COMMENTS AND RESPONSES

Increased Adiposity at Diagnosis in Younger Children With Type 1 Diabetes Does Not Persist

Response to Clarke et al.

The report by Clarke et al. (1) showed that in pediatric type 1 diabetes, younger age at diagnosis was associated with higher BMI SD scores (SDSs); however, BMI SDSs did not change from 1976 to 2004. In patients longitudinally evaluated and grouped according to age at diagnosis, BMI SDSs were higher in the youngest age-group, while, 5 years later, anthropometric parameters were similar across the three groups.

We report our results regarding BMI SDSs of 174 patients, 106 (60.9%) male and 68 (39.1%) female, insulin treated since diagnosis and followed between 1990 and 2005. BMI, recorded at 2–12 months after clinical diagnosis (2), was converted to SDS according to Rolland Cacherà standards (3). Age at diagnosis ranged from 1 to 15.7 years (mean ± SD 8.1 ± 3.9). Patients were divided into three groups according to age at diagnosis: *1*) 1–4.99 years (n = 49, 28.2%), *2*) 5–9.99 years (n = 63, 36.2%), and *3*) 10–15.7 years (n = 62, 35.6%). In our 174 patients, BMI SDS was not different among these three groups of patients (P = 0.61, F-test ANOVA), and it did not change over the period 1990–2005 (Pearson's correlation coefficient r = 0.052, P = 0.50). There was no significant interaction between age category at diagnosis and category of year of diagnosis (P = 0.75); there was no correlation between age and year at diagnosis (r = 0.09, P = 0.23). Anthropometric data of 92 patients were available after a 5-year follow-up. In 92 longitudinally evaluated patients, BMI SDSs at diagnosis were not higher in group 1 than in groups 2 and 3 (P = 0.75, ANOVA). At 5 years after diagnosis, BMI SDSs were similar across the three age groups (P = 0.46, ANOVA). BMI SDSs increased from diagnosis to 5 years (effect of time: P = 0.001, repeated-measures ANOVA). Our observations do not confirm that obesity is a common finding in younger children at type 1 diabetes diagnosis and do not support the accelerator hypothesis (4), which argues that obesity is an environmental factor that hastens the clinical onset of the disease (5). Obesity-induced insulin resistance may upregulate β-cells, which become more susceptible to autoimmune damage in genetically predisposed individuals (6). In regard to BMI as a risk factor for diabetes, it should be established whether patients are more insulin resistant than healthy subjects and whether greater BMI during childhood could explain insulin resistance (4). BMI could be too crude an indicator of insulin resistance (7). There might be a threshold at which obesity determines earlier onset of type 1 diabetes that was not reached in our Italian patients. The increased BMI SDSs after diagnosis could be related to intensive insulin therapy (8) and represents a risk factor for the late development of microvascular complications (9).

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