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Clinical Report

Successful Treatment of Desert Rose (*Adenium obesum*) Toxicosis in a Blue and Gold Macaw (*Ara ararauna*)

April Romagnano, Kian Martin, Scott Martin, Elliott Leonard, Stephanie Monteith, Lisa Smith, Carolyn Cray, and Laura Stern

Abstract: Desert rose (*Adenium obesum*) is a plant from the family Apocynaceace that can cause cardiac glycoside toxicosis if ingested. A 33-year-old male blue and gold macaw (*Ara ararauna*) ate a desert rose flower from an indoor houseplant. After ingestion, the patient fell onto the floor, regained its balance, and then ran to its owners who witnessed the patient "foaming at the mouth" with its "cheeks turning blue." The macaw proceeded to collapse, continued to foam at the mouth, and had what appeared to be a seizure. Upon presentation 2 hours postexposure, the macaw was moribund, in shock, and recumbent with rigid limbs and had dark blue cheeks and severe bradycardia (40 beats per minute). The bird was placed in flow-by oxygen in a warm incubator and, shortly thereafter, began regurgitating frank blood and passing melena. The patient's treatment protocol included atropine, parenteral fluids, dextrose, calcium-gluconate, vitamins, anti-inflammatory therapy, antibiotics, and activated charcoal gavage. The initial plasma biochemistry panel revealed a severe hyperkalemia (>10 mmol/L) that was treated with furosemide. Diagnostic testing and supportive treatment continued for 12 days, including hyperbaric oxygen therapy. Continuous incubator flow-by oxygen coupled with multiple hyperbaric oxygen treatments significantly improved perfusion, facilitating recovery. Three years following exposure to the desert rose flower, the owners consider the bird to have normal behavior and no clinical signs associated with the toxicosis.

Key words: toxicosis, desert rose, *Adenium obesum*, hyperbaric oxygen, avian, blue and gold macaw, *Ara ararauna*

CLINICAL REPORT

A 33-year-old male blue and gold macaw (*Ara ararauna*) was seen by its caretakers near a desert rose (*Adenium obesum*) house plant. The macaw fell onto the floor and ran to its owners "foaming at the mouth" followed by its "cheeks turning blue." Shortly thereafter, the macaw seized, collapsed, and became moribund. The owners found that the single pink and burgundy flower and pedicle was missing from their desert rose plant and noted pieces of the flower stuck to the macaw's beak and in the oral foam he produced.

The macaw was presented 2 hours postingestion laterally recumbent, moribund with rigid limbs, dehydrated, with eyes closed bilaterally, with pale mucous membranes and dark blue facial patches, and with a severe bradycardia (40 beats per minute). A weight and blood sample were obtained, following which the patient was immediately placed in a warm incubator with flow-by oxygen. The oxygen regulator was kept at 4 L/min and raised temporarily for 5 to 10 minutes to 8 L/min before and after subsequent handling. Over the next few hours, the patient was intermittently foaming from the oral cavity, regurgitating frank blood, and developed melena. These signs continued for 48 hours following presentation.

Initial treatment included atropine sulfate (0.02 mg/kg IM PRN; Covetrus North America, Dublin, OH, USA) for bradycardia, parenteral fluids 0.9% NaCL (50 mL/kg SQ q24hr × 12 days; ICU Medical, Lake

From the Avian & Exotic Clinic of Palm City, 1508 SW Mapp Rd, Palm City, FL 34990, USA (Romagnano, K. Martin, S. Martin, Leonard, Monteith, Smith); Martin Animal Clinic, 1516 SW Mapp Rd, Palm City, FL 34990, USA (Romagnano, K. Martin, S. Martin); Division of Comparative Pathology, Department of Pathology & Laboratory Medicine, University of Miami, Miami, FL 33136, USA (Cray); and the ASPCA Animal Poison Control Center, 1806A Fox Dr, Champaign, IL 61820, USA (Stern).

