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The Gastrointestinal Side Effects of GLP-1 Therapies: Understanding the Physiology to Prevent Malnutrition

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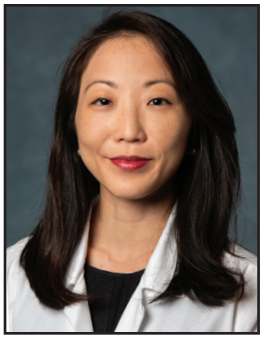

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Glucagon-like peptide-1 (GLP-1) receptor agonists (RAs) have rapidly transformed the management of type 2 diabetes and obesity. While their cardiovascular and metabolic benefits are well established, their effects on the gastrointestinal (GI) system are often underappreciated in routine care. GLP-1 RAs modulate appetite, gastric emptying, and intestinal motility, making the GI tract both the therapeutic target and the primary site of adverse events. Nausea, vomiting, gastroesophageal reflux, diarrhea, and constipation are common, and in susceptible patients these effects may progress to short-term gastroparesis, dehydration, sarcopenia, or malnutrition. This review summarizes current evidence on GLP-1 RA-induced GI effects, examines mechanisms underlying appetite suppression and dysmotility, and highlights strategies for identifying at-risk patients. Included in this review are a comparison of GLP-1 RAs tolerability, a summary of micronutrient deficiencies often found in patients receiving GLP-1 RAs, and recommendations for managing complications.

INTRODUCTION

The introduction of Glucagon-like peptide-1 (GLP-1) receptor agonists (RAs) has reshaped the therapeutic landscape of metabolic disease. Their benefits extend beyond glycemic control and include weight reduction, improved cardiometabolic risk factors, and even renal protection.[1,2] However, the enthusiasm for their use has at times overshadowed the recognition of their gastrointestinal (GI) effects. For healthcare providers, this often creates challenges in clinical practice: they are among the most effective pharmacologic tools for obesity and diabetes, but they may disrupt normal GI physiology and nutrition. Understanding the mechanisms by which GLP-1 RAs alter appetite, motility, and

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nutrient handling is critical for clinicians tasked with balancing efficacy against tolerability, especially with additional single, dual, and triple agonists in development. This article reviews the physiology of GLP-1 RAs, explores the GI side effects commonly encountered, identifies patient populations at increased risk, and outlines evidence-based management strategies to mitigate these adverse effects.

Physiological Role of GLP-1 in Appetite Regulation and Motility

Endogenous GLP-1 is primarily secreted by enteroendocrine L-cells of the distal small bowel and colon at low levels during fasting. Levels rise rapidly within minutes in response to nutrient exposure with additional modulation by neural and hormonal signals. Once released, it has a very short half-life (1-2 minutes) due to rapid degradation by dipeptidyl peptidase-4 (DPP-4).[3] GLP-1 RAs mimic the action of endogenous GLP-1, resist degradation by DPP-4, and thereby achieve extended bioavailability. At a molecular level, GLP-1 RAs exert their effects by binding to GLP-1 receptors distributed throughout the body, including the central nervous system (CNS), vagal afferents, enteric neurons, pancreatic islets, stomach, small bowel, and gallbladder.[4] Such widespread expression of receptors reflects GLP-1's diverse physiological roles including CNS control of appetite, satiety signaling, coordination of gut hormones, and delayed gastric emptying.

CNS Control of Appetite

Appetite is controlled by the brain through a balance of signals that either maintain energy needs (homeostatic feeding) or drive eating for pleasure (hedonic feeding). GLP-1 RAs influence both systems by acting on the brainstem, hypothalamus, and reward pathways.[5–8] In the brainstem, they amplify satiety signals from the gut, reinforcing post-prandial fullness. In the hypothalamus, they promote appetite-suppressing signals while reducing hunger-related messengers, shifting the balance toward reduced intake and higher energy expenditure.[5] In the mesolimbic reward system, GLP-1 RAs reduce dopamine-driven reward responses, lowering the pleasure response to high-calorie foods. Together, these actions limit overeating by curbing both physiological hunger and reward-driven eating.

