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Bromethalin

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Bromethalin, a nonanticoagulant, single-dose rodenticide, is a neurotoxin available as bars (blocks), pellets, seed, and worm. Mole baits are sold as worm containing 0.025% bromethalin, whereas rat and mouse baits contain 0.01% bromethalin. Bromethalin and its main metabolite desmobromethalin are strong uncouplers of oxidative phosphorylation. This results in intra-myelin fluid accumulation, leading to long nerve demyelination and intra-myelin cerebral edema. The net result is cerebral and spinal edema and increased CSF pressure, leading to neurologic dysfunction. In toxicity trials, the oral toxic dose of bromethalin when used as part of bait appears to be much lower than the dose administered as a technical grade agent. For example, in dogs, an average lethal dose of technical grade bromethalin is reported to be 4.7 mg/kg but 2.38 mg/kg in bait. Young dogs (<1 yr old) appear more sensitive; death has been reported at dosages of ~1 mg/kg in bait. Dogs are more commonly involved. Cats are 2–3 times more sensitive than dogs.

Bromethalin can cause either an acute or a subacute/chronic syndrome, depending on the dose ingested. At doses equivalent to or more than the average lethal dose, dogs may develop an acute convulsant (or high-dose) syndrome resulting in clinical signs within 4–36 hr of exposure; such signs include hyperexcitability, muscle tremors, grand mal seizures, hindlimb hyperreflexia, CNS depression, hyperthermia, and death. The paralytic (subacute or chronic) syndrome is seen at lower doses, and clinical signs may not appear for several days (up to 7 days) after exposure. Initial signs may include depression, hindlimb weakness or paresis, decreased propioception, ataxia, and possible tremors. Muscle weakness often progresses from posterior to anterior muscles. Cats typically develop paralytic syndrome irrespective of dose of bromethalin.

Presumptive diagnosis of bromethalin toxicosis is made based on known or suspected history of exposure to the bait, followed by development of neurologic signs within 1–7 days of exposure. Diagnosis can be confirmed by detecting the presence of bromethalin or its major metabolite in liver, kidney, brain, or fat; this analysis is available only in some veterinary diagnostic laboratories in the USA. Based on the history of exposure, bromethalin toxicosis should be considered when there is moderate to acute onset of weakness, hindlimb paralysis, tremors, and seizures. Some other toxicologic and nontoxicologic differential diagnoses should include ethylene glycol toxicosis, marijuana ingestion, 2,4-D and other phenoxyacetic acid herbicide toxicosis, copper head snake envenomation (cats), intervertebral disc problems, spinal cord and CNS trauma, and tick paralysis.

Treatment of bromethalin toxicosis is aimed at early decontamination (induction of emesis and administration of activated charcoal) in an asymptomatic animal and controlling CNS signs (seizures) and providing supportive care in a symptomatic animal. Emesis using 3% hydrogen peroxide solution or apomorphine in dogs and xylazine in cats within 4 hr of ingestion may remove some bait from the gut. Depending on the ingested dose of bromethalin, administration of activated charcoal is considered an effective method to prevent toxicosis. Bromethalin undergoes enterohepatic recirculation, so administration of repeated doses of activated charcoal may be helpful. The following can be used as a guideline to treat bromethalin exposure in dogs and cats:

At a bromethalin dosage of 0.1–0.49 mg/kg in dogs, or 0.05-0.1 mg/kg in cats, emesis alone within 4 hr of exposure may be adequate. If emesis is not successful, or if > 4 hr have elapsed since ingestion, a single dose of activated charcoal at 1–2 g/kg body wt is indicated. Whenever administering activated charcoal, the clinician must remain aware of the risk of aspiration or hypernatremia secondary to fluid shift into the gut. The clinical signs of acute hypernatremia may mimic those seen with bromethalin toxicosis.

At a bromethalin dosage of 0.5–0.75 mg/kg in dogs, or 0.1–0.3 mg/kg in cats, an initial dose of activated charcoal at 1–2 g/kg body wt should be considered. A repeat dose at half the original dose at 8-hr intervals for a total of three doses can be administered, again being aware of and monitoring for possible hypernatremia.

At a bromethalin dosage of ≥0.75 mg/kg in dogs, or ≥0.3 mg/kg in cats, administration of six doses of activated charcoal over 48 hr (repeat every 8 hr) can be considered to reduce the body burden by interrupting enterohepatic recirculation.

Use of mannitol and corticosteroids has been suggested to treat clinical signs, because they may help manage cerebral edema due to other causes. However, this has not been shown to be very helpful, likely because of the presence of intra-myelin edema. Diazepam, barbiturates, and other anticonvulsant medications should be used to control seizures and other CNS signs. Full recovery may require days to weeks of treatment.