Corresponding Author: April Romagnano, draprilr@aol.com

Forest, IL, USA), 50% dextrose (1 mL/kg SQ q12h × 12 days; Covetrus North America), 23% calcium-gluconate (100 mg/kg SQ q12h × 12 days; Covetrus North America), dexamethasone sodium phosphate (4 mg/kg SQ once; Dexium-SP, Bimedia, Le Sueur, MN, USA), ceftazidime (75 mg/kg SQ q12h × 12 days; Sagent, Schaumberg, IL, USA), and activated charcoal-kaolin suspension by gavage (1 g/kg by gavage once; Toxiban, Lloyd, Shenandoah, IA, USA).

Initial biochemistry testing (Chem17 + Lytes4, Catalyst Machine, IDEXX, Westbrook, ME, USA) showed a severe hyperkalemia (>10 mmol/L, reference interval: 2–5 mmol/L) that was treated with furosemide (1 mg/kg SQ q24h × 5 days; Covetrus North America).

An electrocardiogram (Institute of Veterinary Specialists Telemedicine, Gainesville, FL, USA) performed on day 1 after the previously described initial parenteral treatments was considered normal even though a severe sinus bradycardia was auscultated on presentation and intermittently, to a lesser extent, throughout the first half of the 12-day hospitalization. Vital parameters, including heart rate and rhythm, were monitored during handling of the patient before its first morning feeding; during treatments, body weight measurement, and subsequent feedings; and when diagnostic tests were performed. This was done to reduce handling and stress. Radiographic imaging and an echocardiogram were recommended but declined by the owners.

Over the first 7 hours of treatment, the macaw remained recumbent, but its mucous membranes became less pale, it opened its eyes, and it progressed from moribund to minimally responsive; however, its facial patches remained blue. Hyperbaric oxygen treatments (HBOT; Hyperbaric Veterinary Medicine, Lake Worth, FL, USA) were initiated 7 hours after arrival on day 1 with 3 45-minute treatments at 2 atmospheres spaced 4 hours apart. These initial 3 HBOTs resulted in significant clinical improvement. During the first HBOT, the macaw went from lateral recumbency to sternal recumbency; it was up and standing during the second HBOT and taking steps during the third HBOT. During each HBOT, its facial patches turned from dark blue to white and then returned to decreasing shades of blue. The macaw, initially anorexic, began to show interest in food after the first HBOT and started eating after the third. Two additional HBOTs were performed: 1 on day 2 and another on day 8. Over time, the macaw's facial patches were less blue outside the chamber and finally turned back to white after the fifth and final HBOT.

Further supportive treatment, nutritional support, and diagnostics continued for the entire hospital stay of 12 days. Sucralfate (100 mg/kg PO q12h × 12 days; Nostrum Laboratories, Kansas City, MO, USA), milk thistle in lactulose (0.5 mL/kg PO q12h × 191 days; 1.5 mL milk thistle, Nature's Answer, Hauppauge, NY, USA + 15 mL Enulose, Teva Pharmaceuticals, Parsippany, NJ, USA), and enrofloxacin water 0.005% (32.2 mg/mL suspension diluted to 0.05 mg/mL PO ad lib × 12 days; MEDSforVETS, Sandy, UT, USA) were given daily. Nutritional support included tube-feeding EmerAid IC Omnivore (10 mL/kg q12h; EmerAid, Cornell, IL, USA). At discharge, treatments included oral enrofloxacin (15 mg/kg q12h × 32 days; Baytril, Elanco, Shawnee, KS, USA) and milk thistle suspended in lactulose (0.5 mL/kg q12 h × 191 days; 1.5 ml milk thistle, Nature's Answer + 15 mL Enulose, Teva Pharmaceuticals).

