Review

Pediatric Atopic Dermatitis

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Atopic dermatitis (AD or eczema) is one of the most common childhood dermatological conditions, with a reported prevalence peak of 15 to 20% in early childhood in industrialized countries.[1] There is a slight female predominance, 1.3:1.0 female to male ratio. An inherited condition, it is characterized by chronic relapsing flares of intensely pruritic, poorly demarcated, erythematous plaques located typically on the face and extensor surfaces in infants and young children and on the flexural surfaces in older children.[1] Although termed atopic, up to 60% of children with the clinical phenotype do not have demonstrable IgE-mediated sensitivity to allergens.[2]

Diagnosis

Diagnosis of atopic dermatitis is based on a constellation of features described by the Hanifin and Rajka criteria. Major features include pruritis, typical distribution, chronic or chronically relapsing presentation, and personal or family history of atopic disease.[3] Other, minor features of atopic dermatitis include xerosis, periauricular fissures, ichthyosis, hyperlinear palms, keratosis pilaris, IgE reactivity (increased serum IgE, radioallegosorbent test, or prick test positivity), hand or foot dermatitis, cheilitis, scalp dermatitis, susceptibility to cutaneous infections (especially *Staphylococcus aureus* and herpes simplex), perifollicular accentuation (especially in darkly pigmented races), Dennie's lines, and pityriasis alba.[3] In terms of differential diagnosis, the most likely include seborrheic dermatitis, contact dermatitis, scabies, psoriasis, ichthyosis vulgaris, keratosis pilaris, and dermatophytosis.[1]

Pathogenesis

The pathogenesis of atopic dermatitis involves genetic susceptibility resulting in a complex interaction between defects in the immune system, a defective skin barrier, and sensitivity to allergens/microbes leading to a heightened immune response.[1] Skin barrier function is decreased as a result of downregulation of cornified envelope genes (filaggrin/loricrin), reduced ceramide levels, increased levels of endogenous proteolytic enzymes, and enhanced transepidermal water-loss. Soaps/detergents, as well as exposure to house mites/*Staphlococcus*

aureus, increase the activity of endogenous and exogenous proteases, respectively, contributing further to the lack of skin integrity. Compromised barrier function facilitates colonization by allergens and microbes, followed by infiltration of immune cells and release of cytokines/chemokines, causing an inflammatory response.[1]

Genetics

Atopic dermatitis is familial, with a significant maternal influence. The genetic influence is illustrated by a concordance rate of 77% in monozygotic twins and 15% in dizygotic twins.[4] Although there are likely many genes involved in the development of the condition, several connections have been made. A loss of function mutation in filaggrin has been found to be a predisposing factor for the development of AD, by causing impaired water binding capacity and compromised barrier function.[1] Additionally, chromosome 5q31-33 contains a cluster of functionally related cytokine genes, and it has been suggested that there is a genotypic association of the T allele of 590C/T polymorphism of the IL-4 gene promoter region with AD. Furthermore,there have been reports of functional mutation in the alpha subunit of the IL-4 gene being linked to AD development. There is also support for the involvement of CD4+ T cells and dysregulation of Th1 genes in the pathophysiology of AD, as well as a role for host defense genes based on polymorphisms in the NOD1 gene.[1]

Symptoms

Patients typically present with symptoms of pruritis that are often worse at night. Clinical examination reveals crusting, oozing eruptions and, depending on stage of the disease, secondary changes from scratching in the form of thickened, lichenified plaques.[3] AD typically improves with age, and skin becomes pruritic and inflamed only when exposed to exogenous irritants.[1]

Treatment

Treatment of atopic dermatitis requires an individualized, multifaceted approach that includes skin hydration, pharmacological therapy, and elimination of flare triggers. Central to the success of the treatment plan is the commitment of the patient and that of his or her parents. The degree of sleep disturbance, the number and location of involved sites, and the clinical course of the illness are all indicators of severity that likely provide the best basis for making treatment decisions. It is recommended that patients take lukewarm baths daily, pat themselves dry, and immediately apply an occlusive emollient.[5]

In terms of pharmacological treatment, topical corticosteroids are still considered to be first-line-therapy. They are extremely effective and safe when used appropriately. Topical calcineurin inhibitors, such as tacrolimus and pimecrolimus have been shown to be effective in the treatment of atopic dermatitis. They are

most often used as maintenance therapy in well-controlled eczema. Sedative antihistamines are sometimes recommended despite a lack of evidence demonstrating their effectiveness in the treatment of atopic dermatitis. They function mainly to relieve sleep disturbance that results from nocturnal pruritus. Additionally, oral antibiotics are often prescribed for suspected secondary infection, *S. aureus*, being the most common pathogen. In the case of severe flare-ups, a brief course of oral corticosteroids is considered, although the duration of this treatment is as short as possible due to extensive adverse effects.[2,6] Finally, coal tar or UV therapy may be considered for patients who are resistant to corticosteroids and/or topical calcineurin inhibitors.[4,6]

Take-home message

- Atopic dermatitis is a common inherited dermatological condition characterized by chronic relapsing flares of intensely pruritic, poorly demarcated, erythematous plaques
- Differential diagnosis should include seborrheic dermatitis, contact dermatitis, scabies, psoriasis, ichthyosis vulgaris, keratosis pilaris, and dermatophytosis
- Patients should be encouraged to take lukewarm baths daily, pat dry, and immediately apply an occlusive emollient
- Pharmacologically, topical corticosteroids are safe and effective when used appropriately. They are considered the gold standard of treatment for atopic dermatitis
- Atopic dermatitis typically improves with age

Prognosis

Generally, atopic dermatitis is more severe and persistent in young children, and periods of remission become more common with age. Spontaneous remission has been reported to occur in 40 to 60% of patients over the age of five who were affected in infancy[2] and, by the age of 15, remission has been reported to occur in 60 to 70% of cases.[4] Dx

References
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