

Scombroid fish poisoning illness and coronary artery vasospasm

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CASE STUDY

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ABSTRACT

We present an interesting case of a young man with coronary artery vasospasm complicating scombroid fish poisoning illness. The initial presentation included tachycardia and significant hypotension. A 12-lead ECG showed sinus tachycardia with marked widespread ST segment depression and ST elevation in aVR. Symptoms subsequently improved with intravenous fluid rehydration, antihistamines, and glycerol trinitrate. The underlying pathogenesis and treatment of this rarely described manifestation of the fish poisoning illness is discussed.

Key Words

Coronary artery vasospasm, scombroid fish poisoning

Implications for Practice:

1. What is known about this subject?

There is very limited literature describing the association between coronary vasospasm and scombroid fish poisoning illness, with only two other case reports describing this association.

2. What new information is offered in this case study?

This case provides new literature to support the capacity of scombroid fish poisoning illness to induce coronary artery vasospasm mediated by histamine.

3. What are the implications for research, policy, or practice?

This is a rarely reported association; nevertheless, clinicians should be aware of this when assessing and treating patients with the fish poisoning illness because of its potential fatality. This case provides the Australian fishing industry with awareness of a potentially life-threatening manifestation of scombroid poisoning, and the justification to ensure the implementation of safe fishing standards.

Background

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.¹ There has been a recent increase in the awareness of scombroid fish poisoning illness following the death of an Australian mother and daughter in Bali that was thought to be a result of this illness,² although other factors such as asthma may also have contributed to their deaths. This case is presented to illustrate how scombroid fish poisoning^{3,4} illness is capable of inducing the Kounis syndrome, and in particular vasospastic allergic angina.

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.³ that illustrates an acute coronary syndrome due to vasospasm during an episode of this fish poisoning illness. In a series of 71 cases of scombroid poisoning, coronary spasm was not described.⁵ However, in the single case report,³ the patient who suffered scombroid illness and coronary artery spasm in the right coronary artery also had significant coronary artery disease in the left anterior descending artery requiring percutaneous coronary intervention.

The case study by Lionte⁴ revealed three patients with widespread ST segment depression in the setting of scombroid illness and significant hypotension; however, patients described only palpitations with no cardiac ischaemic chest pain or dyspnea. It is possible that coronary vasospasm was involved, however, circulatory failure with coronary hypoperfusion may also have been a mechanism for the ST segment changes. Furthermore, no echocardiography was performed to exclude other pathology such as Takotsubo cardiomyopathy.

Case details

A previously fit and well 30-year-old male presented to the emergency department with widespread erythema, dizziness, profuse sweating, and chest tightness two hours after consuming cooked mackerel fish. The cooked fish 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.

The initial presentation was associated with tachycardia and significant hypotension, which subsequently improved with intravenous fluid rehydration. 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 (Figure 1).

Serial high-sensitivity troponin assay changed from eight 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 (Figure 2).

Serum tryptase level was normal (8.2mcg/L, reference range 0-11mcg/L) with a negative serum allergy test to seafood mix. A transthoracic echocardiogram was performed after resolution of chest tightness and ECG changes; this demonstrated normal left ventricular systolic function, no valvular heart disease, and no pericardial effusion.

In the context of clinical improvement, the patient declined to undergo either invasive coronary angiography or CT coronary angiography for exclusion of significant coronary artery disease. However, the patient agreed to an exercise ECG treadmill test. The patient exercised for 14mins, and was limited by fatigue; at peak exercise myocardial ischaemia was not produced (Figure 3). The patient subsequently discharged himself from the hospital.

Discussion

Given the clinical history, examination, initial biochemical, and immunological tests, the likely diagnosis for the patient described in the case is scombroid fish poisoning.

Mechanism

Scombroid fish poisoning is due to consumption of improperly refrigerated fish belonging to the *Scrombidae* family (mackerel, bluefin and yellowfin tuna, bonito, skipjack), as in the described case. These fish contain histidine within muscle tissue. Bacteria within the fish contain an enzyme histidine decarboxylase, which can convert histidine to histamine at temperatures of 20–30°C.⁶ Implicated bacteria include *Pseudomonas*, *Klebsiella*, *Enterobacter*, *Escherichia*, or *Clostridium*; the bacteria may be either normal flora or contaminants from unhygienic food handling. In the case described, the caught fish were not refrigerated and exposed to sunlight for a prolonged period, creating ideal conditions for histamine formation.

Aside from histamine, other agents derived from the fish that have also been postulated to induce a scombroid reaction include bioamines. These may be considered as potentiators because they may inhibit the catabolism of histamine or increase the amount of histamine absorbed by the gastrointestinal tract.⁷ Examples of these agents include cadaverine and putrescine.

Coronary vasospasm is a sudden and intense vasoconstriction of a major epicardial coronary artery that leads to near or complete occlusion of the vessel. Coronary vasospasm requires the interaction⁸ of:

1. Localised or diffuse abnormality of a coronary artery (including endothelial dysfunction, vascular smooth muscle cell hyper-reactivity), rendering it hyperactive to vasoconstrictor stimuli; and
2. Vasoconstrictor stimulus capable of inducing spasm in a segment of hyperactive coronary artery.

Histamine, a potential stimulus of vasoconstriction, may precipitate coronary vasospasm through direct stimulation of vascular smooth muscle cells in the setting of endothelial dysfunction.⁹ In this case, the histamine derived from histidine within the fish is a likely precipitating factor for coronary vasospasm.

