Conclusion:
Overall hypertension control rate is only 22.8% in hypertension patients with CKD5.

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, hypertension 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% in hypertension patients with CKD5. Diabetic nephropathy, hyperlipidemia and high PTH are independent risk factors in hypertension patients with CKDS.

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Objective: Nonapnea sleep disorders (NASD) and sleep-related problems, which are high prevalent in patients with kidney diseases, are associated with cardiovascular disease, type 2 diabetes mellitus (DM) and chronic kidney disease (CKD). However, whether NASD is associated with acute kidney injury (AKI) development and prognosis have not been thoroughly investigated. The aim of this study is to determine whether NASD is an independent risk factor of AKI by using the database of National Health Insurance Research Database (NHIRD), which is one of the largest medical databases of the world.

Methods: The retrospective study used an 11-year nationwide database, which random sampled of 1,000,000 individuals covered by the National Health Insurance in Taiwan, to analyze the incidence. The patients with NASD were identified through diagnostic and medication codes. Kaplan-Meier and Cox regression analyses were performed.

Results: From 2000 to 2010, 9,316 newly diagnosed NASD cases compared with 27,948 control participants without sleep disorders randomly selected, frequency matched by age, sex, index year from the general population. We demonstrated that the NASD cohort had an adjusted hazard ratio (HR; 95% confidence interval [CI]) = 1.16–1.71) of subsequent AKI 1.44-fold higher than that of the cohort without sleep disorders. Elder age, lower monthly income, hypertension, DM, cerebrovascular disease, CKD status, depression and higher Charlson comorbidity index were significant factors associated with the increased risk of AKI (p<0.05). Male and younger (age<65 year-old) female increased significant risk of AKI development with NASD (HR=2.23; 95% CI = 1.57–3.16, p<0.001).

Conclusion: This nationwide population-based cohort study provides evidence that patients with NASD are at higher risk of developing AKI than people without NASD.

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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 1-7/Ang II type 1 receptor (AT1) axis. In this study, we investigated the role and underlying mechanisms of the ACE2/Ang 1-7/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 examined.

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. Moreover, 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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