Wild honey poisoning: A case report from Central Nepal

Dipendra Dahal[1], Ramesh Lamichhane[2], Rochana Acharya[3], Sagar Panthi[3], Shristi Chhetri[4], Philip Ranjit[1], Saroj Chaudhary[5], Rashmi Aryal[3], and Pradip Khatiwada[3]

[1]Star Hospital, Lalitpur
[2]Nepal Cardio Diabetes and Thyroid Centre
[3]BP Koirala Institute of Health Sciences
[4]Lumbini Medical College
[5]Nepal Cardio Diabetes and Thyroid Center

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Abstract

Introduction

Wild honey has been used traditionally for medicinal and recreational purposes. The toxic symptoms are due to grayanotoxin which prevents the inactivation of sodium channels leading to hyperpolarization clinically manifesting as dizziness, syncope, hypotension, and bradycardia. Most cases are self-limiting and resolve within 24 hours. Complications like bradycardia and hypotension may occur. Symptoms are managed with intravenous fluids and atropine as needed. Refractory hypotension may require vasopressor support.

Case presentation

We report a case of wild honey poisoning in a 45-year old male who presented to our emergency department with complaints of dizziness followed by loss of consciousness and with multiple episodes of vomiting and excessive sweating.

Interventions and outcome

He was stabilized successfully at first with intravenous fluids but later developed hypotension during the observation period and was managed with vasopressor.

Relevance and impact

Although fluid resuscitation remains the mainstay of management in wild honey poisoning, observation for complications such as refractory hypotension should be done in a setting where complications can be managed, which is not possible in most rural settings.

Keywords: *case report; wild honey; mad honey; poisoning, Nepal.*

Introduction

Honey is a naturally produced food having different compositions depending upon geography and species of bee [1]. Wild (mad) honey differs from commercial honey as it contains grayanotoxin [2]. Wild honey poisoning has been reported in Nepal, Turkey, and Korea [3]. Its symptoms range from dizziness, vomiting, syncope, and sweating to more serious effects like bradycardia and hypotension. These effects are usually self-limiting and resolve within 24 hours [2]. Here, we report a case of a wild honey poisoning in the capital city in Central Nepal presenting with hypotension that was initially stabilized with fluid resuscitation but

later again deteriorated which required vasopressor support with nor-adrenaline. This case report has been prepared in accordance with the CARE guidelines [4].

Case Presentation

A 45-year-old male was brought to the emergency department (ER) due to dizziness followed by loss of consciousness for 20 minutes. He also had multiple episodes of vomiting and excessive sweating. As per the patient, he had consumed nearly 100 ml of wild honey twenty minutes prior to the onset of symptoms. He had a laceration under his chin due to the fall which was actively bleeding. He was diabetic and hypertensive and was taking Metformin 500 mg twice daily and Losartan 25 mg once daily.

On examination, the patient was conscious and oriented but agitated. His blood pressure was 70/50 mm Hg, pulse rate was 58 beats/minute and regular, respiratory rate was 24 breaths/min, and oxygen saturation was 98%. There were no additional sounds during auscultation. A 12-lead ECG was obtained (Figure 1) promptly which showed sinus bradycardia with prolonged QT interval (481 ms) with non-specific ST-segment changes. Cardiac biomarkers however were negative. A random blood glucose assessment at the ER revealed blood glucose of 448mg/dL. He received 6 units of subcutaneous regular insulin stat at the ER. Blood glucose was monitored hourly. Routine blood investigations were sent which are given in Table 1.

Based on the history and presentation, diagnosis of wild honey poisoning was made. Treatment was immediately started with intravenous (IV) normal saline, IV hydrocortisone 100 mg (stat and three times a day), and IV atropine (SOS, if heart rate <40 beats/min). Laceration was repaired. After 2 hours at the ER, his vitals stabilized (BP: 110/80mm Hg, pulse 70/min). Thereafter, he was shifted to ICU for observation. His blood pressure dropped again after few hours of shifting to ICU (BP: 85/65mm hg). As a result, he was started on vasopressor support with nor-adrenaline at 2.5 ml/hr. His blood pressure stabilized within the next hour and the infusion was stopped altogether. The patient's vitals were stable overnight. Hyperglycemia was managed with infusion of regular insulin. He was shifted to the ward the next day and discharged the day after.

