CrossMark

Laparoscopic repair of paraesophageal hernia with anterior gastropexy: a multicenter study

Christopher R. Daigle · Peter Funch-Jensen ·
Dan Calatayud · Peter Rask · Bo Jacobsen ·
Teodor P. Grantcharov

Abstract

Background The approach to repair of paraesophageal hernias (PEHs) is controversial. Recent data suggest that mesh repair leads to recurrence rates similar to non-mesh approaches, while subjecting patients to mesh-associated complications. Routine fundoplication during PEH repair has been favored despite significant dysphagia rates. We present our multicenter prospective data on laparoscopic PEH repairs using a modified Boerema anterior gastropexy without fundoplication.

Methods We prospectively followed patients after modified Boerema PEH repair at three institutions. Patient demographics, perioperative data, and postoperative outcomes were evaluated. Subjective and objective outcomes were assessed via clinical assessment, follow-up questioning, endoscopy, and radiographic swallow studies.

Results A total of 101 patients were followed a mean of 10.8 (median, 12) months. We encountered 9 (8.9 %) intraoperative complications and 13 (12.9 %) postoperative complications. There was no mortality. Reflux symptoms were absent in 71 patients (70.3 %) postoperatively. Of the remaining subjects, 8 (7.9 %) had mild intermittent reflux without the need for proton pump inhibitors (PPI), 12 (11.9 %) had moderate reflux necessitating PPI as needed, and 10 (9.9 %) had reflux requiring daily PPI. Our recurrence rate, assessed at postoperative endoscopy/barium swallow, was 16.8 %. Of these, 10 (9.9 %) were small segmental recurrences and 7 (6.9 %) were large recurrences.

Conclusion Herein, we demonstrate a favorable recurrence rate while avoiding the potential major complications associated with mesh hiatoplasty. Our data tend to support a tailored approach to incorporation of fundoplication during PEH repair. Postoperative acid reflux was absent in most of our patients, and pharmacotherapy alone was sufficient for those experiencing reflux symptoms.

Keywords Laparoscopic · Paraesophageal hernia · Boerema · Gastropexy · Fundoplication

C. R. Daigle (✉) · T. P. Grantcharov
Division of General Surgery, St. Michael's Hospital, University of Toronto, 30 Bond Street, Toronto, ON M5B 1W8, Canada
e-mail: daiglec2@ccf.org; dr.crdaigle@gmail.com

P. Funch-Jensen
Aleris-Hamlet Hospital and Clinical Institute, Aarhus University Hospital, Aarhus, Denmark

D. Calatayud
Division of Surgery/Transplantation, Rigshospitalet, Blegdamsvej 9, Copenhagen, Denmark

P. Rask
Surgical Gastroenterological Dept. L, Aarhus University Hospital, Aarhus, Denmark

B. Jacobsen
Copenhagen Private Hospital, Copenhagen, Denmark

The optimal approach to the repair of paraesophageal hernias (PEHs) remains a topic fraught with controversy. Repairs were initially done by simple gastropexy, as described by Boerema, with high recurrence rates [1, 2]. Hernia sac reduction was then found to be invaluable in preventing anatomical and symptomatic recurrence [3]. Approaches eventually focused on hiatus repair using either suture or mesh hiatoplasty. Like most areas of surgery, the debate between open and laparoscopic approaches

began with the weight of the literature now supporting the safety of minimally invasive approaches [4]. Currently, the discussion continues over three main issues: the use of mesh for cruroplasty, the routine incorporation of an anti-reflux procedure, and the role of anterior gastropexy. Oelschlager et al. and Frantzides et al. published randomized trials that showed a significant reduction in recurrence rates at early follow-up with mesh cruroplasty [5, 6]. However, Oelschlager's 5-year results showed no significant difference in recurrence rates between biologic mesh and non-mesh groups [7]. While improved results have been seen with non-absorbable mesh repairs, the potentially disastrous consequences of mesh cruroplasty coupled with Oelschlager's 5-year findings have brought its use back into question [8–10].

