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How to Improve the Outcome of Peritoneal Dialysis? Salt Is a New Poison for PD

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High salt intake is a primary cause of over-hydration in CKD patients. Inflammation is a predictor of both all-cause and cardiovascular mortality in pre-dialysis CKD patients and dialysis patients; however, the pathogenesis of inflammation remains unclear. Sodium storage in tissues has recently emerged as an issue of concern. The binding of sodium to tissue glycosaminoglycans and its subsequent release regulates local tonicity. Many cell types express tonicity-responsive enhancer-binding protein (TonEBP), which is activated in a tonicity-dependent manner. Macrophage infiltration was observed in the pre-dialysis CKD human peritoneum, and in the heart, peritoneal wall, and para-aortic tissues in salt-loading uremic mice. TonEBP was increased leading to upregulation of inflammatory mediators associated with cardiac fibrosis and peritoneal membrane dysfunction. The potential for direct sodium toxicity in PD patients has gained increased interest in recent years. At the time of PD initiation, the baseline peritoneal solute transport rate (PSTR) varies, and a correlation exists between high PSTR and a poor prognosis in PD due to inadequate ultrafiltration. Recent studies have enabled the visualization of sodium accumulation in the lower extremities. 23Na MRI showed sodium storage in the interstitial tissue of the skin, subcutaneous tissues, and the muscles of the leg in disease conditions. These human studies using 23Na MRI suggest that sodium content in the skin is related to inflammation and associated with higher mortality risk. In addition, the baseline peritoneal solute transport rate in PD is related to the sodium storage and inflammation in renal failure conditions. High salt intake induces tissue inflammation associated with organ dysfunction in CKD patients. TonEBP appears to play a crucial role in the process of cardiac fibrosis and peritoneal deterioration induced by sodium overload. Strategies to prevent sodium accumulation and treatments to normalize the sodium content in tissues appear to be important.

Keywords: sodium storage, inflammation, organ dysfunction, TonEBP, IL-6