

Introduction

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Urticaria is one of the most frequently observed dermatological conditions: 15 to 20% of individuals in Europe and America has at least one acute eruption during his or her lifetime, resulting in 1 to 2% of dermatological and allergological consultations. Urticaria is considered chronic when the eruption lasts for longer than 6 wk. It can last for an average of 3 to 5 yr. Because the origin and pathogenesis of urticaria are often unclear, management can be frustrating to both patient and physician.

In this issue, the authors provide the most recent data and ideas regarding this disconcerting disease.

Hennino and colleagues specify how urticaria may result from different pathophysiological mechanisms; immunological reaction mediated by antibodies, and/or T-cells or non-immunological reactions through membrane receptors involved in innate immunity or by direct toxicity of xenobiotics, resulting in mast cell activation. Mast cells from patients with urticaria appear fragile and more sensitive to activation signals than mast cells from normal

individuals. There are three metabolic consequences of mast cell activation: degranulation inducing the immediate release of histamine and serotonin, proteases, proteoglycans, tumor necrosis factor- α ; production of a range of cytokines and chemokines; and synthesis of leukotrienes and prostaglandins. These different mediators explain the great heterogeneity of clinical symptoms and the variety of responses to treatment.

Among involved cells in urticaria, eosinophils have an important role. Staumont-Salle and colleagues provide recent data regarding their role in urticaria. These cells release tissue-damaged proteins as cationic proteins, and also produce many other pro-inflammatory molecules in the form of toxic oxygen radicals and eicosanoid mediators. These molecules may play a critical role in physical urticarias, such as primary cold, solar, or delayed-pressure urticaria. When antihistaminic remains ineffective, these mediators may represent new therapeutic targets, such as leukotriene blockers, cytokine, and anticytokine therapies.

Drugs are a classic cause of urticaria. Mathelier-Fusade discusses the different mechanisms by which drugs can cause and exacerbate urticaria. Acute urticaria may be induced by immunoglobulin E antibodies as allergic reaction to β -lactams and can occur in a nonimmunological context when a drug acts directly on mast cell mediator release. On other hand, in chronic urticaria (CU), drugs more often act by a pharmacological mechanism involving the arachidonic acid metabolism, such as acetyl salicylic acid and nonsteroidal anti-inflammatory drugs, or bradykinin, such as angiotensin converting enzyme inhibitors. Drugs can sometimes cause contact urticaria. This is produced by immunological or nonimmunological mechanisms.

The relationship between urticaria and viral infections is controversial. Cribier suggests that the association between acute urticaria and viral hepatitis is commonly observed. On the other hand, no convincing evidence exists to suggest that hepatitis virus infection causes CU. With the exception of cases involving urticarial vasculitis, systematic investigations in patients with CU probably are not cost-effective.

Irritant dermatitis and eczema are the most prevalent occupational skin diseases. However, contact urticaria is not unusual in some groups of workers. Bourrain discusses physiopathology, epidemiology, and etiologies—par-

ticularly allergy to natural rubber latex. He emphasizes cutaneous exploration and primary and secondary prevention.

Thyroid auto-immunity has been reported to be associated with CU and angioedema. I review the literature about this association. Frequency of antithyroid antibodies is estimated between 10 and 30%, although prevalence of these antibodies in the normal population is generally estimated between 3 and 6%. Sometimes associated with anti-immunoglobulin E receptor antibodies, antithyroid antibodies are not a direct causative agent of CU. Hormonal treatment has not been demonstrated to have an effect on the progression of urticaria.

Epidemiological surveys concur with clinical observations to recognize the possibility that psychological or emotional factors can act as an aggravating, or even as a triggering, factor in certain forms of CU. The same surveys recognize the differing psychological repercussions of urticaria, such as pruritus, visibility, and especially possible lethal risk of laryngeal angioedema. Grob and Gaudy-Marqueste clearly demonstrate that CU severely impairs health-related quality of life at the same levels as other chronic skin conditions, such as psoriasis and atopic dermatitis.

Thanks to these articles, I hope that allergists, dermatologists, internists, and general practitioners will have a fresh view of urticaria.