



Case Report

A case report of hemodialysis management of acute kidney injury in a Russell's viper-venomened dog

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Abstract

A four-year-old Russell's viper-venomened dog presented with severe azotemia and was diagnosed with acute kidney injury (AKI). Russell's viper envenomation-induced AKI is highly fatal and medical treatment alone was insufficient. In the current case, intermittent hemodialysis (IHD) was implemented and was found to be highly efficient in removing uremic toxins from the bloodstream of the venomened dog. After two dialysis sessions, the patient's renal function levels were markedly improved and clinical signs at presentation were resolved. This case report describes the medical treatment and detailed IHD management in a Russell's viper-venomened AKI dog for veterinarians who consider IHD a suitable therapy option for their patients.

Keywords: Acute kidney injury, Dog, Intermittent hemodialysis, Russell's viper

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INTRODUCTION

Acute kidney injury (AKI) is a fatal and common diagnosis in veterinary practice with high morbidity and mortality rates of approximately 50% (Dunaevich et al., 2020; Rimer et al., 2022). It is defined by sudden damage to the renal parenchyma due to a rapid decline in the glomerular filtration rate (GFR), resulting in the retention and elevation of levels of blood urea nitrogen (BUN) and serum creatinine, dysregulation of fluid, electrolytes (mostly hyperkalemia), and acid-base homeostasis (mostly metabolic acidosis), as well as reduced urine output (UOP). The most common etiologies of AKI consist of ischemic/inflammatory (58%), infectious (8%), nephrotoxicosis (including envenomation, 6%), and others (5%) (Rimer et al., 2022).

Eastern Russell's viper (*Daboia siamensis*) is a WHO category 1 medically-important venomous snake commonly found in countries throughout Southeast Asia, including Thailand (Warrell, 2010). It is potentially life-threatening and causes AKI. Although its pathogenesis has yet to be fully established, it appears to involve microvascular fibrin deposition, direct nephrotoxicity, and cardiovascular impairment (Gopalakrishnan, 2019).

On its own, medical management is usually insufficient against Russell's viper envenomation-induced AKI. Therefore, renal replacement therapy (RRT) should also be implemented. Intermittent hemodialysis (IHD) is an extracorporeal renal replacement technique for hemofiltration that occurs outside the patient's body via a hemodialysis machine which removes uremic toxins from the bloodstream through a semipermeable membrane in sporadic cycles. IHD is indicated for uremic patients with AKI, toxicosis and/or electrolyte imbalance. After IHD therapy, adequate renal function usually returns within 2-4 weeks after the first dialysis cycle (Sykes et al., 2011).

This case report describes medical treatment and detailed on IHD management in a Russell's viper-envenomed AKI dog, which can aid veterinarians who consider IHD a suitable therapy option for patients.

HISTORY CLINICAL DIAGNOSIS AND FINDINGS

A four-year-old neutered female American Pitbull presented at Kasetsart University Veterinary Teaching Hospital (KUVTH) Hua Hin, Thailand, with clinical signs of mild depression, vomiting, anorexia and an episode of partial seizure. According to the owner, the patient had been bitten by a Russell's viper 5 days before the hospital visit. The patient was an outdoor dog, and its blood profiles had never been assessed.

From the physical examination, the patient had a pink mucous membrane, normal hydration status, normal abdominal palpation, normal heart and lung auscultation sound. Body weight, body condition score, heart rate, respiratory rate, systolic blood pressure and temperature were 26 kg, 6/9, 104 bpm, 28 bpm, 170 mmHg, and 101.66 °F, respectively. Blood samples were collected for hematological, biochemical, blood gas analysis and coagulation assays, which revealed neutrophilic leukocytosis, severe azotemia, hypoalbuminemia, and hyperphosphatemia conditions. According to the IDEXX VetTest Chemistry Analyzer's reference ranges, the abnormal serum biochemical parameters included BUN (152.4 mg/dL), creatinine (19.07 mg/dL), phosphorus (12.8 mg/dL), and albumin (1.6 mg/dL), as shown in Table 1. Complete blood count (CBC) was frequently monitored to observe trends in the hematocrit (HCT) and

