Joyce M.C. Teng
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Therapy in Pediatric Dermatology

Management of Pediatric Skin Disease

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Preface

We are very excited about the publication of the first edition of Therapy in Pediatric Dermatology: Management of Pediatric Skin Disease. Given limited number of high quality clinical trials available in pediatric dermatology, we aim to address the need for comprehensive review of therapeutic options that are known to be efficacious in management of cutaneous diseases in children. This book summarizes evidence-based literature on clinical responses among pediatric patients, including age-appropriate management strategies. It is our wish to create a succinct, user-friendly, and up-to-date therapeutic dermatologic textbook for physicians who care for children with skin disorders.

A significant percentage of the treatments used in pediatric dermatology are currently off-label. Large scale, well-designed clinical trials are lacking for many of the dermatologic treatments that we recommend on a regular basis. For each skin condition discussed in this book, the investigative and treatment recommendations provided are based on extensive review of the literature. The treatment algorithms are provided using evidence scale A to E as recommended by the Center for Evidence-Based Medicine.

There have been extraordinary developments in understanding of the genetics and pathogenesis of many cutaneous disorders during the past decade. Novel therapeutic options and repurpose of old drugs have been investigated for the management of some of the most challenging skin disorders. The quantum of medical information that physicians receive has grown exponentially and is becoming overwhelming. It is our goal that this book will provide unbiased yet concise information that is valuable to practitioners who manage pediatric patients in their practices.

It has been a tremendous opportunity for Joyce, Ann, and Latanya to work with so many experts and trainees in the field. We thank all our contributors who have helped us create this book. We are indebted to our families, colleagues, and administrative staff. We offer special thanks to Kris Arao, our administrative assistant at Stanford, and Cheryl Winters-Tetreau, developmental editor at Springer, who worked tirelessly with many authors and saw the completion of this project. Our deepest thank you goes out to all the challenging patients we see on a daily basis in our busy clinical practices and who serve as the inspiration for the creation of this book. We hope this textbook proves to be an important body of work and resourceful to all who desire a greater understanding of dermatologic therapies and their uses in the pediatric population.

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Abbreviations

AA	Alopecia areata
AAP	American Academy of Pediatrics
ACD	Allergic contact dermatitis
ACE	Angiotensin converting enzyme
ACIP	Advisory Committee on Immunization Practices
ACR	American College of Rheumatology
AD	Atopic dermatitis
AE	Acrodermatitis enteropathica
ALA	Aminolevulinic acid
ALCL	Anaplastic large-cell lymphoma
AMT	Anxiety management training
AN	Anorexia nervosa
ANA	Antinuclear antibodies
AP	Actinic prurigo
APEC	Asymmetric periflexural exanthem of childhood
APKH	Acquired progressive kinking of the hair
aPL	Antiphospholipid antibodies
APS	Antiphospholipid antibody syndrome
ARCI	Autosomal recessive congenital ichthyosis
ARCL	Autosomal recessive cutis laxa
AT	Alopecia totalis
AU	Alopecia universalis
AVM	Arteriovenous malformations
BCCs	Basal cell carcinomas
BCH	Benign cephalic histiocytosis
BCNS	Basal cell nevus syndrome
BD	Behçet's disease
BDD	Blistering distal dactylitis
BDD	Body dysmorphic disorder
BMI	Body mass index
BN	Bulimia nervosa
BP	Bullous pemphigoid
BSA	Body surface area
BSLE	Bullous systemic lupus erythematosus
CAPS	Cryopyrin associated periodic syndrome
CARRA	Childhood Arthritis and Rheumatology Research Alliance
CBC	Complete blood count
CBDC	Chronic bullous disease of childhood
CBT	Cognitive behavioral therapy
CD	Crohn's disease
CDC	Centers for Disease Control and Prevention

CEP	Congenital erythropoietic porphyria
CGCT	Congenital granular cell tumor
CGD	Chronic granulomatous disease
CHS	Chediak-Higashi syndrome
CIE	Congenital ichthyosiform erythroderma
CINCA/NOMID	Chronic infantile cutaneous articular syndrome/neonatal-onset multi-systemic inflammatory disease
CL	Cutaneous leishmaniasis
CL	Cutis laxa
CM	Capillary malformations
CMCC	Chronic mucocutaneous candidiasis
CMN	Congenital melanocytic nevi
CMP	Complete metabolic panel
CMV	Cytomegalovirus
CNS	Central nervous system
CoA	Coenzyme A
CSD	Cat scratch disease
CSF	Cerebrospinal fluid
CT	Computed tomography
CTA	Computed tomography angiography
CTCL	Cutaneous T-cell lymphomas
CXR	Chest X-ray
DEB	Dystrophic epidermolysis bullosa
DEJ	Dermo-epidermal junction
DEXA	Dual-energy X-ray absorptiometry
DFA	Direct fluorescent antibody
DFSP	Dermatofibrosarcoma protuberans
DGI	Disseminated gonococcal infections
DH	Dermatitis herpetiformis
DI	Delusional infestation
DIC	Disseminated intravascular coagulation
DIF	Direct immunofluorescence
DIHS	Drug-induced hypersensitivity syndrome
DISH	Diffuse idiopathic skeletal hyperostosis
DMARD(s)	Disease-modifying antirheumatic drug(s)
DOC	Disorders of cornification
DOT	Direct observed therapy
DPWH	Diffuse partial woolly hair
dsDNA	Double-stranded DNA
EAC	Erythema annulare centrifugum
EB	Epidermolysis bullosa
EBA	Epidermolysis bullosa acquisita
EBS	Epidermolysis bullosa simplex
EBV	Epstein-Barr virus
ECP	Extracorporeal photopheresis
EDS	Ehlers-Danlos syndrome
EDSF	Ectodermal dysplasia with skin fragility
EDV	Epidermodysplasia verruciformis
EF	Eosinophilic fasciitis
EFA	Essential fatty acids
EI	Epidermolytic ichthyosis
EIAs	Enzyme immunoassays
EKV	Erythrokeratodermia variabilis

