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CASE REPORT

Anticoagulant Rodenticide Toxicity and Secondary Haemostatic Disorder in a Dog

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ABSTRACT

A four year old healthy male Labrador retriever was presented at the Emergency Critical Care Unit at Madras Veterinary College, Chennai with a haemostatic disorder due to anticoagulant rodenticide toxicity. Buccal Mucosal Bleeding Time (BMBT), Prothrombin time (PT) Activated Partial Thromboplastin Time (APTT) were severely increased. The recent use of an anticoagulant rodenticide containing brodifacoum by the neighbours was investigated. The dog exhibited signs of moaning, muscle twitching, mild seizures, severe distension of abdomen, epistaxis, hemoperitonium, hemoptyis, hematemesis and melena. The animal did not respond to Vitamin K doses and succumbed to severe bleeding due to the brodifacoum trigger causing severe secondary haemostatic disorder and death in spite of all emergency efforts to control bleeding.

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CASE REPORT

A four year old healthy male Labrador retriever (Fig. 1) weighing 52 kg was presented at the Emergency Critical Care Unit at Madras Veterinary College, Chennai with a history of recent penile bleeding, hematemesis and melena. The dog was treated locally the day before but was found unresponsive with evidence of profuse penile bleeds, lethargy and sternal recumbency.

The dog was presented in lateral recumbency, T-103°5 F, normal mentation, increased pulse rate, tachypnea and tachycardia. Clinical examination revealed frank penile blood (Fig. 2), distended abdomen on palpation, severe bleeding from venipuncture sites. Arterial Blood Gas analysis showed metabolic acidosis and thrombocytopenia of 40,000 platelets/cmm.

RESULTS

Buccal Mucosal Bleeding Time (BMBT) was more than 8 minutes and coagulation profile using the coagulation analyser (Agappe mispaclog, Fig. 3) evidenced severely increased Prothrombin time (PT) >200 and Activated Partial Thromboplastin Time (APTT) >300s.

The initial blood profile revealed marked leucocytosis, mild anemia, severe thrombocytopenic changes (less tha 40,000 cels/cmm), increased PT and APTT and bleeding at venipuncture sites.

FAST ultrasound confirmed free fluid in the abdomen suggestive of haemoperitoneum.(Fig. 4)

Upon suspicion, the owner was requested to check the garden and kennel premises for any other informations. Six dead rats were found and elaborate history confirmed the use of anticoagulant rodenticides containing brodifacoum by the neighbors recently.

Rodenticide poisonings are common episodes in human and veterinary practice owing to the Over the Counter (OTC) sale of these hazardous chemicals. This case report will elaborate on rodenticide toxicity its consequential bleeding and death of the dog unresponsive to emergency treatments.

Therapy was initiated with 20 mls of menadione sodium (vitamin K) but was found not very responsive even after two hours. The dog revealed signs of moaning, muscle twitching, mild seizures, severe distension of abdomen, epistaxis, hemoperitonium, hemoptyis, hematemesis, melena and subsequent death.

Post-mortem revealed severe coagulopathy, mottled hemorrhagic liver, enlarged hemorrhagic prostrate, hemoperitonium, severe ecchymotic patches over the small and large intestine, hemorrhage in kidneys and urinary bladder was filled with blood confirming an acquired secondary haemostatic disorder (Fig. 5).

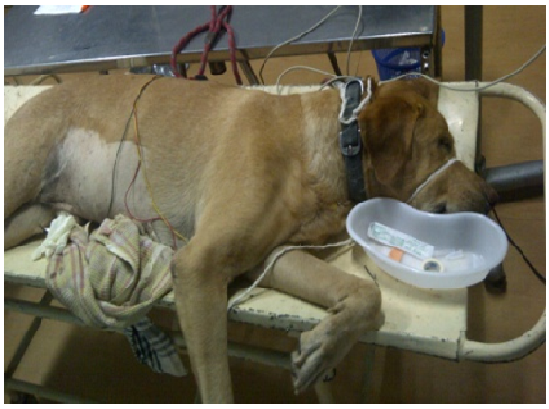

Fig. 1: The recumbent Labrador dog

Fig. 2: A, B- prolonged BMBT, C- penile bleeding

Fig. 3: Coagulation analyser

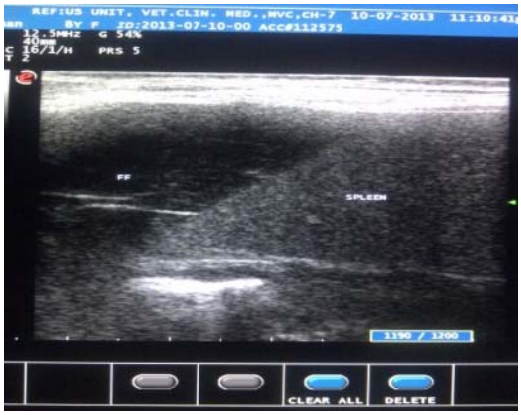

Fig. 4: FAST (Focussed Abdominal Sonograhy Technique) showed free fluid in the abdomen with cellular echogenicity and progressive distension of the abdomen revealed hemoperitoneum.

