

Preface
Urticaria



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Guest Editor

Patients with chronic urticaria and angioedema face a daunting challenge. They have a condition that looks like an allergic reaction yet is rarely due to an allergic process. Urticaria may be due to physical stimuli or may arise without external provocation. Physicians with busy practices often do not have the time to learn the subtleties of the varied presentations of urticaria and may not appreciate the difficulties experienced by the patient. Treatment regimens tend to be symptomatic and suboptimal. Unfortunately, this condition sparks little interest in the general research community, possibly because mortality is rare and there is not an obvious genetic component for most of these conditions.

Yet physicians who care for these patients generally concur that chronic urticaria is an intriguing condition that can be quite distressing and even disabling for many patients. Chronic urticaria can affect the ability to sleep and concentrate, alter self-image, and undermine self-confidence.

Since the last time this subject was reviewed in the *Immunology and Allergy Clinics of North America* (1995), there has been substantial progress in both our understanding of the pathophysiology and the treatment of chronic urticaria, but we are far from a satisfactory endpoint. The introduction of new nonsedating antihistamines, leukotriene pathway modifiers, and COX2 inhibitors has altered our approach to anti-mast cell mediator therapy and improved outcomes for many patients. Immunologic and histologic studies have detailed the nature of the cellular infiltrates in urticarial lesions and provided knowledge about the cytokines that may be important. Controlled trials with the immunomodulator

cyclosporine have further broadened our pharmacologic approach. The development of the autologous serum skin test (ASST) has been an important advance, leading to the observation that a substantial number of patients with chronic urticaria have serum factors that probably contribute to their disease. Furthermore, most of these patients with a positive ASST have autoantibodies that recognize the high-affinity receptor for immunoglobulin E (IgE) or IgE itself and can functionally cross-link the IgE receptor, leading to degranulation of mast cells and basophils. Yet many questions remain. For example, we do not understand the basis for urticaria in the many patients who lack autoantibodies and whose urticaria can be just as intense as those patients who have detectable autoantibodies. Even in patients who have autoantibodies, we do not understand why the hives are localized, why they may be more prevalent one day compared with the next day, and why they may be present for several months, remit, and then recur years later.

The articles in this issue are designed to review the current literature regarding the varied types of urticaria, their pathophysiologic basis, and current pharmacologic approaches. Efforts have been made to discuss controversial areas and to highlight those aspects of this field for which our knowledge is still inadequate.

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