KSN 2021
FULLY VIRTUAL MEETING
September 02 (Thu) - 05 (Sun)

Abstract Submission No. : 2163

Serum levels of Plasminogen Activator Urokinase Receptor and Cardiotrophin-Like Cytokine Factor 1 in serum of patients with nephrotic Syndrome

Natalia Chebotareva, Anatoliy Vinogradov, Venzsin Cao, Alla Gindis
Department of Nephrology, Sechenov University, Russia

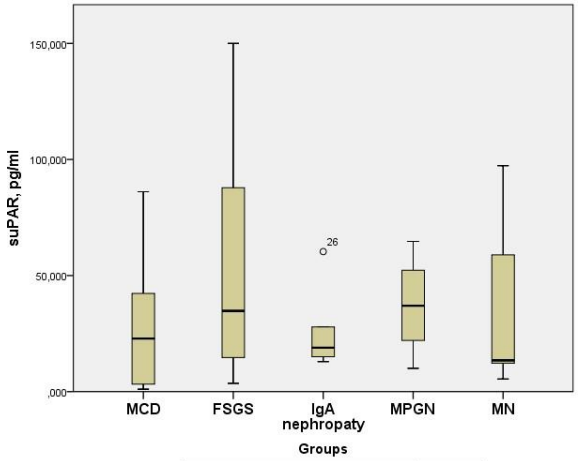
Objectives: The pathogenesis of primary focal segmental glomerulosclerosis (FSGS) and minimal change disease (MCD) remains unknown to date. Some circulating permeability factors are discussed. This work assessed molecule candidates for permeability in serum samples of patients with nephrotic syndrome (NS).

Methods: Forty-one patients with chronic glomerulonephritis (CGN) were included in our study. Seventeen patients had FSGS, 7 patients had MCD, 5 patients had membranoproliferative glomerulonephritis (MPGN), 6 patients had IgA nephropathy, and 6 patients had membranous nephropathy (MN). The laboratory data were compared with the clinical and histological features of nephritis. Serum levels of uPAR and CLCF-1 were measured by ELISA.

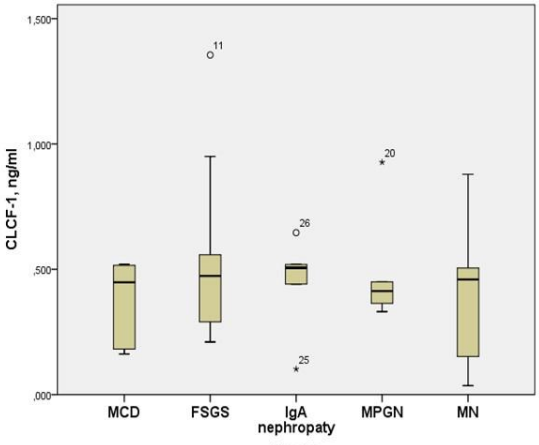
Results: The serum levels of plasminogen activator urokinase receptor (uPAR) were higher in FSGS patients before treatment than in patients with other morphological forms (MCD, IgA nephropathy, MN and MPGN). The levels of uPAR in serum did not correlate with daily proteinuria, serum creatinine/eGFR, arterial hypertension, the number of sclerosed glomeruli or tubulointerstitial fibrosis. No correlations were found between the levels of cardiotrophin-like cytokine factor 1 (CLCF-1) in serum and creatinine levels/glomerular filtration rate, the percentage of sclerosed glomeruli or the severity of tubulointerstitial fibrosis. There were no significant differences between the histological variants of nephritis. However, we found correlations between CLCF-1 levels and proteinuria and lipid levels.

Conclusions: The data indicate an increase in the serum uPAR levels of FSGS before treatment. CLCF-1 levels in serum do not depend on histological forms of CGN, kidney function or immunosuppressive treatment, but they correlate with proteinuria and serum lipids in patients with NS.

Figure



Histological forms of nephritis		p
FSGS vs.	MCD	0.983
	IgA nephropathy	0.056
	MPGN	0.991
	MN	0.953

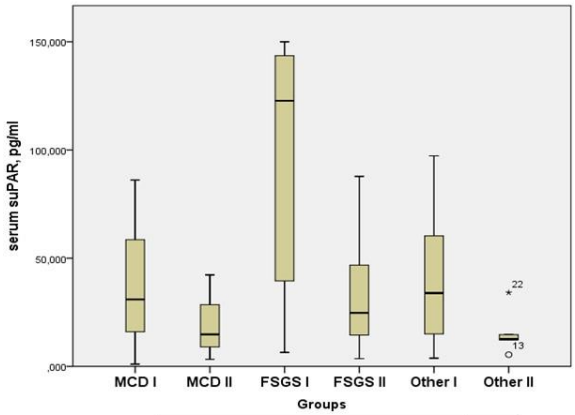


Histological forms of nephritis		p
FSGS vs.	MCD	0.847
	IgA nephropathy	1.000
	MPGN	1.000
	MN	0.999

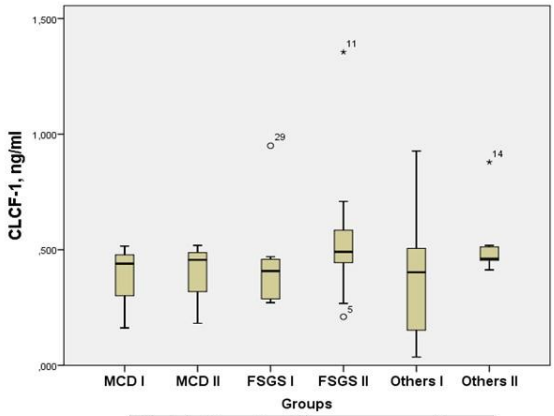
Figure 1. suPAR and CLCF-1 serum levels in patients with nephrotic syndrome

suPAR – soluble Plasminogen Activator Urokinase Receptor; CLCF-1 - Cardiotrophin Like Cytokine Factor 1; MCD – minimal change disease; FSGS – focal segmental glomerulosclerosis; MPGN – membranoproliferative glomerulonephritis; MN – membranous nephropathy.

Figure 2



Histological forms of nephritis		p
FSGS I vs.	MCD I	0.194
	MCD II	0.036
	FSGS II	0.011
	Other forms I (IgAN, MPGN, MN)	0.030
	Other forms II (IgAN, MPGN, MN)	0.003



Histological forms of nephritis		p
FSGS I vs.	MCD I	>0.05
	MCD II	>0.05
	FSGS II	>0.05
	Other forms I (IgAN, MPGN, MN)	>0.05
	Other forms II (IgAN, MPGN, MN)	>0.05

Figure 2. suPAR and CLCF-1 serum levels in patients with nephrotic syndrome depending on therapy

suPAR – soluble Plasminogen Activator Urokinase Receptor; CLCF-1 - Cardiotrophin Like Cytokine Factor 1; MCD – minimal change disease; FSGS – focal segmental glomerulosclerosis; MPGN – membranoproliferative glomerulonephritis; MN – membranous nephropathy; I – blood samples were taken before treatment; II – blood samples were taken during the treatment.