A Surprisingly Under-Recognized Cause of Gastroparesis

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Abstract
Untreated hypothyroidism is an uncommon cause of gastroparesis that manifests with abdominal pain, nausea and vomiting that can mimic gastrointestinal obstruction, especially in a patient with extensive bowel surgery. This article details the workup that led to the diagnosis of hypothyroid-induced gastroparesis, and subsequent management of a patient who presented with the aforementioned symptoms, along with a complicated history of bowel resection, total parental nutrition (TPN) use, and multiple systemic infections.

Introduction
Thyroid hormone status affects gastrointestinal function. Gastroparesis secondary to a hypothyroid state can evoke non-specific symptoms including nausea and weight gain. An extensive history of gastrointestinal surgeries and associated complications made this case of hypothyroid-induced gastroparesis especially challenging.

CASE REPORT
Prior Hospital Course:
A 63-year-old female was referred from a nearby hospital and admitted to our gastroenterology service at University Medical Center for severe refractory nausea and vomiting that had persisted for several days following successful treatment of central line-associated sepsisemia in the referring hospital.

Past Medical and Surgical History:
This patient had a colectomy with ileostomy eight years ago for colonic inertia that had not responded to sigmoidectomy. A later attempt to reverse the ileostomy failed. Ensuing enterocutaneous fistulas necessitated multiple small bowel resections that left her with twelve inches of small bowel exteriorized to a jejunostomy ring and bag. Consequent development of short bowel syndrome rendered her TPN-dependent, for which a vascular catheter and port were placed. Chronic central line use resulted in several subsequent bouts of sepsisemia.

A gastric emptying study was performed to ascertain whether short bowel syndrome could entirely explain her large stool volume. It showed dramatically rapid gastric emptying with 53% emptied by 30 minutes, (normal: <30% emptied by 30 minutes) 82% emptied by 1 hour, (normal: <60% emptied by 1 hour) and 97% by 2 hours (normal: <80% emptied by 2 hours). Inadequate bioavailability of oral thyroxine with such rapid transit along just twelve inches of bowel necessitated parenteral administration.

Admission to Our Service:
On admission to our service, her condition was stable with nasogastric tube in place. Despite mucosal signs of dehydration, she was alert, oriented, and her vital signs were unremarkable. The stoma and the skin surrounding the jejunostomy appeared healthy. She had multiple abdominal surgical scars. Review of systems was positive for nausea, vomiting and intermittent non-radiating epigastric abdominal pain that was not associated with meals. Labs: BUN 68 mg/dL, creatinine 1.6 mg/dL attributable to emesis and dehydration, and ALP 937 units/L, AST 87 units/L, ALT 164 units/L were consistent with a cholestasis secondary to three years of TPN-dependence.

Investigations:
Suspicion of small bowel obstruction based on clinical presentation and the history of extensive bowel surgery prompted an upper GI series, which ruled out obstruction [Figure 1]. Thyroid function: TSH 151 mIU/L (normal range 0.5-5.0 mIU/L) and free thyroxine 0.29 ng/dL (normal range 0.7-1.9 ng/dL). That prompted consultation with the endocrinology service. It was discovered from those investigations after admission to UMC that thyroxine replacement per IV TPN line had not been continued at the referring hospital.

Management:
IV Synthroid was re-instituted to correct the marked thyroid hormone deficiency. TPN continued with vitamin and fluid volume supplementation. Teduglutide [GatteRx®], a newly approved therapy for short bowel syndrome, was also initiated. Resumption of IV Synthroid (dose: 100 mcg daily) normalized thyroid function and resulted in substantial clinical improvement. Increased oral fluid intake contributed to cessation of nausea and vomiting. She was discharged home eight days after admission.

Follow Up:
At a five week outpatient follow up visit, she reported that mild nausea was well controlled with sublingual odansetron [Zofran®]. Symptoms of hypothyroidism have not returned since parenteral thyroxine replacement was instituted. She was eating her usual diet and maintaining weight. Follow up thyroid function tests: TSH 0.16 uIU/mL (nl: 0.35-5.50) and fT4 1.8 ng/dL (nl: 0.80-2.00).

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Figure 1: This upper GI series shows normal emptying of the contrast from the stomach into the duodenum and proximal small bowel. In addition, there is no evidence of small bowel obstruction. Rods can be seen in the thoracic vertebrae from previous spinal surgery. A spinal cord stimulation, which is addressing ongoing back pain, is also pictured.

0.9-1.8 ng/dL), adequately approximate euthyroid status. In the absence of concerning gastroparetic symptoms, a repeat gastric emptying test was unwarranted.

DISCUSSION

Hypothyroidism is defined by subnormal serum free T4 concentration with feedback increased pituitary serum thyroid stimulating hormone secretion. Typical clinical features of hypothyroidism include fatigue, cold intolerance, dry skin, coarse brittle hair & nails, pretibial myxedema, hoarseness, weight gain, and constipation. Reports of persistent nausea and vomiting are uncommon, and reports of hypothyroid-induced gastroparesis are rare. Those nonspecific symptoms can confound a diagnosis. TSH and free T4 are important to narrow in on the diagnosis.

After the confirmatory thyroid function tests in the context of other lab investigations and an upper GI series, ascertainment of the etiology for our patient’s nausea and vomiting can be accredited to an understanding of hypothalamic-pituitary-thyroid axis regulation [Figure 2]. Clinical features and symptoms resolved with thyroid hormone replacement therapy and clinical improvement was sustained at follow up.

Gastroparesis is defined as delayed gastric emptying in the absence of upper GI obstruction, and presents with nausea, vomiting, early satiety, bloating and/or abdominal pain. Approximately 30% of gastroparesis cases are a consequence of diabetes, 20% result from intended or iatrogenic surgical interruption of the vagal nerve, and up to 50% of cases are deemed idiopathic.

In general, the etiologies of gastroparesis can be organized into two categories: reversible and non-reversible. In this case, non-reversible gastroparesis possibilities that might have reasonably been entertained included vagal nerve injury in light of the history of extensive abdominal surgeries, and chronic idiopathic intestinal pseudo-obstruction, which could also have occurred in consequence to her previous surgeries. Potentially reversible etiologies included hypothyroidism, electrolyte abnormalities with acidosis, critical illnesses such as diabetic ketoacidosis, shock, adrenal insufficiency, sepsis, drugs, and narcotics in particular.

Severe nausea and vomiting were nonspecific symptoms of hypothyroidism, but also the most common symptoms of severe gastroparesis. The voluminous nasogastric output in the absence of upper GI obstruction was further indicative of gastroparesis.

Ultimately, the diagnosis of hypothyroidism-induced gastroparesis was based on finding TSH 151 mIU/mL to uncover the history of inadvertent interruption of thyroid hormone replacement. In a study performed with 76 patients with overt hypothyroidism, a positive correlation was found between their serum TSH concentration and the percentage of positive hypothyroid symptoms reported. Due to the complexity of this case, weight gain was not present, and other signs and symptoms of hypothyroidism were overshadowed. Her rapid clinical improvement following resumption of thyroid hormone replacement established hypothyroidism as the cause of gastroparesis.

CONCLUSION

Diagnosis of hypothyroidism is usually straightforward, but this
case illustrates that in the absence of other typical symptoms of hypothyroidism, acute gastroparesis can be a less conspicuous manifestation. Include hypothyroidism in the differential diagnosis of nausea and vomiting in severe gastroparesis.

REFERENCES


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