Common Diagnoses in Pediatric Dermatology:
Empowering the pediatrician and knowing when to refer

1) Atopic dermatitis
   a) *Pathogenesis*
      i) Genetic factors dictate immune response and skin barrier integrity, and environmental influences also play a role
         (1) Most common uniallelic or biallelic loss-of-function mutations in profilaggrin (FLG)
         (2) Environmental factors include irritants, allergens (pollen, foods), heat/cold, microbiome, mechanical injury
            (a) *S. aureus* (usually MSSA), present in 70% of lesional skin – less commensal organisms
      ii) These activate the immune system – specifically Th2 cells express IL-4,-5, and-13, which promote eosinophilia and IgE production but suppress the expression of epidermal barrier proteins as well as antimicrobial peptides
   b) Infantile vs. childhood AD
      i) Infantile AD
         (1) Typically begins on scalp, forehead, cheeks and the diaper often spared, predilection for extensor surfaces
         (2) "Acute" and weeping, may be confused for infection
         (3) Itch may be disguised as fussiness or wiggling
         (4) Saliva a common irritant
         (5) Mimickers: seborrheic dermatitis (not typically as itchy; may have both and both generally require topical steroids if severe)
      ii) Childhood AD
         (1) Classic areas of involvement in this group are the wrists, ankles, hands, feet, neck, and antecubital and popliteal regions
         (2) Facial involvement shifts toward periorbital area
         (3) Lesions more lichenified or thickened over time
         (4) Lymphadenopathy and nail dystrophy may be seen
   c) Common Variants
      i) Follicular eczema – more common in skin of color patients, all hair follicles accentuated in lesional and non lesional skin
      ii) Nummular eczema – coin shaped patches classically more difficult to treat, weeping and may be infected
      iii) Prurigo nodularis – large nodules thickened from recurrent scratching, considered for some patients to be a variant of AD and treated similarly (even with Dupixent)
   d) Treatment
      i) Special consideration: Sudden onset or sudden worsening consider concomitant viral illness, viral infection of the skin, bacterial infection of the skin, or something new coming into contact with the skin (contact dermatitis)
      ii) **Moisturization**

(1) Daily baths hydrate the skin -- water loss is prevented by emollient application within a few minutes after bathing
(2) Prefer a thick emollient (cream) or ointment
(3) Wet wraps of plain water can be applied at night after bathing and after application of moisturizer or topical medication
(4) What about oil?
  (a) Sunflower seed and safflower seed oils preferred
  (b) Some studies also show virgin coconut oil helpful, would avoid olive oil (contact sensitization possible)

iii) **Anti-inflammatory (blue = consider referral if all other things above have failed)**
(1) Topical steroids are our mainstay of treatment
(2) Creams may be more prone to burning but may be less messy/preferred by patients, ointments more effective
(3) If the appropriate topical steroid is chosen, the goal is for rash to clear with twice daily application in 2-3 weeks on any given body surface area
  (a) The goal is always to treat until clear and I would never prescribe anything in a quantity that I do not think is appropriate
  (b) Treating until clear I think leads to less topical steroid use over time
(4) Other topicals like protopic ointment, elidel cream, and eucrisa ointment are best used for maintenance or very mild atopic dermatitis (SE of stinging/burning)
(5)

Body Area	List of topical options
Face	
	Hydrocortisone 2.5% ointment or cream Desonide 0.05% ointment or cream Alclometasone 0.05% ointment or cream Fluocinolone 0.01% oil
Body	
	Fluticasone 0.05% cream Triamcinolone 0.025% cream or ointment Triamcinolone 0.1% cream or ointment **Mometasone 0.1% cream or ointment** **Fluocinonide 0.05% ointment or cream** **Clobetasol 0.05% ointment or cream**

iv) **Anti-itch**
(1) This is a widely debated topical though still generally used among most dermatologists
(2) Sedating antihistamines (hydroxyzine preferred over Benadryl;doxepin) can be useful to help with sleep in addition to controlling dermatitis
(3) Non-sedating antihistamines may be helpful for those with other signs of atopy or urticaria

v) **Anti-infection**
(1) Bleach baths (“swimming pool bath”) can be helpful at decreasing burden of disease flaring (S. Aureus)

