Ethylene glycol poisoning in three dogs: Importance of early diagnosis and role of hemodialysis as a treatment option

A. Schweighauser, T. Francey

Department of Clinical Veterinary Medicine, Vetsuisse Faculty, University of Bern, Switzerland

DOI 10.17236/sat00051

Received: 10.07.2015
Accepted: 09.09.2015

Summary

Poisoning with ethylene glycol as contained in antifreeze can rapidly lead to irreversible acute renal failure and other organ damage. It carries a grave prognosis unless diagnosed early and adequate treatment is initiated within 8 hours of ingestion. Toxicity of ethylene glycol is related to the production of toxic metabolites by the enzyme alcohol dehydrogenase (ADH), leading to early signs of severe polyuria (PU) and polydipsia (PD), gastritis, ataxia and central nervous depression, followed by progressive dehydration, and ultimately oligoanuric renal failure. In addition to general supportive care, therapeutic interventions must include either antidotes blocking ADH-mediated metabolism or blood purification techniques to remove both the parent compound and the toxic metabolites. The goal of this case report is to describe three cases of acute antifreeze intoxication in dogs, and to discuss treatment options available for this poisoning.

Keywords: ethylene glycol, antifreeze, acute kidney injury, hemodialysis, intoxication

Intoxikation mit Ethylenglykol bei drei Hunden: Bedeutung der frühzeitigen Diagnosestellung und Rolle der Hämodialyse bei der Behandlung

Frostschutzmittelintoxikation mittels Ethylenglykol kann in kurzer Zeit zu irreversiblem akuten Nierenversagen und anderen Organschäden führen. Die Prognose ist schlecht, falls die Diagnose nicht sehr früh gestellt und die korrekte Therapie innerhalb der ersten 8 Stunden nach Toxinaufnahme eingeleitet wird. Die Toxizität von Ethylenglykol beruht auf der Produktion von giftigen Metaboliten durch das Enzym Alkoholdehydrogenase (ADH), was in der Frühphase zu hochgradiger Polyurie/Polydipsie, Gastritis, Ataxie und Depression des Zentralnervensystems führt, gefolgt von progressiver Dehydratation und schlussendlich oligoanurischem Nierenversagen. Zusätzlich zu einer allgemein unterstützenden Therapie, können Antidote genutzt werden, die den ADH-übertragenen Metabolismus blockieren, sowie Blutreinigungsverfahren, um die Ursprungssubstanz sowie die toxischen Metaboliten zu entfernen. Das Ziel dieses Fallberichtes ist die Beschreibung von drei Hunden mit akuter Frostschutzmittelvergiftung sowie die Diskussion möglicher Therapieansätze.

Schlüsselwörter: Ethylenglykol, Frostschutzmittel, akute Nierenschädigung, Hämodialyse, Intoxikation

Introduction

Ethylene glycol based antifreeze is a toxic agent reported to cause irreversible damage to the kidneys, and to affect the hepatic, cardiovascular and central nervous system (CNS) (Doty et al., 2006). It is odorless, and is commonly reported to be ingested for its sweet taste (Keller and Goddard, 2005). If appropriate treatment is not started within a few hours, prognosis is grave with progression from acute kidney injury to acute renal failure. Cases of ethylene glycol toxicity in dogs and cats have been regularly reported in the United States (Mueller, 1982; Rowland, 1987; Adams et al., 1991; Connally et al., 1996; Khan et al., 1999). 510 cases of ethylene glycol exposures in animals, 98% being dogs and cats, were compiled by the ASPCA National Animal Poison Control Center in Illinois, USA, from 1995–1997 (Khan et al., 1999). Individual case reports on ethylene toxicity have also been published in other geographical locations including Switzerland (Kupper et al., 2011), South Africa (Keller and Goddard, 2005) and Spain (Goicoa et al., 2003).

Ethylene glycol poisoning in three dogs: Importance of early diagnosis and role of hemodialysis as a treatment option

A. Schweighauser, T. Francey

Antifreeze solutions usually contain approximately 95% ethylene glycol (Thrall et al., 1998). In Switzerland, the law of the EU applies where products containing more than 25% ethylene glycol must be labeled as such as well as for being dangerous for health with an advice to contact a physician in case of ingestion (Ethylenglykol in Frostschutz und Enteisern. Federal Office of Public Health June 2007). While most of the commercially available antifreeze solutions in Switzerland contain propylene glycol, more than 3000 different products registered in our country contain ethylene glycol in variable amounts (personal communication Federal Office of Public Health). The minimum lethal dose of undiluted ethylene glycol is 6.6 ml/kg in the dog and 1.5 ml/kg in the cat (Thrall et al., 1998). Propylene glycol, on the other hand, seems to be less of a risk, although Claus et al. (2011) reported a dog with clinical signs similar to early stage ethylene glycol toxicity.

