Accidental death from acute selenium poisoning

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We report a fatal case of acute selenium poisoning in a 75-year-old man. After reading on the Internet about a possible role of selenium in prostate cancer, the patient ingested 10 g of sodium selenite. Despite intensive care treatment, he suffered a cardiac arrest and died 6 hours after ingestion. This case illustrates the risks of failing to critically evaluate Internet information and exposes the myth that natural therapies are inherently safe. ***(MJA 2006; 185: 388-389)***

Clinical record

After a single test showing a raised level of prostate-specific antigen, a 75-year-old man became concerned about prostate cancer. Without confirmation of the diagnosis, he researched prostate cancer on the Internet and discovered that selenium may have a role in its prevention and treatment. He purchased sodium selenite powder and tablets from two separate pharmacies for supplementation.

He presented to the emergency department with vomiting and diarrhoea at 10:30 am, 3.5 hours after ingesting 10 g of sodium selenite (purity, 96%). He had significant abdominal pain, poor perfusion, and hypotension, with a pulse rate of 76 beats per minute, blood pressure of 75/45 mmHg, and prolonged QT interval. Arterial blood gas examination (breathing 60% O_2) showed pH, 7.325 (reference range [RR], 7.35–7.45); PO_2, 122 mmHg (RR, 85–100 mmHg); PCO_2, 22.4 mmHg (RR, 35–45 mmHg); and HCO_3 concentration, 11 mmol/L (RR, 22–28 mmol/L). He also had hypokalaemia (serum potassium concentration, 3.4 mmol/L [RR, 3.5–5.0 mmol/L]). A blood specimen collected at this time later showed a serum selenium level of 68.0 μmol/L (RR, 0.6–2.3 μmol/L).

The patient was transferred to the intensive care unit, where fluid resuscitation was continued, and a dobutamine infusion was begun. He remained hypotensive (blood pressure, 85/50 mmHg) but conscious. At 12:10, he developed significant ventricular tachycardia, interspersed with normal complexes with ST depression. Repeat blood gas tests showed worsening acidosis and hypokalaemia. Magnesium (10 mmol) and potassium (25 mmol) were administered intravenously over 40 minutes. Dysrhythmia persisted and worsened. Lignocaine was added with no response. The patient suffered a cardiac arrest, and cardiopulmonary resuscitation was begun. Arterial blood gas analysis performed during the arrest revealed a sudden rise in serum potassium level, to 8.8 mmol/L. Throughout the arrest, rhythms varied, from asystole to broad complex bradycardia to some narrow complexes with an output. Despite cardiopulmonary resuscitation, asystole became permanent and unresponsive. The patient's pupils became fixed and dilated, and cardiopulmonary resuscitation was ceased.

The case was referred to the Coroner who confirmed the cause of death to be acute selenium toxicity.

Discussion

The growth in the use of complementary and alternative medicine in Australia has been well documented.[1] Selenium is an essential trace element and an important constituent of the antioxidant glutathione peroxidase.[2,3] It is found in many foods, including seafood, grains and eggs,[2] and dietary intake is usually 20–300 μg/day.[4] An intake below 400 μg/day is considered safe for almost all individuals.[5] In Australia, selenium tablets are marketed as a health supplement, while sodium selenite powder is mainly used as a livestock supplement for animals grazing on selenium-deficient soil. Selenium has been discussed in many epidemiological investigations into prevention and treatment of cancer, especially prostate carcinoma.[6,7]

Excessive doses of selenium result in intoxication, which is characterised by continuous vomiting, garlicky breath, mucosal irritation, abdominal pain, hypersalivation, haemolysis, necrosis of the liver, cerebral and pulmonary oedema, coma and death.[3,8] Selenium presents a nutritional conundrum because of its dual status as an essential but highly toxic trace element.[9]

The exact mechanism of selenium toxicity is as yet unknown.[8,10] It has been suggested that the ready substitution of selenium for sulfur in biochemical reactions may inactivate the sulfhydryl enzymes necessary for oxidative reactions in cellular respiration,[10] contributing to the acute toxic effect.

Cases of selenium poisoning are rare, with Gasmi et al citing only 18 documented cases before 1997, half of them fatal, generally as a result of cardiocirculatory failure and/or pulmonary oedema.[11] Although there is no defined point at which selenium becomes toxic, our patient consumed about 10 g of sodium selenite and, 4 hours after ingestion, had a serum level of 68.0 μmol/L (RR, 0.6–2.3 μmol/L), which proved fatal. The ingested quantity of 10 g was about 10 000 times the recommended daily dose of supplemental selenium. It is almost impossible to rapidly reduce a patient's serum selenium level, as absorbed selenium is mostly contained in erythrocytes or bound to α- or β-globulins. For this reason, haemodialysis is not an established treatment for selenium intoxication, but may reduce serum selenium levels slightly.[8]

In regard to our patient's hyperkalaemia, the extracellular potassium level is expected to increase in metabolic acidosis. However, our patient showed a progressively worsening metabolic acidosis with a falling potassium level. The administration of 25 mmoles of potassium over 40 minutes would be expected to raise the serum potassium level, but did not account for the sudden and dramatic rise, from 3.2 mmol/L to 8.8 mmol/L. This sudden hyperkalaemia has not been described in previous cases of selenium toxicity.

A brief Internet search revealed 287 000 sites discussing the use of selenium in prevention and treatment of prostate cancer. This provides the public with large amounts of information that is not critically evaluated for validity. After reading Internet information on the possible link between selenium and prevention and treatment of prostate cancer, our patient was able to purchase 200 g of sodium

selenite powder without adequate instructions. He selected a dose himself, with catastrophic consequences.

This case highlights the risks associated with failure to critically evaluate Internet material and exposes the myth that natural therapies are inherently safe. Internet sites which fail to disclose the potentially fatal effects of advocated treatments are an emerging threat to health. The World Health Organization has devised guidelines to help consumers evaluate medical information on the Internet, which are available online through the Therapeutic Goods Administration.[12] Adverse outcomes of complementary and alternative medicines should be better publicised and more stringently reported to the Adverse Drug Reactions Advisory Committee (ADRAC), in tandem with adverse outcomes of conventional medications, to create a database of side effects of all current therapies.

Competing interests

None identified.

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(Received 16 Feb 2006, accepted 24 Aug 2006) ❑