(2) Alternative is CLN wash (hypochlorous acid wash)
(3) Dilute vinegar may also be helpful for smaller areas or wounds (1 tbs per cup of water)

vi) Systemic therapy

How to take a bleach bath

What you need:


Bathtub


Water


Measuring cups or spoons


Regular or non-concentrated, unscented household bleach (5–6% sodium hypochlorite)

Recommended water-to-bleach ratio:*

Tub size	Full, standard-size bathtub (~40 gallons)	Half standard-size bathtub (~20 gallons)	Baby-sized bathtub (~4 gallons)
Bleach	½ cup	¼ cup	1 tablespoon
Age	Adults	Adults and children	Children and babies

**This bleach bath recipe has the same level of chlorine as your average swimming pool.*

Directions:

1. Fill your bathtub with lukewarm water. Be sure the water is not too hot.
2. Add bleach to water and mix it in thoroughly. Do not add any other products or ingredients to the bathwater.
3. Get into the tub. Soak your body for 10 minutes. Do *not* submerge your head or face under the water. Avoid splashing and getting water in your eyes. Do *not* soak for longer than 15 minutes.
4. After you're done soaking, rinse your body off with lukewarm water and pat dry.
5. Use your moisturizer of choice to lock moisture into your skin.

Talk to your provider

Before you try a bleach bath for yourself or your child, please consult with your healthcare provider first. They can help you decide if it is a good option for you. For more resources on bathing and moisturizing with AD, visit **NationalEczema.org**.

(1) After failing appropriate trial of topical therapy with appropriate escalation of strength in the steroid ladder and topical steroids cannot be stopped without immediate rebound of rash
(2) Percentage of body surface area involved
(3) Frequent hospitalizations or secondary infection despite preventative measures
(4) Quality of life measures!
  (a) Are they losing sleep?
  (b) Is their itching affecting concentration at school or daycare?

e) Food allergies
  i) No evidence that avoidance of maternal dietary antigens during pregnancy or lactation has a protective effect during the first 18 months of life on the development of AD or on food sensitization!
  ii) Solid foods, including potentially allergenic foods, should not be delayed in at-risk infants
  iii) Early referral to AI for those with moderate to severe AD may help with parental hesitancy for food introduction

iv) Severe AD kids are at highest risk for food allergies due to potential skin exposure of the antigens from environment or foods – so their AD needs to be treated

v) Soriano VX, Ciciulla D, Gell G, Wang Y, Peters RL, McWilliam V, Dharmage SC, Koplin JJ. Complementary and Allergenic Food Introduction in Infants: An Umbrella Review. Pediatrics. 2023 Feb 1;151(2):e2022058380.

vi) Abrams EM, Shaker MS, Chan ES, Brough HA, Greenhawt M. Prevention of food allergy in infancy: the role of maternal interventions and exposures during pregnancy and lactation. Lancet Child Adolesc Health. 2023 May;7(5):358-366.

2) Hidradenitis suppurativa

a) Pathogenesis

i) *Chronic inflammation of apocrine glands (autoinflammatory)*

ii) *Occlusion of hair follicles → rupture → re-epithelize to form tunnels under skin*

b) Demographics

i) Increased prevalence in females and African Americans

ii) Typically develops after puberty (we see it as early as 7 or 8)

iii) Potential association with other inflammatory conditions such as Crohn's disease

c) Clinical presentation

i) Patients develop recurrent tender subcutaneous nodules in axilla, anogenital regions, and under breasts

ii) Some patients will have numerous blackheads or comedones in these areas, this can be another clue

iii) 2 or more lesions in these sites in a lifetime → patient should be referred to dermatology

d) Treatment

i) Approach is stepwise – starting with topical therapy including topical clindamycin and BPO wash or hibiclens (dove also makes an antibacterial soap) to all prone areas

ii) Oral therapy- commonly use doxycycline for longer periods as we would with acne for anti-inflammatory effects; other treatments include clindamycin and rifampin

iii) Biologic therapy – Humira and cosentyx both FDA approved, Humira and infliximab tend to be used for more severe disease, Cosentyx more targeted but useful for more mild disease

iv) Hormone therapy – spironolactone, OCPs, metformin, finasteride

v) Perform deroofing procedures or refer to plastic surgery once disease is under control to remove persistent tunnels/scars

