Science Letters ISSN 2345-5463 – An International Triannually Journal

Review Article
2018 | Volume 6 | Issue 2 | Pages 67-73

ARTICLE INFO

Received
April 04, 2018
Accepted
June 05, 2018
Published
August 10, 2018

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Keywords
Honey poisoning
Cardiogenic Shock
Grayanotoxins
Honey poisoning symptoms

How to Cite
Shrestha R, Shrestha A, Chen SL. Five case reports of wild honey intoxication with symptoms and treatments. Sci Lett 2018; 6(2):67-73

Note
First and second author contributed equally to this manuscript.

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Five Case Reports of Wild Honey Intoxication with Symptoms and Treatments

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Abstract

Honey is one of the daily consuming foods in our life and is being used for centuries. However, honey made from some species of rhododendron plants contains toxins known as grayanotoxins, may be poisoning or even life-threatening. This is also referred as 'wild honey or mad honey'. Wild honey is mostly taken as an alternative medicine for the treatment of various diseases. Many people have a general belief that wild honey can treat gastritis, peptic ulcers, weakness, arthritis, diabetes, hypertension and reduce weight. Wild honey results in intoxication and its symptoms normally include dizziness, hypotension and bradycardia either sinus or atrial-ventricular block. Grayanotoxins interfere with normal sodium channel functioning by maintaining excitable cells in a depolarized state during which, calcium entry into the cells is facilitated and results in continuous stimulation of the vagal nervous system causing various symptoms. In this article, we have presented five cases of wild honey intoxication in the emergency settings. We have discussed symptoms, mechanism and treatment plans for the physician to provide knowledge of its diagnosis and treatment.

Introduction

Wild honey, a natural bee product being used since ancient time for making food, alternative medicines for gastric pain, bowel disorders, weakness, arthritis, diabetes and hypertension and some people also believed to be a weight reducer. It is either used locally or ingested with other food [1, 2]. However, wild honey is not safe every time. Wild honey made from some species of rhododendron containing harmful compounds can cause dramatic effects; grayanotoxins (GTX) is one of those toxins [3]. In one of the studies done by Onat et al. [4], high doses of GTX have been reported in samples collected from patients that consumed two tablespoons of wild honey along with associated symptoms like bradycardia due to vagal stimulation.

GTX is found in the nectar of rhododendrons, a plant species [5]. There are 700 species of rhododendrons in the areas like China, Tibet, Nepal, Myanmar and Assam. Around 300 species are found in tropical Asia and small numbers in Europe and North America. GTX is of 18 forms, GTX I-IV is the unique class of toxic diterpenoids, which are polyhydroxylated cyclic hydrocarbons that do not contain nitrogen. GTX III is a principle toxic isomer in rhododendron [6]. GTX neurotoxin interferes with the transmission of the action potential by blocking sodium channels in the cell membrane, which maintain skeletal, heart muscle, nerves and central nervous system in a depolarized state allowing calcium entry into the cells, finally bringing about sinus dysfunction [7]. We have introduced five cases of wild honey intoxication with various symptoms from mild dizziness to high-grade heart block, which were treated. The aim of this study was to describe the wild honey intoxication cases and to review the literature to address knowledge about its symptoms and treatments to assist physicians.

Case presentation

Case 1

A male patient of 45 years old came with the complaints of chest heaviness, shortness of breath, dizziness, nausea, one episode of syncope along with sweating and weakness after taking wild honey. He denied of taking an overdose of drugs. On presentation no pulse palpable on the radial artery and carotid pulse was feeble. His blood pressure was un-recordable, chest X-ray revealed white opaque haziness in the left upper part of the chest (Fig. 1). The electrocardiograph (ECG) taken the first time showed broad QRS patterns without P waves and the occasional premature beats (Fig. 2). However, ECG repeated later showed sinus bradycardia with ST-segment elevation in limb leads II, III, avf (Fig. 3 & 4). Transthoracic echocardiography (Echo) of this patient stated left ventricular ejection fraction of 65% and no wall motion abnormalities.

