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Non-dipper status and LVH may be the risk factors for the development of CKD in non-diabetic hypertensive patients

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Objectives: Non-dippers are known to be associated with increased incidence of various target organ damages in hypertensive patients. In this study, we hypothesized that non-dipper status would be associated with the development of chronic kidney disease (CKD) in patients treated with antihypertensive medications. **Methods:** This study included 102 non-diabetic hypertensive patients with estimated glomerular filtration rate (eGFR)≥60 mL/min/1.73 m2. Baseline demographic and laboratory data were assessed and 24-hr ambulatory blood pressure (BP) monitoring and echocardiogram were performed at the beginning of the study, and the levels of serum creatinine were followed up. Non-dippers were defined if their nocturnal systolic BP did not decrease by≥10% compared to the average daytime BP, and CKD was defined as a sustained decrease in eGFR of < 60 mL/min/1.73 m2. **Results:** The average duration of follow-up was 51 months (range 13-64 months). Participants (mean age 56.0±10.4 years, 39 men, initial eGFR 81.4±16.1 mL/min/1.73 m2) were divided into two groups, dippers (n=60) or non-dippers (n=42). There were no significant differences in age, duration of hypertension, the average fulltime BP, urine albumin/creatinine (A/C) ratio, and initial eGFR. During the follow-up period, CKD were developed in 11 patients, and there was a significant difference in the CKD incidence between dippers and non-dippers [3 (2.9%) vs. 8 (7.8%) patients, $p<0.05$]. Compared to the patients with eGFR≥60 mL/min/1.73m2, eGFR decline group showed higher urine A/C ratio (52.3±58.6 vs. 17.8±29.3 mg/g, $p<0.05$), presence of left ventricular hypertrophy (LVH) [3 (27.3%) vs. 5 (5.5%) patients, $p<0.05$] and lower serum HDL-cholesterol (41.7±8.3 vs. 50.4±12.4 mg/dL, $p<0.05$). In a multiple logistic regression analysis, non-dipper status (OR 2.4) and the presence of LVH (OR 3.3) were independent risk factors for the development of CKD. **Conclusion:** These findings suggest that non-dipper status and LVH may be the therapeutic targets for the prevention of the development of CKD in non-diabetic hypertensive patients.

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Renal ischemic preconditioning results in the resistance to second ischemic insult by upregulation of Akt

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Background: Renal ischemia is a major cause of acute renal failure. Although reperfusion is essential for the survivial of ischemic tissue,there is good evidence that reperfusion itself causes additional injury. Multiple studies have demonstrated that ischemic preconditioning(IPC) plays a protective role by inhibiting inflammatory response in the kidneys, but the molecular mechanism remains unclear. **Methods:** Adult male Sprague-Dawley rats weighing 160-200 grams were used. Rats were subjected to bilateral ischemia of varying durations (sham, 5,30,45minutes) by clamping both renal pedicles with nontraumatic microaneurysm clamps. According to the result of I/R model with varying duration, new rats are subjected to I/R injury in order to quantify apoptosis. The quantification of apoptosis was performed with DNA laddering and TUNEL method. According to the experiments, we divided new male Sprague Dawley rats into 8 groups. We induced ischemia by bilateral renal pedicle clamping for 45 minutes. Than we induced second ischemia at postischemic 7 days. Proteins were extracted from kidneys and the membranes were incubated with antibodies against phospho-Akt antibody. Immunohistochemistry of kidney for Akt was also done. **Results:** We found that 45 minutes of ischemic reperfusion injury showed resistance to secondary ischemic insults. After 45 minutes of prior ischemia, ischemic preconditioning attenuated the secondary ischemic insults at day 1 and 2 after second operation. Serum creatinine and BUN levels were significatly lower in prior ischemia group than sham group at 48 hours from second injury. Furthurmore, we found that prior ischemia attenuated apoptosis. Electrophoresis of DNA extracted from the kidneys of the rats with prior ischemia showed little DNA laddering as compared to sham groups. We found that Akt was more phosphorylated in ischemic kidney with preconditioning, and localized more phosphorylated Akt in ischemic kidney with preconditioning due to the western blot and immunohistochemistry results. **Conclusion:** Renal ischemic preconditioning results in the resistance to second ischemic insult. Akt may play an important role in the protective effect of renal ischemic preconditioning.