EARLY MARKERS OF TUBULOINTERSTITIAL FIBROSIS IN CHILDREN WITH IDIOPATHIC NEPHROTIC SYNDROME TREATED WITH CYCLOSPORIN A

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BACKGROUND

A glomerulopathy associated with recurrent or persistent proteinuria may lead to tubulointerstitial fibrosis and chronic kidney disease. Early detection of tubulointerstitial fibrosis is important for proper management and prognosis in kidney diseases. The aim of the study was to assess the plasma and urine levels of tubular proteins: annexin V (AnV), uromodulin (UM) and kidney injury molecule-1 (KIM-1) in children with idiopathic nephrotic syndrome (INS) treated with cyclosporine A (CsA). AnV, UM and KIM-1 are suggested to be novel early markers of tubular injury.

MATERIAL AND METHODS

The prospective study included 30 patients with steroid-dependent or steroid resistant INS and 22 healthy children (reference group, R). Plasma and urinary AnV, UM and KIM-1 were determined by ELISA in INS patients three times: before CsA treatment, after 6 and after 12 months of therapy, in the reference group - once.

Table 1. | Characteristics of study groups

	Children with NS n=30 Mean ± SD Median (1 - 3 quartile)	**Reference group n=22** Mean ± SD Median (1 - 3 quartile)	p
Age (years)	9.08 ± 3.99 9.38 (5.9 - 12)	10.50 ± 3.39 10.25 (7 – 14)	0.21
Gender: F/M (%)	8/22 26.67/73.33	7/15 31.81/68.19	0.92

RESULTS

Urinary AnV/creatinine ratio, urinary UM/creatinine ratio and KIM-1/creatinine ratio were significantly higher in INS patients before CsA therapy in comparison to R group ($p<0.001$).

We observed the progressive increase of urinary AnV after 6 and 12 months of therapy ($p<0.01$, despite the remission of NS). Urinary KIM-1 and UM increased after 6 months; next their levels were stable.

Plasma concentrations of AnV, UM and KIM-1 were changed similarly.

No significant correlations were found between plasma and urinary levels of investigated parameters.

The significant relationships between AnV, UM and KIM-1 in urine were noted.

Urinary AnV, UM and KIM-1 were not associated with magnitude of proteinuria.

