

Small Bowel Obstruction Temporally Associated With an Unsupervised Transition From Tirzepatide to Semaglutide After a Prolonged Treatment Interruption: A Case Report

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Sandy Khair¹, Bola Habeb¹

1. Internal Medicine, Ascension Sacred Heart, Pensacola, USA

Corresponding author: Bola Habeb, bolahabeb89@gmail.com

Abstract

Glucagon-like peptide-1 receptor agonists (GLP-1 RAs) are increasingly used for metabolic management, but their effects on gastrointestinal motility raise important considerations regarding treatment interruption and re-initiation. Resuming therapy at a previously used dose or transitioning between agents without gradual dose escalation may increase the risk of gastrointestinal adverse events. We describe a case of acute small bowel obstruction in a patient without diabetes who developed abdominal pain and obstructive symptoms 10 days after independently initiating semaglutide at 1 mg following a three-month interruption in tirzepatide therapy. Imaging demonstrated dilated small-bowel loops with air-fluid levels and a discrete transition point, without an identifiable hernia or mass. The obstruction resolved with conservative management, including nasogastric decompression. Although the temporal relationship raises concern for a potential contribution from GLP-1 RA-associated gastrointestinal dysmotility, the presence of a prior abdominal surgical history and a discrete transition point limits definitive attribution to medication exposure alone.

Categories: Pharmacology, Internal Medicine, Gastroenterology

Keywords: adverse-drug reaction, glucagon-like peptide-1 receptor agonist (glp-1 ra), incretin-based therapy, misuse of semaglutide, obesity and weight loss, physician collaboration or supervision, small bowel obstruction (sbo)

Introduction

GLP-1 receptor agonists (GLP-1 RAs) and dual glucose-dependent insulintropic polypeptide (GIP)/GLP-1 RAs are established pharmacologic therapies for obesity and weight management. Semaglutide and tirzepatide produce substantial weight loss in adults with overweight or obesity, while gastrointestinal adverse effects are among the most frequent treatment-related events [1,2]. The gastrointestinal effects of these agents extend beyond symptoms and include delayed gastric emptying and alterations in intestinal motility [3]. Small bowel obstruction (SBO), a condition characterized by impaired passage of intestinal contents through the small intestine, can lead to significant morbidity and may require urgent intervention. Whether GLP-1-based therapies increase the risk of clinically significant ileus or SBO remains uncertain. A recent systematic review and meta-analysis found no statistically significant overall increase in bowel obstruction or ileus among patients receiving GLP-1 RAs, although heterogeneity among studies limits certainty [4].

Individual cases of clinically significant intestinal dysmotility and obstruction have nevertheless been reported. Javed et al. described semaglutide-associated small bowel pseudo-obstruction and ileitis, while Shimada et al. reported functional SBO temporally associated with tirzepatide in a surgically naïve patient [5,6]. Semaglutide is normally initiated at 0.25 mg weekly and escalated at four-week intervals to improve gastrointestinal tolerability [7-9]. Following a prolonged treatment interruption, resuming therapy at a previously tolerated dose may not be appropriate for every patient, and the optimal reinitiation strategy depends on the clinical context. The prescribing information for semaglutide allows reinitiation with dose escalation after multiple missed doses to reduce gastrointestinal adverse reactions [9]. However, evidence guiding the reinitiation of semaglutide after a prolonged interruption of a different incretin-based therapy, particularly following a transition from tirzepatide, remains limited. Expert guidance on switching between GLP-1 RAs emphasizes individualized dose selection and gradual escalation based on the prior agent, treatment duration, and gastrointestinal tolerability [8].

The increasing availability of GLP-1-based therapies through diverse prescribing and dispensing pathways has also raised safety concerns regarding medication use without adequate clinical supervision, including inappropriate dosing and titration [10]. Such concerns are particularly relevant when patients independently change agents or resume treatment after a prolonged interruption without reassessment of the appropriate starting dose.