white blood cell (WBC) levels (Table 2). The Sysmex CS-2500 Coagulation Analyzer (Siemens Healthcare, Germany) showed that the patient had 15 sec of activated partial thromboplastin time (aPTT; reference range 10-25 sec) and 7.4 sec of prothrombin time (PT; reference range 6-11 sec), which were both within normal limits (Hackner and Rousseau, 2015). However, urine collected via cystocentesis was cloudy, pale yellow, had a specific gravity of 1.010, 5.0 urine pH, and 50 cells/hpf of red blood cells from a microscopic examination (Table 3). On that account, a urine sample was sent for bacterial culture and antimicrobial susceptibility testing (AST). While waiting for the AST result, 20 mg/kg of amoxicillin-clavulanic acid (R.X. Company Ltd., Thailand) was administered every 12 hrs, intravenously. One week later, the AST result was reported revealing positive culture of *E. coli* and *Pseudomonas spp.*, which sensitivity to imipenem and meropenem (Table 4). Hence, amoxicillin-clavulanic acid was replaced with imipenem and bacterial urinary tract infection was indicated. After 5 days on imipenem, another urine sample was sent for bacterial culture and AST, revealing no bacterial growth after 3 days. Thereafter, imipenem treatment was discontinued.

Table 1 Serum biochemistry analyses in a Russell's viper-envenomed dog with acute kidney injury before and after intermittent sessions of hemodialysis (IHD) sessions from Day 0-71.

Day	BUN	Creatinine	Phosphorus	Albumin	Potassium
Reference range*	7-27 mg/dl	0.5-1.8 mg/dl	2.5-6.8 mg/dl	2.3-4 gm%	3.5-5.8 mEq/L
0	152.4	19.07	12.8	1.6	3.59
2 (Pre 1 st IHD)	126	16.91	N/A	1.9	3.59
2 (Post 1 st IHD)	77	10.19	5.3	2.2	2.93
5 (Pre 2 nd IHD)	76.5	11.06	7.5	2.1	3.59
5 (Post 2 nd IHD)	39.8	6.12	4.1	2.1	2.89
7	39	6.72	6.8	N/A	3.61
9	31.3	4.68	4.9	2.3	3.21
14	12.7	2.73	3.3	2.2	N/A
29 (1 st follow-up)	9.9	1.77	2.3	2.1	N/A
71 (2 nd follow-up)	14.1	1.66	N/A	3.4	N/A

Reference ranges of serum parameters analyzed by IDEXX VetTest Chemistry Analyzer, BUN: Blood urea nitrogen, N/A: not applicable.

Table 2 Complete blood count in a Russell's viper-envenomed dog with acute kidney injury before and after intermittent sessions of hemodialysis (IHD) sessions from Day 0-71.

Day	Reference range*	Hematocrit	White blood cells	Platelets	Plasma protein
	30-45%	5.5-19 x 10 ³ /μl	200-500 x 10 ³ /μl	5.8-7.8 gm%	
0	32	26.69	323	6.6	
2	(Pre 1 st IHD)	32	22.79	327	7.0
2	(Post 1 st IHD)	33	N/A	N/A	6.8
3		29	37.48	246	7.0
4		35	N/A	N/A	7.6
5	(Pre 2 nd IHD)	38	32.75	215	7.2
5	(Post 2 nd IHD)	32	N/A	N/A	6.8
7		32	16.93	287	7.2
14		31	10.96	294	6.8
29	(1 st follow-up)	35	9.4	388	7.0
71	(2 nd follow-up)	44	11.41	309	7.4

Reference ranges used at the Laboratory of Veterinary Diagnostic Unit, Faculty of Veterinary Medicine, Kasetsart university, Thailand, N/A: not applicable

Table 3 Urinalysis of Russell's viper-envenomed dog with acute kidney injury before (Day 0) and after (Day 29) hemodialysis sessions.

