Surgical Removal of Cylindrical Batteries 6 Years after Ingestion

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Ingestion of batteries is a well known toxicological entity involving mainly disk batteries in pediatric patients [1]. Early detection is common in such cases and these patients are usually managed soon after ingestion. In contrast, ingestion of cylindrical alkaline batteries is usually intentional and mostly occurs in psychiatric patients [2]. Most reports describe spontaneous expulsion of these batteries after a few days in the gastrointestinal tract [3]. Longer retention is very uncommon and presents a challenging diagnosis requiring a high level of suspicion. We report the unique case of a drug packer and former prison inmate who had cylindrical alkaline batteries in his stomach allegedly for 6 years.

Patient Description

A 38 year old man with a history of drug abuse and previous incarceration presented to a local community clinic with complaints of intermittent diffuse abdominal discomfort of several months duration, and occasional heartburn after meals during the last few years. His medical history included G6PD deficiency and a snakebite (Vipera palaestinae) in childhood. He reported ingesting batteries a few years previously.

Physical examination was unremarkable. An abdominal X-ray revealed no pathology. Since it did not include the upper abdomen, a second X-ray was performed which revealed a radiopaque mass in the stomach compatible with a metal object, possibly batteries [Figure A]. The patient was referred to the emergency department of our hospital. On admission he was alert and complained of abdominal pain. He reported that 6 years earlier, during a leave from prison, he had prepared packets of drugs of abuse and swallowed them for later use in prison. Several days later, when realizing that the drug packets did not emerge, he followed the advice of his fellow inmates and swallowed two pairs of cylindrical alkaline batteries, each pair wrapped in a plastic bag. The rationale was to push the drug packets down the gastrointestinal tract using the weight of the batteries. All drug packets were expelled within several days, as well as at least one pair of batteries. The patient did not verify that all batteries had been expelled. Since then he had self-limited non-progressing heartburn after meals.

Several months prior to admission he began to suffer from abdominal discomfort. The discomfort was mild, diffuse and intermittent and did not radiate. He denied nausea, vomiting, diarrhea and dysuria. He recalled having problems while passing through a metal detector in the airport several months before admission. He noted that the recent appearance of pain as well as the metal detector alarm led him to suspect that some of the batteries he ingested years earlier may not have been expelled, and he decided to seek medical attention.

Blood pressure was 123/83 mmHg, pulse 89/minute regular and temperature 36.9°C. The abdomen was not tender, not distended,
Toxicology

and without any palpable mass and normal peristalsis. Rectal examination was normal. The rest of the physical examination was unremarkable. Hematocrit was 37.4%, white blood cell count 12,200/µl and platelets 222,000/µl. Glucose, creatinine, blood urea nitrogen, K⁺, Na⁺ and amylase were within normal limits.

On gastric endoscopy two cylindrical batteries wrapped together in a thin plastic bag were visualized at the antrum. No sign of leakage could be identified and the stomach looked intact. An attempt was made to retrieve the batteries with forceps but the plastic sheath began to tear and the procedure was discontinued. Because of possible damage to the batteries from the gastric acidity, it was decided to proceed with surgical removal. Laparoscopy was performed under general anesthesia and the two wrapped batteries were removed from the stomach. A biopsy from the margins of the antral gastrointestinal tract was taken. After removing the plastic wrap two rusty VARTA AA batteries were retrieved [Figure B]. Gastric biopsy revealed mild chronic inflammation. The postoperative course was unremarkable. Five days later the patient was discharged without sequelae to ambulatory follow-up and with resolution of the initial problem.

Comment

This report is about a unique case of cylindrical battery ingestion. The patient intentionally ingested two pairs of batteries wrapped in a plastic bag to serve as a weight in order to push down and promote the movement of drug packets along the gastrointestinal tract. To the best of our knowledge such practice has not been reported before. We are not aware of any report on batteries retained in the stomach for a duration of 6 years. During this time the patient had mild symptoms but did not seek medical assistance until abdominal discomfort developed and the alarm sounded on passage through a metal detector.

It is unclear why clinical manifestations developed so slowly. It can be assumed that the plastic bag protected the batteries from the corroding effect of the gastric acidity and at the same time protected the gastric mucosa from a possible leak. However, intermittent leakage of the alkaline content (suggested by the rusty appearance of the batteries) or prolonged pressure on the gastric wall at the antrum cannot be ruled out. These two factors could explain the mild chronic inflammation found in the antral biopsy.

