GUIDELINES FOR TREATMENT OF MONENSIN TOXICOSIS IN DOGS

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/heifer and as an aid in the prevention of coccidiosis in poultry.

The established acute oral LD_{50} in dogs is 10mg/kg in females and 20mg/kg in males (Novilla, 2018). 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 commonly 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 (Novilla, 2018 and Bosch, 2017). Diarrhea is rarely observed in dogs (Novilla, 2018). Persistent/long-term signs include cardiac disease, paresis, and muscle weakness.

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

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 and 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 use:
            1. Apomorphine: 0.03 mg/kg IV (preferred) or 0.04 mg/kg IM—see package insert.
                a. 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 a saline solution (0.9% NaCl) instilled in the conjunctival sac. Remove the tablet and rinse with water or saline solution after emesis (resulting in a dose to effect). After emesis, flushing of the conjunctival sac to avoid protracted vomiting may be recommended.
            2. Ropinirole ophthalmic solution, 30 mg/mL: 1-8 eye drops/dog—see package insert.
        ii. Suggested canine emetic option for at home/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 50 mL/dog. Feeding prior to administration increases likelihood of emesis. [Guidance for local adaptation: The use of hydrogen peroxide, 3%, is recommended for at home emesis by the 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 antiemetics 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.
        i. Re-evaluate laboratory work daily to assess response to therapy and organ damage.
        ii. Hematocrit (HCT) and total protein (total solids) concentration aide in determining hemoconcentration associated with vomiting and dehydration.
        iii. Electrolytes, including an increase in potassium secondary to rhabdomyolysis, are monitored to assist with fluid therapy.
        iv. Creatine kinase (CK) elevation above baseline indicates the onset of muscle damage.
        v. Increasing creatinine, blood urea nitrogen (BUN) and CK elevations are associated with renal damage secondary to rhabdomyolysis and myoglobinuria.
        vi. 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
    a. 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 (SPO_2) if available.
    b. Oxygen supplementation and mechanical ventilation as indicated.
6. Thermoregulation
    a. Persistent hyperthermia >39.7˚C (>103.5˚F) should be treated with cooling measures.
        i. 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.
        ii. Do not actively cool below 39.7˚C (103.5˚F).
7. Physiotherapy and nursing care
    a. 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.
    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 your local emergency clinic or referral center.
9. Other considerations
    a. 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.

This treatment guideline was adapted from:

Bosch L, Bersenas AM, Bateman S. Acute polyneuropathy with respiratory failure secondary to monensin intoxication in a dog. J Vet Emerg Crit Care 2017; 28(1):62-68.

Caldeira C, Neves WS, Cury PM, et al. Rhabdomyolysis, acute renal failure, and death after monensin Ingestion. Am J Kidney Dis 2001; 38(5):1108-1112.

Condon FP, McKenzie RA. Fatal monensin toxicity in a dog after chewing a bovine intra-ruminal slow-release device. Aust Vet Pract 2002; 32(4):179-180.

Langhorn R, Willesen JL. Cardiac troponins in dogs and cats. J Vet Int Med 2016; 30(1):36-50.

Novilla M. Ionophores. In: Gupta R. *Veterinary Toxicology*. 3rd ed. Elsevier, USA; 2018: pp 1073-1091.

Plumb DC. Plumb's Veterinary Drugs. https://plumbs.com/ Accessed June and July 2021.

Prepared by SafetyCall International, LLC, Bloomington, MN, USA. Date of preparation August 2021.