use of topical corticosteroids. Alternative or adjunctive strategies include wet wraps, antihistamines, calcineurin inhibitors, other nonsteroidal anti-inflammatory topicals, nb-UVB phototherapy, and systemic immunosuppressants [94, 95]. Phototherapy and systemic medications are reserved for severe, recalcitrant cases. Cyclosporine is often used as a first-line systemic agent due to its relatively rapid effect, and stopped upon resolution of clinical flaring or after transitioning to safer long-term systemic agents such as methotrexate, azathioprine, or mycophenolate mofetil [100, 101]. Several biologics are in clinical trials at the time of this printing.

Treatment of pediatric AD patients is the same regardless of skin pigmentation; however, while managing AA patients with AD, a few points should be considered. Hypopigmentation is usually postinflammatory but patient/parent concerns over corticosteroid-related hypopigmentation may affect compliance. Thicker plaques usually require more potent corticosteroids or topical treatment under occlusion [95]. Risk of azathioprine toxicity is higher in the AA population due to altered dosing requirements; there is reportedly a 20–30% decreased oral bioavailability and 17% reduction in thiopurine methyltransferase activity in these patients [102]. And finally, higher doses of nb-UVB may be needed to treat AA patients with AD [95].

Allergic Contact Dermatitis

Allergic contact dermatitis (ACD) in children is not uncommon and may even occur in early infancy [103]. Females are affected more than males [104]. Nickel remains the number one antigenic culprit; other common allergens include fragrance mix, lanolin, thimerosal, and neomycin [103, 104]. ACD in skin of color may be missed by clinicians unfamiliar with cultural practices, such as henna body art and early skin piercing, and undertreated by those unfamiliar with lichenification or lichenoid dermatitis as features of ACD in skin of color, or those who do not look for subtle erythema in pigmented skin [6]. Intricate skin art using traditional red henna (leaves of the plant *Lawsonia inermis* crushed into essential oils) is common in the Indian subcontinent and the Middle East, but ACD to red henna is rare; allergic contact dermatitis is more often caused by adulterated products termed 'black henna' in which p-phenylenediamine (PPD) is admixed with red henna [105]. Keloidal lesions, an erythema multiforme-like targetoid reaction, bullous, and generalized dermatitis due to black henna have been reported [105]. Patch testing may be needed to confirm a contact allergen [104], but patients should be cautioned about dyspigmentation in patch test sites. Education, allergen avoidance, and topical corticosteroids or steroid sparing agents for active dermatitis are all important in the management of ACD [106].

Seborrheic Dermatitis

Seborrheic dermatitis (SD) is a very common skin disorder that affects patients of all ages [107]. Incidence in infants reaches 42% [108]. *Malassezia furfur* overgrowth and/or hypersensitivity are thought to contribute to the etiology of SD, especially in adults, but the etiology of infantile SD remains uncertain; metabolic disturbance of essential fatty acids is assumed to play a role in the pathogenesis [109] while other studies have concluded that *Pityrosporum ovale* is likely to be the cause of infantile SD [110]. SD presents with nonpruritic, scaly, erythematous lesions on the scalp, face (eyebrows and paranasal skin), retro-auricular folds,

flexures, and diaper region [107]. In dark-skinned patients, the erythema is not easily distinguished, and patients may present with scalp dandruff and hyperkeratosis that can be mistaken for tinea capitis, atopic dermatitis, or scalp psoriasis [111]. Also, they may present with hyper- or hypopigmented scaly lesions. Topical preparations are usually used to treat infantile SD, e.g., mineral oil or bland emollients to loosen scale, 2% ketoconazole shampoo, and topical steroids used with caution to prevent systemic absorption or atrophy [110].

