URTICARIA, ANGIOEDEMA

WHAT WILL THE FUTURE HOLD? HOW WE WILL TREAT URTICARIA AND ANGIOEDEMA IN 2020 AND BEYOND

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"The history of urticaria represents a fascinating, account of man´s gradually increasing understanting of medicine as such, and of the unique clinical features of the pathomechanisms of urticaria in particular" ("Urticaria",1986) . We can affirm with forcefulness that the most recent pathogenic and therapeutic advances in urticaria are constituted as in a true revolution. Currently, the expectations of therapeutic success for the patient and for the doctor are frankly much better than 10 years ago and will be even better in the immediate future. The success of the management of CSU lies on a perfect strategic plan. The EAACI; GA2LEN;EDF;WAO Urticaria guidelines (2018) define as successful therapy such that obtain complete resolution of signs (hives and angioedema) and symptoms (itch and pain). A basic principle of efficacy and safety is desirable; it is the therapeutic goal, as the clinical experience holds that treatment should continue for extended periods of time, with adaptations according to changes in symptoms. Nowadays the unique recommended third line of treatment consists of adding to the antihistamines an incredible drug for CSU, omalizumab. We learned from our practice and we have data about omalizumab behave: prediction of CSU fast-slow and no response, the need to up dose, relapse and retreatment, use in special populations, efficacy for angioedema and CIndUs, or safety of long term treatment. With the advent of disease-related biomarkers, newer biologic agents are coming forth to revolutionize management of CSU. The wheal is a consequence of mast cell degranulation through different keys, including cross-linkage of immunoglobulin (Ig)E and IgG bound to the high-affinity IgE receptors (FcεRI) on the surface of the mast cells and basophiles. Histamine and other mediators as, Platelet Activating Factor or PGD2 leads to hives and angioedema. Vasodilatation induces erythema and the oedema is consequence of neutrophils, lymphocytes, basophils and mainly eosinophils chemoattraction through leaky capillary. Based in molecular and genetic pathogenic findings several new treatments could also be proposed for CU. Ongoing new therapeutic development includes more potent anti-IgE therapy as liguelizumab. Other drugs targeting different pathogenic pathways are in ongoing research as Spleen tyrosine kinase (Syk) inhibitors, anti-CD20 mAb, Bruton tyrosine kinase (Btk) inhibitors, Chemoattractant receptor homologous molecule expressed on TH2 cells (CRTH2) inhibitors, Siglec-8 inhibitors, anti-PGD2 and some interleukin inhibitors including IL-4/ IL13,

IL-6 and IL-5. We are facing new opportunities to offer a healthy life to CU patients. The identification and validation of reliable biomarkers in CSU and CIndU would be useful to define the patient's disease status leading to a more individualized and personalized treatment and follow-up, improving symptoms control, quality of life and decreasing the burden of the disease. Moreover, the first results about genotype expression in CSU have been published. Their further correlations with CSU phenotypes are reported. New therapeutic developments for CU should be based in the principle defined by efficacy and safety to obtain as fast as possible the complete control of signs and symptoms. Disease improvement means a continuous medical education for which active CU networks (e.g UCARE) are crucial.