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Haemorrhage in seven cats with suspected anticoagulant rodenticide intoxication

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Clinical features were evaluated in seven adult cats (six males, one female) with haemorrhage and presumptive anticoagulant rodenticide intoxication. Haemorrhage appeared as thoracic haemorrhage, otic bleeding, haematoma, melena, haematochezia, and petechiation. The most common other presenting signs were lethargy, anorexia, and tachypnoea or dyspnoea. Six cats were anaemic, four cats were mildly thrombocytopenic (58 000–161 000/µl), and three had slightly decreased plasma protein or albumin values. The prothrombin time (30.3–>100 s, reference range: 16.5–27.5 s) and activated partial thromboplastin time values (32.6–>100 s; reference range: 14–25 s) were markedly prolonged in all cats. All cats received vitamin K_1 subcutaneously or orally (3.7–5 mg/kg body weight initially) and depending on severity of signs five cats were transfused with fresh whole blood. Plasma coagulation times improved in all cats and returned to normal in 1–5 days. Rodenticide poisons represent an important but relatively rare cause of haemorrhage in cats and can be effectively treated.

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Introduction

Intoxications continue to be an important cause of morbidity and mortality for companion animals. According to a survey (1993/1994) of the American Association of Poison Control Centres, 13.6% of all poison exposures occurred in cats and 82.8% in dogs (3.9% in other species). From 17 023 cats exposed to toxic chemicals, most cats had ingested plants (25.2%), followed by insecticides (21.1%), cleaning products (12.6%), foreign bodies (5.5%), hydrocarbons (5%), and rodenticides (4.3%) (Hornfeldt & Murphy 1998). In a poison survey from Brazil (Xavier et al 2002) 29.9% of the cats showed toxicity following ingestion of therapeutic agents (50% non-steroidal antiinflammatory drugs, 42.8% other drugs, and 7.2% antibiotics), 27.6% due to pesticides for farm use (46.1% carbamate, 38.5% organophosphate insecticides, 15.4% others), 14.9% because of domestic pesticides, followed by 10.6% due to rodenticides, and 4.2% because of industrial products (12.8% unknown agents). In a study from Australia the following feline intoxications were diagnosed during a 12-year period: insecticides (39%, mainly organophosphates), snake and insect bites (33%), molluscicides (28%) and anticoagulant rodenticides (22%) (Robertson et al 1992). The most commonly reported rodenticide toxicoses in the United States are those caused by anticoagulant rodenticides, followed by bromethalin, cholecalciferol, strychnine, and zinc phosphide (Hornfeldt & Murphy 1999). In a 3-year German poison survey coumarin intoxication was diagnosed in 26 dogs, but only in one cat; in addition four cats were poisoned with organic hydrocarbons and three cats with thallium (Grünbaum 1990).

Anticoagulant chemicals which are among the most frequently used rodenticides (Mount 1988) continue to be a major cause for morbidity and mortality but should be treatable if detected early, before serious haemorrhage occurred, and the appropriate antidote is used. The coagulation factors II, VII, IX, and X as well as antithrombotic factors (proteins C and S) require the hepatic vitamin K-dependent carboxylation at the glutamic acid of the amino terminus (Murphy & Gerken 1989), as only the carboxylated and activated coagulation factors are capable of binding Ca^{2+} ions. The binding of Ca^{2+} induces a conformational

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change that allows the attachment of coagulation factors to phospholipids on cell membranes. During the carboxylation of these coagulation factors vitamin K is oxidised to the inactive vitamin K epoxide. Normally, vitamin K epoxides are reduced by the vitamin K-dependent epoxide reductase to become reactivated to functional vitamin K. These warfarin-type compounds prevent vitamin K recycling through inhibition of the hepatic vitamin K epoxide reductase (Ren et al 1977). Because of their short half-lives vitamin K deficiency causes a depletion of active factors VII, IX, X, and II (their half-lives are 6, 14, 17, and 41 h in humans, respectively (Hellemans et al 1963), but are not known in small animals); impaired haemostasis can be detected 1 day after exposure to anticoagulant rodenticides in humans and dogs (Woody et al 1992) and clinical signs of haemorrhage can be noticed 2–5 days after exposure (Murphy 2002). These effects may last for weeks depending on the type of anticoagulant rodenticide.

Several clinical case studies on haemorrhage induced by rodenticide intoxication have been reported in dogs (Kammermann-Lüscher 1978, Feldman et al 1981, Mischke 1997, Lewis et al 1997, Robben et al 1998, Sheafor & Couto 1999, Furlanello et al 2000, Reitemeyer et al 2001), but minimal clinical information regarding rodenticide associated haemorrhage in cats exists (Kammermann-Lüscher 1978). In this retrospective case series we describe clinical signs, haematological and radiological changes, and the response to therapy in cats with haemorrhage due to anticoagulant rodenticide intoxication seen at the Free University of Berlin.

