Mini Review

iMedPub Journals
www.imedpub.com

Medical and Clinical Reviews
ISSN 2471-299X

2020
Vol. 6 No. 2: 91

DOI: 10.36648/2471-299X.6.2.91

Update on Atopic Dermatitis: Diagnosis, Severity Assessment, and Treatment Selection

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Citation: Mitwally I, Fawzy M (2020) Update on Atopic Dermatitis: Diagnosis, Severity Assessment, and Treatment Selection. Med Clin Rev. Vol. 6 No. 2: 91.

Abstract

Atopic dermatitis (AD), also known as atopic eczema, is a chronic relapsing inflammatory skin condition. Atopic dermatitis may occur in people of any age but often starts in infants aged 2-6 months. Ninety percent of patients with atopic dermatitis experience the onset of disease prior to age 5 years. Seventy-five percent of individuals experience marked improvement in the severity of their atopic dermatitis by age 14 years; The prevalence of atopic dermatitis in children with one affected parent is 60% and rises to nearly 80% for children of two affected parents. Additionally, nearly 40% of patients with newly diagnosed cases report a positive family history for atopic dermatitis in at least one first degree relative Atopic dermatitis persists into adulthood in 20-40% of children with the condition.

Received: April 21, 2020, **Accepted:** May 04, 2020, **Published:** May 11, 2020

Introduction: Pathophysiology

The pathogenesis of AD is not completely understood, however, the disorder appears to result from the complex interaction between defects in skin barrier function, immune dysregulation, and environmental and infectious agents. Skin barrier abnormalities ap- pear to be associated with mutations within or impaired expression of the filaggrin gene, which encodes a structural protein essential for skin barrier formation. The skin of individuals with AD has also been shown to be deficient in ceramides (lipid molecules) as well as antimicrobial peptides such as cathelicidins. The infectious agent most often involved in AD is Staphylococcus aureus (S. aureus), which colonizes in approximately 90% of AD patients., These early effects lead to increased histamine release from IgE- activated mast cells and elevated activity of the T-helper cell mediated immune system. The increased release of vascular mediators (eg, bradykinin, histamine, slow-reacting substance of anaphylaxis [SRS-A]) induces vasodilation, edema, and urticarial which in turn stimulate pruritus and inflammatory cutaneous changes.

Contact irritants, climate, sweating, aeroallergens, microbial organisms, and stress/psyche commonly trigger exacerbations. Food allergens like egg, soy, milk, wheat, fish, shellfish, and peanut, which together account for 90% of food-induced cases of atopic dermatitis in double-blind, placebo-controlled food challenges. Fortunately, many clinically significant food allergies self-resolve within the first 5 years of life, eliminating the need for long-term restrictive diets [1-3].

Stress may trigger atopic dermatitis at the sites of activated cutaneous nerve endings, possibly by the actions of substance P, vasoactive intestinal peptide (VIP), or via the adenyl cyclase cyclic adenosine monophosphate (cAMP) system.

Pathway for Evaluation/Treatment of Suspected Atopic Dermatitis

The pathway for evaluation/treatment of Suspected Atopic Dermatitis is shown in the below figure (**Figure 1**).

History and examination

History and examination of Suspected Atopic Dermatitis is mentioned in the below table (**Table 1**).

Differential diagnosis for AD

Differential Diagnosis for Atopic Dermatitis is mentioned in the below table (**Table 2**).

Workout (Laboratory Studies)

No definitive laboratory tests

No definitive laboratory tests are used to diagnose Atopic Dermatitis (AD). Elevated serum *immunoglobulin E (IgE)* levels and peripheral blood *eosinophilia*. Prick skin testing to common allergens can help identify specific triggers of atopic dermatitis For accuracy, antihistamines must be discontinued for 1 week and topical steroids for 2 weeks prior to testing [4,5].

