Chapter

Peritoneal Dialysis in Paediatric Acute Kidney Injury in Intensive Care Units: Prescription and Management

Djamila Djahida Batouche, Djilali Batouche, Zakaria-Zoheir Addou, Dalila Boumendil and Fatima Souhila Bouchama

Abstract

Acute kidney injury (AKI) is defined by a rapid decrease in glomerular filtration rate, leading to disruption of physiological functions, including impaired excretion of nitrogenous waste products, hydroelectrolytic disorders, and disturbance of acid-base balance. AKI is a major contributor to morbidity and mortality in severely affected infants and children, and its treatment, apart from symptomatic, etiological treatment, involves renal replacement therapy (intermittent haemodialysis, haemodialfiltration, haemofiltration, and peritoneal dialysis). In paediatric intensive care unit, emergency peritoneal dialysis (PD) is often the only possible technique for renal replacement therapy. It is easy to set up by the intensive care anaesthetist, or paediatric surgeon and uses the mechanisms of diffusion and osmosis (ultrafiltration). The anatomical properties of the peritoneum enable water and solute exchange. Solutions in bag form are available in isotonic or hypertonic concentrations, and their use depends on the clinical indications. PD has many advantages over other dialysis techniques, but there are some complications inherent in PD that need to be addressed by therapeutic protocols.

Keywords: continuous peritoneal dialysis, paediatric emergency, flexible catheter, solutions, intraperitoneal exchanges

1. Introduction

1.1 Acute kidney injury

In 2004, the term acute renal failure (ARF) was replaced by acute kidney injury (AKI), which better expresses the fact that it is a set of symptoms associated with

sudden renal failure. The syndrome is characterised by a reduction in glomerular filtration rate (GFR), retention of urea and other metabolic products such as uric acid and ammonia, and dysregulation of extracellular volume and electrolytes [1–3]. AKI is therefore a syndrome with varied manifestations and not a disease in the strict sense.

AKI is an important factor contributing to morbidity and mortality in severely affected infants and children [4, 5].

Since 2007, four essential definitions for AKI in children have been published: [6–9] see (**Table 1**).

The Paediatric Risk Injury Failure Loss ESRD (pRIFLE) criteria. In parallel with the pRIFLE work, the Acute Kidney Injury Network (AKIN) published a consensus statement on acute kidney injury (AKI).

In 2012, the Kidney Disease Improving Global Outcome (KDIGO) definition was published. The Paediatric Reference change value Optimised for AKI (pROCK) definition was adopted in paediatrics in 2018 from a multicentre study in hospitalised children in multicentre China as creatinine increase beyond RCV of creatinine, which was estimated as the greater of 20 μmol/L or 30% of the initial creatinine level. pROCK It is less sensitive for the detection of AKI but more specific outperformed KDIGO and pRIFLE in predicting the mortality risk especially in children requiring intensive care.

Definitions and comparisons have been difficult between studies in the literature, resulting in a wide range of citations, epidemiologies, morbidities, and mortality rates [10].

Classification	Staging creatinine	Creatinine criteria	Urine output criteria
pRIFLE	Risk eGFR	decreased by ≥25%	0.5 mL/kg/hr. for 8 hr
	Injury	eGFR decreased by ≥50%	0.5 mL/kg/hr. for 16 h
	Failure	eGFR decreased by ≥75% (or < 35 mL/min/1.73 m^2)	0.3 mL/kg/hr. for 24 hr. or anuria for 12
	Loss	Persistent failure >4 wk	
	ESRD	ESRD persistent failure >3 mo	
AKIN	1	Increase in creatinine of ≥50% or an absolute increase in creatinine of 0.3 mg/dL over 48-hr period	
	2	Increase in creatinine of ≥100%	
	3	Increase in creatinine of ≥200%	
KDIGO	1	SCr rise ≥0.3 mg/dL within 48 hr. or an increase in creatinine of≥50% within 7 day	>0.5 and ≤ 1 mL/kg/hr
	2	Increase in creatinine of ≥100%	>0.3 and ≤ 0.5 mL/kg/h
	3	Increase in creatinine of ≥200% or SCr ≥4 mg/dL or receipt of dialysis or eGFR<35 mL/min/1.73 m^2	≤ 0.3 mL/kg/hr

AKIN, Acute Kidney Injury Network; eGFR, estimated glomerular filtration rate; ESRD, end-stage renal disease; KDIGO, Kidney Disease: Improving Global Outcomes; pRIFLE, paediatric risk, injury, failure, loss of kidney function, and end-stage kidney disease; SCr, serum creatinine.

Table 1.
Paediatric acute kidney injury definitions.

In the newborn, there is no consensus definition for AKI. It was suggested that a classification AKI KDIGO for neonates and infants <120 days old [11]:

serum creatinine increase ≥0.3 mg/dL (26.5 mcmol/L) within 48 hours OR

- Stage 1 serum creatinine increase ≥1.5–1.9 times reference serum creatinine within 7 days, defined as the lowest previous serum creatinine value urine output <0.5 mL/kg/hour for 6–12 hours
- Stage 2, either of serum creatinine increase ≥2–2.9 times reference serum creatinine (defined as the lowest previous serum creatinine value)urine output <0.5 mL/kg/hour for ≥12 hours
- Stage 3, any of serum creatinine increase ≥3 times reference serum creatinine (defined as the lowest previous serum creatinine value) serum creatinine ≥2.5 mg/dL, which represents glomerular filtration rate < 10 mL/minute/1.73 m^2initiation of renal replacement therapy

urine output <0.3 mL/kg/hour for ≥24 hours

anuria for ≥12 hours

In the most severe cases, renal replacement therapy (RRT) is initiated to supplement renal function.

Several paediatric publications use peritoneal dialysis as one of the other RRT [12–14].

PD in emergency setting of the paediatric intensive care unit remains the only possible technique for RRT in infants, based on the experience of nursing staff and the unavailability of RRT equipment adapted to the child's weight because of the difficulties of vascular access, the risk of bleeding, and hypotension in an extracorporeal circulation [15–18].

We will focus solely on PD in children with AKI while discussing prescription methods, the complications of PD, and how to manage them.

1.2 Anatomy and physiology of peritoneal membrane

The peritoneum is composed of two adjacent layers: the parietal peritoneum which adheres to the anterior abdominal wall and which covers behind the retro peritoneum and the peritoneum visceral which totally or partially covers the abdominal organs.

It presents folds:

- Ligaments, means of fixing organs, without a main vascular pedicle within them;
- Theomentums that have a free edge;
- Themesos that contain the main vascular pedicle of the organ.

Between the two layers of the parietal and visceral peritoneum, we find the peritoneal cavity, which contains a thin layer of lubricating fluid, thus preventing

friction during the movements of the abdominal organs. This liquid less than 100 ml is produced by the peritoneal layers and is resorbed by lymphatic plexuses that are particularly numerous at the level of the lower surface of the right diaphragm. About 100 mL of a liquid formed from an ultrafiltrate of the peritoneum.

The peritoneal membrane successively comprises, from the blood to the peritoneal dialysate, the basement membrane of the peritoneal capillaries, the supporting connective tissue, the submesothelial basement membrane, and the monolayer of mesothelial cells.

The peritoneal membrane surface area is roughly equal to the body surface area.

PD does not require an extracorporeal blood circuit, and the exchanges between the blood and the dialysis solution (infused into the peritoneal cavity by a catheter) take place through the walls of the rich vascular network of the peritoneal membrane, according to the concentration gradients.

Peritoneal exchanges are based on two fundamental principles: diffusion (dialysis) and convection (ultrafiltration), as well as the three-pore model [19].

Transfer by diffusion occurs under the influence of a concentration gradient: urea, creatinine, calcium, phosphorus, and other plasma waste substances are gradually transferred into the dialysis fluid which is devoid of them, while the transfer is from the dialysis fluid to the blood for glucose, the buffer anion (lactate or bicarbonate), and calcium.

Ultrafiltration: Net ultrafiltration is achieved clinically by creating an osmotic pressure gradient such as glycerol, amino acids, glucose polymers, or icodextrin. Solutes present in body fluids can be carried away by the bulk solvent flow, even in the absence of a concentration difference for net diffusion, which contributes to the overall clearance of solutes. This contribution to net solute clearance has been termed 'convection'.

