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Intoxication in dog

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Abstract: *This case report describes a coumarin anticoagulant intoxication in a Dachshund presented with lethargy, tachypnea, vomiting, and ascites. The diagnosis was established based on medical history, clinical signs, imaging findings, and laboratory confirmation of markedly prolonged prothrombin time (PT). Following infusion therapy and administration of vitamin K, the patient showed significant clinical improvement. Coumarin rodenticide poisoning remains a serious concern in veterinary practice, as ingestion can rapidly lead to life-threatening hemorrhagic complications. Prompt diagnosis and immediate therapeutic intervention are therefore essential. Given the limited availability of effective alternatives for rodent control, minimizing animal access to rodenticide baits remains a key preventive measure to reduce the risk of accidental poisoning.*

Keywords:*dog, rodenticides, poisoning, diagnosis, therapy*

I. INTRODUCTION

Environmental exposure to toxic substances such as herbicides, insecticides (organophosphates, carbamates, strychnine, metaldehyde), or anticoagulant rodenticides (coumarins) poses a serious risk to companion animals [1].

Anticoagulant rodenticides are one of the most common causes of acute toxicosis in dogs and, less frequently, in cats [2]. Intoxication occurs through direct consumption of the bait, toxins dispersed in the environment, or secondarily after consuming a poisoned rodent [3]. The pharmacokinetics of coumarins vary depending on the generation of rodenticides. The first generation (e.g., warfarin, pindone) requires repeated exposure, while second-generation substances (e.g., brodifacoum, bromadiolone, difenacoum) have a long biological half-life and cause prolonged bleeding even after a single ingestion [2]. These lipophilic substances

accumulate in the liver, prolonging their effect [4]. The toxic dose depends on the amount of substance ingested, the species, age, and overall condition of the animal. Dogs are significantly more sensitive than rodents or cats, so even a minimal amount of rodenticide can be fatal [5]. Chemically, these substances belong to the coumarins and their 4-hydroxycoumarin derivatives, which act as vitamin K antagonists by inhibiting epoxide reductase [6]. This disrupts the conversion of vitamin K to its active form and reduces the γ-carboxylation of coagulation factors II, VII, IX, and X, as well as proteins C and S [3]. The result is a secondary hemostasis disorder with a risk of spontaneous bleeding [7].

Clinical signs appear late, usually 2–5 days after ingestion of a toxic dose, as functional coagulation factor reserves are gradually depleted [8]. In dogs, the most common symptoms are lethargy, pale mucous membranes, weakness, loss of appetite, dyspnea, coughing, hemothorax, hemoperitoneum, and bleeding from the mucous membranes [3]. Some patients may develop subcutaneous hematomas or petechiae [6].

The diagnosis is based on medical history, clinical examination, and coagulation tests - prolonged prothrombin time (PT) and activated partial thromboplastin time (aPTT) [6]. If laboratory methods such as liquid (HPLC) or gas chromatography (GC-MS) are available, they can be used to determine the specific substance and its concentration in the liver, blood, or adipose tissue [9]. Modern diagnostic approaches also include RT-qPCR, which can detect rodenticide metabolites at the molecular level [3].

Treatment consists of administering vitamin K (phytonadione) orally for several weeks; in more severe cases, it is combined with a blood transfusion [8]. In addition to therapeutic aspects, it is important to emphasize the preventive importance of proper handling of rodenticides, owner education, and the use of secure bait stations [3].

II. Diagnostics and therapy

History

The patient was a 4-year-old female dachshund (Fig. 1); regularly vaccinated and treated against internal and external parasites. The owners did not observe the presence of ticks on the dog. They also reported feeding the dog bones two days prior. They did not rule out the possibility of the dog ingesting rat bait placed outside by neighbors. They came for examination due to apathy, loss of appetite, and repeated vomiting lasting one day. A private veterinarian immediately administered an antiemetic (Vominil 10 mg/ml inj.; VetViva Richter GmbH, Austria), an anti-inflammatory (Novasul 500 mg/ml inj.; Richter Pharma AG, Austria), ruled out the presence of a foreign object using X-ray, and sent the patient to the emergency at the Veterinary Hospital.

