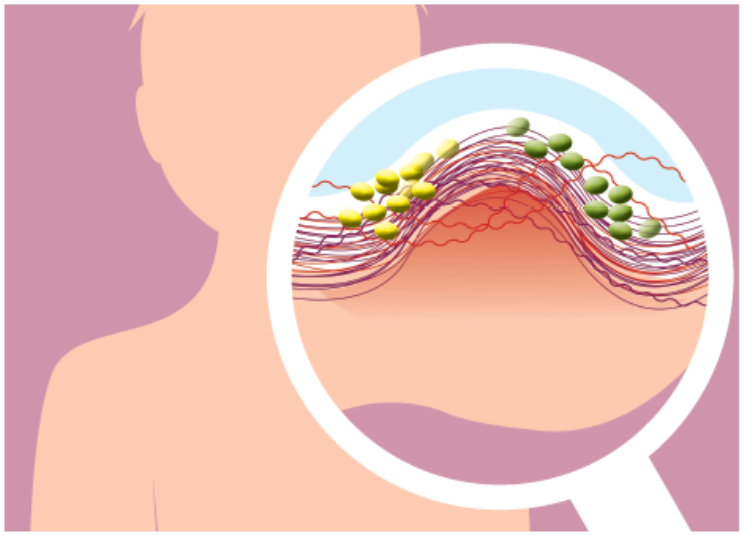

MILD TO MODERATE ATOPIC DERMATITIS:

Pathogenesis and Therapeutic Strategies For Improved Outcomes

Saturday, April 22, 2017

Please complete the preassessment located in your handout before the program begins.

Sponsorship and Support

This educational activity is jointly provided by
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This activity is supported by an educational grant
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To receive credit for your participation in this educational activity:

- Read the objectives and other introductory CME information
- Complete the preassessment ***prior to the start*** of the activity
- Participate in the Atopic Dermatitis presentation
- Complete the postassessment and evaluation ***at the conclusion*** of the activity

Faculty and Disclosures

Activity Cochair

Adelaide A. Hebert, MD

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Dr Hebert has disclosed the following relationships:

Advisory Board: Anacor Pharmaceuticals, Inc; Demira; Galderma Laboratories, LP; GlaxoSmithKline; PharmaDerm; Procter & Gamble; Promius Pharma, LLC; Shionogi, Inc; Stiefel, a GSK Company; Valeant Pharmaceuticals International
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**All research funds paid to the UTHealth McGovern School of Medicine.*

Faculty and Disclosures

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Dr Eichenfield has disclosed the following relationships:

Consultant: Anacor Pharmaceuticals, Inc; Celgene; Genentech, Inc; Lilly Pharma; Maruho Co Ltd; Medimetriks Pharmaceuticals, Inc; Otsuka Pharmaceutical Co Ltd; Pfizer Inc; Regeneron Pharmaceuticals, Inc; Sanofi Pharma; TopMD; Valeant Pharmaceuticals

Investigator: GlaxoSmithKline; Medimetriks Pharmaceuticals, Inc; Otsuka Pharmaceutical Co Ltd; Regeneron Pharmaceuticals, Inc

Levels of Evidence

Two types of grades are provided for any treatment recommendations made in the presentation

Level of Evidence	Strength of Clinical Recommendation
• Used to evaluate available evidence based on the quality of study methodology and the overall focus of the study **I.** Good-quality patient-oriented evidence (ie, evidence measuring outcomes that matter to patients: morbidity, mortality, symptom improvement, cost reduction, and quality of life) **II.** Limited-quality patient-oriented evidence **III.** Other evidence including consensus guidelines, opinion, case studies, or disease-oriented evidence (ie, evidence measuring intermediate, physiologic, or surrogate endpoints that may or may not reflect improvements in patient outcomes)	• Developed based on the best available evidence **A.** Recommendation based on consistent and good-quality patient-oriented evidence **B.** Recommendation based on inconsistent or limited-quality patient-oriented evidence **C.** Recommendation based on consensus, opinion, case studies, or disease-oriented evidence

Source: American Academy of Dermatology Guidelines of Care for the Management of Atopic Dermatitis.

Off-Label Statement

This educational activity may contain discussion of published and/or investigational uses of therapies that are not indicated by the FDA, including roflumilast, OPA-15406, tofacitinib, SB011, and apremilast.

Please refer to the official prescribing information for each product for discussion of approved indications, contraindications, and warnings.

Further, participants are encouraged to consult appropriate resources for any product or device mentioned in this program.

Learning Objectives

At the conclusion of this live activity, practitioners should be better able to:

- Summarize the role of skin barrier dysfunction and inflammatory responses in atopic dermatitis (AD) pathogenesis
- Describe the inflammatory/immunomodulating pathways in atopic dermatitis
- Evaluate the benefits and limitations of current therapies for mild to moderate atopic dermatitis
- Assess the efficacy and safety of emerging therapies and their potential role in treating atopic dermatitis

Epidemiology, Prevalence, and Pathogenesis

What's Your Diagnosis?

- 4-month-old infant presents with erythematous scaling dermatitis of the cheeks bilaterally
- Similar-appearing lesions over the posterior neck and extensor aspects of the extremities

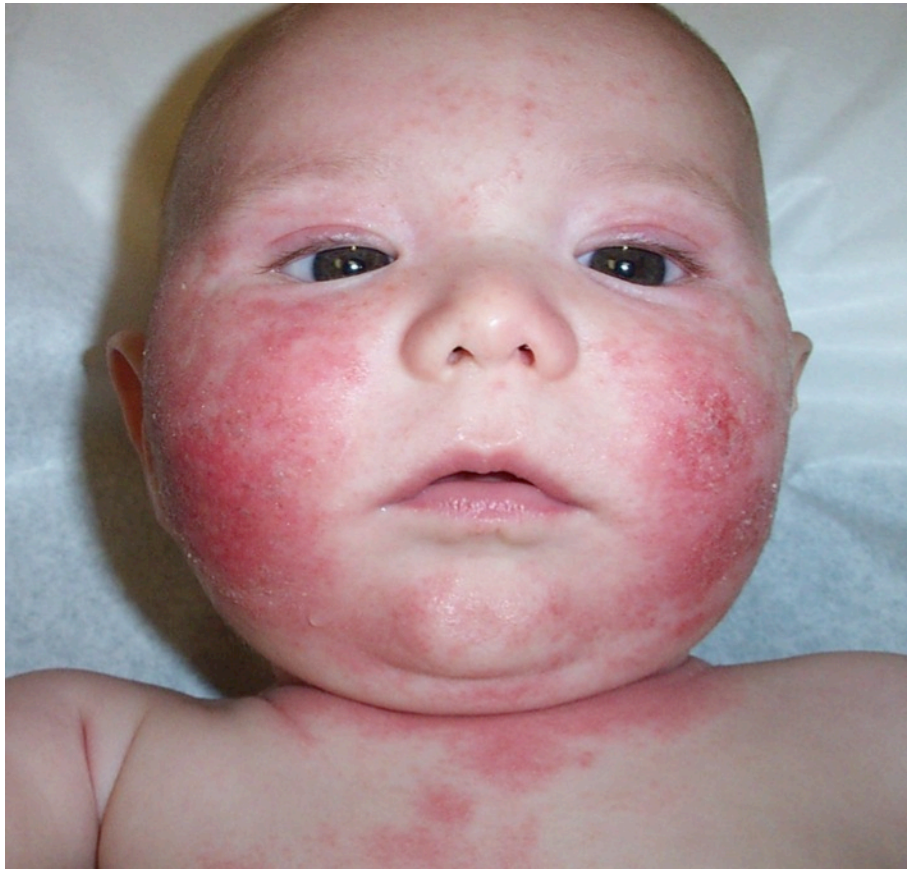


Photo courtesy of Adelaide A. Hebert, MD

Epidemiology in Children and Adolescents

- Affects 10%-20% of school-aged children in the US[1]
- Higher prevalence in African Americans, urban residents, and children living in homes with higher education levels[2]
- AD will persist into adulthood in up to 33% of children[1]

1. Silverberg NB. *Cutis*. 2016;97:267-271. 2. Wolter DY, et al. *Pediatr Clin North Am*. 2014;61(2):241-260.

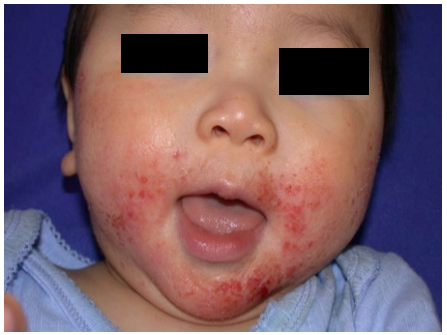
Diagnostic Criteria for Atopic Dermatitis

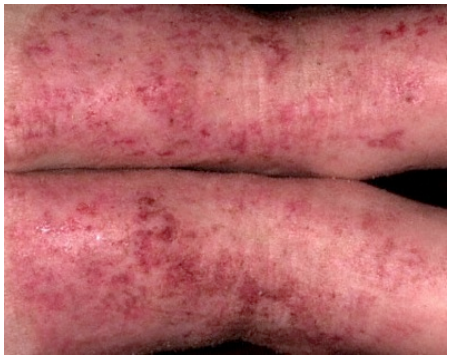
- Pruritus (itching)
- Eczematous changes that are acute, subacute, or chronic
  - Age-specific distribution patterns
  - Intermittent course with flares and remissions

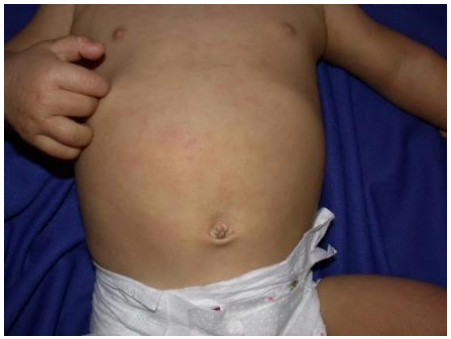
American Academy of Dermatology. *J Am Acad Dermatol*. 2014;70(2):338-351.