ELISA	Enzyme-linked immunosorbent assay
ELVIS	Enzyme-linked virus inducible system
EM	Electron microscopy
EM	Erythema migrans
EN	Erythema nodosum
ENS	Epidermal nevus syndrome
EOS	Early-onset sarcoidosis
EPP	Erythropoietic protoporphyria
EPS	Elastosis perforans serpiginosa
ERA	Enthesitis-related arthritis
ERP	Exposure and response prevention
ERT	Enzyme replacement therapy
ESR	Erythrocyte sedimentation rate
EVHC	Eruptive vellus hair cysts
FDE	Fixed drug eruption
FDH	Focal dermal hypoplasia
FH	Familial hypercholesterolemia
FMF	Familial Mediterranean fever
FSH	Follicle-stimulating hormone
FTI(s)	Farnesyl transferase inhibitor(s)
FWH	Familial woolly hair
GA	Glycolic acid
GA	Granuloma annulare
GAG	Glycosaminoglycan
GCS	Gianotti-Crosti syndrome
GEH	Generalized eruptive histiocytoma
GGA	Generalized GA
GI	Gastrointestinal
GVHD	Graft-versus-host disease
HBV	Hepatitis B virus
HCT	Hematopoietic cell transplant
HEP	Hepatoerythropoietic porphyria
HFMD	Hand-foot-and-mouth disease
HGA	Human granulocytic anaplasmosis
HGPS	Hutchinson-Gilford Progeria Syndrome
HHT	Hereditary hemorrhagic telangiectasia
HIV	Human immunodeficiency virus
HME	Human monocytic ehrlichiosis
HoFH	Homozygous familial hypercholesterolemia
HPLC	High-pressure liquid chromatography
HPV	Human papillomavirus
HRT	Habit reversal therapy
HSCT	Hematopoietic stem cell transplantation
HSP	Henoch-Schonlein purpura
HSV	Herpes simplex virus
HV	Hydroa vacciniforme
HWH	Hereditary woolly hair
IBD	Irritable bowel disease
ICD	Irritant contact dermatitis
IFA	Indirect fluorescence assay
IFAG	Idiopathic facial aseptic granuloma
IFM	Immunofluorescence mapping
IgE	Immunoglobulin E

ILAR	International League of Associations for Rheumatology
ILD	Interstitial lung disease
ILVEN	Inflammatory linear verrucous epidermal nevus
IM	Infantile myofibromatosis
IM	Infectious mononucleosis
IPL	Intense pulsed light
ISD	Infantile seborrheic dermatitis
IVIG	Intravenous immunoglobulins
JDM	Juvenile dermatomyositis
JEB	Junctional epidermolysis bullosa
JIA	Juvenile idiopathic arthritis
JPD	Juvenile plantar dermatosis
JSE	Juvenile spring eruption
jSSc	Juvenile systemic sclerosis
JXG	Juvenile xanthogranuloma
KHE	Kaposiform hemangioendotheliomas
KID	Keratitis-ichthyosis-deafness [syndrome]
KLC	Keratosis lichenoides chronic
KMP	Kasabach Merritt Phenomenon
KMS	Kasabach-Merritt syndrome
LA	Lactic acid
LAD	Leukocyte adhesion deficiencies
LAHS	Loose anagen hair syndrome
LCH	Langerhans cell histiocytosis
LED	Light-emitting diode
LDL	Low-density lipoprotein
LEN	Linear (verrucous) epidermal nevi
LFT(s)	Liver function test(s)
LH	Lutenizing hormone
LI	Lamellar ichthyosis
LM	Lymphatic malformations
LN	Lichen nitidus
LP	Lichen planus
LS	Lichen sclerosus
LSC	Lichen simplex chronicus
LUMBAR	Lipoma, urogenital anomalies/ulceration, myelopathy, bony deformities, anorectal malformations/arterial anomalies, and renal anomalies syndrome
LYP	Lymphomatoid papulosis
MAP	Mitogen-activated protein
MATP	Membrane-associated transporter protein
MCL	Mucocutaneous leishmaniasis
MCRH	Multicentric reticulohistiocytosis
MCTD	Mixed connective tissue disease
MEN 1	Multiple endocrine neoplasia syndrome 1
MF	Mycosis fungoides
MMA	Methylmalonic acidemia
MMRV	Measles, mumps, rubella, and varicella [vaccine]
MMS	Mohs micrographic surgery
MPSs	Mucopolysaccharidoses
MRA	Magnetic resonance angiography
MRI	Magnetic resonance imaging
MRS	Melkersson-Rosenthal syndrome

