Περιεχόμενα:

- **Croup**

Part E: Lower Respiratory Infections

- **Bronchiolitis**
- **Uncomplicated pneumonia**
- **Complicated pneumonia**
- **Recurrent pneumonia**
- **Childhood tuberculosis**

Part F: Cardiac Infections

- **Endocarditis**
- **Myocarditis and Pericarditis**
- **Acute rheumatic fever**

Part G: Gastrointestinal Infections

- **Gastroenteritis**
- **Hepatitis**
- **Antibiotic-associated diarrhea**

Part H: Genitourinary Infections

- **Urinary tract infections**
- **Pelvic inflammatory diseases**

Chapter 62: Infections in Atopic Dermatitis

DEFINITION AND EPIDEMIOLOGY

Atopic dermatitis is a common inflammatory skin disorder that affects 10–20% of children younger wthan 14 years of age.[1,2]

The prevalence of atopic dermatitis has increased two- to threefold since the 1980s in industrialized countries, and it remains the most common dermatitis of childhood. The pathogenesis of atopic dermatitis, although not completely understood, is likely multifactorial, involving complex interactions between environmental triggers, defects in skin barrier function, and systemic and local immunologic responses.[3] atopic dermatitis is often the initial presentation of atopic disease in children, and according to the theory of the "atopic march," poorly controlled atopic dermatitis is believed to contribute to the development of asthma and allergic rhinitis in older children in 50–80% of affected patients.[2,3]

CLINICAL PRESENTATION AND DIAGNOSIS

The diagnosis of atopic dermatitis is made on clinical evaluation. Most cases arise within the first 2 years of life.[4] The key features of atopic dermatitis as defined by Hanifin and Rajka in 1980

and modified in 2001 include a chronic and relapsing course, typical morphology, and distribution of cutaneous findings, and pruritus.[5,6] Pruritus is a universal finding in atopic dermatitis and can be severe, leading to sleep disturbances and irritability. Pruritus also leads to scratching, which causes secondary skin changes such as lichenification (thickening and hyperpigmentation of skin with accentuation of skin lines), excoriations, skin breakdown, and infection.

The cutaneous manifestations of atopic dermatitis may be classified as acute or chronic. In an *acute* exacerbation of atopic dermatitis, erythematous papules and patches associated with scaling, excoriations, and serous exudates are seen (**Figure 62-1**). *Chronic* atopic dermatitis is characterized by variably hyperpigmented, lichenified plaques and nodules that result from chronic rubbing and scratching (**Figure 62-2**). Acute and chronic changes may coexist in the same patient. Most atopic dermatitis patients also have dry, lackluster skin (xerosis), and a significant number also have *ichthyosis vulgaris*, a genetic skin disorder that results from mutations in the gene for filaggrin.[7–9] Filaggrin is a key component of the cornified cell envelope, which forms the epidermal skin barrier. Disruption of the epidermal barrier increases transepidermal water loss and is thought to increase epicutaneous exposure to potential environmental allergens, which can contribute to the development of atopic disease.

FIGURE 62-1. Acute presentation of atopic dermatitis with erythema and scaling.

FIGURE 62-2.

Chronic atopic dermatitis with the development of hyperpigmentation and lichenification.

Chapter 64: Fever of Unknown Origin

DEFINITIONS AND EPIDEMIOLOGY

PHYSIOLOGIC TEMPERATURE VARIATION

The first historical reference to fever was observed on a Sumerian pictogram in the sixth century BC and the first robust scientific study to address normal temperature variation was performed by Carl Reinhold Wunderlich in the nineteenth century. Over 1 million temperature measurements were obtained on approximately 25,000 subjects, establishing a normal value for the healthy human as 37°C.[1] It is now clear that "normal" temperature represents a range of values rather than a single value. Furthermore, diurnal variation in temperature exists such that the lowest body temperature occurs in the early morning (at approximately 4 am) and the highest, in the early evening (at approximately 4 pm). Significant elevations of core body temperature can also occur from endogenous or exogenous factors. Among the exogenous factors that can influence this rate, ambient temperature and humidity are most important.

FEVER SYNDROMES AND DEFINITION OF FEVER OF UNKNOWN ORIGIN

Fever in a child is an everyday occurrence in an office-based pediatric setting and accounts for up to 25-30% of all physician encounters.[2,3] Prolonged fevers account for only a minority of these visits.

The term *fever of unknown origin* (FUO) was first coined in 1961 by Petersdorf and Beeson and directed the evaluation of the adult patient.[4] They introduced the concept that FUO be defined as a temperature higher than 38.3°C on several occasions and lasting longer than 3 weeks, with a diagnosis that remains uncertain after 1 week of investigation. The classic spectrum of disease that they outlined included "no diagnosis," infections, inflammatory diseases, and malignancies. Deep vein thrombosis and temporal arthritis in the elderly were important considerations. Conventional bias at that time held that most adult patients presenting with FUO had serious, potentially life-threatening disease. The traditional approach for evaluation of FUO in the adult patient involved a staged evaluation culminating in tissue diagnosis.

In the first observational survey of children with FUO, Pizzo et al in 1975 described 100 children with fever greater than 38.5°C lasting for >2 weeks and identified infections as the most commonly established diagnoses.[5] Pizzo's experience suggested that an aggressive staged approach to diagnosis was not necessary in most pediatric cases as most patients had reversible or treatable disease, usually an infection. They emphasized that most children recovered, and prognosis was usually good. Diagnoses that were age-based were described, and infectious diseases predominated. A small percent of those younger than 6 years had malignancy (neuroblastoma or leukemia) or rheumatoid disease (juvenile arthritis). Similarly, children aged >6 years occasionally had an oncologic or rheumatologic diagnosis (leukemia, lymphoma, systemic lupus erythematosus). An organized approach in such cases was felt to be essential to identify those children with prolonged fever related to treatable infection or noninfectious disease to avoid morbidity and occasional mortality.