Three-pore model: [19].

There are three types of pore of different sizes in the endothelium of peritoneal capillaries.

The small, intercellular pores are where water and low-molecular-weight molecules such as electrolytes, urea, creatinine, and glucose pass through.

The ultra-small pores, which are the most numerous, are characterised by transcellular channels or aquaporins. They transport only free water, thus diluting the dialysate with a reduction in the initial sodium concentration and transfer from the plasma by concentration gradient or sodium sieving.

The large pores, which are few in number, allow large substances such as proteins and glucose polymers (icodextrin) to pass through the intercellular spaces.

This three-pore model makes it possible to explain peritoneal transfer by convection according to osmotic pressure of crystalloid or colloid origin and according to intraperitoneal hydrostatic pressure.

1.3 Peritoneal dialysis as a treatment for acute kidney injury

Despite the increasing importance of continuous haemofiltration techniques, PD is the most common and simplest method of ensuring the elimination of solutes and water in severe AKI, and has a major role to play in the management of AKI, especially in paediatric units where access to other extra-renal purification techniques is difficult or even impossible. It is very easy to perform in the patient's bed or in the operating theatre and does not require vascular access.

Acute PD is currently the best treatment modality for primary or complicated kidney disease-causing AKI.: The commonest cause was hypoperfusion,

haemolytic-uremic syndrome (HUS), [20], hypernatremic dehydration [21, 22], sepsis infection [23, 24], volume depletion, hypoxia ischemia, intrinsic renal disease, cardiogenic shock, post-surgical [25], and malaria in Africa [26]; peritoneal dialysis was the early treatment for children after cardiac surgery [27]. All AKI secondary to nephrotoxicity and poisoning were treated by PD in children in intensive care [23, 28].

During the COVID-19 pandemic, telemedicine was used to guide patients treated with chronic peritoneal dialysis in adults and has potential to improve patient care quality.

It is safety measures on how to protect PD patients and médical staffs.

The pandemic affected very few children during the first decade; however, some children developed acute kidney injury (AKI) with paediatric multisystem inflammatory syndrome (pMIS), and PD has attracted renewed interest in resource-limited countries, with the advantages of cost-effectiveness, ease of training, and reduced electricity and water requirements making PD the optimal form of therapy.

In both the adult and paediatric populations, there is sufficient evidence to demonstrate that peritoneal dialysis is as effective as other forms of RRT [29–32].

2. Advantages of acute peritoneal dialysis

PD is not associated with dialysis imbalance syndrome and may be more appropriate for patients with elevated intracranial pressure. Vascular access is not required for PD and has a significant advantage in small children where insertion of a peritoneal catheter is often easier than insertion of a dialysis catheter, especially in neonates [33].

PD makes it possible to preserve residual diuresis, and intraperitoneal exchanges ensure good haemodynamic tolerance [33].

In a prospective study, at Week 4 of admission or discharge, early initiation of PD in patients with SA-AKI was associated with early renal recovery and higher eGFR [24].

PD offers other advantages such as: the absence of extracorporeal circulation with its inherent risks (gas embolism, central catheter infection, and hypovolaemia) and the possibility of following a freer diet (especially in the catabolic phase), and it avoids anticoagulation and is inexpensive [3]. In infants, protection of the venous outlets is a crucial factor, especially if there is a high risk of progression to chronic kidney failure.

3. Limitations associated with acute peritoneal dialysis

PD uses the peritoneum as

1. Means of convection and ultrafiltration, so this peritoneum must be intact and free of contraindications to the placement of the peritoneal dialysis catheter, such as

   Recent surgery, peritoneal drains in place, omphalocele, laparoschisis or diaphragmatic hernia in children, fungal peritonitis peritonitis, and abdominal compartment syndrome;

   Even if PD remains effective, it is not without mechanical, infectious, and metabolic complications, which will be detailed in the chapter on complications of peritoneal dialysis.

2. It is more delicate in the event of respiratory distress and makes intubation and support by assisted ventilation necessary, especially in infants and newborns, due to an increase in abdominal volume during the dialysate infusion phase, thus resulting in insufficient ultrafiltration [34].

3. The time required to achieve satisfactory metabolic control and adequate volume control using PD makes this technique unsuitable for life-threatening emergencies—It is not the best modality for RRT in patients with threatening hyperkalaemia and those with hypercatabolism with a high azotemia load as this can only be achieved after 24 hours of dialysis [35]

4. In PD, clearance is limited by the flow rate of the dialysate, the permeability of the membrane, and the surface area coefficient (KoA). In PD, clearance is limited by dialysate flow membrane [36]

PD is less effective than haemodialysis or CRRT in children with inborn errors of metabolism (IEM), such as hyperammoniaemia, where neurological outcome is correlated with the speed and normalisation of blood urea treatment [37].

3.1 Peritoneal dialysis compared with other renal replacement therapy techniques

Observational studies comparing the modalities showed no difference in mortality between children treated with PD and those receiving CRRT to treat AKI.

A retrospective study by Fleming et al. [38] of 34 children who had undergone cardiac surgery showed that CRRT was associated with better fluid control and nutritional support than PD, but there was no significant difference in mortality rates, similarly in Flynn's series [3].

Comparing the safety of PD versus intermittent haemodialysis, a retrospective study by Basu et al. [25] found that the risk of death in patients treated with HD was 75% higher than in patients treated with PD. The children treated with HD in this study experienced frequent episodes of hypotension during treatment, and a risk analysis of the causes of death suggested that fluid and electrolyte changes were possible causes.

Krause et al. [39] found that intermittent HD was associated with a significantly better outcome than PD or haemodiafiltration (HDF) in another retrospective study of 115 children requiring dialysis for AKI. In this study, patients treated with IHD and PD were critical and placed on haemodynamic support, and the number of children treated with PD was significantly greater than in the other renal replacement therapies.

Gabriel et al. [40] then randomised 60 patients between acute high-volume PD and daily haemodialysis. There was no significant difference in terms of mortality.

However, recovery of renal function was observed 3 days earlier in the PD group.

In both the adult and paediatric populations, there is sufficient evidence to demonstrate that peritoneal dialysis is as effective as other forms of renal replacement. Recommendation 1B in adults and 1C in paediatrics, according to which PD is suitable for the treatment of AKI in all settings [41, 42].

4. Indications for acute peritoneal dialysis in children

PD can be carried out as an emergency treatment for AKI with no other organ failure.

- Hyperkalaemia, severe metabolic acidosis, and fluid overload, after failure of medical treatment.
- Risk of progression to kidney failure [43, 44]
- Renal indications and no renal indications of peritoneal dialysis in AKI are summarised in (**Table 2**) adapted by Ansari [45].

5. Efficiency of acute peritoneal dialysis

Numerous case series and randomised trials of PD in AKI demonstrate rapid correction of hyperkalaemia, acidosis, and fluid overload, usually returning to normal levels within 24–48 hours [46, 47].

Cullis et al. [48] recommend that in patients with AKI, a weekly Kt/V of 2.1 may be acceptable.

At present, there are no data correlating clearance and outcome in children undergoing acute PD, so no target dialysis dose can be set. In two paediatric studies of ARF of various aetiologies, the use of a filling volume of 20 ml/kg and a contact time of 60 minutes resulted in a mean weekly creatinine clearance of 75 l/week/1.73m^2.

McNiece et al. [49] measured clearance in five neonates undergoing cardiac surgery using a filling volume of 10 ml/kg and a contact time of 20 minutes. The median weekly creatinine clearance was 74 l/week/1.73 m^2, and the median Kt/V was 4.84.

Finally, Ricci et al. [50] determined that the creatinine clearance of 20 neonates post-cardiac surgery was 34 l/week/1.73 m^2 using a PD filling volume of 10 ml/kg and variable contact times, irrespective of haemodynamic status or drug vasopressor support.

These studies show that despite the low filling volumes and frequent cycles used in paediatrics, the clearances obtained are higher than those recommended in the adult literature [41].

