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Resolution of anuric acute kidney injury in a dog with multiple organ dysfunction syndrome

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Abstract

Objective – To describe the management and resolution of anuric acute kidney injury (AKI) in a dog with multiple organ dysfunction syndrome (MODS) associated with gallbladder wall necrosis.

Case Summary – An 11-year-old neutered female spayed dog was referred for evaluation of anuria following cholecystectomy. Following surgery, the patient became anuric with no response to appropriate medical therapy. During the course of hospitalization, the patient developed MODS as evidenced by alteration in renal function, but also cardiovascular dysfunction, coagulation disorders, and hypoglycemia. Several hemodialysis treatments were performed and, along with intensive care, led to resolution of clinical signs and return of urine production.

New or Unique Information – This report describes resolution of anuria in a dog with AKI and MODS. In this clinical setting, despite a poor prognosis, survival and recovery of adequate renal function were possible with medical management that included hemodialysis.

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Keywords: acute renal failure, canine, hemodialysis, SIRS, sepsis

Introduction

Acute kidney injury (AKI) is characterized by the sudden onset of renal parenchymal injury as a consequence of a broad spectrum of diseases. The term AKI has been adopted to reinforce the concept that AKI encompasses a continuum of functional and parenchymal damage. Acute renal failure (ARF) is associated with rapid hemodynamic, filtration, tubulointerstitial, or outflow injury to the kidneys and subsequent accumulation of metabolic toxins (uremia toxins) and dysregulation of fluid, electrolyte, and acid-base balance. ARF reflects the most severe grade of AKI and it is this subset of patients that have the highest morbidity and mortality and indication for renal replacement therapy.^{1–3} AKI can develop secondary to infectious or toxic insults, as well as hemodynamic and metabolic disorders.^{4–6} AKI also may be caused by a systemic inflammatory response

Abbreviations

AKI	acute kidney injury
AKIN	acute kidney injury network
aPTT	activated partial thromboplastin time
CRI	constant rate infusion
HD	hemodialysis
MAP	mean arterial blood pressure
MCHC	mean corpuscular hemoglobin concentration
MCV	mean corpuscular volume
MODS	multiple organ dysfunction syndrome
N/A	not available
PT	prothrombin time
RIFLE	risk, injury failure, loss, end-stage
RRT	renal replacement therapy

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syndrome associated with a single organ dysfunction or be part of a multiple organ dysfunction syndrome (MODS) with multifactorial pathogenesis. The incidence of AKI in veterinary medicine is not well documented, but AKI is reported to occur in 10% to 67%^{7,8} of human patients in the intensive care setting.

In the most severe forms of AKI, the patient may die from the consequences of acute uremia before there is opportunity for the renal injury to repair unless renal

replacement therapy (RRT) can be performed. Hemodialysis (HD) is a form of extracorporeal RRT that can remove uremic toxins and restore acid-base, fluid, and electrolyte homeostasis.⁹ HD can improve outcome in patients with oligoanuria, by providing renal support while allowing time for the injured kidney to recover. In diverse cohorts of people, the need for RRT is an independent risk factor for in-hospital mortality.¹⁰ In animal patients, the prognosis for AKI is dependent on its underlying etiology and the time to initiate appropriate medical management. Of 124 dogs undergoing HD, survival rates varied from 12% to 76% depending on the underlying etiology.^a

One well recognized etiology of AKI is MODS. While the incidence of AKI in dogs due to MODS has been reported to be approximately 12%,¹¹ the frequency of anuria or the need for RRT in this setting is unknown. Oligo-anuria in a cohort of dogs has been shown to affect mortality⁶ and may lead to patient euthanasia, especially if RRT is not feasible for technical or financial reasons. To the authors' knowledge, this is the first report of resolution of AKI with anuria in a dog with MODS.

