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An unusual case of inferior acute myocardial infarction associated with advanced second grade atrio-ventricular block secondary to scombroid - fish poisoning.

Case Report

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Abstract: Background: Scombroid syndrome (histamine fish poisoning) includes symptoms and signs caused by biogenic amines, mainly due to histamine-containing food. Methods and results: In this report, we describe a 56 year old female who presented in the clinic with symptoms of scombroid syndrome after the ingestion of tuna fish, then gradually developed cardiovascular shock and inferior ST elevation myocardial infarction (STEMI) associated with advanced second grade atrio-ventricular block at the electrocardiogram (ECG) followed by respiratory arrest. Originally, the patient was treated with intravenous fluid infusion, steroids, ranitidine and chlorpheniramine. Following her cardiovascular shock and respiratory arrest, orotracheal intubation was performed and mechanical ventilation was started immediately. The patient was treated with dobutamine and fluid infusion, which has improved her hemodynamic conditions. Emergency cardiac catheterization was performed one hour after the onset of symptoms and coronary angiography did not show a significant coronary artery disease. The clinical picture has improved during the next days, with complete normalization of the ECG. Conclusion: Severe symptoms, including myocardial infarction. may occur in cases of scombroid syndrome.

Keywords: *Scombroid syndrome • Tuna fish intoxication • Atrio-ventricular block • ST elevation myocardial infarction • Cardiac shock*

1. Introduction

Scombroid syndrome (Histamine Fish Poisoning) comprises set of symptoms caused by biogenic amines, mainly the histamine contained in the food.

This disease state occurs generally upon consumption of tuna and other fish belonging to the Scombridae and Scomberesocidae families, such as mackerel, bonito and saury that contain high levels of free histidine in the muscles. However, many clinical cases of scombroid poisoning have also been reported with non-scombroid fish, such as bluefish, mahi-mahi, sardine, anchovy, herring and marlin [1-4]. Histamine and histamine-like substances are generated from histidine by a decarboxylase activity of bacteria such as Proteus, Hafnia aluei, Morganella morganii, Klebsiella pneumonia, Enterobacter, Escherichia coli, Serratia, Aerobacter and, more recently, Photobacterium phosphoreum, which have been isolated from the fish involved in scombroid poisoning incidents [5-8]. Some of these liable bacteria can also be present in the ordinary microbial flora of the fish; many of them derived from contamination that might happen during food processing, handling and/or improper refrigeration [8].

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Histaminosis symptoms occur up to few hours after the poisoning and resemble an allergic reaction [9]. The main clinical manifestations include the skin rash, urticaria, edema and localized inflammation, the gastrointestinal tract symptoms (nausea, vomiting, diarrhea), the haemodynamic changes (arterial hypotension) and alterations in neurological function (headache, palpitations, tingling, burning, itching [7]. The most frequent symptoms reported by Lavon et al., included rash, flushing, gastrointestinal complaints and headaches [10]. In previous clinical studies, there were similar symptoms described, although with a widespread incidence of diarrhea and skin rash [11-13]. While scombroid fish poisoning represents a mild illness that is usually resolved with the administration of antihistamine drugs, in severe cases serious complications may develop such as bronchospasm, cardiac and respiratory distress, in more susceptible individuals [14-18].

2. Case Report

A 56-year-old female with a previous history of arterial systemic hypertension but with no prior cardiac abnormalities, allergies, or other relevant diseases, arrived to the emergency department 30 minutes after eating cooked tuna fish. Ten minutes after ingestion, she experienced flushing, headache, nausea, dispnoea, vomiting and abdominal pain. Physical examination revealed diffuse macular blanching erythema all over the surface of her body, injected conjunctivae, signs of bronchospasm to auscultation of the thorax, tachycardia (100 beats per minute) and arterial systemic hypotension (85/60 mmHg). The first ECG revealed sinus tachycardia without ventricular repolarization abnormalities (Figure 1). The cardiovascular examination revealed normal heart sounds with no murmurs or rub upon auscultation. Neurological and abdominal examination was normal. The patient was initially treated with intravenous fluids (crystalloid solutions 1000 ml in 30 minutes followed by 80 ml/hour infusion), hydrocortisone (1000 mg), ranitidine (50 mg) and histamine H1 receptor antagonist, chlorpheniramine (10 mg); intravenous infusion resulting only in coetaneous rash resolution.