MRSA	Methicillin-resistant S. aureus
MSSA	Methicillin-sensitive S. aureus
MSUD	Maple syrup urine disease
MUD	Minimal urticaria dose
NAAT	Nucleic acid amplification testing
NCM	Neurocutaneous melanosis
NF	Neurofibromatosis
NS	Netherton syndrome
NS	Nevus sebaceous
NSAIDs	Nonsteroidal anti-inflammatory drugs
NTM	Non-tuberculous mycobacteria
NXG	Necrobiotic xanthogranuloma
OCA	Oculocutaneous albinism
OI	Osteogenesis imperfecta
PA	Pityriasis alba
PAH	Phenylalanine hydroxylase
PALCL	Primary cutaneous anaplastic large cell lymphoma
PAN	Polyarteritis nodosa
PAPA	***P***yogenic ***a***rthritis, ***p***yoderma gangrenosum, and ***a***cne conglobata syndrome
PAPASH	Sterile ***p***yogenic ***a***rthritis, ***p***yoderma gangrenosum, ***a***cne and ***s***uppurative ***h***idradenitis syndrome
PBMC	Peripheral blood mononuclear cells
PC	Pachyonychia congenita
PCOS	Polycystic ovarian syndrome
PCR	Polymerase chain reaction
PCT	Porphyria cutanea tarda
PDGF	Platelet derived growth factor
PDL	Pulsed dye laser
PDT	Photodynamic therapy
PF	Pemphigus foliaceus
PG	Pyoderma gangrenosum
PHACE syndrome	Posterior fossa anomalies, Hemangioma, Arterial lesions, Cardiac abnormalities/coarctation of the aorta, Eye anomalies
Phe	Phenylalanine
PIPA	Post-inflammatory pigment alteration
PKU	Phenylketonuria
PL	Pityriasis lichenoides
PLC	Pityriasis lichenoides chronica
PLE	Polymorphous light eruption
PLEVA	Pityriasis lichenoids et varioliformis acuta
PMLE	Polymorphous light eruption
PNH	Progressive nodular histiocytoma
POTS	Postural orthostatic tachycardia syndrome
PP	Pseudoporphyria
PPD	Purified protein derivative [test]
PPGSS	Papular-purpuric gloves and socks syndrome
Ppi	Inorganic pyrophosphate
PPK	Palmoplantar keratoderma
PR	Pityriasis rosea
PRP	Pityriasis rubra pilaris
PSD	Perineal streptococcal dermatitis
PUVA	Psoralen plus UVA

PV	Pemphigus vulgaris
PXE	Pseudoxanthoma elasticum
RAMBA	Retinoic acid metabolism blocking agent
RCT(s)	Randomized, controlled clinical trial(s)
RDFC	Recurring digital fibroma of childhood
ReA	Reactive arthritis
RF	Rheumatoid factor
RMSF	Rocky mountain spotted fever
RP	Relapsing polychondritis
RT-PCR	Reverse transcription-PCR
SA	Salicylic acid
SAPHO	***S***ynovitis, ***a***cne, ***p***ustulosis, ***h***yperostosis, and ***o***steitis syndrome
SCC(s)	Squamous cell carcinoma(s)
SCID	Severe combined immunodeficiency
SGA	Subcutaneous nodules granuloma annulare
SHML	Sinus histiocytosis with massive lymphadenopathy
SJS	Stevens-Johnson syndrome
SLE	Systemic lupus erythematosus
SLICC	Systemic Lupus International Collaborating Clinics
SLS	Sjogren-Larsson syndrome
SPF	Sun protection factor
SPTCL	Subcutaneous panniculitis-like T-cell lymphoma
SSc	Systemic sclerosis
SSRIs	Selective serotonin reuptake inhibitors
SSSS	Staphylococcal scalded skin syndrome
SSTI	Skin and soft tissue infections
TA	Tufted angiomas
TB	Tuberculosis
TBCO	Tuberculosis cutis orificialis
TEN	Toxic epidermal necrolysis
TND	Twenty-nail dystrophy
TNF	Tumor necrosis factor
TSC	Tuberous sclerosis complex
TSS	Toxic shock syndrome
TTM	Trichotillomania
TVC	Tuberculosis verrucosa cutis
TWEL	Transepidermal water loss
Tyr	Tyrosine
UBOs	Unidentified bright objects
ULE	Unilateral laterothoracic exanthem
UV	Ultraviolet
VM	Venous malformations
VZV	Varicella zoster virus
WHN	Woolly hair nevus
WS	Werner syndrome
YAG (laser)	Yttrium aluminum garnet
XD	Xanthoma disseminatum
XLCL	X-linked cutis laxa
XLDPP	X-linked dominant protoporphyria

Contents

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Overview of Dermatologic Care in Children

1

Leslie Potter Lawley

Skin Care of Normal Newborn Skin

Clinical Features

- Full-term infants are born with skin at near normal pH and a natural barrier in the form of the vernix caseosa. A combination of shed epithelial cells, sebum, and lanugo hairs, the vernix caseosa appears as a chalky-white cheesy film on the skin surface of full-term infants. It is not present in post-term neonates, whose skin is drier, and may not have fully formed in a pre-term infant, depending on the age of gestation. The formation of the vernix starts around week 28 and peaks at 33–37 weeks' gestation. The vernix caseosa should not be removed, but allowed to naturally resolve, as it aids in temperature regulation, skin hydration, protection from bacteria, and wound healing. Fine desquamation of the skin develops around 2 days of life for term infants. In preterm infants, desquamation may not be apparent until 2–3 weeks of life, while post-term infants may be born with dry, peeling skin [3, 6].
- Over a period of weeks to months the acid level of stratum corneum increases to transform into the acid mantle. The acidic pH of the acid mantle provides a normal permeability barrier, enhancing cohesion and integrity of the stratum corneum. When cleansing newborns there is balance of removing bacteria, saliva, urine, feces, secretions, and soil while not over-drying the skin or harming the barrier created by the stratum corneum. As the infant ages and the acid mantle develops, skin care should not alter that natural barrier [3].
- Considerations for timing of the first bath for the newborn include stability of the neonate's vital signs. Bathing can lead to hypothermia, increased oxygen demands, and respiratory distress if performed too soon [3].
- At birth, the umbilical cord is clamped, leaving a stump of umbilical tissue. Over a period of weeks (up to 8) it naturally undergoes necrosis and detaches from the body. Typically the process takes an average of 2 weeks. As the umbilical cord stump undergoes this process, the infant is at risk of local secondary infections. Keeping the umbilical stump dry helps reduce this risk.

