

upholstered furniture, triggering atopic sensitization (4). Thus, very high reactivity to indoor allergens of asthmatic patients from west Siberia (Russia) seems to be a man-made problem and could be partly avoided by reducing risk allergen concentration at homes by frequent ventilation and cleaning of flats.

This is the first report on allergic sensitization in asthmatic patients from Russia. It shows the importance of cat, dog and dust mite allergens in maintaining the manifestations of asthma. Our data can serve as a baseline for future studies of risk factors in asthma development in Russia.

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References

1. Gould HJ, Sutton BJ, Beavil AJ, Beavil RL, McCloskey N, Coker HA, et al. The biology of IgE and the basis of allergic disease. *Annu Rev Immunol* 2003;**21**:579–628.
2. Heinzerling L, Frew AJ, Bindslev-Jensen C, Bonini S, Bousquet J, Bresciani M et al. Standard skin prick testing and sensitization to inhalant allergens across Europe – a survey from the GA² LEN network. *Allergy* 2005;**60**:1287–1300.
3. Arbes AJ, Cohn RD, Yin M, Muilenberg ML, Friedman W, Zelding DC. Dog allergens (Can f 1) and cat allergens (Fel d 1) in US homes: results from the National Survey of Lead and Allergens in Housing. *J Allergy Clin Immunol* 2004;**114**:111–117.
4. Munir AKM. Mite sensitisation in the Scandinavian countries and factors influencing exposure levels. *Allergy* 1998;**53**:64–70.

No evidence of tumor necrosis factor- α release in blood of patients with chronic urticaria

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Key words: autologous serum skin test; chronic urticaria; tumor necrosis factor- α .

Mast cells play a key role in chronic urticaria as they release histamine and other inflammatory mediators and cytokines causing urticarial symptoms. In about 30% of chronic urticaria patients, mast cell activation is triggered by autoantibodies directed against the α -subunit of the high affinity IgE receptor; in the other patients the stimuli inducing mast cell activation have not been defined, but an immunological mechanism is also suspected. From a practical point of view, the presence of circulating histamine-releasing factors is demonstrated by *in vivo* autologous serum skin test (ASST), which is considered as a screening test for histamine-releasing autoantibodies (1). Positivity of ASST allows distinguishing patients with chronic autoimmune urticaria from patients with an idiopathic disorder. Tumor necrosis factor (TNF)- α is released by human skin mast cells and other inflammatory cells which can be found at the site of urticarial lesions (2). Therefore, TNF- α is a candidate mediator of urticaria. Tillie-Leblond et al. (3) found TNF- α release during a systemic reaction occurring after cold immersion test in two patients with cold urticaria, and an increased immunoreactivity for TNF- α and IL-3 was detected on endothelial and perivascular cells of the upper dermis in skin lesions from chronic urticaria patients (4). We measured serum TNF- α levels in patients with chronic autoimmune and chronic idiopathic urticaria. Sera were drawn from 62 adult patients diagnosed as having chronic urticaria on

Tumor necrosis factor- α is undetectable in peripheral blood of patients with chronic urticaria.

the basis of recurrent hives for more than 6 weeks. In all cases, known causes of chronic or recurrent urticaria were ruled out by appropriate investigations. Physical urticarias were excluded as well. All patients had active urticaria at the time of the study. Five days after anti-histamine therapy (cetirizine, loratadine, or fexofenadine in all cases) was stopped, an intradermal test with 0.05 ml of fresh autologous serum (ASST) was carried out. ASST was performed and read at 30 min following the method by Sabroe et al. (1). Intradermal injection of saline solution (0.9% weight/volume NaCl) was performed as negative control and skin prick test with 10 mg/ml histamine as positive control. Patients showing a wheal with a diameter at least 1.5 mm greater than the control saline solution were considered positive. Forty patients were strongly positive on ASST and 22 were negative. TNF- α was assayed by a sensitive immunoenzyme method (R & D Systems, Minneapolis, MN, USA) using the same serum samples employed for ASST. Sera from 12 normal adult subjects were used as control. TNF- α concentration was below the sensitivity of the assay (15.6 pg/ml) in all serum samples from chronic urticaria patients and normal subjects. These results indicate that TNF- α is not released in measurable amounts in blood of patients with chronic urticaria, either autoimmune or idiopathic. Although a role for TNF- α as mediator of urticarial lesions cannot be excluded, its release in the skin microenvironment does not lead to a substantial increase in blood concentration.

