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Histamine (Scombroid) Fish Poisoning: a Comprehensive Review

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Abstract Histamine fish poisoning, also known as scombroid poisoning, is the most common cause of ichythyotoxicosis worldwide and results from the ingestion of histamine-contaminated fish in the Scombroidae and Scomberesocidae families, including mackerel, bonito, albacore, and skipjack. This disease was first described in 1799 in Britain and re-emerged in the medical literature in the 1950s when outbreaks were reported in Japan. The symptoms associated with histamine fish poisoning are similar to that of an allergic reaction. In fact, such histamine-induced reactions are often misdiagnosed as IgE-mediated fish allergy. Indeed, histamine fish poisoning is still an underrecognized disease. In this review, we discuss the epidemiology, pathophysiology, evaluation, and treatment of scombroid disease. Because more than 80 % of fish consumed in the USA is now imported from other countries, the disease is intimately linked with the global fish trade (National Marine Fisheries Service, 2012). Preventing future scombroid outbreaks will require that fishermen, public health officials, restaurant workers, and medical professionals work together to devise international safety standards and increase awareness of the disease. The implications of scombroid poisoning go far beyond that of fish and have broader implications for the important issues of food safety.

Keywords Food safety · Scombroid poisoning · Histamine poisoning · Fish

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Introduction

Histamine fish poisoning, also known as scombroid poisoning, is the most common cause of ichythyotoxicosis worldwide and results from the ingestion of histamine-contaminated fish in the Scombroidae and Scomberesocidae families, including mackerel, bonito, albacore, and skipjack. Other non-scombroid fish, such as sardine, bluefish, and rarely salmon, can also cause the disease. Occasionally, cheeses, particularly Swiss cheese, have been implicated [1].

The disease was first described in 1799 in Britain and re-emerged in the medical literature in the 1950s when outbreaks were reported in Japan [1]. In the USA, the first cases were documented in 1968. Since then, cases have been described in a number of different settings, including restaurants, cafeterias, schools, army barracks, and medical conferences. Scombroid poisoning occurs when fish are inadequately frozen, which allows bacteria located in the flesh of the fish to thrive. In the process, bacteria convert histidine to histamine.

The symptoms associated with histamine fish poisoning are similar to that of an allergic reaction. In fact, such histamine-induced reactions are often misdiagnosed as IgE-mediated fish allergy. Indeed, histamine fish poisoning is still an underrecognized disease, though it may account for 39 % of all seafood-associated outbreaks in the USA [2]. This disorder may be well known to allergists, but it is important for other physicians, especially pediatricians, internists, and emergency physicians, to be aware of it when considering a differential diagnosis.

In this review, we discuss the epidemiology, pathophysiology, evaluation, and treatment of scombroid disease. Because more than 80 % of fish consumed in the USA is now imported from other countries, the disease is intimately linked with the global fish trade [3]. Preventing future scombroid outbreaks will require that fishermen, public health officials, restaurant workers, and medical professionals work together to devise

international safety standards and increase awareness of the disease. As allergists, we tend to be predominantly concerned with the clinical spectrum of food sensitivity [4–6], but the larger issue of food safety is even more critical [7].

Epidemiology

Since 1980, fish consumption in the USA has dramatically increased. In fact, US seafood consumption is third in the world, right behind Japan and China [3]. In 2010, the average seafood consumption in the USA was 15.8 lb per person. Paralleling the rise in fish consumption, cases of histamine fish poisoning have increased. In the USA, between 1988 and 1997, histamine fish poisoning was officially reported in 145 outbreaks involving 811 people from at least 20 states [8, 9]. Between 1998 and 2008, there were official reports of 333 outbreaks involving 1383 people, resulting in 59 hospitalizations [10, 11]. Yet, cases of histamine fish poisoning are still vastly underreported, due to misdiagnosis and the inherent barriers in reporting cases to public health organizations. More recently, between 2009 and 2012, there were 40 outbreaks reported, involving 136 people, resulting in one hospitalization [10].The states with the highest number of reported cases were California, Hawaii, and New York. Of note, there has never been a death due to histamine fish poisoning reported in the USA.

