Reversal of acute kidney injury after peritoneal dialysis in a dog: a case report

P.T.C. Guimaraes-Okamoto¹, S.S. Geraldes¹, J.F.A. Ribeiro¹, A.N.L.S. Vieira¹, L.P. Porto¹, P. Barretti², M.L.G. Lourenco¹, A. Melchert¹

¹School of Veterinary Medicine and Animal Science, University Estadual Paulista, Botucatu, Sao Paulo, Brazil
²School of Medicine, University Estadual Paulista, Botucatu, Sao Paulo, Brazil

ABSTRACT: Acute kidney injury is characterised by a sudden injury to the renal parenchyma and causes defects in its excretory, metabolic and endocrine function. Dialysis therapy has been instituted in small animal clinics with the aim of removing the metabolic waste and correcting the electrolyte disturbances stemming from renal dysfunction. Peritoneal dialysis is a therapy based on the use of the peritoneum as a semipermeable membrane through which solutes and fluids are exchanged between blood from the peritoneal capillaries and the dialysis solution. This report describes a case of acute kidney injury stemming from drug therapy in a 13-year-old female mongrel canine. The patient exhibited anorexia, emesis, prostration and anuria and had a history of prolonged treatment with meloxicam. The patient also presented with azotaemia and metabolic acidosis. When anuria continued to persist after drug therapy (volume restoration, chemical and osmotic diuresis and renal vasodilation), peritoneal dialysis was instituted. Three cycles of peritoneal dialysis were performed; during the second cycle, the patient’s anuria was reversed, and at the end of the third cycle she showed a significant reduction in azotaemia, hyperkalaemia and an improved metabolic acidosis. Therefore, peritoneal dialysis showed satisfactory clinical results and reversed anuria, reduced azotaemia and electrolyte disturbances, thereby providing clinical improvement.

Keywords: dialysis therapy; small animals; renal dysfunction; acute kidney injury; blood gases; electrolytes; anuria

Acute kidney injury (AKI) is characterised by a sudden loss of renal capacity to excrete wastes, to concentrate the urine and to maintain the electrolyte balance (Cooper and Labato 2011), and can be caused by many conditions that lead to renal hypoperfusion and hypoxia (Davis 2005). AKI conditions are reported less often when compared to chronic kidney disease and account for about 30% of cases of nephropathy (Davis 2005).

Advances in veterinary medicine have made possible new renal replacement therapies in small animals, such as haemodialysis and peritoneal dialysis (PD) (Dorval and Boysen 2009). These techniques use the principles of diffusion, convection and ultrafiltration to remove metabolic waste and to correct electrolyte disturbances that result from renal dysfunction (Dzyban et al. 2000; Dorval and Boysen 2009).

PD has been used as a treatment of AKI in humans since 1923 (Labato 2000). It involves the swapping of solutes and fluid between the peritoneal blood capillaries and dialysis solution through the peritoneal membrane (Dorval and Boysen 2009). The treatment is instituted in dogs and cats with anuric or oliguric AKI with levels of serum urea above 100 mg/dl, of serum creatinine above 10 mg/dl and in cases refractory to medical therapy (Cooper and Labato 2011).

In veterinary medicine, PD is an alternative treatment for AKI, but its use is also reported in the treatment of pancreatitis, electrolyte disorders and congestive heart failure (Cowgill 2012). The present case report describes the administration and efficacy of PD in the reversal of AKI in a dog.

Case description

The patient was presented at the Veterinary Hospital of the Faculty of Veterinary Medicine
and Animal Science, UNESP Botucatu-SP. It was a 13-year-old canine female mongrel. According to the owner, the patient had been apathetic for about four days, with emesis and anorexia. She reported that the patient had been subjected to a mastectomy surgery approximately 15 days previously at a private veterinary clinic. After the surgery, therapy with meloxicam, a nonsteroidal anti-inflammatory drug, had been prescribed, and the owner continued to give the anti-inflammatory on her own even after the end of the period prescribed by the private veterinarian.

The baseline concentrations of serum urea and creatinine were determined to be 62 mmol/l and 99 µmol/l, respectively. The results of blood gas analysis showed metabolic acidosis and hyperkalaemia. The urinary output of the patient was monitored with a urethral catheter and measured hourly. Dividing this value by the weight of the patient indicated anuria (< 1 ml/kg/h over 6 h).

The clinical presentation, taken together with the background and laboratory test results, indicated a diagnosis of AKI. Fluid therapy was instituted with Ringer’s lactate crystalloid at a rate of 40 ml/kg/h, and two intravenous boluses of 4 mg/kg of furosemide were administered, at intervals of 30 min. After an additional 30 min, a continuous infusion of furosemide was introduced at a rate of 0.7 mg/kg/h and after another 30 min a bolus of 0.5 g/kg of mannitol was administered. As there was no effect, a dopamine infusion of 2.0 mg/kg/min was added to the continuous infusion of furosemide. Even after the attempts detailed above, no reversal of anuria occurred, and urinary output remained at 0 ml/kg/h. Thus, PD was instituted with the main objective of reversing the anuria.

