Check for updates

Received: 15 April 2024 | Revised: 7 October 2024 | Accepted: 26 October 2024

DOI: 10.1111/nmo.14962

CLINICAL REVIEW

Neurogastroenterology & Motility NGM WILEY

Dumping syndrome: Update on pathophysiology, diagnosis, and management

Jan Tack[1,2] | Karlien Raymenants[1] | Cedric Van de Bruaene[1] | Emidio Scarpellini[1]

[1]Translational Research Center for Gastrointestinal Diseases (TARGID), University of Leuven, Leuven, Belgium

[2]Division of Gastroenterology and Hepatology, Leuven University Hospitals, Leuven, Belgium

Correspondence
Jan Tack, Translational Research Center for Gastrointestinal Diseases (TARGID), University of Leuven, University Hospital Gasthuisberg, O&N I—bus 701, Herestraat 49, Leuven 3000, Belgium.
Email: jan.tack@kuleuven.be

Funding information
Onderzoeksraad, KU Leuven; Fonds Wetenschappelijk Onderzoek

Abstract

Background: Dumping syndrome is a complex of gastrointestinal symptoms originally studied in peptic ulcer surgery patients. At present, it is most prevalent in patients who underwent bariatric, upper gastrointestinal cancer or anti-reflux surgery. The symptom pattern comprises early and late dumping symptoms. Several management options have been reported including nutritional, pharmacological and surgical approaches.

Aims and Methods: In this study, we aimed to review the current evidence on dumping syndrome definition, diagnosis and treatment, including preliminary data from newer pharmacological studies.

Results: Current pathophysiological concepts and analyses of provocative tests has led to a clear definition of dumping syndrome, including both early and late dumping symptoms. The term postbariatric hypoglycemia represents a limited focus on late dumping only. The diagnosis relies on recognition of symptoms and signs in a patient with appropriate surgical history; and can be confirmed by provocative testing or registration of spontaneous hypoglycemia. The initial treatment focuses on dietary intervention, to which meal viscosity enhancers and/or the glycosidase inhibitor acarbose can be added. The most effective therapy is the use of short- or long-acting somatostatin analogues, which is however expensive and entails side effect issues. In case of refractory hypoglycemia, diazoxide or SGLT2 inhibitors can be considered, based on limited evidence. In refractory patients, continuous enteral feeding or (rarely) surgical reinterventions have been advocated, although not supported by solid evidence. Therapies under current evaluation include the broad-spectrum somatostatin analogue pasireotide, GLP-1 receptor antagonists, GLP-1 receptor agonists and administration of stable forms of glucagon are currently under study.

Conclusions: Dumping syndrome is a well-defined but probably under-diagnosed complication of upper gastrointestinal, especially bariatric, and surgeries. Diagnosis is confirmed by a provocative test and incremental therapies starting with diet, adding meal viscosity enhancers or glycosidase inhibitors and adding somatostatin analogues in refractory cases. A number of emerging therapies targeting intestinal propulsion, peptide hormone effects and hypoglycemic events are under evaluation.

KEYWORDS
acarbose, diazoxide, dumping syndrome, postbariatric hypoglycemia, somatostatin analogue

 © 2024 John Wiley & Sons Ltd.

1 | INTRODUCTION

Dumping syndrome was first described by Hertz in 1913, who reported the occurrence of 'dumping-like' symptoms after gastroenterostomy.[1] The term refers to symptoms and signs that occur when food reaches the small bowel in a too large quantity and/or too rapidly. Dumping syndrome, which was best-known in the era of peptic ulcer surgeries, commonly occurs after partial or total gastrectomy or surgical resections, now most commonly resulting from bariatric or oncological interventions.[2–4] Under normal circumstances, the stomach together with the duodenum determines the timed release of ingested nutrients into the duodenum through the interplay of antral contractions, pyloric tone, proximal stomach tone and negative feedback from duodenal nutrient sensing, coordinated by the vagus nerve and peptide hormone release.[5] This coordinated function is often compromised after gastric or esophageal surgery, leading to manifestations of dumping syndrome.[2–4]

It has been estimated that dumping syndrome occurs in up to 40% of patients undergoing gastrectomy, and in up to 50% of patients after esophagectomy.[2–6] Dumping syndrome has also been reported after Nissen fundoplication in both children and adults.[7–10] At present, bariatric surgery has become the principal cause of postoperative dumping syndrome, it frequently occurs as a complication of Roux-en-Y gastric bypass (RYGB) as well as the purely restrictive sleeve gastrectomy.[11–13] Furthermore, surgery involving the pylorus, which entails the risk of vagotomy, can potentially lead to dumping syndrome, since subsequent pyloric relaxation and loss of the duodenogastric feedback accelerates transit time.[2,14] However, a study on the safety profile of Gastric per oral endoscopic esophageal myotomy (G-POEM) for gastroparesis, showed only low risk for dumping syndrome, 1.4%, in these patients, all within 1 month post-procedure and manageable by change in diet.[15] A recent systematic review by Vanuytsel et al. assessed the prevalence of early and late dumping after bariatric surgery, which differs according to the technique used.[16] In the case of purely restrictive surgery, such as laparoscopic sleeve gastrectomy (LSG), dumping syndrome is thought to originate from the change in gastric accommodation due to loss of receptive relaxation, and resection of the pacemaker region of the stomach, leading to accelerated gastric emptying.[13,17] After RYGB, dumping seems to originate from the accelerated clearance of food from the stomach pouch. Furthermore, bypassing the pylorus precludes the duodenogastric feedback mechanism, which slows gastric emptying under normal conditions. Evidence for the role of the pylorus in the development of dumping syndrome was found by Vives et al. who found significantly higher gastric emptying rates, following LSG with antral resection versus without.[18]

Furthermore, vagal nerve damage seems to be important, since periprocedural transection of the lesser omentum was associated with higher symptom scores in a post-bariatric questionnaire on dumping symptoms.[19]

Overall, dumping syndrome is least frequent in laparoscopic adjustable gastric banding, ±5%, followed by both LSG, and RYGB with an incidence of approximately 30%.[16,20] A large single-center, questionnaire-based study compared LSG with RYGB, using the modified version of the Sigstad scoring system, and found a significantly higher prevalence for dumping in RYGB, 41.4%, when compared to LSG, 26.5%.[17] However, it should be noted that response rates in this study were low and skewed (30.3% in the RYGB group vs. 22.5% in the LSG group) and that higher prevalences of dumping syndrome are measured by the Sigstad scoring system, when compared to physiological testing.[16]

Key Points

- Dumping syndrome refers to a set of early and late symptoms, occurring after ingestion of a meal in patients who underwent upper gastrointestinal surgery.
- Early dumping syndrome are cardiovascular and gastrointestinal symptoms, due to exposure of the small bowel to excessive caloric and osmotically active content. Late dumping syndrome is hypoglycemia due to overshoot of early insulin release after the meal.
- Management of dumping syndrome is based on recognition of the condition, confirmatory testing and treatment approaches starting with diet, adding agents that slow gastric emptying or inhibit insulin release, and in case of insufficient control, injectable somatostatin analogues.

1.1 | Symptom profile and impact

Two different sets of symptoms and signs have been identified, which are categorized as early and late dumping, respectively (Table 1 and Figure 1). Early dumping symptoms typically occur within the first hour after a meal and include gastrointestinal as well as vasomotor symptoms.[1–4] Gastrointestinal early dumping symptoms include abdominal pain, bloating, borborygmi, nausea, and diarrhea, while vasomotor early dumping symptoms may include fatigue, flushing, palpitations, perspiration, hypotension, and rarely syncope. Early dumping, unlike many other gastroduodenal sensorimotor disorders, triggers in the patient a strong desire or need to lie down after meals.[1–4]

Late dumping consists of symptoms of reactive hypoglycemia which is a consequence of a preceding rapid rise in glycemia during the early dumping phase. The late dumping symptoms comprise perspiration, palpitations, tremor, irritability, hunger, fatigue, weakness, confusion, and may lead to hypoglycemic syncope.[1–4] Its occurrence after bariatric surgery has been recognized in the term "postbariatric hypoglycemia" (PBH),[24] but this fails to reflect the invariably present early dumping as a preceding and initiating factor, and also fails to recognize that (early and) late dumping symptoms also occur

13652982, 2025, 2, Downloaded from https://onlinelibrary.wiley.com/doi/10.1111/nmo.14962 by Capes, Wiley Online Library on [10/03/2025]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

as a consequence of several other types of upper gastrointestinal surgery.[6–9,25,26]

With symptoms occurring on a daily basis, triggered by every meal, dumping syndrome has a major negative impact on quality of life, and this is confirmed in many studies.[21,27,28] A specific post-gastrectomy quality of life instrument was developed, which documented significantly impaired quality of life after gastrectomy.[29] Furthermore, a large proportion of patients with dumping syndrome are unable to work because of the symptoms and are on prolonged or permanent sick leave or unemployment.[21,27–29]

A matter of debate is the extent to which dumping symptoms after bariatric surgery contribute to the weight loss that these interventions aim for. While it is well established that dumping syndrome after various types of surgery may lead to weight loss,[11–13,24] the presence of dumping syndrome is not a determinant of the amount of weight loss after RYGB surgery.[11]

TABLE 1 Early and late dumping symptoms (in order of prevalence).

Early dumping syndrome symptoms
• Abdominal pain
• Bloating
• Dizziness
• Diarrhea
• Sweating
• Palpitations
• Nausea
• Flushing
Late dumping syndrome symptoms
• Drowsiness
• Hunger
• Sweating
• Tremor
• Palpitations
• Irritability
• Unconsciousness

2 | PATHOPHYSIOLOGY

A number of mechanisms have been implicated in the generation of symptoms and signs in dumping syndrome.[2–4,30] The main factor underlying early dumping symptoms is the rapid delivery of nutrients and osmotically active substances into the small bowel, where they overwhelm the physiological absorptive capacity (Figure 2). This is mainly present after surgeries involving (partial) gastrectomy or gastro-intestinal anastomosis, as mentioned earlier. The presence of hyperosmolar small bowel content shifts fluid from the vascular compartment to the intestinal lumen, resulting in a reduced circulating plasma volume, tachycardia, hypotension, and rarely syncope. This is shown as an early rise in hematocrit in dumping provocative tests (see below).[2–4,21–23,30] However, when intravenous fluid

