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BEGINNER

CASE REPORT: CLINICAL CASE

Rare Case of Selenite Poisoning Manifesting as Non–ST-Segment Elevation Myocardial Infarction

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ABSTRACT

A female patient presented with typical angina, as well as dizziness, trembling, and repeated vomiting, after accidental poisoning with sodium selenite. Our case illustrates the role of selenite in myocardial function and provides guidance for cardiovascular management of rare cases of selenite poisoning. (**Level of Difficulty: Beginner.**) (J Am Coll Cardiol Case Rep 2021;3:811–5) © 2021 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

HISTORY OF PRESENTATION

A 74-year-old female patient presented in our chest pain unit (University Hospital Heidelberg, Germany) with acute onset of angina pectoris. She regularly consumed nutrition supplements, including sodium selenite capsules as a cardiovascular preventive measure. On the day of hospitalization, she ran out of selenite capsules and took sodium selenite powder from her husband's laboratory as a substitute. The patient mistakenly consumed 125 mg, which is 1,000 times the capsule dose of 125 μg. Shortly after sodium selenite ingestion, she experienced worsening of her health condition. At first, she noted slight dizziness and trembling. Within 1 h, she had chest pain radiating to the left arm and jaw, as well as vomiting, severe nausea, and soft, frequent bowel movements. There was no collapse or syncope, fever, cough or dyspnea, and no abdominal pain. At the time of admission, the patient had bradycardia and elevated blood pressure (heart rate, 52 beats/min; blood pressure, 180/90 mm Hg).

LEARNING OBJECTIVES

- To identify myocardial injury with angina pectoris–like symptoms and elevated troponin levels as a consequence of acute selenium poisoning.
- To recognize bradycardia and hypertension as possible symptoms of acute selenium poisoning.

MEDICAL HISTORY

The patient's medical history included multiple orthopedic conditions and operations, as well as bronchial asthma, various allergies, euthyroid goiter, and Ménière's disease. Until the day of admission, the patient was not known to have any cardiovascular diseases. Her treatment included levothyroxine, sulpiride, and food supplements such as Ginkgo biloba.

DIFFERENTIAL DIAGNOSIS

There are important differential diagnoses for thoracic pain, ranging from life-threatening

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ABBREVIATIONS AND ACRONYMS

ECG = electrocardiogram

hsTnT = high-sensitivity troponin T

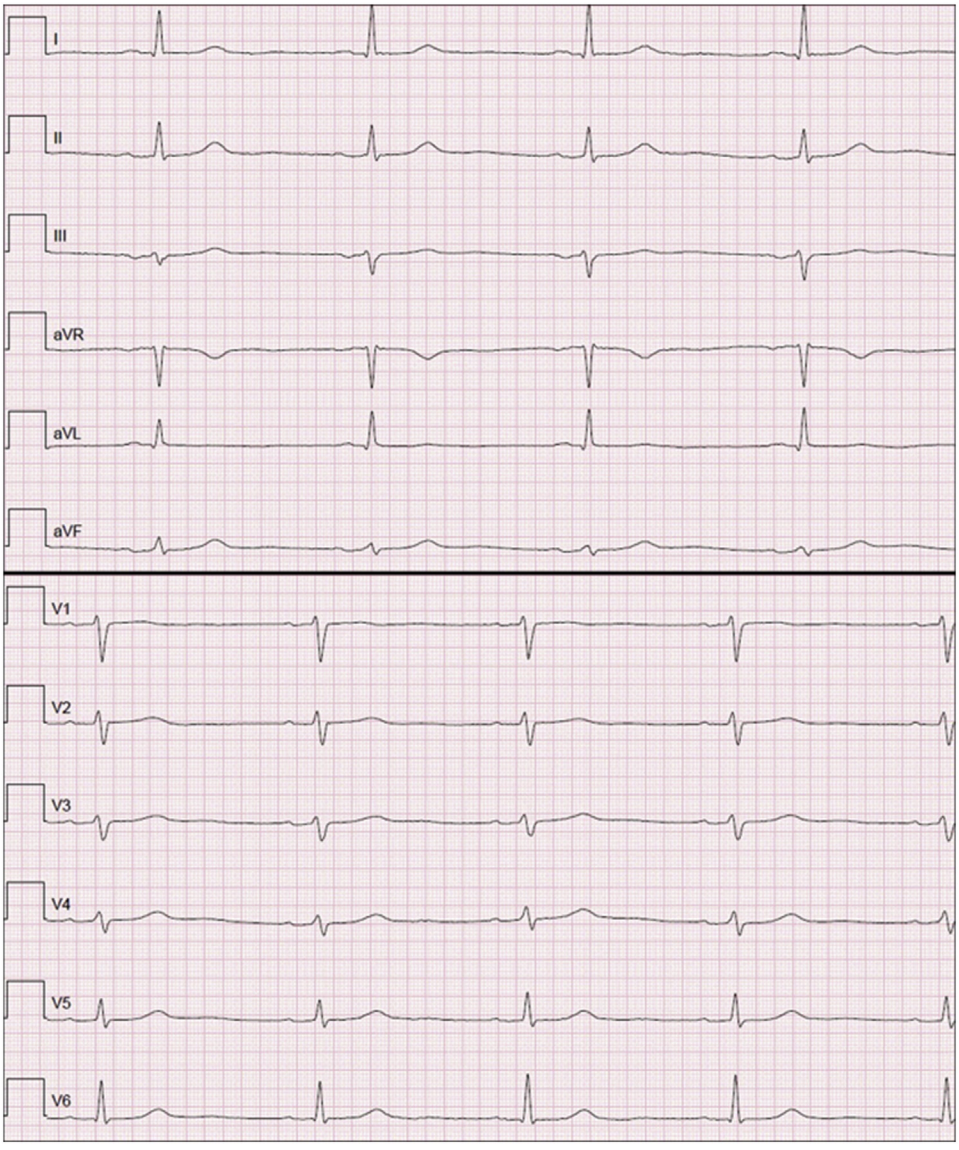
conditions such as acute coronary syndrome or pulmonary embolism to more harmless causes such as intercostal neuralgia.