In order to perform the technique, a PD Tenckhoff catheter (VET Medical, Brazil) was implanted in the abdominal cavity (Figure 1). The catheter insertion occurred in a sterile operating room, the patient was kept in dorsal decubitus, and a local anaesthetic block was administered for the procedure. An omentectomy was not performed as the objective was to administer PD only for a short period; this decision was made in order to reduce time under anaesthesia, and because the animal had electrolyte abnormalities and owing to the fact that the case was acute.

Three cycles of PD were performed with a commercial dialysate solution containing 1.5% of glucose (Fresenius Medical Care, Brazil). Before being introduced into the abdominal cavity, the dialysate was heated to a temperature of between 38 °C and 39 °C which was maintained throughout the procedure.

In each cycle a volume of 30 ml/kg of dialysate was introduced, which remained for 40 min in the peritoneal cavity before its recovery and discharge to a collection bag (Figure 2). During the second cycle, there was a reversal of anuria and in the following 24 h the patient had an average urinary output of 3 ml/kg/h. At the end of the third cycle, serum urea concentration decreased to 60.77 mmol/l and creatinine to 636.48 µmol/l. The serum potassium level fell from 6.02 mmol/l to 4.92 mmol/l, the pH changed from 7.22 to 7.34 and the base deficit from –12.3 mmol/l to –8.6 mmol/l (Figure 3).
In the third cycle it was only possible to recover less than 60% of the content introduced into the abdominal cavity. This retention of dialysate in the abdomen most likely occurred due to a catheter obstruction by the omentum, since the patient was not subjected to an omentectomy. After the three cycles of dialysate were completed, biochemical analysis of the recovered dialysate was performed, and showed 63.43 mmol/l of urea and 1352.52 mmol/l of creatinine. A sample of the last recovered dialysate from the peritoneal cavity was sent for fungal and bacterial culture, which gave negative results.

The PD catheter was removed after two days due to the flow obstruction, after which symptomatic treatment was continued, with crystalloid fluid therapy and nutritional support through a naso-sophageal tube.

**DISCUSSION AND CONCLUSIONS**

AKI is a clinical syndrome characterised by an abrupt increase in serum urea and creatinine (Chew 2005) followed by acid-base and fluid-electrolyte imbalance caused by kidney damage of varying degrees of severity (Cowgill 2012).

The diagnosis of this AKI report was established according to the patient history, clinical signs, haematological evaluation and measurement of urine output. According to Chew (2005), the most common cause of AKI is the chronic use of nonsteroidal anti-inflammatory drugs leading to nephrotoxicity.

Recently, the International Renal Interest Society (IRIS) created a classification system for AKI in dogs and cats, containing grades I to V as well as subgrades, according to the urine output and the need for renal replacement therapy (Cowgill 2012). According to this rating system, the patient described here fits into stage V of AKI, as it presented with azotaemia characterised by serum creatinine levels above 10 mg/dl, anuria and required renal replacement therapy.

Cowgill (2012) states that in the case of a fluid therapy treatment that does not promote optimal diuresis, the use of diuretics and vasodilators is necessary; however, these drugs are not effective in patients with severe stages of AKI. This is in accordance with what occurred in the patient described in this case report, where the AKI was refractory to conventional treatment options, and PD was chosen as an alternative course of treatment.

According to the IRIS, animals with severe renal injury in stage IV or V will most likely die if renal replacement therapies are not performed in addition to conventional therapeutic supports.

There is no consensus in the literature that indicates the best renal replacement therapy in cases of AKI (Gabriel et al. 2008). Haemodialysis would be an effective alternative in this patient; however, as in other hospitals, this was not performed due to structural, financial and location factors that limit the applicability of this technique.

In humans, PD is still the main therapy used in cases of AKI in many countries (Gabriel et al. 2008) due to its availability, ease of execution, excellent cardiovascular tolerance, and lower risk of causing electrolyte imbalances related to haemodialysis (Gallatin et al. 2005). However, PD has limitations such as the integrity of the peritoneal cavity, the risk of peritonitis and protein losses (Gabriel et al. 2008). Moreover the elimination of toxins occurs more slowly when compared to haemodialysis (Gallatin et al. 2005).

Gabriel et al. (2008), in a study with humans, compared PD to daily haemodialysis in patients with AKI. The rate of mortality and the rate of renal function recovery were similar in both groups, and the authors concluded that both are effective treatment alternatives for AKI.

Before starting PD, dehydration and hypotension must be corrected to ensure adequate systemic and
peritoneal blood flow, as low peritoneal blood flow reduces the rate of transport of molecules (Garcia-Lacaze et al. 2002). After preparing the patient, the composition of the dialysate must be decided. The ideal dialysate must promote the removal of solutes with little absorption of osmotic agents, correct electrolyte disturbances, inhibit the growth of microorganisms and be minimally harmful to the peritoneum (Cooper and Labato 2011).

