

The diagnosis of EF was confirmed, and the patient was given prednisone, 20 mg/d. Follow-up 3 weeks later revealed resolution of her lower-extremity swelling and erythema and minimal swelling of her upper extremities. Her eosinophilia had resolved (eosinophil count, 127/mm³).

A rare inflammatory disorder first described by Shulman,² EF is characterized by symmetric painful swelling and induration of the extremities. It has rapid onset and progression. If untreated, flexion contractures may develop. Peripheral eosinophilia and hypergammaglobulinemia are commonly encountered. It can also be associated with various disorders, including monoclonal gammopathies of undetermined significance (MGUS), multiple myeloma, inflammatory arthritis, rheumatoid factor positivity, thyroiditis, aplastic anemia, neuropathies, myeloproliferative and lymphoproliferative disorders, pulmonary fibrosis, lymphoma, leukemia, Sjögren syndrome, myelodysplastic syndrome, eosinophilic colitis, antinuclear antibodies, pericarditis, medium vessel vasculitis, glomerulonephritis, Raynaud phenomenon, myositis, and acquired amegakaryocytic thrombocytopenia.³⁻⁵ Our strongest suspicion for a trigger in this patient was MGUS, and given her age, she is being monitored for the development of a hematologic malignancy.

Our patient had marked peripheral eosinophilia but a paucity of eosinophils in the biopsy specimen, findings not unusual in this entity.⁶ It is possible that the damage was due to mediators released by eosinophils rather than by their physical presence. Timing may also be key in that eosinophils may have been present in the muscle at the beginning of the process, initiating a self-sustaining inflammatory cascade. In support of this possibility is the fact that the patient had elevated inflammatory cytokine levels and eotaxin present in her serum. The potential source of eotaxin is unknown.

In summary, the cardinal feature that led us to consider the diagnosis of EF was the fact that the edema was limited to the muscle planes while sparing the surrounding tissue. This rare but treatable disease should be included in the differential diagnosis of angioedema with eosinophilia.

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HIGH-DOSE SUBLINGUAL IMMUNOTHERAPY IN CHILDREN AT 8-YEAR FOLLOW-UP

Sublingual immunotherapy (SLIT) is efficacious and safe¹⁻³ and prevents both asthma onset and new sensitizations.^{4,5} Because the long-term effects of SLIT have been poorly studied,⁶ we performed a retrospective study in a group of children with rhinoconjunctivitis caused by *Parietaria* pollen.⁷ These children had been included 8 years ago in a randomized, placebo-controlled study and treated with a high dose of SLIT for 2 years.⁷ The aim of this study was to evaluate the long-term effects of SLIT on the clinical course of rhinoconjunctivitis, on the onset of asthma, and on development of new sensitizations.

The original group of patients included 41 children (mean age, 10 years; 20 received SLIT and 21 received placebo).⁷ The SLIT consisted of a *Parietaria judaica* extract (Stallergenes, Antony, France) standardized in index of reactivity (IR) and containing 70 µg/mL of the major allergen Par j 1 in the 100-IR extract. The cumulative dose received by each patient during the 2 years was 150,000 IR (corresponding to 52.5 mg of Par j 1). This dose was approximately 375 times greater than the dose given in an equivalent subcutaneous immunotherapy course.

In the former study, we observed a significant reduction in symptoms of rhinitis and skin reactivity, an increase in the threshold dose for the conjunctival allergen provocation test, and an increase in specific IgG4 levels in the active treatment group when compared with the placebo group.⁷ Eight years later we reevaluated 21 patients (10 active and 11 placebo). The characteristics of the patients are reported in Table 1. We found that the symptom of rhinitis during the *Parietaria* pollen season was similar in the SLIT and placebo groups. Our results do not

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Table 1. Characteristics of Formerly Active and Placebo Patients Reevaluated 8 Years Later

Characteristics	Formerly active	Formerly placebo	Comparison
No. of patients	10	11	
Sex, M/F	6/4	4/7	$\chi^2 = 0.41$
Current mean age, y	21.7	22.9	NS
Asthma (intermittent/mild persistent)	6 (4/2)	7 (5/2)	$\chi^2 = 0.078$
Mean (SD) FEV ₁ , % predicted	97.5 (11.2)	92.6 (16.4)	$t = 0.79$
No. of patients with FEV ₁ <80%	1	3	NS
Mean (SD) FEF _{25%-75%}	79.9 (22.1)	69.9 (16.4)	$t = 1.18$
No. of patients with FEF _{25%-75%} <80%	3	7	$\chi^2 = 1.21$
No. of new sensitizations	19	20	NS

Abbreviations: FEF_{25%-75%}, forced expiratory flow between 25% and 75%; FEV₁, forced expiratory volume in 1 second; NS, not significant.

support the preventive effect of SLIT on asthma because we did not find any difference in the respiratory function (forced expiratory volume in 1 second) between the 2 groups.

