

TERRY DEAN JR. | LOUIS M. BELL



Nelson

PEDIATRICS BOARD REVIEW

CERTIFICATION AND RECERTIFICATION

EDITION

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PEDIATRICS BOARD REVIEW

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SECTION **1**

Allergy

Basic Information

- The atopic diseases include atopic dermatitis (eczema), food allergies, allergic rhinitis, and asthma
- “Atopic march” or “allergic march” refers to the sequential presentation of atopic diseases, typically in the order listed above, from infancy through childhood
- Causes
 - Complex interaction between genetic risk factors and environmental influence
 - Early exposure to endotoxins, pets, farm animals, and daycare may be protective against the development of atopic dermatitis (“hygiene hypothesis”)
 - Maternal diet and breastfeeding do not play a role in the development of atopy
 - Delaying introduction of solid foods past 4 to 6 months does *not* prevent development of food allergies; however, early introduction of peanuts in infants with moderate to severe eczema lowers the risk of development of peanut allergy

Atopic Dermatitis

Basic Information

- Chronic, inflammatory, and pruritic skin disease
- Prevalence: up to 20% prevalence in children and rising, especially in developed nations
- Etiology is multifactorial (epidermal barrier dysfunction, altered skin flora, immune dysregulation)
- Known risk factors include parental history of atopic disease, genetic variants in filaggrin (FLG), environmental exposures
- Distinct from idiopathic nummular eczema, allergic contact dermatitis, chronic hand eczema
- See [Chapter 12](#) for additional information

Clinical Presentation

- Can present as early as 2 to 6 months of age; 85% to 90% present by age 5 years
- Chronic or relapsing course of red macules and papules with intense pruritus and lichenification
 - <2 years: distribution is face and extensor aspects of extremities
 - >2 years: distribution is flexural areas of extremities
- Exacerbated by infections (e.g., *Staphylococcus aureus*), heat, low humidity, chemical irritants, and environmental allergens in some

- Only one-third of patients with severe eczema have foods that consistently exacerbate atopic dermatitis; thus empiric food elimination is not recommended in the treatment of eczema
 - Food elimination for eczema is not recommended due to negative impact on nutrition and increased overall risk of food allergy development

Diagnosis and Evaluation

- Most cases of eczema are diagnosed by history and examination of skin
 - Distribution of eczema usually differs by age (see above)
 - Appearance of eczema can vary depending on skin color
- Biopsy of skin is generally not needed unless eczema is refractory to conservative therapies or other systemic disorder is suspected
- Differential diagnosis:
 - Allergic or irritant contact dermatitis from plants (i.e., poison ivy), metals (nickel-plated jewelry), and chemical products (e.g., perfumes, cleaning products)
 - Immunodeficiency (HIV, Wiskott-Aldrich syndrome, severe combined immunodeficiency, hyper-IgE syndrome)
 - Metabolic disorders (zinc, vitamin B6, or niacin deficiency)

Treatment

- Mainstays of therapy are emollients/moisturizers and topical corticosteroids; choose strength of steroid based on location and severity of eczema
- Daily baths or showers with unscented or mild soap, followed by application of emollient; wet wrap therapy for severe eczema
- Control of itching with oral antihistamines (cetirizine, hydroxyzine)
- Other topical therapies: topical phosphodiesterase-4 inhibitors (crisaborole), calcineurin inhibitors (tacrolimus, pimecrolimus). Refer to Dermatology for cyclosporin, methotrexate, anti-IL-4/anti-IL-13 therapy for severe eczema
- Management of superinfection:
 - *S. aureus* infections of eczema are common
 - Eczema herpeticum: herpes simplex virus infection; treatment with acyclovir

Food Allergies

- See [Chapter 3](#) for more information

Table 4.1 Disorders Associated With Elevated Serum Immunoglobulin E

Allergic disease
Atopic dermatitis (eczema)
Tissue-invasive helminthic infections
Hyperimmunoglobulin E syndromes
Allergic bronchopulmonary aspergillosis
Wiskott-Aldrich syndrome
Bone marrow transplantation
Hodgkin disease
Bullous pemphigoid
Idiopathic nephrotic syndrome

From Table 142-1: Environmental Control of Allergen Exposure. *Nelson's Textbook of Pediatrics*. 20th ed. Elsevier; 2016.



Fig. 4.1 Clinical presentation of urticaria. (Photograph courtesy of Pete Smith, MD, Griffith University, Brisbane, Queensland, Australia.)

- immunosuppression (cyclosporin, tacrolimus), biologics (omalizumab)
- Prognosis is good with resolution in up to 50% within 1 year of onset

Hereditary Angioedema (HAE)

Basic Information

- Most often an autosomal dominant disease due to a deficiency in C1-esterase inhibitor, leading to dysregulation of the complement pathway and intermittent episodes of swelling of various body parts
 - Type 1: 85% of patients, quantitative defect in C1-esterase inhibitor
 - Type 2: 15% of patients, qualitative defect in C1-esterase inhibitor

Clinical Presentation

- Tissue swelling (most commonly skin, upper respiratory tract, GI tract) *without* urticaria or pruritus
 - Majority of patients experience abdominal pain
 - Laryngeal swelling can be fatal
- Episodes may be preceded by trauma
- Untreated, symptoms can last for several days

Diagnosis and Evaluation

- Diagnosis based on clinical history and physical exam during episodes

- Labs: C4 as initial screening test (generally decreased when asymptomatic, absent during attacks), C1 esterase inhibitor protein level and function

