Incidence of prolonged prothrombin time in dogs following gastrointestinal decontamination for acute anticoagulant rodenticide ingestion

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Abstract

Objective: To determine the effect of gastrointestinal (GI) decontamination on the incidence of prolonged prothrombin time (PT) in dogs after anticoagulant rodenticide ingestion.
Design: Retrospective study.
Setting: Urban emergency room.
Animals: One hundred and fifty-one client-owned dogs.
Measurements: Dogs presented to the emergency room within 6 hours of ingestion of an anticoagulant rodenticide and had a PT measured within 2–6 days of toxicant ingestion before initiating vitamin K therapy were included. Dogs were categorized as treated or untreated based on the institution of vitamin K therapy following PT testing. The signalment, body weight, type of rodenticide ingested, time elapsed between ingestion and initial presentation, method(s) of GI decontamination, and the times elapsed between both toxicant ingestion and initial hospital presentation until determination of PT were recorded. The PT results were recorded as well as any treatment received following the recheck examination. Any reported incidents of bleeding or untoward effects between exposure and reexamination were recorded.
Main results: Of 151 dogs, only 11 dogs (8.3%) developed prolonged PT requiring vitamin K supplementation. None of the 11 dogs with prolonged PTs exhibited signs of bleeding or required transfusion therapy. No differences in age, weight, or time elapsed between treated and untreated patients were found.
Conclusions: The incidence of prolonged PT is low in dogs receiving GI decontamination within 6 hours of anticoagulant rodenticide ingestion. Delaying vitamin K therapy until a PT has been assessed 48–72 hours after initial exposure appears to be safe and sensitive in dogs following anticoagulant rodenticide ingestion.

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Introduction

Anticoagulant rodenticides are among the most common toxicants ingested by dogs and are responsible for significant morbidity and mortality in dogs.[1] Anticoagulant rodenticides induce a profound coagulopathy secondary to the antagonism of hepatic vitamin K epoxide reductase, an enzyme that is required to reduce vitamin K epoxide back to active vitamin K.[1] In the absence of vitamin K, carboxylation of procoagulant factors II, VII, IX, and X and anticoagulant proteins C and S to their active forms is inhibited (Figure 1).[2] Because of the relatively short half-lives of factors II, VII, IX, and X (41, 6.2, 13.9, and 16.5 h, respectively[3]), the plasma concentrations of these activated factors become rapidly depleted unless vitamin K is supplemented following anticoagulant rodenticide intoxication.

The initial signs of anticoagulant rodenticide toxicosis are often vague or undetectable until the onset of hemorrhage. Although surface bleeding, such as melena, hematemesis, epistaxis, and petechia or ecchymosis of mucous membranes and skin may occur, bleeding into body cavities is more common with clotting factor deficiencies.[1] Internal hemorrhage into the pleural space, lungs, pericardium, mediastinum, peritoneum, retroperitoneum, joints, fascial planes, urinary bladder, uterus, and ventral hematomas have been documented

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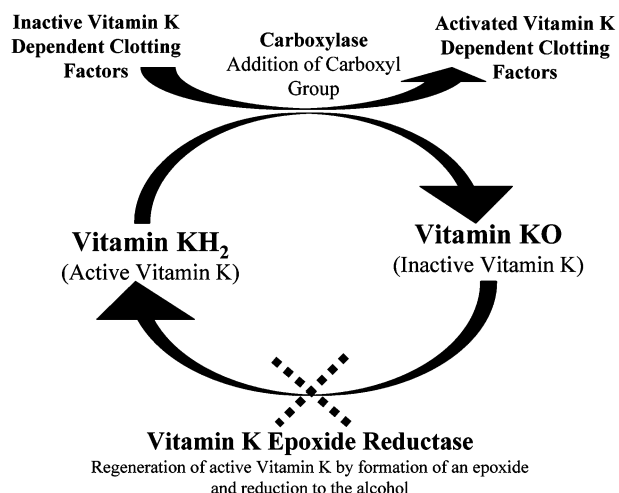
Figure 1: Mechanism of action of anticoagulant rodenticides. Anticoagulant rodenticides inhibit the vitamin K epoxide reductase, which prevents recycling of vitamin K to its active form.