Acute PD is an effective and reliable alternative to renal replacement therapy, particularly for reducing BUN and K+ levels in preterm neonates with AKI, in a retrospective study conducted by Xing et al. [51] including 21 preterm neonates who

Renal indications of peritoneal dialysis in AKI	Nonrenal indications
1. RRT in the treatment of AKI in children	1. Acute pancreatitis
2. Hemodynamically unstable patients	2. Clinically significant hypothermia or hyperthermia
3. The presence of bleeding diathesis or haemorragic conditions contraindicating placement of vascular access for haemodialysis or anticoagulation	3. Refractory heart failure
4. Patients with difficult vascular access placement	4. Liver failure
5. Removal of high molecular weight toxins (10 kD)	5. Infusion of drugs and nutrients as a supportive therapy in critically ill patients

Table 2.
Indications of peritoneal dialysis.

underwent APD in a neonatal intensive care unit (NICU) in Peking University Third Hospital. The median duration of PD was 3 days (range, 1 hour-20 days). Compared with the period before PD, blood urea nitrogen (BUN) and serum K+ levels decreased significantly after PD ($P < 0.05$). Oedema disappeared in 77.8% (n = 14/18) of patients, and 42.9% (n = 9/21) of patients regained normal urine output.

6. Strategies to improve dialysis efficiency in acute peritoneal dialysis

The strategy for improving the effectiveness of peritoneal dialysis will depend on several factors:

6.1 Choice of catheter

The common types of catheters used in acute PD are the rigid stylet catheter, Tenckhoff catheter, and Cook (Cook Medical Inc., Bloomington, IN) Teflon rigid catheter. The semi-rigid catheter can be inserted at the patient's bedside by a resuscitator under local anaesthetic, avoiding the need for a general anaesthetic.

The literature shows the effectiveness of the Tenckhoff catheter compared with other catheters [52].

6.2 Access to the peritoneal cavity

In emergency conditions, the catheter can be placed using a trocar and under peritoneoscopic control, although this technique requires general anaesthesia.

Laparoscopic catheter placement is successfully employed in children in many centres [53–55]. However, catheter placement for long-term peritoneal dialysis treatment is best performed surgically, under rigorous aseptic conditions, by a trained and motivated operator.

The most commonly used catheter entry route is paramedian, slightly below the level of the umbilicus, most often on the right side, the choice being guided by the patient's build.

Before the catheter is inserted, it is essential to reassure the child's parents and get them to sign an informed consent form, explaining the principle of the procedure and how it will be carried out. The catheter is usually inserted under general anaesthetic.

After carefully disinfecting the skin of the abdomen, an incision a few centimetres long is made through the fibres of the rectus abdominis, which are gently pulled apart to just the right length.

The catheter is inserted under visual control using a metal guide, the tip of the catheter being pushed into the lower part of the pelvic cavity, at the level of the cul de sac of Douglas. The tip of the catheter is then in contact with the lateral surface of the rectum. Once the catheter is firmly in place, the first Dacron® flange is fixed at a 45° angle in the peritoneal suture, which is then carefully closed around the catheter and its flange.

A tunnel is then created under the skin, allowing the catheter to pass subcutaneously. The second Dacron® flange is carefully fixed a few centimetres before the exit port, to prevent extrusion.

Check that the catheter is working properly immediately after insertion: after instilling approximately 1 litre of sterile isotonic solution, the liquid should return easily and be perfectly clear.

However, catheter malfunction, that is difficulty in draining the solution introduced into the peritoneal cavity, is not uncommon at the time of insertion. In this case, the position of the catheter should be checked by X-ray of the abdomen: when the peritoneal segment of the catheter has moved and is no longer resting in the cul de sac of Douglas, it may be necessary to intervene surgically to put it back in the correct position.

6.3 Peritoneal dialysis fluid and method of fluid delivery

Although the peritoneal membrane is biocompatible and natural, conventional dialysate fluids containing dextrose can alter the functionality of the peritoneal membrane due to glucose degradation products and the use of lactate as a buffer.

Fischbach et al. [56] demonstrated in children undergoing chronic dialysis that the use of a bicarbonate-lactate buffer gave good results in terms of a reduction in abdominal pressure during filling, alleviation of abdominal pain, and therefore good integrity of peritoneal function.

The composition of the dialysate fluid is summarised (**Table 3**); the components of the dialysate fluids (**Table 4**). Proposed in the Ansari article [45] are multiple and involve the following elements.

Several new PD solutions with low-glucose breakdown products have been introduced, but they are more expensive than conventional solutions containing dextrose and lactate. A recent meta-analysis by Sheng et al. [57] concludes that PD solution with low-glucose breakdown products preserves residual renal function and improves dialysis adequacy without increasing all-cause mortality. Further trials are needed to determine whether this beneficial effect may affect long-term clinical outcomes.

Recommendations on the choice of peritoneal dialysis (PD) fluids in children [58] conclude that the concentration of glucose should be as low as possible in the dialysate fluid. Icodextrin can be applied once a day during prolonged hospitalisation, particularly in children with inadequate ultrafiltration. Infants on PD are at risk of sodium depletion associated with ultrafiltration, while anuric adolescents may experience water and salt overload. The sodium-chloride balance must therefore be closely monitored. In growing children, the calcium balance should be positive, and the calcium in the dialysate should be adjusted according to individual needs.

Amino acid-based PD fluid in children suggests good tolerance. However, the anabolic effect is weak; adequate enteral nutrition is preferable.

Composition of the dialysate fluid
1. Sodium 132–134 (mmol/L)
2. Potassium 0–2 (mmol/L)
3. Calcium 1.25–1.75 (mmol/L)
4. Magnesium0.25–0.75 (mmol/L)
5. Chloride 95–106 (mmol/L)
6. Lactate 35–40 (mmol/L) or HCO3 (34 mmol/L)
7. Glucose 1.5–4.25 (g/dL)
8. pH (Neutral and physiological in newer peritoneal dialysis fluid preparations).

Table 3.
The composition of the dialysate fluide.

Components	Comments
Length of the dialysis session	Length of dialysis session determined by fluid balance, biochemical abnormalities and urine output
Composition of dialysate (% dextrose)	Ultrafiltration rate of 1.5% dextrose and 2.5% dextrose (40 ml/kg dwell) is 50–150 and 200–400 ml/h, respectively. A higher concentration of dextrose solution may be used for initial cycles in children with severe fluid overload.
Exchange volume	Initial fill volume: 10–20 ml/kg; maximum fill volume: 30 ml/kg or 1100 ml/m^2, but <800 ml/m^2 if age < 2 years. The higher the fill volume, the greater the clearance and ultrafiltration.
Exchange time—inflow, dwell, outflow time	Initial exchange time is 1 h—inflow 10 min, dwell 30–40 min, outflow 20 min. Dwell time pf 1–4 h in continuous PD using a flexible catheter. Shorter dwell time for rapid fluid, urea and potassium clearance (e.g. severe hyperkalaemia or pulmonary oedema). If hypernatremia develops, extend the dwell time if feasible or lower glucose concentration in dialysis solution.
Additives to dialysate	Potassium 3–4 mmol/l Heparin 250–1000 U/l
Monitoring	Fluid balance q 4–6 h Serum electrolytes per 6–12 h Blood sugar per 6–12 h (more frequently if using 2.5 or 4.25% dextrose-based dialysis fluid) Blood urea and serum creatinine per 24 h

PD, Peritoneal dialysis.

Table 4.
The components of the dialysate fluids.

In our daily practice in the intensive care unit at the university hospital, the nursing team uses a closed manual system, based on gravity, to deliver the dialysis fluid and drain it from the peritoneal cavity. In neonates, we use burettes to adapt the inflow and outflow of dialysis fluid.

6.4 Regimen of peritoneal dialysis

The different regimes have been described: intermittent peritoneal dialysis (IPD), and modifications to PD have been made recently to improve ultrafiltration, namely continuous equilibration PD, high volume PD, tidal PD, and continuous flow PD [59, 60].

Intermittent peritoneal dialysis (IPD) often used in our hospital for infants, using a rigid catheter. The infusion, contact, and draining times make up 1 cycle of 1 hour. This regime can be carried out manually or using a cycler by prescribing a pre-determined volume of dialysate fluid to drain the peritoneal cavity.

In children, 30 to 40 mL/kg of dialysate, warmed to 37°C, is infused over 10 to 15 minutes, then sucked back into the peritoneal cavity over 30 to 40 minutes, and drained after 10 to 15 minutes. Short exchanges of 1 to 2 L are carried out in sessions lasting 16 to 24 hours, three times a week.

