



Skin Deep: Simplifying Practice Guidelines for Children With Atopic Dermatitis

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KEY WORDS

Atopic dermatitis, eczema, pediatric dermatology, practice guidelines

Atopic dermatitis (AD) is a common chronic inflammatory skin disorder in children. Often called *eczema*, AD is a papulosquamous eruption often characterized by pruritus and then the typical distribution and morphology of the rash (Saavedra et al., 2013). Thus, an idiom often used by health care professionals to describe AD is “the itch that rashes.” Two leading organizations jointly authored an update on AD in 2012 (Schneider et al., 2013), and a third published several guidelines for the management of AD (Eichenfield et al., 2013, 2014, 2015; Sidbury et al., 2014). However, these guidelines are directed predominantly toward dermatologists and allergists rather than toward primary care providers (PCPs), who treat almost half of children with AD (Werner-Busse, Kostev, Heine, & Worm, 2014). Because AD is chronic and tends to relapse, it can significantly affect the quality of life of both the child and the family (Saavedra et al., 2013). The purpose of this article is to simplify the recognized guidelines

for the management of AD in children for use by PCPs such as nurse practitioners.

EPIDEMIOLOGY

Several sources report the incidence of AD in children to be between 12.5% and 20%, with symptoms continuing into adulthood in about 10% of patients (Garg & Silverberg, 2015; Jackson, Howie, & Akinbami, 2013; Schneider et al., 2013). According to Silverberg and Simpson (2014), 67% of children with AD have mild disease, 26% have moderate disease, and 7% have severe disease. Because of the chronicity of AD, children may vacillate between these degrees of disease with flares and use of consistent treatment. Severity is often qualified by the extent of the affected areas. For example, AD that is limited to the flexural surfaces is considered mild by most experts, whereas moderate or severe AD would present with a more generalized distribution or in patients whose symptoms do not abate with basic skin care (Paller & Mancini, 2016). Research has shown that severe AD is more likely in African American and Hispanic children (Silverberg & Simpson, 2014). Studies clearly indicate that AD is a major concern for children and may not improve with age. Therefore, treatment strategies used in childhood may shape therapies needed in adulthood.

ETIOLOGY

Studies have shown a higher incidence of AD in more developed nations, more affluent families, urban dwellers, and those with genetic predisposition (Garg & Silverberg, 2015). AD is also associated with other atopic conditions, such as food allergies, asthma, and allergic rhinitis. However, not all individuals with AD progress to these conditions. Although

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food allergies are common among children with AD, no evidence confirms that food allergies cause AD (Tollefson, Bruckner, & Section of Dermatology, 2014).

Recent scientific advancements postulate that a strong genetic component supports the development of AD (Garg & Silverberg, 2015). Filaggrin gene (*FLG*) mutations have been identified in people with AD (Garg & Silverberg, 2015; Tollefson et al., 2014). Normal skin integrity, where corneocytes and hydrophobic lipid matrix are tightly bonded together, result in a barrier to prevent water loss and stop aeroallergens and microbes from entering the skin. The mutations of the *FLG* protein alter this skin integrity, leading to dryness, inflammation, and pruritus (Garg & Silverberg, 2015; Tollefson et al., 2014). This genetic predisposition, along with environmental exposure, may contribute to the development of AD. Garg and Silverberg found that early exposures to endotoxins, helminthes, animals, and daycare appear to offer protection against developing AD; overbathing with harsh soap and frequent use of antibacterial gels may contribute to the breakdown of skin integrity and result in AD (Garg & Silverberg, 2015).

ASSESSMENT

The following points should remain the focus of the assessment of a child with signs and symptoms of AD.

History of Present Illness/Review of Systems

- Age of child
- What did the rash look like initially? Has it changed?
- When did the rash appear, and how long did it last?
- Where is the rash? Is it spreading? If so, where?
- Does the rash itch? Have you noticed your child scratching or rubbing the rash?
- Is the rash painful, burning, or sore?
- Any fever or other symptoms of illness?
- Any exposure to close contacts with similar symptoms or possible infestation by *Sarcoptes scabiei*?
- Does anything make the rash and/or itching worse? What alleviates the symptoms?

Past Medical History

- Obtain a comprehensive medical history with attention to history of wheezing or symptoms of asthma, allergic rhinitis, or presence of other dermatologic conditions.
- Obtain a thorough family medical history including other disorders such as food and environmental allergies, history of asthma, and others affected by AD or chronic skin problems.
- Any recent or current medications or vaccinations

Physical Examination Findings Typically Seen in AD (Paller & Mancini, 2016)

- Pruritus as evidenced by rubbing, scratching, or parent report

- Eczematous eruption with an age-specific morphology and distribution
 - Infantile (<2 years)
 - More common to find edema and exudative/crusted lesions
 - Typically begins with papules on scalp and face, often sparing the nose and perioral skin
 - Progresses to include the extensor surfaces of extremities and, less commonly, the flexural surfaces
 - Usually spares the diaper area
 - Childhood (2 years to puberty)
 - Dry papules in well-circumscribed patches
 - Lichenification
 - Commonly involves distal extremities, antecubital and popliteal fossae, neck, and around the mouth and eyes
 - Adult (begins in later childhood)
 - Dry and scaly erythema with papules and plaques, possible exudate and crusting
 - Lichenification
 - Affects flexural surfaces, face, neck, fingers, hands, feet, and toes
 - Hyperpigmentation
- Generalized xerosis
- Hypopigmentation secondary to inflammation
- Lymphadenopathy is more common with secondary infection
- Dermatographism
- Prominent follicular pattern or keratosis pilaris

DIFFERENTIAL DIAGNOSES AND DIAGNOSTIC STUDIES

Diagnosis is based on clinical presentation (see **Box**) and the exclusion of other conditions (**Table 1**); no reliable diagnostic tests differentiate AD from other conditions (Eichenfield et al., 2013; Paller & Mancini, 2016; Tollefson et al., 2014). Serum immunoglobulin E testing is nonspecific and may be elevated in children with and without AD; testing is not routinely recommended (Eichenfield et al., 2013). For unrelenting cases or signs of secondary infection, skin cultures might be warranted.

BOX. Clinical presentation of atopic dermatitis

Hallmark features (must be present)

Pruritus
Age-specific eczematous eruption

Secondary features

Early age onset
Personal or family history of atopy
Xerosis

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