Unexplained Weight Loss in Two Growth Hormone Deficient Adolescent Males

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Patient Demographics
Patient A: 15 ½ year old Caucasian male

Clinical Presentation/Diagnosis
Patient A has been followed in the pediatric endocrine clinic since age 18 months. He was diagnosed with growth hormone (GH) and thyroid deficiencies. GH was discontinued at age 15 years and 1 month due to growth completion. He had an appendectomy 1 month prior to his routine endocrine follow-up visit, and complains of diminished energy level and a 15-lb weight loss despite adequate oral intake and absence of gastrointestinal symptoms. No acute illness has been noted by his pediatrician.

Past History
Patient A has the following history:
• Severe growth retardation as an infant and toddler
• Diagnosis of GH deficiency at age 18 months
• Treatment with growth hormone therapy at 0.3mg/kg/wk
• Diagnosis of hypothyroidism in the toddler period
• Maintained on levothyroxine 25 mcg from the time of diagnosis until discontinuation of GH

Evaluation
The following tests have been completed to evaluate patient A:

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>IGF-1</td>
<td>70 (201-609 ng/mL)</td>
</tr>
<tr>
<td>Thyroid function studies (off levothyroxine)</td>
<td>Normal</td>
</tr>
<tr>
<td>Fasting Glucose</td>
<td>80 (56-145 mcg/dL)</td>
</tr>
<tr>
<td>Fasting Cortisol</td>
<td>21 (6.0-23.0 mcg/dL)</td>
</tr>
<tr>
<td>GHmax on insulin tolerance test</td>
<td>0.9 ng/dL</td>
</tr>
<tr>
<td>MRI</td>
<td>Empty sella</td>
</tr>
<tr>
<td>Bone age</td>
<td>16y6m @ 15y1m</td>
</tr>
</tbody>
</table>

Interventions
Patient A was evaluated for adult growth hormone deficiency, with IGF-1 and insulin tolerance test. Growth hormone was restarted at a transition dose of 0.03 mg/kg/day. Return evaluation following two months of growth hormone therapy revealed an 18-lb weight gain, and resumption of his normal energy levels. Levothyroxine was not resumed due to normal thyroid function studies without medication.

References

Patient Demographics
Patient B: 17 year old Caucasian male

Clinical Presentation/Diagnosis
Patient B has been followed in the endocrine clinic since 7 years of age with growth hormone deficiency. GH was discontinued six months prior to his visit due to poor compliance. Bone age was 14 years at 15 years 5 months. He reported a 20-lb weight loss and diminished energy level. He had not had changes in his medical regimen. No other acute illnesses were present.

Past History
Patient B has a complicated medical history including:
• Fetal alcohol syndrome
• Failure to thrive
• Global developmental delay
• Attention deficit hyperactivity disorder
• Gastroesophageal reflux disease
• Eosinophilic esophagitis
• Nissen fundoplication
• Growth hormone deficiency diagnosed at age 7 years

Evaluation
The following tests have been completed to evaluate patient B:

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>IGF-1</td>
<td>129 (209-602 ng/mL)</td>
</tr>
<tr>
<td>Thyroid function studies</td>
<td>Normal</td>
</tr>
<tr>
<td>Fasting Cortisol</td>
<td>21.5 (4.2-38.4) mcg/dL</td>
</tr>
<tr>
<td>GHmax on Insulin Tolerance Test</td>
<td>1.5 ng/mL</td>
</tr>
<tr>
<td>Repeat MRI</td>
<td>Normal</td>
</tr>
</tbody>
</table>

Interventions
Patient B was evaluated for adult growth hormone deficiency, with IGF-1 and insulin tolerance test. He was restarted on GH at a dose of 0.01 mg/kg/day. He has not seen the endocrinologist for follow-up visit since restarting GH therapy; however, a visit with the gastroenterologist 3 months after GH restart revealed a 19-lb weight gain. Phone conversation with the patient’s parent revealed an improvement in his energy level.

Discussion
The usual presentation for adult GH deficiency is an increase in fat mass, and decrease in lean muscle mass, accompanied by weight gain, diminished energy levels, and decreased quality of life(1). The use of GH increases lipolysis, with a decrease in visceral fat with GH treatment(1). Both of these cases are not reflective of the usual presentation for adult GH deficiency. No reports of this phenomenon were found in the literature.