Continuous PD is similar to continuous ambulatory peritoneal dialysis (CAPD), in which manual exchange is performed every 3 to 6 hours depending on patient clearance and fluid elimination.

It requires multiple daily exchanges, either manually or using a cycler. The disadvantage of this regime is that the patient must be hospitalised, and clearance may be insufficient, particularly in hypercatabolic patients, due to a lower dialysate flow rate [35].

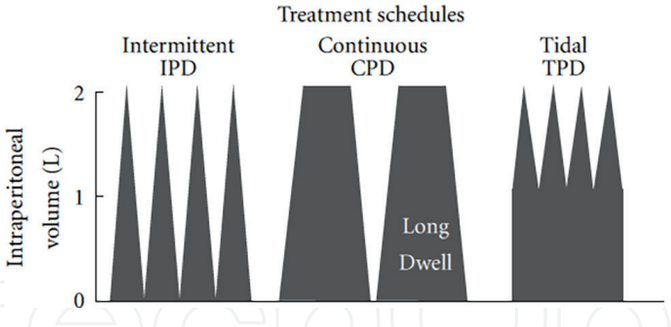

Figure 1.
Basic mechanisms operating in various peritoneal dialysis regimens adapted from Ansari [45].

Tidal peritoneal dialysis (TPD) requires a cycler and is a modality in which only part of the dialysate (25–50% of the infused volume) after a pause is drained during a long session lasting 8 to 10 hours, but optimisation of the clearance of drained solutes is not satisfactory [61, 62].

Basic mechanisms operating in various peritoneal dialysis regimens adapted from Ansari [45] are summarised in **Figure 1**.

6.5 Prescription of peritoneal dialysis

The prescription of PD must take into account the patient's clinical condition, body surface area, and state of hydration in order to achieve better ultrafiltration.

To initiate dialysis, it is recommended that the filling volume be calculated on the basis of body surface area rather than on the basis of the child's weight to avoid the problem of peritoneal membrane hyperpermeability.

It is recommended that the peritoneal cavity be filled with dialysate fluid with a volume of 10–20 ml/kg or 300–600 ml per body surface area in infants until the desired volume is obtained in the order of 30 ml/kg (800 ml per body surface area according to recommendations). There is a gradual increase in volume between d2 and d14 Up to 900 ml/m^2 body surface area for infants and 1400 ml/m^2 in older children.

Increasing the volume of dialysate must be done with caution, because although it improves diffusion and ultrafiltration, it also increases the risk of leakage and therefore of infection, and may create discomfort and thus a compliance defect [63].

In an article, Fischback et al. [64] describe a relationship between intraperitoneal hydrostatic pressure and dialysate volume in children on PD and demonstrate that the relationship between intraperitoneal pressure (IPP) and infused peritoneal volume (IPV) depends on age. In neonates, stable PPI values (3.5 +/− 1.6 cm) were observed for IPVs of 600 to 800 ml/m^2. Mean intraperitoneal pressure values were 8.2 +/− 3.8 cm for a mean peritoneal volume of 990 +/− 160 mL/m^2 body surface area, no significant increase in mean IPP was observed in infants (4.8 +/− 2.6 cm) and children (9.6 +/− 2.1 cm), and the increase in IPP was substantial when IPV increased from 1200 to 1400 mL/m^2 [64].

The dialysis cycle, which may last 60 to 90 minutes, includes a filling time, a pause or contact time of 30 to 40 minutes, and a drainage time.

In the event of threatening hyperkalaemia or a hypercatabolic state, the contact time may be reduced to ensure a high flow rate and therefore effective dialysis, especially in small children.

The duration of contact time in chronic dialysis for children with acute CPP is approximately 3 to 6 hours, which can be shortened to increase the total number of exchanges in order to improve solute clearance.

In acute PD, the duration of dialysis sessions will depend on the doses of dialysis administered, electrolyte and metabolic balance, and the daily kinetics of blood urea and creatinine.

Treatment is monitored on an hourly basis and reports on the amount of fluid recovered during each cycle, the type of dialysate used, its volume, any difficulties encountered, and tolerance. The input and output results and the ionogram enable prescriptions to be adjusted and the effectiveness of PD to be assessed.

Table 4 summarises components of acute peritoneal dialysis prescription adapted by Vasudevan and colleagues [65].

6.6 Complications

Numerous complications are inherent in peritoneal dialysis, and we summarise the main complications associated with this type of APD.

- Infectious complications: Despite improvements in techniques over the last few decades, infectious peritonitis remains one of the most feared complications [66]. Infectious peritonitis may be caused by contamination during handling, infection of the orifice through continuity, bacterial translocation through the digestive wall, or perforation of a hollow organ. The causative organisms are gram-positive and gram-negative bacteria. Peritonitis is suspected when the drainage fluid is cloudy. The diagnosis is confirmed by analysis of the drainage fluid for cell count, culture, and antibiotic sensitivity. Broad-spectrum antibiotic therapy should be initiated as soon as empirically possible to avoid the serious consequences of peritonitis, such as sepsis, and catheter removal.

    - Mechanical complications: Pain on instillation of the liquid or on drainage is a known complication in PD patients, occurring in 13 to 25% of cases [67].

Several factors contribute to this pain: low pH of the dialysate fluid, low temperature, or distension of the tissues around the catheter.

This pain can be minimised by infusing an alkaline PD fluid adding sodium bicarbonate and increasing the temperature of the dialysate while slowing the rate of dialysate infusion [46].

6.7 Intra-abdominal haemorrhage

Mild intra-abdominal haemorrhage is common and may be observed during catheter insertion. Severe intra-abdominal haemorrhage has been reported, particularly with acute semi-rigid catheters.

6.8 Intestinal perforation

Intestinal perforation may occur, particularly with the use of semi-rigid catheters in acute PD. Patients may experience severe abdominal pain, blood-stained peritoneal fluid, intra-abdominal haemorrhage, and (rarely) shock. Treatment consists of stopping treatment for a short period, removing the catheter, administering intravenous antibiotics, and treating the bowel.

Mechanical complications have been reported in several series, including leakage at the catheter insertion site, catheter occlusion, inguinal hernia, catheter malfunction, and fluid leaks [66, 68].

Respiratory complications such as:

- Respiratory distress during infusion of peritoneal fluid in children when the drainage fluid was insufficient, which increases intraperitoneal pressure; recommendations must be taken into consideration when infusing fluid according to the child's size and weight.
- Hydrothorax, generally on the right, is rare and is due to the presence of a congenital diaphragmatic anomaly with subsequent pleuroperitoneal communication, lymphatic drainage, and a pleuroperitoneal pressure gradient. Severe cases may require discontinuation of PD and surgical closure of the septal defect.

Pleurodesis by insufflation of talcum powder or thoracoscopic pleurodesis has been carried out in certain published series [69–71].

6.9 Metabolic complications

Hyperglycaemia can result from a high concentration of glucose in the PD fluid.

- Hypoglycaemia may occur as soon as PD is stopped.
- Hypernatremia: This can be induced by the disproportionate loss of free water in the PD fluid when hypertonic exchanges are frequently used. This is because the aquaporin-1 water channels in the capillaries are activated by the tonicity of the glucose generated by the dialysate. However, if the exchange is of short duration, sodium diffusion can take place early enough, and the patient slowly becomes hypernatremic. Ideally, the duration of the exchange should be corrected for diffusion to occur, or a hypertonic dialysate should be used.

Lactic acidosis is rare, except in patients with end-stage liver failure.

Lactic acidosis can be avoided by the use of peritoneal dialysis fluid containing bicarbonate.

- Hypokalaemia is frequently observed as standard dialysate fluid is at a concentration of 0–2 (mmol/L). Consideration should be given to the empirical addition of potassium to the dialysate solution after 12 hours of continuous PD in order to achieve a concentration of 3–4 mmol/L in the dialysate [42].

Volume changes may occur as a result of the use of hyperosmotic fluid leading to hypovolaemia or ultrafiltration failure leading to hypervolaemia, especially in the event of an episode of acute peritonitis.

This problem of volume imbalance can be resolved by adjusting the dialysis prescription or, in certain situations, necessitating temporary cessation of dialysis.

During episodes of acute peritonitis, hypoalbuminemia may develop due to high protein losses in the dialysate, which may reach 10 to 20 grams per day, requiring protein intake to be increased on a daily basis by infusing albumin.