following anticoagulant rodenticide intoxication, many of which result in hemorrhagic shock.[1,4–8] When presented with clinical signs attributable to anticoagulant rodenticide toxicosis, emergency therapies, such as intravenous volume expansion, oxygen supplementation, and provision of transfusions to replace depleted coagulation factors and red blood cells are often needed. Supplementation of vitamin K_1 is required to compensate for impaired vitamin K epoxide reductase activity.[2,9]

For those animals presented within several hours of anticoagulant rodenticide ingestion, gastrointestinal (GI) decontamination, consisting of inducing emesis, administration of an adsorbent (e.g., activated charcoal), and possibly a cathartic, has been recommended to limit toxicant absorption. Following GI decontamination, there are possible treatment strategies: either preemptive treatment with vitamin K_1 for 1–6 weeks (as dictated by the half-life of the rodenticide ingested)[1,10] or evaluation of the animal's prothrombin time (PT) in 48–72 hours followed by vitamin K_1 therapy only in those animals with altered hemostasis.[11]

One expected advantage to universal preemptive vitamin K_1 prophylaxis following anticoagulant rodenticide ingestion is a decreased risk for hemorrhage as a result of toxicosis. However, vitamin K_1 is costly, may require a lengthy treatment course requiring considerable owner adherence, and includes the potential for allergic reactions to medical therapy.[10,12] Moreover, based on the amount of anticoagulant rodenticide ingested and the efficacy of GI decontamination, vitamin K_1 administration may not be necessary in all dogs that ingest anticoagulant rodenticides.

In dogs with experimental warfarin intoxication, PT prolongation was detected 24–36 hours after inges-

tion.[10,13] The elevation in PT is related to the depletion of factor VII, which has the shortest half-life (6.2 h) of all vitamin K dependent clotting factors.[1,3] Despite prolonging the PT, factor VII depletion is not thought to cause bleeding tendencies, as anticoagulant rodenticides are considered to alter coagulation primarily through reductions in factor II (prothrombin).[9,10] Therefore, the onset of bleeding would not be expected until factor II, with a half-life of 41 hours,[3] is depleted. It takes a minimum of 2 half-lives of factor II, or approximately 3.5 days, for the anticoagulant effects resulting in bleeding to manifest.[9,10,14]

The aim of this study was to retrospectively assess the safety and efficacy of limiting vitamin K_1 supplementation to dogs with prolonged PTs measured within 2–6 days of anticoagulant rodenticide ingestion and GI decontamination. In client-owned dogs that ingested an anticoagulant rodenticide within 6 hours of receiving veterinary care, we hypothesized that delaying vitamin K_1 therapy for 48–72 hours following exposure would be safe and would identify those dogs that absorbed a sufficient amount of toxicant to result in coagulopathy requiring vitamin K_1 therapy.

Materials and Methods

The computerized medical record database of the Matthew J. Ryan Veterinary Hospital at the University of Pennsylvania was searched to identify dogs that were presented to the emergency service between January 2000 and July 2005 with a chief complaint of anticoagulant rodenticide ingestion. Dogs were included in the study if ingestion of an anticoagulant rodenticide was documented, medical evaluation was performed within 6 hours of toxicant ingestion (or, when ingestion time was not recorded, rodenticide was evident in the vomitus), and if the dog was returned for evaluation of a PT within 2–6 days of ingestion.

Dogs were excluded from the study if the rodenticide ingested was not an anticoagulant rodenticide or was unspecified, if the time elapsed between ingestion and presentation to the veterinarian was >6 hours, if vitamin K_1[a] was administered before PT determination, or if the medical record was incomplete.