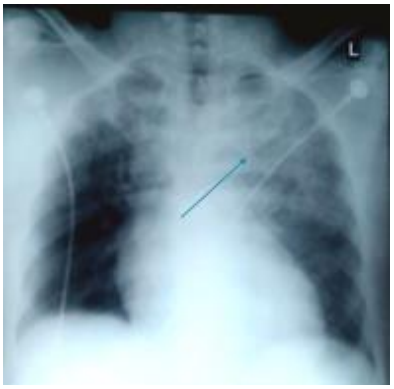

Fig. 1 Chest X-ray of a patient showing white opaque haziness (blue arrow) in the left upper part of the chest.

Cardiac enzyme (CPK MB 42IU/L, CPK NAC: 324 IU/L, and Troponin T: 289 pg/ml), his routine hematological and biochemical parameters were within normal limits. The patient was immediately started with a parenteral normal saline (0.9% sodium chloride) and 1 mg atropine. Under these circumstances, we thought necessary to rule out coronary artery disease. Thus, after slight stabilization of vitals, the patient was taken for angiography. However, the result was normal (Fig. 5). He was monitored in the coronary care unit for the observation. The next day, ECG was repeated and serial cardiac enzymes were checked, which showed a gradual return to normal and clinically asymptomatic, so discharged on that day.

Case 2

A 46 years old female who visited our emergency department complaining of dizziness, nausea, burning sensation of the whole body and muscle, along with joint pain after consumption of the same kind of wild honey with hot water and lemon an hour back. Her heart rate was 50 beats per minute with a feeble pulse in the radial artery. Her blood pressure was 60 mmHg systolic and diastolic could not be measured. Her routine hematological and biochemical parameters were also within normal limits. Her ECG showed sinus bradycardia (Fig. 6 and Fig. 7). She was treated parenterally normal

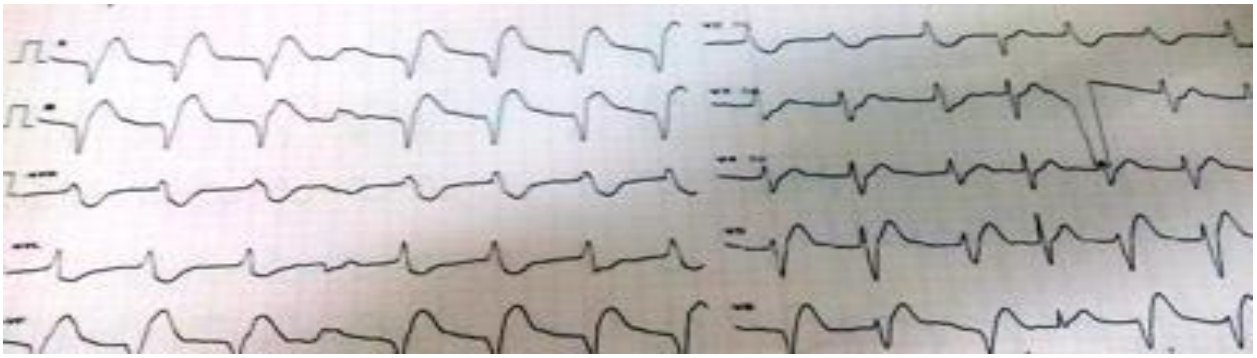

Fig. 2 Electrocardiography **(**ECG) taken the first time showed a broad QRS pattern without P waves and the occasional premature beats in the local hospital.