INVESTIGATIONS

After admission to the chest pain unit, an electrocardiogram (ECG) and troponin assessment were performed. There were no signs of ischemia on the ECG (Figure 1). At first blood draw, the high-sensitivity troponin T (hsTnT) level was 56 ng/l, rising to 216 ng/l at the 3-h control, thus indicating an acute non-ST-segment elevation myocardial infarction or an acute myocardial injury secondary to severe selenium poisoning. No inflammatory constellation and signs of liver or kidney damage were found in the blood tests.

Given that the patient had reservations about undergoing coronary angiography and the symptoms were clearly associated with the selenium

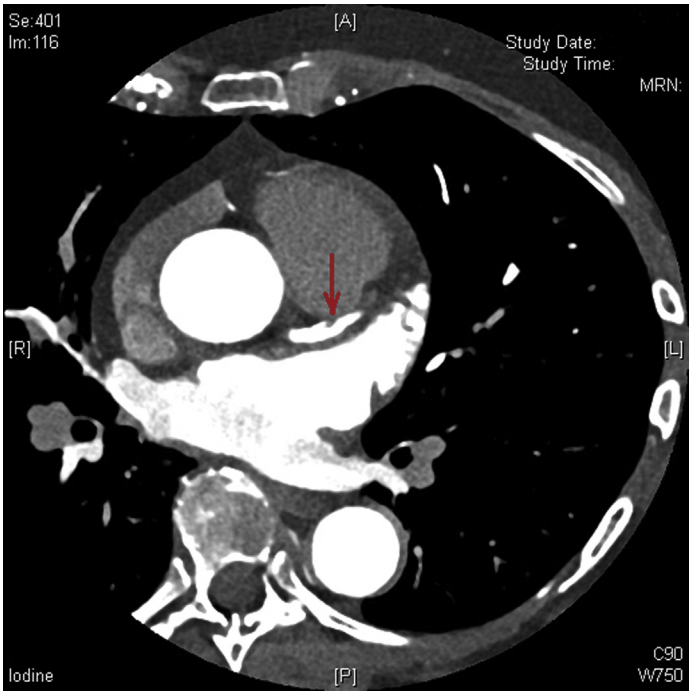
FIGURE 1 12-Lead Electrocardiogram on Admission



The following are shown: sinus bradycardia, 51 beats/min; PQ interval, 166 ms; QRS complex, 89 ms; and QT (QTc), 436 (413) ms.

intoxication, coronary computed tomography was performed. The imaging showed only mild to moderate isolated left anterior descending artery disease (**Figure 2**). The patient explicitly rejected further invasive examination, and cardiac magnetic resonance had to be aborted before administration of the contrast agent because of claustrophobia. In the acquired sequences (**Figure 3**) a normal-sized left ventricle with globally normal systolic function was found, concordant with echocardiography. Pulmonary function testing did not reveal any relevant findings, except for a slightly limited diffusion capacity. Upper abdomen sonography showed fatty liver disease and a renal cyst on the left side.