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References

1. Sabroe RA, Grattan CE, Francis DM, Barr RM, Kobza Black A, Greaves MW. The autologous serum skin test: a screening test for autoantibodies in chronic idiopathic urticaria. *Br J Dermatol* 1999;**140**:446–452.
2. Walsh LJ, Trinchieri G, Waldorf HA, Whitaker D, Murphy GF. Human dermal mast cells contain and release tumor necrosis factor alpha, which induces endothelial leukocyte adhesion molecule 1. *Proc Natl Acad Sci USA* 1991;**88**: 4220–4224.
3. Tillie-Leblond I, Gosset P, Janin A, Dalenne R, Joseph M, Wallaert B et al. Tumor necrosis factor-alpha release during systemic reaction in cold urticaria. *J Allergy Clin Immunol* 1994;**93**:501–509.
4. Hermes B, Prochazka AK, Haas N, Jurgovsky K, Sticherling M, Henz BM. Upregulation of TNF-alpha and IL-3 expression in lesional and uninvolved skin in different types of urticaria. *J Allergy Clin Immunol* 1999;**103**:307–314.

Anaphylactic reaction after aceclofenac intake

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Keywords: aceclofenac; anaphylactic reaction; cross-reaction; diclofenac.

Non-steroidal anti-inflammatory drugs (NSAIDs) are one of the main aetiologies of severe anaphylaxis. The prevalence of severe anaphylaxis because of NSAIDs registered by the Allergy Vigilance Network over 2003–2004 (1) was 13%.

A 75-year-old man with a previous history of osteoarthritis, sporadically treated with various NSAIDs, took aceclofenac (100 mg/day). Clinical symptoms appeared 2 h after the second dose intake. Symptoms included itching in legs without cutaneous lesions, palpebral and labial edema, and dyspnea. The patient was treated with methylprednisolone and dexchlorpheniramine immediately. After

this episode, therapeutic doses of diclofenac as well as other anti-inflammatory drugs, such as Ibuprofen and Metamizol, were tolerated.

Cutaneous tests with aceclofenac, both prick and intradermal tests (30 mg/ml and 3 g/ml, respectively), were performed. The result was positive for the intradermal test. Five controls were tested, with negative results.

Aceclofenac, [o-(2,6-dichloroanilino) phenylacetate glycolic acid ester, is a NSAIDs of the phenylacetic acid group (2). After its absorption, the drug is hydrolyzed to diclofenac (3), also from the phenylacetic acid group (Fig. 1). Alclofenac and fenclofenac, two NSAIDs with similar chemical structure to aceclofenac, were withdrawn in the late 1970s and 1980s because of the high incidence of rash (4).

The most common hypersensitivity reaction described with aceclofenac is hypersensitivity vasculitis (4). A case of photoallergic contact dermatitis (5) and another one of exudative erythema multiforme (6), both related to aceclofenac, have been reported.

Two of these cases, showed no cross-reaction between aceclofenac and diclofenac, as occurred in the case reported above. It suggests that cross-reactions among phenylacetic-derived NSAIDs are not so frequent.

To our knowledge, this is the first report of an anaphylactic reaction after the intake of aceclofenac. Clinical features and cutaneous test with aceclofenac suggest an immunoglobulin E (IgE)-mediated mechanism.

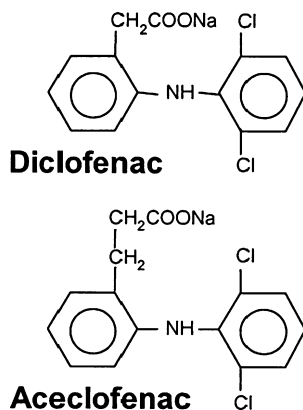


Figure 1. Chemical structure of diclofenac and aceclofenac.

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References

1. Moneret-Vautrin DA, Morisset M, Flabbee J, Beaudouin E, Kanny G. Epidemiology of life-threatening and lethal anaphylaxis: a review. *Allergy* 2005;**60**:443–451.
2. Sweetman SC, editor. Martindale: The Complete Drug Reference: 2002. London: Pharmaceutical Press, 2002.
3. Grau M, Montero JL, Guasch J, Felipe A, Carrasco E, Julia S. The pharmacological profile of aceclofenac, a new non-steroidal anti-inflammatory and analgesic drug. *Agents Actions* 1991;**32** (Suppl):125–129.
4. Morros R, Figueras A, Capella D, Laporte JR. Hypersensitivity vasculitis related to aceclofenac. *Br J Rheumatol* 1997;**36**:503–504.
5. Goday Buján JJ, Garcia Alvarez-Eire GM, Martínez W, del Pozo J, Fonseca E. Photoallergic contact dermatitis from aceclofenac. *Contact Derm* 2001;**45**:170.
6. Ludwig C, Brinkmeier T, Frosch PJ. Exudative erythema multiforme with transition to a toxic epidermal necrolysis after taking aceclofenac. *Dtsch Med Wochenschr* 2003;**128**:487–490.

Sublingual immunotherapy and regulatory T cells

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Key words: bronchial hyperreactivity; IL-10 production; nasal eosinophils; regulatory T cells; sublingual immunotherapy.

Recently, a role for regulatory T cells (Tregs) has been evidenced in patients undergoing specific immunotherapy (SIT) for grasses and mites (1). Thus, it has been pointed out that SIT generates Tregs, secreting both interleukin(IL)-10 and tumour growth factor (TGFβ) and