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PEDIATRIC EBOC
EVIDENCE BASED OUTCOMES CENTER

Atopic Dermatitis Guideline

Definition

Atopic dermatitis is a dermatologic condition that usually occurs in infancy or childhood. The hallmark of atopic dermatitis is pruritus, and it is commonly called "the itch that rashes" as rubbing or scratching the skin can initiate a flare. The rash tends to occur on the face, scalp, trunk, and extensor surfaces in infancy and then transitions to flexural areas of extremities in childhood. The eruption of acute and subacute atopic dermatitis appears as small pink papules coalescing into pink plaques often with vesicles, weeping, crust, and scale. Chronic atopic dermatitis tends to appear as lichenified plaques with dyspigmentation; this is secondary to chronic scratching of the skin. Individuals with atopic dermatitis often have a family history of such, and this condition is commonly seen in conjunction with asthma and allergies (atopic triad). The skin of atopic dermatitis is compromised and thus predisposed to bacterial and viral superinfection.

Epidemiology

- Most common chronic inflammatory dermatologic condition in children, affecting ~ 10% of children in the US[1]
    - 60% present within the first year of life
    - 90% present by age five
    - Affects males and females almost equally[2]
- Majority of children have mild disease[2]
- Increased prevalence in African Americans [2]

Etiology & Pathogenesis

- Three components of pathogenesis:[2]
    - Epidermal barrier dysfunction
    - Immune dysregulation barrier
    - Alteration of skin microbiome
- Genetic predisposition in ~90% early onset AD
    - Fillagrin gene mutations
- Often associated with elevated IgE and personal/family history of atopy (i.e. tendency towards other allergic diseases such as asthma and allergic rhinitis)

Guideline Inclusion Criteria

- Pediatric patients with atopic dermatitis

Guideline Exclusion Criteria

- Patients without atopic dermatitis

Differential Diagnosis

- Seborrheic dermatitis
- Allergic contact dermatitis
- Irritant contact dermatitis
- Psoriasis
- Tinea corporis
- Scabies
- Pityriasis rosea
- Ichthyoses
- Cutaneous T cell lymphoma
- Congenital immunodeficiencies

Diagnosis of Atopic Dermatitis:

Atopic dermatitis (AD) is a clinical diagnosis dependent on rash morphology and patient history.

The **morphology** of the rash changes with acuity:

1. In the acute phase, the rash appears as clustered vesicles and erosions, sometimes with weeping, on a background of erythema. In dark-skinned patients, the erythema can be difficult to appreciate.
2. In the subacute phase, the rash appears as scaly pink papules coalescing into plaques, with or without erosions.
3. In the chronic phase, the rash appears as scaly hyper- or hypopigmented plaques with lichenification (thickening of skin with accentuation of normal skin lines).

The classic **distribution** of the rash changes with age of the patient. **Infants** tend to have AD on the face, scalp, torso, and extensor surfaces of extremities. **Children and adults** are more likely to have it on the flexural surfaces of extremities (antecubital fossae, popliteal fossae).

Pruritus is the hallmark symptom of AD and is necessary for diagnosis. In addition, a personal history of sensitive skin, xerosis, or other atopic disorders (i.e. asthma or allergies) as well as a family history of AD support the diagnosis.

Diagnosis of Superinfection:

The skin of atopic dermatitis is more susceptible to bacterial and viral infections. This is multifactorial and likely due to decreased levels of antimicrobial peptides within the plaques of AD in combination with an inherently compromised skin barrier. This faulty skin barrier is further weakened by repetitive excoriations which facilitates pathogen entry.

Bacterial superinfection: Eczematous plaques may become secondarily impetiginized. This is often a **clinical diagnosis** and appears commonly as areas of honey-crusting within excoriated or

eroded eczematous plaques. Less commonly there may be warmth or increased erythema. **Bacterial culture** can be helpful to confirm this diagnosis.