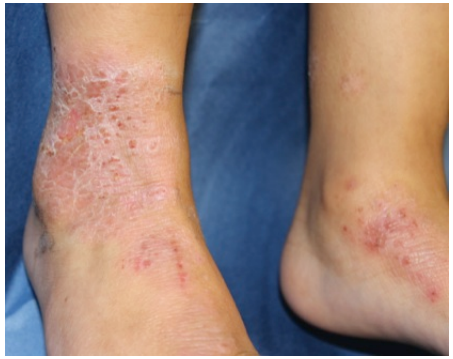
Primary Physical Findings

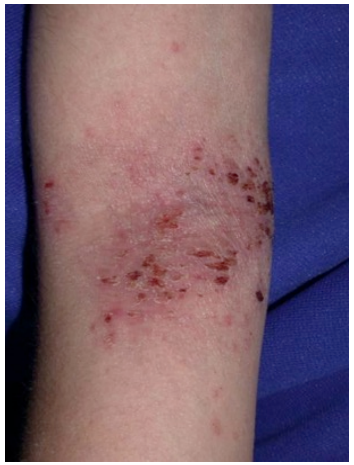
- Erythema
- Papules/plaques
- Excoriations
- Xerosis
- Erosions and crusting
- Lichenification
- Dyspigmentation







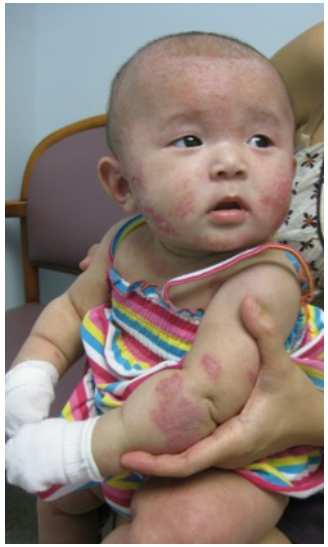


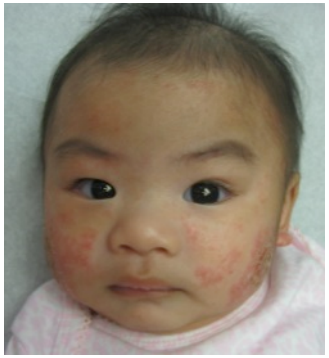


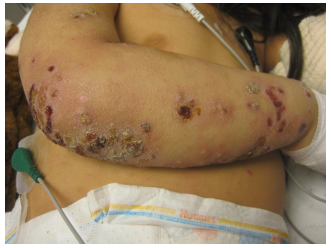
Photos courtesy of
Lawrence F. Eichenfield, MD

Eichenfield LF, et al. *Pediatrics*. 2015;136(3):554-565.
Siegfried EC, et al. *J Clin Med*. 2015;4(5):884-917.

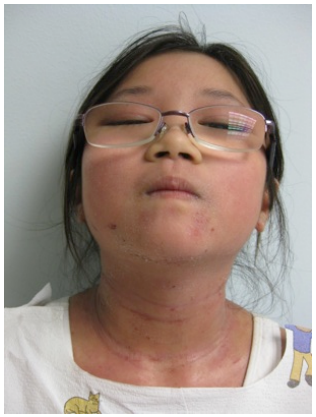
Distribution of Atopic Dermatitis Varies With Age

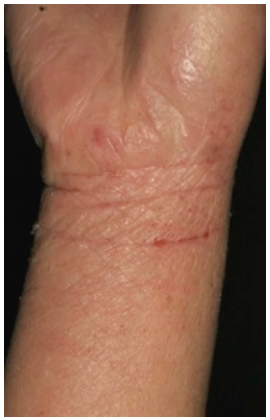


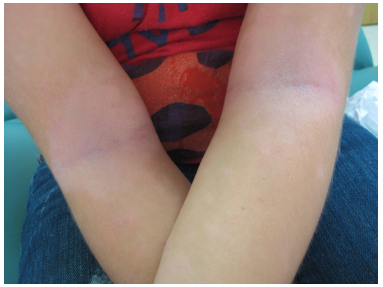


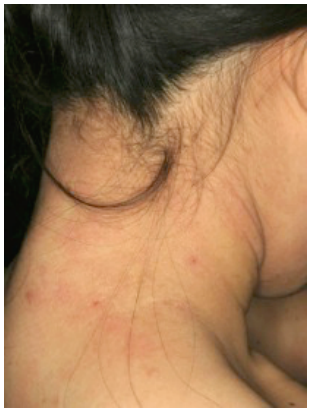


Infants
Face, trunk (except diaper area), extensor extremities









Children
Flexors (wrists, ankles, antecubital/popliteal fossae)

Adolescents
Flexors, neck, wrists, hands, ankles

Photos courtesy of Lawrence F. Eichenfield, MD, and Adelaide A. Hebert, MD

Siegfried EC, et al. *J Clin Med*. 2015;4(5):884-917.
Weidinger S, et al. *Lancet.* 2016;387(10023):1109-1122.

Clinical Presentation in Children

- More than 7 million healthcare provider visits per year for AD[1]
- Approximately 67% of children have mild disease that can be managed by a primary care provider[2]
- Specialist referral is required in moderate (20%) and severe (2%) cases[3]

1. Horii KA, et al. *Pediatrics*. 2007;120(3):e527-e534.
2. Eichenfield LF, et al. *Pediatrics*. 2015;136(3):554-565.
3. Arkwright PD. *J Allergy Clin Immunol*. 2013;1(2):142-151.

