Mad honey poisoning-related asystole

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Mad honey poisoning is well known in the eastern Black Sea region of Turkey. The cause of the poisoning is the toxin grayanotoxin, found in honey obtained from the nectar of *Rhododendron* species on the mountains in the region. A 60-year-old man was brought to the emergency department with dizziness and syncope after eating a few spoonfuls of honey. While the patient was being treated, bradycardia and asystole developed. The patient was given 0.5 mg of atropine, and asystole began and ended. The patient was transferred to the catheter laboratory and a temporary pacemaker was implanted. Mad honey poisoning related asystole has not been previously reported, and the rapid response to atropine is significant.

Mad honey poisoning is a well-known condition in the Black Sea region of Turkey. The cause of the poisoning is the toxin known as grayanotoxin found in honey obtained from the nectar of rhododendron species growing naturally on the mountains in the region. This toxin is a polyhydroxylated cyclic hydrocarbon that does not contain nitrogen.[1 2] Local people are able to distinguish this honey from other varieties. It causes a sharp burning sensation in the throat and thus is also referred to as ''bitter honey''. This form of poisoning has been known since 401 BC; Xenophon refers to soldiers being poisoned by honey in his ''Anabasis''. To date more than 120 cases resulting from ingestion of this honey in this region have been reported[1 2] Mad honey has the potential to cause death if left untreated, although no fatal cases have been reported in the literature. Complete recovery after admission to hospital is normally the rule, because the resulting hypotension usually responds to appropriate fluids, and bradycardia and conduction defects usually respond to atropine treatment. This case report describes a case of honey poisoning related sinus arrest and the patient's response to atropine.

CASE REPORT

A 60-year-old man was brought to the emergency department with dizziness and syncope that arose after eating a few spoonfuls of honey. The patient had no cardiac problem other than chronic hypertension, which was being treated with an angiotensin receptor blocker, a nitrate and indapamide. At examination on admission to the emergency department his blood pressure was 160/60 mm Hg and pulse rate was 75 beats/min. Neurological examination was normal. Bradycardia suddenly developed and asystole (fig 1) increased while a 12 lead electrocardiogram was being administered, and the patient lost consciousness. He was given 0.5 mg of atropine intravenously at the onset of bradycardia, and asystole began and ended, at which point consciousness returned. The patient was transferred to the catheter laboratory where a temporary pacemaker was fitted, and he was admitted to the coronary intensive care unit. During observation, normal sinus rhythm returned that same day, and the temporary pacemaker was removed on the third day. The patient was discharged on the fourth day.

DISCUSSION

Bee production is a widespread activity in the Black Sea region. Local Caucasian bees (*Apis mellifera caucasia*) produce honey from natural flora within a 5 km^2 area. It is thought that of the various rhododendron species widespread in the local regional flora, *Rhododendron luteum* and *Rhododendron ponticum* are the main sources of grayanotoxin.[3] Since these plant species grow in forested areas at specific altitudes and in specific valleys the local inhabitants know which honey produced in which region is mad honey.

There are some 700 different rhododendron species in the area comprising China, Tibet, Myanmar, Assam and Nepal, nearly 300 species in New Guinea, many in Japan, and others in tropical Asia from Indochina to Indonesia and the Philippines, while a small number occur in Europe and North America. Purple-flowered (*R ponticum*) and yellow-flowered (*R luteum*) rhododendrons are widespread along the Black Sea coast in northern Turkey.[3 4] The former is also known as ''black poison'' and the latter as ''yellow poison''.[4]

The grayanotoxins bind to the sodium channels in cell membranes. These compounds cause activation or inactivation of the channels; excitable cells are thus maintained in a state of depolarisation, during which entry of calcium into the cells may be facilitated.[1–3]

Since cases of mild poisoning are well-known to the local inhabitants, they do not generally visit health institutions when affected. However, patients with severe symptoms do go to hospital, and patients whose heartbeat and vital signs improve are discharged after 3–6 h.[1]

Although no case of asystole has previously been reported, well-known toxic effects of honey poisoning include bradycardia, cardiac arrhythmia, hypotension, nausea, vomiting, sweating, salivation, dizziness, weakness, loss of consciousness, fainting, blurred vision, chills, cyanosis and convulsions. Poisoning is frequently seen in this region,[1] and patients generally recover spontaneously without having to go to hospital. The majority of those who do seek medical care recover with administration of 0.5–1.0 mg atropine and controlled fluid replacement. In rare, life-threatening cases, cardiac arrhythmias requiring cardiac pacing may occur.

