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Dumping Syndrome after Bariatric Surgery: A Systematic Review of Current Evidence

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Abstract

Importance: Dumping syndrome is a clinically relevant complication of bariatric surgery with gastrointestinal, vasomotor, and glycemic manifestations. Variability in definitions, diagnostic methods, and outcomes complicates estimates of its frequency and the establishment of standardized treatment. **Objective:** To synthesize contemporary evidence on the definition, pathophysiology, clinical manifestations, diagnosis, and treatment of dumping syndrome after bariatric surgery, emphasizing the distinction between early dumping and post bariatric hypoglycemia. **Evidence Review:** PubMed was systematically searched for original studies published from 2017 through 2026. Studies of patients undergoing bariatric procedures were eligible if they assessed pathophysiologic mechanisms, diagnostic criteria, clinical manifestations, or nutritional, pharmacologic, technology-assisted, endoscopic, or surgical interventions. The narrative synthesis included 47 original research reports. Five reviews, consensus statements, or clinical guidelines were used exclusively to contextualize the evidence and were not counted among the included studies. Heterogeneity in populations, definitions, and outcomes required narrative synthesis. **Findings:** Early dumping typically occurs within 1 hour after food intake and reflects rapid transit of hyperosmolar contents, intravascular fluid shifts, and gastrointestinal mediator release. Postbariatric hypoglycemia typically develops 2 to 4 hours after meals and involves rapid glucose absorption, excessive incretion, and hyperinsulinemia. Confirmed postbariatric hypoglycemia requires Whipple's triad: compatible symptoms with concurrent blood glu-

cose below 54 mg/dL (3.0 mmol/L) and symptom resolution after glucose correction, following exclusion of alternative causes. Mixed-meal tolerance testing has been used to characterize postprandial responses in research, and continuous glucose monitoring can reveal glycemic variability and sensor-detected glucose lows but does not replace biochemical confirmation. Nutritional modification is first-line treatment. Acarbose has the most consistent evidence among conventional medications, whereas avexitide and dasiglucagon have shown promise in short-duration trials. Continuous glucose monitoring may reduce sensor-recorded exposure to low glucose concentrations and support self-management. For refractory symptoms with a dilated gastrojejunal anastomosis, endoscopic outlet reduction is less invasive than surgery. Gastric bypass reversal carries substantial morbidity and should be reserved for carefully selected patients. **Conclusions and Relevance:** Dumping syndrome after bariatric surgery is heterogeneous, and management should be tailored to the predominant phenotype. Evidence supports a stepwise approach integrating nutritional intervention, individualized pharmacologic therapy, glucose-monitoring technologies, and endoscopic procedures before surgical reversal. Multicenter studies using standardized definitions and outcomes are needed to establish comparative effectiveness and durability.

Keywords

Dumping Syndrome, Bariatric Surgery, Roux-en-Y Gastric Bypass, Post Bariatric Hypoglycemia, Postprandial Hypoglycemia, Continuous Glucose Monitoring, Endoscopic Treatment

1. Introduction

Bariatric procedures deliberately alter gastric capacity, gastric emptying, and nutrient transit. Although these changes contribute to their therapeutic effects, they also promote the development of gastrointestinal and metabolic complications. Among these is dumping syndrome, a common but probably underdiagnosed disorder caused by the accelerated delivery of incompletely digested nutrients to the small intestine. Its clinical presentation includes early and late manifestations that may occur independently or coexist. Early dumping develops within the first hour after food intake and may include gastrointestinal symptoms, including abdominal pain, bloating, borborygmi, nausea, and diarrhea, and/or vasomotor manifestations, such as flushing, palpitations, diaphoresis, tachycardia, hypotension, and, in severe cases, syncope [1]. These symptoms are associated with rapid fluid shifts into the intestinal lumen, intestinal distention, and the release of gastrointestinal and vasoactive mediators. By contrast, late dumping generally occurs 1 to 3 hours after carbohydrate ingestion and is attributed to disproportionate incretin and insulin responses followed by hypoglycemia and autonomic or neuroglycopenic symptoms. This temporal and pathophysiologic distinction is clinically relevant because dumping syndrome is not a uniform disorder and should not be regarded

solely as synonymous with postprandial hypoglycemia [2].

The clinical relevance of dumping syndrome has increased alongside the expansion of bariatric surgery, particularly Roux-en-Y gastric bypass and sleeve gastrectomy. However, its actual frequency remains uncertain because of differences in surgical procedures, follow-up periods, measurement instruments, and diagnostic criteria. A systematic review reported a mean prevalence of early dumping of 15.7% during the first 6 months after surgery and rates of study-defined late dumping of up to 25% between 1 and 5 years after the procedure. In individual studies conducted after gastric bypass, overall estimates reached 76% when symptom-based questionnaires were used. These figures are not directly comparable because studies have used different clinical scales, provocative tests, glucose measurements, and diagnostic thresholds and have assessed early, late, or combined manifestations inconsistently [3]. Beyond its frequency, dumping syndrome may impair quality of life, lead to restrictive eating patterns, and produce nutritional, functional, and emotional consequences. Clinical recognition begins with the temporal relationship between food intake and symptoms, although available questionnaires have not been uniformly validated and provocative tests have limitations in reproducibility and interpretation. Similarly, although dietary modification is the initial intervention and pharmacologic alternatives are available for persistent cases, several diagnostic and therapeutic strategies are supported primarily by small studies, heterogeneous populations, and consensus recommendations. Evidence regarding endoscopic or surgical interventions for refractory disease is even more limited [4].

