Atopic Dermatitis

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KEYWORDS

• Atopic dermatitis • Eczema • Pruritus • Diagnosis • Complications
• Management recommendations

KEY POINTS

- Atopic dermatitis (AD) is a chronic inflammatory skin condition characterized by intense pruritus.
- Patients with AD require periodic physician assessment of disease state, comorbidities, and complications.
- Treatment of AD requires a multimodal approach using intensive patient education, anti-inflammatories, antibacterial intervention, and psychological support.

Video of Pruritus in atopic dermatitis accompanies this article at http://www.pediatric.theclinics.com/

OVERVIEW

Atopic dermatitis (AD) is a common chronic inflammatory skin condition characterized by intense pruritus and a waxing and waning course. This condition most often presents in infancy and childhood and can persist, in one form or another, throughout adulthood. The exact cause of AD is unknown, but it likely reflects an interplay between genetic and environmental factors. AD affects up to 20% of children in the United States, and the prevalence may be increasing.[1] Treatment can be very effective in alleviating symptoms but serves only to manage the disease, not cure it. Appropriate therapy can also prevent significant complications, such as infection, sleep disturbance, behavioral problems, and growth impairment.

EPIDEMIOLOGY

Population studies have demonstrated an increasing prevalence of AD throughout the world. In the United States, it affects approximately 10% to 20% of children younger than 18 years, and these numbers are rising. Affected children are more likely to be black, urban, and living in homes with higher education levels.[1] As a chronic disease,

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AD has a significant impact on health care resource utilization, similar to asthma or diabetes. There were an estimated 7.4 million outpatient physician visits for AD during the 7-year-period between 1997 and 2004, amounting to an estimated health care cost of US $364 million to $3.8 billion annually.[2,3]

PATHOPHYSIOLOGY

The exact cause of AD is unknown. However, it is generally agreed that AD results from a combination of genetic and environmental factors. Twin studies support a high rate of concordance; identical twins have a 7-fold increased risk for AD, and fraternal twins have a 3-fold increased risk.[4]

Healthy skin acts as a barrier to both outside influences and transepidermal water loss. Current theory holds that a genetically compromised barrier allows for penetration of environmental factors (irritants, allergens, and bacteria) with resultant immune dysregulation. A mutation in the filaggrin gene, responsible for an important component of the barrier, can be found in up to 10% of people of European ancestry.[5] Filaggrin is an epidermal protein that acts as waterproof "mortar" between keratinocytes in the outermost layer of the skin. Mutations in this protein cause ichthyosis vulgaris and are positively associated with more severe or persistent AD.[6]

Other prevalent theories of pathogenesis in AD focus on immune dysfunction. One observation in support of the role of immune dysfunction is that many primary immunodeficiency syndromes are characterized by early onset of diffuse eczematous eruptions and are caused by genetic mutations resulting in disruption of various immune functions, such as hyper-immunoglobulin E (IgE) syndrome, severe combined immunodeficiency, Wiskott-Aldrich syndrome, and Omenn syndrome. In the 1980s, a popular theory, termed the "hygiene hypothesis," emerged in an attempt to explain the fact that atopy tends to affect individuals from developed nations and those in a higher socioeconomic status.[7] This hypothesis asserts that the lack of childhood exposure to infectious agents results in an immune response favoring atopy, whereas early exposure to infectious agents triggers a T helper 1 (T_H1) response, thus diverting the immune system away from a T helper 2 (T_H2) "atopic" response. There is somewhat conflicting data in support of this hypothesis, and in truth, the interplay between T_H1 and T_H2 is likely more complex than previously thought.[8,9] More rigorous studies into causation are needed.

PREVENTION AND PROGNOSIS

Many studies have investigated primary prevention strategies and their effect on AD. These studies have examined the effect of early exposure to environmental and dietary factors such peanuts, eggs, soy, and animal dander or early supplementation of probiotics, breast milk, and vitamin D as related to development of AD.[10,11] There is currently no convincing evidence that any of these strategies are helpful.

The natural history of AD is variable. Based on population studies, a significant proportion of affected children "outgrow" the disease, as only 1% to 3% of adults are affected.[12] Patients with the most severe disease are more likely to have persistent disease.[13]

PRESENTATION

History

Pruritus, or itch, is defined as an unpleasant sensation that provokes the desire to scratch. A history of pruritus is required to establish the diagnosis of AD. Young