We report a case of SBO occurring approximately 10 days after unsupervised administration of semaglutide 1

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mg following a three-month interruption of tirzepatide therapy. Unlike previously reported cases of GLP-1-associated pseudo-obstruction or functional obstruction, this case demonstrated a discrete radiographic transition point with distal decompression, followed by complete resolution with conservative management and no persistent structural lesion identified. The case highlights the diagnostic uncertainty surrounding SBO in the setting of incretin-based therapy and underscores the importance of supervised medication transitions and individualized dosing after prolonged treatment interruptions. Although the temporal association and pharmacological plausibility raise the possibility of a medication-related contribution, a causal relationship cannot be established.

Case Presentation

A 57-year-old woman with a medical history of anxiety and a prior cerebrovascular accident treated with thrombolytic therapy approximately four years earlier, without residual neurologic deficits, presented to the emergency department with a five-hour history of acute abdominal pain accompanied by persistent nausea and nonbilious, nonbloody vomiting.

The patient had been receiving tirzepatide subcutaneously once weekly for approximately one year for weight management, with the dose gradually titrated to 12.5 mg weekly. During this period, her BMI decreased from 36.8 kg/m² at treatment initiation to 32.4 kg/m² at presentation, representing an approximately 12% reduction. She independently discontinued tirzepatide approximately three months before presentation and remained off incretin-based therapy during this interval. Approximately 10 days before presentation, a friend provided her with semaglutide (Wegovy) 1 mg, which she self-administered as a single subcutaneous injection without clinician supervision or dose titration following the prolonged treatment interruption. Within the days following the injection, she developed vague, dull, generalized abdominal discomfort that persisted over the following days. Her symptoms acutely worsened the night before presentation, with persistent nausea and multiple episodes of nonbilious, nonbloody vomiting over approximately five hours.

Her surgical history was notable only for a remote laparoscopic cholecystectomy performed approximately 20 years earlier. She had no history of previous SBO, inflammatory bowel disease, abdominal malignancy, or known abdominal hernia. She denied tobacco, alcohol, and illicit drug use. Her home medications included aspirin 81 mg daily and escitalopram 5 mg daily. She denied over-the-counter medications, herbal supplements, or other nonprescription agents.

Clinical findings

On presentation, she was afebrile and hemodynamically stable, with a temperature of 36.8°C, blood pressure of 122/82 mmHg, heart rate of 96 beats/min, respiratory rate of 16 breaths/min, and oxygen saturation of 97% on room air. She appeared uncomfortable but was in no acute cardiopulmonary distress. The abdomen was soft with generalized tenderness and hypoactive bowel sounds, without significant distention, guarding, rebound tenderness, or palpable masses. No clinically evident abdominal hernia was present.

Diagnostic assessment

Laboratory results on admission are demonstrated in Table 1.

Parameters	Patient's values on admission	Reference range, adults
Hemoglobin (g/dL)	11.8	12.0–15.5
Hematocrit (%)	35.4	34.9–44.5
White cell count (×10 ³ /μL)	5400	3500–10500
Platelet count (×10 ³ /μL)	398,000	150,000–450,000
Sodium (mEq/dL)	141	135–145
Potassium (mEq/dL)	4	3.5–5.1
Bicarbonate (mEq/dL)	24	22–29
Blood urea nitrogen (mg/dL)	14	12–21
Creatinine (mg/dL)	0.66	0.7–1.2
Calcium (mg/dL)	9.4	8.4–10.8
Lactate (mmol/L)	1.6	0.5–2.2
Alkaline phosphatase (units/L)	109	55–142
Aspartate aminotransferase (units/L)	32	5–34
Alanine aminotransferase (units/L)	34	11–55
Total bilirubin (mg/dL)	0.6	0.1–1.1
Lipase (units/L)*	1077	0.0–64
Triglycerides (mg/dL)*	176	<150
TSH (mIU/mL)	2.32	0.35–4.94
Glucose (mg/dL)	105	70–105
Hemoglobin A1c (%)	5.3	≤6.5

TABLE 1: Laboratory data obtained on admission.
*Abnormal laboratory values.

