Common Rodenticide Toxicoses in Small Animals

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• Rodenticide • Anticoagulant • Bromethalin • Cholecalciferol

This article focuses on the 3 most commonly used rodenticide types: anticoagulants, bromethalin, and cholecalciferol. Since there are multiple types of rodenticides available on the market and the color of the bait is not coded to a specific type of rodenticide, it is important to verify the active ingredient in any rodenticide exposure. Additionally, many animal owners may use the term "D-con" to refer to any rodenticide regardless of the actual brand name or type of rodenticide. Rodenticide baits are most typically formulated as bars. Loose bait such as pellets are no longer produced for consumer sale according to new Environmental Protection Agency (EPA) risk mitigation rules; however, this form (loose bait) may be seen for quite some time while older products are used up. The EPA released their final ruling on rodenticide risk mitigation measures in 2008 and all the products on the market had to be compliant by June 2011. The purpose of the measures is to reduce exposures to children and nontarget species including wildlife. After June 2011, consumer products may not contain the second-generation anticoagulants brodifacoum, difethialone, difenacoum, and bromadiolone and instead must contain either first-generation anticoagulants or nonanticoagulants including bromethalin and cholecalciferol.[1] These regulations are likely to cause an increase in the number of bromethalin and cholecalciferol cases seen in veterinary clinics.

ANTICOAGULANT RODENTICIDES

The discovery of the causative agent of sweet-clover poisoning in cattle, dicoumarol, led to the development of the anticoagulant rodenticides. Cattle suffering from this type of poisoning developed internal bleeding; therefore, dicoumarol was tested as a rodenticide. Warfarin, named after the Wisconsin Alumni Research Foundation (WARF), was the first compound marketed as an anticoagulant rodenticide. The

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first-generation anticoagulants were created during the 1940s and 1950s. They required continuous exposure to achieve rodent control. The second-generation anticoagulant rodenticides (SGARs), including brodifacoum, difethialone, difenacoum, and bromadiolone, were developed in the subsequent decades as rodents developed resistance to the first-generation anticoagulants. SGARs were formulated to be more palatable to rodents, more effective, faster, and longer acting.[2] Although chlorophacinone and diphacinone were developed after warfarin like the SGARs listed above, they differ structurally and the EPA has not placed the same restrictions on their use.[1,2]

Warfarin and pindone are short-acting anticoagulants with shorter half-lives (<24 hours) compared to the long acting products whose half-lives are up to 6 days.[3] The long-acting anticoagulants include diphacinone, difethialone, chlorophacinone, brodifacoum, and bromadiolone. Veterinarians are well-trained to use their knowledge and judgement to make treatment decisions for their patients according to each unique case. As a general guideline, the ASPCA Animal Poison Control Center (APCC) recommends decontamination if and as needed and monitoring (prothrombin PT] time or activated partial thromboplastin time [APTT]) or treatment with vitamin K_1 (if and as needed) when the ingested dose of warfarin is greater than 0.5 mg/kg and other anticoagulants is greater than 0.02 mg/kg.

Exposure in domestic pets occurs through ingestion of the product from the bait container or from the environment to which the rodents have carried the bait. Now that the EPA is requiring consumer products be contained in a tamper-resistant bait stations and has prohibited the sale of pelleted formulations to consumers, hopefully pets will be protected from finding a rodent's hoard of product. For anticoagulants, toxicosis from a pet ingesting rodents poisoned by the bait (also called relay toxicosis) is of limited concern since the amount of rodenticide in the rodent is small. However, if the pet is very small and ingests a large number of the poisoned rodents, relay toxicosis is possible. For example, a barn cat that preys on rodents as its main source of nutrition could become intoxicated if those rodents were poisoned by an anticoagulant rodenticide.[3]

Pathophysiology and Clinical Signs

The anticoagulant rodenticides cause their effects by interfering with the production of the clotting factors II, VII, IX, and X by the liver. In the normal production of these factors, vitamin K_1 is converted to vitamin K_1 epoxide. The enzyme vitamin K_1 epoxide reductase then converts vitamin K_1 epoxide back to the active form of vitamin K_1. This cycle repeats over and over to create active clotting factors. The anticoagulants inhibit vitamin K_1 epoxide reductase, thereby leading to depletion of active vitamin K_1 and the halt of the production of active clotting factors.[3,4]

During the first 36 to 72 hours following ingestion of the anticoagulant, the patient is usually clinically normal as the clotting factors are slowly depleted. Usually within 3 to 5 days, enough clotting factors are depleted for hemorrhage to develop. It is possible in some patients with underlying illnesses (such as preexisting bleeding disorders or hepatic disease), depletion of coagulation factors may occur sooner, resulting in hemorrhage as early as 24 to 48 hours following exposure.

Many poisoned animals are not presented to the veterinarian until clinical signs develop. It is important to remember that hemorrhage can occur anywhere in the body; however, the most common clinical signs are dyspnea, coughing, lethargy, and hemoptysis.[3] Bleeding into body cavities such as the chest, abdomen, and joints is also common. Many patients present with vague clinical signs of lethargy, weakness, and anemia without any overt external hemorrhage, although some animals may