DOI: https://doi.org/10.4038/jccp.v56i2.8109 **Short review**

Dumping syndrome following bariatric surgery: an update

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Journal of the Ceylon College of Physicians, 2025, **56**, 133-137

Abstract

Dumping syndrome refers to symptoms and signs that occur after eating in patients who have undergone gastric bypass or gastrectomy. The symptoms can also occur as complications of oesophageal surgery and, rarely, in the absence of surgery. The prevalence of dumping syndrome after bariatric surgery varies widely, ranging from about 20% to over 75% depending on the surgery type, definition, and diagnostic criteria used. There are two main presentations: early dumping and late dumping syndrome. Early dumping syndrome comprises both gastrointestinal and vasomotor symptoms, triggered by the rapid emptying of a large amount of hyperosmolar food into the small intestine, leading to rapid fluid shifts from the plasma into the bowel and excessive secretion of gastrointestinal vasoactive peptides, causing vasodilation and increased gastric motility. This phase usually occurs within the first hour of food intake. In contrast, late dumping symptoms are of reactive hypoglycaemia due to rapid absorption of glucose, causing reactive hyperinsulinemia, which usually manifests 1-3 hours following a meal. While many patients experience mild symptoms that improve with diet, between 1% and 5% may have severe symptoms. Management of dumping syndrome often needs multiple measures. However, management can be challenging in some patients and may lead to intractable symptoms. Adopting a dumping diet is the first step in the management. Those who do not respond well to dietary modification may benefit from pharmacological treatment with acarbose or somatostatin analogues. Patients resistant to dietary and pharmacological measures may be considered for surgical intervention, but the outcome of surgery is not predictable.

Key words: dumping syndrome, bariatric surgery, hypoglycaemia, management, update, pathophysiology

Introduction

Dumping syndrome refers to symptoms and signs that occur when food reaches the small intestine too rapidly,[1] a condition observed more frequently with the increasing number of bariatric procedures for extreme obesity. Although it is a well-recognised complication of bariatric surgery, severe symptoms may occur in 1-5% patients.[2] Dumping syndrome can also occur from non-surgical causes that disrupt normal regulation of gastric emptying, such as diabetic autonomic neuropathy and Zollinger-Ellison syndrome, but this is much less common. The prevalence of dumping syndrome after bariatric surgery varies widely, ranging from about 20% to over 75% depending on the surgery type, definition, and diagnostic criteria used. It is most common after Roux-en-Y Gastric Bypass (RYGB), with rates up to 75%, and less frequent after sleeve gastrectomy (SG), with rates around 15% to 40%.[3,4] Its prevalence varies with the centre the procedure performed; 31.4% in one centre in Saudi Arabia (2019-2020),[5] 26.3% in a centre in Sweden (2023-2024),[6] and 20-50 % in some centres in America.[7] There is no data on the prevalence of dumping syndrome following gastrointestinal surgeries in Sri Lanka. Symptoms of dumping syndrome are often debilitating and emotionally distressing; they are associated with a substantial reduction in quality of life, and some patients may experience intractable symptoms lifelong.[8] Although the pathophysiology is understood, management continues to be challenging and requires multiple strategies.

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Received 25 August 2025, accepted 10 November 2025.

A cascade of events explains the pathophysiology. Upper gastrointestinal surgery can reduce gastric volume, impair the function of the pyloric sphincter, or affect gastric motor function via vagal denervation. These changes lead to a more rapid delivery of food into the small bowel. The rapid transfer of a large volume of osmotically active, solid food to the small intestine can cause symptoms of dumping syndrome (Figure 1). The symptoms can be classified as early or late, depending on their onset after a meal. Early dumping occurs 15 to 60 minutes post-meal and is caused by a rapid shift of fluid into the small intestine, while late dumping occurs 1 to 3 hours after eating.[3] Early dumping syndrome appears to have a higher incidence compared to late dumping syndrome.[1]

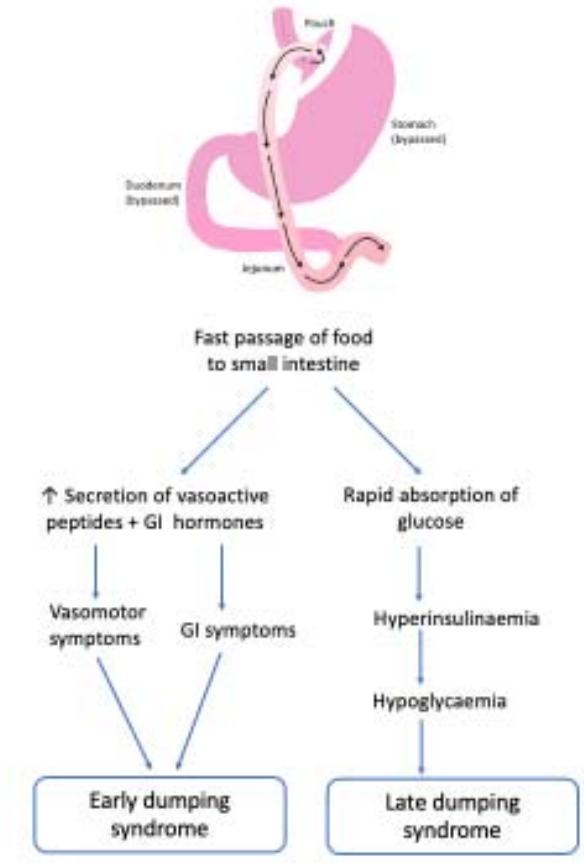

Figure 1. Pathophysiology of dumping syndrome.

