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# Post-bariatric hypoglycaemia

CLINICAL REVIEW

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**Awareness of post-bariatric hypoglycaemia has increased in recent years. The condition appears to be more prevalent than previously estimated, when mild to moderate cases are taken into account. Most patients respond well to dietary**

**adjustments, while a small proportion may require assistance or experience seizures or syncope. Hypoglycaemia is associated with both weight-dependent and weight-independent mechanisms. Key factors include improved insulin sensitivity and increased secretion of incretin hormones such as glucagon-like peptide 1 (GLP-1). The primary goal of treatment is to minimise the postprandial fluctuations in blood glucose and insulin levels. Although certain medications may help to alleviate symptoms, they are rarely used. This clinical overview aims to raise awareness of hypoglycaemia following bariatric surgery and to highlight key considerations for healthcare professionals who encounter these patients.**

Hypoglycaemia is a well-known side effect of diabetes medication but is otherwise a rare condition (1, 2). Blood glucose levels can also drop in cases of severe illness, malnutrition/nutritional deficiency, physical exertion, alcohol consumption, insulin overproduction, liver, kidney or adrenal insufficiency, and with certain medications (2, 3). The clinical presentation ranges from mild symptoms such as dizziness, perspiration and tremors to seizures and loss of consciousness.

The rise in bariatric surgery has been accompanied by an increased awareness of hypoglycaemia as a postoperative complication (4, 5). Bariatric surgery has been an established treatment option in the Norwegian specialist health service since 2004. Before the COVID-19 pandemic, around 3000 patients underwent bariatric procedures annually, and according to the Norwegian Obesity Surgery Registry (SOReg-N), this figure has dropped to just over 2100 (6). An estimated 40,000–50,000 patients have undergone bariatric surgery in Norway, and an unknown number of Norwegians have had surgery abroad. The most common procedures are gastric bypass (50 %) and vertical sleeve gastrectomy (40 %). Postoperative hypoglycaemia is most common after gastric bypass (7, 8). In this procedure, the top of the stomach is divided and a small pouch is attached to the small intestine. Hypoglycaemia is also a known complication of other types of gastric surgery.

Classic postoperative hypoglycaemia usually occurs 1–3 hours after eating and is referred to as 'postprandial hyperinsulinaemic hypoglycaemia'. The phenomenon known as 'early dumping' typically occurs within 20–30 minutes of eating and can present with similar symptoms but is caused by autonomic responses unrelated to hypoglycaemia (7, 9).

Doctors in the specialist health service, general practitioners (GPs) and other healthcare professionals should be aware of hypoglycaemia as a post-bariatric complication, as well as key preventive measures. This clinical review describes the phenomenon and discusses the presumed underlying mechanisms and current treatment options.

The article is based on original research and review articles identified through non-systematic searches in PubMed and UpToDate, as well as reference lists. Search terms included 'hypoglycemia', 'hypoglycaemia', or 'dumping' in combination with 'obesity surgery', 'bariatric surgery', 'prevalence', 'causes', and/or 'treatment'. The author's personal experience with the patient group and published works have also been considered.

## Incidence

Data on the incidence of post-bariatric hypoglycaemia is limited and inconsistent (10). Several studies have reported severe cases with syncope, seizures and hospital admissions in < 1 % of the patients (5). The phenomenon is also associated with acute postoperative mortality (11).

More targeted studies have found a higher incidence, but with a predominance of mild to moderate cases. A meta-analysis included eight studies with continuous glucose monitoring in interstitial fluid (12). Hypoglycaemic values in the range < 3.0–3.9 mmol/L were recorded in 54 % of 280 patients, with no difference between gastric bypass and vertical sleeve gastrectomy. Another large-scale study ($n$ = 333) with oral glucose tolerance testing and a glucose threshold of < 2.8 mmol/L found a higher incidence after gastric bypass (33 %) compared to vertical sleeve gastrectomy (23 %), with no cases of pre-operative hypoglycaemia (13). The risk of hypoglycaemia appears to be higher in women, younger age groups and patients with significant weight loss, lower baseline weight and lower baseline glucose levels (14, 15).

## Symptoms and diagnosis

Symptoms of postprandial hypoglycaemia tend to be mild to moderate. Rapid and severe symptom onset is relatively uncommon. More severe neurological symptoms of hypoglycaemia, such as loss of consciousness and seizures, can be mistaken for epilepsy.

The level of glucose at which hypoglycaemic symptoms occur varies, and different thresholds have been used for diagnosis. By definition, hypoglycaemia is not present when blood glucose is ≥ 4.0 mmol/L in a normal population (1, 2, 5). A drop in glucose initially triggers autonomic symptoms via the central nervous system, including trembling, cold sweats, dizziness and palpitations. Glucose levels below 3.0 mmol/L can trigger neuroglycopenic symptoms, such as concentration problems, memory loss, irritability, lethargy, altered consciousness and more serious clinical outcomes such as arrhythmias, seizures and coma. To meet the diagnostic criteria for hypoglycaemia, three signs must be present: low plasma glucose concentration, symptoms of hypoglycaemia and resolution of these symptoms when plasma glucose concentration is normalised (Whipple's triad) (1, 5, 8).

