

Honey intoxication as a cause of unconsciousness and respiratory failure; Case reports with review of literature

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Abstract

Honey intoxication is caused by the consumption of honey produced from the nectar of rhododendrons. The grayanotoxins causes the intoxication. We describe the honey intoxication in two patients. We aimed to present the clinic, radiologic, diagnostic and therapeutic features of “mad honey syndrome”. This case series suggest that the history of eating honey should be questioned in a person who had sudden onset of bradycardia, hypotension and hypoventilation, especially in older patients. “Mad honey intoxication” diagnosis can't be made unless specifically questioned.

Keywords

honey intoxication; bradycardia; respiratory failure; unconsciousness

Introduction

The dark, reddish, “mad honey,” known as “*deli bal*” in Turkey, contains an ingredient from rhododendron nectar called grayanotoxin - A natural neurotoxin that, even in small quantities, brings about light-headedness and sometimes, hallucinations. When over-imbibed, however, the honey can cause low blood pressure and irregularities in the heartbeat that result in nausea, somnolence, blurred vision, fainting, hallucinations, seizures, and in rare cases even death. This toxicity seen rarely, however it may lead life-threatening hemodynamic instability mentioned above and may be confused with various diseases, as seen in cerebrovascular, cardiac and respiratory problems including hypoxemic/hypercapnic respiratory failure, ischemic heart and central nervous system diseases [1-3]. For these reasons the exact diagnosis and treatment of this intoxication is seems very important. It is called as “mad honey syndrome” and we aimed to present the clinic, radiologic, diagnostic and therapeutic features of “mad honey syndrome” in two cases. The consent forms of patients were obtained for using dataclinic informations and test results.

Case 1

A 80 years old man admitted to the emergency department with complaints of drowsiness and syncope after breakfast at home. Physical examination revealed the somnolence, tending to sleep and shallow breathing. Hypotension (systolic blood pressure 60mm Hg and diastolic blood pressure 30 mmHg) and bradycardia (heart rate 42/min) were noted. ECG showed the bradycardia without ischemic findings and chest roentgenography demonstrated the cardiomegaly and nonspecific reticular opacities

(Figure 1A and 1B). We asked the honey consumption and we learned that he ate “mad honey” for breakfast. “Mad honey syndrome” is defined according to findings and supportive treatment with IV fluid replacement and theophylline. The patient had fully recovered and discharged, one day later.

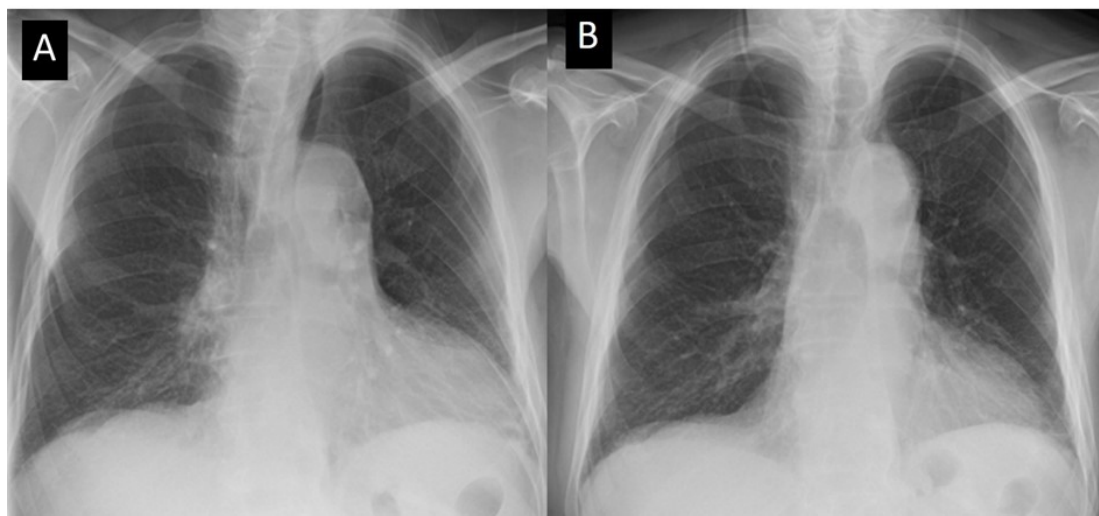


Figure 1: Chest roentgenography showing nonspecific reticular opacities due to bradycardia and hypotension (A) and recovered after the treatment (B).

Case 2

A 82 year old woman admitted to the emergency department with syncope at home after breakfast. Hypotension (systolic blood pressure 80mmHg and diastolic blood pressure 50 mmHg), bradycardia (heart rate 68/min) and unconsciousness were observed. Respiratory failure caused from hypoventilation was noted. Arterial blood gases were pH:7.4, pO₂:43 mmHg, pCO₂: 34.1mmHg and SaO₂ 78.3%. She was intubated for respiratory depression at emergency room. Chest roentgenography demonstrated the pulmonary congestion with nonspecific reticular opacities, as in first case (Figure 2A and 2B). The Electrocardiogram (ECG) showing bradycardia and ischemic heart diseases were excluded.