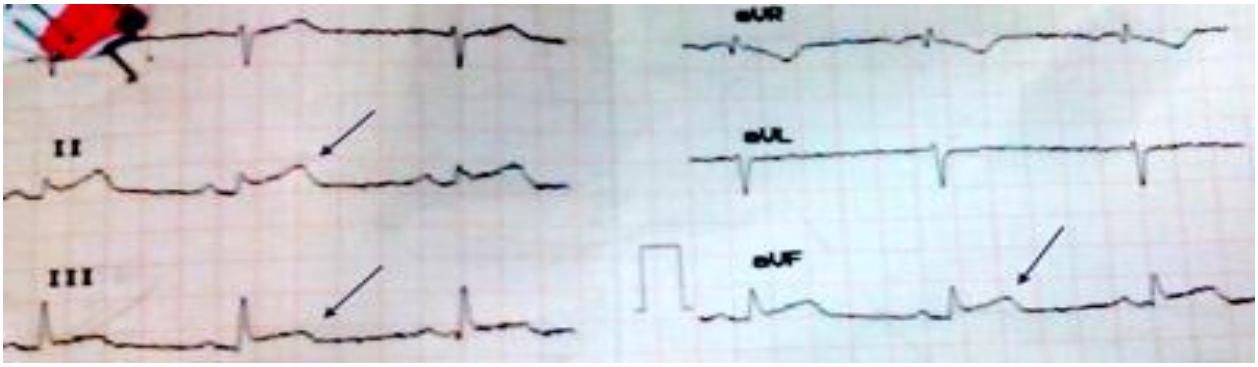

Fig. 3 Electrocardiography **(**ECG) repeated in our hospital later showed sinus bradycardia with ST-segment elevation in limb leads II, III, avf (black arrows).

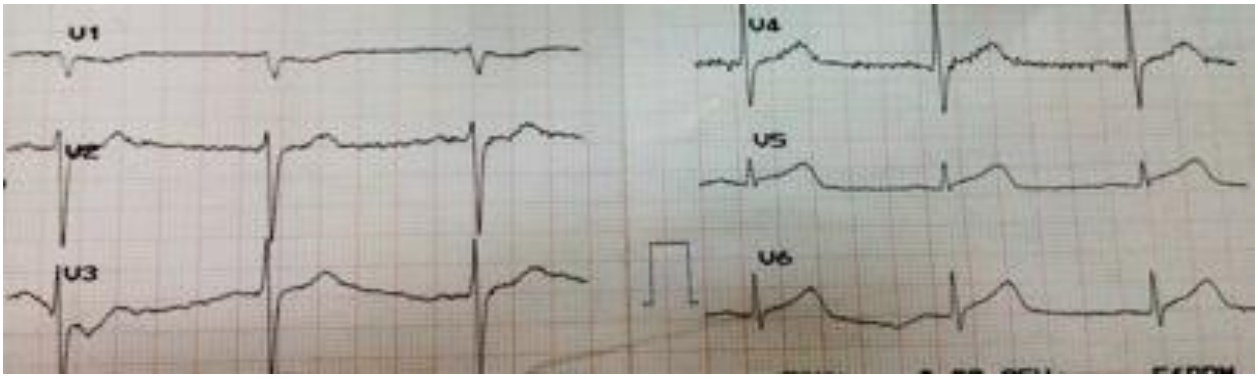

Fig. 4 Electrocardiography **(**ECG) of the same patient showing no abnormalities.

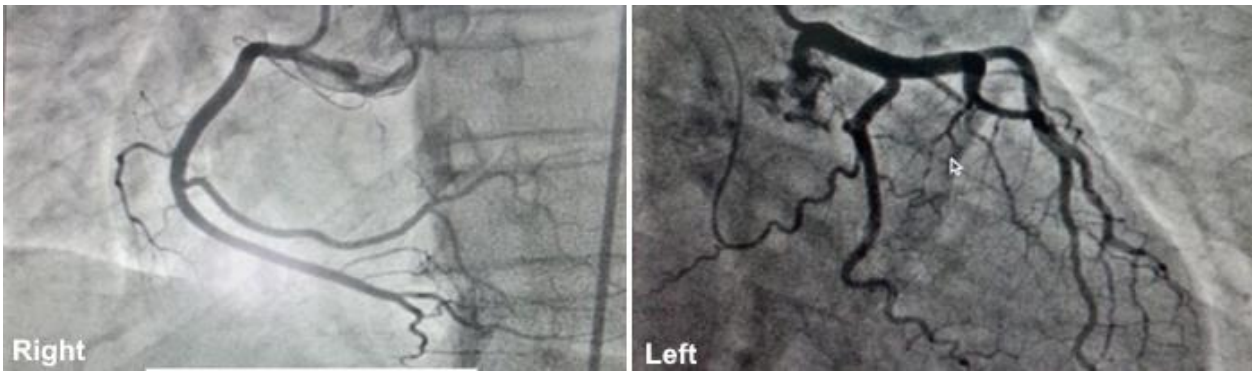

Fig. 5 Normal images of right and left coronary artery angiography of the patient.

