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Peritoneal dialysis in acute kidney injury: Your questions answered

Jonathan S Chávez-Iñiguez[1,2], Jahir R Camacho-Guerrero[1,2] and Daniela Ponce[3,4]

Abstract

In acute kidney injury, no dialytic method has been shown to be superior in terms of mortality or recovery of kidney function. Peritoneal dialysis is an excellent treatment option, since it is continuous and can be adapted to clinical needs, it offers hemodynamic stability, adequate solute clearance, correction of electrolyte and acidosis disorders, appropriate ultrafiltration in volume overload, it is cheaper, does not require anticoagulation, provides calories, can be performed at the patient's bedside, its prescription is simple and does not require such sophisticated machinery. And when compared with other modalities, it has been shown to be equally efficient and safe, although its use is limited, partly due to the lack of knowledge and experience with this modality. Existing clinical evidence has consistently shown that this modality has very similar results in terms of the most relevant objectives evaluated in AKI. This modality offers certain advantages in specific contexts of acute kidney injury, such as cardiorenal syndrome, hepatorenal syndrome, in unstable patients on vasopressors, and in neurocritical patients. For all these reasons, we believe that peritoneal dialysis in acute kidney injury has sufficient arguments to be implemented more frequently and receive the value it deserves.

Keywords

Acute kidney injury, kidney replacement therapy, peritoneal dialysis

Take home points

- With acute kidney injury (AKI), one in ten patients will need to start kidney replacement therapy (KRT), this incidence increases in the intensive care unit (ICU).
- The most rigorous clinical evidence has demonstrated the efficacy and efficiency of PD in the most relevant outcomes in the management of patients with AKI, when compared with continuous, hybrid or intermittent KRT.
- PD in AKI offers hemodynamic stability, appropriate solute control, correction of acid-base complications, adequate ultrafiltration and can be dosed according to clinical needs.
- It is essential to know the benefits and limitations of PD in AKI; understanding and knowing how to treat complications associated with PD in AKI will result in better outcomes and expand its use.
- In AKI, peritoneal dialysis (PD) is an economical option, the necessary material is available in most hospitals, the catheter can be placed at the patient's bedside, it does not require anticoagulation, and it has been tested in diverse causes of AKI.

Case vignette

A 68-year-old, 65-kg man with chronic kidney disease G4 A3 (eGFR 28 mL/min/1.73 m^2 and albuminuria 323 mg) was admitted to the intensive care unit for pneumonia and septic shock. Two hours after the furosemide tubular test 120 mL of urine were reported. Two days later, he developed acute kidney injury KDIGO 3, serum creatinine 5.2 mg/dL, BUN 120 mg/dL, hyperkalemia of 5.2 mEq/L, metabolic acidosis, and fluid overload by VExUS 2, which had not improved despite management. Hence, the nephrology team was consulted.

[1]Nephrology Service, Hospital Civil de Guadalajara Fray Antonio Alcalde, Guadalajara, Jalisco, Mexico
[2]University of Guadalajara Health Sciences Center, Guadalajara, Jalisco, Mexico
[3]Botucatu School of Medicine, University of Sao Paulo State- UNESP, Botucatu, Brazil
[4]Nephrology Service, Clinical Hospital from Botucatu School of Medicine, Sao Paulo, Brazil

Corresponding author:
Jonathan S Chávez-Íñiguez, Nephrology Service, Hospital Civil de Guadalajara Fray Antonio Alcalde, Hospital 278, Colonia Centro. C.P. 44150. Guadalajara, Jalisco, Mexico.
Email: jonarchi_10@hotmail.com

What is the role of peritoneal dialysis in the management of acute kidney injury?

Approximately 8% of patients with acute kidney injury (AKI) start kidney replacement therapy (KRT).[1] Owing to the advent of new technologies, quality improvement strategies, and the understanding of this syndrome, the probability of death in this scenario has been reduced by 40%[1]; approximately 57% of these people die during follow-up.[2] In the case of AKI that warrants the start of KRT, choosing the best modality is a complex decision that involves multiple clinical, laboratory, and infrastructure capacities and, above all, knowledge of KRT techniques. These include hemodynamic instability, cerebral edema, fluid overload, uremic toxins, bleeding risk, mobility, and the need for rapid correction of uremic emergencies.[3]

Peritoneal dialysis (PD) has been a viable option for almost 100 years and has consistently shown efficiency, effectiveness, and acceptable results compared to other techniques in AKI. However, we believe PD's role in AKI has been historically underappreciated. In this review, we aim to discuss the reasons for this and highlight the potential advantages of PD in the management of AKI.

In general, it is perceived that PD for AKI is inefficient. In a survey of nephrologists and intensivists focused on treating AKI, only 5% used PD as a KRT modality,[4] which is in line with that reported in Latin America, where PD in AKI It is the modality that is used least frequently, and 79% of respondents reported not using it.[5] The PD modalities used to treat AKI are diverse, but the so-called "high volume PD" (HVPD) has been the most explored. This involves rapid volume exchanges within a short time and is prescribed according to uremia severity and fluid overload,[6] this prescription is practically unique for AKI scenarios and has been modified as a result of clinical trials demonstrating that PD is effective even with lower doses. This can be performed using a cycler machine or manually.

