

Case Report

A NOT SO HEALTHY MUESLI: A CASE REPORT

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ABSTRACT

We present a patient with severe anaphylaxis, angioedema, hypotension and shock. The near fatal allergic reaction was caused by eating yogurt with muesli containing pine nuts. The patient developed an acute infero-posterolateral myocardial infarction due to systemic hypotension and shock. Food allergy to pine nut was demonstrated by dosage of specific IgE to pine nut.

55 year old man who skipped his regular breakfast and replaced it by yogurt with muesli provoking severe anaphylaxis.

CASE REPORT

A 55-year old male was admitted to the emergency department because of angioedema, hypotension, and chest pain shortly after eating yogurt with muesli (Activia natural yoghurt with muesli®, Danone, Belgium). A few minutes after starting the meal he sensed an itchy feeling in his throat quickly worsening with swelling of his lips and face, shortness of breath and dizziness. He was a current smoker with a smoking history of 50 packs-year. He had no history of pollinosis. He was not on angiotensin converting enzyme inhibitors nor beta-blokkers.

On physical examination he was afebrile. He had angioedema of the face and upper trunk without urticaria. His blood pressure was 80/50 mmHg, heart rate 125/min, percutaneous oxygen saturation while breathing room air 87%. Bilateral wheezes were present over the chest. Slow intravenous epinephrine 0.1 mg of a 1/10000 solution, intravenous corticosteroids (methylprednisolone 125 mg), and antihistamines (promethazine 50 mg IM) were administered. ECG revealed acute inferolateral ischemia. Creatinine kinase was 315 U/L (nl < 145) with a creatine kinase muscle-brain fraction of 28 U/L (nl < 16). Troponin levels were slightly elevated 0.09 µg/L (nl < 0.08). An urgently performed coronarography showed no coronary lesions. No late allergic reaction appeared. Standard chest-X-ray performed after pressure resuscitation was normal. A pulmonary function test, done before discharge, was normal.

INTRODUCTION

Food is the leading cause of outpatient anaphylaxis¹. Although most patients are aware of their food allergy, over half are unaware of the allergen content of the food they eat². So still a lot of patients present to the emergency department with acute anaphylaxis after eating hidden triggers. We report the case of a

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The patient's history revealed no previous events of allergy or oedema in relation to food. It was the first time he ate yoghurt with muesli.

Total serum IgE was 1385 kIU/L (nl <100) with a positive specific IgE to pine nut 98.5 kU/L (nl < 0.35 kU/L) and hazelnut 4.44 kU/L. No specific IgE could be found against grass pollen, tree pollen and moulds. No specific IgE to latex was present. He was sensitized to house dust mite (specific IgE 0.68 kU/L) and cat (specific IgE 1.18 kU/L) but without history of rhinitis or asthma. Dosage of specific IgE to other components of the muesli (wheat, barley, almond) was negative.

Based on the clinical history and the immunoassay results a diagnosis of pine nut anaphylaxis was made.

He received a prescription of a self injectable epinephrine syringe (0.3 mg) and a list of foods containing pine nut.

DISCUSSION

Anaphylaxis is an acute onset systemic allergic reaction³. It is important to keep in mind that any food can cause anaphylaxis. In adults, most commonly implicated foods that cause this serious allergic reaction are peanuts, tree nuts, fish and shellfish¹. Although there are many reports on peanut, many other nuts also are able to induce this type of reaction.

A lot of people like to eat yogurt because it contains friendly bacteria which help digest lactose. Some of these brands even contain probiotics, as in the yoghurt of our patient. *Bifidobacterium lactis regularis* is added in the belief that this has beneficial effects on colonic digestion. Some reports even attribute anti-allergic effects to these probiotics, especially in infant atopic eczema if taken long enough.⁴ An allergic reaction to this component of the yoghurt is very unlikely.

To turn the yoghurt into a breakfast or a snack, muesli is added. Muesli is a mixture of cereals, (dried) fruit and nuts. Our patient's muesli made up 4.4% of the total meal and contained honey, barley- and wheat flakes, wheat bran, almond, walnut, pine nut and hazelnut. These last two components made it into a dangerous mixture for our patient. Although it is possible that the unique combination of yoghurt and the different components of the muesli were responsible for changes in the enzymatic digestion and presentation of the allergenic proteins⁵, we were only able to demonstrate specific IgE against hazelnut and pine nut.