Mild Atopic Dermatitis

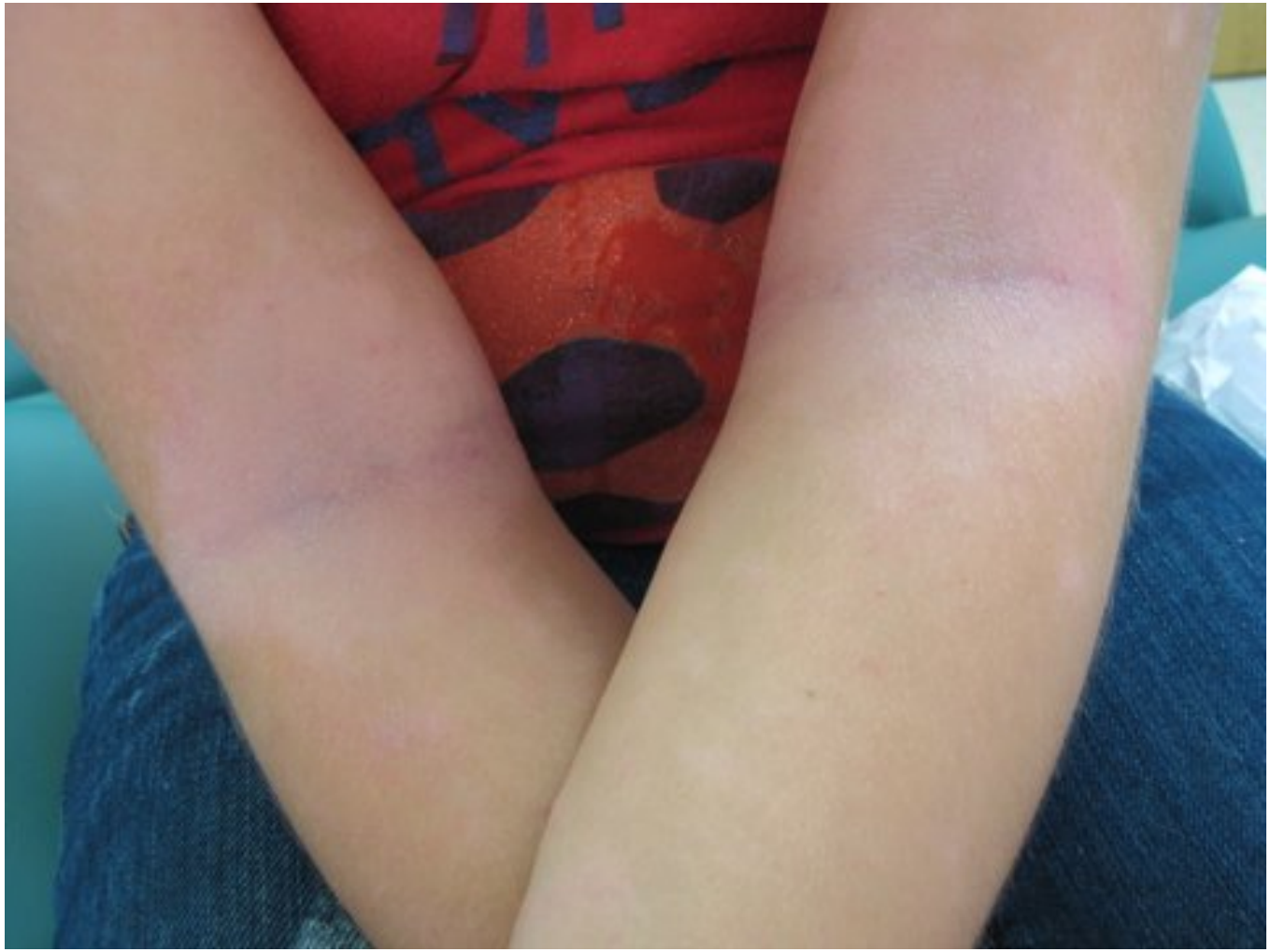


Photo courtesy of Adelaide A. Hebert, MD

Moderate Atopic Dermatitis

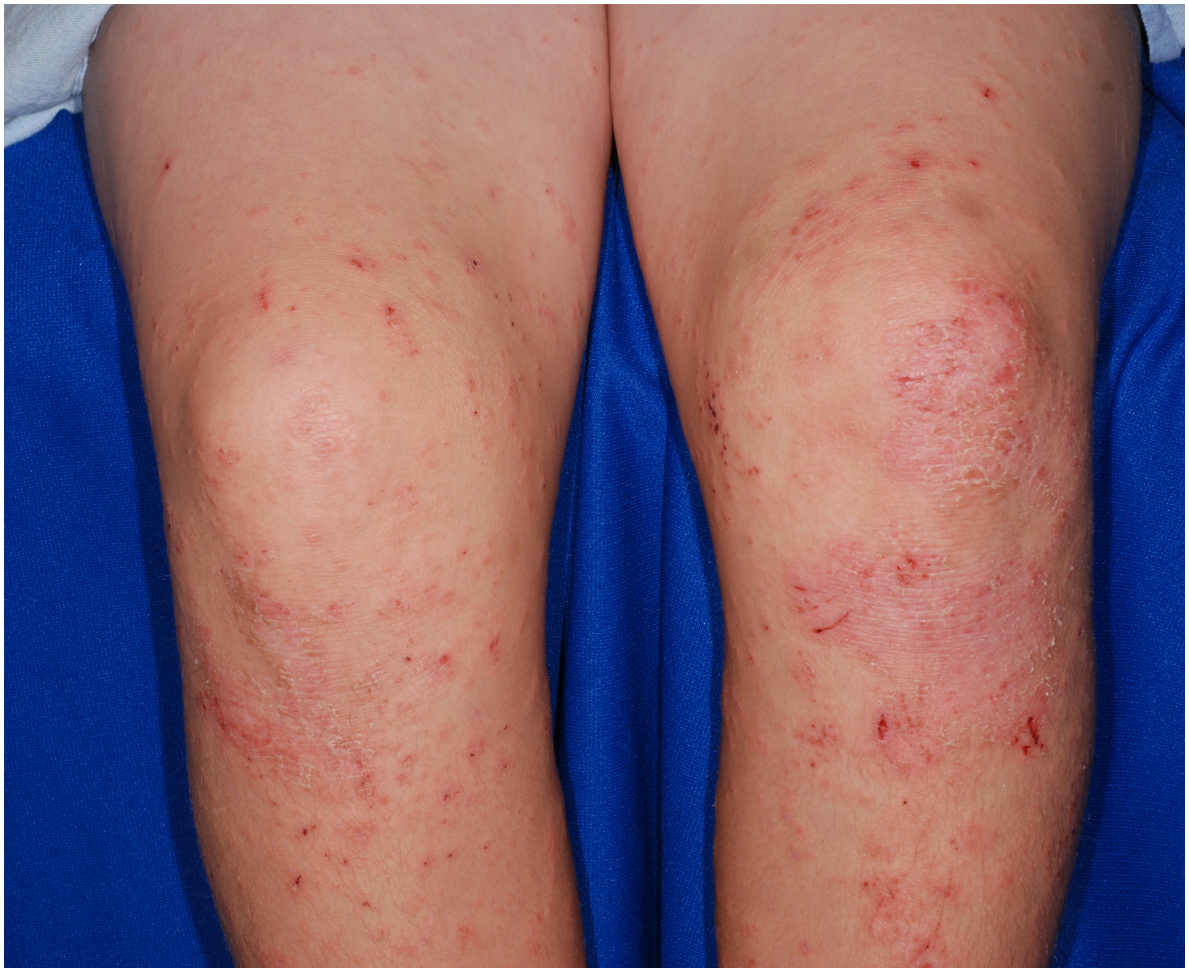


Photo courtesy of Anthony J. Mancini, MD

Severe Atopic Dermatitis

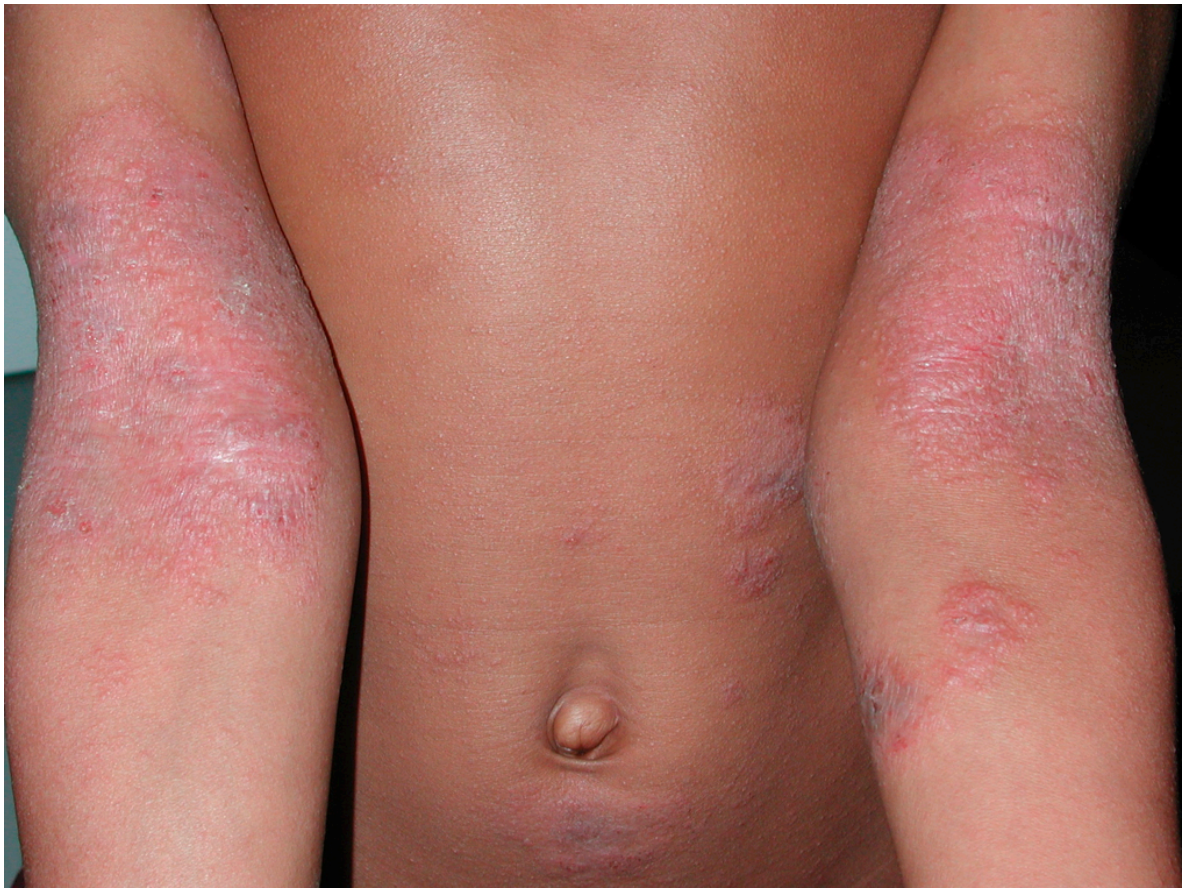
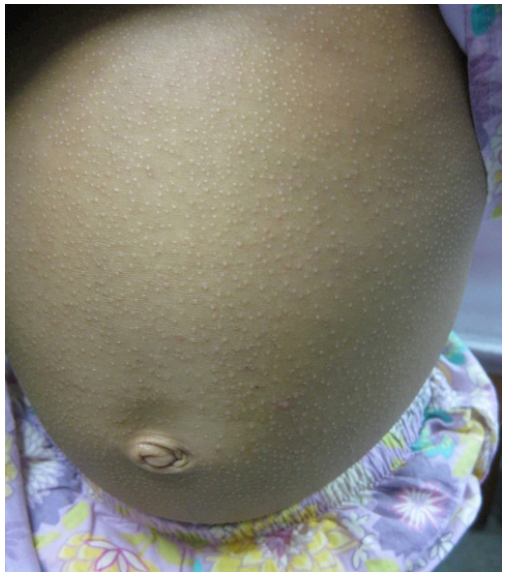
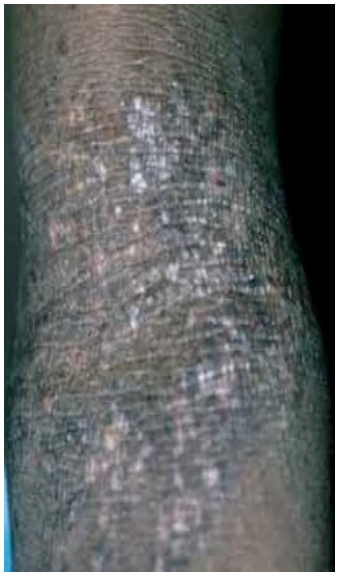


Photo courtesy of Lawrence F. Eichenfield, MD

Features in Darker Skin Types





Photos courtesy of Lawrence F. Eichenfield, MD, and Adelaide A. Hebert, MD

Follicular accentuation

Pityriasis alba

Erythema (hard to see due to pigmentation)

Marked lichenification

Differential Diagnosis of Atopic Dermatitis: Common Disorders

- Seborrheic dermatitis
- Scabies
- Impetigo
- Contact dermatitis (allergic and irritant)
- Psoriasis
- Ichthyosis vulgaris
- Tinea corporis
- Keratosis pilaris

