Evaluation of Castor Bean Toxicosis in Dogs: 98 Cases

Castor beans (*Ricinus communis*) contain ricin. Ricin is a glycoprotein reported to cause hypotension, gastroenteritis, depression, and death. However, few deaths are reported following castor bean ingestion in animals. From January 1987 to December 1998, the American Society for the Prevention of Cruelty to Animals–National Animal Poison Control Center received 98 incidents of castor bean ingestion in dogs. The most commonly reported clinical signs were vomiting, depression, and diarrhea. Death or euthanasia occurred in 9% of the cases. The severity of clinical signs following castor bean ingestion may depend on whether the beans were chewed or swallowed whole. **J Am Anim Hosp Assoc 2000;36:229–33.**

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Introduction

Ricinus communis (i.e., castor bean, mole bean, Palma Christi, Wonder tree, African coffee tree, Mexico weed) is an ornamental plant introduced from the West Indies. It has been naturalized to temperate regions but can also be grown in many parts of North America during the warmer months.[1–3] In addition to being a very large and decorative plant, castor bean produces an oil that is used for industrial and medicinal purposes (e.g., as a high-grade industrial lubricant, in the manufacturing of inks and dyes, and as a medicinal purgative). The plant produces spiny seed-pods that burst open when dry, scattering ornamentally colored beans [Figure 1]. These seeds have a coloring that resembles markings seen on some ticks or beetles [Figure 2]. Hence, the Latin name for this plant (i.e., *Ricinus*), meaning insect.[1,2]

In humans, the ingestion of anywhere from one to eight seeds is estimated to be lethal, and all exposures are considered dangerous, especially in children.[4] Ricin is 100 times more toxic parenterally than orally and has been used as a homicidal poison.[5] Castor beans have poisoned all types of livestock, horses, poultry, as well as both dogs and cats.[1,2] While all parts of the castor bean plant contain ricin, the seeds are extremely toxic since they contain more ricin and are most often associated with clinical toxicosis.[2]

Clinical signs associated with castor bean toxicosis in animals often occur several hours after ingestion of the beans.[2,4] The beans must be crushed or broken in order for the toxalbumin to be released.[4] Initially, gastrointestinal irritation resulting in vomiting, diarrhea, and abdominal pain are the major complaints. These signs often progress to hemorrhagic gastroenteritis. The toxic effects of ricin include hypotension, increased cardiac output, hemorrhage, and myocardial necrosis.[1,2,4] Human deaths were often from hypovolemic shock.[6] In addition, the cytotoxic effects of ricin extend to the liver and kidney, causing hepatic enzyme elevation and renal failure.[2] Other signs reported include dehydration, melena, lethargy, seizures, and death.[1,2,4,7] Castor bean pulp is also highly allergenic and can cause dermatitis, rhinitis, and asthma. The dust liberated from the extraction of castor bean oil affects not only human workers but those living close to the oil mills.[8]

In spite of the severity of signs and toxicity of ricin, cases of human death are rare after the ingestion of the seeds.[6,9] The apparent discrepancy

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Figure 1—Castor bean plant. Note the spiny seedpods that burst open when dry, scattering the castor bean seeds.

between the extreme toxicity of ricin and the low mortality rate is thought to be due to the degree of mastication of the beans. Chewing of the beans is vital in releasing the toxalbumin. In many cases of human castor bean ingestion, the beans were not known to have been chewed or were only partially chewed.[9]

The purposes of this report are to review past cases of castor bean ingestion in dogs, compare clinical signs with those reported in the literature, and evaluate the severity of castor bean ingestion in companion animals. Effective treatment and decontamination procedures following castor bean ingestion are also discussed.

Materials and Methods

Epidemiological and clinical information was obtained from the American Society for the Prevention of Cruelty to Animals (ASPCA) National Animal Poison Control Center's (NAPCC) case record database. The NAPCC receives calls on incidents of animal poisoning from practicing veterinarians and animal owners throughout the United States and Canada. A staff veterinarian collects the following information on affected animals: breed, sex, age, weight, number exposed and at risk, number affected, source of exposure, location, amount

Figure 2—Castor bean seeds. Note the decorative pattern on the seed coat, which resembles some ticks or beetles.

of plant material ingested, and assurance of exposure (i.e., whether observed or evidenced). Information regarding time of onset, type, severity, and duration of clinical signs, clinical serum biochemistry alterations, pathological findings, and response to treatment is obtained. Follow-up calls are made as deemed necessary in order to update records about progression of clinical signs, response to treatment, and final outcome.

