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The possibility of renal regeneration by prostaglandin in IgA nephropathy and FSGS

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Objectives : Glomerulonephritis (GN) is one of the major causes of chronic kidney disease. Injured renal tissue is attributed to proteinuria and loss of renal function. Recently, inhibition of 15-hydroxyprostaglandin dehydrogenase (15-PGDH), a prostaglandin-degrading enzyme, was proven to potentiate tissue regeneration. The majority of GN treatment focused on anti-proteinuric effect to prevent disease progression. We wanted to know the possibility of renal regeneration by 15-PGDH in GN and planned to review the COX2 protein and renin-angiotensin-aldosterone (RAAS) system through the bio specimen.

Methods : We collected blood and urine samples during the renal biopsy for 1 year from Aug 2015. Among them, we selected specimens with IgA nephropathy and focal segmental glomerular sclerosis (FSGS). Plasma COX2 protein (RayBio® Human COX-2 ELISA Kit, RayBiotech, Norcross, GA) and urine angiotensinogen (Human total Angiotensinogen Assay Kit, IBL, Japan) were analyzed and compared with pathologic degree (sclerosis, tubular atrophy), proteinuria degree with spot urine sample (I <0.5, 0.5 ≤ II <1, 1 ≤ III <3, IV ≥ 3) and other basic parameters.

Results : Sixty nine patients (44 of IgA nephropathy, 25 of FSGS) were analyzed. There was no sexual differences between the groups, although IgA nephropathy group was younger (36.1 vs. 43.4 years, p = 0.05). The average amount of proteinuria was 1.406 g/g for IgA nephropathy and 3.471 g/g for FSGS (p = 0.07). Urinary angiotensinogen was 529.27 ng/mL for IgA nephropathy and 1738.5 ng/mL for FSGS (p = 0.25) and plasma COX2 protein was 41.35 ng/mL for IgA nephropathy and 43 ng/mL for FSGS (p = 0.8) without any significant difference between groups. There was no clinical correlation with COX2 protein and urinary angiotensinogen level in both group (IgA nephropathy; p = 0.63, FSGS; p = 0.47). In IgA nephropathy, COX2 protein was correlated with proteinuria degree (r = 0.28, p = 0.06). Also, Urinary angiotensinogen was correlated with proteinuria degree (r = 0.7, p <0.01), hypertension (r = 0.48, p <0.01), histologic change (sclerosis; r = 0.34, p= 0.02, tubular atrophy; r = 0.38, p = 0.01). In FSGS, only urinary angiotensinogen was related with proteinuria (r

= 0.63, p = 0.01).

Conclusions : According to this study, tissue regeneration by 15-PGDH seemed to be impossible with IgA nephropathy and FSGS because prostaglandin had positive or no relationships with proteinuria. The control of intra-renal RAAS by RAAS blockade is a mainstay in dealing with this GN. However, we can try COX2 inhibitor to reduce proteinuria with normotensive and hypotensive patients IgA nephropathy patients who cannot use RAAS blockade and with heavy proteinuric IgA nephropathy patients in combination with RAAS blockade.

Keywords : PGDH, COX, Glomerulonephritis