Scombroid fish poisoning: latest evidences of a still unknown disease

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Abstract

Foodborne diseases are common but often under-notified causes of morbidity in the United States and Italy, and fish foods are often involved in these diseases. The scombroid fish poisoning is a chemical poisoning characterized by symptoms that appear from 10 minutes to 2 hours after the consumption of scombroid and not scombroid fish. The scombroid fishes naturally contain high levels of histidine which is converted to histamine as a result of the histidine decarboxylase contained in bacteria, which grow and multiply if there are no appropriate conditions to control their growth. Symptoms are characterized by rashes on the face, neck and upper chest, flushing, sweating, nausea, vomiting, diarrhea, abdominal cramps, headache, dizziness, palpitations, oral burning sensation, metallic taste and hypotension, which usually resolve within 24 hours. It was recently reported in literature (2015), a case of Kounis syndrome with coronary vasospasm precipitated by scombroid fish poisoning. The rapid cooling of the fish, and the strict maintenance of the cold chain until consumption are essential to prevent the degradation of the fish, the proliferation of bacteria, the histidine decarboxylase activation, the conversion of histidine to histamine and the development of an anaphylactoid reaction resulting in ingestion.

KEY WORDS: food poisoning, scombroid poisoning, epidemiology, prevention.

Introduction

Foodborne diseases are among the most common and underreported sources of morbidity in the United States (1) and in Italy, and seafood products are often implicated in some of these illnesses (2). Ichthyosarcootoxism is an English term for intoxication resulting from the ingestion of the flesh of poisonous fish. There are four major types of ichthyosarcootoxism:

• ciguatera due to ingestion of ciguatoxin, present in some fishes (in particular Gambierdiscus toxicus). The syndrome is often associated with the consumption of fish from tropical or subtropical seas although rare cases have also been reported after the consumption of fish from the Mediterranean Sea;
• tetraodon, or puffer, poisoning, caused the prohibition of puffer fish trade in Italy since 1992 and consequently the preparation of fugu, the famous dish prepared with this fish;
• scombroid poisoning;
• gymnothorax poisoning, due to ingestion of poison contained in morays.

All of these have many characteristics in common, but they differ as to the predominance of certain types of symptoms (3). Although relatively rare, scombroid poisoning has been known for many years and was first reported in 1799 in Britain (4).

Originally, the illness was associated with scombroidea fish (e.g., large dark meat marine tuna, albacore, mackerel); however, the Centers for Disease Control and Prevention (CDC) disproved this belief and showed how nonscombroid fishes are the major causes of the disease. The Scombridae family is composed by yellow fin tuna, skipjack, bonito and mackerel; non scombroid fishes are the mahi-mahi (Coryphaena hippurus), bluefish, herring, sardines and anchovies (5). In recent years, some cases of intoxication have been reported after the consumption of mahimahi (dolphinfish) (6). Although scombroid poisoning is more common in nations with a warm water fishing industry, the illness occurs worldwide (7-10).
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Clinics

SFP is a chemical intoxication and symptoms occur within 10 minute to 2 h after consumption of pre-formed histamine in scombroid fish. The histamine that is released is not acquired from the marine environment, but is produced directly in the flesh of fish as a result of an action of enteric bacteria that decarboxylate the histidine. This process occurs when the fish is not properly refrigerated after the catch (11).

Symptoms are rash on the face, neck and upper chest, flushing, sweating, nausea, vomiting, diarrhoea, abdominal cramps, headache, dizziness, palpitations, oral burning sensation, metallic taste and hypotension. Symptoms usually resolve within 24 h. Although most cases do not seek medical attention, in some instances symptoms may be of sufficient severity to prompt cases to seek urgent medical attention, where antihistamine drugs may be used. Scombroid fish naturally contain high levels of histidine which is converted to toxic histamine as a result of histidine decarboxylase-producing bacteria if storage conditions are inadequate to control bacterial growth. Histamine is heat stable and survives subsequent processing, including canning, and ingestion of fish with histamine at levels in excess of 1000 ppm (100 mg/100 g) can result in illness. Bacterial spoilage and production of histamine may occur at any stage in the food chain (i.e. from landing the fish, at the processing plant or in the distribution system or in catering premises or homes) and adequate temperature control is the key in preventing bacterial growth and histamine formation. For control of fish belonging to the scombroid family the permissible level set by EU legislation for each batch of fish is an average histamine concentration lower than 100 ppm (10 mg/100 g) (12, 13).