Case report

An 11-year-old, 19 kg, neutered female German Shepherd Dog-mix was referred to the William R. Pritchard Veterinary Medical Teaching Hospital for evaluation of anuria following cholecystectomy. The patient was presented to the primary care veterinarian (Day 0) for evaluation of lethargy, anorexia, and vomiting of 2 days duration. Serum biochemistry revealed the following: alanine aminotransferase activity (ALT) 1,541 IU/L (reference interval 12–118 IU/L), alkaline phosphatase activity (ALP) 6,801 IU/L (reference interval 5–131 IU/L), gamma-glutamyl transferase (GGT) 68 IU/L (reference interval 1–12 IU/L), and bilirubin 90.6 $\mu\text{mol/L}$ [5.3 mg/dL] (reference interval 1.1–5.1 $\mu\text{mol/L}$ [0.1–0.3 mg/dL]). The serum potassium concentration was 3.3 mmol/L (reference interval 3.6–5.5 mmol/L), and the blood urea nitrogen (BUN) and serum creatinine concentrations were 5.0 mmol/L [14 mg/dL] (reference interval 2.1–11.1 mmol/L [6–31 mg/dL]) and 61 $\mu\text{mol/L}$ [0.8 mg/dL] (reference interval 38.1–122.0 $\mu\text{mol/L}$ [0.5–1.6 mg/dL]), respectively. The urine specific gravity at that time was 1.013. Abdominal ultrasound examination performed by the primary veterinarian revealed an enlarged gall bladder and peritoneal effusion. The patient subsequently was referred to a specialty practice later that day. Serum biochemistries at the specialty practice revealed hypoglycemia (serum glucose 2.8 mmol/L [51 mg/dL], reference interval 3.3–6.4 mmol/L [60–115 mg/dL]), and azotemia [BUN 13.6 mmol/L [38 mg/dL] (reference interval 3.6–9.3 mmol/L [10–26 mg/dL]) and

creatinine 282.9 $\mu\text{mol/L}$ [3.2 mg/dL] (reference interval 44.2–115.0 $\mu\text{mol/L}$ [0.5–1.3 mg/dL]). The patient was administered IV isotonic crystalloids with 2.5% dextrose and 2 mg/kg of meperidine subcutaneously. An abdominal ultrasound performed by a board-certified internal medicine specialist confirmed the marked gall bladder distension with intraluminal hyperechoic material, but there was no evidence of mucocoele. The liver parenchyma was reported to have a normal ultrasonographic appearance. A moderate amount of peritoneal effusion was present, and the mesentery was assessed as diffusely hyperechoic. The urinary tract showed no abnormality. The patient was anesthetized with oxymorphone, propofol, and isoflurane and an exploratory celiotomy was performed by a board-certified surgeon. The wall of the gall bladder appeared necrotic and ruptured during surgical manipulation. The liver was uniformly purple to brown with multifocal subcapsular hemorrhages. Cholecystectomy was performed, and the abdomen was lavaged at the end of the procedure. Culture of peritoneal fluid collected at surgery yielded no bacterial growth. During surgery, the patient developed hypotension with a mean arterial blood pressure (MAP) of 40 mm Hg, as assessed with an oscillometric blood pressure monitor. Intravenous crystalloids, colloids (canine fresh frozen plasma and hetastarch^b), and an IV constant rate infusion (CRI) of dopamine (started at 3 $\mu\text{g/kg/min}$ titrated up to 7 $\mu\text{g/kg/min}$) were administered in an attempt to stabilize the blood pressure. The patient maintained a MAP of 50 mm Hg during most of the 75-minute surgery. It was reported that the size of the urinary bladder did not change during the procedure. At the time of extubation, 1 hour after the procedure, the dopamine CRI was discontinued. At that time, the patient had a MAP of 64 mm Hg. Ninety minutes later, the patient had a MAP of 72 mm Hg.

Postoperatively, the patient was treated with IV hetastarch^b (0.6 mL/kg/h), isotonic crystalloids with 5% dextrose (3 mL/kg/h), fentanyl (4 $\mu\text{g/kg/h}$), ampicillin/sulbactam (20 mg/kg q 8 h), enrofloxacin (10 mg/kg q 24 h), metoclopramide (0.05 mg/kg/h), and famotidine (0.5 mg/kg q 12 h subcutaneously). Seven hours after the last biochemical panel, the BUN and serum creatinine concentrations were 17.9 mmol/L [50 mg/dL] and 267 $\mu\text{mol/L}$ [3.5 mg/dL], respectively. The serum potassium concentration was 3.6 mmol/L (reference interval 3.8–5.3 mmol/L). Prothrombin time (PT) was 21 seconds (reference interval 12–17 s) and activated partial thromboplastin time (aPTT) was 146 seconds (reference interval 71–102 s). Several hours after recovery, the patient was anuric as measured with a urinary catheter and closed collection set.