A computed tomography (CT) scan of the abdomen and pelvis with intravenous contrast demonstrated multiple dilated small-bowel loops throughout the left abdomen, measuring up to 3.6 cm, with multiple air-fluid levels. A transition point was identified in the left lower quadrant with distal small-bowel decompression, consistent with SBO (Figure 1). No hernia, mass, or other apparent structural cause was identified. The pancreas appeared normal without peripancreatic inflammatory changes or other CT findings suggestive of acute pancreatitis.

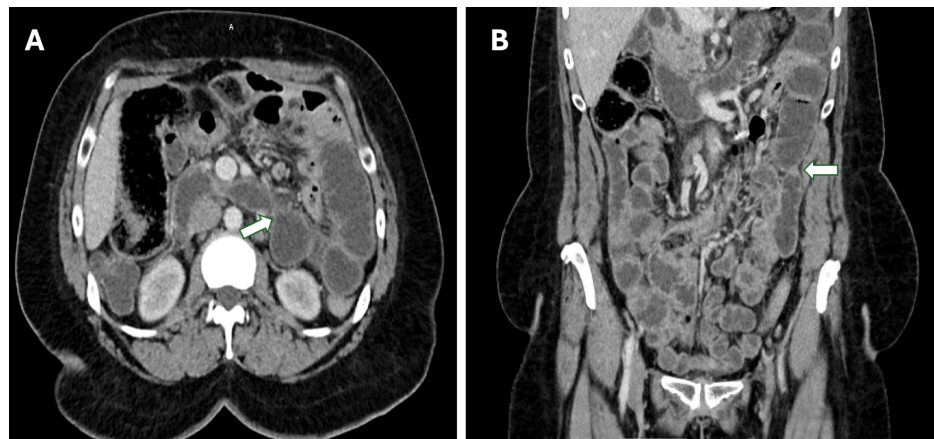


FIGURE 1: Contrast-enhanced CT of the abdomen and pelvis demonstrating small-bowel obstruction. (A) Axial and (B) coronal images show multiple dilated, fluid-filled small-bowel loops with an abrupt transition point in the left lower quadrant (arrows), associated with decompression of the distal small bowel.

Management

The patient was admitted for conservative management. A nasogastric tube was placed for decompression, initially yielding approximately 480 mL of bilious output, and she was maintained nil per os. Intravenous fluids were administered for hydration. The surgical service recommended continued nonoperative management with nasogastric decompression and close monitoring.

On hospital day 3, she had a bowel movement, and small-bowel follow-through demonstrated resolution of the obstruction. Sequential imaging showed no persistent stricture, nodularity, mucosal thickening, or transition zone. No abnormal separation of bowel loops was identified; the terminal ileum was unremarkable, and small-bowel transit time was approximately one hour (Figure 2).



FIGURE 2: Small bowel follow-through demonstrating resolution of the small bowel obstruction, without a persistent transition zone or other apparent structural abnormality, with normal small-bowel transit time.

The nasogastric tube was removed following return of bowel function, and oral intake was gradually resumed without recurrent nausea, vomiting, or abdominal pain. The lipase level decreased to 294 U/L. She was discharged home on hospital day 4. All GLP-1-based therapies were discontinued, and she was advised not to resume these medications without clinician-directed evaluation and appropriate dose titration. At the eight-week follow-up, she remained asymptomatic, with no recurrence of abdominal pain, nausea, vomiting, or other symptoms suggestive of recurrent bowel obstruction.

The overall clinical course, including prior tirzepatide therapy, treatment interruption, subsequent unsupervised semaglutide administration, symptom onset and progression, diagnostic evaluation, conservative management, resolution of the obstruction, and eight-week follow-up, is summarized in Figure 3.

