How to Perform Continuous Peritoneal Dialysis in an Adult Horse

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1. Introduction
Acute renal failure (ARF) in horses can be caused by a multitude of etiologies, but all result in a decrease in glomerular filtration rate (GFR) that leads to azotemia. Intrarenal azotemia must be distinguished from the other two categories of azotemia: pre-renal and post-renal. Pre-renal azotemia may result from any condition that leads to hypovolemia or hypotension that causes decreased renal perfusion. Post-renal failure is caused by obstruction of urinary flow, and in equids, it is rare, except in cases of neonatal bladder rupture. Intrinsic causes of renal failure include ischemic or toxic damage to the tubules, tubular obstruction, glomerulonephritis, or tubulointerstitial edema. Myoglobinurinuria secondary to exertional rhabdomyolysis may cause intrinsic pigment nephropathy and tubular obstruction, which can lead to acute renal failure such as that seen in the case reports described below.

The diagnosis of ARF is arrived at through historical information, clinical findings, and laboratory data. ARF is most often associated with oliguria and, rarely, anuria or polyuria. Other clinical signs are similar to uremia (anorexia and depression) and fluid and electrolyte imbalances. The most common laboratory finding is azotemia, which reflects the decrease in GFR. Electrolyte and acid-base abnormalities typically include acidemia, hyponatremia, hypochloremia, hyperkalemia, and hypercalcemia. If urine is being produced, urine specific gravity, urinalysis, and fractional excretions may help to confirm the presence of renal failure or determine its cause. With intrinsic renal failure, the urine is often isosthenic, and the fractional sodium excretion is >1.0. Renal ultrasound may also be a useful diagnostic tool in cases of ARF. Normal kidneys are the least echogenic organs in the abdomen, although they may become more echogenic with age. With acute renal failure, the kidneys may appear enlarged or perirenal edema may be present. Color-flow Doppler may also be used to assess renal perfusion.

Routine treatment for ARF is the judicious use of IV fluids (slightly higher than the maintenance rate of 5% of body weight for an adult horse over 24 h).
until oliguria is resolved. Fluid overload is a primary concern in oliguric/anuric patients. Euvolemia may be monitored by assessing the conjunctiva for edema and, more objectively, measuring and maintaining central venous pressures between 7.5 and 12 cm H2O. Electrolytes should also be carefully monitored and corrected accordingly, because disturbances are very common and represent a significant cause of morbidity/mortality (arrhythmias, dystrophic mineralization, muscular weakness). Hyperkalemia is the most common electrolyte imbalance in the oliguric horse and may be treated with dextrose (5%, IV), insulin (0.1–0.5 IU/kg, SC), calcium (1 mL/kg, IV in fluids), and sodium bicarbonate (mEq = 5 × wt kg × 0.3; do not combine with calcium-containing fluids). The use of 0.9% NaCl as a maintenance/replacement fluid choice is advisable to decrease the potassium load. The diuretics furosemide (1 mg/kg, q 6 h, IV) and mannitol (1 mg/kg, IV as a 20% solution) are frequently used to promote urination, but their efficacy in the treatment of ARF has been called into question. Furosemide is often ineffective in increasing renal perfusion and GFR in horses with ARF. To reach its active site in the tubular lumen, furosemide depends on established GFR and tubular secretion, two things that are lacking in the oliguric horse. The accumulation of cellular debris or pigments in the tubular lumen may also interfere with furosemide’s ability to cause diuresis. Furosemide has additionally failed to alter outcomes in a recent human multicenter trial. Despite this, judicious use of furosemide is still recommended. Mannitol does increase renal perfusion and GFR as well as enhance tubular flow and urine output, but it also increases tubular oxygen demand, which may potentiate ischemic injury. Dopamine, another agent anecdotally used in the treatment of ARF, used at a constant rate (3–7 μg/kg/min, IV) may selectively improve renal afferent blood flow and therefore, GFR; however, horses must be carefully monitored for systemic hypertension. Dopamine’s efficacy in improving human patient outcome has not been shown, and its use in the intensive care unit for the treatment of ARF is decreasing. Another supportive measure that should be implemented in cases of ARF is omeprazole (4 mg/kg, q 24 h, PO), which prevents uremic ulcers and ulceration secondary to anorexia and total or partial parenteral nutrition for anorexic patients.

