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Dapagliflozin reduces kidney injury markers in patients with chronic kidney disease

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Objectives: Sodium glucose cotransporter2 inhibitors (SGLT2i) reduce the risk of chronic kidney disease (CKD) progression independent of diabetes mellitus (DM). Therefore, we investigate the effect of dapagliflozin on kidney injury makers in CKD on standard of care (SOC).

Methods: We prospectively enrolled healthy volunteers (HV) (n=27) and CKD (n=29) with or without DM. Dapagliflozin (10mg) treatment was started in the CKD group. Urine and serum samples were collected before dapagliflozin treatment after 3 and 6 months. We measured urinary kidney injury molecule 1 (KIM-1), interleukin-1β (IL-1β) and mitochondrial DNA (mtDNA) copy number in the treatment group.

Results: The mean age of patients with CKD and HVs did not differ (54.2 ± 15.2 and 51.8 ± 8.3 years, respectively, p=0.485). Among CKD, 21 (72%) were men and 4 (13%) had underlying DM, and 24 (82 %) were taking an angiotensin receptor blocker. IgA nephropathy (n=12, 41%) was the most common etiology of CKD. The mean eGFR in the CKD group was 59.5 ± 23.8 mL/min and the urinary albumin to creatinine ratio (ACR) was 683.6 ± 768.4 mg/g. The levels of KIM-1, IL-1β and mtDNA, COX3 copy number were higher than those in HV. During the study period, dapagliflozin did not change ACR (p=0.948). eGFR decreased at 3months (p=0.020) and recovered. However, KIM1, IL-1β, COX3 were reduced at 6 months after treatment (p< 0.001, p<0.001 and p=0.026 respectively).

Conclusions: These results suggest that SGLT2i have an additional effect in reducing kidney damage CKD on SOC. This effect requires at least 6 months of SGL2i treatment period.