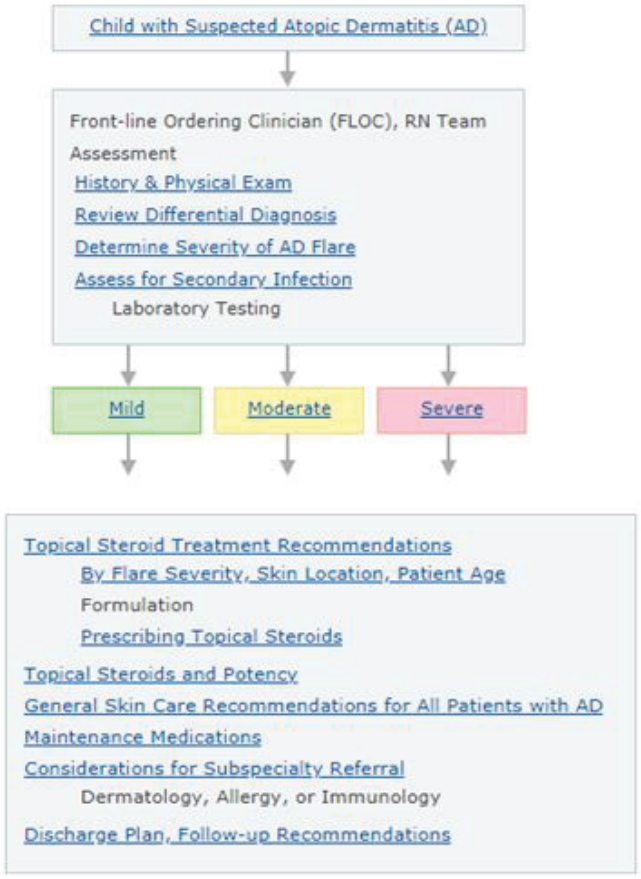

Figure 1 Pathway for Evaluation/Treatment of Suspected Atopic Dermatitis.

Table 1 History and examination of Suspected Atopic Dermatitis.

History	Duration of current symptoms
	Acute, sub-acute, chronic
	Prior history of atopic dermatitis
	Last flare, regions of body usually affected
	Current medications for AD
	Routine skin care
	Bathing, shampoo, lotions, detergents
	Exposures, environment
	Pet dander, new environment, season change, etc.
	Sleep disturbance, behavioral changes
	History of
	Skin infections
	Allergies (food, seasonal, other environmental)
	Asthma
Physical Exam	1. Diagnostic criteria for AD
	Major criteria Patient must have
	An itchy skin condition (or parental/caregiver report of scratching or rubbing in a child)
	Minor criteria
	Plus three or more of the following minor criteria
	History of itchiness in skin creases (e.g., folds of elbows, behind the knees, front of ankles, around the neck)
	Personal history of asthma or allergic rhinitis
	Personal history of general dry skin in the last year
	Visible flexural dermatitis (i.e., in the bends or folds of the skin at the elbow, knees, wrists, etc.)
	Onset under age 2 years
	History of atopic disease in a first-degree relative
	Eczema of cheeks, forehead and outer limbs
	2. Total skin examination
	General Findings
	Keratosis pilaris on the upper arms, thighs and cheeks
	Hyper linear palms
	Darkening around eyes or scaling
	Lesions
	Note color, size, shape, texture, location
	Distribution of lesions
	Infants, toddlers: cheeks, extensor surfaces
	Older children: flexural creases
	Severity
	Mild: pink lesions with thin scale
	Moderate: thicker, more scale, skin may be darker in these areas, often widespread
	Severe: lichenified lesions, often on neck, wrists, ankles Patient may look red head to toe
	Sign of infection
	Crusting, scaling, weeping, erythema, increased pain
	Systemic symptoms

Table 2 Differential diagnosis for AD.

Irritant Dermatitis	Due to drooling, lip licking, diapers, clothing
Contact Dermatitis	Fragrances, metals, plants, chemicals, other
Seborrheic Dermatitis	Characterized by greasy scale on:
	o scalp, eyebrows, central face, neck, chest, axilla, inguinal creases
	Occurs in infants, pre-adolescents, adolescents
	Infants can have an overlap between AD and seborrhea, this combined dermatitis may appear moderate to severe
Psoriasis	In infants, usually affects the diaper area in contrast to AD
	In older children, usually has a thicker scale and affects scalp and extensor surfaces of the skin
Staphylococcal Infection	Peeling skin related to infection
	Impetigo, Staph Scalded Skin
Molluscum Contagiosum	Small skin colored papules with central umbilication caused by a pox virus
	Can develop an underlying dermatitis
Scabies	Infestation causes acute new itchy, widespread dermatitis
	May include folded areas of skin, especially the genital area and interdigital spaces of the hands and feet
Dermatophyte Infections	Can affect the scalp, skin or nails
	Scalp lesions are associated with hair loss, itch, scale or boggi- ness
	Lesions on the body appear as scaly, annular patches

Histologic findings

Acute eczematous lesions show histologic markings of hyperkeratosis, and acanthosis with a decreased or absent granular cell layer.

General Skin Care Recommendations for All Patients with AD

General skin care recommendations for all patients with Atopic Dermatitis is mentioned in the below table (**Table 3**).

Topical Steroids Treatment Recommendations by Flare Severity, Skin Location, Patient Age

Topical Steroids Treatment Recommendations by Flare Severity, Skin Location, Patient Age is mentioned in the below table (**Table 4**).

Maintenance Medications for Outpatient Providers

Two weeks after the patient's initial flare, consider switching to non-steroidal mainte- nance medication for patients who flare frequently such as one of the below (**Table 5**) [6].

Table 3 General Skin Care Recommendations for All Patients with AD.

