

October 2002 • Volume 110 • Number 4

Disappearance of aspirin intolerance with antiasthma treatment

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To the Editor:

The latest international guideline for asthma management, the Global Strategy for Asthma Management and Prevention,¹ states that once nonsteroidal anti-inflammatory drug intolerance develops, it is present for life. This might be misleading because I cared for a patient with aspirin-induced asthma (AIA) whose aspirin intolerance disappeared with antiasthma treatment.

A 14-year-old girl had dyspnea and cough in November 1995. She was treated with antiasthma medications only for a short time. In April 1996, she experienced severe dyspnea about 2 hours after an ingestion and about 4 hours after another ingestion 3 days later of over-the-counter cold drugs, including 400 mg of ibuprofen tablets. In the first episode her symptoms subsided soon after treatment, but in the second episode she experienced ventilatory failure (Paco₂ of 49.4 mm Hg) and continued to experience dyspnea and wheezing in spite of treatment for 17 days. She was referred to Chonnam National University Hospital.

Physical examination did not reveal nasal polyps, but sinus radiographs showed thickening of both maxillary antral walls. Peripheral blood eosinophil counts and serum total IgE levels

were within normal limits, and allergy skin prick test responses were negative.

FEV₁ was 2.76 L (77% of predicted value), and the provocative concentration of methacholine causing a 20% fall in FEV₁ (PC₂₀) was 0.31 mg/mL. One month later, the patient ingested 25 mg of aspirin at 9 AM and 50 mg of aspirin at 11 AM, and FEV₁ was 2.73, 2.88, 2.42, and 2.13 L (21.9% fall from the baseline value) at 9, 10, and 11 AM and 12 noon, respectively. At noon, she began to complain of dyspnea and wheezing.

Four hundred micrograms of budesonide, 50 µg of salmeterol, 4 mg of nedocromil, 200 mg of slow-releasing theophylline, and 1 mg of ketotifen twice daily, respectively, were prescribed for 2 weeks. The self-monitored peak expiratory flow values during the 2 weeks were approximately 200 to 470 L/min. Her asthma was controlled soon, and the asthma control was relatively well maintained, even with reduced medications. In July 1997, peak expiratory flow was approximately 410 to 430 L/min with 200 µg of budesonide twice daily and 25 µg of salmeterol twice daily.

Withholding salmeterol 7 days before challenge, methacholine PC₂₀ was still 0.31 mg/mL, but lysine-aspirin inhalation test results² were negative. Aspirin oral challenges were not conducted, and therefore it is possible that the patient would have reacted to oral challenges with large doses of aspirin. One year later, her asthma controller was reduced to 200 µg of budesonide twice daily only, and the next year, she took no medications at all. In January 2001, the patient's methacholine PC₂₀ was 1.99 mg/mL, and results of lysine-aspirin inhalations and subsequent oral ingestions of 250 and 500 mg of aspirin at 2-hour intervals were negative. No naso-ocular symptoms developed after the aspirin challenges.

Generally, AIA begins with rhinitis that is followed by asthma and then aspirin intolerance. About half of the patients are oral steroid dependent.³ The airways of patients with AIA show marked eosinophilic inflammation before treatment. Because the classic aspirin intolerance develops after asthma is established and severe, it can be speculated that the aspirin intolerance might be improved as asthma is controlled.

Corticosteroids, cromolyns, leukotriene modifiers, aspirin desensitization, and surgical treatment of the accompanying upper airway inflammatory disease are potentially effective treatments for asthma in aspirin-sensitive subjects. However, few investigators have reported a cure of aspirin intolerance. Jankowski et al⁴ reported 4 cases of AIA in which the aspirin reactivity threshold was increased after a radical ethmoidectomy.

AIA is a chronic disease that runs a protracted course. Szczeklik and Stevenson⁵ suggested that persistent airway inflammation in AIA could result from autoimmunity, latent viral infection, or both. However, this young woman showed atypical clinical features, including cure of aspirin insensitivity. This case demonstrates the potential variability of aspirin sensitivity and asthma.

1/8/128014

doi:10.1067/mai.2002.128014

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ABSTRACT

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