Heller oesophagomyotomy as treatment for achalasia after gastric bypass for morbid obesity

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ABSTRACT

When achalasia is unrecognised during the preoperative phase in patients who have undergone bariatric procedures, a late Heller oesophagomyotomy may be used as the treatment modality to prevent the development of megaesophagus. We present the case of a 66-year-old man with achalasia 3 years after a Roux-en-Y gastric bypass procedure.

KEYWORDS

Achalasia – Obesity – Heller myotomy – Roux-en-Y gastric bypass

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Achalasia in morbidly obese patients is particularly infrequent. Classic symptoms of achalasia include dysphagia and weight loss. In the morbidly obese, this is paradoxical. It is well documented that 1% of bariatric patients have achalasia with regurgitation, aspiration and coughing but without dysphagia or weight loss.¹ Co-morbidities affecting respiratory functions such as chronic obstructive pulmonary disease (COPD) or cardiac diseases may obscure typical symptoms of achalasia, which can delay diagnosis.² Hong *et al* postulated that the low threshold of hunger in the obese can be responsible for the lack of weight loss and decreased symptoms of oesophageal dysmotility.³ We present a case of a patient with achalasia who was treated with a laparoscopic Heller myotomy (LHM) three years after a laparoscopic Roux-en-Y gastric bypass (RYGB) procedure.

Case history

A 66-year-old man (body mass index [BMI] 36.16kg/m²) with a 2-year history of symptomatic oesophageal spasms presented with chest pain, difficulty swallowing solid food and regurgitating liquids. He had had a laparoscopic RYGB three years earlier (BMI 58.11kg/m²), resulting in an excess weight loss percentage (%EWL) of 58.66%. His co-morbidities were hypertension, sleep apnoea on bilevel positive airway pressure and COPD (non-smoker). Pneumatic dilation and endoscopic botulinum toxin injection (EBTI) were administered for seven months with minimal improvement. An upper gastrointestinal study demonstrated an area of narrowing (8mm) at the gastro-oesophageal junction (Fig 1) that was not visualised during the oesophagogastroduodenoscopy (OGD) carried out previously. A hypertensive lower oesophageal sphincter was

evident on manometry following contraction, with oesophageal spasms from the mid to distal segments.

The decision for revision and LHM was made by the bariatric team and discussed with the patient. Extensive amounts of adhesion between the small bowel, left lobe of the liver and anterior wall of the stomach were taken down until the gastro-oesophageal junction was exposed. A 32Fr gastric lavage tube was advanced into the pouch. An oesophagomyotomy, 12cm in length, was performed, reaching approximately 2cm into the gastric pouch. An intraoperative OGD was carried out to verify the patency of the gastro-oesophageal junction and to examine the mucosa. The diaphragmatic crus were closed with 2/0 polypropylene non-absorbable unidirectional barbed sutures. The patient tolerated the procedure well with minimal blood loss.

An upper gastrointestinal study on postoperative day 1 demonstrated no signs of perforation (Fig 2). A soft diet was tolerated on day 3 and the patient was discharged home on day 4. At the one-month follow-up appointment, swallowing and oesophageal symptoms were significantly improved with solid food. BMI and %EWL were 37.89kg/m² and 56.69% at one year, with complete resolution of symptoms.

Discussion

This patient had a history of COPD as a non-smoker and sleep apnoea on bilevel positive airway pressure that may have caused a delay in the diagnosis of achalasia. He asserted that prior to the laparoscopic RYGB surgery three years earlier, he had mild symptoms of dysphagia but did not address the problem because the symptoms were not affecting his quality of life. An increase in progression of

Figure 1 Upper gastrointestinal study with water soluble contrast showing narrowing of 8mm in distal oesophagus, 'bird beak' appearance and distal tapering of the gastro-oesophageal junction

Figure 2 Postoperative upper gastrointestinal study with water soluble contrast demonstrating no signs of perforation, obstruction, leak or fistula

the dysphagia started a few months after undergoing the RYGB.