One of the main conceptual challenges is distinguishing late dumping from postbariatric hypoglycemia, terms that have been used interchangeably in parts of the literature even though they do not always describe populations defined according to the same criteria. The Society for Endocrinology guidelines propose defining postbariatric hypoglycemia as the presence of symptoms compatible with hypoglycemia after bariatric surgery, accompanied by a concurrent blood glucose concentration below 54 mg/dL (3.0 mmol/L) and symptom resolution after restoration of euglycemia, consistent with the Whipple triad and after alternative causes have been excluded. This complication typically develops 2 to 4 hours after food intake, whereas early dumping occurs sooner and does not require hypoglycemia for diagnosis. The timing windows reported for late dumping and postbariatric hypoglycemia overlap and should not be used as mutually exclusive diagnostic boundaries. Although both conditions may coexist and share accelerated intestinal nutrient exposure as an antecedent, distinguishing them prevents the indiscriminate attribution of identical mechanisms, diagnostic criteria, and treatments. The extent of this difficulty is reflected in frequencies reported using different assessment methods, which range from 25% to 75% in continuous glucose monitoring studies, from 19% to 30% with provocative testing, and from 0.1% to 0.9% in clinical registries. These differences are primarily explained by the detection method and definition used; sensor-detected or provoked glucose lows alone do not estab-

lish Whipple-confirmed postbariatric hypoglycemia [5]. In this context, the present systematic review aims to synthesize the available evidence on the epidemiology, risk factors, pathophysiology, clinical manifestations, diagnosis, and treatment of dumping syndrome after bariatric surgery, distinguishing early dumping from postbariatric hypoglycemia when permitted by the diagnostic definitions and outcome measurements reported in the original studies.

2. Methods

A systematic review of the literature indexed in PubMed/MEDLINE from January 1, 2017, through August 26, 2026, was conducted without language restrictions. The search strategy combined MeSH terms and free-text terms related to *dumping syndrome*, *early dumping*, *late dumping*, *postbariatric hypoglycemia*, *postprandial hypoglycemia*, *bariatric surgery*, *gastric bypass*, *Roux-en-Y*, *sleeve gastrectomy*, *one-anastomosis gastric bypass*, *biliopancreatic diversion*, and *duodenal switch*. Original studies of adults were eligible when dumping syndrome or postbariatric hypoglycemia was the primary focus. Eligible designs included clinical trials and observational, mechanistic, or diagnostic accuracy studies that used an explicit clinical or biochemical definition and reported sufficient data for interpretation. Uncontrolled therapeutic intervention studies, including small case series, were eligible when they met these criteria; their findings were interpreted as exploratory evidence. Pediatric and animal studies, investigations of nonbariatric gastrointestinal surgery, case reports, protocols, and publications in which the outcome was assessed only secondarily or incidentally were excluded, as were studies focused exclusively on weight loss, pregnancy, or general postoperative complications. When multiple publications originated from the same cohort and evaluated the same outcome, the most comprehensive report was retained. The search identified 96 records. The narrative synthesis included 47 original research reports. Five reviews, consensus statements, or clinical guidelines were used exclusively to contextualize the evidence and were not counted among the included studies. Data extracted from each eligible study included study design, population, bariatric procedure, diagnostic definition, duration of follow-up, outcomes, and principal findings.

For the revised synthesis, early dumping was defined as gastrointestinal and/or vasomotor symptoms within 60 minutes after meals, without requiring hypoglycemia. Late dumping denoted postprandial hypoglycemia, typically 1 - 3 hours after meals; a glucose nadir below 50 mg/dL (2.8 mmol/L) was the interpretive criterion when modified oral glucose tolerance testing was used. Confirmed postbariatric hypoglycemia required Whipple's triad: compatible symptoms, a concurrent blood glucose concentration below 54 mg/dL (3.0 mmol/L), and symptom resolution after glucose correction, following exclusion of alternative causes; episodes typically occur 2 - 4 hours after meals. Venous plasma confirmation was preferred. Timing windows overlap. Original study definitions and thresholds were retained; symptom-based findings and isolated glucose lows detected by tests or

sensors were distinguished from confirmed post bariatric hypoglycemia.

Because of clinical and methodological heterogeneity, the findings were integrated using a structured narrative synthesis.

3. Clinical Definition, Epidemiology, and Associated Factors

Estimates based on symptom questionnaires should be distinguished from postbariatric hypoglycemia confirmed by concurrent blood glucose measurement and fulfillment of Whipple's triad. In a cohort of 351 patients evaluated approximately 27 months after Roux-en-Y gastric bypass, the prevalence of moderate or severe symptoms suggestive of early dumping was 18.8%, while that of symptoms suggestive of late dumping was 11.7%. Although 20.1% reported low postprandial glucose concentrations obtained through self-monitoring, only 5.4% had required assistance from another person and 2.6% had needed medical care. Both symptom patterns were associated with poorer quality of life, greater anxiety, and more depressive symptoms, indicating that their clinical relevance is not determined solely by episode frequency [6].

Prevalence estimates also vary according to the diagnostic instrument and surgical procedure. In a longitudinal study of 220 patients, the Sigstad score identified symptoms suggestive of dumping in 33% at 6 months, including 50% of patients after bypass procedures and 25% after sleeve gastrectomy. After adjustment, bypass was associated with an approximately 3-fold higher likelihood of dumping symptoms than sleeve gastrectomy, while female sex was associated with a nearly 5-fold higher likelihood. Affected patients also had poorer mental health-related quality of life and more symptoms of anxiety or depression, although these associations do not establish causality [7]. By contrast, a questionnaire-based assessment of 122 patients after sleeve gastrectomy found mild symptoms suggestive of hypoglycemia in 65.6% and symptoms suggestive of early dumping in 41.0%, with both patterns coexisting in 33.6%. No participant reported severe symptoms, and only 1 experienced moderate hypoglycemic manifestations. Younger age and lower low-density lipoprotein cholesterol concentrations showed exploratory associations that became unstable after some adjustments and therefore cannot be regarded as confirmed predictors. These findings illustrate that a high prevalence of mild symptoms does not imply a comparable frequency of Whipple-confirmed postbariatric hypoglycemia [8].