Papulosquamous Disorders

Psoriasis

Psoriasis is a common chronic inflammatory skin disorder that affects children with an annual incidence of 40.8/100,000 found in one study of the US Midwest [112]. Similar to adults, the most common type of psoriasis in children is plaque-type psoriasis [113, 114]. Guttate, inverse, palmoplantar, and pustular psoriasis are also seen in pediatric patients [114, 115]. Guttate psoriasis is more often seen in children, tends to follow streptococcal infection more often than in plaque-type pediatric psoriasis, and tends to predict more severe psoriatic disease [116]. Scalp psoriasis was found to be more prevalent in girls while nail psoriasis was more common among boys. Nail psoriasis does not correlate with disease severity in pediatric patients, in contrast to what is observed in adults [113, 115]. Darker skin pigmentation may complicate the assessment of psoriasis severity beyond the greater difficulty observing erythema; one study found thicker plaques and more dyspigmentation in psoriasis in patients of color as compared to Caucasians [117]. Also, scalp psoriasis lesions in dark-skinned children may be more hyperkeratotic than in patients with lighter skin [111].

Pediatric psoriasis is generally treated with emollients, keratolytics, corticosteroids, and steroid sparing agents [118]. No standard guidelines exist for phototherapy, systemic, and biologic drugs for pediatric psoriasis [118]; many protocols are derived from adult psoriasis treatment regimens and other specialists' experience with biologics and systemic drugs used to treat children with rheumatoid arthritis, hematological malignancies, and Crohn's disease [118–120]. Psychological support is crucial for children with psoriasis, given that it is associated with obesity, hypertension, diabetes mellitus, and psychosocial stress [118, 121].

Pityriasis Rosea

Pityriasis rosea (PR) is a papulosquamous skin disorder of uncertain etiology that affects young individuals worldwide [122, 123]. It may be associated with human

herpes virus 6 and 7. PR typically manifests as mildly pruritic erythematous thin papules and plaques with few scales on the trunk, erupting within a few weeks of the appearance of a *herald patch*—a single, relatively larger patch or plaque with raised scaly margins [122]. Atypical variants have been described, including inverse, unilateral, erythema multiforme-like, and generalized papular and papulovesicular PR [124–127]. Case reports and studies of dark-skinned individuals with this eruption have reported reduced or absent erythema, thicker scale, and significant itching in comparison to PR in light-skinned patients [123]. In addition, generalized papular, follicular, and erythema multiforme-like variants were noticed to be more prevalent among dark-skinned and Indian patients [124, 126, 127]. Facial distribution is also observed among African Americans with pityriasis rosea [123, 125]. About two-thirds of dark-skinned patients with PR heal with hyperpigmentation while postinflammatory hypopigmentation tends to be noticed after the papular variant resolves [123]. Pityriasis rosea tends to heal spontaneously; treatment is mainly to control the itching using antihistamines, emollients, and topical steroids. Phototherapy, systemic antibiotics, and antivirals have been used in severe and recalcitrant cases with different results [123, 128, 129]. The dyspigmentation left by this benign condition can be very obvious and quite upsetting to patients and parents.

Lichen Planus

Lichen planus (LP) is a chronic pruritic skin disorder that mainly affects adults [130]; however, studies from different countries have shown increasing rates among children, reaching ∼11% of cases [130–134]. In one study of 36 children from the United States, childhood LP affected females twice as often as males, and 72% were African American [135]. Childhood lichen planus is not uncommon in India, Middle East, and Mexico [131, 133, 135]; a retrospective study in the United Kingdom showed that more than two-thirds of the children with lichen planus came from Indian descent [134]. The exact etiology remains unknown and many theories including autoimmune, infectious, genetic, Hepatitis B infection, and/or vaccination triggers have been suggested [130, 136]. Classic LP is the most common presentation of childhood lichen planus [135], but linear, annular, mucosal, bullous, and lichen planopilaris subtypes are also reported [133, 135, 137, 138]. In darker skinned patients, lichen planus lesions tend to be gray in color rather than violaceous, with persistent and severe postinflammatory hyperpigmentation [139]. Actinic lichen planus predominates in tropical areas [130, 132]. Treatment of childhood lichen planus is mainly achieved by the use of topical corticosteroids under occlusion and topical tacrolimus. Systemic drugs and phototherapy are reserved for eruptive, bullous, and recalcitrant cases [130, 138].

