



## Honey Poisoning Case with Stroke-like Symptoms *İnme Benzeri Semptomlarla Başvuran Bal Zehirlenmesi Olgusu*

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### Summary

A special type of honey produced in Blacksea region in Turkey, known as the “mad honey”, causes hypotension and dizziness. We herein report a patient presented with symptoms suggesting posterior cerebrovascular disease following mad honey ingestion. (*Turkish Journal of Neurology* 2014; 20:13-5)

**Key Words:** Mad honey, stroke-like symptoms, poisoning

### Özet

Daha çok Karadeniz bölgesinde üretilen ve halk arasında “deli bal” olarak bilinen balın hipotansiyon, vertigo yaptığı bilinmektedir. Bu çalışmada bal yedikten sonra acil polikliniğimize arka sistem serebrovasküler hastalık şeklinde semptom veren bir olgu sunulmuştur. (*Türk Nöroloji Dergisi* 2014; 20:13-5)

**Anahtar Kelimeler:** Deli bal, inme benzeri semptom, zehirlenme

### Introduction

Consumption of honey produced from plants in Rhodendron family causes the clinical profile of honey poisoning. This profile is most commonly seen after the consumption of the type of honey, colloquially known as “mad honey”, produced in Blacksea region of Turkey. This honey is produced by bees who gather nectar from plants that belong to Rhodendron family (1). A family of substances called grayanotoxins naturally occurring in the nectar causes the symptoms of poisoning after the consumption of honey. The symptoms can vary: burning on the skin and throat, itchy mouth and nose, redness in the skin and eyes, vertigo, headache, nausea, vomiting, stomach pain, gastroenteritis bradycardia, hypotension, delirium or even life-threatening complete atrioventricular block may take place (1). This condition can rarely result in death (2). The symptoms generally disappear spontaneously after 24 hours. Most of time, support treatment proves to be sufficient. In this article, we present a case with hypotension, bradycardia and cerebrovascular

disease symptoms following honey consumption in the absence of any other etiological reasons.

### Case

A sixty six year old woman without an existing medical condition came into the emergency service with vertigo, double vision, numbness in mouth and tongue, nausea, vomiting and sporadic right arm weakness starting after she had breakfast. She had an unremarkable medical history. An interview with the patient's family revealed that she had a teaspoon of honey that was specially procured. Her complaints reportedly started about half an hour after the breakfast. In the physical examination, her blood pressure was 103/60 mmHg, pulse was 40/min, breathing rate was 20/min and her fever was 96.8°F. In the cardiovascular system examination, heart was rhythmic with normal sounds; there were no murmurs but it was bradycardic. In the neurological examination, she was sleepy, partially cooperative and disoriented; the pupils were isochoric, IR

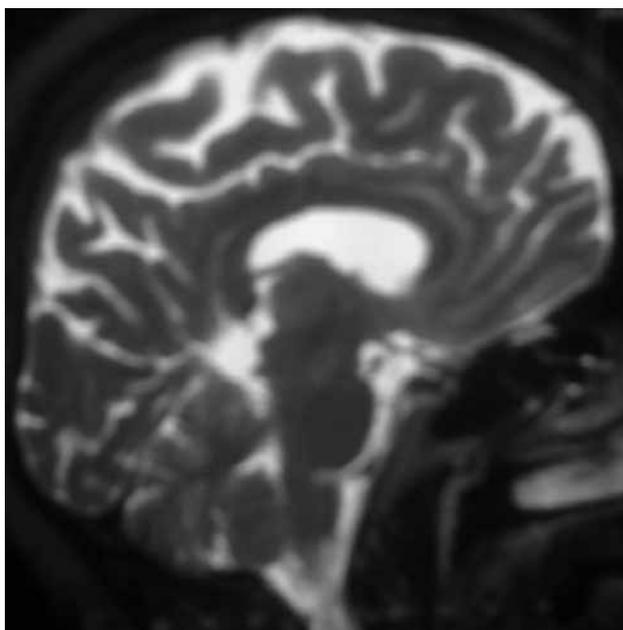
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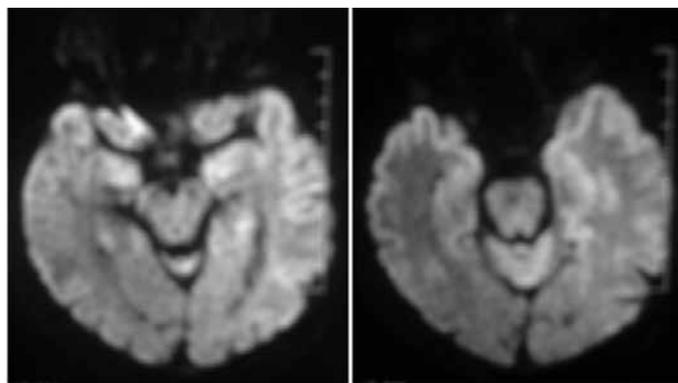
+/, eye movements were unrestricted and facially symmetrical. The motor examination was normal. Deep tendon reflex was present bilaterally. There were no pathological reflexes. Her cardiac enzymes and other biochemical tests were normal. There was sinus bradycardia in the electrocardiogram. Cranial magnetic resonance (MR) and diffusion MR were normal (Figures 1, 2, 3). The patient was monitored in conjunction with the internal medicine clinic and given low molecular weight heparin. All of her complaints and the clinical findings spontaneously disappeared over 24 hours. Her electrocardiogram and carotid-vertebral Doppler ultrasound were normal.

