

Common Skin Diseases in Pediatric Practice

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Abstract

Skin disease is common cause of outdoor visits in pediatric population. In India, infections and infestations are the most common skin problems. Many of these diseases could be treated by pediatrician itself. Basic knowledge of these conditions, differential diagnosis and management is necessary to avoid delay in treatment and associated complications.

Keywords: Pediatric skin conditions, OPD skin conditions

Introduction

Skin disease is common cause of outdoor visits in pediatric population. In India, infections and infestations are the most common skin problems¹⁻³. Many of these diseases could be treated by pediatrician itself⁵. Basic knowledge of these conditions, differential diagnosis and management is necessary to avoid delay in treatment and associated complications.

Accurate diagnosis of cutaneous disease requires careful inspection, evaluation, and some knowledge of dermatologic terminology and morphology to develop a prioritized differential diagnosis.⁴ Cutaneous lesion could be primary or secondary.

Primary Lesions

The term primary refers to the most representative of disease. It might not necessarily be the earliest lesion. Primary lesion could be

Macule: flat, circumscribed change of skin, of size <1 cm.

Papule is circumscribed, nonvesicular, nonpustular, elevated lesion measuring less than 1 cm diameter.

Patch: Flat, circumscribed lesion with color change that is >1 cm in size.

Plaque: Broad, elevated, disk-shaped lesion that occupies an area of >1 cm.

Nodule: Circumscribed, elevated, usually solid lesion that measures 0.5 to 2 cm in diameter. It involves the dermis and may extend into the subcutaneous tissue with its greatest mass below the surface of the skin.

Wheal: Distinctive type of elevated lesion characterized by local, superficial, transient edema.

Vesicle: Sharply circumscribed, elevated, fluid containing lesion that measures ≤1cm in diameter.

Bulla: Larger, circumscribed, elevated, fluid containing lesion that measures >1 cm in diameter.

Pustule: Circumscribed elevation <1 cm in diameter containing purulent exudate.

Abscess: Circumscribed, elevated lesion >1 cm in diameter, often with a deeper component and filled with purulent material.

Secondary Lesions

Secondary lesions represent evolutionary changes that occur later in the course of disease.

Crust: Dried remains of serum, blood, pus, or exudate overlying areas of lost or damaged epidermis. Crust is yellow when formed by dried serum, green or yellowish - green when formed by purulent exudate, and dark red or brown when formed by bloody exudative serum.

Scale: Formed by an accumulation of compact desquamating layers of stratum corneum as a result of abnormal keratinization and exfoliation of cornified keratinocytes.

Fissure: Dry or moist, linear, often painful cleavage in the cutaneous surface that results from marked drying and long-standing inflammation, thickening, and loss of elasticity of the integument

Erosion: Moist, slightly depressed vesicular or bullous lesions in which part or all of the epidermis has been lost. Because erosions do not extend into the underlying dermis or subcutaneous tissue, healing occurs without subsequent scar formation.

Excoriation: Traumatized or abraded (usually self-induced) superficial loss of skin caused by scratching, rubbing, or scrubbing of the cutaneous surface.

Ulcer: Necrosis of the epidermis and part or all of the dermis and/or the underlying subcutaneous tissue.

Atrophy: Cutaneous changes that result in depression of the epidermis, dermis, or both. Epidermal atrophy is characterized by thin, almost translucent epidermis, a loss of the normal skin markings, and wrinkling when subjected to lateral pressure or pinching of the affected area. In dermal atrophy the skin is depressed.

Lichenification: Thickening of the epidermis with associated exaggeration of skin markings which occurs due to chronic scratching or rubbing of a pruritic lesion.

Scar: A permanent fibrotic skin change that develops after damage to the dermis.

The skin of Newborn full-term infant is normally soft and smooth. Desquamation of neonatal skin occurs 24 to 36 hours after delivery. Desquamation at birth is an abnormal phenomenon and is indicative of post maturity, intrauterine anoxia, or congenital ichthyosis.

Acrocyanosis is bluish discoloration of extremities, which is due to increased tone of peripheral arterioles, leading to vasospasm, secondary dilation, and pooling of blood in the venous plexuses, resulting in cyanotic appearance to the involved areas of the skin.