Viral superinfection: The most common viral superinfection to occur within AD plaques is due to the herpes virus (most often HSV-1). This condition is known as **eczema herpeticum** and appears as monomorphic, rounded, punched-out vesicles and shallow erosions on background eczema. There may be honey-crusting indicating impetiginization as well. Usually the patient appears non-toxic, however this condition can be very serious in infants and young children. Diagnosis is often made clinically, however it can be confirmed with an **HSV viral swab**. Until all of the lesions have crusted over, the patient should be considered to be infectious and at risk for transmitting the virus to others.

Clinical Management:

There is no cure for atopic dermatitis, rather the focus is on **disease management** including trigger avoidance and alleviation of symptoms. The main approach to management is **skin-directed therapies**, which includes 4 major components:

1. A gentle skin care regimen is important to repair and maintain a healthy skin barrier. Ways to maintain skin hydration involve moisturizers, wet wraps, and bathing. **Moisturizers** should be fragrance-free and thick. Ointments and creams are more effective than lotions. Ointments are clear and greasy (i.e. Vaseline or Aquaphor). Creams often come in a jar or a tub and are greasy like ointments but white in color. Examples of good creams for eczema include: Cerave Moisturizing Cream, Cetaphil Moisturizing Cream, Aveeno Eczema Therapy, Vanicream Moisturizing Skin Cream. Both ointments and creams are thick and form a barrier to protect the skin and prevent water loss. This is in contrast to lotions. While lotions spread easily and dry quickly, they are less preferable as their increased water and alcohol content makes them more drying. Lotions often come in a tall bottle with a pump. Moisturizers should be applied immediately after bathing while the skin is still wet and frequently throughout the day. **Wet wraps** are based on a similar principle. To perform a wet wrap, the patient or parent should first apply the topical steroid or moisturizer to the area of eczema. This is then occluded by a damp roll of gauze or damp cotton garments, such as pajamas, followed by a layer of dry garments on top. The dry layer can be another pair of pajamas or even a sweatsuit. Both garments are left in place for 2-3 hours then removed at which time an additional layer of moisturization is applied. This can improve hydration, absorption of topical steroids, and pruritus. **Bathing** should take place in warm (not hot) water with gentle, fragrance-free soaps and cleansers (Ex. Dove sensitive skin bar, Cerave cleansing bar or body wash, Cetaphil gentle cleansing bar). Soaps and cleansers should only be applied to the areas of the body that become malodorous, i.e. the axilla, groin, genitals, buttocks. Applying soap to the rest of the body is not necessary unless it is visibly soiled, and doing so strips the skin in these areas of its natural moisturizers. After bathing, the skin should be patted semi-dry and never rubbed or scrubbed. Immediately after bathing, while the skin is still damp, it is crucial to apply a thick moisturizer to lock in the hydration and prevent the water from evaporation, which leads to the increased dryness and irritation that many patients associate with bathing.

2. Anti-inflammatory medications treat eczema by decreasing the immune response in the skin. **Topical steroids** are frequently used, and the choice of agent depends on the severity and body surface area involved. **During flares** it is best to use the topical steroid twice daily. **For maintenance** and **prevention of flares** it is recommended to use the topical steroid two to three times weekly (i.e. once daily on Monday, Wednesday, Friday) to "hot spots" which are areas of the body that frequently flare. This can help keep the eczema at bay and prevent more frequent, severe flares. A helpful way to think about this is to imagine eczema as a fire. When the fire is big and dangerous, you should throw water on it aggressively. In the same way, when AD is flaring, one should treat it more aggressively with twice daily topical steroid use. However, even when the fire is calm, it is best to sprinkle water on it every so often to keep it at bay. Similarly, even when AD is not flaring, it is prudent to "sprinkle" some topical steroids on the "hot spots" a few times per week to keep it calm. In general, it is recommended to avoid twice daily use for longer than two weeks at a time as long-term, uninterrupted steroid use can have side effects (skin thinning, dyspigmentation and stretch marks). That being said, many patients with severe atopic dermatitis do need to use their topical steroids for longer than two weeks at a time, and many do not develop any unwanted side effects. In general, if the patient needs to continue twice daily use for more than two weeks, it is best to take a break for 5-7 days then restart the course. Alternatively, they can also use non-steroidal options during their break periods. **Non-steroidal topical agents** do not cause the same unwanted side effects that topical steroids can cause. They are good options to use for maintenance and prevention of flares, during break periods from topical steroids, and for sensitive areas of skin such as the face, groin, and axillae. **Topical calcineurin inhibitors** (i.e. pimecrolimus, tacrolimus) are non-steroidal agents that are very useful in atopic dermatitis, however they often require prior authorization and can occasionally burn with application. In addition, the **topical PDE-4 inhibitor crisaborole** is another non-steroidal option. Light therapy in the form of narrowband UVB is a good treatment option for those with more severe or recalcitrant disease, however this is usually done in the office and requires two to three visits per week which is difficult for children. Lastly, in severe cases of atopic dermatitis, systemic agents such as dupilumab, methotrexate, cyclosporine, and others are required to manage disease.