Table 5 shows the complications associated with peritoneal dialysis and possible suggestions for their management, adapted by Nourse [42].

Initial prescription	Fill volume: 10–20 ml/kg Total cycle: 60–90 min. Fill: 5–10 min; dwell: 30–60 min; drain: 10–20 min Initial glucose concentration: 2.5% Heparin 500 IU/I PD over a full 24-h for 1–3 days
Problem	*Modification to prescription*
Poor ultrafiltration	1. Rule out access issues or peritoneal leak 2. Increase glucose concentration (1.5% → 2.5% → 4.25%) (or 1.36, 2.27 and 3.86% in some areas) 3. Decrease exchange duration by reducing dwell time by +25% (reduce fill and drainage times to a minimum) 4. Increase fill volumes 30–40 ml/kg (800–1100 ml/m^2) 5. Consider CFPD
Hyperkalaemia (emergency treatment required)	1. Reduce dwell time to 15–30 min (reduce fill and drainage times to a minimum) 2. Monitor potassium levels regularly
Serum potassium <4 mmol/l	Add 4 mmol/l of potassium to PD fluid
Difficulty with ventilation/increased intra-abdominal pressure	1. Reduce fill volume incrementally by 5 ml/kg. 2. Position patient in semi-fowlers position (30° head up) 3. Consider measuring intra–abdominal pressure to guide fill volume (either intravesical pressure with a transducer *via* a urinary catheter or directly from the PD catheter with a manometer) 4. Consider CFPD with very low fill volumes
High phosphate	1. Tolerate if not problematic and limited duration expected. If problematic: 2. Increase dwell times to >60 min 3. Increase fill volumes 30–40 ml/kg (800–1100 ml/m^2)
Hypernatraemia secondary to rapid cycling	1. Increase dwell time to >60 min 2. Reduce dialysate glucose concentration, if possible
Hypernatraemia, AKI and requiring dialysis	1. Add hypertonic sodium (3% or 5%) to PD fluid to within 15 mmol of patient's sodium to allow a gradual reduction in the serum sodium
Lactic acidosis AND hepatic dysfunction OR shock OR neonate AND/OR not responding to lactate-based fluids	Use bicarbonate-based PD fluids
Hyperglycaemia >20 mmol/l	1. Reduce glucose concentration in PD fluid if possible and/or increase exchange duration 2. If not working or not possible: Insulin infusion (start 0.05 IU/kg/h) OR 3. Add insulin to PD bags (see text Section 3.1)

Development of new pleural effusion	1. Consider extracorporeal dialysis modality if available
	2. Insert chest drain and check fluid for glucose
	3. Position patient in semi-fowlers position (30° head up)
	4. Reduce volume of PD per cycle
	5. Measure volume of fluid coming from chest drain and add to fluid balance

PD: peritoneal dialysis; AKI: acute kidney injury; CFPD: continuous flow peritoneal dialysis.

Table 5.
Complications associated with peritoneal dialysis and possible suggestions for their management.

7. Timing of initiation of peritoneal dialysis

Apart from the severity criteria for AKI requiring an emergency dialysis session, in the paediatric literature there is no optimum time to start a PD session.

Very few studies have evaluated the potential benefits of starting EBRT early.

The paediatric series found in the literature are debatable. Early initiation of renal replacement therapy in paediatric heart surgery is associated with lower mortality.

In a retrospective study of 146 newborn babies and children treated with peritoneal dialysis following cardiac surgery in a referral hospital, the authors noted that mortality was 28.1% at 30 days and 36.3% during follow-up. Early dialysis was associated with a 46.7% reduction in 30-day mortality and a 43.5% reduction in 90-day mortality compared with late dialysis. All other short-term variables were similar. Initiation of peritoneal dialysis on the day of surgery or the first day after surgery was associated with a significant reduction in mortality [72].

Another study conducted by Sanchez-de-Toledo et al. showed that early dialysis reduced mortality after paediatric cardiac surgery, and thus the relationship between RIA and morbidity and mortality after paediatric cardiac surgery. A single-centre retrospective study of children who underwent paediatric cardiac surgery between April 2010 and December 2012 in a tertiary hospital included 480 patients. Of these, 109 (23%) were neonates, and 126 infants and children (26%) developed AKI within the first 72 hours postoperatively. RRT techniques were used in 32 (6.6 %) patients (16 %) neonates and (3.8 %) infants and children; $p < 0.01$, with 78% receiving peritoneal dialysis (PD) and 22% continuous EER (CRRT). Patients treated with PD within the first 24 hours postoperatively had lower mortality than those in whom PD was initiated later [4/16 (25%) vs. 4/9 (44.4%)]. Mortality in patients who received CRRT late was 28.6%; no deaths were reported in patients treated with CRRT within the first 24 hours postoperatively [73].

In a meta-analysis conducted by Karvellas et al. [74], 15 studies (2 randomised, 4 prospective cohorts, and 9 retrospective cohorts) were identified out of 1494 citations. The overall methodological quality was poor. Early treatment of ERA compared with late treatment was associated with a significant improvement in 28-day mortality (odds ratio (OR) 0.45; 95% confidence interval (CI), 0.28 to 0.72). However, the limitations of this meta-analysis revealed significant heterogeneity as patients were stratified into medical and surgical patients. The prospective and retrospective study design and the early time to initiation of dialysis were not well defined.

8. Impact of PD on outcomes in children with AKI

There are limited data on the effect of EER modality on survival in paediatric AKI patients.

A retrospective study by Fleming et al. [38] compared HDF (n = 21) and PD (n = 21) in 42 children following repair of congenital heart defects. They concluded that haemodialfiltration (HDF) was superior to PD in terms of ultrafiltration and better nutritional support but no survival benefit was demonstrated between the modalities.

Bunchman et al. [75] in a retrospective study over 7 years reviewed survival outcomes in 226 paediatric patients receiving various forms of RRT, including PD, IHD, and CRRT.

A total of 106 patients were treated with CRRT, 61 with HDF, and 59 with PD. About 54% of the total population studied survived: 40% in the haemofiltration group. About 49% in the group treated with peritoneal dialysis, and 81% in the group treated with intermittent haemodialysis. Despite the clear superiority of IHD over the other modalities in this study, the authors concluded that haemodynamic instability better predicted greater mortality than the EER modality.

Regarding PD regimen: A prospective study by Gabriel et al. [76] was performed on 30 AKI patients who were assigned to continuous high-dose PD (Kt/V = 0.65 per session) *via* a flexible catheter (Tenckhoff) and to automated PD with a cycler. Fluid removal, pH and metabolic control, protein loss, and patient progress were assessed. Patients received 236 sessions of continuous PD, with standardised values for creatinine clearance and urea Kt/V of 110.6+/−22.5 L/week/1.73 m(2) body surface area and 3.8+/−0.6, respectively. Regarding the outcome of AKI, 23% of patients recovered renal function, 13% remained on dialysis after 30 days of follow-up, and 57% died.

A multicentre cohort study [77] was conducted on patients over 15 years of age admitted to an intensive care unit and diagnosed with acute renal failure. The aim of the study was to determine the effects of renal replacement therapy (RRT) modalities on 30-day mortality and renal recovery in patients with AKI whose main aetiology is sepsis.

Intermittent haemodialysis (IHD), continuous renal replacement therapy (CRRT), peritoneal dialysis (PD), or sustained low-efficiency dialysis (SLED) were the treatment modalities (of the 2844 patients with AKI, 449 cases (8.1%) received CRRT). There were no significant differences in 30-day mortality between patients initially treated with CRRT (52%), PD (51.6%), and SLED (55.6%) compared with those treated with HDI. Renal recovery was similar for each RRT mode.

9. Conclusion

The choice of technique for the treatment of AKI depends on the size of the patient, the availability of vascular access, the integrity of the peritoneal membrane and the abdominal cavity, as well as the clinical experience and expertise of the practitioner.

However, in most developing countries, as is the case in Africa, access to paediatric haemodialysis is difficult, so PD remains the only option available for the treatment of AKI.

PD still has its place in paediatric intensive care, particularly in patients with AKI due to a disease at risk of chronic renal failure.

To limit complications, particularly peritonitis, surgical placement of the catheter and strict compliance with a care and monitoring protocol are recommended.

