CORRELATION OF PODOCYTE ULTRASTRUCTURAL CHANGES AND LEVEL OF PROTEINURIA IN PATIENTS WITH DIFFERENT FORMS OF PRIMARY GLOMERULOPATHIES



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Objectives:

Methods:

Loss of podocytes in primary glomerulopathies is crucial for glomerulosclerosis progression which leads to end-stage renal failure in such patients. The mechanism of direct replacement of injured podocytes does not exist so the only way to compensate the integrity of glomerulus is change of cells shape to cover the glomerular tuft with a smaller number of podocytes. Foot process effacement is the typical morphological sign of podocyte respond to stress. Podocyte detachment (PD) from glomerular basement membrane (GBM) develops when podocyte hypertrophy is unsufficient. The aim of investigation was estimation of relationship between range of foot process width (FPW), PD and level of daily proteinuria in patients with primary variants of glomerulopathies.

with biopsy primary patients proven glomerulopathies were included in the study. According to the the results of light and electron microscopy 17 (40,5%) patients had membranous nephropathy, 8 (19,0%) - focal segmental glomerulosclerosis, 12 (28,6%) - minimal change disease and 5 (11,9%) - proliferative variants of glomerulonephritis (2- lgA-nephropathy, 3 - membranoproliferative glomerulonephritis). Standart laboratory and instrumental investigations were performed. Samples of serum and urine were obtained in the day of byopsy. FPW and PD were measured using Image J software (NIH, 1997). FPW was counted as ratio of GBM length to amount of foot processes in every electronogramm using correction factor π/4 as described in previous works. PD was calculated as percentage of bare areas of GBM.