The concentration of dextrose in the dialysate determines the osmotic gradient intensity and rate of movement of fluid in the peritoneal cavity (Garcia-Lacaze et al. 2002). The dextrose concentrations range from 1.5% to 4.25%. When the quick removal of a solute that endangers life is desired or the purpose is the treatment of fluid overload, dialysate with high concentrations of dextrose (2.5–4.5%) should be used; however, use may result in hypovolaemia, dehydration and electrolyte disturbances (Cooper and Labato 2011). Dextrose concentrations between 1.5% and 2.5% are used to avoid sudden changes of fluids and electrolytes (Garcia-Lacaze et al. 2002). In the patient in this study, a 1.5% solution was used, since the patient was normovolaemic.

The dialysate must be warmed to body temperature before being introduced into the abdomen (Garcia-Lacaze et al. 2002). The recommended infusion volume is in the range from 30 to 40 ml/kg (Dzyban et al. 2000), and the content should remain in the abdominal cavity for 30 to 40 min before being drained into a collection bag (Labato 2000).

According to Lacaze-Garcia et al. (2002), the volume recovered from the abdomen should be at least 90% of what was introduced. In the patient in this study, only about 60% of the input content was recovered in the third cycle of dialysis, after which the dialysis catheter was removed. According to Cooper and Labato (2011), retention of the dialysate in the abdomen caused by flow occlusion of the catheter occurred in 22% to 77% of the animals in several retrospective studies. The authors state that the most common causes of catheter occlusion include the presence of the omentum and the accumulation of fibrin in the catheter.

In this study the omentum was not removed due to the intention to conduct PD in a period of a few days. Partial omentectomy may decrease the occurrence of occlusion of the catheter by omentum (Garcia-Lacaze et al. 2002). Therefore, omentectomy is recommended when the goal is to perform peritoneal dialysis for more than three days (Cooper and Labato 2011).

In addition to occlusions of catheter flow, PD can lead to complications such as hypoalbuminaemia, peritonitis, leakage of dialysate to extraperitoneal sites, dyspnoea due to increased intra-abdominal pressure, dehydration or over-hydration and electrolyte abnormalities (Dzyban et al. 2000; Gallatin et al. 2005). To prevent these complications, the patient should be monitored during the procedure and catheter manipulation and dialysis bags should be sterile (Cooper and Labato 2011).

Crisp et al. (1989), in a study in which they performed PD in twenty five dogs and two cats, observed hypoalbuminaemia as the most common complication in eleven animals, followed by retention of dialysate due to catheter obstruction in eight animals. Of all animals studied, six survived to be discharged from the clinic. The authors attributed the low rates of survival to the severity of kidney disease that afflicted these patients.

Beckel et al. (2005) conducted a study in five dogs with AKI caused by leptospirosis, submitted to peritoneal dialysis treatment. Azotemia decreased in all dogs, four dogs became polyuric within 24 h after the start of PD and one dog became polyuric 72 h after the start of PD. These data suggest that AKI arising from leptospirosis is associated with a better prognosis, as it showed the best results when compared to other studies.

Patient selection is the key to success of PD, and it should be prescribed for patients with severe azotaemia refractory to conventional therapy for 24 h (Labato 2000). It is also important to assess the temperament of the animal and the owner’s commitment to that patient (Gallatin et al. 2005).

The goal of PD in AKI is not to normalise azotaemia immediately. The general objectives include an improvement of haemodynamics and electrolyte imbalance and a reduction of azotaemia (Cooper and Labato 2011), which were also observed in this study.

Converting an anuric or oliguric state to a non-oliguric state is an important advance in AKI therapy (Cowgill 2012). The patient described in this report left the AKI state; however, there was no full recovery of renal function, a situation also described by Cowgill (2012), who states that, in some cases, depending on the aetiology and extent of renal injury, it is not possible to recover renal function completely and the patient may develop a chronic condition.
The low success rates of PD in veterinary medicine may be because it is considered as a last alternative in patients with a poor prognosis, and also due to improper application of the technique by inexperienced professionals (Dzyban et al. 2000).

In this case, peritoneal dialysis showed satisfactory results by reversing anuria and reducing azotaemia and electrolyte disturbances, thereby providing clinical improvement. Because the treatment is easy to perform and does not require a dialysis machine, peritoneal dialysis can be an affordable and effective therapeutic option in cases of AKI refractory to conventional therapeutic options.

REFERENCES


Received: 2016–02–04
Accepted after corrections: 2016–05–23

Corresponding Author:
Priscylla Tatiana Chalfun Guimaraes-Okamoto, University Estadual Paulista, School of Veterinary Medicine and Animal Science, Botucatu, Rubiao Junior s/n, Sao Paulo, 18618970 Brazil
E-mail: tatiana@fmvz.unesp.br