Clinical information extracted from the medical record included: signalment, body weight (kilogram), type of rodenticide ingested, time elapsed (hours) between ingestion and initial presentation to the veterinarian, method(s) of GI decontamination (e.g., emesis, activated charcoal, cathartic), and the times elapsed (hours) between both toxicant ingestion and initial hospital presentation until determination of PT. PT results from the recheck examination and any treatment received following the recheck examination were

recorded. Lastly, any reported incidents of bleeding or untoward effects between exposure and reexamination were recorded.

Dogs were categorized as either treated or untreated based on the attending veterinarian's interpretation of PT results and use of vitamin K_1 therapy. Over the 5-year study period, several different coagulation analyzers were used. Some analyzers reported the PT as a patient value compared with a healthy reference canine control value run at the same time,[b] while others were reported as a patient value compared with a reference interval that had been established for the coagulation analyzer.[c,d] In order to standardize results, all PTs were reported as a percent prolongation above reference according to the following formula: ([patient value – control value]/control value) × 100. When canine control values were reported, the percent prolongation was calculated using the control value provided. When a reference interval was reported, the control value was calculated as the mid-point value from the reference interval. For this study, prolongation of the PT was defined as a patient value exceeding the reference value by at least 25%.[14,15]

Telephone follow-up was attempted for all dogs included in the study. If successfully contacted, the owner was asked to recall if their dog needed to see a veterinarian at any time related to the ingestion of the anticoagulant rodenticide aside from the initial visit and recheck PT test (either before treatment or following treatment). Dogs were considered lost to follow-up if the client's phone number was disconnected or 3 phone messages were left without a response.

All data were visually inspected and evaluated for normality by Kolmogorov–Smirnov testing. All data analyses were performed using commercially available statistical software.[e] Non-parametric data are reported as the median (25th, 75th quartiles). Continuous data were compared using the Mann–Whitney test, because the data were not normally distributed. Categorical data were compared using the χ^2 test. Statistical significance was set at $P < 0.05$.

Results

The medical records database search identified 578 dogs that were presented to the emergency service for rodenticide exposure between January 2000 and July 2005. Following review of the medical records, 427 (73.9%) dogs were excluded for the following reasons: 4 had a repeat visit within 48 hours for additional exposure to anticoagulant rodenticide, 58 had incomplete records, 59 ingested a non-anticoagulant rodenticide, 79 received preemptive vitamin K_1 therapy, and 227 did not return to the hospital for PT determination. The remaining 151 dogs (26.1%) were included in the study.

All 151 dogs were seen within 6 hours of ingestion of an anticoagulant rodenticide (or had evidence of poison in the vomitus), received some form of GI decontamination, and returned 2–6 days later for PT testing. Based on the attending clinician's interpretation of PT results obtained at re-examination, 92.7% of the dogs (140/151) were not treated with vitamin K_1 and 7.3% of the dogs (11/151) were treated with vitamin K_1.

The population characteristics of the 151 dogs included in this study, including age, weight, sex, and modes of GI decontamination are summarized in Table 1. There were no significant differences in any of the population characteristics between treated and untreated dogs.

While all 151 dogs were known to have been exposed to some type of anticoagulant rodenticide, the specific toxicant was recorded in only 90/151 dogs (59.6%).

Table 1: Population characteristics of dogs with anticoagulant rodenticide exposure

	Untreated (n = 140)		Treated (n = 11)		
	Median	25th, 75th Percentiles	Median	25th, 75th Percentiles	P-value
Age (days)	604.5	216.5, 1479.5	904	290.8, 2427.8	0.463
Weight (kg)	12.00 (n = 103)	6.71, 21.75 (n = 103)	7.08	4.18, 14.10	0.065
Sex – male intact	38		1	*	*
Sex – male neutered	33		3	*	*
Sex – female intact	20		4	*	*
Sex – female neutered	48		3	*	*
Sex – female unknown	1		0	*	*

Dogs were categorized as treated or untreated based on the institution of vitamin K therapy following prothrombin time testing. Sex is reported as total number of animals in each category.
*Because of small sample size, statistical analysis was not available for the marked categories.