Scabies

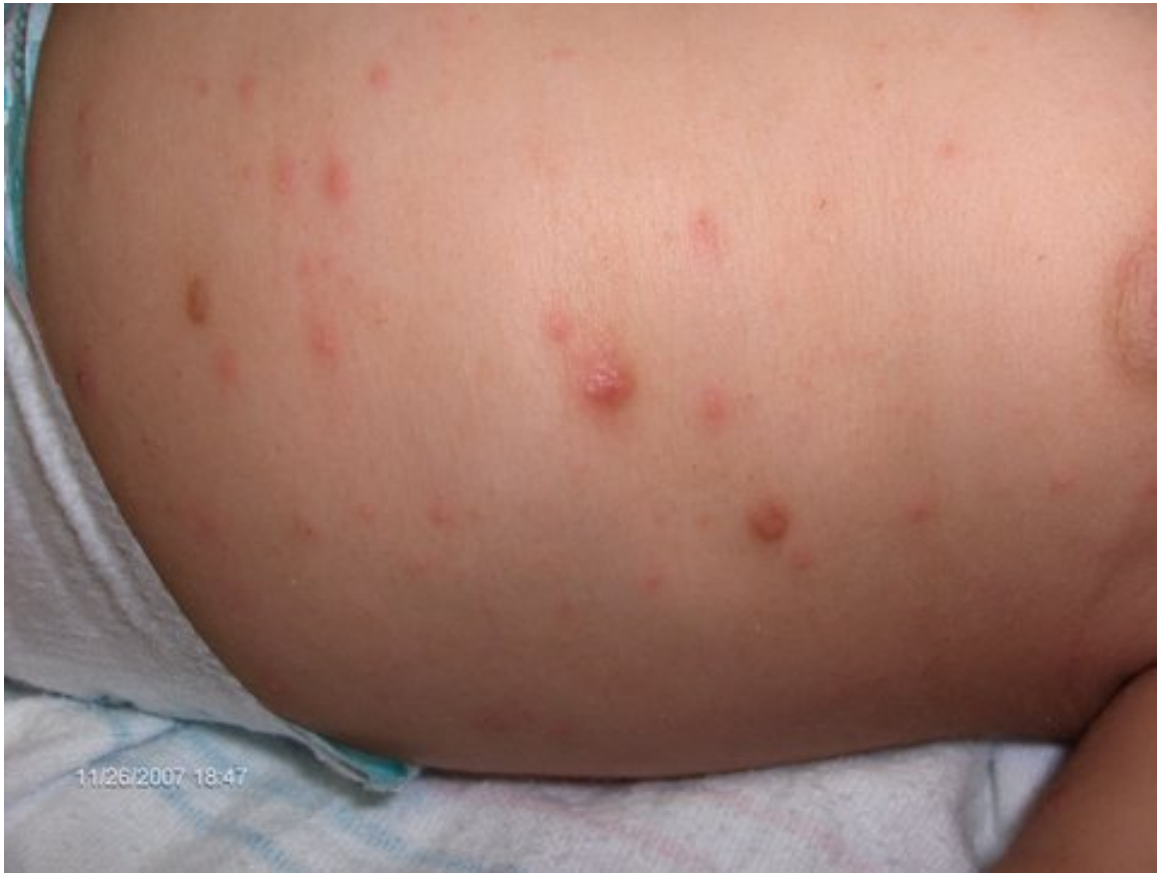


Photo courtesy of Adelaide A. Hebert, MD

Impetigo

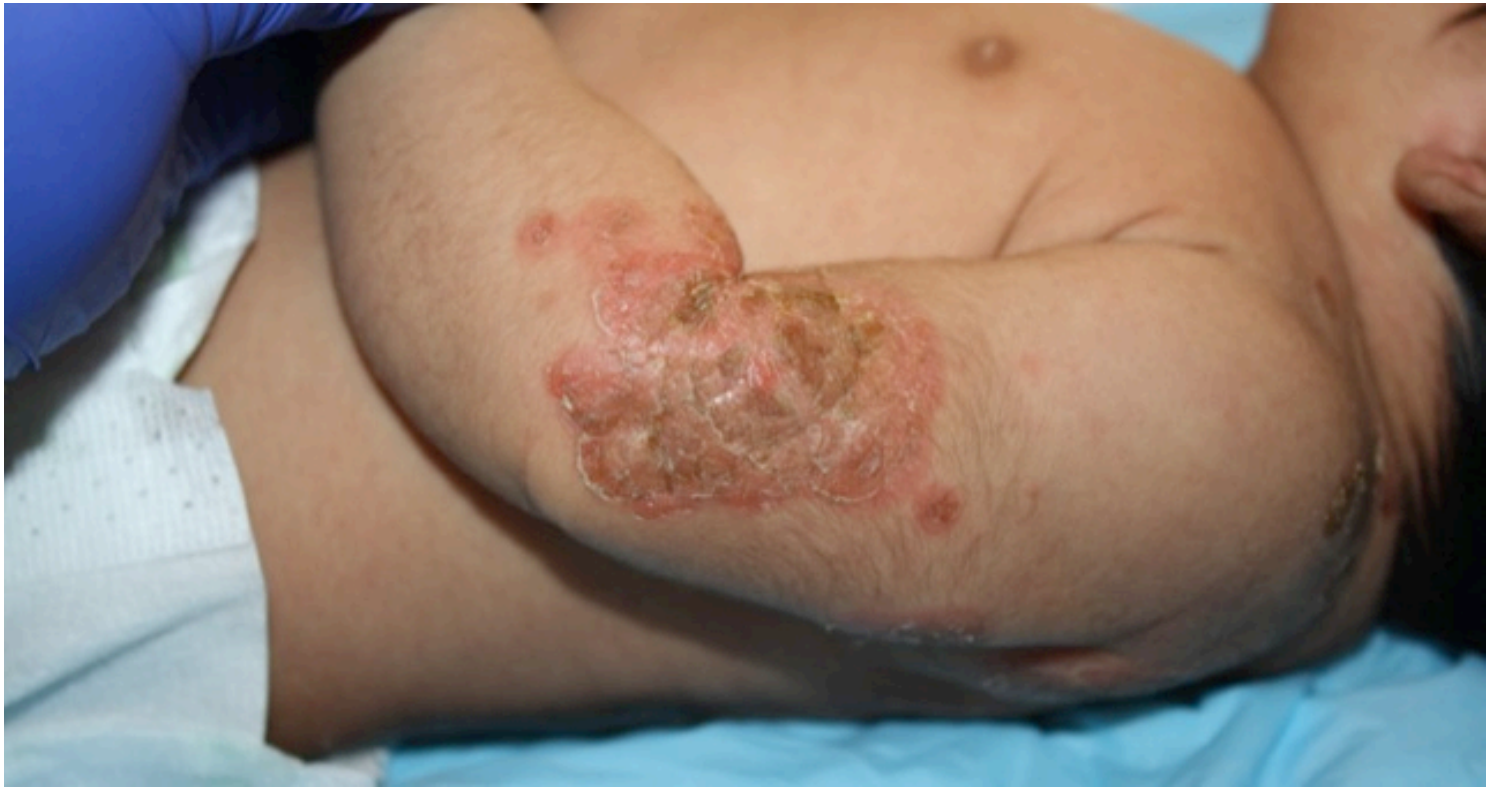


Photo courtesy of Lawrence F. Eichenfield, MD

Differential Diagnosis of Atopic Dermatitis: Rare Disorders in Infancy and Childhood

Metabolic/nutritional/genetic disorders

- Acrodermatitis enteropathica
- Zinc deficiency (prematurity; breast milk deficient in zinc; cystic fibrosis)
- Other nutritional deficiencies (biotin, essential fatty acids)
- Netherton syndrome
- Phenylketonuria
- Omenn syndrome
- Prolidase deficiency
- Gluten sensitivity-related dermatitides
- Hurler syndrome

Differential Diagnosis of Atopic Dermatitis: Rare Disorders in Infancy and Childhood

Immune disorders

- Hyperimmunoglobulin E syndrome
- Severe combined immunodeficiency disorder
- Wiskott-Aldrich syndrome
- Agammaglobulinemia
- Ataxia-telangiectasia
- Neonatal lupus erythematosus

Proliferative disorders

- Langerhans cell histiocytosis

Differential Diagnosis of Atopic Dermatitis: Rare Disorders in Adolescents and Adults

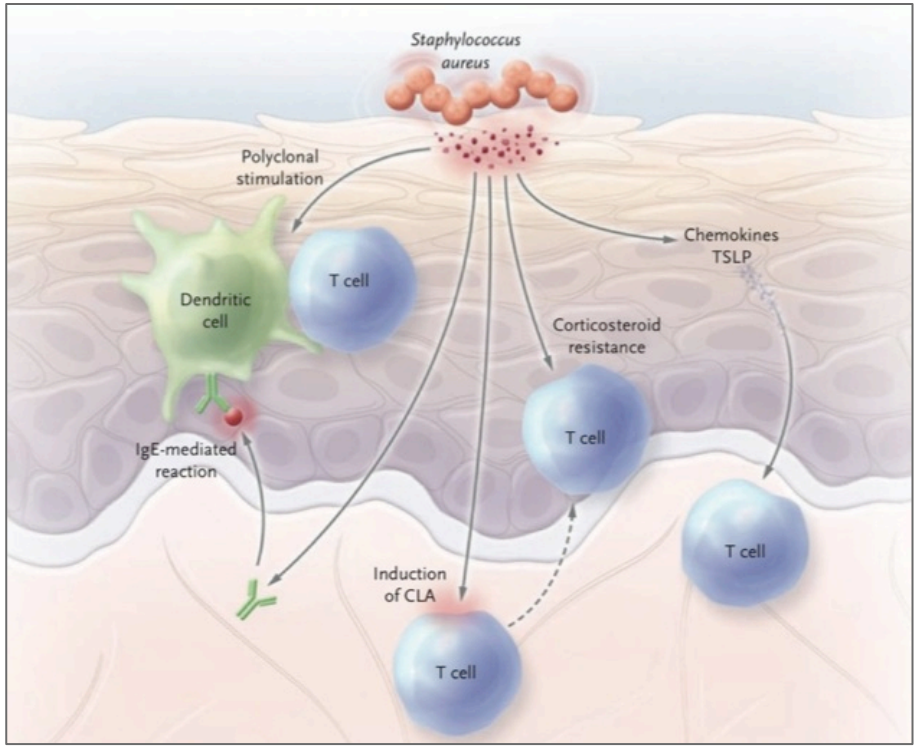
- Cutaneous T-cell lymphoma (Mycosis fungoides or Sézary syndrome)
- HIV-associated dermatoses
- Dermatomyositis
- Graft-versus-host disease
- Lupus erythematosus
- Pemphigus foliaceus
- Drug eruptions

Pathogenesis

Complex, heterogeneous pathogenesis

- Skin barrier dysfunction
  - Filaggrin mutations
  - Diminished ceramides
- Inflammation
- Pruritus/scratching
- Microbial colonization
- Allergy

Colonization by *Staphylococcus aureus*

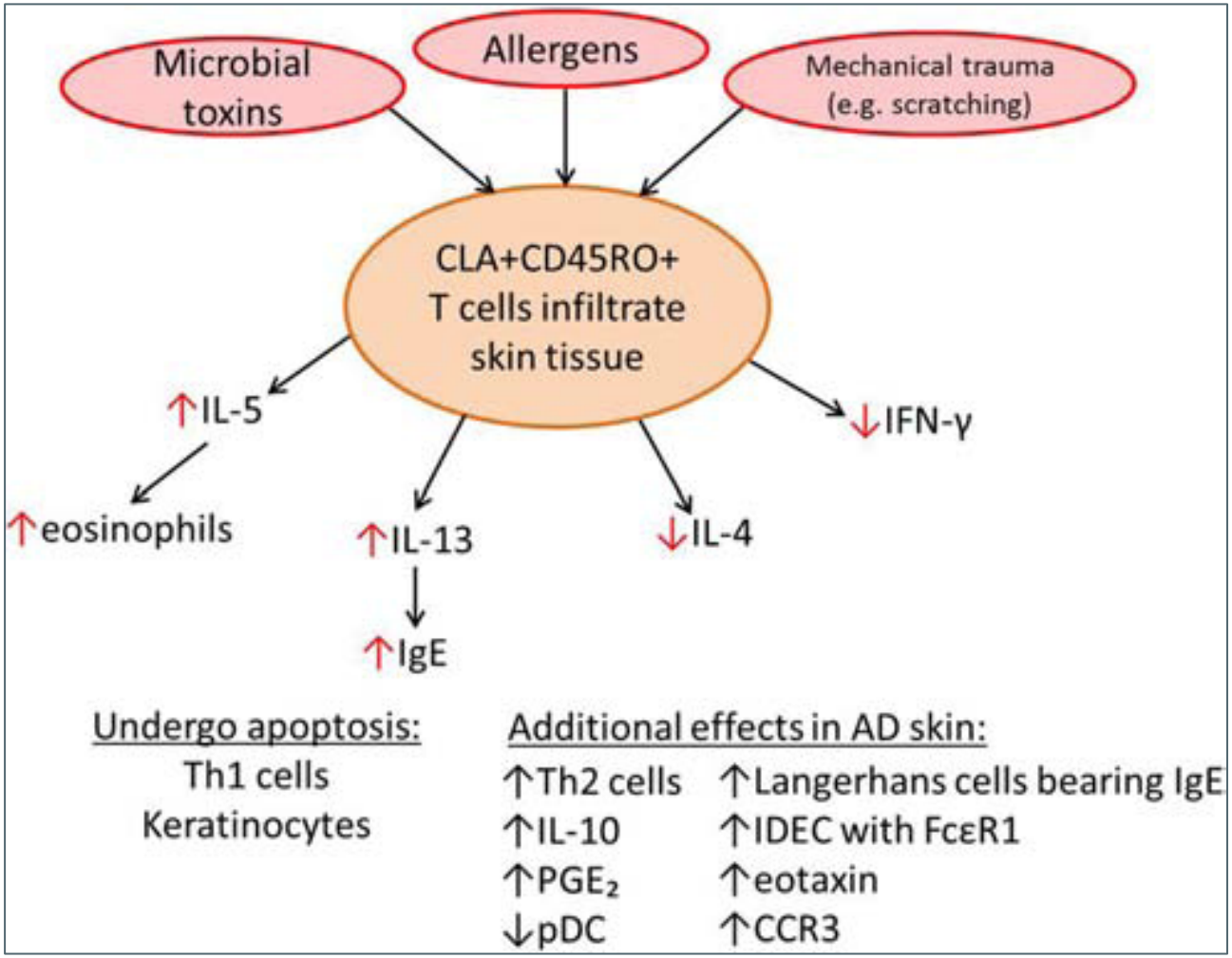


- Worsens disease status
- Renders disease harder to control
- Patients do not have to be infected to be adversely impacted by *S. aureus*
- Skin that is colonized has a true trigger for disease flares

Bieber T. *N Engl J Med*. 2008;358(14):1483-1494.