Day of collection	Appearance	Sp. Gr.*	pH	Urine strip finding	Microscopic finding
Day 0	Light yellow, cloudy	1.010	5.0	Blood 3+	Rbc* 50 cells/hpf*
Day 29	Light yellow, clear	1.005	6.5	No abnormalities	No abnormalities

*Sp. Gr.: Specific gravity, Rbc: Red blood cell, hpf: high power field.

Reference ranges of parameters analyzed by ZOETIS VETSCAN UA Urine Analyzer.

Abdominal ultrasound (Xario 100MX, Canon Medical System Japan) was scanned in the dorsal recumbency position via the ventrolateral approach to assess the patient's renal morphology. The sizes of both kidneys were found to be within normal limits. However, they were markedly hyperechoic in composition compared to the liver and the spleen. Furthermore, the renal corticomedullary distinction was poor (Figures 1a and 1b), suggesting interstitial and glomerulonephritis, acute tubular nephrosis or necrosis (Barr et al., 1989; Adams et al., 1991; Forrest et al., 1998; Eubig et al., 2005).

From a compilation of the patient's history, clinical signs, laboratory test results and ultrasound findings, AKI and bacterial urinary tract infection were diagnosed.

Table 4 Sensitivity to different antibiotics of *E. coli* and *Pseudomonas* spp. isolated from urine which collected via cystocentesis in a dog with acute kidney injury and cystitis.

Antibiotic	<i>E. coli</i>	<i>Pseudomonas</i> spp.	<i>E. coli</i>	<i>Pseudomonas</i> spp.
Amoxicillin	R*	R	Doxycycline	R
Amoxicillin-clavulanic acid	R	R	Enrofloxacin	S
Azithromycin	R	R	Gentamicin	R
Ceftriaxone	R	I	Imipenem	S
Cephalexin	R	R	Marbofloxacin	S
Cephazolin	R	R	Meropenem	S
Ciprofloxacin	R	R	Norfloxacin	I
Clindamycin	R	R	Sulfa-trimethoprim	R

*S: Sensitive, I: Intermediate, R: Resistance.

According to disc diffusion clear zone diameter interpretative standards for *E. coli* ATCC

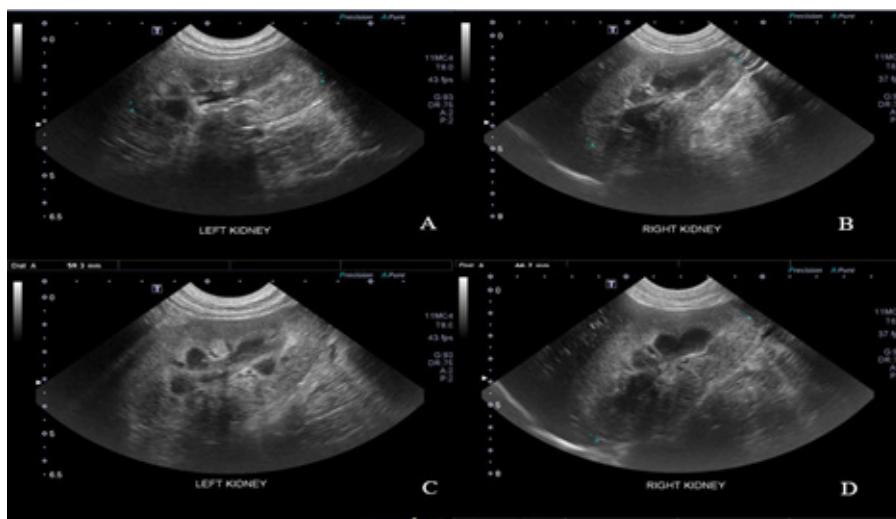


Figure 1 Longitudinal ultrasound images of the left kidney (A) and the right kidney (B) in a dog with acute kidney injury (AKI) caused by Russell's viper envenomation before intermittent hemodialysis (IHD) sessions. Ultrasound images of the left kidney (C) and the right kidney (D) in a dog with AKI caused by Russell's viper envenomation 14 days after IHD