Blood was collected and tested on days 1, 5, and 12 of hospitalization as well as on days 32 and 191 at the recheck examinations. The initial biochemistry testing (see previous results) was performed in house, but later biochemical testing was done with an Ortho Vitros 5600 analyzer (Ortho-Clinical Diagnostics, Rochester, NY, USA) at the reference laboratory (University of Miami, Miami, FL, USA). Whereas analytical methods may vary, reference intervals from the University of Miami are presented (Table 1). The white blood cell count was determined with the hemacytometer method and phloxine stain. The differential was performed with Wright Giemsa-stained smears and reviewed at 1000×. On day 1, a severe hyperkalemia (>10 mmol/L) was present; however, it could not be further quantified because it was higher than the dynamic range of the analyzer (Table 1). Normal results were obtained by day 5 (2.6 mmol/L), but a refractory increase was observed on day 12 (6.5 mmol/L) (Table 1). Also, on day 5, the creatine phosphokinase (CPK) activities were over the dynamic range (1600 U/L) of the analyzer, but decreasing activities were noted by day 12 (826 U/L) (Table 1). The elevation in CPK activity was observed in tandem with an elevated aspartate aminotransferase (Table 1). On day 1, there was a moderate increase in glucose (346 g/dL) and total protein (5.9 g/dL) (Table 1) although the protein fractions determined by electrophoresis remained normal throughout the evaluation period (Table 1).

A moderate elevation in total white blood cell count was present on day 5 ($31.0 \times 10^3/\mu l$) with the presence of 1+ toxic heterophils and immature heterophils ($4.3 \times 10^3/\mu l$) (Table 1). The latter decreased

Table 1. Summary of the clinical pathology test results for a 33-year-old male blue and gold macaw (*Ara ararauna*) that was presented following desert rose (*Adenium obesum*) ingestion. The blood testing was done over 12 days of hospitalization and 2 follow-up revisits (days 32 and 191).

Parameter	Day 1	Day 5	Day 12	Day 32	Day 191	Reference interval[d]
Potassium (mmol/L)	>10[a]	2.6	6.5	3.3	2.1	2.0–5.0
WBC ($\times 10^3$/μl)	nd[b]	31.0	8.4	12.7	10.4	6.0–16.4
PCV (%)	25	30	30	47	47	40–51
Polych/100RBC[c]	nd[b]	14	34	5	4	1–5
AST (U/L)	nd[b]	8860	1455	118	115	105–324
CPK (U/L)	nd[b]	>1600[a]	826	206	363	101–300
Glucose (mg/dL)	346	223	249	289	271	228–325
Total protein (g/dL)	5.9	3.1	3.5	4.4	4.3	2.6–5.0
GGT (U/L)	3	15	10	<10	<10	1–30
Cholesterol (mg/dL)	302	207	371	534	264	100–390
Bile acids (μmol/L)	nd[b]	16.1	33.7	26.6	27.0	8–60

Abbreviations: WBC, white blood cell count; PCV, packed cell volume; Polych, polychromasia; AST, aspartate aminotransferase; CPK, creatine phosphokinase; GGT, gamma-glutamyl transferase; nd, not done.

[a] Greater than dynamic range of analyzer.

[b] Not done.

[c] Number of polychromatic red blood cells per 100 red blood cells.

[d] Reference interval (University of Miami Avian & Wildlife Laboratory, Miami, FL, USA).

to 0.25 $\times$ 10^3/μl by day 12. Concomitant to the low packed cell volume, a significant number of polychromatic red blood cells (RBC) were observed on days 5 (14/100 RBC) and 12 (34/100 RBC) with normalization of both by day 32 (5/100 RBC).