In addition to surgical technique, preexisting characteristics may modify risk. In a cohort of 590 patients, cholecystectomy was associated with a higher frequency of dumping symptoms after primary gastric bypass, with a relative risk of 1.63. The association was stronger among patients with excess weight loss of 70% or greater, although subgroup analysis and reliance on self-reported symptoms limit the precision of this estimate. After sleeve gastrectomy, cholecystectomy was also associated with a greater risk of symptoms compatible with postbariatric hypoglycemia, but the confidence interval was wide and the finding should be considered hypothesis-generating [9]. The influence of postoperative anatomy was

similarly observed in a study of 127 patients assessed with the Sigstad score approximately 20 months after surgery. The proportion of patients with Sigstad-defined dumping symptoms was 15.6% after sleeve gastrectomy, 56.4% after Roux-en-Y gastric bypass, and 42.9% after one-anastomosis gastric bypass; however, the small group sizes and retrospective design preclude interpreting these differences as precise population-level estimates [10]. Longitudinal data from 1448 patients undergoing Roux-en-Y gastric bypass and 511 treated with gastric banding showed that hypoglycemic symptoms increased predominantly after bypass. Among patients without diabetes, the proportion increased from 2.8% before surgery to 36.4% during follow-up, whereas reported episodes requiring emergency care or hospitalization remained between 2.6% and 3.6% at 5 years. Preoperative symptoms were the most consistent predictor, and the use of serotonin or norepinephrine reuptake inhibitors was also associated with greater risk in some subgroups. Nevertheless, the questionnaire could not reliably distinguish documented hypoglycemia from other postprandial manifestations [11].

Preservation of pyloric function may partly explain the differences among surgical procedures. In a multicenter cohort of 919 patients who underwent biliopancreatic diversion with duodenal switch or single-anastomosis duodenoileal bypass with sleeve gastrectomy, dumping syndrome was reported in 7 patients during a mean follow-up of 31.5 months, corresponding to a reported incidence of 0.8%. More than half of the cases occurred during the first 6 months, and all responded to dietary modification. However, the retrospective identification of only 7 events precluded a precise assessment of associated factors [12]. The temporal course was examined in another study using 742 questionnaires administered before surgery and at different points during follow-up. After Roux-en-Y gastric bypass, the prevalence of symptoms highly suggestive of dumping increased from 4.9% before surgery to 26.3% at 5 years, while symptoms compatible with postbariatric hypoglycemia increased from 1.4% to 19.3%. The increase in hypoglycemic symptoms became apparent from the second postoperative year onward. No significant change was observed after sleeve gastrectomy, and no patient with persistent type 2 diabetes reported symptoms highly suggestive of hypoglycemia. Because the instrument was designed for screening, its results identify patients who require further evaluation but cannot replace biochemical confirmation [13].

Eating behaviors may also contribute to the clinical expression of dumping syndrome. In a cross-sectional study of 240 patients, the prevalence of dumping symptoms estimated using a modified Sigstad score was 31.4%. Women had a higher prevalence than men, and symptoms were associated with consuming large meals and drinking liquids with meals. Because the analysis was bivariate and did not adequately control for confounding, these characteristics should be interpreted as potentially modifiable factors rather than independent determinants [14]. A retrospective multicenter cohort included 952 of 1712 screened patients, all with at least 12 months of follow-up. Ascertainment relied on medical records from routine care or unscheduled clinic or emergency department visits, with data col-

lected at 12, 15, and 18 months. The study defined postbariatric hypoglycemia using clinician-documented symptoms, concurrent capillary or venous glucose of 54 mg/dL or lower, and symptom relief after carbohydrate intake. The authors reported cumulative incidences of 25.9% at 12 months and 35.4% at 18 months. Female sex, Roux-en-Y gastric bypass, and vitamin B1 or B12 deficiencies were associated with greater risk, whereas preexisting type 2 diabetes, higher glycated hemoglobin levels, and longer diabetes duration were inversely associated with risk. These findings support the existence of a susceptibility phenotype characterized by greater insulin sensitivity and preserved pancreatic β-cell function, although the nutritional associations require prospective confirmation. However, the publication lists 317 cases among 952 participants (33.3%), elsewhere mentions 1009 patients, and does not specify the denominators underlying the time-specific incidences. These reporting discrepancies and the selection of clinical records limit direct comparison with lower frequencies reported in other clinical cohorts [15]. The available evidence does not support a single prevalence estimate because surgical techniques, follow-up periods, and diagnostic criteria differ across studies. The most consistent findings indicate a higher frequency of dumping or hypoglycemic symptoms after procedures that bypass the pylorus, a progressive increase in hypoglycemic symptoms after gastric bypass, and a lower frequency among patients with persistent diabetes. Accordingly, symptom-based frequencies, self-reported glucose lows, and incidence estimates based on Whipple's triad should be interpreted as distinct epidemiologic outcomes.

4. Pathophysiology

The pathophysiology of dumping syndrome originates from the loss of anatomic and functional control over nutrient delivery to the small intestine. After Roux-en-Y gastric bypass, reduced reservoir capacity and exclusion of the pylorus promote the rapid delivery of partially digested, hyperosmolar contents to the jejunum, resulting in luminal distention and fluid shifts from the intravascular compartment. Although this osmotic mechanism accounts for some of the early gastrointestinal and vasomotor manifestations, its independent contribution appears limited. In a mixed-meal tolerance test involving 46 patients, those who developed symptoms consistent with early dumping had higher postprandial concentrations of glucagon-like peptide 1 (GLP-1) and peptide YY (PYY), without substantial differences in blood pressure, heart rate, glucose, insulin, or absolute hematocrit. These findings identify exaggerated activation of intestinal L cells as a central component of the syndrome and challenge the premise that plasma volume contraction alone can account for its full clinical expression [16].

Early dumping and postbariatric hypoglycemia represent temporally distinct manifestations arising from the same initial alterations in nutrient transit and enteroendocrine signaling, although they should not be considered clinically equivalent entities. In a cohort of 47 patients who underwent mixed-meal tolerance testing, those who developed early symptoms exhibited higher GLP-1 and PYY

concentrations during both the early and late phases of the study, together with significantly lower glucose levels at 90 and 120 minutes. The correlations among these hormones, symptom severity, and the subsequent decline in glucose support the existence of a pathophysiologic continuum in which accelerated intestinal nutrient exposure first triggers gastrointestinal and vasomotor responses and, in susceptible individuals, culminates in insulin-mediated hypoglycemia [17].

