Herniation of an abdominal antireflux fundoplication into the chest: what does it mean?

Yannick Deswysen, Francesco Volonté, Christian Gutschow, Renato Romagnoli, Paolo Strignano, Aous Ouazzani, Luc Verstraete, Charles De Gheldere, Maximillien Thoma, Vincent Uluma, Felicia Ungureanu, Jean-Yves Mabrut and Jean-Marie Collard*

Unit of Upper Gastro-Intestinal Surgery, Cliniques Universitaires Saint-Luc, Université Catholique de Louvain, Brussels, Belgium

* Corresponding author. Unit of Upper Gastro-Intestinal Surgery, Cliniques Universitaires Saint-Luc, Avenue Hippocrate 10, 1200 Brussels, Belgium. Tel: +32-2-7642213; fax: +32-2-7648918; e-mail: jean-marie.collard@uclouvain.be (J.-M. Collard).

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Abstract

OBJECTIVES: The specific contribution of the herniation of an abdominal antireflux fundoplication into the chest to symptomatic and therefore surgical failure remains unclear.

METHODS: The study was conducted in 189 consecutive fundoplication patients, categorized as patients reoperated on for chest herniation of either an abdominal 360° (Group 1; n = 95) or a partial (Group 2; n = 10) fundoplication, and patients having undergone an intrathoracic 360° fundoplication for short oesophagus (Group 3; n = 84; reference group). There were four subgroups in Group 1: 1A: wrap still complete and perioesophageal; 1B: wrap still complete but perigastric; 1C: wrap still perioesophageal but partially disrupted and 1D: wrap perigastric and partially disrupted.

RESULTS: The prevalence of defective symptoms (heartburn and regurgitation) was significantly lower (P < 0.0001) in Group 3 (0.0%) and Subgroup 1A (3.7%) than in Subgroups 1B (84.4%), 1C (86.7%) and 1D (100%) and Group 2 (100%). The prevalence of obstructive symptoms (dysphagia, chest pain, necrosis and perforation) was significantly higher (P < 0.0001) in Subgroup 1A (100%) than in Subgroups 1B (57.8%), 1C (60.0%) and 1D (25.0%). The prevalence of a short oesophagus, an abdominal wall hernia repair and high abdominal pressure episodes in reoperated patients were 13.7, 36.2 and 67.2%, respectively.

CONCLUSIONS: Unlike perigastric or partial fundoplication, a 360° perioesophageal abdominal fundoplication, when herniated into the chest, is still effective against reflux. Obstructive symptoms are due to either diaphragmatic strangulation or perigastric migration of the wrap (slipknot effect). Short oesophagus, weakness of the abdominal wall and high abdominal pressure episodes favour the herniation process.

Keywords: Reoperation of fundoplication • Chest • Oesophagus • Gastroesophageal reflux disease • Hiatal hernia

INTRODUCTION

Various anatomical anomalies following antireflux fundoplication have been described [1-9], each one explaining the failure to provide the expected symptomatic relief to patients suffering from gastroesophageal reflux disease (GERD). Most commonly described are too long or too tied to wrap, construction or slip down of the wrap around the stomach, breakdown of the fundoplication, absence or breakdown of the crura closure, the latter made too snug, construction of the fundoplication with the greater curve of the stomach rather than with the gastric fundus, excessive fibrosis of the hiatal sling post-cauterization and herniation of the wrap into the chest. These anatomical anomalies mostly coexist at reoperation, rendering the specific contribution of each one of them to the failure process unclear.

The present study aims at specifying the role of chest herniation of the fundoplication in the genesis of defective or obstructive oesophageal symptoms occurring either immediately after operation or at follow-up, i.e. after a symptom-free period. A second objective is to evaluate to which extent other factors may influence the failure pattern in these patients. Contributing factors are concomitant partial disruption of the fundoplication, concomitant location of the fundoplication around the stomach instead of around the oesophagus, the presence of a short oesophagus [10], a history of sudden increases in abdominal pressure and diffuse weakness of the abdominal wall (which had justified wall-repairing surgery).

