



A Case of Ascending Sensory Neuropathy Due to Acute Onset Thiamine Deficiency After Intra-gastric Balloon Placement

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Abstract

Endoscopic bariatric therapies (EBTs) are emerging as nonsurgical weight loss interventions that are being used with increasing frequency. Similar to the potential for early and late complications from bariatric surgeries, metabolic derangements and nutritional deficiencies can also be seen after EBTs. In particular, thiamine deficiency is a well-known complication typically presenting with neurological symptoms, commonly Wernicke encephalopathy or dry beriberi. However, such a deficiency developing after placement of a temporary device such as an intra-gastric balloon is not only unique, but the clinical presentation of isolated ascending sensory neuropathy is very rare, both of which were seen in our patient.

Keywords Thiamine deficiency · Vitamin B₁ deficiency · Nutritional deficiency · Endoscopy · Endoscopic bariatric therapy · Intra-gastric balloon · Neuropathy · Ascending sensory neuropathy

Introduction

Treatment options for obesity have increased with the advent of endoscopic bariatric therapies (EBTs). One such therapy—the intra-gastric balloon (IGB)—is favorable as the procedure is less invasive and nonsurgical. IGB therapy entails the placement of a fluid-filled balloon in the stomach under endoscopy. There, it stays in the stomach promoting early satiety and delaying gastric emptying, resulting in weight loss [1]. As EBTs become more popular in clinical practice, potential short- and long-term complications such as nutritional deficiencies are important to be aware of, even during the course of a temporary device such as the IGB. In particular, vitamin B₁ deficiency is a known complication of bariatric surgery. Also known as thiamine, this important vitamin is required for vital body functions and cellular processes including growth, development, and function. Thus, thiamine deficiency is associated with a wide range of clinical symptoms but

presents commonly as Wernicke encephalopathy or dry beriberi [2]. Pure sensory ascending neuropathy as a presenting complaint, however, has rarely been reported. Here, we describe such a case presenting in the acute setting after endoscopic IGB placement.

Case Presentation

A 48-year-old woman with obesity, body mass index (BMI) of 37.4 kg/m², and otherwise healthy underwent an uncomplicated placement of an IGB for weight loss therapy. On post-operative day 1, patient endorsed nausea and abdominal cramping which was initially well-controlled with an anti-emetic and anti-spasmodic medication. However, symptoms returned and continued to progress to significant nausea and intermittent emesis secondary to delayed gastric emptying. Again, this mildly improved with adherence to a strict liquid diet; however, symptoms once again returned with reintroduction of solid foods, and the patient was trialed on erythromycin and buspirone without much improvement. During these initial weeks post-procedure, patient's daily calorie intake on average was 300–500 kcal/day. At her 9-week follow-up, patient's BMI had decreased from 37.4 kg/m² pre-procedure to 30.5 kg/m² with a total body weight loss of 18%. Despite the success of weight reduction, she continued to experience persistent nausea and vomiting, and therefore, the decision was

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made to prematurely remove the IGB at 3 months post-placement. Post-removal, patient endorsed immediate relief from her symptoms of nausea, vomiting, and abdominal discomfort.

However, prior to removal of the IGB, at 8 weeks post-procedure, patient initially noticed symmetric numbness which started at her ankles and ascended rapidly. Due to progression of symptoms in 4–5 weeks, the patient sought medical care and underwent a thorough neurologic evaluation. Exam was significant for symmetrically decreased light touch, pinprick, and temperature in the distribution of the medial malleoli, inner thighs, and abdomen/thorax to the level of T6 between the mid-clavicular lines. The upper extremities, dorsal surface of the trunk, and feet were spared. Weakness, areflexia, and respiratory compromise were not noted. MRI imaging of the brain and spine did not reveal myelopathy, inflammation, or other gross abnormalities. Expanded nutritional labs revealed low serum thiamine at 57 nmol/L (reference range 70–80 nmol/L). Otherwise, labs were normal. Patient was prescribed oral thiamine supplementation. Additionally, the IGB had already been removed by the time this diagnosis was determined, and oral intake had improved. Within weeks of supplementation, she reported improvement in symptoms with almost complete resolution of numbness at 1-year follow-up.

Discussion

Although bariatric surgery has multiple benefits via weight reduction and improvement of obesity-related complications, many possible early and late complications exist as a result of these procedures, including metabolic derangements and nutritional deficiencies. In addition to pre-existing micronutrient deficiencies in patients with overweight or obesity, poor dietary compliance and malabsorption post-operatively can result in nutritional deficiencies. These can be exacerbated by persistent vomiting, as was the case in this patient.

In particular, thiamine, or vitamin B₁, deficiency is a well-known late complication after bariatric procedures; however, it can also occur early post-operatively due to low storage reservoirs in the body. Deficiency has been reported in bariatric patients experiencing inadequate dietary intake, persistent vomiting, and rapid weight loss. As thiamine is absorbed from the duodenum and proximal jejunum, deficiencies are more commonly seen in patients who undergo surgical alteration of the intestinal tract such as a Roux-en-Y gastric bypass and biliopancreatic diversion/duodenal switch [2, 3]. However, in the case described, the patient presented with a thiamine deficiency post-IGB placement due to poor dietary intake and persistent vomiting.

Thiamine deficiency can clinically present with various symptoms due to the significant nature of its function.

Thiamine is required for vital body functions and cellular processes including growth, development, and function. Thus, the recommended daily amount of thiamine intake is 1.2 mg for adult men and 1.1 mg for adult women. Patients who are unable to ensure adequate intake or absorption are prone to develop a deficiency and the associated wide range of symptoms [4]. Classically, patients present clinically with neurological symptoms, most commonly Wernicke encephalopathy or dry beriberi. Isolated ascending sensory neuropathy is a very rare presentation, which was seen in our patient.

After excluding common etiologies of neuropathy via thorough examination, imaging, and labs, a diagnosis of ascending sensory neuropathy secondary to thiamine deficiency can be made in the setting of low serum thiamine. Patients with mild symptoms of isolated ascending sensory neuropathy can be treated with oral thiamine supplementation instead of high doses of intravenous thiamine, which are typically used for severe deficiency and serious manifestations of Wernicke's encephalopathy or dry beriberi. In this case, the resolution of vomiting after IGB removal, improvement in dietary intake, and thiamine supplementation led to symptomatic improvement.

Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

Ethical Approval This article does not contain any studies with human participants or animals performed by any authors.

Informed Consent For this type of retrospective study, formal consent is not required.

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