GLP-1 provides the most firmly established causal link between altered intestinal anatomy and postbariatric hypoglycemia. Its rapid and pronounced release enhances glucose-dependent insulin secretion during the initial rise in glucose, but the insulinotropic response may persist after nutrient absorption has begun to decline. In a double-blind crossover study of 8 patients, intravenous administration of exendin 9 - 39, a GLP-1 receptor antagonist, increased the postprandial glucose nadir by more than 70% and prevented hypoglycemia in all participants. Receptor blockade reduced the insulin area under the curve by 57%, decreased the insulin secretion rate by 71%, and attenuated autonomic and neuroglycopenic symptoms, providing experimental evidence that hyperactivity of this axis is not merely a marker of the disorder [18]. Subcutaneous administration of the same antagonist confirmed this mechanism in 9 women with recurrent hypoglycemia after gastric bypass. All doses evaluated prevented hyperinsulinemic hypoglycemia, increased the mean glucose nadir by 66%, reduced peak insulin concentrations by 57%, and decreased neuroglycopenic symptoms by 80%, with a relationship observed between drug exposure and metabolic response [19].

Individual susceptibility does not appear to depend exclusively on GLP-1. Proteomic analysis of patients with postbariatric hypoglycemia identified fibroblast growth factor 19 (FGF-19), an intestinal hormone regulated by bile acid signaling through the farnesoid X receptor, as the protein with the largest postprandial difference compared with asymptomatic patients who had undergone the same procedure. Its concentration at 120 minutes was 2.4-fold higher on proteomic analysis and 3.5-fold higher when validated by immunoassay. The absence of a consistent relationship between FGF-19 and pharmacologic manipulation of GLP-1 suggests that this hormone may participate in insulin-independent pathways of glucose utilization, although the observational design and sample size preclude establishing a causal relationship [20]. An interaction between the rapid rise in glucose and the inflammatory response has also been described. In a double-blind, placebo-controlled crossover trial involving 12 patients, both inhibition of sodium-glucose cotransporter 2 with empagliflozin and blockade of the interleukin 1 receptor with anakinra reduced postprandial insulin release and decreased the number of episodes requiring rescue glucose from 7 of 12 patients during placebo treatment to 2 of 12 with each intervention. The hyperreactivity observed in isolated monocytes supports a mechanism in which rapid glucose exposure induces interleukin 1β and amplifies insulin secretion, although these findings require replication in larger populations [21].

The magnitude of the decline in glucose also depends on the capacity to limit

insulin action and activate counterregulatory responses. Among 23 patients who had undergone gastric bypass, those who developed hypoglycemia during a mixed-meal tolerance test exhibited a smaller early increase in glucagon and subsequently higher concentrations of insulin, C-peptide, and GLP-1. After adjustment for hyperinsulinemia, the initial glucagon response remained associated with a higher glucose nadir, indicating that early glucagon release may protect against hypoglycemia and account for some of the variability among patients exposed to similar anatomic changes [22]. This protection may deteriorate when episodes become recurrent. In 11 women with documented postbariatric hypoglycemia, the decline in glucose during a mixed-meal tolerance test elicited only a modest increase in glucagon, while the epinephrine increase occurred late and no immediate responses in norepinephrine, pancreatic polypeptide, or cortisol were observed. The more evident activation of these hormones during greater declines in glucose suggests a shift in the counterregulatory threshold, possibly related to recurrent exposure to hypoglycemia [23].

Continuous glucose monitoring indicates that this metabolic instability is not entirely confined to the immediate postprandial period. In a comparison of 40 patients with hypoglycemia after gastric bypass and 38 healthy controls, mean glucose concentrations were similar, but the affected group had a higher coefficient of variation, a lower nadir, and more than twice as much time with glucose levels below 70 mg/dL. The difference was particularly pronounced at night, suggesting the additional involvement of changes in insulin sensitivity, hepatic glucose production, or counterregulation [24]. The heterogeneity of the disorder was also demonstrated in a longitudinal study of 63 patients evaluated before and 1 year after gastric bypass, 21 of whom developed hypoglycemia. Although GLP-1, FGF-19, and FGF-21 increased after surgery, their concentrations did not differ between patients who developed hypoglycemia and those who did not. Metabolomic analysis instead identified 20 discriminating metabolites, including carnitine and acylcholines, indicating that the emergence of the clinical phenotype depends on broader metabolic reorganization rather than on an isolated increase in intestinal hormones [25].

Metabolomics has also provided preliminary evidence that serotonin metabolism may be involved. Patients with postbariatric hypoglycemia had lower fasting concentrations of tryptophan, serotonin, and other related metabolites, whereas their plasma serotonin concentrations after a meal were 1.9-fold higher than those of asymptomatic patients. In murine models, serotonin administration reduced glucose levels and increased insulin and GLP-1 concentrations, whereas pharmacologic blockade of serotonin receptors prevented induced hypoglycemia. The combination of human and experimental evidence identifies a potentially relevant pathway, although its clinical contribution and interaction with the incretin axis still require confirmation in human intervention studies [26]. Finally, the hypothesis that hyperinsulinemia results from expansion of pancreatic β-cell mass has lost support. *In vivo* quantification using positron emission tomography with ra-

diolabeled exendin showed no difference in β-cell mass between patients with and without hypoglycemia after gastric bypass, despite lower pancreatic volume in the affected group. The available evidence therefore favors a functional disturbance of insulin secretion and its enteroendocrine, inflammatory, and counterregulatory control rather than structural proliferation of pancreatic β cells [27].

5. Clinical Presentation and Diagnostic Evaluation

5.1. Clinical Presentation and Biochemical Correlation

Postbariatric hypoglycemia usually manifests after an asymptomatic interval and is characterized by recurrent postprandial episodes, with glucose concentrations generally remaining normal during fasting. Its clinical presentation includes autonomic symptoms such as tremor, diaphoresis, hunger, palpitations, and paresthesias, as well as neuroglycopenic manifestations including weakness, visual disturbances, confusion, speech impairment, syncope, and seizures. This clinical diversity makes it difficult to distinguish postbariatric hypoglycemia from early dumping and other causes of postprandial discomfort, particularly because symptom awareness does not necessarily reflect the magnitude or frequency of glucose declines. In a pilot study of 13 patients with symptoms after gastric bypass, flash glucose monitoring combined with food and symptom records identified interstitial glucose concentrations below 54 mg/dL for at least 1% of monitored time in 8 participants. The low blood glucose index showed 100% sensitivity and specificity at a cutoff of 4.6, although these results were obtained from a small sample and still require external validation. These sensor-based findings do not independently document Whipple's triad. The same study documented reduced symptom awareness in the participant with the most frequent and severe episodes, a finding consistent with impaired awareness of hypoglycemia [28].