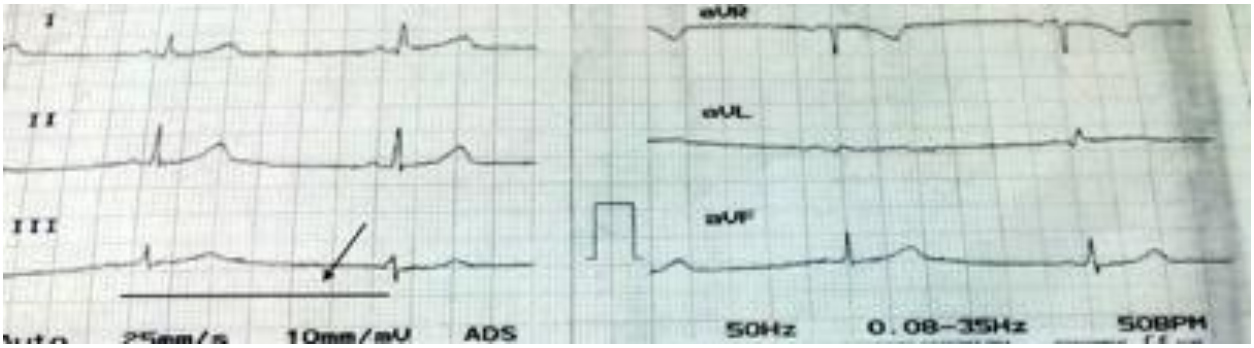

Fig. 6 Patient electrocardiography during symptoms showing sinus bradycardia (black arrow)**.**

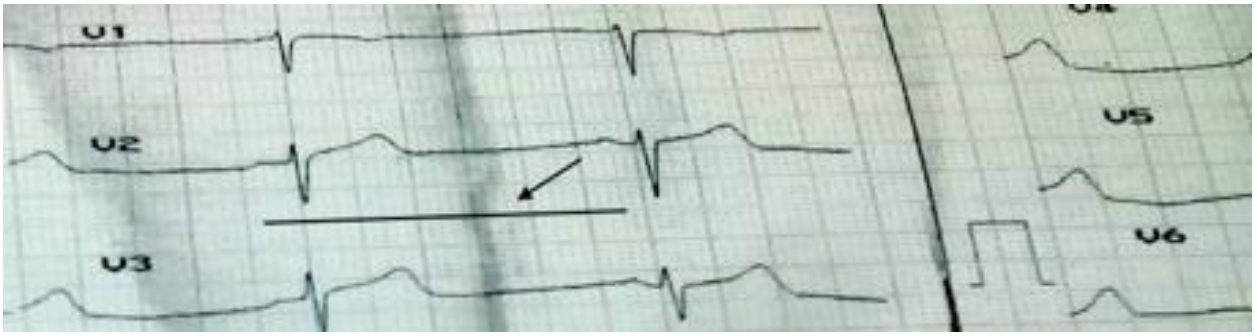

Fig. 7 Patient electrocardiography during symptoms showing sinus bradycardia (black arrow).

saline (0.9% sodium chloride) given fast and another 500 ml slowly after blood pressure measured to 80/60 mmHg. She was given other supportive medicine like an antacid. Like in the first case, she was also discharged another day after stabilization with all normal reports.

Case 3

Male patient, 54 years old visited the emergency department with the complaint of sudden onset of loss of consciousness for about 10 min. He also stated blurring of vision and dizziness prior to it. He had multiple episodes of non-bilious, non-projectile vomiting in the emergency department. All his symptoms started after the consumption of one teaspoon of wild honey. His pulse was 44 beats per minute, blood pressure systolic 60 mmHg and diastolic could not be measured, afebrile with cardiac enzyme: CK MB 40 IU/L, and no abnormalities seen in the routine hematological and biochemical parameters. The ECG showed complete heart block (Fig. 8**)**. Echocardiography revealed normal left ventricular ejection fraction.

