KSN 2016 Abstract Submission

Clinical Nephrology

KSN2016ABS-1043

Apoptosis-dependent kidney damages in children with primary and secondary proteinuric diseases

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Background: Research shows that the level and type of proteinuria (whether the urinary proteins are albumin only - albuminuria) are a good indicator of the extent of kidney damage and a predictor of the irreversible kidney damage. Proteinuria may occur as a sing of kidney disease that are primary proteinuric i.e. nephrotic syndrome and in kidney diseases that develop proteinuria secondary to primary injury (diabetic nephropathy).
Purpose. The aim of the study was to investigate the level of metabolic-hypoxic disorders and the condition of apoptosis controlling system in children with nephrotic syndrome and diabetic nephropathy.
Methods: The study involved 34 children with an active stage of nephrotic syndrome and 23 children with primary diagnosed Diabetes Mellitus type I (T1D) and diabetic nephropathy. The affinity of hemoglobin to oxygen determined by spectrophotometric method. The levels of the marker of cellular hypoxia HIF-1alfa, levels of the apoptotic factor caspase-3 were studied using Western Blotting technique. Immunohistochemical examination of pro-apoptotic factor Bax was done. Comparison of the level of these parameters between the different segments of nephron at different stages of glomerulosclerosis in patients with nephrotic syndrome has been performed.
Results: Measurement of the pro-apoptotic factor Bax in kidney slices obtained from children with morphological form of nephrotic syndrome focal segmental glomerulosclerosis (FSGS) showed the presence of high level of Bax in both glomerular and tubule-interstitial segments. Higher immunosignal of Bax was evaluated in glomeruli with FSGS I-II st. as compared to tubular segment. When FSGS III-IV st. observed higher expression of Bax was detected in surrounding tubule-interstitial segment. Activation of the pro-apoptotic factor Bax occurred in parallel with the gradual increase of the HIF-1alfa depending on the level of FSGS in all nephrotic patients.
In the group of children with newly diagnosed T1D an increased rate of dissociation of oxygen and hemoglobin compared to the control group was detected. Children with DN the levels of this index were significantly lower than the control group. We show stage-dependent manner of the cellular hypoxia and apoptotic effector caspase-3 levels increase. Both markers detected at significantly higher rate in DN patients as compared to T1D.
Conclusion: Thus, the development kidney damage in children with DN is associated with violation of Hb/oxygen dissociation, a sign of the high degree of the Hb glycosylation resulted in formation of cellular hypoxia and activation of apoptosis. The same manner of damages occurred in children with primary proteinuric kidney disease (nephrotic syndrome).

Keywords: None