Management Strategies

- Protect the skin barrier while maintaining cleanliness.

Investigations Recommended [3]

Evaluation
Prior to first bath after birth, measure temperature, oxygen status, respiratory rate and heart rate and ensure these signs are stable for 2–4 h

Therapies [3, 6, 9, 11]

Bathe neonates every 2–3 days
Do not scrub to remove the vernix caseosa, but allow it to resolve naturally
Sponge bath until the umbilical cord naturally falls off
Immersion bathing is less harmful to temperature stability in newborns
Utilize baby washes with neutral pH, free of fragrance and dyes
In situations of home birth where hygiene sterile practices may be suboptimal, a one-time cleansing with chlorhexidine wipes as soon after birth as possible may reduce the mortality rate in low birth-weight babies (no significant change in mortality for normal weight neonates)
Apply petrolatum-based emollients (fragrance-free, preservative-free, dye-free) every 6–12 h as needed for dryness of the skin
Remove emollient from container with a clean tongue blade or other tool to prevent contaminating the moisturizer with bacteria

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Therapies: Daily Umbilical Cord Care [6]

Cleanse area with chlorhexidine. Then remove any excess soap to minimize local necrosis and absorption
Consider the powder form of chlorhexidine which allow for more drying
Avoid isopropyl alcohol or hexachlorophene
Fold diaper away from stump to keep dry

Principles of Pediatric Skin Care

Clinical Features

- When considering cleansing skin, there are no significant differences between infant and adult skin. Infants produce skin surface lipids similar to adults, with a varied ratio of sebum to keratinocyte lipids compared to adults. During childhood these lipids are decreased. Starting in pre-adolescence, increases in circulating hormones (adrenal androgens followed by gonadal androgens) stimulate production of sebum. In many adolescents the increased sebum leads to more oily complexion and hair, contributing to acne and seborrhea [7].
- Most studies on tolerance of skin care products have been in children with certain disease states such as atopic dermatitis. We lack evidence as to how often to bathe children with a normal skin barrier, what cleansers to use, how often to shampoo hair, and how often to use emollients. At this point we can only extrapolate information from studies on infants and children with skin barrier defects and atopic dermatitis to determine skin care for those children with normal skin. It must be considered that children with skin barrier defects have drier, more sensitive skin. Most soaps are made of animal or vegetable fats to remove dirt and oils and are alkaline in nature. Because the skin surface is acidic (acid mantle), synthetic detergents have been developed to protect the pH of the skin. The use of these neutral and even acidic pH cleansers are likely more important for children with skin barrier defects. Most normal children can tolerate the soap and shampoo their parents prefer to use [3, 7].
- Sun protection throughout childhood and adolescence is important. Studies indicate that at least 25–50 % of lifetime sun exposure occurs before age 18 years. In the first 2 years of life melanin production is limited, and skin may be more susceptible to UV damage. After 2 years, the skin of a child is similar to an adults' skin, however, the dermal papillae may be closer to the skin surface, leading to increased UV exposure to the basal layer. As children enter pre-adolescent ages they often become more independent in their skin care, so education of proper sun protection at that time is paramount to preventing damage from UV that can lead to skin cancers later in life [5].
- To date there is not evidence that any of the sun filters used in sunscreen harm children, however, controversy exists over the safety of organic protectors such as oxybenzone (benzophenone-3). Studies have shown absorption of oxybenzone with excretion in the urine, however, no known adverse effects have been seen. Allergic contact dermatitis can develop to organic blockers, leading to advice to avoid them, especially in children with skin barrier defects. Risk is minimized by using inorganic sunscreens (zinc oxide and titanium dioxide), where even the micronized nanoparticle forms do not absorb systemically through the skin, and no cytotoxic or genotoxic effects have been found. The combination of N,N-dimethyl-meta-toluamide (DEET) insect repellent and sunscreen leads to increased absorption of the DEET. For this reason, the combination sunscreen-insect repellent products should be avoided, as well as concomitant application of both products. In addition, sunscreen needs to be applied more frequently than insect repellent [5, 7].

Management Strategies

- No evidence-based guidelines are available for pediatric skin care. Goals are to maintain clean skin and protect against drying and sun damage.

Investigations Recommended

For diagnosis
None

Table 1.1 First line therapies [1, 2, 4, 5, 10]

Bathe every day to every other day in childhood using a mild cleanser. Bathe after heavy perspiration from sports or playing outdoors on hot days
Adolescents should bathe every day and wash their faces once to twice daily using a mild cleanser
Oily hair can be washed daily, whereas dry hair should be washed less frequently, up to once every 1–2 weeks
Apply moisturizers to dry skin and hair
Heavier emollients can be used in the winter (ointments and creams)
Lighter emollients should be used in hot and humid weather to prevent miliaria (lotions)

Table 1.1 (continued)

Follow good sun protection:
Cover up with clothing (lightweight long sleeves and pants, tightly woven fabric), hats (3-in. brim or greater), and sunglasses
Direct sun exposure should be avoided prior to 6 months age
For areas not covered by clothing, apply adequate amount of broad-spectrum sunscreen of SPF 30 or greater (1 oz/application for an adult)
Sunscreen should be reapplied every 2 h or sooner if needed
Use inorganic broad spectrum sun filters to minimize any risk of allergic reaction (zinc oxide and titanium dioxide)
Avoid sun exposure during the peak sun hours (11 am–4 pm), seek shade
Avoid combination sunscreen–insect repellent products

Premature Skin

Clinical Features

- The epidermis in premature infant skin is 55% thinner than term infant skin. The stratum corneum alone is only one cell thick compared to 15 cells in a term infant stratum corneum. The structures that anchor the epidermis cell together (desmosomes, anchoring filaments, anchoring fibrils, and hemidesmosomes) are smaller and fewer in number. This difference translates to increased fragility of the skin and increased permeability (increased transepidermal fluid loss, electrolyte imbalance, increased heat evaporation, and increased absorption of topically applied products). The skin matures quickly after birth, over a period of 2–3 weeks, to reach stages seen in term newborns, unless the skin is in a high-humidity environment or covered with occlusive materials, which can slow maturation [3, 8].