Alkaline batteries contain metals (e.g., zinc, manganese, mercury) and an alkaline electrolyte solution (e.g., potassium hydroxide). These batteries are not resistant and leakage can cause corrosive injury in the form of erosions, ulcers and even perforations [2]. Metal toxicity and hypersensitivity is another possible risk [1]. Disk batteries can contain mercury as well as lithium and manganese, but overt toxicity is unlikely after ingestion due to its resistant cover. Elevated blood and urine levels of mercury are rare and can be observed when batteries split and radiopaque droplets are evident in the gut. Other mechanisms of injury are pressure necrosis, esophageal lodging and aspiration [1]. Litovitz and Schmitz [1] reported a series of 2382 cases where cylindrical and button batteries were ingested. They concluded that early symptoms and diameter are unreliable indicators for esophageal lodging.

The passage of sharp or large objects (e.g., alkaline batteries) is more likely to injure the gastrointestinal tract. It is estimated that foreign objects wider than 2.5 cm or longer then 6 cm in adults, and objects wider than 2 cm and longer then 4 cm in children are more likely to be retained in the stomach [3]. The failure of passage or appearance of symptoms and signs such as abdominal pain, obstruction, bleeding or perforation indicates invasive intervention [2].

The diagnosis of ingestion of batteries and other foreign bodies in adults is challenging. The diagnosis is suspected in drug abusers, prisoners and psychiatric patients. O'Sullivan et al. [4] reviewed 36 cases of deliberate foreign body ingestion by prisoners or psychiatric patients, 30 of whom were institutionalized at the time of ingestion. Although most objects passed spontaneously without complications, upper gastrointestinal endoscopy was required in four patients and an intragastric foreign body was identified in two patients. Laparotomy was performed in two patients for unresolving mechanical intestinal obstruction [4]. Lim and co-workers [2] reported the case of a 60 year old patient with a 10 day history of nausea, vomiting and epigastric pain. Endoscopy revealed two 3 volt Duracell batteries in the stomach, later retrieved, as well as multiple deep ulcers. Inspection of the batteries revealed visible leakage from the copper top and destruction of the grommet seals and of the positive terminals. The psychiatric diagnosis was factitious disorder with the secondary gain of obtaining medical care [2]. It is important to consider foreign body ingestion in the differential diagnosis of abdominal pain of unknown etiology [3].

Long-term retention of a foreign body in the gastrointestinal tract is rarely encountered. Misra et al. [5] reported the case of a 46 year old clerk who complained of abdominal pain. An abdominal X-ray revealed a needle in the stomach, which was accidentally ingested 32 years previously. It was removed endoscopically.

The patient's report that the batteries had been retained in the stomach for 6 years seemed to be reliable. His story was consistent, he made no attempt to conceal his past, and the metal detector alarm incident in the past all support the possibility of obtaining medical care [2]. It is important to consider foreign body ingestion in the differential diagnosis of abdominal pain of unknown etiology [3].

Once the diagnosis of battery ingestion is suggested it can be easily confirmed by adequate X-rays of the neck, chest and abdomen as they are radiopaque. Failure to perform adequate imaging can result in delayed diagnosis and treatment. Intentional or late diagnosed foreign body ingestion can cause serious problems (e.g., perforation). For these cases, the invasive approach including endoscopy or surgical removal is warranted [3,4].

In conclusion, we report an uncommon case of a prisoner who intentionally ingested cylindrical alkaline batteries to promote gastrointestinal passage of drug packets. The batteries were retained in the stomach for 6 years and were eventually removed...
Cylindrical Battery Ingestion
by laparoscopy. Intentional ingestion of batteries and other foreign objects should be suspected in drug abusers, prisoners and psychiatric patients presenting with abdominal manifestations, especially if they are of prolonged duration. Battery ingestion can result in corrosive injury or mechanical pressure on the mucosa. Gastric injury can be expected even if the batteries are wrapped in a plastic bag or sheet.