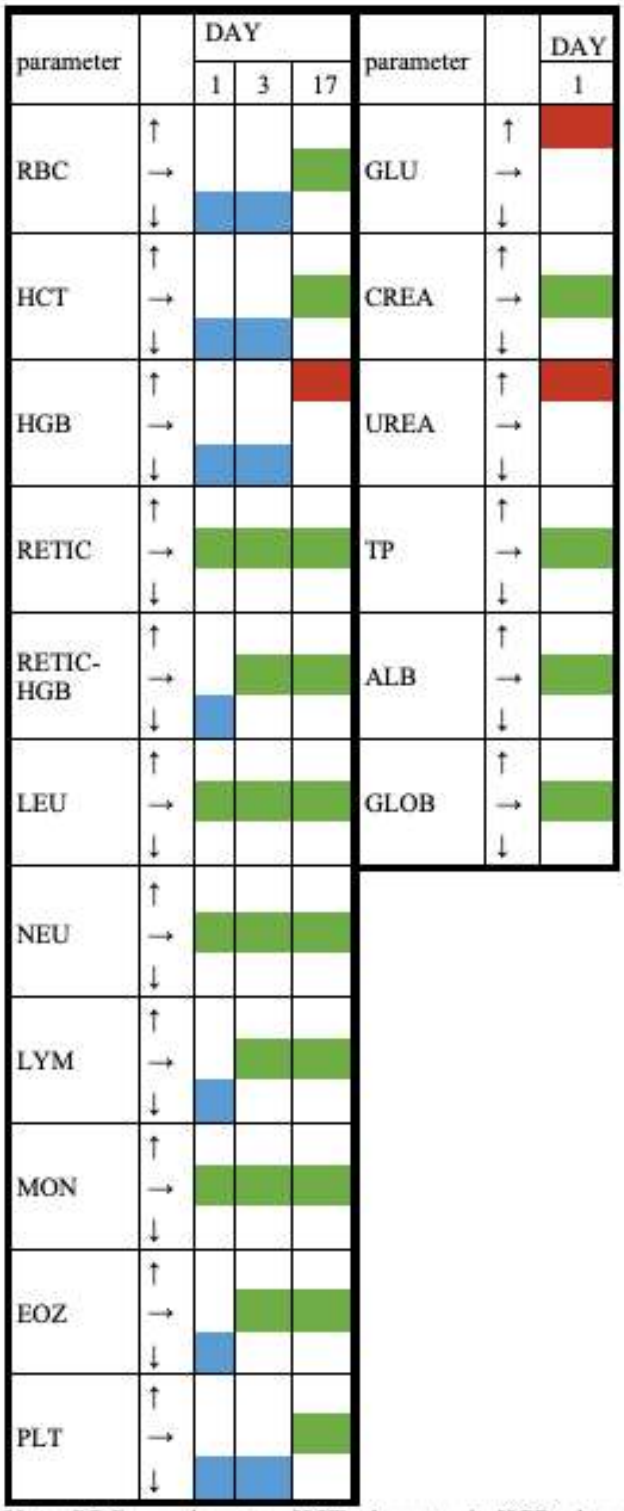
Clinical examination

The dog's condition upon admission was good (BCS 3/5). The examination revealed pale pink conjunctiva and mucous membranes, capillary refill time (CRT) of 4 s, tachypnea, vomiting of foam without blood, abdominal pain in the cranial region; other systems without pathological changes.

Laboratory and imaging examinations

Hematological examination on admission showed mainly moderate non-regenerative anemia and thrombocytopenia (Fig. 2). Coagulation tests evaluated the Quick test (prothrombin time), which was significantly elevated (Table 1). Biochemistry showed mild elevations in urea, phosphorus, glucose, and a decrease in K. USG examination revealed hypoechoic fluid throughout the abdominal cavity (Fig. 3).

