This case report describes treatment of a feline patient with urethral obstruction. After admittance to the intensive care unit, he was also found to have unilateral ureteral calculi. He developed acute kidney injury (AKI), characterized by post renal azotemia, which was nonresponsive to fluid therapy. After 72 hours of hospitalization, he underwent cystotomy and right ureterocystostomy.

Peritoneal Dialysis Following Urethral Obstruction and Acute Kidney Injury

ACUTE KIDNEY INJURY (AKI) is the abrupt inability of the kidneys to regulate solute and water balance. AKI includes oliguric renal failure, anuric renal failure, and severe uremia that is unresponsive to fluid therapy. Image courtesy of shutterstock.com/Deyan Georgiev

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VITAL SIGNS
His AKI persisted after surgery (characterized by worsening azotemia, oliguria that progressed to anuria, and evidence of fluid intolerance), which ultimately required implementation of peritoneal dialysis (PD).

**SIGNALMENT**

The patient was a 12-year-old neutered male domestic shorthair cat that weighed 7.5 kg.

**HISTORY AND PRESENTING COMPLAINT**

The patient was presented to the emergency department for acute-onset anorexia, retching, and being abnormally vocal. When his owner picked him up at home, he seemed painful and bloated. His owner reported that he was uncomfortable throughout the evening. His owner also reported that he had recently been urinating outside the litter box. She believed that her husband’s recent disability caused him stress. The patient was an indoor-only cat with no prior health concerns.

**INITIAL ASSESSMENT/PHYSICAL FINDINGS**

On entry, the patient was bright, alert, and responsive. He was normothermic at 100.2°F, vocal, and ambulatory. He was eupneic at 30 beats/min, with clear lung sounds. He had a relative tachycardia at 220 beats/min, with strong, synchronous femoral pulses and no murmur appreciated on auscultation. His mucous membranes were pink and tacky with a capillary refill time of less than 2 seconds. He was normotensive; the Doppler systolic blood pressure was 162 mm Hg. He was painful on abdominal palpation, and he had a large, firm bladder.

**NURSING CARE TREATMENT PLAN**

I placed a 20-gauge cephalic IV catheter and started a 13-mL/kg (1/8 shock) IV crystalloid bolus. Blood was collected for point-of-care venous blood gas (VBG) measurement, as well as for a complete blood count. The VBG analyzer provided information about acid-base status, electrolytes, ventilation, lactate, glucose, PCV/total protein (TP), and blood urea nitrogen (BUN)/creatinine values. IV crystalloids were continued at 6.5 mL/kg per hour after the bolus, and 0.02 mg/kg IV buprenorphine (a partial opioid agonist) was administered for pain. Preliminary abdominal radiographs were also obtained (FIGURE 1).

The patient was sedated with 0.2 mg/kg IV midazolam for urinary unobstruction. He was preoxygenated and then induced with 2 mg/kg IV propofol slowly titrated to effect. After intubation, flow-by oxygen was provided via mask around his endotracheal tube. He remained anesthetized with 1.5-mg/kg boluses of IV propofol as needed, for a total dose of 4 mg/kg. An electrocardiographic monitor was placed during the procedure to ensure normal heart rate and rhythm. By using sterile technique, I passed an open-ended tomcat catheter; hemorrhagic urine with grit-like material was noted. A sterile urine sample was collected for urinalysis. I was able to gently retropulse the urethra with sterile saline, and eventually placed a 3.5-Fr red rubber urinary catheter. I connected the catheter to a sterile closed collection set (FIGURE 2) and obtained an additional lateral radiograph to confirm placement.

All vital signs remained within normal limits, and the patient’s anesthetic recovery was uneventful. After urinary catheterization, the patient was receiving 0.02 mg/kg IV buprenorphine q8h, and IV crystalloids were adjusted q4h on the basis of hourly urine output. His urine output...
was quantified every 4 hours. Serial weights were measured every 6 to 8 hours. He was started on 30 mg/kg IV ampicillin-sulbactam (bactericidal antibiotic) q8h because of concern for infection.

VBG revealed azotemia with a BUN of 42 mg/dL (reference range, 18 to 34 mg/dL) and creatinine of 2.6 mg/dL (reference range, 1.0 to 2.1 mg/dL). He had a normal acid-base status with a pH of 7.361 (reference range, 7.24 to 7.4), partial pressure of CO₂ of 26.1 mm Hg (reference range, 25.9 to 41.7 mm Hg), and HCO₃ of 14.9 mmol/L (reference range, 14.7 to 22.1 mmol/L). Electrolytes, lactate, and PCV/TP were within normal limits. His complete blood count was normal except for an elevated white blood cell count of 18,000 cells/μL (reference range, 2870 to 17,020 cells/μL). Urinalysis showed too numerous red blood cells to count, 1 to 3 white blood cells per high-power field, urine specific gravity of 1.021, and pH of 7.0. No crystals, casts, or bacteria were noted on sediment review. The initial abdominal radiographs showed cystic calculi and a urinary outflow obstruction secondary to the accumulation of crystalline debris and calculi within the distal urethra; the material was subsequently displaced into the bladder after catheterization. The patient had right renal mineralization in the retroperitoneal space (concerning for ureteral calculi); abdominal ultrasonography was recommended.