Graphs and tables

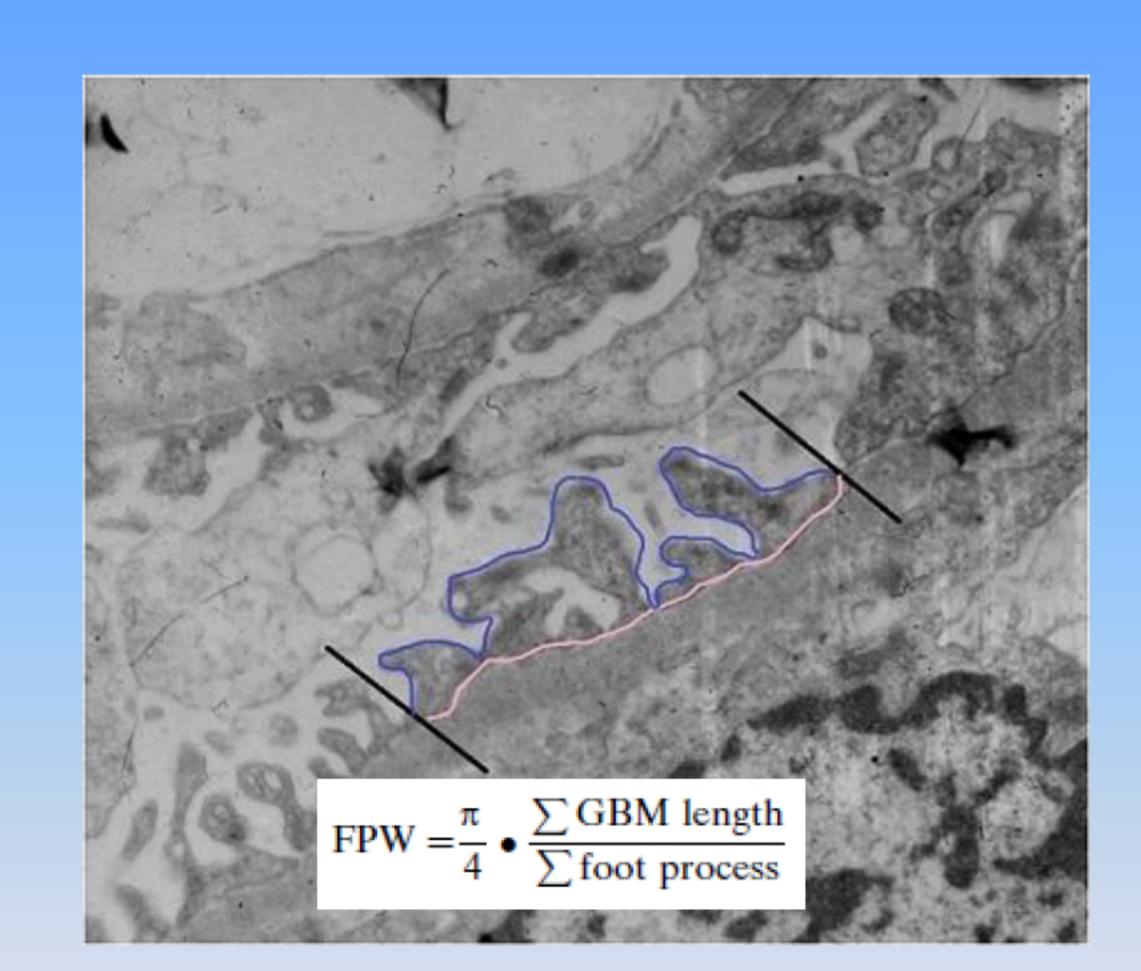


Figure 1: FPW estimation

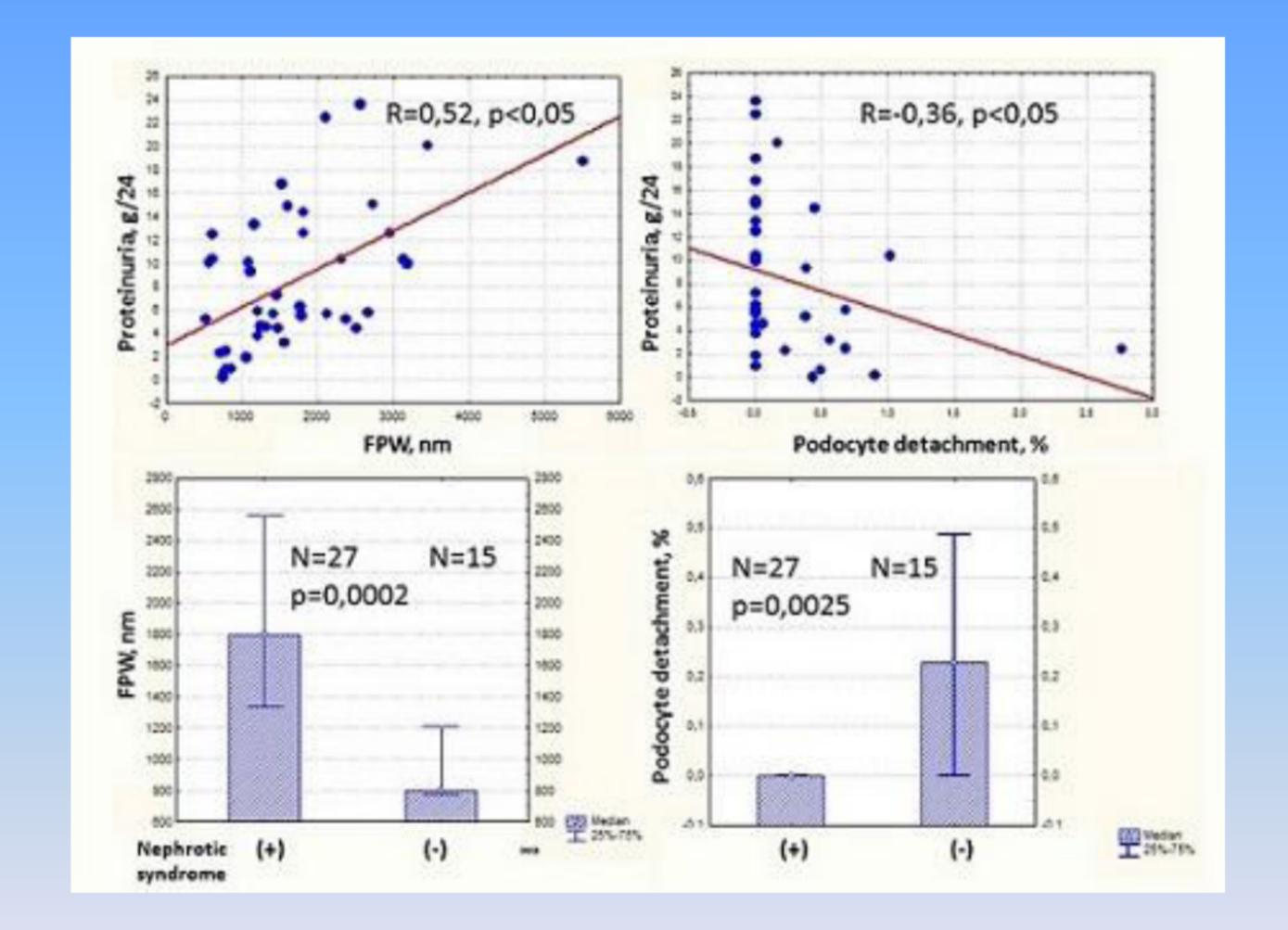


Figure 2: Correlation of FPW and PD with daily proteinuria and in patients with or without nephrotic syndrome

Results:

There were no statistically significant differences between FPW and PD in patients with different forms of glomerulopathies (p>0,05). There was negative correlation between FPW and PD (r=-0,31, p<0,05). Daily proteinuria rate positively correlated with FPW (r=0,52, p<0,05) while inverted relation with level of PD was found (r=-0,36, p<0,05). The same pattern was detected comparing groups of patients with and without nephrotic syndrome. The level of daily proteinuria was higher in patients with more expressed hyaline droplet degeneration of tubular epithelial cells.

Conclusions:

Unlike data published in recent works we found no difference of FPW and PD rate in patients with different forms of glomerulonephritis. Strong positive correlation of FPW with proteinuria range confirms the role of podocytes in development of high proteinuria and nephrotic syndrome, considering that there were no abnormalities in tubular reabsorbtion of protein. Interestingly the detachment of podocytes from GBM does not increase proteinuria range, more over inverse relationship was detected. Probably this fact can be explained by unknown mechanisms of transcellular transport of protein rather than directly through bare parts of GBM.



