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Deep Characterization of Pancreas Volume of New-Onset Type 1 Diabetes Patients Reveals Puberty-Specific Patterns and New Topographic Correlations with Pancreatic Functions.

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There is a current understanding of type 1 diabetes (T1D) as being the result of a heterogeneous disease of whole pancreas. So far, descriptions of pancreas alterations suggested a global involvement of inflammation throughout the organ, characterized by abnormal age-specific patterns (i.e. > or <7 yo). In our DIATAG study, we thoroughly characterized pancreatic structure modifications in new-onset T1D patients and investigated whether these correlated with pancreatic functions during the first postdiagnosis year. Pediatric patients with new-onset T1D (n=31; 11.2±2.9 years) underwent abdominal MRI scan at diagnosis (i.e. 40±37 days postdiagnosis). Exocrine function (i.e. serum trypsinogen) and residual endocrine secretion estimates (i.e. fasten = CPEPBASAL, stimulated model = CPEPEST (1)) were evaluated on fasten blood tests at diagnosis (Δ) and Δ+3, +12 months, respectively. Clinical parameters of glucose homeostasis (i.e. A1C, insulin daily dose, insulin-dose adjusted A1c [IDAA1C]) were collected at Δ+3, +6, +9 and +12 months. We compared pancreas volumes (i.e. PV, PVHEAD, PVBODY, PVTAIL) and ratios (pancreas volume ratio [PVR] = PVHEAD/PVBODY +TAIL; pancreas index [PI] = PV/body weight) to retrospectively identify age-, BMI- and sex-matched controls (n=29). Compared to control group, T1D patients demonstrated a homogeneous (PVR, 1.39±0.7 vs 1.46±0.4, p>0.05) decrease of PI (0.6±0.2 vs 1.1±0.3 mL/kg,

p<0.0001) independently of pancreatic subregion. Interestingly, prepubertal children with T1D (n=15) exhibited greater pancreas atrophy of both the body-tail (10 ±5.6%, p<0.05) and head (7.2±3%, p<0.05) subregions compared to pubertal group (n=16; PI difference of mean, 0.64 vs 0.48 mL/kg). Next, we evaluated correlations between pancreatic volumes and functions during the natural evolution of T1D using multivariate and univariate linear regressions. PV and PI correlated moderately with exocrine function (trypsinogen; R=0.46, R=0.52, p<0.001) but not with residual endocrine function evaluated at Δ+3 months. Interestingly, we observed using topographical analysis that PVHEAD correlated with trypsinogen (R=0.5, p<0.001) but not with any endocrine estimates (p>0.05). PVTAIL positively correlated with CPEPEST and CPEPBASAL at Δ+3 months (R=0.55, p=0.002; R=0.47, p<0.01) and not with clinical parameters or trypsinogen (p>0.05). PVBODY did not correlate with any pancreatic function. Finally, we identified multivariate models that included PV and predicted pancreatic endocrine function at Δ+6 and +12 months (i.e. IDAA1C, HbA1C). MRI performed early after T1D onset helped us define specific pancreas alterations and demonstrate for the first time: (1) the increased pancreas atrophy in prepubertal children; (2) correlations between pancreatic subregion volumes and their respective residual functions early after diagnosis and (3) the improvement of endocrine function prediction models by adding PV estimates as predictors.

(1) Wentworth et al. 2019;62(1): 33–40

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