Hypertensive Nephropathy

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Analysis of Risk Factors in Hypertension Patients with Chronic Kidney Disease Stage 5
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Objective: To analyse the risk factors for hypertension patients with chronic kidney disease stage 5 (CKDS).

Methods: Collecting 390 hypertension patients with CKDS, recording the basic information. Gender, age, primary disease, dialysis method, BMI, complications (hyperlipidemia, high uric acid, cardiac insufficiency), level of education, PTH were first examined by using univariate analysis. Univariate variables that showed statistical significance were then subjected to the multivariate analysis (logistic regression) to identify the risk factors for hypertension patients with CKDS; evaluating the level of DDD according to hypertension segmented standard.

Results: Overall hypertension control rate is 22.8%. (1) Univariate analysis showed the following variables had significant differences: > 40 years old, male, diabetic nephropathy, hypertensive nephropathy, hemodialysis, hyperlipidemia, high PTH (P < 0.05). (2) Logistic multivariate analysis showed that diabetic nephropathy, hyperlipidemia, high PTH were the major risk factors for hypertension patients with CKDS. (3) In hypertension segmented standard, the level of DDD was no different between 0 and 1 standard (P >0.05), DDD of 2 and 3 paragraphs were increased significantly compared with 0 and 1 paragraphs (P <0.05).

Conclusion: Overall hypertension control rate is only 22.8%. Male and younger (age < 65 year-old) female increased significant risk of AKI development with NASD (HR = 2.3; 95% CI = 1.57–3.16, p<0.001).

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ACE2/Mas Double Deficiency Promotes Angiotensin II-induced Renal Fibrosis by Enhancing the ERK1/2 MAPK-Smad3 Crosstalk Pathway
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Objective: Angiotensin II (Ang II) has been recognized as a key mediator in chronic kidney disease (CKD), particularly in hypertension-associated nephropathy. Increasing evidence shows that the angiotensin-converting enzyme 2 (ACE2)/Ang1-7/Mas receptor (Mas) axis plays a protective role in CKD by counter-regulating the pathogenic actions of the ACE2/Ang II type 1 receptor (AT1) axis. In this study, we investigated the role and underlying mechanisms of the ACE2/Ang1-7/Mas axis in Ang II-mediated renal injury.

Methods: A mouse model of hypertension was induced in ACE2 knockout (KO), Mas KO, double ACE2/Mas KO and their littermate wild-type (WT) mice by subcutaneous infusion of Ang II (1.0 mg/kg/day) or control saline for 28 days via osmotic mini-pumps. Blood pressure, serum creatinine (Scr) and creatinine clearance (Ccr), renal fibrosis including α-SMA and collagen matrix deposition, and signaling pathways related to Ang II-mediated renal injury were assessed.

Results: Deletion of ACE2 or Mas developed higher levels of blood pressure compared to WT mice, which was further increased in mice with double ACE2/Mas deletion at day 7 after Ang II infusion. Compared to single ACE2 or Mas KO mice, mice with double ACE2/Mas KO developed more severe hypertensive nephropathy as demonstrated by higher levels of Scr, a fall in Ccr, and progressive renal fibrosis with a marked accumulation of α-SMA+ myofibroblasts and collagen I matrix. Further study revealed that the development of more severe kidney injury in double ACE2/Mas KO mice was associated with enhanced AT1-dependent activation of ERK/MAP kinase-Smad3 crosstalk pathway.

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