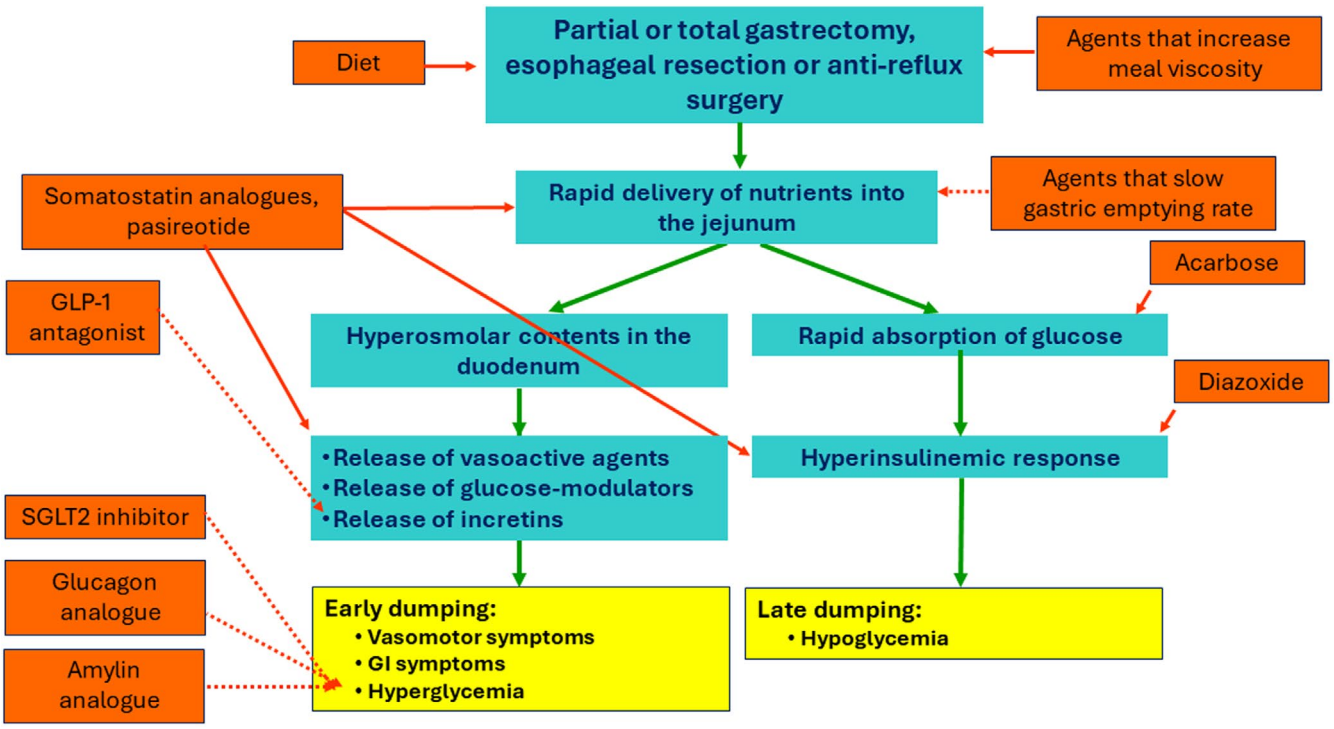

FIGURE 1 Percentage of patients experiencing symptoms in the early and late dumping phase ($n = 56$). Unpublished data from baseline symptom scores in interventional trials with long-acting somatostatin analogues and pasireotide.[21–23] AP, abdominal pain; B, bloating; DZ, dizziness; DR, diarrhea; N, nausea; FL, flushing; SW, sweating; P, palpitations; SL, sleepiness; H, hunger; TR, tremor; AG, agitation.

13652982, 2025, 2, Downloaded from https://onlinelibrary.wiley.com/doi/10.1111/nmo.14962 by Capes, Wiley Online Library on [10/03/2025]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

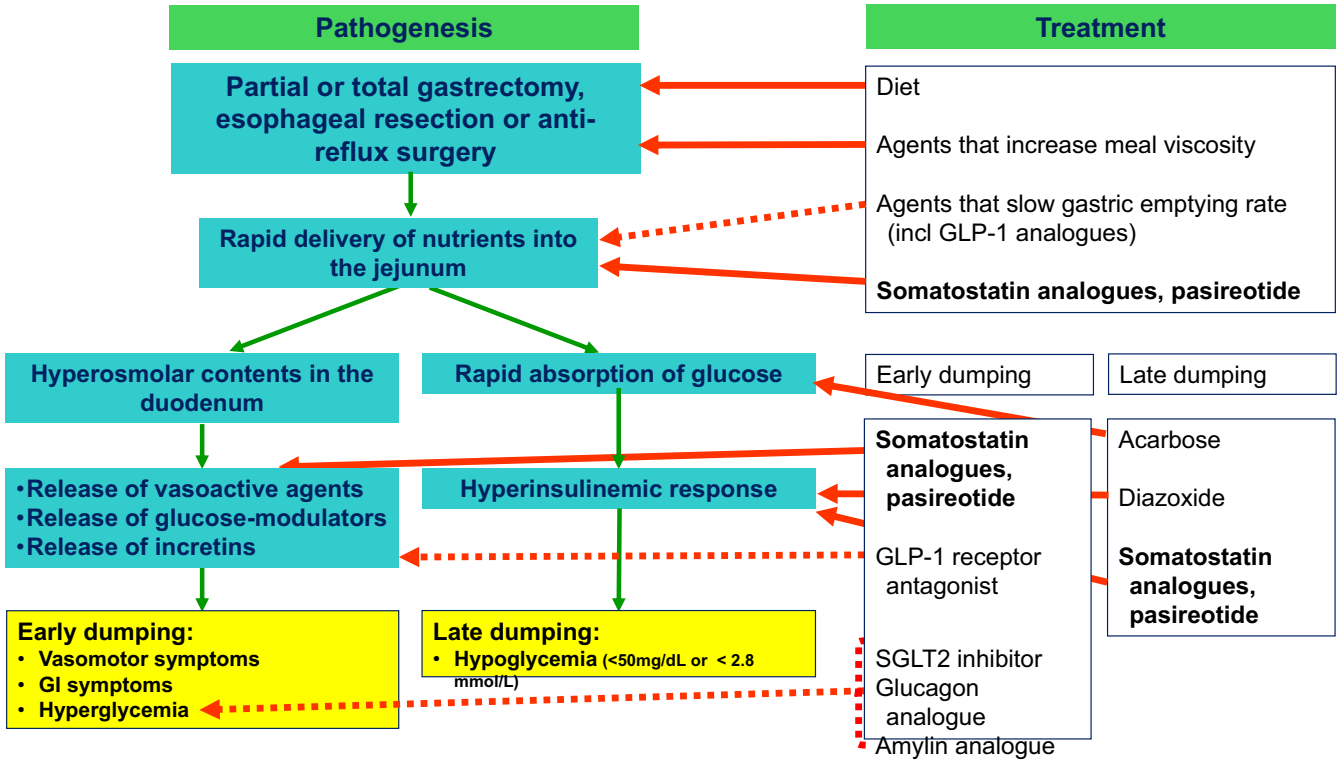

FIGURE 2 Summary of the pathophysiological mechanisms and treatment targets in dumping syndrome.

is administered to compensate for the volume shift, early dumping symptoms persist,[31] indicating involvement of other factors. For example, accumulation of fluid in the small bowel may induce distention leading to symptoms such as cramping, pain and bloating. Indeed, dilated small bowel loops are often observed during barium radiography in patients with dumping syndrome, and duodenal balloon distention is able to induce upper abdominal symptoms in healthy volunteers, similar to patients with dumping syndrome.[32–34]

However, the most important mechanism underlying early dumping symptoms is enhanced release of several gastrointestinal hormones including vasoactive agents (e.g., neurotensin and vasoactive intestinal peptide), incretins (e.g., glucagon-like peptide [GLP]-1), peptide YY, glucose-dependent insulinotropic polypeptide and glucose modulators (e.g., insulin and glucagon).[2–5,30] The release of these peptides and signaling molecules may underlie gastrointestinal as well as cardiovascular symptoms. A systematic analysis of the literature identifies GLP-1 as the gut peptide with the most consistently elevated postprandial levels in post-bariatric hypoglycemia.[35,36] However, the level of insulin secretion is also an important contributing factor and besides GLP-1, PYY has also been implicated in the early dumping phase.[37] PYY is known to delay gastric emptying and mouth to cecum transit time, and inhibit gastric acid secretion.[38] Several studies have shown increased levels of postprandial PYY after bariatric surgery, which may involve both the proximal and distal small bowel, and PYY has been suggested to contribute to inhibition of upper gastrointestinal propulsive motility, pancreatic endocrine and exocrine secretion and satiety-related signaling in the brain in patients with dumping syndrome.[39]

In addition to loss of resistance to passage of nutrients to the small bowel, the aforementioned gastric volume capacity is another important determinant of dumping syndrome symptoms. This is illustrated both by the occurrence of dumping after sleeve gastrectomy and also after a Nissen fundoplication in children and adults.[7–9,12,13,30]

Early dumping symptoms driven by osmotic and peptide hormone release are already present 30 min after meal ingestion.[2–4,21,30] Late dumping symptoms are caused by a hyperinsulinemic response leading to hypoglycemia (Figure 2). Rapid delivery of carbohydrates to the small intestine during the early dumping phase generates elevated glucose concentrations which trigger a hyper-insulinemic response, and subsequent hypoglycemia.[2–4,22,23,25,26,30,35–37,40] Within this pathophysiological mechanism, early dumping and insulin sensitivity are the prerequisites for late dumping—i.e. hypoglycemia—to occur. Indeed, clinical signs of hypoglycemia often occur or are recognized only several months after bariatric surgery, perhaps because of the need for insulin sensitivity to recover as weight loss occurs.[28] However this has never been investigated in a prospective trial. Analyses of early and late dumping symptom occurrence support the view that both are linked, and that the early dumping phase is the determining factor, presumably leading to occurrence of late dumping in those who are sensitive to hypoglycemic factors.[25,26]

3 | DIAGNOSIS

Diagnosing dumping syndrome is based on clinical awareness and recognition of the symptoms in an appropriate clinical context,

13652982, 2025, 2, Downloaded from https://onlinelibrary.wiley.com/doi/10.1111/nmo.14962 by Capes, Wiley Online Library on [10/03/2025]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

exclusion of confounding factors (e.g., mechanical obstruction) and confirmatory testing, as needed.[2–4,30] Clinical suspicion should be raised by a suggestive symptom pattern in a patient with a history of upper gastrointestinal surgery for bariatric (including sleeve gastrectomy, RYGB), oncological (including esophagectomy, (partial) gastrectomy) or other reasons. Both meal-induced gastrointestinal and cardiovascular symptoms, as well as symptoms suggestive of hypoglycemia, may occur. Profound fatigue after meal ingestion, with the need to lie down, is an important clinical clue suggestive of dumping syndrome, and is absent in patients with meal-related symptoms due to functional dyspepsia or gastroparesis. An overview of the diagnostic sequence can be found in Figure 3.

In the 1970s, a symptom-based diagnostic index was proposed by Sigstad, mainly based on observations in patients who had undergone vagotomy[3,41] (Table 2). The diagnostic performance of this score in current-day dumping syndrome patients has not been established. A recent study in 271 patients post-barriatric surgery provided evidence of the contrary, as there was no correlation between Sigstad diagnostic index score and late dumping symptoms.[42] Mechanical obstruction in the postoperative setting needs to be excluded by appropriate imaging investigations.