The symptom profile of postprandial hypoglycaemia resembles the hypoglycaemic symptoms seen in diabetes mellitus. A medical history of diabetes or previous bariatric surgery therefore provides important information about the underlying cause. In diabetes, hypoglycaemia is typically associated with the use of insulin or other antidiabetic medications.

A characteristic feature of post-bariatric hypoglycaemia is meal-related fluctuations and imbalances in glucose and insulin levels, as well as the time it takes for postprandial symptoms to appear (5, 8). Plasma glucose concentration often rises excessively within the first 30 minutes, triggering a supraphysiological insulin response. It subsequently drops, potentially low enough to cause hypoglycaemic symptoms, typically 1–3 hours after a meal. Symptoms often emerge 1–3 years after surgery. Fasting blood glucose levels are usually normal. Hypoglycaemic episodes have also been observed independently of meals, including during fasting or at night.

Symptoms of early dumping occur sooner after eating and are often also triggered by carbohydrates, but they may also be caused by high-fat foods or by eating too much or too quickly. The symptom profile typically includes vasomotor and gastrointestinal symptoms such as a warm sensation, palpitations, nausea, diarrhoea and abdominal pain (9).

In acute cases and recurrent episodes, capillary blood glucose self-monitoring is recommended, along with recording symptoms and prior food intake (3). Table 1 shows suggested glucose thresholds, clinical interpretations and interventions. Raising awareness about diet is essential, and follow-up with a clinical nutritionist may be necessary. In the specialist health service, a meal challenge provocation test is preferred for diagnosis over an oral glucose tolerance test (3, 5). Continuous glucose monitoring in interstitial fluid is sometimes used, but this method has limitations due to time lags and reduced accuracy at low glucose levels.

## Glucose homeostasis and mechanisms

Tissue sensitivity to insulin is reduced in people with obesity, but increased insulin secretion often helps maintain normal blood glucose levels (2). The mechanisms behind insulin resistance are not fully understood, but fat accumulation, oxidative stress and inflammation are considered key factors.

Reduced blood glucose levels after bariatric surgery are attributed to both weight-dependent and weight-independent mechanisms. Weight loss increases insulin sensitivity, allowing more glucose to be transported into tissues. Weight loss also improves β-cell function (5, 16).

Postoperative anatomical changes result in faster food transit to the small intestine. This leads to more rapid and increased absorption of carbohydrates, which is believed to be a key factor in the rise in blood glucose levels (5, 8). There is also a characteristically rapid and enhanced release of incretin hormones from the gut (8). These hormones affect the β-cells in the pancreas and increase postprandial insulin secretion beyond what glucose levels would

indicate. Glucagon-like peptide-1 (GLP-1) is considered the most significant of the incretins, particularly after gastric bypass surgery. Figure 1 illustrates how a meal test affects blood glucose, insulin and GLP-1 levels after gastric bypass compared with normal anatomy (17). Studies have shown up to a tenfold increase in postprandial GLP-1 levels following gastric bypass (5, 8). This increase correlates with hyperinsulinemia and hypoglycaemia.