Figure 1. Patient during hospitalization



Note: RBC – erythrocytes, HCT – hematocrit, HGB – hemoglobin, RETIC – reticulocytes, RETIC – HGB – reticulocyte hemoglobin ratio, LEU – leukocytes, NEU – neutrophils, LYM – lymphocytes, MON – monocytes, EOZ – eosinophils, PLT – thrombocytes, GLU – glucose, CREA – creatinine, UREA – urea, TP – total protein, ALB – albumin, GLOB – globulin

Figure 2. Comparison of hematological and biochemical parameters before and during treatment

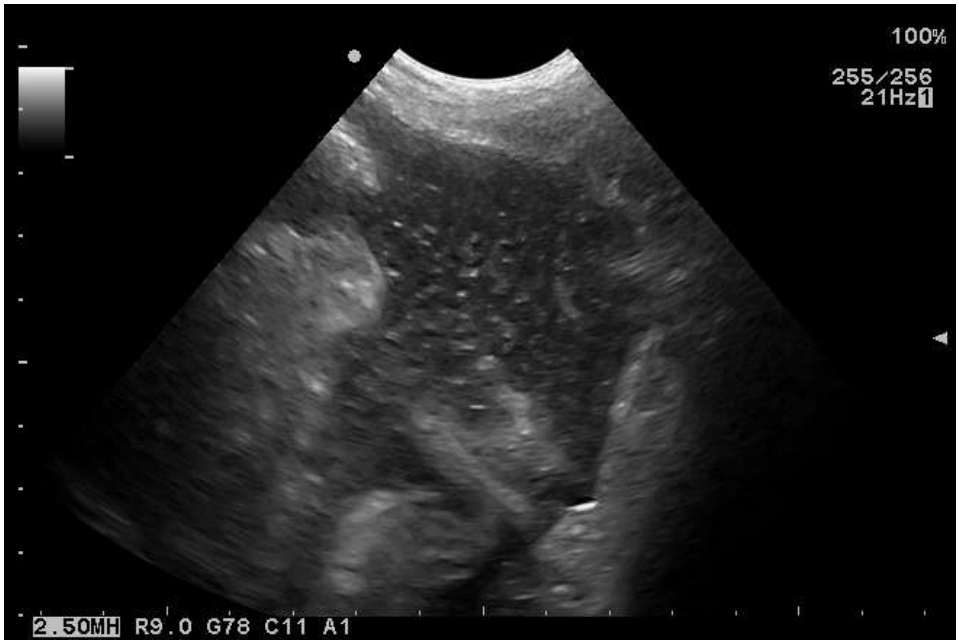


Figure 3. USG

Table 1. Coagulation testing

parameter	Reference range	1st day	2nd day	17th day	28th day
PT	6 – 10 s	>100 s	10 s	14 s	7 s

Diagnosis

The patient was diagnosed with coumarin poisoning based on his medical history, clinical examination, laboratory tests, and imaging tests.

Differential diagnosis

The clinical manifestations of intoxication are often nonspecific and may mimic other diseases, such as trauma, spleen rupture, disseminated intravascular coagulopathy (DIC), neoplasms, hepatopathies, congenital hematological disorders, etc. [6].

Therapy and progression of the disease

After examination, slow infusion therapy was immediately initiated in the patient: 80 ml of physiological solution (0.9% sodium chloride infusion; B. Braun, Germany) with vitamin K (Kanavit 10 mg injection; Saneca Pharmaceuticals, a.s., Slovakia) at a dose of 10 mg/kg without anaphylactic reaction, followed by infusion with Ringer's lactate (Ringer Lactate inf.; B Braun, Germany) and plasma substitute (Gelofusine sol. inf.; B. Braun, Germany). On the second day of hospitalization, the patient was no longer vomiting, but was not eating. A repeat PT test was within normal limits (Table 1). Vitamin K administration continued in oral form at a dose of 5 mg/kg. On the third day of treatment, the dog began to eat again; An ultrasound examination showed resorption of a large amount of fluid from the abdomen; anemia persisted. The dog was discharged to home care. Subsequent maintenance therapy included daily administration of vitamin K in the form of drops (Kanavit 20 mg/ml gtt.; Saneca Pharmaceuticals, a.s., SR). A check-up after 14 days showed a slight elevation in the PT test, and it was recommended to prolong the therapy for another 10 days, after which no abnormalities were found in the clinical findings or laboratory values.