Overnight, the patient’s urine output was normal, ranging from 1 to 2 mL/kg per hour. Repeat VBG showed worsened azotemia, with a BUN of 51 mg/dL (reference range, 18 to 34 mg/dL) and creatinine of 3.7 mg/dL (reference range, 1.0 to 2.1 mg/dL). Electrolytes, acid-base status, and PCV/TP were all still within normal limits. Abdominal ultrasonography confirmed that the patient had a right ureteral obstruction with concurrent bilateral pyelonephritis. Throughout the day, the patient’s urine output steadily declined to the point of oliguria (urine output < 1 mL/kg per hour), and he gained 10% to 12% of body weight. Given the ultrasonographic findings and poor urine output, surgery for ureteral reimplantation and placement of a drain for possible PD was discussed. Because of their finances and the invasiveness of the procedure, the owners elected to continue medical management at this time.

Because of the patient’s pyelonephritis, antibiotic coverage was increased by adding 5.5 mg/kg IV enrofloxacin q24h. Ampicillin-sulbactam frequency was decreased to q12h as a result of limited renal excretion. A trial dose of 1 mg/kg IV furosemide, a loop diuretic, was given to encourage polyuria; if urine output improved, additional doses or a constant rate infusion (CRI) would be considered. At 24 hours of hospitalization, VBG showed worsened azotemia, with a BUN of 63 mg/dL (reference range, 18 to 34 mg/dL) and creatinine of 5.2 mg/dL (reference range, 1.0 to 2.1 mg/dL); all other values were within normal limits. By 48 hours of hospitalization, the patient remained oliguric.

Three days after presentation, the patient had surgery with a board-certified specialist, and the patient underwent cystotomy; a stone in his right ureter was dilated proximal to the lesion, so right
ureterocystostomy (ureteral reimplantation of the right kidney) was done. A Jackson-Pratt drain was placed for possible PD. The patient did well under anesthesia, and his recovery was uneventful. His weight also improved, decreasing to a 5% gain over his entry weight (this change was suspected to have resulted from reestablishment of a patent urinary system and use of furosemide). After the procedure, VBG showed normal values with the exception of a BUN of 83 mg/dL (reference range, 18 to 34 mg/dL); creatinine was improved at 4.7 mg/dL (reference range, 1.0 to 2.1 mg/dL). The patient was started on IV fentanyl (a 100 times more potent opioid analgesic) CRI at 4 mcg/kg per hour. Jackson-Pratt drain production was quantified every 4 hours, and IV crystalloids were continued at 3.5 ml/kg per hour, to be adjusted according to urine output.

After ureterocystostomy, the patient continued to have severe oliguria, verging on anuria (lack of urine production). He also began to develop peripheral and SC tissue edema. His owners were informed that despite surgery, the patient’s urine output was minimal, renal values continued to worsen, and evidence of fluid intolerance was developing. The team reiterated that PD was the only option available, and the inherent risks (electrolyte shifts, hyperglycemia, peritonitis) were discussed; his owners approved.

I placed a 5-Fr nasoesophageal tube, as well as a 5-Fr · 13-cm triple-lumen jugular catheter (FIGURE 3). His IV crystalloids were discontinued because of evidence of fluid overload. IV antibiotics and fentanyl CRI were continued as previously; 1 to 2 mg/kg IV furosemide was to be given as needed. Enteral nutrition was started at one quarter of his resting energy requirement (calculated as [body weight in kg · 70]0.75), and IV ketamine (a dissociative agent that works well on somatic pain and is accentuated by opioids) CRI was added at 2 mcg/kg per minute for multimodal analgesia.

The patient’s dialysate (the mixture that passes through the membrane during dialysis) prescription was prepared by using 250 IU heparin + 2.5% dextrose in 1 L of lactated Ringer’s crystalloid solution (FIGURE 4). Heparin was added to prevent occlusion of the peritoneal catheter from fibrin, and dextrose was added as the osmotic agent.

By using strict aseptic technique, I connected the dialysate infusion line to the Jackson-Pratt drain tubing (the bulb was removed) with a 3-way stopcock (FIGURE 5). I connected the third port on the 3-way stopcock to a separate sterile collection bag so that the dialysate/peritoneal fluid could accumulate and be
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quantified. The Luer lock port sites were wrapped with sterile chlorhexidine-soaked gauze (FIGURE 6). The dialysate was infused over 15 minutes, then dwelled intra-abdominally for 30 minutes. Drainage via gravity was allowed for an additional 15 minutes. The dialysate infusion/drainage process was to be done every 3 hours.

