

Churg–Strauss syndrome in a case of asthma

L. Richeldi*, G. Rossi, M. P. Ruggieri,
L. Corbetta, L. M. Fabbri

Key words: anti leukotriene; asthma; macrolide; vasculitis.

• CHURG–STRAUSS syndrome (CSS) has been reported in patients taking cysteinyl leukotriene (LT)-receptor antagonists (LTRAs), even though no causal relationship has yet been determined between LTRAs and CSS (1,2). Little attention has been paid to concurrent medications in *asthmatic* patients taking LTRAs, which might be associated with the emergence of CSS.

An 18-year-old woman presented with fever, general malaise, dry cough and chest tightness. She had IgE-mediated asthma (3) from childhood and was on regular inhalator treatment with fluticasone and formoterol. She was also on zafirlukast 20 mg twice daily (started 6 months before). Antibiotic therapy (oral rokitamicin 400 mg twice daily) was started 10 days before she was admitted with a fever of up to 38°C, dry cough and headache. After antibiotic treatment, the fever persisted and the cough and dyspnoea increased. At admission, body temperature was 37.5°C and physical examination of the chest was negative. Dry cough, general malaise, chest pain and diffuse myalgia were present. Radiological examination of the chest revealed bilateral patchy infiltrates in the upper and middle zones. Erythrocyte sedimentation rate was elevated, white cell count was $15.9 \times 10^9/l$, with an absolute eosinophil count of 10.3 (64.9%). Bronchoalveolar lavage (BAL)

revealed a marked eosinophilic alveolitis ($600 \times 10^9/l$ total cells, 78% eosinophils) (Fig. 1, upper panel). A transbronchial biopsy showed pneumonia-like involvement, characterized by prominent extravascular infiltration by eosinophils (Fig. 1, lower panel). Bilateral sinusitis was diagnosed by CT: a biopsy of the paranasal sinuses also showed marked tissue eosinophilia and vasculitis. LTRA and antibiotic therapies were discontinued and

steroid therapy (80 mg/day of intravenous methylprednisolone) was started. One week later, the white cell count was $13.6 \times 10^9/l$ with an absolute eosinophil count of 0.7 (5.0%). Fever and cough disappeared, the patient rapidly recovered, and the chest radiograph after three weeks was negative.

In this case of CSS in a young asthmatic patient, both macrolide and LTRA therapies were in use when the

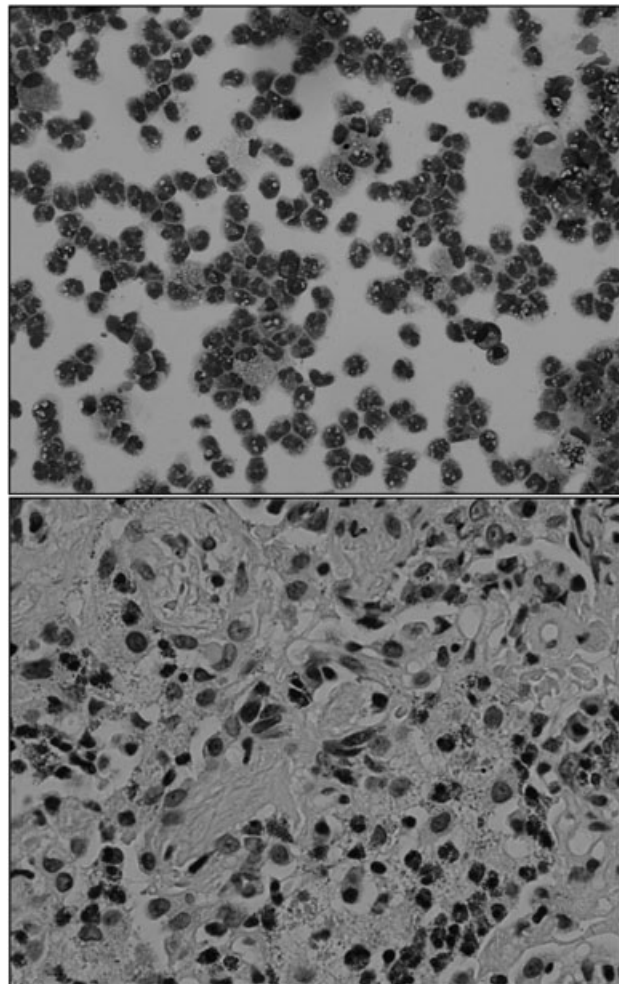


Figure 1. Upper panel: bronchoalveolar lavage cells are, for the vast majority, represented by eosinophils (May Grünwald Giemsa, 25×). Lower panel: formalin-fixed, paraffin-embedded transbronchial biopsy showing an eosinophilic pneumonia-like tissue characterized by prominent extravascular infiltration by eosinophils (Pagoda Red, 40×).

nonallergic drug hypersensitivity occurred (3). Since both classes of drugs have been associated with CSS (2,4,5), we were unable to identify a single pharmacological cause (possibly linked to the induction of the vasculitic reaction).