The patient was immediately injected parenteral 1mg atropine and normal saline (0.9% sodium chloride) until blood pressure measured to 80/60 mmHg. His coronary angiography was done which revealed normal coronary arteries (Fig. 9**)**. Patient blood pressure gradually normalized to 130/70 mmHg and pulse of 88 beats per minutes.

Case 4

Fifty-five years old male patient with a past history of hypertension, chronic kidney disease stage II, polycystic kidney disease, dyslipidemia, and prostatomegaly came to the emergency department with the complaints of altered sensorium for half an hour and vomiting for 2 episodes after the consumption of wild honey; he also experienced dizziness during that time. Pulse 42 beats per minutes, blood pressure 80/40 mmHg, afebrile, respiration, and oxygen saturation normal (Fig. 10). He denied taking an overdose of the prescribed drugs. Renal function test: serum creatinine 2.9 mg, other parameters including potassium were all under normal ranges. Cardiac enzymes were also normal. Echocardiography revealed normal left ventricular ejection fraction of 60%, borderline thickening of the left ventricle, mildly dilated left atria, mild mitral and aortic regurgitation with mildly thickened aortic valves. The patient was given parenteral 1mg of atropine and parenteral normal saline (0.9% sodium chloride) 1000 ml and his medication for hypertension was stopped till his blood pressure came to 140/80 mmHg. The next day, the patient was clinically asymptomatic with

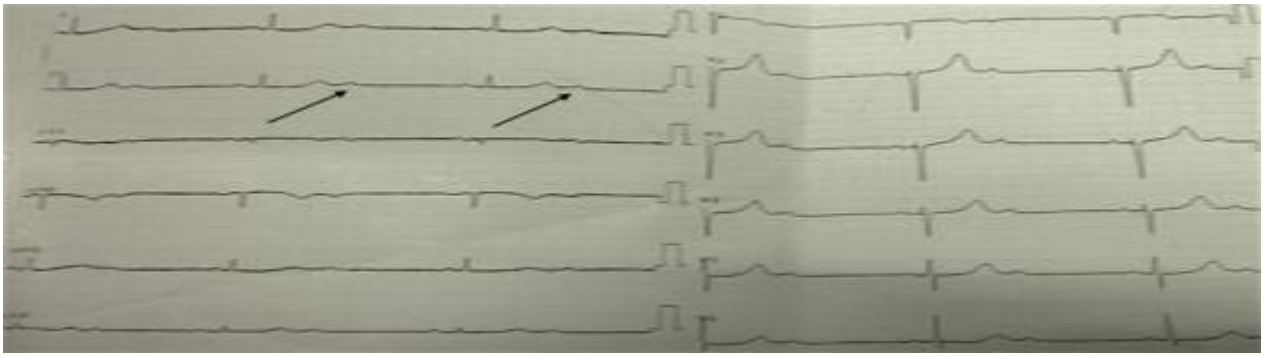

Fig. 8 Patient electrocardiography during symptoms showing complete heart block (black arrow showing p waves alone).