Animals and methods

Records of cats with haemorrhage and suspected rodenticide intoxication admitted to the Clinic for Small Animals at the Free University of Berlin during a 6-year period (April 1996 to June 2002) were analysed. Cats were included when their medical records were adequate to assess and if they had signs of haemorrhage together with abnormalities of coagulation parameters, which normalised following administration of vitamin K_1.

The signalment of the patients, the clinical signs, alterations of coagulation parameters as well as haematological and biochemical values, radiographic findings, route of administration and dosage of vitamin K_1 (Konakion®, Roche, Grenzach, Germany), the need for whole blood transfusions, and treatment outcome were monitored.

If a blood transfusion was needed the blood groups of the patients were determined prior to transfusion. The expected haematocrit (Hct) rise caused by a transfusion was calculated by the formula: expected Hct increase (%)=transfusion volume (ml):bodyweight (kg)×2 (Griot-Wenk & Giger 1995) and compared to the observed Hct rise.

EDTA-anticoagulated blood was used to assess the complete blood cell count by impedance cell counting (Celldyn 3500, Abbott, Wiesbaden, Germany), and the platelets were counted manually using a commercially available test kit (Thrombo Plus®, Sarstedt, Nümbrecht, Germany). Biochemical parameters were determined in heparin-anticoagulated plasma (Electrolyte 14+ Analyser, Nova Biomedical, Rödermark, and Cobas Mira plus, Roche, Grenzach, Germany). For the measurement of the prothrombin time (PT) and activated partial thromboplastin time (aPTT) assay 0.9 ml of whole blood was collected into a plastic tube with 0.1 ml 3.13% sodium citrate, and after centrifugation, the plasma was quickly separated. The PT was determined by the Hepato Quick® Test (Boehringer, Mannheim, Germany), the aPTT was measured with the Pathromtin® test kit (Behring, Marburg, Germany) by a fibrometer (Schnitger and Gross, Amelung, Lemgo, Germany; Mischke & Nolte 1999). Plasma fibrinogen levels were determined with the method of Clauss (1957) using as a coagulation activator (Multifibren®, Behring, Marburg, Germany). The results of the PT, aPTT, and fibrinogen measurements were compared to reference values of the clinical laboratory of the Free University of Berlin.

Results

Clinical findings

During a 6-year period, anticoagulant rodenticide intoxication was suspected in seven European shorthair cats presented with bleeding. All of them lived mostly outdoors. Six cats were males, four of them castrated, and one was a spayed female cat. Their age ranged from 1 to 10 years (mean age±SD, 2.8±3.2). None of the cats had been seen to ingest poison by the owners but one owner reported having seen his cat eating a mouse 3 days before admission to the clinic.

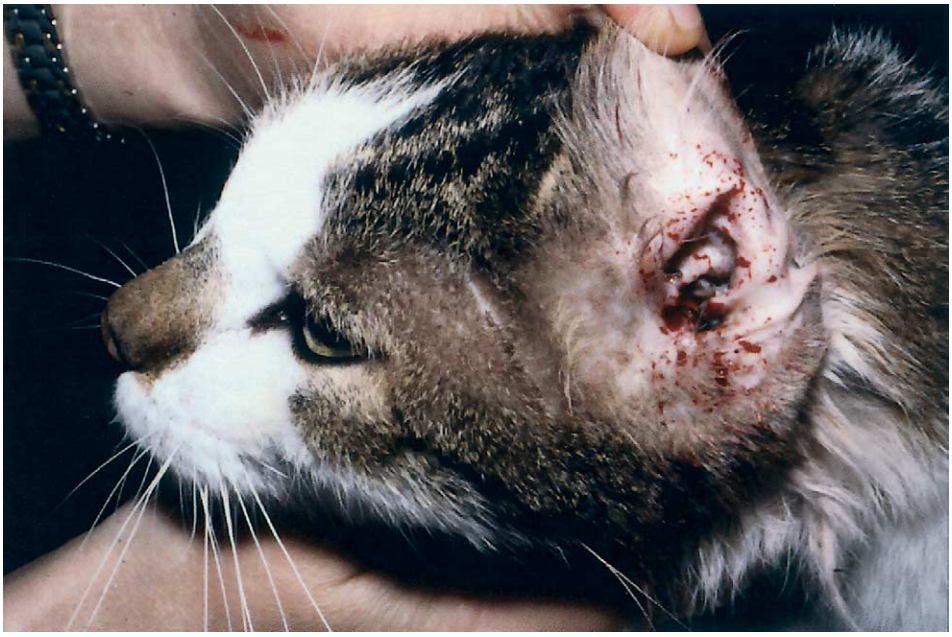

Fig 1. Severe otic haemorrhage in a cat with anticoagulant rodenticide intoxication.

