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Management of acute renal failure in cats using peritoneal dialysis: a retrospective study of six cases (2003–2007)

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Information regarding the use and success of peritoneal dialysis (PD) in the management of acute renal failure (ARF) in cats is lacking. The purpose of this retrospective study is to describe the indications, efficacy, complications and outcome of cats undergoing PD for ARF. Six cats that underwent PD for treatment of ARF of various etiologies were included. PD effectively replaced renal function in all cats and allowed renal recovery in 5/6 cats. Five cats were discharged and one cat died. Complications were reported in all cats and included subcutaneous edema ($n = 5$), hyperglycemia ($n = 4$), dialysate retention ($n = 3$), and hypoalbuminemia ($n = 3$). A novel technique consisting of a Blake surgical drain and an intermittent closed suction system was used, which appears to be a viable option for PD in cats. Although complications are common, PD is an effective renal replacement therapy for ARF in cats and carries a reasonable prognosis in selected cases.

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Advancements in veterinary medicine have introduced a number of renal replacement therapies including continuous renal replacement therapy (CRRT), intermittent hemodialysis (IHD) and peritoneal dialysis (PD). These therapies use principles of diffusion, ultrafiltration and convection to remove metabolic waste products from the bloodstream and to re-establish acid–base, electrolyte and fluid imbalances that have resulted from renal dysfunction. To date, only IHD, and more recently, CRRT, have been reported with success in the treatment of acute renal failure (ARF) in cats.[1–3] These techniques require specialized equipment and trained personnel, and therefore, tend to be limited to referral and academic veterinary hospitals. PD, however, tends to be less technologically demanding, and because most veterinary hospitals already have the equipment necessary to perform PD, it often remains the only accessible method to replace renal function and prevent systemic imbalances from occurring during the period of renal recovery.

PD has been used to treat ARF in humans since 1923.[4] It involves the exchange of solutes and fluid between the peritoneal capillary blood and the dialysis solution across the peritoneal membrane. Waste products in higher concentration in the blood diffuse across the peritoneum into the dialysate and are removed with each fluid exchange. Although the veterinary literature describes the use of PD for many conditions, information is lacking concerning its use in the management of naturally occurring ARF in cats.[4–6] One retrospective study mentions the use of PD in the treatment of ARF in two cats but fails to show its efficacy in replacing renal function in the two cases. Moreover, no explanation was apparent for the failure of PD in either case.[7]

The objectives of this retrospective study are to describe the application, efficacy and clinical features of cats treated with PD for ARF of different etiologies.

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Materials and methods

Criteria for selection of cases

The medical records of cats managed with PD for ARF between January 2003 and December 2007 at the University of Montreal Veterinary Teaching Hospital were reviewed. All cats managed with PD for ARF during the study period were included. Only cats diagnosed with ARF and a potentially reversible underlying disease were selected for PD. Cats with signs suggestive of chronic kidney disease and cats believed to have an irreversible underlying disease were not selected for PD.

Data collection

The following information was recorded: age, sex, breed, clinical signs and history at presentation, suspected or definitive cause of ARF, treatment prior to PD, urine output and indication for PD. Indications for PD included presence of severe azotemia (blood urea nitrogen (BUN) >100 mg/dl and creatinine >10 mg/dl) that is unresponsive to more than 24 h of medical management (rehydration, osmotic or chemical diuresis), worsening azotemia with signs of fluid overload, and persistent oliguria (<1 ml/kg/h) or anuria (0 ml/kg/h).[4,5,8] Overhydration was defined as the presence of >pleural effusion, pulmonary edema, ascites, subcutaneous edema or a combination of these factors. Severe hyperkalemia ($K+>8$ mmol/l) and/or severe acid–base disturbances ($pH<7.2$) were considered supporting indications for PD.[6] Serum biochemistry and venous blood gas results before, during and after PD were recorded. The technique for PD catheter placement, type of dialysis catheter, type of dialysate, number and volume of exchanges, duration of PD, urine output during PD, complications associated with PD and duration of hospitalization were also recorded. Abdominal sonography, abdominal and thoracic radiography, urinalysis, and final outcome of the patients were collected. One-year follow-up was recorded when available. An acute onset of clinical signs, elevated renal parameters with low urine specific gravity (USG) and the absence of ultrasonographic evidence of chronic renal disease were defined as ARF. Outcome was classified as discharged, died or euthanased.