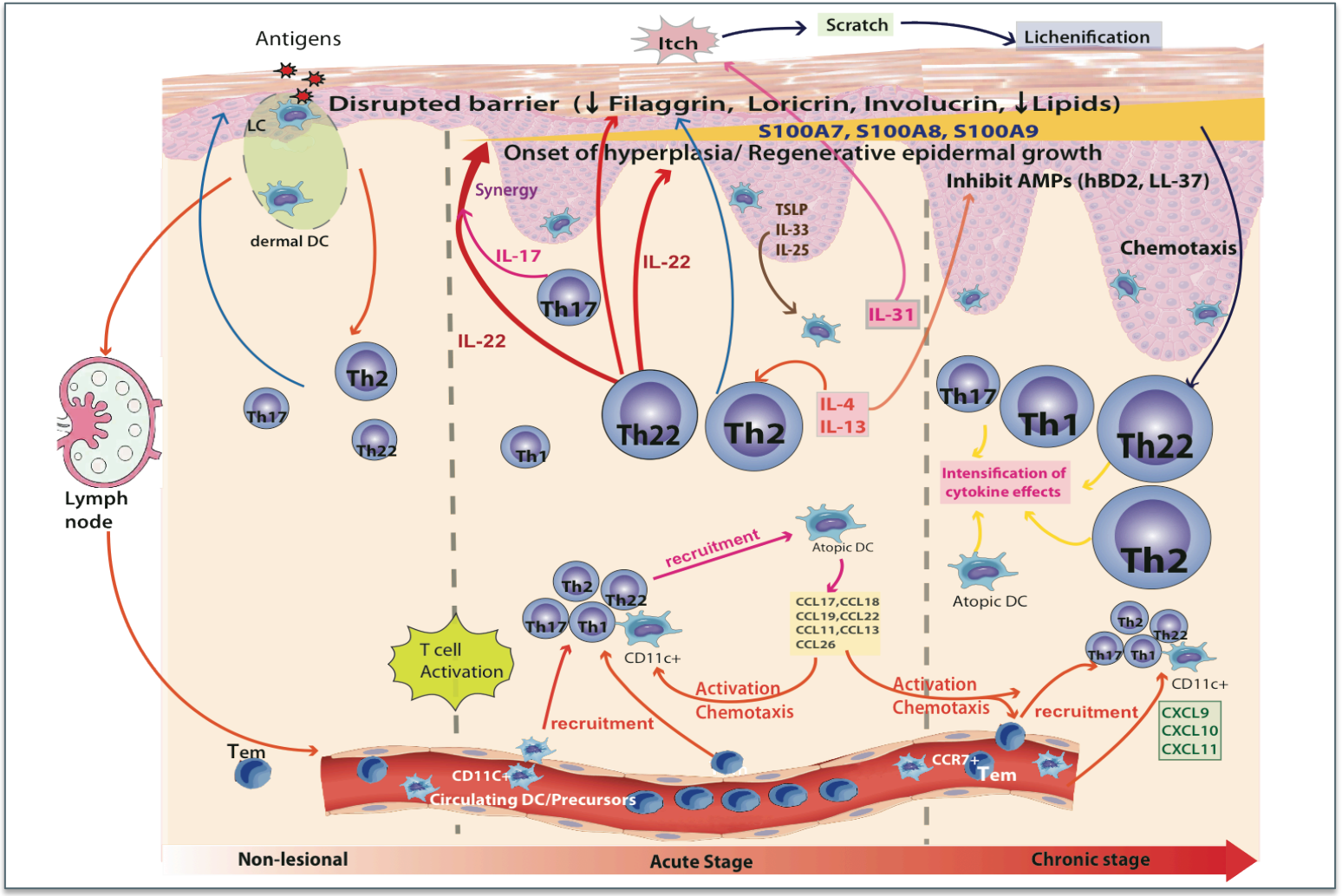
Boguniewicz M, et al. *J Allergy Clin Immunol*. 2010;125:4-13.

Immunopathogenesis of Atopic Dermatitis



Harskamp CT, et al. *Semin Cutan Med Surg*. 2013;32:132-139.

Pathogenesis of Atopic Dermatitis



Gittler JK, et al. *J Allergy Clin Immunol*. 2012;130(6):1344-1354.

Burden of Disease

- **Quality of Life**
  - An average of 9 flares per year, each lasting 15 days[1]
  - Poor quality of sleep[2]
    - Sleep disturbances ~7.3 nights per flare[1]
    - Increased co-sleeping (up to 30% in one study)[3]
    - Polysomnography showing high number of arousals in AD children, independent of scratching[4]
  - Itching[5]
    - 87% experience itching daily
    - Itching lasts ≥ 18 hours in 41.5% of patients

1. Zuberbier T. *J Allergy Clin Immunol.* 2006;118:226-232.
2. Shani-Adir A, et al. *Pediatr Dermatol*. 2009;26(2):143-149.
3. Chamlin SL, et al. *Arch Pediatr Adolesc Med*. 2005;159:745-750.
4. Reuveni H, et al. *Arch Pediatr Adolesc Med*. 1999;153:249-253.
5. Simpson EL, et al. *J Am Acad Dermatol.* 2016;74(3):491-498.

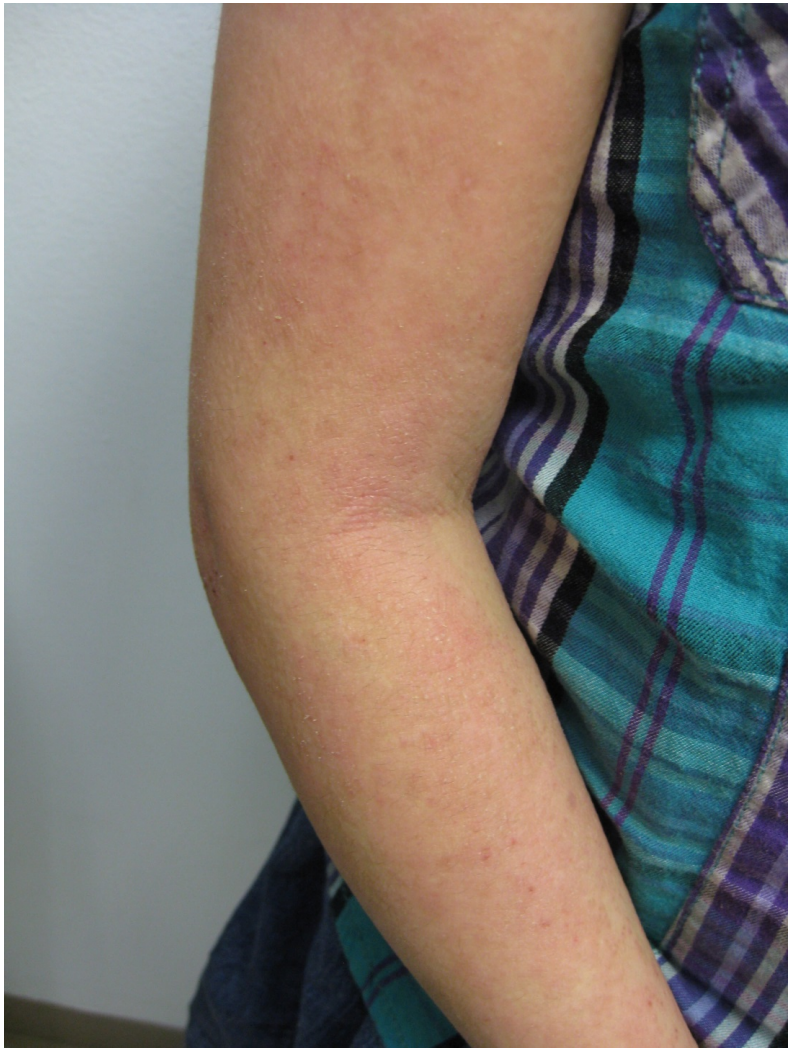
Impact of Comorbidities

- Asthma
- Allergic rhinitis
- Food allergy
- Contact dermatitis
- Emerging comorbidities
  - Obesity
  - Hypertension

Simpson EL. *Curr Dermatol Rep*. 2012;1(1):29-38.

Available Therapies and Management Strategies

Case 2



Case 2: Therapy Recommendations

- Apply mild to moderate potency topical steroids twice daily (A,I) for 1-2 weeks, several days beyond clearing for flare control
- Emollients 2 to 3 times per day (A,I)
- Sedating antihistamines can be considered if sleep is disturbed….BUT…skin-directed therapy should be emphasized!

*Against use of systemic antihistamines: sedating C, III, and nonsedating A, II.

Eichenfield LF, et al. *J Am Acad Dermatol*. 2014;71:116-132.
Sidbury R, et al. *J Am Acad Dermatol*. 2014;71:327-349.
Stein SL, et al. *JAMA*. 2016;315:1510-1511.

Maintenance Therapies for Atopic Dermatitis

- Skin care
  - Liberal and frequent application of moisturizers
  - Warm baths/showers (<5 min) using nonsoap cleansers or mild soaps
- Antiseptic measures
  - Dilute bleach baths
- Trigger avoidance

Eichenfield LF, et al. *Pediatrics*. 2015;136(3):554-565.

Management of Acute Flares

- Avoid trigger factors
- Restore barrier integrity
- Control itching
- Treat infection/control colonization

Eichenfield LF, et al. *Pediatrics*. 2015;136(3):554-565.

Barrier Defect

Repairing Barrier Integrity Requires Fundamental Skin Care

- Gently cleanse twice a day
- Use mild, nonsoap cleansers (syndets)
  - Eg, CeraVe®, Cetaphil®, Equate®, etc
- Use an effective moisturizer every day after cleansing

Eichenfield LF, et al. *Pediatrics*. 2015;136(3):554-565.
Nicol NH. *Cutis.* 2005;76(suppl 6):26-31.

Water: Irritant or Treatment?

- **Water *irritates* skin IF:**
  - Skin is frequently wet, without immediate application of effective moisturizer
  - Moisture evaporates, causing skin barrier to become dry, irritated
- **Water *hydrates* skin IF:**
  - Effective moisturizer is applied and hydration is retained, keeping skin barrier intact and flexible

Importance of Barrier Integrity

Topical Corticosteroids (TCS): Benefits and Limitations

Benefits:

- Highly effective at treating inflammation
- Rapid onset of action
- Multiple potency and delivery vehicles
  - Varied potency frequently required per patient

Limitations:

- Product-specific age limits (although often used off-label)
- Potential for local and systemic side effects (but rare when used appropriately):
  - Local: striae, telangiectasias, skin atrophy, dyspigmentation, periorificial dermatitis, acne rosacea
  - Systemic: HPA axis suppression
  - Periorbital administration: cataracts, glaucoma

HPA, hypothalamic-pituitary-adrenal.

Eichenfield LF, et al. *J Am Acad Dermatol*. 2014;71:116-132.
Stein SL, et al. *JAMA*. 2016;315:1510-1511.

Table V. Relative potencies of topical corticosteroids

Class	Drug	Dosage form(s)	Strength (%)
I. Very high potency	Augmented betamethasone dipropionate	Ointment	0.05
	Clobetasol propionate	Cream, foam, ointment	0.05
	Diflorasone diacetate	Ointment	0.05
	Halobetasol propionate	Cream, ointment	0.05
II. High potency	Amcinonide	Cream, lotion, ointment	0.1
	Augmented betamethasone dipropionate	Cream	0.05
	Betamethasone dipropionate	Cream, foam, ointment, solution	0.05
	Desoximetasone	Cream, ointment	0.25
	Desoximetasone	Gel	0.05
	Diflorasone diacetate	Cream	0.05
	Fluocinonide	Cream, gel, ointment, solution	0.05
	Halcinonide	Cream, ointment	0.1
	Mometasone furoate	Ointment	0.1
	Triamcinolone acetonide	Cream, ointment	0.5
III-IV. Medium potency	Betamethasone valerate	Cream, foam, lotion, ointment	0.1
	Clocortolone pivalate	Cream	0.1
	Desoximetasone	Cream	0.05
	Fluocinolone acetonide	Cream, ointment	0.025
	Flurandrenolide	Cream, ointment	0.05
	Fluticasone propionate	Cream	0.05
	Fluticasone propionate	Ointment	0.005
	Mometasone furoate	Cream	0.1
	Triamcinolone acetonide	Cream, ointment	0.1
V. Lower-medium potency	Hydrocortisone butyrate	Cream, ointment, solution	0.1
	Hydrocortisone probutate	Cream	0.1
	Hydrocortisone valerate	Cream, ointment	0.2
	Prednicarbate	Cream	0.1
VI. Low potency	Alclometasone dipropionate	Cream, ointment	0.05
	Desonide	Cream, gel, foam, ointment	0.05
	Fluocinolone acetonide	Cream, solution	0.01
VII. Lowest potency	Dexamethasone	Cream	0.1
	Hydrocortisone	Cream, lotion, ointment, solution	0.25, 0.5, 1
	Hydrocortisone acetate	Cream, ointment	0.5-1

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Includes representative examples and not all available agents.