Eighty-five of 140 dogs (60.7%) in the untreated group had a specific anticoagulant toxicant recorded and only 5 of 11 (45.5%) dogs in the treated group had a specific anticoagulant toxicant recorded. Brodifacoum (52/90, [57.7%]) was the most common toxin, occurring in 58.8% (50/85) of the known ingestions for the untreated group and 40% (2/5) of the known ingestions for the treated group. In the untreated group, 31.8% (27/85) ingested bromadiolone while 40% (2/5) of the treated group ingested bromadiolone. The other reported intoxicants for the untreated group included diphacinone (3/85, [3.5%]), an unspecified coumarin derivative (1/85, [1.2%]), difethialone (3/85, [3.5%]), and an unspecified indandione derivative (1/85, [1.2%]). The other reported intoxicant for the treated group was difethialone (1/5, [20.0%]). There was no statistical difference in types of anticoagulant ingested and incidence of treatment with vitamin K_1.

GI decontamination was accomplished by either successfully inducing emesis or administration of activated charcoal in all dogs. Emesis induction combined with activated charcoal administration was performed in 87.4% (132/151) of dogs, while emesis induction without activated charcoal administration occurred in 3.97% (6/151) of dogs, and activated charcoal administration was the sole therapy in 8.6% (13/151) of dogs. The dose of activated charcoal administered was recorded for only 21 of 145 dogs (14.5%), with a mean dose of 2.1 g/kg. There was no statistical difference when comparing the method of GI decontamination between patients in the untreated group and the treated group (Table 2).

The time elapsed from ingestion of the anticoagulant rodenticide to the initial presentation to the hospital was known for 129 of 151 dogs (85.4%) (121 in the untreated category and 8 in the treated category). All 22 dogs that had an unknown time elapsed between ingestion and initial presentation had evidence of poison in the vomitus. There was no difference in the median time elapsed from ingestion to initial hospital presentation between the non-treatment group, 1.00 hours (0.63 h, 2.50 h), and the treatment group, 2.13 hours (1.50 h, 3.19 h). (P = 0.125).

Table 2: Method of gastrointestinal decontamination in 151 dogs with anticoagulant rodenticide exposure

	Untreated (*n* = 140)	Treated (*n* = 11)*
Emesis only	6	0
Activated charcoal only	12	1
Emesis and activated charcoal	122	10

*There were no significant differences between treated and untreated dogs when comparing the method of gastrointestinal decontamination.

The median time elapsed between ingestion and evaluation of the PT for dogs in the untreated group was 52.3 hours (46.3 h, 64.9 h), which was not different from the median time of dogs that were treated, which was 52.2 hours (42.4 h, 59.5 h). This data was available for 121 of 140 (86.4%) dogs in the untreated group and 8 of 11 (72.7%) dogs in the treated group.

The median time elapsed between the initial presentation and evaluation of the PT for dogs in the untreated group was 50.1 hours (44.8 h, 61.1 h), which was not different from the median time of dogs that were treated, which was 49.7 hours (42.6 h, 51.0 h). These data were available for all 151 dogs included in the study.

PTs were available for 122 of 140 (87.1%) untreated dogs and 11 of 11 treated dogs. For the 18 untreated dogs where results were not available in the medical record, discharge instructions stated that the PT was normal. The PT was 4.5% (0.0%, 15.6%) prolonged in the 122 untreated dogs and 72.4% (44.0%, 114.9%) prolonged in the 11 treated dogs, which was statistically different (P < 0.001).

Prolongation of the PT by >25% of the reference value was present in 18 of 151 dogs (11.9%). Seven of these dogs were in the untreated group and 11 were in the treated group. The median percentage PT prolongation in the 7 dogs that were not treated was 38.8% (32.8%, 40.7%) compared with the 72.4% (44.0%, 114.9%) median prolongation seen in the 11 dogs that were treated with vitamin K_1. Of the 7 dogs that were not treated with vitamin K_1, 6 returned for an additional PT measurement 24–48 hours later. One dog did not return for a repeated PT and was lost to follow-up. Of the 6 dogs that returned, 3 had PTs <25% prolonged and remained asymptomatic. The remaining 3 dogs had PTs >25% prolonged, 1 with a percent prolongation of 28.4%, and 2 with 35.8% percent prolongation. All 3 remained untreated because the magnitude of prolongation had decreased from the previous visit. Only 1 of these 3 dogs' owners were successfully contacted – this dog was reported to remain asymptomatic.

