REVIEW ARTICLE

A Comprehensive Review on Effects of Peritoneal Dialysis in the Treatment of Acute Renal Damage

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Abstract

Aim and objective: To evaluate the prevalence and impact of physical symptoms in peritoneal dialysis (PD) patients, assess the effectiveness of PD in managing acute kidney injury (AKI) during COVID-19 and in patients who are suffering from congestive heart failure (CHF), investigate the incidence of vascular calcification and the role of biocompatible PD solutions (BPDSs) in improving patient outcomes.

Background: When treating acute renal failure, PD involves inserting a catheter into the abdomen, and a special fluid is introduced to draw waste and excess fluids from the blood through the peritoneal membrane. This process helps to balance electrolytes and remove toxins from the body. Compared to hemodialysis, PD offers advantages such as flexibility in scheduling, fewer dietary restrictions, and preservation of residual kidney function. However, potential complications include infection, hernias, and fluid overload. Overall, PD can be an effective option for managing acute renal failure, but careful monitoring and management of complications are essential.

Conclusion: Peritoneal dialysis is a practical and safe alternative to hemodialysis for patients without established access. It offers acceptable complication rates and patient and method survival. Comprehensive renal replacement therapy (RRT) programs should include a tailored educational program for a successful PD urgent start.

Keywords: Biocompatible peritoneal dialysis solution, Dialysis adequacy, Encapsulating peritoneal sclerosis, Erythropoiesis-stimulating agents, Metabolic syndrome, Peritoneal dialysis, Peritoneal permeability, Peritoneal equilibration test.

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Introduction

Within seven days, there is a sharp decline in kidney function known as acute kidney injury (AKI), caused by prerenal, intrinsic, and postrenal factors such as infections, dehydration, heart failure, cirrhosis, medications, and kidney stones.[1] Acute kidney injury diagnosis involves patient symptoms, serum creatinine tests, urine output assessment, urine microscopy, electrolytes, renal ultrasonography, and kidney biopsy for postrenal etiology.[1] Acute kidney injury affects 10–15% of hospital admissions and 50% of critical care patients, causing complications like metabolic acidosis, increased potassium, uremia, fluid imbalance, organ system effects, and mortality.[2] Uremia, previously known as acute kidney damage or uremic poisoning, is a condition characterized by diminished urine flow, also known as oliguria, caused by urine mixing with blood.[3] Acute tubular necrosis (ATN), a condition causing rapid kidney function decline, was first identified in the UK in the 1940s. It decreased during the Korean and Vietnam conflicts.[4] Acute kidney injury symptoms include exhaustion, lack of appetite, headache, nausea, vomiting, irregular cardiac rhythms, and fluid balance, regardless of blood pressure.[5] Kidney disorders can cause flank pain due to stretching of the fibrous tissue capsule. Dehydration may cause fluid depletion and thirst. Physical examination may reveal underlying etiology indicators like rash or palpable bladder.[6] Patients with kidney failure undergo renal replacement therapy (RRT) through peritoneal dialysis (PD). using the abdomen's peritoneal membrane as a filter. It introduces a dialysis solution, drains, and repeats daily or at night. Peritoneal dialysis offers flexibility and home-based treatment options.

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Materials and Methods

A thorough search of the literature was done to find research on the effects of PD done to treat acute renal damage Utilizing distinct combinations of MeSH keywords associated with PD and acute renal damage, all observational studies that reported effects of PD in acute renal damage treatment were found using PubMed, Scopus, DOAJ, and Google Scholar.

Method of Doing Peritoneal Dialysis

Peritoneal dialysis practices should assess patients' understanding of the process and support mechanisms before starting, educate them on catheter care, monitor patients for issues, develop treatment plans, and emphasize infectious control.[7]

The belly must first be cleaned in order to medically insert a tube. The other part of the tube needs to stick out from the skin.[8]