Outside the USA, histamine fish poisoning is most frequently reported in Japan and the UK. In fact, the largest outbreak ever recorded, involving 2656 people, was recorded in Japan in 1973 [1]. Cases have also been documented in Australia, Bermuda, Canada, China, Croatia, Czech Republic, Egypt, France, Germany, Indonesia, Israel, Italy, Korea, New Zealand, Netherlands, Norway, Oman, Papua New Guinea, Poland, Romania, Senegal, Spain, Sri Lanka, South Africa, Sweden, Switzerland, Taiwan, and the former Yugoslavia (Table 1). Only one death has been noted worldwide [1].

Between 1998 and 2012, the fish most frequently involved in histamine fish poisoning reactions in the USA, according to the CDC, are tuna, mahi mahi, escolar, marlin, and salmon. In fact, tuna and mahi mahi alone make up more than 80 % of reported cases (Fig. 1).

Clinical Features

Histamine fish poisoning is usually mild, of short duration, and self-limiting. Given that symptoms result from excess amounts of histamine, the physical manifestations of histamine fish poisoning are similar to those of an allergic reaction. Symptoms typically occur within 20–30 min of fish ingestion and most commonly include facial flushing, abdominal pain, diarrhea, headache, and palpitations (Table 2). Other reported symptoms include flushing of the neck and torso or generalized flushing, nausea, vomiting, urticaria, dry mouth, lightheadedness, and rarely wheezing or loss of consciousness due to hypotension. Some patients with scombroid poisoning also note a metallic, bitter, or peppery taste. Most symptoms resolve within 6–8 h, but feelings of malaise can last for a day or more. In a case series of histamine fish poisoning involving 71 French soldiers who ate yellowfin tuna, 87 % reported warmth sensation, 70 % reported weakness, 86 % reported

Table 1 Geoepidemiology of histamine fish poisoning

US outbreaks (1998–2008)	333
US cases (1998–2008)	1383
US hospitalizations (1998–2008)	59
Prevalence of histamine fish poisoning outbreaks relative to all food outbreaks	2.5 % (333/13,405)
Total deaths from histamine fish poisoning in the literature	1
Countries with documented reactions	Australia, Bermuda, Canada, China, Croatia, Czech Republic, Egypt, France, Germany, Indonesia, Israel, Italy, Japan, Korea, New Zealand, Netherlands, Norway, Oman, Papua New Guinea, Poland, Romania, Senegal, Spain, Sri Lanka, South Africa, Sweden, Switzerland, Taiwan, USA, USA, Yugoslavia
Israel (2005–2007)	21 events involving 46 cases
Switzerland (1966–1991)	76 cases
Japan (1998–2008)	150 cases each year
New Zealand (2002–2004)	12 events involving 73 cases
UK (1992–2004)	56 events involving 296 cases
Australia (1988–2010)	38 events involving 148 cases

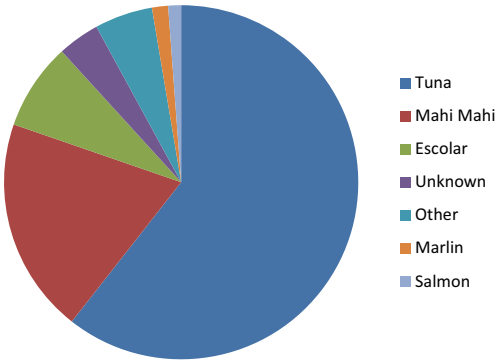

Fig. 1 Proportion of each fish causing histamine fish poisoning reactions, 1998–2012 (CDC Food Outbreak Online Database)

flushing, 83 % reported headache, and 57 % reported diarrhea [12].

Severe reactions result in hypotension, bronchospasm, respiratory distress, myocardial infarction, and even refractory myocardial dysfunction requiring biventricular assist devices [13, 14].Of the 71 French soldiers, 19 % reported dyspnea, 5 % reported mouth swelling, and 12 % reported difficulties swallowing [12].

Atypical presentations have also been reported. For instance, a 60-year-old male presented with vision loss and atrial tachycardia with block after eating tuna [15]. In another case, mackerel ingestion caused isolated severe hypotension, with a blood pressure of 60/40, in an 80-year-old woman [16].