Early dumping syndrome

Excessive release of intestinal and pancreatic vasoactive substances causes early dumping symptoms by triggering vasodilation, fluid shifts, and altered gut motility.[1] Symptoms mainly fall into two categories: gastrointestinal, such as borborygmi, abdominal pain, diarrhoea, bloating, nausea, and vasomotor, including flushing, palpitations, perspiration, tachycardia, hypotension, syncope, and dizziness.[9] Vasoactive intestinal peptide (VIP) and neurotensin, potent vasodilators, are responsible for vasomotor symptoms. Their increased secretion results in a rapid and significant diversion of blood flow to the splanchnic circulation, leading to a decrease in overall circulating blood volume through fluid shift and a reduction in blood pressure, which causes tachycardia, hypotension, and syncope. Serotonin, Peptide YY, and enteroglucagon-like hormones induce gastrointestinal symptoms by increasing gut motility and secretion alterations, leading to abdominal cramps, pain, diarrhoea, nausea, and bloating.[9,10]

Late dumping syndrome

Rapid absorption of glucose, leading to hyperinsulinaemia, causes symptoms of late dumping syndrome. This process is mainly driven by the exaggerated release of incretin hormones such as Glucagon-Like Peptide-1 (GLP-1) and Glucose-dependent insulinotropic polypeptide (GIP), which, in turn, cause an excessive and rapid release of insulin from the pancreas, ultimately leading to hypoglycaemia.[10] Symptoms include either adrenergic symptoms (sweating, palpitations, hunger, weakness, confusion, tremors) or neurological symptoms (diplopia, confusion, slurring speech, ataxia, seizure) of hypoglycaemia.

Diagnosis

Dumping syndrome is a clinical diagnosis. Diagnosis relies on compatible symptoms in individuals with a relevant surgical history; however, dumping physiology and symptoms can rarely be observed in people who have never undergone GI surgery, such as in autonomic neuropathy in diabetes mellitus. Because dumping syndrome is often overlooked, it should be suspected in all those with the typical systemic and abdominal symptoms alongside a suggestive surgical history.[11] There are no established guidelines for diagnosis and management of dumping syndrome; therefore, an international consensus was developed using the Delphi method,[9] Accordingly, symptom recognition is essential and the diagnosis can be confirmed by provocative testing with a modified oral glucose tolerance test.[9,10]

Different rating scales, such as the Sigstad diagnostic index or self-assessment questionnaires, can be used to identify patients with severe symptoms based on their history.[3] However, gastric emptying testing, which uses a radioactive tracer in a meal, is

not employed for diagnosis, as it has low sensitivity and specificity for dumping syndrome.[9] An increase in haematocrit >3% or in pulse rate >10 beats per minute, 30 min after start of the glucose intake during the modified oral glucose tolerance test is diagnostic of early dumping syndrome, and a nadir hypoglycaemia level <50 mg/dl is diagnostic of late dumping syndrome.[9] A mixed meal test is used to diagnose dumping syndrome, particularly late dumping syndrome, when patients experience symptoms of hypoglycaemia one to three hours after eating.[10] It helps identify the cause of symptoms by assessing the body's response to a meal containing carbohydrates, fats, and proteins, which also aids in the subsequent management of the patient.[12,13] Unlike the modified oral glucose tolerance test, which involves only a pure glucose drink,[9] the mixed meal test involves consuming a special liquid meal containing carbohydrates, fats, and proteins after an overnight fast. During the test, blood samples are taken every 30 minutes for up to 2 hours to monitor blood glucose and insulin levels, and the patient is also asked to report symptoms.[9,10]

Management

The management of dumping syndrome depends on the symptoms, as the main aim of treatment is to alleviate them. Dietary adjustment is the most essential and initial step in management and is especially effective for mild to moderate symptoms.[14,15] While patients with moderate to severe symptoms tend to consume small amounts of food, it is vital for patients with dumping syndrome to adopt a proper diet to prevent malnutrition.[11] Early dumping syndrome often resolves on its own within three months with dietary adjustments.[9,12]