This fish poisoning illness is also associated with self-limiting symptoms of skin flushing, pruritus, throbbing headache, dizziness, nausea, vomiting, abdominal cramps, and diarrhoea.⁶ Food allergy is less likely as a cause in the case

described given the rapid resolution of symptoms, the consumption of fish in the *Scrombidae* family, absence of prior fish food allergy, presentation with generalised erythema rather than discrete urticarial wheals, normal initial tryptase level, and negative serum allergy test to seafood mix. The illness would be further confirmed by the detection of high levels of histamine in the fish; in this case such investigations were not possible.

The case we have presented is unique in comparison to prior cases of scombroid illness and associated ischaemic ECG changes for the following reasons:

1. Presence of ischaemic chest pain; and
2. Prompt resolution of chest pain and profound ischaemic ECG changes with glyceryl trinitrate.

Thus, our case is perhaps more specific for the development of coronary vasospasm in the setting of scombroid fish poisoning illness.

Management

Scombroid poisoning is a self-limiting illness that lasts a few hours. Treatment is supportive, including the use of antihistamines, antipyretics, and intravenous fluid rehydration.⁹ Specifically, H₁ receptor antagonists are recommended in the literature for treatment of the illness.⁷

The treatment of the acute angina episode related to coronary vasospasm includes sublingual or intravenous nitroglycerine.¹⁰ Given the presence of an acute resolving vasoconstricting factor, agents such as calcium channel antagonists and nitrates would not necessarily be required for long-term prevention of coronary vasospasm. Lifestyle factors associated with a risk of comorbidity, including smoking cessation should be advised.

The presented case is important as it highlights a life-threatening cardiac manifestation of scombroid fish poisoning illness. It is also important for the Australian fishing industry to promote correct fish handling in their process of safeguarding fishing industry standards and thereby increasing awareness of the industry standards for best practice.

Conclusion

In summary, we present a rare case of Kounis syndrome with coronary vasospasm precipitated by scombroid poisoning. This is a rarely reported association and clinicians

should be aware of this when assessing and treating patients who have the fish poisoning illness.

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PEER REVIEW

Not commissioned. Externally peer reviewed.

CONFLICTS OF INTEREST

The authors declare that they have no competing interests.

PATIENT CONSENT

The authors, Anastasius M, Yiannikas J, declare that:

1. They have obtained written, informed consent for the publication of the details relating to the patient(s) in this report.
2. All possible steps have been taken to safeguard the identity of the patient(s).
3. This submission is compliant with the requirements of local research ethics committees.

Figure 1: 12-lead ECG taken from 30-year-old old male at the time of chest tightness

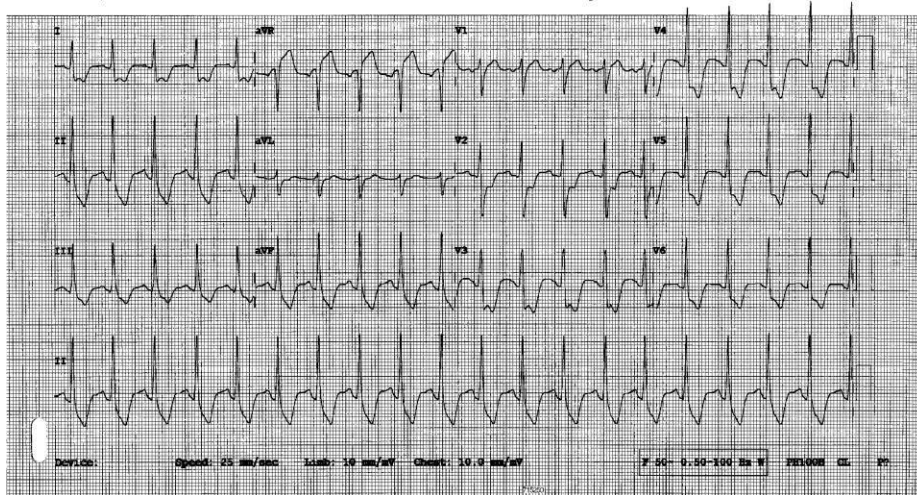


Figure 2: 12-lead ECG taken from 30-year-old male following resolution of chest tightness

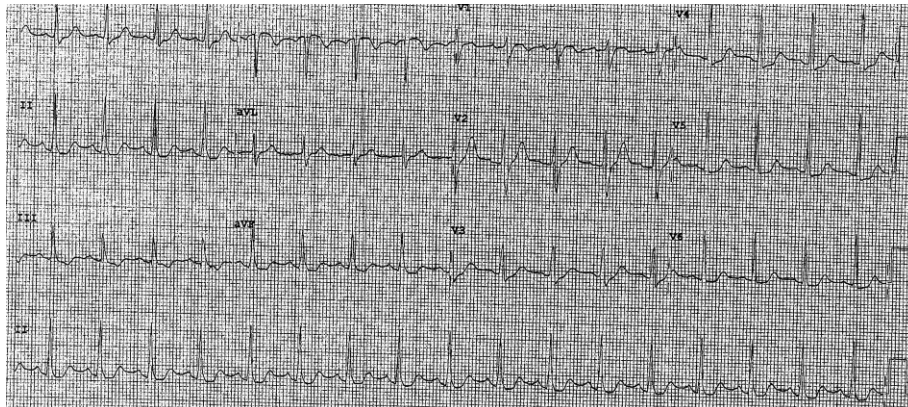


Figure 3: Exercise ECG performed to submaximal workload

