

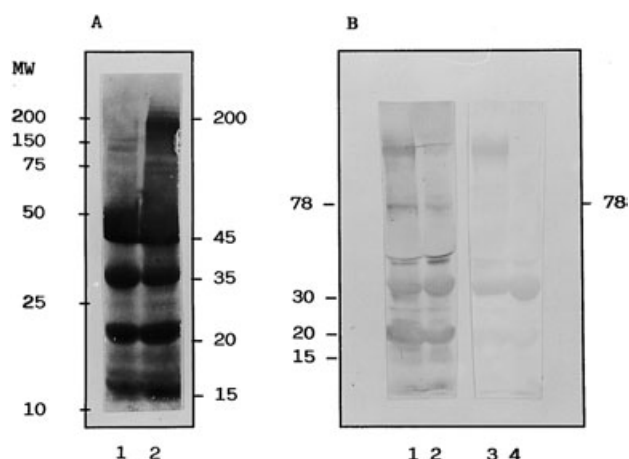


Figure 1. A) Coconut SDS-PAGE, without (lane 1) or with (lane 2) β_2 -mercaptoethanol. B) Coconut IgE immunoblotting: lanes 1 and 3 reduced, lanes 2 and 4 unreduced; lanes 1 and 2 patient serum, lanes 3 and 4 control.

Allergy to walnut was ruled out by prick test and CAP. An open oral challenge with walnut was negative. Skin prick test and CAP to latex were both negative.

In conclusion, we present an exceptional case of anaphylaxis induced by the ingestion of a coconut ice-cream due to an IgE-mediated reaction to coconut, which was demonstrated by both *in vivo* skin tests and *in vitro* tests. In the two previously reported cases of combined allergy to nuts and coconut the main allergens involved had molecular weights of 35–36 kDa (1). A different allergen pattern was identified in our patient probably because he was monosensitized to coconut.

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Unusual NSAID hypersensitivity

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Key words: aspirin; conjunctivitis; NSAID intolerance.

• THE appearance of conjunctivitis as the sole manifestation of an adverse reaction to a drug administered by the oral route is an uncommon phenomenon (1). We report a case of conjunctivitis induced by non-steroidal anti-inflammatory drugs (NSAIDs). To the best of our knowledge, no similar selective ocular reactions have been previously reported. A non-atopic 53-year-old female reported several episodes of ocular pruritus, eye redness, and lacrimation, always affecting her left eye. All the episodes started in the first 2 h following the intake of NSAIDs, such as aspirin, metamizol, ibuprofen, and dextetoprofen, and resolved spontaneously after 2–6 h. She denied involvement of the right eye, as well as rhinitis, bronchial or skin symptoms. A complete ophthalmologic examination, including slit lamp biomicroscopy and conjunctival impression cytology (2) of both eyes, was normal in basal conditions.

Isolated conjunctivitis after oral intake.

The patient gave written informed consent to the reintroduction of NSAIDs in order to study her reported adverse reactions. All drugs were given following a single-blind placebo-controlled procedure. Two hours after the administration of aspirin 125 mg she presented pruritus, hyperemia of the conjunctiva and lacrimation of her left eye. An impression cytology taken at that moment revealed an important neutrophil infiltrate. No reaction was observed (ophthalmologic examination, impression cytology) of the right eye. The reaction subsided spontaneously in 4 h, and the infiltrate in the impression cytology also disappeared. Two weeks later the patient received diclofenac, an NSAID she had never taken before, and 2 h after the intake of 25 mg she presented a left eye conjunctivitis with a neutrophil infiltrate. Two hours after the application of fluorometolone 1 mg/ml eye-drops the inflammatory response of the left eye subsided and the infiltrate in the impression cytology disappeared. Skin, nasal or bronchial involvement were absent in these two positive challenges, and no significant changes of basal FEV_1 were observed. Diclofenac eye-drops (1 mg/ml) were applied to both eyes. Thirty minutes later conjunctival hyperemia and lacrimation of the left eye appeared, but no reaction was observed in the right eye. In contrast, paracetamol (up to 1000 mg) and nimesulide (up to 300 mg) were well tolerated.