Once admitted to the Emergency Room, arterial blood gas (ABS) showed a pH = 7.098, an arterial oxygen pressure = 65 mmHg, an arterial carbon dioxide pressure = 52 mmHg, an oxygen saturation = 86%, lactate concentration = 3,6 mmol/L, and CHCO3 =–12.3 mmol/L. Gradually the cardiovascular status has worsened, with the emergence of severe arterial hypotension (70/40 mmHg) and cardiogenic shock signs associated with an advanced second grade atrio-ventricular block at the ECG (Figure 2) followed by respiratory arrest. Endotracheal intubation, mechanical ventilation, and inotropic therapy (dobutamine: 10 mcg/Kg//min) were performed. Echocardiogram showed akinesis of the left ventricular apex and of the inferior wall with an overall left ventricular systolic function being markedly impaired with an ejection fraction (EF) of 40%. The patient has also received intravenous atropine (1 mg) and epinephrine (0.5 mg sol. 1:1000), achieving a rapid resolution of the atrio-ventricular block at the ECG (Figure 3).

Since the clinical picture was consistent with inferior acute myocardial infarction, the patient was brought to the Cardiac Catheterization Laboratory, one hour after the symptom onset, to undergo catheterization. However, coronary angiogram showed rather normal coronary vessels, without atherosclerotic lesions or intravascular thrombosis.

Blood analysis revealed an increase in the creatine kinase MB fraction (plasma peak was 38,90 ng/ml; normal range: 0,1 – 4,5 ng/ml), a significant positive detection in troponin T (plasma peak was 278 ng/ml; normal range: 0,1–14 ng/ml), and a white blood cell count of 25320 per microliter with 88,3% of neutrophilis.

Gradually the hemodynamic, metabolic, and respiratory parameters improved. Within the fist day the ventilatory support was removed and following 26 hours of admission into the coronary care unit, dobutamine therapy was gradually reduced until no longer needed. The ECG revealed normalization of the elevated ST segment without inversion of T wave in the inferior leads on the second day (Figure 4). Four days after hospitalization, the echocardiogram showed complete normal left ventricular kinesis, with regression of the akinesis, previously evidenced, and an EF of 70%. At the fifth day of hospitalization the patient was discharged in good health.

3. Discussion

Scombroid-fish poisoning is the most common cause of itchthytoxicosis worldwide [19]. The pathogenesis of scombroid poisoning has not been clearly elucidated**;** it is generally associated with high histamine concentrations in bacterially contaminated fish, because histamine is heat stable and therefore not destroyed by different cooking methods [10].

Histamine toxicity is potentiated through inhibition of metabolizing enzymes , which detoxify histamine, and through the presence of putrescine and cadaverine [25]. Histamine, putrescine and cadaverine are formed postmortem in the muscular tissue of fish, through the action of certain microorganisms. Histamine interacts with

Figure 1. The ECG revealed a sinus tachycardia without ventricular re-polarization abnormalities

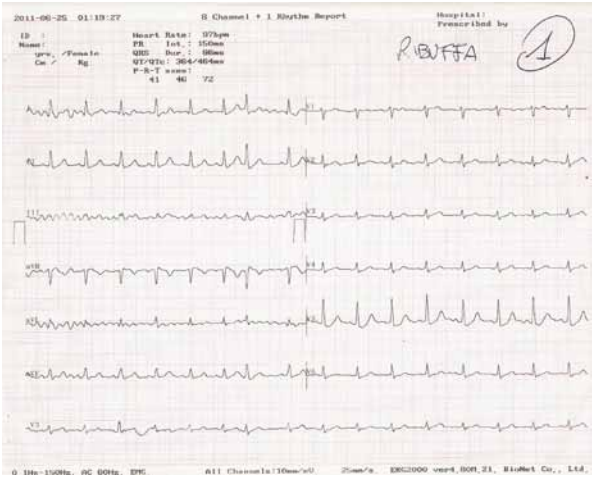


Figure 2. The ECG revealed a ST elevation in inferior leads with advanced second grade atrio-ventricular block.

