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Wild Honey Poisoning: A Case Report from Remote Mountains

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Wild Honey Poisoning: A Case Report from Remote Mountains

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Abstract- Wild honey is consumed as a tonic in different parts of the world with a belief of increasing libido and treating various musculoskeletal, gastrointestinal and cardiovascular symptoms. However, honey produced from the nectar of several species of the Ericaceae (Rhododendron) family may contain grayanotoxins which act on sodium ion channels and place them in partially open state which causes symptoms like sweating, dizziness and altered sensorium owing to their effect on cardiac muscles and nervous system. We report a case of 60 years male who consumed wild honey as a pain reliever and later presented to the emergency room of Manang District Hospital with bradycardia, hypotension and altered mental status.

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I. Introduction

Wild Honey hunting is an old age tradition in Manang and other Himalayan parts of Nepal. Historically, wild honey has been used for gastritis, peptic ulcer disease, hypertension, wound healing, common cold, and diabetes. Studies have suggested about the possible health benefits of honey as an antihypertensive [1], antidiabetic, antioxidant [1], cardioprotective [2], antitussive and anti-bacterial [3]. However honey poisoning is caused by the consumption of wild honey (mad honey) made by bees from certain species of rhododendron [4]. Grayanotoxin is a naturally occurring sodium channel toxin found in honey made by bees from the pollen and nectar of the Ericaceae family of the Rhododendron [5]. Grayanotoxin binds the voltage-dependent sodium (Na) channel from the cytoplasmic side of excitable cells in human body in its open state after binding to the receptors preventing inactivation of Na channel and thus, increases the membrane permeability of Na channels. As a result, the membrane hyperpolarizes and leads to increase in refractory period. This leads to decrease in firing rates of pacemaker cells in the heart and decrease in cardiac contractility [6]. Thus, hypotension and bradycardia are the most common physical finding. Cardiac arrhythmias including sinus bradycardia (commonest), nodal rhythms and atrioventricular block are the usual electrocardiographic findings [7]. This report details a case of accidental wild honey poisoning presented with bradycardia and hypotension and its successful management in the emergency room.

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II. Case Report

A 60-year male without any past comorbid illness, presented in the emergency unit of Manang District Hospital, Chame, Manang, Nepal with history of ingestion of wild honey about 200 ml in amount. After around 1 hour of ingestion, the patient had multiple episodes of vomiting containing ingested food particles, generalized tingling, burning sensation, dizziness and altered sensorium. The patient had consumed the honey as a musculoskeletal pain reliever following physical exertion. On presentation to emergency, the patient was ill looking with Glasgow Coma Scale of 12 (E3M4V5) and was vomiting. The patient had blood pressure of 70/50 mm of Hg, heart rate of 52 beats/minute, axillary temperature of 97^0 Fahrenheit, respiratory rate of 22 breaths per minute and capillary oxygen saturation of 85% in room air. Immediate twelve lead ECG showed sinus bradycardia with rate of 50 beats per minute (Figure 1). All the laboratory investigations including hemoglobin, total leukocyte count, differential leukocyte count, serum sodium, potassium, urea and creatinine and serum level of liver enzymes were within normal range (Table 1).