The temporal relationship with food intake guides the diagnosis but does not confirm it. In a cohort of 60 patients referred for symptoms attributed to hypoglycemia, 91.7% reported postprandial episodes and 78.9% described their onset as occurring 1 to 3 hours after food intake. Autonomic symptoms were present in 86.7% and neuroglycopenic symptoms in 46.7%; however, only 6 patients fulfilled the Whipple triad, and only 4 were classified as having postbariatric hypoglycemia under the study's criteria. This low concordance indicates that improvement after carbohydrate consumption is also nonspecific because it may occur in patients with postprandial symptoms without documented hypoglycemia. The evaluation should assess medication use, dietary restriction, alcohol consumption, and other disorders capable of causing hypoglycemia, particularly when episodes occur during fasting, begin shortly after surgery, or do not have a reproducible relationship with meals [29].

5.2. Diagnostic Confirmation and Test Selection

Diagnostic confirmation requires Whipple's triad: compatible symptoms accompanied by blood glucose below 54 mg/dL (3.0 mmol/L) and symptom resolution

after glucose correction, following exclusion of alternative causes [5]. Whenever possible, a venous sample should be obtained during a spontaneous event, with simultaneous measurement of glucose, insulin, and C-peptide and screening for insulin secretagogues when clinically warranted. The Society for Endocrinology guidelines advise against provocative testing for diagnosing postbariatric hypoglycemia [5]. When spontaneous biochemical confirmation cannot be obtained, mixed-meal testing may be considered selectively as an individualized specialist decision outside the diagnostic pathway recommended by that guideline. Testing requires clinical supervision, concurrent symptom assessment and blood glucose sampling, and access to hypoglycemia treatment; an isolated provoked glucose low does not establish Whipple's triad**.** Published studies have used mixed-meal tolerance tests to relate postprandial glucose concentrations to insulin responses and symptoms. In a direct comparison involving 15 patients with previously documented Whipple's triad after gastric bypass, 8 asymptomatic patients who had undergone the same procedure, and 9 nonsurgical controls, the mixed-meal tolerance test had greater sensitivity than continuous glucose monitoring for detecting glucose concentrations below 50 mg/dL—71% vs 47%—as well as greater specificity—100% vs 88%. Glycemic variability remained elevated in both surgical groups regardless of symptoms; therefore, an isolated excursion recorded by a sensor was insufficient to establish the diagnosis [30].

The diagnostic yield of these tests depends on the population studied, the glucose threshold, meal composition, and protocol duration. Among 20 patients with recurrent symptoms after gastric bypass, a mixed-meal tolerance test extended to 240 minutes detected glucose concentrations below 54 mg/dL in 60%, whereas 7 days of continuous glucose monitoring identified such concentrations in 75%. The detected glucose lows were predominantly asymptomatic. The mixed-meal tolerance test detected glucose concentrations below the study threshold in 88.9% of patients who had undergone surgery more than 3 years earlier, compared with 36.4% of those with a shorter postoperative interval; in one-quarter of cases, the glucose nadir occurred after 120 minutes. Continuous glucose monitoring revealed predominantly nocturnal and asymptomatic episodes. These detection percentages do not establish the frequency of episodes fulfilling Whipple's triad. The two methods characterized different glycemic profiles, with continuous monitoring providing information about everyday exposure and unrecognized glucose lows. Sensor accuracy decreases in the hypoglycemic range, and unexpectedly low values should be confirmed by capillary or plasma glucose measurement before diagnostic significance is assigned [31].

The pretest probability of disease should be considered before prolonged testing is requested. In a cohort of 3487 individuals who underwent bariatric surgery, 90 had hypoglycemia documented through chart review using the study's Whipple-based criteria, corresponding to a cumulative frequency of 2.6%. Of these patients, 98% had undergone gastric bypass, and the first episode occurred after a median of 40.6 months. Thirty-two patients developed severe neuroglycopenic manifes-

tations, but their presence was not consistently associated with the glucose nadir, episode frequency, or time of onset. These findings distinguish clinical severity from biochemical glucose nadirs and reinforce the need for objective documentation of events, particularly when syncope, altered mental status, or seizures occur [32]. Some routinely available parameters may help identify patients who warrant further evaluation, although they do not replace diagnostic confirmation. Among 35 patients with clinical suspicion who underwent a standardized mixed-meal tolerance test, 19 fulfilled the study's Whipple-based definition during testing, with exaggerated insulin and C-peptide responses. Test-positive participants had lower glycated hemoglobin levels and greater absolute and relative weight loss. An index calculated by dividing weight loss in kilograms by glycated hemoglobin achieved an area under the curve of 0.784, with 68% sensitivity and 94% specificity. However, its single-center derivation and the absence of a validation cohort limit its use as an independent diagnostic tool [33].

Symptom questionnaires and the classic physiologic criteria for dumping also lack sufficiently consistent concordance. In 56 patients referred for postprandial symptoms, 58 solid-meal tests tailored to postoperative nutritional recommendations were analyzed. Although all patients reported clinically relevant symptoms in everyday life, only 28% achieved a Sigstad score of 7 or higher during testing. Criteria consisting of a heart rate increase greater than 10 beats per minute and a hematocrit increase greater than 3% classified several tests as consistent with early dumping, but these changes were not associated with the symptom score. No participant had a glucose concentration below 50 mg/dL, and declines below 70 mg/dL were also unrelated to symptoms. These results question the isolated use of the Sigstad score and hemodynamic changes to establish the diagnosis, although the relatively low carbohydrate content of the test meal may have reduced its ability to provoke late hypoglycemia [34].

