A REVIEW OF FERRET TOXICOSES

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Abstract

Within the past 20 years, ferrets have steadily gained popularity as a household pet. Owing to their small size, ability to climb, and explore under furniture and behind appliances, curious ferrets are often exposed to misplaced items. This ability to search secluded areas within the house provides opportunities for the animal to interface with toxic substances, of which the owner may be totally unaware. Owing to their low body weight, ferrets are at a higher risk of toxicosis since they obtain higher doses of the toxin on a mg/kg basis. Improperly applied flea and tick products, labeled for other pets, may also result in an adverse drug reaction. Copyright 2015 Elsevier Inc. All rights reserved.

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Treatment of toxicoses in pet ferrets is similar to other mammals, initially evaluating and stabilizing the patient.[1] The clinician should also determine if the owner actually witnessed the ferret's exposure to the toxin or if it is an assumed intoxication. If the owner did not observe the exposure, performing a diagnostic workup is recommended to gain a better assessment of the patient's condition.[1] The owner should provide a list of all possible plants, medications, and other possible toxicants that the patient may have contact with on a daily basis.

Treatment of a ferret that has been exposed to a toxic substance depends on the specific toxin involved and clinical disease conditions related to the body's reaction to that substance. When appropriate, ingested substances that will cause adverse physiological reactions may be initially removed from the body by inducing emesis with 3% hydrogen peroxide or apomorphine.[2] If corrosive materials are ingested, the ferret should be administered water or milk, orally, in small volumes (0.25 to 0.5 teaspoons), to dilute the substance[1]; induction of emesis in these cases is contraindicated. The ferret dose for activated charcoal (AC) is 1-3 g/kg,[1] if indicated based on patient history and physical examination. When administering AC there is a risk to the patient for aspiration and hypernatremia. It is recommended to monitor for hypernatremia 3 to 4 hours after each dose of AC.[2] AC does not bind all toxins, including heavy metals, xylitol, hydrocarbons, and alkali materials (including bleach).

Some of the more common ferret exposures reported to the American Society for the Prevention of Cruelty to Animals Animal Poison Control Center, January 2005 to January 2015, are rodenticides, ibuprofen, detergents, topical flea products, acetaminophen, and venlafaxine.

RODENTICIDES

There are 3 commercially available rodenticide products that are commonly purchased for indoor or outdoor use: anticoagulants, bromethalin, and cholecalciferol. Most of the commercial rodenticides have added attractants (e.g., peanut butter) to entice mice. Unfortunately, ferrets are also attracted to the scents added to the final product.

Anticoagulant rodenticides can cause coagulopathies within 3 to 7 days after ingestion.[3] Treatment of ferrets that have ingested anticoagulant rodenticide–containing compounds begins with inducing emesis if the

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exposure has been within 4 hours of presentation. Vitamin K_1 can be given in observed or suspected ingestions. The dose is 2.5-5 mg/kg divided q8-12hr. Vitamin K1 is given for 14 days for warfarin exposure, 21 days for bromodiolone exposure, and 30 days for other anticoagulants.[4,11] If the ferret is at risk because of previous exposures to the rodenticide, monitor a baseline prothrombin time (PT). The patient's PT should also be monitored for 48 to 72 hours after the last dose of vitamin K_1. Once the ferret is released from the hospital, the owner should observe the animal at home for any changes in activity levels, appetite, sleep, and any visible petechiation or bruising. If the ferret does develop signs of continued toxicosis, monitor PT and activated partial thromboplastin time as well as a packed cell volume and total protein. Normal PT in a ferret is 10.3 $\pm$ 0.1 seconds and activated partial thromboplastin time is 18.4 $\pm$1.4 seconds.[5] The ferret may require a plasma transfusion or whole blood transfusion if initial treatment is unsuccessful.

Bromethalin and the metabolite desmethylbromethalin uncouple oxidative phosphorylation in mitochondria of the central nervous system (CNS). This can lead to edema of myelin sheaths within the CNS. By ingesting small amounts of bromethalin or desmethylbromethalin, mild clinical signs (gastrointestinal [GI] upset, lethargy, and ataxia) may be observed.[6] When increased amounts of the rodenticide are ingested, clinical signs become more severe and include paresis, paralysis, seizures, and death.[6,7] Clinical signs associated with bromethalin or desmethylbromethalin toxicosis are usually progressive. Once the ferret is exhibiting CNS signs that may be classified as seizure activity and/or paralysis, the prognosis is poor. As one would expect, treatment for bromethalin or desmethylbromethalin toxicosis in ferrets primarily involves decontamination and supportive care based on the patient's clinical condition. The ferret may need to receive several doses of AC based on the dose ingested to imitate the decontamination process. The owner must monitor the animal for any CNS signs for several days postexposure. If the ferret does develop CNS signs after the rodenticide exposure, control of cerebral edema with mannitol, dexamethasone, and furosemide may temporarily lower CSF pressure, but the clinical condition may remain and progress once these treatments are discontinued.[8] Seizure activity can be controlled with diazepam, barbiturates, or general anesthesia. Supportive care is also crucial to recovery, with fluid therapy and nutritional and environmental support (critical care unit) being important aspects of this treatment plan. Ginko biloba has been shown to reduce the development of cerebral edema in rats given toxic doses of bromethalin and may be worth considering in ferrets that are presented with CNS signs.[9]

Cholecalciferol ingestion can cause hypercalcemia and hyperphosphatemia. The elevated mineral levels will often be the underlying cause of secondary soft tissue mineralization which results in acute renal failure (ARF). Initial management of cholecalciferol exposure consists of decontamination by inducing emesis, AC doses, obtaining baseline renal values, and monitoring serum calcium and phosphorous concentrations every 24 hours for a total of 4 days. The owner must monitor the ferret at home for disease signs consistent with ARF (e.g., polydipsia, polyuria, vomiting, anorexia, lethargy) for 4 days. If the ferret does develop clinical signs of cholecalciferol toxicosis, start treatment with saline diuresis, furosemide, prednisolone, phosphate binders, and a low calcium diet.[8] In some cases it may be necessary to use pamidronate[10] or calcitonin.[11]

IBUPROFEN

Ibuprofen, a common non-steroidal anti-inflammatory drug, can be purchased "over the counter" or obtained through prescription. Non-steroidal anti-inflammatory drug products are available as tablets, liquid filled capsules, chewable tablets, film coated tablets, and suspensions (40 and 100 mg/ml).[12] When an overdose occurs, ibuprofen inhibits prostaglandins that protect the gastric mucosa, potentially leading to GI ulcerations.[13] In dogs and cats ibuprofen intoxication can lead to GI ulceration, ARF, and CNS signs. However, in ferrets the most common clinical signs associated with ibuprofen intoxication are neurological (e.g., ataxia, tremors, depression, and coma), followed by GI and renal disease.[14] A single 200 mg ibuprofen tablet may be enough to provide a lethal dose (220 mg/kg)[13] in a 900 g ferret. Treatment for ferrets diagnosed with ibuprofen toxicosis consists of supportive care and addressing clinical disease problems. Decontamination and AC may have to be delayed until the ferret is stable, although treatment to protect the GI mucosa should be started as soon as possible with sucralfate and famotidine. Renal values should be monitored every 24 hours for at least 48 hours, and preferably 72 hours. Fluid diuresis at twice maintenance is recommended for a minimum of 48 hours to maintain renal blood