No possible source of anticoagulant could be identified in any of the households.

Cats were presented because of lethargy and inappetance/anorexia. In addition six cats had obvious signs of bleeding or haematoma, four experienced dyspnea or tachypnea for 1 day, three cats had collapsed, and one cat had vomited once. Two cats had severe unilateral otic haemorrhage (Fig 1). Other signs of haemorrhage in different cats were melaena, petechiae of the tongue, a large haematoma in the sternal region (Fig 2), haematochezia, and haematoma on the back developing after a bite wound. In three cats thoracic haemorrhage was suspected based upon thoracic radiographs. Other physical examination findings were pale mucous membranes (6), hypothermia (3, 36.5–37.3°C), pyrexia (1, 40.2°C) due to an infected bite wound, a systolic heart murmur (grade II to III/VI; $n=2$), and tachycardia (2). Abnormalities of chest radiographs were recognised in three cats. One cat had a pleural effusion, and two cats had a precardial opacity with widening of the mediastinum, and a mediastinal bleeding was suspected (Fig 3a). None of the abdominal radiographs available from six cats revealed any abnormalities.

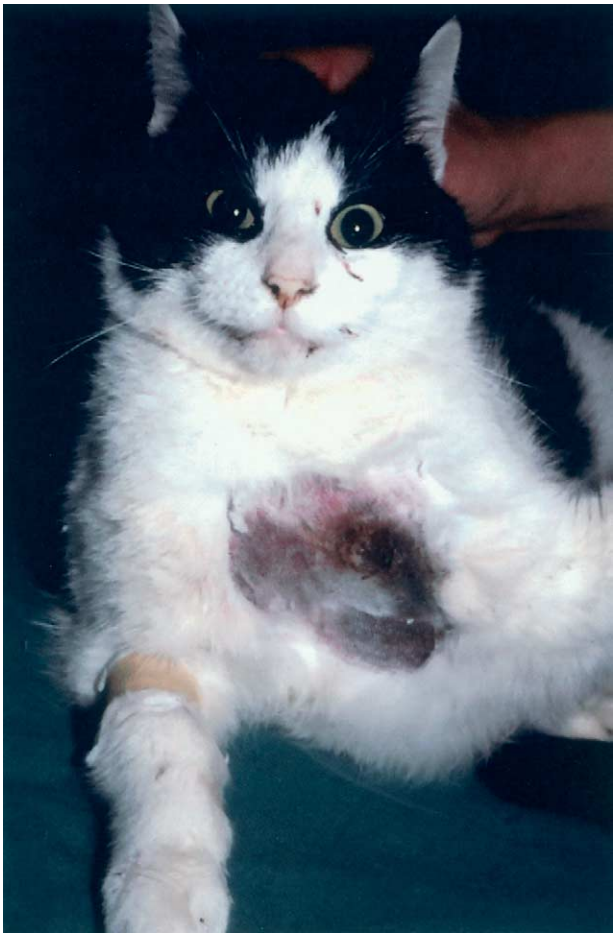

Fig 2. Large haematoma in the sternal region in a cat with anticoagulant rodenticide intoxication.

Laboratory findings

At the time of admission, four of the seven cats were severely anaemic with Hct values of 8–12% (mean 10±1.6). Two had moderate anaemia