Discordance between symptoms and glucose concentrations is also observed outside the laboratory. Among 44 adults evaluated 1 year after gastric bypass using blinded continuous glucose monitoring and repeated symptom records over 10 days, 30 had at least 1 sensor-detected event involving an interstitial glucose concentration between 41 and 69 mg/dL, and 10 recorded episodes between 41 and 54 mg/dL. The total number of symptoms did not predict the events detected by the sensor. However, tremors, dizziness, and impaired coordination occurred more frequently during periods coinciding with low glucose concentrations, whereas common manifestations such as hunger and somnolence lacked a specific association. These findings should be interpreted cautiously because the primary analysis used a threshold below 70 mg/dL and sensor readings were not systematically confirmed using capillary glucose measurements. The reported frequencies therefore describe sensor-defined events, without establishing Whipple-confirmed postbariatric hypoglycemia. Nevertheless, the results show that some individual symptoms are more informative than the overall symptom burden [35].

6. Nutritional and Pharmacologic Management and Supportive Technologies

Treatment should be tailored to the predominant phenotype because early dumping and postbariatric hypoglycemia are related manifestations of accelerated nutrient transit but require different therapeutic targets. In early dumping, the goal is to limit the immediate gastrointestinal and vasomotor response, whereas in postbariatric hypoglycemia, treatment should attenuate the rapid rise in glucose and subsequent excessive insulin secretion. Nutritional modification is the initial intervention for preventing recurrent symptoms in both scenarios, although it should not be limited to eliminating refined carbohydrates. In a crossover trial involving 12 patients who had undergone Roux-en-Y gastric bypass, carbohydrate-rich and fat-predominant meals produced a comparable overall burden of dumping symptoms despite eliciting different glycemic and insulinemic responses. Fat caused more nausea and abdominal bloating, indicating that the overall composition of the meal should be considered when identifying triggers of early dumping [36].

For prevention of recurrent episodes in patients with postbariatric hypoglycemia, reducing carbohydrate intake while increasing protein intake can attenuate experimental postprandial glucose excursions. A crossover trial involving 10 patients found that a diet providing 30% of energy from carbohydrates and 30% from protein reduced peak glucose after breakfast and lunch, increased the glucose nadir, and limited insulin secretion compared with a conventional diet providing 55% of energy from carbohydrates and 15% from protein. These changes did not result from alterations in β-cell function or insulin clearance but from reduced glycemic and incretin stimulation and a more favorable glucagon response. These findings concern short-term glycemic and hormonal responses [37].

Acute treatment is distinct from dietary prevention. In conscious patients who can swallow safely, confirmed hypoglycemia can be treated initially with 10 - 15 g of dextrose, followed by a blood glucose check after 15 minutes and repeat dosing if hypoglycemia persists. A low-glycemic-index snack may follow recovery; sucrose should be avoided during acarbose treatment. Severe neuroglycopenia, seizures, or inability to swallow safely require urgent assistance and parenteral rescue with glucagon or intravenous glucose, according to the clinical setting [5].

Strategies for correcting low plasma glucose have also been evaluated experimentally. In a crossover trial involving 8 patients, 15 g of glucose reduced the time spent at plasma glucose concentrations below 3.9 mmol/L compared with a bar containing 10 g of protein, but produced higher glycemic and insulinemic peaks than either 5 g of glucose or the protein bar. Furthermore, 3 of the 8 administrations of 15 g of glucose were followed by another decline below 3.0 mmol/L, whereas no rebound episodes occurred with the other interventions. However, glucose remained below 3.0 mmol/L at 40 minutes in 2 participants after 5 g of glucose and 3 after the protein bar. The primary outcome, postcorrection time within the target range of 3.9 - 5.5 mmol/L, did not differ significantly between

strategies. These findings do not establish the superiority of 5 g of glucose or protein-containing foods and cannot be extrapolated to rescue treatment for severe neuroglycopenic episodes [38].

When preventive nutritional intervention does not control the episodes, pharmacotherapy may be directed toward delaying carbohydrate absorption, reducing insulin secretion, or counteracting the decline in glucose. In a crossover trial involving 11 women, acarbose and pasireotide increased the glucose nadir and reduced exposure to study-defined low glucose concentrations during a mixed-meal tolerance test. Acarbose also reduced the glucose peak, whereas pasireotide produced pronounced and sustained hyperglycemia. Sitagliptin lowered the glucose nadir, and verapamil showed no benefit. Liraglutide also did not modify hypoglycemia during the test, although it reduced hyperglycemia and glycemic variability during outpatient monitoring. None of the interventions reduced hypoglycemia exposure measured by continuous glucose monitoring. Acarbose therefore showed the most balanced experimental profile, although sustained clinical effectiveness was not demonstrated [39]. Pramlintide has also failed to produce consistent results. In an 8-week open-label study, 14 of the 20 patients who initiated treatment completed follow-up, and no changes were observed in the glucose nadir, insulin secretion, symptoms, or sensor-recorded time spent below 70 mg/dL. Tolerability varied, and 3 participants discontinued treatment because of nausea or worsening hypoglycemia, findings that do not support its routine use [40].

Interventions targeting glucagon-like peptide 1 signaling and on-demand glucagon administration have shown improvements in specific trial outcomes. In the PREVENT trial, 18 women with severe postbariatric hypoglycemia that was refractory to nutritional treatment received subcutaneous avexitide according to 2 dosing regimens. Compared with placebo, 30 mg twice daily and 60 mg once daily increased the glucose nadir by 21% and 26%, respectively, and reduced peak insulin concentrations by 23% and 21% during mixed-meal testing. Outpatient outcomes were assessed separately using self-monitored blood glucose thresholds below 70 and 54 mg/dL and severe events requiring assistance. Reductions were reported across these domains without clinically relevant hyperglycemia, although each regimen was administered for only 14 days [41]. In another double-blind, placebo-controlled crossover trial, 24 patients administered 120 μg of dasiglucagon when glucose concentrations below 3.9 mmol/L were detected by continuous monitoring and verified by self-monitored blood glucose. During 4 weeks of treatment, dasiglucagon reduced sensor-recorded time below 3.9 mmol/L by 33% and below 3.0 mmol/L by 54%. Self-monitored blood glucose increased above 3.9 mmol/L within 15 minutes after 97.3% of dasiglucagon administrations, compared with 29.1% of placebo administrations, without evidence of rebound hyperglycemia. Nausea was more frequent with dasiglucagon, and the short follow-up period precludes determining the safety and sustainability of repeated use [42].

