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INJURIES CAUSED BY RODENTICIDE INTOXICATION IN A CANINE: CLINICAL CASE REPORT

Gabriela Espino-Moreno

Academic Unit of Veterinary Medicine and Zootechnics, Universidad Autónoma de Zacatecas, Zacatecas, Zac ORCID: 0009-0003-6436-5711

Juan Antonio Ramírez-Chequer

Academic Unit of Veterinary Medicine and Zootechnics, ``Universidad Autónoma de Zacatecas``, Zacatecas, Zac ORCID:0000-0001-6400-0390

Victoria Elizabeth Castrellón-Ahumada

Academic Unit of Veterinary Medicine and Zootechnics, ``Universidad Autónoma de Zacatecas``, Zacatecas, Zac ORCID: 0000-0003-2581-2278



All content in this magazine is licensed under a Creative Commons Attribution License. Attribution-Non-Commercial-Non-Derivatives 4.0 International (CC BY-NC-ND 4.0). Abstract: Anticoagulant rodenticides are highly toxic compounds that are used to control rodent plagues, but they can also endanger the health of pet animals such as dogs (Vivancos Cuadros, MD, & Guillén Monzón, N. 2015). This is mainly due to the fact that many of these animals can be exposed to snakes accidentally, or by ingestion of poisoned animals. (Stevers & Palau, 2002). Anticoagulant rodenticides are agents designed specifically for the elimination of rodents (although they can affect any mammal) by inducing coagulopathy by inhibiting the "recycling" of vitamin K (Gómez Baute et al., 2011). Many animals exposed to toxic doses of these rodenticides remain asymptomatic until the exhaustion of active coagulation factors, which is why clinical signs are not observed until 5 months. post-ingestion day. In the case of pets, these are most commonly animals that live in rural areas, plots, hunting dogs or lactating dogs of intoxicated females (Talcott & Murphy, 2013). The most frequent clinical signs are: dysnea, lethargy, coughing, anorexia, apathy, depression, pallor of mucous membranes and weak pulse. In addition to other signs, it is not common, but is the result of a severe lack of coagulation such as tachycardia, pulmonary edema, pleural and pericardial effusion, spontaneous hemorrhages due to encías and saliva, hematomas in bone projections, melena, hematuria, hematochecia, ecchymosis, cojera, paralysis (epidural or subdural hemorrhage), airway obstruction, convulsions upper hemorrhage), (cerebral acute collapse (general hemorrhage). (Sheafor & Couto, 1999; Waddell, et al., 2013). A male canine patient (enteral) Chihuahua cross aged 2 years old weighing 3.5 kg was remitted. He presents himself for consultation with hypersalivation, dysnea and temblor; the patient comes from a rural area of the Community of San Ramón, Guadalupe, Zac., México.

Keywords: Rodenticide, Canine, Intoxication, Coagulopatía.

TEXT

The patient has dysnea, incoordination, vomiting, and no general lesion was observed. The patient sells his house without supervision and has access to the entire property; The owner commented that there is currently a rodent plague where the patient is located, which is why I use rodenticides to prevent and control it. The differential diagnosis includes: poisoning by rodenticides. insect bite poisoning (black widow or alacran), parvovirus and intestinal obstruction. If the patient was treated for possible intoxication, unaware of the origin (active ingredient), it is believed that he accidentally consumed rodenticides.

The treating doctor administered atropine sulfate at 0.15 mg/kg intravenously to inhibit the effect of possible intoxication. state of unconsciousness and continued with the indicated therapy, however, the patient did not respond to treatment, suffering a cardiac arrest and later death.

DISCUSSION

After the information provided by the doctor, there was confusion about the cause of the dog's death. They relate the lesions found in the differential diagnosis and presumptive diagnosis, highlighting the presumptive diagnosis in not finding evidence that supports it; Pathological hallmarks suggest poisoning due to ingestion of rodenticides which presents similarities with clinical signs found in dogs such as vomiting, bloody diarrhea followed by weakness, dysnea, hypersalivation and convulsions. (Sheafor & Couto, 1999; Waddell, et al, 2013) It is important to highlight that coagulopathy is produced by rodenticides that act as anticoagulants; Its mechanism of action is the inhibition of vitamin K1 epoxide

reductase, preventing clot formation (Talcott & Murphy, 2013).

CONCLUSION

The witnesses found suggested that the cause of death was poisoning by rodenticides, which was consumed by the animal within the same property. If the canine ingested a less aggressive toxic agent, it could present a picture of kidney disease, lung collapse, problems in the gastrointestinal system and liver damage. Injuries to these organs would be seriously harmful.

The time factor is very important when an intoxication occurs, which is why it is essential to act quickly to identify the signs present, contact the veterinarian and even identify the agent that caused the damage.

Commonly in clinical practice of small species there are cases of ingestion of rodenticide poisons; Due to the accidental consumption of these or good, to feeding on animals contaminated with this type of poisons.

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DESCRIPTION OF HALLAZGOS IN NECROPSY

EXTERNAL INSPECTION

Mark of blood on the right anterior limb from which treatment was administered.



Image 1. Canine position of right lateral cubit.

INTERNAL INSPECTION



Image 2. Disposition of organs (generalized hemorrhage).



Image 3. Ventral view, Hemorrhagic lungs.



Image 4. Round heart.



Image 5. Hemorrhagic digestive system.



Image 6. Ventral view of the liver, red-dark and hemorrhagic coloration.



Image 7. Portion of duodenum, with hemorrhages and presence of yellow content.



Image 8. Cut the kidney evenly, deepens the black color.

OTHER HALLAZGOS DURING THE NECROPSY

The blood did not clot during the time during which the necropsy was carried out and was probably due to the presence of a toxin, causing a coagulopathy.

HISTOPATHOLOGY



Image 9. Spleen, necrosis and lymphoid depression. (H&E100x)



Image 10. Liver, scattered cytoplasmic material, few nuclei, hepatocytes with vacuoles without cytoplasmic membrane and pyknosis (H&E100x).



Image 11. Kidney, cells with loss of cytoplasmic membrane (necrosis), severe diffuse hemorrhage (H&E40x).



Image 12. Small intestine, multifocal hemorrhages in vellosities, necrosis (H&E40x).



Image 13. Small intestine, presence of eosinophils. (H&E 40x)



Image 11. Heart, degeneration of muscle fibers and severe hemorrhage. (H&E40x).



Image 12. Lung interstitial pneumonia, emphysema, atelectasis, mild edema, lymphoid proliferation, moderate congestion (H&E40x).



Image 13. Lung, lymphocyte infiltrated pneumonia (H&E100x).



Image 14. Brain, neuronal hyperchromasia, satelitosis (H&E40x).



Image 15. Spinal cord, severe generalized congestion, pyknosis, neuronal hyperchromasia, neuronophagia. (H&E 40x).

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