FIGURE 2 Coronary Computed Tomography Angiogram Revealing Mild to Moderate Isolated Left Anterior Descending Coronary Artery Disease



The area of 50% stenosis is marked with an **arrow**. A = anterior; L = left; P = posterior; R = right.

MANAGEMENT

Directly after admission, the poison information center in Freiburg (Germany) was consulted. According to the colleagues at the center, our patient's symptoms were in potential agreement with severe selenium intoxication. Because intake had occurred 2 h earlier and the patient had spontaneously vomited multiple times, no specific measures were available. The patient was monitored until she was free of symptoms. In addition, serial troponin and selenium serum measurements were made (**Figures 4A and 4B**).

The patient showed rapid clinical improvement and a good level of mobility. Her initial muscle complaints and concentration issues regressed.

With a spontaneous and significant drop in troponin, we adjudicated the event as acute myocardial injury in the context of sodium selenite poisoning. Finally, hsTnT normalized at day 9, and the patient was free of any symptoms. Besides sinus bradycardia, no arrhythmic events occurred during monitoring of this patient. Because single-vessel coronary artery disease was detected, aspirin and atorvastatin were administered. The drugs were well tolerated by the patient.

DISCUSSION

Selenium is a trace element that plays an important role in the regulation of the immune system and cardiovascular protection because of its pronounced antioxidant effect (1). Unfortunately, the assumed cancer-preventive effects of selenium could not be verified. Furthermore, the SELECT (Selenium and Vitamin E Cancer Prevention Trial) found a possible association between selenium supplements and an increased risk of type 2 diabetes (2).

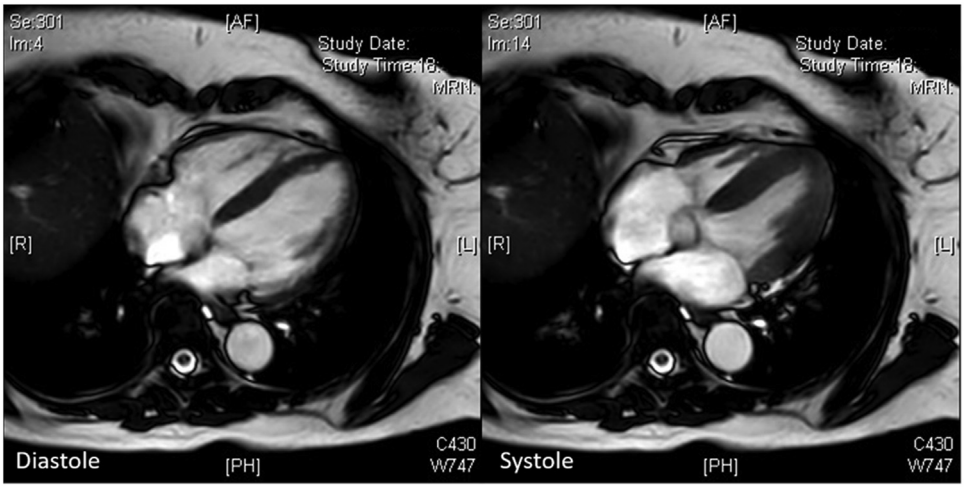
Selenium deficiency leads to impairment of the immune system and muscle function and, in extreme cases, to Keshan disease (a rapidly progressive cardiomyopathy) or Kashin-Beck disease (a type of osteochondropathy) (3). Long-term selenium overdose leads to selenosis, with a garlic smell of the breath, hair loss, and typical nail changes (4).

Only few cases of acute selenium poisoning in humans have been described so far. Patients were reported to develop tachycardia, hypotension, gastroenteritis, hemorrhages, nonspecific ECG changes (prolonged QT interval, atrioventricular nodal block, frequent premature ventricular complexes, and ST-wave changes), and deterioration of cardiac function, including reduced ejection fraction and cardiovascular collapse (5). In case reports of acute selenium intoxication in animals, severe myocardial injury with elevated troponin levels and microscopic changes of myocardium such as edema or fibrosis were repeatedly described (6).

There is evidence that selenium in high concentrations displaces sulfur from organic compounds such as sulfhydryl enzymes, which are particularly important for the oxidative reactions in cellular respiration, thus leading to cell damage and death (7).