Fifty-one dogs in the untreated group and 4 in the treated group returned for a PT earlier than 48 hours following ingestion. The median time for dogs in the untreated group that returned early was 42.7 hours (23.3 h, 47.8 h), while the median time for dogs in the treated group that returned early was 41.4 hours (40.9 h, 44.8 h). All 4 dogs in the treated group already demonstrated prolongation of the PT. Of the 51 untreated dogs that returned early, 4 had additional PT values performed approximately 48 hours later, none of which were prolonged >25%. Follow-up was available in 25 of 51 (49%) of dogs in this category. None of these dogs

required further veterinary care related to the rodenticide ingestion.

Telephone follow-up was available in 72 of 140 (51.4%) dogs in the untreated group and 6 of 11 (54.5%) dogs in the treated group. For the untreated dogs, none required vitamin K_1 administration or transfusions at a later date, nor were any complications from anticoagulant rodenticide ingestion reported. Of the 6 dogs in the treated category that required vitamin K_1 therapy, none required transfusion therapy (red blood cells or fresh/fresh frozen plasma), nor did they develop clinical signs indicative of a coagulopathy.

Discussion

Anticoagulant rodenticide ingestion is a common intoxication in dogs that can lead to considerable morbidity and mortality.[1] Early identification of toxicant ingestion and GI decontamination are important to reduce the risk of development of a coagulopathy or need for antidotal vitamin K_1 therapy. This study demonstrates that immediate institution of vitamin K_1 therapy is not necessary in all dogs following anticoagulant rodenticide ingestion. One hundred and fifty-one dogs that presented to the emergency room within 6 hours of ingestion of an anticoagulant rodenticide were evaluated, received some form of GI decontamination (emesis or activated charcoal), and were returned within 2–6 days for PT testing to assess coagulation. No dog received prophylactic vitamin K_1 therapy before PT testing. Only 7.3% (11/151 dogs) ultimately absorbed a sufficient quantity of the toxicant to result in coagulopathy necessitating antidotal vitamin K_1 therapy. While all 11 of these dogs demonstrated prolonged PTs, none exhibited any untoward effects from the anticoagulant rodenticide between the initial presentation for GI decontamination and the follow-up visit for PT testing.

While the inclusion criteria for the study specified that the PT measurement was within 2–6 days of toxicant ingestion, 94.7% (143/151) presented for PT measurement within 72 hours (3 days) and only 5.3% (8/151) dogs presented for measurement after 72 hours (7 untreated, 1 treated). The 1 patient in the treated group had a percent prolongation of 34.9% and may not have required treatment. Unfortunately follow-up was not available for this patient. Given the small number of patients evaluated after 72 hours and the known half-lives of the clotting factors affected by anticoagulant rodenticides, the authors can only safely draw conclusions about patient safety up to 72 hours and would not recommend waiting longer than 72 hours for PT evaluation.

In this study, there were no statistical differences detected in any of the population characteristics, the time

elapsed between ingestion of the toxicant and the emergency visit for GI decontamination, or the type of GI decontamination performed between the dogs that were untreated or treated with vitamin K_1. While no statistical differences were detected, there was a tendency for body weight ($P = 0.065$) to be lower in dogs that required treatment (7.08 kg) *versus* those that were not treated (12.00 kg). Smaller dogs that ingest a specific amount of toxicant would ingest a greater milligram per kilogram dose, and therefore be more likely to ingest a toxic dose compared with larger dogs. Were there a greater number of dogs enrolled to increase the power of this analysis, it is possible that weight would have been significantly different.

