CASE REPORTS

Trouble with tuna: two cases of scombrototoxin poisoning

Ian M Stell

Abstract

Two cases of scombrototoxin poisoning after exposure to tuna are described, the second being unusual in that tinned, rather than fresh, tuna was involved. This relatively rare condition needs to be distinguished from an allergic reaction, for which it can easily be mistaken.

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Keywords: scombrototoxin poisoning; tuna fish.

Scombrototoxin poisoning occurs when certain fish, particularly members of the scombroid family, undergo bacterial spoilage, with the production of large quantities of histamine. About 50 episodes are reported annually in the United Kingdom.¹ Small outbreaks involving up to five people are not uncommon.

Case reports

CASE 1

A 45 year old male presented with a three hour history of face and throat swelling, flushing, headache, and diarrhoea. The symptoms began 45 minutes after eating cooked fresh tuna at a wine bar, and settled spontaneously over a few hours. The next day samples from the same fish were obtained; of the three portions sold, two were known to have caused illness, including this case. The samples had levels of histamine > 200 mg/100 g of fish (normal safe limit 5 mg/100 g, potentially toxic from 50 mg/100 g).

CASE 2

A 31 year old male presented with a sudden onset of a red rash involving the face and upper body. It began an hour after eating tuna mayonnaise sandwiches. The six other staff in his office had also eaten the same sandwiches and became ill shortly afterwards, with symptoms including headache, flushing, rash, and diarrhoea. All symptoms settled within a few hours of onset. Samples of the tuna mayonnaise were obtained the same afternoon; these showed levels of histamine of > 250 mg/100 g. The tin which had contained the tuna had been opened about 6 am in the morning, the tuna mixed with mayonnaise, and the sandwiches made and sold at about 1 pm.

Discussion

Scombrototoxin poisoning is also referred to as histamine poisoning because of the central role that histamine plays in the condition.² How-

ever, questions still remain as to whether it is the histamine alone which is responsible for the condition.³

The commonest fish implicated in outbreaks in the United Kingdom are tuna and mackerel, members of the scombroid family. However, non-scombroid fish—herrings, pilchards, sardines, and anchovies—have also been responsible. Scombroid fish have red meat, and very high levels of free histidine are found in the muscle. The free histidine is thought to act as a buffer to the large amounts of acidity produced by short bursts of activity (personal communication, professor I Johnston, Gatty Marine Laboratory). The imidazole side chain in histidine has a pKa close to neutral pH, making it an effective buffer.

This free histidine can undergo metabolism to histamine by bacteria which contain histidine decarboxylase. This enzyme is not widespread, and only some strains of certain bacteria contain it. These bacteria either contaminate the fish meat from the gut, or from handlers, or the general environment. Fish that have been affected by these bacteria look and smell normal; however, they may have a peppery or hot taste. Histamine is stable in storage and to heat. The conversion of histidine to histamine can occur at varying temperatures, depending upon the bacteria involved, but will generally not occur with storage below 4°C. Nearly all cases of scombrototoxin poisoning relate to fish contaminated after catching or in processing. However, contamination of tinned tuna after opening can occur in the right conditions, as appears to have happened in our second case, where high levels of histamine were reached within seven hours of a tin of tuna being opened. It could be argued that this histamine was already present at the time the fish was canned, but as all the other tins tested from the same batch were free of histamine, this is unlikely. One possible, although purely speculative, explanation for the histamine production is the mixture of the tuna with mayonnaise. Mayonnaise can contain lactobacilli, some strains of which produce histidine decarboxylase; had these bacteria been present in this mayonnaise/tuna mixture, this might explain the histamine production.

Although a few other cases of such “post-processing contamination” have been recorded, they are unusual; in cases arising from

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Tuna fish, a member of the scombroid family.

canned fish, poor handling from catching to canning is more usually the problem.⁴

Symptoms may begin in the mouth almost immediately, with numbness, tingling, or burning. This may be followed within minutes to a few hours by headache, a red rash over the face, neck and upper trunk, nausea, and diarrhoea. Complete recovery within 12 hours is usual. Antihistamines lead to a rapid resolution of symptoms: H₁ receptor antagonists such as chlorpheniramine are effective, but H₂ receptor antagonists such as cimetidine may be better.⁵

The exact mechanism whereby histamine reaches the circulation in scombrototoxin poisoning is not known as pure histamine given by

mouth is rapidly metabolised by gut enzymes. If, as is usually assumed, the histamine in the fish is responsible for the symptoms, then some other factor must inhibit its metabolism in the gut, perhaps other amines. Another suggested mechanism is that some other toxin in the fish is absorbed from the gut and causes endogenous histamine release, perhaps from basophils.

Cases of scombrototoxin poisoning have been reported throughout the world, but are commonest in the United Kingdom, the USA, and Japan.

CONCLUSION

Scombrototoxin poisoning should be considered in any patient who suffers the typical symptoms after eating fish. It may be that many episodes are ascribed to food sensitivity, and the reported numbers may not reflect the true incidence of this condition.

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Non-operative management of a splenic laceration in a patient with the Proteus syndrome

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Abstract

An adult patient with the Proteus syndrome sustained a grade III splenic laceration after falling off a horse. Clinical features of this rare disorder include subcutaneous and visceral hamartomatous tumours. The patient also suffered from chronic intravascular coagulation associated with extensive haemangiomatosis (Kasabach-Merritt syndrome). Considering the visceral anomalies and abnormal coagulation, a non-operative approach was preferred despite considerable transfusion requirement, and the patient successfully underwent embolisation of the splenic artery. This is the first reported case of splenic injury in a patient with Proteus syndrome.

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Keywords: splenic injury; embolisation; Kasabach-Merritt syndrome; Proteus syndrome.

The spleen is the most frequently injured intra-abdominal organ following blunt abdominal trauma. In recent years, a less aggressive approach toward splenic laceration is accepted as being as safe as urgent splenectomy in selected cases. Attempts have been made to identify and test criteria for safe non-surgical management of splenic injuries. We present an adult patient with the Proteus syndrome and associated coagulation abnormalities due to large subcutaneous hamartomas (Kasabach-Merritt syndrome) who sustained a laceration of the spleen following blunt injury.

Case report

A 23 year old male patient with the Proteus syndrome since childhood fell off a horse. He did not immediately seek medical attention. The morning after, however, he woke up with severe upper abdominal pain and fainting, and was admitted to the emergency department.

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