As the primary nurse in the patient’s initial stabilization, my involvement continued through PD therapy. Initial nursing care focused on shock IV fluids, analgesia, total IV anesthesia, electrocardiographic monitoring, urinary catheter placement, laboratory testing, and diagnostic radiography. Once unobstructed, my focus was on close monitoring of parameters essential in renal patients, including mentation, vital signs assessed every 4 hours, Doppler blood pressure measured every 4 to 8 hours, measurement of urine output every 4 hours, urinary catheter care, and weight measurement every 6 hours. After surgery, I assessed pain by using the Colorado State University’s pain scale every 4 hours, adjusted analgesic CRIs as needed for pain control, continued to measure urine output, placed a nasoesophageal feeding tube and triple-lumen jugular catheter, calculated and started enteral nutrition, used 3-syringe technique for venous sampling, and had knowledge of multiple drug/fluid type interactions.

During PD, I provided one-on-one nursing care and management (FIGURE 7). For each PD infusion, I ensured strict sterility and accurate record keeping (volume in [medications, nutrition], dialysate in, dialysate out, urine output). I closely monitored the peritoneal catheter insertion site dressing for change in position or strikethrough. I also closely monitored for ongoing or worsening signs of fluid overload (development of nasal discharge, changes in respiratory rate/effort/pattern, changes in lung/heart sounds on auscultation, and spreading/worsening of peripheral edema). I also massaged the patient’s limbs and used passive range-of-motion exercises as part of physiotherapy to improve peripheral edema.

OUTCOME

After initiation of PD, the patient’s urine output progressively improved. A furosemide CRI was started;
restarting IV crystalloids was to be considered if the patient moved from oliguria to polyuria. Furosemide is a diuretic that works primarily on the loop of Henle portion of a nephron; it inhibits the body’s ability to reabsorb sodium, leading to increased excretion of water in urine. The furosemide CRI at 0.5 mg/kg per hour, in conjunction with increasing PD frequency to every 2 hours, resulted in the patient’s transitioning to polyuria. Unfortunately, he had sustained azotemia (BUN, 111 mg/dL [reference range, 18 to 34 mg/dL]; creatinine, 8.0 mg/dL [reference range, 1.0 to 2.1 mg/dL]). Because of the owners’ financial constraints, concern for residual renal function, and concern for long-term quality of life, his owners elected to humanely euthanize.

DISCUSSION

AKI is the abrupt inability of the kidneys to regulate solute and water balance. AKI includes oliguric renal failure, anuric renal failure, and severe uremia that is unresponsive to fluid therapy. Azotemia is characterized by abnormally high levels of body waste compounds, primarily urea and creatinine. Azotemia can further be classified as prerenal, intrinsic renal, and postrenal. As it implies, prerenal refers to “before” the kidney, meaning azotemia is caused by other physiologic factors or disease processes (ie, hypovolemia, hypotension, dehydration, cardiac compromise, systemic inflammatory response syndrome). Intrinsic renal refers to direct damage to the kidney (ie, nephrotoxins, ischemia, infectious causes). Postrenal refers to azotemia caused by an outflow obstruction in which urine cannot be eliminated from the body (ie, urethral obstruction, urolithiasis, neoplasia). This patient’s AKI was from postrenal causes (urethral and ureteral obstruction).

Dialysis transfers water and solute from one compartment to another by means of diffusion across a semipermeable membrane (SPM). In PD, the peritoneum serves as the SPM between the peritoneal cavity and the blood within the peritoneal capillaries. Dialysate (mixture that passes through the membrane) is prescribed to maximize elimination of uremic toxins, prevent depletion of normal blood solutes, replenish depleted solutes, and minimize physiologic and metabolic disturbances during and after dialysis sessions. PD is accomplished by instilling the dialysate mixture into the abdomen through a peritoneal catheter. The dialysate is allowed to dwell for a prescribed period and then is drained into a waste bag. During the dwelling/draining period, fluid and solutes (primarily waste products: urea and creatinine) are drawn across the membrane through diffusion (solute move across an SPM from an area of high to low concentration) and convection (movement of solutes with the flow of fluid).

PD is mostly indicated in patients with AKI and is considered a temporary therapy to replace the function of the kidneys, giving them time to heal. Other dialysis options available in veterinary medicine are intermittent hemodialysis (IHD) and continuous renal replacement therapy (CRRT). IHD is the removal of a patient’s blood so that it can be run through an artificial kidney machine (dialyzer). Uremic toxins are removed by diffusion across an SPM within the dialyzer (blood is circulated on one side, and dialysate is circulated on the other). The entire blood volume of the patient is treated, then returned. IHD takes 4 to 6 hours per treatment, with treatments done a set number of times per week. CRRT is essentially the same process as IHD except that it is done continuously. Limiting factors of IHD and CRRT include client cost and regional availability.

CONCLUSION

This case was interesting in that it started as a routine condition that progressed to a dedicated, one-on-one nursing case. The importance of a thorough workup was essential in managing this patient’s whole disease process. Even though the outcome was not what we had hoped, this case demonstrated many aspects of renal care, management, and complications. TVN