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The balance of proinflammatory cytokines and Treg cells in chronic glomerulonephritis

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Objectives: Chronic glomerulonephritis (CGN) with nephrotic syndrome is an immune-mediated disease with high activity of immune inflammation. An imbalance between the T helper cells and cytokines is suggested to induce CGN.

Methods: Adult patients with CGN (n=98) were recruited. 47 of the patients had proteinuria that ranged from 1 to 3 g/day, and 51 patients were diagnosed with nephrotic syndrome (NS). Half of the patients had kidney dysfunction, due to high nephritis activity. 10 healthy subjects were evaluated as a control. 18 patients had focal segmental glomerulosclerosis (FSGS), 11 -membranoproliferative glomerulonephritis (MPGN), 20 - IgA nephropathy, and 16 - membranous nephropathy (MN). We used the commercial ELISA kits to determine the levels of cytokines (IL-17A, IL-6, and TNF-α) in urine and we studied the number of Treg cells in the interstitium of the renal cortex with the immunohistochemical method.

Results: The levels of TNF- α, IL-10, IL-17A and IL-6 in the urine of patients with CGN were significantly higher than that in the healthy individuals. IL-17 and TNF-α levels in the urine were significantly increased in the patients with MCD/primary FSGS compared to other forms. The IL-17A and Il-6 levels were significantly higher in the patients with a renal dysfunction with eGFR less than 60 ml/min/1.73 m2 and in the patients with advanced interstitial fibrosis compared to levels of TNF-α. We revealed a decrease in the number of Treg FoxP3-positive cells in the tubulointerstitial compartment in FSGS and MN with NS compared with that in other histological variants of CGN.

Conclusions: The data indicate that cytokine imbalance was the most pronounced in the activation of IL-17 and TNF-α; a decrease in the regulatory anti-inflammatory link (Treg) in the kidney tissue was observed in FSGS, the most severe form of CGN.

Figure 1

Fig. 1. IL-17A, IL-6 and TNF-α in the urine of patients with different stages of tubulointerstitial fibrosis (TIF)

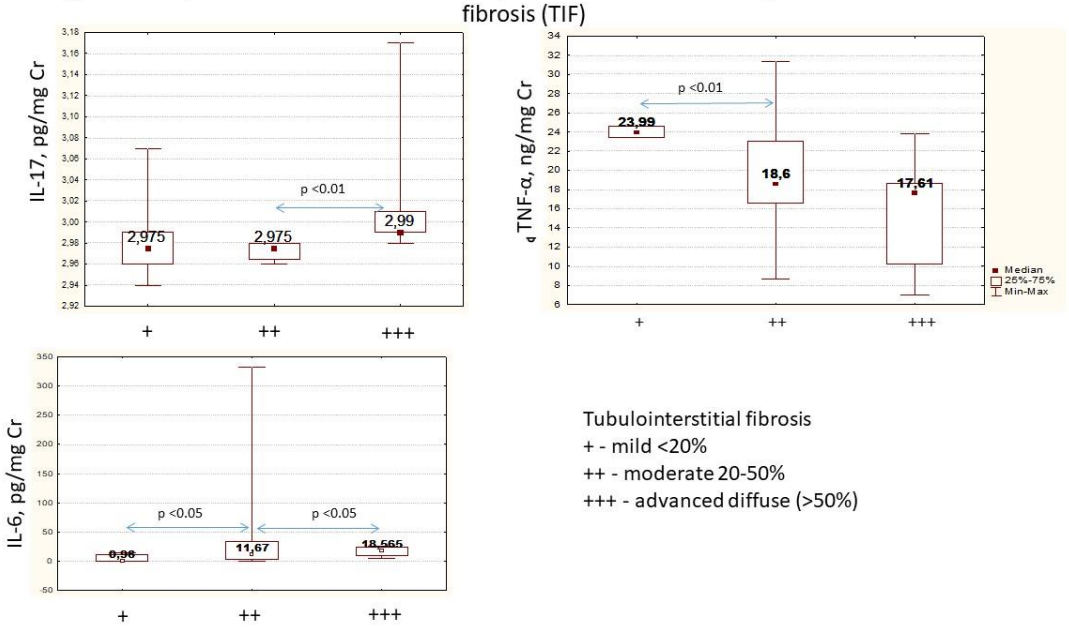
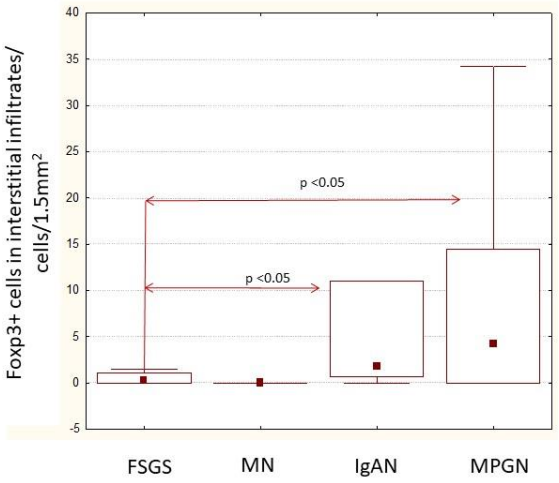


Figure 2

Fig.2 Treg Foxp3+ cells in interstitial infiltrates in various CGN forms



FSGS – focal segmental glomerulosclerosis,
MN – membranous nephropathy,
IgAN – Immunoglobulin A nephropathy,
MPGN – membranoproliferative glomerulonephritis