Before each injection, cleanse the tube and monitor movement. About 2–3 liters of dialysis liquids are pumped into the belly for 10–15 minutes, called stays. The fluid may contain medication and can be up to 3 liters in volume.[9] Waste products from blood vessels spread over the peritoneum, requiring 4–6 hours of fluid removal and refilling. Automatic peritoneal dialysis (APD) In contrast to CAPD, which has four dwells every day lasting 2–3 liters and 4–8 hours in the stomach, APD alternates between three and ten dwells per night. Continuous ambulatory dialysis (CAPD) can be used for daily maintenance and refilling. The dialysis process in PD involves sodium chloride, lactate, bicarbonate, and glucose to ensure hyperosmolarity.[10] The amount of dialysis depends on stay capacity, exchange frequency, and fluid content. Automatic peritoneal dialysis alternates between three and ten dwells per night, while CAPD contains 4 dwells/day, which lasts 2–3 liters as well as lasting 4–8 hours in the stomach.[11] The parietal peritoneum is the more significant component, making up around four-fifths of the membrane's surface area. The three-pore model and dispersed model explain the importance of capillaries in PD. Osmotic ultrafiltration (UF) forces fluid from peritoneal vessels into the peritoneal cavity due to elevated glucose levels.[12] Dialysate glucose diffuses into the bloodstream rapidly, However, the glucose osmotic gradient becomes too low to sustain further osmotic UF after 4–6 hours. The plasma colloid osmotic pressure, 18–20 mm Hg higher than the peritoneal colloid osmotic pressure, induces dialysis solution to be reabsorbed from the peritoneal cavity to the capillaries. Lymphatic absorption also assists in fluid reabsorption from the abdominal cavity to the plasma.[13]

Causes of Acute Kidney Injury

Prerenal

Prerenal azotemia is common in elderly and hospitalized individuals, as kidneys filter blood and produce pee to remove debris, but blood filtration decreases with decreased blood flow (Table 1).

Acute kidney failure, often prerenal azotemia, is caused by an accumulation of nitrogen waste products like creatinine and urea, which harms tissues and reduces system performance. It could be brought on by any illness that lowers blood supply to the kidney, such as:

Conditions that cause liquids to leak from the circulation include:

- Burns
- Prolonged nausea, diarrhea, or blood
- Hot weather
- Lower drink consumption (dehydration)
- Blood volume loss
- NSAIDs, or nonsteroidal anti-inflammatory drugs, along with ACE inhibitors, which are medications for high blood pressure and heart failure, are examples of certain pharmaceuticals.[6]

Intrinsic or Renal

Intrinsic AKI, caused by internal disease processes, can occur in various kidney components like glomeruli, renal tubules, or interstitium. Common causes include glomerulonephritis, ATN, AIN, tumor lysis syndrome, and rhabdomyolysis. Drug classes like tacrolimus can also cause intrinsic AKI.[14]

Table 1: Causes of acute kidney injury

Causes of acute kidney injury			
Cause	*Processes/subgroup*	*Examples*	*References*
Prerenal	Intrarenal vasoconstriction	Drugs: Tacrolimus, cyclosporine, NSAIDS, laxatives, vasoconstrictors, antihypertensives, diuretics, and antihypertensives.	Kaufman et al.
	Systemic vasodilation	Sepsis, neurogenic shock.	Holley JL
	Volume depletion	Diuretic overuse, osmotic diuresis, vomiting, diarrhea, burns, sweating, blood loss.	Ashley C and Holt S
Intrinsic	Glomerular	Drugs: Chlorpropamide, dapsone, gold, allopurinol halothane, levamisole, hydralazine, NSAIDs, penicillamine, penicillin, probenecid, procainamide, psoralen, quinidine, rifampicin, thiazides, tolbutamide.	Ashley C and Holt S; Rahman et al.
	Interstitial	Drugs: Acyclovir, allopurinol, aminosalicylates, bumetanide, cephalosporins, cimetidine, cotrimoxazole, furosemide, gold, interferon, isoniazid, lithium, NSAIDs, penicillin analogues, phenytoin, PPIs, quinolones, rifampicin, thiazides Infections: bacterial, fungal or viral, Systemic disease: Sarcoidosis, lupus.	Ashley C and Holt S; Rahman et al.
	Tubular	Ischemic: Prolonged hypotension, nephrotoxic exogenous toxins including acyclovir, aminoglycosides, amphotericin, cisplatin, contrast media, cyclosporine, ethylene glycol, foscarnet, ifosfamide, lithium, mannitol, NSAIDs paracetamol, tacrolimus, vancomycin Endogenous toxins such as hemolysis, rhabdomyolysis, tumor lysis syndrome, myeloma.	González et al.
	Vascular	Renal vein thrombosis, scleroderma renal crisis, renal atherothrombotic disease, malignant hypertension, renal infarction.	Holley JL; Rahman et al.
Postrenal	Ureteral/Bladder	Prostate hypertrophy, neurogenic bladder, retroperitoneal fibrosis, cancer of bladder, prostate, or cervix.	Rahman et al.
	Pelvic	Transitional cell carcinoma, pelvic malignancy leading to extrinsic compression of ureters, inflammatory aortic aneurysm.	Rahman et al.
	Intrarenal obstruction	Stones, crystals (acyclovir, indinavir), clots, tumors, paraproteins.	Rahman et al.