Measurement of spontaneous hypoglycemia at a time of symptoms can confirm suspected hypoglycemia, although the threshold low value of glycemia which can be considered abnormal has not been

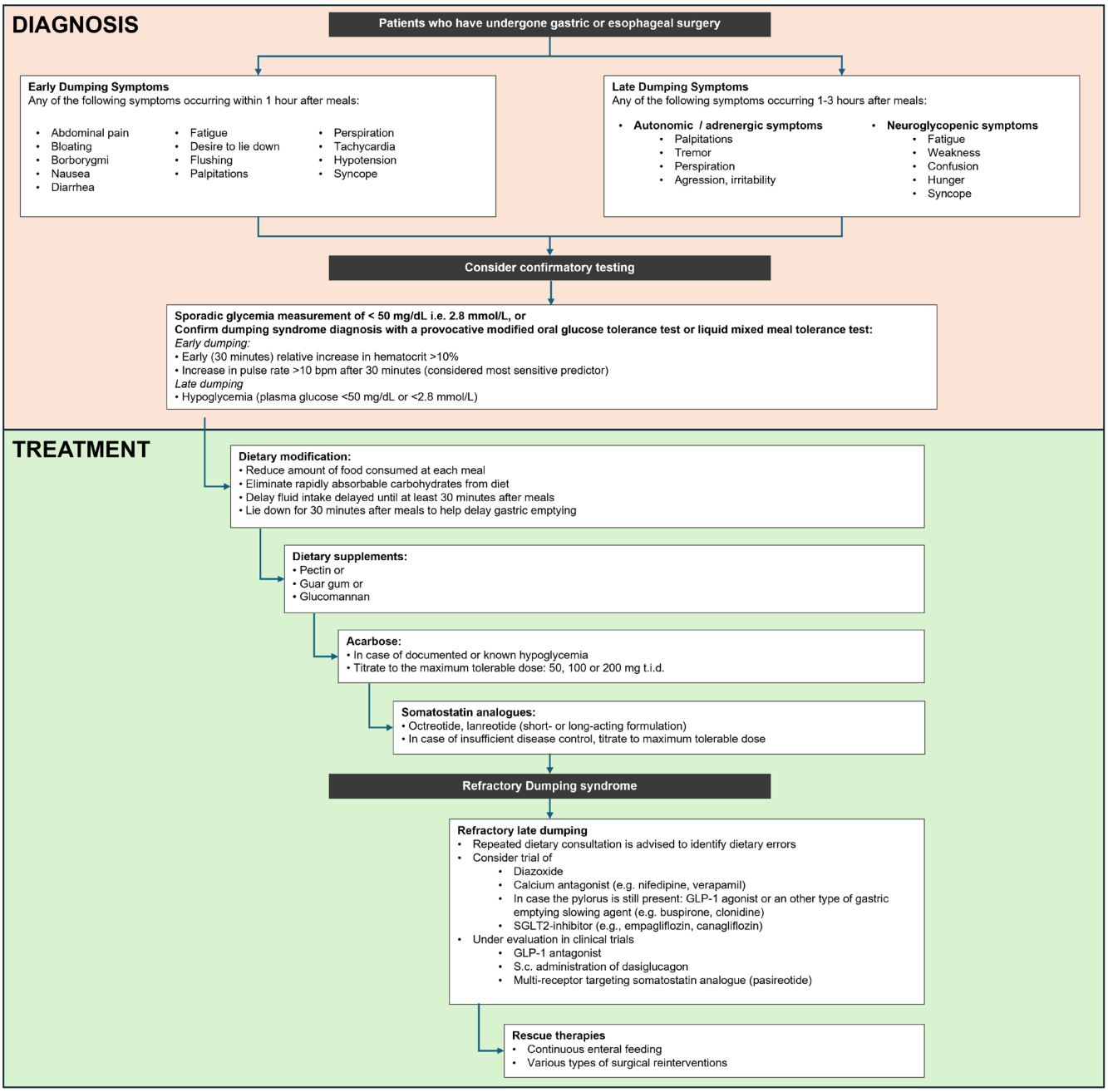

FIGURE 3 Overview of the management sequence (diagnosis and treatment) in dumping syndrome.

13652982, 2025, 2, Downloaded from https://onlinelibrary.wiley.com/doi/10.1111/nmo.14962 by Capes, Wiley Online Library on [10/03/2025]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

TABLE 2 Sigstad index for dumping syndrome.

Postprandial symptoms	Score[a]
Shock	+5
Fainting, syncope, unconsciousness	+4
Desire to lie down	+4
Dyspnea	+3
Weakness	+3
Sleepiness, apathy	+3
Palpitations	+3
Restlessness	+2
Dizziness	+2
Headaches	+1
Warm, clammy skin or pallor	+1
Nausea	+1
Abdominal fullness	+1
Borborygmi (abdominal rumbling/gurgling)	+1
Eructation (belching)	−1
Vomiting	−4

[a]A score of >7 is suggestive of dumping syndrome. A score of <4 is suggestive of an alternate diagnosis.

fully established. An international multidisciplinary Delphi consensus supported a threshold glycemia of 2.8 mmol/L (50 mg/dL) or lower as abnormal.[30] A provocative test can be used to confirm clinical suspicion. The best established test in the literature is based on the oral glucose tolerance test (OGTT), involving the ingestion of 75 g glucose in solution after an overnight fast, with measurement of glycemia, pulse and hematocrit at 30 minutes interval up to 3 h after ingestion.[2–4,21,30] The test is positive for early dumping in case of an increase in hematocrit >3% or an increase in pulse rate >10 beats/min after 30 min, and positive for late dumping in case of hypoglycemia (<2.8 mmol/L) after 60–180 min post-ingestion. Although highly reproducible,[23] the glucose provocative test has been criticized as it might lack specificity and lead to over-diagnoses of dumping syndrome.[43–45] It is indeed not evident to define a certain postprandial hypoglycemia value as cutoff for late dumping. To increase specificity, the presence of hypoglycemia in association with symptoms, relieved by carbohydrate ingestion, can be used to indicate late dumping (Whipple's triad).[46]

As an alternative, a mixed meal provocative test has been advocated, mainly as a measure to induce late dumping (hypoglycemia).[34,36,40] Patients ingest a mixed liquid meal containing carbohydrates, fats, and proteins after an overnight fast, and blood samples are collected at 30-min intervals for up to 2 h. The mixed-meal tolerance test is considered positive for late dumping in patients who develop hypoglycemia between 60 and 180 min after meal ingestion.

Anecdotal reports suggest that continuous glycemia monitoring may help to demonstrate hypoglycemia (late dumping), although the diagnostic accuracy in comparison to that of dumping provocative tests has not been fully determined.[47,48] In addition, although increasingly used in clinical trials, its value as primary outcome parameter is yet to be established.[49–51]

Although rapid gastric emptying is a key feature in the pathophysiology of dumping syndrome, gastric emptying testing with solid and liquid component lacks diagnostic value.[2–4,30] After total gastrectomy, the test has no value and while initial rapid gastric emptying may suffice to trigger early dumping symptoms, the overall value of gastric emptying rate is most often within the normal range, decreasing its utility in the assessment of presumed dumping syndrome.

4 | TREATMENT

4.1 | Dietary adjustment

After a suspected or confirmed diagnosis of dumping syndrome, the first-line treatment is dietary adjustment, which comprises a reduction in the amount of food ingested at each meal, postponement of fluid intake until at least 30 min after the meal (aimed at early dumping, to limit gastric emptying of the first fluid component) and elimination of rapidly absorbable carbohydrates (aimed at late dumping).[2–4,30] While the dietary approach is well accepted in clinical practice, the literature lacks convincing outcome data. Although it would seem beneficial, the advantage of referral to an experienced dietician over physician recommendations has not been established. It has been estimated that close to 50% of patients have a clinically relevant response to the dietary interventions.[2–4]

4.2 | Dietary supplementation with viscosity enhancing agents

Based on the assumption that increased viscosity of the meal slows the release of nutrients to the small intestine and may thus improve dumping syndrome symptom triggering, studies have evaluated the impact of adding guar gum, pectin and glucomannan to meals (Table 3). Small studies showed that ingestion of up to 15 g of guar gum or pectin with each meal is able to slow gastric emptying, reduce the release of gastrointestinal hormones, improve hyperglycaemia (and thus subsequent hypoglycemia) and control symptoms of dumping syndrome.[52–59,61–63] A single study reported that glucomannan improved glucose in children after a variety of gastric surgical procedures.[60] In clinical practice, the tolerance and palatability of these supplements limit their applicability beyond the short term. In addition, these supplements are not readily available as approved pharmaceutical products at these high doses and may require specific pharmacists' preparation.

In patients not responding to dietary intervention and/or supplementation, pharmacotherapy is the recommended next step. A number of pharmacological interventions have been evaluated, potentially showing higher efficacy as they were mainly studied in non-responders to diet.[2–4,30]

13652982, 2025, 2, Downloaded from https://onlinelibrary.wiley.com/doi/10.1111/nmo.14962 by Capes, Wiley Online Library on [10/03/2025]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

TABLE 3 Summary of studies evaluating pectin, guar gum and glucomannan in dumping syndrome.

Study	*n*	Treatment	Result
Jenkins et al.[52]	9	Pectin 14.5 g, single administration prior to OGTT	Improved symptoms and glycaemia levels (normalized in 46%) during OGTT
Jenkins et al.[52–54]	11	Pectin 14.5 g, single administration prior to OGTT	Improved postprandial levels of glucose, insulin and enteroglucagon. Reduced hypoglycaemia
Leeds et al.[55]	11	Pectin 15 g, single administration prior to OGTT	Improved vasomotor symptoms and glycaemia levels, lower insulin levels and slower gastric emptying during OGTT
Lawaetz et al.[56]	4	Pectin 15 g, single administration prior to OGTT	Reduced vasomotor symptoms, lower levels of insulin, glucagon, neurotensin and GIP and slower initial gastric emptying during OGTT
Harju et al.[57]	11	Guar gum 5 g with meals	Improvement of symptoms
Harju et al.[58]	11	Guar gum 5 g with meals	Slowing of gastric emptying
Harju et al.[59]	11	Guar gum 5 g with a glucose challenge meal	Improvement of symptoms and hyperglycaemia after a glucose challenge meal
Kneepkens et al.[60]	8	Glucomannan 1.3 g, single administration prior to OGTT	Improvement of glucose tolerance, no effect on glucose absorption; however, no consistent effect on symptoms was seen
Andersen et al.[54]	5	Pectin 5 g, single administration prior to muffin meal	No effect on symptoms or gastric emptying rate
Snook et al.[61]	9 healthy volunteers	Duodenal instillation of 150 mL glucose with 5 g guar gum	Failure to modify experimental dumping through intraduodenal instillation of a hypertonic glucose meal

Abbreviations: OGTT, oral glucose modified tolerance test; GIP, Gastric Inhibitory Peptide.

