

CLINICALLY RELEVANT CROSS-REACTIVITY BETWEEN LATEX AND PASSION FRUIT

A 36-year-old woman reported that she had experienced generalized urticaria, oropharyngeal pruritus, tongue swelling, dysphagia, dysphonia, cough, rhinorrhea, sneezing, lacrimation, and ocular itching immediately after drinking a 330-mL can of mango and passion fruit juice. A year before the reaction, she had experienced an episode of oropharyngeal pruritus minutes after eating a chestnut. She reported local pruritus and erythema whenever she used latex gloves during the last 5 years. The patient eats avocado and banana with no adverse symptoms.

After obtaining informed consent, we conducted skin prick testing with passion fruit–mango juice, passion fruit, mango, and chestnut by the prick-prick method. Then, 9- and 22-mm wheals were observed in the patient after testing with passion fruit–mango juice and passion fruit, respectively; skin test results were negative with mango and chestnut. The skin prick test result was positive with a latex extract (wheal diameter, 26 mm). Specific IgE levels (UniCAP assay; Phadia, Uppsala, Sweden) to passion fruit, chestnut, latex, and rHev b 6.02 (hevein) were 2.17, 0.72, 10.50, and 14.30 kUA/L, respectively. No specific IgE was detected with mango extract or rHev b 1, 3, 5, 8, 9, and 11. By UniCAP-inhibition experiments, the patient's IgE antibody to passion fruit was completely inhibited by latex extract (0.1 mg/mL), but IgE binding to latex and rHev b 6.02, inhibited with passion fruit (1 mg/mL), yielded a partial inhibition (58% and 63%, respectively). Sodium dodecyl sulfate–polyacrylamide gel electrophoresis/immunoblotting studies of passion fruit extract showed IgE binding components of 16 to 54 kDa. A complete dose-dependent inhibition of passion fruit was observed after preincubation with latex extract (Fig 1).

A double-blind, placebo-controlled food challenge was performed with chestnut. Increasing doses of a liquid vehicle (custard, milk, coffee, sugar, and wheat germ) containing (verum) or not (placebo) ground chestnut were administered on separate days. A cumulative dose of 5.4 g of chestnut elicited a clinical response. The result of an open food provocation with fresh mango was negative. Symptoms induced by the offending juice precluded further challenges with passion fruit.

Cross-reacting IgE antibodies recognizing latex and passion fruit allergens were first demonstrated by radioallergosorbent inhibition tests.¹ In addition, putative class 1 chitinases have been reported to be relevant cross-reactive components in foods associated with the latex-fruit syndrome.² Our investigation confirms the cross-reactivity between passion fruit and latex in a patient with IgE (to hevein) from latex and its clinical relevance. Therefore, it could be important to evaluate the risk of reaction to passion fruit in latex-allergic patients because exposure to this exotic fruit, even as a component of soft drinks, could not be exceptional and might be a risk for allergic reactions.

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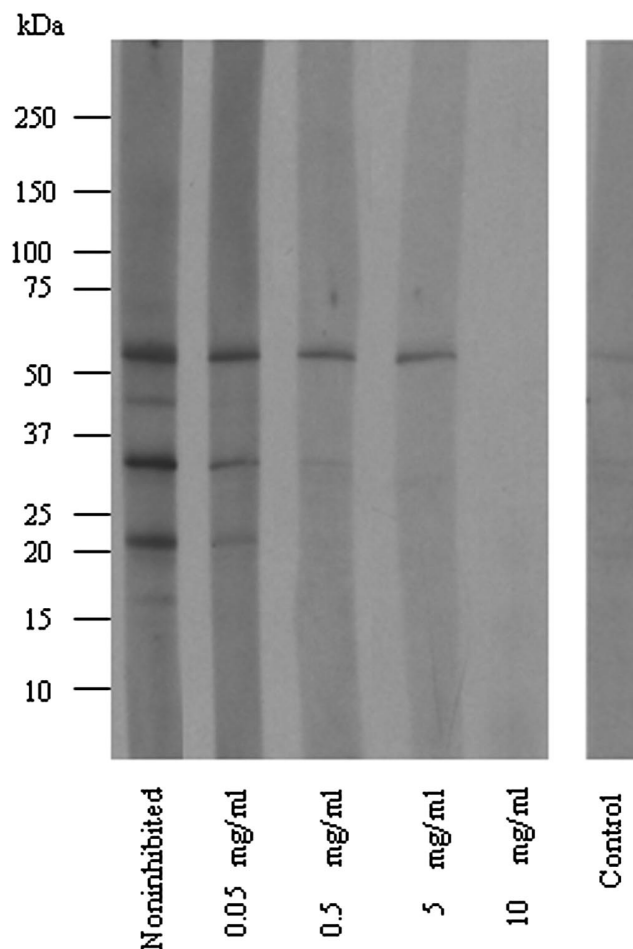


