Commentary
Volume 13:1, 2023

Journal of Nephrology & Therapeutics

ISSN: 2161-0959 Open Access

Short Commentary on Peritoneal Dialysis and Inflammation

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Introduction

Patients with end-stage renal illness can get kidney replacement therapy called peritoneal dialysis (PD) (ESRD). It is based on the peritoneal semipermeable membrane's capabilities. Ultrafiltration (UF) and solute transport across the peritoneal membrane are made available by PD by infusing a dialysis solution into the peritoneal cavity. Dialysis is advised once a patient reaches stage 5 of chronic kidney disease (CKD), which is indicated by a glomerular filtration rate (GFR) less than 15 mL/min/1.73 m2, along with clinical signs of malnutrition, an overloaded volume that is unresponsive to diuretics, or other signs and symptoms associated with uremia. Before stage 5, the therapy may be started due to particular clinical factors and kidney failure problems.

Description

Through peritoneal dialysis, accumulated solutes from the blood, including urea, creatinine, potassium, phosphate, and water, are removed (PDS). The interchange of water and solutes between the interstitial capillaries and PDS is regulated by the peritoneal membrane (PM), which performs as a comparable dialyzer. The peritoneal equilibration test assesses the PM's ability to transport solutes and its capacity for ultrafiltration in PD patients (PET). PET is a semi-quantitative method for evaluating the peritoneal membrane's ability to transfer substances. It is measured by tracking the levels of glucose and creatinine in the plasma and dialysis solution over time [1-3].

This test divides patients into four groups: (a) high peritoneal solute transfer rates, which are likely to have insufficient ultrafiltration to standard PD; (b) high-average peritoneal solute transport, which is still responsive to standard PD even after losing residual renal function; and (c) low-average and particularly (d) low peritoneal transport, which is likely to develop symptoms and signs of insufficient dialysis. Despite all recent technological advancements made in this field, the rate of mortality in patients undergoing peritoneal dialysis is higher than that of hemodialysis. Although there are several factors linked to mortality in PD patients, inflammation is one of the most critical ones. Due to the high concentration of glucose, the acidic pH (5.5), and the lactate used to treat the metabolic acidosis [4], long-term peritoneal dialysis promotes chronic inflammation processes, such as those related to catheter access infections, dialysate contamination, inadequate dialysis, high concentration of uremic toxins, release of plastic materials, and bioincompatibility of dialysis solution.

Peritoneal fibrosis and neoangiogenesis typically follow inflammation linked to PDS usage. As a result, it is shown that the transport of tiny solutes has increased, which causes UF failure during the course of the dialysis therapy. However, it is still unknown how the peritoneal membrane alterations are controlled. The discovery of alternative PDSs with improved biocompatibility and lower risk of peritoneal membrane (PM) damage was sparked by the toxicity associated with long-term use of standard peritoneal dialysis solution [5]. The ability of PM to remain functional and without major clinical modification even after a lengthy duration of therapy can be used to define biocompatibility.

Inflammation brought on by traditional PDS

As an osmotic gradient enhancer and buffering agent, respectively, high glucose and lactate concentrations are present in conventional PDS. Glucose concentrations range from 1.5% to 4.25% to 2.3% to 2.5%. Due to glucose's direct metabolic effect of hyperosmolarity, the traditional PDS is primarily bioincompatible with glucose. Additionally, the chemical instability of glucose causes elevated amounts of poisonous GDPs (methylglyoxal, glyoxal, and 3-deoxyglucosone) to be produced during heat sterilising and storage, which worsens the negative effects of PDSs.

