Early Endothelial disorders in children with diabetic nephropathy

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Background: Vascular endothelial cells play a major role in maintaining cardiovascular homeostasis. In diabetes mellitus type I (T1D) progression of cardio-renal disorders, i.e. arterial hypertension and its complications, diabetic nephropathy (DN), is still the most important side-effect. There are data about the role of Vitamin D in T1D and it's complications in adults. However, this issue remains to be open in pediatric practice.

Aim of the study: To study the levels of Vitamin D, Endothelin-1 in children with T1D and DN and to find out the network of these markers inter-relation.

Material and methods: 36 children T1D aged 6 to 17 years hospitalized in Endocrinology unit in Children Clinical Hospital №6 (Kyiv, Ukraine) studied. Vitamin D3 levels measured using ELISA assay and commercially available kit (Vitamin D3 (human) ELISA kit (BioVision, USA). Endothelin-1 levels measured using ELISA assay and commercially available Endothelin-1 ELISA kit (Abcam, USA). Results processed using STATISTICA 6.0 and non-parametric statistical method (Mann-Whitney test).

Results: In our study normal level, insufficiency and deficiency of the Vitamin D defined as - ≥ 30 ng/mL, 21-29 ng/mL and ≤ 20 ng/mL, respectively. All patients included into the study during the period September-May. We show that the most prominent Vitamin D3 deficiency detected in the group of patients with diabetic nephropathy (DN). In control group Vitamin D3 was detected at level 35.68 ± 1.56 ng/mL, in patients with T1D – 32.37 ± 5.1 ng/mL, in patients with DN – 19.39 ± 1.76 ng/mL (p<0.01 as compared to control group). Analysis of the Vitamin D3 levels and the disease course show negative correlation (R=-0,79, p<0,001).In all children with T1D and DN increased level of ET-1 measured.

Conclusion: Our data show the prominent deficiency of Vitamin D in T1D patients and patients with DN, increased ET-1 level. We hypothesize that Vitamin D deficiency is a result of toxic effect of glucose. Increased ET-1 in all patients is a sign of early microvascular changes and resistant vessels damage leading to DN progression and arterial hypertension. All mentioned above changes accompanied by reduced O2-Hb dissociation as a result of increased level of HbA1C and may be a reason of cellular hypoxia.

Biography

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