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CASE REPORT

Mad Honey Mimicking Acute Coronary Syndrome

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Abstract
A 56-year-old Nepalese man presented to the hospital with syncope, bradycardia, and hypotension. Throughout his admission, he was hemodynamically unstable, with associated electrocardiographic changes and fluctuations in consciousness. This followed the unintentional overdose of the traditional medicine "mad honey" containing grayanotoxins with hallucinogenic properties. Following treatment with an anticholinergic infusion, he made a full recovery within 48 hours. This case underlines the importance of considering atypical causes for presentations such as bradycardia and hypotension, especially in patients from cultures where mad honey usage is common, and highlights the potential adverse events of this traditional medicine to be life-threatening.

Background
Approximately 300 types of honey are recognized worldwide. "Mad honey" contains a specific class of toxin called *grayanotoxin*. It is produced by honeybees that gather nectar from the *Rhododendron* species, from the Ericaceae family, most used in the Black Sea region of Turkey. However, it is also native to several other countries, such as Japan, Nepal, North America, and Brazil (1). Its use dates back as far as 2100 BCE, originally used as a biological weapon, highlighting its longstanding importance in traditional medicinal practices (1).

Case Report
A 56-year-old man presented to the emergency department following an episode of syncope and ongoing dizziness. On arrival, he was hypotensive, with an initial systolic blood pressure of 52 mm Hg and bradycardic, with an initial heart rate of 37 beats/min. A 12-lead electrocardiogram showed sinus bradycardia (Figure 1). Reassuringly, 2 troponin blood tests taken over 3 hours apart came back with the following levels: 4 ng/L and 3 ng/L (Figure 2). Similarly, other than a raised lactate level of 1.6 mmol/L, his venous blood gas test showed no outstanding results (Figure 2).

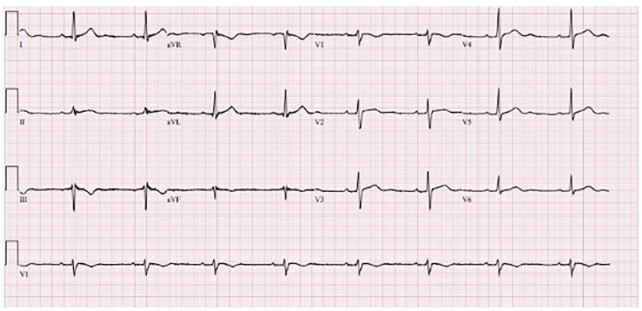

Figure 1. This 12-lead electrocardiogram demonstrates the patient's sinus bradycardia (rate, 49 bpm), recorded prior to intervention.

VENOUS BLOOD GAS		
pH	7.38	
pCO_2	6.0	kPa
Na^+	138	mmol/L
K^+	3.9	mmol/L
Cl^-	102	mmol/L
Ca^{++}	1.24	mmol/L
Hct	43	%
Glucose	5.7	mmol/L
Lactate	1.6	mmol/L
tHB	145	g/L
O_2Hb	73.8	%
HHb	24.6	%
BE (B)	1.0	mmol/L
HCO_3^- std	25.0	mmol/L

LABORATORY RESULTS		
Hb	136	g/L
WBC	7.9	x 10^9/L
RBC	4.61	x 10^{12}/L
Platelets	211	x 10^9/L
Neutrophil	4.4	x 10^9/L
Creatine Kinase	208	IU/L
Urea	7.7	mmol/L
Creatinine	134	mmol/L
Sodium	142	mmol/L
Potassium	4.0	mmol/L
eGFR-EPI	51	mL/min/1.73^2
Hs Troponin I	4	ng/L

Figure 2. Venous blood gas results showing a raised lactate level of 1.6 mmol/L. Otherwise, the patient's test results were normal. Serial troponin levels at 0 and 3 hours were 4 ng/L and 3 ng/L, respectively, showing no significant rise.

During the patient's assessment, a friend who was with him at the time of change in consciousness disclosed that he had ingested Himalayan mad honey. The attending team members were unfamiliar with this potential toxin and reviewed the management on TOXBASE. Following this review, he received an initial dose of 600 mcg of intravenous atropine as well as fluid resuscitation, with a total of 3 L of 0.9% sodium chloride via a large cannula given over a few hours, resulting in an improved heart rate of approximately 70 beats/min and a blood pressure of 120/77 mm Hg. On further assessment, his airway remained patent and his chest was clear on auscultation.