On the basis of exposure history and clinical information provided, expected clinical signs of toxicosis as described in the literature, amount ingested, and previous experience dealing with the agent, the NAPCC veterinarian categorizes each incident. An incident is categorized as toxicosis if all temporal, clinical, and historical data is consistent with the expected syndrome. If clinical signs are characteristic of the expected syndrome, but some data is not available, the incident is categorized as a suspected toxicosis. If only a few signs are consistent with the expected syndrome, the reported incident is categorized as a possible toxicosis. When clinical signs and exposure history are not consistent with the expected syndrome, the incident is categorized as a doubtful toxicosis. Finally, an incident is categorized as an exposure only when signs were not observed at the time of the call and no signs developed on subsequent follow-ups. Information received at the NAPCC regarding castor bean ingestion was not confirmed by analytical methods or by direct observation. The NAPCC database was searched for records of dogs ingesting castor beans between January 1987 and December 1998.

Results

During the last 11 years, 98 incidents were reported to the NAPCC concerning castor bean ingestion in dogs. Twenty-one incidents categorized as toxicosis, 50 incidents categorized as suspected toxicosis, and three incidents categorized as possible toxicosis were included in this report. Twenty-four incidents were reported of castor bean ingestion, but the animals never showed any signs. Thus, 76% of animals with castor bean exposures developed signs. Doses of castor beans ingested by the

Table

Frequently Reported Clinical Signs in 98 Incidents of Castor Bean Ingestion in Dogs

Sign	Frequency
Vomiting	80%
Depression	45%
Diarrhea	37%
Diarrhea with blood	24%
Abdominal pain	14%
Anorexia	16%
Vomiting with blood	10%
Death/euthanasia	9%
Weakness	8%
Hyperthermia	7%
Ataxia	5%
Hypersalivation	5%
Recumbency	5%
Tachycardia	5%

animals were often unknown, and insufficient data was available to estimate a lethal dose. However, the ingestion of just one castor bean was sufficient to cause clinical signs in some dogs. There were no significant breed, sex, or age differences in the animals ingesting the beans. Incidents of castor bean ingestion occurred in all months of the year, but were most frequently reported in the months of April, June, October, and November.

The most commonly reported clinical signs associated with castor bean ingestion in the dogs of this study are listed in the Table. Although deaths occurred in seven (9%) cases, three of the seven animals were euthanized. Other less frequently described signs in dogs included coma, tremors, seizures, dehydration, pallor, dyspnea, polydipsia, jaundice, hyperthermia, and vocalization. Signs developed most frequently within six hours following ingestion (range, 0.5 to 42 hrs). The duration of these signs was from 1.5 to 5.5 days.

Laboratory values were checked infrequently in the cases reported to the NAPCC. The most common abnormal laboratory test results were a high hematocrit, an increased white blood cell (WBC) count, and elevated alanine aminotransferase (ALT) and/or aspartate aminotransferase (AST) serum enzyme levels. Additional laboratory abnormalities were increased serum bilirubin and globulin levels, urinary casts, and hyponatremia in one dog.

Discussion

The toxic component in the castor bean plant is the water-soluble, heat-labile toxalbumin ricin. Fortunately, the castor bean oil, if properly extracted, does not contain ricin.[1,2,4] This toxalbumin is one of the most deadly substances known. Lethal doses range from 0.025 mcg given intraperitoneally in mice to 1 mg/kg orally in humans.[4,5] The ricin molecule is composed of two glycoprotein chains. One chain facilitates endocytosis, while the other chain, once in the cell, inhibits protein synthesis and causes cell death.[4,5,10,11]

The target organs for signs that are caused by ricin in the body depend on the route of administration.[5] In the cases discussed in this report, the ricin toxin was ingested. Thus, signs involving the gastrointestinal tract were more predominantly reported. Because ricin is poorly absorbed, additional signs (including death) require large doses of ricin to be ingested.[2] The authors' experience, as well as the experience of others reporting castor bean exposures, suggest that ingestion infrequently results in death despite ricin being one of the most potent toxins known.[6,9] A possible explanation for this inconsistency may be that sometimes castor beans are swallowed whole. The toxalbumins in castor beans are not likely to be released unless the seed coat is damaged or compromised. If the ricin is not released and absorbed, signs (including death) will not develop.

Ricin is a cellular toxin, and the mechanism of toxicity in mammalian cells has been extensively studied.[4,5,7,10–13] The B chain of the ricin molecule binds to galactoside-containing proteins on the cell surface, allowing internalization of the toxin. Mammalian cells possess a large number of glycoproteins and glycolipids with galactose residues that are available for the B chain of ricin to attach.[10] This ensures that most, if not all, ricin molecules present at the cell surface will enter the cells by endocytosis. Once in the cell, some of the ricin is transported to lysosomes or back to the cell surface. In a lysosome or outside the cell, ricin can produce no cellular damage.[10] However, some ricin is moved from the endosomes to the trans-Golgi network, resulting in retrograde transport of the ricin through the Golgi stack. Ultimately, the ricin A chain is translocated into the cytosol and works its way back to the endoplasmic reticulum. Once in the endoplasmic reticulum, the A chain depurinates 28S ribosomal ribonucleic acid (rRNA) by the removal of a specific adenine residue.[4,5,7,10] Protein synthesis cannot occur, and the cell dies. Cell death in the gastrointestinal tract results in vomiting, abdominal pain, diarrhea, and gastrointestinal hemorrhage.[1,2,4]