Acneiform Eruptions

Acne Vulgaris

Acne vulgaris is one of the most common skin disorders, affecting patients from all ethnic backgrounds and skin pigmentation [7, 140–142]. Due to the practice of applying heavy oils and thick pomades to their hair, African American patients tend to have more comedonal acne on the forehead, also known as *pomade acne* [143]. Newer trends in African American hair care practices such as chemical relaxation led to a reduction in the use of thick hair oils, eventually decreasing the incidence of pomade acne [139]. Postinflammatory hyperpigmentation is very common among patients with darker skin, and the application of cosmetic products used to cover the hyperpigmentation can lead to acne cosmetica among those groups [140–142]. Treatment of acne vulgaris in skin of color does not differ from that for other ethnic groups; however, patient education and counseling are crucial, as well as sensitivity toward the psychological impact of the hyperpigmentation and/or acne-related keloidal scarring [141, 144].

Periorificial Dermatitis

Periorificial dermatitis is an eruption of erythematous, flesh-colored papules in association with skin desquamation and rare pustules around the mouth, eyes, nose, and occasionally the vulva or perineum [145–147]. Topical corticosteroid application may play a role in the pathogenesis [145]. Histology may reflect a granulomatous dermatitis, as seen in reports of childhood granulomatous periorificial dermatitis, sarcoid-like granulomatous dermatitis, and facial Afro-Caribbean childhood eruption [145, 147]. Treatment includes discontinuing steroid application, topical 0.75% metronidazole [148], topical calcineurin inhibitors, and mupirocin [145]. Oral antibiotics such as tetracycline, doxycycline, and erythromycin have been used [145, 147].

Hair Disorders

Human hair can be categorized based on sulfur protein content, keratin type, and amino acid composition, but it is generally classified based on ethnic background, differentiating Caucasian, Asian and African American (AA) hair. AA hair shafts are elliptical in cross section, with a variable diameter along the hair shaft, and also characterized by random reversals and twisting. AA hair follicles are curved, with less elastic fiber content attaching it to the dermis, resulting in spiral, curly hair and higher risk for hair shedding and breakage [149]. AA hair dryness is due to

decreased sebaceous gland activity and lower moisture content compared with other ethnic groups [149].

Trichorrhexis Nodosa

Trichorrhexis Nodosa is a congenital or acquired hair shaft disorder. Congenital cases are seen among various genetic diseases including trichothiodystrophy and Menkes kinky hair syndrome, while acquired cases are caused by excessive heat, forceful combing and prolonged chemical use. Patients usually present with complaints of brittle, dull hair appearance, and white spots along the hair shaft. Microscopic hair shaft exam reveals what looks like two paintbrushes facing each other, which represent the whitish dots along the hair shaft. Treatment of acquired trichorrhexis nodosa is mainly through reducing chemical and physical hair shaft trauma [149].

Traction Alopecia

Traction is a common cause of hair loss that occurs as a result of prolonged tension applied on the hair, leading to clinical or subclinical inflammation of the hair follicles. It usually starts during childhood and might progress to permanent loss of the hair follicles. Traction alopecia predominantly affects AA females, with onset reportedly as young as 8 months of age. In one clinic population the prevalence of traction alopecia in AA females between the ages of 5 and 14.5 years was 18% [150]. Another study showed that African schoolchildren with traction alopecia increased from 8.6% in the first year of school to 21.7% in the last year of high school [151]. Traction alopecia is usually marginal frontal and/or temporal, with the presence of fringe hair. Hair casts in a biopsy indicate ongoing traction alopecia. Treatment is mainly through prevention and education.

