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Letter to the Editor

Chronic Kidney Disease due to Acute Kidney Injury and Acute Kidney Injury as a Risk Factor of CKD: Earlier Initiation of Hypotensive Therapy with a Focus on Long-Term Prognosis

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Preexisting chronic kidney disease (CKD) is the most important risk factor of acute kidney injury (AKI). Many studies have reported that the risk of AKI increases as the CKD stage advances [1-3]. Furthermore, although attention has been paid to CKD due to AKI [4-6], as treatment at the time of AKI onset is usually performed by emergency or intensive-care physicians and because kidney function recovers after AKI in most cases, awareness that these patients may someday develop CKD is lacking. Therefore, from the standpoint of renal protection, patients should be referred to internists immediately after the onset of AKI and given hypotensive agents known to have renal-protective effects, such as calcium channel blockers (CCB) or angiotensin receptor blockers (ARB). For CCB, an understanding of the different subtypes is important. Drugs that block not only L-type but also T- and N-type calcium channels have been found have a renal protective effect [7,8]. For ARB, we performed a 24-week prospective study in 12 patients with hypertension who were receiving maintenance dialysis. For the first 12 weeks, they received valsartan 80 mg/day, which was then changed to olmesartan 20 mg/day for the second 12 weeks. We measured blood pressure, plasma renin activity, blood aldosterone concentration, and plasminogen activator inhibitor 1 (PAI-1) as a marker of vascular endothelial damage. No significant difference in blood pressure and plasma renin activity were observed before and after the drug was changed. However, blood aldosterone concentration and PAI-1 levels declined significantly after the drug was changed. This was not a crossover study, so whether these changes could have been caused by ARB administration itself could not be determined. Still, olmesartan has been reported to have better effect in terms of increasing endogenous Ang1-7 level via ACE2 activity [9,10]. This suggests that the renal-protective action as mediated by ACE2/Ang1-7/Mas receptors may differ depending on the type of ARB, which is something that has received attention recently. While it is important to administer hypotensive agents to patients immediately after AKI from the standpoint of renal protection, the above-mentioned findings indicate that this should be based on an understanding of the class effects of CCB and ARB, as well as the effects of the individual drugs.

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