CASE MANAGEMENT

After the initial assessment, the patient was hospitalized. Initially, urine output (UOP) was monitored every 4 hrs, which remained consistently within the normal range. The patient was treated with intravenous administration of 0.9% normal saline solution (NSS, General Hospital Products Public Co., Ltd., Thailand) at a rate of 50 mL/hr based on the calculated average insensible (respiratory) fluid loss, obtained UOPs and ongoing losses (estimated volume loss from emesis). This helped return renal perfusion to normal levels to prevent further damage to the patient's kidneys and boost the excretion of uremic toxins. After the AST was reported, imipenem (Siam Bheasach Co., Ltd., Thailand) was selected and administered every 8 hrs, intravenously, at a reduced dosage of 10 mg/kg to minimize its nephrotoxicity due to AKI. The antimicrobial dosage was adjusted according to the following formula:

$$\text{Reduced dosage} = \text{normal dosage} \times \left(\frac{\text{normal serum creatinine}}{\text{patient serum creatinine}} \right)$$

The dosage for the antimicrobial was adjusted according to the varied daily creatinine level. After 36 hrs of intensive medical treatment, the severe azotemia condition still persisted. Thus, IHD was regarded as necessary to improve the condition and reduce its complications.

To instigate IHD, an 11 Fr x 20 cm Double-lumen catheter (Amornwit Medical Co., Ltd, Thailand, [Figure 2b](#)) was placed with aseptic technique using a Seldinger technique while the patient was under general anesthesia. Due to the fact that the patient had a skin lesion on the right side of the neck, the patient was placed in the right lateral recumbent position to install the IHD access point via the left jugular vein. First, the introducer needle enters the blood vessel, the guidewire is then advanced through the lumen of the introducer needle. Once it is in place, the introducer needle is withdrawn. The dilator is then passed over the guidewire into the vessel in the direction that is most parallel to the vessel as possible, and the dilator is withdrawn. The catheter is then passed over the guidewire into the vessel. Intraoperative fluoroscopy was then used to guide the catheter along the guidewire into the junction of the cranial vena cava and the right atrium of the patient. Afterwards, the guidewire is removed. Each lumen of the catheter is then inspected to make certain that there is no blood flow obstruction.

A bolus of 1:1 dilution of 50 units/kg unfractionated heparin (GLAND PHARMA Ltd., India) and 0.9% NSS was added into the double-lumen catheter to prevent blood coagulation in the catheter. We then calculated a maintenance dose of heparin CRI at the dose of 50 units/kg/hr for a total of 1.5 hrs which is the length of the dialysis session to prevent clotting of the extracorporeal circuit. Furthermore, an esophagostomy tube was placed to ensure that the daily energy requirement was met. Human albumin transfusion was also given approximately 12 hrs before initiating the first and second IHD therapy sessions to help minimize possible complications.

The IHD was initiated by a Fresenius 4008B hemodialysis machine ([Figure 2e](#)) in 2 intermittent sessions approximately 36 hrs after the patient initial medical treatment. The hemodialysis prescription used included a pediatric blood tubing set (Kawasumi Laboratories Co., Ltd., Thailand, [Figure 2c](#)) and a cellulose triacetate membrane dialyzer (Nipro sureflux-90E, [Figure 2a](#)) in 0.9 m² surface area. Dialysate (HD Medical Co., Ltd., Thailand, [Figure 2d](#)) flow rates of 300 mL/min and 500 mL/min were used in the first and the second dialysis sessions, respectively. The blood flow rates were 6 mL/kg/min (150 ml/min) in a countercurrent direction with dialysate flow in both 90 min sessions. Ultrafiltration volume in each session was set at 0.7 L and 0.5 L, respectively. During hemodialysis, heart rate, respiratory rate, temperature, mucous membrane color, systolic blood pressure and pulse oxygen saturation level (SpO₂) were monitored every 30 min; all readings were normal throughout the dialysis treatments. Three hours following the first dialysis treatment, the patient experienced head tremors, a neurological manifestation of Dialysis disequilibrium syndrome (DDS) due to a drastic decline in serum osmolarity. Hence, 0.5 g/kg of 20% mannitol (A.N.B. Laboratories Co., Ltd., Thailand) was introduced intravenously. Sporadic episodes of head tremors resolved approximately 6 hrs later.