3. Controlling pruritus is an important component of atopic dermatitis and is a crucial component of quality of life for patients with AD. **Sedating antihistamines** such as diphenhydramine or hydroxyzine are helpful for itching that interferes with sleep. Non-sedating antihistamines are usually ineffective. In addition, a behavioral psychologist can help patients and families discover coping mechanisms and distraction techniques for chronic itching.

4. Managing superinfection with empiric antibiotics and antivirals is a necessary component of AD treatment. For mild, localized bacterial superinfection, treatment with topical mupirocin 2% ointment three times a day for seven to 10 days, or until all the yellow crusted areas have healed, is often sufficient. For more severe impetiginization, treatment with oral antibiotics with good staph and strep coverage (i.e. cephalexin) is required. Empiric coverage for MRSA is usually not necessary unless the patient has a

history of MRSA infections or does not respond to first-line antibiotics. For viral superinfection, treatment with acyclovir or valacyclovir is required.

Prevention:

Counseling patients on maintaining a gentle skin care routine, using topical steroids 2-3x weekly for maintenance, and avoiding triggers are all important components in AD prevention. Each patient has different triggers, however some common ones include season changes, dry climates, temperature extremes, harsh chemicals/soaps, fragrance-containing products, lotions, and rough clothing material like wool.

Atopic Dermatitis Pathway (ED)

DCMC Evidence-Based Outcomes Center

Step #1 Topical Steroids

Mild, moderate, or severe eczema? Use Figure 1a to determine severity

Yes

Treat by body region and severity. Refer to Table 1a/1b

Step #2 Infection

Evidence of bacterial infection (erosions, crusting, pustules, fever, abrupt worsening)? See Figure 1b

No → **Evidence of viral infection (vesicles or small, punched-out erosions)?** See Figure 1b

No → Go to Step #3

Yes

Swab contents of vesicle by deroofing intact vesicle. Otherwise, swab an open, wet erosion. Send for order "HSV 1/2 Amplification Cutaneous/ Mucocutaneous" (see picture to right)

Signs of systemic infection?

Yes → Consider hospital admission, IV acylovir, and +/- Derm consult See Table 2a for dosing.

No

Consider discharge with topical steroids and PO antivirals. See Table 2a for dosing.

→ Go to Step #3

Yes

Swab purulent material if present, otherwise swab open, wet erosion Send for order "Culture Bacterial Aerobic No Smear"

Signs of systemic infection?

No → Consider topical steroids and topical vs PO antibiotics See Table 2b and Table 4.

Yes

Consider hospital admission, coverage for MRSA, and IV antibiotics See Table 2b for dosing.

Step #3 Itch

Itching disruptive to sleep and/or activities of daily living?

No → Go to Step #4

Consider trial of first generation antihistamines See Table 2c for dosing.

Step #4 Gentle Skin Care

Is the patient following a gentle skin care routine?

