



The Gastric Migration Crisis in Obesity Surgery

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The term intra-thoracic migration of the stomach (ITM) has been applied for postoperative hiatal hernias after laparoscopic sleeve gastrectomy (LSG). ITM is underrecognized because symptoms are often unspecific when they relate to entrapment rather than reflux. ITM is also underreported [1] because of the infrequent use of (3D)-CT which is the best method to detect staple lines above the diaphragm. Many obesity surgeons are not fully aware of the increasing numbers of ITM and the challenges associated with its prevention and therapy.

Post-bariatric ITM results from persistence or progression of a pre-existing hiatal hernia or from de novo formation. The mechanisms responsible for this outcome are not well understood but they appear to differ from the mechanisms leading to hiatal hernias in the non-obese population. A gastric sleeve is not much wider than the esophagus itself and can easily be drawn through the hiatus. This may be facilitated by the melt down of visceral and hiatal fat. At some point, the diaphragmatic hiatus surrenders and the stomach is dragged upwards by the abdomino-thoracic pressure gradient and the contractile forces of the esophagus. This anatomical alteration severely impairs the function of the gastroesophageal junction leading to gastroesophageal reflux. After LSG, the prevalence of ITM and gastroesophageal reflux disease (GERD) increases with time up to 50% and 45%, respectively [2]. This correlation underlines that migration of a gastric sleeve regularly results in symptomatic reflux. Systematic endoscopy 5 years after LSG results in a high rate (18.8%) of Barrett's esophagus [3] and some cases with Barrett carcinoma have recently been published. A worrying scenario is an upcoming flood of symptomatic and complicated ITM following the recent tide of LSG.

The rate of ITM after bypass surgery is unknown because most patients with pre-existing or de novo hiatal hernia remain free of reflux and thus do not undergo an endoscopic or

radiologic evaluation. GERD is reported in less than 5% of patients after the Roux-en-Y gastric bypass (RYGB). The common explanation is the combination of a low-pressure environment in the gastric pouch and its downward traction by the gastrojejunostomy. The positive effect makes RYGB the surgical procedure of choice for obese patients with GERD.

Similarly, the One Anastomosis Gastric Bypass-Mini Gastric Bypass (OAGB-MGB) is also thought to protect from GERD. The anti-reflux property is attributed to the low intragastric pressure and the neutralizing effect of bile. Objective functional measurements, however, are inconclusive on this matter. Tolone et al. examined 15 patients free of GERD before and 1 year after omega-loop bypass [4]. Gastroesophageal junction function was not compromised and the number of reflux events was dramatically decreased. On the contrary, 24 h pH-metry in 11 patients demonstrated increases in the DeMeester score, the esophageal acid exposure and the number of non-acid reflux episodes 12 months after OAGB [5]. De novo GERD developed in two patients and worsening of pre-existing GERD developed in all four patients with preoperative evidence of GERD. Further objective studies are needed to clarify the anti-reflux concept of OAGB-MGB as chronic reflux of acid and bile has mutagenic implications on the esophageal epithelium.

There is little reason to believe that the stomach cannot migrate after OAGB-MGB because there is no downward traction of the long gastric pouch and the surgical dissection of the cardia region is not that much different to that in LSG. The diameter of the gastric pouch is larger than that of the gastric sleeve but hardly large enough to prevent migration through the diaphragmatic hiatus.

A 2012 consensus-based best practice guideline concluded that surgeons should routinely dissect the phrenoesophageal membrane during sleeve gastrectomy and inspect the greater curvature side of the stomach for the presence of a hiatal hernia, and if present appropriate closure of the crus should be performed [6]. Recent observations contradict this recommendation. For example, Dakour Aridi et al. found no effect on GERD symptomatology after SG with or without

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concomitant hiatal hernia repair [7]. The rates of GERD remission (21.3% vs. 29.7%) and new-onset GERD symptoms (41.4% vs. 46.2%) were comparable. Samakar et al. observed a 65.4% rate of persistent complaints and a 15.6% rate of de novo complaints in 58 patients who underwent SG with hiatal repair [8]. An increase in the prevalence of typical GERD symptoms by concomitant hiatal hernia repair was found in 78 patients when compared with 102 patients with SG only [9]. These recent observations suggest that aggressive manipulation of the cardia may even predispose to ITM.

A pre-existing hernia is usually not repaired during RYGB. The first consensus conference on OAGB-MGB did not reach agreement on routine crural approximation [10]. Rutledge does not repair hiatal hernias simultaneously during OAGB-MGB [11], whereas Carbajo proposes its repair [12]. Others simply retract small hernias without crural repair.

It is surgical wisdom not to operate on asymptomatic patients, but controversy exists on the best approach when ITM becomes symptomatic. GERD is markedly improved by conversion of a sleeve into a RYGB and by diversion of an OAGB-MGB, respectively. Symptoms that are related to entrapment such as volume reflux, pain, or dysphagia require anatomical correction rather than conversion alone but published results are lacking. There is good agreement on mobilizing the esophagus over a sufficiently long distance for tension-free subphrenic relocation. There is also agreement on repairing the hiatus but the technique is not standardized. The crura are approximated around a calibrating nasogastric tube of varying diameter. Some surgeons combine posterior and anterior sutures and others advocate mesh reinforcement. There is no agreement on whether to fix the cardia to the diaphragm or the ligamentum teres hepatis. Mimicking a fundoplication with the bypassed fundus or with a so-called Nissen/Sleeve have been proposed, but the evidence is small.

Conclusion

Post-bariatric ITM is on the rise. Symptoms relate to gastroesophageal reflux and entrapment, and chronic reflux may have mutagenic effects even when clinically silent. Prevention and treatment of ITM are increasingly important issues in obesity surgery. Bariatric surgeons should quickly take up the challenge before others will.

Compliance with Ethical Standards

Conflict of Interest The author declares no conflict of interest.

Informed Consent Statement Does not apply.

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