Metaldehyde Toxicosis in Dogs

Jessica M. Kitagaki, BS, CVT, VTS (CP-C/F)
The Parkway Veterinary Hospital
Lake Oswego, Oregon

Metaldehyde is an organic compound commonly used as bait (molluscicide) to control unwanted snail and slug populations in yards. Metaldehyde toxicosis is commonly reported in western coastal areas of the United States, where slugs and snails are more prominent. Many commercial slug-bait products contain at least 3.5% metaldehyde and come in many forms: liquid, pellets, powder, and granules (FIGURE 1). Dogs are most commonly exposed to metaldehyde through direct ingestion. To attract snails and slugs, metaldehyde is combined with bran and molasses for palatability, unfortunately making it attractive to dogs. Liquid forms may be inadvertently ingested through grooming after walking over treated areas. The lethal dose is 100 to 300 mg/kg, which equates to about 1 tsp per 4.5 kg of body weight, but doses of ≥2 mg/kg warrant decontamination.

Mechanism of Action
The gastrointestinal (GI) tract is a common route of metaldehyde absorption in dogs, but the respiratory tract (inhalation) and the dermis (contact) are other potential paths. When exposed to gastric secretions, metaldehyde is partially converted into acetaldehyde, which undergoes enterohepatic circulation, but the exact mechanism of action is unknown. Metaldehyde and acetaldehyde readily cross the blood-brain barrier, resulting in a decrease in the neurotransmitters serotonin, noradrenaline, and γ-aminobutyric acid. Serotonin and γ-aminobutyric acid are inhibitory neurotransmitters that calm the brain, while noradrenaline is excitatory and stimulates the brain. As these neurotransmitters decline, neuronal excitability increases proportionally, decreasing the seizure threshold. Metaldehyde is eliminated in the feces and urine; only 1% of the oral dose remains unchanged in the urine. The elimination half-life of metaldehyde is 27 hours; therefore, intensive care may be lengthy, sometimes more than 4 days.

Clinical Signs and Patient Assessment
Neurologic effects are the primary complication of metaldehyde toxicosis. Typically, clinical signs appear 1 to 4 hours after ingestion, but their onset is highly correlated with GI transit time. The clinical signs are primarily neurologic, including ataxia, agitation/anxiousness, and hyperesthesia (BOX 1). The most common clinical sign in dogs is severe muscle tremors, which, if untreated, can progress to continuous tonic convulsions. Hyperthermia due to increased muscle activity can be severe. Organ necrosis at the cellular level follows once body temperature exceeds 107°F (41.7°C). Other clinical signs include vomiting (metaldehyde is a gastric irritant), diarrhea, hypersalivation, tachypnea (respiratory compensation of metabolic acidosis), and tachycardia. Metabolic acidosis occurs due to metaldehyde metabolites and the accumulation of lactic acid due to muscle tremors and/or seizures. Untreated, high-level exposure to metaldehyde can result in death by respiratory failure within 4 to 24 hours after ingestion. Patients that survive this period may succumb to liver failure 2 to 3 days after ingestion.

Box 1. Clinical Signs of Metaldehyde Toxicosis

- Agitation/anxiousness
- Muscle tremors
- Ataxia
- Seizures
- Hyperesthesia
- Tachycardia
- Hypersalivation
- Tachypnea
- Hyperthermia
- Vomiting/diarrhea

Figure 1. Commercial slug baits are attractive to dogs due to palatability and a kibble-like appearance.
Metaldehyde Toxicosis in Dogs

Diagnosis
The diagnosis of metaldehyde toxicosis is usually presumptive because ingestion is often not witnessed and specific toxicologic testing is not readily available. A presumptive diagnosis is based on history of possible exposure, physical examination findings, and response to therapy. In some patients, a mild formaldehyde-like odor may be noted in the vomitus or on the breath. Additionally, finding blue or green pellets in the stomach contents can be indicative of slug bait, but many formulations mimic dog kibble. Some slug-bait formulations contain organophosphates or arsenic in addition to metaldehyde, which can obscure the diagnosis. A serum biochemical profile may reveal complications due to dehydration (i.e., an increased total protein concentration) and increased muscle activity (i.e., an increased creatine kinase concentration). Acid-base status may reveal metabolic acidosis with compensatory respiratory alkalosis. Necropsy findings are generally nonspecific.