Management Strategies

- Replace fluids, prevent fluid loss, maintain electrolyte balance, maintain environmental temperature.

Investigations Recommended [3, 6]

For diagnosis
Measure sensible fluid losses (urine, feces) and insensible fluid losses
Correct caloric intake to support growth and energy losses
Monitor electrolytes

Table 1.2 First line therapies [3, 6]

Humidified incubator with radiant warmer
Semipermeable skin dressings
Bathe every 2–3 days with a pH neutral, fragrance-free, preservative-free, dye-free cleanser if weight over 1,000 g; if weight under 1,000 g, bathe in water alone

Therapies: Daily Umbilical Cord Care [6]

Cleanse area with chlorhexidine; then remove any excess soap to avoid local necrosis and absorption
Consider the powder form of chlorhexidine which allows for more drying
Do not use isopropyl alcohol or hexachlorophene
Fold diaper away from stump to keep dry

Therapies [3, 6, 8, 9]

Apply petrolatum-based moisturizers (fragrance-free, preservative-free, dye-free) every 12 h for the first 2–4 weeks, then as needed for dryness
Remove emollient from container with a clean tongue blade or other tool to prevent contaminating the moisturizer
Gently remove any unnecessary adhesives
Caregivers should use good hand hygiene
Avoid potentially hazardous compounds (see Appendix)

Special Considerations for Newborn Skin

Clinical Features

Certain transient skin phenomena occur in newborns and infants, but not later in life.

- Harlequin color change represents a transient alteration in blood flow on one side of the body with a sharp cutoff in the midline. One side of the body becomes impressively more erythematous for seconds to minutes and resolves with position change. It occurs more often in premature infants and when the infant is lying on one side. There is no disease state or underlying systemic disorder related to the phenomena and it spontaneously resolves.
- Cutis marmorata and acrocyanosis: The cutaneous vasculature of infants is immature and may not respond normally to environmental changes. Vasomotor instability leads to vasoconstriction of skin vessels, producing a reticulated pattern on the skin called cutis marmorata. In some infants acral vasoconstriction in the setting of colder

temperatures results in acrocyanosis, a bluish discoloration of the lips, hands, and feet. Both cutis marmorata and acrocyanosis occur more frequently in premature infants. No diagnostic studies are needed. These vascular changes improve with re-warming of the skin.

- Sebaceous gland hyperplasia: Hormonal stimulation in utero leads to hypertrophy of sebaceous glands on the face of newborns. Yellow-white, smooth papules result, most often symmetrically spaced on the nose and upper lip. Up to 50 % of newborns present with sebaceous gland hyperplasia. No diagnostic studies are indicated, and treatment is not needed as the hyperplasia spontaneously resolves.
- Sucking blisters: Vigorous sucking in utero may lead to a sucking blister present at birth. Most often located on the hand or forearm, a solitary, non-inflamed vesicle or bullae is present at birth and spontaneously resolves within 2 weeks. Diagnostic studies are not needed unless the morphology is not typical, or additional vesicles develop after birth to indicate other etiologies (i.e. herpes virus, impetigo, epidermolysis bullosa, etc.).
- Diaper dermatitis is a common manifestation, increasing in frequency after 1 month of age with a prevalence of 4–5 %. It is an irritant dermatitis resulting from a combination of an occlusive moist environment, resulting in macerated skin with increased skin permeability, reduced acid mantle protection (due to alkaline urine, thus activating fecal enzymes), and growth of microorganisms. Typically the dermatitis spares the inguinal folds.
- Seborrheic dermatitis (aka cradle cap) affects up to 10 % of infants. In most cases the scalp is involved with development of greasy, yellow scales, hence the colloquial term "cradle cap." Intertriginous areas may also be involved and, rarely, the dermatitis can be generalized in more severe cases.

Investigations Recommended

Evaluation
The presence of pustules, crusts, hemorrhagic crusts, or ulcerations should prompt cultures and biopsy to investigate for secondary infection or Langerhans cell histiocytosis

Management Strategies

- Diaper dermatitis: Supra-absorbant diapers help reduce the moisture and prevent irritant diaper dermatitis. Once the dermatitis develops, use of non-medicated, fragrance-free wipes is advised, along with frequent diaper changes. A zinc oxide-based diaper paste should be applied as a barrier with every diaper change [3].
- Seborrheic dermatitis: The treatment of cradle cap depends on severity. Most cases respond to a mild anti-dandruff shampoo containing zinc or selenium, or an anti-yeast shampoo such as ketoconazole. Oils may be used to gently lift scales from the scalp.

Table 1.3 First line therapies [2, 3]

Seborrheic Dermatitis Treatment
Salicylic acid should be avoided, due to risks of percutaneous absorption in the newborn. For more inflammatory cases, a low-potency topical corticosteroid may be implemented

Skin Care Products to Avoid

Clinical Features

- Infants and children have increased skin surface-to-body weight ratio which can increase relative absorption of topical medications, and potentially lead to adverse events and toxicity. In addition, the skin has increased absorptive capacity in premature and newborn infants that should be considered when choosing topical therapies. Certain compounds are known to be more hazardous to preterm infants compared to term neonates. For example, providone-iodine may cause local cutaneous necrosis and also hypothyroidism due to absorption of the iodine in premature infants. Salicylic acid used as a keratolytic can be absorbed, leading to salicylism in infants and children [2, 3, 8].