Dietary management includes eating small meals, eating slowly, chewing food thoroughly, drinking fluids between meals instead of during meals, and delaying fluid intake until at least 30 minutes after eating. Limiting fluid intake with meals to half a cup at the start, then increasing fluids gradually as tolerated, is advised. However, it is recommended to drink around 1.5-2.0 litres of fluid daily to maintain adequate hydration, carefully timing it around meals. Lying down for 30 minutes after meals may help alleviate cardiovascular symptoms by slowing gastric emptying.[12,14,16] Adjusting the composition of the diet to include more proteins and complex carbohydrates while limiting rapidly absorbed simple sugars is beneficial, especially in alleviating symptoms of late dumping. Educating the patient about foods with high glycaemic indices and avoiding alcoholic beverages is needed, and referral to a clinical nutritionist would be helpful.[11] Increasing intake of high-fibre foods is advised. Edible gums such as Guar gum, pectin supplements, or Glucomannan can help relieve symptoms by increasing chyme viscosity, delaying the transit of food, and reducing peptide release.[11,12,14,17] In addition to treating dumping syndrome, to minimise nutritional deficiencies caused by reduced food intake and impaired nutrient absorption, supplementation with several vitamins and minerals is recommended. The specific regimen should be determined on an individual basis by the treating physician, based on the signs, symptoms and investigations. Generally, supplementation with multiple vitamins, calcium, and iron is indicated.[18]

People who do not respond to dietary modifications are considered for pharmacological management with acarbose or somatostatin analogues. Acarbose, an alpha-glucosidase hydrolase inhibitor, can delay the breakdown of oligosaccharides into monosaccharides in the small bowel.[15] Taken with meals, three times daily, at a dose of 50-100 mg, acarbose has been shown to reduce postprandial hyperglycaemia, reactive insulin release, and symptoms of late dumping syndrome.[9,12,19] However, side effects such as bloating, flatulence, or diarrhoea resulting from the fermentation of remaining carbohydrates in the colon may limit its use.[11] Somatostatin analogues, such as octreotide, are reserved for patients who do not respond to dietary management and acarbose.[8,20] Somatostatin can be administered alone or in combination with acarbose. They work by reducing gastric emptying rate, small bowel transit time, gastrointestinal hormone secretion, insulin secretion and vasomotor symptoms.[8] Rapid-acting octreotide is more effective than long-acting preparations, and it is more effective when administered subcutaneously about 15-20 minutes before meals. However, taking 3 to 4 injections per day and its high cost are limitations for long-term use. The main side effects of somatostatin analogues include pain at the injection site, gallstone formation and steatorrhoea.[21] Furthermore, the effects of somatostatin analogues may not be sustained for long. Therefore, long-term therapy after an initial three months is recommended for those who experience substantial symptom improvement with this treatment.

Diazoxide, a drug used to treat specific, severe, refractory hypoglycaemic conditions, which acts by inhibiting calcium-induced insulin release from the pancreas, is not usually used for the management of routine hypoglycaemias or dumping syndrome.[22] Theoretically, Glucagon-Like Peptide Receptor Agonists (GLP-1 RA) should help counteract symptoms of dumping syndrome in several ways, but mainly by delaying gastric emptying and synchronising insulin release and glucose peaks. Additionally, it suppresses

appetite, stimulates glucagon release, and reduces small bowel motility. Although there is some evidence of the benefit of GLP-1 RA in the treatment of dumping syndrome, there is insufficient data to draw firm conclusions.[23] The next drug under investigation for the treatment of dumping syndrome would be a combined GLP-1 and GIP (glucose-dependent insulinotropic polypeptide) agonist by further modulating gut hormones in addition to the effects of GLP-1.[11,23,24]

Despite all these measures, a significant number of patients do experience refractory symptoms, and surgical intervention remains the next option for them. Depending on the type of initial surgery, several procedures, including narrowing of the anastomoses and reconstruction of the pylorus, are performed. Still, these surgical interventions can be challenging and complex, and the outcomes are unpredictable.[9,11]

Failing all these measures, the rescue therapy is continuous enteral feeding through a feeding jejunostomy. Although its role in management is limited due to complications and its invasive nature, case reports show that this method can help avoid symptoms triggered by meals while providing essential nutrients for survival.[11,20]

Conclusion

Dumping syndrome occurs when food enters the small intestine rapidly, usually after procedures such as gastric bypass or gastrectomy. It manifests as early dumping with gastrointestinal and vasomotor symptoms, or late dumping with hypoglycaemia-related symptoms, depending on the timing after a meal. Diagnosis is clinical and supported by tests such as the modified oral glucose tolerance test or a mixed meal test. Management starts with dietary changes-small meals, slow eating, reduced simple sugars, and delayed fluid intake. If symptoms persist, medications such as acarbose or somatostatin analogues (e.g. octreotide) may be used, although they have limitations. In refractory cases, surgical options or continuous enteral feeding might be necessary.

Author declaration

Conflict of interests

The authors declare that they have no conflict of interests.

Criteria for authorship

CM conceptualised and drafted the paper. CM and PS revised and approved the manuscript.

Funding

None.

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