When traditional therapies do not result in resolution of azotemia, other treatment methods must be considered. Hemodialysis and peritoneal dialysis are two therapies that have been used in the treatment of both humans and animals. Hemodialysis, although used in small-animal veterinary medicine, is cost and time prohibitive in large animals. Peritoneal dialysis has been used in small animals for the treatment of acute renal failure, metabolic and electrolyte imbalances, severe temperature extremes, and toxicities. Intermittent peritoneal dialysis or lavage has been used in large animals for the treatment of peritonitis and management of uremia in a foal with a ruptured bladder. The continuous peritoneal dialysis system described here for use in an adult horse with acute renal failure was first reported by Gallatin et al. in 2005. The usefulness of peritoneal dialysis relies on the permeability of the peritoneal membrane. The dialysate equilibrates with the plasma across the peritoneal membrane and allows for the diffusion of metabolic wastes and electrolytes out of the bloodstream and into the dialysate. The hypertonicity of the dialysate, because of the addition of dextrose to the fluids, causes ultrafiltration; this allows further transfer of solutes beyond what diffusion alone could permit.

2. Materials and Methods

1. Restrain and sedate horse.
2. Clip and aseptically scrub 8 × 8 cm areas for ingress and egress tubes. The ingress portal is placed in left paralumbar fossa. The egress portal is placed ventrally to the right of midline. Both areas are infiltrated with 5–10 mL lidocaine (Fig. 1).
3. Acquire two 32-French thoracic tubes with stylets for the ingress and egress portals. Other types of catheters have been used including a T-fluted catheter and a spiral fenestrated catheter.
4. Administer Buscopan (1.5 mL/100 kg) to decrease rectal contractions before placement of the ingress tube.
5. Use a #10 scalpel blade to incise through the skin, SC tissues, and superficial muscle layers of the left paralumbar fossa. The 32-French thoracic tube is then inserted through the incision and advanced toward the abdominal cavity. A release in tension

Fig. 1. Materials: (clockwise from 9 o’clock) Ioban surgical dressing, Elastikon, athletic tape, Christmas tree adaptors, scalpel blades, needle drivers, #2 Vicryl, 32-French thoracic tubes, and suction tubing.
is detected twice: first, with passage through the abdominal aponeurosis, and second, when the tube enters the peritoneum. An assistant should provide transrectal shielding of the spleen and abdominal viscera during insertion and confirm the tube’s placement in the peritoneal space. It is important to be confident that the tube is in the peritoneal space, because it is very easy to leave it retroperitoneally, in which case the dialysate may still flow very freely. When placement is confirmed, the stylet is removed. The tube is secured with a purse-string suture followed by a Chinese fingertrap using #2 vicryl. A Christmas tree adaptor with a female end is used to connect the tube to a large-animal, fluid-coil delivery system. All unions are secured with white athletic tape (Fig. 2).

6. Use a dialysate solution of 1.5% dextrose (150 ml 50% dextrose/5 l) in a balanced polyionic fluid (Normosol-R, 0.9% sodium chloride, Lactated Ringer’s solution, or Hartmann’s solution). To float the abdominal viscera away from the ventrum, infuse 10 l of the dialysate into the abdomen before attempting placement of the egress tube.

7. Place the egress tube in the same location that an abdominocentesis would be performed. A #15 blade is used to incise the skin and superficial layers of the abdominal wall. The chest tube with stylet is then advanced through the peritoneum until fluid is obtained. The stylet is removed, and the tube is sutured into place as above. This tube is connected to suction tubing with a Christmas tree adaptor and further secured with white athletic tape. Another Christmas tree adaptor is used to connect the tubing to an empty 5-l sterile fluid bag (Fig. 3).

8. Cover both the egress and ingress tube portals with Ioban surgical dressing and secure with Elastikon tape, and superglue.

9. Secure the collection bag at the level of the withers using a surcingle or suspend it from a braided mane. By keeping the collection bag higher than the egress port, enough back pressure is obtained to float the omentum off the egress tube. The collection bag is emptied when the contents have reached 4 l. Always leave at least 1 l of fluid in the bag after emptying to maintain intraperitoneal pressure. Be careful to preserve the sterility of all connections.

10. Continuously infuse the dialysate solution of 1.5% dextrose in a sterile balanced polyionic fluid through the ingress at a rate of 3 l/h. The dialysate fluids are suspended with IV fluids from an overhead suspension apparatus. A coiled delivery device provides easy delivery of the fluids at a high rate of administration. Fluid choices should be dictated by the patient’s electrolyte status.

11. Because of concerns regarding peritonitis, use broad-spectrum antibiotics that are not nephrotoxic, such as cefazolin and enrofloxacin, throughout treatment.

