A Sea Change in the Treatment of Chronic Urticaria☆

Quién lo ha visto y quién lo ve? Tratando la urticaria crónica

Chronic urticaria is primarily an autoimmune disease, mediated either by immunoglobulin (Ig) E or IgG, acting on the high-affinity IgE receptor (FcεIgERI), which is overexpressed both in skin mastocytes and circulating basophils. Different exacerbating factors contribute to the manifestation of the condition as well as its conversion to a chronic process. Regardless of whether the spontaneous or inducible type of disease is present, the chosen therapy should be effective and safe, and it needs to be maintained for long periods of between 1 to 5 years. The therapeutic goal is to keep the patient completely free of symptoms so that she or he can lead a normal life. With this aim, omalizumab has become an essential addition to the therapeutic armamentarium for the treatment of chronic urticaria. The authors of the commented article reflect on their experience with the use of this monoclonal antibody in daily clinical practice in accordance with the recommendations of the most recent treatment guidelines.[1] In daily clinical practice, their patients achieve better therapeutic outcomes than in clinical trials, measured in terms of the urticaria activity score (UAS) in the 7 days before the study visit. Only 1 phase II study, EXCUISITE, reports percentages of complete control similar to those observed in clinical practice. That study only included patients with IgE anti-peroxidase; that is, patients were selected for an FcεIgERI activation mechanism or autoallergy. This suggest that this type I autoimmunity mechanism is the predominant one. In contrast, IgG anti-FcεIgERI, implicated in type 2 b autoimmunity mechanism, is less frequent. The authors of the commented article analyzed clinical response and complete response at 3 and 6 months of treatment and found higher percentages of responders than those reported in the clinical trials. To define predictors of response, they link later complete response with those patients with longer-standing urticaria (≥ 18 months), who are perhaps the most refractory patients. The article does not discuss studies that suggested that this group of patients might be affected by type IIb autoimmunity as indicated by a positive autologous serous skin test (ASST) or positivity for functional serum autoantibodies. The authors report agreement with previous studies such as the study by Curto-Barredo et al.[2] that the slowest response to omalizumab can also be linked to the use immediately beforehand of immunosuppressive medication, such as ciclosporin A. This was the observation of the authors of the Xarxa d'Urticària Catalana i Balear (XUrCB) in a cohort of 286 patients in a bivariate analysis. To finish, we would like to congratulate the authors on the efforts to systematize their experience in the management of chronic urticaria using omalizumab. This is the only way to properly evaluate our day-to-day work.

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