Management Strategies

Avoid the use of agents known to have increased absorption and that lead to toxicity in newborns, infants, and children, or those with a defective skin barrier. Be cautious in using topical agents in newborns, infants, and children.

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Management of Skin Disorders of the Newborn

2

Nika Finelt and Brandi M. Kenner-Bell

Evidence Level

A: Double blind study
B: Clinical Trial ≥20 subjects
C: Clinical trial <20 subjects
D: Series ≥5 subjects
E: Anecdotal case reports

Neonatal Cephalic Pustulosis

For diagnosis
Clinical–facial eruption of pustules that begins at age 5–30 days. It resembles neonatal acne but lacks comedones and is associated with colonization with Malassezia
Giemsa stain–yeast form, neutrophils
For treatment
None, self-limiting

Table 2.1 First line [1, 2]

Ketoconazole 2 % cream applied topically twice a day	D

Neonatal/Infantile Acne (Fig. 2.1)

For diagnosis
Clinical–comedones and inflammatory papules and/or comedones. It usually begins in the first year of life and is secondary to a physiologic increase in adrenal and gonadal androgens.
If severe consider work-up for hyperandrogenism
For treatment
Treat to avoid formation of pitted scarring from acne

Table 2.2 First line [3–6]

Gentle cleansing	E
Mild retinoid (adapalene 1 % gel or tretinoin 0.025 % cream)	C
Benzoyl peroxide 2.5 % cream	D

Fig. 2.1 Neonatal acne

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For Severe Acne Consider

Oral erythromycin, azithromycin, or trimethoprim-sulfamethoxazole	D
Oral isotretinoin	D

Seborrheic Dermatitis

For diagnosis	
Clinical–greasy yellow scale and erythematous patches on the face, scalp, ears, and intertriginous areas. Appears between 2 and 10 weeks of age and may be associated with colonization of *Malassezia furfur*	
Consider culture for bacteria and candida for any weeping intertriginous area	
KOH preparation and fungal culture to exclude superficial dermatophyte infection	
For treatment	
None, self-limiting within a few weeks to months	

Table 2.3 First line [6–15]

Emollients. For thick scale, application of mineral or baby oil followed by gentle scalp massage with a soft toothbrush	A
Frequent shampooing with gentle shampoo or anti-seborrheic shampoo	E
Bifonazole 1 % shampoo	B
Ketoconazole 2 % shampoo	A
Ketoconazole 2 % cream	B
Hydrocortisone 1 % cream	B

Acrodermatitis Enteropathica

For diagnosis	
Clinical–well demarcated scaly perioral and acral plaques, which may be accompanied by alopecia and diarrhea. It is secondary to an autosomal recessive mutation in intestinal zinc-specific transporter gene SLC39A4. Acquired forms of zinc deficiency will have the same clinical presentation and may be secondary to inadequate intake, excessive losses, malabsorption, and increased demands. Mothers may have low zinc secretion into milk caused by a mutation in the SLC30A2 gene, which encodes the transporter responsible for secreting zinc into breast milk	
Blood plasma or serum zinc levels, fasting; serum zinc levels <50 µg/dl	
Low alkaline phosphatase levels	
Urine zinc excretion	
Physical exam and routine laboratory evaluation, lipid profile, copper levels	
Genetic testing	
For treatment	
Blood plasma or serum zinc levels q 3–6 months	

Table 2.4 First line therapy [16–21]

Oral zinc supplementation	A
Inherited deficiency – 3 mg/kg/day for life (50 g elemental zinc per 220 mg zinc sulfate)	
Acquired deficiency – 0.5 to 1.0 mg/kg/day until corrected	

Table 2.5 First line for acropustulosis of infancy [22, 23]

Potent topical corticosteroids	D
Oral antihistamines	E

Acropustulosis of Infancy

For diagnosis	
Clinical–pruritic recurrent pustules on palms and soles	
Rule out scabies	
Skin biopsy–intraepidermal pustules with neutrophils, occasional eosinophils	
For treatment	
None	

Table 2.6 Second line [24]

Oral dapsone	E

Aplasia Cutis Congenita [14] (Figs. 2.2 and 2.3)

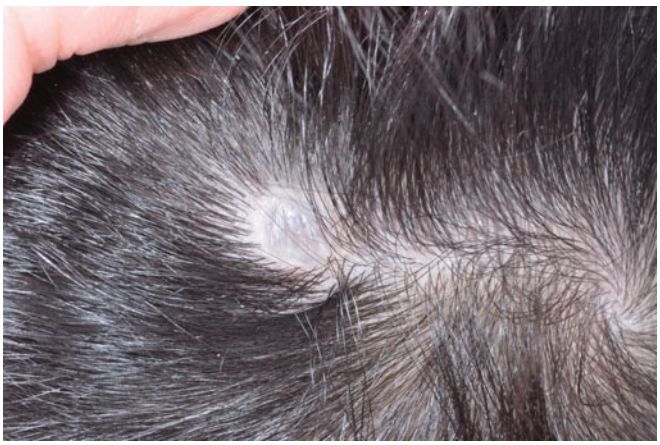
For diagnosis	
Clinical–discrete ovoid defect covered with a membrane that may be bullous or flat at birth that eventually heals with a scar. Aplasia cutis congenita may present sporadically or may be inherited as part of a syndrome	
Thorough history and physical for other developmental anomalies	
If hair collar sign (ring of longer, darker hair around the defect) and midline, need evaluation for underlying neural tube defect; ultrasound or MRI if concerned	
For treatment	
Follow-up proper formation of scar	