Inhibition of sodium-glucose cotransporter 2 has produced less conclusive results. Among 22 patients treated for 20 days, empagliflozin did not modify the

peak-to-nadir excursion during a mixed-meal tolerance test or reduce sensor-recorded time spent at glucose concentrations below 3.0 mmol/L. Although it modestly reduced glycemic variability and exposure to concentrations above 10 mmol/L, these effects were not accompanied by symptom improvement [43]. The HypoBar I trial compared 4 weeks of acarbose, canagliflozin, and placebo in 11 analyzed participants. Neither active treatment reduced the primary outcome of sensor-recorded time spent below 3.9 mmol/L, although canagliflozin reduced reported symptomatic events associated with glucose lows detected by continuous monitoring in secondary analyses. Adverse events, predominantly gastrointestinal, occurred in 91% of participants during acarbose treatment and 82% during canagliflozin treatment, compared with 54% during placebo. The small sample, low baseline hypoglycemia burden, and premature termination of recruitment make these findings exploratory and do not support the routine use of sodium-glucose cotransporter 2 inhibitors [44].

Continuous glucose monitoring may have a therapeutic role by displaying the direction and rate of glycemic change, permitting earlier intervention, and facilitating the identification of triggering meals. In a study of 22 patients, access to glucose values and alarms was associated with a reduction in median sensor-recorded time spent below 60 mg/dL from 1.4% to 0.7% and the time below 70 mg/dL from 4.7% to 2.9%. It also increased the time within the range of 70 to 180 mg/dL by approximately 4 percentage points. The sequential design, use of 2 different sensor models, and absence of systematic capillary confirmation limit the causal attribution of these changes [45]. A subsequent randomized crossover trial compared 10-day periods of visible and blinded monitoring in 14 patients, including 13 after Roux-en-Y gastric bypass and 1 after Nissen fundoplication. During visible monitoring, symptom-triggered episodes without alarms and with capillary glucose below 70 mg/dL were 6-fold less frequent. Symptomatic events below 54 mg/dL and overall sensor-recorded time below the hypoglycemic thresholds did not decrease significantly. The glucose nadir increased by more than 8 mg/dL, and hypoglycemia-related concerns decreased. A greater number of glucose excursions was interpreted by the authors as evidence of dietary liberalization [46]. Despite these findings, continuous glucose monitoring does not independently establish postbariatric hypoglycemia fulfilling Whipple's triad because interstitial glucose measurement becomes less accurate in the hypoglycemic range. Its greatest utility appears to be among patients with recurrent episodes, rapid glucose declines, or impaired awareness of hypoglycemia.

Overall, the evidence supports a stepwise management approach beginning with nutritional intervention tailored to the type of dumping and a separate strategy for correcting acute episodes. Persistent hypoglycemia warrants consideration of individualized pharmacologic treatments and alert technologies, although most studies included small samples predominantly comprising women and used brief treatment periods. Treatment effects should be interpreted according to the outcome measured: symptom relief, experimental glucose responses, sensor-recorded

exposure, or blood glucose-documented episodes. Consequently, no pharmacologic option can be considered universally effective, and therapies with promising results require longer trials evaluating severe hypoglycemia, safety, quality of life, and adherence under outpatient conditions.

7. Endoscopic and Surgical Interventions for Refractory Dumping Syndrome

Anatomic interventions should be reserved for patients with disabling symptoms that persist despite nutritional and pharmacologic treatment, after confirming an association between the clinical manifestations and accelerated transit through a dilated gastrojejunal anastomosis. Transoral outlet reduction (TORe) aims to reduce the anastomotic diameter through mucosal ablation and endoscopic suturing, thereby delaying gastric pouch emptying without restoring the original gastrointestinal anatomy. In a cohort of 87 patients, 58 had dumping symptoms defined by a Sigstad score of at least 7. Symptom-resolution rates, defined as a score below 7, were 68.9%, 66.7%, and 57.2% at 6, 12, and 24 months, respectively. Among those who completed 2 years of follow-up, the median Sigstad score decreased from 15 to 2, and the early and late symptom domains of the Arts Dumping Score also improved. This benefit was accompanied by a mean total body weight loss of 8.1% at 24 months. Only 1 serious adverse event was reported, consisting of a perigastric collection treated with antibiotics. However, the absence of a control group and the availability of 2-year data for only 64.4% of the cohort limit certainty regarding the durability of the effect [47].

The usefulness of TORe for postprandial hypoglycemia was specifically evaluated in an uncontrolled series of 11 patients with symptoms refractory to dietary modification and medications. All patients reported fewer episodes attributed to hypoglycemia during a mean follow-up of 409 days, and the 3 patients with a history of seizures experienced no further seizures after the intervention. The only 3 participants who underwent continuous glucose monitoring before and after the procedure showed a mean reduction of 11.5 percentage points in sensor-recorded time spent below 70 mg/dL. No complications required hospitalization, although 1 patient developed persistent nausea and vomiting that resolved after dilation of the anastomosis. These findings are exploratory because the small sample, absence of a control group, lack of uniform outcome confirmation using Whipple's triad, and concomitant use of glucagon-like peptide 1 receptor agonists by most participants limit causal attribution and generalizability [48]. A subsequent cohort of 71 patients, including 26 treated for isolated dumping or dumping associated with weight regain, provided additional information regarding recurrence. The anastomotic diameter was reduced from approximately 28 to 9.5 mm, and 22 of the 26 patients experienced improvement in dumping symptoms. Four patients had recurrent symptoms during a mean follow-up of 27 months, and 3 required surgical revision. The overall perioperative complication rate was 11.3% and included transient dysphagia, hematemesis, and 3 perforations. Two perforations were

closed endoscopically, and 1 required laparoscopic repair. The clustering of these events early in the institution's experience suggests a clinically relevant learning curve, but the retrospective design and absence of standardized scales precluded attributing symptom improvement specifically to early dumping or Whipple-confirmed postbariatric hypoglycemia [49].