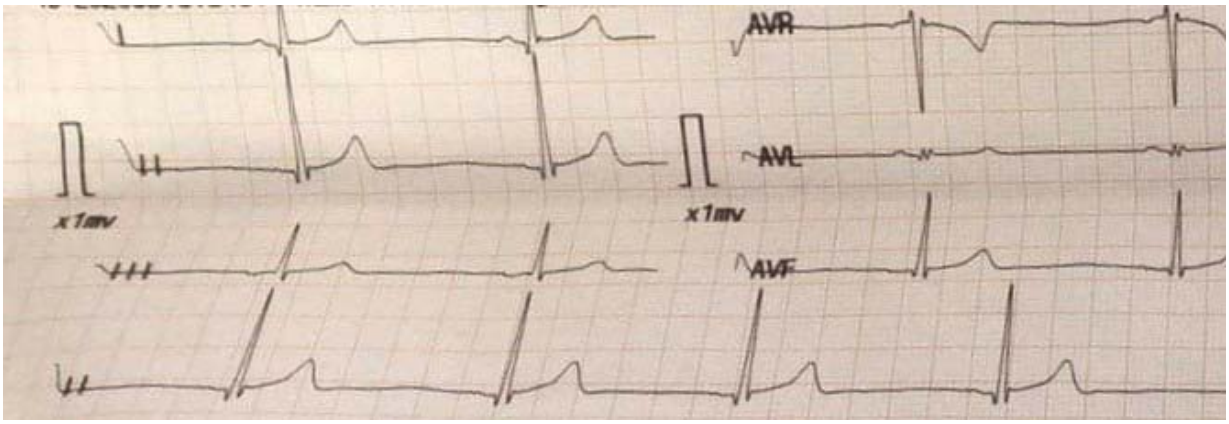

Figure 1: Electrocardiogram showing sinus bradycardia.

Table 1: Value of lab parameters.

Lab Parameters (units)	Value at admission
Total Leukocyte Count (cells/mm^3)	8100
Differential Leukocyte count (% of Total Leukocyte count)	Neutrophil 65% Lymphocyte 35%
Hemoglobin (g/dl)	13.2
Serum urea (mg/dl)	25
Serum Creatinine (mg/dl)	0.7
Serum Sodium (mmol/L)	140
Serum Potassium (mmol/L)	3.8
Serum Total Bilirubin (mg/dl)	1.2

The patient was provided with supplemental oxygen at the rate of 2l/min, given bolus intravenous crystalloids at 20ml/kg within one hour and was kept in Atropine infusion to maintain mean arterial pressure above 65 mmHg and heart rate above 50 beats per minute. The patient was kept nil per oral and received maintenance fluid at 80 ml/hour, injectable proton pump inhibitor and antiemetic. After about one hour of treatment, the Glasgow Coma Scale of the patient improved to 15/15. Atropine infusion was withheld after 8 hours of hospital stay, and his heart rate normalized to 70 to 75 beats per minute and twelve-lead electrocardiography showed normal sinus rhythm which persisted throughout hospital stay. The patient also made adequate urine output of 60 ml per hour over this time and continued to do so during hospital stay. Symptoms of vomiting also subsided over this time. The patient was discharged after 48 hours of observation in medical ward without complications.

III. Discussion

Grayanotoxins are found in leaves and flowers of plants of the family Ericaceae including *Rhododendron, Agarista and Kalmia* genera [8] which are available in Manang and other hilly regions of Nepal [9]. Grayanotoxin binds to the voltage gated sodium channels which prevents inactivation of the channels and increases their membrane permeability leading to hyperpolarization. The toxic effects of grayanotoxin are rarely fatal and usually last for not more than 24 hrs. Generally, they induce symptoms of dizziness, weakness, perspiration, salivation, nausea, vomiting and signs of hypotension, bradycardia, atrioventricular block and syncope [10].

In our patient, symptomatic emergency care with appropriate fluids and low dose atropine improved altered mental status, hypotension and bradycardia within short period of time. Patient with acute coronary syndromes can also present with similar symptomatology but with ischemic changes in ECG, serum positivity for cardiac injury biomarkers and vessels abnormality in coronary angiogram. Similarly organophosphate poisoning with cholinergic excess can also present with similar signs and symptoms with deranged cholinesterase enzyme activity on laboratory analysis.

Estimation of cholinesterase enzyme level in serum can be done to rule out organophosphate poisoning, which has a quite similar manifestation of cholinergic excess. The enzyme level is not affected by mad honey poisoning [11]. However, in the rural setup like ours, such laboratory analysis and cardiac catheterization facility are unavailable. Patient's history of consumption of wild honey along with suggestive ECG is ultimate for diagnosis.

Hunting and consumption of wild honey is a traditional and common practice in Manang and other Himalayan parts of Nepal. However, proper studies have not been done in these parts about the presence of toxic components in the plants which are available in this region belonging to the Ericaceae family. Also, the studies illustrating the therapeutic uses of such plants not available. These sort of accidental poisoning warrants such studies and creation of awareness among residents of areas where wild honey consumption practice is common. Also, with timely intervention and observation, patient presenting with this poisoning can be treated with readily available drugs with good prognosis. Physicians working in areas with wild honey hunting and consumption tradition should be aware of this poisoning.

Conflict of Interest: None.

Consent: Case Report Consent Form was signed by the patient and the original article is attached with the patient's chart.

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