This study demonstrates that withholding vitamin K_1 therapy until the results of a PT have been evaluated 2–3 days following ingestion is safe. The earliest time that a PT prolongation was detected in the dogs that were treated with vitamin K_1 was 40.9 hours, with a median time of 49.7 hours. None of the 11 dogs in the treatment group, despite PT elevations ranging from 31.0% to 417.2% over the reference value demonstrated any clinical evidence of bleeding nor did they require transfusion of either red blood cells or plasma. The dog with the most dramatic PT prolongation (417.2%) had the PT performed 44.8 hours after initial evaluation.

Although there were 7 dogs in the untreated group that had PT prolongations >25% over the reference value, 3 had PTs <25% prolonged and remained asymptomatic on a follow-up examination 1–2 days after the initial PT. Three dogs continued to have a prolonged PT on a follow-up examination 1–2 days after the initial PT, although the magnitude of the prolongation had decreased from the previous test. One dog was not available for follow-up. Furthermore, all 7 of these dogs demonstrated prolonged PTs with the same analyzer.[c] In dogs with minor PT prolongations (<30%), one option may be to recheck a PT test in 24 hours to better determine the need for vitamin K_1 therapy.

Based on the half-lives of the vitamin-K-dependent coagulation factors, an increase in the PT should not occur until approximately 36 hours after ingestion.[1,9,10] The initial effect is related to the decrease of factor VII, which has a short half-life *in vivo* of 6.2 hours.[3] This prolongation in the PT is not associated with a clinically important effect, as anticoagulant rodenticides are thought to exert their effect mostly through reductions in factor II (prothrombin).[9,10] If the ability of anticoagulant rodenticides to impair clot growth and prevent further clot formation depends in principal on clearance of factor II with a half-life of 41 hours, then it should take a minimum of 2 prothrombin half-lives, or about 3.5 days, to express the antithrombotic effect in patients.[9,10] Following experimental intoxication, initial

changes in the coagulogram were noted at 3 days, while clinical illness was not documented until 4–6 days after toxicant ingestion.[16] Experimentally, delayed toxicosis following ingestion has been documented.[13]

In addition to being safe, there are several other reasons to consider PT testing before vitamin K_1 therapy. At the authors' hospital, the cost for a 4-week course of vitamin K_1 therapy (2.5 mg/g PO q 12 h) for an average-sized dog based on the weights recorded in this study (10 kg) is $47.60. The cost for a PT test is $28, which should be performed in both circumstances, either 48–72 hours after initial toxicant exposure, or 2–3 days following the completion of the prophylactic treatment course. Therefore, based on this study, 92.7% (140/151) of clients would have saved an average of $47.60, and they would have been relieved of the stress associated with medicating their dog twice daily for 4 weeks, not to mention that the treatment course was potentially unnecessary.

One limitation to this study is that not all dogs had telephone follow-up available. Only 51.4% (72/140) of the dogs that did not require treatment had telephone follow-up. However, every owner that was contacted indicated that medical attention was not required at any latter point related to the ingestion of the anticoagulant rodenticide. While a potential limitation, there was no statistical significance when comparing time from initial visit to the PT test in untreated dogs that were available for follow-up (52.3 hours) and dogs that were lost to follow-up (49.1 hours). Therefore, if dogs that were lost to follow-up followed the same statistically pattern in developing signs related to anticoagulant rodenticide toxicosis it is unlikely that significant clinical signs developed in these dogs either.

Another limitation to this study is the small number of dogs ($n = 11$) in the group requiring treatment with vitamin K. This may have made detection of differences in population characteristics or times elapsed between exposure and GI decontamination or PT testing more difficult to detect. Despite this limitation, the low incidence of coagulopathy supports the rationale for withholding vitamin K_1 therapy unless there is clinical evidence that a sufficient dose of toxicant was absorbed.

