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Short Communication

Fetal death of dogs after the ingestion of a soil conditioner

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ABSTRACT

Castor beans (*Ricinus communis*) contain ricin, which is one of the most toxic substances of plant origin. Ricin toxicosis has been reported in different countries with usually ingestion of castor beans or plants in both animals and humans. However, ricin toxicosis by ingestion of some products containing castor oil cake has rarely been reported. This paper describes outbreaks of dog death by ricin toxicosis after accidental ingestion of the same soil conditioner. Fifteen dogs showed toxic symptoms such as severe vomiting, abdominal pain and hemorrhagic diarrhea, and then thirteen dogs died in a few days. The soil conditioner dogs ingested consisted of 10% castor oil cake containing ricin. On the basis of clinical signs, laboratory and pathologic findings, a diagnosis of ricin toxicosis was established in the present case. In comparison with previous cases by ingestion of castor beans, the dogs' morbidity was very high in the present case. The ingestion of castor oil cake may be more dangerous to life than the castor beans. It is because mortality by ingestion of castor beans depends on the degree of mastication of the beans, whereas ricin in oil cake is easily absorbed from the stomach and the intestines. As ricin is a heat-labile toxin, products containing ricin or oil cake should be properly treated with heat and have written caution sentences about toxicosis, and be kept out of reach of domestic animals and children.

Introduction

Castor bean (*Ricinus communis*) is a widely used ornamental vine and usually grown on a large scale for castor oil production (Wexler and Gad, 1998; Klaassen, 2001). The castor bean contains 40% oil, 1–5% ricin and 0.3–0.8% ricinin (Johnson et al., 2005). Ricin is one of the most toxic substances of known plant origin, and its toxicity results from the inhibition of protein synthesis, which leads to cell death (Winder, 2004). The toxicity of castor beans has been known since ancient times, and all species of animals including humans are susceptible to the toxic effects of ricin (Rauber and Heard, 1985). In April 2007, fifteen dogs showed toxic symptoms including severe vomiting, abdominal pain and hemorrhagic diarrhea after accidental ingestion of the same soil conditioner, and then thirteen dogs were within a few days. The soil conditioner dogs ingested consisted of 10% oil cake, which is pomace after the commercial separation of oil from castor beans, and contained the ricin (Dreisbach, 1983). On the basis of clinical signs, pathological and toxicological findings, a diagnosis of ricin toxicosis was established in the present case. Many literatures associated with ricin toxicosis have been reported in different countries with ingestion of castor beans or plants in both animals and humans (Albretsen et al., 2000; Palatnick and Tenenbein, 2000; Aslani et al., 2007; Mouser et al., 2007); however, ricin toxicosis by ingestion of some products containing castor oil cake has rarely been reported to the author's knowledge. The present report describes ricin toxicosis of the dogs in clinical, laboratory and pathological aspects after accidental ingestion of a soil conditioner in Korea.

Case report

History and clinical findings

In April 2007, outbreaks of dog death occurred with major clinical signs of severe vomiting and hemorrhagic diarrhea. There were fifteen affected dogs , and thirteen dogs died within a few days despite intensive supportive care in veterinary clinics. The history of these dogs was that all of them accidentally ingested the same soil conditioner that was freely distributed from a famous cafe as a commemorative event (Fig. 1). The main clinical signs were vomiting, abdominal pain, shivering and fever. The dogs showed intermittent vomiting continuously after taking the soil conditioner and then diarrhea. Diarrhea gradually started

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Fig. 1. The soil conditioner dogs ingested. According to the information given by the manufacturing company, it contained 60% used coffee grounds, 20% starch, 10% bio-degradable resin and 10% oil cake in a pack of 90–100 g.

containing blood with malodor. The soil conditioner contained 60% used coffee grounds (the powder that is left after coffee has been filtered out to drink), 20% starch, 10% bio-degradable resin and 10% oil cake in a pack of 90–100g according to the information given by the manufacturing company.

Laboratory findings

Hematology revealed a very high hematocrit (%), indicating severe dehydration and hypotension. Serum biochemistry profile showed high AST and ALT activity, high serum BUN and creatinine concentrations. Table 1 shows the change in serum biochemistry and Complete Blood Count (CBC) of a dog presented to veterinary clinic with toxic symptoms. The dog showed no positive responses to intensive supportive cares in veterinary clinics and died 3 days after ingestion of a soil conditioner.