Two doses of furosemide (at 2 mg/kg IV) and a single bolus of mannitol (at 0.5 g/kg) followed by a CRI

at 60–120 mg/kg/h did not result in any urine production. On day 1, the BUN concentration was 21.4 mmol/L [60 mg/dL] and the serum creatinine concentration was 366.0 μ mol/L [4.8 mg/dL]. The serum potassium concentration was 4.5 mmol/L. Hypoxemia was confirmed via pulse oximetry measurements (SpO₂ 87–90%, fraction of inspired oxygen 21%) and was treated with oxygen supplementation. The dog was referred to the small animal emergency service at the William R. Pritchard Veterinary Medical Teaching Hospital for continued medical management.

On presentation, the patient was stuporous, had peripheral edema of all limbs, and was judged to be clinically mildly overhydrated. The temperature was 37.8°C (100.1°F), and perfusion parameters were adequate. A grade 3/6 left systolic apical heart murmur was auscultated. The patient had a respiratory rate of 36/min, and clear lung sounds were auscultated throughout all lung fields. Abdominal palpation elicited a moderate pain response, and the abdomen was markedly distended.

Upon admission, a serum biochemical analysis and a complete blood count were performed (Tables 1 and 2, respectively). Urine collected from the closed urinary collection system had a specific gravity of 1.019, rare granular casts (3–5 WBC/HPF), and >100 RBC/HPF. Urine culture yielded no aerobic or anaerobic bacterial growth. Cytologic examination of an ultrasound-guided sample of peritoneal effusion was consistent with postoperative nonseptic inflammation and demonstrated no evidence of bile leakage. The systolic blood pressure by indirect oscillometry^c was 65 mm Hg, prompting the placement of an indwelling 20-Ga arterial catheter in a dorsal pedal artery for assessment of direct arterial pressure. Direct arterial blood pressure monitoring^d confirmed a MAP of 60 mm Hg. Central venous pressure at that time was 7 cm H₂O. The patient was treated with a dopamine^e CRI (5–15 μ g/kg/min IV) to maintain a MAP between 70 and 90 mm Hg. Coagulation times were prolonged [PT 14.8 s (reference interval 7.0–9.3 s), aPTT 23.1 seconds (reference interval 10.4–12.9 s)]. A nasogastric tube^f was placed, and 1500 mL of green liquid containing a mild amount of blood were removed from the stomach. The urinary catheter was removed.

Given progressive azotemia and documented anuria, intermittent hemodialysis (HD) was initiated. The patient was sedated with narcotic analgesics, and an 11.5 Fr x 24 cm duel lumen hemodialysis catheter^g was placed in the jugular vein using the modified Seldinger technique. The Gambro Phoenix Hemodialysis System^h was utilized for HD. The hemodialysis prescription included a Gambro pediatric Cartridge Blood Set and a Gambro Polyflux Revaclear dialyzer. The first HD treatment consisted of a Kt/V (Kt/V defines hemodialysis dose)

of 3.5, where K = dialyzer urea clearance, t = treatment time, and V = volume of distribution, as measured by the Diascan on the HD machine. Approximately 0.6 L of ultrafiltrate was removed during the 4.5-hour session, which resulted in a net fluid removal approximately of 150 mL with Qp = 55 L (volume of blood processed during HD). No hypotension occurred during the treatment. Ampicillinⁱ (20 mg/kg IV q 8 h), enrofloxacin^j (5 mg/kg IV q 24 h), hydromorphone^k (0.05 mg/kg IV q 4 h), famotidine^l (0.5 mg/kg IV q 24 h), sucralfate^m (1 g via the nasogastric tube q 8 h), and metoclopramideⁿ (1 mg/kg/day IV CRI) were administered following the HD treatment.

