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Short Communication

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Guidelines for Treatment of Monensin Toxicosis in Dogs

Lynn Hovda^{1,2}, Renee Schmid², Ahna Brutlag^{1,2}, Angelique Rijpert-Duvivier³ and Frédéric Vangroenweghe^{4,5}*

¹Pet Poison Helpline & SafetyCall International LLC, Bloomington, Minnesota, USA

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Abstract

Monensin is a carboxylic ionophore with several labelled applications for use in poultry and ruminants. In Europe and Great Britain, the claims are for the reduction in the incidence of ketosis in the peri-parturient dairy cow and heifer, and as an aid in the prevention of coccidiosis in poultry. In ruminants, monensin is administered through an intraruminal slow release (95 days) device, that may be emitted in some cases. Farm dogs or dogs walked unattended on farmland may come into contact in case emission takes place before all monensin containing tablets inside the device are dissolved. Furthermore, unintended exposure can happen when dogs have access to devices before administration, due to poor storage strategy or due to devices in reach of dogs during the process of administration. This short communication aims to summarize the most important steps in the treatment of monensin intoxication in dogs through contact with the active ingredient present in the intraruminal device.

Keywords: Monensin, Intoxication, Dog, Treatment advice

Introduction

Monensin is a carboxylic ionophore with several labelled applications for use in poultry and ruminants. It is available globally for administration to various species with different indications depending on country or region. In Europe and Great Britain, the claims are for the reduction in the incidence of ketosis in the peri-parturient dairy cow and heifer, and as an aid in the prevention of coccidiosis in poultry. In ruminants, monensin was administered through an intraruminal slow release (95 days) device equipped

with two wings to prevent return of the device upon rumination activity. However, in some cases, the intraruminal device is emitted before the monensin containing tablets are dissolved and dogs may come into contact with the remaining monensin present inside the device. Furthermore, unintended exposure can happen when dogs have access to devices before administration, due to inappropriate storage or due to devices in reach of dogs during the process of administration.



²College of Veterinary Medicine, University of Minnesota, USA

³Elanco Animal Health Netherlands, BU ruminanats & Swine, Van Deventerlaan AG Utrecht, Netherlands

⁴Elanco Animal Health Belgium, BU Ruminants & Swine, Generaal Lemanstraat Antwerpen, Belgium

⁵Department of Internal Medicine-Reproduction-Population Medicine, Ghent University, Belgium

^{*}Corresponding author: Frédéric Vangroenweghe, Elanco Animal Health Belgium, BU Ruminants & Swine, Generaal Lemanstraat Antwerpen, Belgium, Department of Internal Medicine-Reproduction-Population Medicine, Ghent University, Belgium.

Monensin Intoxication in Dogs

The established acute oral LD_{50} in dogs is 10 mg/kg in females and 20 mg/kg in males [1]. Clinical signs of toxicosis develop rapidly, within 4-24 hours, and can persist for days to months, with some potentially lifelong. Affected organs are those found in the musculoskeletal, cardiovascular, gastrointestinal, respiratory, and neurologic systems. Signs most observed in dogs include anorexia, vomiting, muscle weakness, ataxia, progressive paresis/paralysis beginning in the posterior area and moving forward, recumbency, arrhythmias, seizures, and death [1,2]. Diarrhea is rarely observed in dogs [1]. Persistent/long-term signs include cardiac disease, pa-

resis, and muscle weakness [4-6].

There is no antidote for monensin toxicosis. The exact mechanism of toxicity remains unknown, and therefore the treatment consists of aggressive and prompt supportive care until signs have resolved. Prognosis is poor if prompt treatment is not initiated. Prognosis is moderate to good with prompt treatment.

Treatment Advice for Monensin Intoxication in Dogs

Specific treatment advice following monensin toxicosis consists of specific steps, as given in Table 1.

Table 1: Specific treatment guidelines for monensin exposure and toxicosis in dogs.

N°	Action
1	Stabilize
	a. Ensure a patent and protected airway.
	b. Monitor breathing including respiratory rate – volume, and oxygenation.
	c. Monitor cardiac parameters including heart rate – blood pressure. An electrocardiogram (ECG), if available, should be used to monitor for cardiac arrhythmias.
2	Decontaminate
	a. Emesis should not be induced and activated charcoal should not be administered in animals showing clinical signs as there is a potential for aspiration.
	b. Emesis should be induced in asymptomatic animals if the ingestion occurred within 2 hours.
	I. Canine emetic options for in-hospital.
	1. Apomorphine: 0.03 mg/kg IV (preferred) or 0.04 mg/kg IM-see package insert. If injectable formulations are not available, use of tablets placed in the conjunctival sac can be considered: Place a 6.25 mg tablet (whole or crushed) under the conjunctiva. Alternatively, a crushed tablet (typically 6.25 mg) can be dissolved in saline solution (0.9% NaCl) and instilled in the conjunctival sac. Remove the tablet and rinse with water or saline solution after emesis (i.e., resulting in a dose to effect). After emesis, flush the conjunctival sac to avoid protracted vomiting.
	2. Ropinirole ophthalmic solution, 30 mg/mL: 1-8 eye drops/dog-see package insert.
	II. Suggested canine emetic option for at home or on-farm emergency use by pet owners:
	1. Hydrogen peroxide, 3%: 1 mL/kg PO, can repeat with 2mL/kg dose if no vomiting in 15 minutes. In general, do not exceed the described two doses or 50 mL/dog, whichever is lower. Feeding prior to administration increases the likelihood of emesis. [Guidance for local adaptation: The use of hydrogen peroxide, 3%, is recommended for at-home emesis by veterinary professionals who are specifically trained in toxicology. Use of this product should be determined on a case-by-case basis in consideration of the dog's signalment, time since exposure, toxicant, current health status, and other factors. Adverse effects can include esophagitis and gastric lesions (erosions, ulcerations, etc.). Use of this product as a canine emetic may not be customary in all countries, nor will it be appropriate for all at-home situations.]
	c. Following induction of emesis, if applicable, and prior to the administration of activated charcoal, administer an anti-emetic (e.g., maropitant citrate 1mg/kg subcutaneous (SC), ondansetron 0.5-1mg/kg intravenous (IV), metoclopramide 0.2-0.5mg/kg SC or intramuscular (IM)) to minimize the likelihood of additional vomiting. Be mindful that latent emesis in dogs with concurrent gastrointestinal obstruction from ingestion of intraruminal devices may be masked by antiemetics. If obstruction is suspected, use anti-emetics judiciously.
	d. Administer activated charcoal (1-2 grams/kg) with a cathartic (e.g., sorbitol, lactulose) to asymptomatic animals. Due to enterohepatic recirculation of monensin, two additional doses of activated charcoal without a cathartic should be given every 6-8 hours to asymptomatic animals only. Be alert to the development of hypernatremia secondary to charcoal administration. Co-administration with IV fluids and monitoring of serum sodium concentrations is advised.