NSAIDs, nonsteroidal anti-inflammatory drugs; PPI, proton pump inhibitors

Postrenal

Postrenal AKI is primarily caused by urinary tract blockage, BPH, urinary catheter blockage, kidney stones, bladder, ureters, bladder stones, or prostate cancer.[14]

Diagnosis

The doctor may advise specific tests and treatments to confirm the diagnosis if the patient's signs and symptoms point to acute renal failure. They may consist of:

- *Urine output measurements:* By monitoring how much urine you produce over the course of a day, your doctor may be able to determine the cause of your kidney failure.[15]
- *Urine tests:* The analysis of a urine sample, known as a urinalysis, may reveal irregularities that indicate renal failure.[15]
- *Blood tests:* Two substances that are used to assess renal function and can be found in your blood in rapidly increasing amounts are urea and creatinine.[15]
- *Imaging tests:* Your doctor may use imaging tests like computed tomography and ultrasound to help them visualize your kidneys.[15]
- *Electrolytes in urine:* Measuring fractional excretion of sodium (FENa) in oliguric individuals can help differentiate between prerenal and inherent renal causes of acute kidney damage. The following algorithm gives the definition of FENa:

Online tools indicate prerenal causes of acute kidney damage with a FENa value of less than 1%, while a value of more than 2% indicates inherent renal causes. Diuretic treatment may cause natriuresis, making FENa levels less accurate. Fractional elimination of urea is useful. FENa levels less than 1% are not diagnostic for prerenal causes of acute kidney damage.[15]

Methods of Treatment

To manage AKI, the root cause must be identified and treated. Preventing cardiovascular collapse and mortality and requesting specialized counsel from a nephrologist are the primary goals of early care. Management of AKI often involves avoiding nephrotoxins, or chemicals poisonous to the kidneys, in addition to treating the underlying illness. They include NSAIDs like ibuprofen or naproxen, numerous antibiotics like gentamicin, iodinated contrasts like those used in CT scans, and a variety of other drugs.[16]

It is standard practice to monitor urine output and serial serum creatinine readings to determine how well the kidneys are functioning. In the hospital, the placement of the urinary catheter aids in monitoring the flow of urine and removes any potential blockage of the bladder outlet, which would occur in the case of an enlarged prostate.[16]

Prerenal Kidney Failure

Intravenous fluid infusion is frequently the first step toward restoring kidney function in prerenal AKI without any overload of the fluid. With a central venous catheter, volume status can be monitored to prevent giving too much or insufficient fluid replacement.[17]

Vasopressors (drugs that raise blood pressure), like norepinephrine, as well as inotropes (drugs that improve the pumping ability of the heart), such as dobutamine, may be used to improve the blood flow to the kidney if there persists low blood pressure even though after providing the patient with enough intravenous fluid. Despite being a helpful vasopressor, there is little evidence that dopamine offers any unique advantages, and it may even be harmful.[17]

Intrinsic Kidney Failure

Intrinsic AKI, caused by vasculitis or glomerulonephritis, requires specialized treatments like steroid therapy, cyclophosphamide, and plasma exchange, while discontinuing offending substances can alleviate toxin-induced prerenal AKI.[18] Furosemide is one of the diuretics that is frequently used and has some utility in treating fluid excess. It has no relation to increased mortality (risk of death), decreased mortality, or duration of stay in an ICU or hospital.[19]

Postrenal Kidney Failure

If a urinary tract obstruction is the cause, it may be imperative to clear the obstruction (either with a urinary catheter or nephrostomy).

Renal Replacement Therapy

In cases of AKI, RRT, such as hemodialysis, may be initiated AKI. The effectiveness of continuous renal replacement therapy (CRRT) and intermittent renal replacement therapy (IRRT) has been the subject of conflicting research. Continuous veno-venous hemofiltration (CVVH) treatment does not improve outcomes in critically ill patients. However, CRRT is more cost-effective and associated with a reduced risk of chronic dialysis in acute renal damage patients.[20]

Complications

Treatment options for metabolic acidosis, hyperkalemia, and pulmonary edema may include sodium bicarbonate, anti-hyperkalemic drugs, and diuretics.[21]