Patient Management
There is no antidote for metaldehyde poisoning, so treatment is aimed at controlling clinical signs until the toxin is eliminated from the body. Metaldehyde metabolism can take 4 days or longer, but patients generally require 24 to 72 hours of hospitalization. If ingestion was recent and the patient is asymptomatic, emesis should be induced using hydrogen peroxide or apomorphine. Patients presenting with severe muscle tremors or seizures must be anesthetized for gastric lavage due to the increased risk of aspiration. Decontamination is followed by administration of activated charcoal, preferably with an osmotic cathartic (i.e., sorbitol or sodium sulfate) to reduce absorption of the toxin from the GI tract. For treating acute toxicosis, the recommended dose of activated charcoal is 1 to 4 g/kg PO, which may be repeated if desired. If activated charcoal is used with sorbitol, the initial dose should not be repeated because osmotic diuretics are known to cause hypernatremia. Multiple cool-water enemas are indicated to rid the colon of the toxin and to treat hyperthermia.

Muscle tremors and seizures are treated with muscle relaxants, benzodiazepines, and, if necessary, general anesthesia. Methocarbamol, a muscle relaxant that acts through the central nervous system (CNS), controls muscle tremors. It is administered at a dose of 44 to 150 mg/kg IV, as needed, to control tremors, but the dose must not exceed 330 mg/kg/d. Methocarbamol is not sufficient to control tremors or if the patient is experiencing seizures, a benzodiazepine such as diazepam or midazolam should be administered. Diazepam is used to control anxiety and convulsions and to induce muscle relaxation. The dose of diazepam is 2 to 5 mg/kg IV, to effect, to treat metaldehyde toxicosis. Seizures that are resistant to treatment with benzodiazepines are treated using general anesthesia (e.g., administration of propofol, isoflurane, or sevoflurane). The use of barbiturates, such as pentobarbital, is contraindicated in patients with metaldehyde toxicosis because this drug class competes with acetaldehyde plasma-binding sites, leading to increased toxicosis.

It is not unusual for a patient to present with a temperature of 108°F (42.2°C) after ingesting slug bait. Hyperthermia is secondary to increased muscle activity and is treated with basic cooling measures. Cool-water baths and enemas, application of isopropyl alcohol to the plantar surface of the feet, and intravenous fluid therapy are effective at managing an increase in body temperature. As with all animals being treated for hyperthermia, it is important to monitor for hypothermia and discontinue cooling treatments when the patient's temperature reaches 103°F (39.4°C). Body temperature should be monitored regularly because even mild muscle spasms over long periods can dramatically increase patient temperature.

Glossary

Arsenic—a highly poisonous metal used in producing pesticides, herbicides, and insecticides
Ataxia—diminished or absent voluntary muscle movement
Cathartic—a substance that is administered to accelerate defecation
Dyspnea—difficulty breathing
Enterohepatic circulation—circulation of substances, such as bile or drugs, from the liver to the small intestine and back to the liver
Half-life—the time it takes a drug to reach half of its pharmacologic activity
Hyperesthesia—abnormal increased sensitivity of the senses, such as sight, hearing, or touch
Hypersalivation—increased production of saliva
Hyperthermia—increased body temperature caused by the absorption or production of more heat than the body can dissipate
Metabolic acidosis—increased plasma acidity
Molluscicide—a pesticide used to bait mollusks such as snails and slugs
Neurotransmitter—a chemical that transmits signals from one neuron to the next across a synapse
Organophosphate—an insecticide that causes inhibition of acetylcholinesterase, resulting in an increased level of acetylcholine in the body
Respiratory alkalosis—decreased carbon dioxide in the blood due to excessive respiration
Tachycardia—an increased heart rate
Tachypnea—an increased respiratory rate
Tonic convolution—a convulsion in which muscle rigidity is prolonged, also known as a grand mal seizure
Key Points

- Metaldehyde is the active ingredient in many commercial slug and snail baits and is extremely attractive to dogs due to its palatability and appearance.
- Signs of metaldehyde toxicosis (e.g., muscle tremors, hyperthermia, seizures) generally appear 1 to 4 hours after ingestion.
- There is no antidote for metaldehyde poisoning, so treatment is aimed at limiting further absorption and controlling clinical signs.

