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Case report

Ricin poisoning after oral ingestion of castor beans: A case report and literature review

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ABSTRACT

Introduction: > 1000 ricin poisoning cases secondary to intentional castor bean consumption have been reported in the literature since the late 1800s. The lethality of ricin poisoning after oral ingestion is determined by a few factors.

Case report: We present a case that highlights the erratic absorption of ricin after accidental oral ingestion. On admission, the physical examination found a somnolent patient, with miosis, and a generalized abdominal tenderness. Her blood tests showed metabolic acidosis. Thanks to her early management, the discharge was possible three days later.

Discussion: The toxicity of ricin is dependent on the dose delivered and the route of the exposure. Supportive care is the mainstay of treatment. As shown in our case, early management is crucial for a good outcome.

African relevance

- Ricin poisoning is a challenge for the emergency physicians, especially when little or no clinical history is available.
- Castor oil is also found in many industrial products in African countries.
- Its management needs no specific treatment.

Introduction

Ricin is a protein toxin derived from the castor bean plant, '*Ricinus communis*', that grows in many tropical and subtropical regions. Ricin is contained in the bean pulp; its ratio in a single castor bean has been reported to be 1–5% [1,2]. The beans are oblong and light brown, mottled with dark-brown spots. The castor oil produced from the beans is commonly used as a laxative. Castor oil is also found in many industrial products, such as nylon, cosmetics, paints, and automotive lubricants [3,4]. > 1000 ricin poisoning cases secondary to intentional castor bean consumption have been reported in the literature since the late 1800s [5,6]. The lethality of ricin poisoning after oral ingestion is determined by a wide spectrum of factors and, as a result, its clinical symptoms are quite varied. This poses a challenge for the emergency physicians, especially when little or no clinical history is available. We present a case that highlights the erratic absorption of ricin after accidental oral ingestion.

Case report

A 2-year-old female child presented to the emergency department 4 h after an accidental ingestion of an undetermined quantity of castor beans. Prior to her admission, she was already suffering from weakness, abdominal pain, nausea, and several vomiting episodes followed by drowsiness. On admission to the intensive care unit, the physical examination found a somnolent patient, with a Glasgow Coma Scale (GCS) score at 14. She had miosis and presented a generalized abdominal tenderness, with no fever. However, she had a stable hemodynamic and respiratory state. In addition, the chest X-ray and electrocardiogram were normal. The biology tests revealed moderate hypokalaemia (3.2 mmol/l), metabolic acidosis and increased lipase. Renal and hepatic functions, and the haemostasis balance were normal (Table 1).

The patient received symptomatic treatment: aggressive intra-venous hydration, an antidiarrheal, an antiemetic and a supplementation of hydro electrolytic losses. Within the first 24 h after admission, the patient resumed her state of consciousness and no further gastro-intestinal (GI) symptoms were noted. Her liver and kidney function tests were normal. The medical staff decided to prolong the surveillance for at least 72 h to early detect any eventual secondary complications. As her general status improved gradually, she was discharged from the hospital three days after admission.

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Table 1
Results of the biological tests.

	On admission	At h-24 of hospitalisation	Reference Range
Sodium, mmol/L	139	138	136–145
Potassium, mmol/L	3.7	4.3	3.5–5.1
Chloride, mmol/L	100	105	98–107
PH	7.28	7.38	7.38–7.42
PaCO2, mmHg	36	36	38–42
PaO2, mmHg	87	82	≥80
bicarbonates level, mmol/l	16	23.6	22–26
Base excess	−8.6	0.3	
Creatinine, µmom/l	24	22	62–106
Alanine transaminase, IU/L	32	37.7	10–41
Aspartate transaminase, IU/L	34	36.8	10–37
Lactate, mmol/L	0.68	0.79	0.50–2.20
Lipase, U/L	247	49	13–60

Discussion

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 disulphide 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].