Ethylene glycol toxicosis is potentially lethal, and early diagnosis and treatment are crucial. After ingestion, it is rapidly absorbed from the gastrointestinal tract with peak plasma concentrations in dogs 2–3 hours post-ingestion. It is subsequently metabolized primarily by the liver (Grauer et al., 1984; Grauer et al., 1987). Its toxicity is not related to the parent compound, but to the production of toxic metabolites by the enzyme ADH. At the end of the cascade, oxalic acid chelates calcium, leading to the formation of insoluble calcium oxalate complexes. These are freely filtered by the kidneys and will ultimately deposit in the renal tubules causing acute kidney injury and to a lesser extent, in the vasculature of the brain, heart and other organs (Gaynor and Dhupa, 1999a; Rietjens et al., 2014).

The goal of this case report is to highlight the fact that acute ethylene glycol poisoning also occurs in Switzerland, to describe the clinical picture in dogs, as well as the possible treatment options.

Case histories

Case 1

A 2–year-old male intact German Shepherd, BW 30.4 kg, was presented to the Small Animal Teaching Hospital in Bern with a history of witnessed ingestion of ethylene glycol-containing antifreeze (Agrola Frostschutz II xl 50/50) in an estimated amount of several deciliters approximately 3 hours prior to presentation. Clinical signs reported by the owners included ataxia, disorientation and acute onset of severe PU/PD shortly after ingestion. On initial clinical examination, the dog showed moderate depression, severe generalized ataxia, disorientation and limited responsiveness. Cardiovascular parameters were normal except for a bradycardia of 52 bpm with a sinus rhythm on ECG. Blood pressure measured with an oscillometric device (Cardell Veterinary monitor 9403, Midmark Animal Health, USA) was systolic 112, diastolic 63, and mean arterial pressure 86 mmHg on average of 3 consecutive measurements. Serum creatinine and urea were within normal limits (Tab. 1). Treatment goals at this time included prevention of ethylene glycol metabolism and enhancement of its elimination. Ethanol 5% (Ethanol 20%, Christoffel Apotheke Bern, diluted in a 5% glucose solution, Glucosum 5% "Bichsel", Laboratorium Dr. G. Bichsel AG) was infused at 22 ml/kg iv over 30 minutes. Additionally, high-clearance hemodialysis was initiated to rapidly remove the small molecular weight ethylene

Table 1: Selected laboratory data of 3 dogs with ethylene glycol toxicity
Hct, hematocrit; iCa^{2+}, ionized calcium; AG, anion gap; p.i., post ingestion of ethylene glycol; a, pre dialysis; b, post dialysis

	Hct %	Creatinine µmol/l	Urea mmol/l	Total Ca mmol/l	iCa^{2+} mmol/l	K^+ mmol/l	AG mmol/l
Dog #1							
3h p.i.[a]	40	78	4.8	2.28	1.20	3.4	15.9
11h p.i.[b]		15	0.8	2.06	n/a	3.4	n/a
24h p.i.		83	5.4	2.36	n/a	3.9	n/a
15d p.i.		95	5.3	n/a	n/a	n/a	n/a
Dog #2							
9h p.i.	47	70	2.7	2.88	n/a	4.7	n/a
30h p.i.	36	433	23.9	n/a	0.91	6.6	35.2
Dog #3							
24h p.i.	45	324	10.3	2.42	n/a	4.8	n/a
48h p.i.	n/a	742	22.6	2.17	n/a	5.0	n/a
54h p.i.	26	n/a	n/a	n/a	1.15	n/a	24.6
Ref. range	39–57	53–120	3.5–11.1	2.50–2.93	1.09–1.38	4.1–5.3	14.8–19.0