At the end of each session, blood samples were taken for hematological, biochemical, blood gas analysis and coagulation assays to monitor response to treatment as well as any possible complications. Moreover, dialysis efficacy was measured using the urea reduction ratio (URR) after each session following the equation below:

$$\text{URR} = (\text{BUN}_{\text{pre}} - \text{BUN}_{\text{post}}) / \text{BUN}_{\text{pre}}$$

When comparing the first postdialysis serum biochemical profile results to the predialysis assessment, the serum BUN level decreased to 77 mg/dL, creatinine decreased to 10.19 mg/dL, and phosphorus decreased to 5.3 mg/dL (Table 1), yielding the first dialysis URR of 0.39. Moreover, after the first dialysis sessions coagulation assay showed that the patient had 18.2 sec of aPTT (reference range 10-25 sec) and 8.4 sec of PT (reference range 6-11 sec), which were both within normal limits. However, hypokalemia and anemia were observed. The serum potassium level decreased from 3.59 to 2.93 mEq/L and the HCT level decreased from 32% to 29%. The conditions were corrected before resuming the second dialysis session by administering 0.3 mEq/kg/hr potassium chloride (KCL) (Atlantic Laboratories Co., Ltd., Thailand) infused into 500 mL of NSS and administered at the rate of 50 ml/hr, intravenously and fresh whole blood transfusion was also provided. Three days after the first dialysis, the second dialysis was repeated. The serum BUN level decreased to 39.8 mg/dL, while creatinine decreased to 6.12 mg/dL, and phosphorus decreased to 4.1 mg/dL, URR of 0.48 was then calculated. After the second dialysis sessions coagulation assay showed that the patient had 16 sec of aPTT (reference range 10-25 sec) and 8.4 sec of PT (reference range 6-11 sec), which were both within normal limits. In other hand, hypokalemia and anemia were also observed after the second dialysis session (Table 1 and Table 2), and a correction with KCL was provided accordingly. After 5 days of intravenous imipenem therapy, another urine sample was sent for bacterial culture and AST. The result revealed no bacterial growth after 3 days of incubation. Thereafter, intravenous imipenem therapy was discontinued. One week after the second IHD session, the serum BUN level decreased to 12.7 mg/dL, creatinine decreased to 2.73 mg/dL and phosphorus decreased to 3.3 mg/dL. Consequently, the double-lumen catheter was removed, and the abdominal ultrasound was repeated. After 16 days of hospitalization, the patient was discharged and maintained on a renal prescriptive diet. 27 days after the first dialysis session, the patient's kidney function levels returned to within normal limits. However, a second abdominal ultrasound revealed retained kidney damage (Figures 1c and Figures 1d), prompting a diagnosis of chronic kidney disease (CKD).



Figure 2 Main supplies and equipment used in hemodialysis, including, a dialyzer (Nipro sureflux-90E) in 0.9 m² surface area (A), an 11 Fr x 20 cm Double-lumen catheter (B), a pediatric blood tubing set (C), dialysates (D) and a Fresenius 4008B hemodialysis machine (E).

DISCUSSION

This is the first report on IHD for managing AKI in a Russell's viper-venomened dog. IHD, as well as peritoneal dialysis (PD) from a previous study (Poppinit and SungThong, 2021), were found to be successful and imperative in stabilizing Russell's viper envenomation-induced AKI. In IHD, blood is passed through straw-like semipermeable membranes, whereas dialysate flows around the straws in a concurrent or countercurrent direction. In contrast, PD utilizes the peritoneum as a semipermeable membrane across which fluid and uremic solutes are exchanged. To this date, there is no consensus regarding the most suitable RRT for managing AKI. Nevertheless, the elimination of toxins occurs more gradually in PD (Gallatin et al., 2005) and is only about one-eighth to one-fourth as efficient as hemodialysis (Winchester et al., 2007).