Bathing	Soak daily in lukewarm water, 5-10 minutes, if child enjoys bathing
	If not, or if irritated skin, do every 2-3 days
	Use gentle, fragrance-free soap
	Avoid bubble bath
	Avoid wipe-down with washcloths, sponges, loofahs, baby wipes
	Pat skin dry after bath, leaving skin damp
Emollients	Moisturize at least twice daily with cream or ointment (not lotion), more often as needed
	Use petroleum jelly, or other fragrance-free moisturizer
	Apply within 3 minutes of bathing
	Apply topical steroid medication 30-60 minutes before emollient
	Application sooner dilutes medication effect
Considerations to decrease irritation	Keep fingernails short
	Use cotton clothing
	Wool, nylon, etc., may irritate the skin
	Avoid fragrances, perfumes around patients
	Use mild detergent
	all® Free and Clear, Tide® Free
	Avoid dryer sheets, fabric softeners
	Give diphenhydramine or hydroxyzine before bed to help sleep, ease itching as needed
Wet wraps	Increase penetration of emollients and topical medicines
	Decrease water loss
	Provide physical barrier against scratching
	Procedure:
	Apply emollient
	Wrap skin with layer of wet bandage or clothing
	Apply dry bandage or clothing over wet layer
Bleach baths (for patients who are prone to superin- fection)	Add 1/4 cup unscented, regular, not concentrated Clorox bleach into 1/2 of regular sized bathtub
	Bathe 5-10 minutes
	1-3 times weekly
	Bleach Bath +

Table 4 Topical Steroids Treatment Recommendations by Flare Severity, Skin Location, Patient Age.

Severity	Location	Age < 3 yrs Ointment covered by most insurance payers	Age ≥ 3 yrs Ointment covered by most insurance payers	Potency Class
Mild	Face/Genital s	2.5% Hydrocortisone	2.5% Hydrocortisone	Lowest Class VII
Mild	Body	2.5% Hydrocortisone	0.025% Triamcino- lone acetonide	Lower- Medium Class VI
Moderate	Face/Genital s	0.025% Triamcino- lone acetonide	0.025% Triamcino- lone acetonide	Lower- Medium Class VI
Moderate	Body	0.025% Triamcino- lone acetonide	0.1% Triamcino- lone acetonide	Medium Class V
Severe	Face/Genital s	0.025% Triamcino- lone acetonide	0.025% Triamcino- lone acetonide	Lower- Medium Class VI
Severe	Body	0.1% Triamcinolone acetonide	0.05% Fluoci- nonide	High Class II

Table 5 Maintenance Medications for Outpatient Providers.

Medication	Age Range	Considerations
Crisaborole 2% (Eucrisa®) Cream	≥ 2 years	Can be used as treatment or maintenance
		Side effect: burning sensation
		Often not covered by insurance or is a step therapy requiring prior authorization
Pimecrolimus (Elidel®) Cream	≥ 2 years	Can be used for 4-6 weeks after acute flare medication
		Side effect: stinging/burning
Tacrolimus (Pro-topic®) Ointment	2-16 years: 0.03% ointment ≥ 16 years: 0.1% ointment	Can be used for 4-6 weeks after acute flare medication
		Side effect: stinging/burning
		Often not covered by insurance or is a step therapy requiring prior authorization
		Keep in refrigerator to reduce burning effect
Anti-IL-4Ra thera- py (dupilumab) Dupilumab	Children >12 years	Sc injection administered every 2 weeks.

Table 6 Considerations for Subspecialty Referral.

Dermatology Re- feral	Uncertain diagnosis
	Possibility of: Contact dermatitis Psoriasis Fungal Infection
	History of recurrent skin infections
	Extensive/severe disease
	Management to date has not controlled symptoms
	Need for Class 1 topical steroid
Allergy Referral	Mod/severe atopic dermatitis and known/suspected food allergies
	Environmental triggers suspected
	Concomitant moderate or severe asthma
	Infants with severe atopic dermatitis
Immunology Re- feral	Immunodeficiency or suspected immunodeficiency
	History of recurrent fractures or infections in older patients
	Infants with severe atopic dermatitis and growth concerns, infections

Considerations for Subspecialty Referral

Considerations for subspecialty referral is mentioned in the below table (**Table 6**) [7,8].

Outpatients

Most patients with AD do not need urgent consultation or outpatient referrals to Dermatology or Allergy/Immunology. Consider an expedited referral request to dermatology/allergy for:

- Severe or refractory disease
- Unclear diagnosis
- Suspected food allergy
- Suspected immunodeficiency

Inpatients

- Consult for severe or refractory disease requiring escalation of treatment
- Initiation of systemic agents to control disease
- Severe or recurrent skin infections
- Concern for underlying immunodeficiency

Conclusion

AD is a complex disorder that requires both a genetic predisposition and exposure to poorly defined environmental factors. Disease results from a defective skin barrier and immune dysregulation. Effective treatment requires therapies targeted to both restoring barrier function and controlling inflammation. Treating both defects is crucial to optimal outcomes for patients with moderate to severe disease. Education of patients regarding the underlying defects and provision of a comprehensive skin care plan is essential.

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