Treatment

- First-line therapy: replacement of C1 esterase inhibitor is needed
- Second-line therapy (if above not available): fresh frozen plasma
- Symptoms DO NOT improve with antihistamines, steroids, or epinephrine
- In the acute setting of an attack: consult allergist on-call and protect airway, with supportive management for dehydration and pain if present

Mastocytosis

Basic Information

- Two forms:
 - Cutaneous mastocytosis (predominantly children), three subcategories:
 - Urticaria pigmentosa (also maculopapular cutaneous mastocytosis, MPCM), most common form of cutaneous mastocytosis in children
 - Diffuse cutaneous mastocytosis (DCM)
 - Mastocytoma of the skin
 - Systemic mastocytosis (predominantly adults)
 - Majority of patients will have cutaneous involvement

Clinical Presentation

- Cutaneous mastocytosis
 - Eighty percent of patients will have brown or red skin lesions. See [Fig. 4.2](#)
 - Darier's sign (wheal or reddening of the skin with mechanical stroking or rubbing). See [Fig. 4.3](#)
 - Less than 10% of patients develop systemic symptoms
- Systemic mastocytosis
 - Previously mentioned cutaneous symptoms
 - Systemic symptoms: idiopathic anaphylaxis, flushing, hives, angioedema, diarrhea, fatigue, bone pain, wheezing

Diagnosis and Evaluation

- Cutaneous mastocytosis
 - Clinical diagnosis: skin biopsy if diagnosis is unclear
 - No bone marrow biopsy needed in children
- Systemic mastocytosis
 - Bone marrow biopsy and biopsy of the affected organs
 - Serum tryptase level, KIT D816V mutation analysis

Treatment

- Cutaneous mastocytosis
 - Topical corticosteroids for pruritic lesions
 - Oral second-generation antihistamines for pruritus
- Systemic mastocytosis
 - Management per hematologist

Hypereosinophilia

Basic Information

- Hypereosinophilia: absolute eosinophil counts (AEC) >500 cells/ μ L



Fig. 4.2 Subforms of CM. Cutaneous manifestations in mastocytosis are categorized into MPCM, presenting with disseminated brown lesions (A); DCM, presenting with generalized erythema and thickened skin (B); and mastocytoma, presenting with a brown or red elevated lesion (C). (From Hartmann K, Escribano L, Grattan C, et al. Cutaneous manifestations in patients with mastocytosis: consensus report of the European Competence Network on Mastocytosis; the American Academy of Allergy, Asthma & Immunology; and the European Academy of Allergology and Clinical Immunology. *J Allergy Clin Immunol*. 2016;137(1):35–45. <https://doi.org/10.1016/j.jaci.2015.08.034>.)



Fig. 4.3 Darier's sign. (A–C) A wheal-and-flare reaction develops upon stroking of a CM lesion with a tongue spatula. Darier's sign is a highly specific diagnostic feature of CM. (From Hartmann K, Escribano L, Grattan C, et al. Cutaneous manifestations in patients with mastocytosis: consensus report of the European Competence Network on Mastocytosis; the American Academy of Allergy, Asthma & Immunology; and the European Academy of Allergology and Clinical Immunology. *J Allergy Clin Immunol*. 2016;137(1):35–45. <https://doi.org/10.1016/j.jaci.2015.08.034>.)

- Hypereosinophilic syndrome: hypereosinophilia plus organ dysfunction due to eosinophilia
- Mild, transient eosinophilia common in pediatric population
 - Severity
 - Mild: AEC 500–1500 cells/ μ L
 - Moderate: AEC 1500–5000 cells/ μ L
 - Severe: AEC >5000 cells/ μ L
- Primary causes:
 - Hypereosinophilic syndrome (HES): types include myeloproliferative, lymphocytic, overlap (hypereosinophilia with eosinophilic disease of one organ system), familial (autosomal dominant), idiopathic
- Secondary causes (more common in children)
 - Infections: parasites (e.g., strongyloides, toxocariasis, hookworm, scabies), fungal infections (e.g., coccidiomycosis), mycobacterial infection, HIV
 - Allergic disorders:
 - Atopic disease (asthma, atopic dermatitis, allergic rhinitis)
 - Drug-induced, DRESS
 - Allergic bronchopulmonary aspergillosis
 - Eosinophilic disorders
 - Eosinophilic gastrointestinal disease
 - Eosinophilic granulomatosis with polyangiitis
 - Primary immunodeficiencies
 - Autosomal dominant hyper-IgE syndrome (Job syndrome), Omenn syndrome

- Malignancy-related (leukemia or lymphoma, some solid tumors)
- Other: autoimmune disorders (e.g., sarcoidosis), adrenal insufficiency, graft-versus-host disease

Clinical Presentation

- Primary HES: usually insidious with skin rash, respiratory (cough, shortness of breath), GI, cardiac and/or neurologic symptoms
- Secondary HES: eosinophilia may be incidental finding during workup for underlying condition

Diagnosis and Evaluation

- Repeat CBC to determine whether eosinophilia is transient, persistent, or rising
- Physical examination and lab testing (e.g., CBC and smear, electrolytes, liver function tests) to evaluate for end-organ involvement
- Primary HES: bone marrow biopsy may be necessary for diagnosis
- Secondary HES: testing for secondary causes is disease specific, will depend on history and physical examination
- IgE may be elevated for certain diseases associated with eosinophilia (see Table 4.1)

Treatment

- Primary HES is treated only if there is end-organ involvement
- Secondary causes are treated for the underlying disorder

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