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사구체손상의 예측인자로 요중 Synaptopodin 농도의 유용성

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Urinary Synaptopodin Excretion as a Predictor of Glomerular Disease Progression

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Intriduction and Aims: Podocytes play an important role in maintaining glomerular filtration barrier, and make slit diaphragm that is essential for preventing albumin loss and filtering water and solutes. Diabetic kidney disease is progressive kidney failure caused by intraglomerular hypertension showing heavy proteinuria. Urinary podocyte loss is associated with diabetic kidney disease progression, but it is not clear that the amount of podocyte or podocyte slit diaphragm protein can reflect clinical significance of glomerular damage. We investigated the correlation between the amount of urinary slit diaphragm proteins and renal function and albuminuria.

Methods: Total 40 patients with diabetic chronic kidney disease and glomerular disease patients were enrolled, and the amount of urinary podocyte and slit diaphragm proteins were measured by Western blot analysis. Amount of urinary nephrin, podocalyxin, podocin, synaptopodin and beta actin were measured by Western blot band density. Each urine sample was loaded as same amount of total sample protein, and adjusted by beta-actin band density in the setting of same beta-actin band density. We measured serum creatitine and sopt urine albumin/creatinine ratio as a marker of renal damage, and compared correlation of urinary podocyte protein between disease groups or renal disease progression.

Results: 15 Patients were diabetes, 25 were glomerulonephritis and others were unspecified chronic kidney disease. Mean age was 34±15.8 years old, mean serum creatinine was 2.01±1.37 mg/dL, mean albumin/creatinine ratio was 4.65±3.60. Urine albumin excretion showed no difference between diabetes and non-diabetes ($p=0.26$), and that showed no correlation with serum creatinine level ($p=0.81$). The reference beta-actin band density showed no significant difference between diabetes and non-diabetes ($p=0.60$), and band density of podocalyxin/actin and nephrin/actin and showed no significant difference between patient group ($p=0.31$, 0.58). However, synaptopodin/actin and podocin/actin ratio between diabetes and non-diabetes showed remarkable difference (6.43±3.79 vs. 2.79±3.29, 8.46±9.43 vs. 4.47±7.67, respectively, $p<0.01$). Serum creatinine level showed significant correlation only with urinary synaptopodin/actin ratio ($r=0.58$, $p<0.001$) in contrast to nephrin, podocalyxin and podocin showed no significant correlation.

Conclusions: In conclusion, amount of urinary synaptopodin excretion is increased in diabetic kidney disease comparing with glomerulonephritis, and it showed significant correlation with serum creatinine elevation in glomerulonephritis. We suggest that urine synaptopodin level can be a predictor of glomerular damage regardless of urine albumin excretion.

Key Words: 시냅토포딘, 단백뇨, 사구체손상
Synaptopodin, Proteinuria, Glomerular damage