4.3 | Acarbose

Acarbose is a luminally acting alpha-glycosidase inhibitor that slows the release of monoglycerides from nutritional carbohydrates. The available studies show that acarbose at 50–100 mg t.i.d. doses is able to improve glucose tolerance, reduces gastrointestinal hormone release and reduces the incidence of hypoglycaemia (namely, the manifestation of late dumping syndrome)[8,50,60,63–73] (Table 4). Efficacy on early dumping is not expected and has not been documented in the available studies. The main side effects associated with acarbose use are flatulence, bloating and diarrhea, due to colonic fermentation of non-absorbed carbohydrates.

4.4 | Diazoxide

Diazoxide is a potassium channel activator that inhibits calcium-induced insulin release at doses of 100–150 mg t.i.d. Its potential to improve the hypoglycaemia of late dumping syndromeis supported by a few case reports and a retrospective case series in the literature.[74–76] One small prospective series, published as abstract only, showed improvement of late hypoglycemia but none of the other parameters in dumping syndrome.[77] Hence, the use of diazoxide should probably only be considered in case of refractory late dumping (hypoglycemia).

4.5 | Somatostatin analogues

Activation of somatostatin receptors has multiple effects of potential benefit in dumping syndrome: slowing of gastric emptying, slowing of small bowel transit, inhibition of the release of gastrointestinal hormones, and inhibition of insulin secretion and inhibition of postprandial vasodilation. Taken together, this is a mechanistic profile that allows to improve both early and late dumping symptoms (Figure 2).

Short-acting somatostatin analogues, administered subcutaneously three times daily, have shown efficacy in improving symptoms of dumping syndrome, both for early and for late dumping symptoms (Table 5). The doses used, octreotide mostly 50–100 μg s.c., are substantially lower than the ones used in other indications for somatostatin analogues.[21,30,78–85,88,89] At present, this is likely the most effective approach to manage dumping symptoms, but the need for daily repeated injections limits the long-term patient adherence to this treatment.[21,30,86,90] Moreover, somatostatin analogues are expensive treatments, the cost of which can be to some extent offset by using multi-draw vials in countries where they are available.

Long-acting formulations, both octreotide L.A.R. and lanreotide, administered at 4-week intervals, respectively, intramuscularly or subcutaneously, have been evaluated in dumping syndrome patients (Table 5). Both early and late dumping syndrome symptoms improve with these preparations, and they are preferred by patients, most

13652982, 2025, 2, Downloaded from https://onlinelibrary.wiley.com/doi/10.1111/nmo.14962 by Capes, Wiley Online Library on [10/03/2025]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

TABLE 4 Summary of studies evaluating acarbose in dumping syndrome.

Study	*n*	Treatment	Result
McLoughlin et al.[67]	10	Acarbose 100 mg single administration prior to OGTT	Improved symptoms and hyperglycaemia and hypoglycaemia during OGTT, reduced rise in plasma levels of GIP and insulin, no change in gastric emptying rate
Speth et al.[63]	9	Acarbose 50–100 mg, pectin 4.2 g, acarbose 50 mg plus pectin 4.2 g, placebo, after standard breakfast	Acarbose and acarbose plus pectin inhibited postprandial hyperglycaemia and hypoglycaemia, acarbose plus pectin inhibited hyperinsulinemia, acarbose, pectin and combination therapy reduced hypoglycaemic symptoms
Gerard et al.[64]	24	Acarbose 100 mg single administration prior to OGTT	Improved hyperglycaemia and hypoglycaemia during OGTT, reduced rise in plasma levels of insulin, inhibition of glucose-induced glucagon suppression
Lyons et al.[66]	13	Acarbose 50 mg single administration prior to standard breakfast	Significant attenuation of hyperglycaemia, reduced rise in plasma levels of GIP, enteroglucagon and insulin, no influence on plasma levels of VIP and somatostatin, no significant effect on symptoms
Hasegawa et al.[65]	6	Acarbose 50–100 mg t.i.d. before meals for a month	Attenuation of glucose fluctuations and improvement of dumping syndrome symptoms (uncontrolled)
Ozgen et al.[69]	21	Acarbose 150 mg per day before meals for 2 weeks and 300 mg per day for the remainder of the 3-month treatment period	Reduced early hyperglycaemic and hyperinsulinemic response, reduced reactive hypoglycaemia
Ng et al.[8]	6	Acarbose 12.5 mg before a meal	Improved postprandial hypoglycaemia
De Cunto et al.[51]	4	Acarbose 25–100 mg before meals	Stabilized postprandial levels of glucose
Valderas et al.[70]	8	Acarbose 100 mg before a meal	Avoided postprandial hypoglycaemia, reduced hyperinsulinaemic response, reduced GLP1 secretion
Ritz et al.[50]	8	Acarbose 50–100 mg, 3 times per day for 6 weeks	Eliminated dumping syndrome symptoms and improved CGM profile
Cadegiani et al.[72]	25	Acarbose 50 mg, 4–5 times per day for 6 months	Decrease in number of early and late dumping episodes, complete resolution in 84%, increase in ability to perform resistive exercise (uncontrolled)
Øhrstrøm et al.[73]	11	Cross-over study with acarbose 50 mg 1 week, sitagliptin 100 mg 1 week, verapamil 120 mg 1 week, liraglutide 1.2 mg 3 weeks, pasireotide 300 µg single dose	Increased nadir glucose levels and reduced time in hypoglycaemia, reduced peak glucose levels and time in hyperglycaemia, reduced insulin and c-peptide levels.

Abbreviations: CGM, continuous glucose monitoring; GIP, gastric inhibitory peptide; GLP1, glucagon-like peptide 1; OGTT, oral glucose modified tolerance test; t.i.d., three times per day;VIP, vasoactive intestinal polypeptide.

likely because of less frequent administration.[21,27,30,86,87] Besides local reactions at the injection site, the use of somatostatin analogues can be associated with steatorrhea and gastrointestinal discomfort in the short term, and gallstone formation in the long term.[2–4,30]

4.6 | Treatment sequence and other therapeutic options

Once a diagnosis of dumping syndrome is established, dietary intervention should always be the first-line approach. If available, an experienced dietician can be involved and the focus should be on limiting liquids with meals and avoidance of rapidly-absorbable carbohydrates. Rescue therapy for hypoglycemic events should be a rapidly-absorbable glucose source, combined with a slowly- absorbable carbohydrate source, for example, a fiber-containing bar. In case of insufficient response to dietary intervention, acarbose can be added in those with documented late dumping, and guar gum (or pectin or glucomannan) can be considered for all patients. In case of persisting impactful symptoms, especially late dumping (hypoglycemia), somatostatin analogues are the most efficacious option. Cost and side-effect issues should be carefully considered. While the short-acting formulation provide the best symptom control, long-acting preparations are easier for patient acceptance, especially for long-term maintained use. Diazoxide should only be considered in case of refractory late dumping. An overview of treatment sequence can be found in Figure 3.

4.7 | Refractory patients

A number of therapeutic approaches to dumping syndrome are available but relatively poorly studied, are emerging, or still under

13652982, 2025, 2, Downloaded from https://onlinelibrary.wiley.com/doi/10.1111/nmo.14962 by Capes, Wiley Online Library on [10/03/2025]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

TABLE 5 Summary of studies evaluating somatostatin analogues in dumping syndrome.

Study	*n*	Treatment	Result
Short-acting somatostatin analogues			
Hopman et al.[78]	12	Octreotide 50 μg versus placebo prior to OGTT	Improved symptoms of dumping syndrome and suppression of postprandial rise in pulse rate, reduced peak insulin and higher nadir glycaemia, slowing of gastrointestinal transit
Primrose and Johnston[79]	10	Octreotide 50 versus 100 μg versus placebo prior to OGTT	Reduced symptoms of early dumping syndrome and abolished symptoms of late dumping syndrome, suppression of early dumping-associated changes in hematocrit and pulse rate, inhibition of hypoglycaemia
Tulassay et al.[80]	8	Octreotide 50 μg versus placebo prior to OGTT	Suppression of rise in pulse rate and hematocrit, suppression of rise in plasma levels of VIP, inhibition of postprandial hypoglycaemia; inhibition of rise in plasma levels of insulin and GIP
Geer et al.[81]	10	Octreotide 100 μg versus placebo prior to a dumping provocative meal	Prevention of development of symptoms of dumping syndrome and diarrhea, prevention of late hypoglycaemia and of the rise in plasma levels of glucose, glucagon, pancreatic polypeptide, neurotensin and insulin, delayed gastric emptying and intestinal transit
Richards et al.[82]	6	Octreotide 100 μg versus placebo prior to a dumping provocative meal	Prevention of symptoms of dumping syndrome, induction of migrating motor complex phase 3 in the small intestine, less postprandial intestinal motor activity
Gray et al.[83]	9	Octreotide 100 μg versus placebo prior to a dumping provocative meal	Suppression of rise in pulse rate, inhibition of insulin release, prevention of hypoglycaemia, inhibition of symptoms of dumping syndrome
Hasler et al.[84]	8	Octreotide 50 μg versus placebo prior to OGTT	Suppression of rise in pulse rate, inhibition of symptoms of dumping syndrome and diarrhea, no influence on change in hematocrit, inhibition of insulin release, prevention of hypoglycaemia, no influence on gastric emptying rate
Vecht et al.[85]	9	Octreotide 25 μg s.c. prior to OGTT versus placebo	Prevention of activation of RAAS and decrease in plasma ANP as manifestation of hypovolemic state
Vecht et al.[86]	20	Long term follow-up with octreotide 25 to 200 μg per day for 37 ± 9 months	Initial relief of symptoms, persistent symptom relief in 80% after 3 months, increase of mean body weight, increase in fecal fat excretion
Arts et al.[21]	30	Octreotide 50 μg prior to OGTT	Suppression of rise in pulse rate and hematocrit, inhibition of postprandial hypoglycaemia, inhibition of rise in plasma levels of insulin, improvement of symptoms of early and late dumping syndrome
Long-acting somatostatin analogues			
Penning et al.[27]	12	Octreotide L.A.R. 10 mg I.M. every 4 weeks for 6 months	As effective as short acting Octreotide in suppressing symptoms, led to increase in body weight (open label)
Arts et al.[21]	30	Octreotide L.A.R. 20 mg I.M.	Suppression of rise in pulse rate and hematocrit, inhibition of postprandial hypoglycaemia, inhibition of rise in plasma levels of insulin, improvement of symptoms of early and late dumping syndrome and quality of life, preferred by patients over short-acting formulation
Wauters et al.[87]	24	Cross-over study with placebo or somatuline 90 mg I.M.	Improvement of symptoms of early but not late dumping syndrome

Abbreviations: ANP, atrial natriuretic peptide; GIP, gastric inhibitory peptide; I.M., intramuscular; OGTT, oral glucose modified tolerance test; RAAS, Renin-Angiotensin-Aldosteron-System; S.C., subcutaneous; VIP, vasoactive intestinal polypeptide.

investigation (Table 6). Beneficial effects of calcium antagonists on hyper-insulinemic hypoglycemia have been reported, possibly through a direct inhibitory effect on the pancreatic β-cell glucose-induced insulin release.[73,102] In a Spanish case series, partial control of PBH with the use of calcium channel blockers (nifedipine, verapamil) was obtained in a subset of patients.[76] Calcium channel blockers may also contribute to symptom control through their ability to slow gastric emptying and decrease small bowel propulsive motility.