Aerobic choanal and cloaca cultures were submitted on day 32 at the macaw's first recheck examination. Bacterial culture was elected at this time due to the extreme stress associated with the toxic condition and recovery. No pathogenic bacteria were isolated from the choanal culture but a light growth of *Pseudomonas aeruginosa* was identified from the cloacal swab. The *P aeruginosa* organism was susceptible to ceftazidime (75 mg/kg IM once) and enrofloxacin (15 mg/kg IM once; Norbrook Laboratories, Newry, Northern Ireland, UK). At the time of this first recheck examination, the macaw was clinically normal except for a moderately elevated cholesterol (534 mg/dL) (Table 1). The patient was continued on milk thistle suspended in lactulose (0.5 mL/kg q 12h $\times$ 191 days) until the second recheck examination. Daily probiotics (0.5 mL/kg; Bene Bac Bird and Reptile, Pet-Ag, Hampshire, IL, USA) were prescribed at this time. On day 191, a second recheck examination was done, and the macaw was again clinically normal.

DISCUSSION

Desert rose, a member of the dogbane family (*Apocynaceace*), contains more than 30 cardiac glycosides, most notably oleandrigenin beta-gentiobiosyl-beta-D-thevetoside.[1–5] The entire plant contains cardiac glycosides.[2–5] It is so potent that its sap has been used to create poison arrows to hunt large game in Africa.[4] Desert rose has been reported to be potentially toxic to dogs and cats due to the presence of cardiac glycosides though no case report of desert rose toxicosis was identified in a thorough literature search.[6] This case describes desert rose toxicosis and successful treatment in a psittacine bird.

Clinical signs and biochemical abnormalities associated with desert rose toxicosis are similar to other cardiac glycoside–containing plants and cause arrhythmias and lesions in heart tissue.[2–5,7–9] Cardiac glycoside extracts have historical and evolving medicinal applications, including antineoplastic potential.[2–4] Cardiac glycosides affect the heart by binding to and inhibiting the myocardial form of the Na+/K+ ATPase enzyme that pumps sodium ions into the bloodstream and potassium ions into the cardiomyocyte.[2,3,5,9] Under these conditions, excess intracellular sodium is exchanged for calcium. The resultant high myocardial calcium increases myocardial contractility.[2,3,5,9] This disruption of the normal sodium/potassium exchange also causes hyperkalemia.[5,9] Cardiac glycosides have direct effects (positive inotrope and negative chronotrope resulting in fewer, more forceful contractions) and indirect effects (hyperkalemia resulting in bradycardia) on the myocardium.

Cardiac glycosides are, thus, associated with cardiovascular toxicity, resulting in both bradyarrhythmias and tachyarrhythmias as well as lesions in the heart tissue.[2,4,5,9] Other clinical signs reported in the

Table 2. Clinical signs for dogs, cats, and birds following exposure to desert rose (*Adenium obesum*) reported to the American Society for the Prevention of Cruelty to Animals Poison Control Center from 2010 to 2024.

Clinical Signs	Dogs (n = 128)	Cats (n = 307)	Birds (n = 13)
Gastrointestinal Signs			
Vomiting	33 (25.8%)	43 (14%)	0
Hypersalivation	89 (69.5%)	281 (91.5%)	0
Regurgitation	0	0	6 (46.2%)
Diarrhea	11 (8.6%)	2 (0.7%)	2 (15.4%)
Cardiac Signs			
Bradycardia	1 (0.8%)	3 (1%)	3 (23.1%)
Neurologic Signs			
Lethargy	10 (7.8%)	7 (2.3%)	9 (69.2%)

literature include nausea, salivation, vomiting or regurgitation, abdominal pain, dyspnea, ataxia, hemiplegia, and mydriasis.[2,4,5] Hypoglycemia has been described in an oleander (*Nerium oleander*) cardiac glycoside toxicosis in a dog and is thought to have been attributed to both inhibition of alpha-glucosidase and the inhibition of the Na+/K+ ATPase enzyme causing a decrease in the sodium gradient available for glucose transport.[2,10] Onset of clinical signs can occur up to 6 hours postingestion.[5] Enterohepatic recycling of the toxin also occurs, warranting hospitalization and monitoring of vital parameters, including potassium concentrations.[2,5,9]