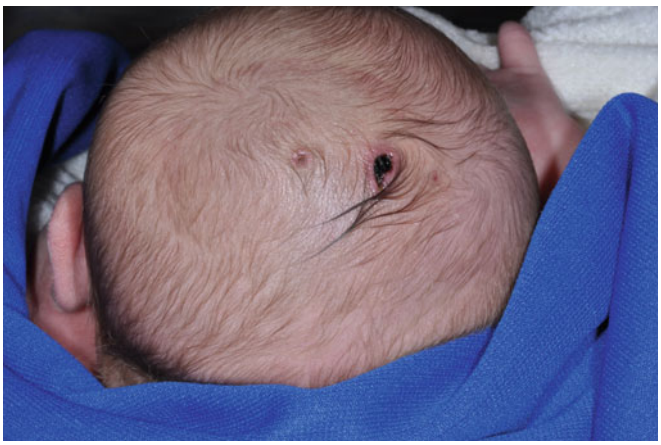
Bronze Baby Syndrome

For diagnosis	
Clinical–hyperpigmentation of the skin, serum, and urine during treatment for neonatal jaundice with phototherapy. It occurs in infants with cholestasis and elevated levels of unconjugated and conjugated bilirubin	
Evaluate for underlying cause of jaundice	
Evaluate for underlying hepatocellular disease	
For treatment	
Monitor for jaundice, cholestasis, hepatocellular disease	

Congenital/Neonatal Herpes Simplex Virus (HSV)

For diagnosis	
Clinical–vesicles on an erythematous base; three recognized syndromes: skin, eyes, and mouth infection (SEM); disseminated infection; central nervous system infection	
Viral Culture (swab from mouth, nasopharynx, conjunctiva, anus, and any vesicles), Viral DFA or immune peroxidase slide test, PCR (skin vesicle, CSF, blood), Tzanck Preparation, ALT elevation, skin biopsy	





Figs. 2.2 and 2.3 Aplasia cutis congenita

Must rule out CNS disease
CT brain, MRI brain, EEG
For treatment with acyclovir
Serial absolute neutrophil count (ANC) twice weekly
Adjust dose of Acyclovir for renal failure or sustained ANC <500 mm^3
For infants with CNS disease- consider daily suppressive therapy with oral acyclovir for 6 months after parenteral regimen

Table 2.7 First line for Bronze Baby Syndrome [6, 14, 25]

Improves with discontinuation of phototherapy	E

Table 2.8 First line [6, 26, 27]

Oral Acyclovir–20 mg/kg IV q8 × 14 (SEM)-21 days (disseminated or CNS)	B

Congenital/Neonatal Candidiasis

For diagnosis
Clinical; congenital–pustules on palms and soles birth to first few days of life, may have respiratory distress; neonatal–diaper (beefy erythema and satellite pustules or vesicles), oral thrush (white plaques on oral mucosa), and also commonly associated with intertrigo (erythema and maceration in skin folds). This warrants a high index of suspicion in premature and immunocompromised infants
Smear of pustule with KOH, Giemsa, Gram, or calcofluor stain (budding yeast or pseudohyphae), fungal culture
PCR, restriction fragment endonuclease digestion of chromosomal DNA, electrophoretic karyotyping, Southern blot hybridization analysis with DNA probes, B-glucan assay, gas chromatography mass spectrometry for D-arabinitol, buffy coat smear microscopy
CBC (leukocytosis), Glucose (elevated)
Congenital–evaluate placenta and umbilical cord for lesions; if suspect disseminated systemic disease (premature and low birth-weight), must culture blood, urine, cerebrospinal fluid
For treatment
Monitor for sepsis
Follow monitoring guidelines for any PO or parenteral antifungals

Table 2.9 First line [28–37]

Thrush–nystatin solution (100,000 u/ml) applied to oral mucosa 4×/day; fluconazole (2–3 mg/kg/day)	A
For localized disease–topical anti-yeast preparations, Imidazoles, nystatin, allylamines	A
Invasive–oral fluconazole, itraconazole, amphotericin	A, B (amphotericin)
Prophylaxis in infants <1 kg-Fluconazole IV	A

Table 2.10 Second line [28–37]

Thrush–Itraconazole (2 mg/kg/day)	D
Localized–1 % gentian violet, or 2 % eosin	E

Staphylococcus Aureus Pustulosis (Fig. 2.4)

For diagnosis
Clinical–discrete vesicles and pustules, or superficial erosions and crust
Bacterial culture and gram stain of fluid from vesicle, pustule, or beneath crust. Nasal swabs to evaluate for S. Aureus carriage
Consider work-up for deeper or systemic infection if any constitutional signs of illness (fever, temperature instability, irritability, lethargy, etc.)
For treatment
Monitor for signs of deeper or systemic infection
Monitor sensitivities of culture
Consult with colleagues and local resources regarding resistance patterns in your hospital and community

Table 2.11 First line [6, 38–44]

Oral antibiotics–choose agent based on sensitivity and local resistance patterns	A
Topical antibiotic ointment (mupirocin, fusidic acid, or retapamulin ointments)	A
Decolonization of neonates and close contacts	A (adults– trial results pending in neonates)

