

mucosa. Supplementary medical treatment is always necessary to prevent recurrence (1). Treatment with oral corticosteroids is sometimes referred to as "medical polypectomy" because steroids have good repressive effects on nasal polyps (2), but there is also a strong tendency to recurrence within 5 months after successful oral therapy (3). However, oral corticosteroids should be used only for short-term treatment due to the risk of systemic side-effects. Intranasal corticosteroids are reserved for the treatment of mild cases (e.g., nasal polyposis). In the past 2 years leukotrienes have been implicated in the mediation of bronchoconstriction and inflammatory changes in asthma. The leukotriene-receptor antagonist montelukast has been shown to produce subjective and objective improvement in patients with mild to moderate asthma (4). Leukotriene levels have also been shown to be elevated in patients with sinonasal polyposis and sinusitis. Recently, an objective alleviation, or at least stabilization, of sinonasal polyposis was reported in 50% of the patients after application of the leukotriene-synthesis inhibitor zileuton or the leukotriene-receptor antagonist zafirlukast (5).

On the basis of these findings, we evaluated the efficacy of short-term oral corticosteroid therapy combined with montelukast in a daily dosage of 10 mg as maintenance therapy in controlling symptoms of severe sinonasal polyposis. Outcome measures included subjective interviews, serial endoscopic examinations, and MRI. We treated nine patients with massive bilateral sinonasal polyposis. All but one patient had repeated surgical polypectomy in the past. Recurrence was observed after 3 weeks ( $n=1$ ), 8 weeks ( $n=3$ ), 12 weeks ( $n=1$ ), and 1 year ( $n=3$ ) after surgery. Nasal blockage (100%) was found to be the major complaint before therapy. Nearly half of the patients initially

suffered from rhinorrhea. In the first 5 days of treatment, 40 mg methylprednisolone was given orally followed by a progressive reduction. Montelukast in a daily dosage of 10 mg was also administered. After day 12, the oral steroid therapy was discontinued. The follow-up period for the first treated patients is now up to 1 year. Meanwhile seven patients experienced subjective improvement of their symptomatology and four out of these seven were even free of symptoms. Anterior rhinoscopy was performed before treatment and then monthly. All patients showed endonasal polyps at the initial visit, five patients reached a polyp-free nasal cavity under treatment, and one patient showed clear regression of nasal polyps. Three patients presented with unchanged findings under anterior rhinoscopy. After 3 months regressive disease was documented by MRI in four patients, whereas in three patients stabilization of sinonasal polyposis was documented. Endoscopic nasal examination findings and MRI results were consistent with the reports of overall benefit ( $n=7$ ). Side-effects of this treatment concept were not observed. Two patients showing strong benefit of this therapy chose to discontinue treatment, and recurrence was observed 4–8 weeks later. All patients, even the two patients with only limited benefit of this therapeutic approach, preferred this therapy to surgical intervention.

We conclude that this new concept in the management of diffuse sinonasal polyposis might be a viable alternative to long-term oral steroid therapy and repeated surgical debridement. According to these preliminary data, oral application of montelukast should not be discontinued due to the risk of recurrence. None of our patients developed progressive disease; therefore, our data show that the leukotriene-receptor antagonist montelukast might play a significant role in the prevention of nasal polyps after surgery.

In view of these promising results, a clinical trial comparing our new therapy concept group to a control group receiving only topical steroids is in progress.

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### Severe reaction to lysine aspirin

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**Key words:** lysine aspirin; severe reaction.

- ADVERSE reactions to aspirin range from rhinoconjunctivitis, urticaria, and

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/mm<sup>3</sup>. 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 FEV<sub>1</sub> of 2.27 l (75% of predicted value) with an FEV<sub>1</sub>/FVC ratio of 77%. Methacholine PC<sub>20</sub> 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). FEV<sub>1</sub> 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 FEV<sub>1</sub> 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 FEV<sub>1</sub> 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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## 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).