Because case reports regarding desert rose toxicosis are scarce for any animal species, data from cases that were established with the American Society for the Prevention of Cruelty to Animals (ASPCA) Poison Center were obtained for review (January 2005 to December 2024). This included cases with a history consistent with exposure to desert rose and no other agents. These cases were obtained from the ASPCA Poison Center AnTox program. Cases within the database were selected via a nonstratified collection method with cases assessed as either a medium or high likelihood of desert rose causing the reported clinical signs for the case. Clinical signs reported in 10% or more of cases are listed in Table 2. There were 13 unique cases of birds being exposed to desert rose that fit the selection criteria. The data regarding the macaw from the present case was excluded. The most common clinical signs reported in birds were lethargy 9/13 (69.2%); regurgitation 6/13 (46.2%); bradycardia, depression, and weakness 3/13 (23.1%); and ataxia, diarrhea, and retching 2/13 (15.4%). There were 307 unique desert rose cases in cats that fit the selection criteria. The most common clinical signs reported in cats were hypersalivation 281/307 (91.5%) and vomiting 43/307 (14%). There were also 128 unique desert rose cases in dogs that fit the selection criteria. The most common clinical signs reported in dogs were hypersalivation 89/128 (69.5%), vomiting 33/128 (25.8%), and head shaking 15/128 (11.7%). Signs of cardiotoxicity were rare in cats and dogs.

Whereas gastrointestinal signs were common in dogs and cats that ingested desert rose, more severe signs, including cardiovascular signs, were reported in birds. This is perhaps due to a unique sensitivity in birds but may also be due to a bird's small size in relation to dogs and cats. A similar exposure to a flower would be expected to result in a much higher dosage of cardiac glycosides per kilogram of body weight in birds. Whereas the macaw and other birds with reported toxicity experienced more severe signs than reported for dogs and cats, the signs seen in birds were consistent with toxicosis reported from other plants that produce cardiac glycosides, including marked gastrointestinal signs (eg, foaming from the oral cavity, regurgitation of frank blood, melena, loose feces) over the initial 48 hours. This macaw also experienced hyperkalemia, resulting in a severe bradycardia and sinus bradycardia arrythmia.[11] The heart rate measured at presentation (40 beats per minute) likely contributed to the cyanosis identified on the physical examination. As the patient's oxygen concentrations likely further decreased, the bird seized, collapsed, and became moribund 2 hours before presentation. The macaw also had elevated CPK activity, which was likely associated with the clinical conditions of bradycardia, arrythmia, cyanosis, rigid limbs, history of seizure activity, and the state of shock. Supportive care, including a flow-by oxygen incubator and HBOT, appeared to significantly improve perfusion and facilitate recovery.

The initial potassium concentration was markedly elevated, but by day 5 it was within the reference interval. However, on day 12, it had again increased to hyperkalemic concentrations. The cause for this second elevation in potassium is unknown and potentially spurious. Fortunately, on days 32 and 191, potassium concentrations had returned to normal.

Anemia was noted from days 1–12. Whereas the initial loss of blood through the gastrointestinal tract explains the moderate reduction in the packed cell volume to 25%, a vigorous regenerative response was observed within the early period of recovery (days 5–

12). To the authors' knowledge, anemia has not been previously described in any desert rose toxicosis cases.

Other notable blood parameters that were abnormal on day 1 included glucose and TP concentrations. Notably, the hyperglycemia was noted prior to the administration of parenteral treatments, including dextrose and dexamethasone. It is possible that the hyperglycemia was secondary to stress and not a direct effect of the cardiac glycosides.

