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Commentary

The Significance of Renal Replacement Therapy in Chronic Kidney Disease Management

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DESCRIPTION

The cost-effective, life-saving renal replacement therapy known as Peritoneal Dialysis (PD) is being used by an increasing number of people with chronic kidney disease globally. In comparison to hemodialysis patients, it has important advantages in terms of early patient outcome and quality of life. Overall, patient outcomes are still subpar, and only a small percentage of patients in most nations use PD. The primary drawback of PD, in addition to infectious problems, particularly bacterial peritonitis, is the inability to remove enough fluid and toxins, as well as the leakage of peritoneal proteins. While the effect on technique survival is yet unknown, several studies have shown that peritoneal protein losses indicate worse outcomes, including as cardiovascular events, peritonitis, and mortality.

Conventional PD Fluids (CPDFs), which have hazardous Glucose Degradation Products (GDP) and high glucose contents, have an acidic pH and unphysiologically high lactate concentrations. The incredibly unphysiological makeup of PD fluids causes significant peritoneal membrane change that eventually leads to PD failure. GDPs are quickly absorbed and lead to higher systemic concentrations of AGEs.

Double-chamber PD fluids have a physiological pH, include less GDPs, and isolate the glucose from the buffer molecule, which is either lactate or bicarbonate. Despite this, they still cause significant peritoneal toxicity and quickly change the peritoneum. Uncertainty still exists over whether superior long-term peritoneal membrane preservation and superior preservation of residual renal function lead to better PD patient outcomes.

The addition of protecting chemicals to PD fluids is an alternate strategy to prevent PD-related local and systemic toxicity and to maintain and improve peritoneal membrane transport properties. *In vitro* and in rats, Alanyl-Glutamine (AlaGln) improves the stress response and immunocompetence of mesothelial cells. In patients with chronic PD, adding 8 mM AlaGln to CPDF for a single 4-hour stay showed that peritoneal cellular stress responses would be restored, sterile inflammation would be reduced, and peritoneal host defence would be strengthened. A subsequent randomized crossover study showed improved *ex vivo* (LPS and Pam3Cys) stimulated IL-6 release from effluent cells, improved *ex vivo* (LPS and Pam3Cys) stimulated phosphate, uric acid, and potassium removal, and significantly improved peritoneal membrane semipermeability, i.e., higher dialysis removal of phosphate, uric acid, and potassium along with 20% lower peritoneal protein leakage.

The peritoneal endothelium is regarded as the primary barrier restricting the clearance of tiny and large solutes and fluids based on experimental research and mathematical modelling. A worldwide AQP-1 knockout mouse model has shown that AQP-1 channels are responsible for 50% of the water removal from the PD fluid; however, it is unknown what molecules are responsible for the removal of the remaining water and solutes. *Ex vivo* experiments in sheep and human parietal peritoneum have demonstrated that sodium channel blocker amiloride has a similar impact to vasogenic substances like adrenaline and endothelin-1 in reducing transperitoneal permeability.

Transcellular and paracellular routes determine peritoneal permeability, with the latter being more common in situations involving leaky membranes like the peritoneum. The paracellular barrier is mostly composed of Tight Junctions (TJ), which are found between neighbouring epithelial and endothelial cells. TJs indirectly regulate the paracellular but also transcellular transport features of the barrier. Currently, Claudin 5 (CLDN5), which mediates the cell-cell interaction in endothelial monolayers, is thought to be the most significant TJ protein.

CONCLUSION

The intracellular scaffold protein Zonula Occludens-1 (ZO-1) that connects the TJ claudins to the actin cytoskeleton is also an important part of the TJs. The maintenance of the endothelium's physiological permeability properties depends on TJs functioning properly. In order to improve appropriateness for chronic PD, postulated that AlaGln works on the peritoneal membrane barrier by altering peritoneal tight junction components and lowering mid- and large-size molecule transfer.

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