Satiety Signaling

Leptin, a hormone produced by adipocytes, helps signal the brain to reduce appetite and increase energy expenditure. Although patients with obesity have higher circulating levels of leptin due to increased adipose tissue, resistance often blunts its response. GLP-1 RAs may improve leptin sensitivity, allowing the brain to respond more effectively to leptin's satiety signals leading to stronger appetite suppression.[5,9]

Gut Hormone Regulation

Gut hormones such as ghrelin, cholecystokinin (CCK), and peptide YY (PYY) are key regulators of appetite and digestion, working in coordination with GLP-1 signaling.[4–6] Ghrelin, produced primarily by the stomach during fasting, stimulates hunger, increases food intake, and promotes fat deposition. GLP-1 RAs may reduce circulating ghrelin levels, thereby decreasing hunger signals.[10] CCK is released from the small bowel in response to fat and protein intake and promotes digestion by stimulating pancreatic enzyme secretion and gallbladder contraction while slowing gastric emptying. GLP-1 RAs enhance these CCK-mediated effects, reinforcing satiety. PYY, secreted from the distal small bowel and colon after eating, also acts as a potent satiety hormone that slows gastric emptying and reduces appetite.[11] GLP-1 RAs augment the release and activity of PYY, prolonging fullness and contributing to better control of food intake.[12,13] Through the hypothesized combined modulation of these hormones, suppressing ghrelin while enhancing CCK and PYY activity, GLP-1 RAs coordinate multiple peripheral and central pathways that promote satiety and regulate energy balance.

Gastric Emptying

GLP-1 and GLP-1 RAs (dose-dependently) slow gastric emptying through coordinated effects on the smooth muscles and neural pathways of the GI tract.[4,5] They relax the gastric fundus, increase gastric compliance, inhibit antral contractility, and increase pyloric tone, all of which delay the passage of food from the stomach to the small

bowel. Activation of the vagus nerve further reduces gastric contractions and prolongs food retention in the stomach. The effects of delayed gastric emptying enhance satiety, decrease overall food intake, and contribute to the reduction of postprandial glucose elevations, supporting both glycemic control and weight management.[6]

Nutritional Risks of Therapy

Common side-effects alter nutrient intake and fluid balance

GLP-1 RAs are commonly associated with adverse GI effects. These effects include nausea, diarrhea, vomiting, constipation, and abdominal pain, with respective incidences of 25-44%, 19-30%, 8-24%, 17-24%, and 9-20% (Table 1).[14–16] Patients generally tolerate these symptoms, with discontinuation rates due to adverse effects of less than 10% in many of the clinical trials.[17] If these symptoms persist, they can impact individuals' feeding habits and thus nutrient intake along with fluid balance.

While GI side-effects are often transient, persistent symptoms can reduce oral intake, leading to unintentional nutritional deficits. In the context of a severely restricted diet, as noted in malnourished anorexia nervosa patients, there is a high prevalence of deficiencies of zinc, vitamin D, copper, selenium, vitamin B1, vitamin B12, and vitamin B9.[18] Individuals with obesity may already be deficient in vitamin D, vitamin B12, folate, zinc, iron, selenium, and thiamine, in addition to having inadequate intake of vitamins A, E, and C plus calcium and magnesium,[19,20] meaning further restriction could worsen pre-existing deficiencies. In individuals with obesity, these deficiencies are thought to result from a combination of altered metabolism, tissue distribution, and/or inadequate oral intake.[14,21] The most common micronutrients of concern for patients taking GLP-1 RAs and supplementation recommendations are found in Table 2.[22] Interestingly, while clinical trials suggest treatment emergent adverse events related to nutrient deficiencies are uncommon,[23] real-world evidence notes over 20% of patients develop nutritional deficiencies within a year of starting GLP-1 RAs.[24]

Persistent GI intolerance can further precipitate clinically significant hypovolemia and electrolyte derangements. If patients experience significant vomiting and/or diarrhea, they may develop a negative fluid and electrolyte balance. Additionally, GLP-1 RAs have been demonstrated to independently suppress water intake in rodents.[25] Poor hydration is associated with several adverse outcomes across multiple organ systems.[26] These adverse outcomes are particularly concerning for older adults, who may be at increased risk for falls and resultant serious injury in the setting of orthostatic hypotension.[27]

Therapeutic Mechanisms Further Drive Reduced Caloric Intake

Peripherally, GLP-1 RAs slow gastric emptying, leading to potential over-restriction in oral intake. At the extreme end of the spectrum, GLP-1 RAs are associated with an elevated risk of gastroparesis,[28] which is associated with an increased incidence of avoidant/restrictive food intake disorder symptoms.[29] Failing to improve oral food tolerance can lead to many of the nutritional and electrolyte disturbances previously discussed in addition to unhealthy caloric restriction.