Pathologic findings

At necropsy (Fig. 2), there were markedly severe congestion and hemorrhage in both the mucosal and subserosal layers of the gastrointestinal tracts and mesentery with hemorrhagic ascite. Congestion and hemorrhagic lesions were well-observed in serosal surface than in the mucosal one. The liver was a little pale, and some petechias were observed. On microscopic examination, the liver showed a great dilation of the sinusoids with congestion, which caused a marked compression of the rows of hepatocytes. There were toxic hepatocellular degeneration and necrosis with several pyknotic nuclei. Many hemosiderin phagocyting macrophages presented in sinusoids (Fig. 3**A**). Renal tubular epithelium showed vacuolar degeneration and necrosis (Fig. 3**B**). In the gastrointestinal tracts, marked congestion was observed in submucosa and muscular layers more than mucosa. Desquamation of necrotic epithelial cells mingled with the other intestinal contents and piled up on the surface of congested mucosa (Fig. 3**C**). There were many degenerated intestinal epithelial cells with karyorrhectic nuclei (Fig. 3**D**). In the mesenteric lymph nodes (Fig. 3**E**), there were hemorrhage and multi-focal necrosis. Numerous karyorrhectic and karyopyknotic nuclei of lymphocytes presented due to cell death. In the spleen (Fig. 3**F**), splenic contraction was observed due to a decrease of red pulp and there was infiltration of a number of hemosiderin-laden cell. Megakaryocytes with a lobulated nucleus in a large cytoplasm presented occasionally.

Table 1
Serum biochemistry and Complete Blood Count (CBC) of a dog after ingestion of a soil conditioner.

Measurement	1 day	3 day	Reference range
Serum biochemistry			
Glucose (mg/dl)	94		75–128
BUN (mg/dl)	10.8	74	9.2–29.2
Creatinine (mg/dl)	0.5	2	0.4–1.4
T-Cholesterol (mg/dl)	346		111–312
T-Bilirubin (mg/dl)	0.3	1.6	0.1–0.5
Ca (mg/dl)	12.4	11.5	9.3–12.1
P (mM)	2.6		1.9–5.0
Total protein (g/dl)	6.9	4.8	5.0–7.2
Albumin (g/dl)	3.9	2.2	2.6–4.0
AST (U/L)	97	354	17–44
ALT (U/L)	70	91	17–78
Creatine kinase (U/L)	146		49–166
Amylase (U/L)	5670		269–2299
GGT (U/L)	7	1	5–14
ALP (U/L)	243	1306	47–254
Na (mmol/L)	131	151	141–152
K (mmol/L)	3.3	3.8	3.8–5.0
Cl (mmol/L)	105	116	102–117
CBC			
WBC ($\times 10^9$/L)	8980	19,720	6000–17,000
Neutrophil	7860	11,960	3000–11,800
Lymphocyte	360	2210	1000–4800
Monocyte	140	470	200–2000
Eosinophil	610	4700	100–1300
Basophil	0	380	0–500
RBC ($\times 10^{12}$/L)	9.79	8.67	5.0–8.5
Hemoglobin (g/dl)	24	15.1	12–18
Hematocrit (%)	71.4	64.2	37–55
MCV (fL)	72.9	74	60–74
MCH (pg)		17.4	19.5–24.5
MCHC (g/dl)		23.5	31.0–36.0
PLT ($\times 10^9$/L)	492	446	200–500

Discussion

There is no specific antidote for ricin toxicosis, and supportive and symptomatic treatment in all species is recommended (Albretsen et al., 2000; Doan, 2004). The recognition of clinical pathologic characters and clinical signs of ricin toxicosis can help rapid diagnosis, and it can lead the patient to have more effective treatment. However, recognition of the ricin poisoned patient will likely be difficult because the symptoms that are presented tend to be non-specific, such as airway inflammation and other respiratory problems regarding inhalations, or abdominal problems regarding ingestion (Rosenbloom et al., 2002; Audi et al., 2005). Therefore, a diagnosis of ricin toxicosis is generally based on observing ingestion or emesis of the castor bean or plant components, or in the context of an outbreak of severe gastrointestinal or respiratory illness in animals so far (Albretsen et al., 2000; Palatnick and Tenenbein, 2000; Soto-Blanco et al., 2002; Aslani et al., 2007; Mouser et al., 2007; Botha and Penrith, 2009), although detection of ricin in blood or bodily fluids by the radioimmunoassay and enzyme-linked immunosorbent assay (ELISA) has been described (Doan, 2004; Mouser et al., 2007). In the present case, a diagnosis of ricin toxicosis was also established on the basis of clinical signs, laboratory, pathologic findings and