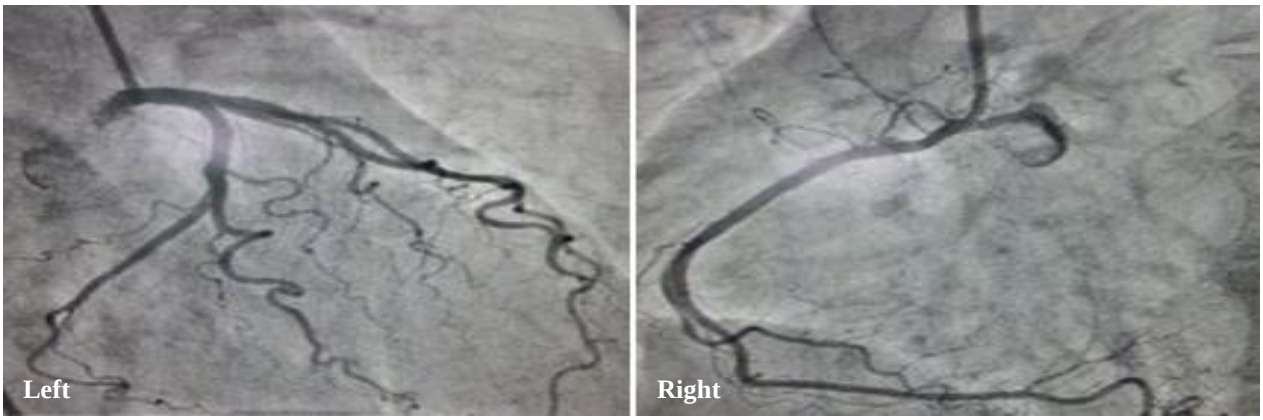

Fig. 9 Angiography images of a patient with normal left and right coronary artery.

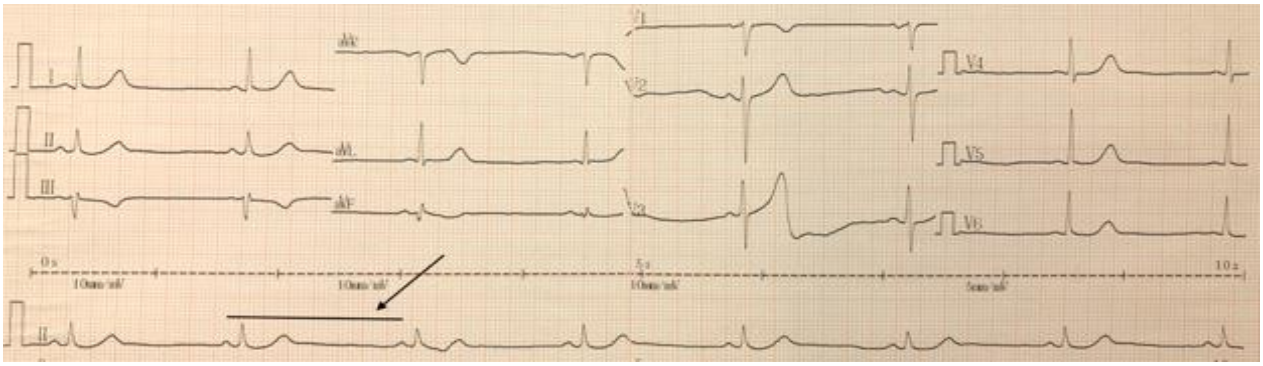

Fig. 10 Patient electrocardiography during symptoms showing sinus bradycardia with a heart rate of 42 beats per minutes.

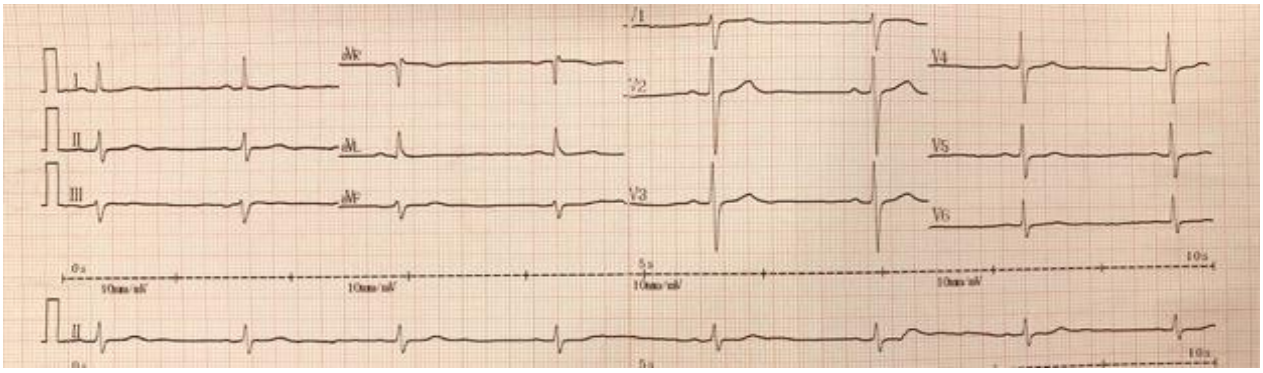

Fig. 11 Patient electrocardiography during symptoms showing sinus bradycardia with a heart rate of 45 beats per minute.

normal vitals and discharged on his previous medication without any complications.