References

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Imatinib Mesylate® (Gleevec) is an inhibitor of the tyrosine kinase domain of the Bcr-Abl oncoprotein and prevents the phosphorylation of the kinase substrate by ATP. This agent is mainly used to treat chronic myelogenous leukemia. Recent basic science and animal data have suggested its effect in systemic sclerosis (SSc), a chronic systemic autoimmune disorder that is characterized by vasculopathy, which leads to accumulation of extracellular matrix in skin and viscera, and thus causes fibrosis. Pathogenesis includes overproduction and accumulation of collagen and extracellular matrix protein such as fibronectin, tenasin, fibrillin-1, and glycosaminoglycans. Humoral immunity, one of the features associated with pathogenesis, involves production of a number of cytokines, including transforming growth factor-beta (TGFβ). TGFβ has many roles, including stimulation of fibrosis. Known as one of the major players in SSc, TGFβ binds to serine threonine c-Abl kinase receptor on the surface of fibroblasts, phosphorylates and Smad1 protein, which activates transcription of CCN2-DNA sequence encoding for connective tissue growth factor A. It has been demonstrated that Imatinib mesylate strongly inhibits the synthesis of major extracellular matrix protein by dermal fibroblasts and efficiently prevented the development of fibrosis in the mouse model of bleomycin-induced dermal and pulmonary fibrosis (Arthritis Rheum 2008;58:2528). Smad 1 pathway is activated in SSc fibroblasts and is targeted by Imatinib mesylate. A number of in vitro studies evaluating the role of Smad1 signalling in systemic fibrosis concluded that activation of Smad1 signalling occurs in a subset of SSc patients and contributes to the persistent activation of SSc fibroblasts. It was also shown that the Smad1/CCN2 pathway is blocked by imatinib mesylate and that c-Abl is required for the activation of Smad1 pathway in SSc fibrosis. TGFβ was shown to stimulate CCN2 promoter activity. In conclusion, this study suggests that SSc patients with activated Smad1 signalling may benefit from Imatinib mesylate treatment. In two known cases, Imatinib mesylate was used as an antifibrotic agent to treat patients with severe lung fibrosis. In one, with therapy-resistant and progressive SSc as well as pulmonary involvement, the skin tightness subsided and the pulmonary involvement stabilized after treatment with Imatinib mesylate. (p. 2549). The second patient had mixed connective tissue disease and pulmonary involvement, and showed a significant improvement in several outcome measurements following treatment with Imatinib mesylate (p. 2538).

Rozalia Tverskaya

Give me the luxuries of life and I will willingly do without the necessities
Frank Lloyd Wright (1867-1919), American architect, interior designer, writer and educator. Wright promoted organic architecture. Already well-known during his lifetime, he was recognized in 1991 by the American Institute of Architects as "the greatest American architect of all time."

Capsule
Mechanism and antifibrotic effect of Imatinib mesylate
Imatinib Mesylate® (Gleevec) is an inhibitor of the tyrosine kinase domain of the Bcr-Abl oncoprotein and prevents the phosphorylation of the kinase substrate by ATP. This agent is mainly used to treat chronic myelogenous leukemia. Recent basic science and animal data have suggested its effect in systemic sclerosis (SSc), a chronic systemic autoimmune disorder that is characterized by vasculopathy, which leads to accumulation of extracellular matrix in skin and viscera, and thus causes fibrosis. Pathogenesis includes overproduction and accumulation of collagen and extracellular matrix protein such as fibronectin, tenasin, fibrillin-1, and glycosaminoglycans. Humoral immunity, one of the features associated with pathogenesis, involves production of a number of cytokines, including transforming growth factor-beta (TGFβ). TGFβ has many roles, including stimulation of fibrosis. Known as one of the major players in SSc, TGFβ binds to serine threonine c-Abl kinase receptor on the surface of fibroblasts, phosphorylates and Smad1 protein, which activates transcription of CCN2-DNA sequence encoding for connective tissue growth factor A. It has been demonstrated that Imatinib mesylate strongly inhibits the synthesis of major extracellular matrix protein by dermal fibroblasts and efficiently prevented the development of fibrosis in the mouse model of bleomycin-induced dermal and pulmonary fibrosis (Arthritis Rheum 2008;58:2528). Smad 1 pathway is activated in SSc fibroblasts and is targeted by Imatinib mesylate. A number of in vitro studies evaluating the role of Smad1 signalling in systemic fibrosis concluded that activation of Smad1 signalling occurs in a subset of SSc patients and contributes to the persistent activation of SSc fibroblasts. It was also shown that the Smad1/CCN2 pathway is blocked by imatinib mesylate and that c-Abl is required for the activation of Smad1 pathway in SSc fibrosis. TGFβ was shown to stimulate CCN2 promoter activity. In conclusion, this study suggests that SSc patients with activated Smad1 signalling may benefit from Imatinib mesylate treatment. In two known cases, Imatinib mesylate was used as an antifibrotic agent to treat patients with severe lung fibrosis. In one, with therapy-resistant and progressive SSc as well as pulmonary involvement, the skin tightness subsided and the pulmonary involvement stabilized after treatment with Imatinib mesylate. (p. 2549). The second patient had mixed connective tissue disease and pulmonary involvement, and showed a significant improvement in several outcome measurements following treatment with Imatinib mesylate (p. 2538).

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