THE KOREAN SOCIETY OF NEPHROLOGY
APCN & KSN 2024
June 13(Thu) - 16(Sun), 2024 Coex, Seoul, Korea
Promoting Sustainable Kidney Health: The Asia-Pacific and Beyond

Abstract Submission No.: A-1252

Effects of Dietary Salt Intake on Intrarenal Immune cells And Sodium Buffer in Ischemic Acute Kidney Injury

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Objectives : Kidney immune cells play a role in ischemic acute kidney injury(AKI). Considering the known immunomodulatory capacities of sodium, we hypothesized that changes in dietary salt intake affect ischemic AKI outcomes by altering kidney immune cells and sodium buffer.
Methods : C57BL/6 mice were treated with normal(0.8%NaCl), low-salt(0.3%NaCl), or high-salt(8%NaCl) diet. After 6 weeks of allocated diet, we induced ischemic AKI followed by reperfusion and obtained kidneys at baseline(steady-state), 3 days(early), and 14 days(late). Kidney mononuclear cells were isolated and studied with flow cytometry. To compare kidney sodium buffer, glycosaminoglycan(GAG) concentration was measured from kidney protein extracts.
Results : Mice treated with high-salt and low-salt diets exhibited more severe injury after ischemic AKI(at 24h, serum creatinine, normal 0.91±0.20, low-salt 2.12±0.12, P<0.001; high-salt 1.75±0.19 mg/dL, P=0.003/ BUN, 55.7±13.8; 138.6±4.4, P<0.001; 127.8±6.1 mg/dL, P<0.001). $CD4^+$ T cells from mice fed with high-salt diet skewed toward effector-memory phenotype($CD44^{high}CD62L^{low}$) not only in steady-state kidneys(of total $CD4^+$ T cells, 44.2±2.6%; 57.1±3.5%, P=0.020; 62.1±2.5%, P=0.002) but also in post-ischemic kidneys at early(38.9±2.4%; 45.0±8.6%, P=0.605; 57.5±2.02%, P=0.028) and late time-points(62.9±3.6%; 76.2±2.9%, P=0.020; 76.9±2.6%, P=0.010). Post-ischemic kidneys from high-salt diet group had higher proportions of neutrophils(of total $CD45^+$ cells, early 14.9±1.14%; 22.3±3.6%, P=0.205; 29.9±4.2%, P=0.004/ late 16.2±3.2%; 35.1±3.4%, P=0.002; 33.7±3.0%, P=0.003) and lower B cells(of total lymphocytes, early 32.8±2.5%; 17.5±1.3%, P=0.093; 14.0±6.1%, P=0.037/ late 25.5±1.9%; 10.6±1.7%, P<0.001; 9.6±2.1%, P<0.001), compared to those from normal-diet group. Kidney GAG concentration increased during early after AKI(steady-state 0.82±0.09; early 2.04±0.40, P=0.032; late 0.45±0.23 μg/mg, P=0.627). High-salt diet induced GAG upregulation in steady-state kidneys(0.88±0.13; 1.06±0.07, P=0.374; 1.46±0.07 μg/mg, P=0.001), but AKI abolished this effect, showing comparable concentrations between groups in post-ischemic kidneys.
Conclusions : Dietary salt induced changes in kidney sodium buffer and kidney immune cell compositions, affecting ischemic AKI outcomes. Modulation of dietary salt intake could be a promising approach to potentially mitigate AKI.