13652982, 2025, 2, Downloaded from https://onlinelibrary.wiley.com/doi/10.1111/nmo.14962 by Capes, Wiley Online Library on [10/03/2025]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

TABLE 6 Summary of studies evaluating experimental agents in dumping syndrome.

Study	*n*	Treatment	Result
Pasireotide			
Deloose et al.[22]	9	Cross-over placebo or pasireotide 300 μg for 2 weeks	Inhibition of postprandial hypoglycaemia, slowed gastric emptying rate
Tack et al.[23]	43	3 month dose-escalation study with pasireotide 50 to 200 μg s.c. followed by extension with monthly long acting 10 or 20 mg injections	Improvement of symptoms of late and early dumping syndrome and signs on the OGTT
Øhrstrøm et al.[73]	11	Cross-over study with acarbose 50 mg 1 week, sitagliptin 100 mg 1 week, verapamil 120 mg 1 week, liraglutide 1.2 mg 3 weeks, pasireotide 300 μg single dose	Increased nadir glucose levels and reduced time in hypoglycaemia with pasireotide, during MMTT, increased peak glucose levels and time in hyperglycaemia, reduced insulin, c-peptide, GLP-1 levels
Exendin 9–39			
Craig et al.[91]	8	Cross-over placebo or Exendin 9–39 during OGTT on 2 separate days	Decreased time to peak glucose and rate of glucose decline during OGTT, normalizing postprandial nadir glucose, preventing hypoglycaemia, decreased insulin AUC and secretion rate, reduced symptoms.
Craig et al.[92]	9	Ascending dose study with Ex-9 s.c. during OGTT	All doses effectively prevented hyperinsulinaemic hypoglycaemia and reduced symptoms.
Craig et al.[93]	18	Placebo-controlled crossover study with avexitide 30 mg b.i.d or 60 mg o.d. versus placebo, each treatment given for 14 days in random order	Increased glucose nadir and decreased insulin peak after MMTT, reduction in hypoglycaemia.
Glucagon			
Laguna Sanz et al.[94]	7	Administration of 300 μg glucagon depending on CGM and hypoglycaemia prediction algorithm	Improvement in glucose time above hypoglycaemia threshold
Mulla et al.[95]	12	Placebo-controlled cross-over trial using a closed-loop glucose-responsive automated glucagon delivery system and CGM	Inhibition of postprandial hypoglycaemia after MMTT, higher nadir plasma glucose, no rebound hyperglycaemia after glucagon delivery
Nielsen et al.[96]	10	Placebo-controlled cross-over using dasiglucagon 80 μg, 200 μg or placebo	Raised nadir plasma glucose and reduced time in hypoglycaemia
Nielsen et al.[97]	24	Placebo-controlled s.c. dasiglucagon 120 μg for 4 weeks	Reduced time of hypoglycaemia using CGM
SGLT2 inhibitors			
Hepprich et al.[98]	12	Placebo-controlled cross-over using empaglifozin and anakinra versus placebo	Reduced postprandial insulin release and hypoglycaemia after MMTT with empaglifozin and anakinra
Ciudin et al.[99]	21	Canaglifozin 300 mg daily for 2 weeks	Reduction in plasma glucose levels and insulinemia after OGTT, reduction in hypoglycemia
Ferreira et al.[49]	22	Placebo-controlled cross-over using empaglifozin 25 mg for 20 days	Increased glucose excursion during MMTT, reduced hyperglycaemia on CGM but no reduction in hypoglycaemia
Amylin analogue			
Sheehan et al.[100]	20	Open label trial with Pramlintide 8 weeks	No difference in glucose, glucagon or insuline after MMTT, no change in symptoms
GLP-1 analogues			
Abrahamsson et al.[101]	5	Open label liraglutide	Elimination of symptoms
Øhrstrøm et al.[73]	11	Cross-over study with acarbose 50 mg 1 week, sitagliptin 100 mg 1 week, verapamil 120 mg 1 week, liraglutide 1.2 mg 3 weeks, pasireotide 300 μg single dose	No effect on hypoglycaemia after MMTT, decreased hyperglycaemia and glycaemic variability during continuous glucose monitoring

Abbreviations: b.i.d., twice per day; CGM, continuous glucose monitoring; I.M., intramuscular; MMTT, mixed meal tolerance test; o.d., once per day; OGTT, oral glucose modified tolerance test; S.C., subcutaneous.

13652982, 2025, 2, Downloaded from https://onlinelibrary.wiley.com/doi/10.1111/nmo.14962 by Capes, Wiley Online Library on [10/03/2025]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

Especially patients in whom dumping occurs after esophageal surgery and thus who still have an intact distal stomach including pyloric functions, seem to benefit from a slowing in gastric emptying.

Clonidine, an alpha-2 adrenergic agonist with antihypertensive properties, and buspirone, a 5-HT_{1A} agonist indicated for the treatment of anxiety disorders, are able to significantly delay gastric emptying rate and might be considered in refractory dumping syndrome patients with an intact distal stomach, although data in dumping syndrome are lacking.[103,104]

In spite of the variety of treatment approaches and mechanisms outlined above, a considerable subgroup of patients with dumping syndrome fails to respond to the treatment. In these patients, surgical re-intervention or continuous enteral feeding are treatment options that can be considered.

Most of the reports on surgical reintervention for dumping syndrome symptoms in the literature are case reports or limited size case series. Applied surgical techniques include gastric bypass reversal, gastric pouch restriction, and anti-peristaltic interposition of a jejunal loop.[105–112] In the past, hypoglycemia after gastric surgery was attributed to pancreatic nesidioblastosis, for which partial pancreatic resection was proposed.[74,113–117] Although partial pancreatectomy was used initially to reduce islet mass, recurrence of hypoglycemia was observed.[118] The concept of nesidioblastosis has now been largely abandoned.[2–4,30,119] Complications of surgical re-intervention for dumping syndrome include persisting symptoms, diabetes and weight gain.

Given the small case series in the literature, presumably including highly selected patients, and a lack of systematic follow-up with dumping provocative testing, the evidence for efficacy of surgical reintervention is limited. Hence, conservative approaches should be maximized before surgery can be considered. Moreover, dumping syndrome may spontaneously improve over time.[2–4,30]

Providing a constant relatively slow supply of nutrients through an enteral feeding tube or jejunostomy prevents the activation of the dumping syndrome cascade of events and is able to control the occurrence of hypoglycemia.[2–4,30] This is an impactful intervention, however, and the long-term evolution in patients in whom one needs to resort to this approach has not been reported in the literature.

5 | THERAPIES UNDER INVESTIGATION

5.1 | Pasireotide

Pasireotide is a multireceptor-targeted somatostatin analogue, approved for the medical therapy of acromegaly and Cushing's disease. Pasireotide has a high affinity for 4 of the 5 somatostatin receptor subtypes, and on this basis has a potential to be more effective for the treatment of dumping syndrome than the earlier studied somatostatin analogues. A pilot study showed that pasireotide improves objective markers of both early (increase in pulse rate) and late dumping syndrome (hypoglycemia), slows gastric emptying and inhibits secretion of insulin and glucagon during OGTT.[84] A phase 2 dose-escalation study of subcutaneous pasireotide followed by open label long-acting release pasireotide in dumping syndrome showed improvement of postprandial hypoglycemia, pulse rate and hematocrit and inhibition of release of insulin, glucagon, GLP-1, and GIP, representing early as well as late dumping events.[23] These effects were paralleled by improvement in symptoms and quality of life. In another study comparing different treatment options for late dumping, these effects of Pasireotide were confirmed, although also an increased time in hyperglycaemia was noted.[73] Pasireotide, subcutaneous as well as long-acting, was well tolerated. Pasireotide is currently under evaluation in a controlled multicenter trial for post-bariatric hypoglycemia (ClinicalTrials.gov ID NCT05928390).

5.2 | GLP-1 receptor antagonists

In a double-blind cross-over trial, intravenous administration of the GLP1 receptor antagonist exendin 9–39 amide was able to decrease insulin secretion, improve hypoglycemia and neuroglycopenic symptoms during OGTT in 8 patients with post-bariatric hypoglycemia (PBH).[91] No effects on early dumping were evaluated. In a follow-up study, subcutaneously administered exendin 9–39 was also able to prevent hyper-insulinaemic hypoglycaemia and improved associated symptoms during an OGTT.[92]

In a 2-week multi-center phase 2 placebo-controlled trial in 18 women with PBH, b.i.d. subcutaneous exendin 9–39 (avexitide) 30 or 60 mg improved nadir glycemia during a mixed meal test and improved the occurrence of hypoglycemic events.[93] None of the studies with exendin 9–39 evaluated the early dumping component in these patients, although there is a potential for efficacy on these symptoms.

5.3 | GLP-1 receptor agonists

Paradoxically, given the implication of GLP-1 release in the pathogenesis of dumping syndrome symptoms, beneficial effects of the GLP-1 receptor agonist liraglutide have been reported in patients with dumping syndrome.[73,101,120] In a preliminary report, 12 patients with dumping syndrome underwent oral glucose tolerance testing before and during treatment with liraglutide, showing improvement only in those who had a preserved antro-pyloric region, suggesting that the effect may at least in part be attributable to a slowing of gastric emptying.[121]

5.4 | Glucagon (analogues)

Glucagon, the endogenous pancreatic hormone that prevents hypoglycemia, has a short plasma half-life and its stability in solutions is problematic. Biotechnological advances have allowed the creation of stable glucagon solutions or derivatives. In a controlled cross-over study with

13652982, 2025, 2, Downloaded from https://onlinelibrary.wiley.com/doi/10.1111/nmo.14962 by Capes, Wiley Online Library on [10/03/2025]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

12 PBH patients, a glucose-responsive automated glucagon intravenous delivery system was able to reduce hypoglycemia and increase nadir glycemia in a mixed meal challenge test.[94,95] A pilot controlled cross-over study in 10 PBH patients demonstrated the ability of s.c. administered dasiglucagon to prevent hypoglycemia and increase nadir glycemia after a mixed meal test.[96] In a follow-up randomized, double-blind, placebo-controlled, crossover study of 2 weeks in 24 PBH patients, s.c. administration of dasiglucagon in combination with continuous glucose monitoring significantly decreased the occurrence of hypoglycemia.[97] It is likely that glucagon will not address the early dumping component, but this was not evaluated in these studies.