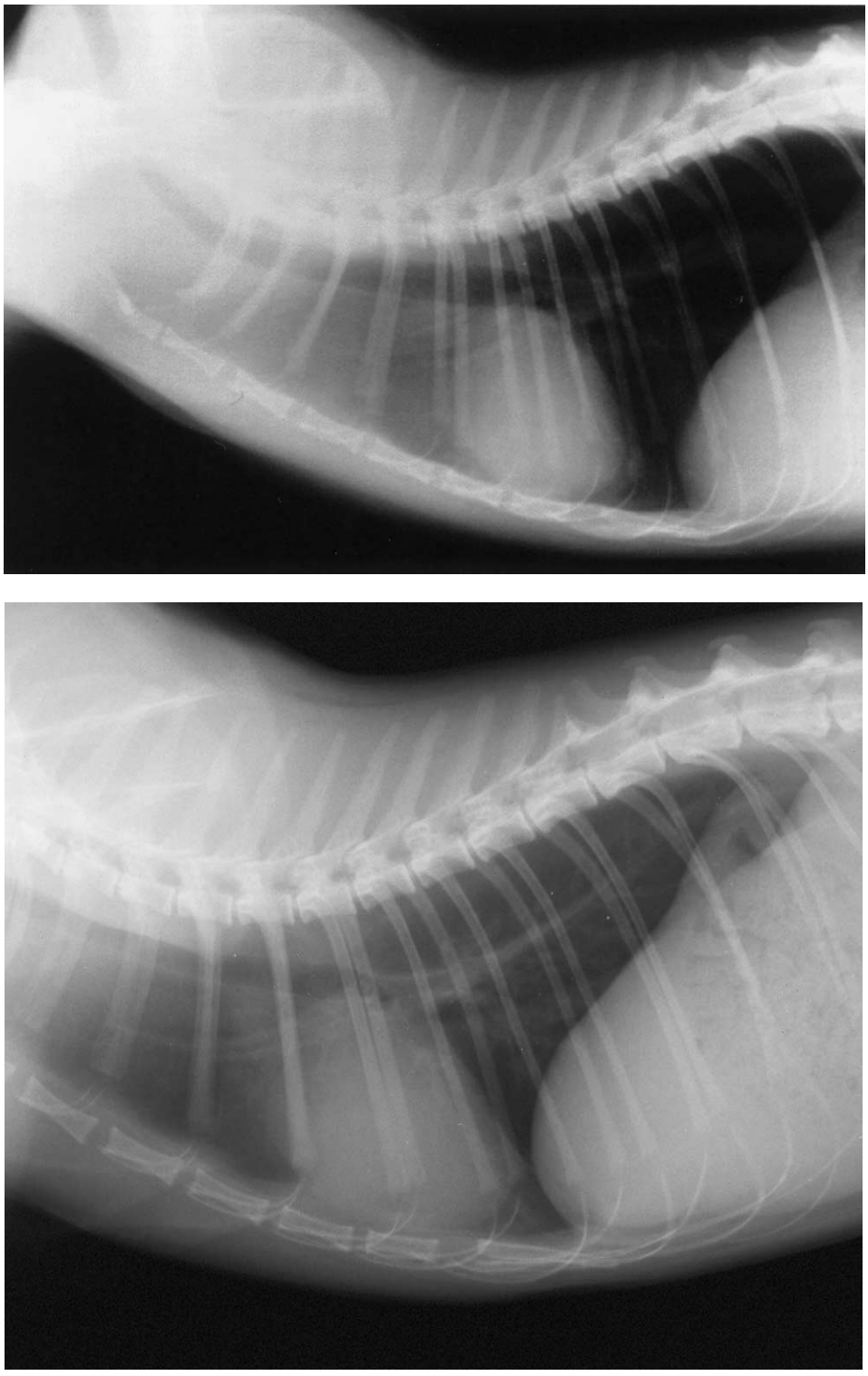

Fig 3. Increased precardial radiodensity suggesting mediastinal bleeding in a cat with anticoagulant rodenticide intoxication before (a) and 5 days after (b) initiating therapy with vitamin K_1 and fresh whole blood.

(Hct 19 and 23%), and one cat had a low normal Hct of 33% (reference range 30–44%). The MCV ranged from 38 to 51 fl (mean 43.1±4.5, reference range 40–55 fl) and the MCHC ranged from 31–38 g/dl (mean 35.6±2.6, reference range 31–35 g/dl).

Aggregate and punctate reticulocyte counts measured on the day of admission or the day after were increased in three of the four cats assessed: 6.8% aggregate (absolute 102 680/µl) and 7.2% punctate reticulocytes (cat 2), 3.4% aggregate (absolute 131 240/µl) and 5.0% punctate (cat 3), 1.6% aggregate (absolute 43 520/µl) and 3.6% punctate (cat 4), and 0.6% aggregate (absolute 29 760/µl) and 0.4% punctate in cat 7 (reference range aggregated reticulocytes <0.4%/40 000/µl and punctate reticulocytes 1.4–10.8%) (Tvedten 1994). Based upon a manual platelet count, a mild or moderate thrombocytopenia was initially present (58 000–161 000/µl) in four of five tested cats (reference values 180 000–500 000/µl), the two other cats had adequate platelet counts on a microscopic blood smear evaluation. In one cat with a normal platelet count on presentation (203 000/µl), the platelet count fell to 90 000/µl within 2 days. In all cases plasma PT and aPTT were initially prolonged. Values of the PT ranged between 30.3 and >100 s (reference values 16.5–27.5 s) and the aPTT ranged from 32.6 and >100 s (reference values 14–25 s) at the time of admission.

On presentation plasma protein concentrations ranged between 56 and 76 g/l (reference values 57–77 g/l) in six cats. The plasma albumin levels ranged from 22 to 39 g/l (reference values 30–46 g/l) in five cats with two cats having decreased serum albumin values (22 and 25 g/l, respectively).

Therapy and outcome

All cats were given vitamin K_1 subcutaneously (6) or orally (1) at 3.7–5.0 mg/kg bodyweight (mean 4.6) on day 0. A switch from parenteral to oral treatment occurred either on the first (n=4) or on the third day (n=2), respectively, when the cats were eating again. After 1 day the vitamin K_1 dose was continued at a dose of 1.4–3 mg/kg bodyweight twice daily (mean 2.1 mg/kg±0.5) and was only slightly reduced to a dose of 1–2 mg/kg body weight per os twice daily (mean 1.5 mg/kg±0.3) for 31–61 days (mean 42.5 days±11.3). Different treatment regimes were followed for these patients, and, thus, the dose of vitamin K_1 varied.

