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CASE REPORT

Nearly Fatal Wild Honey Intoxication A Case Report of Seven Cases

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ABSTRACT

Honey is used as a common breakfast item all over the world. Honey is also used in cooking and baking, is used as a spread on breads and is added to beverages such as tea, or as a sweetener in commercial beverages.

In Nepal, there is a fancy for the use of wild honey. Honey is also used in weight reduction. Studies reported elsewhere pinpoint that wild honey is poisonous. This poisoning is well known from ancient times, right from the time of Xenophon. This wild honey is derived from the flowers of *Rhododendrons* (the national flower of Nepal). Honey consumption toxicity was noted in a few medical students who were treated and the current paper describes the outcome and the cause of toxicity.

Key Words: ICCU Intensive care unit, NMCTh. Nepal Medical College teaching hospital, GT. graynotoxin

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Introduction

Honey is a sweet aliment produced by honey bees [1] and is derived from the nectar of flowers. According to the United States National Honey Board, "honey is a pure product that is not contaminated with any other substance. [2] Most micro-organisms do not grow in honey due to a low water activity of 0.6[3] except dormant endospores of the bacterium *Clostridium botulinum*, which can be dangerous and may even cause death [4].

For at least 2700 years, honey has been used by humans to treat a variety of ailments through topical application. Recently, honey has been found to have some antiseptic and antibacterial

properties. Wild honey toxicity symptoms include dizziness, weakness, excessive perspiration, nausea, and vomiting. Less commonly, low blood pressure, shock, heart rhythm irregularities and convulsions may occur, with rare cases resulting in death if consumed in large quantities.

Supportive therapies are the principal management strategies. Morphological identification of the poisonous plant is helpful. Laboratory studies can sometimes determine the toxins. The current report is a series of cases of grayanotoxin poisoning after the ingestion of wild honey derived from the flowers of *Rhododendron simsii*. The diagnosis was established by the typical clinical features and the detection of the toxin in the specimen of wild honey consumed by doctors and medical students, which was procured by the first author after examining the cases in the hospital ICCU.

Case Report Of Wild Honey Intoxication

In this series, seven cases of medical students (age 20-25 years, males) are reported. All of them went to the same restaurant, nearly on an empty stomach for dinner. In spite of the warning from the owner, they consumed large

varying amounts of wild honey [10 tablespoons full to 2-3 tablespoons full] before consuming their dinner. The one who consumed the largest amount of honey could eat only half the meal before gastro-intestinal (GI) symptoms developed. In the rest of the students, there was a latent period of 15-20 min. before G.I. symptoms developed. One of the authors consumed 1 tablespoon full of the same sample of honey later, but he developed no symptoms (included in Table 1.)

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History Of Present Illness

Except one case, all of them developed symptoms after 15-20 minutes of wild honey consumption. All of them developed a burning sensation in the throat, excessive salivation, nausea and vomiting. These were followed by eye symptoms of diplopia, blurring of vision, dilated pupils and a sluggish reaction to light and the cardio-vascular symptoms of palpitation and hypotension. Lastly, symptoms of CNS developed in the form of unconsciousness, deep gasping respiration, respiratory depression and apnea. The severity of the symptoms depended upon the quantity of wild honey consumed. The detailed symptomatology is given in [Table/Fig 1.

(Table/Figure 1) Systemic Manifestations of *Grayanotoxin* Toxicity

AMOUNT OF HONEY IN TABLE SPOONS	SYMPTOMS			
	G.I.	EYE	CVS	CNS
1 (15ml.)	(-)	(-)	(-)	(-)
2 (30ml.)	(+)	(+)	(-)	(-)
3 (45ml.)	(++)	(++)	(+)	(-)
4-5 (60ml.)	(++)	(++)	(++)	(+)
10 (150 ml.)	(+++)	(+++)	(+++)	(+++)

GI SYMPTOMS: Burning in throat and epigastrium, Vomiting. Vomit contained food particles & honey, along with frothy fluid.

EYE SYMPTOMS: Diplopia, Blurring of vision, dilated pupil, and sluggish reaction to light.

CVS SYMPTOMS: Palpitation, hypotension

CNS: Black out, unconsciousness, deep gasping, apnea

Physical Examination

The general condition of all cases was fair. The pulse ranged from 48-82/min, B.P. 80-110/ 40-80 mm of Hg. The rest of the systemic examination was normal except for papillary signs, respiratory depression, gasping unconsciousness, nausea and vomiting. The vomit consisted of food particles and wild honey, but did not contain blood.

Investigations

Blood T.L.C. = 4300-9800 cubic mm (4.3-10.8 x10³ mm³), DLC= N= 58-78% (40-80%), L= 22-42%(20-40%), Hb 11.8-15% gm% (13- 18 gm%), Blood Urea =18.8- 33mg% (20-40mg%), serum createnin= 0.9-1.4(0.6-1.2 mg/dl), Na+ = 139-143 meq/L(135-14.5 meq/L), K+ = 3.7- 5.0 meq/L (3.5-5.0 meq/L), The blood group in one case was group A, in three cases was group B, total bilirubin was 0.8-0.9(0.8-0.9), , direct bilirubin was 0.2, SGPT was 16-26(7-56 U/L)s, SGOT was 19-31(5-35 U/L) , A.L.P.was 172-209 (20- 160 U.L) and random blood sugar was 88- 108 mg/dl(70-110 mg/dl).