The standard incorporation of anti-reflux procedures is another topic of significant controversy in the literature. Many surgeons add a fundoplication to their PEH repairs to prevent postoperative acid reflux. Predisposition to reflux and dissection at the gastroesophageal (GE) junction have been quoted as the justification for this [11]. However, most PEH patients deny reflux preoperatively, and the postoperative reflux encountered after repair without fundoplication can be treated medically [3]. At the same time, fundoplication has not been shown to reduce the recurrence rate of PEH repairs, and its use can lead to unwanted clinical outcomes, like dysphagia, which can be difficult to manage postoperatively [12, 13]. Furthermore, re-operation for a symptomatic recurrence can be very difficult in the presence of an anti-reflux procedure.

The aim of this study was to report the multicenter outcomes of a prospective series of 101 PEH repairs using a modified Boerema anterior gastropexy without mesh cruroplasty or an associated fundoplication.

Methods

Between October 2002 and December 2008, a total of 101 patients underwent laparoscopic PEH repair via anterior gastropexy at 1 of 3 institutions (Aarhus University Hospital in Arhus, Denmark, Gentofte Hospital in Copenhagen, Denmark, and St. Michael's Hospital in Toronto, Canada) after obtaining informed consent. Cases were done by 1 of 4 staff physicians with the aid of fellows and/or residents. Patients were offered an associated fundoplication if there was subjective or objective clinical evidence of severe acid reflux, and thus, were excluded from the main study cohort ($n = 7$). Patients were then followed prospectively, and post-procedural objective and subjective data were obtained via physician assessment, standardized questionnaires, gastroscopy, and radiological swallow study. Patient demographics, preoperative symptoms, perioperative data, related

complications, and postoperative parameters were evaluated and analyzed. The primary outcomes were evidence of recurrence at endoscopy/barium swallow study and need for acid reflux pharmacotherapy after repair. Statistical analysis was completed using IBM's SPSS $^{®}$ analytical software.

Surgical procedure

A Veress needle is introduced in the left subcostal area and is used to establish 15 mmHg capnoperitoneum. An optical trocar is then placed 15 cm below the xiphoid process, followed by three additional trocars in the left and right paramedian areas and the left anterior axillary line. A Nathanson retractor is then introduced through a 5-mm incision below the xiphoid and used to elevate the left lobe of the liver, exposing the diaphragmatic hiatus. Using an ultrasonic dissector, the dissection begins through the pars flaccida of the lesser omentum and continues toward the right crus. The peritoneum overlying the right crus is incised, and the space around the hernia sac is entered. Dissection continues in a circumferential fashion around the hernia sac. Adhesions to both pleurae are taken down with a combination of blunt and sharp dissection. Adhesions between the pleura, thoracic aorta, and the distal esophagus are carefully dissected sharply; it is important to ensure that the posterior aspects of the hernia sac and distal esophagus are fully mobilized. A Penrose drain is placed around the GE junction to provide retraction and adequate exposure. This dissection usually provides sufficient length of the intra-abdominal esophagus without tension. At the end of the dissection, all landmarks should be clearly identified: the esophagus, the thoracic aorta, and the entire length of both crura, including the posterior confluence. Non-absorbable sutures (2-0 Ethibond^{TM}) are used to approximate the diaphragmatic crura. If the defect is particularly large or the crura attenuated, this step is not performed (avoids significant tension). Six non-absorbable sutures (2-0 Ethibond^{TM}) are placed in two rows of three along the lesser curvature and are extracted using a suture passer in the left upper quadrant; it is important to ensure optimal GE angulation when placing these sutures. If using a laparoscopic approach, the abdomen is desufflated and the sutures are tied under direct visualization.

Results

Patient characteristics

A total of 101 patients were included in the study (76.2 % females, $n = 77$). The mean age was 69.3 ± 12.0 years, with a median age of 69 years (range, 37–88). Fifty-seven patients (56.4 %) had no previous abdominal or thoracic

surgery. Thirty-three participants (32.7 %) had prior lower abdominal surgery, 10 (9.9 %) had upper abdominal surgery, and 6 (5.9 %) had both. Two patients (2.0 %) had a previous thoracic PEH repair. The distribution of hernia types was 18.8 % (n = 19) type II, 68.3 % (n = 69) type III, and 12.9 % (n = 13) type IV [14].

