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An Inappropriate Snack

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A woman ate some "seeds" from a plant outside her home and now has severe gastrointestinal distress. The implicated toxin is the castor bean, which can be used for both benign and deadly purposes.

Case

A previously healthy 60-year-old woman presents to the emergency department (in December) complaining of nausea, vomiting, and diffuse abdominal pain. She states that 12 hours earlier she chewed and swallowed three "seeds" that had been growing on a plant in her backyard. She acknowledges that she did this because "they looked tasty" and she did not have any intent of self-harm. She has brought several seeds with her to the emergency department. The gastrointestinal distress began 6 hours postingestion and she waited 6 more hours before coming to the emergency department. In the emergency department her abdominal pain is minimal, but she has one episode of emesis. Her vital signs are as follows: blood pressure, 136/75 mm Hg; heart rate, 79 beats/min; respiratory rate, 20 breaths/min; and temperature, 98.1°F. Her oxygen saturation is 100%. On physical examination, her mucous membranes are dry but other findings are normal.

What kind of seeds did the patient ingest, and how are they toxic?

The castor bean plant (*Ricinus communis*) is indigenous to tropical regions but is also grown worldwide as an ornamental plant. It grows rapidly, exceeding 8 ft in a single temperate growing season, and has a burgundy stem with leaves that are large (>1 ft across), dark green, and deeply scalloped (Figure 1). Towards the autumn, spiked seed pods grow and eventually mature to contain three individual seeds each (Figure 2). The seeds, called castor beans, are not by some definitions true beans since they are inedible. Castor beans (Figure 2) are elliptical in shape, brown,

FIGURE 1. Castor bean plant.

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and variegated, resembling a tick (*ricinus* is Latin for "tick"). The shell of the bean, while soft, is tough and water impervious. Castor beans are the source of castor oil, which has multiple uses, including as a laxative and a lubricating oil for jet engines (it was the initial Castrol motor oil). It is also used as a solvent for paints and varnishes.

Ricin is the toxin that remains when castor oil is separated from the bean; the oil is toxin-free. Most of the ricin is in the seeds.[1] It is a protein toxin consisting of 2 chains: an A chain and a B chain linked by a disulfide bond. The B chain has binding sites for cell surface galactose residues, which allows ricin to bind to cell surfaces. After binding to glycoproteins and glycolipids, ricin is endocytosed and transported within endosomes. Some of the ricin molecules enter the endoplasmic reticulum, where the A and B chains are cleaved. The A chain translocates to the cytosol and binds ribosomal RNA (rRNA), inhibiting protein synthesis irreversibly, leading to cell death.[2-4]

What other seeds can be included in the toxicologic differential diagnosis?

A similar toxin, abrin, is found in the rosary pea, derived from the plant *Abrus precatorius.* These 1-cm oblong, red seeds with a black "cap" at one end (Figure 2) are commonly strung together for use as prayer beads. They are also used to decorate small trinkets and other objects. The hole in the shell from the stringing should heighten concern for abrin release following ingestion. Abrin has a mechanism of action similar to that of ricin, but is more potent.[5]

Jimson weed (*Datura stramonium*), which grows naturally throughout the United States, also develops spiked pods towards the end of its growing season (Figure 2). The flowers of this plant are large and fluted (thus the moniker "angel's trumpet"). There are dozens of small seeds within the pods that contain anticholinergic alkaloids such as atropine and scopolamine (Figure 2). Since jimson weed is often proffered online as a "natural hallucinogen," it is commonly sought by teens who are unaware of the other

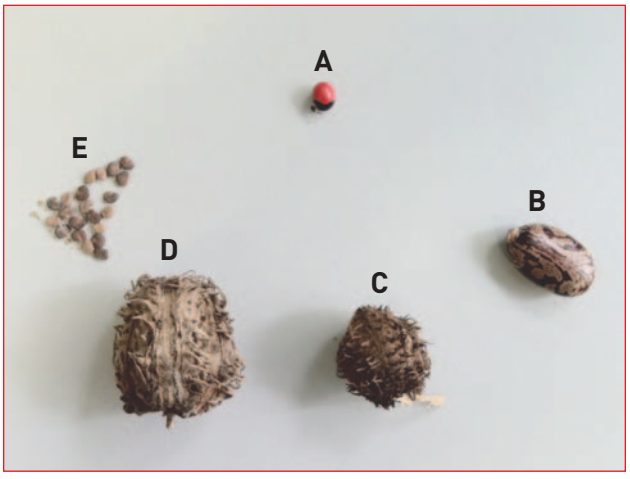

FIGURE 2. A. Rosary pea (*Abrus precatorius*). **B.** Castor bean (*Ricinus communis*). **C.** Dried castor bean pod. **D.** Dried Jimson weed pod (*Datura stramonium*). **E.** Jimson weed seeds.

significant clinical effects (the anticholinergic toxidrome) associated with its ingestion.[2]

What is the typical clinical picture that occurs after ricin ingestion?

