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Multiple sensitivity to NSAID

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Key words: cross-reactivity; drug allergy; nonsteroidal anti-inflammatory drugs; urticaria.

● It is generally believed that chemically unrelated nonsteroidal anti-inflammatory drugs (NSAID) may induce urticaria relapses in patients with chronic urticaria (CU), whereas normal subjects who experience acute urticaria after taking NSAID are monosensitive and may take other NSAID with impunity (1). However, several studies (2–5) suggest that some individuals without CU might have multiple NSAID sensitivity as well. If this were demonstrated, we should change, at

least in part, our approach to NSAID-intolerant patients.

A total of 261 patients (78 males, 183 females; aged 14–75 years, mean 42 years) without CU but with a history of urticaria induced by NSAID were studied. Exactly 179 of them agreed to undergo single-blind, placebo-controlled peroral tests with acetaminophen (A), nimesulide (N), and/or floctafenine (F) in order to detect reaction to at least one alternative drug (3–5). The following doses were given 1 h apart: A: 125, 250, 500 mg; N: 25, 50, 100 mg; F: 50, 100, 200 mg. Drugs under investigation were challenged at least 1 week apart. Only the appearance of unequivocal urticaria was considered a positive response.

A total of 336 peroral challenges (166 with A, 149 with N, 21 with F) were performed; 29/179 (16%) patients experienced a total of 36 urticaria/angioedema reactions (15, 17, and 4 after A, N, and F, respectively). All reactions were mild and immediate, and occurred after the first or second provocative dose.

Of the patients, 178/261 (68%) had a history of intolerance to a single NSAID. Aspirin and pyrazolones were most frequently involved (Table 1). Exactly 11/126 (9%) patients did not tolerate at least one challenged drug (A, N, and F in five, seven, and one case, respectively); 83/261 (32%) patients had a history of multiple NSAID intolerance (63 subjects to two drugs, 19 to three, and one to four drugs).

Aspirin and pyrazolones were most frequently involved (Table 1). Exactly 18/53 (34%) patients did not tolerate at least one challenged drug (A, N, and F in 10, 10, and three cases, respectively). Reactions were much more frequent among patients with a history of multiple NSAID intolerance than among patients with a history of intolerance to a single NSAID ($P<0.001$) (Table 1). A history of multiple NSAID intolerance was a significant risk factor for intolerance to one of the challenged drugs ($RR=5.4$). On the basis of both clinical history and challenge tests, at least 94/261 (36%) patients were finally considered to have multiple NSAID intolerance.

These findings suggest that multiple NSAID intolerance is rather common also in individuals without CU; interestingly, the proportion of multiple NSAID reactors is very similar to that found in patients with CU (1). Thus, all patients with a history of NSAID-induced urticaria should undergo tolerance tests with at least two chemically unrelated compounds in order to detect subjects prone to multiple reactivity, and to prevent potentially severe adverse reactions with full dose therapies. In otherwise normal patients, false positive results are very unlikely and a single-blind, placebo-controlled protocol seems adequate. Finally, peroral challenges seem to be quite safe, and can be easily performed in all normally equipped settings.

Table 1. Offending drugs and challenge tests results

	SI (n=178)	MI (n=83)	P	Total (n=261)
Aspirin	71	75		146
Pyrazolones	68	62		130
Diclofenac	21	16		37
Propionic acid derivatives	9	23		32
Oxicams	7	10		17
Indomethacin	2	1		3
No. submitted to tolerance tests	126	53		179
No. positive on tolerance tests	11 (9%)	18 (34%)	<0.001	29 (16%)

SI: patients with history of single drug intolerance; MI: patients with history of multiple drug intolerance.

Acknowledgments I thank the nurses of the allergy center, Stefania Arienti, Ombretta Dolcino, and Aurelio Tirloni, for their skillful assistance.

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Accepted for publication 17 April 2000
Allergy 2000; 55:893–894
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ISSN 0105-4538

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Serum IgE levels in HIV-infected patients in Poland

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Key words: chemokines; HIV; serum IgE.

● DAMAGE to functional cellular mechanisms may be observed during the progression of HIV infection. The absolute number of CD4+ T (CD4+) cells decreases in the later phases of HIV infection. Despite

People with high IgE may be less susceptible to HIV.

the immune deficit, the progression of HIV infection is accompanied by the stimulation of antibody synthesis; e.g., IgE. IgE overproduction may have clinical effects, such as predisposition to IgE-mediated allergic reaction, an effect which could partially explain the higher susceptibility to allergic reactions in HIV-infected (HIV+) patients, especially in the later stages of infection. This study aimed to evaluate the total serum IgE level (sIgE), its individual dynamics, and the dependence on CD4+ in a group of HIV+ patients from Poland.

The study group comprised 177 adult subjects from Poland, in different stages of HIV infection, whose sIgE concentration and CD4+ in peripheral blood were determined. In this group, 33 HIV+ subjects

had two and 15 three determinations of the above-mentioned parameters at average intervals of 18 months. The control group included 144 healthy blood donors. The analysis included the differences in sIgE (lnIgE) distribution in HIV+ groups in various stages of the disease according to CD4+/μl; the differences in CD4+ distribution in HIV+ groups with increased and normal IgE; and the frequency of increased sIgE in the control group and HIV+ groups in various stages of disease according to CD4+. Similar analysis was also done in the subgroups of patients who had several determinations of parameters.

Serum samples were checked for anti-HIV antibodies with commercial test kits (Vironostica HIV Uni-Form II plus O, Organon Teknika, The Netherlands). Each positive enzyme-linked immunosorbent determination result was checked again and confirmed by immunoblot (HIV-1 Western-Blot Kit, Organon Teknika, The Netherlands). The CD4 T-cell subset was determined by flow cytometry (Simultest IMK Plus, Becton Dickinson, USA). Serum IgE antibodies were determined by the fluoroallergosorbent commercial test (Total IgE II Fast Test, Biowhittaker, Inc., USA). The increased serum IgE concentrations corresponded to the above 135 IU/ml. The absolute serum IgE concentrations were also transformed into logarithms.

sIgE (lnIgE) distribution differed between the HIV+ group with CD4+ <199 and the

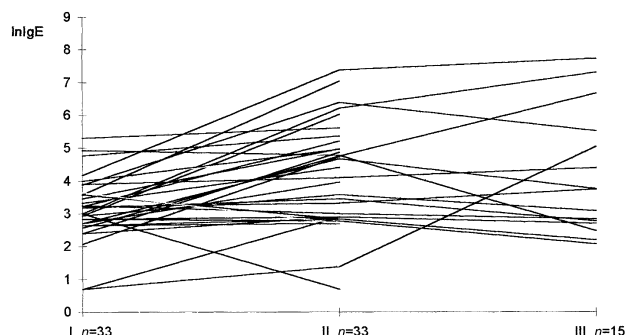


Figure 1. Individual dynamics of lnIgE in succeeding determinations in HIV-infected patients.