

Evaluation of serum spondin 2 levels in the different etiologies of glomerular diseases

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Objectives:

Spondin 2 (SPON2, mindin) is a member of the Spondin 2/F-spondin family of extracellular matrix proteins. SPON2 plays important roles in multiple processes, such as the promotion of neuron outgrowth, inhibition of cancer and angiogenesis, involvement in the immune response and inflammatory processes, and inhibition in the development of obesity, hepatic steatosis, inflammation and insulin resistance. Recent studies have shown that podocyte injury plays a role in the pathogenesis of various glomerular diseases, including diabetic nephropathy (DN). Mindin is excreted in the urine in patients with type 2 diabetes and DN, and may be related to podocyte injury in these patients. In this study, we investigated serum SPON2 levels and its correlation with urine albumin excretion in different glomerular diseases.

Methods:

The cohort included 144 consecutive adults with persistent proteinuria (>300 mg/day) and 22 healthy controls. The serum SPON2 levels were determined using a commercial sandwich enzyme-linked immunosorbent assay kit from Cloud-Clone Corp (Houston, TX, USA).

Results:

The etiologies of diseases are focal segmental glomerulosclerosis (FSGS, n:26), membranous glomerulonephritis (MGN, n:30), IgA nephropathy (IgAN, n:17), mesangioproliferative glomerulonephritis (MPGN, n:5), diffuse proliferative glomerulonephritis (DPGN, n:5), lupus nephritis (n:5), amyloidosis (n:22), polycystic kidney disease (APKD, n:16) and DN (n:18). There was a significant difference between the age distribution, hsCRP, daily urinary protein excretion, serum uric acid and albumin levels of groups. The gender ratios, serum BUN, creatinine and lipid profile were similar. Serum SPON2 levels in MGN (67 ± 36 ng/mL), FSGS (57 ± 34 ng/mL), IgAN (57 ± 34 ng/mL), DPGN (52 ± 10 ng/mL) and amyloidosis (58 ± 41 ng/mL) groups were higher than those of the control (28 ± 13 ng/mL) and APKD groups (22 ± 28 ng/mL). SPON2 levels in DN (35 ± 18 ng/mL) were lower than MGN, FSGS and IgAN groups and higher than that of APKD group. The patients with glomerulonephritis were divided by two groups as proliferative (IgAN, MPGN, DPGN and lupus nephritis) and non-proliferative (MGN and FSGS). Serum SPON2 levels of proliferative (52 ± 32 ng/mL) and non-proliferative (62 ± 35 ng/mL) groups were comparable. Also SPON2 levels of non-proliferative group were significant higher than DN group. Serum SPON2 levels were inversely correlated with serum total protein levels ($r=-0.2$, $p=0.024$) and positively with urinary protein excretion ($r=0.33$, $p<0.001$, figure 1), but not with age, hsCRP and other studied parameters.

Conclusions:

Finally, our study firstly revealed that there were differences between serum SPON2 levels in different glomerular diseases. Serum SPON2 increase was more prominent in MGN, FSGS, IgAN and amyloidosis groups, respectively, and correlated with increment in proteinuria. SPON2 may be produced by damaged podocytes and also serve as a biomarker of the progression of glomerular diseases.

Figure 1:

