

(SCORAD) was 45 (2). Prick tests to cow's milk and soya milk were negative, so diet was not modified till he reached 10 months of age. At this time AD was generalized and patch tests were carried out (3). Positivity to soya milk led to its withdrawal. Despite feeding with a casein extensive hydrolysate, the infant failed to improve: daily

rashes were accompanied by vomiting and diarrhoea. An amino acid formula (AAF)

IgE-mediated allergy to an elemental amino acid formula.

was prescribed. The digestive symptoms resolved but SCORAD remained at 34.

This AAF contains a soy lipidic emulsion. Skin tests with separate components of AAF were carried out. Patch tests were positive to the soy oil emulsion only. An oral challenge to the soy oil emulsion (30 ml in 240 ml of water) induced a rash four hours later.

The AAF was stopped for three days. A partial regression of the inflammatory state was observed. Conversely, the reintroduction of 240 ml of AAF on the fourth and fifth days provoked a rash in the afternoon of day four, and exacerbation of the eczema on day five. SCORAD rose from 27 to 45.

Neocate Advance® that does not contain soy lipidic emulsion was substituted. A complete recovery was observed six weeks later.

Following IgE-dependent allergy to cow's milk, the child developed T cell mediated allergy to soy (1). In such cases, replacing cow's milk with extensive hydrolysates (EH) is advised (4). However, allergy to casein EH occurs in 8% of cases (5). Substitution with AAF is usually effective, and safety is excellent (6). A case of allergy to AAF was attributed to contamination (7).

The role of soy oil was suspected on the basis of the T cell-mediated allergy to soy. Indeed, allergens in soy lecithins and soy oil have been evidenced (8,9) and allergic reactions to soy lecithin have been observed (10).

AAFs are used specifically for infants presenting with severe CMA, often associated with multiple food intolerance or with allergy to hydrolysates. These infants can be considered to be the most atopic subjects, reacting to traces of allergens. It would seem advisable to

replace the soy oil by other vegetable lipids in AAFs.

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References

1. JOHANSSON SGO, O'B HOURIHANE J, BOUSQUET J, et al. A revised nomenclature for allergy. An EAACI position statement from the EAACI nomenclature task force. *Allergy* 2001;**56**:813–824.
2. EUROPEAN TASK FORCE ON ATOPIC DERMATITIS. Severity scoring of atopic dermatitis: the SCORAD index. *Dermatology* 1993;**186**:23–31.
3. ISOLAURI E, TURJANMAA KE. Combined skin prick and patch testing enhances identification of food allergy in infants with atopic dermatitis. *J Allergy Clin Immunol* 1996;**97**:9–15.
4. HÖST A, KOLETZKO B, DREBORG S, MURARO A, WAHN U, AGGETT P, et al. Dietary products used in infants for treatment and prevention of food allergy. *Arch Dis Child* 1999;**81**:80–84.
5. DE BOISSIEU D, DUPONT C. Time course of allergy to extensively hydrolyzed cow's milk proteins in infants. *J Pediatr* 2000;**136**:119–120.
6. ISOLAURI E, SÜTAS Y, MÄKINEN-KILJUNEN S, OJA SS, ISOSOMPI R, TURJANMAA K. Efficacy and safety of hydrolyzed cow milk and amino-derived formulas in infants with cow milk allergy. *J Pediatr* 1995;**127**:550–557.
7. NILSSON C, OMAN H, HALLDEN G, LILJA G, LUNDBERG M, HÄRFÄST B. A case of allergy to cow's milk hydrolysate. *Allergy* 1999;**54**:1322–1326.
8. AWAZUHARA H, KAWAI H, BABA M, MATSUI T, KOMIYAMA A. Antigenicity of the proteins in soy lecithin and soy oil in soybean allergy. *Clin Exp Allergy* 1998;**28**:1559–1564.
9. ZITOUNI N, ERRAHALI Y, KANNY G, MOUTETE F, METCHE M, MONERET-VAUTRIN DA, et al. Soy allergens are detected in some edible soy oils. *J Allergy Clin Immunol* 2001; **107**:S188.
10. PALM M, MONERET-VAUTRIN DA, KANNY G, DENERY-PAPINI S, FRÉMONT S. Food allergy to egg and soy lecithins. *Allergy* 1999;**54**:116–117.

Natural course of AEDS

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Key words: asthma; atopic dermatitis; childhood; intrinsic type; natural course.

• WHAT WAS previously called atopic dermatitis (AD) is a common inflammatory skin disease that often develops in early childhood. Its natural course over the time varies considerably from case to case: the skin manifestation can disappear after infancy, childhood or puberty, but it can also persist in the adolescent and in the adult phase (1,2). Of children with AD 30–60% will at a later age develop respiratory diseases such as asthma bronchiale (AB) or allergic rhinitis (AR). Recently, the term 'atopic eczema/dermatitis syndrome' (AEDS) was proposed by the EAACI nomenclature task force (3) to underline the fact that AD is not one single disease but rather an

Children with nonallergic variety of allergic eczema/dermatitis syndrome rarely get asthma.

aggregation of several diseases with certain clinical characteristics in common (4). The nonallergic AEDS (former called 'intrinsic' type of AD) is characterized by eczematous skin lesions in typical localization similar to the IgE-associated subgroup of AEDS, but is not associated with a specific sensitization either in the skin tests nor in RAST/CAP to common environmental allergens (5).

In this study we performed clinical and allergological investigations: skin prick tests (SPT), total and specific IgE determinations against an inhalant (Sx1) and a food mix (Fx5)) in a group of 22 children with AEDS seen at the age of 2–4 years.

