

angioedema to asthma (1). The most distinctive features of aspirin sensitivity are (1) nasal polyposis, (2) sinusitis, and (3) bronchial asthma, more commonly known as the aspirin triad (2). The prevalence of aspirin sensitivity in patients with asthma varies with the population studied and the methods used to detect the sensitivity. Aspirin can induce bronchoconstriction in 4–19% of adult asthmatic patients (3). Of asthmatic patients with pansinusitis and nasal polyps, 30–40% have aspirin-sensitive intolerance and asthma (4).

Inhalation challenge with lysine aspirin is a reliable, sensitive, specific, reproducible, and safe method for detection of aspirin-sensitive asthma (5, 6). We report the case of a patient with anaphylactoid reaction to lysine aspirin during the bronchial challenge test.

A 56-year-old man was referred to our department for shortness of breath, which started 3 h after oral ingestion of Brufen® (Ibuprofen) in over-the-counter medication. The patient had suffered from similar reactions previously one or two times in relation to headache due to drug over 3 years. He was treated in a local clinic with diphenhydramine and methylprednisolone with eventual resolution of symptoms.

He had no history of allergic diseases. On admission, laboratory data showed his serum IgE level to be 89.2 U/ml (normal range 0–100 U/ml). Blood count was normal at 50 eosinophils/mm³. Skin prick tests were performed with 55 common allergen extracts (Allergopharma Co, Germany). The positive and negative tests were histamine and saline solution, respectively. The patient had no positive reactions to allergens. Baseline spirometry showed a FEV₁ of 2.27 l (75% of predicted value) with an FEV₁/FVC ratio of 77%. Methacholine PC₂₀ was 0.36 mg/ml. Paranasal sinus radiologic study was negative.

The lysine aspirin (L-ASA) bronchial challenge test was performed by a method modified from those described previously (5, 6). FEV₁ was measured before and during aspirin challenge. L-ASA in a solution containing 900 mg of lysine acetylsalicylate with 100 mg of glycine (Synthelabopharma, Lausanne, Switzerland) was made up fresh on challenge day. All medication including theophylline, bronchodilators, and steroids was stopped 24 h before the procedure. The test solutions were delivered by a De Vilbiss 646 nebulizer (De Vilbiss Co, Somerset, PA, USA) and connected to a compressed air source (5 l/min). Normal saline was inhaled as a placebo solution. If there was less than a 10% fluctuation of FEV₁ with placebo inhalation, the L-ASA bronchial provocation test from 56.0 up to 450 mg/ml was performed. The patient was asked to breathe the nebulized aspirin aerosol to tidal volume for 2 min.

After starting the challenge test, the patient had facial flush, and presented generalized pruritus. During inhalation of 450 mg/ml of L-ASA, he complained of shortness of breath, cold sweating, and wheezing. His blood pressure dropped from 130/80 mmHg to 70/50 mmHg, and he was drowsy. His FEV₁ fell from 2.27 to 0.56 l (24.6% of actual value). He was rapidly administered epinephrine, oxygen, a short-acting bronchodilator by inhaler, methylprednisolone, and volume expander, and he recovered after 4 h.

This case is consistent with asphyxia and hypotonia following severe bronchoconstriction induced by inhaled aspirin in a sensitive patient. Our patient was advised to avoid administration of aspirin.

In summary, this is, to our knowledge, the first case of severe reaction induced by inhaled L-ASA. Such reactions have been thought to be caused by a non-IgE-mediated hypersensitivity reaction. Therefore, we should observe patients carefully during the L-ASA challenge test.

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Accepted for publication 19 June 2000
Allergy 2000; 55:1092–1093
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ISSN 0105-4538

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Washing feather and synthetic pillows

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Key words: allergen avoidance; Der p 1; Der p 2; feathers; pillows; synthetic; washing method.

• BED mattresses are known to be the main reservoir for mite allergens in dwellings (1).

Recent studies have demonstrated that both synthetic and feather-filled pillows contain mite allergens (Der p 1 and Der p 2), but the allergens appear in a significantly higher degree in synthetic pillows (2, 3). The aim of the present study was to evaluate the effectiveness of a commercially available washing method to reduce the mite-allergen content of pillows.

The fillings of 26 pillows were inspected for this study: of these, 17 were feather filled (1–15 years old) and nine were synthetic filled (1–5 years old). Before and after washing, the pillows were vacuumed with a dust trap for 2 min on both sides. Mites were counted with a microscope. After the fine dust was extracted, Der p 1 and Der p 2 were analyzed by sandwich ELISA (4), and the total protein content was determined.

The washing procedure was as follows:

- 1) prewash
- 10-min washing at maximum 40°C and pH 6–7
- centrifugation step (2×3 min, 1000 rpm)
- 2) main wash
- 20-min washing at maximum 60°C and pH 7.4–7.8
- centrifugation step (3 min)
- rinsing step – 3×5 min at 40°C (pH level fell from 6.5 to 4.5–5.0)
- centrifugation step (3 min)
- rinsing step – 5 min at 50°C and pH 4.5
- centrifugation step (7 min)
- 3) drying.

The drying was done under constant

moving at a maximum of 65°C until the moistness reached 2% less than the ambient air.

Only one dead mite was found in one of the 17 feather-filled pillows before the washing procedure; all the other samples contained neither living nor dead mites before and after washing. Table 1 presents the allergen and protein content (median; 25/75th percentile) per gram fine dust. Feather pillows contained a higher allergen (Der p 1) content than synthetic pillows, but the difference was not significant. For Der p 1, a reduction of 98.8% in feather and 80.3% in synthetic pillows was achieved; for Der p 2, there was a reduction below the determination limit of 0.1 µg/g dust (100%). The overall protein content was reduced by 66.3% for feather and 82.1% for synthetic pillows. Because of the wide variation, all results shown in Table 1 are given as calculated median, so that some of them are below the detection limit of the method. For better understanding, the range of variation is also shown.