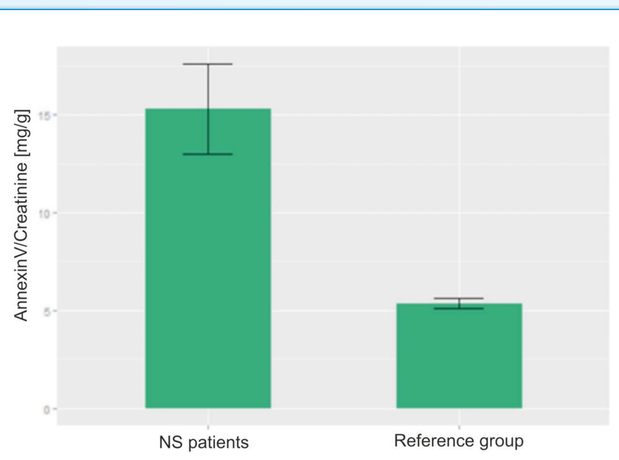

Fig. 1 | Urinary concentration of AnV before CsA therapy

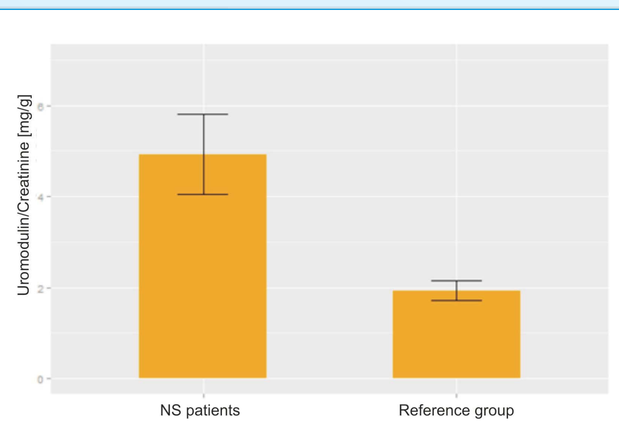

Fig. 2 | Urinary concentration of UM before CsA therapy

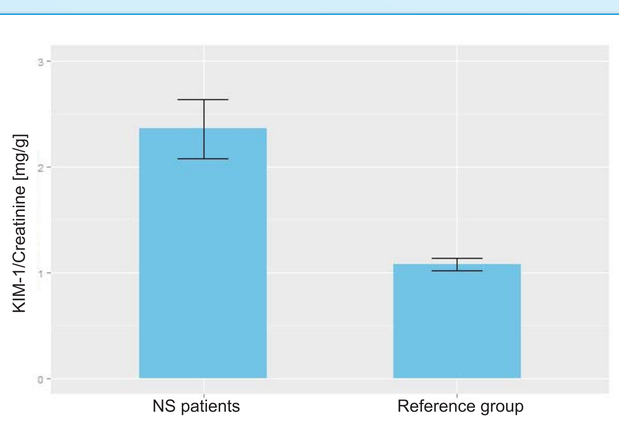

Fig. 3 | Urinary concentration of KIM-1 before CsA therapy

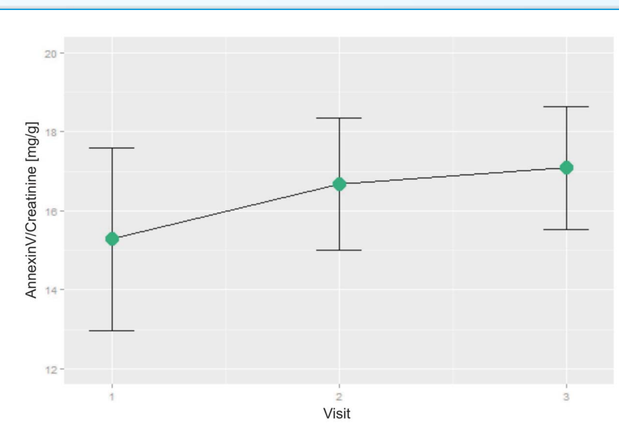

Fig. 4 | Urine concentration of An V in NS patients before CsA treatment, after 6 and after 12 months of therapy

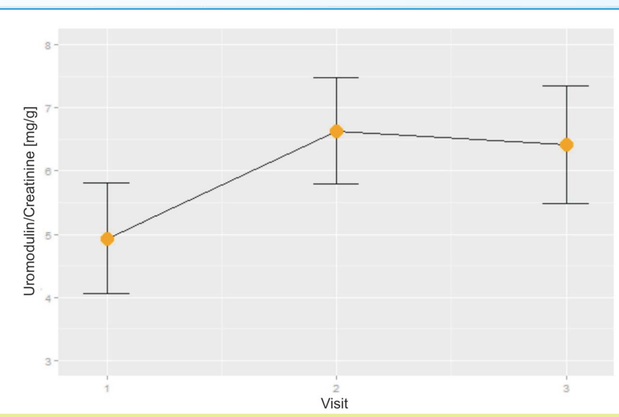

Fig. 5 | Urinary concentration of UM in NS patients before CsA treatment, after 6 and after 12 months of therapy

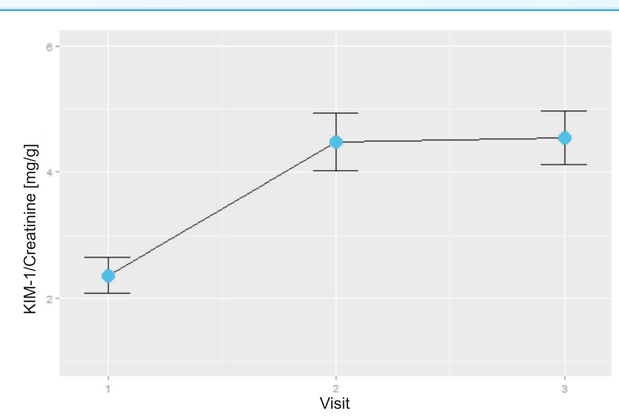

Fig. 6 | Urinary concentration of KIM-1 in NS patients before CsA treatment, after 6 and after 12 months of therapy

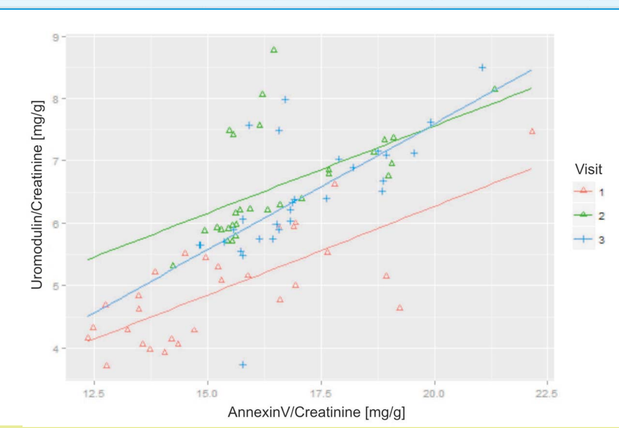

Fig. 7 | Correlation between urinary concentrations of AnV and UM in NS patients in three time points

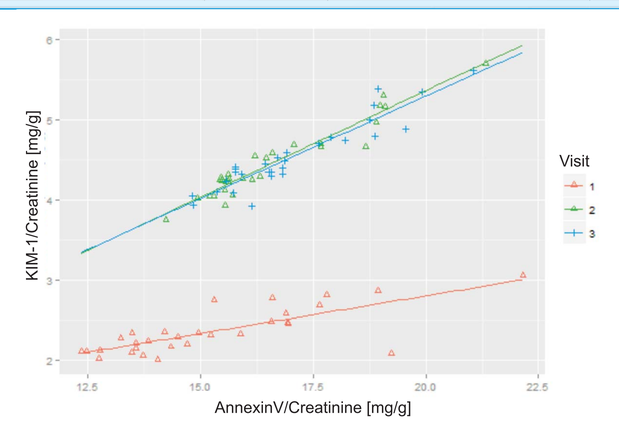

Fig. 8 | Correlation between urinary concentrations of AnV and KIM-1 in NS patients in three time points

CONCLUSION

Increased urinary excretion of annexin V, KIM-1 and uromodulin in children with INS treated with CsA suggests that tubulointerstitial fibrosis develops early in the course of INS, initially without kidney function impairment. AnV seems to be a more sensitive indicator of tubular damage in the course of CsA therapy than other investigated markers; however, large multicenter studies are needed to confirm these observations.

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