Case 5

Female patient, 50 years old, feverish along with chills and rigors came to the emergency department. She had one episode of non-projectile vomiting contained only food particles. She also had symptoms of nausea, sweating, palpitations and burning sensation in the epigastric region after taking wild honey. On physical examination, her bilateral limbs were cold. Blood pressure systolic was 90 mmHg and diastolic couldn't be measured. A pulse rate of 45 beats per minutes with normal renal function test was found. The ECG showed sinus bradycardia (Fig. 11). The patient was managed with parenteral normal saline (0.9% sodium chloride) and other supportive medicines like antiemetic and antacids. Patient blood pressure increased to 110/70 mmHg with a pulse of 62 beats per minutes. She was monitored in the coronary care unit. Next day, after patient vitals normalized and clinically asymptomatic was discharged without any complications.

Discussion

Wild honey poison is not rare in some geographical areas or its diverse symptoms. Although wild honey poison has no potential for morbidity, and mortality symptoms are dose-related from mild to severe life-threatening so we should diagnose in time and treat in time adequately. In a study done by Yilmaz et al., the honey amount of 5g to 30g is enough to cause poisoning along with its diverse symptoms [8]. Symptoms related to wild honey poison are dose-dependent, lasts for minutes to hours, no more than 24 hours as it is metabolized and excreted rapidly [9,10]. The most common symptoms are hypotension, syncope, bradycardia, atrioventricular block, to mild weakness, dizziness, nausea, vomiting along with perspiration. In severe cases, high degree atrioventricular block, severe sinus bradycardia with hypotension and signs of posterior ischemic changes in ECG [8-10]. Likewise, in a study done by Yilmaz et al., 17.6% of the patients with syncope, 45.4% with nausea and 31.8% with vomiting were found. In this study, all the patient's symptoms resolved completely within 24 hours with IV fluids and atropine, and none died [8]. Yavuz et al. studied nausea-vomiting in 91% and dizziness in 74% of patients [10]. Similarly, like other studies, patient symptoms resolved within 24 hours with IV fluids and atropine. Aygun et al. stated bradycardia and hypotension, the most common clinical symptoms were dizziness in 60% of patients, syncope in 56% and vomiting in 28%. Other clinical symptoms vary depending upon age, gender, and other additional diseases. All the patients received the saline solution and 1-2 mg atropine IV as support therapy and were discharged with stable vital sign after 24 hours [11]. All of the patients described above were treated with adequate fluid therapy (saline 500-1000 ml), IV atropine (0.5 to 2 mg depending upon the blood pressure and heart rate) and those patients with severe hypotension used inotropes for complete recovery. In addition, all patients were safely discharged after 1-2 days of cardiac monitoring.

GTX found in rhododendron flower species are collected by the bees from the nectar and pollens then retained in the honey. GTX are neurotoxins which interfere the transmission of the action potential by blocking sodium channels in the cell membrane, which maintains skeletal, heart muscle, nerves and central nervous system in a depolarized state, allowing calcium entry into the cells and finally brings about sinus dysfunction [3, 12]. Many kinds of cases of wild honey intoxication during emergency settings can be diagnosed as variant angina and pathologic sinus dysfunction and would be prescribed anti-angina medicine to temporary pacemaker implantation, but with designing treatment and thorough history will assist the physician to diagnose wild honey intoxication precisely and timely.

Conclusions

Wild honey poison is not as infrequent as considered. It can be missed in emergency settings; thorough history is a must. We have to keep in mind that it may progress to higher degree conduction block and bradycardia. However, most of the patients need symptomatic treatment and close observation.

Conflict of Interest

The authors declare that they have no conflict of interest to declare.

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