Fluid overload, metabolic acidosis, therapy-resistant hyperkalemia, and lack of improvement after fluid resuscitation may call for artificial support in the form of dialysis or hemofiltration.[21] Although the impact of a fluid load is quite unpredictable, oliguria when under anesthesia may be able to anticipate AKI. It is pointless to try to avoid AKI by achieving a predetermined urine production objective.[22,23]

Early Recovery of AKI

Based on the inverse of the AKI Kidney Disease: Improving Global Outcomes (KDIGO) serum creatinine criterion, the recovery from AKI may be divided into phases I–III.[24]

Physical Symptoms of Patients Receiving PD

Peritoneal dialysis has been found to affect the pathophysiology of underlying symptoms in renal patients, with a higher percentage of diabetics and a larger male population. The study found that stomach symptoms were not distinct, but appetite loss and constipation may be linked to the illness. Physical symptoms were more frequent in PD patients as well as hemodialysis patients. People with chronic illnesses often express feelings of weakness or fatigue, which is a continuum between vitality and sleepiness. Fatigue is the most common symptom in end-stage renal disease and is experienced by 50–70% of dialysis patients.

Muscle cramps were more common in patients receiving both hemodialysis and PD, with a frequency of 43% in a group of patients receiving both treatments. The study's sample size, patients' confinement to a single unit, and the use of a questionnaire were limitations. Despite these limitations, the study is significant as it is the only one to precisely evaluate PD patients' symptoms.[25–27]

Peritoneal Dialysis is a Viable Choice for Managing AKI in Patients with COVID-19

Continuous PD is a type of continuous kidney replacement therapy (CKRT) used in patients with severe illness, such as those with severe lung disease, for example, those found in COVID-19. The PD's effectiveness in managing AKI is comparable to hemodialysis and potentially hemodiafiltration.[28,29] However, during high morbidity situations like the current pandemic, PD may not significantly impact all-cause mortality or kidney function recovery compared to other extracorporeal therapies.[30]

The potential for an extended pandemic presents risks of equipment shortages along with supply chain deficiencies, especially in nations with inadequate economic means. In these areas, PD may provide distinct benefits compared to the need for workers with low training and the necessary infrastructure to provide treatment. Additionally, most countries have extensive expertise in implementing PD policies, making it a valuable and efficient method of dialysis in severe illness situations.[31,32]

Prescription PD strategies for AKI are extensively divided, with ISPD recommendations suggesting aiming for a weekly Kt/V urea of 3.5, which produces outcomes equivalent to daily HD. However, this dosage may not be essential for all patients, and a lower objective of weekly Kt/V close to 2.1 may be appropriate. Continuous APD may be the best modality in the COVID-19 crisis due to its reduced number of connections and disconnections, and less adjustments to prescriptions.[33]

Ponce et al. published PD prescription guidelines with a focus on brief cycles for patients experiencing symptoms of uremic symptoms, metabolic acidosis, severe hyperkalemia, or fluid overload.[34] Tidal PD may reduce medical staff exposure and shorten patient monitoring times. Experts advise against using PD as the first option for RRT when ARDS develops, but PD can be started early before patients experience symptoms of the disease. Because of the technical difficulties and difficulties in using the mechanical ventilator, additional RRT techniques should be taken into consideration.[35]

Peritoneal Dialysis and Congestive Heart Failure (CHF)

A possible treatment for individuals with severe CHF is peritoneal UF. The treatment of CHF patients with severe edema, frequent hospitalizations, and significantly decreased cardiac reserve may benefit from peritoneal UF, a relatively simple method for long-term salt and water removal.[36] It is noteworthy that our group of patients with severe diuretic-resistant heart failure also had a similar decreased incidence of rehospitalizations. We observed improved quality of life in our diuretic-resistant heart failure patients during the follow-up period with CAPD. After PD was started, the hospital readmission rate significantly decreased.[37] As a result, four of the five patients passed away 16 months after the PDC was implanted due to complications from advanced heart failure; however, the quality of life for all patients improved significantly as a result of PD. We found a correlation between improved left ventricular ejection fraction (LEVF) and reduced hospitalization and improved quality of life as a result of Parkinson's disease (PD). Severe heart failure patients frequently have renal impairment, which is a recognized independent prognostic factor in these patients. However, the level of renal dysfunction had no effect on the survival rate; on the other hand, PD-related improvement in LEVF was linked to a higher survival rate.[38] Under certain conditions, acute PDC implantation may be appropriate even in cases of severe hepato-cardio-renal pathology or liver cirrhosis.