After discussion, it was decided to start KRT, considering the hemodynamic instability with the need of vasopressors, metabolic complications and overload, PD was prescribed. According with the desire expressed in the nephrology consultation 6 months before, in the scenario that it was necessary due to the progression of CKD. The nephrology team decided implanted the PD catheter in the ICU.

Is it feasible to urgently start peritoneal dialysis in acute kidney injury?

Yes, PD can be started immediately after catheter placement in people with AKI. It is critical to consider the circumstances that could interfere with appropriate PD treatment and therefore should be thoroughly discussed. Box 1.

Furthermore, in the context of AKI, some patients are critically ill in the ICU, and the placement of the PD catheter can be performed in bedside using local anesthesia or mild sedation without the need to go to the operating room. At many centers nephrologists are involved in placement of catheter in patients, although the surgical team and interventional radiologists are also involved. The procedure usually takes approximately 30 min, has a low risk of bleeding, does not require specialized equipment, and requires minimal disposable material. Soft, straight catheters with double Dacron cuffs (in pediatrics single Dacron cuffs are used) are preferred.[7]

Owing to the need to urgently initiate PD in AKI, it was started immediately after catheter placement. The most commonly used techniques for peritoneal catheter placement are percutaneous, laparoscopic and open surgery. The choice depends on local practice, the type of catheter to be placed and the characteristics of the patient. The percutaneous technique with or without a peel-away sheath has the advantage of being able to be placed at bedside, it is a minimally invasive procedure, and the puncture can be guided with ultrasound to ensure proper placement. The laparoscopic technique is minimally invasive and allows for omentopexy, rectus sheath tunneling, and adjunctive procedures such as adhesiolysis, resection of epiploic appendices, salpingectomy, and colopexy. Another advantage is that the catheter placement is observed at all times. Open surgery is usually performed by surgeons, it is appropriate for those who have had multiple abdominal surgeries, in people with obesity and where the abdominal cavity can be visualized, it may be the most expensive technique.[8]

Here, we describe a technique that has been performed at our hospital for 15 years. We used a Tenckhoff-type catheter, a peritoneal catheter made of silicone, with a length of 42 cm, including, two cuffs, Biosil ®, a titanium connector, and a Y-shaped transfer line. The PD catheter was placed at bedside. It was administered percutaneously with prophylactic ceftriaxone, local anesthesia with 2% lidocaine, and, if needed, conscious sedation with dexmedetomidine. A straight silicone catheter was placed in the right pelvic cavity guided by ultrasound through a paramedial incision 3 and 4 cm from the umbilical scar; one cuff was located in the rectus abdominis and the other in the subcutaneous tissue. To ensure the proper functioning of PD, cycles began immediately after catheter placement. Initially, three manual exchanges were performed, input by output, followed by automated exchanges using the Pacifica® machine. These are tailored to the patient's needs, with dwell times ranging from 1.5 to 2.0 h, adjusted at the discretion of the nephrology team.[9] The glucose concentration used was determined based on the presence of fluid overload.

If flexible catheters are not available, it is recommended to use a rigid stylet catheter. These allow for an appropriate flow of dialysis fluid, can be placed on the patient's bedside, and are very inexpensive, but have the disadvantage of presenting more mechanical dysfunctions and increase the risk of intestinal perforation during placement.[10] There are

alternatives in case the recommended catheters are not available, such as inserting nasogastric tubes with side holes cut into the tube prior to insertion into the peritoneal cavity, intercostal drainage tubes, hemodialysis catheters and percutaneous cavity drainage catheters.[11,12] Although these strategies have saved lives, they have significant drawbacks such as flow problems and leaks. They should be reserved for cases where no other option is available.

With the intention of correcting uremia, fluid overload, hyperkalemia, and metabolic acidosis, PD sessions were prescribed immediately after catheter placement, with a cycling machine.

How should we prescribe the dose of peritoneal dialysis in acute kidney injury?

The PD dose for treating patients with AKI is higher than the usual dose for patients with end-stage kidney disease (ESKD). The goal is rapid and efficient correction of uremic and electrolyte emergencies, metabolic acidosis, or congestion. For context, patients with ESKD are dosed at a Kt/V close to 0.24 per day, while in cases of AKI, it is almost double.

The adequacy of the dose of HVPD depends on clinical needs, but it is estimated that the first days of initiation should meet the requirements proposed by the Brazilian group,[13–15] which is to prescribe a Kt/V of 0.5 per day of treatment, which can be estimated with the equation in the Box 2.