Up to 72% of peanut or tree nut allergic patients report symptoms during their first known exposure, al-

though these patients must have been sensitized previously through unknown exposures⁶. Pine nuts are the seeds of *Pinus pinea* and are consumed as roasted nuts or added to vegetable salads and to different kinds of sauces (e.g. Italian pesto). Hazel nuts are a popular additive to chocolate spread. It is clear that this content should be labelled on food to avoid unforeseen exposure. This labelling was present on the yoghurt of our patient.

Is this patient's anaphylaxis an IgE mediated reaction to a unique tree nut allergen or is it a cross reactivity to so-called pan-allergens such as profilins, heveins and lipid transfer proteins which share identical domains with pollen, seed, fruit and vegetable allergens?⁵ The main pine nut allergen is not well characterized by immunoblotting assays but is probably a 17-kDa allergen⁷. Immunoblotting of *Artemisia vulgaris* and pine nut recently revealed two matching diffuse bands just below 14-kDa and 30-kDa suggesting cross-reactivity⁸. The major allergen of hazelnut is an 18-kD protein homologous to Bet v 1, an allergen present in birch pollen⁹. Some publications describe cross-reactivity between pine nut and other nuts¹⁰. We could not demonstrate sensitisation to common inhalant allergens as weed pollen, grass pollen, moulds or latex, making cross-reactivity to major pollen allergens less likely. Our patient is sensitized to cat and house dust mite but no cross-reactivity has been described with tree nut storage proteins (vicilins, legumins, and 2S albumins) responsible for most tree nut allergies⁵.

Different markers of mast cell degranulation, such as tryptase and carboxypeptidase, with a longer half-life than tryptase, are elevated in a subset of anaphylactic patients. But these tests lack sensitivity and specificity for diagnosing anaphylaxis¹¹. The clinical diagnosis is based on history and physical examination¹. The severity of the symptoms in our patient advised against an oral challenge test to confirm diagnosis. Acute systemic reactions immediately after skin testing with fresh pine nut have been described, so caution with in vivo testing is warranted^{12;13}. On the basis of the patient's history and immunoassay we were able to prove type I allergy to pine nuts probably in combination with hazelnut.

The time of onset between ingestion of the allergen and anaphylaxis has been reported to be approximately 25-35 minutes (range 10 min till 6 hours)¹. Factors predicting anaphylaxis to tree nuts are incompletely understood. Recent work suggests an increased likelihood for predominantly asthmatic symptoms of anaphylaxis if the victim has underlying severe atopic

disease. Severe pharyngeal oedema is more associated with severe allergic rhinitis and tree nut allergy¹⁴. This last association might be due to the cross reactivity between certain tree pollen and tree nut allergens¹⁵.

Pharmacologic treatment is symptomatic and can be life-saving. Prompt injection of a dose of 0.01 mg/kg epinephrine IM up to 0.5 mg is recommended¹⁶. A disadvantage of commercialised syringes is that they only deliver a standardized dose (e.g. Epipen® 0.15 mg and 0.3 mg). In severe reactions IV administration may be necessary, although high serum levels can be achieved through the intramuscular route³.

The use of corticosteroids is controversial. Some authors propose to administer them to prevent late response reactions. Antihistamines, which alleviate especially cutaneous symptoms, are considered second-line treatment for anaphylaxis¹.

Anaphylaxis precipitating coronary vasospasm leading to myocardial infarction is uncommon and only sporadic cases have been reported¹⁷. It is not clear whether mast cell degranulation itself or the IV use of a low dose of epinephrine triggered the AMI in our patient.

Coronary vasospasm due to potent vasoactive mast cell derived mediators released from perivascular and interstitial cardiac mast cells is one proposed mechanism¹⁸. Catecholamine induced damage due to a low dose intravenous epinephrine is another possibility¹⁹. Finally, stress cardiomyopathy, also referred to as Tako-Tsubo cardiomyopathy, with typical acute reversible apical ventricular dysfunction, in relation to high levels of epinephrine is another explanation²⁰. In our patient the typical apical dysfunction was lacking.

In summary we presented a patient who developed an acute anaphylaxis after eating yoghurt with muesli. We hypothesized that the reaction was triggered through an IgE mediated reaction against pine nut and hazelnut. The anaphylaxis was complicated by an acute myocardial infarction.

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