An interesting case of postprandial hypoglycaemia

Ishwar Bhattarai, Cheuk Mun Au

CASE
A woman aged 55 years presented to her general practitioner with intermittent episodes of dizziness, sweating and shakiness for the past four months. These episodes lasted for a few minutes and resolved spontaneously or after lying down. Lately, these episodes had become more common, especially after sugary meals. She had no chest pain, breathlessness or loss of consciousness during these episodes. Her medical history included anxiety, asthma, type 2 diabetes and obesity, for which she had undergone gastric bypass surgery three years ago. She was a non-smoker who drank alcohol socially. Her only medication was escitalopram 20 mg daily. Her clinical examination was unremarkable and vital signs were normal. Her body mass index was 32 kg/m².

QUESTION 1
What are the provisional and differential diagnoses?

ANSWER 1
The provisional diagnosis is dumping syndrome. Other differentials are:
• postprandial hypotension
• pheochromocytoma
• insulinoma
• carcinoid syndrome
• functional neurological disorder.

QUESTION 2
What is dumping syndrome and what are its causes?

ANSWER 2
Dumping syndrome is a cluster of gastrointestinal and vasomotor symptoms due to rapid gastric emptying after a meal. Dumping syndrome is classified as early and late type. Early dumping syndrome presents within 30 minutes of a meal, and symptoms include palpitations, fatigue, sweating, lightheadedness, hypotension, abdominal fullness, nausea and diarrhoea. Late dumping syndrome occurs 1–3 hours after a meal and is an incretin-driven hyperinsulinaemic response.¹ This leads to reactive hypoglycaemia, which presents with adrenergic and neuroglycopenic symptoms such as tremor, sweating, anxiety, fatigue, hunger and confusion.¹

Dumping syndrome is common after bariatric surgeries such as gastric bypass or sleeve gastrectomy.² There are cases of dumping syndrome associated with non-surgical causes such as diabetes mellitus.³,⁴

QUESTION 3
What is the pathophysiology of dumping syndrome?

ANSWER 3
Dumping syndrome is typically due to rapid transit of gastric chyme into the intestinal lumen. This leads to intestinal distension and release of many gastric hormones. These hormones decrease splanchnic vascular resistance, shifting fluids from general circulation to gastrointestinal (GI) circulation,⁵ which leads to vasomotor symptoms such as hypotension, fatigue, dyspnoea and faintness. Sympathetic activation then causes palpitations and sweating.

In late dumping syndrome, rapid transit of gastric chyme causes the release of other GI hormones such as gastric inhibitory peptide and glucagon-like peptide.⁵,⁶ These hormones stimulate the release of insulin from the pancreas, leading to reactive hypoglycaemia manifested as sweating, palpitations, light-headedness and fainting.

QUESTION 4
How is dumping syndrome diagnosed?

ANSWER 4
A classic presentation with a history of gastric surgery is highly suggestive of dumping syndrome. There is a symptoms-based scoring system available to diagnose dumping syndrome (Table 1).⁶ An oral glucose tolerance test (OGTT) is typically confirmatory; however, there is risk of severe hypoglycaemia, so it is advisable that the test is conducted under clinical supervision. A haematocrit rise of 3% or an increase in pulse rate >10 beats/min after 30 minutes is suggestive of early dumping syndrome, while hypoglycaemia after 2–3 hours of glucose ingestion suggests late dumping syndrome.⁷

A gastric emptying test using radionuclide scintigraphy can demonstrate rapid gastric emptying quantitatively.⁸

CASE CONTINUED
The patient underwent an OGGT; the results are shown in Table 2. Her thyroid, kidney and liver functions and electrolytes were within normal limits. She was diagnosed with late dumping syndrome.

QUESTION 5
What is the treatment for dumping syndrome?

In late dumping syndrome, rapid transit of gastric chyme causes the release of other GI hormones such as gastric inhibitory peptide and glucagon-like peptide.⁵,⁶ These hormones stimulate the release of insulin from the pancreas, leading to reactive hypoglycaemia manifested as sweating, palpitations, light-headedness and fainting.
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Lifestyle and dietary modifications are the first-line treatment for dumping syndrome. Smaller and more frequent meals of low-calorie dense food help to control the symptoms. A high-fibre diet of unprocessed carbohydrates and low glycaemic index food can stabilise blood glucose fluctuation as seen in late dumping syndrome. Other dietary modifications include avoiding liquids within 30 minutes of solid food meals, avoiding simple sugars (juices/lollies) and processed calorie-dense foods (donuts/chocolate), and increasing intake of protein.

Pharmacological interventions are reserved for severe cases uncontrolled by lifestyle and dietary modifications. Currently available options are alpha-glucosidase inhibitors (eg acarbose) and somatostatin analogues (eg octreotide). Acarbose inhibits alpha-glucosidase in the small intestine and delays the intraluminal digestion of carbohydrates that leads to insulin surge and reactive hypoglycaemia. Octreotide improves dumping syndrome by multiple mechanisms: delaying gastric emptying, slowing small intestine transit and decreasing release of gastrointestinal hormones including insulin secretion.

### Table 1. Sigstad score: A score of ≥7 is highly suggestive of dumping syndrome

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Score</th>
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<tbody>
<tr>
<td>Pre-shock or shock</td>
<td>+5</td>
</tr>
<tr>
<td>Loss of consciousness, fainting</td>
<td>+4</td>
</tr>
<tr>
<td>Desire to lie down or sit</td>
<td>+4</td>
</tr>
<tr>
<td>Dyspnœa</td>
<td>+3</td>
</tr>
<tr>
<td>Physical fatigue, exhaustion</td>
<td>+3</td>
</tr>
<tr>
<td>Sleep, listlessness, blurred vision</td>
<td>+3</td>
</tr>
<tr>
<td>Palpitation</td>
<td>+3</td>
</tr>
<tr>
<td>Restlessness, agitation</td>
<td>+2</td>
</tr>
<tr>
<td>Dizziness, vertigo</td>
<td>+2</td>
</tr>
<tr>
<td>Headache</td>
<td>+1</td>
</tr>
<tr>
<td>Feeling hot, sweating, paleness, clammy skin</td>
<td>+1</td>
</tr>
<tr>
<td>Nausea</td>
<td>+1</td>
</tr>
<tr>
<td>Abdominal distension, meteorism</td>
<td>+1</td>
</tr>
<tr>
<td>Borborygm</td>
<td>+1</td>
</tr>
<tr>
<td>Eruption</td>
<td>-1</td>
</tr>
<tr>
<td>Vomiting</td>
<td>-4</td>
</tr>
</tbody>
</table>

### Table 2. Oral glucose tolerance test results

<table>
<thead>
<tr>
<th>Time</th>
<th>Glucose (mmol/L)</th>
<th>Reference range (mmol/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fasting</td>
<td>4.4</td>
<td>3.0–6.0</td>
</tr>
<tr>
<td>One hour</td>
<td>9.3</td>
<td>&lt;10</td>
</tr>
<tr>
<td>Two hours</td>
<td>2.0</td>
<td>Up to 7.7</td>
</tr>
</tbody>
</table>

### Key points
- Dumping syndrome is common after bariatric surgeries.
- Reactive hypoglycaemia is a typical feature of late dumping syndrome.
- An OGTT and gastric scintigraphy are diagnostic tests.
- Management is based on lifestyle and dietary modification, failure of which necessitates medication such as acarbose or octreotide.

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### References