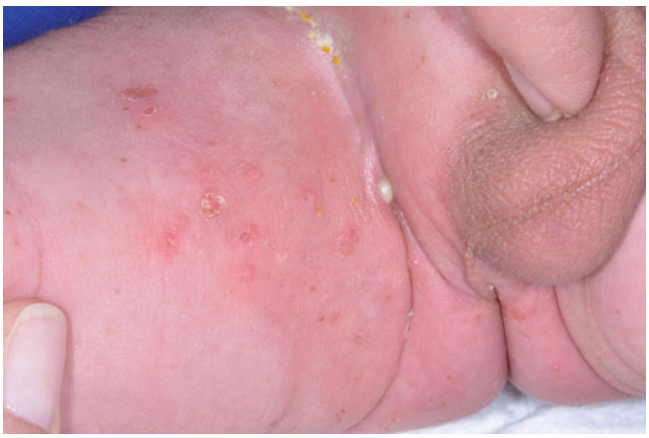


Fig. 2.4 Staphylococcal pustulosis

Neonatal Scabies (Fig. 2.5)

For diagnosis
Clinical–pruritic contagious infestation of the Sarcoptes scabiei mite that commonly involves the palms, soles, and axillae but also may involve the scalp of infants. Burrows, vesicles, erythematous papules, and nodules may be present
Mineral oil examination–apply a drop of mineral oil and scrape with No. 15 blade then smear contents onto glass slide, cover with mineral oil and evaluate for mite, eggs, or feces
Dermoscopy
For treatment
May have pruritus for 1 month following treatment

Table 2.12 First line [14, 45]

Permethrin 5 % cream–approved for over 2 months of age but commonly used under 2 months of age, traditionally applied to all skin from neck down and rinsed after 8 h, but scalp must also be treated in infants; should be repeated after 1 week	A
Sulfur 6 % ointment–safe in infants and pregnant women; apply as above for 3 consecutive nights and rinse 24 h after last application	D
Treat all close contacts	E
Launder all linens following treatment	E

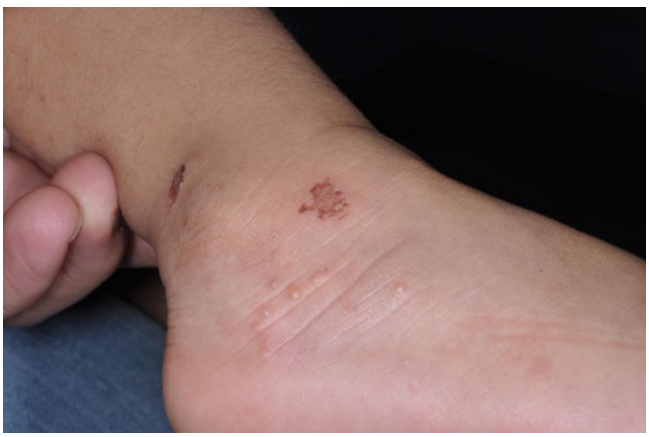


Fig. 2.5 Neonatal scabies

Cutis Marmorata Telangiectatica Congenita (Figs. 2.6 and 2.7)

For diagnosis
Clinical–localized or generalized fixed reticulate erythema, may see limb hypo or hyperplasia over affected extremity
Evaluate for other vascular anomalies, macrocephaly (rule out macrocephaly-capillary malformation syndrome)
Consider ophthalmology referral for facial involvement
Consider neurology referral for neurological symptoms
For treatment
Monitor as above

Table 2.13 First line [46, 47]

Monitor for signs of hypoplasia or hyperplasia	D

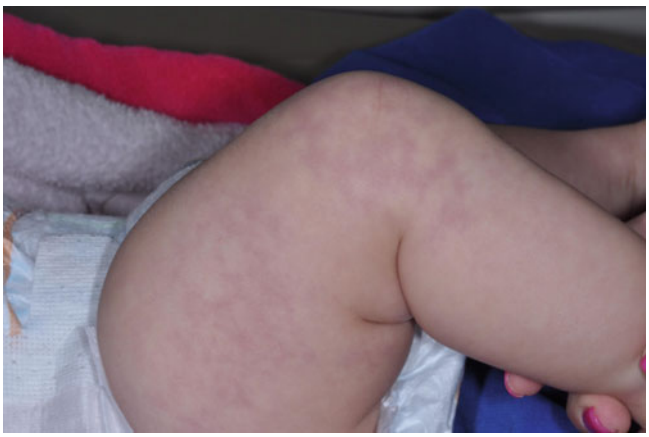


Fig. 2.6 Cutis marmorata telangiectatica congenita

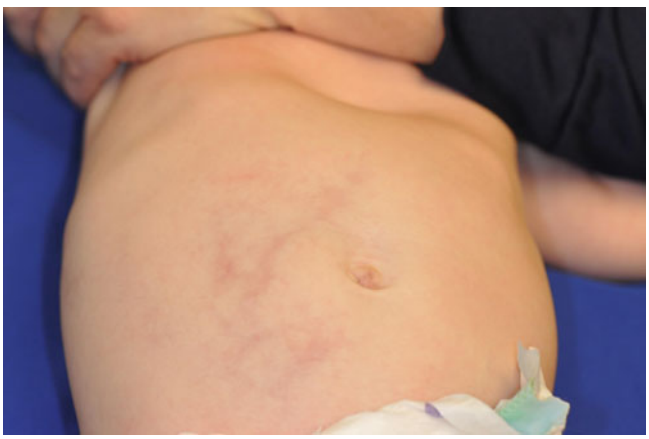


Fig. 2.7 Cutis marmorata telangiectatica congenita

Eosinophilic Pustular Folliculitis (Fig. 2.8)

For diagnosis
Clinical–Pruritic follicular based pustules, commonly on the scalp, that recur in crops
Culture with sensitivity from pustule
Gram stain and mycology from touch prep, Tzanck or scraping