Atopic Dermatitis in Children

Clinical Features, Pathophysiology, and Treatment

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KEYWORDS

• Atopic dermatitis • Eczema • Allergy • Netherton syndrome • Hyper-IgE syndrome

KEY POINTS

- Atopic dermatitis is a complex disorder resulting from gene-environment interactions.
- Defective skin barrier function and immune dysregulation are paramount to disease pathogenesis.
- Pruritus is universal, is a major comorbidity, and is poorly responsive to antihistamines.
- Effective treatment requires therapies targeted to restore both barrier function and to control inflammation.
- Education of patients regarding the principal defects and provision of a comprehensive skin care plan is essential.

INTRODUCTION

Atopic dermatitis (AD) is a chronic, relapsing, and highly pruritic dermatitis that generally develops in early childhood, and has a characteristic age-dependent distribution. AD is relatively common, affecting 10% to 20% of children in developed countries.[1] Patients with AD frequently have elevated total immunoglobulin E (IgE) levels, sometimes markedly elevated, the level of which appears to correlate with disease severity.[2] Patients with AD also can have elevated allergen-specific IgE levels, indicating sensitization, but not necessarily clinical allergy, an area of great confusion for patient management, particularly with regard to food allergy.[3] The major medical comorbidities associated with AD are infections, including *Staphylococcus aureus*

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superinfection and eczema herpeticum; however, chronic pruritus and sleep loss, as well as the time and expense associated with treatment, are often most distressing for patients and families. AD has been associated with poor school performance, poor self-esteem, and family dysfunction.[4–7]

The causes of AD are still poorly understood, although genetic predisposition in the setting of inciting environmental factors appears critical. Similar to asthma and other complex chronic disorders, AD should be viewed as a common end manifestation of many different genetic defects, resulting in impaired epidermal barrier function and immune dysregulation. Additional identification and characterization of genetic defects among patients with AD is needed; this may lead to better characterization of the disease and development of more effective therapies.

For now, management is based on targeting the known defects in AD, namely skin barrier dysfunction and cutaneous inflammation, along with treatment (in some cases prophylactically) of associated infections. The pruritus associated with AD is often the most distressing symptom and is treated with skin hydration and topical anti-inflammatories, but is poorly responsive to antihistamines in most patients. Behavioral interventions, such as biofeedback and relaxation techniques, also can be helpful in controlling scratching. Although a comprehensive treatment plan with extensive education is effective is controlling AD in most patients, better treatments are needed, particularly disease-modifying therapies that can be initiated in early childhood.

CLINICAL FEATURES

AD is characterized by a chronic, relapsing dermatitis that is pruritic, begins in the first 5 years of life in 90% of patients (but not in the first weeks of life, as seen in the autosomal dominant hyper-IgE syndrome), and usually presents in a characteristic age-dependent distribution with facial, scalp, and extensor involvement in infants and young children, and predominant flexural involvement in older children and adults. Pruritus is universal and xerosis is a common feature in children with AD. Acute lesions are characterized by pruritic papules with erythema, excoriations, and serous exudate, whereas chronic AD is characterized by areas of lichenification and fibrotic nodules, often accompanied by acute lesions (**Fig. 1**).

Because pathognomonic lesions are not present to definitively diagnose AD, diagnostic criteria have been described; the most widely cited being the "Hanifin and Rajka" criteria[8] and subsequent modifications, including the UK Working Party's Diagnostic Criteria for Atopic Dermatitis (**Box 1**).[9] Five major clinical features based

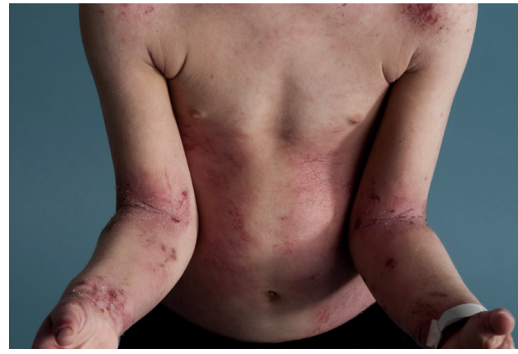

Fig. 1. Typical distribution of skin lesions in a child with AD.

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