The usual workup for dysphagia after RYGB involves an oesophagography and upper endoscopy. The main anatomical abnormalities investigated include anastomotic strictures and ulceration, pouch size, length of Roux limb and jejunal blind end, and gastrogastic fistulas. If significant reflux symptoms are present in the setting of normal RYGB anatomy, a 24-hour pH study is necessary to distinguish between acid and alkaline reflux. The oesophagography for this patient revealed an 8mm stricture in the distal part of the oesophagus with no evidence of extravasation. The OGD did not demonstrate any evidence of gastro-oesophageal reflux disease or strictures resulting from the RYGB. Computed tomography (CT) is not a routine investigation for these patients and does not help with the diagnosis of achalasia. However, patients with similar symptoms after RYGB surgery often undergo CT to rule out other more common pathologies such as gastrogastic fistulas, jejunojejunostomy strictures, internal hernias and intussusception.

Definitive diagnostic tests for achalasia must also include a manometry. Upper gastrointestinal series and the manometry results supported the diagnosis of achalasia in this patient. EBTI was performed on three different occasions but resulted in a relapse of symptoms. Furthermore, EBTI may increase the difficulty of a subsequent LHM owing to scarring in the lower oesophageal sphincter.

Little evidence exists for the indication of LHM to treat achalasia in the morbidly obese because of the rarity of

this combination. In our case, the lack of improvement of symptoms led to surgical management in the form of a LHM. Combining a LHM with a bariatric procedure or performing a late LHM is achievable but choosing the appropriate procedure is contentious.

Suboptimal outcomes of LHM may result from the failure to adequately extend the myotomy 2cm on to the stomach and to a minimum of 4cm along the oesophagus from the lower oesophageal sphincter. When executing both procedures concurrently, we recommend performing the LHM before the RYGB to create an adequate gastric pouch. An intraoperative OGD is advocated for direct confirmation of adequate length of the myotomy. Gastric banding is suspected to reduce oesophageal dysfunction.⁴ The restriction of the upper stomach in gastric banding may interfere with a LHM and can predispose patients to perforations.

Owing to the gradual improvement in clinical symptoms, follow-up studies were not indicated in the postoperative management of our patient. In hindsight, the presence of gastro-oesophageal reflux disease after a LHM in a prior gastric bypass could be managed with a fundoplication, using the fundus of the excluded stomach, as described by some authors.⁵ However, based on our experience with other LHM cases performed without fundoplication, proton pump inhibitor therapy is very effective for reflux symptoms.

Conclusions

The occurrence of achalasia in a morbidly obese patient is rare and difficult to treat in the long term. LHM after RYGB

can be performed safely and is a feasible treatment option for achalasia when pneumatic dilation or EBTI produces no improvement in symptoms. With the exception of bilio-pancreatic diversion with or without duodenal switch and gastric banding, the literature has supported performing both the weight loss surgery and a LHM concurrently to avoid significant adhesion in the foregut and additional time in the operating room. However, we recommend not waiting to surgically treat achalasia when symptoms appear early in a bariatric candidate patient. Further studies are needed to evaluate the morbidly obese for achalasia prior to any weight loss surgery.

References

1. Almog G, Anthone GJ, Crookes PF. Achalasia in the context of morbid obesity: a rare but important association. *Obes Surg* 2003; **13**: 896–900.
2. Koenig SM. Pulmonary complications of obesity. *Am J Med Sci* 2001; **321**: 249–279.
3. Hong D, Khajanchee YS, Pereira N *et al*. Manometric abnormalities and gastroesophageal reflux disease in the morbidly obese. *Obes Surg* 2004; **14**: 744–749.
4. Petermac D, Hauser R, Weber M, Schöb O. The effects of laparoscopic adjustable gastric banding on the proximal pouch and the esophagus. *Obes Surg* 2001; **11**: 76–86.
5. Kawahara NT, Alster C, Maluf-Filho F *et al*. Modified Nissen fundoplication: laparoscopic antireflux surgery after Roux-en-Y gastric bypass for obesity. *Clinics* 2012; **67**: 531–533.