In addition to the vitamin K_1 therapy, five blood type A cats were transfused with blood type compatible fresh whole blood on day 0 (4.3–15 ml/kg bodyweight, mean 9.2±4.4 ml/kg) because of serious bleeding and anaemia, and two of them received a second transfusion the following day (4.3 and 5 ml/kg) because of continued anaemia and bleeding. In all but one cat, the Hct rose by 3–9% after the transfusions, whereas in one case with continuous bleeding the Hct decreased by 1%. Based upon the volume transfused the difference between the expected Hct increase and the observed Hct change post transfusions in each cat ranged between −3.3 and 6.5% (−3.3, −3.3, −2.5, 1.9, 2.2, 4.9, and 6.5%; mean 0.8±4.4%). All animals received also Ringer lactate infusions (Sterofundin®, Braun, approximately 50 ml/kg/day) for dehydration, shock treatment or a lack of water intake.

One cat with dyspnoea was supplemented with oxygen, and the dyspnoea and tachypnoea resolved within the first day of treatment. Three animals with hypothermia were placed under a red light warming lamp for the first day, and their body temperature normalised within 24 h. A single intravenous dose of prednisolone-21 hydrogensuccinate (Solu Decortin®, Merck, Darmstadt, 10–30 mg/kg, mean 16.3±9.2 mg/kg) was given to four cats with hypovolemic shock on day 0. In addition six of seven cats were also receiving antibiotics for the treatment (infected bite wound in one cat) or prevention of secondary bacterial infections. One animal with melena was given ranitidine additionally (1 mg/kg IV BID for 6 days; Ranitidin®, Ratiopharm, Ulm), and two cats with large haematomas received the analgesic buprenorphin (0.01 mg/kg subcutaneously TID for 3 days; Temgesic®, Essex Pharma, München).

The plasma coagulation times improved in all animals within the first 24 h and returned to the normal range after 1–3 days (Fig 4). Values of PT and aPTT returned to normal after 1 day in two cats, in another two cats after 2 days, and within 4 days in the remaining cats (Fig 5). (Day 0 was the day of presentation.) Five cats, who were monitored at the Small Animal Clinic, also had normal clotting times after 5, 7, 9, and 12 days, and these times remained normal at least 3 days after vitamin K withdrawal on day 38, 42, and 61 in three cats, respectively. An influence of vitamin K dosage on the normalisation of PT and aPTT was not observed. The mild thrombocytopenia resolved within 2 days in three cats and in the other two cats after 5 days.

Radiographically, the precardial opacity appeared markedly reduced on day 5 (Fig 3b). The duration of hospitalisation lasted 4–7 days and all animals survived. However, one cat was presented as an emergency 36 days after the first visit with a haematoma on the left front leg, and the PT