Yes → Go to Step #5

No

Instruct patient/family to use lukewarm water when bathing, mild fragrance-free soap (i.e. Dove Sensitive Skin bar), and thick moisturizing cream or ointment (Vaseline, Aquaphor, Cerave) applied immediately after bath and multiple times throughout day. *Please reference patient discharge materials for more information.*

Step #5 Triggers

Instruct patient/family to avoid triggers such as:

- harsh soaps/chemicals
- products with fragrance/preservatives/alcohols
- wool clothing
- temperature extremes

Note: Please review Table 3 to determine appropriate referral. Provide patient with AD discharge education materials and encourage them to reference the educational video which is accessible via the QR code on their paperwork.

Updated August 9, 2021

Atopic Dermatitis Pathway

Figure 1a. Atopic Dermatitis Severity Atlas

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Mild

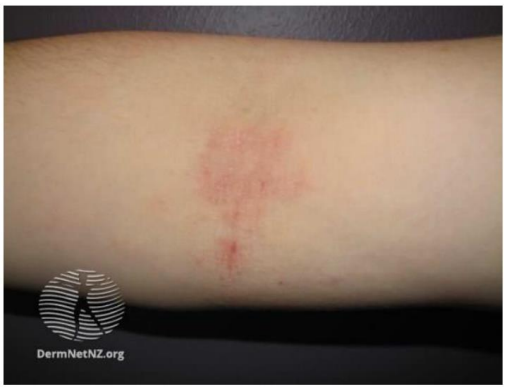


Photo courtesy of DermNet NZ. No changes made.

Moderate

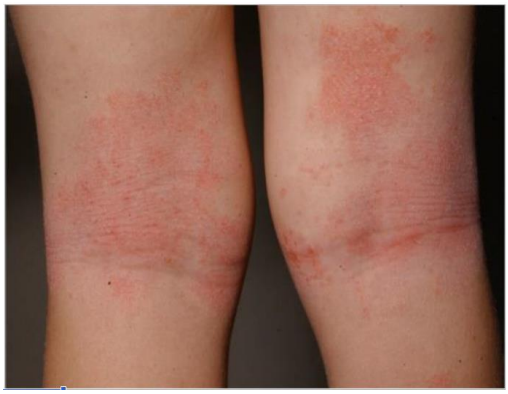
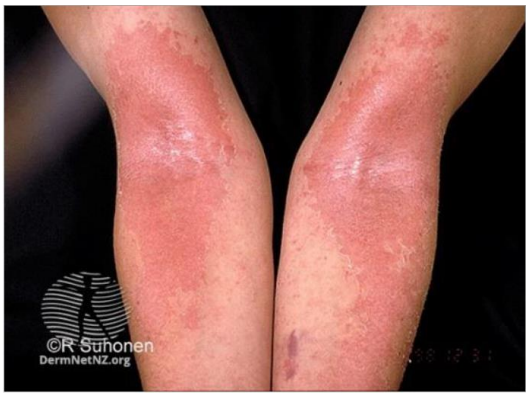


Photo courtesy of DermNet NZ. No changes made.

Severe



Salava A, Lauerma A. Role of the skin microbiome in atopic dermatitis. Clin Transl Allergy. 2014;4:33. Published 2014 Oct 17. doi:10.1186/2045-7022-4-33

Mild

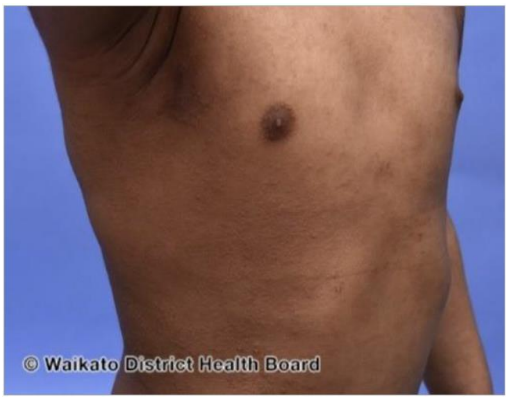
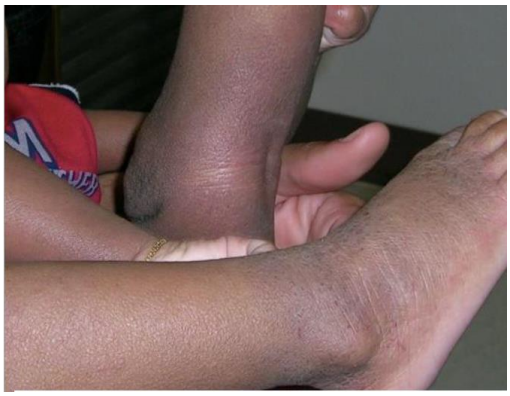


Photo courtesy of DermNet NZ. No changes made.

