LETTER TO THE EDITOR

Outcome after depot gonadotrophin-releasing hormone agonist treatment for central precocious puberty: effects on body mass index and final height

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In their interesting paper, Arrigo et al. (1) investigated longitudinally body mass index (BMI) evolution and prevalence of obesity in girls with idiopathic central precocious puberty (CPP) who were treated with luteinizing hormone-releasing hormone (LHRH) analogs (LHRHa) for at least 2 years. They demonstrated that suppression of the pituitary–gonadal axis function was accompanied by a significant decrease in being overweight. They observed that girls with CPP are frequently obese at diagnosis, suggesting that the pretreatment increase in BMI is due to pubertal hormonal changes and secondary changes in body fat, rather than being a result of CPP (1).

We have retrospectively reviewed 29 female patients with idiopathic CPP. The main criteria for diagnosis of idiopathic CPP were (1) onset of breast development and/or menses before 8 years of age, (2) pubertal luteinizing hormone (LH) response to exogenous LHRH (LH peak of > 10 IU/l), (3) no evidence of hypothalamo–pituitary lesions at magnetic resonance imaging and (4) no additional conditions that might affect BMI. Predicted height was calculated using a computerized Bylley–Pinneau method (2). We assessed height SDS, BMI and BMI SDS (3) before, 1 year after the beginning of therapy, at the end of therapy and 2.5 years after the end of therapy. A group of 45 healthy girls with normal onset of puberty was matched for age or for end of therapy. A group of 45 healthy girls with CPP, we found an increasing trend in BMI after the first year of therapy, and this did not change after therapy was stopped. With regard to BMI SDS, few changes were observed during the first year of therapy, while an increasing trend was observed at the end of therapy and a complete recovery after 2.5 years of the end of therapy. Nevertheless, in both girls with CPP and the control group, the mean BMI increased; in fact the value in the patients was as much as in the control group during therapy and at the end of therapy. Mean BMI SDS, different in the two groups, was significantly higher in the patients at the end of therapy than in controls, suggesting that the difference was due to the treatment. The data available in the literature on the effects of LHRHa on hypothalamic and/or leptin-mediated control of body weight (4). Some authors have highlighted the fact that obesity is frequent among children with CPP, but does not appear to be related to the long-term pituitary–gonadal suppression induced by LHRHa therapy (5–7). On the other hand, Feuillan et al. (8) showed the association of both CPP and LHRHa with obesity in patients who had increased BMI at initial presentation and during therapy: this condition persisted after discontinuation of therapy and progresses to frank obesity (8).

References
Table 1 Auxological findings and statistical analyses in girls with CPP (n = 29) and controls (n = 45) at diagnosis, after 1 year of therapy, at the end of therapy and 2.5 years after the end of therapy.

<table>
<thead>
<tr>
<th>Time</th>
<th>Mean chronological age (range)</th>
<th>Mean height SDS (range)</th>
<th>Mean BMI (range)</th>
<th>Mean BMI SDS (range)</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>CPP</td>
<td>Controls</td>
<td>CPP</td>
<td>Controls</td>
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<tr>
<td>Diagnosis</td>
<td>7.9 (6.6–8.6)</td>
<td>9.2 (6.5–10.5)</td>
<td>1.3 (0.5–4.4)</td>
<td>0.66 (1.35–3.97)</td>
</tr>
<tr>
<td>After 1 year</td>
<td>8.8 (7.6–9.6)</td>
<td>9.4 (8.2–11.6)</td>
<td>1.4 (0.9–4.4)</td>
<td>–1.46/3.97</td>
</tr>
<tr>
<td>End</td>
<td>11 (9.6–11.7)</td>
<td>11.3 (10.3–13)</td>
<td>1.2 (2/4)</td>
<td>–1.46/3.97</td>
</tr>
<tr>
<td>After 2.5 years</td>
<td>13.5 (12.1–14.2)</td>
<td>14.3 (11.5–17.1)</td>
<td>0.4 (2.3/4)</td>
<td>–1.76/1.84</td>
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</tbody>
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