Training of care teams and close collaboration with paediatric resuscitators and paediatric nephrologists are essential.

Conflict of interest

The authors declare no conflict of interest.

Author details

Djamila Djahida Batouche[1,2,3*], Djilali Batouche[4], Zakaria-Zoheir Addou[1,2], Dalila Boumendil[2] and Fatima Souhila Bouchama[2]

1 Paediatric Neonatal Intensive Care Unit EHU, Oran, Oran, Algeria

2 Faculty of Medicine Oran 1, Oran, Algeria

3 Research Laboratory LERMER, University Oran 1, Oran, Algeria

4 Clinical Research Multihealth and Pharmacovigilance, Company Freelance, Paris, France

*Address all correspondence to: batouchedjamiladjahida@gmail.com

References

[1] Prandota J. Clinical pharmacology of furosemide in children: A supplement. American Journal of Therapeutics. 2001;**8**(4):275-289. DOI: 10.1097/00045391-200107000-00010

[2] Warady BA, Bunchman T. Dialysis therapy for children with acute renal failure: Survey results. Pediatric Nephrology. 2000;**15**(1-2):11-13. DOI: 10.1007/s004670000420

[3] Flynn JT, Kershaw DB, Smoyer WE, Brophy PD, McBryde KD, Bunchman TE. Peritoneal dialysis for management of pediatric acute renal failure. Peritoneal Dialysis International. 2001;**21**(4):390-394. PMID: 11587403

[4] Goldstein SL, Currier H, Graf CD, Cosio CC, Brewer ED, Sachdeva R. Outcome in children receiving continuous venovenous hemofiltration. Pediatrics. 2001;**107**:1309

[5] Batouche D-D, Kerboua K. Pronostic prognosis-of-acute-renal-failure-in-children-in-intensive-care-unit-a-pilot-study. Current Pediatric Research. 2019;**20**(2):194-119

[6] Akran-Arikan A, Zappitelli M, Loftis LL, Washburn KK, Jefferson LS, Goldstein SL. Modified RIFLE criteria in critically ill children with acute kidney injury. Kidney International. 2007;**71**:1028e35

[7] Mehta RL, Kellum JA, Shah SV, Molitoris BA, Ronco C, Warnock DG, et al. Acute kidney injury network: Report of an initiative to improve outcomes in acute kidney injury. Critical Care. 2007;**11**(2):R31

[8] KDIGO Clinical Practice Guideline for Acute Kidney Injury. Kidney International Supplements. Vol. 2, No. 1. 2012. DOI: 10.1038/kisup.2012.1

[9] Xu X, Nie S, Zhang A, Jianhua M, Liu HP, Xia H, et al. A new criterion for pediatric AKI based on the reference change value of serum creatinine. Journal of the American Society of Nephrology: JASN. 2018;**29**(9):2432-2442. DOI: 10.1681/ASN.2018010090

[10] Sutherland SM, Byrnes JJ, Kothari M, Longhurst CA, Dutta S, Garcia P, et al. AKI in hospitalized children: Comparing the pRIFLE, AKIN, and KDIGO definitions. Clinical Journal of the American Society of Nephrology. 2015;**10**:554-561

[11] Selewski DT, Charlton JR, Jetton JG, Guillet R, Mhanna MJ, Askenazi DJ, et al. Neonatal acute kidney injury. Pediatrics. 2015;**136**(2):e463-e473

[12] Williams DM, Sreedhar SS, Mickell JJ, Chan JC. Acute kidney failure: A pediatric experience over 20 years. Archives of Pediatrics & Adolescent Medicine. 2002;**156**(9):893-900. DOI: 10.1001/archpedi.156.9.893. PMID: 12197796.

[13] Mehta P, Sinha A, Sami A, Hari P, Kalaivani M, Gulati A, et al. Incidence of acute kidney injury in hospitalized children. Indian Pediatrics. 2012;**49**:537-542. DOI: 10.1007/s13312-012-0121-6. Epub 2011 Dec 17. PMID: 22317984

[14] Raina R, Chauvin AM, Bunchman T, Askenazi D, Deep A, Ensley MJ, et al. Treatment of AKI in developing and developed countries: An international survey of pediatric dialysis modalities. PLoS One. 2017;**12**(5):e0178233. DOI: 10.1371/

journal.pone.0178233. PMID: 28557999; PMCID: PMC5448754

[15] Coulthard MG, Vernon B. Managing acute renal failure in very low birthweight infants. Archives of Disease in Childhood. 1995;**73**:F187-F192. DOI: 10.1136/fn.73.3.f187. PMID: 8535880; PMCID: PMC2528481

[16] Rasmussen SK. An overview of pediatric peritoneal dialysis and renal replacement therapy in infants: A review for the general pediatric surgeon. Seminars in Pediatric Surgery. 2022;**31**:151193. Epub 2022 May 29. PMID: 35725048

[17] Diarrassouba G, Adonis-Koffy L, Niamien E, Yaokreh JB, Coulibaly PA. Acute peritoneal dialysis in African pediatric area experience of pediatric nephrology unit of Yopougon university hospital (Abidjan, Côte d'Ivoire). Blood Purification. 2015;**39**:141-144. DOI: 10.1159%2F000368938. Epub 2015 Jan 20. PMID: 25660135

[18] Menaouri M, Batouche D, Elhalimi K, Hadjou F, Lahmer M, Okbani R, et al. Insuffisance rénale aiguë chez le nouveau-né et son pronostic. Néphrologie & Thérapeutique. 2022;**18**(5):454. DOI: 10.1016/j.nephro.2022.07.057

[19] Rippe B. A three-pore model of peritoneal transport. Peritoneal Dialysis International. 1993;**13**(Suppl. 2):35S-38S. PMID: 8399608

[20] Thiongane A, Ndongo AA, Ba ID, Boiro D, Faye PM, Keita Y, et al. Syndrome hémolytique et urémique de l'enfant au Centre Hospitalier Universitaire (CHU) de Dakar: à propos de quatre observations [Hemolytic-uremic syndrome (HUS) in children at the University Hospital Center in Dakar: about four cases]. Pan African Medical Journal. French. 10 Jun 2016;**24**:138. DOI: 10.11604/pamj.2016.24.138.8822. PMID: 27642476; PMCID: PMC5012731

[21] Moritz ML, Del Rio M, Crooke GA, Singer LP. Acute peritoneal dialysis as both cause and treatment of hypernatremia in an infant. Pediatric. Nephrology. 2001;**16**(9):697-700. DOI: 10.1007/s004670100644. PMID: 11511979

[22] Yildiz N, Erguven M, Yildiz M, Ozdogan T, Turhan P. Acute peritoneal dialysis in neonates with acute kidney injury and hypernatremic dehydration. Peritoneal Dialysis International. May-Jun 2013;**33**(3):290-296. DOI: 10.3747/pdi.2011.00211. Epub 2012 Nov 1. PMID: 23123669; PMCID: PMC3649898

[23] Genc G, Bicakci U, Gunaydin M, Tander B, Aygun C, Ozkaya O. Temporary peritoneal dialysis in Newborns and children: A single-center experience over five years. Renal Failure. 2012;**34**(9):1058-1061. DOI: 10.3109/0886022X.2012.715574. Epub 2012 Aug 20. PMID: 22906229

[24] Tomar A, Kumar V, Saha A. Peritoneal dialysis in children with sepsis-associated AKI (SA-AKI): An experience in a low- to middle-income country. Paediatrics and International Child Health. 2021;**41**(2):137-144. DOI: 10.1080/20469047.2021.1874201. Epub 2021 January 17. PMID: 33455545

[25] Basu B, Mahapatra TK, Roy B, Schaefer F. Efficacy and outcomes of continuous peritoneal dialysis versus daily intermittent hemodialysis in pediatric acute kidney injury. Pediatric Nephrology. 2016;**31**(10):1681-1689. DOI: 10.1007/s00467-016-3412-7

[26] Mwaba C, Munsaka S, Bvulani B, Mwakazanga D, Chiluba BC, Fitzwanga K, et al. Malaria is the leading

cause of acute kidney injury among a Zambian paediatric renal service cohort retrospectively evaluated for aetiologies, predictors of the need for dialysis, and outcomes. PLoS One. 2023;**18**(10):e0293037. DOI: 10.1371/journal.pone.0293037. PMID: 37878602; PMCID: PMC10599569