Pathophysiology

The clinical symptoms of histamine fish poisoning are not surprising given its causative agent, histamine [2-(3-imidazolyl)-ethylamine], an endogenous amine found in many tissues in the body, particularly the liver and spleen. Many cells in the body, such as mast cells, basophils, and gastric enterochromaffin-like cells, produce histamine [17]. Histamine becomes active through its four receptor subtypes and has many disparate effects throughout the body, including cell proliferation, cell differentiation, vasodilation, hematopoiesis, embryonic development, wound healing, and neurotransmission. At the same time, histamine influences cognition, memory, vigilance, and the sleep-wake cycle. Of relevance to histamine fish poisoning, activation of the H1 receptor results in the immediate allergic response, while H3 receptor activation modulates neurotransmitter release in the central nervous system, resulting in nausea, vomiting, and headaches [17, 18].

Table 2 Diagnostic features of histamine fish poisoning

Suggestive symptoms	-Facial flushing, abdominal pain, diarrhea, headache, palpitations -Metallic, bitter, or peppery taste in the mouth
Details in the history	-Symptoms occur within 20 min to 1 h of fish ingestion -What type of fish was ingested -No previous history of food allergies -Medications: isoniazid or monoamine oxidase inhibitors -Symptoms are self-limited and resolve within 6–8 h -Symptoms improve with antihistamines -Other patrons in the same location become ill with similar symptoms
Laboratory data	-Fish histamine level >50 mg/100 kg -Skin prick testing (see text for details) -Normal tryptase level -Negative 5-HIAA, metanephrines, urinary PGD2

Histamine was first suggested as the implicated agent in scombroid poisoning in the 1940s. Yet, for half a century afterward, studies suggested that histamine could not be absorbed via the gastrointestinal tract in sufficient quantities to reach an appreciable amount in the systemic circulation [19]. Consequently, the role of histamine in scombroid toxicity was seriously doubted. It was not until 1991, when volunteer subjects knowingly ingested spoiled marlin and urinary histamine levels were subsequently measured, that researchers were able to conclusively demonstrate that excess histamine was the culprit for scombroid toxicity.

Histidine decarboxylase, found in bacteria that reside in fish gills and gastrointestinal tracts, is the enzyme responsible for converting histidine to histamine. Bacteria species with histidine decarboxylase activity include enterics such as *Proteus*, *Enterobacter*, *Serratia*, *Citrobacter*, and *Escherichia coli*, as well as *Clostridium*, *Vibrio*, *Acinetobacter*, *Pseudomonas*, and *Photobacterium* [20, 21]. The exact composition of histamine-forming bacteria in each fish is unique and varies according to geographical location, fish feeding habits, nets, season, water temperature, water quality at harvest, handling processes, and the cleanliness of the locale where the fish is sold. At the same time, the efficacy of histidine decarboxylase is dependent on temperature, pH, and sodium concentration [22]. Of note, once histamine is formed, it is highly resistant to tampering, so cooking, smoking, freezing, and canning cannot prevent histamine fish poisoning reactions.

Ideally, fish should be kept a temperature of 0 °C or less, so that the bacteria cannot grow and histidine decarboxylase is not activated. Left at adequately high temperatures for just a few hours, bacteria on the fish can multiply to Malthusian proportions. For instance, toxic levels of histamine can form in 2 to 3 h in fish stored at 20 °C or greater [23]. In fact, histidine decarboxylase can continue to function even when the bacteria are no longer viable [18].

Paradoxically, some cases of histamine fish poisoning have occurred even when histamine levels in the fish were low. One hypothesis for this discrepancy is that certain proteins, possibly cadaverine and/or putrescine, simultaneously act as histamine potentiators and inhibit histamine-metabolizing

enzymes [24]. Other hypotheses include intolerance, unknown substances that act as histamine agonists, or that a protein, such as urocanic acid, causes the endogenous release of histamine via mast cell degranulation [25]. Ultimately, histamine may be necessary, but not sufficient, to cause the syndrome of histamine fish poisoning.

Evaluation and Diagnosis

The evaluation of histamine fish poisoning begins with a thorough history. Attention should be given to the type of fish ingested, whether the fish was cooked or raw, whether these reactions have occurred in the past, and the time frame between fish ingestion and the onset of symptoms. Patients on isoniazid (INH) and monoamine oxidase inhibitors (MAOIs), which inhibit histamine metabolism, may be at an increased risk for histamine fish poisoning [26]. Other foods ingested prior to the reaction, aside from fish, should be noted. Moreover, comorbidities such as unstable asthma or heart disease can result in a life-threatening histamine fish poisoning reaction.