A case was reported of a patient with selenium blood concentration of 1,430 ng/ml (18.161 μmol/l)

FIGURE 3 Cardiac Magnetic Resonance Showing Normal-Sized Left Ventricle With Globally Normal Systolic Function

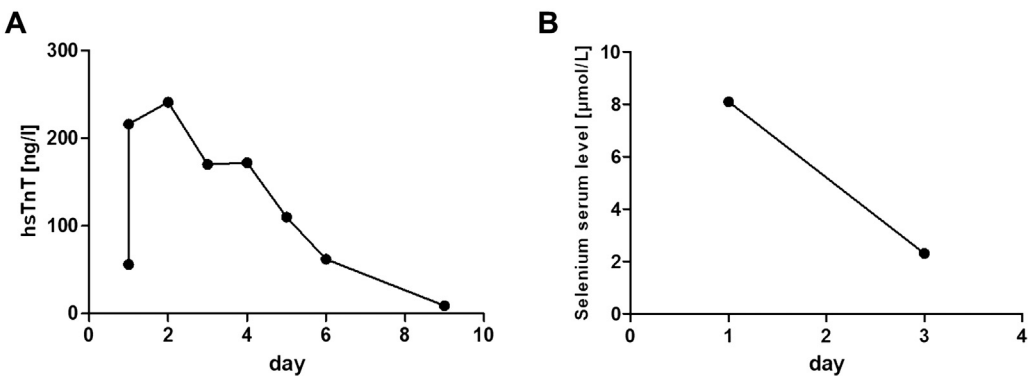


(Left) Diastole. **(Right)** Systole. AF = anterior-foot; L = left; PH = posterior-head; R = right.

who developed diarrhea and vomiting and who subsequently died of pronounced heart failure. The autopsy showed massive lung congestion, focal fibromatosis of the heart, and cerebral edema. Other cases of patients ingesting lower selenium concentration reported nonlethal symptoms (4). In most patients, symptoms develop triphasically: first gastrointestinal complaints, later muscle weakness, and finally cardiopulmonary complaints with multiorgan failure and death (5).

The German Society for Nutrition recommends an intake of 60 μg selenium/day for adult women. According to the European Food Safety Authority, 300 μg of selenium/day is a dose tolerable for adults (8). Thus, our patient took 400 times the upper tolerable dosage, and we detected a selenium serum concentration of 8.10 μmol/l (639.9 ng/ml) shortly after ingestion. Her symptoms were in accordance with the triphasic course described earlier, except for pulmonary symptoms. Troponin first showed a significant elevation from 56 to 241 ng/l, thereby indicating myocardial damage and correlating with angina pectoris, and it then decreased rapidly according to the decrease of selenium concentration in

FIGURE 4 Troponin and Selenium Concentrations After Admission



(A) Troponin and **(B)** selenium serum concentrations after admission (high-sensitivity troponin [hsTnT] normal range <14 ng/l; normal range of selenium serum concentration, 0.75 to 1.8 μmol/l).

the blood (**Figures 4A and 4B**). Although it cannot be ruled out that the troponin elevation in our patient was also caused by pre-existing coronary artery disease, given the only moderate plaque size and a clear time-symptom correlation between selenium intake and angina pectoris symptoms, we assumed that intoxication was the leading cause of cardiac symptoms in this patient. This diagnosis was confirmed by the finding that the symptoms improved after reduction of the serum selenium concentration and did not recur during exercise both in the hospital and at the time of follow-up despite persistent plaque in the left anterior descending artery.

Our patient also presented with hypertension as well as bradycardia, which to our knowledge has not been described at the beginning of acute poisoning so far. Here a "U-shaped response curve" may play a role, with extremely high (in fatal poisoning cases) or low (Keshan disease) blood selenium concentrations leading to tachyarrhythmias, whereas the concentrations in between could possibly exert antiarrhythmic and bradycardic effects. In addition, a certain synergistic effect of *Ginkgo biloba* through irreversible HCN-channel blockage cannot be ruled out (9).

Because there are no antidotes, and the chelators seem to increase the toxicity of selenium, the therapy of choice consists of stopping the exposure and alleviating symptoms. Recommendations for emesis induction are cautious, especially if a caustic compound such as selenite has been consumed (4). In accordance with this, we treated our patient conservatively under strict monitoring.

FOLLOW-UP

The patient was clinically stable at 1-month follow-up, without any symptoms presented after the ingestion. Furthermore, there were no symptoms of selenosis.

CONCLUSIONS

In this case report, the clinical presentation and management of a patient after acute sodium selenite intoxication with subsequent severe damage to the heart muscle are described. We confirm that this case report received proper ethical oversight.

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Dr. Meder received funding from the Informatics for Life, Heidelberg University Hospital, Im Neuenheimer Feld 669, 69120 Heidelberg, Germany. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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KEY WORDS chest pain, drug abuse, myocardial infarction