

진행성 신장질환에서 RAAS 억제제의 효과

전남대학교 의과대학 내과학교실

배 은 희

RAAS blockade in Progressive Renal Disease

Eun Hui Bae

Departments of Internal Medicine, Chonnam National University Medical School, Gwangju, Korea

The renin–angiotensin–aldosterone system (RAAS) play a dominant role in the pathophysiology of hypertension, diabetes mellitus and chronic kidney disease. Therefore, drugs that block key components of the RAAS such as ACE inhibitors and ARBs have gained wide clinical use for these indications. Similarly, RAAS inhibitors have been used increasingly in patients with underlying renal disease as mounting evidence has pointed to the agents having an antiproteinuric effect that is independent of blood pressure lowering. This effect is considered important in light of two key observations. Several large interventional studies have uniformly found that proteinuria is a major risk factor for the progression of renal disease. Furthermore, the reduction of proteinuria in patients with underlying diabetic nephropathy has been associated with a decreased risk for a renal end–point. A similar observation was seen in patients with nondiabetic kidney disease with lower levels of baseline proteinuria, thereby demonstrating the importance of lowering protein excretion in a variety of renal disorders and across a broad spectrum of protein excretions. Despite progress, the morbidity and mortality of patients treated with ACEi or ARBs remain high. Small molecules that directly inhibit renin (DRI) and are orally active have also been developed and one such drug, aliskiren, was introduced into clinical use for treatment of hypertension in 2007. The information already gained with aliskiren, raises questions regarding the advantages of DRIs as monotherapy compared to marketed ACEis and ARBs, their potential added value in combination with other RAAS modulators and other unproven benefits in relation to prorenin and rennin receptor biology.