An unusual type of food poisoning is commonly seen in the Black Sea coast of Turkey attributable to andromedotoxin containing toxic honey ingestion. This study is a retrospective case series of 19 patients admitted to an emergency department in 2002, poisoned by “mad” honey. All of the patients had the complaints of nausea, vomiting, sweating, dizziness, and weakness, several hours after ingesting “mad” honey. Physical examination showed hypotension in 15 patients, sinus bradycardia and conduction defects in 15, and complete atrioventricular block (AVB) in four patients on admission. Two patients with bradycardia and two with AVB fell and injured their heads. Three of them presented with local haematoma. One patient had a 6 cm cut on his head without any neurological deficit and his cranial computed tomography imaging was normal. Hypotension and conduction disorders resolved with atropine treatment, resulting in complete recovery within 24 hours.

Turkish honey from the Black Sea coast of Turkey, occasionally contains concentrations of acetylandromedol high enough to cause poisoning. Bradycardia, atrioventricular block (AVB), and arterial hypotension have the potential to cause death in untreated cases but no fatalities have been reported in the literature. Complete recovery after hospital admission is normally the rule because, hypotension usually responds to the administration of fluids, correction of bradycardia and conduction defects, which usually respond to atropine treatment. Most of the cases have been reported from Turkey. Sutlupinar et al reported 11 cases admitted to a hospital in Istanbul, between 1983 and 1988.1 Von Malottki et al reported a case from Germany and Gossinger et al reported two cases from Austria all treated successfully.2 3 Geroulanos et al from Switzerland reported that about eight cases of intoxication induced by honey have been reported in the hospital of Trapezunt every year.4

Symptoms of poisoning occur after a dose dependent latent period of a few minutes to two or more hours. In severe intoxication, loss of coordination and progressive muscular weakness result. Atrioventricular and intraventricular conduction disturbances also may occur. Convulsions are reported occasionally.5

CASE REPORTS
The study is designed as a series of cases analysed retrospectively. Nineteen patients (12 men, seven women) aged between 22–61 years, were admitted to the emergency department with the symptoms of nausea, vomiting, hypotension, bradycardia, and fainting in the year 2002 (table 1). History was obtained from the patients except for four who had collapsed before admission. The relatives supplied the history in these four cases. Each patient ingested about 30 to 180 grams of honey several hours before admission. Fifteen of the patients had previously been diagnosed with a duodenal ulcer in the internal medicine or gastroenterology departments of the local hospitals. The reason for the honey ingestion may be the belief that honey heals a duodenal ulcer when consumed continuously. Initial physical examination showed that all patients had bradycardia and 15 had hypotension (arterial systolic blood pressure <90 mm Hg). In four patients symptoms began suddenly while they were standing. These four patients sustained head trauma after falling due to possible sudden hypotension and collapse. Three patients had small (less than 5 cm in diameter) haematoma on their heads and neurological examinations were normal. One patient had a 6 cm cut on his head. He had cranial computed tomography (CT) for possible intracranial haemorrhage after a neurological consultation. The result was normal. In three patients symptoms were comparatively mild because of a slight decrease in blood pressure and sinus bradycardia. In two patients bradycardia was severe and four patients had complete AVB (fig 1A).

Figure 1 (A) Complete atrioventricular block in a patient after honey ingestion. (B) Sinus rhythm restored after 0.5 mg atropine administration in the same patient.
Cardiac emergencies and honey ingestion

All patients were monitored in the coronary care unit for 24 hours. Sixteen patients received 0.5 mg of atropine. Administration criteria were symptomatic hypotension and complete AVB. Two patients with symptomatic bradycardia and three patients with AVB were given a second dose of 0.5 mg atropine five minutes after the initial dose because heart rate and blood pressure could not be restored (fig 1B). In all patients, heart rate and blood pressure returned to normal limits within two to nine hours. A basal rate of intravenous sodium chloride infusion (100 cc/h) was continued for 24 hours. The authors of the study do not recommend infusion of larger volumes of fluid for patients recovering from symptomatic hypotension because the main cause of hypotension in these patients are the underlying conduction disorders in which atropine is effective in most cases. All patients were discharged healthy the next day.

**DISCUSSION**

Grayanotoxins, mainly grayanotoxin I (andromedotoxin) occurs only in Erichaceae plants, are the compounds responsible for poisoning. They are extracted by bees from the leaves and flowers of rhododendron species. From the 18 forms of grayanotoxins, grayanotoxin I is responsible for honey poisoning. The grayanotoxins bind to sodium channels in cell membranes preventing inactivation and maintaining the cell in a state of depolarisation, during which entry of calcium into the cells may be facilitated.