5.5 | SGLT2 inhibitors

In a pilot study, glucose-induced secretion of IL-1beta was implicated in the pathogenesis of postprandial hypoglycemia after gastric bypass surgery.[98] A placebo-controlled, randomized, double-blind, crossover study with the IL-1 receptor antagonist anakinra and the SGLT2-inhibitor empagliflozin in 12 patients showed that both agents were able to reduce postprandial insulin release and prevent postprandial hypoglycemia and related symptoms during a mixed meal test.[98] However, in a follow-up controlled cross-over treatment trial of 20 days empagliflozin 25 mg daily or placebo, evaluated by continuous glucose monitoring, the SGLT2 inhibitor decreased hyperglycemia time but did not affect hypoglycemic events and their duration, and the associated symptoms.[49] In another pilot study, empaglifozin 300 mg daily was given for 2 weeks to 22 patients and 5 healthy controls. OGTT with 100 g glucose was done at baseline and after the treatment, with a significant reduction of plasma glucose levels and insulinemia 30 and 60 min after the meal, and a significant reduction in the rate of hypoglycemia 180 min after the meal. The effect on symptoms was not assessed in this study.[99] Currently, a randomized clinical trial is running, comparing canagliflozin with acarbose and placebo regarding their effect on glycemia.[122]

5.6 | Amylin analogue

Amylin is a peptide, released from the pancreas, which reduces the magnitude of postprandial glycemic rise and slows gastric emptying. In an 8-week open label study with pramlintide, a stable analogue of amylin, in 20 PBH patients, no significant effect was observed on glycemic profiles, insulin, and glucagon during the mixed meal test, on symptoms and hypoglycemia on continuous glucose monitoring.[100]

6 | CONCLUSIONS

Dumping syndrome was a well-known complication from the era of peptic ulcer surgery, but is now common after surgery for gastric or esophageal malignant disease, after anti-reflux fundoplication and especially after bariatric surgical interventions. Two different sets of symptoms and signs comprise the syndrome, which are categorized as early and late dumping, respectively. Early dumping is a mixed gastrointestinal and cardiovascular symptom response to over-exposure of the small bowel to nutrients and osmotically active contents, mediated by peptide hormone release and cardiovascular reflex responses. Late dumping is characterized by hypoglycemia, mediated by exaggerated insulin release, triggered by small intestinal glucose overload in the early dumping phase.

Over the last few years, some researchers and pharmaceutical industry have focused in the latter group on "postbariatric hypoglycemia," but based on current pathophysiological concepts and analyses of provocative tests, this is merely the late component of dumping syndrome. Diagnosis relies on clinical alertness in patients with appropriate antecedents and confirmation by dumping provocative testing (OGTT or mixed meal test), or registration of spontaneous hypoglycemia. Initial treatment focuses on dietary intervention. In a next step, dietary supplementation with viscosity enhancing agents and/or acarbose can be considered. The most effective therapy, but expensive, injected and not devoid of relevant side effects, is the use of short- or long-acting somatostatin analogues. The former are most effective, but patients prefer the latter. For refractory hypoglycemia, diazoxide or SGLT2 inhibitors can be considered, although the evidence for their efficacy is very limited. Rescue therapies comprise continuous enteral feeding or various types of surgical reinterventions, both of which are poorly studied.

Therapies under evaluation include the broad-spectrum somatostatin analogue pasireotide, GLP-1 receptor antagonists, SGLT2 inhibitors and administration of stable forms of glucagon. The latter two approaches mainly target late dumping symptoms.

AUTHOR CONTRIBUTIONS

All authors: drafting of article, critical review of the text.

FUNDING INFORMATION

Jan Tack is supported by a Methusalem grant from the Onderzoeksraad, K.U.Leuven. Karlien Raymenants is supported by a PhD grant from F.W.O. (Fonds Wetenschappelijk Onderzoek) Vlaanderen.

CONFLICT OF INTEREST STATEMENT

Jan Tack has given Scientific advice to Ricordati. Jan Tack and Cedric Van de Bruaene are investigators in a controlled trial of pasireotide for dumping syndrome. The other authors have no conflict of interest.

DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

ORCID

Karlien Raymenants https://orcid.org/0000-0003-0520-4734

REFERENCES

1. Hertz AF. The cause and treatment of certain Unfavourable after-effects of gastro-enterostomy. *Proc R Soc Med*. 1913;6:155-163.

13652982, 2025, 2, Downloaded from https://onlinelibrary.wiley.com/doi/10.1111/nmo.14962 by Capes, Wiley Online Library on [10/03/2025]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

2. Tack J, Arts J, Caenepeel P, De Wulf D, Bisschops R. Pathophysiology, diagnosis and management of postoperative dumping syndrome. *Nat Rev Gastroenterol Hepatol*. 2009;6(10):583-590.
3. Tack J, Deloose E. Complications of bariatric surgery: dumping syndrome, reflux and vitamin deficiencies. *Best Pract Res Clin Gastroenterol*. 2014;28(4):741-749.
4. van Beek AP, Emous M, Laville M, Tack J. Dumping syndrome after esophageal, gastric or bariatric surgery: pathophysiology, diagnosis, and management. *Obes Rev*. 2017;18(1):68-85.
5. Farre R, Tack J. Food and symptom generation in functional gastrointestinal disorders: physiological aspects. *Am J Gastroenterol*. 2013;108(5):698-706.
6. Glasgow R, Mulvihill S. *Surgery for Peptic Ulcer Disease and Postgastrectomy Syndromes*. Fourth Edition; 2009:276-282.
7. Bufler P, Ehringhaus C, Koletzko S. Dumping syndrome: a common problem following Nissen fundoplication in young children. *Pediatr Surg Int*. 2001;17(5–6):351-355.
8. Ng DD, Ferry RJ Jr, Kelly A, Weinzimer SA, Stanley CA, Katz LE. Acarbose treatment of postprandial hypoglycemia in children after Nissen fundoplication. *J Pediatr*. 2001;139(6):877-879.
9. Raymenants K, Boeckxstaens V, Broers C, Pauwels A, Tack J. Late dumping syndrome as a complication of a Nissen fundoplication in adults.
10. Palladino AA, Sayed S, Levitt Katz LE, Gallagher PR, De León DD. Increased glucagon-like peptide-1 secretion and postprandial hypoglycemia in children after Nissen fundoplication. *J Clin Endocrinol Metab*. 2009;94(1):39-44.
11. Banerjee A, Ding Y, Mikami DJ, Needleman BJ. The role of dumping syndrome in weight loss after gastric bypass surgery. *Surg Endosc*. 2013;27(5):1573-1578.
12. Tzovaras G, Papamargaritis D, Sioka E, et al. Symptoms suggestive of dumping syndrome after provocation in patients after laparoscopic sleeve gastrectomy. *Obes Surg*. 2012;22(1):23-28.
13. Papamargaritis D, Koukoulis G, Sioka E, et al. Dumping symptoms and incidence of hypoglycaemia after provocation test at 6 and 12 months after laparoscopic sleeve gastrectomy. *Obes Surg*. 2012;22(10):1600-1606.
14. Berg P, McCallum R. Dumping syndrome: a review of the current concepts of pathophysiology, diagnosis, and treatment. *Dig Dis Sci*. 2016;61(1):11-18.
15. Baret F, Jacques J, Pioche M, et al. Evaluation of the safety profile of endoscopic pyloromyotomy by G-POEM: a French multicenter study. *Ther Adv Gastroenterol*. 2022;15:17562848221122472.
16. D'Hoedt A, Vanuytsel T. Dumping syndrome after bariatric surgery: prevalence, pathophysiology and role in weight reduction–a systematic review. *Acta Gastroenterol Belg*. 2023;86(3):417-427.
17. Ahmad A, Kornrich DB, Krasner H, et al. Prevalence of dumping syndrome after laparoscopic sleeve gastrectomy and comparison with laparoscopic roux-en-Y gastric bypass. *Obes Surg*. 2019;29(5):1506-1513.
18. Vives M, Molina A, Danús M, et al. Analysis of gastric physiology after laparoscopic sleeve gastrectomy (LSG) with or without antral preservation in relation to metabolic response: a randomised study. *Obes Surg*. 2017;27(11):2836-2844.
19. Frantzides CT, Carlson MA, Shostrom VK, et al. A survey of dumping symptomatology after gastric bypass with or without lesser omental transection. *Obes Surg*. 2011;21(2):186-193.
20. Kalarchian MA, Marcus MD, Courcoulas AP, Cheng Y, Levine MD. Self-report of gastrointestinal side effects after bariatric surgery. *Surg Obes Relat Dis*. 2014;10(6):1202-1207.
21. Arts J, Caenepeel P, Bisschops R, et al. Efficacy of the long-acting repeatable formulation of the somatostatin analogue octreotide in postoperative dumping. *Clin Gastroenterol Hepatol*. 2009;7(4):432-437.
22. Deloose E, Bisschops R, Holvoet L, et al. A pilot study of the effects of the somatostatin analog pasireotide in postoperative dumping syndrome. *Neurogastroenterol Motil*. 2014;26(6):803-809.
23. Tack J, Aberle J, Arts J, et al. Safety and efficacy of pasireotide in dumping syndrome-results from a phase 2, multicentre study. *Aliment Pharmacol Ther*. 2018;47(12):1661-1672.
24. Rossini G, Risi R, Monte L, et al. Postbariatric surgery hypoglycemia: nutritional, pharmacological and surgical perspectives. *Diabetes Metab Res Rev*. 2024;40(2):e3750.
25. Ohrstrom CC, Worm D, Kielgast UL, Holst JJ, Hansen DL. Evidence for relationship between early dumping and postprandial hypoglycemia after roux-en-Y gastric bypass. *Obes Surg*. 2020;30(3):1038-1045.
26. van Furth AM, de Heide LJM, Emous M, Veeger N, van Beek AP. Dumping syndrome and Postbariatric hypoglycemia: supporting evidence for a common etiology. *Surg Obes Relat Dis*. 2021;17(11):1912-1918.
27. Penning C, Vecht J, Masclee AA. Efficacy of depot long-acting release octreotide therapy in severe dumping syndrome. *Aliment Pharmacol Ther*. 2005;22(10):963-969.
28. Emous M, Wolffenbuttel BHR, Totte E, van Beek AP. The short- to mid-term symptom prevalence of dumping syndrome after primary gastric-bypass surgery and its impact on health-related quality of life. *Surg Obes Relat Dis*. 2017;13(9):1489-1500.
29. Nakada K, Takahashi M, Ikeda M, et al. Factors affecting the quality of life of patients after gastrectomy as assessed using the newly developed PGSAS−45 scale: a nationwide multi-institutional study. *World J Gastroenterol*. 2016;22(40):8978-8990.
30. Scarpellini E, Arts J, Karamanolis G, et al. International consensus on the diagnosis and management of dumping syndrome. *Nat Rev Endocrinol*. 2020;16(8):448-466.
31. Johnson LP, Sloop RD, Jesseph JE. Etiologic significance of the early symptomatic phase in the dumping syndrome. *Ann Surg*. 1962;156(2):173-179.
32. Sigstad H. Post-gastrectomy radiology with a physiologic contrast medium: comparison between dumpers and non-dumpers. *Br J Radiol*. 1971;44(517):37-43.
33. Simren M, Vos R, Janssens J, Tack J. Acid infusion enhances duodenal mechanosensitivity in healthy subjects. *Am J Physiol Gastrointest Liver Physiol*. 2003;285(2):G309-G315.
34. Whitehouse GH, Temple JG. The evaluation of dumping and diarrhoea after gastric surgery using a physiological test meal. *Clin Radiol*. 1977;28(2):143-149.
35. Jalleh RJ, Umapathysivam MM, Plummer MP, Deane A, Jones KL, Horowitz M. Postprandial plasma GLP-1 levels are elevated in individuals with postprandial hypoglycaemia following roux-en-Y gastric bypass–a systematic review. *Rev Endocr Metab Disord*. 2023;24(6):1075-1088.
36. Lee CJ, Clark JM, Egan JM, et al. Comparison of hormonal response to a mixed-meal challenge in hypoglycemia after sleeve gastrectomy vs gastric bypass. *J Clin Endocrinol Metab*. 2022;107(10):e4159-e4166.
37. Herzig D, Schiavon M, Tripyla A, et al. Unraveling, contributing factors to the severity of postprandial hypoglycemia after gastric bypass surgery. *Surg Obes Relat Dis*. 2023;19(5):467-472.
38. Ballantyne GH. Peptide YY(1-36) and peptide YY(3-36): part I. Distribution, release and actions. *Obes Surg*. 2006;16(5):651-658.
39. Steinert RE, Feinle-Bisset C, Asarian L, Horowitz M, Beglinger C, Geary N. Ghrelin, CCK, GLP-1, and PYY(3-36): secretory controls and physiological roles in eating and Glycemia in health, obesity, and after RYGB. *Physiol Rev*. 2017;97(1):411-463.
40. Wijma RB, Emous M, van den Broek M, Laskewitz A, Kobold ACM, van Beek AP. Prevalence and pathophysiology of early dumping in patients after primary roux-en-Y gastric bypass during a mixed-meal tolerance test. *Surg Obes Relat Dis*. 2019;15(1):73-81.

