GLP-1 Agonists and Gastroparesis: Ozempic Initiation in a Case of Chronic GI Issues

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Gastroparesis is a chronic disorder involving delayed emptying of the stomach after a meal. Symptoms include nausea, vomiting, belching, bloating, and early satiety. Glucagon-like peptide-1 (GLP-1) receptor agonists function like the incretin hormone GLP-1 that is released into the intestine after consumption of food. GLP-1 agonists can serve as pharmacologic agents to treat type 2 diabetes, as they increase insulin sensitivity, decrease glucagon secretion, and slow gastric emptying. At this time, there is inconclusive data about the association between gastroparesis and GLP-1 medications. We report a patient with a history of numerous abdominal surgeries and prior bowel obstructions who presented with gastric outlet obstruction after starting Ozempic 5 weeks prior.

A 68 year old female presented to the ED with epigastric abdominal tenderness for 2 days and 3 episodes of non-bloody emesis of undigested food after eating breakfast. Prior to this, she reported doing well aside from worsening reflux and 1 episode of emesis. She has a history of coronary artery disease, CVA, diabetes mellitus, peptic ulcer disease, small bowel obstruction, abdominal hernias, gastrectomy, gastric bypass, and a 30 pack-year smoking history. Patient denied hematemesis, bloody stool, melena, or diarrhea. She mentioned starting Ozempic 5 weeks earlier with previous ability to tolerate it well. On physical exam, she had mild abdominal distension and tenderness in the epigastric area. Labs were unremarkable upon admission. Surgery was consulted for admission and gastroenterology was consulted for CT findings of a large volume of gastric contents and mildly distended loops of fluid and gas-filled small bowel. Placement of a nasogastric tube for gastric decompression was planned but unsuccessful due to anatomic difficulty and patient refusal. EGD was performed showing a large amount of food in the stomach but no gastric outlet obstruction with ability to visualize the jejunum. While in the hospital, the patient was given supportive care, including intravenous fluids and antiemetics in addition to holding her Ozempic dosing. Patient reported having bowel movements and no further nausea or vomiting. She was ultimately discharged following toleration of a regular diet.

The recent initiation of Ozempic in this patient can raise the question of whether her gastroparesis was medication induced. There is no significant literature that demonstrates a link between GLP-1 medications and gastroparesis. The most common side effects of GLP-1 agonists are diarrhea, nausea, and vomiting. Clinical trials have shown that these adverse gastrointestinal reactions can be alleviated by starting patients on a low dosage and gradually increasing the dose to allow patients to develop tolerance. Additionally, hyperglycemia can slow gastric emptying, with gastroparesis most commonly seen in patients with a 10+ year history of diabetes. Fundoplication and bariatric surgeries can also cause gastroparesis due to entrapment of the vagus nerve. In this patient, with a significant history of multiple abdominal surgeries, bowel obstructions, diabetes, and impaired healing due to a significant smoking history it remains unclear if Ozempic was the direct cause of her slowed gastric emptying. However, as GLP-1 agonists are more frequently used this is something that needs further research and should be considered in cases of otherwise unexplained delayed gastric emptying.