Moderate



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Severe

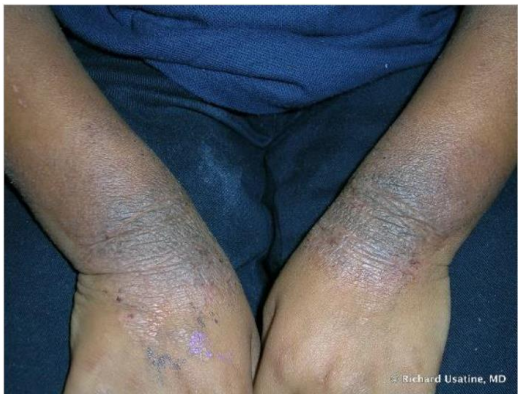

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Figure 1b. Atopic Dermatitis Superinfection Atlas

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Viral Superinfection (Eczema Herpeticum)

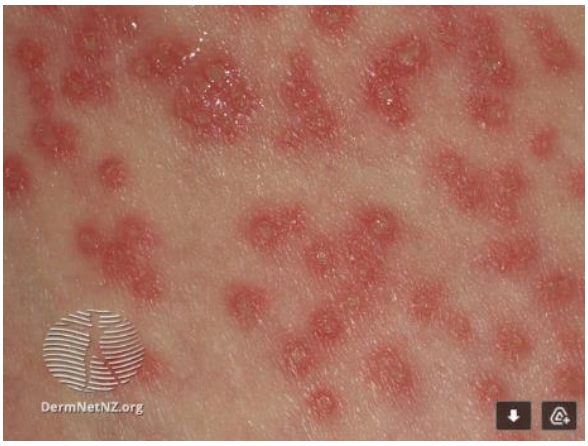

Photo courtesy of DermNet NZ. No changes made.

Bacterial Superinfection

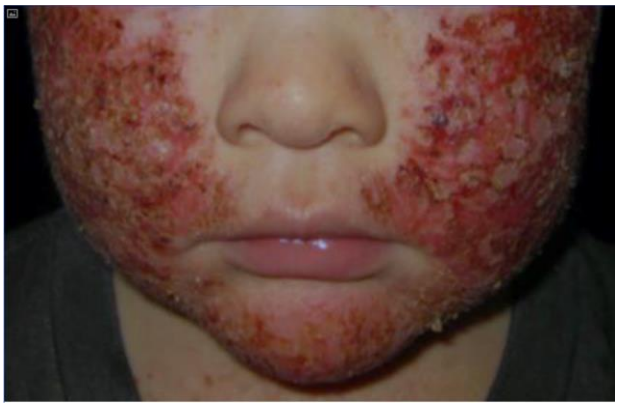

Image courtesy of the American Academy of Dermatology. All Rights Reserved.

Atopic Dermatitis Pathway

Description of Severity

Determine Severity of AD Flare

Severity is determined by skin findings in addition to the patient's level of discomfort which can impact quality of life.

