

mined. Cross-reactivity between pine nuts from different trees such as *Pinus edulis* (the antigen used for specific IgE determination) and *P. cembra* (nuts found in bird food used by bird fanciers) has been reported (6, 7). But there has been no mention of cross-reactivity between pine nuts and other nut species (7), pine pollen, or pine resin (2, 4). Patient 2 presented generalized urticaria when eating Brazil nut and had positive prick tests for Brazil nut, chestnut, and walnut. Patients 1 and 3 were asymptomatic and tested negative for other nut species. The allergenicity of pine pollen is controversial. Some authors classify pine pollen as nonallergenic because of its large size, while others believe it could cause allergy with sufficient exposure (1, 4). Since these three patients were not sensitive to pine pollen, it is unlikely that they became sensitized by prior contact with conifers. It is also unlikely that patients 1 and 3 became sensitized to pine nuts by prior ingestion of other nut species. Pine nuts are often hidden in food. Thus, patients allergic to pine nuts should be aware of their potential presence in pesto sauce, salad sauce, pastries, desserts, and pasta. Pine nuts should be classified as a food causing acute life-threatening anaphylaxis. Allergy to it should be considered when investigating anaphylaxis of apparently unknown origin. Strict avoidance and the carrying of an emergency kit with epinephrine (injectable and aerosol) are presently the only recommendations.

Key words: anaphylaxis; food; pine nuts; pine seed.

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Detection of aspirin reactivity in patients with pyrazolones-induced skin disorders

PYRAZOLONES, one of the most widely prescribed drug classes throughout the world, are frequently involved in severe, unpredictable adverse reactions. From an allergologic point of view, three types of pyrazolones-induced adverse reactions can be distinguished:

- 1) allergic reactions clinically characterized by anaphylactic shock and/or urticaria, and by tolerance of therapeutic doses of different nonsteroidal anti-inflammatory drugs (NSAID) (1, 2)
- 2) pseudoallergic reactions, which can present as asthmatic attacks or as acute urticaria/angioedema (U/A), small amounts of chemically unrelated NSAID inducing acute asthma or U/A, respectively, in these patients, probably via cyclooxygenase inhibition (1-3)
- 3) pathogenetically unclear skin and/or mucosal reactions, as in Stevens-Johnson syndrome (SJS) or toxic epidermal necrolysis, which can be induced by both pyrazolones and NSAID.

Administration of full-dose aspirin (ASA) or other NSAID in patients who experienced pyrazolones-induced skin reactions might be hazardous. This study assessed the prevalence of ASA intolerance among patients who experienced pyrazolones-induced skin disorders.

The study was performed on 91 subjects (M/F 25/66, mean age 37.5 ± 15.6 years, range 10-81 years) seen at this allergy center between March 1992 and December 1996 for skin reactions unequivocally caused by the ingestion of one or more of the following pyrazolones: noramidopyrine, propiphenazone, or feprazone. Some 78, 12, and two patients experienced U/A, SJS, and anaphylactoid reactions, respectively. One patient experienced both U/A and SJS.

On the basis of clinical history, two groups of patients were distinguished:

- 1) Forty-six patients with clinical history of pyrazolones intolerance only (U/A, SJ, and anaphylaxis in 34, 11, and one case, respectively) who underwent peroral challenges with AS.
- 2) Forty-five patients with clinical history of adverse reactions caused by both pyrazolones and other NSAID (ASA in most cases), 44 cases of U/A (one associated with SJS), and one case of anaphylaxis were recorded. Every possible effort

was made to establish which drug class, whether pyrazolones or NSAID, caused the first intolerance episode in these subjects. Patients from this group were used as controls.

ASA was challenged in a single-blind, placebo-controlled fashion (4); the following doses were given 1 h apart: day 1: 50 mg, 125 mg; day 2: 250 mg, 500 mg.

Placebo was given first in each session. Patients were controlled for at least 1 h after the last provocative dose and were newly seen on day 3 in order to detect possible late reactions. Patients gave informed written consent before each challenge session. All subjects who tolerated ASA were strongly recommended to contact us as soon as possible in the case of subsequent NSAID-induced adverse reactions.

Some 3/46 (6%) patients had U/A reactions after ASA administration; all reactions took place on day 1 after the 50- or the 125-mg dose, and were easily controlled by conventional therapy. All positive patients had a clinical history of U/A caused by pyrazolones administration (3/34, 9%); by contrast, ASA was tolerated in all patients with a history of SJS or anaphylaxis reactions ($n=12$).

In the control population, the first adverse reaction was induced by NSAID other than pyrazolones in 34 (87%) cases and by pyrazolones in five (13%) cases. In the remaining six cases, it was not possible to ascertain the causative agent of the first adverse reaction. To date, no patient who tolerated ASA on placebo-controlled challenge has contacted us to report NSAID intolerance.

The present study confirms that most patients with clinical history of adverse skin reactions induced by pyrazolones can take ASA and other NSAID safely (1). However, both the prospective and the retrospective parts of this study show that in about 10% of these patients, particularly those who experienced U/A reactions, pyrazolone intolerance reactions represent the first episode of more general NSAID intolerance. A simple SBPCDC with ASA may prevent potentially severe adverse reactions in these cases; such a test should be recommended in all patients with a history of pyrazolones-induced urticaria.

About 10% of patients with skin reactions to pyrazolones develop general NSAID hypersensitivity.

Key words: adverse reactions; aspirin; NSAID; pyrazolones.

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Pollen exposure and sensitization

SEVERAL pieces of evidence from the literature support the hypothesis that sensitization to aeroallergens depends upon allergen exposure. Following the same track, we compared skin reactivity and prevalence of allergic symptoms in 933 school-children living at high altitude or at sea level in two medium-sized towns. The same team of field-workers performed skin tests at both locations. As expected, due to low mite-allergen exposure, the percentage of children sensitized to mites was much lower in those living at high altitude, especially in children born there (1). Surprisingly, the percentage of children sensitized to grass and mugwort pollens was much higher at high altitude than at sea level. The percentage of skin sensitivity to grass pollen was equal to 21.7% in children born and living at high altitude, whereas it was equal to only 8.5% in children living at sea level. Figures for mugwort sensitivity were 7.6% and 1.0%, respectively. We wondered whether the higher prevalence of sensitization to pollen at high altitude was related to a higher exposure to pollens.

■ Thus, we compared pollen counts in both settings. Pollen grains were trapped with a Burkart apparatus located either in Briançon (French Alps, altitude: 1350 m) or Martigues (near Marseille, southeastern

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