Tinea Capitis

Tinea capitis is the most common fungal infection affecting the pediatric population. AA children have the highest incidence rate, in males more than females. *Trichophyton tonsurans* is an anthropophilic organism; it remains the most common causative agent of tinea capitis in North America. *Microsporum canis* is the most frequent cause of zoophilic tinea capitis [152]. In AA patients, tinea capitis may manifest as non-inflammatory disease with patches of hair loss, hyperkeratosis, or breakage of the hair shaft at the scalp surface (black dot tinea). Another clinical presentation is kerion, a tender, boggy, sometimes purulent plaque in a patient who

may also have fever, malaise, and occipital lymphadenopathy. Bright green fluorescence with Wood's lamp is seen only in ectothrix fungi such as *M. canis*. It is negative in the presence of *T. tonsurans*, an endothrix infection. Dermoscopic examination reveals comma-shaped and corkscrew hair shafts. Hair culture is negative in up to 50% of cases [111, 153] so clinical features and history are important. Differential diagnoses of non-inflammatory tinea capitis with scalp hyperkeratosis include seborrheic dermatitis and atopic dermatitis, or rarer cause such as collagen vascular diseases and Langerhans cell histiocytosis, where erythema is difficult to appreciate in dark skin [111].

Tinea capitis is frequently undertreated and physicians tend to prescribe low doses of antifungal medication, resulting in frequent relapses once treatment is discontinued. While terbinafine, fluconazole, and pulsed itraconazole can be used for shorter or more convenient regimens, griseofulvin is still widely used by pediatric dermatologists due to its cost and safety profile. In cases of *M. canis* fungal infection, a longer duration of therapy is usually required [153].

Piedra

Piedra is a superficial fungal infection characterized by easily detachable gritty white or yellowish nodules (white piedra) or black nodules (black piedra) on hair shafts, usually of the axilla or scalp [154–156]. Black piedra is caused by *Piedraia hortae* while white piedra is caused by *Trichosporon* species. Most reported cases in the United States have been in immigrants from tropical regions [155]. Piedra affects females more than males, although in genital hair white piedra is more prevalent in black males [156]. Humid climate, poor hygiene, and the practice of veil hair covering have been implicated in the pathogenesis [157, 158]. They present easily detachable gritty nodules on the scalp, facial, or genital hair [154–156]. Diagnosis is based on the clinical presentation, KOH, and fungal cultures [152, 153]. Treatment is mainly achieved through hair shaving, although this is usually not desirable for scalp involvement. Oral azoles and topical antifungal shampoos have been successfully used with variable reported recurrence rates [154–157].

Vesicular and Pustular Diseases in Newborns

Transient neonatal pustular melanosis and infantile acropustulosis are seen more often in skin of color. Conversely, miliaria is less common, due to more efficient body temperature regulation with less sweating compared to white skin [6].

Transient Neonatal Pustular Melanosis

Transient neonatal pustular melanosis (TNPM) predominantly affects black new-borns with a 4.4–15% incidence rate [6, 159, 160]. It usually presents at birth as innumerable, superficial pustules without surrounding erythema. Pustular lesions last up to 2 days, and then heal leaving delicate collarettes of scale around hyperpigmented macules, which take from 2 weeks to months to fade [159]. Pustules may not be seen due to disruption perinatally. An infant born with multiple hyperpigmented macules that fade away over weeks to months (unlike true lentigines that do not fade) can be presumed to have had TNPM pustules in utero [160]. A smear of pustular contents yields abundant neutrophils with rare eosinophils and Gram's stain is negative (ruling out staphylococcus bullous impetigo) [6]. Biopsy is rarely necessary for diagnosis but shows corneal and subcorneal pustules with abundant neutrophils and rare eosinophils, while hyperpigmented macular lesions show basal layer hyperpigmentation and hyperkeratosis [6, 159]. The main differential diagnosis is erythema toxicum neonatorum (ETN), which can be differentiated by the presence of erythema surrounding the pustules, although this may be difficult to recognize in darkly pigmented infants [6]. In ETN, eosinophils predominate on smear and histopathologic exams [6, 160].

