

Method: Data on treatment-emergent AEs were pooled from six randomized, double-blind studies, in which patients with symptomatic allergic rhinitis received DL 2.5–20 mg/day or placebo (PBO) for 2–12 week. Participants had to be free of other clinically significant disease that would interfere with study evaluations. In the present analysis, data were pooled for all participants aged ≥ 55 years. AEs assessed in this analysis included: (i) those reported in the package insert as occurring more frequently in DL-treated than PBO-treated subjects (COM-AE), (ii) those that may be related to anticholinergic activity (ACh-AE) and (iii) those associated with drug hypersensitivity (HS-AE). AE rates are summarized descriptively; no statistical analysis was conducted.

Result: The pooled population was 150 subjects (mean age 61.3 year) for DL and 100 (mean age 61.3 year) for PBO. Total incidence of AEs was 43% for DL and 42% for PBO. Several of the COM-AEs occurred as or less frequently with DL than PBO (Table 1); fatigue was more common in DL-treated subjects (DL 7% *versus* PBO 1%). Gastrointestinal and mucosal ACh-AEs were infrequent in DL-treated and PBO-treated subjects (Table 2). HS-AEs were generally less frequent in DL-treated *versus* PBO-treated subjects (Table 3). No serious AEs were reported for DL or PBO.

Conclusion: This analysis adds to the limited published data on the safety and tolerability of second-generation antihistamines in older adults with AR and demonstrates that DL was well tolerated in this patient population. The pattern and frequency of AEs, including those that have been associated with DL or with the antihistamine class, were similar between patients treated with DL and those who received PBO.

Table 1. Incidence of most frequent COM-AEs. (for abstract 1823)

	DL (%)	PBO (%)
Fatigue	7	1
Dry mouth	4	6
Somnolence	2	2
Nasopharyngitis	1	3
Myalgia	1	1

Table 2. Incidence of gastrointestinal and mucosal ACh-AEs. (for abstract 1823)

	DL (%)	PBO (%)
Diarrhea	2	0
Vomiting	2	0
Constipation	1	1
Nausea	1	2
Dry nose	1	0
Dry throat	1	0

Table 3. Incidence of HS-AEs. (for abstract 1823)

	DL (%)	PBO (%)
Dyspnea	1	3
Pruritus	1	3
Wheezing	1	0
Asthma	0	1
Cough	0	1
Urticaria	0	3

1824

Positive patch tests to palladium are not mere cross-reactivities to nickel but relevant diagnostic findings

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Background: Palladium (Pd) is present in every automobile catalytic converter, computer, mobile phone, or LCD television. Palladium alloys are used in dentistry and orthopaedics. Following EU restrictions on nickel (Ni) use in jewellery, Pd has replaced Ni in 'white gold' alloys. This has led to increase in numbers of patients with contact allergy to Pd. Until recently, positive patch tests to Pd were mostly regarded as a mere cross-reactivity with Ni. The aim of the present study was to analyse clinical characteristics of patients with primary allergy to palladium.

Methods: In June 2010, the Polish Baseline Series was supplemented with propolis and palladium. Since then, I had the opportunity of testing 85 patients to this series (19 males, 65 females, aged 8–70, median 37 years) with suspicion of allergic contact dermatitis (ACD).

Results: In the analyzed group, 28 (32.9%) patients were Ni-positive and 17 (20.0%) Pd-positive; 15 (17.6%) patients were positive to Ni but negative or doubtful to Pd. Four (4.7%) patients reacted to Pd but not Ni. Two patients (2.4%) were found with reactions to Pd clearly stronger than to Ni, which suggests that their primary hypersensitivity was Pd allergy, while response to Ni – a cross reaction. All the six persons were women aged 18–70 years. In three of them, it seems apparent that testing to Pd provided information pivotal to their diagnostic process (Table 1): To most doctors, history given by Ms 'A' and would be a 'dead sure' indicator of nickel allergy – except the rather surprising fact that the patch test to nickel remained negative, along with chromium and cobalt – two other metals included into European Baseline Series. Instead, positive patch test to palladium (and gold) has solved the case (Pd-Au alloy is jewellery 'white gold'). In Ms 'B', all the evil would be ascribed to cobalt, if not tested with Pd. If Ms 'C', a cashier, was tested only to Ni, the positive reaction would seem a sound explanation of her case. However, patch test to Pd resulted in an extreme reaction, suggesting that Ni was a mere cross-reactivity rather than the main cause. The relevance of Pd patch tests in the three remaining patients remains unclear.

Conclusion: Over past years, palladium has become broadly present in our surroundings. Combined with its sensitizing properties, this hapten has acquired big importance for allergists and should be included into routine patch testing.