Argon plasma coagulation is a technically simpler alternative when the objective is to induce scar-mediated contraction of the anastomosis without endoscopic suturing. In a cohort of 25 patients with confirmed postprandial hypoglycemia that had persisted for at least 3 months despite pharmacologic treatment, eligibility required Whipple's triad and blood glucose below 54 mg/dL, documented by capillary measurement and/or a 240-minute mixed-meal test. A mean of 2 coagulation sessions was performed. The anastomotic diameter decreased from 26.8 to 16.4 mm, and the Sigstad score declined from 8.2 to 0.9 at 12 months. All patients reported clinical improvement, and 84% were able to discontinue pharmacotherapy during the 24-month follow-up. Follow-up included pre- and postprandial capillary glucose measurements and measurements during symptoms. Adverse events were mild and included pain, transient vomiting, minor bleeding, and excessive narrowing of the anastomosis, with none requiring hospitalization. However, the need for as many as 4 sessions, the retrospective single-center design, and the absence of endoscopic confirmation of diameter stability at the end of follow-up preclude determining whether this technique provides outcomes equivalent to full-thickness endoscopic suturing [50].

The response to TORe is not uniform and depends on both the anatomy and the predominant mechanism underlying the symptoms. In a small, uncontrolled implementation study published in 2026, 17 patients with a Sigstad score of at least 7 were selected after conservative management had failed. The procedure was completed in 15 patients and abandoned in 2 because of unfavorable anatomy. Among the 14 patients with follow-up data, 3 reported complete symptom resolution, 5 partial improvement, 2 initial resolution followed by recurrence, and 4 no improvement. Among the 5 patients with partial improvement, 4 reported episodes every 5 or 6 weeks and 1 reported a reduction from daily to weekly symptoms. Recurrence occurred at 6 and 8 months in the 2 patients with an initial response. No ulcers, strictures, or other procedure-related complications were reported. These symptom-based responses do not establish remission of Whipple-confirmed postbariatric hypoglycemia. The small sample and absence of a control group make these findings exploratory and limit causal attribution and generalizability. The variable clinical response suggests that a technically satisfactory reduction in anastomotic diameter may be insufficient for sustained symptom resolution, supporting careful endoscopic assessment and multidisciplinary patient selection [51].

Reversal of gastric bypass to normal anatomy is the most radical intervention and should be considered only after less invasive alternatives have failed. In a multicenter analysis of 48 gastric bypass reversals, dumping syndrome was the pri-

mary indication in 33.3% of cases. All procedures were initiated laparoscopically, although 8.3% required conversion to open surgery. During the first year, morbidity reached 50%, with Clavien-Dindo grade III or IV complications in 16.7%, anastomotic leakage in 8.3%, reoperation in 10.4%, and 1 postoperative death. Body mass index increased by a mean of 18%, from 23.9 to 28.2 kg/m^2, and patients who underwent reversal because of dumping experienced some of the greatest weight regain. The study did not systematically assess syndrome resolution or quality of life and therefore could not relate the substantial surgical risk to a quantifiable clinical benefit [52]. Consequently, the available evidence favors endoscopic reduction of the anastomosis before reversal is considered, provided that gastrojejunal dilation is present and other causes of the symptoms have been excluded. Surgery should be limited to carefully selected patients with severe, refractory disease and performed at centers experienced in complex revisional bariatric procedures.

8. Limitations

This review has limitations related to both the search strategy and the quality of the available evidence. The search was restricted to PubMed and to the period from 2017 through 2026; therefore, relevant studies indexed in other databases or earlier investigations that contributed to the initial understanding of the syndrome may have been excluded. The possibility of publication bias should also be considered, particularly for pharmacologic, endoscopic, and surgical interventions, whose results frequently originate from specialized centers and may favor the reporting of positive experiences. Methodologic heterogeneity precluded a reliable quantitative synthesis and required the findings to be integrated narratively.

Comparisons across studies were limited by substantial differences in the definition of the syndrome, the timing of symptom onset, and the criteria used to confirm hypoglycemia. Studies used variable glucose thresholds, nonuniform provocative tests, continuous glucose monitoring devices with different levels of accuracy, and clinical scales that did not always distinguish early dumping from postbariatric hypoglycemia. Most investigations included small samples composed predominantly of women who had undergone Roux-en-Y gastric bypass, with limited representation of sleeve gastrectomy and other bariatric procedures. In addition, many studies were retrospective, lacked a control group, or had short follow-up periods. These limitations are particularly relevant to therapeutic interventions because reductions in sensor-detected episodes were not always accompanied by plasma confirmation, symptom improvement, or fewer severe events. Consequently, estimates of effectiveness should not be interpreted as direct comparisons among treatments or uniformly extrapolated to the entire post bariatric population.

9. Conclusions

Early dumping presents gastrointestinal and/or vasomotor symptoms within 1

hour after meals, whereas post bariatric hypoglycemia typically occurs 2 to 4 hours afterward. Confirmation requires Whipple's triad: compatible symptoms, concurrent blood glucose below 54 mg/dL (3.0 mmol/L), and symptom resolution after glucose correction, following exclusion of alternative causes. Questionnaires and sensor-detected glucose lows do not independently establish this diagnosis. Mixed-meal testing has characterized postprandial responses in research.

Management should follow a stepwise approach beginning with nutritional modification, followed by individualized pharmacotherapy and monitoring support. Acarbose remains a conventional treatment option, while avexitide and dasiglucagon have promising short-term evidence. Continuous glucose monitoring may support self-management. Endoscopic anastomotic reduction may improve refractory symptoms when gastrojejunal dilation is present, although durability remains uncertain. Gastric bypass reversal should be reserved for carefully selected patients because of substantial morbidity and weight regain.

Evidence remains limited by small samples, heterogeneous definitions, and short follow-up or uncontrolled designs. Multicenter studies should use standardized definitions, distinguish symptom improvement and sensor outcomes from biochemically confirmed hypoglycemia, and assess severe events, quality of life, and treatment durability.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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