De novo fish allergy in adults is rare but can occur. The estimated prevalence of fish allergy is 0.6 %, with little difference across age and ethnic groups [27]. In some cases of new-onset fish allergies, sensitization via inhalation of fish allergens may be a factor [27]. Nevertheless, if other people who ate at the same restaurant become ill, then the cause is likely to be histamine fish poisoning rather than new-onset fish allergies.

While patient symptoms may be suggestive of histamine fish poisoning, testing the fish itself helps to definitively diagnose the toxic etiology. An assessment of spoiled fish based on appearance and odor alone does not aid in diagnosis. Instead, it is necessary to test the histamine levels directly. Fresh fish contain minimal amounts of histamine, less than 0.01 mg/100 g; histamine poisoning only occurs when the histamine levels are orders of magnitude greater [22]. As a general rule, >50 mg of histamine per 100 g of fish causes histamine fish poisoning. In the outbreak involving 71 French soldiers reference earlier, the mean histamine concentration in the spoiled tuna was 490 mg/100 g. To ensure a wide margin of error, food regulations in the USA require that histamine levels not exceed 5 mg/100 g of fish, while in Europe levels cannot exceed 10 mg/100 g [28].

Histamine levels are dependent on both temperature and the length of incubation. For example, at 26 °C, the histamine concentration in mahi mahi, skipjack, and tuna can reach 50 ppm after 12 h. At 35 °C, however, 50 ppm of histamine is reached in only 9 h, and by 12 h, histamine concentration exceeds 500 ppm [29]. Importantly, there can be an uneven distribution of histamine throughout a single fish. Thus, multiple samples from the same fish should be analyzed.

In the past two decades, many technologies, with varying strengths and weaknesses, have been developed to measure histamine levels. The most widely used and official method of histamine analysis in the USA is reverse-phase high-performance liquid chromatography (HPLC). Because HPLC is impractical outside the laboratory, rapid tests, including flow injection analysis (FIA) and commercial quantitative ELISA kits, are used to make real-time diagnoses. Indeed, during histamine fish poisoning outbreaks, regulatory agencies favor the use of FIA and quantitative ELISA.

In the allergy clinic, where in vitro testing for tissue levels of histamine in fish is not available, skin prick testing (SPT) can help diagnose histamine fish poisoning [30]. For instance, in our clinic, a patient may report a reaction to seared ahi (tuna) that they cooked at home. First, a sample of the suspected tuna that caused the reaction should be obtained, but also samples of a flash-frozen tuna filet available in many locales, or if not available, several different samples of tuna from different sources that are as fresh as possible and on ice or frozen. Then, the patient is skin tested, using the prick-prick method, to the various tuna samples as well as to commercial fish extracts, including tuna. If all the tests are positive on the patient to tuna, then the development of a new-onset fish allergy is highly likely. However, if the SPT is positive only to the implicated tuna sample, histamine fish poisoning or a new food allergy to something used in the food preparation may be to blame (for example, sesame or macadamia nuts that should also be part of the skin prick testing panel). The physician should then also test a normal control subject with histamine, saline, one of the fresh tuna samples and the implicated sample. If the normal control person also develops a positive skin test to the implicated tuna, this is very likely histamine fish poisoning and the state health department should be notified and provided the sample for quantitative testing and follow-up. We then recommend that patients then undergo repeat SPT to tuna that has been handled properly as far as known, followed by open food challenge in clinic. If this second SPT is negative and the patient subsequently passes an oral food challenge to tuna, then histamine fish poisoning likely caused the patient's symptoms.

The differential diagnosis for histamine fish poisoning reactions include an allergic reaction to finned fish, myocardial infarction, anisakiasis, IgE-mediated reactions to *Anisakia* larvae, and flushing disorders such as carcinoid syndrome and mast cell activation syndrome. Urinary *N*-methylhistamine can be elevated in both histamine fish poisoning and anaphylaxis, while serum tryptase and urinary PGD2 and 9a-11B PGF2, a PGD2 metabolite, will be normal in histamine fish poisoning because mast cell degranulation does not occur. Moreover, tryptase and urinary prostaglandin metabolites, 5-HIAA, or urinary metanephrines can help diagnose mast cell activation syndrome, carcinoid syndrome, and pheochromocytoma, respectively.