In a more simplified way, PD in AKI could be prescribed as recommended by the current International Society of Peritoneal Dialysis Guideline in 2021, which recommends prescription according to weight in kg; an acceptable dose is given with a cycling machine, using 2 L bags; in a person weighing <60 kg, 60–80 kg and >80 kg, they would need 1.5 L for 2 h, 2 L for 2 h, and 2 L every 1.5 h, respectively.[10] These HVPD doses may not be strictly necessary; a clinical trial revealed that giving half the dose, i.e., Kt/V of 0.38 compared to 0.62, is sufficient to maintain adequate metabolic control and the same rate of survival,[16] as shown in Figure 1.

In the absence of cyclers, PD can be performed manually. In the absence of commercially prepared dialysis solution, it can be manufactured locally, the appropriate volume of 50% dextrose is drawn up into a syringe, and injected into a bag of lactated ringer's solution, to produce 1.5%, 2.5%, or 4.25% dextrose dialysate. A cohort reported that clinical evolution and mortality were similar in AKI patients with PD when comparing the artisanally prepared bags, compared to the commercial ones.[17] This provides an accessible option to prescribe a safe and efficient PD in low-resource settings.

The dose of PD in AKI should be dynamic and adjusted to the clinical trajectory and patient needs. In addition to Kt/V, the serum electrolytes, BUN, acid-base status and fluid overload should be evaluated. If the goals of solute reduction are still not reached, the dose can be increased, with short cycle times (1–2 h), they may be increased to 4–6 hourly once the above are controlled and facilitate clearance of larger molecular sized solutes. In the case of volume overload, the glucose concentration must be increased.

In critically ill patients with shock or liver failure, it is common to find alterations in lactate metabolism with hyperlactatemia. In these cases it is recommended to use DP bags with bicarbonate. These DP solutions have been shown to increase pH and serum bicarbonate more quickly.[18]

What is the performance of peritoneal dialysis compared to other kidney replacement therapies in the management of acute kidney injury?

Encouraging results were observed when comparing PD with other techniques in the treatment of AKI as shown in Table 1. A matched cohort study comparing the 30-day risk of death in patients with AKI receiving intermittent hemodialysis (HD) (3/week) and low-dose PD (<18 L/day) found the same mortality and KRT dependency at follow-up.[19] HVPD in critically ill patients with AKI has excellent performance, stabilizing solutes such as blood urea nitrogen (BUN), serum creatinine, serum bicarbonate, pH, and potassium in just 3–4 days, very similar to that observed with other KRT techniques.[20] Comparing HVPD with daily HD in the ICU, 120 patients were randomized, 62% of them had hemodynamic instability with a mean APACHE II score of 27, the trajectory of uremic solutes and electrolytes was similar, as was the overall survival at 30 days ($p > 0.05$). However, PD was associated with two fewer days of treatment and a faster resolution of AKI by three days (both $p < 0.05$).[21] In a clinical trial that randomized 75 patients in the ICU, 75% received vasopressors and mechanical ventilation (MV) to receive low-volume PD (18–24 cycles/day), compared to intermittent HD (3/week), and observed the same kidney recovery at 28 days (40%), metabolic control, and ultrafiltration goals ($p > 0.05$ in all).[22] Another clinical trial by the same group of researchers compared HVPD with prolonged intermittent renal replacement therapy (PIRRT), randomizing 143 critically ill patients; 45% had sepsis, 86% had MV, 67% had oliguria, and 70% had fluid overload. The results were similar between the groups in terms of solute control, electrolytes, and ultrafiltration during the 5 days of treatment, with no differences in survival and recovery of kidney function.[23] When comparing HVPD with continuous renal replacement therapies (CRRT) in a clinical trial of 125 patients in the ICU, PD was administered 25 L per day, compared with CRRT at a dose of 30 mL/kg/hr. PD is associated with fewer infections, better kidney function recovery, faster resolution of AKI, 10 fewer days in the ICU and lower mortality (30 vs. 53%).[24]