Figure 1. Inhibition of passion fruit immunoblot by latex extract at different concentrations. The control indicates inhibition of passion fruit immunoblot by passion fruit extract (1 mg/mL).

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FATAL ANAPHYLACTIC SHOCK AND TAENIA SOLIUM INFESTATION: A POSSIBLE LINK?

While performing a retrospective analysis of forensic-autopsied cases in the last 25 years, we found an interesting case of anaphylaxis that occurred in 1985. A 26-year-old, healthy, male farmer was found dead. Death occurred during sleep and did not seem linked to any previous known diseases. The man's relatives and physician reported that the man had not previously presented with any type of allergy or history of drug or alcohol abuse.

Cadaveric examination showed copious dark blood with clots in the heart cavities; dark red surface of the lungs with a moderate

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amount of blood; hyperemic, edematous laryngeal and tracheal mucosa, covered by scarce frothy mucus; increased, dark red spleen; and hypertrophic hyperemic mucosa of the stomach with raised folds. In the intestinal loops several meters of a tapeworm with a long series of proglottids were found (Fig 1); the worm was identified as *Taenia solium*. Histologic examination revealed hyperemic and edematous laryngeal mucosa; occlusion of the pulmonary alveoli by protein material mixed with erythrocytes; partially obstructed bronchi with hyperplastic epithelium, contracted walls, and enlarged, congested vessels; sporadic area of acute emphysema; edematous interstitial dissociation in the heart, with width-ways interrupted myocardial fibers with preserved structure; marked dilation and repletion of liver veins and capillaries; hypertrophic spleen follicles with dilation and repletion of the sinuses, some in the hemorrhagic area; and a moderate number of eosinophils in the spleen cords.

The cause of death was hemorrhagic pulmonary edema, conspicuous heart edema, and multivisceral congestion. The concluding cause of death was anaphylactic reaction. The target of the shock was the respiratory tract, as histologically displayed by the laryngeal edema and hyperemia and contraction of the bronchial walls.

Postmortem diagnosis of anaphylactic death is usually based on exclusion and circumstantial evidence.¹ However, in this case, swelling of the larynx, mucous plugging in the airways, pulmonary emphysema, visceral congestion, and splenic eosinophilia supported an allergic reaction.¹ Serum tryptase concentration was not measured because before the year 2000 this test was not used routinely for suspected anaphylaxis.² The most common causes of anaphylaxis (drugs, foods, bee sting) were excluded by interviews with the man's relatives, external examination of the body, and cadaveric examination.

T solium can be responsible for 2 different pathologic conditions: taeniasis and cysticercosis. In taeniasis an adult worm is present in the intestine, and the infestation is usually asymptomatic and generally recognized when segments of proglottids are found in stool specimens. Cysticercosis is caused by dissemination of the *T solium* larval form (cysticercus). Cysticercus consists of a fluid-filled bladder elongate and oval, measuring 5 to 15 mm with a single protoscolex.³

It is possible that an autoinfection of larval forms by ingestion of eggs present on the man's dirty hands or by reverse peristalsis had occurred. The larval forms were released by the digested eggs, penetrated the intestinal wall, reached the bloodstream, and developed into cysticercosal cysts. The rupture of a unknown cyst may have triggered the anaphylaxis. At the time of necroscopy, cysticercosal cysts were not searched in the examined tissues, but the presence of *T solium* in the intestine, the pathologic findings at autopsy, and the absence of other possible causes of anaphylaxis make the association between anaphylaxis and cysticercosis plausible.

In absence of a well-documented causality between *T solium* infestation and anaphylaxis, our hypothesis is speculative and needs to be confirmed by other reports. Therefore, cysticercosis should be taken into account in the case of fatal anaphylaxis of unknown reason and concomitant gastrointestinal worm infestation.



Figure 1. *Taenia solium* found in the intestinal loops.

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