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, cerebrovascular 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 sulfydryl 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

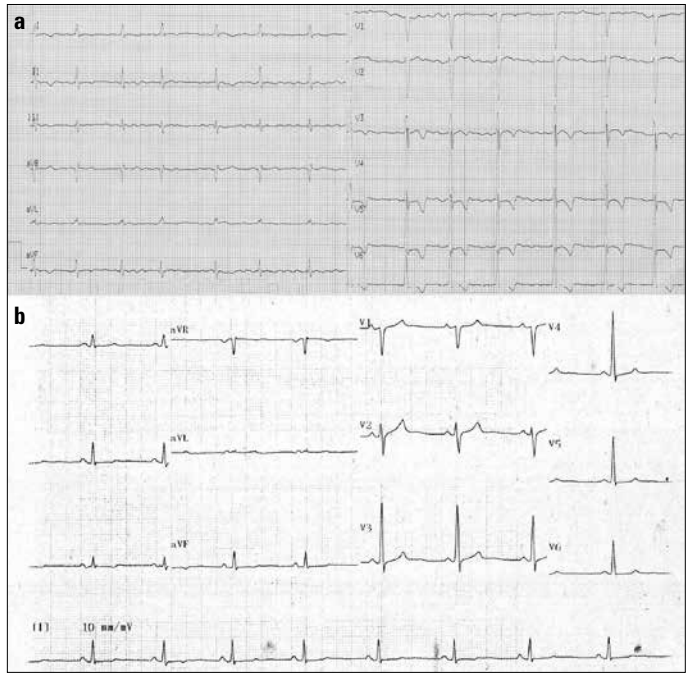

Figure 1. a, b. ECG at presentation shows rhythm to be atrial fibrillation and T-wave inversion in leads V3–V6 (a). ECG on the third day after DMPS therapy. Reversion to sinus rhythm and normalization of T wave (b)

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

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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DOI:10.5152/akd.2015.6282