Gastroparesis is a disorder in which the stomach takes too long to empty its contents. Also known as delayed stomach emptying, it is most often a complication associated with Type 1 diabetes, although it sometimes occurs in people with Type 2 diabetes. Up to 50 per cent of people with diabetes develop gastroparesis, though the vast majority will only experience it to a mild extent.

Gastroparesis occurs when the nerves to the stomach are damaged or stop working. The vagus nerve controls the movement of food through the digestive tract. If this is damaged, the muscles of the stomach and intestines do not work normally and the movement of food is slowed or stopped.

Diabetes can damage the vagus nerve if blood glucose levels (BGL) remain high over a long period of time. High blood glucose can cause chemical changes in nerves and can damage the blood vessels that carry oxygen and nutrients to the nerves.

### Symptoms

These include nausea, vomiting, an early feeling of fullness when eating, weight loss, abdominal bloating and abdominal discomfort. The symptoms of gastroparesis may be mild or severe and are usually worse after a meal.

If food lingers too long in the stomach, it can cause problems like bacterial overgrowth from the fermentation of food. Also, the food can harden into solid masses (called bezoars) that may cause nausea, vomiting, and obstruction in the stomach. These can be dangerous if they block the passage of food into the small intestine.

Gastroparesis can make diabetes worse by adding to the difficulty of controlling BGL. When food that has been delayed in the stomach finally enters the small intestine and is absorbed, BGL rise. Since gastroparesis makes stomach emptying unpredictable, a person's BGL can be erratic and difficult to control. Hypoglycaemia may be a particular problem due to a mismatch between insulin action and glucose absorption.

### Complications

- Bacterial overgrowth - treated with antibiotics.
- Bezoars ('hair balls') - these can induce nausea, vomiting and obstruction. They may be broken down and removed by the use of endoscopic tools or dissolved with injected treatment. Alternatively, they may be surgically removed.
- Erratic glycaemic control - it is harder to predict the timing of post-prandial blood glucose peak and therefore match insulin accordingly.
- Hypoglycaemia – insulin peaks before absorption of glucose.
- Hyperglycaemia – glucose is absorbed after peak insulin action or patients may reduce/omit insulin due to fear of hypos. Oral hypoglycaemic agents may not be absorbed effectively.
- Acute variability in glycaemia may exacerbate gastroparesis.

### Diagnosis

- If the patient has unexplained hyper and / or hypoglycaemia, an upper gastro-intestinal (GI) endoscopy should be considered to exclude gastric outlet or duodenal obstruction (Horowitz et al., 2002).
- Patients presenting with upper GI symptoms suggestive of delayed gastric emptying, should have a comprehensive history/examination performed, followed by appropriate investigation to identify other causes of upper GI symptoms (Horowitz et al., 2002).
- Diagnosis is based on the presence of upper GI symptoms in combination with objective evidence of delayed gastric emptying.
PATIENT SERVICES

DIABETES DROP-IN SESSIONS

DIABETES DROP-IN SESSIONS

Pharmacological Treatment (better tolerated in liquid form)

- Prokinetics are prescribed to accelerate gastric emptying and these form the mainstay of gastroparesis treatment. Prokinetics include Domperidone, a ‘first-line’ agent (Chang et al., 2011), Metoclopramide and Erythromycin.

Diagnostic Tests

- In general, diagnostic tests are not readily available or standardised between centres.

- Scintigraphy is regarded as the ‘gold standard’ diagnostic test but other investigations used to diagnose gastroparesis include the scintigraphic breath test, capsule telemetry, ultrasonography, magnetic resonance imaging and electrogastrography (Keld et al., 2011).

Non-Pharmacological Treatment

- Gastric electrical stimulation (GES) - this involves the implantation of electrodes in the gastric wall. These are connected to a subcutaneous pulse generator which triggers stomach contractions (NICE, 2004).

- Botox - this involves injecting the pylorus muscle which enlarges the outlet between the stomach and the intestine allowing the release of more food.

Glycaemic Control

- Due to delayed gastric emptying, quick-acting hypo treatment should be provided in a form that can be absorbed through the oral mucosa e.g. Glucogel or sucked boiled sweets.

- Recent evidence suggests that Continuous Subcutaneous Insulin Infusion (CSII) or insulin pump therapy can help to reduce glycaemic excursions, including the frequency of hypoglycaemic episodes, which in turn may improve the symptoms of gastroparesis (Sharma et al., 2011).

- Sharma et al. (2011) also found that CSII can reduce hospital admissions and the number of inpatient bed days, potentially being more cost effective in the longer-term, despite initial high costs to commence CSII.

Dietetic Management

- Dietary recommendations should focus on measures that promote gastric emptying or, at a minimum, do not retard emptying (DMUK, 2011).

- Dietary assessment should examine the time, content and size of meals eaten and the type and consistency of foods tolerated. Nutritional and fluid status should be assessed and the risk of nutritional deficiencies examined (Keld et al., 2011). A review of glycaemic control should also form part of the dietary assessment.

- Dietary advice may involve the avoidance of large volume meals and excessive intakes of dietary fat and fibre as these can impede gastric emptying (Keld et al., 2011). Alcoholic & carbonated drinks should also be discouraged as they may exacerbate symptoms (Chang et al., 2011; Brodie, 2007).

- Advice may be given regarding the consistency of foods eaten. In mild cases, adequately chewing food may be helpful, but in severe cases, a pureed/liquid diet may be required to reduce symptoms (Keld et al., 2001).

- A failure of dietary and drug therapies to maintain nutritional status and improve symptoms will warrant the use of artificial (post-pyloric) feeding. The naso-jejunal route is an appropriate feeding route in the short term, but if long-term feeding is necessary, a jejunostomy or gastrojejunostomy is recommended. Parenteral nutrition should be considered in a non-accessible / non-functioning gut with impaired nutrient absorption (Keld et al., 2011).

References