The toxicity of ricin is dependent on both the dose delivered and the route of the exposure [7,8]. The median lethal dose (LD50) is the dose that would kill at least 50% of the people who have ingested such a dose. The smaller the LD50 is, the less material is needed to kill the average person. For ricin, the LD50 is lowest for inhalation, and increases respectively for intravenous, intraperitoneal, subcutaneous, and intragastric administration [8]. Local pulmonary and/or systemic effects following ricin inhalation were reported as well [9]. Ingestion of ricin is less likely to lead to toxicity due to the poor gastrointestinal absorption and to the potential enzymatic degradation of the protein within the gastrointestinal tract [8]. Contact allergies have been reported from dermal exposure (castor bean necklaces) [10]. Dermal absorption of ricin is poor [8,7] but systemic toxicity remains possible [11].

Gastrointestinal (GI) administration is the most frequent route of exposure encountered by emergency physicians. The orally ingested ricin initially damages the GI system, causing GI tract symptoms (e.g., nausea, vomiting, bloody diarrhoea), leading to severe dehydration, visceral organ damage (e.g., liver, kidney, and spleen), and possible death in severe cases [2]. The visceral organ damage can be explained by previous animal studies, which showed that orally ingested ricin damages intestinal epithelium and is absorbed within 2 h through intestinal blood vessels and lymphatics, leading to its accumulation in the liver and spleen [6]. According to the previous clinical case reports of castor bean oral ingestion, the lethal dose of ricin falls within a wide range, between 1 and 20 mg/kg. One report stated that oral ingestion of two castor beans was lethal [2]. A significant variation in the lethality after castor bean oral ingestion has been reported in the literature and might be explained by several factors. One such factor is the fact of chewing the castor beans prior to an oral ingestion. Indeed, ricin needs to be released from the ingested castor beans through mastication to exert toxicity. The gastric-content volume may also influence the degree of ricin toxicity [6]. Some authors have suggested that much more ricin is required to achieve lethality by oral route due to the relatively large molecular size of the compound and its degradation through the GI tract, leading to a poor intestinal absorption [6,12]. When ingested, as it was in our case, it is extremely difficult to estimate its toxicity and lethality based on the number of orally ingested beans alone [12]. Lopez Nunez et al. reported the case of a male patient who presented to the emergency department (ED), eight hours after he had ingested 200 castor beans mixed with juice in a blender. He had light-headiness, nausea, and several vomiting episodes. The symptoms disappeared within three days [13]. In our case, the patient presented to the emergency department after accidental ingestion of an undetermined quantity of castor beans with weakness, abdominal pain, nausea, and several vomiting episodes followed by drowsiness.

Other pathways of intoxication have also been reported in the literature. It was reported few cases of localized pain within the soft tissues and regional lymphadenitis induced by intra-muscular and subcutaneous injections of high doses of ricin [14]. Georgi Markov, the assassinated Bulgarian exiled dissident, received an estimated 500 mg of ricin which was inoculated into his right thigh. He developed immediate pain at the injection site and complained of weakness within 5 h of the injection, approximately as in our case [14]. He became febrile within 24 h, with nausea and vomiting. On admission, 36 h after the injection, he had fever, tachycardia, and inguinal lymphadenopathy with an induration around the puncture site. As it was described in our case, his hemodynamic status was stable. His condition worsened three days after the injection with a multivisceral dysfunction and he died on the fourth day [14]. Targosz et al. reported the case of a 20-year-old man who injected himself with the extracted liquid of castor beans subcutaneously and developed the same clinical presentation with death ensuing within several days of the injection [15].

The only human data available for inhalational ricin exposure involves workers exposed to castor bean dust in castor oil processing plants. These patients developed nasal and throat congestion, irritation of the conjunctivae, urticaria, chest tightness, and bronchospasm with favourable outcomes [16].

