ВЕТЕРИНАРНІ НАУКИ

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MODERN APPROACHES TO THERAPY AND RESUSCITATION OF DOGS IN CASES OF COUMARIN ANTICOAGULANT POISONING

Abstract

Poisoning with coumarin-type anticoagulants in dogs remains a pressing issue in modern veterinary medicine, requiring prompt diagnosis and effective therapy. These substances, widely used as rodenticides, disrupt blood coagulation by inhibiting the synthesis of vitamin K-dependent factors, leading to hemorrhagic syndrome with a high risk of severe internal bleeding. This article analyzes the pathogenesis, clinical manifestations, diagnostic criteria, and current treatment methods for animals exposed to coumarin anticoagulants. A comparative review of two large-scale clinical studies – a 23-year experience from the Western College of Veterinary Medicine (Canada) and practical case reports from veterinary clinics in Ukraine – reveals key features of etiopathogenesis and supports optimization of therapeutic approaches. The main antidote is vitamin K1, whose efficacy is supported by evidence-based clinical data; however, treatment must be comprehensive, including blood transfusions, symptomatic support, and coagulation status monitoring. The article also outlines recommendations for the timing and dosing of vitamin K1, as well as management strategies for severe poisoning cases. The results highlight the need to implement standardized treatment protocols and enhance clinicians' vigilance regarding the latent course of symptoms, alongside the importance of long-term monitoring of coagulation profiles even after apparent clinical recovery. The developed recommendations offer practical value for veterinarians, toxicologists, and clinicians, and can serve as a foundation for further experimental studies in toxicology, pharmacology, and veterinary medicine.

Key words: *anticoagulant rodenticides, coumarin derivatives, dog poisoning, dicoumarol, coagulopathy, prothrombin time, vitamin K1, intensive therapy, resuscitation, coagulation monitoring.*

Introduction. Poisoning of dogs with coumarin-based anticoagulants is a pressing issue in veterinary medicine, associated with the widespread use of these substances as rodenticides in both domestic and agricultural settings. Coumarin anticoagulants inhibit the synthesis of vitamin K-dependent clotting factors, leading to the development of hemorrhagic syndrome, which may result in severe internal bleeding and fatalities. A characteristic feature of such poisonings is a prolonged incubation period, complicating early diagnosis and timely treatment. In veterinary practice, a comprehensive

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approach is crucial, involving the identification of clinical symptoms, laboratory assessment of coagulation parameters, and the selection of optimal therapeutic strategies based on the severity of the poisoning.

In recent decades, numerous clinical studies have been conducted to better characterize the mechanisms of action of coumarin anticoagulants in dogs. Notable among them is the experience of the Western College of Veterinary Medicine (Canada) from 1999 to 2022, along with analyses of case reports from veterinary clinics in Ukraine. These studies highlight the importance of timely diagnosis and the rational administration of vitamin K1 as an antidote, which remains a cornerstone of therapy.

The issue of poisoning necessitates the continuous professional development of veterinarians, the refinement of treatment protocols, and the development of guidelines for prevention and early detection of toxic exposure cases. Scientific advancements are aimed at optimizing therapeutic measures, reducing mortality rates, and improving the quality of life for affected patients.

Aim of the Study. The aim of this research is to analyze the clinical manifestations, diagnostic criteria, and treatment efficacy in dogs poisoned with coumarin-based anticoagulants, based on a review of current scientific data and practical experience. The objectives include identifying key features of the pathogenesis, developing optimal treatment protocols involving vitamin K1 and resuscitation measures, as well as summarizing recommendations for veterinary practice.

Results and Discussion. Anticoagulant rodenticides (ARs) are among the most commonly used poisons for rodent control; however, they also pose a significant toxicological risk to domestic animals, particularly dogs. These compounds disrupt vitamin K metabolism, which plays a crucial role in the synthesis of blood clotting factors, leading to coagulopathy and internal hemorrhage.