Treatment

Once the patient's history supports a diagnosis of histamine fish poisoning, immediate use of antihistamines is the mainstay of treatment. No double-blind, placebo-controlled trials have been performed to validate treatments or the superiority of one antihistamine or combination of antihistamines over others. Instead, recommended treatments are drawn from case reports and review articles. For mild to moderate symptoms, effective oral H1 antagonists include diphenhydramine, cetirizine, and chlorphenarimaine (Table 3). Cetirizine is preferred because it is less sedating. H2 blockers such as cimetidine, famotidine, or ranitidine can also be added. If nausea is present, intravenous promathazine can be used and intravenous fluids are indicated for diarrhea. The patient's symptoms should completely resolve in 6–8 h.

For more serious presentations, especially if a patient is unable to tolerate oral medications, intravenous diphenhydramine and ranitidine or famotidine are the drugs of choice. Intravenous fluids are indicated in treatment of hypotension. If the symptoms are particularly severe, then IM epinephrine should be considered, or low dose pressors titrated to effect, though this is extremely rare, given that scombroid poisoning is mast-cell- and basophil-independent.

It is possible that being on H1 or H2 blockers may prevent histamine fish poisoning reactions from occurring in the first place. Yet, because of the rarity of the disease, prescribing histamine antagonists for prophylactic purposes is not warranted. This could be considered for patients on INH or MAO inhibitors who have had a first episode. Furthermore, patients should be educated that histamine fish poisoning is a distinct clinical entity from fish allergy.

Health Policy Initiatives

In 1996, the FDA implemented the Hazard Analysis and Critical Control Point (HACCP) program, which is a set of rules that governs time and temperature requirements at critical control points along the entire supply chain (Center for Food Safety and Applied Nutrition, FDA, September 1996, *Fish and fisheries product hazards and control guide*). The principles of HACCP are now being applied in other countries. However, it is difficult to ensure that boats are adhering to HACCP. In 2002–2003, for instance, only 5–7 % of 8500 companies importing seafood were investigated by FDA regulators, uncovering low compliance rates with HACCP regulations (FDA 2002/2003). Because so much of the fish sold in the USA has international origins, standardization of seafood safety protocols is becoming increasingly important.

Notably, some fish sold in the USA—more than 20 %, according to one figure—are caught by sports fishermen whose boats are exempt from strict federal guidelines that regulate commercial fisheries [31]. As a result, several histamine fish poisoning outbreaks have been traced back to fish caught on private boats. Thus, implementing time and temperature standards for privately caught fish could help further reduce the incidence of histamine fish poisoning.

In the majority of cases, the source of the fish is never determined. However, through impressive feats of sleuthing, the cause of an outbreak can sometimes be traced back to its original source. In 2006, for example, the CDC identified an outbreak in Louisiana in which six employees at an oil refinery became ill after ingesting tuna steaks [23]. The tuna in question were caught in Indonesia, shipped to Boston, and subsequently transported to Louisiana. In another instance, people in Tennessee became ill after eating tuna that was harvested in Vietnam. Indeed, the route from the fishing net to a restaurant's refrigerator can be tortuous.

The medical community can also do its part. The first step is educating health care providers about this underrecognized disease, particularly first-line providers such as emergency room physicians and primary care doctors. Second, physicians should report histamine fish poisoning cases to local and state health agencies, so that outbreaks can be closely monitored and corrective actions instituted.

Table 3 Treatment

Mild to moderate symptoms	-Oral H1 blockers: diphenhydramine, chlorpheniramine, cetirizine, or IV diphenhydramine -Oral H2 blockers: ranitidine, cimetidine, famotidine, or IV ranitidine or famotidine -Intravenous fluids -Promethazine for nausea
Severe symptoms	-Epinephrine IM or IV epinephrine or dopamine titrated to effect -Methylprednisolone
Prevention	-Rigorous food safety -H1 or H2 blockers prior to eating seafood

Future Directions

Although FIA and ELISA can accurately detect elevated histamine levels, both tests have drawbacks that prohibit their widespread implementation in the field. Thus, future research should focus on the creation of a test that is inexpensive, portable, and easy to use. If such a test is devised, then the diagnosis of histamine fish poisoning could become standardized worldwide and prevention could be enhanced.

At the same time, some aspects of the pathophysiology of scombroid poisoning still need to be clarified. For instance, which bacteria are most culpable for the conversion of histidine to histamine? What role do cadaverine and putrescine play? And, are histamine agonists involved? We eagerly await the results in future studies.

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