The complications of IHD include hypotension and hypovolemia, as well as problems with vascular access, and respiratory, hematologic, gastrointestinal, and neurologic complications (Elliott, 2000). Hypotension and hypovolemia make up approximately 50% of previously reported complications (Langston et al., 1997; Langston, 2002), though neither occurred in the current case. Instead, the complications observed were DDS, anemia, and hypokalemia.

As previously mentioned, IHD was chosen because it is the most effective method for lowering the level of severely elevated BUN. Even so, it causes a rapid decline in BUN level, which may lead to a fatal plunge in plasma osmolarity while the osmolarity of brain tissue remains elevated above that of

plasma. The disequilibrium causes an influx of water in plasma into the brain, leading to cerebral edema and DDS signs during or after a dialysis session, e.g. agitation, disorientation, vomiting, seizure, coma, or death (Rosen et al., 1964). To reduce the risk, the initial session should be a low-efficiency dialysis with a shorter length session, smaller dialyzer surface area, slower blood flow rates, slower dialysate flow rates, and concurrent direction of flow for blood and dialysate. One way to calculate the estimated efficiency of the dialysis session based on the severity of the BUN level is via the URR to measure a certain reduction in the BUN level (Langston et al., 2010). The recommended URR for the initial treatment is no greater than 0.1 URR per hour (Cowgill and Francey, 2012). However, the URR of the first session in this case was 0.39 per 1.5 hours (0.26 per hour), which was higher than anticipated. This was due to the limitation of the available hemodialysis machine, which has a minimum dialysate flow rate of 300 mL/min. Typically, the blood flow rate is set at half of the chosen dialysate flow rate. Hence, the calculated patient's suitable blood flow rate was 150 mL/min or 6 mL/kg/min, whereas it should have been only 1 to 2 mL/kg/min for animals with predialysis BUN levels greater than 180 mg/dL (Cowgill, 2011).

As the extracorporeal blood flows through the dialysis tube and the dialyzer unit, the coagulation cascade may be activated and induce thrombosis. Consequently, routine anticoagulation assay monitoring is required during the dialysis session (FISCHER, 2007). Unfractionated heparin is one of the most commonly used anticoagulants for IHD, which uses Automated activated clotting time (ACT) to prescribe and monitor safe heparin requirements. The constant rate infusion (CRI) of heparin is adjusted based on ACT measured every 30 to 60 mins to maintain the ACT within the desired range of 160 to 200 seconds (Langston et al., 2010). Regardless, other coagulation time assays, such as aPTT and PT, can also be used with equal reliability (Cowgill and Francey, 2012). In this case, a bolus of heparin was injected directly into the extracorporeal blood before the filter and directly followed by a CRI of heparin. aPTT and PT were then measured, although only before and after each dialysis session due to economic limitations.

According to Elliott (2000), the extracorporeal volume should not exceed 10% of the dialysis patient's blood volume to prevent problems with hypotension and hypovolemia. In the current case, the patient weighed approximately 25 kg, meaning its estimated blood volume was 2000 ml. Approximately 136 ml of the patient's blood was transported into the extracorporeal circuit (56 ml as the priming volume of the dialyzer unit and 80 ml in the blood tubing), accounting for 6.8% of the patient's blood volume, which was within the recommended limit. In addition, the patient's predialysis HCT level was within the normal range. Thus, 0.9% NSS was selected as the priming solution. After the first dialysis session, the patient's HCT level dropped to 29%. Although it may not have been a significant drop, a fresh whole blood transfusion was provided to diminish the risk of hypotension and hypovolemia. In the case of small patients weighing less than 7 kg, the blood circuit would contain more than 10% of the patient's blood volume, and it is beneficial to prime the circuit with type-matched whole blood or colloidal fluid to minimize complications (Elliott, 2000).