13652982, 2025, 2, Downloaded from https://onlinelibrary.wiley.com/doi/10.1111/nmo.14962 by Capes, Wiley Online Library on [10/03/2025]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

41. Sigstad H. A clinical diagnostic index in the diagnosis of the dumping syndrome. Changes in plasma volume and blood sugar after a test meal. *Acta Med Scand*. 1970;188(6):479-486.
42. Rieck JH, Heidinger H, Schlöricke E, Schmidt N, Krause S. Is Sigstad's score really capable of detecting post-surgical late dumping syndrome? *Langenbeck's Arch Surg*. 2024;409(1):94.
43. Kim SH, Liu TC, Abbasi F, et al. Plasma glucose and insulin regulation is abnormal following gastric bypass surgery with or without neuroglycopenia. *Obes Surg*. 2009;19(11):1550-1556.
44. Lev-Ran A, Anderson RW. The diagnosis of postprandial hypoglycemia. *Diabetes*. 1981;30(12):996-999.
45. Scarpellini E, Arts J, Vanuytsel T, Tack J. Reply to: late dumping syndrome or postprandial reactive hypoglycaemic syndrome after bariatric surgery. *Nat Rev Endocrinol*. 2021;17(5):317-318.
46. Emous M, Ubels FL, van Beek AP. Diagnostic tools for post-gastric bypass hypoglycaemia. *Obes Rev*. 2015;16(10):843-856.
47. Lupoli R, Lembo E, Rainone C, et al. Rate of post-bariatric hypoglycemia using continuous glucose monitoring: a meta-analysis of literature studies. *Nutr Metab Cardiovasc Dis*. 2022;32(1):32-39.
48. Ramos-Levi AM, Rubio-Herrera MA, Matia-Martin P, et al. Mixed meal tolerance test versus continuous glucose monitoring for an effective diagnosis of persistent post-bariatric hypoglycemia. *J Clin Med*. 2023;12(13):4295.
49. Ferreira A, Schonenberger KA, Potoczna N, et al. Randomized, double-blind, placebo-controlled crossover trial of once daily Empagliflozin 25 mg for the treatment of postprandial hypoglycemia after roux-en-Y gastric bypass. *Diabetes Technol Ther*. 2023;25(7):467-475.
50. Ritz P, Vaurs C, Bertrand M, Anduze Y, Guillaume E, Hanaire H. Usefulness of acarbose and dietary modifications to limit glycemic variability following roux-en-Y gastric bypass as assessed by continuous glucose monitoring. *Diabetes Technol Ther*. 2012;14(8):736-740.
51. De Cunto A, Barbi E, Minen F, Ventura A. Safety and efficacy of high-dose acarbose treatment for dumping syndrome. *J Pediatr Gastroenterol Nutr*. 2011;53(1):113-114.
52. Jenkins DJ, Gassull MA, Leeds AR, et al. Effect of dietary fiber on complications of gastric surgery: prevention of postprandial hypoglycemia by pectin. *Gastroenterology*. 1977;73(2):215-217.
53. Jenkins DJ, Jenkins AL. The clinical implications of dietary fiber. *Adv Nutr Res*. 1984;6:169-202.
54. Andersen JR, Holtug K, Uhrenholt A. Trial of pectin-enriched muffins in patients with severe dumping syndrome after gastric resection. Observations on symptoms and gastric emptying pattern. *Acta Chir Scand*. 1989;155(1):39-41.
55. Leeds AR, Ralphs DN, Ebied F, Metz G, Dilawari JB. Pectin in the dumping syndrome: reduction of symptoms and plasma volume changes. *Lancet*. 1981;1(8229):1075-1078.
56. Lawaetz O, Blackburn AM, Bloom SR, Aritas Y, Ralphs DN. Effect of pectin on gastric emptying and gut hormone release in the dumping syndrome. *Scand J Gastroenterol*. 1983;18(3):327-336.
57. Harju E, Larmi TK. Efficacy of guar gum in preventing the dumping syndrome. *JPEN J Parenter Enteral Nutr*. 1983;7(5):470-472.
58. Harju E, Heikkila J, Larmi TK. Effect of guar gum on gastric emptying after gastric resection. *JPEN J Parenter Enteral Nutr*. 1984;8(1):18-20.
59. Harju E, Nordback I. Postprandial hyperglycemia after different carbohydrates in patients with total gastrectomy. *Surg Gynecol Obstet*. 1987;165(1):41-45.
60. Kneepkens CM, Fernandes J, Vonk RJ. Dumping syndrome in children. Diagnosis and effect of glucomannan on glucose tolerance and absorption. *Acta Paediatr Scand*. 1988;77(2):279-286.
61. Snook JA, Wells AD, Prytherch DR, Evans DH, Bloom SR, Colin-Jones DG. Studies on the pathogenesis of the early dumping syndrome induced by intraduodenal instillation of hypertonic glucose. *Gut*. 1989;30(12):1716-1720.
62. Harju E, Makela J. Reduction in symptoms after proximal selective vagotomy through increased dietary viscosity. *Am J Gastroenterol*. 1984;79(11):861-863.
63. Speth PA, Jansen JB, Lamers CB. Effect of acarbose, pectin, a combination of acarbose with pectin, and placebo on postprandial reactive hypoglycaemia after gastric surgery. *Gut*. 1983;24(9):798-802.
64. Gerard J, Luyckx AS, Lefebvre PJ. Acarbose in reactive hypoglycemia: a double-blind study. *Int J Clin Pharmacol Ther Toxicol*. 1984;22(1):25-31.
65. Hasegawa T, Yoneda M, Nakamura K, et al. Long-term effect of alpha-glucosidase inhibitor on late dumping syndrome. *J Gastroenterol Hepatol*. 1998;13(12):1201-1206.
66. Lyons TJ, McLoughlin JC, Shaw C, Buchanan KD. Effect of acarbose on biochemical responses and clinical symptoms in dumping syndrome. *Digestion*. 1985;31(2–3):89-96.
67. McLoughlin JC, Buchanan KD, Alam MJ. A glycoside-hydrolase inhibitor in treatment of dumping syndrome. *Lancet*. 1979;2(8143):603-605.
68. Moreira RO, Moreira RB, Machado NA, Goncalves TB, Coutinho WF. Post-prandial hypoglycemia after bariatric surgery: pharmacological treatment with verapamil and acarbose. *Obes Surg*. 2008;18(12):1618-1621.
69. Ozgen AG, Hamulu F, Bayraktar F, et al. Long-term treatment with acarbose for the treatment of reactive hypoglycemia. *Eat Weight Disord*. 1998;3(3):136-140.
70. Valderas JP, Ahuad J, Rubio L, Escalona M, Pollak F, Maiz A. Acarbose improves hypoglycaemia following gastric bypass surgery without increasing glucagon-like peptide 1 levels. *Obes Surg*. 2012;22(4):582-586.
71. Zung A, Zadik Z. Acarbose treatment of infant dumping syndrome: extensive study of glucose dynamics and long-term follow-up. *J Pediatr Endocrinol Metab*. 2003;16(6):907-915.
72. Cadegiani FA, Silva OS. Acarbose promotes remission of both early and late dumping syndromes in post-bariatric patients. *Diabetes Metab Syndr Obes*. 2016;9:443-446.
73. Øhrstrøm CC, Worm D, Højager A, et al. Postprandial hypoglycaemia after roux-en-Y gastric bypass and the effects of acarbose, sitagliptin, verapamil, liraglutide and pasireotide. *Diabetes Obes Metab*. 2019;21(9):2142-2151.
74. Patti ME, McMahon G, Mun EC, et al. Severe hypoglycaemia post-gastric bypass requiring partial pancreatectomy: evidence for inappropriate insulin secretion and pancreatic islet hyperplasia. *Diabetologia*. 2005;48(11):2236-2240.
75. Thondam SK, Nair S, Wile D, Gill GV. Diazoxide for the treatment of hypoglycaemic dumping syndrome. *QJM*. 2013;106(9):855-858.
76. Vilarrasa N, Goday A, Rubio MA, et al. Hyperinsulinemic hypoglycemia after bariatric surgery: diagnosis and management experience from a Spanish multicenter registry. *Obes Facts*. 2016;9(1):41-51.
77. Vanuytsel T, Bisschops R, Holvoet L, et al. Diazoxide, an insulin release inhibitor, improves late hypoglycemia in dumping syndrome. *Gastroenterology*. 2013;144:S732.
78. Hopman WP, Wolberink RG, Lamers CB, Van Tongeren JH. Treatment of the dumping syndrome with the somatostatin analogue SMS 201-995. *Ann Surg*. 1988;207(2):155-159.
79. Primrose JN, Johnston D. Somatostatin analogue SMS 201-995 (octreotide) as a possible solution to the dumping syndrome after gastrectomy or vagotomy. *Br J Surg*. 1989;76(2):140-144.
80. Tulassay Z, Tulassay T, Gupta R, Cierny G. Long acting somatostatin analogue in dumping syndrome. *Br J Surg*. 1989;76(12):1294-1295.
81. Geer RJ, Richards WO, O'Dorisio TM, et al. Efficacy of octreotide acetate in treatment of severe postgastrectomy dumping syndrome. *Ann Surg*. 1990;212(6):678-687.