Cutis marmorata is reticulated bluish mottling of the skin seen on the trunk and extremities of infants and young children. This phenomenon, a physiologic response to chilling with resultant dilation of capillaries and small venules, usually disappears as the infant is rewarmed. If mottling does not resolve with warming, other conditions should be considered, including shock, sepsis and hypothyroidism.⁸

Sclerema neonatorum is a diffuse, rapidly spreading, wax-like hardening of the skin and subcutaneous tissue that occurs in premature or debilitated infants during the first few weeks of life. The disorder, usually associated with a serious underlying condition such as sepsis other infection, congenital heart disease, respiratory distress, diarrhea, or dehydration.

Differentiation of the epidermis and its appendages, particularly in the premature infant, is often incomplete at birth. As a result, a high incidence of sweat-retention phenomena may be seen in the newborn. They are characterized by a vesicular eruption with subsequent maceration and obstruction of the eccrine ducts. Miliaria crystalline (sudamina), which consists of clear superficial pinpoint vesicles without an inflammatory areola; Miliaria rubra (prickly heat), represents deeper level of sweat gland obstruction and characterized by small discrete erythematous papules, vesicles, or papulovesicles. The incidence of miliaria is greatest in first few weeks of life due to relative immaturity of the eccrine ducts, which favors poral closure and sweat retention. Therapy is directed toward avoidance of excessive heat and humidity. Use of light-weight cotton clothing and cool baths is only treatment required.⁹ Application of emollients must be avoided, especially in warm, humid climates or in the winter when infants are bundled under heavy clothing

MILIA: small retention cysts commonly occur on the face of newborns as tiny 1-to2-mm pearly white or yellow papules. Particularly prominent on the cheeks, nose, chin, and forehead, they may be few or numerous and are often grouped. They usually disappear spontaneously during the first 3 to 4 weeks of life and accordingly require no therapy.

Erythema Toxicum Neonatorum

Erythema toxicum neonatorum (ETN), is an idiopathic, asymptomatic, benign, self-limiting, cutaneous eruption in full-term newborns. Lesions consist of erythematous macules, papules, and pustules,

or a combination of these, and may occur anywhere on the body, especially the forehead, face, trunk, and extremities, sparing palms and soles may be explained by the absence of pilosebaceous follicles in these areas. Cytologic examination of a pustule smear that with Wright or Giemsa staining reveals a predominance of eosinophils.⁹

Seborrheic Dermatitis: Is a common, self-limiting condition of the scalp, face, ears, trunk, and intertriginous areas characterized by greasy scaling, redness, fissuring, and occasional weeping. It usually presents in infants with a scaly dermatitis of scalp termed cradle cap which may spread over face, including the forehead, ears, eyebrows, and nose. Its predilection for areas of high sebaceous gland density and the correlation of activity with increased hormonal levels during the first year of life¹⁰ and adolescence suggests a relation to sebum and sebaceous glands. Seborrheic dermatitis of adolescence and adulthood has been attributed to *Pityrosporum ovale*.¹¹ Management of seborrheic dermatitis includes topical antifungal (ketoconazole), topical steroid and keratolytic.¹²

Diaper Dermatitis: Occurs due to combination of factors, most important of which is prolonged contact with urine and feces, skin maceration, and, in many cases, secondary infection with bacteria or *Candida Albicans*.¹³ Three most common types of diaper dermatitis are chafing dermatitis, irritant contact dermatitis, and diaper candidiasis.

Chafing dermatitis: Generally present on areas where friction is the most pronounced (inner surfaces of the thighs, the genitalia, buttocks, and the abdomen). Eruption presents as mild redness and scaling and tends to wax and wane quickly. This type responds quickly to frequent diaper changes and good hygiene.

Irritant contact diaper dermatitis: Usually involves the convex surfaces of the buttocks, the vulva, the perineal area, the lower abdomen, and the proximal thighs, with sparing of the intertriginous creases. The disorder may be attributable to contact with proteolytic enzymes in stool and irritant chemicals such as soaps, detergents, and topical preparations. Other significant factors appear to be excessive heat, moisture, and sweat retention associated with the warm local environment produced by the diaper.