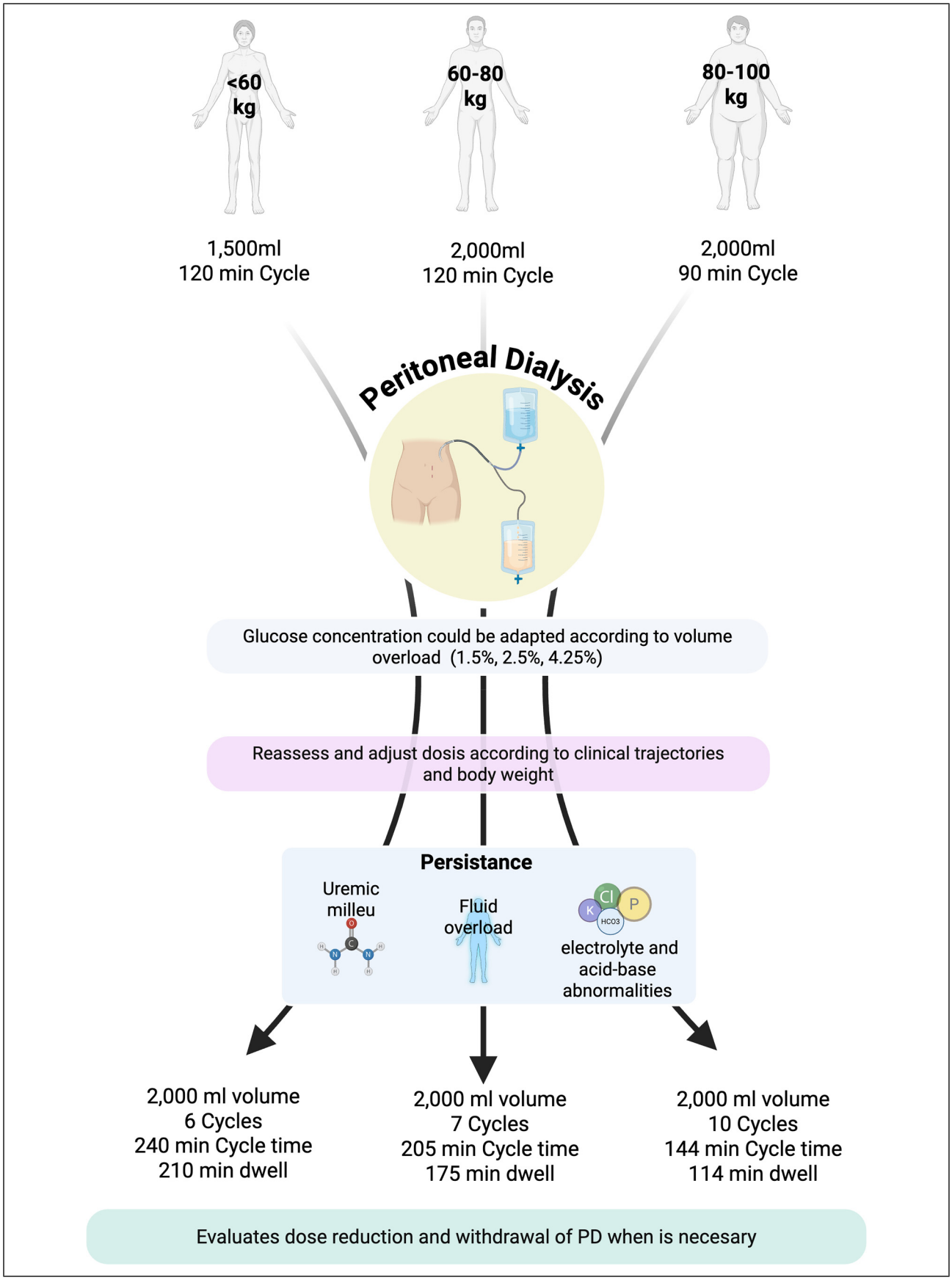


Figure 1. PD dose in AKI.

Table 1. Outcomes between PD with other techniques in the treatment of AKI.

Author	Type of modality against which it was compared	Study design	n	Characteristics	Results	Comments
DP Gabriel et al. 2008[28]	HVPD vs DHD	RCT	120	AKI due to ATN	Mortality: HVPD 58% vs. HD 53% ($p = 0.71$) Kidney function recovery: HVPD 83% vs. HDD 77% ($p = 0.84$)	– No differences in infections, mechanical complications. – HVPD was associated with faster recovery vs. HDD (7.2 ± 2.6 vs. 10.6 ± 4.7 days, $p = 0.04$)
Al-Hwiesh *et al.* 2018[29]	PD Vs. CRRT	RCT	125	Hemodynamically unstable	Mortality: PD 19% vs. CVVHD 33% ($p = 0.0028$) Kidney function recovery: PD 38% vs. CVVHD22% ($p = 0.0056$)	Fewer days for Kidney function recovery PD 5 vs. CVVHD 8 ($p = 0.0044$) More infections PD 6% vs. CVVHD 11% ($p = 0.0084$)
Ponce *et al.* 2012[30]	HVPD vs EHD	RCT	143	AKI due to ATN, hemodynamically unstable	Mortality: HVPD 63.9% vs. HD 63.4% ($p = 0.94$) Kidney function recovery: HVPD 29.6% vs. HD 26.9% ($p = 0.11$)	Despite better BUN and sCr control ($p = <0.05$) with HD, there were no differences in mortality or need for chronic dialysis
Phu *et al.* 2002[31]	PD vs HF	Open Randomized Comparison	70	Malaria falciparum 48 patients Sepsis due to other causes 22	Metabolic control: sCr control and acidosis 2 times higher in hemofiltration ($p = <0.005$) Mortality: PD 47% vs. Hemofiltration 15% ($p = 0.005$)	In the DP group, a rigid catheter was used, with a dwell time of 30 min and manual exchanges
Parapiboon 2017[32]	PD high dose (36L) vs. low dose (18L)	Randomized Pilot Study	75	AKI due to sepsis and cardiorenal syndrome	Mortality: HVPD 79% vs. PD low dose 63%, $p = 0.13$ Dialysis dependence: HVPD 2.6% vs. PD low dose 8.3%; $p = 0.29$	HVPD Kt/v 0.67 PD Low dose Kt/v 0.36 -Same outcomes for a lower Kt/v, with a tendency towards a lower risk of infection, 15.3% vs. 8.3% ($p = 0.34$)
Parapiboon 2024[33]	PD minimum standard dose (18L-24L) Vs. iHD 3 times a week	RCT	157	AKI sepsis	Mortality: PD minimum standard dose 50% vs. iHD 49%, RR 0.60, 95% CI, -15.0–16.3	PD minimum dose Kt/v 2.11 week iHD Kt/v 2.87
George *et al.* 2011[34]	DP Vs. CVVHDF	Randomized Pilot Study	50	AKI sepsis, ATN	Composite correction of urea, sCr, acidosis, hyperkalemia and fluid overload: Continuous PD 56% vs. CVVHDF 48% ($p = >0.05$) Mortality: PD 72% vs. CVVHDF 84% ($p = 0.49$)	– Faster acidosis correction in PD ($p = <0.001$) – Higher ultrafiltration in HDFCVV group 20.31L vs. PD 5.31 L