13652982, 2025, 2, Downloaded from https://onlinelibrary.wiley.com/doi/10.1111/nmo.14962 by Capes, Wiley Online Library on [10/03/2025]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

82. Richards WO, Geer R, O'Dorisio TM, et al. Octreotide acetate induces fasting small bowel motility in patients with dumping syndrome. *J Surg Res*. 1990;49(6):483-487.
83. Gray JL, Debas HT, Mulvihill SJ. Control of dumping symptoms by somatostatin analogue in patients after gastric surgery. *Arch Surg*. 1991;126(10):1231.
84. Hasler WL, Soudah HC, Owyang C. Mechanisms by which octreotide ameliorates symptoms in the dumping syndrome. *J Pharmacol Exp Ther*. 1996;277(3):1359-1365.
85. Vecht J, Gielkens HA, Frölich M, Lamers CB, Masclee AA. Vasoactive substances in early dumping syndrome: effects of dumping provocation with and without octreotide. *Eur J Clin Investig*. 1997;27(8):680-684.
86. Vecht J, Lamers CB, Masclee AA. Long-term results of octreotide-therapy in severe dumping syndrome. *Clin Endocrinol*. 1999;51(5):619-624.
87. Wauters L, Arts J, Caenepeel P, et al. Efficacy and safety of lanreotide in postoperative dumping syndrome: a phase II randomised and placebo-controlled study. *United European Gastroenterol J*. 2019;7(8):1064-1072.
88. Long RG, Adrian TE, Bloom SR. Somatostatin and the dumping syndrome. *Br Med J (Clin Res Ed)*. 1985;290(6472):886-888.
89. Morz R, Prager J, Pointner H. Influence of somatostatin (SS-14) on early dumping reaction in patients after partial gastrectomy. *Z Gastroenterol*. 1982;20(6):299-304.
90. Didden P, Penning C, Masclee AA. Octreotide therapy in dumping syndrome: analysis of long-term results. *Aliment Pharmacol Ther*. 2006;24(9):1367-1375.
91. Craig CM, Liu LF, Deacon CF, Holst JJ, McLaughlin TL. Critical role for GLP-1 in symptomatic post-bariatric hypoglycaemia. *Diabetologia*. 2017;60(3):531-540.
92. Craig CM, Liu LF, Nguyen T, Price C, Bingham J, McLaughlin TL. Efficacy and pharmacokinetics of subcutaneous exendin (9-39) in patients with post-bariatric hypoglycaemia. *Diabetes Obes Metab*. 2018;20(2):352-361.
93. Craig CM, Lawler HM, Lee CJE, et al. PREVENT: a randomized, placebo-controlled crossover trial of Avexitide for treatment of Postbariatric hypoglycemia. *J Clin Endocrinol Metab*. 2021;106(8):e3235-e3248.
94. Laguna Sanz AJ, Mulla CM, Fowler KM, et al. Design and clinical evaluation of a novel low-glucose prediction algorithm with mini-dose stable glucagon delivery in post-bariatric hypoglycemia. *Diabetes Technol Ther*. 2018;20(2):127-139.
95. Mulla CM, Zavitsanou S, Laguna Sanz AJ, et al. A randomized, placebo-controlled double-blind trial of a closed-loop glucagon system for postbariatric hypoglycemia. *J Clin Endocrinol Metab*. 2020;105(4):e1260-e1271.
96. Nielsen CK, Ohrstrom CC, Kielgast UL, et al. Dasiglucagon effectively mitigates Postbariatric postprandial hypoglycemia: a randomized, double-blind, placebo-controlled, crossover trial. *Diabetes Care*. 2022;45(6):1476-1481.
97. Nielsen CK, Ohrstrom CC, Houji IJK, et al. Dasiglucagon treatment for postprandial hypoglycemia after gastric bypass: a randomized, double-blind, placebo-controlled trial. *Diabetes Care*. 2023;46(12):2208-2217.
98. Hepprich M, Wiedemann SJ, Schelker BL, et al. Postprandial hypoglycemia in patients after gastric bypass surgery is mediated by glucose-induced IL-1beta. *Cell Metab*. 2020;31(4):699-709 e5.
99. Ciudin A, Sánchez M, Hernandez I, et al. Canagliflozin: a new therapeutic option in patients that present postprandial hyperinsulinemic hypoglycemia after roux-en-Y gastric bypass: a pilot study. *Obes Facts*. 2021;14(3):291-297.
100. Sheehan A, Goldfine A, Bajwa M, et al. Pramlintide for post-bariatric hypoglycaemia. *Diabetes Obes Metab*. 2022;24(6):1021-1028.
101. Abrahamsson N, Engstrom BE, Sundbom M, Karlsson FA. GLP1 analogs as treatment of postprandial hypoglycemia following gastric bypass surgery: a potential new indication? *Eur J Endocrinol*. 2013;169(6):885-889.
102. Sanke T, Nanjo K, Kondo M, Nishi M, Moriyama Y, Miyamura K. Effect of calcium antagonists on reactive hypoglycemia associated with hyperinsulinemia. *Metabolism*. 1986;35(10):924-927.
103. Tack J, Caenepeel P, Corsetti M, Janssens J. Role of tension receptors in dyspeptic patients with hypersensitivity to gastric distention. *Gastroenterology*. 2004;127(4):1058-1066.
104. Van Oudenhove L, Kindt S, Vos R, Coulie B, Tack J. Influence of buspirone on gastric sensorimotor function in man. *Aliment Pharmacol Ther*. 2008;28(11–12):1326-1333.
105. Campos GM, Ziemelis M, Paparodis R, Ahmed M, Davis DB. Laparoscopic reversal of roux-en-Y gastric bypass: technique and utility for treatment of endocrine complications. *Surg Obes Relat Dis*. 2014;10(1):36-43.
106. Carter CO, Fernandez AZ, McNatt SS, Powell MS. Conversion from gastric bypass to sleeve gastrectomy for complications of gastric bypass. *Surg Obes Relat Dis*. 2016;12(3):572-576.
107. Huang CK, Wang MY, Das SS, Chang PC. Laparoscopic conversion to loop duodenojejunal bypass with sleeve gastrectomy for intractable dumping syndrome after roux-en-Y gastric bypass-two case reports. *Obes Surg*. 2015;25(5):947.
108. Huang CK, Wang MY, Hsin MC, Chang PC. Conversion to modified duodenal switch for relieving intractable dumping syndrome and constipation after laparoscopic roux-en-Y gastric bypass. *Obes Surg*. 2015;25(5):946.
109. Lakdawala M, Limas P, Dhar S, et al. Laparoscopic revision of roux-en-Y gastric bypass to sleeve gastrectomy: a ray of hope for failed roux-en-Y gastric bypass. *Asian J Endosc Surg*. 2016;9(2):122-127.
110. Lee CJ, Brown T, Magnuson TH, Egan JM, Carlson O, Elahi D. Hormonal response to a mixed-meal challenge after reversal of gastric bypass for hypoglycemia. *J Clin Endocrinol Metab*. 2013;98(7):E1208-E1212.
111. Sawyers JL, Herrington JL Jr. Superiority of antiperistaltic jejunal segments in management of severe dumping syndrome. *Ann Surg*. 1973;178(3):311-321.
112. Vilallonga R, van de Vrande S, Himpens J. Laparoscopic reversal of roux-en-Y gastric bypass into normal anatomy with or without sleeve gastrectomy. *Surg Endosc*. 2013;27(12):4640-4648.
113. Service GJ, Thompson GB, Service FJ, Andrews JC, Collazo-Clavell ML, Lloyd RV. Hyperinsulinemic hypoglycemia with nesidioblastosis after gastric-bypass surgery. *N Engl J Med*. 2005;353(3):249-254.
114. Clancy TE, Moore FD Jr, Zinner MJ. Post-gastric bypass hyperinsulinism with nesidioblastosis: subtotal or total pancreatectomy may be needed to prevent recurrent hypoglycemia. *J Gastrointest Surg*. 2006;10(8):1116-1119.
115. Alvarez GC, Faria EN, Beck M, Girardon DT, Machado AC. Laparoscopic spleen-preserving distal pancreatectomy as treatment for nesidioblastosis after gastric bypass surgery. *Obes Surg*. 2007;17(4):550-552.
116. Mala T. Postprandial hyperinsulinemic hypoglycemia after gastric bypass surgical treatment. *Surg Obes Relat Dis*. 2014;10(6):1220-1225.
117. Mathavan VK, Arregui M, Davis C, Singh K, Patel A, Meacham J. Management of postgastric bypass noninsulinoma pancreatogenous hypoglycemia. *Surg Endosc*. 2010;24(10):2547-2555.
118. Vanderveen KA, Grant CS, Thompson GB, et al. Outcomes and quality of life after partial pancreatectomy for noninsulinoma pancreatogenous hypoglycemia from diffuse islet cell disease. *Surgery*. 2010;148(6):1237-1245.
119. Meier JJ, Butler AE, Galasso R, Butler PC. Hyperinsulinemic hypoglycemia after gastric bypass surgery is not accompanied by islet hyperplasia or increased beta-cell turnover. *Diabetes Care*. 2006;29(7):1554-1559.

13652982, 2025, 2, Downloaded from https://onlinelibrary.wiley.com/doi/10.1111/nmo.14962 by Capes, Wiley Online Library on [10/03/2025]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

120. Chiappetta S, Stier C. A case report: Liraglutide as a novel treatment option in late dumping syndrome. *Medicine (Baltimore)*. 2017;96(12):e6348.
121. Vanuytsel T, Bisschops R, Holvoet L, Deloose E, Andrews C, Tack J. Liraglutide, a Long-acting GLP-1 agonist, improves parameters of late dumping syndrome during Ogtt in patients without gastrectomy. *Gastroenterology*. 2013;144:S731.
122. Lobato CB, Winding CT, Bojsen-Møller KN, et al. Canagliflozin or acarbose versus placebo to ameliorate post-bariatric hypoglycaemia–the HypoBar I randomized clinical trial protocol. *Diabet Med*. 2024;41(6):e15320.

How to cite this article: Tack J, Raymenants K, Van de Bruaene C, Scarpellini E. Dumping syndrome: Update on pathophysiology, diagnosis, and management. *Neurogastroenterology & Motility*. 2025;37:e14962. doi:10.1111/nmo.14962

13652982, 2025, 2, Downloaded from https://onlinelibrary.wiley.com/doi/10.1111/nmo.14962 by Capes, Wiley Online Library on [10/03/2025]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License