Scombroid poisoning is not an allergic reaction and patients should be advised that they may eat similar types of fish in the future. The urticaria, flushing, and occasional bronchospasm have led to the term “pseudo-fish allergy” for scombroid poisoning, and victims are often incorrectly told that they are allergic to seafood. It is important for emergency physicians to be able to rapidly recognize the manifestations of scombroid poisoning so that proper therapy can be instituted. In addition, timely diagnosis of scombroid poisoning made by recognizing multiple patients with similar signs and symptoms or identification of an index case allows the most efficient utilization (13).

Scombroid poisoning is a clinical diagnosis made by association of the symptom complex with the ingestion of the types of fish listed above. Laboratory confirmation may be made by quantification of histamine levels in suspected fish. Normal fish has less than 10 ppm of histamine (1 mg per 100 grams of flesh). Illness is usually associated with 1,000 ppm (100 mg per 100 grams of flesh), but levels as low as 200 ppm (20 mg per 100 grams of flesh) may cause illness in susceptible individuals (14).

Treatment consists of administration of parenteral antihistamine therapy with diphenhydramine and/or cimetidine, IV fluids, and possibly steroid therapy (15).

Histidine decarboxylase and its role in accumulation of histamine in fishes

Some scombroid fishes such as tuna, bonito, and mackerel have histamine-producing bacteria (HPB) which are responsible of the intoxication (16, 17). HPB isolated from fish implicated in actual HFP incidents reported to date include Raoultella planticola, Morganella organii, Hafnia alvei, and Photobacterium phosphoreum (8, 18). Enteric bacteria such as R. planticola and M. organii are reported to be the dominant HPB in fish, whereas P. phosphoreum and Photobacterium damsela are frequently isolated from fish subjected to appropriate examination for marine bacteria, such as by the addition of NaCl to the culture medium and avoidance of exposure of the bacterium to high temperatures (18-20). It has been shown that accumulation of histamine by HPB occurs after the level of bacterial growth exceeds 107 CFU/ml in culture medium (21).

Histidine decarboxylase and its role in accumulation of histamine in fishes

Several cases of scombroid poisoning occurred in the world in the last years. Scombroid fish poisoning accounted for 4.5% of all cases of food poisoning reported to the Centers for Disease Control (25). Between 1973 and 1987, 116 outbreaks (757 cases) of scombroid fish poisoning were reported to the Centers for Disease Control. Of these, 109 (94%) involved mahimahi, tuna, or bluefish (26). No deaths have been reported.

Numerous reports were connected to the consumption of improperly stored fish from private noncommercial or recreational catches (16, 27). In 1998 in Pennsylvania a case of HFP had an interesting dynamic; in fact, the cause of the intoxication was related to the improper treatment of the fish in the early stages of fisheries, made using a method based on 60 miles long fishing-line and on a series of hooks (up to 3000). With this system the fish can remain hooked even 20 hours before being recovered. In this episode the water temperature was almost 28.5°C, and the only phase which was not granted by the HACCP procedure was the time between the hooking.
of the fish and landing; in all the other steps the cold chain has been respected. Between 1992 and 2004, 56 outbreaks of intoxication from scombroid fish were recorded by the Health Protection Agency in England and Wales (0-10 incidents per year) involving 296 people. The epidemiological analysis conducted between 1992 and 1999 on many cases of poisoning linked to marine organisms, showed that scombroid fish poisoning accounted for 32% of these cases and that intoxication is more frequent in the summer months. Recently there has been a steady increase in the number of reported incidents. Between December 2004, and August 2005, 24 SFP outbreaks and incidents (46 ill, 3 hospitalized) were reported to the HPA from several regions within England and Wales. Sixteen of the outbreaks occurred between December to June and the remaining eight occurred during the summer months up to August. A seasonal increase during the summer has been observed previously; however, the increase seen in the winter and spring months was unusual since outbreaks are more likely to occur during warmer weather after consumption of fish that has been improperly stored, handled and prepared (28, 29).

The outbreaks were associated with 21 catering premises (sandwich shops, restaurants, hotels and public houses) and three domestic setting, of which 23 were caused by consumption of tuna. Six of the catering premises were supplied with tuna from the same supplier in sealed foil vacuum packs that had low levels of histamine (<30 ppm), but remnants of tuna and prepared food (e.g. tuna mayonnaise) contained toxic levels (>3000 ppm). This suggests poor food handling and inadequate refrigeration during storage at these premises after the tuna packs were opened. In one of the other outbreaks, toxic histamine levels (>1970 ppm) were present in both sealed packs of raw and cooked tuna indicative of poor temperature control at some stage post-harvest, storage or transportation. Maintenance of microbiological quality from postharvest until the moment of consumption is essential if SFP associated with fish is to be avoided (28, 29).