ATN: acute tubular necrosis; CVVHD: continuous veno-venous hemodialysis; CVVHDF: continuous veno-venous hemodiafiltration; DHD: daily hemodialysis; EHD: extended daily hemodialysis; HF: hemofiltration; HVPD: high volume peritoneal dialysis; ICU: intensive care unit; iHD: intermittent hemodialysis; iPD: intermittent peritoneal dialysis; NS: no significant; PD: peritoneal dialysis; RCT: randomized control trial; RR: relative risk; sCr: serum creatinine; TPD: tidal peritoneal dialysis.

Table 2. Mechanical complications of PD in AKI.

Complication	Definition	Mechanism	Clinical presentation	Treatment
Leaks	Fluid leakage outside the catheter lumen and/or peritoneal cavity	Disruption of the integrity of the cavity due to increased intra-abdominal pressure or a catheter defect.[28]	Decreased ultrafiltration Clear leak from exit site Wall edema	Peritoneal rest for 24 h (if the patient's condition allows it).[29] Decrease infused volume Supine position Perform a drawstring at the insertion site in the parietal fascia of the rectus abdominis muscle
Flow alterations	Changes in entry and/or exit times in peritoneal dialysis flows	Output: Catheter tip migration (constipation), bladder distension (extrinsic catheter obstruction) Bidirectional: Fibrin, obstruction, Kinking	Delay in infusion time and/or drainage of peritoneal fluid	Laxatives Vigorous flushing of the catheter to remove fibrin Infuse fibrinolytic agent Alpha maneuver[30] Removal and repositioning of the catheter in refractory cases

The non-inferiority of PD in terms of mortality compared with other KRT (intermittent HD, PIRRT, or CRRT) has also been supported in three meta-analyses.[25–27]

Is it feasible to use peritoneal dialysis in acute kidney injury and other comorbidities such as cardiorenal syndrome, acute pancreatitis, acute-on-chronic liver disease, cerebral edema and COVID?

PD in acute cardiorenal syndromes

In congestive heart failure (CHF) that is refractory to diuretics, PD has been positioned as an excellent treatment option to improve overload and the uremic environment. It has been shown to promote decongestion, provide calories, reduce polypharmacy, improve functional class, quality of life, cardiac remodeling, reduce rehospitalization, deactivate the renin-angiotensin-aldosterone axis, remove more sodium mass, and improve kidney function.[35,36] In these cases, to maximize UF and achieve rapid and effective decongestion, the speed of exchanges can be increased, and PD fluid can be prescribed with the highest amount of glucose (2.25% or 4.5%), which will result in higher UF volumes.

Ponce et al. explored the role of HVPD in cardiorenal syndrome (CRS) type 1 patients (acute cardiac and kidney injury) in relation to metabolic and fluid control and outcome.[37] Sixty-four patients were treated by HVPD (prescribed Kt/V = 0.50/session), flexible catheter and cycler. Mean age was 68.8 ± 15.4 years. 54.7% needed intravenous inotropic agents and/or intravenous vasodilators, 31.2% were on MV, acute coronary syndrome (ACS) was the main cause of acute disease heart failure (ADHF) 48.3%, median left ventricular ejection fraction (LVEF) was 38% and the main dialysis indications were uremia and hypervolemia. BUN and creatinine levels stabilized after 4 sessions at around 50 and 4 mg/dL, respectively. Negative fluid balance (FB) and ultrafiltration (UF) increased progressively and stabilized around 2.6 L and −2.5 L/day, respectively. Weekly-delivered Kt/V was 3.0 ± 0.42, and 32.8% died. There was a significant difference between the survivors and non-survivors in age (71.4 ± 15.7 vs 63.6 ± 17.6, $p < 0.001$), main diagnosis of ADHF (ACS: 76.2 vs 34.8%, $p = 0.04$), MV (52.4 vs 20.1%, $p = 0.03$), fluid overload (FO) at predialysis moment (52.4 vs 25.6%, $p = 0.04$), and FB and UF from the 2nd to 5th dialysis session.

