Acute Tellurium Toxicity From Ingestion of Metal-Oxidizing Solutions

Mark C. Yarema, MD*‡, and Steven C. Curry, MD*‡§

ABSTRACT. Tellurium is an element used in the vulcanization of rubber and in metal-oxidizing solutions to blacken or tarnish metals. Descriptions of human toxicity from tellurium ingestion are rare. We report the clinical course of 2 children who ingested metal-oxidizing solutions containing substantial concentrations of tellurium. Clinical features included vomiting, black discoloration of the oral mucosa, and a garlic odor to the breath. One patient developed corrosive injury to the esophagus secondary to the high concentration of hydrochloric acid in the solution. Both patients recovered without serious sequelae, which is typical of tellurium toxicity. An awareness of situations in which children may be exposed to tellurium and its clinical presentation may assist clinicians in the diagnosis of this rare poisoning.


ABBREVIATIONS. HR, heart rate; RR, respiratory rate; BP, blood pressure.

Tellurium is a naturally occurring element found most commonly as a byproduct from the electrolytic refining of copper. Its main uses are in the vulcanization of rubber (in which it increases resistance to heat, abrasion, and aging) and in alloys of copper, steel, lead, and bronze (by making them more resistant to corrosion). In addition, tellurium is used in metal-oxidizing solutions to blacken or tarnish metals (eg, in jewelry manufacturing). Historically, tellurium was used in the treatment of syphilis and leprosy.

Acute human ingestions of tellurium are rare. The majority of descriptions of toxicity from tellurium are either from intravenous or inhalational exposure.1–4 We present 2 cases of toxicity in young children from ingestion of metal-oxidizing solutions that contained substantial concentrations of tellurium.

CASE REPORTS

Case 1
A 20-month-old boy, weighing 11.2 kg, was noted by his father to ingest an unknown quantity of Silver-Black metal-oxidizing solution (Fig 1). There was no child-proof cap on the bottle. His father immediately washed the child’s face and hands. The child then experienced several episodes of hematemesis and developed progressive odynophagia, followed by a refusal to swallow. He was taken to the emergency department.

On arrival at the referring hospital 1 hour postingestion, his vital signs were: heart rate (HR), 123 beats per minute; respiratory rate (RR), 22 respirations per minute; oxygen saturation, 96% on room air; and temperature, 36.5°C (97.7°F). Blood pressure (BP) was not measured. His episodes of emesis continued. Burns were noted to his upper and lower lips. No comment was made about discoloration to his oral mucosa. The remainder of his examination was normal. He was rehydrated with intravenous normal saline and transferred to our children’s hospital for additional management.

On examination 7 hours postingestion, his vital signs were: BP, 92/73 mm Hg; HR, 126 beats per minute; RR, 24 respirations per minute; and temperature, 36.5°C (97.7°F). His emesis had resolved, and he appeared well hydrated. Burns were noted to his upper and lower lips. A distinct garlic odor to his breath was
Clinical features of acute tellurium toxicity include a metallic taste, nausea, vomiting, blackened oral mucosa, and corrosive gastrointestinal tract injury from acidic solvents. Although tellurium-induced inhibition of sweating has been described, we were unable to locate any objective studies or measurements documenting this symptom in the scientific literature. The discoloration of the oral mucosa and skin is thought to be a result of deposition of elemental tellurium in the dermis and subcutaneous tissue and may involve the webs of the fingers as well as the neck after topical contact. Our patients exhibited many of the characteristic features of tellurium toxicity, namely, vomiting, garlic odor of the breath, blackened oral mucosa, and benign clinical course. The caustic injury noted on upper gastrointestinal endoscopy in patient 1 was likely related to the low pH of the solution secondary to the high concentration of hydrochloric acid.

Toxicity from exposure to hydrogen telluride gas is different from other forms of tellurium. Small exposures may result in only mucous membrane and pulmonary irritation. In animals, large exposures to hydrogen telluride have resulted in hemolysis, hemoglobinuria, anuria, jaundice, and pulmonary edema, a presentation similar to toxicity from inhalation of arsine or stibine.

Fatalities from tellurium exposure are rare. Keall et al reported 3 poisonings in individuals accidentally injected with sodium tellurite during retrograde pyelography. Two of these patients died. Their clinical course before death comprised a garlic odor, cyanosis, vomiting, loss of consciousness, and apnea.

Tellurium, selenium, and, to a lesser extent, arsenic toxicity are characterized by a garlic odor secondary to their respective metabolites (dimethyl telluride, dimethyl selenide, monomethyl and dimethyl arsenic acid). After the ingestion of alcohol, the garlic-breath odor of tellurium has been stated to increase from the formation of ethyl telluride. The differential diagnosis of tellurium toxicity includes toxicity from selenium and arsenic. Similar to tellurium salts, acute ingestions of arsenic salts or selenium salts also produce emesis and a garlic odor. The corrosive action of the acid solvent commonly used in tellurium-containing metal-darkening solutions could mimic the more severe gastroenteritis and shock accompanying severe arsenic poisoning. Pulmonary edema, coma, respiratory failure, hypotension, and death may be seen in children who accidentally ingest as little as 5 mL of gun-bluing solutions containing 2% seleniumic acid. A garlic odor may also result from exposures to organic and inorganic phosphorous compounds, lewisite, pyridine, dimethyl sulfoxide, and garlic.

Treatment of tellurium toxicity is supportive. Amdur reported the use of 2,3-dimercaptopropanol (British antilewisite) for chelation of 3 patients with inhalational tellurium toxicity. However, the diagnosis was delayed for at least 48 hours. By this time their symptoms included only mild headache, epigastric pain, and garlic odor to the breath and sweat. No benefit from chelation in these patients was demonstrated. Ascorbic acid may remove the garlic odor by reducing tellurite and tellurate to elemental tellurium; however, it is not commonly recommended.
Patients exposed to tellurium must be informed that the garlic odor to their breath and urine may persist for several weeks or months after exposure. Reisert\(^\text{10}\) reported the persistence of a garlic-breath odor 237 days after ingestion of 3 doses of 5 mg of tellurous oxide over the course of 6 hours. Similarly, Muller et al\(^\text{6}\) reported a persistent garlic-breath odor for 10 months after ingestion of a small amount of meat contaminated with 800 to 1000 μg/kg of tellurium. His patient also developed temporary alopecia. To our knowledge, alopecia has not been otherwise associated with tellurium toxicity.

CONCLUSIONS

The clinical courses of 2 children accidentally exposed to tellurium in metal-oxidizing solutions are presented. Clinical features included vomiting, black discoloration of the oral mucosa, and a garlic odor to the breath. Both patients had a benign clinical course typical of tellurium toxicity. An awareness of situations in which people may be exposed to tellurium and its clinical presentation may assist clinicians in the diagnosis of this rare poisoning.

REFERENCES

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*Pediatrics* 2005;116;e319
DOI: 10.1542/peds.2005-0172 originally published online July 1, 2005;
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