ARs are divided into two main categories: First Generation Anticoagulant Rodenticides (FGARs), which include compounds such as warfarin and zoocoumarin, and Second Generation Anticoagulant Rodenticides (SGARs), which were developed to overcome rodent resistance to warfarin. SGARs include substances such as difenacoum, brodifacoum, coumatetralyl, dicoumarol, coumafuryl, and bromadiolone. Second-generation anticoagulants are significantly more toxic and have a prolonged effect – their half-life may reach 3 to 7 days, which far exceeds the approximately 14-hour half-life of warfarin [4; 14, p. 337].

The main pathogenic mechanism of ARs involves inhibition of the enzyme vitamin K1 epoxide reductase, which is essential for the activation of vitamin K1. This leads to a gradual reduction in the levels of active coagulation factors II, VII, IX, and X, resulting in hypocoagulability and a tendency to bleed. Clinical signs of poisoning usually appear 2 to 5 days after ingestion of the toxicant due to the progressive depletion of the coagulation system.

In dogs, the primary routes of intoxication remain accidental ingestion of rodenticide baits or, less commonly, secondary poisoning through consumption of poisoned rodents or non-target species (e.g., birds, invertebrates) [1, p. 5–15; 5]. Anticoagulants are rapidly absorbed through the gastrointestinal tract, and SGARs, in particular, can accumulate in the liver, maintaining their toxic effect for an extended period due to enterohepatic recirculation.

Effective treatment of AR poisoning in dogs is primarily based on the administration of vitamin K1 at therapeutic doses (either orally or parenterally), which enables the restoration of clotting factor synthesis. In severe cases, infusion therapy, plasma or whole blood transfusions, hemodynamic support, and close monitoring of coagulation parameters (PT, aPTT) are indicated [5, p. 5–15]. Modern treatment strategies also include coagulometric monitoring, individualization of the duration of vitamin K1 therapy depending on the type of AR ingested (first- or second-generation), and consideration of potential rodent resistance to specific rodenticides [6, p. 1460–1466; 11, p. 175–190].

Poisoning with coumarin-based anticoagulants remains a pressing issue in veterinary toxicology, requiring practicing veterinarians to have a clear understanding of the toxicant's mechanism of action, the pathogenesis of intoxication, and up-to-date therapeutic and resuscitation algorithms based on the most recent data from 2021–2025. Studying the clinical management experience of such cases is a critical step toward improving treatment efficacy and reducing mortality rates in veterinary patients.

The clinical signs of coumarin anticoagulant intoxication in dogs typically develop after a latent period of 2 to 5 days following ingestion of the toxicant. The duration of the incubation period and the severity of clinical symptoms primarily depend on the dose and the type of compound ingested (first- or second-generation anticoagulants) [1, p. 5–15].

The main manifestations of intoxication are symptoms of coagulopathy, including subcutaneous hemorrhages, hematomas, epistaxis, gingival bleeding, hematuria, melena, dyspnea due to hemothorax or hemopericardium, pallor of mucous membranes, weakness, and collapse. The occurrence of unexplained bleeding or atypical hematomas is often a key reason to suspect AR poisoning [2, p. 1015–1020; 8, p. 112–117].

In the early stages of intoxication, dogs may exhibit general signs of hemorrhagic syndrome and systemic intoxication, which can progress to a severe clinical condition [3, p. 45–59].

To refine the diagnosis, assess the severity of intoxication, and select the optimal treatment strategy, it is advisable to use a combination of laboratory and instrumental diagnostic methods. The main components of the diagnostic process and therapeutic algorithm are presented in Table 1.

From Table 1, it is evident that the key stages in managing dogs with coumarin anticoagulant poisoning include early diagnosis using prothrombin time, specific therapy with vitamin K1, and an individualized approach to the duration of treatment, depending on the type of toxicant, its dose, and the patient's response. In severe cases, therapy is supplemented with plasma transfusion, fluid therapy, and continuous coagulation monitoring.