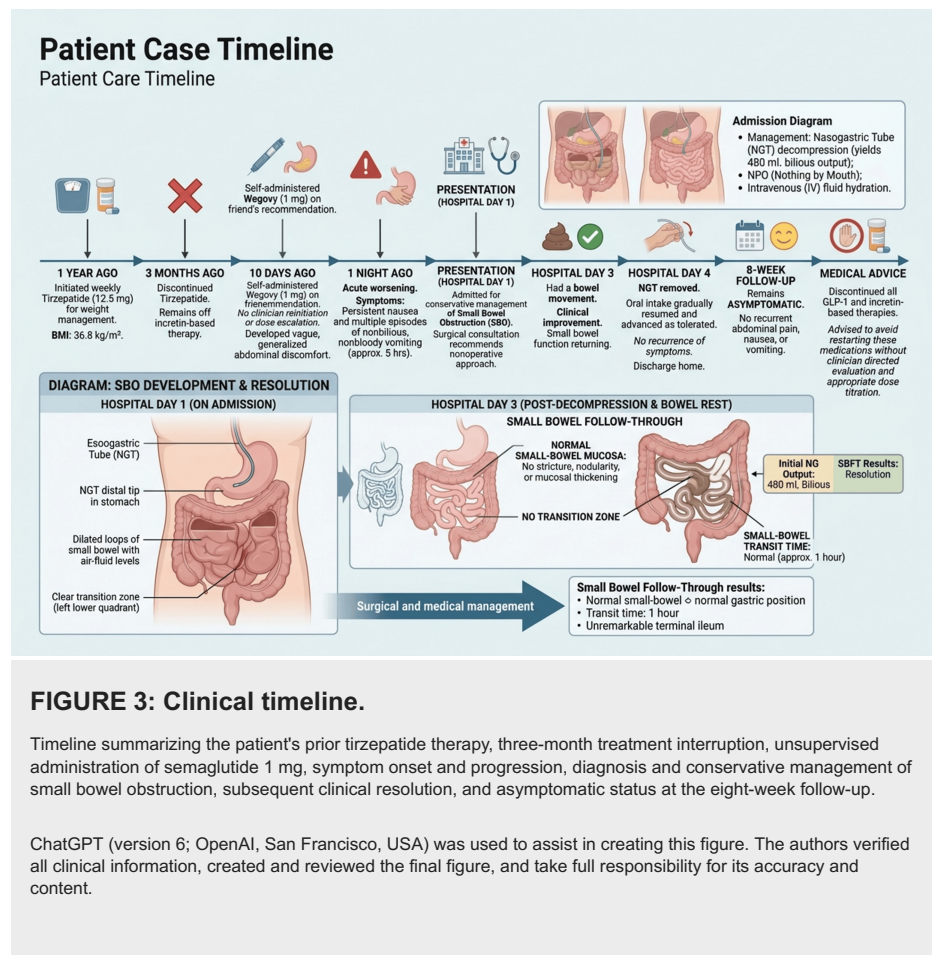


FIGURE 3: Clinical timeline.

Timeline summarizing the patient's prior tirzepatide therapy, three-month treatment interruption, unsupervised administration of semaglutide 1 mg, symptom onset and progression, diagnosis and conservative management of small bowel obstruction, subsequent clinical resolution, and asymptomatic status at the eight-week follow-up.

ChatGPT (version 6; OpenAI, San Francisco, USA) was used to assist in creating this figure. The authors verified all clinical information, created and reviewed the final figure, and take full responsibility for its accuracy and content.

Discussion

This case describes transient CT-confirmed SBO approximately 10 days after unsupervised administration of semaglutide 1 mg following a three-month interruption of incretin-based therapy. The patient had previously tolerated tirzepatide 12.5 mg weekly for approximately one year, but semaglutide was subsequently administered at 1 mg as the first dose after treatment interruption. CT demonstrated dilated small-bowel loops, air-fluid levels, an abrupt transition point, and distal decompression, establishing an obstructive pattern rather than uncomplicated ileus. However, no fixed structural lesion was identified; the transition zone was absent on subsequent follow-through, and the obstruction resolved with conservative treatment.

Gastrointestinal effects of incretin-based therapy

GLP-1 receptor activation affects gastrointestinal motility, with delayed gastric emptying being the best-characterized effect and additional effects on intestinal motor activity and transit described in the literature [3,11]. Gastrointestinal adverse events, particularly nausea, vomiting, constipation, and abdominal discomfort, are common with semaglutide and tirzepatide, especially during treatment initiation and dose escalation. The prescribing information for semaglutide (Wegovy) also warns of more serious gastrointestinal complications, including acute pancreatitis, ileus, intestinal obstruction, and severe constipation, including fecal impaction [1,2,7]. These effects provide biologic plausibility for altered intestinal transit contributing to gastrointestinal symptoms or functional obstructive physiology, but they do not establish a causal relationship with mechanical SBO.