Band 158, Heft 2, Februar 2016, 109–114, © GST | SVS

glycol and its metabolites. For this purpose, a 12-French 20 cm double lumen central venous catheter (Arrow Swiss GmbH, Baar, Switzerland) was placed in the right jugular vein under deep sedation with butorphanol (Morphasol, Dr. E. Gräub AG, 0.3 mg/kg iv) and propofol (Propofol 1% MCT, Fresenius Kabi Schweiz AG, iv to effect after an initial bolus of 3 mg/kg). Dialysis therapy was performed on a Gambro AK 200^{R} Ultra S machine using a Polyflux^{R} 140H filter and Gambro BL200BD blood tubings with a total extracorporeal volume of 240 ml. Bicarbonate based dialysate was used (A Component 283 and BiCart^{R}, Gambro Lundia AB, Sweden) and systemic heparinization (Heparin Bichsel 1000 IU/ml, Laboratorium Dr. G. Bichsel AG) was provided as an initial bolus of 50 IU/kg followed by a constant rate infusion of 50 IU/kg/h titrated to double the activated clotting time compared to baseline. A urea reduction ratio of 83% with an ionic fractional clearance of urea, Kt/V, of 4.1 was reached by processing 126 L of blood (4.1 L/kg BW) in 398 minutes. For relevant details of blood parameters at the end of the hemodialysis treatment, see Table 1.

Under extracorporeal blood purification the dog became progressively brighter, alert and responsive. Ataxia resolved and he was clinically unremarkable at the end of the hemodialysis treatment. A balanced crystalloid electrolyte solution (Plasma Lyte A, Baxter AG) was administrated at a rate of 4 ml/kg/h iv to enhance diuresis, and treatment with omeprazole (Teva Pharm AG; 20 mg po q24h) and ranitidine (Christoffel Apotheke Bern; 0.5 mg/kg iv q12h) was initiated due to expected gastric irritation by ethylene glycol. The dog recovered uneventfully, and iv fluids could be reduced progressively and then discontinued 27 hours later. Serum chemistry was reevaluated on day 2 and all parameters were within normal limits. The dog was discharged on day 3 in excellent clinical condition with omeprazole 20 mg po q24h for 4 days. A recheck was performed by the referring veterinarian after 16 days and revealed normal renal parameters on bloodwork (Tab. 1).

Case 2

A 7-year-old male intact Jack Russell Terrier was referred to the Small Animal Teaching Hospital in Bern with a history of acute onset of severe ataxia and mental disorientation followed by severe acute PU/PD immediately after the dog had been unsupervised for a short period of time and suspected to have ingested some unidentified toxic substance. He had been presented to an emergency veterinarian 3 hours post suspected ingestion and was treated with apomorphine and dexamethasone. His general condition worsened over the next few hours and he was presented again to another veterinarian 6 hours later, this time in shock, hypothermic (<35 °C rectal temperature), severely obtunded and ataxic. Treatment for cardiovascular stabilization was initiated with iv crystalloid infusion. Due to progression of obtundation to stupor, oliguria, and development of azotemia, the dog was then referred to the clinic approximately 30 hours post suspected ingestion. On presentation, the dog was in severely reduced general condition, stuporous with intermittent signs of mental disorientation and vocalization. Perfusion and hydration were considered adequate and results of emergency bloodwork are summarized in Table 1. The dog showed moderate azotemia (creatinine 433 µmol/l, reference range: 53–120) and severe high anion gap metabolic acidosis with venous pH 7.064 (7.320–7.517), $HCO3^{-}$ 8 mmol/l (18.3–26.7), pCO2 19.6 mmHg (24.9–46.9) and anion gap 35.2 mmol/l (10.5–19.0). Serum osmolality (Mikro-Gefrierpunkt Osmometer OM-806, Sysmex Digitana AG, Horgen) was 371 mOsm/kg (300–310) and calculated osmolality was 352 mOsm/kg (264–292; Silverstein and Hopper, 2009). Resulting osmolar gap was 19 mOsm/kg (10+/–6; Grauer et al., 1984). Urinalysis revealed isosthenuric urine specific gravity (USG) at 1.010, pH 5, trace proteins and no crystals. Urine osmolality was 361 mOsm/l (500–1200 mOsm/l; Silverstein and Hopper, 2009). Abdominal ultrasound showed markedly hyperechoic renal cortices bilaterally with a distinct medullary rim sign, highly suggestive of ethylene glycol toxicity. Based on the severe clinical picture and a guarded prognosis for renal recovery, owners opted for euthanasia.