Centrally, GLP-1 RAs lead to reduced hunger and increased satiety, further promoting reduced caloric intake. While this mechanism promotes healthy weight loss, there are cases where accelerated weight loss can negatively impact health, even with higher BMIs.[30] Various malnutrition diagnostic criteria can help identify nutritional risk with weight loss, such as consuming less than 50% of estimated needs for one week, or less than 75% of estimated needs for one month.[31] Of note, minimum energy intake recommendations for individuals taking GLP-1 medications are generally set at 1,200 kcal/day for females and 1,500 kcal/day for males with an appropriate calorie deficit of 500-1,000 kcal/day,[19,32] but because individuals with an elevated BMI may require more energy than the population on average,[33,34] they still may be at risk of malnutrition even when meeting the minimum energy intake guidelines.

Elevated Likelihood of Skeletal Muscle Wasting

Lastly, a key concern with GLP-1 RA weight loss is a loss of lean body mass. Trials suggest loss of fat-free mass, of which skeletal muscle accounts for 50% of by mass, drives 25-40% of

Table 1. Comparison of GLP-1 RA Tolerability[14–16,21]

GLP-1 RA	Brand Name(s)	Indication(s)	Route(s) of Administration	Frequency of Administration	Mechanism of Action	Incidence of Nausea
Liraglutide	Victoza® Saxenda®	T2DM, weight management	SQ	Daily (short-acting)	GLP-1	15%
Semaglutide	Ozempic® Rybelsus® Wegovy®	T2DM, weight management	PO SQ	Daily for PO, Weekly (long-acting) for SQ	GLP-1	22%
Dulaglutide	Trulicity®	T2DM	SQ	Weekly	GLP-1	10%
Exenatide	Bydureon® Byetta®	T2DM	SQ	Weekly	GLP-1	32%
Tirzapetide	Mounjaro® Zepbound®	T2DM, weight management	SQ	Weekly	GLP-1/GIP	25%

Abbreviations: T2DM, type 2 diabetes mellitus; PO, per os; SQ, subcutaneous injection; GIP, glucose-dependent insulinotrophic polypeptide

the weight loss associated with GLP-1 RAs.[35] The literature is heterogenous related to if these body changes are substantially different from lifestyle measures or bariatric surgery.[36] Nonetheless, given a lesser percentage reduction in fat-free mass with some lifestyle measures, this has led to claims of treatment-induced "frailty" that have yet to be consistently documented.[30]

Identifying At-Risk Patients

Not all patients are equally vulnerable to nutritional complications. Those at highest risk include:[37]

- Older adults and frail patients with low muscle mass and increased risk of sarcopenia
- Patients with pre-existing gastroparesis, motility disorders, or dyspepsia in whom GLP-1 RAs may further disrupt motility
- Post-bariatric surgery patients, already predisposed to malabsorption and restricted intake. Given many post-op patients are often prescribed GLPs if there is weight recurrence or lack of response, surgical candidacy for patients with nutrient deficiencies or poor intake at baseline should be carefully reviewed
- Patients with a history of foregut surgery given increased risk for vagal nerve dysfunction
- Patients with chronic kidney disease (CKD) or congestive heart failure (CHF), for whom dehydration from vomiting or diarrhea is poorly tolerated. Given management of these co-morbidities can benefit from GLP-1s, these benefits should be balanced against the risks of nutritional complications.
- Patients with cancer whose protein and energy needs are elevated while appetite is often suppressed (of note, GLP-1 RAs are contraindicated in patients with medullary thyroid carcinoma and/or MEN2)
- Patients on opioids, anticholinergics, and/or diuretics, which can compound GLP-1–related motility slowing or fluid loss
- Patients with a current or past history of eating disorders

Management Strategies

Clinical evaluation in conjunction with multidisciplinary care is imperative to exclude alternative causes of symptoms and optimize management in patients experiencing unwanted GI adverse effects. Management requires a combination of patient education, dietary modification, pharmacologic support, and dose

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adjustment.[38,39] If weight loss is greater than expected, clinicians should rule out secondary etiologies including endocrine, psychiatric, and oncological disorders. Weight loss that exceeds expectations may also be related to nutrition knowledge deficits and a lack of awareness of food intake. In some cases, patients are not attuned to hunger cues and may require intentional reminders to consume regular meals. Other contributing factors include unrealistic weight loss expectations and the presence of disordered eating behaviors or eating disorders.

