Influence of birth parameters on growth response and metabolic effects of growth hormone (GH) therapy in GH-deficient children and adolescents

INTRODUCTION

Growth depends on growth hormone (GH) secretion and on individual sensitivity to its action. The effects of birth parameters on growth and metabolic status are well documented in small-for-gestational-age (SGA) children, but in children with GH deficiency those associations are not clear. Taking into account that GH-deficient children are not a homogenic group of patients, the importance of an individual approach to GH doses and the assessment of the effects of GH therapy is emphasised.

THE AIM OF THE STUDY

We investigated the associations between birth weight (BW) and length (BL) and gestational age (GA) and anthropometric and biochemical parameters in GH-deficient children before and in the first year of GH therapy.

<table>
<thead>
<tr>
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<th>Baseline</th>
<th>6 months</th>
<th>12 months</th>
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</thead>
<tbody>
<tr>
<td>Adiponectin (ng/ml)</td>
<td>18.658.2 ± 10.211.9</td>
<td>21.480.5 ± 8.461.9</td>
<td>16.783.8 ± 7.292.7</td>
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<tr>
<td>Resistin (ng/ml)</td>
<td>3.8 ± 1.3</td>
<td>3.8 ± 1.1</td>
<td>3.6 ± 0.8</td>
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<tr>
<td>Fasting glucose (mg/dl)</td>
<td>82.8 ± 8.8</td>
<td>85.4 ± 10.1</td>
<td>85.4 ± 7.9</td>
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<tr>
<td>Fasting insulin (µIU/ml)</td>
<td>3.7 ± 3.4</td>
<td>6.6 ± 4.9</td>
<td>8.5 ± 6.1</td>
</tr>
<tr>
<td>IGF-1 (SDS)</td>
<td>-0.4 ± 1.0</td>
<td>1.6 ± 2.4</td>
<td>0.9 ± 1.2</td>
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</tbody>
</table>

Table 1

RESULTS

1) BW correlated with baseline height SDS in prepubertal patients (R = 0.38, p = 0.029) and with height SDS after 12 months of GH therapy (R = 0.73, p = 0.016) in pubertal patients.
2) GA was associated with both weight SDS and BMI SDS at baseline and after 6 and 12 months of GH therapy in prepubertal patients.
3) In prepubertal children GA was associated with adiponectin (R = 0.39, p = 0.029) and fasting glucose (R = -0.45, p = 0.008) at baseline and with resistin (R = -0.49, p = 0.015), IGF-1 SDS (R = 0.44, p = 0.009) and with increase in IGF-1 SDS (R = 0.47, p = 0.018) after the first 6 months of GH therapy.
4) In pubertal patients we only found that baseline resistin was adversely associated with GA and BL (R = -0.64, p = 0.035; R = -0.65, p = 0.031, respectively).
5) No correlations with insulin, HOMA-IR, QUICKI and lipid profile were found.

CONCLUSIONS

BW and GA seemed to be important factors affecting height deficit and nutritional status in GH-deficient children, especially before puberty. Higher GA is associated with better IGF-1 response to GH therapy, lower resistin and glucose levels at baseline and during GH therapy.

REFERENCES


The authors declare no conflicts of interest