Asthmatic patients who are reducing oral corticosteroids and/or introducing new anti-asthmatic (notably LTRA) or antibiotic (notably macrolide) treatments, should be monitored carefully for the appearance of a multisystem disease, by measurement of erythrocyte sedimentation rate, C reactive protein and peripheral blood eosinophil count.

*Respiratory Disease Clinic
University of Modena and Reggio Emilia
Via del Pozzo 71
41100 Modena
Italy
Tel: +39 059 4222655/4222198
Fax: +39 059 4224231
E-mail: richeldi.luca@unimo.it

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References

1. WECHSLER ME, PAUWELS R, DRAZEN JM. Leukotriene modifiers and Churg–Strauss syndrome: adverse effect or response to corticosteroid withdrawal? *Drug Saf* 1999;**21**:241–251.
2. WECHSLER ME, FINN D, GUNAWARDENA D et al. Churg–Strauss syndrome in patients receiving montelukast as treatment for asthma. *Chest* 2000;**117**:708–713.
3. JOHANSSON SG, HOURIHANE JO, BOUSQUET J et al. A revised nomenclature for allergy. An EAACI position statement from the EAACI nomenclature task force. *Allergy* 2001;**56**:813–824.
4. DIETZ A, HUBNER C, ANDRASSY K. Macrolide antibiotic-induced vasculitis (Churg–Strauss syndrome). *Laryngorhinootologie* 1998;**77**: 111–1114.
5. KRANKE B, ABERER W. Macrolide-induced Churg–Strauss syndrome in patient with atopy. *Lancet* 1997;**350**:1551–1552.

Allergen in soy oils

Y. Errahali, M. Morisset, D.-A. Moneret-Vautrin, G. Kanny, M. Metche, J.-P. Nicolas, S. Frémont*

Key words: food allergy; immunoblot; protein; soy oil.

• SOY ALLERGY is of particular importance because of the ubiquity of soy proteins in contemporary foods. This allergy appears to affect 6% of atopic children (1) and 14% of children with cow's milk allergy (2). Soy, which has been responsible for several fatal and severe reactions, is probably an underestimated cause of food anaphylaxis (3). Several proteins have been identified in soy oil (4), but to date, the allergenicity of proteins from deodorized soy oil has never been observed. In this paper we show that allergenic proteins are present in both cold-pressed soy oil and deodorized soy oil. The allergenicity of at least one of these proteins has been demonstrated by Western blot.

The serum was taken from a soy-sensitized woman with a high level of soy specific IgE (18.9 kU/l) (RAST, CapSystem, Pharmacia, Uppsala, Sweden). Two kinds of soy oil were tested: deodorized (type A) and cold-pressed (type

B) soy oils were supplied by the same company. The proteins were extracted from 500 ml of each batch using PBS buffer at 40°C and stirred for 3 h. The aqueous phase was dialyzed for 2 days.

The total protein contents of oils A and B were 0.32 µg/ml and 1.8 µg/ml, respectively. The SDS-PAGE of deodorized and cold-pressed soy oils showed the same pattern (Fig. 1a). The immunoblots of batch A and batch B, conducted with the serum of a soy-sensitized patient, showed two bands at 28 and 56 kDa, respectively, the latter being the main band, whereas the immunoblot using serum of a nonsensitized patient did not exhibit any binding (Fig. 1b).

In conclusion, we showed that allergenic proteins were present not only in cold-pressed but also in deodorized soy oil. We are currently trying to find out whether this allergen is clinically relevant.

*Dr S. Frémont
Laboratory of Medical and Pediatric Biochemistry
Faculty of Medicine
BP 184
Vandoeuvre les Nancy
France
Tel: +33 03 8359 2708
Fax: +33 03 8359 2718
E-mail: fremont@lbmp.u-nancy.fr

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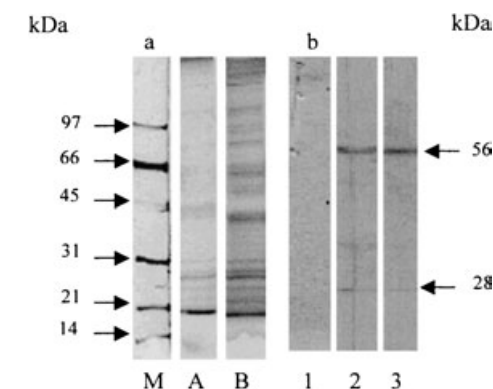


Figure 1. (a) Silver stained SDS-PAGE patterns of proteins from deodorized (A) and cold-pressed (B) soy oils. M: molecular weight marker. (b) IgE immunoblots of proteins from each batch of soy oil. (1) negative control; (2) cold-pressed soy oil (B); (3) deodorized soy oil (A).