For this study, those patients who had their initial abdominal fundoplication herniated into the chest were extracted from the senior author’s series of patients who underwent redo antireflux surgery following unsatisfactory symptomatic outcomes.
MATERIALS AND METHODS

Patients

The present study is based on 189 consecutive patients operated on between 1986 and 2010 (Fig. 1). They were 72 women and 117 men ranging in age from 23 to 82 years (mean: 52 years; SD 13.24). Among those, 105 patients were reoperated on by the senior author (Jean-Marie Collard) for herniation into the chest of either an abdominal 360° fundoplication (Group 1, n = 95) or an abdominal partial fundoplication (Group 2, n = 10). The remaining patients underwent an intrathoracic 360° fundoplication as first antireflux operation for GERD linked to a short oesophagus (reference group, Group 3, n = 84). Group 1 patients were categorized clinically as those who never experienced any symptomatic relief following antireflux operation (early failure, n = 31) and those who became symptomatic again after a symptom-free period ranging from 1 to 300 months (late failure, n = 64). Group 1 patients were further subdivided into four subgroups according to anatomical findings at reoperation: Subgroups 1A: herniation of an intact 360° periesophageal fundoplication into the chest (n = 27); 1B: herniation of a 360° fundoplication that also had slipped down around the stomach (n = 45); 1C: herniation of a 360° fundoplication that had partially broken down (n = 15) and 1D: herniation of a 360° fundoplication that had partially broken down and slipped down around the stomach (n = 8).

The number of previous antireflux operations in combined Groups 1 and 2 was 1, 2 and 3 in 88, 14 and 3 patients, respectively. The last antireflux operation had been carried out by either laparotomy (n = 46) or laparoscopy (n = 1), or left thoracotomy (n = 13). Three were performed as emergency operations for acute strangulation of the fundoplication at the hiatal level with (n = 2) or without (n = 1)

The time elapsed between the previous antireflux operation and the reoperation ranged from 1 day to 305 months (median: 24 months). The local ethics committee approved the study and waived patient written consent.

Preoperative work-up

Radiological anatomy of the gastroesophageal (GE) junction in all patients was assessed by a preoperative barium swallow study including radiographs taken in both the upright and supine positions [12, 13]. The oesophagus was considered short whenever the junction between upper gastric folds and the unwrinkled oesophageal mucosa was above the diaphragm, while the oesophageal body was straight in the upright position.

Ninety-four patients, i.e. 84 of Group 1 and all 10 of Group 2, underwent upper gastrointestinal (GI) endoscopy in search of erosive oesophagitis or Barrett’s.

One hundred and twenty-five patients underwent 24-h oesophageal pH monitoring either before the reoperation in Group 1 (n = 54) and in Group 2 (n = 5) or after the intrathoracic 360° fundoplication in Group 3 (n = 66). Data were analysed using DeMeester’s criteria [14] in terms of percentage of time that the oesophageal pH was <4 during the total, upright and supine periods of recording (95th percentile of normal volunteers: 4.5, 8.4 and 3.5%, respectively).

Intraoperative findings and surgical technique

Antireflux reoperations were performed by laparotomy (n = 91), laparoscopy (n = 1) or left thoracotomy (n = 13). Three were performed as emergency operations for acute strangulation of the fundoplication at the hiatal level with (n = 2) or without (n = 1)
gastric perforation. The thoracic approach was used whenever oesophageal shortening was suspected from the preoperative barium swallow study. The GE junction was considered surgically irreducible whenever it could not be replaced in the abdomen without undue tension, despite extended surgical mobilization of the oesophageal body up to the aortic arch. The actual anatomy of the residual fundoplication was assessed by the senior author with regard to its completeness or partial breakdown and to its location around the stomach or around the oesophagus. For this, the residual fundoplication was carefully freed from any adhering tissue and progressively taken down.