The retrospective nature of this study made it impossible to quantitate the amount of poison ingested. While attempts have been made to calculate the likelihood of a toxic ingestion,[17] it is frequently noted that owners either do not know or are unable to precisely quantitate the amount of poison ingested. Anticoagulant rodenticides are produced by a number of different manufacturers with numerous active ingredients and are packaged in a variety of sizes and quantities. For this reason, it is difficult to determine if the amount of toxicant ingested by the dogs in this study or if the GI decontamination performed prevented sufficient absorption to result in prolongation of the PT.

This study demonstrated that owner adherence following emergency treatment for anticoagulant rodenticide ingestion is poor despite written discharges instructing the owners of the needed follow-up care. The initial medical records search found 578 dogs that were seen for acute anticoagulant rodenticide ingestion, but 227 dogs (39%) were excluded from the study because the owner did not return the dog for PT testing following GI decontamination. One possible explanation for this low level of adherence is that owners elected to have this test performed by their local veterinarian, although this was not confirmed by telephone contact. Despite this poor adherence, of the 227 excluded dogs, none re-presented to our hospital for clinical bleeding following the initial presentation. Even if owners did not have PT times performed, the incidence of coagulopathy should be similar to the incidence reported. If, however, this truly indicates that 39% of clients were not adherent, veterinarians must strongly emphasize the importance of returning for bloodwork when adopting this protocol. The non-compliant owners may also be less likely to follow medication instructions further stressing the importance of educating owners of the need for appropriately monitoring and treating anticoagulant rodenticide toxicosis. The percent of patients that did not follow the veterinarian's discharge instructions greatly emphasizes the importance of veterinarian–client communication in both the emergent and non-emergent setting.[18,19]

Anticoagulant rodenticide ingestion is a common intoxication that could provide the means for several future prospective studies. Controversy exits regarding the efficacy of different methods of GI decontamination in many toxicoses.[20] The delayed onset of clinical signs (coagulopathy) and the availability of an effective antidote (vitamin K_1) in anticoagulant rodenticide intoxication provide an ideal toxicosis to determine the efficacy of different methods of GI decontamination with little chance of serious adverse effects or harm to animals.[21,22] One possible prospective study would be to compare GI decontamination and repeat visit within 2–3 days for a PT test (as studied here) to a similar protocol that does not include GI decontamination. The safety of managing anticoagulant rodenticide toxicosis without GI decontamination and vitamin K_1 treatment has already been addressed and found to be a safe and effective protocol in the human medical literature.[20–22] Another possible extension of this study would be to prospectively compare the efficacy of the various methods of GI decontamination in dogs exposed to anticoagulant rodenticides. Traditional GI decontamination using both emesis and activated charcoal could be

compared with emesis only or activated charcoal without emesis using a larger number of dogs in each treatment group. In this study, there was no difference in the incidence of coagulopathy in the 132 dogs that received traditional GI decontamination compared with the 6 dogs that only had induced emesis, or the 13 dogs that only received activated charcoal.

In summary, in acute anticoagulant rodenticide ingestion, withholding vitamin K_1 unless prolongation of the PT is documented 2–3 days later appears safe and cost effective in dogs. This study demonstrates that dogs that are presented within 6 hours of anticoagulant rodenticide ingestion, that are treated with GI decontamination and return within 2–3 days for a repeat PT test, remain free of clinical signs as a result of anticoagulant rodenticide ingestion, whether the PT was normal or prolonged. While clinical bleeding may occur 2–6 days following ingestion, returning for a PT test within 2–3 days appears safe and should detect a prolongation in PT before onset of clinical bleeding in those dogs that absorbed a sufficient quantity of toxicant warranting treatment with vitamin K_1.

Footnotes

[a] Phytonandione, Butler Animal Health Supply LLC, Dublin, OH.
[b] BBL Fibrosystem Precision Coagulation Timer, Becton Dickinson Microbiology Systems, Cockeysville, MD.
[c] STA-Compact, Diagnostica Stago Inc., Parsippany, NJ.
[d] SCA 2000 Veterinary Coagulation Analyzer, Synbiotics, San Diego, CA.
[e] SigmaStat 3.0, SPSS Inc., Chicago, IL.

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