Table 1. For abstract 1824

Patient	Age	Pd	Ni	Other metals	Indications for patch tests	Metal-related symptoms	Exposure to metals
Ms 'A'	32 y.o.	(+)	(-)	Cr(-), Co(-), Au(+), Cu(+), Mn(+)	Intolerance of jewellery, planned dental implants	Eczema to jewellery, including gold and non-precious metals	Typical everyday exposure
Ms 'B'	18 y.o.	(++)	(-)	Co(++)	Intolerance of metals and detergents	Itching provoked by earrings	Typical everyday exposure
Ms 'C'	27 y.o.	(+++)	(++)	Cr(-), Co(-)	Intolerance of metals and cosmetics	Reactions to wrist watch, earrings, jeans buttons	Cashier
Ms 'D'	38 y.o.	(+)	(-)	Cr(-), Co(-), Au(+), Ag(-)	Intolerance of cosmetics, household detergents, and textile finishes	None reported	Typical everyday exposure
Ms 'E'	70 y.o.	(+)	(-)	Co(-), Au(+)	Intolerance of household detergents	None reported	Typical everyday exposure
Ms 'F'	46 y.o.	(++)	(+)	Cr(-), Co(IR), Hg(-)	Intolerance of cosmetics	None reported	Cashier

IR, irritant reaction.

1825

Severe eczema, failure to thrive and developmental delay in a highly atopic cohort of infants

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Background: Severe eczema in infancy may be associated with allergic enteropathy and subsequent failure to thrive. The clinical characteristics of these infants have not been extensively investigated, particularly with respect to head growth and development. We sought to characterise a cohort of 20 infants presenting to a tertiary paediatric allergy referral centre who had severe eczema, IgE sensitization to multiple foods in association with failure to thrive.

Methods: Infants presenting to the our department with severe eczema, multiple food allergies and failure to thrive were retrospectively assessed with respect to growth and developmental parameters, skin prick test reactivity and allergen-specific IgE.

Results: Twenty infants were identified with severe eczema, IgE sensitization to multiple foods and failure to thrive. At presentation, the majority of the infants were being breast fed and showed evidence of chronic intestinal symptoms, immune dysregulation and hypereosinophilia. Most required treatment with potent topical steroids and the introduction of an amino acid-based formula. Following appropriate treatment, all infants gained weight rapidly. In nine infants, failure to thrive was associated with a failure of head growth, and these infants tended to have the highest food-specific IgE levels [KU/l, geometric mean and standard deviation; 978 (261–3661), $n = 5$ versus 220 (56–862),

$n = 5$; $P = 0.12$]. They were significantly more likely to be skin prick test positive to wheat ($P = 0.011$) and peanut ($P = 0.0001$) compared to infants in which head growth was maintained. There was no difference between groups in mean age at presentation, family history of atopy, presence of gastrointestinal symptoms, serum globulin, white cell count or differential. Four of these infants displayed developmental delay manifesting itself mainly in failure to achieve motor milestones.

Conclusions: Infants with multiple food allergies and severe eczema are at risk of failure to thrive and poor head growth. Highly atopic infants are most at risk and head growth may be sufficiently severe as to affect motor development. Early recognition and appropriate treatment are required to optimise growth and development in these infants.

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Complement levels in patients with chronic idiopathic urticaria

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Background: A role of complement in pathogenesis of chronic idiopathic urticaria (CIU) is a still matter of debate. Some groups have found hypocomplementaemia associated with CIU, other have found no significant abnormalities in complement values. It was also observed presence of autoantibody in patients with CIU. Aim of this study was to determine serum complement levels in patients with CIU and its relation with presence of some autoantibody and clinical picture.

Methods: We include in the study 44 (eight male, 36 female) patients with CIU. Duration of CIU was 1–12 years. Complement examination (C3, C4, CH 50, C1q, C1 inhibitor) as well as autoantibodies [antinuclear antibody (ANA), anti-neutrophil cytoplasmic antibody (ANCA), anti-mitochondrial antibody (AMA), anti-smooth muscle antibody (ASMA), anti-thyroperoxidase antibody (antiTPO), anti-thyroglobulin antibody (ATGA)] was determined in all patients. We also monitor presence of these parameters in correlation with duration and severity of clinical picture as well as response to therapy.

Results: Low complement levels were found in 14/44 patients. Decreased concentrations of C3 was found in four patients, C4 in six, C1q in one, low functional activity of C1 inhibitor in four. All decreases in complement levels are slight. CH 50 was normal in all patients. Only in one patient C4 and C1 inhibitor functionality was slightly decreased. In our group of patients 18/44 patients had autoantibodies: ANA in 3, AMA in 2, ASMA in 2, all in low titers. Anti TPO was detected in seven and ATGA in five patients, with variability of titers. No patient had positivity of ANCA. One patient had ANA and AMA positivity. Only two patients had decreased C4 levels and ATGA antibodies, and one low C3 level and ANA. No patient developed systemic disease during follow up period (minimum 1 year).

Conclusion: Although presence of low complement levels and autoantibodies are more frequent in patients with CIU it is not clear connection with presence of autoantibodies, contribution in clinical picture of CIU or difference in response to therapy.