Infantile Acropustulosis

Infantile acropustulosis (IA) is a rare disorder that primarily affects black males 2–10 months of age [6, 159]. It presents with recurrent 1–3 mm acral vesicles and pustules that appear in crops, occasionally at non-acral sites [160]. Lesions are intensely pruritic, last for 1–2 weeks, and then spontaneously remit until the next crop of lesions appears. IA eventually resolves within 2–3 years [159]. It is frequently misdiagnosed as scabies; other differential diagnoses include dyshidrotic eczema, hand–foot–mouth disease, transient neonatal pustular melanosis, and erythema toxicum neonatorum [159, 161]. If biopsied, histopathologic exam reveals subcorneal and intraepidermal neutrophilic pustules, mild perivascular lymphohistiocytic infiltrate and rare eosinophils [160]. Treatment includes topical steroids and antihistamines for intense pruritus [160, 162]. IA has been successfully treated with dapsone 2 mg/kg/day with tapering over the following few months to prevent relapse [6, 162].

Pediatric Malignancies

Skin cancers are rare in children. Most of the malignancies in darker skinned patients are described in the literature as case reports. Hypopigmented mycosis fungoides (MF) is more prevalent in patients of color. It has a chronic course that rarely progresses and responds well to topical steroids and phototherapy; however,

recurrence after successful treatment is often seen [163]. Malignant melanoma has been reported within nevus of Ito [27] and nevus of Ota [25]. Malignant blue nevus arising within giant blue nevus in an infant has been described [28]. Childhood subungual malignant melanoma in situ is rare; no case of invasive subungual melanoma has been reported in children [47]. It has been argued that these reported cases of childhood subungual malignant melanoma in situ may in fact have been atypical melanocytic nevi because pediatric pigmented lesions in general display more atypia compared to lesions in adults [48].

Conclusion and Psychosocial Considerations

For the pediatric patient of color, skin conditions that alter pigmentation primarily, such as vitiligo or albinism [164, 165] and birthmarks (Figs. 17.4 and 17.5), or secondary to inflammation, as in atopic dermatitis, psoriasis, or ichthyosis, can significantly impair quality of life [165]. Devastating effects have been recorded cross-culturally and interracially. Children with visible differences are vulnerable to discrimination and bullying [166]. They often face other social challenges involving peer relationships during games and sports, as well as reduced academic performance [165, 167, 168]. These incidents influence future social engagement; fear of reliving a traumatizing event sometimes results in "safety-seeking behaviors" such as concealing the affected area with clothing or camouflage make-up, and avoiding activities that require skin exposure, such as swimming [169]. In addition, these conditions have an impact upon confidence, self-consciousness, shyness, and general happiness [165, 167, 168, 170, 171]. If prolonged, negative social and psychological experiences may precipitate mental illness; Picardi et al. found that 25% of patients with diverse skin conditions experience significant psychological distress and psychiatric morbidity [172]. While some children and young adults with skin conditions are remarkably confident and resilient, others may experience depressive symptoms, true clinical depression, anxiety, and suicidal ideation [165, 168, 173–178]. It is important to keep in mind that a clinician's objective assessment of a patient's skin condition does not correlate with the patient's self-perception and distress [179].

In children with atopic dermatitis there is a direct relationship between dermatologic disease severity and psychological disturbances [176]. Maladaptive effects may be due to a combination of the physical lesions, the signature pruritus, and secondary dyspigmentation. In some cultures, postinflammatory hyperpigmentation on the neck is sometimes called "atopic dirty neck," which adds the misperception of poor hygiene to the already challenging social experience of this condition [180]. Chronic pruritus can also cause fussiness, irritability, and significant problems with sleep and concentration [176, 181].

Fig. 17.4 Blaschkoid epidermal nevus

Appearance can represent one's public representation of his or her personal self in society, and it is often heuristically linked to membership within social and cultural groups [182]. This association creates another challenge for children of color: among African Americans with vitiligo, the hypopigmentation can be misinterpreted as "turning white" [183]. Vitiligo and albinism can thus threaten racial identity and cultural membership.

Psychiatric comorbidities can complicate dermatologic disease when each condition exacerbates the other [178]. Furthermore, parental coping, mental health, and skin caretaker ability are critical in pediatric dermatology [184, 185]. Thus, multidisciplinary healthcare initiatives and patient- and family-centered interventions

Fig. 17.5 Extensive organoid nevus

that reduce psychosocial stress, increase self-acceptance, and strengthen protective relationships with peers and family have the potential to significantly improve health outcomes.

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