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A Case of Mad Honey Poisoning Presenting with Convulsion: Intoxication Instead of Alternative Therapy

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An unusual type of food poisoning, commonly seen in the eastern Black Sea region, is caused by toxic honey (1,2). Grayanotoxins, which are extracted by bees from the leaves and flowers of rhododendron species, are the main compounds responsible for this poisoning (3,4). The honey is well known and is named 'mad honey' by local people.

Here we report a pediatric case of honey intoxication, in which the parents were aware that the honey was 'mad honey'. Regarding this patient, we discuss the reasons for common 'voluntary' ingestion of this well-known toxic product in terms of socio-cultural factors.

Case Report

An eight-year-old previously healthy boy was admitted to our hospital with suddenly developed nausea, vomiting, weakness, dizziness and impaired consciousness. There was no history of trauma or drug ingestion. The symptoms began within 1 h of drinking three spoons of honey in the form of sherbets for his abdominal pain. He was sweating diffusely. Body temperature was 36.4 OC, heart rate 45 beats/min, and arterial blood pressure 85/45 mmHg. All routine biochemical tests were within normal limits. The electrocardiogram showed sinus bradycardia. Parenteral fluid was administered, and atropine and dopamine were given for symptomatic treatment of hypotension and bradycardia. Half an hour after admission he had a generalised tonic and clonic seizure lasting two minutes. Seven hours after admission he began to regain consciousness. He had no history of convulsion and an electroencephalogram taken 12 h later showed no abnormality. He was discharged at the end of the first day.

An unusual type of food poisoning seen in the eastern Black Sea region is caused by honey containing grayanotoxin (1-4). Although 18 forms of grayanotoxins have been isolated from the leaves and flowers of the rhododendron and some other plants, grayanotoxin I is responsible for honey poisoning (3,4). Of the seven endemic kinds of rhododendron in Turkey, Rhododendron luteum and Rhododendron ponticum are the species growing in the eastern Black Sea mountains (5). Toxic substances are obtained by bees from these plants and incorporated into their honey.

The well-known toxic effects of honey poisoning are bradycardia, cardiac arrhythmia, hypotension, nausea, vomiting, sweating, salivation, dizziness, weakness, loss of consciousness, fainting, blurred vision, chills and cyanosis. Cardiac disturbances are the main signs in this poisoning. Commonly reported cardiac findings are arrhythmias such as bradycardia, atrioventricular block and sinus bradycardia (1,2,4,6). Onat et al. showed that atropine sulphate alleviated bradycardia due to grayanotoxin, and AF-DX 116, a selective M2-muscarinic receptor antagonist, restored heart rate. They suggested that M2-muscarinic receptors were involved in the cardiotoxicity of grayanotoxin (7).

To the best of our knowledge the generalised convulsion noted in our case has not previously been reported in humans. Animal studies showed that grayanotoxin I increased the membrane permeability to Na+ in Na+ dependent excitable membranes and maintained those cells in a state of depolarisation. In addition to peripheral excitable cells, it also played a role in central nerve cells (8,9). The centrally acting properties of grayanotoxin I may explain the convulsion noted in our case.

In general the severity of the honey poisoning depends on the amount ingested. Fatal cases of honey poisoning are extremely rare. Administration of parenteral fluid, and symptomatic treatment are sufficient. Since grayanotoxins are metabolised and excreted rapidly the patients regain consciousness and feel better within hours and heart rate and blood pressure usually return to normal within 24 h (1,2). Honey poisoning should be kept in mind in any patients admitted with unexplained hypotension, bradycardia and other rhythm disturbances, and patients eating honey from the Black Sea region must be examined carefully.

Beekeeping is a common and honourable work among the local people of the eastern Black Sea area. The native Caucasian bees, which can fly only about five kilometres square, are used in the traditional method of honey production. Therefore each honey contains one valley's flora. Since rhododendrons are long-living plants beekeepers know which honey contains toxic substances; in other words they know which honey is 'mad'. Honey produced in springtime is more toxic and sometimes contains higher concentrations of grayanotoxin than that produced in other seasons (10). Since 'mad honey' is used for alternative therapy, it is considered very valuable, especially when produced from some special valleys. It is thought to be helpful in healing some gastrointestinal diseases such as gastritis and peptic ulcer. Sütlüpinar et al. (4) noted this reason for toxic honey ingestion in their 11 patients. Additionally it is believed that mad honey reduces coronary heart diseases and increases life expectancy when used continuously. Since grayanotoxins decrease blood pressure this belief should not be dismissed completely as blind faith. The reasons mentioned above explain why grayanotoxin intoxication cases do not decrease in spite of awareness about mad honey. We think that a warning label containing the ingredient flora and a consumption guide on the honey jar may be helpful for avoiding intoxication.

To the best of our knowledge the generalized convulsion noted in our case has not previously been reported. Therefore we wanted to share our experience and call attention to the wide use of honey in alternative therapy.

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