Eight years later the same children, now aged 10–12 years, were re-evaluated. In the follow-up we investigated the persistence of the skin disease after early childhood and the development of respiratory allergies. In particular, we were interested to know how many patients with the first diagnosis of nonallergic AEDS were still

Table 1. Clinical and allergological data of children with AEDS at the first visit and at follow-up ($n = 22$)

	First visit	Follow-up
Age (years)	2–4	10–12
AEDS	100% (22/22)	68% (15/22)
Nonallergic AEDS	32% (7/22)	18% (4/22)
Asthma bronchiale (AB)	9% (2/22)	45% (10/22)
Allergic rhinitis (AR)	0% (0/22)	41% (9/22)
Positive SPT*	54% (12/22)	77% (17/22)
Elevated IgE levels†	41% (9/22)	81% (18/22)
Positive Sx1 (CAP FEIA)‡	50% (11/22)	80% (18/22)
Positive Fx5 (CAP FEIA)§	41% (9/22)	27% (6/22)

*. *D. pteronyssinus* and *D. farinae*, cat, dog and horse epithelia, grass, rye, birch, alder, ash, hazel and mugwort pollen, Alternaria, Aspergillus, Cladosporium, Penicillium moulds (ALK, Hørsholm, Denmark); hen's egg and cow's milk (Stallergènes, Fresnes, France).

†. According to age values (7).

‡. *D. pteronyssinus*, cat, and dog epithelia, timothy, rye, birch and mugwort pollen, Cladosporium (Pharmacia, Uppsala, Sweden).

§. Egg white, cow's milk, fish (cod), wheat, peanut, soy (Pharmacia, Uppsala, Sweden).

SPT and IgE negative. The clinical and allergological data of these patients are shown in Table 1.

At re-evaluation eight years later 68% of the children still suffered from AEDS, 45% from AB and 41% from AR. The percentage of sensitization against common inhalant allergens (Sx1) increased from 50% to 80% and against food (Fx5) decreased from 41% to 27%. Four of the seven children with nonallergic AEDS were still without any detectable sensitization, but three evolved into an IgE-associated type. One of them developed asthma; however, the risk of developing asthma is much higher in IgE-associated AEDS (9/15; 60%) than in nonallergic AEDS (1/7; 14%).

Our long-term observations in a small group of young children with AEDS confirm the recent large study of Novembre et al. (6) on 111 children with AD who re-evaluated the patients about nine years later. A group of children with initially a nonallergic type of AD can develop into an IgE-associated type that underlines the necessity of repeated allergological investigations over the time. However, in adults there is still a percentage of patients with AEDS without specific IgE sensitization (1,5). The classification into an allergic, IgE-associated and a nonallergic AEDS at

each age phase (infancy, childhood, teenage, and adult phase) is essential for the allergological management of the patients (allergen avoidance, secondary allergy prevention). For the long-term prognosis it's therefore important to know that the risk of the development of an IgE-mediated respiratory disease is much lower in the nonallergic AEDS.

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References

1. WÜTHRICH B. Clinical aspects, epidemiology and prognosis of atopic dermatitis. *Ann Allergy Asthma Immunol* 1999;**83**:464–470.
2. KISSLING S, WÜTHRICH B. Verlauf der atopischen Dermatitis nach dem Kleinkindalter. *Hautarzt* 1993;**44**:569–573.

3. JOHANSSON SGO, O'B HOURIHANE J, BOUSQUET J, BRUIJZEEL-KOOMEN C, DREBORG S, HAAHTELA T, KOWALSKI ML, MYGIND N, RING J, VAN CAUWENBERGE P, VON HAGE-HAMSTEN M, WÜTHRICH B. Position paper. A revised nomenclature for allergy. An EAACI position statement from the EAACI nomenclature task force. *Allergy* 2001;**56**:813–824.
4. WOLLENBERG A, BIEBER T. Atopic dermatitis: from the genes to skin lesions. *Allergy* 2000;**55**:205–213.
5. SCHMID-GRENDELMEIER P, SIMON D, SIMON H-U, AKDIS CA, WÜTHRICH B. Epidemiology, clinical features, and immunology of the 'intrinsic' (non-IgE-mediated) type of atopic dermatitis (constitutional dermatitis). *Allergy* 2001;**56**:841–849.
6. NOVEMBRE E, CIANFERONI A, LOMBARDI E, BERNARDINI R, PUCCI N, VIERUCCI A. Natural history of 'intrinsic' atopic dermatitis. *Allergy* 2001;**56**:453.
7. WÜTHRICH B, BENZ A, SKVARIL F. IgE and IgG4 levels in children with atopic dermatitis. *Dermatologica* 1983;**166**:229–235.

Allergic contact eczema/dermatitis from cosmetics

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Key words: allergic contact eczema/dermatitis; contact eczema/dermatitis; cosmetics; irritant/toxic contact eczema/dermatitis.

● WE CARRIED out a descriptive study of 351 consecutive patients pertaining to the year 2000. Our incidence area is the northwest of Spain. These subjects were tested with the standard battery of GEIDC tests and with specific series of cosmetics and fragrances. Tests were read according to ICDRG.

A diagnostic suspicion of Allergic Contact Eczema/Dermatitis (ACED) (1) from cosmetic usage existed in 53 (15%) (45 female). The average age was 40.6 years (14–70 years) and personal or family atopy was not significant. The main occupational categories were housewives (32.5%), office workers (16%) and agricultural workers (8.3%).

Of the 53 patients, 34 (64.1%) were found to be positive for one or more