Candidal diaper dermatitis: Presents as a widespread, beefy-red erythema on buttocks, lower abdomen, and inner aspects of the thighs. Characteristic features include a raised edge, sharp marginalization with white scales at the border, and pinpoint pustulovesicular satellite lesions (the diagnostic hallmark). Infants harbor *C. Albicans* in the lower intestine, and it is from

this focus that infected feces present the primary source for candidal diaper eruptions

Seborrheic dermatitis of the diaper area may be recognized by the characteristic salmon-colored, greasy plaques with a yellowish scale and a predilection for intertriginous areas. Coincident involvement of the scalp, face, neck, and postauricular and flexural areas helps to establish the diagnosis.

Pityriasis Alba is a common cutaneous disorder characterized by asymptomatic hypopigmented patches, usually on the face, neck, upper trunk, and proximal extremities. Individual lesions vary from 1 cm or more in diameter and may show a fine scale. Effective moisturization during drier months may help prevent recurrence of the pityriasis alba in subsequent summers.

Intertrigo is a superficial inflammatory dermatitis that occurs in areas where the skin is in apposition. As a result of friction, heat, and moisture, the affected areas become intensely erythematous in a well-demarcated pattern, macerated, and often secondarily infected by bacteria or *Candida*, or in adolescents by dermatophytes **Mongolian spots** are flat, deep brown to slate gray or blue-black, often poorly circumscribed, large macular lesions generally located over the lumbosacral areas, buttocks, and occasionally the lower limbs, back, flanks, and shoulders of normal infants. Mongolian spots are present at birth, tend to fade during the first 2 to 3 years of life, and only occasionally persist into adulthood.

Pyoderma is a superficial bacterial infection of the skin, which includes impetigo, folliculitis, furuncle, ecthyma and several others.

Impetigo is the most frequently encountered bacterial skin infection, which is caused by *Staphylococcus aureus* and *Streptococcus pyogenes*^{6,7}. It is common in infants and children. It occurs in two clinical forms, the non-bullous impetigo or impetigo contagiosa and bullous impetigo. Although impetigo contagiosa can occur on any body surface, the exposed parts, especially face and the extremities are frequently affected. It begins with a 1- to 2-mm erythematous papule or pustule that soon develops into thin-roofed vesicle or bulla surrounded by a narrow rim of erythema. The vesicle ruptures easily with release of a thin, cloudy, yellow fluid that subsequently dries, forming a honey-colored crust, the hallmark of nonbullous impetigo.

Bullous Impetigo occurs on the face and moist intertriginous areas. Clinically, it presents as thin walled, flaccid, subcorneal blister that ruptures rapidly leaving behind an erythematous and moist erosion, surrounded by a pathognomonic peripheral remnant of blister roof. Impetigo may also develop on pre-existing atopic eczema or scabies lesions (secondary impetiginization).

Folliculitis is a superficial staphylococcal infection of the follicular ostia, which involves scalp, buttocks and perineum. It presents as asymptomatic, superficial erythematous papules and pustules with perilesional

erythema centered on the hair follicles. Unlike in folliculitis, **furuncle** is a deep seated infection involving the hairfollicle and its surrounding soft tissue, forming a tender nodule or abscess. Children with folliculitis and furuncle have no constitutional features.

Treatment of pyodermas: For localized superficial infections, topical mupirocin or fusidic acid can be used. For multiple lesions and deeper infections, systemic antibiotics are recommended for a period of 7–10 d. antibiotics used are cloxacillin/ dicloxacillin 50–100 mg/kg/d 4 times a day; cephalexin 25–50 mg/kg/d 3–4 times a day; amoxicillin/ clavulanic acid (40 mg of amoxicillin/ kg/d) 2–3 times a day. Failure to respond to antibiotic treatment may suggest methicillin resistant organisms, which are treated with clindamycin, vancomycin, or trimethoprim/ sulfamethoxazole^{6,7}.