Statistical analysis

Results are presented as mean (±SD) or median and range. Statistical comparisons of pre- and post-dialysis serum BUN and creatinine were performed using a Wilcoxon signed rank test; $P \leq 0.05$ was considered significant.

Results

Six cats met the criteria for study inclusion. Breeds included four domestic shorthair, one Maine Coon, and one domestic longhair cat. There were four castrated males and two spayed females. Median age was 6.5 years (range 2–9). All cats were indoor cats. Clinical signs at presentation included anorexia ($n=6$), vomiting ($n=6$), lethargy ($n=5$), painful abdomen ($n=2$), dyspnea ($n=2$), dysuria ($n=1$), weight loss ($n=1$), facial twitch ($n=1$) and cardiac arrhythmias ($n=1$). Polyuria/polydipsia were not reported in any of the cats. All cats were referred for evaluation of ARF and possible PD. All cats were treated with intravenous fluids and furosemide (Salix; Intervet) for a median of 2.5 days (range 14 h to 7 days) prior to initiation of PD. One cat received mannitol (Osmitrol 20%; Baxter). Dopamine was not used in any of the cats.

All cats had a biochemistry profile, venous blood gas and urinalysis done pre- and post-dialysis. Median USG prior to PD was 1012 (range 1010–1016). Values for BUN, creatinine, potassium and urine output are reported in Table 1. Hypoalbuminemia was present in one cat (cat 5) prior to starting PD, which got worse during PD. Hypoalbuminemia developed during PD in two other cats (cats 3 and 4). All cats had metabolic acidosis at the time PD was initiated, five of which were considered severe (median 7.2, range 7.05–7.33). Several diagnostic tests were performed in an effort to identify the underlying cause of ARF including urinalysis ($n=6$), urine culture and sensitivity ($n=6$), kidney cytology ($n=3$) and pyelocentesis ($n=3$). The urinalysis revealed abnormalities in five cats, which included hematuria ($n=4$), pyuria ($n=2$), and glucosuria ($n=2$). There was one positive urine culture (*Escherichia coli*). Kidney cytology was unremarkable for two cats and non-diagnostic for one cat. Urine cytology following pyelocentesis was suggestive of pyelonephritis (pyuria) in one case and was normal in two cases. These findings were similar to the urinalysis of the respective cats. Leptospirosis serology was negative for the one cat in which it was evaluated. Snap tests for feline immunodeficiency virus and feline leukemia virus were negative for all cats. Abdominal ultrasound revealed abnormalities in all six cats which included dilated renal

Table 1. Duration of PD, and renal parameters at base-line, start of PD (following at least 24 h of medical management for ARF), at 12, 48 and 72 h after starting PD, and at the time of discharge