Between 2005 and 2007 in Israel 21 cases of illness were registered, involving 46 people. Among these 30 needed medical interventions. Also in this case the tuna was the most responsible fish (30). In 1999, in Catania (Italy), 12 persons ate the same dish (tuna cooked at home), 7 (58%) presented symptoms related to scombroid syndrome and 1 of these showed severe symptoms. From 24 to 28 July 1996, two serious cases were registered in the hospital of Palermo (Italy). The first – a 23-year-old woman who ate roast tuna three hours before presenting to the emergency room – required the admission to the intensive care unit because of an acute pulmonary edema. The second – a 75-year-old man with a history of chronic obstructive bronchitis and ischemic heart disease, who ate fresh cooked tuna – required hospitalization in the coronary unit; his wife showed mild symptoms after eating the same dish (31). In January 2005 an extreme gravity case occurred, due to the consumption of tuna. The case was unusual for the severity of symptoms (the admission to the coronary care unit was necessary), but also for the high concentration of histamine found in the analyzed dish (> 7000 ppm). The intoxication can lead to an infarct syndrome, without the anatomical changes in myocardial and coronary arteries (confirmed by the negativity in the test to detect markers of myocardial necrosis and by coronary angiography). Electrocardiography showed “modest subendocardial damage”, due to the action of histamine as a vasoconstrictor and to the profound hypotension. During the diagnostic process infectious diseases were excluded: all the analyses for protozoa, helminths, Salmonella, Campylobacter, Shigella, were negative (32).

Scombroid syndrome and coronary vasospasm: new evidences from the literature

It was recently reported in the literature (2015) a case of Kounis syndrome with coronary vasospasm precipitated by scombroid fish poisoning (33).

Kounis syndrome is an acute coronary syndrome that includes acute myocardial infarction, coronary vasospasm, and stent thrombosis. It is associated with mast cell activation in allergic or hypersensitivity processes and may be initiated by histamine, the putative inflammatory mediator in scombroid poisoning (34).

The rare association between scombroid poisoning and coronary artery vasospasm has not been widely described. We are aware of one case report by Coppola et al. that illustrates an acute coronary syndrome due to vasospasm during an episode of this fish poisoning illness (35). A recent case report describes the history of a fit and well 30-year-old male which presented to the emergency department with widespread erythema, dizziness, profuse sweating, and chest tightness two hours after consuming cooked mackerel fish. He cooked fish that was caught on a fishing trip along the New South Wales south coast. The fish had not been refrigerated and was exposed to sunlight for a prolonged period. There was no prior history of fish allergy, other food allergy, or atopic illness. The patient did not develop facial, lip, or tongue swelling. In light of the chest tightness a 12-lead ECG was performed, showing sinus tachycardia with marked widespread ST segment depression and ST elevation in aVR. Serial high-sensitivity troponin assay changed from 8 to 80. The patient was treated with antihistamines, which was followed by complete resolution of the presenting symptoms. In particular, the chest tightness and global ST segment depression promptly resolved with glycerol trinitrate. Histamine is a potential stimulus of vasoconstriction and may precipitate coronary vasospasm through direct stimulation of vascular smooth muscle cells in the setting of endothelial dysfunction (36). In this case, the histamine derived from histidine within the fish is a likely precipitating factor for coronary vasospasm.

The presented case is unique in comparison to prior cases of scombroid illness because of the presence...
of ischaemic chest pain, the prompt resolution of chest pain with glyceryl trinitrate and profound ischaemic ECG changes.

Conclusions

The rapid cooling of the fish, and the strict maintenance of the cold chain until consumption are essential elements to prevent the degradation of the fish; the proliferation of bacteria, the histidine decarboxylase activation, the conversion of histidine to histamine and the development of an anaphylactoid reaction after the ingestion. Local Health Unit should post educational messages about the need for adequate refrigeration at docks and other locations where vessels are chartered, along with information on how to report occurrences of scombroid fish poisoning. If surveillance confirms substantial underreporting, the incidence of scombroidtotoxic disease may warrant a regulatory role for Local Health Unit in ensuring that chartered vessels have adequate refrigeration capability to engage in commercial recreational fishing. It’s important to remember that the inspection based on human sensory perception, such as fish appearance and odor, is of no value in detecting and controlling health risks. State should also provide advisory bulletins on scombroidtoxicism as an integral component of the licensing process for recreational fishers.

The diagnosis of scombroid fish poisoning is mainly based on clinical history, symptoms and epidemiological and it is fundamental a differential diagnosis with a classic allergic reaction. A prompt identification of the poisoning and the anti-histamines are essential if symptoms appear. Although this is a rather rare disease, clinicians should be able to recognize it, especially because recent studies have shown a connection between intoxication and the development of coronary vasospasm, as a result of a vasoconstrictor stimulus induced by the histamine.

References