

Abstract Submission No. : IL-9079

Interpretation and adaptation of new AHA guideline for diagnosis and management of hypertension in CKD patients.

Hong Sang Choi

Chonnam National University Hospital, Korea, Republic of

Hypertension is the most common comorbidity of patients with chronic kidney disease (CKD) and is also one of the most important causes of CKD. The prevalence of hypertension in CKD patients is known to be 67-92% and the prevalence increases with declining renal function. Hypertension may occur as a result of CKD, and conversely, hypertension accelerates the progression of CKD. It is well known that hypertension is an independent risk factor for the development of end-stage renal disease, not only in patients with CKD but also in the general population. Therefore, treatment of hypertension is an important means to prevent kidney functional decline.

In the 2017 ACC / AHA hypertension guideline, the definition of hypertension was lowered to >130/80 mmHg when compared to the previous guidelines. The results of the Systolic BP intervention (SPRINT) trial, published in 2015, was the most important background for changing the definition of hypertension. In SPRINT trial, intensive BP control with systolic blood pressure (BP) less than 120 mmHg improved primary cardiovascular composite outcome and all-cause mortality compared to standard control with systolic BP less than 140 mmHg. However, subgroup analysis showed no significant improvement in primary outcome and renal outcome in patients with CKD at baseline. Incidence of incident CKD and acute kidney injury was also significantly increased in the intensive control group. Therefore, SPRINT trial showed not only the lack of significant renoprotective effect of intensive BP lowering but also it could be harmful. Nevertheless, the 2017 ACC / AHA guideline adjusted the BP goal to lower than 130/80 mmHg regardless of the presence or absence of proteinuria in patients with CKD.

Since the SPRINT trial was published, a number of post-hoc analyzes of kidney disease patients have been made. In the CKD subgroup, intensive BP lowering improved the all-cause mortality ($p=0.04$) and cardiovascular mortality ($p = 0.06$). In the intensive BP lowering group, eGFR was rapidly decreased in the first 6 months, but it was considered to be a hemodynamic effect because there was no difference in the renal damage marker in another published study. Similar trends were observed in patients with incident CKD. The incidence of AKI was also higher in the intensive treatment group, but in most cases, mild and over 90% of patients were in complete resolution.

Although intensive BP control may lead to a slightly faster renal function decline and more AKI events, these adverse outcomes were reversible and clinically mild. Thus, the cardiovascular benefit and reduction of mortality risk of intensive BP control outweigh the adverse renal effects.