

IgE-mediated anaphylaxis to antacid

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● A 22-year-old male driver presented a history of allergic rhinitis and atopic dermatitis. After a meal, he took one tablet of Gaviscon® (Ferring, Malmö, Sweden) antacid. Two hours later, his face became erythematous and his eyes red and wet. He started

to cough and his breathing became difficult. He went to an

IgE antibodies were found to the tablet and to seaweed.

emergency clinic, where he was diagnosed as having anaphylaxis and subsequently treated.

In prick tests, weak allergic reactions (3 mm) were seen to timothy, meadow fescue, dog, garlic, agar-agar (seaweed product), and molds (*Acremonium kiliense*, *Aspergillus versicolor*, *Cladosporium cladosporoides*, *Fusarium culmorum*, and *Phoma herbarum*). Prick-prick tests with the antacid tablet gave a 20-mm reaction, but alginic acid was negative. Skin tests repeated 3 months later with the same batch and two different batches obtained from the manufacturer gave 7–8-mm positive reactions. Other antacids, also all chewable tablets, gave allergic prick reactions: Novaluzid mint (3 mm; AstraZeneca), Rennie (3 mm; Roche), Link (16 mm; Alparma), Balancid Novum (3 mm; AstraZeneca), and Topalkan (18 mm; Pierre Fabre Medicament). None of the controls had positive reactions to the antacids tested.

The immunospot method was used to detect serum IgE antibodies to the Gaviscon antacid tablet (2). For this,

extracts were made from three batches of the preparation (see above), other antacids, and alginic acid, and 27 skin prick tests and other allergen preparations were done. The fruit flavors in the Gaviscon tablet were not available. A serum from a nonatopic subject and three serum pools from atopic patients were used as controls.

Distinct binding of IgE to all three batches of the Gaviscon preparation was demonstrated from the patient's serum. Binding was also detected to seaweed extract, but not to other materials tested. No distinct binding was demonstrated from the control sera. Only a faint binding to the tablet extract was demonstrated from the pooled serum from mold-allergic patients.

Gaviscon is a chewable antacid taken for gastritis and esophagitis. According to the manufacturer, one tablet contains alginic acid (USP, 350 mg), aluminum hydroxide dried gel (USP 100 mg), sodium hydrogen carbonate (Ph Eur, 120 mg), mannitol, saccharin sodium, polyvidone, talc, magnesium stearate, and the flavors lemon, raspberry, and vanilla.

Pulmonary hypersensitivity and precipitating antibodies to seaweed have been reported in the alginate industry (4). Alginic acid was negative in our testing. Other seaweed products commonly used are agar-agar and carrageenan, produced from different red seaweeds (Rhodophyceae). Indeed, skin tests and IgE tests were positive with agar-agar and seaweed extract. A possible contamination of alginic acid with seaweed ingredients cannot be excluded. Our patient's sera showed strong IgE binding to seaweed in immunospot, as well as to the Gaviscon tablet itself.

One possibility is that our patient was allergic to the artificial fruit flavorings

used in antacids. These, however, were not available for testing. Interestingly, our patient reacted to other antacid tablets in prick tests, too. However, confirmation of IgE-mediated mechanism by *in vitro* binding studies was achieved only with chewable Gaviscon, not with other antacids. The patient stated that he had been able to take the other antacids he had tried (Link and Novaluzid Mint) without any symptoms, even after his initial anaphylactic reaction. It is known that skin tests may be positive also via non-IgE-mediated mechanisms, and nonspecific histamine release cannot be excluded. After he stopped taking the Gaviscon antacid tablets, his skin prick test response fell from 20 mm to 7 mm, and the patient has been symptomless for over 20 months.

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