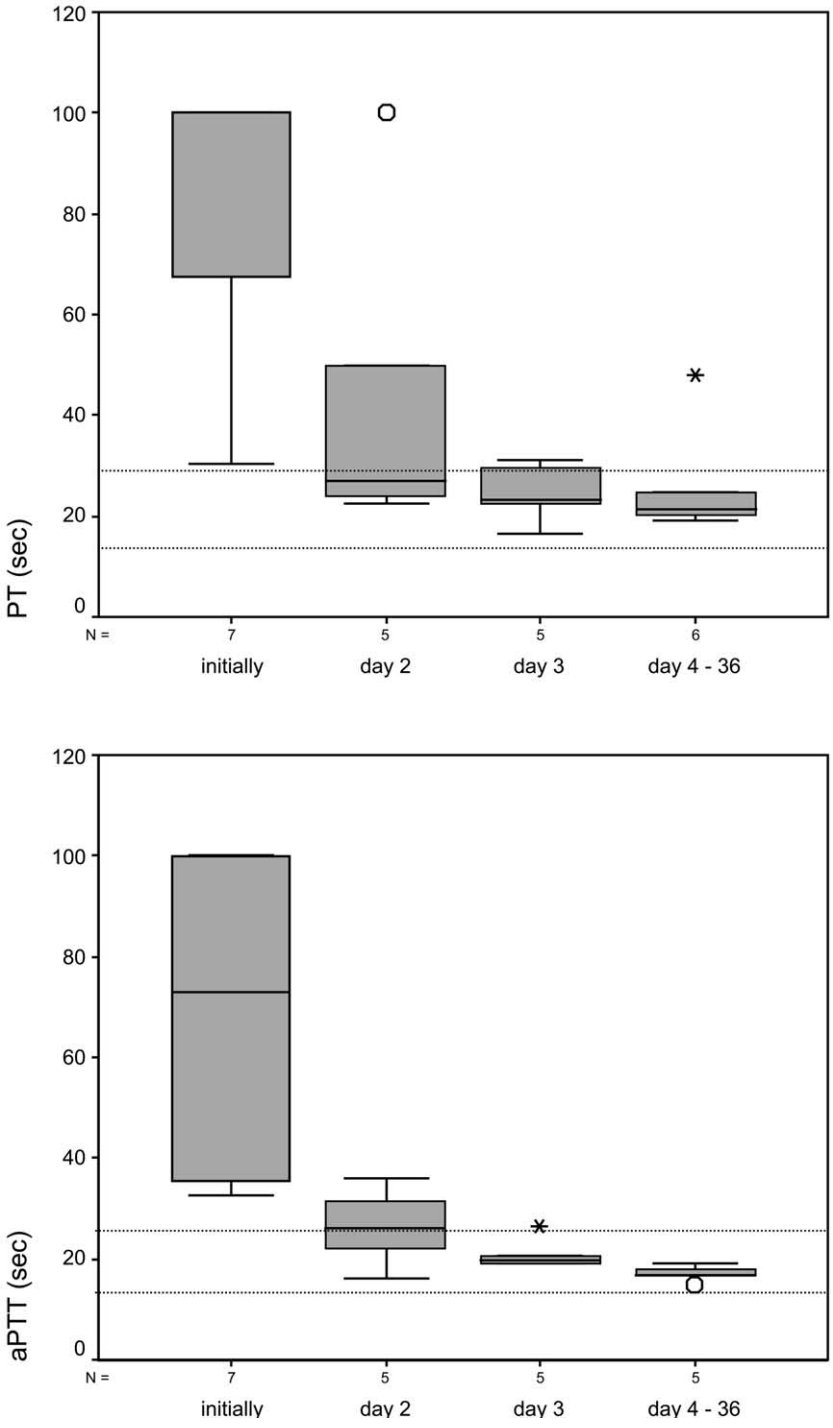

Fig 4. PT and aPTT values before and after initiating treatment with vitamin K_1 and fresh whole blood. ○, PT still >100 s, *, prolonged PT on day 36 in one cat (after discontinuation of vitamin K therapy).

was markedly prolonged (48 s). The owners of the animal had discontinued the vitamin K_1 therapy after 6 days of treatment and the cat was again allowed to go outdoors. After retreating with vitamin K_1 the PT value returned to the reference range within a day.

Discussion

Whereas haemorrhage due to anticoagulant rodenticide intoxication has been well-recognised and characterised in dogs (Kammermann-Lüscher 1978, Feldman et al 1981, Mischke 1997,

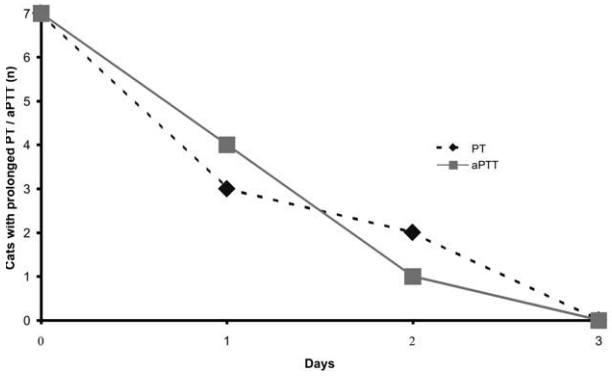

Fig 5. Number of cats with prolonged PT or aPTT caused by anticoagulant rodenticide intoxication during the course of treatment.

Lewis et al 1997, Robben et al 1998, Sheafor & Couto 1999, Furlanello et al 2000, Reitemeyer et al 2001), rodenticide-induced bleeding is less commonly described in cats. Based upon toxicology surveys (Grünbaum 1990, Hornfeldt & Murphy 1998) and clinical reports (Kammermann-Lüscher 1978, Reitemeyer et al 2000) including a case series from Berlin anticoagulant rodenticide poisoning seems to occur five to 20 times less frequently in cats compared to dogs. This may be related to housing conditions, since cats may be kept more often in human living quarters (apartments) away from rat poisons, their selective eating habits (rat/mice bait as well as poisoned dead rodents may not be appealing to cats, and ingested dead mice may not contain high quantities of poison), and a resilient haemostatic system or lack of trauma (safe living quarters/lifestyle). However, all cats of this study were living in part outdoors and at least one was seen recently to have eaten a mouse. Based on this report it appears that male cats are more frequently intoxicated than female cats. The number of cats in this report may affect this statistic as the population is really too small to draw conclusions.

