

Correspondence

Systemic and local responses in aspirin-exacerbated respiratory disease: Same or different disease?

To the Editor:

In their interesting study, Makowska et al¹ demonstrated that specific bronchial challenge with aspirin can induce systemic symptoms and bone marrow activation of leukocytes and eosinophil progenitor cells.

It is not an easy task to explain how an organ-specific (ie, bronchial) provocation can elicit nasal and skin responses. Aspirin dosage seems to play a key role because those patients affected with the “most severe” degree of aspirin-exacerbated respiratory disease (AERD) had a positive bronchial test response with a median of 6.8 mg of aspirin, with no extrapulmonary symptoms recorded, whereas “less-sensitive” individuals reaching a 10-fold aspirin cumulative dose showed both bronchial and systemic responses.

The complementary nasal aspirin challenge provides a good model to evaluate dosage and safety on this issue. We have studied nasal responses to aspirin challenge over the last 3 years, with a total of 55 nasal provocation tests (NPTs) completed.^{2,3} Twenty-six of 30 patients with AERD had a positive (rhinomanometric and clinical criteria) test result, whereas the 25 aspirin-tolerant asthmatic subjects (control group) had a negative NPT result. Most of the nasal responses were ipsilateral (13/26) or bilateral (12/26), whereas 1 patient had a contralateral response. Twenty-two of the 26 patients with aspirin-induced asthma were given diagnoses of a maximal cumulative dose of 14 mg of aspirin, and 4 of 26 subjects had a positive NPT result with only 7 mg. Only patients with AERD had positive clinical scores showing exclusively nasal symptoms (rhinorrhea, congestion, sneezing, and/or pruritus). Nine of the 26 patients with positive NPT results required medical treatment, and oral antihistamines were the most frequent medication (9/10 prescribed drugs), followed by topical corticosteroids (only 1/10 prescribed drugs). No inhaled β -agonists, systemic corticosteroids, or adrenaline were needed at any time, and hospital admissions were not required.

Because 6.8 mg of aspirin applied to the bronchi and 14 mg applied to the nose are far less than the 181.98 mg described by

Makowska et al,¹ it would be desirable to identify a threshold of aspirin exposure to produce accurate and safe organ-specific diagnoses in AERD to avoid the risk of systemic responses. The authors also point out that late bronchial responses can be linked to eosinophil progenitor recruitment in those patients with extra-bronchial symptoms. In our previous experience with nasal aspirin challenge, 19 (73%) of the 26 positive NPT results were diagnosed within 50 minutes (48.07 ± 27.71 minutes) after the lysine-aspirin application, 7 of 26 had positive results occurred within 2 hours, and no late responses were recorded. Again, one might speculate on the aspirin exposure dosage and the effect on bone marrow progenitor mobilization.

The Makowska et al¹ study provides an objective method to assess the systemic reactions after lysine-aspirin bronchial challenge in patients with AERD. More studies are needed to clarify whether the systemic and local responses to aspirin in these patients are caused by specific and different pathophysiologic mechanisms or if this is a dose-dependent phenomenon.

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