Preoperative symptoms

Subjective "heartburn" was experienced in only 13.9 % (n = 14) of patients preoperatively. Regurgitation was present in 34.7 % (n = 35), with recurrent emesis in 53.5 % (n = 54). Significant dysphagia was reported by 55.4 % (n = 56) of participants preoperatively. Pulmonary symptoms were prevalent in this population, with 52.5 % (n = 53) complaining of dyspnea and 3.0 % (n = 3) recurrent pneumonia. Signs of gastrointestinal (G.I.) bleeding were present in 19 subjects (18.8 %). Twenty-eight patients (27.7 %) reported significant weight loss.

Operative data

Only 5 of 101 cases (5.0 %) required conversion to laparotomy. Operative times were recorded for 89 of 101 cases with a mean of 109.8 min. Primary crural approximation was achieved in 94 patients (93.1 %); the remaining 7 patients had no crura repair due to poor tissue quality and significant tension. Blood loss was negligible in 92.6 % of the cases, and only two cases had blood loss as high as 500 cc (both were splenic capsule injuries). The vast majority of cases were without intraoperative complications (n = 92; 91.1 %). Intraoperative complications included 6 (5.9 %) small pneumothoracies, 1 (1.0 %) gastrotomy (recognized and repaired), and 2 (2.0 %) splenic capsule injuries (hemostasis achieved without splenectomy).

Postoperative data

The mean length of stay in this series was 5.8 days (median, 3). Of 101 patients, 88 (87.1 %) were uncomplicated, and there was no mortality. The postoperative complications encountered are summarized in Table 1. After discharge, patients were followed for an average of 10.9 months (median, 12; range, 3–60). Symptoms related to their disease and/or complications of the procedure were assessed using a standardized symptom questionnaire and are summarized in Table 2. Reflux symptoms were absent in 71 patients (70.3 %). Of the remaining subjects, 8 (7.9 %) had mild intermittent reflux without the need for proton pump inhibitors (PPI), 12 (11.9 %) had moderate reflux necessitating PPI as needed, and 10 (9.9 %) had reflux requiring daily PPI. Recurrence was assessed via postoperative endoscopy/barium swallow study at 3 and

Table 1 Complications after PEH repair

Postoperative complication	Number (%)
No complications	88 (87.1 %)
Wound infection	3 (3.0 %)
Unspecified vascular incident	3 (3.0 %)
Myocardial infarction	2 (2.0 %)
Atrial fibrillation	2 (2.0 %)
Intra-abdominal abscess	1 (1.0 %)
Bleeding	1 (1.0 %)
Pneumonia	1 (1.0 %)

Table 2 Subjective symptoms at follow-up

Follow-up symptoms	Number (%)
Asymptomatic	70 (69.3 %)
Acid Reflux (mild to severe)	30 (29.7 %)
Emesis (occasional or recurrent)	10 (9.9 %)
Bloating	8 (7.9 %)
Dysphagia	6 (5.9 %)
Dyspnea	1 (1.0 %)

12 months and all but 5 (n = 96, 95.1 %) patients completed at least the 3-month assessment (5 patients lost after 3 month follow-up visit; no clinical evidence of recurrence). In total, 72 (71.3 %) patients had a second endoscopy/barium study at 1 year as per the protocol; the remaining patients declined repeat testing since they were without symptoms suggestive of recurrence. In total, we found endoscopic or radiographic evidence of recurrence in 17 patients (16.8 %). Of these, 10 (9.9 %) were small segmental recurrences and 7 (6.9 %) were large recurrences. Of the 17 subjects who were found to have PEH recurrence, six ultimately had a revisional surgery.

Of note, 7 patients were excluded from the main study cohort because of preoperative signs of clinically severe acid reflux (debilitating symptoms, daily PPI, esophagitis on gastroscopy). These patients were given an anterior gastropexy with associated Toupet fundoplication. This small subset of patients (mean age, 59.7; 85.7 % female) had no perioperative morbidity or mortality and was followed for a mean of 9.7 (median, 12) months. Gas bloating was prominent in 1 (14.3 %) patient, reflux in another (14.3 %), and there was no subjective dysphagia. Recurrence was objectively observed in 1 of 7 (14.3 %) patients.