On the morning of day 2, an approximate 6 cm urinary bladder was palpable. Serum biochemistry analysis and complete blood count were repeated (Tables 1 and 2, respectively). Arterial blood gas measurements demonstrated a PaO₂ of 85 mm Hg and a PaCO₂ of 34 mm Hg on room air. The blood pressure and blood glucose concentration remained within normal limits without IV dopamine or dextrose support. Enteral nutrition^o was initiated via the nasogastric tube with 50% of the daily resting energy requirement provided over 4 meals per 24 hours.

On day 3, serum biochemistry analysis and complete blood count were repeated (Tables 1 and 2, respectively). The patient had a 5 cm bladder palpable on physical examination and urinated approximately 150 mL (7.5 mL/kg) overnight. The patient received a second HD treatment on that day. Ondansetron^p (0.2 mg/kg IV q 24 h) was added to the previous therapeutic plan, as the patient appeared nauseous.

On day 4, the patient received a third HD treatment due to progressive azotemia (Table 1). On day 5, thoracic radiographs revealed alveolar pulmonary infiltrates, ventrally in the right cranial, and middle lung lobes, consistent with aspiration pneumonia. A diffuse interstitial pattern was also present and was attributed to noncardiogenic pulmonary edema. The patient was placed back on supplemental oxygen for the following 96 hours until pulse oximetry recordings were above 94% on room air. On day 6, the patient received the last HD treatment. Urine output had increased to approximately 2 mL/kg/h.

The patient developed polyuria during recovery and required IV fluid administration to maintain normal electrolyte and hydration status. Supportive care was provided until day 13 when IV fluids could be tapered. The patient was discharged from the hospital on day 14 with the following medications: amoxicillin^q (20 mg/kg PO q 12 h for 14 days), enrofloxacin^j (5 mg/kg PO q 24 h for 14 days), aluminium hydroxide^r (12.5 mg/kg PO q 12 h with meals), omeprazole^s (1 mg/kg PO q 24 h for 7 days), and ondansetron^t (0.4 mg/kg PO q 12 h as needed).

Table 1: Results of serum biochemical analysis in a dog with anuric acute kidney injury and multiple organ dysfunction syndrome during hospitalization

Parameter	BUN (mmol/L) [mg/dL]	Creatinine (μ mol/L) [mg/dL]	Potassium (mmol/L)	ALP (IU/L)	GGT (IU/L)	Bilirubin (μ mol/L) [mg/dL]	ALT (IU/L)	AST (IU/L)
Reference interval	3.9–11.8 [11–33]	70.7–132.6 [0.8–1.5]	3.6–4.8	14–91	0–5	0–3.4 [0–0.2]	21–72	20–49
Day 1*	34.3 [68]	503.9 [5.7]	4.1	3,260	55	102.6 [6.0]	747	271
Day 1 [†]	1.43 [4]	88.4 [1]	3.5	N/A	N/A	N/A	N/A	N/A
Day 2	6.1 [17]	194.5 [2.2]	4.1	3,223	51	97.5 [5.7]	596	183
Day 3	22.1 [62]	468.5 [5.3]	3.1	5,168	63	83.8 [4.9]	445	121
Day 4*	34.6 [97]	6–1.1 [6.8]	3.0	9,532	97	87.2 [5.1]	399	163
Day 4 [†]	1.07 [3]	53.0 [0.6]	2.7	N/A	N/A	N/A	N/A	N/A
Day 5	10.0 [28]	256.4 [2.9]	3.4	8,970	91	27.4 [1.6]	296	98
Day 6*	17.5 [44]	371.3 [4.2]	3.5	N/A	N/A	N/A	N/A	N/A
Day 6 [†]	3.4 [9.6]	88.4 [1]	3.0	N/A	N/A	N/A	N/A	N/A
Day 11	12.1 [34]	256.4 [2.9]	4.9	2,237	19	6.8 [0.4]	45	37
Day 13	10.4 [29]	221.0 [2.5]	4.6	N/A	N/A	N/A	N/A	N/A

*Data were collected prior to hemodialysis treatment.