	Base-line*	Start of PD (>24 h medical therapy)	12 h	24 h	48 h	72 h	Discharge	PD duration, h
Cat 1								64
BUN (mg/dl)	120	325	277	114	53	30	17.3	
Creat (mg/dl)	13.7	27.7	24.7	9.7	5.0	3.3	2.0	
K+ (mEq/l)		8.2		4.2	3.6	3.9		
Urine output (ml/kg/h)		0.5	0.3	4.0	7.8	4.4		
Cat 2								72
BUN (mg/dl)	164	164	N/A	111	140	105	98.1	
Creat (mg/dl)	17.5	17.7	N/A	12.7	12.2	10.2	6.7	
K+ (mEq/l)	3.7	4.2		4.2	3.9	4.0		
Urine output (ml/kg/h)		3.0	2.7	4.8	7.2	6.8		
Cat 3								78
BUN (mg/dl)	64	202	N/A	127	99	44.8	22.3	
Creat (mg/dl)	6.5	18.0	N/A	14.7	10.5	3.8	1.7	
K+ (mEq/l)		10.9		8.8	4.5	3.9		
Urine output (ml/kg/h)		0	0	0	2.5	6.3		
Cat 4								96
BUN (mg/dl)	49	121	59	38	28	N/A	28	
Creat (mg/dl)	3.0	6.2	3.4	N/A	1.6	N/A	1.5	
K+ (mEq/l)	4.57	6.11	3.7	3.7	4.0		3.2	
Urine output (ml/kg/h)		4.0	3.0	2.0	N/A	N/A		
Cat 5								80
BUN (mg/dl)	N/A	303	N/A	189	168	127	25.9	
Creat (mg/dl)	20.1	24.0	N/A	19.5	17.0	14.6	2.3	
K+ (mEq/l)	4.7	6.2		6.1	4.4	4.4		
Urine output (ml/kg/h)		0	0	0	0	1.0		
Cat 6								53
BUN (mg/dl)	89	268	149	101	107	Died	Died	
Creat (mg/dl)	21.9	26.5	16.2	N/A	12.0			
K+ (mEq/l)	7.2	8.11		5.9	4.6			
Urine output (ml/kg/h)		0.7	N/A	1.7	1.2			

Creat = creatinine, N/A = not available, K+ = potassium. Reference ranges: BUN 12–30 mg/dl, Creat 0.6–2.0 mg/dl, K+ 3.6–5.3 mEq/l.

*Base-line values were taken prior to or during medical management, and medical management was continued for at least 24 h following these values (with the exception of cat 6 that received only 14 h of therapy prior to initiating PD).

pelvises ($n = 4$), increased renal cortical echogenicity ($n = 4$), renomegaly ($n = 3$), nephrolithiasis ($n = 2$), perirenal effusion ($n = 2$), abdominal effusion ($n = 2$) and renal mineralization ($n = 1$). No cat showed echographic abnormalities compatible with chronic kidney disease. An antemortem diagnosis for the ARF was determined in three cats, suspected in one cat and not determined in two cats. Suspected or definitive diagnosis, and indications for PD for each cat are reported in Table 2.

One dialysis drain was placed in each cat using sterile technique and general anesthesia. As previously described, a ventral abdominal incision 2–3 cm caudal to the umbilicus and just off the midline was used to place the drains.[4] A Blake silicon drain, Sil-Med, attached to a closed intermittent negative pressure system (J-Vac; Ethicon) was used for all cats. The Blake drains were connected to warmed dialysate solution and a closed suction system using a Y-connector, similar to what has been previously described.[5]

Table 2. Diagnosis, indication for, and complications noted during PD

Diagnosis	Indication for PD	Complications during PD
Cat 1		
Pyelonephritis (*E coli*) Nephroliths	Severe azotemia despite medical therapy Oliguria Overhydration Severe hyperkalemia Severe metabolic acidosis	Dialysate retention Subcutaneous edema
Cat 2 Easter lily intoxication		
	Severe azotemia despite medical therapy Overhydration	Dialysate retention Subcutaneous edema Dialysate leakage Hypokalemia
Cat 3 Suspected pyelonephritis		
	Severe azotemia despite medical therapy Anuria Overhydration Severe hyperkalemia	Loss of J-VAC pressure Hypoalbuminemia
Cat 4		
Traumatic ARF following bilateral pyelectomies Nephroliths	Progressive azotemia despite medical therapy Overhydration	Subcutaneous edema Hyperglycemia Hypoalbuminemia Progression of pleural effusion
Cat 5		
Undetermined Suspected obstruction	Severe azotemia despite medical therapy Anuria Severe metabolic acidosis	Dialysate retention Subcutaneous edema Hyperglycemia Hypoalbuminemia Pleural effusion Cardiopulmonary arrest
Cat 6		
Undetermined Chronic severe tubulointerstitial nephropathy with subacute suppurative inflammation Pulmonary adenocarcinoma	Severe azotemia despite medical therapy Oliguria Severe metabolic acidosis Severe hyperkalemia	Loss of JVAC pressure Subcutaneous edema Hyperglycemia Facial twitch Cardiopulmonary arrest

J-VAC Suction Resevoir (Ethicon, Somerville New Jersey).