Discussion

The operative approach for PEH has evolved over the years. Initially, most PEHs were repaired via an open

thoracic or abdominal approach with simple visceral reduction and gastropexy. Recurrence rates were high in these early reports and this, perhaps unjustly, led to decreased acceptance of gastropexy as a suitable repair [1, 2]. Later reports indicated that hernia sac reduction was essential in order to prevent recurrence, and this has now become a key step in the repair of PEH [3]. The discussion eventually centered on open versus minimally invasive techniques, with the weight of the literature now supporting the safety and effectiveness of the laparoscopic approach [4]. However, much debate still exists over the intraoperative approach that will have the lowest recurrence rates with an acceptably low complication rate. Two of the most controversial current topics are the role of mesh cruroplasty and the standard use of anti-reflux procedures.

Mesh versus primary cruroplasty

Randomized trials by Oelschlager et al. and Frantzides et al. showed promising reductions in recurrence rates at early follow-up when mesh was used versus primary cruroplasty (24 vs. 9 % and 22 vs. 0 %, respectively) [5, 6]. However, Oelschlager's 5-year data showed no significant difference in recurrence rates between biologic mesh and primary crural repair groups [7]. It should be noted, however, that superior recurrence rates have been reported with non-absorbable mesh hiatoplasty when compared to biologic mesh, but the long-term results remain to be seen [15]. The reported complications of mesh hiatoplasty, while infrequent, are potentially catastrophic, especially when considering the demographics of the patient population (typically old and frail) [8–10]. Esophageal stenosis, erosions, aortal bleeding, and dense fibrosis have been reported with some patients requiring re-operation for mesh removal, partial gastrectomy, or partial esophagectomy [10]. Furthermore, re-operation for clinically significant recurrence can be challenging given the local inflammatory response associated with mesh incorporation [10].

The recurrence rate in this series, assessed via upper endoscopy and upper GI study at 3 and 12 months, was 16.8 %. The majority of these were small segmental recurrences without any associated symptoms. As previously mentioned, some ($n = 24$) patients did not complete the second objective recurrence assessment at 1 year; however, none of these patients had subjective or symptomatic evidence of recurrence. Postoperative symptoms were subjectively reported in 31 subjects (30.7 %). Symptoms included reflux in 30 patients (29.7 %), occasional-to-recurrent emesis in 10 patients (9.9 %), gas bloating in 8 patients (7.9 %), dysphagia in 6 patients (5.9 %), and shortness of breath in 1 (1.0 %).

The repair of PEH is associated with a significant recurrence rate regardless of the surgical approach used for hiatoplasty. Diagnostic imaging can overestimate PEH recurrence, and perhaps this "inherent recurrence rate" is higher than is clinically significant [16]. As such, our study tends to support the use of primary hiatoplasty (with gastropexy) if crura integrity allows. Attenuated crural defects associated with thin/weak crura were left open, and a corresponding gastropexy was used. This method is associated with a reasonable recurrence rate and avoids the potentially morbid long-term complications reported with mesh usage.

Role of anti-reflux procedures

There is significant variation in the reported prevalence of acid reflux in preoperative PEH patients. Rates varying from 13 to 60 % or greater have been reported [17]. Only 13.9 % (14 of 101) of patients in this cohort reported preoperative reflux symptoms [21 of 108 (19.4 %) including the 7 patients who had an adjunctive Toupet fundoplication]. Despite this heterogeneity in prevalence, many surgeons still routinely incorporate an anti-reflux procedure as part of their PEH repair. Predisposition to reflux and dissection at the GE junction have been quoted as the justification for this; however, the role of routine fundoplication in patients who deny or have mild reflux symptoms preoperatively is unclear [11]. Fundoplication has not been shown to reduce the recurrence rate of PEH repairs, and therefore, its consideration should only be with regards to treatment or prevention of acid reflux [13].

The supradiaphragmatic herniation of stomach seen in a PEH creates an "acid pocket" that sits above and across the highly dynamic diaphragm. Boeckxstaens demonstrated that when the "acid pocket" is located above the diaphragm or is extending into the hiatal opening, 70–85 % of all transient lower esophageal sphincter relaxations (TLESR) are accompanied by acidic reflux. In contrast, when the "acid pocket" is located below the diaphragm prior to a TLESR, only 7–20 % have a corresponding acidic reflux episode [18]. Thus, the "acid pocket" is a major risk factor for significant reflux events, which could explain why some PEH patients do suffer from reflux symptoms. One would assume that a successful PEH repair should remove this "acid pocket" as a noxious contributor once the stomach is returned to its intra-abdominal location.