Of note, the multidisciplinary team should include a registered dietitian (RD) specializing in weight management. Dietitians can support patients with individualized nutrition and lifestyle strategies to help minimize symptoms, support weight loss, and promote sustainable, long-term eating habits. In the clinical trials for both semaglutide and tirzepatide, nutrition counseling by a nutrition expert was provided.

Nutrition and Hydration

For patients experiencing post-prandial symptoms and/or loss of appetite, adaptation of gastroparesis dietary strategies is recommended (Table 3).[19,40] Patients may benefit from small, frequent, low-fat meals to minimize gastric stasis. Liquids and soft-textured foods are often better tolerated than solids. Protein intake should be emphasized to help preserve muscle mass and protein powders, shakes, or bars considered for those unable to meet daily targets. Keeping a food diary may be useful in identifying foods or meal timings that exacerbate symptoms. In severe cases, medication should be stopped.

In those with diarrhea, avoidance of dairy products, laxative juices, coffee, alcoholic drinks, high-sugar drinks and products with sweeteners ending in "ol" (sorbitol, mannitol, xylitol, maltitol) can be helpful with the addition of soluble fiber. Monitoring urine output and orthostatic symptoms can also help detect early dehydration. Encourage structured fluid goals of 1.5–2 L/day, adjusted for

Table 2. Micronutrient Deficiencies Associated with GLP-1 RA, Obesity and a Restricted Diet[22]

Nutrient Deficiency	Common Supplementation
Calcium	Chronic deficiency: 1.2-2.4 g daily in split doses Prophylaxis: 1.2-1.5 g daily in split doses
Iron	Deficiency: 150-300 mg elemental iron two to three times daily
Vitamin A	Severe Deficiency: 100,000 units for 3 days, then 50,000 units daily for 2 weeks, then 10,000-20,000 units daily for 2 months
Vitamin B1 (thiamine)	Treatment: 100-500 mg daily for 5-7 days Prophylaxis: 100 mg daily
Vitamin B9 (folate)	Maintenance: 0.4 mg daily Pregnancy: 0.8 mg daily
Vitamin B12	Treatment: 1,000 mcg SQ daily for 7days, then every 7 days for 4-8 weeks, then 1,000 mcg monthly for life Prophylaxis: 1,000 mcg by mouth daily
Vitamin C	Treatment: 200 mg IV at 33 mg/min daily for 7 days. Oral (off-label): 1 to 2 g daily for 2 days, then 500 mg daily for 1 week.
Vitamin D	Deficiency in people without obesity: 50,000 IU weekly for 8-12 weeks followed by 1500-2000 IU daily maintenance dose Deficiency in individuals with obesity: 6000-10,000 IU daily for 8 weeks followed by 3000-6000 IU daily maintenance dose Prophylaxis (off-label): 1000-2000 IU daily
Vitamin E	Treatment: 15-25 IU daily
Zinc	Treatment: 0.5-1 mg/kg/day elemental zinc daily (up to 60 mg two to three times daily)

*Oral doses unless otherwise stated
Abbreviations: SQ, subcutaneous injection; IU, international units; IV, intravenous

comorbidities (i.e., CKD/CHF). Oral rehydration solutions are useful for those with vomiting or diarrhea. Isotonic drinks (i.e., sports drinks) should be avoided.

Combining medication use with exercise may mitigate the effects of reduced muscle mass.[41] As such, in addition to nutritional interventions focusing on protein content, patients may benefit from starting an exercise program when initiating these medications.

Furthermore, a well-balanced high quality (e.g., nutrient rich) diet can prevent some adverse effects of GLP-1 RAs. There is also the consideration of micronutrient supplementation like a multivitamin when clinically indicated and/or the addition of fiber, both in its soluble and insoluble forms.[30]

Pharmacological Support

Short courses of antiemetics can control nausea. Ondansetron is effective in reducing nausea but also slows down motility and may worsen constipation. Other antiemetics (i.e., promethazine, aprepitant, scopolamine, trimethobenzamide) can also be considered depending on availability, versus prokinetics such as metoclopramide or erythromycin. Laxatives or soluble fiber supplementation can be effective for constipation, whereas anti-diarrheal medications such as loperamide can be used for diarrhea.