In a clinical trial, patients with CRS type 1, an average serum creatinine of 3.1 mg/dL and a LVEF of 30%, refractory to diuretics, were randomized to tidal PD at 12–14 cycles per day compared to extracorporeal ultrafiltration sessions of 75–120 mL/h, it was observed that creatine, LVEF, body weight, and hospitalizations were lower in the PD group.[38]

Moreover, achieving a negative volume balance in the first 5 days of PD treatment in people with CRS type 1 is associated with better survival.[39] These results are in line with a meta-analysis of 20 studies, where PD in people with diuretic-refractory CHF was associated with a 4% increase in LVEF, 1.3 points in functional class, and reduced days of hospitalization, with a mortality rate of 37% per year.[40]

PD in AKI with acute pancreatitis and in acute-on-chronic liver disease (ACLD)

Little evidence is available supporting the use of PD in the treatment of pancreatitis-related AKI. It has been proposed

Table 3. Metabolic complications of PD and AKI.

Complication	Definition	Physiopathology	Clinical presentation	Treatment
Protein loss	Protein loss in dialysate (6–12 gr per day, up to 48 gr in peritonitis)	Transport of macromolecules through highly permeable peritoneum (exacerbated in peritonitis).[29]	Does not affect serum albumin High transport phenotype (adequate management of solutes, with volume overload)	Diet 1.2 gr/kg/protein day Treatment of pro-inflammatory state (eg. Peritonitis)
Hyperglycemia	Glucose >180 mg/dL	Absorption of glucose from dialysis fluid (Absorption from 35.3% to 50%).[31] Systemic Inflammation is a risk factors	Glucose >180 mg/dL	-Subcutaneous, intravenous and/or intraperitoneal insulin
Hypernatremia	Serum sodium > 145 mmol/L	Volume loss due to osmotic effect of dialysis fluid, without sufficient time for sodium equilibrium between plasma and dialysis fluid.[31] Risk factor: short exchanges	nausea, vomiting, disorientation, gait disturbance, seizures	Avoid short exchanges whenever possible, to ensure an adequate balance between plasma and dialysate Intravenous or oral hyposmolar replacement fluids
Hypokalemia	Serum potassium <3.5 mmol/L.[32]	Low potassium intake Renal or intestinal potassium losses Potassium loss in dialysate due to low potassium concentration	Muscle weakness, cramps, electrocardiographic changes, sudden cardiac death	Eliminate triggering factors (diuretics, diarrhea) Correct hypomagnesemia Intraperitoneal, intravenous and/or oral replacement depending on severity Spironolactone has shown an increase potassium of up to 0.7 mmol/l

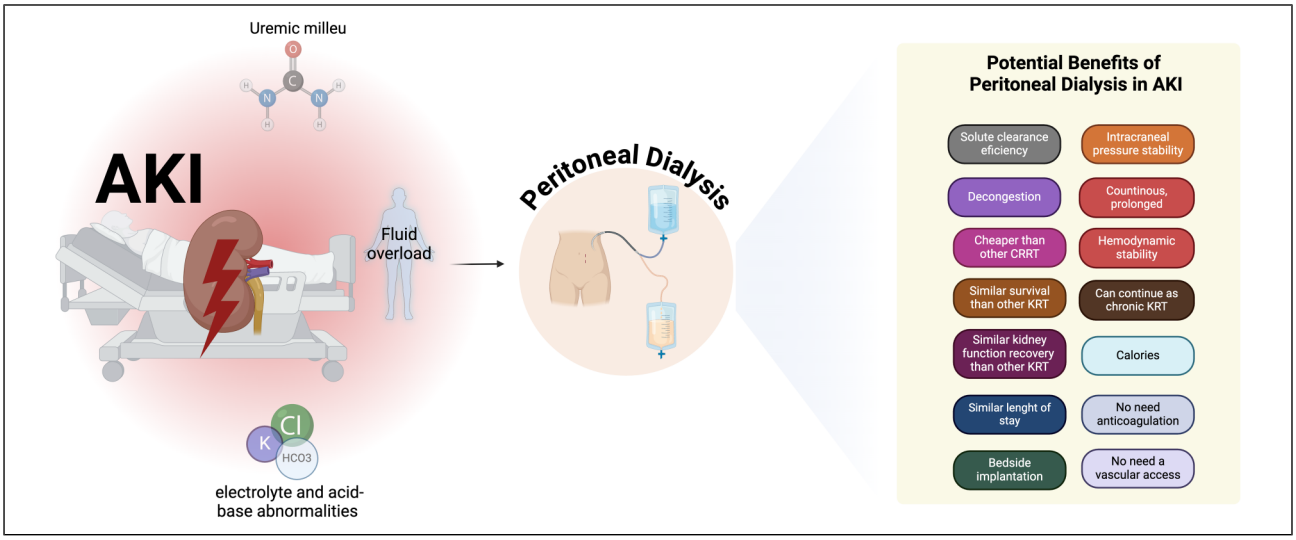


Figure 2. Potential benefits of DP in AKI.

