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Esophagogastric Junction Outflow Obstruction and Hiatal Hernia: Is Hernia Repair Alone Sufficient?

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ABSTRACT

Introduction: Esophagogastric junction outflow obstruction (EGJOO) is attributed to primary/idiopathic causes or secondary/mechanical causes, including hiatal hernias (HH). While patients with HH and EGJOO (HH+EGJOO) may undergo HH repair without myotomy, it is unclear if an underlying motility disorder is missed by therapy which addresses only the secondary EGJOO cause. The goal of this study was to determine if HH repair alone is sufficient management for HH+EGJOO patients.

Methods: A retrospective review of patients who underwent HH repair between January 1, 2016 and January 31, 2020 was performed. Patients who underwent high-resolution esophageal manometry

(HREM) within one year before HH repair were included. Patients with and without EGJOO on pre-operative HREM were compared.

Results: Sixty-three patients were identified. Pre-operative HREM findings included: 43 (68.3%) normal, 13 (20.6%) EGJOO, 4 (6.3%) minor disorder or peristalsis, 2 (3.2%) achalasia, and 1 (1.6%) major disorder of peristalsis. No differences between patients with EGJOO or normal findings on pre-operative manometry were found in pre-operative demographics/risk factors, pre-operative symptoms, and pre-operative HREM, except higher integrated relaxation pressure in EGJOO patients. No differences were noted in length of stay, 30-day complications, long-term persistent symptoms, or recurrence with mean follow-up of 26-months. Of the 3 (23.1%) EGJOO patients with persistent symptoms, 2 underwent HREM demonstrating persistent EGJOO and none required endoscopic/surgical myotomy.

Conclusion: Most HH+EGJOO patients experienced symptom resolution following HH repair alone and none required additional intervention to address a missed primary motility disorder. Further study is required to determine optimal management of patients with persistent EGJOO following HH repair.

Key Words: EGJOO, Esophageal motility, Hiatal hernia, Manometry, Myotomy.

INTRODUCTION

Esophagogastric junction outflow obstruction (EGJOO) is an esophageal motility disorder defined by the Chicago Classification Version 30.0 as an elevated lower esophageal sphincter (LES) integrated relaxation pressure (IRP) without significant derangements in peristalsis which would meet the criteria for achalasia.[1] Since the institution of the new guidelines in 2014, the rate of diagnosis of EGJOO on high-resolution esophageal manometry (HREM) studies has increased tenfold with the reported incidence on HREM now ranging from 3% to 25%.[2–4] It is well recognized that the diagnostic criteria for EGJOO comprises a heterogenous group of clinical subsets which are broadly classified into two categories, either primary (idiopathic) EGJOO and secondary (mechanical) EGJOO.[5] Furthermore, manometric artifact, chronic opioid use, and even patient positioning during HREM can lead to isolated elevation of the IRP and can add to uncertainty in the etiology of EGJOO.[1,6,7]

Primary EGJOO, also termed "functional" or "idiopathic" EGJOO, is the diagnosis assigned when no anatomic cause for esophageal outflow obstruction is found. Because of manometric, clinical, and histologic overlap

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between EGJOO and achalasia, many experts consider primary EGJOO as a precursor or variant of achalasia.[1,8,9] The rate of conversion to achalasia is unknown, though numerous studies cite examples of patient progression from EGJOO to achalasia.[4,10] Secondary EGJOO, also termed "structural" or "mechanical" EGJOO, is caused by the presence of an anatomic abnormality leading to outflow obstruction. Causes include hiatal hernias, prior fundoplications, and esophageal pathology including strictures, webs, or diverticulae.[8,11,12] The published rate of EGJOO cases with a secondary anatomic cause is widely variable, ranging from 13% to 65%.[3,8,11–13]

In cases of secondary EGJOO, hiatal hernia (HH) is the most commonly cited anatomic cause and accounts for up to 70% of secondary EGJOO cases.[12] For surgeons managing HH, several key questions are unanswered by the current body of literature in regard to patients with the pre-operative finding of EGJOO. First, while a significant proportion of EGJOO cases are related to HH, the incidence and clinical significance of EGJOO in patients with HH (HH+EGJOO) is unknown. Secondly, while HH+EGJOO patients often undergo HH repair without myotomy, it is unclear if an underlying or developing primary motility disorder is missed by therapy which addresses only a secondary EGJOO cause. The surgical outcomes of HH+EGJOO patients undergoing HH repair have not been published to date. Therefore, the goal of this retrospective review was to identify HH+EGJOO patients and characterize their outcomes after HH repair with the hypothesis that HH repair alone would be sufficient treatment in this subset of patients.