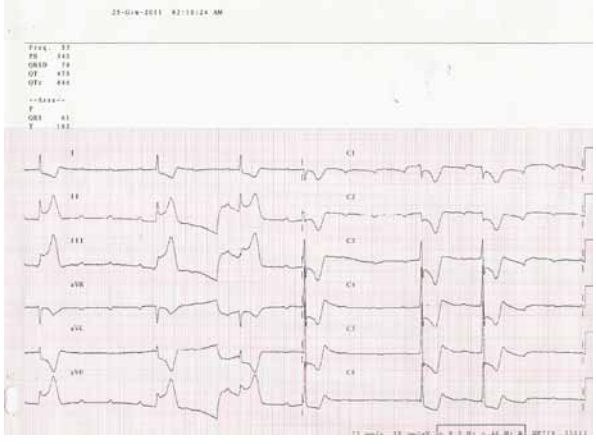


Figure 3. The AV block resolved after atropine and epinephrine injection.

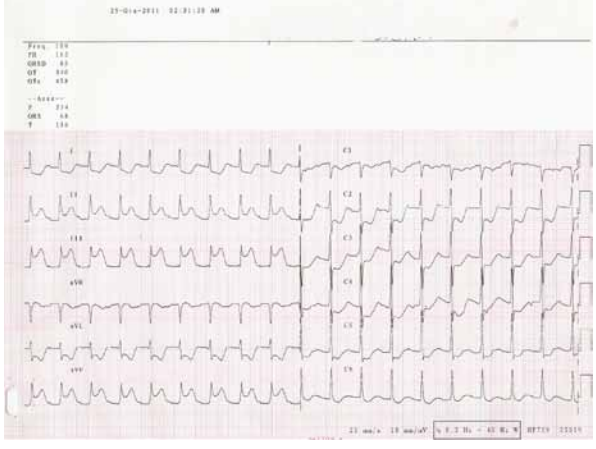
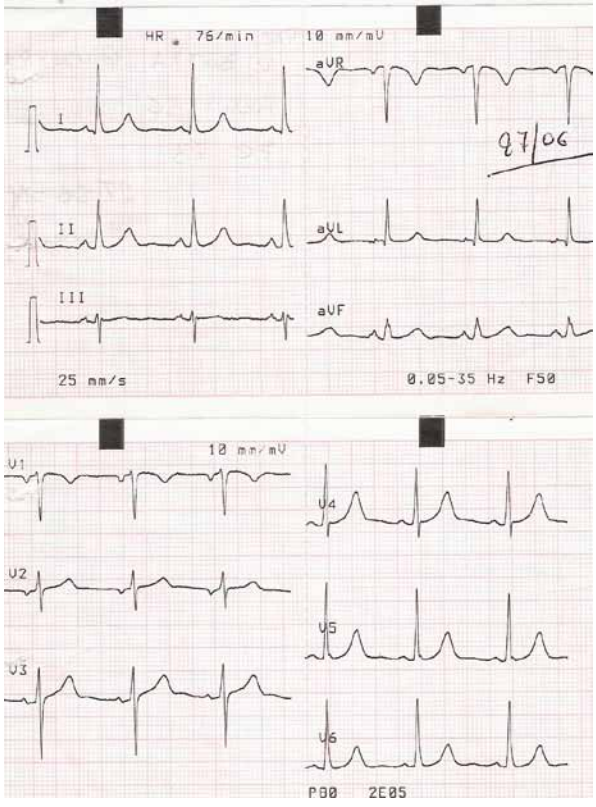