3 Diagnostics

- a. Complete blood count (CBC), chemistry profile, electrolytes, cardiac troponin I levels (CTnI), urinalysis.
- b. Re-evaluate laboratory work daily to assess the response to therapy and organ damage.
- c. Hematocrit (HCT) and total protein (total solids) concentration aide in determining hemoconcentration associated with vomiting and dehydration.
- l. Electrolytes, including an increase in potassium secondary to rhabdomyolysis, are monitored to assist with fluid therapy.
- e. Creatine kinase (CK) elevation above baseline indicates the onset of muscle damage.
- f. Increasing creatinine, blood urea nitrogen (BUN) and CK elevations are associated with renal damage secondary to rhabdomyolysis and myoglobinuria.
- g. CTnI elevation indicates nonspecific myocardial damage. While not well studied in dogs with monensin toxicosis, serial CTnI concentrations may be used to guide therapy and provide a prognosis. Low concentrations remaining stable over the sampling period are generally associated with a better cardiac outcome. Typically, a baseline CTnI should be obtained, followed by additional samples at 24 to 48 hours, 72 hours, and 7 days.
- 4 | Fluid therapy

Standard isotonic crystalloids at a 1.5-2 X maintenance rate to maintain normal perfusion, protect against acute renal injury associated with myoglobinuria, and to treat dehydration/hemoconcentration. The rate and choice of fluids and electrolyte supplementation should be adjusted based on laboratory results and clinical condition of the animal.

- 5 Ventilation support
 - a. Monitor oxygen saturation (SPO₂) if available.
 - b. Oxygen supplementation and mechanical ventilation as indicated.
- 6 Thermoregulation

Persistent hyperthermia $>39.7^{\circ}$ C ($>103.5^{\circ}$ F) should be treated with cooling measures.

- a. Suggested methods include: recumbency on a cool surface, clipping haircoat on the trunk, tepid water over the skin, ice packs in high blood volume areas (axillary and inguinal regions), ice water rectal enemas for severe refractory hyperthermia.
- Do not actively cool below 39.7°C (103.5°F).
- 7 Physiotherapy and nursing care

Passive range of motion and physical therapy due to significant muscle weakness.

- 8 Intralipid emulsion therapy
 - a. Intravenous lipid emulsion (ILE) therapy can be considered, however, the effectiveness in dogs with monensin toxicosis is currently unknown [6].
 - b. Recommended dosage is 1.5-4mL/kg of 20% ILE given IV as a bolus over 1-2 minutes, followed by 0.25mL/kg/min for 30-60 minutes. Repeat 1.5mL/kg IV bolus every 4-6 hours as long as the serum is not lipemic. Discontinue if no improvement after 24 hours of use.
 - c. ILE: If you are not familiar with this product, contact animal poison control center, referral center.
- 9 Other considerations

Dogs ingesting the intraruminal device may be at risk for gastrointestinal obstruction.

These are guidelines only. Treatment should be based on a complete history, physical examination, and patient assessment by a veterinarian.

Discussion with Elanco Animal Health and / or use of this treatment guideline should in no way replace consultation with appropriate veterinary emergency or referral center personnel.

Acknowledgement

None.

Conflict of Interest

None.

References

1. Novilla M (2018) Ionophores. In: Gupta R., Veterinary Toxicology. 3rd

edition. Elsevier: 1073-1091.

- Bosch L, Bersenas AM, Bateman S (2017) Acute polyneuropathy with respiratory failure secondary to monensin intoxication in a dog. J Vet Emerg Crit Care 28 (1): 62-68.
- C Caldeira, WS Neves, PM Cury, P Serrano, MA Baptista, et al. (2001) Rhabdomyolysis, acute renal failure, and death after monensin Ingestion. Am J Kidney Dis 38: 1108-1112.
- Condon FP, McKenzie RA (2002) Fatal monensin toxicity in a dog after chewing a bovine intra-ruminal slow-release device. Aust Vet Pract 32: 179-180
- Langhorn R, Willesen JL (2016) Cardiac troponins in dogs and cats. J Vet Int Med 30: 36-50.
- 6. (2025) Plumb DC. Plumb's Veterinary Drugs.