Treatments in this case included atropine, furosemide, dexamethasone sodium phosphate, subcutaneous fluids, calcium gluconate, gavage feeding, flow-by oxygen incubator therapy, and HBOT. Atropine was administered to improve atrioventricular conduction because it increases the heart rate by blocking parasympathetic pathways.[3,5,7] Furosemide was given to treat hyperkalemia because it has a direct effect on driving potassium intracellularly.[5,7] Dexamethasone sodium phosphate was only administered once on day 1 to also help decrease potassium through renal and gastrointestinal excretion and by driving it intracellularly; however, its efficacy in psittacine birds is unknown.[2,3,5,7]

Many acute cardiac glycoside toxicity treatments used in humans are not generally applied to veterinary patients because research is limited or not available, including antidigoxin Fab (digoxin-specific antibody). This compound has a high affinity for digoxin and removes it from the Na+/K+ ATPase enzyme, thus rapidly reducing toxicity and making it particularly helpful in acute toxicity cases. However, patients must be monitored for the development of severe hypokalemia and/or anaphylaxis if the patient is allergic to sheep, papain, bromelain, or papaya.[5,12] Antidigoxin Fab was not available for this case, nor is it known to be safe in psittacine birds. This compound has been used to treat a dog and a cat with oleander toxicosis.[13,14] Per anecdotal evidence from the ASPCA Poison Center, antidigoxin Fab was used to treat an eclectus parrot (*Eclectus roratus*) exhibiting cardiotoxic signs from desert rose that reportedly responded favorably with a full recovery. Insulin-dextrose is often used in the treatment of hyperkalemia in mammals, but in the first author's (AR) opinion, that is neither safe nor recommended in glucagon-dependent psittacine birds.[5] Another consideration was the use of cholestyramine, a bile acid sequestrant that binds bile acids and then prevents the reabsorption of bile and any associated cardiac glycosides that had been excreted in the bile through enterohepatic circulation.[5,15] No studies regarding the use of cholestyramine are available in avian species.[5] There is a potential mechanism for reducing cardiac glycoside concentrations with the administration of cholestyramine following exposure to other cardiac glycoside containing plants; however, it is unknown whether cardiac glycosides from desert rose undergo enterohepatic recirculation in birds. Hence, cholestyramine was not administered in this case due to the need for fasting prior to administration and a lack of proven safety and efficacy in birds.

Of the above listed treatments used in this case, exogenous calcium may be considered controversial.[2,3,16] Generally, calcium is thought to be cardiac sparing in cases of heart conditions.[3,16] However, given the mechanism of action for cardiac glycoside toxicosis, exogenously administered calcium is generally considered to be contraindicated for the treatment of desert rose toxicosis. Because the macaw responded very well to fluid therapy, calcium gluconate, atropine, and furosemide, the more aggressive treatments, such as digoxin-specific antibody fragments and insulin-dextrose therapy, were ultimately not pursued.

It is the first author's opinion (AR) that the constant flow-by oxygen in an incubated environment, coupled with HBOT, significantly improved perfusion and, thus, facilitated recovery of this macaw safely.[17–20] A study in guinea pigs showed that HBOT was cardioprotective at 1, 2, and 3 atmospheres when ouabain, a plant-derived cardiac glycoside, was administered. The proposed protective mechanism was thought to be increased dissolved oxygen in the blood.[21] Hyperbaric oxygen treatment delivers oxygen to the alveoli under increased atmospheric pressure, resulting in large increases of dissolved oxygen. This increases oxygen saturation to >110% of normal for 3 hours posttreatment and increases peripheral and cerebral oxygen content 10 times.[19] Over the past 15 years, HBOT has been used in our clinics for treatment of hypoxia from respiratory compromise due to infection, trauma, or toxins in thousands of psittacine birds with consistent positive results. Hyperbaric oxygen therapy has been shown to hasten tissue repair, decrease pain, increase appetite, and potentiate fluoroquinolone and anti-inflammatory drugs.[17–20] This theory is derived from this macaw's improving clinical status throughout hospitalization, characterized by its decreasing cyanosis by which the blue in its facial patches decreased and turned a more normal shade of white, and improvements in its activity, appetite, drinking, and vocalizations. The macaw has been reexamined numerous times over the past 3 years and

has not had any long-term sequalae to the desert rose toxicosis.