Factors Influencing Vascular Calcification in PD Patients

Patients receiving hemodialysis (HD) experience vascular calcification (VC) at rates of 80–85%, while PD patients experience VC at rates of 65%. This is related to the total time taken for the dialysis; each year, HD patients have a 15% level of increased risk of VC. This study reported a 50.55% incidence of abdominal aortic calcification (AAC) particularly in patients with Parkinson's disease.[38–41]

Increased mortality rates are a result of common abnormal bone metabolism and also the vascular calcification. Significant alterations in the bone as well as mineral hormone axis occur as chronic kidney disease-mineral and bone disorder (CKD-MBD) advances. These changes result in altered bone turnover and clinical manifestations, including fractures, increased fragility, and decreased bone mass, along with associated vascular and valvular calcification. Similar to the development of cartilage and bone, "vascular calcification is thought to be an actively regulated process, however, the signaling pathways involved are not fully" understood.[42]

A key regulatory role in bone metabolism is played by the protein called sclerostin (SOST), which is produced by bone cells.[43] Under normal conditions, SOST inhibits bone growth and, by blocking the Wnt signaling pathway, decreases the differentiation of osteoprogenitor cells into osteoblasts, which in turn decreases the production of new bone. This way, SOST regulates bone metabolism.[44] Sclerostin expression levels are, however, frequently elevated in chronic renal disease, particularly during or after CKD stage III.[45] This study also shows that SOST increases the risk of vascular calcification in people with Parkinson's disease.

Atherosclerotic plaques have been reported to contain sclerostin, which may play a role in the development of vascular calcification and atherosclerosis.[46] Prior studies have examined the incidence and progression of coronary artery or aortic arch calcification, as well as their relationship to death rates.[47] In this "study, there was no discernible relationship between sclerostin and survival in patients with Parkinson's disease after adjusting for dialysis vintage, vascular calcification, and comorbidities".

The study's findings have a significant impact on clinical practice because identifying these risk factors enables the identification of Parkinson's disease patients who are at high risk and the implementation of appropriate therapies to delay or prevent the development of vascular calcification.[48] Techniques aimed at regulating the amount of Sclerostin in the bloodstream could reduce vascular calcification and improve patient outcomes.[49] To determine the precise "mechanisms by which Sclerostin encourages vascular calcification and evaluate the efficacy of Sclerostin-targeting treatments in clinical" settings, more research is necessary.

Cardiovascular Risk Potential in PD Patients Utilizing Biocompatible Solutions

The study confirms that the type of Parkinson's disease treatment does not significantly affect the prognosis of patients. Biocompatible peritoneal dialysis solutions (BPDSs) have advantages such as reducing membrane damage, maintaining peritoneal UF capacity, and reducing the risk of cardiovascular disease (CVD) due to fluid overload. Biocompatible peritoneal dialysis solutions can also maintain endothelial dysfunction indicators and residual renal

function. Further research is needed to understand the effects of BPDSs and clinical parameters on patients' prognoses. The relationship between peritoneal permeability and Parkinson's disease prognosis is complex, with high transporter patients receiving automated peritoneal dialysis (APD) having better technique survival and survival rates.[50–54]

Future Directions of PD in AKI

When it comes to treating AKI, PD appears to be on par with other modalities, just like in patients with CKD. Even though the Al-Hwiesh report suggests better outcomes than CKRT, adults in wealthy resource-rich nations will not be willing to use it until a multicenter randomized trial verifies this. The SYL program and ISN recommend it because the data is unquestionably sufficient to support its preferred use in low-resource settings and pediatrics where there are significant advantages over alternative treatments.

If research findings are confirmed, the following suggested study topics could help PD become more widely accepted:

- Comparing the activation of the inflammatory cascade in extracorporeal circuits and Parkinson's disease using the body's biocompatible membrane.
- Tidal APD and its impact on outcomes, such as greater clearance of larger molecules.
- For the treatment of liver failure and hepatorenal syndrome, biocompatible fluids containing bicarbonate as well as more recent solutions are being tested.
- The relative frequency of gastrointestinal and cardiac hypoperfusion in patients receiving extracorporeal therapy for Parkinson's disease.

Conclusion

In patients who require immediate initiation of hemodialysis but lack established hemodialysis access, PD may be a practical and safe alternative to hemodialysis (HD). It offers acceptable complication rates as well as patient and method survival. We discovered that this strategy, which permits genuine free individual choice of the modality, is a crucial component of comprehensive RRT programs in every reference dialysis unit. A good RRT educational program that is tailored to the unplanned setting is also required for the PD urgent start program to be successful. This will allow participants to make an informed decision regarding the RRT approach that best suits their lifestyle.

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