Eichenfield LF, et al. *Pediatrics*. 2015;136(3):554-565.

Topical Corticosteroids

- Low potency: hydrocortisone 1%-2.5% or desonide 0.05%
- Mid potency: triamcinolone 0.1%
- High potency: fluocinonide 0.05%

Eichenfield LF, et al. *J Am Acad Dermatol*. 2014;71:116-132.

Topical Calcineurin Inhibitors (TCI) Benefits

- Extensive clinical trials experience
- Steroid-sparing
- Good efficacy for mild, moderate, and severe AD
- Used for acute and maintenance therapies
- Little systemic absorption
- Can be applied to face (including periorbital regions), extremities, and genital area

Eichenfield LF, et al. *J Am Acad Dermatol*. 2014;71:116-132.
Stein SL, et al. *JAMA*. 2016;315:1510-1511.

Available TCIs

TCI	Vehicle	Indications
Pimecrolimus (1%)	Cream	Approved for mild to moderate AD (2 years and older)
Tacrolimus (0.03% and 0.1%)	Ointment	Approved for moderate to severe AD (0.03%: 2 years and older; 0.1%: 15 years and older)

- Both TCIs were shown to be more effective than vehicle in short-term (3-12 weeks) and long-term studies (up to 12 months) in adults and children with active disease
  - Decline in Eczema Area and Severity Index (EASI) score
  - Decrease in percent body surface involved
  - Reduction in patient evaluated symptoms and signs of disease

Eichenfield LF, et al. *J Am Acad Dermatol*. 2014;71:116-132.
Stein SL, et al. *JAMA*. 2016;315:1510-1511.

TCI Limitations and Potential Adverse Events

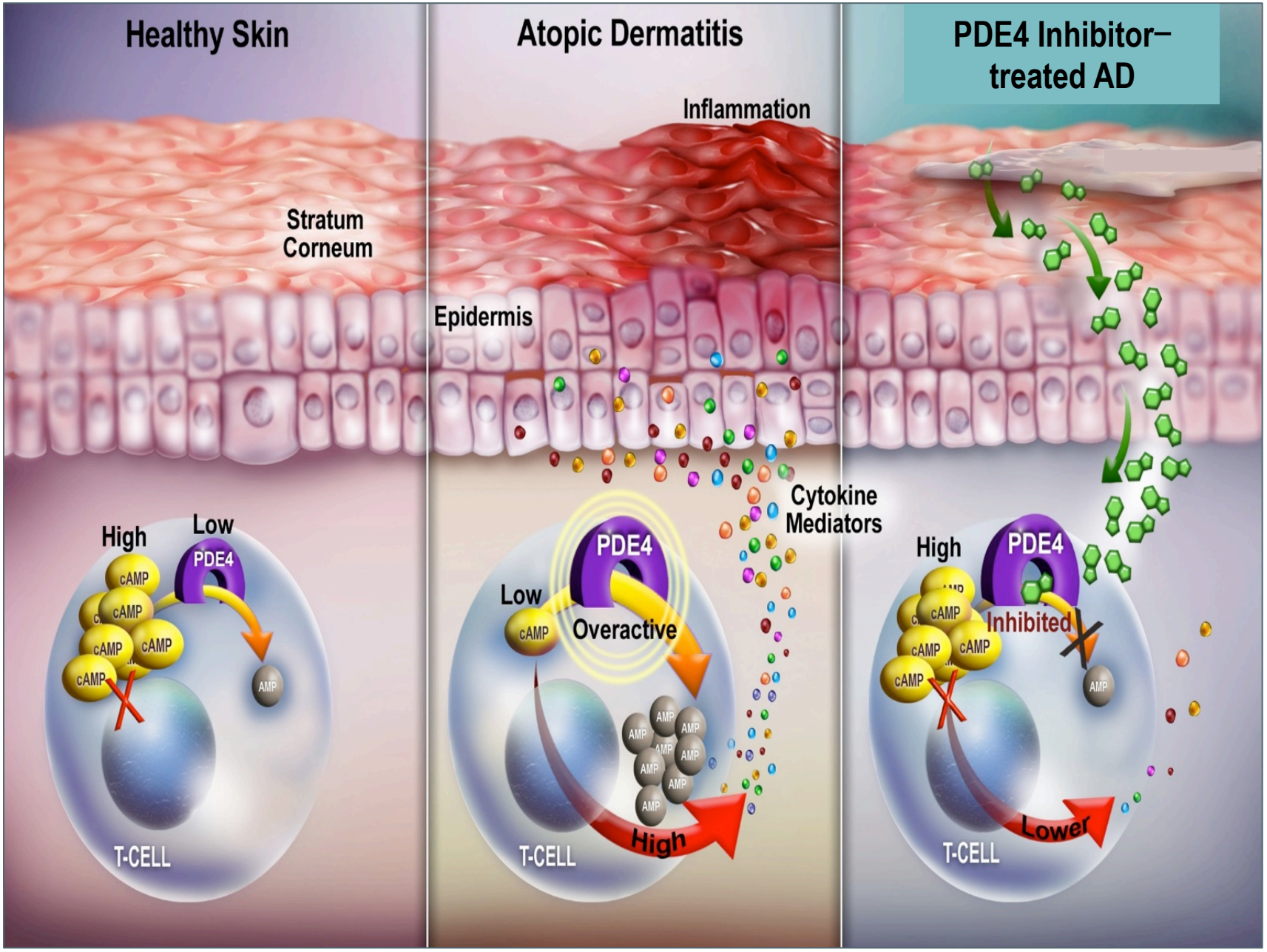
- Not indicated for use in children <2 years of age
- Not indicated for long-term continuous therapy
- Second-line agents
- Limited range of vehicles available vs TCSs
- Stinging and burning in a small subset of patients
- FDA-mandated black box warning and medication guide
- The only time in FDA history that a black box was given for potential risk

Eichenfield LF, et al. *J Am Acad Dermatol*. 2014;71:116-132.
Stein SL, et al. *JAMA*. 2016;315:1510-1511.

Phosphodiesterase Type 4 (PDE4)

- Elevated in patients with AD compared with control patients[1]
- Reduces intracellular cyclic adenosine monophosphate (cAMP) and suppresses protein kinase A, leading to increased levels of proinflammatory cytokines[1]
- Topical and oral PDE4 inhibitors currently under clinical investigation

1. Hanifin JM, et al. *J Invest Dermatol*. 1996;107:51-56.



Jarnagin K, et al. *J Drugs Dermatol*. 2016;15(4):390-396.

Crisaborole Topical Ointment

- A nonsteroidal, boron-based PDE4 inhibitor
- Approved for mild to moderate AD in adults and children ≥2 years in December 2016
- Reduces inflammation and itching
- Maintains skin barrier

Crisaborole Topical Ointment

	AD-301 (Crisaborole/Vehicle) N = 503/256	AD-302 (Crisaborole/Vehicle) N = 513/250
Primary Efficacy Endpoint[1] • Percentage of patients who achieved success in ISGA (defined as score of 0 [clear] or 1 [almost clear] with a minimum 2-grade improvement) at Day 29	**32.8%/25.4%** (*P* = .038)	**31.4%/18.0%** (*P* < .001)
Secondary Efficacy Endpoint[1] • Percentage of patients achieving ISGA clear (0) or almost clear (1) at Day 29	**51.7%/40.6%** (*P* = .005)	**48.5%/29.7%** (*P* < .001)

50% of patients treated with crisaborole achieved improvement in pruritus by 1.37 days (compared with 1.73 days for the vehicle group, *P* = .001)[2]

ISGA, Investigator's Static Global Assessment.

1. Paller AS, et al. Presented at: Fall Clinical Dermatology Conference; October 1-4, 2015; Las Vegas, NV.
2. Hebert AA, et al. Presented at: Fall Clinical Dermatology Conference; October 1-4, 2015; Las Vegas, NV,

Crisaborole 48-Week Safety Study

- Open-label study (after Phase 3), 517 patients
- Disease severity assessed every 4 weeks using ISGA scale
- Patients received 4-week cycles of crisaborole as needed
- Safety measures: local tolerability, adverse events, serious adverse events, clinical laboratory results, vital signs, physical examinations

Eichenfield LF, et al. Presented at: 13th ESPD; May 26, 2016; Paris, France.

Crisaborole Safety Profile

- Favorable safety profile over 48-week study
  - Treatment-related TEAEs in ≥1% of patients: AD (3.1%), application site pain (2.3%), application site infection (1.2%)[1]
  - TEAEs in at least 5% of patients: AD (11.2%), upper respiratory tract infection (10.3%), nasopharyngitis (7.7%), cough (6.8%), and pyrexia (5.6%)[1]
  - Limited systemic exposure[2]
  - No atrophy, telangiectasia, hypopigmentation

TEAE, treatment-emergent adverse event.

1. Eichenfield LF, et al. Presented at: Winter Clinical Dermatology Conference; January 15-20, 2016; Koloa, HI.
2. Tom WL, et al. *Pediatr Dermatol*. 2016;33(2):150-159.

Dupilumab

- Approved March 2017
- Injectable biologic therapy
- Blocks cytokines IL-4 and IL-3
- Indicated for adults with moderate to severe AD

Beck LA, et al. *N Engl J Med*. 2014;371:130-139.

Controlling the Itch

- Frequent moisturization to reduce dryness
- Apply low- to mid-potency TCSs to control inflammation
- Antihistamines are not effective at alleviating itching
  - Sedating antihistamines can be used to improve sleep

Tollefson MM, et al. *Pediatrics*. 134(6):e1735-e1744.

Antihistamines in Atopic Dermatitis

Agent	Vehicle	Properties
Pramoxine	**C, L**	Topical anesthetic – blocks nerve conduction and impulses by inhibiting depolarization of neurons
Diphenhydramine*	**Oral**	Sedating antihistamine
Hydroxyzine	**Oral**	Sedating antihistamine
Doxepin	**Oral, L**	Sedating antihistamine
Cetirizine*	**Oral**	Non-sedating antihistamine

C, cream; L, lotion.
*Available over the counter.

Dilute Bleach Baths

- Mechanism of action still unclear
  - Anti-inflammatory actions or suppression of *S. aureus* overgrowth?[1]
- Preparation: ⅛ -½ cup of bleach per standard bathtub, at least 2 times per week[2]
- Supporting evidence: see references 1-3

1. Hon KL, et al. *J Dermatolog Treat*. 2016;27:156-162.
2. Huang JT, et al. *Pediatrics*. 2009;123:e808-e814.
3. Wong SM, et al. *J Dermatol*. 2013;40:874-880.