Figure 4. The ECG revealed normalization of the elevated ST segment without inversion of T wave in inferior leads on the second day.



several receptors and induces a variety of effects like clinical poisoning (histamine concentration of 20 mg/100 g is considered to be the threshold) or severe poisoning (histamine levels over 100 mg/100 g) [10,20]. The Food and Drug Administration allows maximum histamine levels of 5 mg/100 g of fish [21]. Due to its important role as a chemical mediator of inflammation, vasodilatation, increased vascular permeability, decreased peripheral resistance, airway smooth muscles contraction, gastric acid secretion and induction of pain and itching through sensory nerves stimulation, histamine levels should be tightly controlled. Acting at H1 – H2 receptors, histamine induces the vascular endothelium to release nitric oxide, leading to vasodilation, erythema, increased vascular permeability and edema [22]. Vasodilatation and reduced peripheral resistance may contribute to a significant fall in blood pressure [23]. Histamine can cause, directly or indirectly coronary spasm, which is proposed as the main underlying mechanism of allergy-induced coronary syndromes [15-17]. Profound drop in contractility and disrhythmias such as sinus tachycardia or idioventricular rhythm during exposure to an allergen, are in proportion to the amount of released histamine [24]. The ectopic and sinoatrial node automatism stimulation is H2 receptor dependent, whereas conductance

disturbances are H1 receptor mediated [25]. Histamine effect on H1 receptor could explain the atrio-ventricular block that was observed in our case. Just as during the course of anaphylactic reaction, myocardial ischemia may result also from the instability in circulatory system (drop in coronary perfusion pressure) and from the pathophysiology due to disturbances, shock, or other etiology [24].

Scombroid poisoning can nevertheless mimic other conditions. A correct diagnosis is deemed important, since prompt identification and reporting will assist officials in preventing others from becoming ill. There are a variety of laboratory methods developed to test the levels of histamine in fish and fish products. In contrast to many other more potent seafood toxins, the relatively high action levels established for histamine in fish, has allowed for the detection of histamine levels and its use, involving a variety of different approaches. These range from simple and inexpensive thin layer chromatography procedures to labor-intensive and more powerful liquid chromatography coupled with mass spectrometric detection methods [26]. Unfortunately, in here reported case, it was not possible to perform the tests using tuna fish eaten by the patient.

People who present to an emergency department with acute symptoms suggestive of allergy should also be questioned about consumption of dark-fleshed fish in the hours before onset of symptoms. The diagnosis is based on taking a good food history and ruling out other causes of the usually dramatic symptoms. However, measurement of plasma histamine concentration is not widely available in clinical laboratories. Treatment is supportive nevertheless and use of antihistamines and in some case corticosteroids may relieve the symptoms, although they may resolve within few hours, even without specific treatment. Major scombroid poisoning can induce severe hypotension requiring intravenous fluids and inotropic support with dobutamine and/or dopamine infusion and intravenous epinephrine [27]. In severe cases**,** urticaria and bronchospasm may occur, but death is unusual [28]. Scombroid poisoning can be prevented by refrigerating the dark-fleshed fish (fresh or canned fish that has been opened) and by ensuring that such fish is not consumed if it has been kept opened for several days. Since the toxins that cause scombroid poisoning are heat stable [29], cooking of contaminated fish will not eliminate them nor reduce the risk of illness.

4. Conclusion

Scombroid syndrome may occasionally be very severe and trigger significant cardiovascular symptoms. In this case, the patient required intensive care in order to hasten a good outcome. Our report serves to highlight a rare case of one severe scombroid syndrome triggering acute coronary syndrome complicated by atrio-ventricular block and cardiogenic shock requiring mechanical respiratory assistance and continuous intravenous inotropic with clinical resolution only several days later.

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