Intrathoracic fundoplication [15, 16] for Group 3 was performed by left thoracotomy according to a previously described surgical technique [17, 18]. The most critical steps of the operation include further enlargement of the hiatal sling by a 3-cm radial incision of the diaphragm to prevent any strangulation of the intrathoracic stomach at the hiatal level, and anchoring of the intrathoracic fundoplication to the hiatal sling using numerous sutures placed so as to prevent any tiring out of the gastric wall during diaphragmatic movements, especially when the patient is coughing after the operation.

Data analysis

The clinical charts of all patients were carefully reviewed for the presence of defective (i.e. heartburn and regurgitation) or obstructive symptoms (i.e. dysphagia, chest pain, gastric necrosis or perforation) before the antireflux reoperation in Groups 1 and 2 by the first author and after the intrathoracic fundoplication in Group 3 by the second author.

Fifty-eight patients belonging to Groups 1 and 2 were interviewed by the first author with a view to uncovering any previous abdominal wall hernia repair and any history of sudden or repetitive increase in abdominal pressure following the initial antireflux operation.

Statistical analysis

Categorical variables were analysed using Fisher’s exact and $\chi^2$ tests. Mann-Whitney U- and Kruskal-Wallis rank-sum tests were used for continuous variables. A $P$-value of <0.05 was considered statistically significant (Statistica 5.0, StatSoft, Inc., Oklahoma, USA).

RESULTS

Symptoms

The prevalence of defective symptoms significantly increased ($P < 0.0001$) from 0% ($n = 0$) in Group 3 to 3.7% ($n = 1$) in Subgroup 1A, to 84.4% ($n = 38$) in Subgroup 1B, to 86.7% ($n = 13$) in Subgroup 1C, to 100% ($n = 8$) in Subgroup 1D and to 100% ($n = 8$) in Group 2 (Fig. 2). As shown in Fig. 3, the prevalence of obstructive symptoms was significantly higher ($P < 0.0001$) in Subgroup 1A (100%; $n = 27$) than in Group 3 (5.9%; $n = 5$). In Group 1, it was significantly higher ($P < 0.0001$) in Subgroup 1A (100%; $n = 27$) than in Subgroups 1B (57.8%; $n = 26$), 1C (60.0%; $n = 9$) and 1D (25.0%; $n = 2$). There was no significant difference in terms of either defective or obstructive symptoms between the four subgroups belonging to Group 1 depending on whether they had experienced early or late failure (Table 1).

Upper gastrointestinal examinations

The prevalence of erosive oesophagitis at upper GI endoscopy was significantly lower ($P = 0.0008$) in Subgroup 1A (18.5%) than in Subgroups 1B (78.0%), 1C (66.7%) and 1D (62.5%), and in Group 2 (50.0%).

As shown in Fig. 4, oesophageal acid exposure at 24-h oesophageal pH monitoring expressed as the percentage of total time that oesophageal pH < 4 (mean ± standard error of the mean, [SEM]) was significantly ($P < 0.0001$) lower in Group 3 (0.50 ± 0.10) and Subgroup 1A (2.63 ± 1.27) than in Subgroups 1B (8.09 ± 1.45), 1C (10.18 ± 2.39) and 1D (7.42 ± 2.40) and in Group 2 (9.20 ± 4.37).

Intraoperative findings: early vs late failures

As shown in Fig. 5, perigastric fundoplication was as common ($P = 0.90$) in patients who had experienced early failure (54.8%) as in those with late failure (56.3%). There was a substantial trend
towards a higher prevalence of breakdown of the antireflux repair in late failure patients (29.7%) compared with those with early failure (12.9%). Short oesophagus, a condition that was present in 13 of the 95 Group 1 patients (13.7%), was significantly more common ($P = 0.0025$) in patients who experienced early failure (29%) than in those with late failure (6.3%). All 13 patients had their residual fundoplication found to be located around the stomach rather than around the oesophagus at reoperation.