12. Continue IV fluids at a maintenance rate of 5% body weight per day and parenteral nutrition, if indicated.

3. Results
This system has been used at our hospital to treat an 8-yr-old Paso Fino stallion that was in ARF subse-
quent to an episode of exertional rhabdomyolysis. On presentation, his serum blood urea nitrogen (BUN) was 92 mg/dl, and his serum creatinine was 10.3 mg/dl. The horse was treated with fluids, furosemide, dopamine continuous rate infusion, and mannitol. He converted from anuria to oliguria, but his azotemia became more severe. By the third day, his BUN was 112 mg/dl, and his creatinine was 13.8 mg/dl. The peritoneal dialysis system was installed on day four and remained in place for four days. As the peritoneal BUN and creatinine increased, the serum BUN and creatinine decreased until the two equilibrated. By the time the system was removed, the horse’s serum BUN was 32 mg/dl, and his creatinine was 5.0 mg/dl (Fig. 4). Over the next several days, the azotemia resolved with additional IV fluids, and he was discharged with normal renal parameters. The stallion is currently active in show competitions and has a successful breeding career.

Our system was derived from a report on a 15-yr-old Paso Fino gelding who was successfully treated for ARF using a continuous-flow peritoneal dialysis system. This horse had also developed ARF subsequent to exertional rhabdomyolysis and was refractory to conventional treatment with fluids, furosemide, total parenteral nutrition, and dopamine. Intermittent peritoneal dialysis was instituted on day 3 by infusing 10–15 l of warmed fluids into the abdomen one time per day and then draining the fluids after 30 min. The horse remained azotemic after 6 days of treatment, including 4 days of intermittent peritoneal dialysis. A continuous-flow peritoneal dialysis system was initiated on day 7 and maintained for 72 h. At discharge, serum BUN was 23 mg/dl, and serum creatinine was 3.0 mg/dl. Three months after discharge, the horse had normal renal parameters and was resuming athletic activity.

4. Discussion

Acute renal failure in adult horses is a frustrating disease to treat. Although many animals will respond to treatment with fluids with or without diuretics, some will remain refractory. The prognosis for horses with ARF depends on the underlying cause, duration of azotemia, response to initial treatment, and development of secondary complications. Those disease processes that result in disruption of the tubular basement membrane, such as acute interstitial nephritis and aminoglycoside toxicity, are most associated with patients who do not recover renal function. After 72 h have passed without adequate response to treatment, the prognosis for any horse with ARF is grave. When conventional treatment fails, continuous peritoneal dialysis is a viable option. Case selection is important for the success of continuous peritoneal dialysis. Horses with ARF after an episode of exertional rhabdomyolysis are probably the best candidates, because they rarely have other underlying systemic illnesses. ARF secondary to other disease processes, such as colitis, that cause systemic illness will present many challenges and potential complications to the clinician. Horses with chronic renal failure generally make poor candidates for peritoneal dialysis as a curative treatment option, although this therapy may be considered for palliative care.

Patient, owner, clinician, and nursing compliance are also all critical to the success of continuous-flow peritoneal dialysis. This is a relatively non-technically demanding system to install, but it does de-
mand ongoing nursing support. There are several potential complications that may be encountered. The egress tube may become obstructed with omentum on a daily basis and require replacement. Any omentum that is adhered to the tube may be safely removed by blunt dissection or surgical scissors at the time of tube replacement. Small obstructions may be dislodged by attaching a 1-l bag to the egress tube and back-flushing the line. In human medicine, laparoscopic omentectomy has been used to treat the blockage of peritoneal dialysis catheters and may be considered in the future of equine medicine and laparoscopic surgery. Localized peritonitis and endotoxemia were also concerns that our patient encountered along with laminitis. In small-animal medicine, peritoneal dialysis has a reported rate of peritonitis of 22%. One may assume that the incidence would be equal if not higher in large animals because of the technical demands of keeping the system clean in a barn environment. The Ingress and egress tube entrances should be covered with Ioban surgical dressing to prevent wound contamination. Strict sterile technique must also be maintained whenever tubes or connections are being changed. Throughout maintenance of the system, the patient should receive broad-spectrum non-nephrotoxic antibiotics. Culture and sensitivities from the collected dialysate should be performed if the patient begins to show signs consistent with peritonitis (abdominal splinting, fever, symptoms attributable to endotoxemia). Endotoxemia may be addressed with standard therapies of hyperimmune-plasma (4 ml/kg, IV), flunixin meglumine (0.25 mg/kg, q 6 h, IV), and polymyxin B (6000 IU/kg, q 12 h, IV), depending on clinician preference.

Horses with ARF with possible peritonitis or endotoxemia are prime candidates for the development of laminitis and should, therefore, be maintained in ice boots.

Despite minor complications, we would recommend continuous peritoneal dialysis as a treatment option for horses in acute renal failure that are refractory to other treatment. Furthermore, we strongly recommend this treatment for anuric/oliguric patients where aggressive fluid therapy is contraindicated. In the past, peritoneal dialysis has been considered a “last-resort” treatment, but we contend that early initiation in horses who have not responded to conventional treatment after 36–72 h may show reduced morbidity and mortality.

References and Footnotes