Case 3

A 5-year-old female spayed Border Collie, BW 15.8 kg, was referred to the Small Animal Teaching Hospital in Bern with a history of acute kidney injury after witnessed ingestion of ethylene glycol-containing antifreeze in an estimated amount of several milliliters 2 days prior to presentation. The dog had shown neurological signs including ataxia and disorientation initially, and now presented with worsening vomiting and diarrhea. Initial PU/PD progressed to oliguria and later to anuria. Treatment with iv fluids, metoclopramide, ranitidine, dopamine and glucose was initiated, and the dog was referred to the clinic due to progressive renal azotemia (Tab. 1). Upon initial clinical examination, the dog was lethargic, weak, and mildly ataxic. He was estimated 5% overhydrated and despite this, oral mucous membranes were dry and tacky, compatible with uremic xerostomia. Cardiovascular parameters were within normal limits. Upon abdominal palpation, the bladder was empty, kidneys were painful, and vomiting was elicited. Emergency bloodwork was performed as depicted in Table 1. The dog showed a high anion gap metabolic acidosis with venous pH 7.234 (7.320–7.517), $HCO3^{-}$ 10.3 mmol/l (18.3–26.7), pCO2 24.9 mmHg (24.9–46.9) and anion gap 24.6 mmol/l (10.5–19.0). Abdominal ultrasound revealed bilateral severe renal cor-

tical hyperechogenicity. Due to poor prognosis for renal recovery, owners elected euthanasia.

Discussion

The clinical signs of ethylene glycol intoxication are classically described in three fairly distinct clinical stages, summarized in Table 2 (Gaynor and Dhupa, 1999a). Marked and progressive PD within 1 hour of ethylene glycol ingestion, as reported in dogs #1 and 2, is typically a hallmark of this intoxication and is thought to result from stimulation of the thirst mechanism by the acute rise in serum osmolality as seen in dog #2. This hyperosmolality is normally a strong stimulus for vasopressin release and renal water conservation, but it has been suggested that ethylene glycol may inhibit vasopressin release in a manner similar to that of ethanol. In addition, severe PU due to marked ethylene glycol-induced osmotic diuresis is common in dogs initially. All these mechanisms can rapidly lead to severe dehydration, hypovolemia and prerenal azotemia (Gaynor and Dhupa, 1999a). Dehydration is further amplified by the animal's inability to drink due to CNS depression. All 3 dogs in this case series displayed variable degrees of mental affection. CNS depression may diminish after 12 hours mimicking clinical improvement before progressive uremia affects mentation.

Azotemia is an important diagnostic and prognostic parameter in dogs with ethylene glycol poisoning. Dog #1 was presented within a short time after intoxication and did not display azotemia. As the dog was severely polydiptic, he may still have been compensating for his increased fluid losses at that time. It is important, however, to distinguish between early onset of azotemia which is mostly prerenal and thus potentially reversible with adequate fluid treatment, and true renal azotemia due to oliguric acute renal failure as witnessed in dogs # 2 and 3. Acute renal failure typically develops after 24–72 hours and has progressively less potential of recovery. Since the urine specific gravity may be isosthenuric as early as 3 hours post ingestion and render the differentiation between prerenal and renal azotemia almost impossible, correct assessment of the respective stage of intoxication may be difficult (Grauer et al., 1984; Dial et al., 1994). Furthermore, individual differences in progression of symptoms may exist, and knowledge of the approximate timing of ingestion is therefore a key point for prognostic recommendations (Gaynor and Dhupa, 1999a).

Echographic changes are not pathognomonic for ethylene glycol nephrosis, but they are often highly suggestive and thus supportive of diagnosis. In a study by Adams et al. (1991) investigating 12 dogs and 3 cats with oxalate nephrosis due to ethylene glycol intoxication, ultrasonographic changes varied from mild to marked increases in renal cortical echogenicity, similar to findings in dogs #2 and 3 in our case series. Medullary rim sign, as seen in dog #2, has also been described in ethylene glycol toxicity (Biller et al., 1992). Actual ingestion of ethylene glycol is frequently not observed by the owner, such as in dog #2, and the ultrasonographic picture may therefore be an important complement to the puzzle of clinical and clinicopathological findings and thus enable correct treatment decisions.

