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SGLT-2 inhibitor in Diabetic Nephropathy

*Eun young LEE

Internal Medicine, Soonchunhyang University Cheonan Hospital, Korea,South

Metabolic and hemodynamic components are the important players in the development and progression of diabetic nephropathy towards end-stage kidney disease. Control of blood glucose and blood pressure are the mainstays to prevent kidney damage and slow its progression. There is emerging evidence that some hypoglycemic agents may have renoprotective effects which are independent of their glucose-lowering effects.
Of the new glucose-lowering drugs, the sodium-glucose co-transporter-2 (SGLT2) inhibitor is of particular interest to the nephrologists. First, the kidney is the main site of action for SGLT2 inhibitors, which waste glucose in the urine by blocking sodium-coupled glucose reabsorption in the proximal tubule. Second, SGLT2 inhibitors increase afferent arteriolar tone and decrease intraglomerular pressure by increasing distal tubular sodium delivery and stimulating tubuloglomerular feedback. In addition, SGLT2 inhibitors lead to modest decreases in weight and BP, presumably through natriuretic effects. Decreased sodium reabsorption could also plausibly affect proximal tubular cell energetics and therefore other functions of these metabolically active cells. Ultimately, recent advances in diabetes treatmemt should help prevent kidney disease by directly targeting the kidney, providing an expanded menu of options to effectively and safely control glycemia, or both. New glucose-lowering medications will hopefully also improve outcomes for patients with diabetes and established chronic kidney disease though further studies in this population are needed, as are new classes of drugs targeting other injury pathways leading to CKD progression.
In conclusion, SGLT2 inhibitor slowed the progression of renal disease in patients with type 2 diabetes, and SGLT2 inhibitor may confer renoprotective effects independently of its glycemic effects. Therefore, SGLT2 inhibitor may offer a novel therapeutic option for patients with type2 diabetes who are at a high risk of kidney failure.