[27] Kwiatkowski DM, Krawczeski CD. Acute kidney injury and fluid overload in infants and children after cardiac surgery. Pediatric Nephrology. 2017;**32**(9):1509-1517. DOI: 10.1007/s00467-017-3643-2. Epub 2017 Mar 30. PMID: 28361230

[28] Li H, Yang S, Jin L, Wang Z, Xie L, Lv J, et al. Peritoneal dialysis treatment in small children with acute kidney injury: Experience in Northwest China. Blood Purification. 2019;**48**(4):315-320. DOI: 10.1159/000502079 [Epub 2019 Jul 29]. PMID: 31357204

[29] McCulloch M, Abugrain K, Mosalakatane T, Coetzee A, Webb K, Scott C. Peritoneal dialysis for treatment of acute kidney injury in a case of paediatric inflammatory multisystem syndrome temporally associated with SARS-CoV-2. Peritoneal Dialysis International. 2020;**40**(5):515-517. DOI: 10.1177/0896860820953716. Epub 2020 Sep 2. PMID: 32875970

[30] Parapiboon W, Ponce D, Cullis B. Acute peritoneal dialysis in COVID-19. Peritoneal Dialysis International. 2020;**40**(4):359-362. DOI: 10.1177/0896860820931235. Epub 2020 Jun 19. PMID: 32552550

[31] Al-Hwiesh AK, Mohammed AM, Elnokeety M, Al-Hwiesh A, Al-Audah N, Esam S, et al. Successfully treating three patients with acute kidney injury secondary to COVID-19 by peritoneal dialysis: Case report and literature review. Peritoneal Dialysis International. 2020;**40**(5):496-498. DOI: 10.1177/0896860820953050. PMID: 32998645

[32] Chen W, Caplin N, El Shamy O, Sharma S, Sourial MY, Ross MJ, et al. NYC-PD Consortium. Use of peritoneal dialysis for acute kidney injury during the COVID-19 pandemic in New York City: A multicenter observational study. Kidney International. 2021;**100**(1):2-5. DOI: 10.1016/j.kint.2021.04.017. Epub 2021 April 28. PMID: 33930411, PMCID: PMC8079266

[33] Golej J, Kitzmueller E, Hermon M, Boigner H, Burda G, Trittenwein G. Low-volume peritoneal dialysis in 116 neonatal and paediatric critical care patients. European Journal of Pediatrics. 2002, 2002;**161**(7):385-389. DOI: 10.1007/s00431-002-0919-7. Epub 2002 May 9. PMID: 12111191

[34] Bunchman TE, Meldrum MK, Meliones JE, Sedman AB, Kershaw DB. Pulmonary function variation in ventilator dependent critically ill infants on peritoneal dialysis. Advances in Peritoneal Dialysis. 1992;**8**:75-78. PMID: 1361858

[35] Chitalia VC, Almeida AF, Rai H, Bapat M, Chitalia KV, Acharya VN, et al. Is peritoneal dialysis adequate for hypercatabolic acute renal failure in developing countries? Kidney International. 2002;**61**(2):747-757. DOI: 10.1046/j.1523-1755.2002.00177.x. PMID: 11849419

[36] Chionh CY, Soni S, Cruz DN, Ronco C. Peritoneal dialysis for acute kidney injury: Techniques and dose. Contributions to Nephrology. 2009;**163**:278-284. DOI: 10.1159/000223811. Epub 2009 Jun 3. PMID: 19494626

[37] Schaefer F, Straube E, Oh J, Mehls O, Mayatepek E. Dialysis in neonates with

inborn errors of metabolism. Nephrology, Dialysis, Transplantation. 1999;**14**:910-918. DOI: 10.1093/ndt/14.4.910. PMID: 10328469

[38] Fleming F, Bohn D, Edwards H, Cox P, Geary D, McCrindle BW, et al. Renal replacement therapy after repair of congenital heart disease in children. A comparison of hemofiltration and peritoneal dialysis. The Journal of Thoracic and Cardiovascular Surgery. 1995;**109**(2):322-331. DOI: 10.1016/S0022-5223(95)70394-2. PMID: 7853885

[39] Krause I, Herman N, Cleper R, Fraser A, Davidovits M. Impact of dialysis type on outcome of acute renal failure in children: A single-center experience. The Israel Medical Association Journal (IMAJ). 2011;**13**(3):153-156

[40] Gabriel DP, Caramori JT, Martim LC, Barretti P, Balbi AL. High volume peritoneal dialysis vs daily hemodialysis: A randomized, controlled trial in patients with acute kidney injury. Kidney International. 2008;**73**(Suppl. 108):S87-S93. DOI: 10.1038/sj.ki.5002608. PMID: 18379555

[41] Cullis B, Al-Hwiesh A, Kilonzo K, McCulloch M, Niang A, Nourse P, et al. ISPD guidelines for peritoneal dialysis in acute kidney injury: 2020 update (adults). Peritoneal Dialysis International. 2021;**41**:15-31. DOI: 10.1177/0896860820970834. Epub 2020 Dec 3. PMID: 33267747

[42] Nourse P, Cullis B, Finkelstein F, et al. ISPD guidelines for peritoneal dialysis in acute kidney injury: 2020 update (paediatrics). Peritoneal Dialysis International. Mar 2021;**41**(2):139-157. DOI: 10.1177/0896860820982120 [Epub 2021 Feb 1]. PMID: 33523772

[43] De Galasso L, Picca S, Guzzo I. Dialysis modalities for the management of pediatric acute kidney injury. Pediatric Nephrology. 2020;**35**:753-765. DOI: 10.1007/s00467-019-04213-x. Epub 2019 Mar 18. PMID: 30887109

[44] Strazdins V, Watson AR, Harvey B. European Pediatric Peritoneal Sialysis Working Group. Renal replacement therapy for acute renal failure in children: European guidelines. Pediatric Nephrology. 2004;**19**(2):199-207. DOI: 10.1007/s00467-003-1342-7. Epub 2003 Dec 18. PMID: 14685840; PMCID: PMC1766478

[45] Ansari N. Peritoneal dialysis in renal replacement therapy for patients with acute kidney injury. International Journal of Nephrology. 2011;**2011**:739794. DOI: 10.4061/2011/739794. PMID: 21716704; PMCID: PMC3118664 [Epub 2011 Jun 8]

[46] Phu NH, Hien TT, Mai NT, Chau TT, Chuong LV, Loc PP. et al. Hemofiltration and peritoneal dialysis in infection associated acute renal failure in Vietnam. The New England Journal of Medicine. 2002;**347**:895-902. DOI: 10.1056/NEJMoa020074. PMID: 12239258

[47] Al-Hwiesh A, Abdul-Rahman I, Finkelstein F, Divino-Filho J, Qutub H, Al-Audah N, et al. Acute Kidney Injury in Critically Ill Patients: A Prospective Randomized Study of Tidal Peritoneal Dialysis Versus Continuous Renal Replacement Therapy. Therapeutic Apheresis and Dialysis. Aug 2018;**22**(4):371-379. DOI: 10.1111/1744-9987.12660. PMID: 29575788 [Epub 2018 Mar 25]

[48] Cullis B, Abdelraheem M, Abrahams G, Balbi A, Cruz DN, Frishberg Y, et al. Peritoneal dialysis for acute kidney injury. Peritoneal Dialysis International. 2014;**34**(5):494-517.