	Skin Findings	Quality of Life
Mild	Patches of scaly pink to red skin Mild, intermittent itching	Little impact on daily activities or sleep
Moderate	Multiple patches of scaly pink to red skin Frequent itching Excoriation or localized areas of thickened skin may be present	Some impact on daily activities and sleep due to frequent itching and skin discomfort
Severe	Widespread scaly skin Incessant itching Open, cracked areas of skin Lichenified skin Bleeding, oozing Skin color changes	Limits daily activities Impacts sleep and can affect daily mood

Determine Severity of AD Flare. In: Pathway for Evaluation and Treatment of Suspected Atopic Dermatitis. https://www.chop.edu/clinical-pathway/atopic-dermatitis-determine-severity-flare. Posted: October 2018 Authors: L. Castelo-Soccio MD; K. Woo Castelo CRNP; M. Jen MD; C. Tucker MD; K. Gupta MD; C. Yun MD; J Hart MD; E. Delgado MD; L. Wilson RN; B. Johnson RN; A. Verma MD; M. Perman MD; J. Treat MD The Children's Hospital of Philadelphia Philadelphia, PA 19104

	Skin Findings
Viral Superinfection (Eczema Herpeticum)	Round, punched-out, shallow erosions
Bacterial Superinfection	Erosions or ulcerations with erythema, honey-crusting, warmth, +/- purulence

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Table 1a: ABBREVIATED STEROID TABLE FOR PATIENTS USING DCMC PHARMACY:

Severity	Location	Age<2 y/o[a]	Age>2y/o[a]	Potency
Mild	Face/axilla/genitals	Hydrocortisone acetate 2.5%	Hydrocortisone acetate 2.5%	Low
	Body	Hydrocortisone acetate 2.5%	Hydrocortisone acetate 2.5%	Low
Moderate	Face/axilla/genitals	Hydrocortisone acetate 2.5%	Hydrocortisone acetate 2.5%	Low
	Body	Triamcinolone 0.025%	Triamcinolone 0.1%	Medium
Severe	Face/axilla/genitals	Hydrocortisone acetate 2.5%	Triamcinolone 0.025%	Low-medium
	Body	Triamcinolone 0.1%	Triamcinolone 0.1% Mometasone 0.1%	Medium-high

[a] When multiple per category, steroids are listed in order of increasing strength.

Table 1b: ABBREVIATED STEROID TABLE FOR OUTPATIENT PHARMACY USE:

Severity	Location	Age<2 y/o[a]	Age>2y/o[a]	Potency
Mild	Face/axilla/genitals	Hydrocortisone acetate 2.5%	Hydrocortisone acetate 2.5% Desonide 0.05%	Low
	Body	Hydrocortisone acetate 2.5% Desonide 0.05%	Hydrocortisone acetate 2.5% Desonide 0.05% Triamcinolone 0.025%	Low
Moderate	Face/axilla/genitals	Hydrocortisone acetate 2.5% Desonide 0.05%	Hydrocortisone acetate 2.5% Desonide 0.05% Triamcinolone 0.025%	Low
	Body	Triamcinolone 0.025%	Triamcinolone 0.1% Fluticasone 0.005%	Medium
Severe	Face/axilla/genitals	Hydrocortisone acetate 2.5% Desonide 0.05% Triamcinolone 0.025%	Desonide 0.05% Triamcinolone 0.025%	Low-medium
	Body	Triamcinolone 0.1%	Triamcinolone Mometasone 0.1% Betamethasone dipropionate 0.05% Clobetasol 0.05%	Medium-high/ Very High

[a] When multiple per category, steroids are listed in order of increasing strength.

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Table 1c: QUICK TIPS FOR PRESCRIBING TOPICAL STEROIDS:

Strength	• If a patient is using high potency steroid and flaring, do not decrease strength if possible. Send a refill of the high potency steroid to *outside* pharmacy **OR** prescribe triamcinolone 0.1% in a 454 g jar, especially if there is extensive skin involvement.
Formulation	• OINTMENT>cream>lotion • Ointments are the most effective vehicle for topical steroid use in atopic dermatitis, however some patients may have an aversion to the texture and prefer a cream instead.

