Post Gastric Bypass Hypoglycemia: Four Cases Treated Successfully with Alphaglucosidase Inhibitor Therapy

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Introduction
- Roux-en-y gastric bypass (GB) surgery has been increasingly employed to treat the morbidities of obesity
- In 2005, Service et al (1) reported hyperinsulinemic hypoglycemia in 6 GB patients who were found to have anatomic changes in islet cells
- Since that time, postprandial hypoglycemia has become increasingly recognized as a late complication of GB
- Carbohydrate (CHO) restriction is recommended as initial therapy but is not always effective in reducing hypoglycemia
- Alphaglucosidase inhibitor (AGI) therapy has been suggested as a second line therapy
- We report the first collection of GB hypoglycemic patients who were successfully treated with long-term AGI therapy

Case 1
- 42 F who underwent GB 3 years prior presented for evaluation of hypoglycemia
- Patient met criteria for Whipple’s Triad:
  - Experienced sweating, palpitations, tremor and neuroglycopenic symptoms 2-4 hours postprandial
  - Documented capillary glucose (BG) of 35-50 mg/dl
  - Symptoms resolved with small CHO meals
- Baseline testing ruled out adrenal, thyroid, renal and hepatic dysfunction
- Mixed 75g meal endocrine testing revealed:
  - Fast bg 63 mg/dl
  - 115 minute symptomatic BG of 42 mg/dl with simultaneous serum insulin 14 uU/mL (<29.2 uU/mL)
  - C-peptide 4.2 ng/ml (0.8-9.6 ng/ml)
  - Beta hydroxybutyric acid of 0.08 mmol/L (0.0-0.42 mmol/L)
- Sulfonylurea screen negative
- CT negative for pancreatic mass
- Because her hypoglycemia was exclusively postprandial, empiric therapy was instituted rather than pursuing invasive testing to rule out insulinoma
- She started a 30-gm per meal CHO restricted diet and initially responded
- However increasing CHO intake resulted in symptomatic hypoglycemia
- Pre-meal AGI therapy of 50mg TID was initiated with meals
- At 3-year follow-up patient denies symptomatic postprandial hypoglycemia except when she omits AGI therapy

Discussion
- Most recent review of symptomatic hypoglycemia in GB patients has described an incidence between 0.2-6% (2)
- To date 89 cases have been reported (Table 2)

Table 1
<table>
<thead>
<tr>
<th>Case 1</th>
<th>Case 2</th>
<th>Case 3</th>
<th>Case 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>PPG &amp; BG nadir (55-90 mg/dL)</td>
<td>14.2</td>
<td>32</td>
<td>56</td>
</tr>
<tr>
<td>Insulin (&lt;29.2 uU/mL)</td>
<td>14.2</td>
<td>32</td>
<td>56</td>
</tr>
<tr>
<td>C-peptide ng/ml</td>
<td>6.2</td>
<td>2.8</td>
<td>10.8</td>
</tr>
<tr>
<td>CT Abdomen</td>
<td>Negative</td>
<td>Negative</td>
<td>Positive</td>
</tr>
</tbody>
</table>

**Conclusions**

- We are the first to demonstrate the long-term effectiveness of AGI therapy in GB patients with postprandial hypoglycemia who fail dietary carbohydrate restriction

References:

Table 2

<table>
<thead>
<tr>
<th>Case</th>
<th>Mean age (years)</th>
<th>Time of Onset after GB* (months)</th>
<th>Lowest Glucose Value (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Current Series</td>
<td>61</td>
<td>42</td>
<td>(mixed meal)</td>
</tr>
</tbody>
</table>

**Discussions (cont.)**

- Glucagon like peptide-1 levels were found to be significantly increased in GB patients with symptomatic hypoglycemia compared to asymptomatic GB patients (4)
- CHO restriction is the first step instituted to treat symptomatic GB hypoglycemia
- Kellogg et al (5) described 10 out of 12 GB patients had complete or partial improvement of symptomatic hypoglycemia with low CHO diet
- Acarbose has been shown to decrease postprandial hyperglycemia after mixed meal testing as well as attenuate the rise in insulin and GLP-1 levels (6)
- Kellogg et al (5) placed 2 patients on AGI therapy after failing low CHO diet therapy with 1 experiencing improvement
- Moreira et al (7) added AGI to verapamil therapy to a single patient with resolution of hypoglycemia
- Haruna et al (8) reported the use of AGI therapy in a single patient decreased hypoglycemia after an 8 month follow-up

**Conclusions**

- The etiology of GB hypoglycemia remains unclear
- Symptoms from dumping syndrome can mimic post-prandial hypoglycemia
- However, dumping syndrome occurs weeks to months after surgery versus 1-2 year delay in GB hypoglycemia patients
- Some patients were found to have changes in islet cells consistent with noninsulinomas pancreaticoblastoma (NIPS) (1)
- However, Meier et al (3) did not find islet cell hyperplasia or hyperplasia
- Attention has focused on exaggerated secretion of incretins after meal ingestion

**References:**