Table 1. Key Diagnostic Methods and Therapeutic Approaches for Coumarin Anticoagulant Poisoning in Dogs

Component	Indicators / Methods	Features / Comments
Diagnosis of coagulation status	Prothrombin time (PT)	Sensitive test for early detection of factor VII deficiency; increases first; key for early diagnosis of AR poisoning and for monitoring therapy effectiveness.
	Activated partial thromboplastin time (aPTT)	Increases at later stages of intoxication, when other coagulation factors are depleted.
Extended confirmatory tests	Vitamin K content or serum blood analysis	Detection of ARs using high-performance liquid chromatography; used in forensic veterinary and severe clinical cases.
	Postmortem liver and kidney tissue analysis	Identification of toxicants and clinical evaluation after death.
Primary therapy	Administration of vitamin K1	The only specific antidote; restores synthesis of vitamin K-dependent clotting factors.
Factors influencing therapy duration	Presumed toxicant dose	Often unknown, so duration must be determined individually.
	Type of compound (FGAR vs SGAR)	SGARs have longer half-lives; require more prolonged and intensive treatment protocols.
	Individual patient response	Taken into account when determining the length of therapy and during coagulation monitoring.
Recommended duration of vitamin K1 therapy	2–4 weeks	For FGARs and mild cases of intoxication.
	6 weeks or more	For SGAR intoxication or in severe clinical cases.
Complex therapy (additional measures)	Plasma or whole blood transfusion	For severe hemorrhages.
	Hemodynamic support	To stabilize hemodynamic parameters.
	Monitoring coagulation parameters over time	Mainly PT; requires regular monitoring during treatment.
	Patient condition monitoring	Throughout the course of treatment and for several days after completion.

Source: author's own contribution based on [1; 5; 6; 10; 11]

To deepen the practical understanding of AR poisoning in dogs, we analyzed clinical studies that reflect current treatment practices in various countries.

The first of these studies was conducted by a team of veterinary specialists at the Western College of Veterinary Medicine (WCVM) in Saskatoon, Saskatchewan, Canada. The study spanned a substantial time frame of 23 years and included 349 dogs with confirmed anticoagulant rodenticide poisoning of various types. This scale of observation enabled the researchers to formulate reliable conclusions regarding the clinical course, diagnostic strategies, and treatment efficacy [9, p. 496–503].

A thorough analysis of medical records was performed for each case. The evaluation included the general clinical condition, coagulation test results (prothrombin time – PT and activated partial thromboplastin time – aPTT), platelet count, anemia parameters, and the biochemical blood profile. Confirmation of poisoning was based on history of bait ingestion or consumption of rodents, laboratory findings, and imaging techniques such as thoracic and abdominal FAST exams and abdominal ultrasound. Postmortem examinations were performed when necessary. Coagulation testing (PT/aPTT) was considered the standard method for assessing hemostatic disorders.

Diagnosis was established by considering case history, clinical signs of coagulopathy (bleeding of various localizations, anemia), laboratory findings, and positive response to vitamin K1 therapy, which confirmed anticoagulant intoxication. The main treatment strategy involved administering vitamin K1 at 2–5 mg/kg, either subcutaneously or orally. For second-generation anticoagulants, therapy lasted up to 4 weeks. In cases of severe clinical presentation, hospitalization for 1–2 weeks was required, during which the dogs received intensive supportive therapy.

Symptomatic treatment included anemia correction, fluid and electrolyte balance maintenance, and administration of furosemide, vitamin C, glucose, and antibacterial therapy (amoxicillin or amoxiclav). In 49 out of 104 cases with severe hemorrhages, fresh frozen plasma or whole blood transfusions were administered. Treatment outcomes were favorable: 86% of dogs with clinical signs survived to discharge. Importantly, after completion of vitamin K1 therapy, prothrombin time monitoring was mandatory for several days to prevent late-onset recurrence of coagulopathy.

A valuable complement to the Canadian experience was a study conducted in Ukraine, as part of a thesis by M.A. Vlasenko at Dnipro State Agrarian and Economic University, in collaboration with the private veterinary clinic "Kolibri" (Dnipro) [16, p. 79]. This practice-oriented study assessed the efficacy of different treatment protocols in dogs

with acute AR poisoning. The study included 10 dogs of various breeds whose owners had witnessed bait ingestion or found baits in the living environment.