Available/ Suggested Quantities	For diffuse BSA: 454 g (only available for hydrocortisone 2.5% and triamcinolone 0.1%)
	For localized BSA: 30-60 g
	• DCMC
	  ○ Hydrocortisone 2.5%: 28.35 g, 454 g
	  ○ Triamcinolone 0.025%: 80 g
	  ○ Triamcinolone 0.1%: 80 g, 454 g
	  ○ Mometasone 0.1%: 45 g
	• Retail pharmacies
	  ○ Hydrocortisone 2.5%: 28.35 g, 454 g
	  ○ Desonide 0.05%: 60 g
	  ○ Triamcinolone 0.025%: 80 g
	  ○ Triamcinolone 0.1%: 80 g, 454 g
	  ○ Fluticasone 0.005%: 30 g, 60 g
	  ○ Mometasone 0.1%: 45 g
	  ○ Betamethasone valerate 0.05%: 45 g
	  ○ Clobetasol propionate 0.05%: 30 g, 45 g, 60 g

Sig	• Apply twice daily to affected areas until clear then 2-3x weekly for maintenance

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Table 2a: Antiviral Treatment for HSV Superinfection (i.e. Eczema Herpeticum)

Drug [a, b]	Route	Dosing	Max Dosage
Acyclovir[a]	IV	>3 mo age: 5 mg/kg/dose q8hrs x 5-7 days	1000mg/dose
Acyclovir	Oral	20mg/kg/dose 4x/day x 5-7 days	800mg/dose, 3200mg/day
Valacylovir[b]	Oral	20mg/kg/dose BID x 5-7 days	1000mg/dose

[a] For patients <3 mo age: 20 mg/kg/dose IV q8hrs x14 days due to risk of complications/CNS involvement.
If signs of systemic infection consider IV acyclovir. Maintain adequate hydration while using IV acyclovir. Consider IV fluids (5-10cc/kg bolus or maintenance rate continuous fluids) prior to IV acyclovir use, and monitor renal function.
Dosing in this table is for patients with normal renal function. Please contact pharmacy for assistance with dosing in renal insufficiency. Acyclovir Dosing Guide for DCMC

[b] There is limited data for use of valacyclovir in patients <2 years of age. Acyclovir is the preferred drug in this age group.

Table 2b: Antibiotic Treatment for Bacterial Superinfection

Indication	Drug [a, b, c, d]	Route	Dose	Max dose
Suspected MSSA	Cephalexin[a]	PO	25mg/kg/dose q8hrs x 5-7 days	1000mg/dose
	Cefazolin	IV	33mg/kg/dose q8hrs x 5-7 days	6g/day
Suspected MRSA	Clindamycin, [b]	PO	10mg/kg/dose q8hrs x 5-10 days	450mg/dose
	Clindamycin[c]	IV	13mg/kg/dose q8hrs x 7-10 days	600mg/dose
	Doxycycline[d]	PO	≤ 40kg: 2.2mg/kg/dose q12hrs x 5-10 days >40 kg: 100mg BID x 5-10 days	
	Bactrim	PO	5mg/kg/dose (for TMP component) q12hrs x 7-10 days	320mg TMP/dose
Localized Infection	Mupirocin	Topical	TID x 10-14 days	NA

[a] Clindamycin is the preferred empiric choice for purulent infections or personal/family history of MRSA.

[b] Round to nearest cap size (75mg, 150mg, 300mg). Caps may be opened and contents sprinkled into food (i.e. pudding, applesauce, yogurt). Also available as a 15 mg/mL oral solution.

[c] If signs of systemic infection, consider IV Vancomycin (until culture and sensitivity information is available).

[d] Do not use in children < 8 years of age.

Hospital Antibiogram link

Table 3c: Anti-itch Medications-Antihistamines

Drug	Age	Route	Dosing	Max dosage
Hydroxyzine	-	Oral	0.5 mg/kg q6hrs	25mg/dose

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Table 3: Pediatric Dermatology Referral/Consultation Criteria

Routine Outpatient Referral	Expedited Outpatient Referral*	Inpatient/ED Consultation
Severe AD	Severe AD with multiple ED visits	Severe AD
Mild/moderate AD not improving with appropriate PCP management after 8-12 weeks	Severe AD not improving with appropriate PCP management after 8-12 weeks	Moderate persistent AD not responding to appropriate treatments
Mild/moderate AD requiring multiple ED visits	Severe AD with superinfection not improving on appropriate therapy	Diagnosis in question
AD with recurrent superinfection		AD with severe or extensive superinfection or any superinfection not improving on appropriate therapy
		AD with immunosuppression

*To request an expedited referral:
During clinic hours: Call SFC Pediatric Dermatology nurse line (512-628-1920, option 4).
Outside clinic hours: Page after hours dermatology (512-203-1210).