to exert beneficial effects by reducing intra-abdominal pressure and improving systemic inflammation. In a cohort of 75 patients treated with PD for AKI, seven (9.3%) had pancreatitis, five survived, and two died during follow-up.[41] In general, reports on PD in AKI with pancreatitis are scarce, and no important alterations have been reported during pancreatitis or in its evolution compared to other groups of patients.[42,43]

The use of PD in ACLD, including hepatorenal syndrome (HRS) is very limited and in the absence of liver

transplantation, the mortality associated with HRS is higher than that of any other AKI etiology in cirrhosis. Case reports of successful treatment have been published.[44,45]

Ponce et al. treated 53 ACLD patients using PD (prescribed Kt/V = 0.40/session), with a flexible catheter, tidal modality, and lactate as a buffer.[46] The mean age was 64.8 ± 13.4 years, model of end stage liver disease (MELD) was 31 ± 6, 58.5% were in the intensive care unit, 58.5% needed intravenous inotropic agents including terlipressin, 69.5% were on MV, alcoholic liver disease was the main cause of cirrhosis and the main dialysis indications were uremia and hypervolemia. Blood urea and creatinine levels stabilized after four sessions at around 50 and 2.5 mg/dL, respectively. Negative FB and UF increased progressively and stabilized around 3.0 L and −2.7 L/day, respectively. Weekly-delivered Kt/V was 2.7 ± 0.37, and 71.7% of patients died.

The hypothesized benefit could be the hemodynamic stability offered by PD, the reduction of intra-abdominal pressure with the extraction of ascitic fluid by PD cycles, and the removal of inflammatory particles.[47] Similar to CRS, in cases where there is congestion, UF can be maximized by increasing the rate of PD exchanges and prescribing PD fluid bags with higher glucose concentrations. We believe that, despite the limited existing evidence, PD presents a good rationale in these scenarios and may be an interesting option for further exploration in studies involving larger patient population.

PD in specific AKI scenarios such as cerebral edema and COVID

In patients with AKI and cerebral edema, the KDIGO guidelines recommend using continuous KRT, PD meets these specifications and can provide some additional benefits, such as no anticoagulation, less increase in intracranial pressure, fewer changes in serum sodium levels, and reduction of extracellular glutamate.[48]

A Brazilian study treated by PD 58 neurocritical AKI patients (prescribed Kt/V = 0.40/session) using a flexible catheter, a cycler and lactate as a buffer.[49] The mean age was 61.8 + 13.2 years, 65.5% were in the ICU, 68.5% needed intravenous inotropic agents, 72.4% were on MV, APACHE II was 16 + 6.67. The main dialysis indications were uraemia and hypervolemia. Negative FB and UF increased progressively and stabilized around 2.1 + 0.4 L /day. Weekly delivered Kt/V was 2.6 + 0.31. Peritonitis and mechanical complications were not frequent (8.6% and 10.3%, respectively). Mortality rate was 58.6%.

In patients undergoing MV, it is reasonable to consider that the increase in intra-abdominal pressure due to the peritoneal fluid infused during PD might negatively impact respiratory mechanics and patient outcomes. However, this was not the case in a cohort study of 20 patients with AKI. Pulmonary mechanics were measured before and after initiating PD, revealing intra-abdominal pressure increased from 8 to 12 mmHg on the first day of PD but then decreased to 9 mmHg on the third day of treatment. Furthermore, the pulmonary static compliance improved on the third day and increased significantly from 30 to 54 mL/cm H2O, and pulmonary resistance remained stable throughout this period.[50] It is very important to consider that the cohort study of mechanics tested the lung mechanics when the abdomen was empty thereby limiting the results. However, PD was associated with two fewer days of treatment and a faster resolution of AKI by three days (both $p < 0.05$).[51] In patients with COVID-19, the high incidence of AKI and the lack of supplies necessary to provide KRT have caused many centers worldwide to use PD, which has performed satisfactorily in these scenarios. In a cohort involving 37 patients in ICU on MV, no deterioration in ventilatory or hemodynamic parameters was observed, with a favorable clinical evolution.[52] In a report of 21 patients with COVID-19, 75% were on MV, and PD improved the potassium, bicarbonate, urea, and serum creatinine levels.[53] A review of 6 articles reported that in patients treated with PD for AKI during the COVID-19 pandemic, a mortality of 43–63% was observed. Kidney recovery occurred in 33–55% of cases, MV did not worsen, and PD was used in prone patients.[54]

After 72 h of PD, 5200 mL of volume was ultrafiltered, serum potassium was 4.0 mEq/L, BUN was 62 mg/dL, VExUS score was 0, serum glucose had been consistently high with values ranging from 134 to 320 mg/dL. The nursing staff reported intermittent problems with peritoneal fluid infusion.

What are the expected complications in people who start peritoneal dialysis in the treatment of acute kidney injury?