[†]Data were collected at the end of hemodialysis treatment.

BUN, blood urea nitrogen; ALP, alkaline phosphate; GGT, gamma-glutamyl transferase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; N/A, not available.

Table 2: Results of complete blood counts in a dog with anuric acute kidney injury and multiple organ dysfunction syndrome during hospitalization

Parameter	Leukocytes (/ μ L)	Bands cells (/ μ L)	Neutrophils (/ μ L)	Platelets (/ μ L)	Hematocrit (%)	MCV (fL)	MCHC (g/dL)	Reticulocytes (/ μ L)
Reference interval	6,000–13,000	Rare	3,000–10,500	150,000–400,000	40–55	65–75	33–36	7,000–65,000
Day 1	45,861	459	39,000	190,000	31.1	61.1	36.3	N/A
Day 2	55,770	11,154	34,020	73,000	32.4	15.6	34.0	29,800
Day 3	49,880	8,978	34,417	91,000	33.1	65.8	32.6	27,900
Day 4	56,800	2,840	43,736	150,000	34.0	68.3	32.1	35,300
Day 5	40,870	0	28,200	170,000	32.4	65.6	33.0	28,600
Day 10	26,890	261	21,663	263,000	24.7	65.5	33.2	60,200

MCV, mean corpuscular volume; MCHC, mean corpuscular hemoglobin concentration; N/A, not available.

Histopathologic examination of the gall bladder demonstrated severe multifocal fibrinosuppurative cystitis, necrosis, hemorrhage, multifocal granulation tissue, and mucinous hyperplasia. Analysis of the liver biopsy revealed severe neutrophilic hepatitis with multifocal necrosis, hemorrhage, and thrombosis.

Two months after discharge, re-examination at the primary care veterinarian revealed the ALT concentration was 135 IU/L (reference interval 12–118 IU/L), the ALP concentration was 141 IU/L (reference interval 5–131

IU/L), the BUN was 8.2 mmol/L [23 mg/dL] (reference interval 2.1–11.1 mmol/L [6–31 mg/dL]), and the serum creatinine concentration was 132.6 μ mol/L [1.5 mg/dL] (reference interval 44.2–141.4 μ mol/L [0.5–1.6 mg/dL]).

Discussion

To the authors' knowledge this is the first report of resolution of AKI with anuria in a critically ill dog with concomitant MODS. Two major classification schemes

for AKI have been proposed in people and transposed to veterinary medicine:^{4,12} RIFLE (risk, injury, failure, loss, and end-stage) and AKIN (acute kidney injury network). Both schemes use an acute increase in creatinine concentration and decrease in urine output as criteria. For the patient in this report, the diagnosis of AKI was based on the lack of urine production and a greater than 50% rise in creatinine concentration from initial presentation. There however was suspicion of AKI on original presentation as the patient was isosthenuric at that time. As opposed to dogs, AKI in people frequently occurs during hospitalization, and in many situations, human scoring systems might be difficult to apply to veterinary patients as baseline creatinine or glomerular filtration rate (GFR) are often unknown at presentation. The International Renal Interest Society (IRIS) has recently proposed a grading system for AKI.¹¹ In accordance with that system, the patient in this report would be classified in AKI grade I at initial presentation (based on the urine specific gravity) and progressed to a grade IV (serum creatinine 451.0–884.0 $\mu\text{mol/L}$ [5.1–10 mg/dL]) O (oligoanuric) upon presentation to the William R. Pritchard Veterinary Medical Teaching Hospital (based on the creatinine measurement and the urine output).