Chronically being exposed to a high glucose load in a standard peritoneal dialysis solution dramatically increases the PM's inflammatory state. Advanced glycation end-products (AGEs), which cause an upregulation of the AGE receptors, are produced as a result of this type of solution, as are fibroblast growth factor, transforming growth factor- (TGF-), and vascular endothelial growth factor (VEGF) (RAGEs). These elements work together to cause mesothelial fibrosis and neoangiogenesis. Additionally, glucose breakdown products generate oxidative stress, which damages human peritoneal mesothelial cells (HPMCs), resulting in apoptosis and mesothelial denudation [6]. Oxidative stress then intensifies the inflammation. Human peritoneal mesothelial cells' viability and function are lost during PD using a standard peritoneal dialysis solution, and AGEs are also created in the blood as a result of GDPs' passage from the peritoneal cavity to the systemic circulation. High levels of glucose degradation products, which are precursors to advanced glycation end products; cause an inflammatory response in the peritoneum, which causes HPMC to apoptose and the peritoneum submesothelial layer to fibrose. The synthesis of TGF- is increased when AGEs attach to their receptors.

Utilising PDS for inflammation with low GDPs concentration and pH

When utilising PDSs with lower glucose degradation products and neutral pH compared to normal PDSs, it has been demonstrated that extracellular matrix components such IL-6 and hyaluronic acid, respectively, are less vulnerable to PD treatment, indicating improved biocompatibility. Data from 121 peritonitis incidents in patients receiving various peritoneal dialysis treatments were evaluated by Naqvi SB, et al. [7]. Among these, patients using traditional PDSs experienced 107 events, while patients utilising PDSs with lower GDPs experienced just 14. As a result, these patients had a much decreased peritonitis rate.

Icodextrin is a glucose polymer that is water soluble. For more than ten years, this high molecular weight starch derivative has been used as PDS in therapeutic settings. Icodextrin is approximately 100 times bigger than glucose, which hinders its absorption from the peritoneal cavity and offers a colloid osmotic UF. Because of this, its diffusion across the peritoneal capillary endothelium is constrained, allowing an osmotic pressure that is largely constant. This peritoneal dialysis solution contains lower GDPs than glucose PDSs and is isosmotic to plasma. The icodextrin solution fully or partially remedied three key traits of the previous solutions that were connected to their biocompatibility. In order to better understand the involvement of various peritoneal dialysis solution types in the development of inflammation in patients with chronic peritoneal dialysis, additional well planned and overseen

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Received: 28 December, 2022, Manuscript No. JNT-23-89023; **Editor Assigned:** 30 December, 2022, PreQC No. P-89023; **Reviewed:** 11 January, 2023, QC No. Q-89023; **Revised:** 16 January, 2023, Manuscript No. R-89023; **Published:** 23 January, 2023, DOI: 10.37421/2161-0959.2023.13.431

clinical trials including a larger sample size are necessary. The selection of the proper PDS in accordance with the patient profile will reduce the likelihood of inflammation or at least slow down the process, enhancing the quality and length of life of these patients.

Mammalian cells contain large amounts of the sulfonic beta-amino acid taurine, also known as 2-aminoethanesulfonic acid. This physiological compound controls ion transport across cells and osmotic balance. Compared to glucose, taurine has a low molecular weight (125 vs 180 Da, respectively). Taurine also has high water solubility, low lipophilicity, and high dipole constants, which help it to maintain a neutral pH and have a potent buffering action. When in solution, taurine has a powerful buffering action and keeps the pH at a neutral level. Additionally, it is regarded as a safe substance. Even when it was given to rats at the highest dose, animal investigations did not reveal any harmful consequences. This reasonably priced and easily accessible amino acid works well as a nutritional supplement to help manage hypertension and dyslipidemia. Taurine was chosen as a strong contender as an osmotic agent in PD solutions due to all of these factors.

Conclusion

Numerous studies have revealed that PDSs with lower GDPs have advantages over conventional PDSs in terms of biocompatibility and have less of an impact on the inflammatory process. However, bigger prospective randomised trials are required, especially given their higher cost when compared to the standard peritoneal dialysis solution, to validate the decreased incidence of inflammation and the higher integrity of the peritoneal membrane in patients utilising these solutions. Although there have been some debates, several dialysis facilities in Europe have already judged that the benefits may outweigh the costs and have made the PDS with lower glucose degradation products their regular course of action.

Acknowledgement

Not applicable.

Conflict of Interest

There is no conflict of interest by author.

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How to cite this article: Dare, Hamilton. "Short Commentary on Peritoneal Dialysis and Inflammation." *J Nephrol Ther* 13 (2023): 431.