### Abdominal pressure and weakness

Of the 58 patients belonging to Groups 1 and 2 for whom the information was available, 39 (67.2%) had a history of sudden or repetitive increases in abdominal pressure due to carriage of heavy loads ($n = 25$), forceful vomiting ($n = 4$), pregnancy ($n = 2$), prostatism ($n = 2$) or strain while awaking from anaesthesia ($n = 6$).

Twenty-one of these 58 (36.2%) patients were operated on for an abdominal wall hernia that was incisional, inguinal, umbilical, epigastric and diaphragmatic in 15, 1, 2, 2 and 1 patients, respectively.

### DISCUSSION

This single-centre retrospective study identifies three critical conditions for an antireflux fundoplication to function properly in the chest in order for a patient to be asymptomatic [18, 19]. First, the fundic wrap must be complete, covering 360° of the oesophageal circumference. Secondly, it must be located proximal to the GE junction, around the lower oesophagus. Thirdly, the hiatal sling must be large enough to give a wide berth to the fundoplication. These three conditions are met when the fundoplication is primarily constructed in the chest around a short oesophagus (Fig. 6A) or when an abdominal fundoplication herniates into the chest while remaining both complete and perioesophageal and when spontaneous disruption of the crura closure is also complete (Fig. 6B). Should one of these three conditions be lacking, the patient is bound to get defective or obstructive symptoms and to seek further medical consultation. Initial placement or subsequent downward migration of the fundoplication around the stomach as well as its spontaneous breakdown, and above all, the combination of the two, generate defective symptoms witnessing a loss in efficacy against GE reflux. The study shows indeed that neither a perigastric nor a partial fundoplication can function in the chest, as shown by pathological oesophageal acid exposure at 24-h oesophageal pH monitoring: ~90% of the patients studied had defective symptoms requiring antireflux reoperation. In this respect, Toupet [20], 50 years ago, interestingly emphasized the need for anchoring the partial fundoplication to the diaphragm. He
described, presuming that the latter, should it herniate into the chest, could no longer function properly. The fact that a 360° fundoplication may still be effective against reflux when it herniates into the chest at follow-up is a strong argument favouring the performance of a complete rather than a partial abdominal fundoplication in routine antireflux surgery.

The study shows that there are two conditions for the onset of obstructive symptoms once an abdominal fundoplication has herniated into the chest. First, incomplete spontaneous disruption of the crura closure (Fig. 7A) creates a strangulation effect on the gastric wall, usually responsible for dysphagia or chest pain, sometimes leading to life-threatening complications such as gastric bleeding from collar ulcers, or even necrosis of the intrathoracic portion of the stomach and subsequent gastric perforation into the lower mediastinum. This pinching effect at the hiatal level, although it accounted for obstructive symptoms in all studied subgroups, is less common when the fundoplication has both partially broken down and slipped down around the stomach. This observation probably reflects a smaller amount of gastric tissue at the hiatal level in these patients in comparison with those with a complete fundoplication herniating into the chest. Secondly, downward migration of a still complete fundoplication around the stomach as part of the herniation process generates a slipknot effect on the gastric wall (Fig. 7B), so that the proximal part of the stomach cannot expand enough to accommodate ingested food and the patient subsequently suffers from either dysphagia or lower retrosternal pain during meals. In this respect, perigastric location of the wrap in early failure patients reflects either its initial misplacement or its early downward migration along the stomach, whereas in late failure patients, it may be assumed that the initially perioesophageal wrap with time gradually migrated along the stomach.

There are three conditions identified in the study that predispose to the herniation of an abdominal fundoplication into the chest.