DOI: 10.3747/pdi.2013.00222. PMID: 25074995; PMCID: PMC4114667

[49] McNiece KL, Ellis EE, Drummond-Webb JJ, Fontenot EE, O'Grady CM, Blaszak RT. Adequacy of peritoneal dialysis in children following cardiopulmonary bypass surgery. Pediatric Nephrology. 2005;**20**(7):972-976. DOI: 10.1007/s00467-005-1894-9. Epub 2005 May 5. PMID: 15875216

[50] Ricci Z, Morelli S, Ronco C, Polito A, Stazi GV, Giorni C, et al. Inotropic support and peritoneal dialysis adequacy in neonates after cardiac surgery. Interactive Cardiovascular and Thoracic Surgery. 2008;**7**(1):116-120. DOI: 10.1510/icvts.2007.165118. Epub 2007 Nov 30. PMID: 18055480

[51] Xing Y, Sheng K, Liu H, Wu S, Wei H, Li R, et al. Acute peritoneal dialysis is an efficient and reliable alternative therapy in preterm neonates with acute kidney injury. Translational Pediatrics. 2021;**10**(4):893-899. DOI: 10.21037/tp-20-469. PMID: 34012838; PMCID: PMC8107877

[52] Chadha V, Warady BA, Blowey DL, Simckes AM, Alon US. Tenckhoff catheters prove superior to cook catheters in pediatric acute peritoneal dialysis. American Journal of Kidney Diseases. 2000;**35**:1111-1116. DOI: 10.1016/s0272-6386(00)70048-5. PMID: 10845825

[53] Daschner M, Gfrorer S, Zachariou Z, Mehls O, Schaefer F. Laparoscopic Tenckhoff catheter implantation in children. Peritoneal Dialysis International. 2002;**22**(1):22-26. PMID: 11929139

[54] Mattioli G, Castagnetti M, Verrina E, Trivelli A, Torre M, Jasonni V, et al. Laparoscopic-assisted peritoneal dialysis catheter implantation in pediatric patients. Urology. 2007;**69**:1185-1189. DOI: 10.1016/j.urology.2006.12.033. PMID: 17572212

[55] David VL, Mussuto E, Stroescu RF, Gafencu M, Boia ES. Peritoneal dialysis catheter placement in children: Initial experience with a "2+1"-port laparoscopic-assisted technique. Medicina. 2023;**59**(5):961. DOI: 10.3390/medicina59050961. PMID: 37241193, PMCID: PMC10223083

[56] Fischbach M, Warady BA. Peritoneal dialysis prescription in children: Bedside principles for optimal practice. Pediatric Nephrology. 2009;**24**(9):1633-1642. Epub 2008 Sep 20. PMID: 18807074, PMCID: PMC2719743

[57] Chen S, Jia J, Guo H, Zhu N. The benefits of peritoneal dialysis (PD) solution with low-glucose degradation product in residual renal function and dialysis adequacy in PD patients: A meta-analysis. Investigación Clínica. Sep 2022;**63**(3):283-303. DOI: 10.54817/ic.v63n3a07

[58] Schmitt CP, Bakkaloglu SA, Klaus G, Schröder C, Fischbach M, European Pediatric Dialysis Working Group. Solutions for peritoneal dialysis in children: Recommendations by the European pediatric dialysis working group. Pediatric Nephrology. 2011;**26**:1137-1147

[59] Kim YH, Resontoc LP. Peritoneal dialysis in critically ill children. In: Deep A, Goldstein SL, editors. Critical Care Nephrology and Renal Replacement Therapy in Children. New York: Springer; 2018. pp. 307-323

[60] Kontesis AKP, George E DM-S, Symvoulidis DA, Komninos Z. Continuous Equilibration Peritoneal Dialysis (CEPD) in Hypercatabolic

Renal FAILURE. Peritoneal Dialysis International. 1983;**3**(4):178-180. DOI: 10.1177/089686088300300404

[61] Balaskas EV, Izatt S, Chu M, Oreopoulos DG. Tidal volume peritoneal dialysis versus intermittent peritoneal dialysis. Advances in Peritoneal Dialysis. 1993;**9**:105-109. PMID: 8105900

[62] Piraino B, Bender F, Bernardini J. A comparison of clearences on tidal peritoneal dialysis and intermittent peritoneal dialysis. Peritoneal Dialysis International. 1994;**14**(2):145-148. PMID: 8043667

[63] Fischbach M, Stefanidis CJ, Watson AR, European Paediatric Peritoneal Dialysis Working Group. Guidelines by an ad hoc European committee on adequacy of the paediatric peritoneal dialysis prescription. Nephrology, Dialysis, Transplantation. 2002;**17**:380-385. DOI: 10.1093/ndt/17.3.380. PMID: 11865081

[64] Fischbach M, Dheu C, Seugé-Dargnies L, Delobbe JF. Adequacy of peritoneal dialysis in children: Consider the membrane for optimal prescription. Peritoneal Dialysis International. 2007;**27**(Suppl. 2):S167-S170. PMID: 17556298

[65] Vasudevan A, Phadke K, Yap HK. Peritoneal dialysis for the management of pediatric patients with acute kidney injury. Pediatric Nephrology. 2017;**32**(7):1145-1156. DOI: 10.1007/s00467-016-3482-6. Epub 2016 October 28. PMID: 27796620

[66] Bakal U, Sarac M, Tartar T, Aydin M, Kara A, Gurgoze MK, et al. Peritoneal dialysis in children infectious and mechanical complications experience of a tertiary hospital in Elazığ, Turkey. Nigerian Journal of Clinical Practice. 2022;**25**(8):1227-1232. DOI: 10.4103/njcp.njcp_1529_21

[67] Ogunc G, Tuncer M, Tekin S, Ersoy F. An unexpected complication in CAPD: Severe abdominal pain. Peritoneal Dialysis International. 2001;**21**:84

[68] Coccia P, Ramírez F, Suárez A, Alconcher L, Balestracci A, Chervo L. Acute peritoneal dialysis, complications and outcomes in 389 children with STEC-HUS: A multicenter experience. Pediatric Nephrology. 2021;**36**(6):1597-1606. DOI: 10.1007/s00467-020-04876-x

[69] Yim AP, Lee TW, Wan IY, Ng C. Images in cardiothoracic surgery. Pleuroperitoneal fistula. The Annals of Thoracic Surgery. Apr 2002;**73**(4):1327. DOI: 10.1016/s0003-4975(01)02743-6. PMID: 11996291

[70] Ramaema DP, Mpikashe P. Pleuroperitoneal leak: An unusual cause of acute shortness of breath in a peritoneal dialysis patient. Case Reports in Radiology. 2014;**2014**:614846. DOI: 10.1155/2014/614846. Epub 2014 Aug 4. PMID: 25165608; PMCID: PMC4137701

[71] Jonny J, Violetta L. Bilateral pleural effusion in continuous ambulatory peritoneal dialysis managed by vats pleurodesis. European Journal of Case Reports in Internal Medicine. 2024;**11**(4):004343. DOI: 10.12890/2024_004343. PMID: 38584902; PMCID: PMC10997387

[72] Bojan M, Gioanni S, Vouhe PR, Journois D, Pouard P. Early initiation of peritoneal dialysis in neonates and infants with acute kidney injury following cardiac surgery is associated with a significant decrease in mortality. Kidney International.

2012;**82**(4):474-481. DOI: 10.1038/ki.2012.172. PMID: 22622499

[73] Sanchez-de-Toledo J, Perez-Ortiz A, Gil L, Baust T, Linés-Palazón M, Perez-Hoyos S, et al. Early initiation of renal replacement therapy in pediatric heart surgery is associated with lower mortality. Pediatric Cardiology. 2016;**37**(4):623-628. DOI: 10.1007/s00246-015-1323-1. PMID: 26687178 Epub 2015 Dec 21

[74] Karvellas CJ, Farhat MR, Sajjad I, Mogensen SS, Leung AA, Ron W, et al. A comparison of early versus late initiation of renal replacement therapy in critically ill patients with acute kidney injury: A systematic review and meta-analysis. Critical Care. 2011;**15**(1):R72. DOI: 10.1186/cc10061

[75] Bunchman TE, McBryde KD, Mottes TE, Gardner JJ, Maxvold NJ, Brophy PD. Pediatric acute renal failure: Outcome by modality and disease. Pediatric Nephrology. 2001;**16**:1067-1071. DOI: 10.1007/s004670100029. PMID: 11793102

[76] Gabriel DP, Nascimento GV, Caramori JT, Martim LC, Barretti P, Balbi AL. High volume peritoneal dialysis for acute renal failure. Peritoneal Dialysis International. May-Jun 2007;**27**(3):277-282. PMID: 17468475

[77] Panaput T, Peerapornratana S, Sirivongrangson P, Kulvichit W, Lumlertgul N, Jonny J. Modalities of renal replacement therapy and clinical outcomes of patients with acute kidney injury in a resource-limited setting: Results from a SEA-AKI study. Journal of Critical Care. 2021;**65**:18-25. DOI: 10.1016/j.jcrc.2021.05.006. PMID: 34058688. Epub 2021 May 23