The toxic effects of honey poisoning are bradycardia, cardiac arrhythmia, hypotension, nausea, vomiting, sweating, salivation, dizziness, weakness, loss of consciousness, fainting, blurred vision, chills, and cyanosis. A case of generalised convulsion has been reported in the literature. Cardiac disturbances are the main signs in this poisoning and Onat et al showed that atropine sulphate alleviated bradycardia attributable to grayanotoxin, and AF-DX 116, a selective M2-muscarinic receptor antagonist, restored heart rate. They suggested that M2-muscarinic receptors were involved in the cardiotoxicity of grayanotoxin.

“Mad” honey is used in the Black Sea region as an alternative medicine for the treatment of gastrointestinal, bowel disorders, hypertension, and it is believed to be a sexual stimulant. The precise amount for a toxic dose is not known, but previously reported cases have suggested that “one teaspoonful” toxic honey may cause intoxication. In general the severity of the honey poisoning depends on the amount ingested. Our findings showed that as the amount ingested increases, the probability of development of AVB increases. However, this is just an observation. It should not be proposed that “mad honey” ingestion lower than 30 g is safe. The concentration of grayanotoxin ingested may differ greatly from case to case. As grayanotoxins are metabolised and excreted rapidly the patients generally regain consciousness and feel better within hours and heart rate and blood pressure usually return to normal within 24 hours.

Because of the increasing preference for natural products, intoxication induced by consumption of honey will increase in the future especially with products bought directly from the beekeeper. Cases of honey intoxication should be considered in every country because of an increase in the consumption of imported, unprocessed honey. All people are believed to be susceptible. Honey poisoning should be kept in mind in previously healthy patients admitted with unexplained hypotension, bradycardia, and complete AVB. Inquiry should be made about the possibility of honey ingestion. Close surveillance and symptomatic treatment should be carried out and physicians should be alert for sudden worsening of bradycardia and progressive conduction disturbances.

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Atrial fibrillation after electrical shock: a case report and review

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A 52 year old man was admitted to an emergency department with a fast ventricular rate atrial fibrillation after an electrical shock. Electrical cardioversion was attempted after echocardiographic examination. This failed, but the heart rate slowed. Successful pharmacological cardioversion was achieved after 16 hours of amiodarone infusion. Pre-excitation syndrome was detected on baseline echocardiograph. Serum cardiac specific markers were all within normal limits. No abnormal findings were detected by chest radiography, echocardiographic, or coronary angiographic investigations. Acute onset atrial fibrillation after electrical injury is discussed.

A 52 year old man was admitted to our emergency department with palpitation and shortness of breath after an electrical shock. He had touched a live cable carrying 220 volts during repair of a washing machine. He had lost consciousness for a while after the accident, but was conscious on admission. He had no history of cardiac disorder and there were no indications of coronary artery disease. On physical examination, his blood pressure was 90/70 mm Hg and his heart rate was about 220 beats per minute, with irregular rhythm. Lung examination revealed rales. The entry wounds were two contact abrasions located proximally on the second and third fingers of the right hand. The exit wound was a small burn injury on the right side of the tongue (fig 1A).

Cardiac monitoring showed atrial fibrillation with faster heart rate on the monitor of DC shock device was 220 bpm). We started heparin 1000 unit/h after an initial 30 minutes. We started intravenous metoprolol 5 mg three times over 12 hours intervals. All other biochemical and haematological investigations were normal. Chest radiography was normal. Echocardiographic examination of cardiac structures was normal. Coronary artery disease was excluded by coronary angiography. The patient rejected electrophysiological study and catheter ablation. We treated the patient with acetylsalicylate acid 300 mg/day for two months and he has no further complaints.

DISCUSSION

Domestic electrical supplies operate at 220–240 volts, and alternating current is more dangerous than direct current. Electrical shocks generally result from contact with live wires or lightning. Cardiovascular effects include myocardial infarction, transient accelerated hypertension, left ventricular dysfunction, cardiac rupture, and arrhythmias. Premature ventricular contractions, ventricular tachycardia, ventricular fibrillation, atrial tachycardia, atrial fibrillation, bundle branch, and complete heart block may be occur after electrical shock. Follow up studies show that the prevalence of arrhythmias after electrical injury varies between 10% to 36%. Atrial fibrillation after electrical shock is extremely rare. In a study of 182 cases of electrical injury over a 20 year period, only two instances of atrial fibrillation were reported and one of them required cardioversion.

The mechanism of electrical current induced arrhythmias is not clear. Because of differences in electrical resistance, current travels preferentially along blood vessels and nerves, making the heart most susceptible to injury. Patchy necrosis can be seen in heart muscle biopsy specimens after electrical injuries, the fibrotic tissue being a potential chronic arrhythmogenic focus. Increased cardiac sodium/potassium pump activities and an increase in potassium concentration have also been described. Cardiac arrhythmias may occur at the time of electrical shock or later, but mostly within first day after injury.

Arrowsmith et al retrospectively evaluated 145 patients with electrical injury in the same centre over the five year period; 128 (88%) had suffered low voltage injury and 17 (12%) had suffered high voltage injuries (>1000 V). The...