## Discussion

Honey poisoning is frequently seen in western Blacksea and Southeastern Anatolia regions in Turkey but they are not confined to those areas. Grayanotoxins are nitrogen deficient polyhydroxy-



**Figure 1.** There were no ischemic findings in the T2-weighted MRI



**Figures 2-3.** There were no ischemic findings in the axial diffusion-weighted MRI

cyclic hydrocarbons soluble in oil (3). With 18 different types previously defined, it is reported that the first type is responsible for causing the cardiac symptoms (3).

It is not clear whether the effect of the toxin is dependent on the dose or it is idiosyncratic. Grayanotoxin possibly binds to the voltage-gated sodium channels on the cell membrane and suppresses the inactivation of the channels. Consequently, the cells constantly depolarize and produce fewer action potentials. The involvement of vagus nerve causes weaker action potentials in the sinoatrial node, therefore causing clinical symptoms such as hypotension, bradycardia and slowed breathing rate through sinus node dysfunction (4,5). Based on the observation, smaller doses only cause dizziness, hypotension and bradycardia while higher doses cause more serious symptoms such as loss of consciousness, seizures and atrioventricular block. The real relationship between the dosage and toxicity is largely unknown but it was shown that even small doses do cause poisoning (2). Our case experienced the symptoms after consuming only one teaspoon of honey.

The symptoms appearing after oral consumption of honey can start anywhere between a few minutes and two hours after consumption (2). In our case, this duration was half an hour.

The most common symptoms are gastrointestinal such as nausea, vomiting and diarrhea. However, life-threatening cardiovascular symptoms can also be seen. In rare cases, conditions that require a pacemaker, such as varying degrees of atrioventricular block, sinus bradycardia, nodal rhythm, asystole, long QT syndrome, and acute coronary syndrome (4,6) were also seen (2). Central nervous system symptoms are extremely rare. Blurry vision and delirium can be experienced (2,4). Since the reasons for her ER visit were vertigo, impaired vision, numbness around the mouth and tongue, and weakness in the right arm, a hemodynamic dysfunction in the vertebrobasilar system was suspected. Low blood pressure and sinus bradycardia were detected in the physical examination. The cranial MR and diffusion MR were normal despite the suspicious honey consumption. The biochemical examinations to discover the etiology were normal. At the same time, echocardiogram and carotid-vertebral Doppler ultrasound were also normal. Magnetic resonance angiography was not conducted since there were no findings indicating ischemia in the cranial and diffusion MR, and carotid-vertebral arteria Doppler ultrasound examinations conducted to detect potential cerebrovascular diseases.

Honey intoxication has been known since the old ages. It is told that 3 battalions of the roman general Pompeius poisoned after eating the bowls of mad honey left on the mountains of Blacksea and were subsequently butchered by their enemies in B.C. 67 (7).

The toxic effect always ends within 24 hours. Therefore only supportive therapies are recommended for honey poisoning cases. Sinus bradycardia and conduction defects generally respond well to atropine treatment. Blood pressure treatment requires hydration support. In our case, sinus bradycardia and hypotension accompanied cerebral symptoms. Since there were no other

symptoms indicative of a cerebro-vascular pathology except for the honey intoxication, the patient was closely monitored and hemodynamically stabilized. Her physical and neurological examinations at the 24<sup>th</sup> hour were normal.

To our knowledge, there has not been another case reporting cerebrovascular disease-like symptoms due to honey poisoning. We conclude that possibility of honey poisoning should be taken into consideration when obtaining medical history.

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