

Scombroid fish poisoning: A potentially life-threatening allergic-like reaction

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Scombroid fish poisoning, most often caused by the consumption of tuna or any other scombroid fish, has been attributed to the metabolic products of bacterial degradation of blue fish flesh. Symptoms are produced by histamine release¹ and resemble those encountered with IgE-mediated food allergies. The illness is characterized by flushing, headache, dizziness, burning sensation in the mouth and throat, abdominal cramps, nausea, vomiting, and diarrhea. Urticaria and generalized pruritus are often manifest. In more severe cases bronchospasm and respiratory distress may develop. Histamine results from enzymatic decarboxylation of histidine, which occurs optimally between 20° and 30° C and is prevented if bacterial growth is inhibited by chemicals or refrigeration. Food with histamine concentrations exceeding 50 mg per 100 gm is generally considered to be hazardous.² This illness has short incubation periods, ranging from a few minutes to a few hours.

Here we report a collective tuna fish poisoning with severe clinical manifestations, which were developed in November 1995.

CASE REPORTS

Case 1

On November 14, a previously healthy 45-year-old man was admitted to the emergency department for treatment of dyspnea, vomiting, diarrhea, generalized erythema, and pruritus, which started 20 minutes after tuna ingestion. Hypotension, tachycardia, bronchospasm, cyanosis, hypoxia, and metabolic acidosis were found. The patient was thought to be experiencing acute anaphylaxis caused by fish allergy, and he was treated with epinephrine, fluids, oxygen, steroids, and antihistamines. Symptoms disappeared in 15 to 17 hours.

Case 2

On November 15, a 30-year-old man was first seen 30 minutes after tuna ingestion with erythema, cutaneous pruritus, facial flushing, palpebral angioedema, sweating, palpitations, dizziness, hypotension, headache, nausea, vomiting, and dyspnea; he was diagnosed with acute anaphylaxis and was treated with epinephrine, fluids, oxygen, steroids, and antihistamines. Eradication of the symptoms occurred in hours.

Case 3

On November 15, a 54-year-old man was first seen a few minutes after tuna ingestion with urticaria, facial flushing, headache, and glottis angioedema; epinephrine was required for resolution of symptoms.

Case 4

On November 15, the wife of the patient described in case 2 had only diarrhea and headache and did not require any treatment.

Cases 5 to 7

On November 17, four persons were first seen with headache and urticaria after tuna ingestion. Symptoms disappeared with administration of antihistamine.

After the first case, an alert was given to Public Health and Emergency Services in the Health Area. Six more cases were found. The epidemiologic investigation showed that in all cases the individuals had eaten tuna (*Thunnus thynnus*) from the same grocer's shop.

Serum and urine histamine were measured by radioimmunoassay, urine methyl-histamine by chromatography, and serum tryptase by radioimmunoassay (IRMA) in cases 2 and 3. In the rest of the patients it was impossible because the patients were initially seen after the first 24 hours. In the two studied patients, increased levels of serum histamine (115.4 and 89.6 µg/dl), urine histamine (74 and 57 µg/l), and 24-hour urine methylhistamine (250 and 213 µg/24 hr, respectively) were observed, with undetectable tryptase serum levels (<2 UI/L). All patients were studied in Allergy Consultants to rule out fish allergy, through the determination of specific IgE in vivo and in vitro by prick test and ELISA, respectively. All the results were negative, but when the prick test was carried out by using the affected tuna, all of the results were positive in patients and control subjects. Quantification of histamine content of the fish was elevated (2000 mg per 100 gm of fish).

DISCUSSION

Some authors have previously described scombroid poisoning with slight symptoms.^{3,4} Three of our patients exhibited severe symptoms with potentially life-threatening events, so we consider it important to report their cases. Severe symptoms may be related to the high levels of histamine found in the implicated fish flesh. Although cases 1 and 2 were initially diagnosed as allergic reactions, food poisoning was

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promptly suspected and preventive measures were adopted early. We insist on the importance of recognizing the syndrome as an intoxication rather than an allergic reaction so that the source of the toxin can be identified and additional cases prevented.

Atopic disease in human beings results primarily from the activity of tissue mast cells and circulating basophils with histamine liberation, which is quickly metabolized in blood. This substance can come from ingested food and can be produced by different cells. Tryptase is located in secretory granules from mast cells and is released at the same time as histamine during degranulation. Tryptase levels are an indicator of mast-cell activation, which can be detected by immunoassay.⁵ Then the quantification of tryptase release in vivo allows us to make a precise valuation of mast cell activation. The low levels of tryptase in our patients reject an

anaphylactic reaction and favor poisoning by an exogenous source of histamine.

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