It has been reported that 4% to 7% of the human hospital population develops AKI,^{13,14} with this number being higher in critically ill patients.¹⁵ In the ICU setting, RRT is required in 72% of patients with AKI.⁵ The associated mortality rate for AKI in critically ill people when RRT is required is 60%, and 14% of the patients who survive are dialysis dependent at discharge.⁵ The survival rate in the canine population with AKI depends on the etiology, and ranges from 12–76%.^{4,12,16,17,a}

Systemic inflammatory response syndrome is due to either sterile or infectious etiologies (sepsis) and can result in organ dysfunction. When more than one organ is dysfunctional as a result of systemic inflammation distant to the site of inflammation, a diagnosis of MODS can be made.¹⁸ The rates of individual organ failure with MODS in veterinary medicine are unknown. AKI can be associated with a wide array of disorders, including infections, thrombosis, metabolic disturbances, intoxication, drugs, and hemodynamic instability. Sepsis with either single or multiple organ dysfunction is the leading cause of AKI in humans,¹⁹ accounting for approximately 50% of all AKI incidents.⁵ For the patient of this report, renal dysfunction was likely part of MODS, incited by gall bladder necrosis with associated peritonitis. Other potential aggravating factors for the AKI were anesthesia-induced hypotension and hetastarch administration.

While this patient had evidence of MODS with dysfunction of the renal (AKI), cardiovascular ($\text{MAP} < 65 \text{ mm Hg}$ with central venous pressure of 7 $\text{cm H}_2\text{O}$),

and coagulation systems (prolonged PT/aPTT with a declining platelet count), as well as hypoglycemia, it is unknown if this was due to a septic or nonseptic etiology. In a study from Crews *et al.*,²⁰ 11/45 dogs with gallbladder disease had positive bile bacterial cultures. Out of those 11 dogs, 9 had gallbladder wall necrosis and 5 had gallbladder rupture. Seventy to 81% of dogs with documented gallbladder wall necrosis had positive bile bacterial cultures.^{20,21} Gallbladder wall necrosis and rupture also were found in the dog in this report; however, no bile culture was performed. Peritoneal cultures from the time of surgery showed no evidence of bacterial growth, but this does not rule out a septic process originating from the biliary tract.

MODS is a condition associated with a high mortality rate. Individual mortality rate is affected by the severity of the underlying disease and correlates with the number of dysfunctional organs. In a study of adult human ICU patients, a 9% mortality rate was reported when no organ failure was present upon admission, whereas mortality increased to 83% for patients with 4 or more failing organs.²² In another study,²³ patients with single organ dysfunction had a mortality rate of 28%, and those with multiple organ failure had a mortality rate of 38%. In a pediatric ICU study,²⁴ the mortality for patients with MODS was 42% compared to 5% in patients who did not have MODS. In a cohort of human patients with AKI and MODS requiring RRT, 92% of patients that survived were not dependent on RRT at the time of discharge.²⁵

In veterinary medicine, similar observations have been made. The mortality rate for septic dogs increased as the number of dysfunctional organs increased, with mortality rates of 16%, 31%, 54%, 76%, 91%, and 100% for 0, 1, 2, 3, 4, and 5 organs dysfunctional, respectively.¹¹ This study did not assess glycemic imbalance as a criteria for organ dysfunction; therefore, according to those criteria our patient had evidence of 3 dysfunctional organs (cardiovascular, renal, and coagulation). Although the patient was diagnosed with pulmonary dysfunction, this was thought to be due to aspiration pneumonia and not acute lung injury or acute respiratory distress syndrome. The same study also demonstrated that dysfunction of the respiratory, cardiovascular, renal, or coagulation systems (3 of which were affected in our patient) significantly increased the odds of death. AKI in the setting of MODS has reported survival rates ranging from 32% to 57% in humans^{24–27} and 14% in septic canine patients.¹¹

The patient of this report also developed significant hypotension during anesthesia, and this likely contributed to the renal damage, but it was unlikely to have been the sole cause, especially since azotemia developed before anesthesia. The degree of hypotension in this