In Germany the most commonly used anticoagulant rodenticides include diphacinone, brodifacoum, and bromadiolone (Beratungsstelle für Vergiftungserscheinungen und Embryonaltoxikologie, Berlin, 2000, personal communication), all belonging to the second generation, long acting group of coumadin derivatives. The ingested rodenticides can be identified by high-pressure liquid chromatography in a blood sample (Robben et al 1998), a method rarely used in clinical practice. Furthermore, low blood rodenticide levels may not be detectable, and only commonly used rodenticides can be traced. Similarly with most canine cases ingestion of rodent bate was not observed and the rodenticide was not identified in most canine case series. Thus, the diagnosis of rodenticide-induced haemorrhage in the cats reported here was presumptive and based on the combination of clinical signs, haemostatic laboratory test abnormalities, and response to therapy.

All cats presented with signs of haemorrhage. As with intoxicated dogs (Lewis et al 1997, Robben et al 1998, Sheafor & Couto 1999, Reitemeyer et al 2001) and typical for coagulopathies, cats presented with haematomas and thoracic haemorrhage in the form of pleural effusion or mediastinal haemorrhage associated with tachypnoea and dyspnoea. In addition, two cats also had severe otic haemorrhage, which interestingly has also been observed in Devon rex cats with a hereditary vitamin-K-dependent coagulopathy (Giger, personal observation). However, surface bleedings including melaena, haematochezia, and petechiation were also observed. Three of the four cats with superficial bleedings had a mild thrombocytopenia. Although the platelet count was not below 40 000/µl, combined with a coagulopathy and anaemia, this mild thrombocytopenia may have resulted in surface haemorrhage. Similarly, in a canine study on rodenticide intoxication eight out of 20 dogs had surface bleeding and three of them had a mild to moderate thrombocytopenia (Reitemeyer et al 2001). Lewis et al (1997) described eight dogs with anticoagulant rodenticide-induced haemorrhage and thrombocytopenia. Beside these varied signs of haemorrhage, unspecific clinical signs including lethargy, anorexia, vomiting, hypo- and hyperthermia, and hypovolemic shock were observed in the cats of this report which are likely related to the intoxication and blood loss and have also been reported in intoxicated dogs (Sheafor & Couto 1999, Reitemeyer et al 2001).

Because vitamin K antagonists affect factor II, VII, IX, and X, all three (intrinsic, extrinsic and common) pathways of the coagulation cascade are affected. Values of PT and PTT were markedly prolonged, in fact the PT and PTT were indefinitely prolonged (>100 s) in five of seven cats at the day of admission. Severe PT and PTT prolongations have been documented in dogs and humans after rodenticide intoxication (Hellemans et al 1963, Green et al 1979, Woody et al 1992, Reitemeyer et al 2001) and more than threefold increases in PT were found to be highly suggestive of rodenticide intoxication compared to other coagulopathies (Rozanski et al 1999).

Proteins induced by vitamin K antagonists/absence (PIVKA) tests have been advocated in veterinary medicine for detection of rodenticide toxicosis as in theory the test should detect the altered carboxylation prior to elongation of the PT. However, the commercially available test, also known as Thrombotest (Nyegaard and Co., Oslo, Norway), is actually a modified PT test using diluted plasma and a specific thromboplastin for activation of factor VII leading to very prolonged clotting times in case of extrinsic coagulation cascade disorders (Mount et al 1986, Rozanski et al 1999). The PIVKA test is not specific for rodenticide intoxication as other conditions such as isolated hereditary factor VII or X deficiencies also cause PIVKA time prolongation (Rozanski et al 1999). However, marked prolongation of the PT/PTT and PIVKA are strongly suggestive of rodenticide intoxication. Immunoassays to detect PIVKA would be more specific, but are not routinely available.

The thrombin time is the only coagulation screening test unaffected by rodenticides, as this test only assesses the quantity and function of fibrinogen to form fibrin after the exogenous addition of thrombin (Factor IIa). Thus, a normal thrombin time in light of severely prolonged PT and PTT is highly suggestive of rodenticide intoxication, and allows for differentiation from other multifactor coagulopathies such as liver disease and DIC (Giger 1995). It should be noted that fibrinogen split products (FSP) and D-dimers are not only elevated in DIC, but can also be increased in animals with internal bleeding such as caused by rodenticide intoxication, because of the breakdown of fibrinogen and fibrin in haematomas and effusions in dogs (Griffin et al 2002). These parameters were not assessed in the cats of the current report.

Although anticoagulant rodenticides induce a coagulopathy, they apparently also cause thrombocytopenia and thereby surface bleeding as shown here in two cats. The mechanism for the thrombocytopenia, which had also been reported in dogs with rodenticide intoxication (Mischke 1997, Lewis et al 1997, Sheafor & Couto 1999, Furlanello et al 2000, Reitemeyer et al 2001) remains unknown, but may include platelet loss due to haemorrhage, a consumptive process, or impaired thrombopoiesis (Lewis et al 1997).