The time frame between ingestion of ethylene glycol and treatment is crucial, as irreversible damage develops rapidly. Symptomatic treatment including aggressive intravenous crystalloid fluid therapy is essential to combat severe dehydration and hypoperfusion (Gaynor and Dhupa, 1999b). Specific medical treatment aims at prevention of metabolism of ethylene glycol to its toxic metabolites by inhibiting ADH, and typically includes early and prolonged therapy with ethanol or fomepizole (4-methylpyrazole, 4-MP) (Dial et al., 1994; Connally et al., 1996; Gaynor and Dhupa, 1999b). Most dogs are expected to recover if treatment is initiated within 8 hours after ingestion (Dial et al., 1994; Connally et al., 1996). Although efficacy is reported to be superior with 4-MP compared to ethanol, the former is usually cost prohibitive for veterinary patients and may be of limited availability (Grauer et al., 1987). While 4-MP in regular doses appears to be safe, potential adverse effects of ethanol may be challenging. They include severe CNS and respiratory depression, and exacerbation of ethylene

Table 2: Clinical stages of ethylene glycol poisoning in the dog
*Rarely recognized in dogs

Stage	Clinical signs	Main laboratory changes	Timing
I	Ataxia Vomiting PU/PD Hypothermia	Hyperosmolality, high osmolar gap High anion gap Normochloremic metabolic acidosis ± Prerenal azotemia Calcium oxalate crystalluria	30 min–12 h
II	Cardiopulmonary manifestations*		12–24 h
III	Acute kidney injury or oligo-anuric acute renal failure	Renal azotemia Hyperkalemia	within 24–72 h

glycol-induced serum hyperosmolality and osmotic diuresis (Grauer et al., 1987).

Alternatively, within about 8 hours of ingestion, ethylene glycol and its toxic metabolites can be effectively removed with blood purification techniques such as hemodialysis or peritoneal dialysis, considered the treatment of choice for severe intoxications in humans (Rietjens et al., 2014). Prompt, pre-azotemic dialytic therapy, as initiated in dog #1, is therefore highly likely to be curative (Rollings et al., 2004). Additional time may be gained by combining early medical treatment with ethanol before hemodialysis can be started. In addition, hemodialysis also aims at correcting ethylene glycol induced metabolic acidosis and electrolyte disturbances (Rietjens et al., 2014). Rollings et al. (2004) described 26 dogs with ethylene glycol intoxication that underwent hemodialysis treatment. All non-azotemic dogs (n=6) and 20 % of the azotemic dogs (n=20) achieved non-dialysis-dependent survival. None of the non-azotemic dogs developed azotemia at a later stage. Prognosis for renal recovery decreases significantly if animals present in true oliguric acute renal failure but differentiation from pre-renal azotemia may be challenging in these dogs (Di Bartola et al., 1985; Thrall et al., 1998). In a report on the use of hemodiaylsis for acute renal failure in 124 dogs, Francey and Cowgill (2002) described 42 dogs with ethylene glycol intoxication, of which only 5 dogs (12%) survived to become non-dialysis dependent. Based on the grave prognosis for timely renal recovery, owners of dogs # 2 and 3 elected for immediate euthanasia.

In conclusion, dogs with acute onset of CNS and gastrointestinal signs, possibly in conjunction with acute PU/PD, must raise a suspicion of ethylene glycol toxicity. Emergency treatment must be initiated within 8 hours to prevent progression to acute kidney injury. Hemodialysis as a highly effective and at that point likely curative treatment option should be considered.

Acknowledgments

The authors gratefully acknowledge the assistance of the staff involved as well as the referring veterinarians, and the Robmar Foundation for their support of the NephroBern program.

References

Adams W. H., Toal R. L., Breider M. A.: Ultrasonographic findings in dogs and cats with oxalate nephrosis attributed to ethylene glycol intoxication: 15 cases (1984–1988). J. Am. Vet. Med. Assoc. 1991, 199: 492–496.

Biller D. S., Bradley G. A., Partington B. P.: Renal medullary rim sign: ultrasonographic evidence of renal disease. Vet. Radiol. Ultrasound 1992, 33: 286–290.

Claus M. A., Jandrey K. E., Poppenga R. H.: Propylene glycol intoxication in a dog. J. Vet. Emerg. Crit. Care 2011, 21: 679–683.