Dose Adjustment

GI adverse events are most common during dose escalation. Extending each titration step by two to four weeks often helps with tolerability. If symptoms persist despite the measures above, temporary dose reductions or drug holidays (1-2 weeks off therapy) allow symptoms to resolve before cautiously restarting at a lower dose. For

Table 3. Dietary Recommendations for GLP-1 RA-Related GERD and Gastroparesis[19,40]

	GERD	Gastroparesis
Overall Aim	Reduce reflux and discomfort without compromising overall intake.	Reduce nausea, early satiety, and fullness while preserving calories and protein.
Meal Pattern	Smaller, more frequent meals; avoid very large evening meals or "feast" days.	Small, frequent meals or snacks (about 5–6 per day); avoid large portions at any one time.
Texture/Particle Size	Encourage thorough chewing; avoid very large, tough or dry pieces of food.	Prefer soft, small-particle foods (minced, mashed, puréed, soups, smoothies) to ease gastric emptying.
Fat Content	Limit very high-fat meals (fried foods, heavy cream sauces, fast food), especially at night.	Choose low-fat options at each meal; use small amounts of added fats spread across the day.
Fiber	If symptoms worsen with big salads or very coarse fiber, trial more cooked/soft vegetables and fruits.	Limit coarse, high-fiber foods (large salads, peels, nuts, seeds); choose cooked, peeled, soft fruits and vegetables.
Hydration	Promote regular fluids; avoid very large volumes with meals and reflux-trigger beverages (carbonated, very acidic, or alcoholic drinks).	Take frequent small sips of non-carbonated fluids through the day; consider small volumes of oral nutrition drinks if solid intake is poor.
Protein Focus	Lean, softer proteins (fish, eggs, yogurt, tofu, ground meats) to support satiety and weight maintenance.	Soft, easily digested proteins (eggs, yogurt, milk or protein shakes, soft tofu, tender fish); spread protein across multiple small meals.
Position/Timing & GLP-1 Dosing Days	Stay upright after meals and avoid lying down within 3–4 hours of eating; be extra cautious with meal size and fat content on injection days. Walk for 15 minutes after eating.	Remain upright after meals; consider gentler, softer, lower-fat meals on GLP-1 injection days and during dose increases when symptoms peak.

Abbreviation: GERD, gastroesophageal reflux disease

refractory cases, switching to a more tolerable agent can be considered. If nutritional compromise persists, discontinuation is warranted to prevent complications from malnutrition.

CONCLUSIONS

GLP-1 RAs are among the most effective therapies for diabetes and obesity, but their GI side effects may compromise nutrition and hydration. Their physiological mechanisms, central satiety and delayed gastric emptying, are the same forces driving intolerance. For healthcare providers, recognizing at-risk patients, applying nutrition-centered management, and engaging in multidisciplinary care are the keys to balancing efficacy with safety. ■

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Answers to this month's crossword puzzle:

1 D	I	2 G	E	3 S	T	4 I	V	5 E	S	Y	S	6 T	E	7 M
I	■	A	■	T	■	T	■	E	■	■	■	W	■	O
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E	■	E	■	O	■	H	■	■	■	I	■	■	■	E
11 R	E	S	12 U	L	13 T	■	14 A	B	S	C	O	15 P	A	L
T	■	■	S	■	O	■	G	■	■	■	■	E	■	■
I	■	16 R	E	17 M	O	18 V	E	19 D	■	20 H	E	R	B	S
C	■	E	■	A	■	O	■	I	■	I	■	I	■	■
21 U	22 N	C	O	N	F	I	R	M	23 E	D	■	24 S	A	25 P
26 L	E	O	■	O	■	D	■	■	27 M	E	28 A	T	■	Y
I	■	29 V	I	M	■	■	30 K	31 U	S	S	M	A	U	L
32 T	33 I	E	■	E	■	■	■	S	■	■	■	L	■	O
34 I	R	R	I	T	A	35 B	L	E	■	36 L	A	S	E	R
37 S	E	E	■	R	■	A	■	U	■	E	■	I	■	I
■	■	38 D	R	Y	■	39 H	O	P	K	I	N	S	■	■