Management of diabetic kidney disease: intraglomerular pressure matters and the beneficial effects of Aliskiren

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Activation of the renin–angiotensin–aldosterone system (RAAS) plays a key role in the progression of Diabetic kidney disease (DKD). RAAS inhibitors, such as angiotensin converting enzyme inhibitors (ACEis) and angiotensin II receptor blockers (ARBs), decrease the rate of DKD progression and are first-line anti- hypertension therapies for DKD. Although these agents are highly effective, current therapeutic strategies are unable to sufficiently suppress the RAAS and stop CKD progression. Since angiotensin II inhibits renin release, a large increase in plasma renin activity occurs with the administration of either ACEis or ARBs by feedback mechanisms. After several weeks of ACEi or ARB therapy, plasma aldosterone returns to pretreatment levels in up to 30–40% of patients. This phenomenon has been termed aldosterone breakthrough. Patients who demonstrate aldosterone breakthrough have a worse clinical prognosis than those who do not. Aliskiren, the first in a new class of RAAS-inhibiting agents (direct renin inhibitors, DRI) has been approved to treat hypertension and provides dose-dependent efficacy and sustained 24-hour blood pressure control. The inhibition of renin by aliskiren is associated with a reduction in circulating levels of angiotensin I and II, which may possibly eliminate the breakthrough effect. Therefore, DRI theoretically exerts better renal and/or cardiovascular protective effect in patients receiving ACEi or ARB treatment who demonstrate aldosterone breakthrough.