Mechanical complications of PD in AKI

PD in patients with AKI was associated with complications. We recommend reading the excellent review by Cheng et al.[55] Because exchanges are initiated immediately prior to catheter placement, mechanical complications such as inflow may occur. Approximately 25% of the patients are estimated to experience this event. This may be due to the migration of the catheter from the pelvic cavity, kinking of the catheter, obstruction due to fibrin, clots, or constipation. Therefore, obtaining abdominal radiographs is essential. When it is caused by migration or obstruction, it may be corrected by passage of a guidewire, sometimes it may be necessary to mobilize the patient or reposition the catheter. Some intraluminal mechanical obstructions may be improved by vigorous flushing of the catheter to remove fibrin mechanically, this strategy does not dissolve the tissue.

Another common complication is fluid leakage through the wound, especially when large volumes of

Box 1. Conditions that may interfere with adequate PD treatment in patients with AKI and should therefore be thoroughly discussed.

Recent abdominal surgery, a potentially new abdominal surgery, active intra-abdominal neoplasia, abdominal stomas, peritoneum-scrotal or pleural fistula, ventriculo-peritoneal shunt, active peritoneal tuberculosis, severe chronic obstructive pulmonary disease, morbid obesity, uncorrectable bleeding disorders, severe psychiatric illness, diverticular disease of the colon, complicated umbilical hernia, or symptomatic scrotal hernia

Box 2. Equation to prescribe PD in AKI.

K = Urea clearance by the peritoneal membrane (constant 0.5) in liters
T = is the duration of treatment (24 h = 1)
V = is the volume of distribution of urea in liters

- Recommended Kt/V = 0.50 per session
- Dialysis bag solution (sodium 135 mEq/L, calcium 3.5 mEq/L, potassium 0 mEq/L, magnesium 1.5 mEq/L, acetate 40 mEq/L and 1.5% glucose. Glucose concentration may vary according to ultrafiltration needs 1.5%, 2.5%, 4.25%).
- Performed using a PAC-Xtra 2 cycler (any other brand can be used).
- 2 L bags solution with a duration of 35–50 min (total 32–44 L/day = 15 to 22 exchanges).
- In the example of a 70 kg man treated with 2 L bags, clearing the equation gives the following results.
- **K** = dialysis volume in 24 h (mL) × 0.5 (consider medium peritoneal transport)
- **t** = 1 (day)
- **V** = volume of urea distribution (mL) (Watson formula).
- K = 21,000
- T = 1
- V = 42,000
- **K t/V** = 21 × 1/42
- **K t/V = 0.5**
  Prescription: PD 42l (21 exchanges of 2 liters in 24 h)

intraperitoneal fluid are used (2000 mL). This complication could potentially be mitigated by using smaller volumes (1250 or 1500 mL), allowing a few hours or days for wound healing, and recommending that the patient remain in a supine position during exchanges. Another complication is hydrothorax associated with peritoneal fluid due to diaphragmatic defects. Hernia defects of the abdominal wall are usually more noticeable with peritoneal fluid infusion. Complications, clinical presentations, and possible solutions are described in Table 2.

It is important to note that most mechanical complications can be easily corrected, allowing treatment to continue safely and without interruption.

Metabolic complications of PD in AKI

Among the aspects that must be considered when prescribing a PD in AKI is the large amount of glucose that will be administered, which depends on the glucose concentration of each solution bag. However, a bag of 1.5% glucose is estimated to administer approximately 1100 kcal in 1 day, equivalent to 250 g of glucose, thus increasing glycemia and making glucose monitoring necessary.[35] Another complication may be the tendency to increase serum sodium levels and potentially hypernatremia because during rapid fluid exchange, aquaporin channels promote the UF of water rather than sodium. Although rare (<7%), peritonitis should always be considered as a differential diagnosis. As a preventive strategy, a 2% mupirocin ointment should be applied to the catheter exit site, and strict sterile precautions should be taken during connection and disconnection. Hypokalemia can be observed mainly when using PD bags with low potassium concentrations owing to a lack of intake, diuretics, or diarrhea, this complication can be treated by adding potassium to the PD pellets, either orally or intravenously. (Table 3).

After 4 days of PD, the patient reached the predicted goals, and the septic shock was resolved. The PD dose was reduced to 3 manual changes per day with 2000 mL of 1.5% glucose, maintaining a good trajectory. The patient was discharged, and in a follow-up in the clinic 2 weeks later, the eGFR returned to baseline, his functional status improved, and the PD catheter was removed.

Conclusions

Among the KRT options for patients with AKI, PD is an excellent treatment option compared to other techniques. PD offers several resource-saving benefits: it is less expensive, continuous, does not require a central vein, provides calories, improves fluid overload, uremic conditions, and electrolyte imbalances. Additionally, PD is similar in terms of long-term survival and kidney function recovery as shown in Figure 2. Therefore, training persons involved in AKI management is necessary to disseminate the benefits and risks of PD, thereby improving the prognosis of this syndrome.

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Authors' contributions

JSCI, JRCG and DP conceived and designed the review and wrote the original draft. All authors approved the final version of the manuscript and agree to be accountable for all aspects of the work.

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ORCID iD

Jonathan S Chávez-Iñiguez https://orcid.org/0000-0003-2786-6667

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