JGPEMN
ISSN 2091-279X
eISSN 2362-1168

CASE REPORT

Mad honey (grayanotoxin) poisoning in Ghalegaun, Lamjung, Nepal: a case report

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Date of submission: 17 Sep 2025

Date of acceptance: 9 Dec 2025

Date of Publication: 31 Dec 2025

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How to cite:
Poudel P, Khanal A. Mad honey (grayanotoxin) poisoning in Ghalegaun, Lamjung, Nepal: a case report. J Gen Pract Emerg Med Nepal. 2025 Dec;12(20):57-59.

Online information
DOI:
https://doi.org/10.59284/jgpeman374

OPEN ACCESS

Abstract

Mad honey, derived from Rhododendron nectar, contains grayanotoxins that can precipitate an acute cholinergic-like toxidrome characterized by bradyarrhythmias, hypersalivation, and sweating. Nepal, particularly Himalayan districts such as Lamjung, has culturally significant wild-honey harvesting and periodic intoxications. We report an adult from Ghalegaun, Lamjung, who developed dizziness, vomiting, hypersalivation, hypotension, and sinus bradycardia within two hours after ingesting locally harvested wild honey for recreational purposes. Symptoms resolved within 48 hours with oxygen, intravenous fluids, ondansetron, and atropine. Recognition of the characteristic symptom cluster (sudden gastrointestinal upset, bradycardia, and hypotension after honey ingestion) enables prompt supportive management and prevents unnecessary investigations. Community education in honey-harvesting regions is warranted.

Keywords: Bradycardia; Cholinergic toxidrome; Grayanotoxin; Hypotension; Mad honey poisoning; Nepal

INTRODUCTION

Mad honey intoxication results from grayanotoxins derived from Rhododendron species; these toxins keep voltage-gated sodium channels in an activated state, leading to vagotonia, bradyarrhythmias, and vasodilation.[1] Typical manifestations include nausea, vomiting, dizziness, syncope, hypotension, and sinus bradycardia or AV block.[2,3] Most cases resolve with supportive care, intravenous fluids, and atropine.[3,4] Nepal is a recognized source of such honey, with traditions of cliff honey hunting among Gurung communities.[2] Periodic case series from Nepal have highlighted both the frequency and clinical relevance of these intoxications, especially in Lamjung and surrounding Himalayan districts.[5] Ghalegaun, a high-altitude tourism village, is one such location where wild honey is locally traded.[6]

CASE REPORT

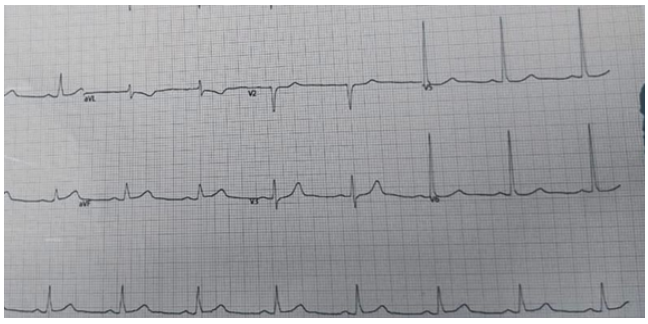
A forty-seven-year-old previously healthy male presented to our center with acute-onset dizziness and vomiting approximately two hours after ingesting locally harvested wild honey. The dizziness was described as a sensation of lightheadedness and near-syncope. Vomiting was sudden, non-bilious, and non-bloody.

There was no preceding chest pain, palpitations, or visual disturbances. He did not report fever, headache, abdominal pain, diarrhea, cough, or shortness of breath. There was no significant past medical or surgical history, and no history of recent medication use, pesticide or organophosphate exposure, or ingestion of other toxins

The patient was drowsy but arousable, with a Glasgow Coma Scale (GCS) of 12/15 (E3V4M5). His vitals revealed: Blood pressure (BP): 90/60 mmHg, Heart rate (HR): 35 bpm (regular), Respiratory Rate (RR): 16/min, SpO_2: 90% on Room Air, Temperature: Afebrile.