Mechanical obstruction versus dysmotility

The imaging findings support SBO rather than uncomplicated ileus. Multiple dilated small-bowel loops, air-fluid levels, an abrupt transition point, and distal decompression were present. These findings are important because the established gastrointestinal effects of GLP-1 RAs would more readily be expected to produce delayed transit or functional obstructive physiology than a discrete mechanical transition point.

Nevertheless, the transition point did not correspond to an identifiable fixed structural lesion. No hernia, mass, bowel wall thickening, stricture, or other obstructing lesion was identified on CT, and subsequent small-bowel follow-through demonstrated resolution without a persistent transition zone or structural abnormality. The episode may therefore have represented a transient mechanical obstruction related to an

intermittent adhesion, transient angulation, or focal narrowing. Alternatively, altered intestinal motility associated with incretin therapy could have contributed to impaired transit or functional obstruction. The latter mechanism remains speculative.

The remote laparoscopic cholecystectomy provides an additional possible adhesive etiology. Adhesive SBO can occur after laparoscopic abdominal surgery, although the risk is lower after laparoscopic than open cholecystectomy [12]. In a population-level analysis of more than 1.6 million patients, the five-year incidence of adhesive SBO after cholecystectomy was 0.65% after laparoscopic surgery versus 2.2% after open surgery, with the greatest risk occurring during the first two years [13]. Because adhesions are not reliably visualized on CT, an intermittent adhesive process cannot be excluded. However, the remote timing of surgery, absence of a persistent transition zone or fixed lesion, and spontaneous resolution prevent confirmation of an adhesive cause.

Treatment interruption and unsupervised transition

The patient received semaglutide 1 mg as her first dose after approximately three months without incretin exposure. This was substantially above the recommended starting dose of 0.25 mg weekly for Wegovy, which is increased at four-week intervals to improve gastrointestinal tolerability [9]. Although previous tolerance of tirzepatide 12.5 mg may have influenced the patient's perception of the appropriate dose, tolerability of one incretin-based agent does not establish an equivalent starting dose for another agent after prolonged treatment interruption.

A potential, although unproven, explanation is loss of gastrointestinal adaptation during the prolonged interruption. Some gastrointestinal effects of GLP-1 receptor stimulation, particularly slowing of gastric emptying, demonstrate tachyphylaxis with sustained exposure [11,14]. After several months without therapy, this adaptation may no longer have been present to the same degree. Re-exposure to an incretin-based agent at a relatively high starting dose could therefore produce more pronounced gastrointestinal effects than gradual escalation. This remains a hypothesis, as there are no established clinical data demonstrating that treatment interruption increases intestinal sensitivity or specifically predisposes to SBO.

The timing of symptom onset is pharmacologically relevant. Subcutaneous semaglutide reaches maximum plasma concentration approximately 1-3 days after administration and has an apparent elimination half-life of approximately one week [9]. Therefore, although the patient's presentation approximately 10 days after injection occurred beyond the expected time to peak concentration, clinically meaningful drug exposure would be expected to persist. This temporal association, together with the potential for gastrointestinal adverse effects, provides pharmacological plausibility for a possible contribution of semaglutide to the clinical presentation. However, causality cannot be established from the temporal relationship alone, particularly given the confirmed SBO as an alternative explanation for the patient's symptoms. The improvement observed with conservative management is consistent with resolution of the obstruction but does not independently establish or exclude a medication-related contribution. Accordingly, semaglutide may have contributed to the clinical presentation, but a causal relationship cannot be confirmed.