The general therapeutic principles of rodenticide intoxication include: (1) emesis and prevention of further toxin exposure within the first hours of ingestion, (2) stop and prevent further haemorrhage and reverse coagulopathy by the application of vitamin K_1 and the transfusion of whole blood or fresh (fresh frozen) plasma, and (3) correct the anaemia and organ failures. Because the cats of this report presented with bleeding, intoxication must have occurred at least 2 days previously. Therefore, induction of emesis would not only be ineffective, but is contraindicated, because of the risk for inducing further life-threatening haemorrhage. However, animals should be kept under close observation to monitor and prevent further intoxication. It is possible that the one cat with recurrent haematoma formation 1 month after initial presentation of this report may have been reintoxicated.

Similar to canine studies most cats showed a normalisation of clotting times after 2 days (Sheafor & Couto 1999, Reitemeyer et al 2001). The cause of the individually different duration until normalisation of coagulation times remains unclear. It may depend on the type of toxin, the dose of ingested poison, the dose and absorption of vitamin K and the hepatic function of the animals. One of the cats in this report had again a prolonged PT 5 weeks after the initial presentation and 4 weeks after termination of vitamin K treatment. This may be due to a very high poison dose and involvement of ultralong acting anticoagulant or more likely due to toxin reexposure. It is prudent to advise clients to minimise the future possibility of reexposure.

Five of the seven cats were severely anaemic with Hct between 8 and 19% and three of them had hypoproteinemia or hypoalbuminemia, which is consistent with external blood loss anaemia. They received blood type matched fresh whole blood during the first or second day, and aside the administered blood volume, the observed Hct rise varied, likely due to ongoing blood loss, infusion-related haemodilution and fluid shifts as four cats also presented in hypovolemic shock. However, an initial transfusion volume of 10 ml/kg fresh whole blood should be effective in providing sufficient erythrocytes for oxygen carrying capacity, but may need to be repeated depending on the type and severity of bleeding. As fresh whole blood also contains functional platelets, coagulation factors, and other plasma proteins, the bleeding tendency can thereby be immediately corrected, which is pivotal in seriously bleeding animals.

Vitamin K_1 is the key antidote and is readily absorbed by the gastrointestinal tract (Murphy & Gerken 1989) and effective in carboxylating newly synthesised factors in the liver, but not in the plasma. Vitamin K_1 rather than K_3 should be

administered as Vitamin K_3 need to be metabolised and is not reliable in its activity in dogs. Based upon the kinetic a normalisation of the coagulation times could be expected in 1–2 days, which is what was observed in the cats of this study as well as in dogs (Reitemeyer et al 2001). In very weak, anorectic patients or with vomiting, initial subcutaneous application is preferred as intramuscular injections may cause serious haemorrhage. As opposed to the study of Sheafor & Couto (1999), who noted urticaria and abscess in two dogs after subcutaneous administration, the cats of this study did not show any adverse effects of vitamin K_1 therapy nor were they observed in dogs in a similar study (Reitemeyer et al 2001). The intravenous application of vitamin K_1 seems unnecessary because of the rapid absorption from the gastrointestinal tract or subcutaneous tissue and is not recommended, because of the risk of systemic anaphylactic responses, possibly with a lethal outcome (Clark & Halliwell 1963, Kammermann-Lüscher 1978). However, the vitamin K formulation may have recently been changed (adjuvant) to be also possibly safe for intraveneous administration.

Because the rodenticide was not identified in any cats of this study, a high dose and protracted long time course was chosen. Doses of vitamin K_1 of 4 mg/kg daily for 5 days have been reported to cause Heinz bodies in one out of seven dogs (Fernandez et al 1984). However, none of the cats appeared to experience any red cell changes, despite the fact that feline haemoglobin is particularly sensitive to oxidative injury due to the high number of free thiol groups (Christopher 2000). A lower dose of vitamin K_1 for a shorter time period than what has been used in this study may have been sufficient. It is prudent to monitor the response to treatment with coagulation times such as PT, aPTT, or activated clotting time (ACT). ACT tube assays are somewhat difficult to perform in cats, however point-of-care instruments to perform PT and PTT in practice have become available and facilitate monitoring and prompt therapeutic adjustments (Tseng et al 2001).

Although much less common than in dogs, rodenticides may cause serious haemorrhage in cats. Fortunately, all cats of this study survived, presumably because of the rapid specific and supportive therapy. In larger studies of intoxicated bleeding dogs, the survival rates have also been high with 82–90% (Mischke 1997, Sheafor & Couto 1999, Reitemeyer et al 2001). In conclusion, if a rodenticide intoxication is detected and the cat is treated intensely with vitamin K_1 and blood components as well as supportive care early enough, a favourable outcome can be predicted.

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