IHD removes potassium from the extracellular fluid compartment as a result of diffusion by 85% of dialytic potassium clearance (Locatelli et al., 2015). Hence, the rate and amount of potassium removal are largely dependent upon the serum-to-dialysate potassium gradient. Since patients that undergo dialysis usually have hyperkalemia conditions, most dialysate is designed with lower potassium concentrations (mostly 2 mEq/L or less) compared to the serum (Kovesdy et al., 2007). As the patient in this report was isokalemic before the initial dialysis, hypokalemia was observed after both dialysis sessions. Therefore, the authors recommend avoiding low-potassium dialysate (less than 2 mEq/L) in patients with known predialysis serum potassium levels less than or equal to 5 mg/dL. Furthermore, the serum potassium level should be frequently monitored to minimize the plunging of potassium during treatment. After two sessions of IHD, the patient's BUN and creatinine levels were markedly improved and all clinical signs at presentation were resolved. The patient was discharged and maintained on a renal prescriptive diet. Throughout the course of monthly follow-ups, the CBC and kidney function parameters remained stable. Nonetheless, the patient was still classified as an International Renal Interest Society (IRIS) CKD stage 2 patient.

This case provides details on IHD management in a dog with AKI, which could also be implemented in the management of other conditions, such as medically unresponsive oliguria or anuria, intoxications, life-threatening electrolyte disturbances, severe metabolic acidosis, or fluid overload.

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AUTHOR CONTRIBUTIONS

The dog patient in this case was first presented to P.T. After realizing that the patient was not responding to medical treatment, P.T. offered the option of hemodialysis (HD) to the dog owner, who later gave consent on performing the procedure. Consequently, P.T. conceived the idea of reporting the procedure protocol and findings in this case. S.C. assisted P.T. in the diagnosis of this patient via abdominal ultrasound. Both the authors then decided to collaborate together. From that point on, both P.T. and S.C. equally contributed on this case report–research; protocol drafting; patient care and monitoring; and writing the manuscript until it reached completion.

CONFLICT OF INTEREST

All authors declare no conflict of interest.

REFERENCES

Adams, L.G., Polzin, D.J., Osborne, C.A., O'Brien, T.D., 1991. Comparison of fractional excretion and 24-hour urinary excretion of sodium and potassium in clinically normal cats and cats with induced chronic renal failure. *Am. J. Vet. Res.* 52(5), 718-722.

Barr, F.J., Patteson, M.W., Lucke, V.M., Gibbs, C., 1989. Hypercalcemic nephropathy in three dogs: sonographic appearance. *Vet. Radiol. Ultrasound.* 30, 169-173.

Cowgill, L., Francey, T., 2012. Hemodialysis and extracorporeal blood purification. In: DiBartola, S.P. (Ed.), *Fluid, electrolyte and acid-base disorders in small animal practice*, (4th edition). Saunders Elsevier, St. Louis, Missouri, pp. 680-713.

Cowgill, L.D., 2011. Urea kinetics and intermittent dialysis prescription in small animals. *Vet. Clin. North. Am. Small. Anim. Pract.* 41(1), 193-225.

Dunaevich, A., Chen, H., Musseri, D., Kuzi, S., Mazaki-Tovi, M., Aroch, I., Segev, G., 2020. Acute on chronic kidney disease in dogs: Etiology, clinical and clinicopathologic findings, prognostic markers, and survival. *J. Vet. Intern. Med.* 34(6), 2507-2515.

Elliott, D.A., 2000. Hemodialysis. *Clin. Tech. Small. Anim. Pract.* 15(3), 136-148.

Eubig, P.A., Brady, M.S., Gwaltney-Brant, S.M., Khan, S.A., Mazzaferro, E.M., Morrow, C.M., 2005. Acute renal failure in dogs after the ingestion of grapes or raisins: a retrospective evaluation of 43 dogs (1992-2002). *J. Vet. Intern. Med.* 19(5), 663-674.