There was a significant correlation (Spearman) between Der p 1 and Der p 2 content before the washing process ($r=0.684$; $P=0.00$). Therefore, determination of only one allergen, usually Der p 1, seems to be sufficient to characterize the mite-allergen content in pillows. There was no correlation between the total protein content and the amount of specific allergens. Although the proportion of proteins was higher in the synthetic material, the difference between the two

types of pillow was not significant.

Regarding the lack of mite detection on the pillow surfaces, one can speculate that the mite allergens in the samples were of mattress origin.

The allergen content of the pillows tested varied widely, independently of the filling material. This indicates that a subject's exposure to mite allergens depends on individual conditions. The washing procedure used in our study was effective in reducing both allergens and proteins independently of the type of filling material. Although the allergen concentration in the pillows tested in the present study was low compared with other studies (2, 3), because the washing procedure led to a major reduction in proteins, we assume that also a larger quantity of allergens can be removed by this method.

In summary, the described washing procedure is an effective allergen-avoidance measure for both feather and synthetic pillows, and it allows mite-allergic patients to choose their preferred pillow material.

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Accepted for publication 16 June 2000
Allergy 2000; 55:1093–1095
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ISSN 0105-4538

Table 1. Allergen and all protein content in pillows before and after washing

	Feather pillows (n=17)			Synthetic pillows (n=9)		
	Before washing	After washing	Reduction (%)	Before	After	Reduction (%)
Der p 1 (ng/g)	295.4	3.6	98.8	230.7	45.5	80.3
Range	(66.5–8854)	(0–166.6)		(12.4–5184)	(0–152.1)	
Der p 2 (ng/g)	27.5	0	100	25	0	100
Range	(0–1772)	(0–45.2)		(0–1540.8)	(0–2.2)	
Protein content (mg/g)	15.9	5.4	66.3	21.9	3.9	82.1
Range	(8.7–33.6)	(2.9–16.2)		(7.7–48.5)	(5.4–11.7)	

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Delayed-type allergy to systemic corticosteroids

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Key words: allergy; corticosteroids; cross-reactivity; dermatitis; dexamethasone; drug allergy.

• WITH reference to the recently published paper on anaphylactoid symptoms due to paramethasone (1), we would like to draw attention to another type of steroid-intolerance reaction, which has been very rarely reported.

A 59-year-old woman, with no past medical history of allergies or intolerances, and suffering from encephalomyelitis disseminata, was put on oral dexamethasone during an acute phase of her disease. On the third day after starting

Positive patch tests in a patient treated for encephalomyelitis.

this medication, she developed a macular rash with an exfoliate component involving the face, the upper chest, and intertriginous areas. Dexamethasone treatment was immediately stopped; subsequently, the skin disorders resolved over several days. The patient had never received corticosteroids before. An allergic reaction to the administered corticosteroid was suspected as the cause of the rash, and allergologic investigations were initiated.

Patch testing revealed a positive crescendo reaction to dexamethasone, betamethasone, and clobetasol-17-propionate. Other tested corticosteroids, including prednisolone, hydrocortisone-17-butyrate, methylprednisolone, and triamcinolone-acetonide, were negative. Prick tests using the same set of substances were completely negative. Finally, we tested oral exposure with methylprednisolone to confirm tolerance to this alternative treatment for future exacerbations of encephalomyelitis disseminata, and the patient tolerated this drug without complications.

Allergic reactions, particularly delayed reactions to applied systemic corticosteroids, are rarely reported in the literature (2). In this context, two main types of systemic allergic reactions have been described: an anaphylactoid type, with symptoms such as angioedema, bronchospasm, shock, or urticaria, occurring minutes or hours after systemic exposure; and a so-called delayed type of systemic reaction, which is described as generalized macular or maculopapular exanthematous dermatitis, characteristically appearing several days after the start of systemic corticosteroid medication (3, 4).

In our case, the clinical presentation already suggested a delayed systemic reaction, as skin changes occurred 2 days after oral dexamethasone treatment was started, and were not accompanied by anaphylactoid symptoms. Moreover, the

typical exanthematous rash with an exfoliate component, which our patient developed, fitted into this picture. Finally, we could confirm the diagnosis by a positive result in the patch test for dexamethasone.

Interestingly, the patch test was also positive for betamethasone and clobetasol-17-propionate, suggesting a cross-reaction. According to Coopman et al., cross-reactions occur primarily (6–7 times more often) within certain groups of corticosteroids (5). Four different groups of structurally related substances are distinguished:

- 1) group A comprising hydrocortisone-related steroids such as methylprednisolone
- 2) group B comprising triamcinolone acetonide and related compounds
- 3) group C comprising betamethasone-like substances including dexamethasone
- 4) group D comprising steroid esters such as clobetasol-17-propionate.

Our patient showed a cross-reaction between substances of group C and group D. This interesting phenomenon (especially as both groups are considered rarely to cause delayed-type reactions [6]) can be explained by the similar molecular structure of these steroids (7, 8). The fact that dexamethasone, betamethasone, and clobetasol-17-propionate all have a methyl-group at the C16 position in the D-ring seems to be associated with the reaction (9). This explains why, in our patient, methylprednisolone, a member of group A, which is not substituted on C16, was tolerated well upon oral exposure.

In general, corticosteroid allergy, especially to group-C corticosteroids, is difficult to establish, as skin reactions in allergologic testing may be suppressed by the anti-inflammatory and immunosuppressive properties of the tested substance (8). Therefore, it is important to keep the possibility, and the whole spectrum, of corticosteroid allergy in mind.