patient may have been over- or underestimated, as oscillometric blood pressure has been shown to be unreliable in anesthetized hypotensive dogs.²⁸ While hypotension is a well reported contributor to the onset of AKI,^{29–31} the magnitude and duration of hypotension required to trigger damage to the kidney is unknown and is most likely multifactorial. Anesthetic induced intraoperative hypotension leading to AKI is poorly described in the general human hospital population,³² although AKI following cardiopulmonary bypass surgery is well documented. Multiple animal models have been used to investigate ischemia injury to the kidneys. In these studies, renal artery clamping with no blood flow for 22 to 60 minutes was used and better mimics AKI during renal transplantation, as opposed to the patient in this report.^{33–35} Hypotension during anesthesia, however, is more likely to be related to reduced rather than to zero-flow scenarios. One study in geriatric human patients undergoing elective hip replacement showed no difference in renal complications between patients with marked hypotension (MAP 45–55 mm Hg) and mild to moderate hypotension (MAP 55–70 mm Hg); however, no control group of normotensive patients was available for comparison.³⁶ In healthy anesthetized dogs, renal blood flow remains autoregulated until the MAP decreases below 75 mm Hg and then decreases progressively with an additional 13% decrease of MAP to 65 mm Hg.³⁷ In the setting of systemic inflammation, autoregulation of blood flow in the kidney is compromised.³⁸ In a canine model of endotoxemic shock, renal blood flow has been shown to be altered even with MAP >75 mm Hg.³⁹ As the patient was critically ill and had signs of systemic inflammation, it is plausible that the extent of hypotension present during anesthesia was sufficient to contribute to renal injury.

The patient also was administered synthetic starch-based colloids during and after anesthesia. While there are no reports in veterinary medicine of starch-based colloid induced kidney injury, multiple studies in people have raised serious concerns. In a randomized multicenter study of human patients with severe sepsis,⁴⁰ hetastarch 10% (200/0.5) was found to be associated with dose dependent higher rates of AKI and need for RRT than was Ringer's lactate. The negative renal effects of synthetic colloid administration are relative to the cumulative dose,⁴¹ making caution necessary when used in patients at risk for AKI. The patient in this report received a cumulative dose of hetastarch of 9 mL/kg, which has been associated with AKI in people.⁴⁰ It is speculative that the use of hetastarch in this patient also contributed to the development of renal injury; however, based on clinical experience this was unlikely to be the initiating factor.

Conclusion

To the authors' knowledge this is the first report of resolution of anuric AKI in a critically ill patient due to systemic inflammation with MODS. This report confirms that despite the presence of severe illness and multifactorial AKI, resolution of anuria and return of adequate renal function are possible, if appropriate treatment is administered.

Footnotes

- ^a Francey T, Cowgill LD. Use of hemodialysis for the management of acute renal failure in the dog: 124 cases (1990–2001). *J Vet Intern Med* 2002; 16:352.
- ^b Hetastarch: 6% Hetastarch, Hospira, inc., Lake Forest, IL.
- ^c Cardell: Model 9401, Cardell Veterinary Monitor, Tampa, FL.
- ^d Spacelabs: Model 90602, Spacelabs, Redmond, WA.
- ^e Dopamine: DOPamine.
- ^f Nasogastric tube: Nasogastric feeding tube, Mila International, Inc., Covington, KY.
- ^g HD catheter: 11.5 Fr, 24 cm, Medcomp, Harleysville, PA.
- ^h Phoenix Hemodialysis System, Gambro, Lakewood, CO.
- ⁱ Ampicillin: Ampicillin, Sandoz Inc., Princeton, NJ.
- ^j Enrofloxacin: Baytril, Bayer, Shawnee Mission, KS.
- ^k Hydromorphone: Hydromorphone.
- ^l Famotidine: Pepcid, Merck, Whitehouse Station, NJ.
- ^m Sucralfate: Carafate, Nostrum Laboratories, Kansas City, MO.
- ⁿ Metoclopramide: Metoclopramide.
- ^o Vital HN: Vital HN, Abbott Nutrition, Columbus, OH.
- ^p Ondansetron IV: Ondansetron, West-Ward Pharmaceuticals, Eatontown, NJ.
- ^q Amoxicillin PO: Amoxi Tabs, Pfizer, New York, NY.
- ^r Aluminium hydroxide: Aluminium hydroxide, Rugby, Duluth, GA.
- ^s Omeprazole: Omeprazole, Procter and Gamble, Cincinnati, OH.
- ^t Ondansetron PO: Ondansetron, Teva Pharmaceuticals, Sellersville, PA.
- ^u Cowgill L. Clinical Staging of Acute Kidney Injury. In: *Proceedings of the Advanced Renal Therapies Symposium*; 2012 : New York, USA. pp. 81–84.

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