

systemic allergic reaction, independent of mugwort sensitization. Although a double-blind placebo-controlled food challenge is the gold standard for the diagnosis of a food allergy (6), open oral challenge was our best viable option in this study.

In conclusion, we report a case of severe anaphylactoid reaction caused by *C. lanceolata* root ingestion in a patient with allergic rhinitis. This reaction was not mediated by IgE, but rather by direct histamine releasing effects via basophils.

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Anaphylactic shock caused by silkworm pupa consumption in China

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Key words: anaphylactic shock; food allergen; silkworm pupa; tourist.

Materials derived from the bodies of the domesticated silkworm *Bombyx mori*, including silks, contain allergens which can trigger or aggravate allergic diseases such as allergic asthma, hypersensitivity pneumonitis, allergic rhinitis and atopic dermatitis (1). The silkworm life cycle includes four distinct developmental stages: ova, larva, pupa, and imago.

Silkworm pupae are nutrient-rich, comprised of ~50% protein with abundant amounts of essential amino acids for humans (1). As a result of the cultural influences of traditional Chinese food and medicine, Chinese people often eat oil-fried pupa, water-boiled pupa or ground pupa powder. The use of silkworm pupa as food may be a custom unique to China.

Silkworm pupa can be allergenic (1). It is estimated that each year in China, there are over 1000 patients who suffer anaphylactic reactions after consuming silkworm pupa, 50 of whom received emergency room treatment for severe anaphylactic reaction. Hence, we previously purified protein allergens from a crude silkworm pupa extract through ion exchange chromatography, gel filtration and sodium dodecyl sulphate polyacrylamide gel electrophoresis. Our subsequent immunoblot analysis revealed an ~30-kDa protein as silkworm pupa's major allergen (1).

Severe allergic shock caused by silkworm pupa consumption.

Here we report the case of a French national visiting China who went into anaphylactic shock after consuming silkworm. A 37-year-old French man, with a history of allergic rhinitis, ate oil-fried silkworm chrysalis in his dinner during his visit to China in August, 2003. Thirty minutes later, he felt an itchy sensation in his mouth and on his face, accompanied by mild nausea and by flushing and swelling of his face. He proceeded to the Hospital Emergency Department, where he began to experience difficulty breathing. Upon physical examination, the emergency room physician noted cardiovascular signs of shock, including low blood pressure (70/40 mmHg) and an elevated heart rate 105/min. A diagnosis of anaphylactic shock caused by a silkworm pupa food allergic reaction was made and the patient was immediately treated with a hypodermic injection of epinephrine 1 mg. The patient was also given supplemental oxygen by mask and intravenous infusion of vitamin C 3 g and hexadecadrol 10 mg. He recovered within 3 days of the incident.

A summary of 13 prior cases of severe anaphylactic reaction caused by silkworm pupa consumption is provided in Table 1 (2–5). Treatment of this condition is consistent with the general principles of treating allergic shock generally (6). Following proper and timely therapy, patients who suffer an allergic reaction to silkworm do not exhibit sequelae (2–5).

In the absence of treatment, anaphylactic shock can lead to death within minutes. A portion of the increasing number of western tourists who visit China choose to try traditional Chinese foods. However, regulation of food allergen labeling has not yet to be achieved in China. The present case highlights the need for foreign nationals visiting China to be alerted to the allergenic risks of local foods, such as silkworm pupa, in order to reduce the risk of negative consequences on their health.

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Table 1. Thirteen cases of severe anaphylactic reaction caused by silkworm pupa consumption in China

Reference	No. of cases Sex: age (years)	History of previous allergy	History of previous allergic shock	First time SP consumed	Symptoms after SP ingestion	Reported time of onset of symptoms (h)	Diagnosis of reported case(s)	Treatment in emergency department
(2)	1 case 1 male: 21	–	None	Yes	P, U, FA, H, F, UC	0.5	Anaphylactic shock	Supplemental oxygen; Epinephrine 1 mg (HDI); hexadecadrol 10 mg (IVI).
(3)	3 cases 1 male, 19 2 females: 37,54	–	None	Yes	U, FA, HA, H, AP, V, D	2–3	Anaphylactic shock	Supplemental oxygen; Epinephrine 1 mg (HDI); hexadecadrol 10 mg (IMI); Chlorphenamine Maleate 10 mg (IMI); Cetacort 300 mg (IVI).
(4)	1 case 1 male: 20	–	None	Yes	P, U, FA, H, F, N, UC	0.5	Anaphylactic shock	Supplemental oxygen; Epinephrine 1 mg (HDI); hexadecadrol 10 mg (IVI).
(5)	8 cases 5 males: 9–46 3 females: 9–46	None	None	Yes	P, U, FA, A, AP, V, N, D	0.5–4	Severe anaphylactic reaction	Supplemental oxygen; Diprazine 25 mg (IMI) plus hexadecadrol 5 mg (IMI). After 2 h, hexadecadrol 10 mg (IVI) plus VtC 3 g (IVI) and VtB6 0.2 g (IVI) (only for 2 cases).

A, angioedema; AP, abdominal pain; D, dyspnoea; F, fainting; FA, flushed appearance; H, hypotension; HA, headache; N, nausea; P, pruritus; U, urticaria; UC, unconsciousness; V, vomiting; IMI, intramuscular injection; IVI, intravenous injection; HDI, hypodermic injection; SP, silkworm pupa.

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Dissociation between biological and clinical response to oral challenge in children with food allergy

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Key words: food allergy; food challenge; inflammatory markers; pediatrics.

Controlled oral food challenges are considered as the golden standard for the diagnosis of food-related symptoms (1), the diagnosis being carried out based on the symptoms after a challenge.

In children with food allergy, a biological response to food ingestion persists even when the clinical tolerance is acquired.

The retrieval of stable mediator metabolites from urine provides a valuable indication of the synthesis or release of mediators such as leukotrienes (LT) and prostaglandins (PG). LTE₄ is a stable urinary product considered as an integrated measure of overall LT production in the body, while 9α,11β-PGF₂ is one of the major urinary metabolites of PGD₂, the main cyclo-oxygenase product released on mast-cell activation (2).

The aim of our study was to compare LTE₄ and 9α,11β-PGF₂ urinary concentrations in children with food allergy and in healthy controls and to evaluate the change in these biological mediators before and after a controlled food challenge.

We recruited 31 children with food sensitization and a history of immediate reaction after food ingestion (mean age 3.6 years) and 16 healthy controls (mean age 3.3 years). All patients had been on an elimination diet and had serum positive for specific IgE (>0.35 kU/l) for the offending food. Thirty-one open food challenges with cow's milk (*n* = 24), egg (*n* = 5), wheat (*n* = 1)