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Surgeon at work

## Simplified laparoscopic Hill repair for the treatment of symptomatic sliding hiatus hernia after bariatric surgery

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Sliding hiatus hernia (SHH) is a frequent condition associated with obesity [1]. After Roux-en-Y gastric bypass (RYGB) and sleeve gastrectomy (SG), SHH may be asymptomatic, may predispose to gastroesophageal reflux disease (GERD) [2], and occasionally may trigger painful dysphagia. The pathophysiology of pain is thought to be related to the rubbing of the gastric staple line on the left diaphragmatic pillar, transmitted by the left phrenic nerve [3].

Workup of symptomatic postbariatric SHH includes upper gastrointestinal contrast study (UGI), esophagogastropscopy, manometry, and pH-metry. If results of these examinations are interpreted as normal, an abdominopelvic computed tomography (CT) may help to identify even small SHH by visualization of the ascension of the gastric staple line above the diaphragmatic crura (Fig. 1).

Revisional surgery might become necessary to treat symptomatic SHH when medically refractory GERD or painful dysphagia are present. An ideal surgical technique should provide hernia reduction and ensure the prevention of recurrence. In the general population this can be achieved by fundoplication [4]. In postbariatric patients the gastric fundus is absent or inaccessible for an antireflux valve

creation; therefore, we adopted the laparoscopic Hill repair (LHR) as the revisional technique of choice for the treatment of symptomatic SHH. With the aim of reproducibility and technical simplicity, we do not perform horizontal cardioplication under intraoperative manometry, as described in the original description by Hill et al. [5], and refer to our procedure as simplified LHR (sLHR). The technique is illustrated in a patient who developed painful dysphagia 3 months post-SG. The SG was performed over a 36-Fr bougie without buttressing material and the staple line was not oversewn. Workup before revisional surgery included abdominal CT with oral contrast, esophagogastropscopy, and manometry. A SHH was identified, and other potential causes of dysphagia, such as esophageal motility disorders, gastric ulcer, gastric volvulus, midgastric stenosis, or gastric bezoar, were ruled out. We believe that SHH formation was triggered by the SG via the dissection of the suspensor tissue around the esophagogastric junction (EGJ) and by fundus removal in a high pressure abdomen. The symptomatology of dysphagia for solid food with a herniated gastric sleeve identified by CT is compatible with the pathophysiological hypothesis of mechanical friction of the herniated staple line on the left diaphragmatic pillar. To relieve the patient's discomfort and to avoid malnutrition, we proceeded to revisional surgery by sLHR.

**Surgical technique (video)**

We administer a preoperative single dose of antibiotic (cefuroxime) and standard antithrombosis prophylaxis. The patient is placed in reverse-Trendelenburg position with the

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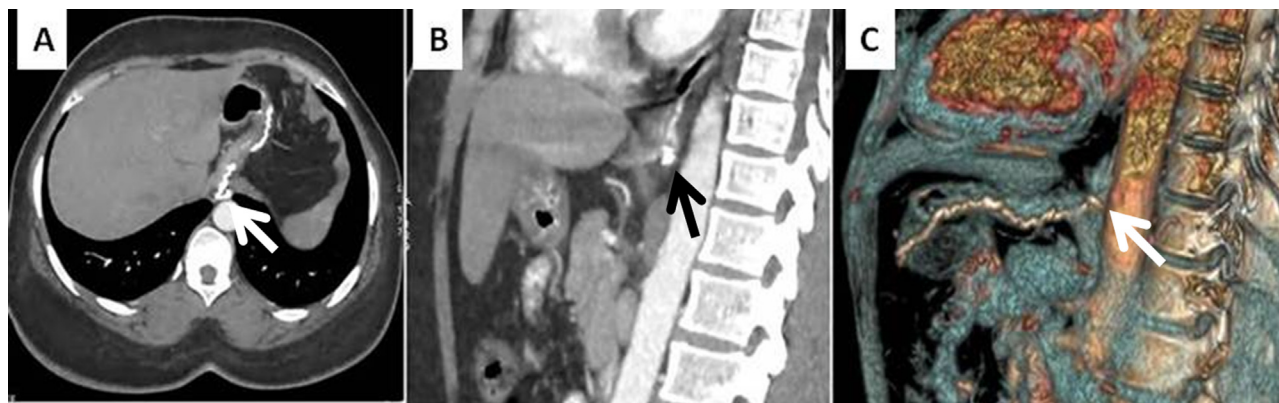


Fig. 1. Ascension of the gastric staple line above the diaphragmatic crura after sleeve gastrectomy on abdominal computed tomography. The arrows point at the summit of the gastric sleeve. (A) Axial view. (B) Saggital view. (C) Three-dimensional saggital reconstruction.

legs apart. The surgeon stands between the patient's legs. Five trocars are placed into the abdomen in a semicircular shape (Fig. 2).

The main steps of the sLHR are the following:

1. Adhesiolysis around the left liver lobe and dissection of the phrenoesophageal membrane (Fig. 3A).
2. Identification of the summit of the gastric staple line above the diaphragmatic crura.
3. Takedown of the attachments between the gastric staple line and left diaphragmatic pillar. Care should be taken to avoid gastric injury during dissection. We recommend the use of ultrasound scissors and blades to decrease heat dissemination during coagulation and to avoid accidental formation of electric arcs on the metallic staple line (Fig. 3B).
4. Posterior closure of the esophageal hiatus by nonabsorbable suture over a 36-Fr bougie. In our department this bougie size is used for primary SG, in accordance with studies on optimal bougie size [6].
5. Mobilization of at least 3 cm of distal esophagus into the abdominal cavity (Fig. 3C).



Fig. 2. Position of ports for the simplified laparoscopic Hill repair.

6. Fixation of the seromuscular layer of the EGJ to the median arcuate ligament by 2 stitches of nonabsorbable suture (Fig. 3D).
7. Verification of the patency of the EGJ with a 36-Fr bougie
8. Additional anterior closure of the esophageal hiatus if the anterior angle of the hiatus is steep and the gap is still >2 cm when the 36-Fr bougie is in the stomach.

The procedure can be coupled with gastric pouch resizing by vertical linear stapling or with conversion of SG to RYGB. Postoperatively, patients are started on semisolid diet on day 1 and have routine UGI on day 3. One month of proton-pump inhibitor treatment is prescribed systematically.

## Discussion

High intra-abdominal pressure due to obesity is the main cause of SHH development in the bariatric population [7]. SG and RYGB may add contributing factors, such as dissection of the suspensor tissue around the EGJ, flattening of the His angle due to fundus removal, and reduced formation of adhesions after laparoscopic surgery [8,9]. Later, when weight loss is achieved, the shrinkage of the pericardial fat pad relatively enlarges the hiatal orifice, which further contributes to the occurrence of SHH [10].

Based on abdominal CT findings, Baumann et al. observed a 37% prevalence of SHH 6 months after SG [11]. In their study, preoperative symptoms of GERD or proton-pump inhibitor use were contraindications for SG, whereas the rate of SHH at baseline was not described. However, at follow-up, 4 of 10 patients (40%) with a herniated sleeve complained of regurgitation, compared with only 2 of 17 in the group with a correctly positioned sleeve (12%). Tai et al. endoscopically identified SHH in 27% of SG patients at 1 year [12]. The real incidence of SHH after RYGB is probably underestimated. Fornari et al. found a 16% prevalence of new-onset SHH by UGI in previously unaffected RYGB patients at 6 months postoperatively [13].

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