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Selected Topics: Toxicology

RICIN POISONING AFTER ORAL INGESTION OF CASTOR BEANS: A CASE REPORT AND REVIEW OF THE LITERATURE AND LABORATORY TESTING

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☐ **Abstract—Background: Ricin is a protein toxin derived from the castor bean plant *Ricinus communis*. Several cases secondary to its consumption have been published and, more recently, its use as a potential bioterrorism agent has also been reported. Oral absorption of ricin is highly erratic, leading to a wide spectrum of symptoms. In addition, conventional urine drug screening tests will not be able to detect this compound, posing a diagnostic challenge. Case Report: A male teenager intended to die by ingesting 200 castor beans after mixing and blending them with juice. Eight hours later, he presented with weakness, light-headedness, nausea, and vomiting and sought medical treatment. The patient was admitted and treated conservatively. An immune-based standard urine toxicology drug screen panel was reported as negative. A comprehensive untargeted urine drug screen test showed the presence of ricinine, a surrogate marker of ricin intoxication. He was transferred to the psychiatric service 3 days after admission. Why Should an Emergency Physician Be Aware of This?: This case highlights the importance of knowing the peculiar pharmacokinetic properties of ricin after oral ingestion of castor beans and toxin release through mastication. Emergency physicians should be aware that oral absorption of ricin is dependent on several factors, such type and size of seeds and the geographic harvesting region, making it extremely difficult to estimate its lethality based solely on the number of ingested beans. Finally, comprehensive untargeted urine drug screening testing is highly valuable as a diagnostic tool in this context.**

☐ **Keywords—castor bean; clinical chemistry tests; ricin; ricinine; toxicology**

INTRODUCTION

Ricin is a protein toxin derived from the castor bean plant *Ricinus communis*. The ricin content of castor beans has been reported to be 1–5% (1,2). Under the assumption that the average seed weight is 100 mg, the total ricin content would be 1–5 mg per seed. The beans are oblong and light brown, mottled with dark-brown spots (Figure 1). The castor oil produced from the beans is popularly used as a purgative. The castor oil is also found in many industrial products, such as nylon, cosmetics, paints, and automotive lubricants (1,3,4). Ricin is contained in the bean pulp. Because ricin is inactivated during oil extraction under heated conditions, it is not expected to be found in the conventional castor bean oil or its related products (1). More than 1000 ricin poisoning cases secondary to intentional castor bean consumption have been reported in the literature since the late 1800s (5,6). Because of its historical use as a weapon, ricin has been categorized by the United States (US) Centers for Disease Control and Prevention (CDC) as

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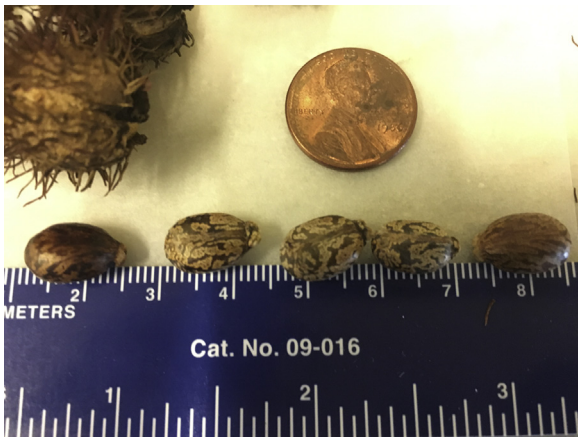

Figure 1. A photo of castor beans and a seed capsule (*Ricinus communis*). The seeds weigh between 53 and 194 mg (mean 103 mg).

a category B biothreat agent (second-highest priority), and its possession, transfer, and use are subject to domestic and international regulations (1,5). The lethality of ricin poisoning after oral ingestion is determined by a wide spectrum of factors and, as a result, clinical symptoms are quite variable. This poses a challenge for the emergency physician, especially when little or no clinical history is available. We present a case that highlights the erratic absorption of ricin after oral ingestion and the value of gas chromatography−mass spectrometry (GC-MS) as a diagnostic aid in those circumstances.

CASE REPORT

A male teenager with a history of depression and suicide attempts, presented to the emergency department after intending to die by ingesting 200 castor beans mixed with juice in a blender. Eight hours after ingestion, he developed weakness, light-headedness, nausea, and several vomiting episodes. He was no longer suicidal and sought medical treatment. The patient was admitted to the toxicology service and received aggressive i.v. hydration. An immune-based standard urine toxicology drug screen panel was reported as negative. A comprehensive untargeted drug screen test demonstrated the presence of ricinine compound in urine. In the first 24 h after admission, the patient presented with a single fever episode (38.3°C) and leukocytosis (19,000/mm^3). No further gastrointestinal (GI) symptoms were noted. His liver and kidney function tests were normal and his overall status improved quickly. Three days after ingestion, the patient was medically cleared for psychiatric disposition. The laboratory test results upon admission are summarized in Table 1.

Table 1. Basic Metabolic Profile and Complete Blood Counts of the Patient upon Admission

Test	Result	Reference Range
Sodium (Na), mmol/L	138	136–146
Potassium (K), mmol/L	3.8	3.5–5.0
Chloride (Cl), mmol/L	107	98–107
CO_2, mmol/L	24	21–31
Anion gap, mEq/L	11	7–15
Blood urea nitrogen (BUN), mg/dL	13	8–26
Creatinine (Cr), mg/dL	0.5	0.5–1.4
Glucose, mg/dL	88	70–99
Calcium (Ca), mg/dL	8.8	8.4–10.2
Albumin, g/dL	3.9	3.4–5.0
Total protein, g/dL	6.8	6.3–7.7
Billirubin total, mg/dL	0.4	0.3–1.5
Billirubin direct, mg/dL	0.1	0.1–0.5
Alanine transaminase, IU/L	53	17–63
Aspartate transaminase, IU/L	29	15–41
Alkaline phosphatase, IU/L	73	38–126
Lactate, mmol/L	1.1	0.5–2.2
Lipase, U/L	6	15–70
White blood cells, $\times 10^9$/L	10.7	3.8–10.6
Red blood cells, $\times 10^{12}$/L	4.74	4.13–5.57
Hemoglobin, g/dL	14.9	12.9–16.9
Hematocrit, %	44	38.0–48.8
Platelets, $\times 10^9$/L	228	156–369
Neutrophils, %	72	44–77
Lymphocytes, %	15	13–44
Monocytes, %	10	4–13
Eosinophils, %	2	0–6
Basophils, %	1	0–1

DISCUSSION

Structure of Ricin

Ricin is a protein toxin with a molecular weight of 60–65 kDa. It is composed of two chains, A and B, linked by a disulfide bond (2,7). Ricin preferentially binds to the abundant galactose-containing glycoproteins and glycolipids that line the surface of the cells (5). This is followed by internalization and its retrograde transport through the Golgi apparatus toward the endoplasmic reticulum. The A chain inhibits protein synthesis by irreversibly inactivating ribosomes (1,2,5). The B chain is catalytically inactive, but is essential for cell binding. Other mechanisms include induction of apoptosis, direct cell membrane damage, electrolyte imbalances, and release of cytokine inflammatory mediators (1,2,6). Only a single ricin A-chain molecule is required to inactivate > 1500 ribosomes per minute and kill the cell (1,2,5).

Toxidrome and the Route of Exposure

A ricin toxidrome depends on the route of exposure and the dose the patient received. The median lethal dose of ricin intoxication is reported as 3–5 μg/kg of body weight (BW) for inhalation, 5–10 μg/kg of BW for injection, and 20–30 mg/kg of BW for oral ingestion, based on animal