Case 3

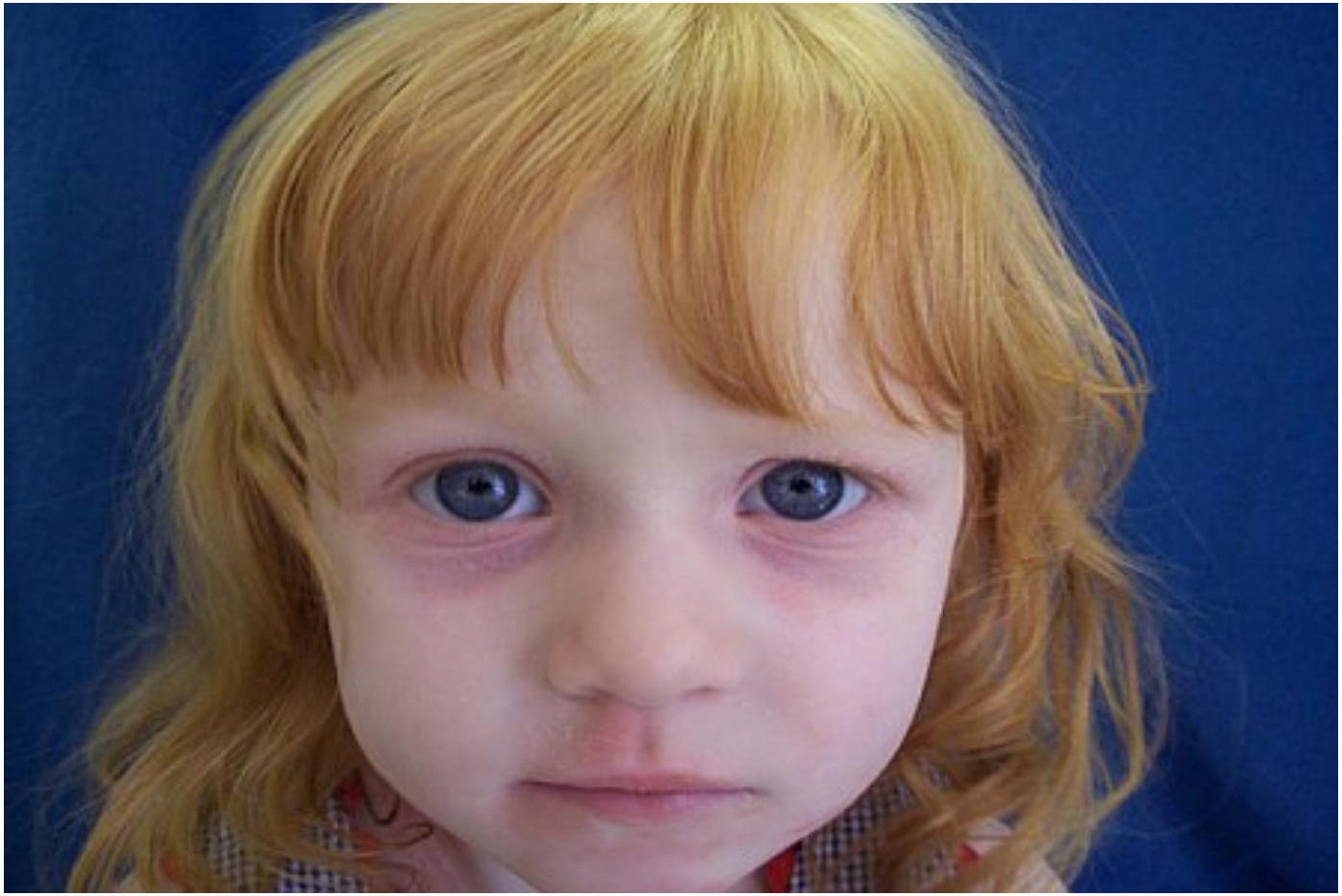


Photo courtesy of Adelaide A. Hebert, MD

Case 3

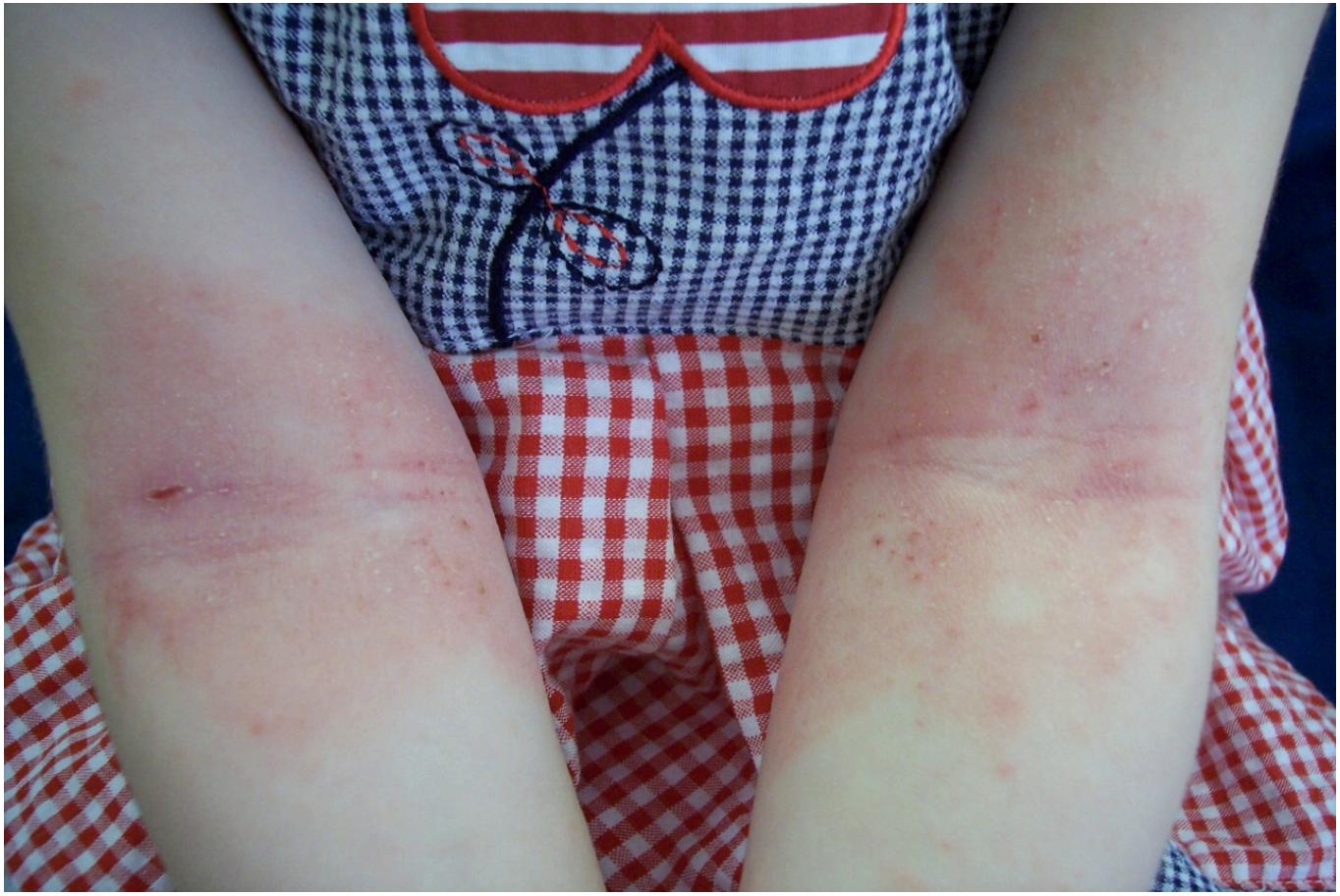


Photo courtesy of Adelaide A. Hebert, MD

Case 3

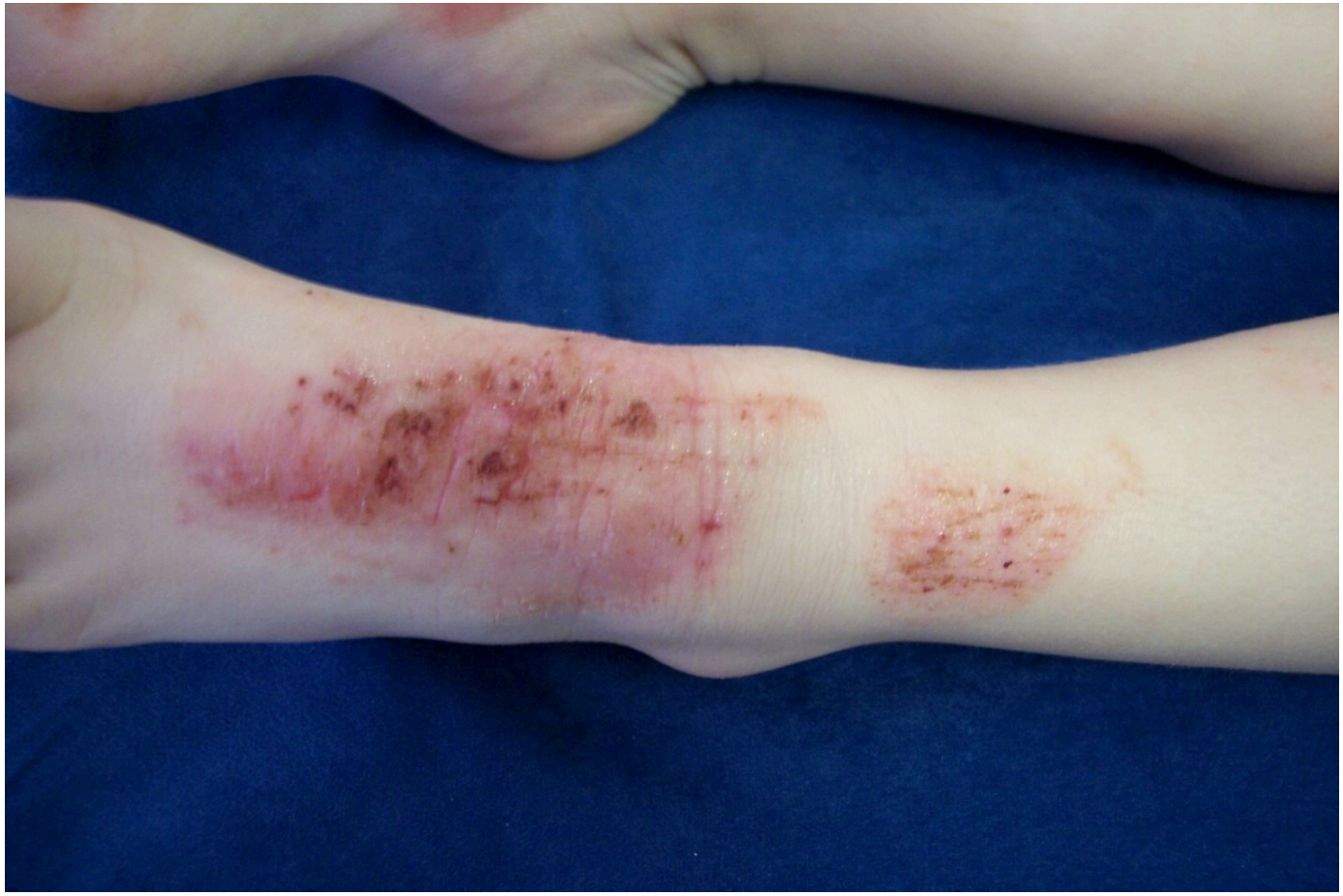


Photo courtesy of Adelaide A. Hebert, MD

Case 3: Therapy Recommendations

Initial therapy

- Disease control
  - Face: low-potency TCS or TCI (A,I)
  - Body: mid-potency TCS, with or without wet wraps (A,I)
- Emollients 2-3 times per day (A,I)
- Antihistamines if necessary/desired*
- Dilute bleach baths if skin is red and crusted (B,II)

After-flare control

- Intermittent treatment with TCS, TCI (A,I)

*Against use of systemic antihistamines: sedating C, III, and nonsedating A, II.