Discussion

The common factors of the above seven cases were; All of them were either medical students or young doctors of 22- 25 years, males, non smokers, but occasional users of alcohol.

As they were medical persons who had a better understanding of their signs and symptoms and as they stayed near the hospital, they reported early to the hospital. The signs and symptoms and the onset of illness were proportional to the amount of honey consumed, as shown in [Table/Fig 1].

It was reported by the restaurant owner that the wild honey was brought from Sagarmatha, Dolakha jilla, from the jungles (south of Jari) from Lincon Bazaar, near which Rhododendrons grew in abundance.

Xenophon described the odd behaviour of Greek soldiers after having consumed honey in a village surrounded by rhododendrons. Later, it was discovered that the honey resulting from these plants have a slightly hallucinogenic and laxative effect. The suspect rhododendrons are *Rhododendron ponticum* and *Rhododendron luteum* (formerly *Azalea pontica*), both of which are found in northern Asia Minor. Eleven similar cases have been documented in Istanbul Turkey during the 1980s [5].

In October 2004, in the Emergency Medical Journal, a similar report was published from Turkey about a series of 19 cases of honey poisoning. [6]. A neurotoxin found in the nectar of certain species of rhododendrons (*Rhododendron* spp.) and laurels (*Kalmia* spp.) and in unprocessed foods produced from the nectar such as unpasteurized honey, was found to cause temporary effects such as nausea, vomiting, dizziness and irregular heartbeat if ingested.[7].

Several cases of grayanotoxin poisonings in humans have been documented in the 1980s. These cases were reported from Turkey and Austria. The Austrian case resulted from the consumption of honey that was brought back from a visit to Turkey. From 1984 to 1986, 16 patients were treated for honey intoxication in Turkey. The symptoms started approximately 1 hr after 50 g of honey was consumed. In an average of 24 hrs, all of the patients recovered. The case in Austria resulted in cardiac arrhythmia, which required a temporal pacemaker to prevent further decrease in the heart rate. After a few hours, pacemaker

simulation was no longer needed. The Austrian case shows that with increased travel throughout the world, the risk of grayanotoxin poisoning is possible outside the areas of Ericaceae-dominated vegetation, namely, Turkey, Japan, Brazil, United States, Nepal, and British Columbia. In 1983, several British veterinarians reported an incident of grayanotoxin poisoning in goats. One of the four animals died. The post-mortem examination showed grayanotoxin in the lumen contents. [8]

Another report from Turkey in 2007 describes asystoles in a 60 year old patient who ingested wild honey derived from the flowers of rhododendron. [9] This was followed by another report from the same authors in which they questioned the necessity of hospitalization following wild honey poisoning [10]

Our cases are similar to previously published reports. According to FDA, a neurotoxin found in the nectar of certain species of rhododendrons (*Rhododendron* spp.) and laurels (*Kalmia* spp.) and in unprocessed foods produced from the nectar, such as unpasteurized honey, was found to cause temporary effects such as nausea, vomiting, dizziness, and irregular heartbeat if ingested. The intoxication is rarely fatal and generally lasts for no more than 24 hours. Generally, the disease induces dizziness, weakness, excessive perspiration, nausea and vomiting shortly after the toxic honey is ingested. Other symptoms that can occur are low blood pressure or shock, bradyarrhythmia (slowness of the heart beat associated with an irregularity in the heart rhythm), sinus bradycardia (a slow sinus rhythm with a heart rate less than 60), nodal rhythm (pertaining to a node, particularly the atrioventricular node), Wolff-Parkinson-White syndrome (anomalous atrioventricular excitation) and complete atrioventricular block.[8]

Mechanism Of Action Of Grayanotoxin

The grayanotoxins bind to sodium channels in cell membranes. The binding unit is the group II receptor site, localized on a region of the sodium channel that is involved in the voltage-dependent activation and inactivation. These compounds prevent inactivation; thus, excitable cells (nerve and muscle) are maintained in a state of depolarization, during which entry of calcium into the cells may be facilitated. This action is similar to that exerted by the alkaloids of veratrum and aconite. All of the observed

responses of skeletal and heart muscles, nerves, and the central nervous system are related to the membrane effects. [8]

Relative Frequency Of Poisoning

Grayanotoxin poisoning most commonly results from the ingestion of grayanotoxin-contaminated honey, although it may result from the ingestion of the leaves, flowers, and nectar of rhododendrons. Not all rhododendrons produce grayanotoxins. *Rhododendron ponticum* grows extensively on the mountains of the eastern Black Sea area of Turkey. This species has been associated with honey poisoning since BC 401. A number of toxin species are native to the United States. Of particular importance are the western azalea *Rhododendron occidentale* which is found from Oregon to southern California, the California rosebay *Rhododendron macrophyllum* which is found from British Columbia to central California, and *Rhododendron albiflorum* which is found from British Columbia to Oregon and in Colorado. In the eastern half of the United States, grayanotoxin-contaminated honey may be derived from other members of the botanical family Ericaceae, to which rhododendrons belong. Mountain laurel (*Kalmia latifolia*) and sheep laurel (*Kalmia angustifolia*) are probably the most important sources of the toxin. [8]

Conclusion

The increased desire of the public for natural (unprocessed) foods may result in more cases of grayanotoxin poisoning. Individuals who obtain honey from farmers who may have only a few hives are at increased risk. The pooling of massive quantities of honey during commercial processing generally dilutes any toxic substance which may be present. The public should consume only reliable branded forms of honey which are obtained from reliable sources in order to avoid honey intoxication.

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