Oral absorption of ricin is commonly associated with a wide spectrum of symptoms and can be misdiagnosed. Besides, conventional urine screening tests will not detect this compound [13]. According to forensic reports, it has been proposed that any level below 0.08 to 10 ng/ml 48 h after exposure should imply a less-than-lethal dosage [2]. However, ricinine concentrations should be interpreted cautiously. In a case report, urinary ricinine concentrations were quantified for up to 63 h post-exposure with decreasing concentrations until 130 ng/ml [2,17]. Urinary screening is proposed to be used only to identify patients who are exposed and those who are not exposed [2].

Supportive care is the mainstay of treatment. Hypotension should be treated with aggressive fluid replacement and direct-acting vasopressors. There is no evidence that any individual vasopressor is superior to another. However, there is evidence of increased endogenous norepinephrine release following ricin administration in rabbits, as well as decreased vascular responsiveness to norepinephrine [18,19]. Given this, vasopressin may be a reasonable alternative. Similarly, replenishing of electrolytes presents an important component. Vomiting and diarrhoea should be handled with barrier and splash precautions (gown, eye shield, mask, gloves), but is unlikely to produce a vapour or to expose healthcare workers to contamination [20]. For inhalation exposure, treatment also includes oxygen administration, bronchodilators, endotracheal intubation, and supplemental positive end-expiratory pressure as needed [1,2,6]. In our case, treatment was symptomatic: aggressive intra-venous hydration, an antidiarrheal, an antiemetic, and a supplementation of hydro electrolytic losses.

Patients who are exposed to ricin by way of liquid, powder, or aerosol must have their clothing removed and their skin thoroughly washed for 5–6 min with water before admission to the hospital to avoid further absorption and caregivers' contamination [21]. In case of ingestion, or if the pathway of the intoxication is unknown,

gastrointestinal decontamination with gastric lavage and activated charcoal may be appropriate. However, gastric lavage is not likely to remove a significant amount of ricin unless it is performed within 1 h of ingestion [22]. Ricin is poorly adsorbed by activated charcoal because of its large size [20].

At this time, there is no effective antidote for ricin poisoning and dialysis has been proven to be ineffective because of the large molecular size of ricin [1].

Our patient did not receive a gastric lavage no activated charcoal because she consulted tardily.

Conclusion

Ricin poisoning has been reported after inhalation, parenteral injection, and oral ingestion. Successful absorption of ricin after oral ingestion is determined by multiple variables. Therefore, a wide spectrum of clinical manifestations is expected, and while some patients might present with only mild GI complaints, others can demonstrate severe clinical manifestations. This can be misleading for the physician, especially when little clinical information is available.

Dissemination of results

The details of this observation were shared with staff members at the data collection group through an informal presentation.

Authors' contribution

Authors contributed as follow to the conception or design of the work; the acquisition, analysis, or interpretation of data for the work; and drafting the work or revising it critically for important intellectual content: OCW contributed 40%; MB contributed 35%; NR contributed 15% and EG and SB contributed 5% each. All authors approved the version to be published and agreed to be accountable for all aspects of the work.

Declaration of competing interest

The authors declared no conflicts of interest.

