

An arrhythmic episode after mercury exposure and successful treatment with chelation therapy: A case report

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Introduction

People are exposed to mercury in daily life by means of food, batteries, broken thermometers and fluorescent lamps, and amalgam. Neurotoxicity and reproductive toxicity of mercury are well known. However, the relationship between mercury poisoning and its effects on the cardiac conduction system has not been clearly identified. Sodium 2, 3-dimercaptopropane-1-sulfonate (DMPS) is a metal chelating agent approved for oral or intravenous use to treat poisoning with mercury (1). In this case, a patient with neuromuscular symptoms and an arrhythmic episode that started after exposure to mercury and alleviated with DMPS therapy is presented.

Case Report

A 32-year-old female patient presented to our out-patient clinic with a 1-week history of malaise, fatigue, weakness of the lower and upper extremities, atypical chest pain, and palpitation. The clinical history was not significant for any systemic disease. On presentation, she was tachypneic and tachycardic with a heart rate of 140 bpm; pulse was irregular. Electrocardiography (ECG) disclosed atrial fibrillation with T-wave inversion in lateral precordial leads (Fig. 1a). Transthoracic echocardiography revealed normal left ventricular systolic and diastolic functions. The heart chambers and valvular functions were within normal limits. Laboratory investigation was normal for electrolytes; renal, liver, and thyroid functions; and cardiac biomarkers. Arterial blood gas analysis was also within normal limits. Electromyography revealed normal sensorial and motor functions with no signs of polyneuropathy.

When clinical history was repeated, it was found that 1 day prior to the initiation of her symptoms, she broke a fluorescent lamp while trying to replace a damaged one. Toxicological blood analysis showed the blood mercury level to be 4.2 µg/L (<10 µg/L), spot urine mercury level to be 61.3 µg/L (<10 µg/L), and 24-h urine mercury level to be 344 µg/L (<15 µg/L). The patient was subsequently admitted with a diagnosis of mercury poisoning. DMPS was given intravenously at a dose of 3 mg/kg, three times a day for 4 days. In addition, the patient was anticoagulated with enoxaparin throughout the course of admission. Anti-arrhythmic or beta blocking agents were not used. Table 1 shows mercury levels in blood, spot urine, and 24-h urine that were measured daily during hospital stay. Neurological symptoms disappeared after DMPS therapy, and ECG taken on the third day of admission showed sinus rhythm and normalization of negative T waves (Fig. 1b). The patient was discharged on the fifth day, with anticoagulation planned for 1 month.

Discussion

The cardiovascular consequences of mercury toxicity include hypertension, coronary heart disease, carotid artery obstruction, cere-

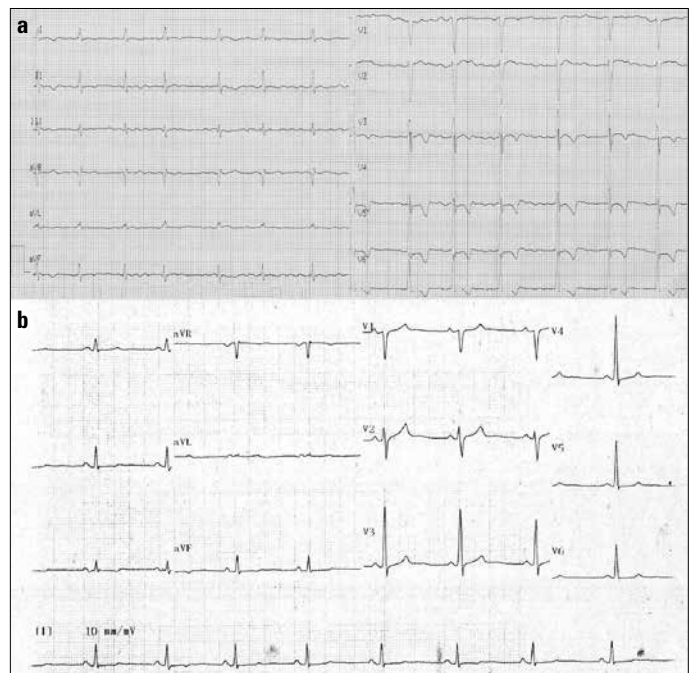


Figure 1. a, b. ECG at presentation shows rhythm to be atrial fibrillation and T-wave inversion in leads V3–V6 (a). ECG on the third day after DMPS therapy. Reversion to sinus rhythm and normalization of T wave (b)

Table 1. Blood, spot urine, and 24-h urine mercury levels measured daily during admission

