Pathophysiology and Management of Mild to Moderate Pediatric Atopic Dermatitis CE

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ABSTRACT

Atopic dermatitis (AD), or eczema, is a chronic inflammatory skin condition characterized by relapsing pruritic and dry, scaly lesions. AD affects 10% to 20% of children in the United States and significantly affects the quality of life of patients and their families. Primary care providers (PCPs) are often the first point of contact for the management of AD symptoms. As many as 70% of patients with mild to moderate disease can be managed by a PCP, underscoring the need for these providers to understand basic AD pathophysiology and current standards of care. This article will discuss the basic principles of AD diagnosis and management that PCPs need to optimize patient care, including AD pathogenesis, appropriate use of currently available topical therapies, basic skin care practices, and patient/caregiver counseling points. This article is sponsored by Spire Learning and supported by an educational grant from Pfizer Inc. J Pediatr Health Care. (2018) *32*, S2-S12.

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KEY WORDS

Atopic dermatitis, corticosteroid, eczema, emollient, skin barrier, topical immunomodulatory

OBJECTIVES

1. Summarize the role of skin barrier dysfunction and inflammatory pathways and responses in AD pathogenesis.
2. Identify management strategies for mild to moderate AD based on patient and caregiver preferences, adherence, and concerns.
3. Describe the efficacy and safety of currently available therapies and their potential role in the management of mild to moderate AD.
4. Implement into practice shared decision-making strategies to increase patient and caregiver involvement in AD management.

INTRODUCTION

Atopic dermatitis (AD) is a chronic, relapsing inflammatory skin disease that affects approximately 10% to 20% of American children and typically presents in patients older than 2 years of age (Shaw, Currie, Koudelka, & Simpson, 2011; Siegfried & Hebert, 2015). Children with AD often experience persistent itching, which often interferes with sleep and significantly affects the quality of life of the patient and family (Pustišek, Vurnek Živković, & Šitum, 2016). Primary care providers (PCPs) are usually the first points of contact for most patients with AD, and as many as 70% of patients have mild to moderate disease that can be effectively managed by a PCP (Eichenfield et al., 2015). This article will discuss AD diagnosis and management that PCPs need to optimize patient care, including AD

pathogenesis, appropriate use of currently available topical therapies, basic skin care practices, and patient/caregiver counseling points.

DIAGNOSING AD

A diagnosis of AD is made clinically and is based on a personal or family history of atopy and clinical presentation of a chronic or relapsing pruritic dermatitis exhibiting typical morphology and age-specific patterns (Eichenfield, Tom, Chamlin, et al., 2014; Simpson, Irvine, Eichenfield, & Friedlander, 2016). Patients with acute flares present with erythematous, scaly lesions, and widespread excoriations. Papules and/or spongiotic vesicles are present in more severe cases. Dyspigmentation and lichenification are hallmarks of chronic disease. In darker skin types, the skin may have a grayish-white "ashy" appearance, and erythema may be difficult to see, whereas follicular accentuation, lichenification, and postinflammatory dyspigmentation are more conspicuous (Siegfried & Hebert, 2015).

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Although AD can appear anywhere on the body and at any age, it has a characteristic age-distribution pattern that is helpful in confirming the diagnosis. In infants (birth to 12 months of age), lesions are commonly found on the face (except the nose area), upper and lower extremities (including wrists and ankles), and trunk. In toddlers (1–3 years of age), involvement typically includes the face, neck, antecubital and popliteal regions, wrists, and ankles. In older children and adolescents, the face, antecubital and popliteal areas, hands, and feet are more commonly affected (Simpson, Bieber, et al., 2016).

The clinical features of AD are often indistinguishable from other dermatologic conditions that can mimic, overlap, or complicate AD. The most common differential diagnoses to consider are contact dermatitis, psoriasis, scabies, and seborrheic dermatitis (Silverberg & Durán-McKinster, 2017).

PATHOPHYSIOLOGY

Research from the past decade has gleaned evidence of genetic, environmental, and immunologic factors that contribute to the pathogenesis of AD (Figure 1). For instance, a strong family history of atopy, onset of disease before 12 months of age, and mutation of the filaggrin (*FLG*) gene are reliable predictors of disease severity (Eichenfield, Tom, Chamlin, et al., 2014). Environmental factors affecting skin barrier, combined with the patient's genetic profile, aid in predicting risk for developing AD (Simpson, Irvine, et al., 2016; Wen et al., 2009). Exogenous factors such as harsh soaps, detergents, and wool can cause itching and scratching, thereby disrupting the skin barrier and initiating a flare. Endogenous factors contributing to

FIGURE 1. Complex pathophysiology of AD. AD, atopic dermatitis; PDE-4, phosphodiesterase-4; IgE, immunoglobulin E; IL, interleukin; TEWL, transepidermal water loss; Th2, T helper 2.