

Efficacy of omalizumab in delayed pressure urticaria: a case report

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Although the exact mode of action is presently unknown, the efficacy of treatment of chronic urticaria with anti-IgE (Omalizumab, Xolair, Novartis) has been reported recently (1). We present the successful treatment of a patient with severe delayed pressure urticaria (DLP) with omalizumab 150 mg every second week.

The patient is a now 39-year-old female with a 10-year case history of severe debilitating DLP, elicited by even slight pressure preventing the patient from normal daily activities such as manual work, horse riding, walking in

high heels, bicycling, or marital activities. Initially, the patient presented with leucocytosis of unknown origin, which was normalized after treatment with azithromycin 500 mg for 3 days and positive test for autoimmune urticaria (3) (HR-Test, Reflab, Copenhagen, Denmark). Partial relief, only, was obtained with fexofenadine 540 mg [triple dose (2)] and azathioprine 100 mg per day was added for a period of 10 months. On this treatment, partial relief was obtained initially but the effect vanished over time. Azathioprine was substituted with cyclosporine 200–350 mg per day (3–5 mg/kg) with initial marked effect which declined over the next 2 years and was stopped due to side effects (lanugo hair, renal impairment) hereafter.

April 22, 2009: the patient still on fexofenadine was given 150 mg omalizumab subcutaneous (serum IgE 32 kIU/l) and already 2 days after this first injection the patient was totally free from any symptoms of DLP. The injection was repeated every second week and the patient is still free from any symptoms on the same dose today (July 2009) without any side effects. Serum IgE has gradually risen to now 184 kIU/l.

We have followed the clinical improvement with repeated measurements of basophil histamine release, elicited with anti-IgE (KPL, Maryland,

USA) (Fig. 1) and a marked decrease in releasability has been obtained. Serum tryptase and serum ECP (Phadia AB, Uppsala, Sweden) has been measured immediately before and 2 h after each injection without any significant increase neither before/after nor over the treatment period.

This is, to our knowledge, the first report of successful treatment of DLP with omalizumab. The efficacy is paralleled by a decrease in basophil releasability. The down regulation of IgE-mediated basophil activity may indicate that IgE signalling through the basophil is involved in the pathogenesis of delayed pressure urticaria.

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References

1. Kaplan AP, Joseph K, Maykrut RJ, Geba GP, Zeldin RK. Treatment of chronic autoimmune urticaria with omalizumab. *J Allergy Clin Immunol* 2008;**122**:569–573.
2. Zuberbier T, Bindslev-Jensen C, Canonica W, Grattan CE, Greaves MW, Henz BM et al. EAACI/GA2LEN/EDF. EAACI/GA2LEN/EDF guideline: management of urticaria. *Allergy* 2006;**61**:321–331.
3. Platzter MH, Grattan CEH, Poulsen LK, Skov PS. Validation of basophil histamine release against the autologous serum skin test and outcome of serum-induced basophil histamine release studies in a large population of chronic urticaria patients. *Allergy* 2005;**60**:1152–1156.

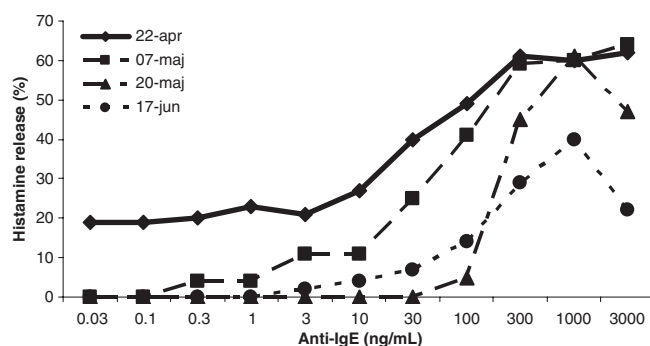


Figure 1. Changes over time in basophil releasability during treatment with omalizumab.