In the former study, 6 patients in the active group and 5 patients in the placebo group were polysensitized, whereas all the others were monosensitized to *Parietaria* pollen. During the follow-up, monosensitized patients developed new sensitizations in both the SLIT and placebo groups, suggesting that SLIT for 2 years is ineffective in preventing new sensitizations.

Taken together, our data indicate that high-dose SLIT for 2 years is not sufficient to modify significantly the natural history of respiratory allergy to *Parietaria* pollen. When our first study was conducted from 1995 to 1997, the knowledge of the effects of SLIT was rather limited, and in particular it was not yet clarified that 3 to 5 years may represent an optimal duration.^{1,8} Furthermore, we can speculate that the prolonged exposure to *Parietaria* pollen (almost perennial in southern Italy) may lead to a greater degree of airway inflammation, which is likely to be associated with asthma onset. The data from our retrospective study indicate the need to perform new studies on SLIT in a larger number of patients with allergic rhinitis associated with *Parietaria* sensitization.

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METABOLIC PROFILE OF PLASMA FREE FATTY ACIDS IN PATIENTS WITH ASPIRIN-INTOLERANT URTICARIA

Acetylsalicylic acid (aspirin) and nonsteroidal anti-inflammatory drugs may act as an aggravating factor or augment urticaria after intake with a specific food.^{1–3} We speculated that aspirin-intolerant urticaria (AIU) may be related to altered fatty acid (FA) composition and investigated plasma FA composition during aspirin provocation.⁴

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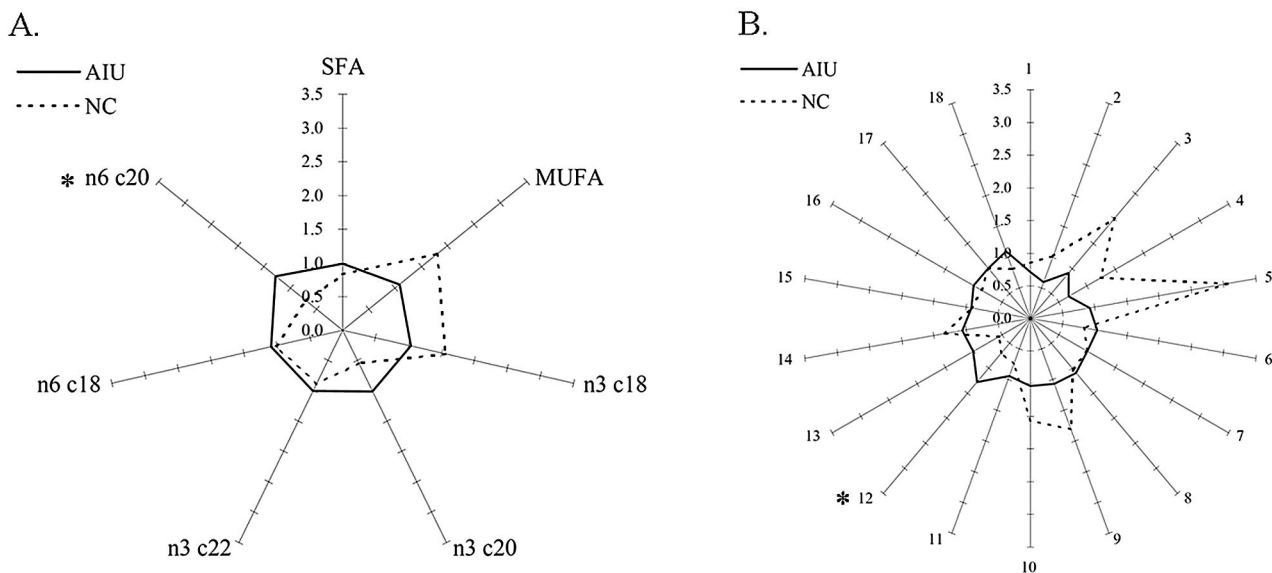


Figure 1. Aspirin-intolerant urticaria (AIU) and control (NC) groups based on the induction folds of the 18 fatty acid composition after normalization to the corresponding basal composition. A, Induction folds of saturated fatty acid (SFA), monounsaturated fatty acid (MUFA), and polyunsaturated fatty acid after aspirin provocation. B, Induction folds of free fatty acids after aspirin provocation. 1 indicates capric acid; 2, lauric acid; 3, myristoleic acid; 4, myristic acid; 5, palmitoleic acid; 6, palmitic acid; 7, γ -linolenic acid; 8, linoleic acid; 9, oleic acid; 10, α -linolenic acid; 11, stearic acid; 12, arachidonic acid; 13, eicosapentaenoic acid; 14, eicosanoic acid; 15, docosahexaenoic acid; 16, docosapentaenoic acid; 17, erucic acid; and 18, nervonic acid. * $P = .034$. Group comparison of fatty acid composition was performed with the Mann-Whitney test.