He was confused, not oriented to time, place, and person, and his Pupils were round, regular, and reactive. Chest examination revealed decreased air entry bilaterally with vesicular breath sounds and bilateral wheeze. His cardiac examination was normal; however, the per abdomen examination revealed mild epigastric tenderness.

ECG showed sinus bradycardia without heart block (Figure 1).



Fgure 1. ECG showing sinus bradycardia without heart block

Table 1. Laboratory Parameters of the patient during the initial visit

Parameter	Result	Reference Range
Hemoglobin	14.2 g/Dl	13.5–17.5 g/dL
WBC Count	7,800 /µL	4,000–11,000 /µL
Platelets	210,000 /µL	150,000–450,000 /µL
Blood Glucose	92 mg/dL	70–110 mg/dL
Creatinine	0.9 mg/dL	0.7–1.3 mg/dL

No additional toxin assays were available due to the rural setting.

The patient received a bolus of 500 mL IV normal saline, followed by 1 L over 4 hours to correct hypotension. Atropine 0.6 mg IV was administered for persistent bradycardia, blocking excessive vagal stimulation at the sinoatrial node. The patient's heart rate rose to 58 beats per minute and BP to 100/60 mmHg within 30 minutes post-atropine, and by 6 hours, HR and BP had normalized (64 bpm and 110/68 mmHg, respectively).

Ondansetron 4 mg IV stat was given to control nausea and vomiting and prevent dehydration. Supplemental oxygen was provided via nasal cannula to maintain adequate tissue oxygenation, particularly in the context of hypotension and bradycardia.

Due to the severity of the initial presentation and the risk of persistent bradyarrhythmias or hemodynamic instability, the patient was referred to a higher center and kept under observation in the ICU for 48 hours. Clinical information from the referral center was obtained through direct review of the patient's discharge summary and communication with the treating team. Continuous monitoring ensured early detection and management of any deterioration.

He remained stable throughout ICU observation and was discharged with counseling to avoid wild honey.

The patient was contacted on Day 7 and Day 14 after discharge. He reported no recurrence of dizziness, vomiting, chest discomfort, palpitations, or syncopal episodes. Home blood pressure readings remained within normal range, and no bradyarrhythmic symptoms were noted. He was advised to avoid consumption of locally harvested wild honey due to the unpredictable concentration of grayanotoxins. The patient and his family received counseling regarding early warning symptoms—progressive dizziness, recurrent vomiting, presyncope, or severe fatigue—that would warrant prompt medical evaluation. No further medical visits were required, and the patient returned to his usual daily activities without limitation.

DISCUSSION

This case illustrates the typical toxidrome: abrupt gastrointestinal upset with bradycardia and hypotension after Rhododendron-derived honey ingestion.[1,3,5] Grayanotoxins prolong sodium channel activation, mimicking cholinergic excess.[2] Diagnosis is clinical,

supported by history and hemodynamic findings, particularly in rural Himalayan settings where assays are not feasible.[5]

Treatment is supportive with oxygen, fluids, antiemetics, and atropine.[3,4] ICU observation is recommended. Rarely, pacing or vasopressors may be required.[2,3] Previous Nepalese reports, including case series from Lamjung and other districts, have highlighted similar presentations and outcomes.[5] Anaphylaxis following honey ingestion has also been reported, though rare.[5]

Public health awareness is crucial, as honey-harvesting remains culturally significant in Nepal. Educational initiatives in endemic regions could prevent intoxication and unnecessary hospitalizations.[7]

CONCLUSION

Mad honey poisoning should be suspected in patients presenting with acute gastrointestinal upset, bradycardia, and hypotension in the Himalayan regions of Nepal. Prompt recognition and supportive management are sufficient in most cases. Community education remains vital to prevent recurrence.

DECLARATIONS

Acknowledgement
None

Conflict of Interest
The authors declare no conflicts of interest.

Funding
No external funding was received for this work.

Ethical Clearance
No external funding was received for this work.

Consent of the Study
Formal written consent for publication was obtained from the patient.

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