Eichenfield LF, et al. *J Am Acad Dermatol*. 2014;71:116-132.
Sidbury R, et al. *J Am Acad Dermatol*. 2014;71:327-349.
Stein SL, et al. *JAMA*. 2016;315:1510-1511.

Patient and Caregiver Education

- Written treatment plan increases likelihood of adherence
- Moisturize frequently throughout the day
- Topical medications do not take the place of moisturizers
- Continue maintenance therapies, even if skin “appears” healthy
- Appearance of AD changes with age

Specialty Referral

- Early referral in the case of severe, persistent disease
- Otherwise, refer if the patient is not responding to conservative measures and standard treatment modalities
- For food allergy evaluation for milk, egg, peanut, wheat, and soy if at least 1 of the following conditions is met:
  - Persistent AD in spite of optimized management and topical therapy
  - Reliable history of immediate reaction after ingestion of a specific food

Eczema Herpeticum

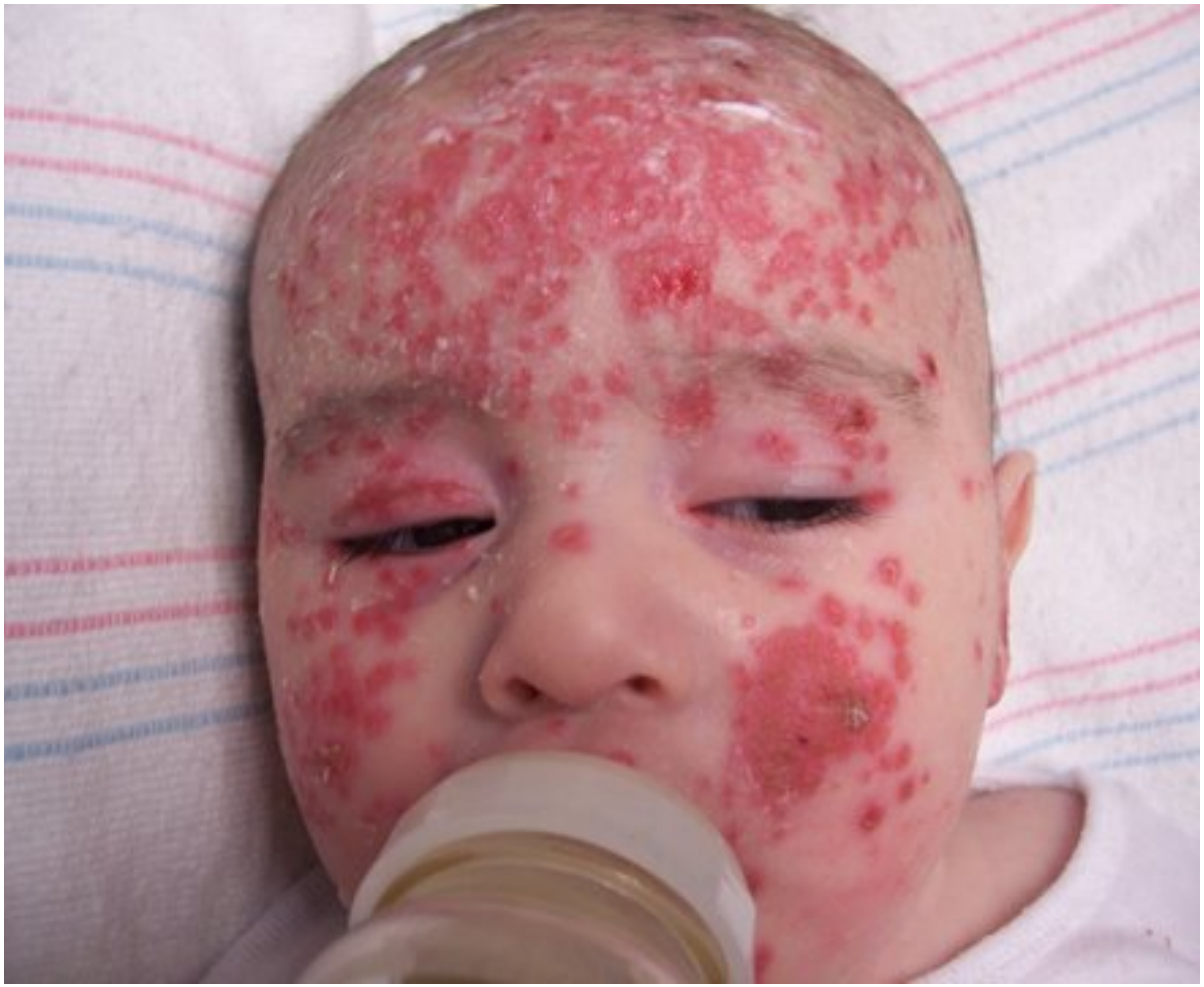


Photo courtesy of Adelaide A. Hebert, MD

Eczema Herpeticum

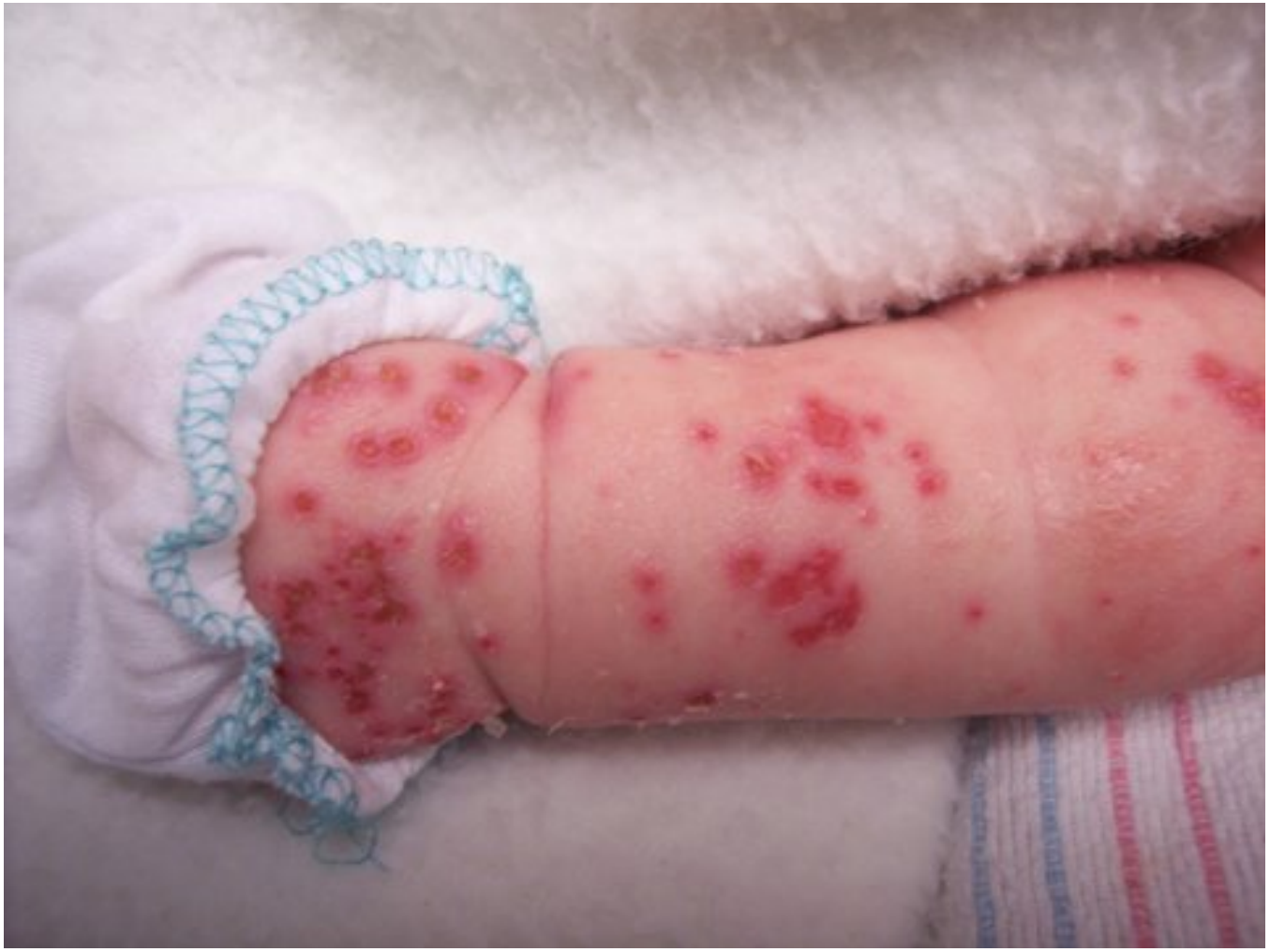


Photo courtesy of Adelaide A. Hebert, MD

Emerging Therapies for Atopic Dermatitis

Other Emerging Treatments for Atopic Dermatitis

- Topical therapies
  - PDE4 inhibitors (eg, roflumilast,[1] OPA-15406[2])
  - Janus kinase inhibitors: tofacitinib ointment[3]
    - Phase 2 trial results (2% ointment, BID)
      - EASI score for tofacitinib -87.7% (compared with -29.9% with vehicle, *P*<0.001)
      - Significant improvements in EASI, PGA, and BSA by week 1, pruritus by day 2 with tofacitinib
      - More AEs were observed for vehicle (55.9%) vs tofacitinib (31.4%)
      - No patients treated with tofacitinib discontinued treatment due to AEs
      - No SAEs were reported in either group
      - Fewer TEAEs reported for tofacitinib (5.7%) vs vehicle (11.8%)
  - Calcineurin inhibitor: SB011[4]

EASI, Eczema Area and Severity Index; PGA, Physician's Global Assessment; BSA, Body Surface Area; AE, adverse event; SAE, serious adverse event; TEAE, treatment-emergent adverse event.

1. ClinicalTrials.gov Identifier: NCT01856764. 2. ClinicalTrials.gov Identifier: NCT01702181.
3. Bissonnette R, et al. *Br J Dermatol*. 2016 Jul 16. [Epub ahead of print]. 4. ClinicalTrials.gov Identifier: NCT02079688.

Other Emerging Treatments for Atopic Dermatitis

- Systemic therapies
  - Apremilast: an oral PDE4 inhibitor[1]
- Other new agents on the horizon that look promising

1. ClinicalTrials.gov Identifier: NCT02087943.

Clinical Pearls

- Do not undertreat the disease
- Stress the importance of moisturization in disease control to patients/parents
- Control infection/colonization
- Oral steroids are very rarely indicated in the treatment of AD

Questions

Thank You

Please complete the **postassessment and evaluation** located in your meeting handout.