The dogs were divided into two groups of five: a control group (treated with vitamin K3 – Vikasol) and an experimental group (treated with vitamin K1 – Konakion). All dogs underwent clinical examination, blood and urine testing, and coagulation profiling. Diagnosis was based on clinical signs of coagulopathy (localized hemorrhages, hematuria, anemia, hypothermia), laboratory data, and differential diagnosis to rule out DIC syndrome, von Willebrand disease, and liver pathology.

Treatment involved two stages. In the first stage (emergency therapy), the control group received vitamin K3, while the experimental group received vitamin K1 (Konakion) at 3–5 mg/kg intravenously or intramuscularly. Both groups also received symptomatic support: Strophanthin K, furosemide, 5% glucose with insulin and ascorbic acid, Contrical, and amoxicillin.

During the second stage (20–30 days), the control group was treated with oral vitamin K3 in combination with tablet vitamin K, while the experimental group continued on vitamin K1 (Konakion) and tablets. Additional treatments included Essentiale, Catosal, Multivitamin 100 (Norbrook), glucose with insulin and ascorbic acid, and Thioprotectin. In the experimental group, clinical stabilization occurred 6–8 hours earlier than in the control group. Hematological values improved significantly in the K1 group: erythrocyte count increased by 16%, hemoglobin by 18,4%, and albumin by 36,5%. Hemorrhagic syndrome resolved more rapidly with vitamin K1, confirming its clinical advantage in treating acute AR poisoning in veterinary practice.

Thus, both studies – the large-scale long-term analysis conducted at the Western College of Veterinary Medicine (Canada) and the practice-based project by M.A. Vlasenko at the "Kolibri" clinic (Dnipro) – clearly demonstrate both common features and differences in diagnostic and therapeutic approaches to coumarin anticoagulant poisoning in dogs. Despite differences in sample size, regional context, and clinical workflow, both studies emphasize the critical importance of timely diagnosis, use of vitamin K1 as the treatment of choice, comprehensive supportive therapy, and post-treatment coagulation monitoring to prevent relapses of coagulopathy.

To better summarize the key characteristics and approaches used in the aforementioned studies, Table 2 below provides a comparative overview that clearly illustrates both the shared principles and the differences between the two clinical models.

Table 2. Comparative Characteristics of Diagnostic and Treatment Approaches for Coumarin Anticoagulant Poisoning in Dogs Based on Canadian and Ukrainian Studies

Criterion	First Study (Canada)	Second Study (Ukraine)
Institution	WCVM, Saskatoon	"Kolibri" Clinic, Dnipro
Years	1999–2022	2019–2021
Number of dogs	349	10
Diagnostic methods	History; clinical signs; PT/aPTT; Thoracic/Abdominal FAST	History; clinical signs; coagulation testing; differential diagnosis
Main treatment protocol	Vitamin K1; plasma/blood transfusions	Vitamin K1 or K3; plus supportive therapy
Key therapeutic conclusions	SGARs → require prolonged K1 course; importance of monitoring	Vitamin K1 more effective than K3; faster clinical improvement
Overall survival rate	86%	100% (all 10 dogs survived)

Source: author's own contribution based on [9; 15]

Analysis of the data presented in Table 2 confirms both the universality of core therapeutic approaches to coumarin anticoagulant poisoning and the specific features of individual clinical models. In particular, the large-scale Canadian study demonstrated the efficacy of a standardized treatment protocol involving vitamin K1 and emphasized the importance of careful coagulation monitoring in cases of prolonged intoxication with second-generation anticoagulants. The Ukrainian experience, in turn, highlights the advantages of vitamin K1 over K3 and underscores the significance of comprehensive supportive therapy in clinical settings.

Summarizing the presented clinical data, a typical pattern of symptom progression in anticoagulant poisoning – depending on whether first- or second-generation compounds are involved – can be clearly delineated. Poisoning with second-generation anticoagulants tends to follow a more aggressive and prolonged course, requiring a more complex clinical approach and extended treatment duration. The comparative table below illustrates the time-dependent development of clinical signs in dogs following ingestion of both types of rodenticides (Table 3).