Omentectomies were not performed. The time required to place the drains was available for 3/6 cats and ranged from 35 to 60 min.

The initial dialysate solution for all cats was lactate Ringer's solution mixed with a variable dextrose concentration (1.25, 2.5 or 4.5%), depending on the volume status of the patient when PD was initiated. Bicarbonate 8.4% was added to the dialysate solution of two cats. Heparin (250−1000 UI/l) was added to the dialysate of all cats. Three cats received 10 ml/kg, two received 17 ml/kg and one cat received 40 ml/kg

of dialysate solution during the initial exchanges. The frequency of dialysate exchanges and dwell times were adjusted for each animal's individual needs. Initial exchanges were performed hourly with a dwell time of 45 min for every cat. Frequency of exchanges were gradually decreased in four cats and stopped acutely in two. Subcutaneous leakage occurred while tapering PD in one cat, which necessitated premature stoppage of PD. In two cats PD was acutely stopped due to cardiopulmonary arrest. Median duration of the PD was 75 h (range 53–96 h). The cat that was unsuccessfully resuscitated had 53 h of PD prior to cardiac arrest. Additional therapies instituted during PD were based on preferences of the attending clinician and included antibiotics, calcium gluconate, famotidine (Famotidine; Omega), Continuous rate infusion (CRI) of insulin (HumulinR; Eli Lilly), butorphanol (Torbugesic; Wyeth), metoclopramide (Metoclopramide; Sandoz), odensetron (Zofran; GlaxoSmithKline) and supplemental oxygen.

There was a statistically significant decrease in the mean pre- and post-dialysis values for BUN and creatinine ($P < 0.03$). The hyperkalemia noted in five cats resolved within 48 h of PD. Adjuvant therapy (dextrose ± regular insulin) for hyperkalemia was administered to four cats. Urine output was measured in all cats. Median urine output before PD was 0.6 ml/kg/h (range 0–4 ml/kg/h). Two cats had urine output > 2 ml/kg/h prior to PD while two were anuric and two were oliguric. The two anuric cats became acutely polyuric (urine output > 2 ml/kg/h) 48 h (cat 3) and 85 h (cat 5) after the initiation of PD. One of the two oliguric cats became polyuric within 17 h of PD (cat 1) while the urine production decreased progressively for the second oliguric cat that eventually arrested (cat 6).

Nutritional support was provided to five cats during PD. A nasoesophagostomy tube was placed in two cats, an esophagostomy tube was placed in one cat, and force-feeding was used in one cat. Partial parenteral nutrition (PPN) was provided for 72 h in one cat, which was subsequently switched to total parenteral nutrition (TPN) for an additional 2 days. One cat arrested before institution of nutritional support. Median time before institution of nutritional support was 48 h (range 24–72 h).

Evidence of overhydration prior to PD was present in four cats and included subcutaneous edema ($n = 4$) pleural effusion ($n = 3$), pulmonary edema ($n = 2$) and ascites ($n = 2$). Two cats that presented with respiratory signs secondary to pleural effusion and pulmonary edema showed clinical (improved respiratory effort and improved breath sounds on auscultation of the thorax) and radiographic improvement within 24 and 48 h of PD, respectively. Progression of pleural effusion occurred during PD in one cat. One cat with normal thoracic radiographs prior to PD developed pleural effusion during dialysis. Subcutaneous edema developed ($n = 2$), remained unchanged ($n = 1$) or progressed ($n = 3$) during PD.

Complications associated with PD are reported in Table 2. Cardiopulmonary arrest occurred in two cats; one that was anuric and one that was oliguric. Cardiopulmonary resuscitation (CPR) was successful in the anuric patient whom eventually left the hospital while CPR was unsuccessful in the oliguric cat. A CRI of regular insulin was used in two cats to treat persistent hyperglycemia (glucose > 17 mmol/l (305 mg/dl)) that developed during PD. One cat responded to the CRI of insulin, which was adjusted to maintain a glucose concentration between 8 and 13 mmol/l (144 and 234 mg/dl), while the other cat showed no response to insulin therapy. Hyperglycemia in a third cat returned to normal with a decrease in the frequency of dialysate exchanges.