First, the existence of a short oesophagus precludes performing a tension-free fundoplication proximal to the GE junction from the abdomen. Should this be attempted, the wrap would be either placed around the subcardiac area of the stomach [21], leaving some secreting gastric tissue proximally [22] or made at all cost, i.e. under undue tension around the oesophagus. In the latter instance, the short oesophagus is predisposed to come back to its natural location in the lower mediastinum. The GE junction is either pushed transhiatally by any increase in abdominal pressure or attracted upward by the negative pressure that exists in the chest [23, 24]. The higher prevalence of a short oesophagus observed in the early failure patients of this study in comparison with the late failure patients probably reflects the fact that thoracic herniation of a GE junction that has been forcefully reduced into the abdomen usually occurs very early following antireflux operation. All this confirms that an oesophagus that never existed or that lost its space in the abdomen is best approached from the chest as far as a perioesophageal fundoplication is concerned.

Secondly, many GERD patients suffer from diffuse weakness of all walls of the abdominal cavity. Indeed, one-third of the patients in the present study had a history of abdominal wall-repairing surgery including surgical repair of an incisional, umbilical, epigastric, diaphragmatic or inguinal hernia. In this respect, we have to bear in mind that the diaphragm with all its oriﬁces is nothing else than the roof of the abdomen. Furthermore, unlike the other parts of the abdominal wall, the upper aspect of this roof is exposed to a negative-pressure environment, i.e. the lower intrathoracic pressure, which certainly adds to the intrinsic weakness of the tissues.

Thirdly, two-thirds of the 58 patients who had been interviewed for the purposes of the study reported a history of sudden or repetitive increases in intra-abdominal pressure due to the carriage of heavy loads, forceful vomiting, pregnancy or strain while awake from anaesthesia just after the previous antireflux operation. This emphasizes the fact that long-term outcomes of an antireflux operation depend not only on the quality of the surgical

![Figure 6: (A) Radiograph showing the absence of any strangulation effect in a Group 3 patient who underwent a primary intrathoracic 360° perioesophageal fundoplication including enlargement of the hiatus by radial incision of the diaphragm for short oesophagus. The dotted line represents the diaphragm. (B) Radiograph showing the herniation of an abdominal perioesophageal fundoplication made under tension in a patient in whom the presence of a short oesophagus had not been detected prior to initial antireflux surgery. Note the gastric slipknot phenomenon (arrow). The dotted line represents the diaphragm.](image-url)

![Figure 7: (A) Radiograph showing strangulation (arrow: diaphragmatic pinching phenomenon) of the herniated fundoplication because of incomplete breakdown of the crura closure in a Group 1 patient. (B) Radiograph showing an initially abdominal fundoplication that had herniated into the chest and was found to be located around the stomach at reoperation in a patient in whom oesophageal shortening had not been detected prior to initial antireflux surgery. Note the gastric slipknot phenomenon (arrow). The dotted line represents the diaphragm.](image-url)
procedure, but also on the patient’s behaviour at follow-up [11]. This observation also questions the indication of an antireflux operation in GERD patients likely to carry heavy loads in their daily life. In any event, proper performance of an antireflux fundoplication only creates an effective barrier against reflux. Surgery neither remedies the pre-existing tissular weakness nor suppresses the risk of high-pressure episodes in the abdomen, two conditions that are also at the origin of a hiatal hernia in most non-operated GERD patients. The higher prevalence in our recruitment of late failures (Group 1: 64 of 95) compared with early failures (Group 1: 31 of 95) together with the trend for a higher prevalence of breakdown of the fundoplication in late failure patients than in those with early failure both confirm the well-known observation that the results of an antireflux operation deteriorate with time, probably reflecting the cumulative effects of the mechanical constraints exerted upon the hiatal area over the years.

In conclusion, symptomatic failure of an antireflux operation reflects a complex anatomical situation in the hiatal area in which several abnormalities coexist. One of these, i.e. the herniation of a 360° fundoplication from the abdomen into the chest, does not pose a real problem unless disruption of the hiatal sling is incomplete, and the wrap concommitantly breaks down or slides along the stomach, because neither a perigastric nor a partial fundoplication functions properly in the chest. Neither GERD patients carrying heavy loads on a regular basis nor those having a short oesophagus are good candidates for an abdominal antireflux fundoplication.

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