



Turner Syndrome: does GH treatment influence glucose homeostasis ?

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Background: Growth hormone (GH) has been shown to reduce insulin sensitivity in Turner girls, however a compensatory increase of insulin secretion by pancreatic beta cells usually occurs, probably stimulated by GH itself. Oral disposition index (ODI) express the capacity of beta cells to adapt to insulin sensitivity.

Objective: to study longitudinally the insulin sensitivity (HOMA-S), the insulin secretion (IGI) and the ODI in a group of girls affected by Turner syndrome (TS) treated with GH.

Methods: we studied 104 GH treated (0.33 mg/kg over 7 days) TS girls (first evaluation at 9.1 ± 3.4 years) for a median period of 7.2 years (range 2.04-13).

Puberty started spontaneously in 46/104 (44%) girls at a mean chronological age of 13.2±2.3 years, however, 41 girls required estrogen treatment to complete pubertal development. In the other 58/104 (56%) estrogens were started at a median age of 15.7 years.

Every year the children underwent an OGTT which was employed to calculate the

HOMA-S ($1/((\text{insulin} \times \text{glucose})/22.5)$),

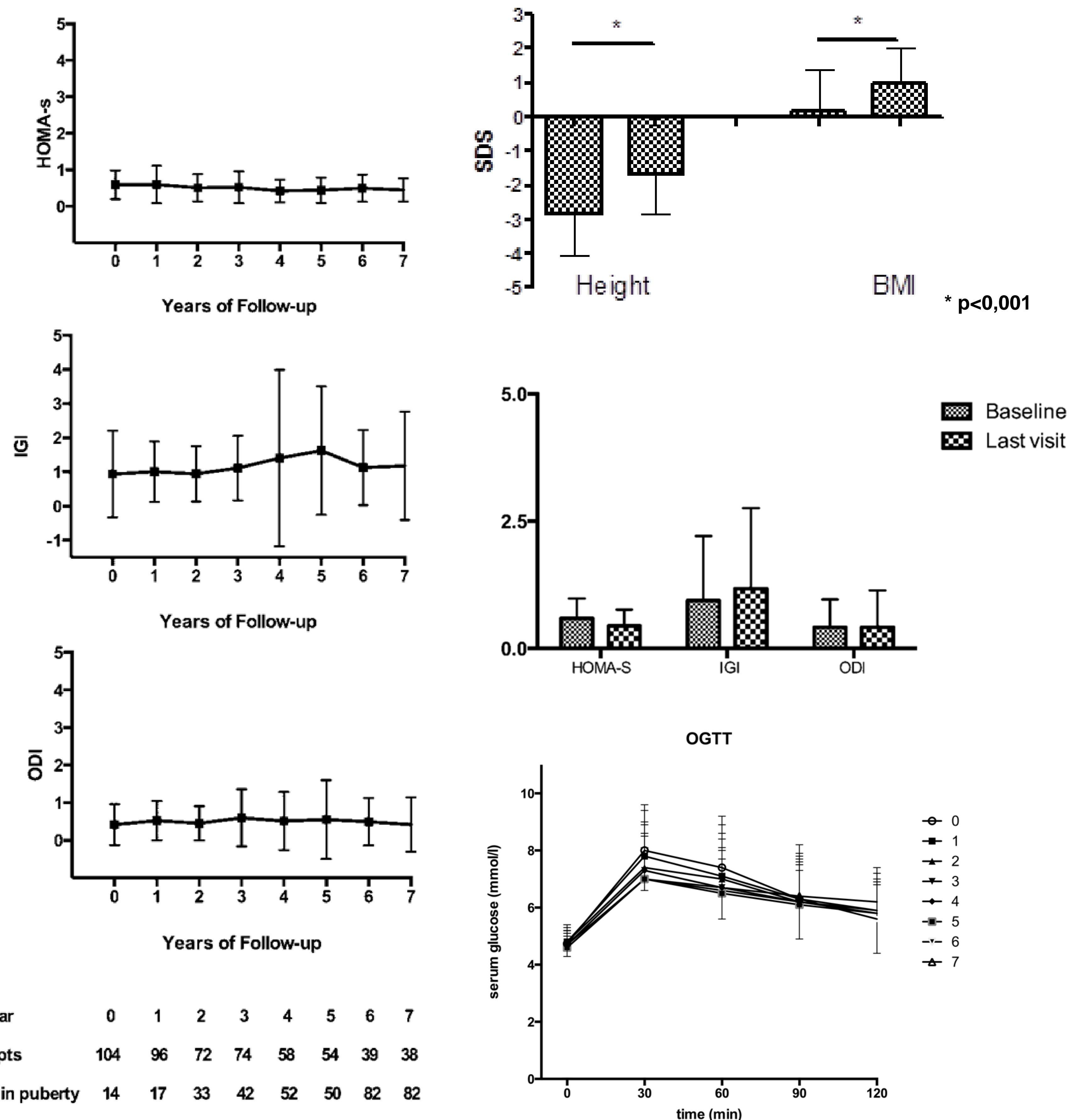
IGI (insulinogenic index) ($\Delta I30/\Delta G30$) and the

ODI (disposition index= $\text{HOMA-S} \times \text{IGI}$).

Results: GH treatment induced a significant increase in height SDS ($p < 0.001$) between the first and the last visit, as BMI SDS ($p < 0.001$).

No significant changes over the years were observed in term of HOMA-S, IGI, or ODI. IGF-I serum level, after 7 years treatment, was 475 ± 203 ng/ml (range 343 to 580 ng/ml) in normal range for age.

No cases of diabetes were found



Conclusion: this study, while further confirming the safety of GH treatment in TS girls, suggests that it is unnecessary to check annually the glucose tolerance in those girls, reserving glucose homeostasis control in selected patients (i.e obese TS). This approach would lead to a significant reduction of the expenses without lowering the quality of care in our patients.