Five cats were discharged from hospital and one died. Median duration of hospitalization was 11.5 days (range 3–15). All cats were continued on intravenous fluids for at least 24 h (median 2 days, range 1–9 days) following PD. Renal parameters of three cats were normal at discharge (see Table 1). Two cats were discharged with supplemental fluid therapy (40 ml/kg sid SQ and 6 ml/kg sid SQ, respectively). The renal profiles of these two cats returned to normal 1 week and 6 months following discharge, respectively. The former was on a tapering dose of subcutaneous fluids (12 ml/kg q48 h) and had normal renal parameters at the time this paper was written (3 months following discharge), while the latter was no longer receiving subcutaneous fluids. Four cats were available for 1-year follow-up: no cat required medical therapy for renal disease, all had normal renal profiles and none were receiving subcutaneous fluids.

Discussion

PD effectively replaced renal function in all cats of this study, and allowed renal recovery in 83% of cases (5/6). Retrospectively, the rapid

development of polyuria and the reversible nature of the underlying diseases likely explain the high success rate of patients treated with PD in our study. These results are more encouraging than what has historically been reported for PD in the veterinary literature. In a study of 25 dogs and two cats treated with PD for ARF or azotemia, only 6/27 patients (22%) survived to discharge.[7] However, a more recent study showed 4/5 dogs (80%) treated with PD for ARF secondary to leptospirosis survived to discharge, suggesting that patients with leptospirosis may have a better prognosis.[9] Unfortunately, data specific to feline ARF treated with PD is limited to experimental studies and a retrospective study that evaluated only two cats.[7,10] The two cats in the latter study had an unsuccessful outcome, and it is unclear if PD decreased renal parameters in these two cases. Although the number of cases in the current study is small, the results are more in line with the success of cats treated with IHD for ARF of various etiologies (60%) and suggest that PD remains a viable option in the treatment of selected cats with ARF.[1–3]

PD was initiated despite the absence of a definitive diagnosis in half of the patients of this study (3/6). A potentially reversible underlying disease and signs suggestive of ARF influenced the decision to perform PD. Transient ureteral inflammation associated with surgical trauma, pyelonephritis and possible transient ureteral obstruction, which are all reversible conditions, were identified or suspected to be the cause of ARF in most of the cats of this study. It remains speculative if these cats might have become polyuric and survived with continued medical therapy if PD had not been initiated. However, given the progressive nature of the azotemia despite more than 24 h of medical therapy prior to initiating PD, and/or the persistence of oliguria/anuria with signs of overhydration, PD was considered necessary to help re-establish homeostasis and manage azotemia until renal function improved. It is anticipated that cases that have more intractable underlying renal disease would have developed more complications as a result of longer-term PD and the prognosis would not be as favorable. In all cases the azotemia, electrolyte and acid–base disorders improved within 24 h of PD. This improvement is likely the result of PD as urine output did not change significantly during the first 24 h of dialysis, with the exception of one cat that developed polyuria after 17 h of PD. Polyuria that developed in two cats within 48 h of starting PD likely contributed to the resolution of fluid overload, electrolyte, and acid–base disorders, however, the same improvements were observed in two cats that did not develop polyuria and remained anuric or oliguric during the same period of time. Our results suggest that signs compatible with ARF (or exclusion of signs compatible with chronic disease), in association with other indications for renal replacement therapy, might be sufficient to support the decision of initiating PD even if the inciting cause cannot be confirmed.

