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Surviving a massive sodium azide poisoning with toxic cardiomyopathy

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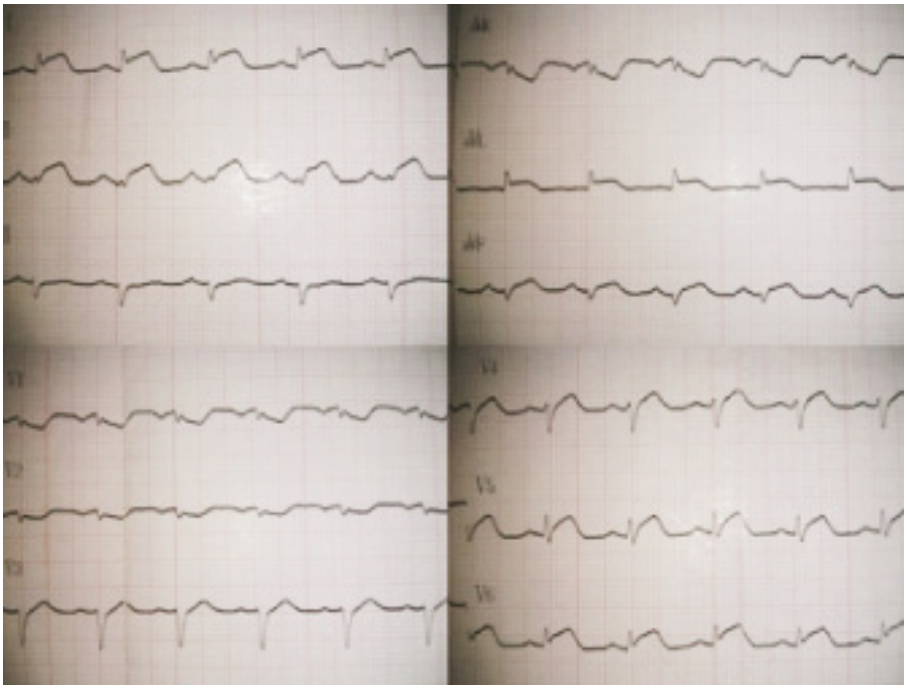
Sodium azide poisoning is rare (~50 case reports) but can be quickly fatal. A systematic review reported the fatal dose in humans to be over 10 mg/kg[1]. A 69 year-old female was admitted to our hospital for voluntary sodium azide poisoning. She ingested a massive dose of a soup spoon (15 g) of pure sodium azide powder with intention to commit suicide without any co-intoxication. Within minutes, she felt nauseous and had several vomiting. She was immediately brought to the hospital.

At admission, 2 hours after the ingestion, despite the absence of hypotension she had widespread marbles on her knees and an extended recoloration time. She had no signs of cardiac insufficiency. Diaphoresis was noticed. Biology showed a balanced metabolic acidosis with (pH of 7.48, bicarbonate 17,2 mmol/L), high blood lactate (6,6 mmol/L) indicating tissue hypoxia, elevated blood glucose (2,1 g/L), normal hypersensitive troponin. The electrocardiogram (ECG) was normal. The patient was immediately treated with gastric lavage and crystalloids. Hyperlactatemia and acidosis resolved within 12 hours.

Figure 1:
ECG showing ST-segment elevation in leads DI, DII, AVL, AVF, and V4 to V6, with reciprocal ST depression in leads AVR and V1.



On the 3rd day after the poisoning the biology showed increased hypersensitive troponine (100 ng/mL, normal < 50 ng/mL). The ECG showed a ST-segment elevation in leads DI, DII, AVL, AVF, and V4 to V6, with reciprocal ST depression in leads AVR and V1 without any chest pain (figure 1). Contemporary blood lactate increased again (4.3 mmol/L), same as liver transaminases (3 times the normal rate) and CRP (80 mg/L). The transthoracic echocardiography showed a decreased left ventricle ejection fraction (LVEF) at 30% with no dilation. Akinesis was noted in the apex, upper third of the lateral and inferior area. The left ventricle was overall hypokinetic. Medium E/E' ratio of 12.5 pointing out elevated left ventricle filling pressure. The right ventricle and the systolic arterial pulmonary pressure were normal. There was no pericardial effusion.

She was immediately transferred for emergency coronary angiography which found no clot or coronary spasm, but a 80% stenosis was noticed in the proximal section of the right coronary artery. No angioplasty was carried out. Nitrates were introduced to improve myocardial perfusion.

A cardiac MRI (Magnetic Resonance Imaging) was carried out 3 weeks after the poisoning. It showed a LVEF of 52%, a slight overall hypokinesis, no pathological enhancement, thus no myocarditis or infarct sequelae. Besides it showed a circumferential pericardial effusion of moderate volume (maximum 17 millimeters facing the left ventricle).We concluded to toxic cardiomyopathy caused by sodium azide.

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The patient's evolution during hospital stay was satisfying: the ST segment elevation on the ECG gradually decreased within a month, the hypersensitive troponine remained stable at 200 ng/mL for several weeks before normalization. The patient was discharged from the ICU 4 weeks after admission and at that time, the transthoracic echocardiography showed that the LVEF had mostly recovered (55%).

Depending on the dose ingested patients most commonly suffer from vomiting, altered consciousness, diaphoresis, acute respiratory distress, acidosis, ventricular fibrillation, hypotension and shock, cerebral edema and ultimately death[1]. Surprisingly our patient had no hypotension. On the contrary, she developed signs of vasoconstriction. A similar case of sodium azide-induced cardiomyopathy was previously reported in 1989[2]. Similarly, the cardiomyopathy appeared on the 3rd day and the most affected part was the lateral area of the left ventricle, but unlike our patient, the poisoning was fatal. Unlike Graham's study on animals[3], our patient's myocardial contractility was severely depressed. Moreover, diastolic function was also impaired. We believe that the severe stenosis on the right coronary artery was only responsible for a minor part of the myocardial damage, and sodium azide potentiated ischemia. Indeed, it inhibits the cytochrome c oxidase (or complex IV) of the mitochondrial respiratory chain, resulting in a blockage of adenosine-triphosphate synthesis. The suggested mechanism of the cardiac toxicity is an increase in intracellular calcium, due to an increased influx, which activates calpain, a protease responsible for intracellular damages causing cell apoptosis through the caspase pathway[4, 5, 6]. Despite interesting results of calcium antagonists in vitro no human case of NaN3 poisoning treated with calcium antagonist has been reported yet.

Today there is no antidote to sodium azide poisoning. We treated our patient with nitrates until the heart recovery, aiming to improve myocardial perfusion. Buys et al. reported in mice a protective effect of nitric oxide against cardiac dysfunction caused by inflammatory shock, known to interfere with mitochondrial respiration, mediated by soluble guanylate cyclase producing cyclic guanosin monophosphate[7]. Cauwels et al. found that nitrite had a protective effect on mitochondrial respiration and improved cellular energetics against TNF-mediated inhibition of complexes I and IV of the mitochondrial respiratory chain[8]. We can hypothesize that the nitrates improved the metabolic tolerance of the sodium azide toxicity on the mitochondrial chain. Further studies are needed to prove this hypothesis.

Statement of ethical publishing

The authors agree to abide by the requirements of the "Statement of publishing ethics of the International Cardiovascular Forum Journal"[9].

Conflict of interest:

The authors declares that there is no conflict of interest.

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