When castor beans are swallowed whole, ricin is not released. However, mastication of the beans prior to swallowing releases ricin. Similarly, rosary peas are toxic only if chewed, although the hole created by stringing the seeds may provide an exit portal for toxin if the seed is ingested. The oral absorption of ricin is poor, and ingestion typically causes gastrointestinal symptoms within 4 to 6 hours of exposure. The most common cause of death following ingestion is hypovolemia from dehydration. Fortunately, castor bean ingestion is rare in humans, although it is more frequent in dogs and other household pets. In humans and animals, absorbed ricin can cause multiorgan dysfunction including hepatotoxicity, nephrotoxicity, cardiovascular toxicity, and hemolysis. In mice, the median lethal dose (LD_{50}) is suggested to be 30 mg/kg.[4] Case reports demonstrate that the lethal oral dose in humans ranges from 1 to 20 mg/kg. About eight beans is commonly considered lethal for

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an adult, although death has been reported after ingestion of as few as two beans.[4]

Parenterally administered ricin carries substantial risk of morbidity and mortality. In the most famous case, Georgi Markov, a Bulgarian dissident, was unknowingly impaled with a tiny metal sphere through an umbrella.[6] Several days later he died of multisystem organ failure. Autopsy revealed the sphere, which had a hole drilled through its diameter, in his popliteal fossa. Speculation that the sphere contained ricin was confirmed after the breakup of the Soviet Union. Recent concerns include the possibility of terrorist activity dispersing airborne powdered ricin that can be inhaled and result in lethal pulmonary toxicity or systemic absorption.

How should ricin poisoning be managed?

There is no specific antidote or treatment for ricin toxicity, regardless of route of exposure. Single-dose activated charcoal can be considered if the poison was ingested, although its efficacy for ricin poisoning is unknown.[7] If the patient presents shortly after ingestion and has not yet had emesis, gastric lavage should be considered, although its utility is unknown. Hemodialysis does not remove ricin.[4] The mainstay of treatment is supportive and directed towards resolution of symptoms and maintenance of normal vital signs. Renal function should be monitored. Once affected, patients are typically symptomatic for up to 36 hours. Patients who are asymptomatic at 12 hours postingestion are unlikely to develop delayed gastrointestinal toxicity,[4] although delayed hepatotoxicity has been reported in one child 48 hours after ingestion.[8]

Development of a ricin vaccine is an area of high military interest. In 2004, such a vaccine was created.[9] The vaccine consists of a genetically modified, enzymatically inactivated ricin chain A. The vaccine completed its phase 1A clinical trial in 2006, where it was shown to be safe and to induce antibodies in healthy human volunteers.[10] This vaccine has been granted orphan drug status, and currently phase 1B trials are under way.

Case Conclusion

The patient received 4 mg of ondansetron to control her nausea, followed by 50 g of activated charcoal. She tolerated the activated charcoal and was admitted to the general medicine unit for overnight observation. By the next morning, her symptoms had resolved and results of all laboratory studies were normal. She was discharged to home with a follow-up visit scheduled with her primary care physician. **EM**

References

1. Coopman V De Leeuw M, Cordonnier J, Jacobs W. Suicidal death after injection of a castor bean extract (Ricinus communis L.). *Forensic Sci Int.* 2009;189(1-3):e13-e20.
2. Palmer ME, Betz JM. Plants. In: Nelson LS, Lewin NA, Howland MA, et al, eds. *Goldfrank's Toxicologic Emergencies.* 9th ed. New York, NY: McGraw Hill; 2011:1537-1560.
3. Lord JM, Roberts LM, Robertus JD. Ricin: structure, mode of action, and some current applications. *FASEB J.* 1994;8(2):201-208.
4. Audi J, Belson M, Patel M, et al. Ricin poisoning: a comprehensive review. *JAMA.* 2005;294(18):2342-2351.
5. Dickers KJ, Bradberry SM, Rice P, et al. Abrin poisoning. *Toxicol Rev.* 2003;22(3):137-142.
6. Papaloucas M, Papaloucas C, Stergioulis A, et al. Ricin and the assassination of Georgi Markov. *Pak J Biol Sci.* 2008;11(19):2370-2371.
7. Chyka PA, Seger D. Position statement: single-dose activated charcoal: American Academy of Clinical Toxicology; European Associated of Poisons Centres and Clinical Toxicologists. *J Toxicol Clin Toxicol.* 1997;35(7):721-741.
8. Palatnick W, Tenebein M. Hepatoxicity from castor bean ingestion in a child. *Clin Toxicol.* 2000;38(1):67-69.
9. Doan LG. Ricin: mechanism of toxicity, clinical manifestations, and vaccine development. A review. *J Toxicol.* 2004;42(2):201-208.
10. Vitetta ES, Smallshaw JE, Coleman E, et al. A pilot clinical trial of a recombinant ricin vaccine in normal humans. *Proc Natl Acad Sci U S A.* 2006;103(7):2268-2273.