Ingestion of a large number of castor beans may allow more ricin to be absorbed. After absorption, or when given parenterally, ricin is preferentially distributed to the liver, spleen, and muscle.[4,5,11] Ricin in these organs results in reversible liver damage, vascular endothelial injury, edema, and myalgias.[11] Ricin also disturbs calcium homeostasis in the cardiovascular system by decreasing calcium uptake by the sarcoplasmic reticulum and increasing sodium-calcium exchange. The deregulation of intracellular calcium homeostasis causes myocar-

dial necrosis and cardiac hemorrhage. In addition, cardiac function is altered, often resulting in hypotension.[7,12,14,15] Alteration of calcium regulation is required for ricin to inhibit protein synthesis in cultured macrophages.[12] When ricin is absorbed through the gastrointestinal tract or when it is given parenterally, multiple organ systems are affected and death is more likely to occur. Pathologically, parenteral ricin administration causes necrosis and hemorrhage in the heart, stomach, lungs, liver, kidneys, and pancreas.[4,7,13–15]

In this report, deaths occurred in seven (9%) cases. This is not consistent with the potency and lethality of ricin. From the information available in the reported incidents, it was not always determined if the animals chewed on the castor beans before ingestion. However, it has been postulated that the reason for the low mortality rate following castor bean ingestion is that the beans were not chewed.[2] In addition, poor absorption of ricin from the gastrointestinal tract is probably a factor. Although clinical serum biochemistry values were not checked often in the cases reported in this paper, elevated ALT, AST, and increased WBCs are consistent with the hepatotoxicity described in some human cases of castor bean ingestion.[6,15]

The castor bean seeds do not mature and release from the spiny pods of the plant until autumn. This probably explains why a large number of reported castor bean ingestions occurred in October and November. However, the castor bean seeds are ornamentally colored and have been used to make craft projects.[2] The beans are thus available at all times of the year for small animals or pets to consume. Indeed, castor bean ingestions have been reported in all months of the year. In the authors' experience, the ornamental use of castor beans can be a source of attraction to dogs.

There are no postingestion antidotes to ricin.[4] Emesis can be beneficial if instituted early after an animal ingests castor beans. The use of hydrogen peroxide (3%; 1 to 5 ml/kg body weight, per os [PO]) or another appropriate emetic is indicated. This should be followed with activated charcoal (1 to 4 gm/kg body weight, PO) and a cathartic (i.e., magnesium sulfate [250 mg/kg body weight, PO] or sorbitol [70%; 3 ml/kg body weight, PO]), unless the animal already has diarrhea.[16,17] Activated charcoal can be beneficial even if given several hours after the castor beans are ingested. Gastrointestinal protectants such as kaolin-pectin or sucralfate (0.25 to 2 grams, *tid* PO) should be used as needed.[18] Appropriate fluid and electrolyte therapy should be considered very important, and hypotension can often be controlled with balanced electrolyte solutions (i.e., lactated Ringer's solution). Fluid administration should account for both daily requirements and ongoing fluid losses.[16] Seizures have been reported and may be controlled with diazepam (0.5 to 1.0 mg/kg body weight, intravenously [IV]).[17]

Baseline complete blood count (CBC) and serum biochemistry values are helpful, but specific elevations of liver or kidney biochemical values may not occur for 12 to 24 hours following castor bean ingestion. When hepatic damage occurs, oral antibiotics, lactulose (0.1 to 0.5 mg/kg body weight, *tid* PO), and/or dietary management along with appropriate fluid therapy can be used to correct the clinical signs associated with hepatic failure.[19] Similarly, signs of renal failure should be treated with appropriate fluid therapy and supportive care. Repeated clinical serum biochemistry evaluations are indicated until enzyme levels return to normal and clinical signs resolve. Due to ricin's effects on the gastrointestinal tract, a soft, bland diet in small amounts should be given frequently for one to four days once the vomiting is controlled.

The effectiveness of glucocorticoid administration in animals following ricin ingestion is not known, although dexamethasone given three days prior to ricin administration prolonged death in mice challenged with lethal doses of ricin. It is postulated that dexamethasone inhibits lipid peroxidation. Since lethal ricin doses result in increased lipid peroxidation, dexamethasone could be the reason for the delayed time of death in the challenged mice.[5]

Conclusion

Although ricin is one of the most potent plant toxins known, few lethal castor bean ingestions have been reported. The more severe signs, including death, caused by castor bean ingestion may depend on whether the beans were masticated or broken. Ingestion of castor beans remains a potential hazard, especially to small animals. Furthermore, past incidents of castor bean ingestion indicate owners are often unaware of the amounts ingested and whether or not the beans were chewed. Consequently, all known or potential ingestions of this plant should be considered serious and treated aggressively to avoid any severe consequences or death to the animals. While clinical signs of castor bean toxicity could be delayed for 48 hours, most animals recover with supportive therapy.

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