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Commentary

The Role of Hemodiafiltration in the Reduction of Cardiac Death Rates

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DESCRIPTION

Hemodiafiltration is capable of eliminating residual small and Medium Molecular Weight (MMW) compounds that accumulate in HD-treated patients with end-stage kidney disease (ESKD). Small water-soluble compounds (WSCs; 500 Da), MMW substances (0.5-40 kDa), and Protein-Bound Toxins (PBTs) are the three primary types of uraemic solutes. While WSCs like urea and creatinine are mostly eliminated through diffusion, convection is the major factor behind the removal of bigger MMW solutes, which penetrate the dialysis membrane through solute drag caused by the transmembrane pressure gradient. WSCs may be removed by any membrane, whereas MMW compounds can only be removed by high-flux dialysers. PBTs are difficult to remove because only the free fraction can pass through the dialysis membrane, which is mostly made up of low Molecular Weight (MW) molecules. Any membrane, once again, can be employed for this purpose.

Urea, a small solute (60 Da) that accumulates in Chronic Kidney Disease (CKD), is widely used in the Kt/Vurea calculation to determine dialysis adequacy. Although Hemodiafiltration improves Kt/Vurea, boosting urea clearance did not improve mortality before. Similar results were reported for creatinine (113 Da), which is most typically used to estimate renal function by measuring creatinine clearance. Phosphorus (95 Da) levels that are elevated are linked to vascular calcifications and Cardiovascular Disease (CVD) mortality.

Although phosphorus is a tiny molecule, the surrounding water mantle causes it to function more like a medium molecule in the biological system. While phosphorus levels and the prescription of oral binders were lower in Hemodiafiltration compared to low-flux HD, blood levels did not differ between the two conditions in two recent RCTs. However, it should be noted that an increased phosphate level is only one component of the multidimensional CKD-Mineral and Bone Disease (MBD), which also includes calcium derangements, vitamin D status and resistance, Parathyroid Hormone (PTH) levels, and Fibroblast Growth Factor 23 levels (FGF23). Because phosphate levels in these patients are also affected by Residual Kidney Function (RKF) and the administration of CKD-MBD-specific medications such as phosphate binders, vitamin D analogues, and calcimimetics, dialysis represents only one aspect of the complicated interplay between these components. Currently, it is unclear if reducing serum phosphorus is connected with improved clinical outcome in ESKD patients.

The MMW chemicals that accumulate in ESKD are mostly tiny peptides, many of which have been linked to inflammation, endothelial damage, smooth muscle cell proliferation, oxidative stress, and interference with the coagulation cascade. Because majority of these processes may contribute to CVD, removing them by convection may improve clinical outcomes. Although Hemodiafiltration significantly improves their elimination, particularly in patients with only marginal RKF, neither the reduction of 2-microglobulin (MW 11.8 kDa), the pro-inflammatory cytokines interleukin 6 (MW 21 kDa) and tumor necrosis factor-α (TNF-α) (MW 25.6 kDa), nor complement factor D (MW 24 kDa) has been shown to underpin the beneficial clinical effects of convective therapies.

In the case of CKD-MBD, elevated PTH (MW 9.4 kDa) levels have been linked to a variety of CVD symptoms. Reduction by medicine, however, did not significantly affect results obtained. In contrast, encouraging outcomes for FGF23 (MW 32 kDa), the first detectable biochemical modification in CKD-MBD, have been found. This phosphatonin is 100-1000-fold greater in ESKD patients than in healthy controls. FGF23 removal was much greater during Hemodiafiltration than during high-flux HD. Because FGF23 has been linked to left ventricular hypertrophy and CVD events, particularly congestive heart failure in individuals with CKD Stages 2-4, lowering FGF23 with Hemodiafiltration may reduce CVD mortality in ESKD.

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