3) Vascular lesions

a) Nevus simplex

i) Ectatic capillaries represent persistent fetal circulatory patterns in skin

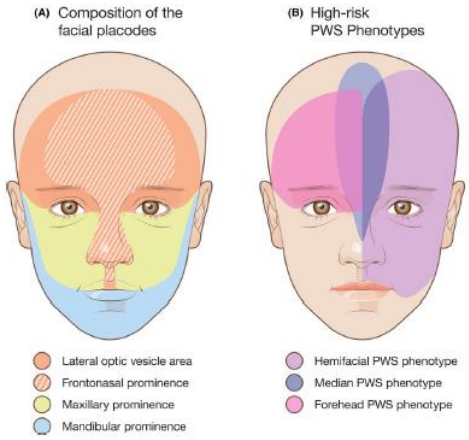
ii) Typically midline and symmetric

iii) Up to 40% of newborns, may be extensive

(1) Usually will have at least one typical site of involvement: glabella (77.8%), nape (59.3%), and eyelids (55.6%)
(2) Additional sites were the scalp, including the vertex, occiput, parietal (66.7%); nose (66.7%); lip (59.2%); lumbosacral skin (55.6%); and upper and mid back (14.8 %)

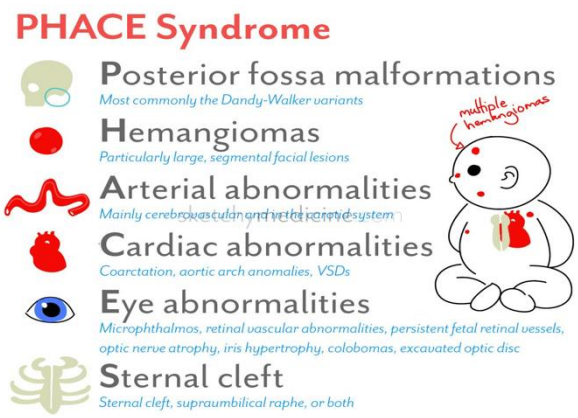
b) Nevus flammeus (port wine birthmark)

i) Typically evident at birth, will not thicken or proliferate at the 2-3 month mark as wound a hemangioma
ii) Over time (several years), lesions thicken and develop vascular blebs that bleed
iii) Earlier intervention with laser shows better outcomes (swaddle until age 2)
iv) Risk of Sturge Weber syndrome
(1) High risk lesions: midline forehead/nose, hemifacial, or more than half of the contiguous hemi forehead (based on embryonic fusion planes/facial placodes)
(2) Ophthalmology exam– glaucoma and diffuse choroidal hemangioma
(3) MRI with contrast - Tram track gyraform calcifications
(4) Neurology evaluation
(5) If + MRI findings:
(a) Early intervention with aspirin and anti-epileptics



c) *Infantile hemangioma*

i) Typically, evident at birth or first few weeks of life
ii) Most rapid growth 2-3 months of age (not adjusted for prematurity)
iii) Plateau around 6-8 months (deeper persist)
iv) Start to involute around age 1, maximum improvement around 5-8 years of age
v) Often residual scar tissue or redness
vi) Eye lesions need ophthalmology eval most of the time
vii) Consider referral to discuss propranolol for any facial lesion larger than 0.5 cm, diaper lesion, trunk lesion >2 cm, trunk lesion in a highly visible or cosmetically sensitive location, ulcerated or painful hemangioma
viii) PHACES syndrome – needs MRI/MRA of the brain and neck, echocardiography, ophthalmologic evaluation



d) Neonatal hemangiomatosis

i) Diffuse (with internal organ involvement) or benign

ii) Most common organ involved – liver
iii) Other organs at risk secondarily – heart and thyroid (The transient hypothyroidism associated with hepatic IH is caused by hemangioma production of the thyroid hormone inactivating enzyme, type 3 iodothyronine deiodinase. Evaluation reveals elevated thyroid-stimulating hormone (TSH), normal to decreased free thyroxine (fT4), decreased free triiodothyronine (fT3), and increased reverse T3 (rT3).) Needs to be checked only for very large multiple lesions or with any signs of growth problems