SSSS is a term used to describe a blistering skin disease caused by the epidermolytic (or exfoliative) toxin (ET)-producing *S. aureus*. The pathogenesis of SSSS relates to the production of ETs, of which there are two serotypes affecting humans, ETA and ETB. The pathogenic mechanisms of ETA and ETB have been clearly elucidated, and they have been shown to target desmoglein-1, a cell–cell adhesion molecule found in desmosomes of the superficial epidermis. SSSS generally begins with localized infection of the conjunctivae, nares, perioral region, perineum, or umbilicus. Separation of perioral crusts often leaves behind radial fissures around the mouth, resulting in the characteristic facial appearance of SSSS. Other infections that may serve as the initial nidus for SSSS include pneumonia, septic arthritis, endocarditis, or pyomyositis. Fever, malaise, lethargy, irritability, and poor feeding subsequently develop, and the generalized eruption begins. The rash is characterized by erythema that progresses to large, superficial fragile blisters that rupture easily, leaving behind denuded, desquamating, erythematous, and tender skin. The eruption is most marked in flexural creases but may involve the entire surface area of skin. The Nikolsky sign (progression of the blister cleavage plane induced by gentle pressure on the edge of the bulla) is positive. The most helpful distinguishing feature of TEN is mucosal involvement, including of the mouth, conjunctivae, trachea, and genital mucosa, which is lacking in SSSS. It must be remembered that the majority of blisters in SSSS are sterile, because they are caused by the hematogenous dissemination of the bacterial toxin and not the bacteria itself. Treatment of SSSS is directed at the eradication of toxin-producing staphylococci, thus terminating toxin production.

A penicillinase resistant penicillin, first- or second-generation cephalosporin, or clindamycin are all appropriate initial choices, with modification based on sensitivity testing.

Scabies is a common infestation caused by the mite *Sarcoptes scabiei* variety *hominis*. It is transmitted by

close direct contact. The earliest symptom of scabies is nocturnal pruritus, which may manifest as extreme irritability in infants. Face, palms and soles are particularly affected in younger children, while waists, wrists, ankles, interdigital spaces, penis and areola are commonly affected in older children. A variety of primary and secondary lesions such as papules, nodules, burrows and vesiculopustules occur in scabies. Of these, burrows and nodules are the specific lesions. Burrows are located just beneath the stratum corneum and consist of linear or serpiginous whitish lesions with a black dot at the end, which represents the location of female mites. Nodular scabies is characterized by severely pruritic reddish brown nodules commonly seen on the trunk, axillae and genitalia. It may persist for several months even after resolution of scabies. Diagnosis is usually clinical.

Head lice usually affect children between 3 and 12 year via direct head to head contact. Itching in scalp is the primary symptom and there may be redness, papules, excoriated lesions and secondary bacterial infections. Occipital and cervical lymphadenopathy may be present. Diagnosis is confirmed by the presence of nits or lice in the scalp and hair. Nits are grey white, 1 mm sized, ovoid eggs which are firmly attached to the hair shaft. Nits can be moved along the hair shaft in contrast to scales (dandruff) which are yellowish and irregular in shape and can easily be removed in any direction¹⁴. The treatment of choice is 1 % permethrin cream, which should be applied to mildly damp hair, followed by rinsing after 10 min. A repeat application is usually recommended after 1 or 2 week. Oral Ivermectin can be used in children older than 5 year^{14,15}. Like in scabies all household or close contacts should be treated

Tinea capitis, the most common dermatophytic infection, is usually seen in pre-pubertal girls (3–7 y). More than 90% of the infections are caused by *Trichophyton tonsurans*^{16, 17}. *T. Capitis* (grey patch) presents with single or multiple patches of alopecia with inflammatory signs of papules, pustules, crusting and scaling. Hair in these patches are easily pluckable. Black dot *T. capitis* is characterized by patches of alopecia with tiny black dots within them. The black dots represent the broken ends of endothrix infected (*T. violaceum*) hair shaft. Kerion, the severe inflammatory type, shows alopecia with boggy swelling and pustules or exudation.

Tinea versicolor is a common skin condition which occurs in older children and young adolescents and is caused by *Malassezia furfur*. It is frequently seen in tropical countries with high humidity. It presents as numerous hypopigmented scaly macules on the upper chest, back and proximal upper extremities. Potassium hydroxide test from the skin scraping demonstrates short non-branching hyphae and yeast cells. The treatment options available includes topical selenium sulfide 2.5% lotion, imidazoles, ciclopirox olamine and sulfur preparations. Oral ketoconazole or fluconazole

400 mg can also be used. Management consists of educating the parents regarding the benign nature of the condition.

