INFLUENCE OF PANCREATIC AUTOIMMUNITY IN THE ONSET AND PROGRESSION OF DIABETES IN PEDIATRIC POPULATION

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Diabetes

Introduction:

Anti-islet autoantibodies are predictive and diagnostic markers for Type 1 Diabetes (T1D). The most frequently determined pancreatic autoantibodies in T1D are anti-glutamic acid decarboxilase (GAD), anti-tyrosine phosphatase (IA-2) and anti-insulin (AAI).

Objectives:

To study whether the pancreatic autoimmunity profile influences the initial presentation of diabetes, its metabolic behaviour and the presence of other autoimmune disorders in T1D.

Methods:

Retrospective study of 210 pediatric patients with T1D. We analyzed age, sex, age at diagnosis, type of clinical presentation: hyperglycemia, ketosis, ketoacidosis (KAD), HbA1c (HPLC-Menarini, NV 5.31 0.31%), C-peptide levels and pancreatic autoimmunity (GAD, IA2, AAI). Additional autoimmune disorders were screened with an antibody array at diagnosis and at follow-up. The metabolic control (last year mean HbA1c and acute complications) were also analyzed.
Data are reported in percentages, median and interquartile ranges. Statistical analysis was performed with SPSS 22.0.

Results:

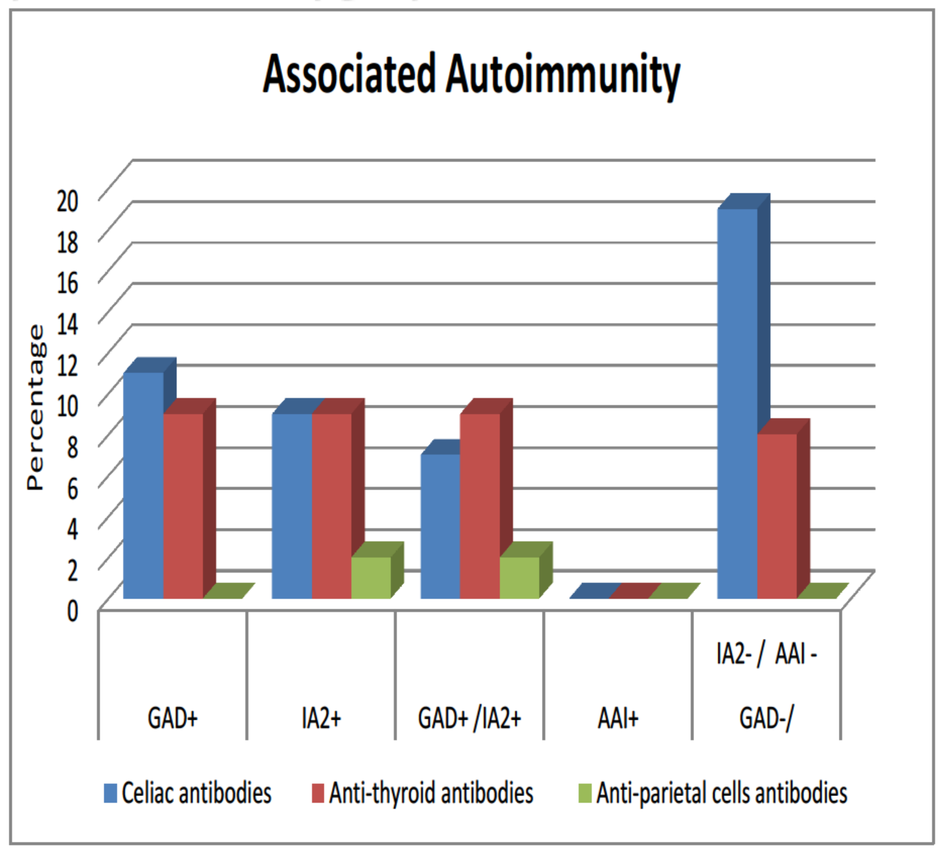
At diagnosis, mean age was 7 years (3.3-10.5), 53% female, Hb1Ac 10.7% (9.6-12.2), C-peptide 0.5 ng/m (0.3-0.7). Initial presentation: hyperglycemia 23%, ketosis 40%, ketoacidosis 37%. In our cohort 88% of patients have pancreatic autoimmunity markers. Associated autoimmunity at follow-up: anti-thyroid antibodies 9%, celiac disease 10%, parietal cells antibodies 2%. Celiac disease was diagnosed in five patients before T1D. GAD+ patients showed more rapid progression to celiac disease. Other autoimmunity markers: one patient had adrenal antibodies (GAD+/IA2) with normal adrenal function, 4% of the patients presented positive ANA, one of them with olygoarthritis (IA2+) and another with associated autoimmune thyroiditis (GAD+/IA2+). Another patient was diagnosed with autoimmune hepatitis 3.7 years after T1D (GAD+). Within the last year follow-up no patient presented episodes of severe hypoglycemia or ketoacidosis. No significant differences were found between patient-groups with isolated, combined or absence of pancreatic autoantibodies. Eleven of children with negative autoimmunity had haplotype analysis and ten of them were HLA of risk of T1D.

Table

	GAD+	IA2+	GAD+ /IA2+	AAI+	GAD-/IA2- / AAI -
n (%)	45 (21%)	45 (21%)	92 (44%)	2 (1%)	26 (12%)
Years at DM1 onset	5.0 (2.1-10.5)	6.8 (3.6-9.8)	8.3 (4.7-10.9)	7.0 (3.8-10.3)	4.1 (2.7-8.2)
Sex (F%)	44	62	52	50	54
DM1 debut (%) HG/Ketosis/KAD	29/38/33	20/32/48	29/38/33	50/0/50	19/58/23
HbA1c at DM1 debut (%)	10.6 (8.7-12.1)	10.7 (9.6-12.6)	10.7 (9.8-11.9)	9.9 (6.8-13.0)	11.0 (9.9-12.1)
Basal C-Peptide (ng/ml)	0.4 (0.3-0.5)	0.5 (0.3-0.6)	0.5 (0.3-0.7)	0.5 (0.4-0.7)	0.3 (0.1-0.6)
Follow up (years)	4.4 (2.6-8.3)	4.5 (2.2-7.8)	4.0 (2.1-6.6)	3.6 (2.6-4.6)	5.3 (3.0-9.2)
Mean HbA1c in the last year (%)	6.5 (6.2-7.2)	6.7 (6.2-7.1)	6.6 (6.3-7.0)	6.4 (6.3-6.5)	6.7 (6.3-7.1)

DM1 debut: HG: hyperglycemia. KAD: ketoacidosis

Graphic: Associated autoimmunity acccording to pancreatic antibody groups



Conclusions:

Pancreatic autoimmunity does not influence the type of disease onset nor evolution or frequency of associated autoimmune diseases.

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ESPE 2015 731--P3

Diabetes 1

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DOI: 10.3252/pso.eu.54espe.2015

Poster presented at: 54th Annual Meeting 1-3 October ESPE 2015 BARCELONA European Society for Paediatric Endocrinology

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