In an attempt to prevent the NSAID-induced inflammatory response of the left eye, several pre-medication strategies were assayed. Disodium cromoglycate (40 mg/ml), sodium nedocromil (20 mg/ml), levocabastine (0.5 mg/ml) and fluorometolone (1 mg/ml) eye-drops were given separately at therapeutic doses for 2 days prior to the administration of aspirin. No protective effect was observed with any of them, although the eliciting dose of aspirin increased to 250 mg after the pre-medication with fluorometolone.

In conclusion, we present an exceptional case of isolated left eye conjunctivitis after the oral (and local) administration of NSAIDs, in which a local idiosyncratic reaction to inhibition of the cyclo-oxygenase (COX) pathway seems to be involved (3). The inflammatory response induced by NSAIDs was mild, transient, and self-

limited, but the patient was advised not to use them again because of the eventual damage which could be induced by repeated episodes of conjunctival inflammation. Perhaps the application of a more potent corticosteroid would have blocked the eye's reactivity, but it would not have been a good therapeutic option. The patient was advised to take paracetamol and nimesulide. New COX-2 inhibitors will probably be a good alternative for this patient.

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Nonallergic anaphylaxis to pantoprazole

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Keywords: Anaphylaxis, pantoprazole, proton pump inhibitors.

● PANTOPRAZOLE is a substituted benzimidazole which inhibits gastric $H^+/K^+ATPase$ of the parietal cells (1). Clinical adverse events were recorded in approximately 7.5% and were generally mild and transient (1,2).

Between December 1998 and July 1999 a 45 year old woman showed four episodes of flushing, Quincke's edema, dyspnea, low

blood pressure and somnolence followed by a loss of consciousness. Initially, the first two events were classified as China–restaurant syndrome because our patient had eaten Chinese food before showing any symptoms.

Additionally, the patient suffered from gastric ulcers, however, with only slight clinical

symptoms. Irregularly, in expectation of an opulent meal, she had taken pantoprazole.

In July 1999 she presented herself in our allergy clinic. A food allergy or food additive allergy was excluded by skin, blood and provocation tests. The test reactions in skin tests to pantoprazole (patch test, prick test [4 mg/ml]) were also negative. Following this initial test a double blind placebo-controlled oral challenge test was performed. Two hours after ingestion of 20 mg pantoprazole, she developed conjunctivitis, flush, dyspnea, and a hypertensive reaction. The challenge tests with the other ingredients of Pantozol® were negative.

Furthermore we performed a cellular antigen stimulation test (CAST), which is useful in the differentiation of allergic and nonallergic reactions to drugs (3). In this case, pantoprazole was used in a concentration of 1 mg/ml in the test with following results: background: 200.25 pg/ml, stimulation control: 4816.75 pg/ml, C5a alone: 246.5 pg/ml, pantoprazole: 888.8 pg/ml, C5a + pantoprazole: 1341.8 pg/ml. We observed a more positive reaction when the drug was applied together with complement component C5a, which is considered a nonallergic response (3).

Based on the patient history, the oral challenge test and *in vitro* test (CAST) we diagnosed a nonallergic anaphylactoid reaction to pantoprazole (4).

The frequency of skin rashes induced by proton pump inhibitors is less than 0.5% (5). Only a few cases of severe allergic or nonallergic anaphylaxis and erythrodermic reaction to omeprazole, lansoprazole or the coating substances were reported (5,6).

In addition Natsch et al. (2000) reported two cases of generalized urticaria anaphylaxis after ingestion of omeprazole and lansoprazole in one

patient and pantoprazole in another. But in this report no oral challenge test was performed (7).

Anaphylactic reactions caused by proton pump blockers are very rare, but can be life threatening. It is difficult to make the diagnosis from the personal history of the patient because related symptoms often occur several hours after ingestion due to the enteric coating of the capsules.

We propose to include reactions to proton pump blockers in the differential diagnosis of food related intolerance reactions.

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