Dehydration, electrolyte imbalances, hyperthermia, and acidosis can be corrected with intravenous fluid therapy. Lactated Ringer solution or 5% dextrose is recommended for treating acidosis (lactate is converted to bicarbonate), preventing liver damage, and helping remove toxins. In severe cases, blood gas analysis may be needed to monitor acidosis. Acidosis may be treated with sodium bicarbonate, but this is recommended only if there is access to blood gas analysis.

The most common cause of death in patients that have ingested metaldehyde is respiratory failure. Some patients may benefit from flow-by oxygen. Endotracheal tubes should be available if severe dyspnea or respiratory arrest develops and artificial respiration is required.

Patients that appear to recover may develop liver failure 2 to 3 days after ingesting metaldehyde. Therefore, liver enzymes should be analyzed and clinical signs of liver disease (i.e., icterus) monitored daily for 2 to 3 days after ingestion.

Prognosis and Prevention

Metaldehyde toxicosis can be fatal, but if it is caught early and treated aggressively, most patients recover. Clients should be educated about household and garden products that are harmful to their pets. If slug bait is used, the unused portion should be kept in a tightly sealed container that prevents access by pets. Dogs must be restricted from treated areas for at least 2 weeks.

Although the US Food and Drug Administration has approved a slug and snail bait (Sluggo, Lawn and Garden Products, Inc., Fresno, CA) for generally safe use, the active ingredient of the product is iron phosphate, ingestion of which can result in iron toxicity. Educate clients to keep pets away from this product as well.

Because there is no pet-safe slug and snail bait, preventing ingestion of these products is crucial.

References

1. Which chemical is not a common ingredient in slug and snail bait?
   - a. iron phosphate
   - b. metaldehyde
   - c. acetaldehyde
   - d. organophosphate

2. What is the lethal dose of metaldehyde?
   - a. 2 mg/kg
   - b. 100 to 300 mg/kg
   - c. 1 tsp per 4.5 kg of body weight
   - d. b and c

3. How do metaldehyde and acetaldehyde affect the central nervous system (CNS)?
   - a. They decrease the neurotransmitters serotonin, noradrenaline, and γ-aminobutyric acid.
   - b. They decrease the activity of the CNS.
   - c. They do not affect the CNS; they only cause liver failure.
   - d. They increase the seizure threshold.

4. What is the primary body system affected by metaldehyde toxicosis?
   - a. the renal system
   - b. the hepatic system
   - c. the neurologic system
   - d. the GI system

5. How is metaldehyde poisoning commonly diagnosed?
   - a. by clinical signs and a history of exposure
   - b. by blood metaldehyde analysis
   - c. by finding an increase in hepatic enzymes on a serum biochemical profile
   - d. by finding metabolic acidosis on a blood gas analysis

6. What is the antidote for metaldehyde poisoning?
   - a. a benzodiazepine such as midazolam or diazepam
   - b. apomorphine
   - c. a barbiturate such as pentobarbital
   - d. There is no antidote.

7. What is a life-threatening, secondary effect of muscle tremors?
   - a. convulsions
   - b. hyperthermia
   - c. hyperesthesia
   - d. respiratory arrest

8. How long can metaldehyde remain active in the body?
   - a. <4 days
   - b. 24 to 72 hours
   - c. >4 days
   - d. >10 days

   - a. 1 to 4 hours
   - b. 12 hours
   - c. 24 to 72 hours
   - d. 4 days

10. Which of the following is a pet-safe slug bait?
    - a. iron phosphate
    - b. organophosphate
    - c. beer
    - d. none of the above; there is no pet-safe alternative