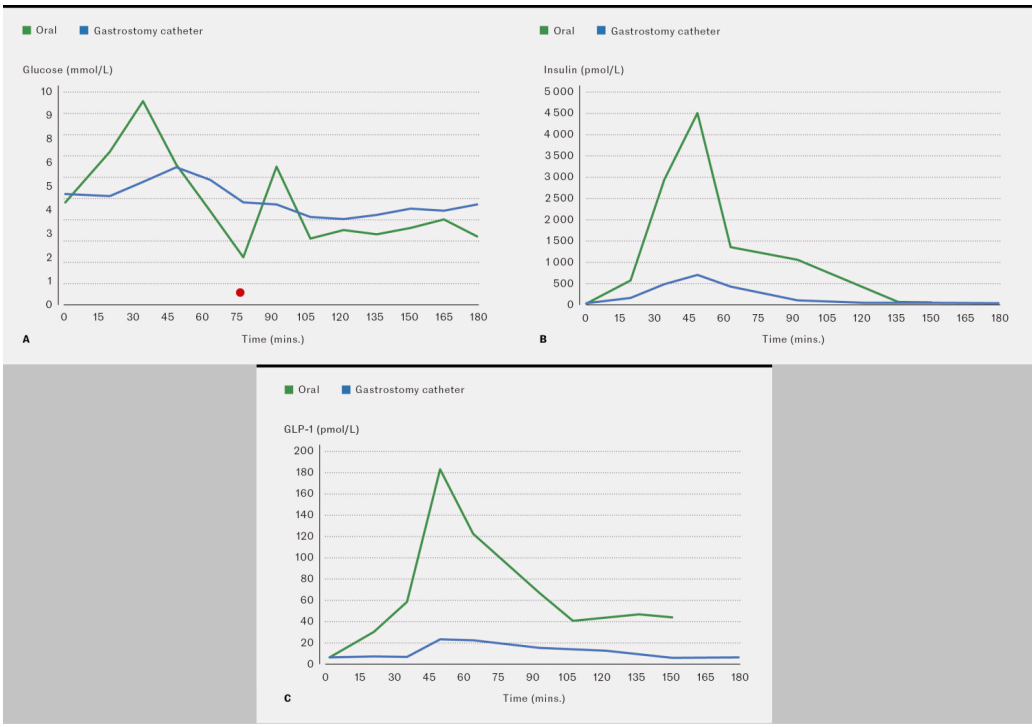


**Figure 1** Meal test in a patient with hypoglycaemia after gastric bypass, with measurements of plasma glucose levels (a), serum insulin levels (b) and plasma levels of glucagon-like peptide 1 (GLP-1) (c). The curves show blood values following oral intake (green) and administration via catheter to the bypassed stomach with pylorus and duodenum (blue). The red circle marks the time point of severe hypoglycaemia, at which point intravenous glucose was administered and the oral test was discontinued. The figure is adapted from Qvigstad (17) and reproduced with permission.

Changes in insulin clearance or in the regulation of insulin secretion are additional plausible mechanisms that can predispose individuals to hypoglycaemia (8). While insulin secretion typically decreases at glucose levels below 4.0–4.5 mmol/L, a further drop in glucose levels triggers increased glucagon secretion. Persistent hypoglycaemia activates several counter-regulatory mechanisms involving both autonomic and hormonal responses. Some data indicate reduced responses of glucagon, adrenaline and cortisol following gastric bypass (8).

## Treatment

Postprandial hypoglycaemia often responds well to dietary interventions, such as consuming smaller and more frequent meals and avoiding rapidly absorbed carbohydrates. Follow-up with a clinical nutritionist is recommended in cases of moderate to severe hypoglycaemia.

Since carbohydrates trigger the strong insulin response and hypoglycaemia, the most important intervention is to reduce carbohydrate intake. We recommend a maximum of 30 grams of carbohydrates per meal. Additionally, foods containing complex carbohydrates and a low glycaemic index lead to a slower rise in blood glucose and a weaker insulin response. Two slices of bread contain approximately 30 grams of carbohydrates, so bread consumption often needs to be limited or replaced with crispbread. Other measures include replacing carbohydrates with salad, vegetables and eggs, as well as foods containing protein and unsaturated fats, which do not affect blood glucose levels. Delaying fluid intake after meals is recommended to slow the passage of food into the intestine. Easily digestible foods can be absorbed quicker in the intestine and intake should be reduced.

A small number of patients require additional treatment. There are currently no medications specifically indicated for hypoglycaemia. However, some are used off-label for symptom relief. The first-line medication is often acarbose – an antidiabetic drug that inhibits the breakdown of carbohydrates in the intestine and delays glucose absorption (7). Many patients experience symptom relief, but gastrointestinal side effects are common. Diazoxide and somatostatin analogues, which inhibit insulin secretion, have been used in some difficult cases. Both medications have side effects that limit efficacy, and evidence from clinical trials is lacking. Such treatment should be managed by a specialist. Anecdotally, some patients have responded to calcium channel blockers and corticosteroids.

Paradoxically, GLP-1 analogues appear to stabilise glucose levels and can help prevent hypoglycaemia (10). One possible explanation is that they enhance satiety even after small meals. However, GLP-1 analogues can also cause gastrointestinal side effects and exacerbate hypoglycaemia. A few studies have shown promising effects of GLP-1 antagonists, but these are not in routine use (8). Glucagon therapy is relevant in cases of severe hypoglycaemia.

In severe cases, surgical reversal of gastric bypass to near-normal anatomy may be considered as a treatment option. It often leads to the patient regaining weight, and many patients are sceptical. An oral meal test, followed by catheter-based delivery of the same meal to the excluded stomach, can be useful before surgical reversal. Changes in blood glucose, GLP-1 and insulin levels indicate whether restoring normal anatomy could reduce postprandial fluctuations (Figure 1).

## Summary

Postprandial hypoglycaemia after bariatric surgery can have serious consequences. Both the patients and healthcare professionals managing this patient group should be aware of the phenomenon. A thorough medical history and blood glucose measurements are essential for an accurate diagnosis. Symptoms usually improve by reducing the intake of fast carbohydrates. Some patients should be referred for further investigation and treatment in the specialist health service.

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