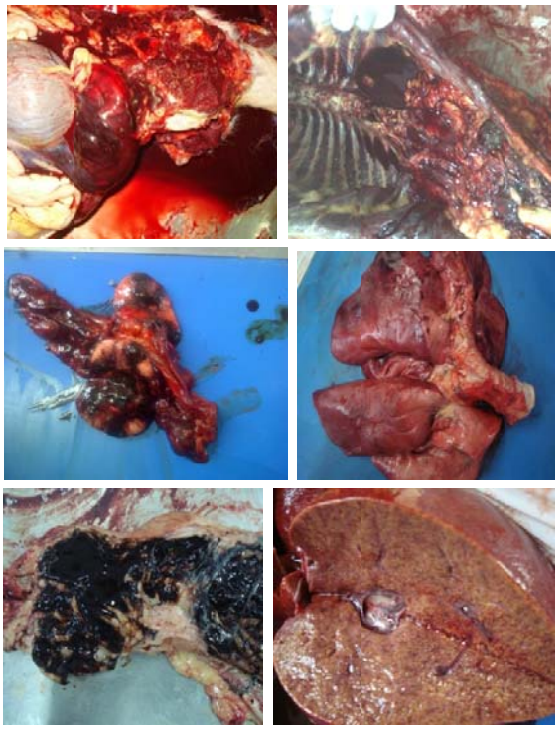

Fig. 5: A- Haemopertioneum with unclotted blood, 5 hrs post death, B- Haemothorax, C- Hemorrhagic prostatitis, D- Haemorrhagic liver, E. Haemorrhagic gastric walls, F- Petichae in the kidneys.

DISCUSSION

Anticoagulant rodenticides are among the most common toxicants ingested by dogs and are responsible for significant morbidity and mortality in dogs. They result in life-threatening coagulopathy by antagonizing vitamin K epoxide reductase in the liver. This enzyme is required to reduce vitamin K epoxide back to active vitamin K. Without appropriate recycling of vitamin K, factors II, VII, IX, and X and the anticoagulant proteins C and S are unable to be carboxylated, a necessary step for them to become active factors (Mount *et al*., 1990).

Brodifacoum is a 4-hydroxycoumarin anticoagulant, used commonly as rodenticides were confirmed in this case and their action is similar to its historical predecessors dicoumarol and warfarin. However, due to very high potency and long duration of action (elimination half-life of 20-130 days), it is characterised as a "second generation" or "superwarfarin" anticoagulant (Murphy, 2002).

Anticoagulant rodenticides inhibit the enzyme *vitamin K epoxide reductase*, needed for the reconstitution of the vitamin K in its cycle from vitamin K-epoxide, and so brodifacoum steadily decreases the level of active vitamin K in the blood. Vitamin K is required for the synthesis of important substances including prothrombin, which is involved in blood clotting. This disruption becomes increasingly severe until the blood effectively loses any ability to clot. (Murphy, 2009).

In addition, brodifacoum (as with other anticoagulants in toxic doses) increases permeability of blood capillaries; the blood plasma and blood itself begins to leak from the smallest blood vessels. A poisoned animal will suffer progressively worsening internal bleeding, leading to shock, loss of consciousness, and eventually death. The anticoagulant is highly lethal to mammals and birds and extremely lethal to fish. It is a highly cumulative poison, due to its high lipophilicity and extremely slow elimination. (Gfeller and Messonnier, 1998).

Brown and Waddell (2009) reviewed the clinical signs which include anorexia, nausea, vomiting (often containing blood), abdominal pain, colic, diarrhea, prostration, lethargy, ataxia), chest tightness, dyspnea (frequent rapid breathing), salivation, excitement, convulsions, paralysis, rigor and coma. In fatal cases there is liver, kidney, heart and brain damage and death is usually due to anoxia (decreased amount of oxygen in organs and tissues).

Mackintosh *et al.*, 1988 recommended vitamin K therapy, 2 mg/kg by tablet or injection, daily for 3 weeks in cases of known or suspected brodifacoum poisoning in dogs after a trial of 20 dogs with brodifacoum poisoning. If a dog is known or thought to have eaten baits or dead animals containing brodifacoum, then vitamin K therapy, 2 mg/kg either by IM injection or oral tablets, should be given daily for 3 weeks. If a dog develops clinical signs of anticoagulant poisoning, it must now be assumed to be due to brodifacoum and treated accordingly. If the dog shows signs of acute massive hemorrhage, then it is advisable to give a transfusion of whole blood or plasma which should immediately supply hemostatic concentrations of clotting factors. The amount transferred should approximate 5-10% of the patient's total blood volume, which is approximately 90 ml/kg (Mount *et al*., 1982).

In cases of hemothorax the blood should be aspirated and autotransfusion (where aspirated blood plus vitamin K is transfused back into the dog) may be performed (Crispin, 1977).

Conclusion

With anticoagulant rodenticide toxicity, hemorrhage can occur in a variety of locations and most typically as body cavity effusions or pulmonary parenchymal bleeding. Dogs can show a myriad of different clinical signs, including both nonspecific (anorexia, lethargy, weakness) and specific manifestations (cough, dyspnea, hemoptysis, lameness, hematuria, bruising, exophthalmus, pharyngeal swelling, CNS signs, epistaxis, melena), signs of shock if the blood loss has resulted in a significant decrease in circulating volume. Other differential considerations for acute coagulopathy including DIC, severe thrombocytopenia, congenital coagulopathy and hepatic failure should be excluded based on history and other diagnostic tests.

As it takes 12-36 hours for regeneration of depleted coagulation factors after initiation of vitamin K therapy, transfusion support is required in patients that are clinically affected. The deficient coagulation factors can be found in frozen plasma, fresh frozen plasma, or fresh whole blood.

In acute cases, vitamin K, (1 mg/kg) diluted with saline may be given by slow intravenous injection, and prior administration of an antihistamine is recommended to prevent histaminoid reactions (Clark & Halliwell, 1963 and Crispin, 1977). This should restore a proportion of clotting factors within 2 hours. Tablets or intramuscular injections of vitamin K may be used initially in less acute cases. Vitamin K therapy by IM injection or oral tablets (2 mg/kg) should continue for 3 weeks.

In this case the Labrador dog succumbed to severe bleeding due to the brodifacoum trigger causing severe secondary haemostatic disorder and death in spite of all emergency efforts to control bleeding.

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