The complication rate associated with PD in cats using the current technique is high (100%). Previous reports of PD in small animals have also reported frequent complications including hypoalbuminemia, hypochloremia, hypokalemia, catheter obstruction, dialysate retention, peritonitis, subcutaneous leakage of dialysate and limb edema.[7,9,10] Comparison between PD and IHD is difficult because of the limited number of veterinary studies and the small number of cats in the present study. In one study, 69% of cats treated with IHD had one or more dialysis-related complications. IHD-related events included hypotension, dialysis dysequilibrium, clotting and bleeding.[1] Anemia, dyspnea, thrombosis in the right atrium, respiratory and cardiopulmonary arrest are among other significant complications reported to occur with IHD.[1,5,11] Significant hypothermia and hypocalcemia were the two major complications encountered in an ARF cat treated with CRRT.[3]

Subcutaneous edema was the most frequent complication encountered in this study (83%) and likely resulted from a combination of dialysate leakage, hypoalbuminemia and overhydration. Intermittent wrapping of the limbs, as previously described, helped to promote mobilization of the edema.[8] Dialysate leakage was evident in one case that resulted in progressive subcutaneous edema and a decrease in the efficacy of the PD. Increased intra-abdominal pressure associated with the volume of dialysate used may have contributed to dialysate leakage. Starting the initial exchange volumes at or below 10 ml/kg, or decreasing the amount of fluid infused throughout dialysis may prevent leakage, although smaller volumes of dialysate could also decrease the efficacy of PD. The use of Dacron cuffs for longer-term dialysis, minimizing manipulations of the PD catheter, optimal surgical technique and good postoperative care may also decrease the risk of dialysate leakage.[8,12]

Hypoalbuminemia occurred in 50% of cats (3/6) in the present study, which is similar to what has previously been reported.[9] Hypoalbuminemia secondary to PD is likely the result of loss of albumin in the dialysate. Gastrointestinal and renal losses, uremic catabolism, concurrent disease, production of acute phase proteins, and low protein intake are additional causes that likely contribute to the development of hypoalbuminemia in uremic patients. The fact that hypoalbuminemia is a frequent complication of dialysis indicates that attention should be given to nutritional intake. In people, there is evidence to suggest that protein malnutrition is a significant risk factor for morbidity and mortality in dialysis patients.[13] All of the hypoalbuminemic cats in the current study received some form of nutritional support, however, given they were anorexic for several days prior starting PD, earlier nutritional support may have prevented or decreased the severity of hypoalbuminemia. In addition, the amount of protein provided via PPN and TPN was 1.5 g/kg/day and 2 g/kg/day, respectively, which may be suboptimal for patients undergoing dialysis.[6] The use of amino acid based dialysate solutions may provide a means to decrease the development of hypoalbuminemia during PD. A study in people evaluating daily protein losses with the use of a 1.1% amino acid dialysate solution showed that the amino acids absorbed during PD more than adequately replaced typical daily dialysate protein losses.[14] Ultrafiltration rates for 1.1% amino acid solutions have been shown to be similar to 1.5% dextrose solutions.[14] Clinical studies evaluating the risks and benefits of amino acid solutions in veterinary patients have not been performed.

Hyperglycemia, which has been reported in cats receiving nutritional support,[15] was present in 3/6 cats. Rapid absorption of glucose from the dialysate into the blood, which is reported at rates of up to 80% in people,[13] likely contributed to the incidence of hyperglycemia in our study. The fact that hyperglycemia tended to develop prior to nutrition initiation also support that PD played a major role in this complication. Hyperglycemia is a documented complication in humans undergoing PD and a negative association between the severity of hyperglycemia and outcome has been reported. For this reason, insulin therapy is sometimes used in non-diabetic people that develop hyperglycemia during PD.[16] Recommendations regarding the treatment of persistent hyperglycemia in cats undergoing PD cannot be made at this time, however, if a CRI of regular insulin is started the patient should be closely monitored for the development of hypoglycemia. Glucose-sparing dialysate solutions containing icodextrin or amino acids have been investigated in people in an effort to avoid the metabolic complications of glucose, however, long-term studies are lacking and dextrose solutions currently remain the most common solution used in people and veterinary patients.[9,12]

Facial twitching was present in one cat at the time of admission, and developed in a second cat shortly after starting PD. Serum calcium and magnesium were assessed in both cats and were normal. Uremic encephalopathy was suspected to be the cause of twitching in both cats as the twitching gradually decreased following initiation or continuation of PD, with no other therapies being administered for the twitching. Dialysis dysequilibrium syndrome could not be ruled out for the second cat as the twitching developed after the initiation of PD and dialysis disequilibrium symptoms may resemble signs of uremic encephalopathy.[17]