4) Other birthmarks and pigmentary disorders

a) Congenital nevi

i) Defined as melanocytic nevi present at birth or within the first few months of life
ii) Neural crest derived
iii) Grow rapidly during early infancy and enlarge in proportion to the child's growth – **thicken during puberty**
iv) Large and giant lesions on the head, neck, and back have risk of neurocutaneous melanosis (also >20 satellite nevi)
(1) Risk of melanoma 10-15% over lifetime, usually <5 years of age and nodular
(2) Risk of melanoma in small or medium size lesion is about 1% and comparable to general population

b) Café au lait macules

i) Having one or two is normal amongst the general population especially in darker skin types
ii) Can be classified as atypical or typical
iii) Signs of NF1 early to consider
(1) 6 or more CALMs > 5 mm in diameter (usually typical with smooth borders)
(2) (Large lesions suspicious for early PNFs)
(3) Axillary freckling*
(4) Tibial bowing, psedoarthrosis
(5) Sphenoid wing dysplasia resulting in pulsatile exophthalmos
(6) Paper determining risk groups based on typical vs atypical CALMS (Ben-Shachar et al. (2017)
(a) **High-risk group:** Patients with 6 or more regular CALMs and under 29 months, or those with 6 or more CALMs under 14 months, faced an **80.4%** chance of developing NF1.
(b) **Intermediate-risk group:** Patients with fewer than 6 CALMs under 29 months, or 6 or more CALMs over 29 months without atypical CALMs, had an **11.5% to 14.3%** chance of developing NF1.
(c) **Low-risk group:** Patients with fewer than 6 CALMs over 29 months, or those older than 29 months with atypical CALMs, **had a 0.9% chance** of developing constitutional NF1.

c) Pigmentary mosaicism

i) Hypo or hyperpigmented lesions along lines of blaschko, can co-exist

ii) Extracutaneous manifestations occurred more often with hypopigmentation and extensive lesions
iii) Other associations: developmental delay (54%), bone issues (38%), seizures or electroencephalogram abnormalities (37%), dysmorphic facial features (31%), and/or psychomotor retardation (16%)

5) Infections and Exanthems
   a) Bullous impetigo - Blistering secondary to exfoliative toxin produced by bacteria (same in SSSS)
      i) Localized disease can treat with mupirocin, often will need oral antibiotics (Keflex)
   b) Staphylococcal scalded skin
      i) **Staphylococcal exfoliative toxin - Systemic effect, bacteria not present in denuded skin unless you are swabbing primary site**
      ii) Binds desmosomes in epidermis → loss of cell-cell adhesion leads to bullae formation and sloughing of skin
      iii) Peri-oral and peri-ocular fissuring, skin folds, then generalized erythema with + Nikolsky sign
      iv) Oral nafcillin, clinda, +/- MRSA coverage
   c) Gianotti Crosti Syndrome
      i) Symmetrically distributed on the face, buttocks, and extremities of children
      ii) Usually with preceding URI-like symptoms
      iii) EBV is believed to be the most common etiology in the United States
      iv) Hep B, CMV, pox virus (molluscum), parvovirus B19, rotavirus, and HHV-6
   d) Molluscum contagiosum
      i) Caused by ***Poxvirus***
      ii) Face, trunk, diaper area common
      iii) Spontaneous remission within 6 months to 2 years
      iv) Lesions themselves may cause scarring as can treatment (consider referral if numerous or symptomatic)
      v) Treatment options: **cryotherapy**, Gotucream, dilute apple cider vinegar, berdazimer gel, **cantharidin**, cimetidine
      vi) Child abuse and sexual transmission
         (1) New genital herpetic lesions in children who have independent toileting are suspicious for abuse and should be reported
         (2) HPV/Condyloma most often acquired through vertical transmission or by contact with adults and young diapered children
            (a) Usually innocent if <3 years of age (long latency period)
         (3) Molluscum contagiosum is most often acquired innocently, sexual transmission is possible (adults)

6) Products

Children's of Alabama