However, within the hour, his hemodynamic state further deteriorated. He then received an additional 600-mcg dose of atropine, which again allowed his blood pressure and heart rate to return to normal limits. The critical care team assessed the patient and recommended observation and monitoring at a higher level of care for a minimum of 48 hours, as he was currently stable. Over the next few hours, he received 2 mg of intravenous isoprenaline in 100 mL 0.9% sodium chloride when his heart rate again went below 40 beats/min. His Glasgow Coma Score remained 15/15 and his condition continued to improve, with no disruption to his urine or cardiac output.

He then disclosed that a friend had recommended mad honey for fatigue, which he consumed with alcohol, leading to unintentional overdose.

He did not report any significant family or medical history. He admitted to being a long-term smoker, smoking nearly 20 cigarettes a day for most of his adult life, and drinking slightly over the recommended limit of 14 units per week. He was not taking any prescribed medications and he did not report any other illicit, unprescribed, herbal, or over-the-counter medication usage. After 48 hours in the critical care unit, his condition improved, he required no further intervention, and he was discharged.

Discussion

In its native regions, mad honey is used as alternative medicine to prevent and treat many disorders, including hypertension, peptic ulcers, and gastritis (2). It is also commonly used for pain relief but there is no evidence that it is used for fatigue or lethargy (3). Recent studies have noted its antimicrobial, blood sugar–lowering and analgesic effects (4, 5). It can even be used to enhance sexual performance (6). It is also used for its hallucinogenic properties, which may be enhanced when consumed with alcohol, which is a common practice (7). However, it has been banned from some countries, including South Korea in 2005, and there were several deaths attributed to mad honey in 2012 in southwestern China (6, 8).

Grayanotoxins, also known as *andromedotoxin*, *acetylandromedol*, and *rhodotoxin*, produced by the *Rhododendron* species, are sodium channel agonists that, when ingested, can cause a range of effects (7). They interfere with the transmission of the action potential by blocking sodium channels in cell membranes and prevent inactivation (8). Poisoning occurs when ingested in honey form in doses greater than 14 g (9). There are many types of honey, including one containing tutin (a neurotoxin from *Coraria* species), which is seen in New Zealand (10).

There is no routine test available to quantify the level of grayanotoxin in the blood of a patient and the severity of adverse effects vary according to the dose ingested. These include nausea, hypersalivation, dizziness, weakness, hypotension, respiratory distress, paralysis, and loss of consciousness (2). The cardiac complications of mad honey poisoning are commonly seen in severe poisoning and include arrhythmias, atrioventricular block, nodal rhythm, atrial fibrillation with a slow ventricular response, transient ST-segment elevation, and QT prolongation (2). These occur as grayanotoxin binds to voltage-gated sodium channels on cell membranes of neurons, leading to increased vagal nerve activation, inducing bradycardia and hypotension (11). This can reduce perfusion to the heart, mimicking a myocardial infarction. Other nonspecific manifestations can include blindness, blurred vision, hypothermia, hepatitis, and convulsions (6). Ingestion of mad honey with antihypertensives or certain cardiac medications, such as beta blockers or calcium channel blockers, worsens its effects (12). It has been noted that the combination of grayanotoxins with alcohol exacerbates symptoms such as light-headedness, hypotension, and syncope (13).

It has been reported that the half-life of mad honey ranges between 256 and 580 minutes according to CakMak-Arslan and colleagues (14). The literature shows that the onset of symptoms of poisoning occurs after a period of a few minutes to a few hours and with prompt treatment usually subsides in 24 to 48 hours (2). Bradycardia and hypotension are the most common physical findings (2). Hospital admission with cardiac monitoring is recommended for at least 6 hours. The benefits of activated charcoal are unclear; prompt treatment of hypotension with intravenous fluids and symptomatic bradycardia with atropine, isoprenaline, or dobutamine is recommended (2). Mad honey may be associated with convulsions, which should be treated with intravenous diazepam. Rarely, temporary pacing can be used if necessary for transient atrioventricular block (7).

Although mad honey and its associated intoxication are rare in the United Kingdom, they remain common in regions such as Turkey and Nepal (2). Other cases may lack a witness to provide helpful context; with increasing migration and travel around the world, health care professionals should be aware of mad honey and its effects, which can often mimic more common diagnoses. The severity of adverse effects from grayanotoxin ingestion should not be underestimated because, although they are rarely fatal, they can persist from minutes to hours (12).

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