The disruption of the anatomic continuity of the LES and the diaphragmatic hiatus can also contribute adversely with respect to reflux, as can impaired esophageal motility [19, 20]. However, many PEH patients deny any symptoms suggestive of acid reflux. This is because many factors contribute to both the integrity and breakdown of the GE

junction pressure system and these factors appear capable of compensation [19, 20]. Cuomo et al. [20] showed that although an altered pressure topography of the GE junction can increase the susceptibility to reflux, only the associated impairment of both the diaphragmatic crura and the LES presents the true risk condition. Thus, a normally functioning LES can compensate for a low pressure defect in the crura, but a failure of both leads to complete breakdown of the system.

Preoperative endoscopic evidence of esophagitis has been reported in 13–47 % of PEH patients, but severe esophagitis or reflux is rare [17]. For these patients, an associated fundoplication should be considered. However, the role of anti-reflux procedures in patients who do not have subjective or objective evidence of acid reflux remains controversial. Unlike sliding hiatus hernias, most PEHs have normal LES pressures capable of compensating pre- and post-repair [21–23]. In the present series, 70.3 % of patients ($n = 71$) had no reflux postoperatively. Intermittent reflux without the need for medical therapy was present in 7.9 % ($n = 8$), while 11.9 % ($n = 12$) had intermittent reflux requiring PPI as needed. Daily PPI were needed in only 10 patients (9.9 %).

A major concern with the standard use of anti-reflux procedures is postoperative dysphagia. A recent meta-analysis demonstrated that 14 % of patients suffered from dysphagia after standard Nissen fundoplication at 2-year follow-up [24]. Dysphagia rates as high as 50 % have been reported after PEH repair with the addition of a fundoplication [25]. Triponez et al. showed that HH patients who underwent fundoplication experienced significantly more dysphagia and gas bloating than patients with HH and controls [26]. Dysphagia following PEH repair with fundoplication can be a very difficult problem sometimes necessitating endoscopic dilation and even surgical intervention. Furthermore, recurrence seems to be a significant issue regardless of the surgical approach taken, and recurrence of PEH after a fundoplication is more likely to be symptomatic and technically challenging to repair. There also seems to be an association between all types of HH and impaired esophageal motility, although it is unclear if this is caused by anatomical distortion or inherent contractile abnormalities [25, 27–29]. Regardless, impaired esophageal peristalsis is commonly seen in a PEH patient population that typically has normal LES function. Routine fundoplication in these patients may lead to significant dysphagia rates. Many publications support a more tailored approach to the addition of fundoplication, and the findings of this study further support this [3, 13, 21, 23, 30, 31]. The present study suggests that if severe reflux or esophagitis is present preoperatively, a fundoplication should be offered after an informed discussion. If subjective or objective evidence of significant acid reflux is not present, a fundoplication, and its associated potential complications, can be avoided. Medical therapy is usually sufficient in treating most reflux symptoms experienced postoperatively.

Role of anterior gastropexy

Boerema first described anterior gastropexy for the repair of a PEH in 1952. His initial reports showed symptomatic improvement in 90 %; thus, he concluded his recurrence rate was 10 % [1]. Later reports showed symptomatic "recurrence" in up to 60 % of patients after Boerema repair [2]. This finding led to decreased acceptance of anterior gastropexy as a suitable repair of PEH and the focus shifted to cruroplasty in an attempt to minimize recurrence. These early procedures were done without hernia sac reduction, a component of the repair now deemed essential when recurrence is considered. Subsequent case reports have shown acceptable recurrence rates using a modified Boerema gastropexy both with and without fundoplication [3, 4, 17, 32–34]. These reports demonstrated a significant correlation between lack of gastropexy and recurrence [3, 4, 33]. In the present study of 101 PEH patients, we found promising results using a modified Boerema gastropexy without mesh hiatoplasty, with an overall recurrence rate of 16.8 % ($n = 17$). Of these, 10 (9.9 %) were small segmental recurrences and 7 (6.9 %) were large recurrences. This rate is comparable to the other recurrence rates in the literature and this tends to support anterior gastropexy as a suitable method for PEH repair. Anterior gastropexy achieves all treatment objectives for patients with symptomatic PEH with minimal morbidity and mortality and avoids the potential long-term complications of mesh cruroplasty and routine fundoplication.