REFERENCES

1. Yamauchi T, Abe F. Cardiac glycosides and pregnanes from *Adenium obesum* (studies on the constituents of Adenium I.). *Chem Pharm Bull (Tokyo)*. 1990;38:669–672.
2. Siroka Z. Toxicity of house plants to pet animals. *Toxins*. 2023;15:346.
3. Oerther SE. Plant poisonings: common plants that contain cardiac glycosides. *J Emerg Nurs*. 2010;37:102–103.
4. Alanazi KM, Ali MA, Kim SY, et al. The cp genome characterization of *Adenium obesum*: gene content, repeat organization and phylogeny. *Saudi J Biol Sci*. 2021;28:3768–3775.
5. Roberts DM, Gallapatthy G, Dunuwille A, et al. Pharmacological treatment of cardiac glycoside poisoning. *Br J Clin Pharmocol*. 2015;81:488–495.
6. Barr CA. Household and garden plants. In: Peterson ME, Talcott PA, eds. *Small Animal Toxicology*; St. Louis, MO. Elsevier; 2013: 345–410.
7. Fitzgerald BC. Cardiovascular disease in pet birds: therapeutic options and challenges. *Vet Clin Exot Anim Pract*. 2022:25:469–501.
8. Burrows GE, Tyrl RJ. *Toxic Plants of North America*. 2nd ed. Wiley Blackwell; 2013.
9. Botelho AFM, Pierezan F, Soto-Blanco B, et al. A review of cardiac glycosides: structure, toxicokinetics, clinical signs, diagnosis and antineoplastic potential. *Toxicon*. 2018;158:63–68.
10. Page C, Murtaugh RJ. Hypoglycemia associated with oleander toxicity in a dog. *J Med Toxic.* 2014;11:141–143.
11. Strunk A, Wilson H. Avian cardiology. *Vet Clin North Am Exot Anim Pract.* 2003;6:1–28.
12. Plumb DC. Digoxin. *Plumb's Veterinary Drugs*. https://plumbs.com/. Accessed May 1, 2023.
13. Pao-Franco A, Hammond TN, Weatherton LK, et al. Successful use of digoxin-specific immune Fab in the treatment of severe *Nerium oleander* toxicosis in a dog. *J Vet Emerg Crit Care.* 2017;27:596–604.
14. Galton AF, Granfone MC, Caldwell DJ. Digoxin-specific antibody fragments for the treatment of suspected *Nerium oleander* toxicosis in a cat. *JFMS Open Rep*. 2020;6:2055116920969599.
15. ASPCA Pro. What to know about cholestyramine resin and pets. https://www.aspcapro.org/resource/what-know-about-cholestyramine-resin-and-pets. Accessed May 1, 2023.
16. Hack JB, Woody JH, Lewis DE, et al. The effect of calcium chloride in treating hyperkalemia due to acute digoxin toxicity in a porcine model. *J Toxico Clin Toxicol*. 2004;42:337–342.
17. Edwards ML. Hyperbaric oxygen therapy. Part 1: history and principles. *J Vet Emerg Crit Care.* 2010;20:284–288.
18. Edwards ML. Hyperbaric oxygen therapy. Part 2: application in disease. *J Vet Emerg Crit Care.* 2010;20:289–297.
19. Romagnano A, Cray C, Phillips L. Extensive eye and face trauma: treated successfully with hyperbaric oxygen therapy post-enucleation. *Proc AAV.* 2017:139.
20. Duke AM, Woodward MC, Tully TN, et al. Evaluation of the safety of hyperbaric oxygen therapy in domestic chickens (*Gallus gallus domesticus*). *Proc ExoticsCon.* 2021:13.
21. Bachand RT, Somani P. Digitalis intoxication: protection with hyperbaric oxygen. *Life Sciences.* 1967;6:739–742.