	Blood Mercury level (µg/L)	Spot Urine Mercury level (µg/L)	24-h Urine Mercury level (µg/L)
Day 1	4.2	61.3	344.0
Day 2	4.8	8.0	81.1
Day 3	4.7	7.6	78.7
Day 4	4.0	4.1	47.5
Day 5	1.3	1.9	2.3

brovascular accident, and generalized atherosclerosis (2). The effects of mercury on the cardiac conduction system and the relationship between mercury toxicity and arrhythmias are not adequately known. There are a few studies that have evaluated the relationship between heart rate variability (HRV), which is an indicator of cardiac autonomic function and thus cardiac arrhythmias, and mercury poisoning (3, 4). Mercury binds to the sulfhydryl group of S-adenosylmethionine, which is a cofactor of catecholamine-O-methyltransferase (COMT) enzyme (5). As a result, COMT is inhibited and blood levels of noradrenaline, adrenaline, and dopamine increase. The resulting sympathetic over-activity may be the underlying mechanism of arrhythmias due to mercury poisoning.

Fluorescent lamps contain mercury and when broken, the mercury can spread in high concentrations in the environment and toxicity signs may be seen similar to the scenario presented in this case (6). Another important point that should be kept in mind is that the clinical manifestations of mercury intoxication vary depending on not only its concentration but also its form, route of ingestion, and the duration of exposure (7). Although the blood mercury level of our patient was below the reference value, it was considered to be mercury poisoning because of clinical presentation consistent with poisoning, high urine mercury

level, and acute and non-occupational exposure. In our patient with neurological and cardiac symptoms on presentation, initial tests for possible etiologies did not lead to a successful diagnosis. When clinical history was intensified, it was observed that the patient's symptoms were due to an incident that can happen in daily life and therapy was successfully administered.

Conclusion

This case presents the relationship between mercury toxicity and cardiac arrhythmias for the first time and it also emphasizes the value of carefully recording the medical history of a patient on the one hand and the hazardous consequences of environmental exposure on the other.

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Short QT syndrome in a 14-year-old patient: The first pediatric case from Turkey

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Introduction

Short QT syndrome (SQTS) is a cardiac channelopathy associated with sudden cardiac death (SCD) and disposition to atrial-ventricular fibrillation (A-VF) (1). An accelerated ventricular repolarization (VR) abnormality develops in the heart due to an electrical stability disorder secondary to increased extracellular potassium flow in the heart (2, 3). Therefore, ventricular arrhythmias develop, which cause syncope, convulsion, and SD (2).

This paper presents a 14-year-old male patient whose elder brother and father had died because of SD and who presented to us with the complaint of syncope. He was identified to have a short QT interval (SQTI) in his electrocardiogram (ECG) result, diagnosed with SQTS following an electrophysiological study (EPS), and implanted with an implantable cardioverter defibrillator (ICD).

Case Report

The 14-year-old male patient presented to our center with the symptom of syncope. His elder brother was found dead after taking a bath (autopsy result negative), and his father was found dead while he was asleep. The physical examination, biochemical parameters, telecardiography, and echocardiography results of the patient were normal. The QT and QTc values were identified to be 310 msec and 320 msec in his ECG result, respectively (Fig.1a). After the family's consent was received, in EPS, the patient had a QTc value of 323 msec and the values for the atrial-ventricular effective refractory period (AERP-VERP) pertaining to the SQTS were found to be short, i.e., 150 msec and 160 msec, respectively. It was observed that the patient easily entered AF based on a programmed stimuli delivered through the right atrium catheter (Fig.1b). The six-step ventricular tachycardia (VT) stimulation protocol was started in such a way that the ventricular catheter was placed first on the right ventricle (RV) apex and then on the RV outflow tract. Upon a triple stimulus at the RV apex, polymorphic VT and VF suddenly developed (Fig. 2a). The VF attack was terminated after the delivery of 100 joules of energy by the defibrillator. His SQTS diagnosis score of >4 (diagnosis score-7) were taken into account. A transvenous, single-chamber ICD was successfully implanted in the patient (Fig. 2b). The patient was started on Sotalol therapy; the result of the genetic study is awaited.

Discussion

According to the HRS/EHRA/APHS specialists' consensus guide, SQTS diagnosis is made if the QTc value is ≤ 330 msec. If the QTc distance is measured as <360 msec for girls and as <350 msec for boys, it is diagnosed in the presence of one or more of the following conditions: pathogenic mutation, family history of SQTS, history of sudden death below the age of 40 years, and surviving a VT/VF episode without any cardiac diseases (3,4). During EPS, AERP and VERP are typically measured to be short. The AERP and VERP values of our patient were measured to be <160 msec.

It is recommended that an ICD can be implanted as a Class I indication in short QT syndrome patients, who have survived a cardiac arrest and have symptoms such as a documented spontaneous sustained VT attack with or without syncope. ICD implantation may be considered as a Class IIb indication in asymptomatic patients with a diagnosis of SQTS and a family history of SCD. Furthermore, quinidine and sotalol treatment