There are limitations to this study. Firstly, this is not a randomized study and 7 patients did get a different procedure than the main study cohort. Secondly, not all patients completed the intended 1-year follow-up period with repeat objective recurrence assessment. As previously stated, while these patients did deny symptomatology suggestive of recurrence, our "recurrence rate" could be underreporting radiographic/endoscopic recurrence. Further randomized prospective trials are needed to establish the true role of the modified Boerema gastropexy in the treatment of PEH.

Conclusion

The repair of PEH appears to have an inherent recurrence rate regardless of the operative approach. Herein, we demonstrate a favorable recurrence rate while avoiding the

potential major complications associated with mesh hiatoplasty. Our data also support a more tailored approach to the incorporation of a fundoplication at the time of PEH repair. Postoperative acid reflux was absent in most of our patients, and pharmacotherapy alone was sufficient for those who did experience reflux symptoms.

References

1. Boerema WJ (1969) Anterior gastropexy: a simple operation for hiatus hernia. Aust N Z J Surg 39(2):173–175
2. Davies CJ (1975) A survey of the results of the Boerema anterior gastropexy for hiatus hernia over a 4-year period. Br J Surg 62(1):19–22
3. Peet DLVD, Klinkenberg-Knol EC, Poza AA, Sietses C, Eijsbouts QAJ, Cuesta MA (2000) Laparoscopic treatment of large paraesophageal hernias: both excision of the sac and gastropexy are imperative for adequate surgical treatment. Surg Endosc 14:1015–1018
4. Poncet G, Robert M, Roman S, Boulez J-C (2010) Laparoscopic repair of large hiatal hernia without prosthetic reinforcement: late results and relevance of anterior gastropexy. J Gastrointest Surg 14(12):1910–1916
5. Oelschlager BK, Pellegrini CA, Hunter J, Soper N, Brunt M, Sheppard B, Jobe B et al (2006) Biologic prosthesis reduces recurrence after laparoscopic paraesophageal hernia repair: a multicenter, prospective, randomized trial. Ann Surg 244(4):481–490
6. Frantzides CT, Madan AK, Carlson MA, Stavropoulos GP (2002) A prospective, randomized trial of laparoscopic polytetrafluoroethylene (PTFE) patch repair vs simple cruroplasty for large hiatal hernia. Arch Surg 137(6):649–652
7. Oelschlager BK, Pellegrini CA, Hunter JG, Brunt ML, Soper NJ, Sheppard BC, Polissar NL et al (2011) Biologic prosthesis to prevent recurrence after laparoscopic paraesophageal hernia repair: long-term follow-up from a multicenter, prospective, randomized trial. J Am Coll Surg 213(4):461–468
8. Zügel N, Lang RA, Kox M, Hüttl TP (2009) Severe complication of laparoscopic mesh hiatoplasty for paraesophageal hernia. Surg Endosc 23(11):2563–2567
9. Tatum RP, Shalhub S, Oelschlager BK, Pellegrini CA (2008) Complications of PTFE mesh at the diaphragmatic hiatus. J Gastrointest Surg 12(5):953–957
10. Stadlhuber RJI, Sherif AE, Mittal SK, Fitzgibbons RJ Jr, Michael Brunt L, Hunter JG, Demeester TR, Swanstrom LL, Daniel Smith C, Filipi CJ (2009) Mesh complications after prosthetic reinforcement of hiatal closure: a 28-case series. Surg Endosc 23(6):1219–1226
11. Casabella F, Sinanan M, Horgan S, Pellegrini CA (1996) Systematic use of gastric fundoplication in laparoscopic repair of paraesophageal hernias. Am J Surg 171(5):485–489
12. Watson DI, de Beaux AC (2001) Complications of laparoscopic antireflux surgery. Surg Endosc 15(4):344–352
13. Furnée EJB, Draaisma WA, Simmermacher RK, Stapper G, Broeders IAMJ (2010) Long-term symptomatic outcome and radiologic assessment of laparoscopic hiatal hernia repair. Am J Surg 199(5):695–701