Table 4: Criteria for Treatment of Bacterial Superinfection with Oral Antibiotics

Infection not responding to topical antibiotics
Signs/symptoms of systemic infection
Superinfection involving large body surface area that is not amenable to topical application
Immunosuppression

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Methods

Existing External Guidelines/Clinical Pathways

Existing External Guideline/Clinical Pathway	Organization and Author	Last Update
Pathway for Evaluation/Treatment of Suspected Atopic Dermatitis	Children's Hospital of Philadelphia	October 2018

Any published clinical guidelines have been evaluated for this review using the **AGREE II criteria**. The comparisons of these guidelines are found at the end of this document. **AGREE II criteria** include evaluation of: Guideline Scope and Purpose, Stakeholder Involvement, Rigor of Development, Clarity of Presentation, Applicability, and Editorial Independence.

Review of Relevant Evidence: Search Strategies and Databases Reviewed

Search Strategies	Document Strategies Used
Search Terms Used:	Atopic dermatitis, eczema
Years Searched - All Questions	1980-2020
Language	English
Age of Subjects	0-18 years old
Search Engines	PubMed
EBP Web Sites	UpToDate
Professional Organizations	American Academy of Dermatology
Joint Commission	
Government/State Agencies	None
Other	

Evidence Found with Searches

Check Type of Evidence Found	Summary of Evidence – All Questions
☐	Systematic Reviews
☐	Meta-analysis articles
x	Randomized Controlled Trials
x	Non-randomized studies
x	Review articles
☐	Government/State agency regulations
x	Professional organization guidelines, white papers, ect.

Evaluating the Quality of the Evidence

The GRADE criteria were used to evaluate the quality of evidence presented in research articles reviewed during the development of this guideline. The table below defines how the quality of evidence is rated and how a strong versus a weak recommendation is established.

Recommendation	
Strong	Desirable effects clearly outweigh undesirable effects or vice versa
Weak	Desirable effects closely balanced with undesirable effects
Type of Evidence	
High	Consistent evidence from well-performed RCTs or exceptionally strong evidence from unbiased observational studies
Moderate	Evidence from RCTs with important limitations (e.g., inconsistent results, methodological flaws, indirect evidence, or imprecise results) or unusually strong evidence from unbiased observational studies
Low	Evidence for at least 1 critical outcome from observational studies, from RCTs with serious flaws or indirect evidence
Very Low	Evidence for at least 1 critical outcome from unsystematic clinical observations or very indirect evidence

References

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DELL CHILDREN'S
EVIDENCE BASED OUTCOMES CENTER

Approved by the Atopic Dermatitis Pediatric Evidence-Based Outcomes Center Team

Revision History
Original Date Approved: August 9, 2021
Next Review Date: August 2024

Pediatric Atopic Dermatitis EBOC Team:
Ila Sehgal, DO
Hana Paladichuk, MD
Nanditha Shivaprakash, MD
Levy Moise, MD
Lucia Diaz, MD
Sujit Iyer, MD
Carmen Garudo, PM

EBOC Committee:
Lynn Thoreson, DO
Tory Meyer, MD
Sarmistha Hauger, MD
Patricia Click, MSN, RN
Sujit Iyer, MD
Meena Iyer, MD
Amanda Puro, MD

Recommendations

Practice recommendations were directed by the existing evidence and consensus amongst the content experts. Patient and family preferences were included when possible.

Approval Process

EBOC guidelines are reviewed by DCMC content experts, the EBOC committee, and are subject to a hospital wide review prior to implementation. Recommendations are reviewed and adjusted based on local expertise.