References

[1] Audi J, Belson M, Patel M, Schier J, Osterloh J. Ricin poisoning: a comprehensive review. JAMA 2005;294:2342–51. https://doi.org/10.1001/jama.294.18.2342.
[2] Musshoff F, Madea B. Ricin poisoning and forensic toxicology. Drug Test Anal 2009;1:184–91. https://doi.org/10.1002/dta.27.
[3] Johnson RC, Lemire SW, Woolfitt AR, et al. Quantification of ricinine in rat and human urine: a biomarker for ricin exposure. J Anal Toxicol 2005;29:149–55. https://doi.org/10.1093/jat/29.3.149.
[4] Hamelin EI, Johnson RC, Osterloh JD, Howard DJ, Thomas JD. Evaluation of ricinine, a ricin biomarker, from a non-lethal castor bean ingestion. J Anal Toxicol 2012;36:660–2. https://doi.org/10.1093/jat/bks077.
[5] Bozza WP, Tolleson WH, Rosado LA, Zhang B. Ricin detection: tracking active toxin. Biotechnol Adv 2015;33:117–23. https://doi.org/10.1016/j.biotechadv.2014.11.012.
[6] Moshiri M, Hamid F, Etemad L. Ricin toxicity: clinical and molecular aspects. Rep Biochem Mol Biol 2016;4:60–5.
[7] Bradberry SM, Dickers KJ, Rice P, Griffiths GD, Vale JA. Ricin poisoning. Toxicol Rev 2003;22:65–70. https://doi.org/10.2165/00139709-200322010-00007.
[8] Franz DR, Jaax NK. Ricin toxin. In: Sidell FR, Takafuji ET, Franz DR, editors. Medical aspects of chemical and biological warfare. Washington (DC): 7 Office of the Surgeon General/TMM publications; 1997. p. 631–42.
[9] Bunner D. Biological warfare agents: an overview. In: Somani S, editor. Chemical warfare agents. San Diego (CA): 7 Academic Press; 1992. p. 387.
[10] Schaumburg H, Ricin Spencer P. Spencer P, Schaumberg H, editors. Experimental and clinical neurotoxicology. 2nd ed.New York 7 Oxford University Press; 2000. p. 1068–9.
[11] Henghold W. Other biologic toxin bioweapons: ricin, staphylococcal enterotoxin B, and trichothecene mycotoxins. Dermatol Clin 2004;22:257–62. https://doi.org/10.1016/j.det.2004.03.004.
[12] Griffiths GD. Understanding ricin from a defensive viewpoint. Toxins (Basel) 2011;3:1373–92. https://doi.org/10.3390/toxins3111373.
[13] Lopez Nunez OF, MD Pizon AF, Tamama K. Ricin poisoning after oral ingestion of castor beans: a case report and review of the literature and laboratory testing. J Emerg Med 2017;53(5):e67–71. https://doi.org/10.1016/j.jemermed.2017.08.023.
[14] Crompton R, Gall D. Georgi Markov: death in a pellet. Med Leg J 1980;48:51–62. https://doi.org/10.1177/002581728004800203.
[15] Targosz D, Winnik L, Szkolnicka B. Suicidal poisoning with castor bean (Ricinus communis) extract injected subcutaneously: case report (abstract). J Toxicol Clin Toxicol 2002;40:398.
[16] Topping M, Henderson R, Luczynska C, et al. Castor bean allergy among workers in the felt industry. Allergy 1982;37:603–8. https://doi.org/10.1111/j.1398-9995.1982.tb02347.x.
[17] Thomas J, Chang A, Johnson RC. Intentional castor bean ingestion with serial ricinine levels. J Clin Toxicol 2006;44:625.
[18] Christiansen VJ, Hsu C-H, Robinson CP. The effects of ricin on the sympathetic vascular neuroeffector system of the rabbit. J Biochem Toxicology 1994;9(4):219–23. https://doi.org/10.1002/jbt.2570090407.
[19] Christiansen VJ, Hsu C-H, Zhang L, Robinson CP. Effects of ricin on the ability of rabbit arteries to contract and relax. J Appl Toxicol 1995;15(1):37–43. https://doi.org/10.1002/jat.2550150109.
[20] Kortepeter M. Ricin. In: Christopher G, Cieslak T, editors. USAMRIID's medical management of biological casualties handbook. 4th ed.Frederick (MD): 7 United States Army Medical Research Institute of Infectious Disease; 2001. p. 130–7.
21. OSHA. Practices for hospital-based first receivers of victims from mass casualty incidents involving the release of hazardous substances Available at www.osha.gov/dts/osta/bestpractices/firstreceivers_hospital.html.
[22] Vale JA. Gastric Lavage (position statement). J Clin Toxicol 1997;35:711–9.