The table indicates that second-generation anticoagulants cause symptoms that appear later but are more sudden and severe compared to those induced by first-generation compounds. This necessitates a longer course of vitamin K1 therapy, regardless of initial clinical improvement.

Table 3. Comparative Dynamics of Clinical Symptom Development in Dogs Poisoned with First- and Second-Generation Anticoagulants

Time Since Toxicant Ingestion	First-Generation Anticoagulants (e.g., Warfarin)	Second-Generation Anticoagulants (e.g., Brodifacoum)
0–24 hours	Symptoms often absent	Symptoms often absent
2–3 days	Onset of initial bleeding; ↑ PT	Symptom onset; PT rises sharply
4–5 days	Progression of clinical signs; weakness	Massive hemorrhage; life-threatening condition
After day 5	Without treatment – deterioration	Plasma/blood transfusion may be required
After day 7	Complications less common	Relapse possible if vitamin K1 discontinued too early

Source: author's own contribution based on [9; 12; 13]

Conclusions. The analysis of clinical data obtained from a long-term study in Canada and practical experience in Ukraine confirms the efficacy of vitamin K1 as the treatment of choice for managing coumarin anticoagulant poisoning in dogs. Vitamin K1 ensures faster normalization of hemostasis and resolution of hemorrhagic syndrome compared to vitamin K3, supporting its rational use in veterinary practice.

Successful management of such cases requires comprehensive supportive therapy, including symptomatic treatment, correction of anemia, maintenance of fluid and electrolyte balance, and, if necessary, plasma or whole blood transfusions. Continuous monitoring of coagulation parameters (PT/aPTT) during and after treatment is essential to prevent recurrence of coagulopathy.

Ongoing accumulation of clinical experience and refinement of treatment protocols will contribute to improved outcomes in veterinary care for dogs with coumarin anticoagulant poisoning.

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СУЧАСНІ ПІДХОДИ ДО ТЕРАПІЇ ТА РЕАНІМАЦІЇ СОБАК ПРИ ОТРУЄННЯХ АНТИКОАГУЛЯНТАМИ КУМАРИНОВОГО РЯДУ

Анотація

Отруєння собак антикоагулянтами кумаринового типу залишається актуальною проблемою сучасної ветеринарної медицини, що потребує своєчасної діагностики й ефективної терапії. Ці речовини, широко застосовувані як родентициди, порушують згортання крові шляхом пригнічення синтезу вітамін К-залежних факторів, що призводить до розвитку геморагічного синдрому з високим ризиком тяжких внутрішніх кровотеч. У статті аналізуються патогенез, клінічні прояви, діагностичні критерії та сучасні методи лікування тварин, які зазнали впливу кумаринових антикоагулянтів. Проведено порівняльний огляд двох масштабних клінічних досліджень – 23-річного досвіду Західного коледжу ветеринарної медицини (Канада) та практичних випадків із ветеринарних клінік України, що виявляє ключові особливості етіопатогенезу та сприяє оптимізації терапевтичних підходів. Основним антидотом є вітамін К1, ефективність якого підтверджена доказовими клінічними даними; проте лікування має бути комплексним, включати переливання крові, симптоматичну підтримку та моніторинг стану згортання крові. У статті також наведені рекомендації щодо термінів і дозування вітаміну К1, а також стратегії ведення важких випадків отруєння. Результати підкреслюють необхідність упровадження стандартизованих протоколів лікування та підвищення пильності клініцистів щодо латентного перебігу симптомів, а також важливість довготривалого контролю показників згортання крові навіть після клінічного одужання. Розроблені рекомендації мають практичне значення для ветеринарів, токсикологів і клініцистів і можуть слугувати основою для подальших експериментальних досліджень у галузях токсикології, фармакології та ветеринарної медицини.

Ключові слова: *антикоагулянті родентициди, кумариновий ряд, отруєння собак, дикумарин, коагулопатія, протромбіновий час, вітамін К1, інтенсивна терапія, реанімація, моніторинг згортання крові.*

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