

Comparison of the occurrence of islet autoantibodies in siblings of patients with type 1 diabetes mellitus to healthy children

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OBJECTIVES

Complex interactions between environmental and genetic factors contribute to the development of type 1 diabetes mellitus (T1DM) in genetically predisposed patients. Islet autoantibodies are markers that appear when insulin producing beta cells in pancreas are damaged. They can be detected a long time before beta cells are completely destroyed.

The aim of the study is comparison of the occurrence of islet autoantibodies in healthy siblings of children with T1DM to healthy children from non-diabetic families.

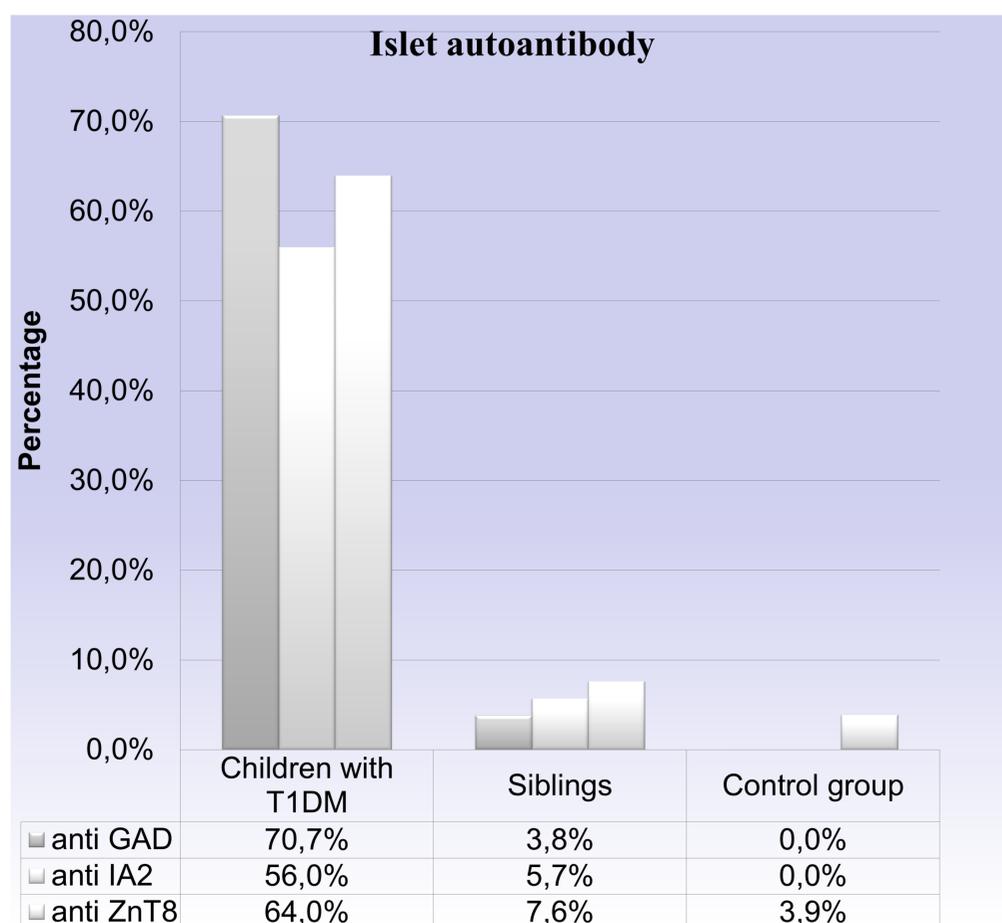
METHODS

Determination of anti-decarboxylase of the glutamic acid antibodies (anti GAD- Ab), anti- protein tyrosine phosphatase (anti IA2), and antibodies against zinc transporter 8 (anti ZnT8) in 75 children with T1DM, their siblings - 105, and 77 healthy children. All antibodies were determined by ELISA. The results were analyzed with the May-Whitney Wilcoxon test.

RESULTS

The highest level of anti-GAD (median 28,2 IU/ml), anti IA2 (median 26,19 IU/ml), and anti ZnT8 (median 94,73 IU/ml) was noted in children with T1DM and significantly lower levels were noticed in their siblings (median: anti GAD 2,61IU/ml, anti IA2 3,53 IU/ml, anti ZnT8 5,14 IU/ml). This level of anti-GAD was statistically significant higher in comparison to the control group of healthy children (anti-GAD 0,21 IU/ml). The levels of anti IA2 antibodies and anti ZnT8 Ab were similar to that in the control group.

In 2 children from the siblings, elevated levels of all antibodies were observed, in 2 siblings, elevation of anti -GAD and anti-ZnT8 Ab was noticed, and in 18 siblings only ZnT8 Ab was increased. Only in 3 healthy children an increase in Zn T8 Ab was observed and no increased anti-GAD or anti IA2 were observed in any healthy child.



CONCLUSIONS

The anti- GAD antibody is most characteristic in siblings of T1DM patients, especially in children with 2 or 3 autoantibodies predisposed to development of T1DM.

References

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The authors declare no conflict of interest.

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