Relationship to existing literature

Population-level evidence has not established a class-wide increase in SBO or ileus with GLP-1 RAs. A recent systematic review and meta-analysis found no statistically significant overall increase, although substantial heterogeneity limited certainty [4]. Individual reports remain informative but cannot establish causality. Javed et al. described semaglutide-associated small bowel pseudo-obstruction and ileitis, whereas Shimada et al. reported functional SBO following tirzepatide [5,6]. The present case differs from those reports because CT demonstrated a transition point with distal decompression, while follow-up imaging showed no persistent structural abnormality. The case therefore lies between classic mechanical SBO and functional dysmotility and should be interpreted accordingly.

Elevated lipase

The admission lipase level was 1,077 U/L, approximately 17 times the upper limit of normal, prompting consideration of acute pancreatitis. However, the available clinical and radiographic findings did not establish this diagnosis. Although the patient presented with generalized abdominal pain, nausea, and vomiting, these symptoms occurred in the setting of a confirmed SBO, which provided an alternative explanation for the clinical presentation. Furthermore, CT imaging demonstrated SBO without pancreatic enlargement, peripancreatic inflammatory changes, or other findings suggestive of acute pancreatitis. According to the revised Atlanta classification, the diagnosis of acute pancreatitis requires at least two of three criteria: characteristic abdominal pain, serum lipase or amylase levels at least three times the upper limit of normal, and characteristic imaging findings [15]. In this case, although the biochemical criterion was met, the available evidence was insufficient to establish the other diagnostic criteria. The lipase level subsequently decreased to 294 U/L as the obstruction resolved with conservative management, further supporting an association between the elevated lipase level and the obstructive process, although this temporal relationship does not establish causality.

Elevated pancreatic enzymes may occur in nonpancreatic abdominal conditions, including SBO. In a retrospective cohort of 344 patients with SBO, 38 patients (11%) had elevated lipase levels after patients with radiographic evidence of pancreatitis were excluded [16]. Proposed mechanisms include increased intraluminal pressure, altered splanchnic circulation, and intestinal inflammation. Semaglutide may also increase pancreatic enzyme concentrations, with the Wegovy prescribing information reporting a mean increase in serum lipase of approximately 39% [9]. However, the magnitude of elevation in this patient substantially exceeded the average treatment-associated increase reported in clinical trials. The temporal decline in lipase alongside clinical improvement is compatible with a transient process but does not establish whether the elevation resulted from SBO, semaglutide exposure, or another mechanism. Accordingly, the marked lipase elevation warrants acknowledgment but should not, in isolation, be interpreted as evidence of acute pancreatitis.

Limitations

The primary limitation of this report is its single-case design, which limits the ability to establish a causal relationship between semaglutide exposure and SBO. Although the temporal relationship between unsupervised semaglutide administration, symptom onset, and subsequent resolution after discontinuation suggests a possible medication-related contribution, causality cannot be confirmed, and an unrelated transient obstructive event cannot be excluded. The lack of a persistent transition zone or structural abnormality on follow-up imaging also limits determination of the underlying mechanism. Semaglutide rechallenge was not performed because of potential clinical risk, and the eight-week follow-up period limits assessment of longer-term recurrence. Additionally, the marked lipase elevation represents a potential confounding finding, although the clinical and radiographic evaluation did not support acute pancreatitis and elevated lipase has been reported in the setting of SBO.

Conclusions

This case describes CT-confirmed SBO occurring approximately 10 days after unsupervised administration of semaglutide 1 mg following a three-month interruption of incretin-based therapy. Although the temporal relationship and subsequent resolution raise the possibility of a medication-associated gastrointestinal contribution, the transition point means that a transient mechanical obstruction, including an intermittent adhesive process related to prior abdominal surgery, cannot be excluded. The case therefore does not establish semaglutide as the cause of the obstruction. It highlights a medication-safety concern associated with unsupervised transition to a different incretin-based agent at a noninitial dose following prolonged treatment interruption. Clinician-directed dose selection and gradual escalation remain important when restarting or transitioning incretin-based therapy.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Bola Habeb, Sandy Khair

Acquisition, analysis, or interpretation of data: Bola Habeb, Sandy Khair

Drafting of the manuscript: Bola Habeb, Sandy Khair

Critical review of the manuscript for important intellectual content: Bola Habeb, Sandy Khair

Supervision: Bola Habeb

Disclosures

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