Atopic dermatitis or eczema is a chronic relapsing inflammatory skin disorder. The hallmark of atopic dermatitis is dry skin (xerosis) and pruritus. It usually starts in the first few weeks or months of life. The clinical phase of atopic dermatitis is divided into 3 groups, based on the onset and distribution of the lesions; infantile; childhood and adult phases^{18, 19}. In the infantile phase, the dermatitis usually begins on the face and then may spread to trunk. The affected infants will be irritable or scratching due to intense pruritus. It is characterized by symmetrical erythematous papules, vesicles, edema, exudation/oozing and crusting. The extensor aspect of the extremities are affected around 8 mo of age when they start crawling. Infantile atopic dermatitis usually spares the diaper area and groin. In the childhood phase, the lesions become chronic and lichenified with a predilection for the flexural areas (cubital and popliteal fossae). In the face, peri-oral and per-orbital areas may also show chronic dermatitis and lichenification. Pigmentary changes may be found in children. Management includes topical emollients and topical corticosteroids.²⁰ Antihistaminics are often used for management of pruritus.

Exanthematous Diseases

Varicella (Chickenpox): Varicella-zoster virus (VZV) is a member of the herpesvirus family, and the causative agent of both varicella (chickenpox) and herpes zoster. Once acquired, VZV becomes established in sensory ganglia in a latent form with intermittent reactivation in a dermatomal distribution, resulting in herpes zoster. Patients are considered contagious until at least 5 days after onset of the rash or until all existing lesions are dry and crusted. Primary varicella begins with a prodrome of fever, chills, malaise, headache, arthralgia, and myalgia. After 24 to 48 hours, the earliest skin lesions become evident, initially as red macules or papules that progresses rapidly to a vesicular phase. The fully developed lesion has been described as “dewdrop on a rose petal” Varicella lesions present initially on the scalp, face, or trunk and then spread to the extremities. Older lesions crust over, and new lesions continue to develop, resulting in the pathognomonic finding of lesions in various stages being present at the same time. The lesions of varicella heal with hypopigmentation and scarring, especially at sites of the initial lesions routine use of antiviral agents for varicella in otherwise-healthy children is not recommended.

Oral antiviral therapy should be considered, however, for healthy individuals who are at risk for moderate or severe disease, such as those over 12 years of age (especially unvaccinated), those with chronic skin or lung disorders, and those receiving corticosteroids (short, intermittent, or aerosolized

courses). When used in the treatment of varicella in otherwise healthy patients, antiviral therapy should be started within 24 hours of the appearance of the rash.

Measles is caused by a single-stranded ribonucleic acid (RNA) virus in the family Paramyxoviridae. Infection begins in the nasopharyngeal epithelium and less commonly through the conjunctivae. Transmission of measles is primarily via respiratory droplets and less commonly by small particle aerosols. The incubation period is around 10 to 14 days. From the initial site of infection, the virus enters the lymph nodes and lymphatics and multiplies within the reticuloendothelial system with a subsequent viremia. The virus is then disseminated to multiple lymphoid tissues and other organs, including the skin, liver, and gastrointestinal tract. Measles immunity includes cell-mediated, humoral, and mucosal responses. Measles antibodies are responsible for protection from future infection or reinfection. Measles classically presents with fever and cough, coryza, and conjunctivitis. The pathognomonic enanthem, Koplik spots, usually occurs during this prodromal period and presents with punctuate, gray-white to erythematous papules distributed on the buccal mucosa. It begins on the face especially the forehead, hairline, and behind the ears, and spreads downward onto the trunk and extremities. The lesions are erythematous to purple-red macules and papules that may become confluent and fade in the same order as their appearance, leaving behind coppery macules and desquamation.

To conclude, these few diseases make up most of outdoor visits to pediatrician. Knowledge of these few pediatric skin condition can reduce unnecessary referrals and associated anxiety

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