Although cases of honey poisoning related bradyarrhythmia have previously been reported, asystole has not, and the response of atropine to asystole caused by mad honey in this case is important.

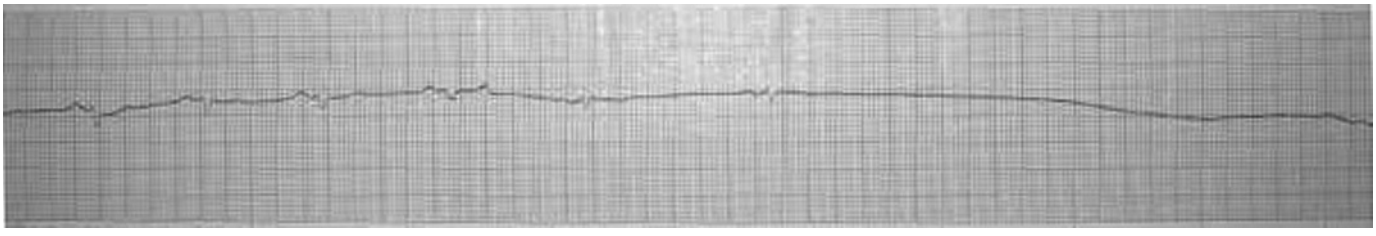

Figure 1 Electrocardiogram showing sinus bradycardia and asystole.

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Images in emergency medicine

Haemoptysis from the pulmonary artery

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A 72-year-old woman presented because of cough with fresh blood for 3 days. She had a history of aortic dissection and underwent aortic arch reconstruction 12 years earlier. On arrival, her vital signs included blood pressure of 150/78 mm Hg and respiratory rate of 24 breathes/min. Physical examination revealed rales over the left hemithorax. Laboratory results included haemoglobin of 10.9 g/dl and platelet count of 113 000/μl. An oblique coronal reformatted image was performed by multislice computed tomography (MSCT) and demonstrated non-tapering distal branches of the left pulmonary artery with an adjacent area of ground-glass attenuation, indicating the culprit lesions (asterisk). The patient was treated conservatively and recovered uneventfully.

Most cases of haemoptysis (90%) originate from the bronchial circulation. MSCT angiography with a combination of multiplanar reformatted images can help identify the origins and courses of arteries that may be responsible for bleeding. Effective trans-arterial embolisation requires such knowledge, particularly for differentiating pulmonary, bronchial or non-bronchial systemic feeder vessels.[1]

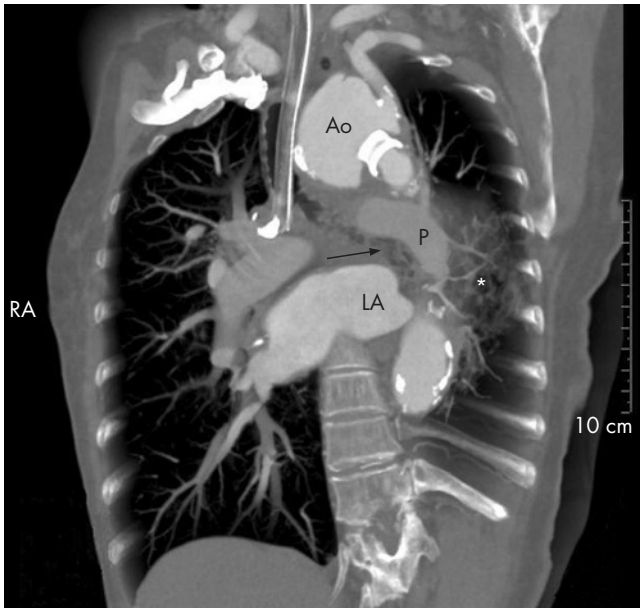

Figure 1 An oblique coronal maximum intensity projection reformatted image, obtained with 64 multislice computed tomography, demonstrating non-tapering distal branches of the left pulmonary artery with an adjacent area of ground-glass attenuation, and indicating the culprit lesion (asterisk: the highest attenuation was 95 HU). Arrow, left main bronchus; Ao, aortic arch; LA, left atrium; P, left pulmonary artery.

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