Connally H. E., Thrall M. A., Forney S. D., Grauer G. F., Hamar D. W.: Safety and efficacy of 4-methylpyrazole for treatment of suspected or confirmed ethylene glycol intoxication in dogs: 107 cases (1983–1995). J. Am. Vet. Med. Assoc. 1996, 209: 1880–1883.

Dial S. M., Thrall M. A., Hamar D. W.: Efficacy of 4-methylpyrazole for treatment of ethylene glycol intoxication in dogs. Am. J. Vet. Res. 1994, 55: 1762–1770.

DiBartola S. P., Chew D. J., Tarr M. J., Sams R. A.: Hemodialysis of a dog with acute renal failure. J. Am. Vet. Med. Assoc. 1985, 186: 1323–1326.

Doty R. L., Dziewit J. A., Marshall D. A.: Antifreeze ingestion by dogs and rats: influence of stimulus concentration. Can. Vet. J. 2006, 47: 363–365.

Francey T., Cowgill L. D.: Use of hemodialysis for the management of acute renal failure in the dog: 124 cases (1990–2001). J. Vet. Intern. Med. 2002,16: 352.

Gaynor A. R., Dhupa N.: Acute ethylene glycol intoxication. Part I. Pathophysiology and clinical stages. Compend. Contin. Educ. Pract. Vet. 1999a, 21: 1014–1023.

Gaynor A. R., Dhupa N.: Acute ethylene glycol intoxication. Part II. Diagnosis, treatment, prognosis, and prevention. Compend. Contin. Educ. Pract. Vet. 1999b, 21: 1124–1133.

Goicoa A., Barreiro A., Peña M. L., Espino L., Pérez-López M.: Atypical presentation of long-term ethylene glycol poisoning in a German shepherd dog. Vet. Hum. Toxicol. 2003, 45: 207–209.

Grauer G. F., Thrall M. A., Henre B. A., Grauer R. M., Hamar D. W.: Early clinicopathologic findings in dogs ingesting ethylene glycol. Am. J. Vet. Res. 1984, 45: 2299–2303.

Grauer G. F., Thrall M. A., Henre B. A., Hjelle J. J.: Comparison of the effects of ethanol and 4-methylpyrazole on the pharmacokinetics and toxicity of ethylene glycol in the dog. Toxicol. Lett. 1987, 35: 307–314.

Khan S. A., Schell M. M., Trammel H. L., Hansen S. R., Knight M. W.: Ethylene glycol exposures managed by the ASPCA National Animal Poison Control Center from July 1995 to December 1997. Vet. Hum. Toxicol. 1999, 41: 403–406.

Keller N., Goddard A.: First report of suspected ethylene glycol poisoning in 2 dogs in South Africa. J. S. Afr. Vet. Assoc. 2005, 76: 116–119.

Kupper J., Meng L., Baumann D., Walser K., Walser M., Del Chicca F., Naegeli H.: Chemical risk after flooding. Schweiz Arch Tierheilkd. 2011, 153, 411–414.

Mueller D. H.: Epidemiologic considerations of ethylene glycol intoxication in small animals. Vet. Hum. Toxicol. 1982, 24: 21–24.

Rietjens S. J., de Lange D. W., Meuenbelt J.: Ethylene glycol or methanol intoxication: which antidote should be used, fomepizole or ethanol? Neth. J. Med. 2014, 72: 73–79.

Rollings C. E., Francey T., Cowgill L. D.: Use of hemodialysis in uremic and non-uremic dogs with ethylene glycol toxicity. J. Vet. Intern. Med. 2004, 18: 416.

Rowland J.: Incidence of ethylene glycol intoxication in dogs and cats seen at Colorado State University Veterinary Teaching Hospital. Vet. Hum. Toxicol. 1987, 29: 41–44.

Silverstein D. C., Hopper K.: Small Animal Critical Care Medicine. Ed. Saunders Elsevier, St.Louis, 2009.

Thrall M. A., Connally H. E., Dial S. M., Grauer G. F.: Advances in therapy for antifreeze poisoning. Calif. Vet. 1998; 52: 18–22.

Corresponding author

Dr. med. vet. Ariane Schweighauser, DACVIM, DECVIM-CA
Departement für klinische Veterinärmedizin, Kleintierklinik
Länggassstrasse 128
3012 Bern
Phone +41 31 631 2629
Fax +41 31 631 2275
E-Mail: ariane.schweighauser@vetsuisse.unibe.ch