Brodifacoum poisoning in backyard chickens

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Introduction. In order to overcome the problem of controlling rodents that become genetically resistant to warfarin, second-generation anticoagulant rodenticides were developed. These rodenticides are much more potent as they have a longer biological half-life/faster action so only low levels are needed to kill rodents after the ingestion of a single lethal dose. Unfortunately, non-target species - mammals (dogs, cats, livestock, wildlife) and birds (domestic poultry/captive wild birds) - can also be affected by these toxic compounds. Chickens can ingest these chemicals accidentally via commercial bait formulations, by malicious intent, or by pecking poisoned rodents; accumulations in their livers may pose a risk to human health. These pesticides block the synthesis of functional vitamin K-dependent clotting factors by affecting the vitamin K enzyme complex in the liver. In birds, depletion of these factors prolongs the extrinsic and common coagulation pathways preventing blood clotting and causing widespread hemorrhage and death due to severe blood loss/hypovolemic shock.

Material and methods/results. In mid-March 2007, one of the chickens from a small backyard flock of 10 birds (fighting cock roosters and layer hens) was brought to our diagnostic laboratory for necropsy. Six birds had died within 2 weeks. Hematemeses was observed in most of the affected birds and, when moribund, whole body shaking was also noted; some were just found dead. Deaths occurred within 24-48 h after birds were noticed sick. Oral activated charcoal was given to the affected birds but none recovered. Necropsy findings included clotted blood on the beak and nares and in the oral cavity and tracheal lumen, dark tarry contents in the esophagus/crop/proventriculus/gizzard, locally extensive pulmonary hemorrhage, paleness of the internal organs, petechiae around the sciatic nerves, multiple ecchymoses in the thymus, bloody contents in the ceca, and presence of a blood clot in the coelomic cavity on the dorsal lobe of the pancreas. Based on the gross lesions, anticoagulant rodenticide poisoning was suspected. Anticoagulant screening on the liver and gizzard contents included testing for warfarin, bromodiolone, coumachlor, brodifacoum, diphacinone, chlorophacinone, and difethialone. Brodifacoum was detected in the liver but not in the gizzard contents. Histopathology confirmed hemorrhage in different organs and tissues. Results of other laboratory tests were either negative or non-significant, except for the presence of a moderate amount of coccidia in the cecal contents. The source of brodifacoum was found to be a nearby dump/storage area where neighbors put anticoagulant baits to get rid of rats. The chickens had free, unlimited access to the area and thus the poison. Deaths ceased right after the access of these chickens to this area was denied.

Discussion/conclusion. Anticoagulant rodenticide poisoning was suspected based on the clinical picture/necropsy findings consistent with a coagulopathy, and was confirmed by the detection of brodifacoum in the liver. No cases of accidental poisoning by brodifacoum or by any other second-generation anticoagulant rodenticide have been documented in chickens previously. Diseases to be considered in the differential diagnosis of anticoagulant rodenticide poisoning in chickens are those characterized by sudden onset, peracute-acute clinical course, high mortality, and in which generalized hemorrhage (petechiation and ecchymoses affecting a multitude of organs and tissues) is observed, e.g., viscerotropic velogenic Newcastle disease, highly pathogenic avian influenza, septicemias, and hemorrhagic syndrome induced by Chicken anemia virus.

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