THE COMPARISON OF THE OCCURRENCE OF BETA CELLS AUTOANTIBODY AND NATURAL KILLER CELLS IN PATIENTS WITH TYPE 1 DIABETES MELLITUS, THEIR SIBLINGS AND HEALTHY CHILDREN.

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Background

Natural killer cells are a type of cytotoxic lymphocyte critical to the innate immune system. NK cells from patients with type 1 diabetes (DM1) have numeric and functional abnormalities. However, little is known about the number of NK cells in healthy siblings of children with DM1. The aim of the study is to compare the population of NK cells and the correlation between NK cells and beta cells autoantibody in healthy siblings of children with DM1 to healthy children from non-diabetic families and to children with DM1.

Methods

Peripheral blood mononuclear blood cells were obtained from 76 children with DM1, their siblings - 101, and 30 healthy children. NK cells were characterized by flow cytometry FACSCalibur (Becton Dickinson, USA). The auto-antibodies were determined by ELISA. The results were analyzed with STATISTICA 10 PL.

Results

The lowest percentage of NK cells was observed in diabetic patients (average percentage 10,59±5,37) and was lower than that in the control group (14,89±7,78) (p=0,002). The number of NK from the siblings was similar to patients with diabetes mellitus type 1, there was no significant difference in the number of NK cells between children with DM1 and their siblings (p=0,11). NK cells from siblings was lower (average percentage 11,93±5,62) than that in the control group (p=0,02).

The levels of anti IA2 and anti ZnT8 antibodies were statistically significant higher in siblings in comparison to the control group (anti IA2 Ab p=0,0000001; anti ZnT8 Ab p=0,00001). The level of anti-GAD in siblings was similar to that in the control group. There was a positive correlation between the reduced number of NK cells and the co-occurrence of anti-GAD and anti ZnT8 Ab (the May-Whitney test Z=-2,02; p=0,04) in the diabetic patients.

Conclusion

The results suggest that the dysfunction of NK cells may contributes to the autoimmune pathogenesis of type 1 diabetes and is connected with genetic predisposition to DM1.