Table 1: Biochemical parameters of the patient **Test**	**Table 1: Biochemical parameters of the patient** **Value**	**Table 1: Biochemical parameters of the patient** **Reference range**
Random Blood Glucose	**450 mg/dL**	70-140 mg/dL
HbA1C	**11.1%**	4.0-5.6: Normal 5.7-6.4: Increased Risk of Diabetes 6.5 and above: Diabetes
Hemoglobin	15.1 g%	13-18 g%
Total WBC count	8900 cells/mm^3	4000-11000 cells/mm^3
Serum Na+	137.1 mmol/L	135-145 mmol/L
Serum K+	4.2 mmol/L	3.5-5.5 mmol/L
Serum Creatinine	0.8 mg/dL	0.4-1.2 mg/dL
Serum Urea	31.9 mg/dL	15-45 mg/dL

Discussion

Traditionally many people from Nepal and other parts of the world use wild honey for medicinal purposes in gastritis, diabetes, hypertension, common cold, wound healing, and sexual stimulant. In addition, it has also been used recreationally; euphoric effects are seen in low doses and with increasing doses, it can be hallucinogenic and potentially fatal. The toxic effects of wild honey are due to grayanotoxin which is found in different species of rhododendron transmitted by wild bees, this condition is characterized as a mad honey disease. This toxin binds voltage-gated sodium channel preventing its inactivation and causing hyperpolarization which results in effects similar to cholinergic excess [5].

The main complaints of wild honey poisoning are dizziness, bradycardia, nausea, vomiting, and pre-syncope

[2]. Although the exact amount of honey required for poisoning is not known, but symptoms have been reported after ingestion of 1 tablespoon of honey [6]. Symptoms have been reported to occur 0.5-3 hours after ingestion [7]. In our case, the patient experienced typical symptoms of dizziness, and vomiting 30 min after ingestion.

The diagnosis of wild honey poisoning is made clinically based on the history of ingestion and clinical feature. There are no routine blood tests available to measure grayanotoxin levels. Sinus bradycardia, atrioventricular block, and nodal rhythms are usually reported on ECG [8]. In addition, findings such as QT prolongation and Wolff-Parkinson-White (WPW) syndrome have also been reported [8, 9]. In our case, the ECG did not show any significant arrhythmia but had mild QT prolongation (QT interval= 480ms). The history of ingestion of wild honey was the only significant finding that led to suspicion of wild honey poisoning. Hence, wild honey poisoning should be suspected in presence of a history of ingestion with unexplained hypotension by other causes.

The toxic effects of poisoning usually last for 24 hours [10]. Treatment involves symptomatic management and observation. Hypotension is treated with normal saline infusion and atropine is used in bradycardia. Transvenous pacing and adrenaline infusion are required in refractory cases (1). Our patient's symptoms recovered in 24 hours. However, during the course of observation patient became hypotensive after initial stabilization. Subsequently, he required a nor-adrenaline infusion to stabilize his blood pressure. In the rural context of Nepal and other parts of the world, facilities to provide nor-adrenaline infusion may not be available. The majority of cases resolve with fluid resuscitation but the patient may deteriorate after initial stabilization, which happened in our case. Therefore, observation and close monitoring should be done in a setting where appropriate intervention can be done.

Wild honey hunting has been a tradition in certain Himalayan parts of Nepal. Moreover, it is also a source of income. Data regarding the toxic components and safety value in these wild honey is not available warranting further research.

Conclusion

In our part of the world, history of wild honey ingestion should be asked in patients presenting with unexplained hypotension or bradycardia. Patients may deteriorate after initial stabilization. Therefore, active surveillance should be done in resource-rich settings in order to provide vasopressor support and cardiac pacing when needed.

Conflict of interest: The authors declare no conflict of interest.

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Patient consent: Informed written consent was taken from the patient in this case report. We also ensured, none of the identifying characteristics are included in the case report.

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Figure

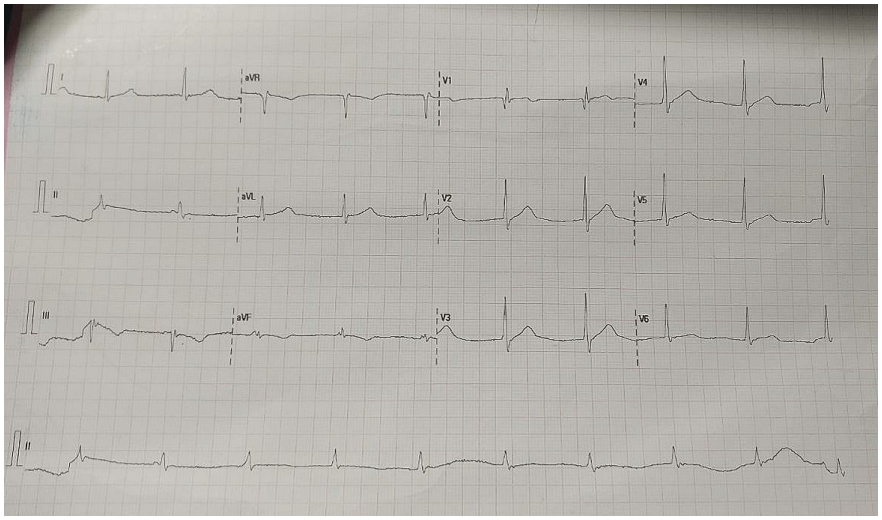

Fig. 1. ECG of patient on admission (Heart rate: 58bpm, QT interval: 480m)

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