14. Hyun JJ, Bak Y-Tae (2011) Clinical significance of hiatal hernia. Gut Liver 5(3):267–277
15. Antoniou SA, Antoniou GA, Koch OO, Pointner R, Granderath FA (2012) Lower recurrence rates after mesh-reinforced versus simple hiatal hernia repair: a meta-analysis of randomized trials. Surg Laparosc Endosc Percutan Tech 22(6):498–502
16. Watson DI (2011) Evolution and development of surgery for large paraesophageal hiatus hernia. World J Surg 35(7):1436–1441
17. Rakić S, Pesko P, Dunjić MS, Gerzić Z (1994) Paraoesophageal hernia repair with and without concomitant fundoplication. Br J Surg 81(8):1162–1163
18. Boeckxstaens GE (2010) Alterations confined to the gastrooesophageal junction: the relationship between low LOSP, TLOSRs, hiatus hernia and acid pocket. Best Pract Res Clin Gastroenterol 24(6):821–829
19. Kahrilas PJ, Lin S, Chen J, Manka M (1999) The effect of hiatus hernia on gastro-oesophageal junction pressure. Gut 44(4):476–482
20. Cuomo R, Grasso R, Sarnelli G, Bruzzese D, Bottiglieri ME, Alfieri M, Sifrim D et al (2001) Role of diaphragmatic crura and lower esophageal sphincter in gastroesophageal reflux disease: manometric and pH-metric study of small hiatal hernia. Dig Dis Sci 46(12):2687–2694
21. Ellis FH, Crozier RE, Shea JA (1986) Paraesophageal hiatus hernia. Arch Surg 121(4):416–420
22. Walther B, DeMeester TR, Lafontaine E, Courtney JV, Little AG, Skinner DB (1984) Effect of paraesophageal hernia on sphincter function and its implication on surgical therapy. Am J Surg 147(1):111–116
23. Geha AS, Massad MG, Snow NJ, Baue AE (2000) A 32-year experience in 100 patients with giant paraesophageal hernia: the case for abdominal approach and selective antireflux repair. Surgery 128(4):623–630
24. Broeders JA, Sportel IG, Jamieson GG, Nijjar RS, Granchi N, Myers JC, Thompson SK (2011) Impact of ineffective oesophageal motility and wrap type on dysphagia after laparoscopic fundoplication. Br J Surg 98(10):1414–1421
25. Swanstrom LL, Jobe BA, Kinzie LR, Horvath KD (1999) Esophageal motility and outcomes following laparoscopic paraesophageal hernia repair and fundoplication. Am J Surg 177(5):359–363
26. Triponez F, Dumonceau J-M, Azagury D, Volonte F, Slim K, Mermillod B, Huber O et al (2005) Reflux, dysphagia, and gas bloat after laparoscopic fundoplication in patients with incidentally discovered hiatal hernia and in a control group. Surgery 137(2):235–242
27. Clouse RE, Eckert TC, Staiano A (1986) Hiatus hernia and esophageal contraction abnormalities. Am J Med 81(3):447–450
28. Gordon C, Kang JY, Neild PJ, Maxwell JD (2004) The role of the hiatus hernia in gastro-oesophageal reflux disease. Aliment Pharmacol Ther 20(7):719–732
29. Kasapidis P, Vassilakis JS, Tzovaras G, Chrysos E, Xynos E (1995) Effect of hiatal hernia on esophageal manometry and pH-metry in gastroesophageal reflux disease. Dig Dis Sci 40(12):2724–2730
30. Hawasli A, Zonca S (1998) Laparoscopic repair of paraesophageal hiatal hernia. Am Surgeon 64(8):703–710
31. Morris-Stiff G, Hassn A (2008) Laparoscopic paraoesophageal hernia repair: fundoplication is not usually indicated. Hernia 12(3):299–302
32. Agwunobi AO, Bancewicz J, Attwood SE (1998) Simple laparoscopic gastropexy as the initial treatment of paraoesophageal hiatal hernia. Br J Surg 85(5):604–606
33. Ponsky J, Rosen M, Fanning A, Malm J (2003) Anterior gastropexy may reduce the recurrence rate after laparoscopic paraesophageal hernia repair. Surg Endosc 17(7):1036–1041
34. Kloek JJ, van de Laar GAG, Deurloo JA, Aronson DC, Benninga MA, Taminiau JAJM, Heij HA (2006) Long-term results of boerema anterior gastropexy in children. J Pediatr Gastroenterol Nutr 43(1):71–76