III. Discussion

If coumarin poisoning is suspected, it is important to take a thorough medical history. The veterinarian should determine whether the dog had access to rodenticide baits, garbage, or could have consumed a dead rodent. Secondary poisoning is not uncommon, especially in hunting or farm dogs [3].

A key diagnostic parameter in rodenticide poisoning is prolonged PT, which is often the first detectable indicator of vitamin K deficiency [3]. In our patient, the initial PT values were significantly high (more than 100) (Table 1). Factor VII has the shortest biological half-life (6–8 hours), and its deficiency primarily causes prolonged PT [4]. With progressive intoxication, aPTT also prolongs, indicating a more severe hemostasis disorder [6].

The spectrum of clinical manifestations is broad and depends on the amount of substance ingested, the type of rodenticide, and the time since exposure. Hemorrhagic manifestations can be observed in virtually every organ – most commonly hemothorax, hemoperitoneum, hematuria, melena, or subcutaneous hematomas [2]. Although we did not observe any external signs of bleeding in our case, an ultrasound examination revealed ascites in the abdominal cavity (Fig. 3). Alveolar bleeding can cause dyspnoea and cyanosis [6]. The dog showed rapid breathing and increased CRT, which also correlates with a hemorrhagic condition.

Treatment should be started immediately after exposure to rodenticide is detected or suspected, even without laboratory confirmation of the toxin [8]. The first-line antidote is vitamin K, administered orally at a dose of 2–5 mg/kg daily for at least three weeks; higher doses are also possible in severe cases [10]. A positive response to treatment is usually seen within 12–24 hours and is detectable by an improvement in PT results [8], as in our case. For more severe forms or intoxications with second-generation coumarins (e.g., brodifacoum, bromadiolone), treatment for 4–6 weeks, sometimes up to two months, is recommended [3]. In our patient, treatment was discontinued after four weeks. In acute conditions with severe bleeding, it is necessary to administer a transfusion of fresh frozen plasma or whole blood to replenish the missing coagulation factors [6]. Vitamin K can be administered subcutaneously; however, intravenous (i.v.) administration is more effective, but carries a higher risk of anaphylactic reaction [11]. The oral form has the advantage of stable absorption and a lower risk of complications. It is recommended to administer vitamin K with food rich in fats, which improves its absorption [6]. Due to the acute condition of the patient, high-dose intravenous administration was chosen, subsequently limited to safer oral administration. Supportive therapy includes oxygen therapy, hematocrit control [3], antioxidants, and, if necessary, hepatoprotectives [4].

The prognosis is generally good if treatment is started early [3]. The most important prognostic factor is the time between exposure and the start of therapy. Dogs that receive vitamin K within 48 hours of ingesting the toxin have an almost 100% chance of survival [6]. The risk of second-generation rodenticide poisoning lies in the possibility of recurrent bleeding, so follow-up examinations and consistent maintenance therapy are essential [3].

In any case of unexplained bleeding, veterinarians should consider anticoagulant poisoning [2]. It is important to emphasize to owners that symptoms appear with a delay, so even a single ingestion of bait requires immediate consultation [10].

The use of safe rodenticide stations that minimize access by domestic animals is recommended. Prevention also includes proper education of breeders on the handling of toxic substances, thorough labeling of baits, and safe storage of rodenticides [2].

IV. Conclusion

Poisoning with coumarin anticoagulants in dogs poses a serious health risk, as it can be fatal. Despite the severity of the clinical picture, the prognosis is favorable if the diagnosis is made early and treatment is started immediately. Important predictors of success include rapid identification of exposure, adequate vitamin K dosing, and monitoring of coagulation parameters throughout treatment. From an environmental perspective, safe use of rodenticides and prevention of exposure are important.

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