We asked the patient about honey consumption and we learned that he ate “mad honey” for breakfast, just like our first case. IV theophylline treatment was given to the patient. Rapid response to treatment was achieved and she was extubated after 1 hour of admission.

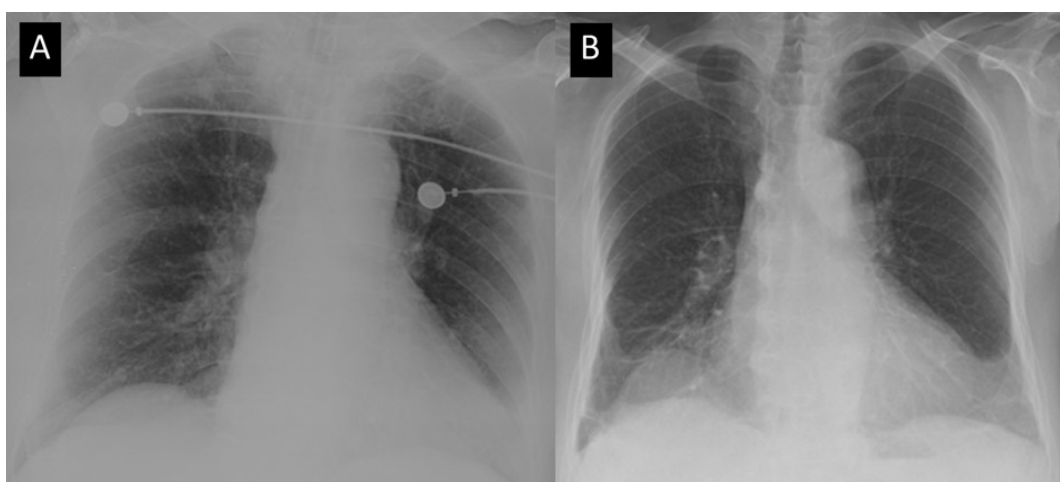


Figure 2: Chest roentgenography showing nonspecific reticular opacities and pulmonary congestion due to bradycardia and hypotension (A) and recovered after the treatment (B).

Discussion

There have been famous episodes of inebriation of humans from consuming toxic honey throughout history. Xenophon, Aristotle, Strabo, Pliny the Elder, and Columella all document the results of eating this "maddening" honey. Historically the toxin in the honey was derived from the pollen and nectar of *Rhododendron luteum* and *Rhododendron ponticum*, which are found around the Black Sea. According to Pliny and later Strabo, the locals used the honey against the armies of Xenophon in 401 BCE and later against Pompey in 69 BCE. When Roman troops under Pompey the Great attacked the Heptakometes in Turkey, and they were poisoned by the honey from honeycombs deliberately placed along their route. The Roman soldiers became delirious and nauseous after eating the toxic honey, leading to an easy defeat [4-6].

Honey containing grayanotoxin known as *deli bal* is still deliberately produced in the Caucasus region of Turkey. In the eighteenth century, this honey was exported to Europe to add to alcoholic drink to give it extra potency.

The grayanotoxins are neurotoxins interfering with the transmission of the action potential by blocking sodium channels in cell membranes. These compounds prevent inactivation; thus, excitable cells (nerves and muscles) are maintained in a state of depolarization, during which entry of calcium into the cells may be facilitated. All of the observed responses of skeletal and heart muscles, nerves, and the central nervous system are related to the membrane effects [7]. The toxic effects of honey poisoning are rarely fatal and generally last for no more than 24 hours. Demir et al. reported the 21 cases with "mad honey syndrome" [3]. Symptoms of poisoning occurs depending on the amount of honey consumed and there is latent period of a few minutes to hours. In mild forms, dizziness, weakness, excessive perspiration, hypersalivation, nausea, vomiting, and paresthesias are present, and close follow-up is enough. However, severe intoxication may lead to life threatening cardiac complications such as complete atrioventricular block. In our cases and reported articles suggested that the symptomatic sinus bradycardia with hypotension is the most frequently reported mad-honey-induced cardiac dysrhythmia [8]. Previously reported patients were responded to intravenous fluid therapy and parenteral atropine. The bradycardia and hypotension were rapidly responded to IV theophylline treatment, in our cases. Interestingly, both of our cases were older patients and elder people may be more susceptible to toxication.

Conclusion

History of eating honey should be questioned in a person who had sudden onset of bradycardia, hypotension and hypoventilation, especially in older patients. "Mad honey intoxication" diagnosis can not be made unless specifically kept in the differential diagnosis and asked from the patient.

References

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