Fischer, K.G., 2007. Essentials of anticoagulation in hemodialysis. *Hemodial. Int.* 11, 178-189.

Forrest, L.J., O'Brien, R.T., Tremeling, M.S., Steinberg, H., Cooley, A.J., Kerlin, R.L., 1998. Sonographic renal findings in 20 dogs with leptospirosis. *Vet. Radiol. Ultrasound.* 39, 337-340.

Gallatin, L.L., Couëtil, L.L., Ash, S.R., 2005. Use of continuous-flow peritoneal dialysis for the treatment of acute renal failure in an adult horse. *J. Am. Vet. Med. Assoc.* 226(5), 756-759.

Gopalakrishnan, N., 2019. Snake envenoming-an underreported cause of acute kidney injury. *Kidney. Int. Rep.* 4(5), 643-646.

Hackner, S.G., Rousseau, A., 2015. Bleeding Disorders. In: Silverstein, D.C., Hopper, K. (Eds.), *Small Animal critical care medicine*, (2nd edition). Saunders Elsevier, St. Louis, Missouri, pp. 554-567.

International Renal Interest Society, 2023. IRIS staging of CKD. Available from: http://www.iris-kidney.com/pdf/2_IRIS_Staging_of_CKD_2023.pdf

Kovesdy, C.P., Regidor, D.L., Mehrotra, R., Jing, J., McAllister, C.J., Greenland, S., Kopple, J.D., Kalantar-Zadeh, K., 2007. Serum and dialysate potassium concentrations and survival in hemodialysis patients. *Clin. J. Am. Soc. Nephrol.* 2(5), 999-1007.

Langston, C., Poeppel, K., Mitelberg, E., 2010. *AMC dialysis handbook*, 5th edition. Animal Medical Center, New York. pp. 3.

Langston, C.E., 2002. Acute renal failure caused by lily ingestion in six cats. *J. Am. Vet. Med. Assoc.* 220(1), 49-52.

Langston, C.E., Cowgill, L.D., Spano, J.A., 1997. Applications and outcome of hemodialysis in cats: a review of 29 cases. *J. Vet. Intern. Med.* 11(6), 348-355.

Locatelli, F., La Milia, V., Violo, L., Del Vecchio, L., Di Filippo, S., 2015. Optimizing haemodialysate composition. *Clin. Kidney. J.* 8(5), 580-589.

Poppinit, T., Sungthong, C., 2021. A case report of peritoneal dialysis for management of acute kidney injury caused by Russell's viper envenomation in a dog. *Vet. Integr. Sci.* 20(1), 1-12.

Rimer, D., Chen, H., Bar-Nathan, M., Segev, G., 2022. Acute kidney injury in dogs: Etiology, clinical and clinicopathologic findings, prognostic markers, and outcome. *J. Vet. Intern. Med.* 36(2), 609-618.

Rosen, S., O'connor, K., Shaldon, S., 1964. Haemodialysis disequilibrium. *Br. Med. J.* 2(5410), 672-675.

Sykes, J., Hartmann, K., Lunn, K., Moore, G., Stoddard, R., Goldstein, R., 2011. 2010 ACVIM small animal consensus statement on leptospirosis: diagnosis, epidemiology, treatment, and prevention. *J. Vet. Intern. Med.* 25(1), 1-13.

Warrell, D.A., 2010. Guidelines for the management of snake-bites. Available online: <https://apps.who.int/iris/rest/bitstreams/1140945/retrieve>

Winchester, J., Boldur, A., Oleru, C., Kitiyakara, C., 2007. Use of dialysis and hemoperfusion in treatment of poisoning. In: Daugirdas, J.T., Blake, P.G., Ing, T.S. (Eds.), *Handbook of dialysis*, (4th edition). Lippincott Williams & Wilkins, Philadelphia, pp. 300-320.

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