Although catheter obstruction has been reported as high as 20–30% in small animals undergoing PD,[7,9] it was not a problem in the cats of the present study. The use of heparin in the dialysate to decrease fibrin formation may have helped prevent obstructions. The use of a Blake surgical drain, which is based on a fluted design, may have also helped prevent catheter obstruction. The Blake surgical drain has been used with success for PD in human infants,[8] but to the author's knowledge has never been used clinically for PD in veterinary patients. This drain functions similar to the fluted T drain which has been investigated in dogs and demonstrated a low rate of obstruction when compared to other PD catheter types.[6] Although not specifically designed for PD, the Blake drain allowed easy ingress and egress of dialysate exchanges with no catheter obstruction. It has also been suggested that omentectomies may be helpful in decreasing the risk of PD catheter obstruction, however, they were not performed in the current study due to the prolonged anesthesia required to perform an omentectomy.

The use of negative pressure during the egress phase of PD has been reported in people[18] and may have further contributed to the success of peritoneal drainage in the cats of the current study. The advantage of intermittent closed suction drainage during PD is that it may help

decrease abdominal dialysate retention by actively removing fluid from the peritoneal cavity during the egress phase. It may also allow more complete dialysate exchanges over a shorter period of time, thereby improving the efficacy of PD.[18] However, there were complications associated with the J-Vac, which included loss of negative pressure due to breakdown of the reservoir material. This was easily corrected by replacing the negative pressure collection system.

Dialysate retention has been described as the failure to recover 90% of the dialysate volume infused and occurred in three cats of the current study.[8] Subcutaneous dialysate leakage and/or abdominal absorption of dialysate solution were believed to be the causes of dialysate retention. The significance of dialysate retention depends upon the hydration status of the patient, the location where the positive fluid balance accumulates, the quantity of dialysate retained, the underlying renal function and the patient's ability to excrete excess fluids, and concurrent underlying disease conditions such as heart failure. In two cats that showed signs of overhydration during PD, dialysate retention was also present, suggesting it may have contributed to the state of overhydration. Promoting ultrafiltration by using higher dextrose concentrations (3.0−4.25%) in the dialysate may be necessary to prevent abdominal absorption of dialysate.

PD may have corrected pre-existing overhydration in two cats (decreased pleural effusion with no increase in urine ouput); however, it likely contributed to the development or progression of pleural effusion in two other cats. In the latter two cases, a combination of hypoalbuminemia, dialysate retention, overhydration, and the passage of dialysate from the peritoneal to the pleural space via diaphragmatic lymphatics may have contributed to the development or progression of pleural effusion during PD. The dialysate is believed to pass through diaphragmatic lymphatics in response to pressure gradients that develop across the diaphragm as a result of adding fluid to the peritoneal space.[5] Given the small number of patients, and the variation in signs regarding improvement versus progression of overhydration, it is difficult to draw conclusion concerning the efficacy of PD to correct overhydration in the cats of our study, and further studies would be required to answer this question. When overhydration occurs, ultrafiltration, through the use of more hyperosmolar dialysate solutions, may be more effective in correcting the problem. However, it should be noted that the rapid absorption of dextrose from the peritoneum into the vascular system may limit the effectiveness of dextrose solutions in achieving ultrafiltration and prevent PD from optimizing volume control.[13]

This is the first study to describe and report positive outcomes in cats treated with PD for ARF. PD represents an effective renal replacement therapy for ARF in cats non-responsive to medical therapy and carries a reasonable prognosis in selected cases. Although short-term complications are common, they can usually be managed and are rarely fatal. Despite the small number of cases, our results suggest complete renal recovery is possible following PD, and that long-term complications associated with its use are uncommon. The Blake surgical drain and an intermittent closed suction system appear to be a reasonable choice for PD in cats. Although morbidity associated with PD in our study was high it remains a viable alternative to veterinarians in private practice that may not have access to IHD or CRRT.

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