METHODS

A single institution retrospective review of the electronic medical record (EMR) was performed for patients who underwent HH repair between January 1, 2016 and January 31, 2020. All operations were performed by one of six board-certified general surgeons at an academically-affiliated tertiary referral center. Institutional review board approval was obtained.

Institutional billing data were queried to identify all patients who had undergone HH repair and HREM. Open, laparoscopic, robotic, and minimally-invasive converted to open techniques were included. Patients were included if they had undergone HREM within one year prior to HH repair. Cases were excluded if the patient was < 18 years old at the time of operation, if HREM was not performed prior to surgery, and if HREM was performed > 1 year prior to surgery.

All HREM cases were performed using a standardized protocol using the Sierra Scientific ManoScan 360 system (Sierra Scientific Instruments, Los Angeles, California, USA).[14,15] All studies were interpreted using definitions provided by the Chicago Classification of esophageal motility disorders, version 30.0.[1] The technical details of the pre-operative work-up and HH repair were determined by the preference of the operating surgeon and were not standardized. The pre-operative characterization of the HH was variably performed based on surgeon preference, and included use of barium swallow, computed tomography, and upper endoscopy. Likewise, the indication and use of pre-operative HREM was determined by the individual surgeon and not standardized. Routine pre-operative HREM was not obtained on every patient undergoing HH repair at our institution during the study period. The operative approach, use of mesh, and use of concurrent fundoplication, gastropexy, and gastroplasty were decided by the surgeon at the time of operation.

Demographic information including age, gender, body mass index (BMI), American Society of Anesthesiologists (ASA) classification, comorbidities, and smoking history were recorded. Data on prior foregut surgery were collected including history of prior HH repair and presence of prior mesh. Pre-operative symptoms were recorded using a standardized clinical document and included the presence of dysphagia, regurgitation, chest pain, respiratory symptoms, and subjective weight loss prior to HH repair. Pre-operative details of the HH including classification (Type I-IV) were collected.[16] Perioperative data were collected including the operative technique, type and number of crural sutures, mesh type and fixation, procedural length; and concurrent procedures including fundoplication, gastropexy, and gastroplasty.

Postoperative follow-up also varied by surgeon but generally included a clinic visit within one month of surgery and additional follow-up based on clinical improvement at that time. Patients did not routinely undergo radiographic surveillance or postoperative HREM following hospital discharge. Postoperative data including length of stay, symptoms reported at follow-up, complications, readmission, and recurrence of HH were collected from the EMR. The primary outcome measure was the presence of persistent symptoms, which was defined as documentation of a patient complaint of symptoms at the standard postoperative visit which prompted an additional clinic appointment or diagnostic test beyond the standard

follow-up practice. Given the nature of postoperative symptoms following foregut surgery, patients with mild or improving symptoms at their postoperative visit were not classified as experiencing "persistent symptoms" unless further follow-up was initiated.

Pre-operative risk factors, operative details, and postoperative outcomes were compared between patients with and without EGJOO on pre-operative HREM utilizing student's T-test and Fisher's exact test. Continuous data is presented as mean ± standard deviation.

RESULTS

Sixty-three patients were identified that met the inclusion criteria. Demographic information of these 63 patients is shown in **Table 1** The mean age was 64.4 ± 11 and included 51 (81.0%) females and 12 (19.0%) males. The mean ASA Class was 2.4 ± 00.5 and the mean BMI at the time of operation was 30.6 ± 4. Forty (63.5%) patients were classified as obese (BMI ≥ 30). **Table 1** also demonstrates the distribution of HH type as determined by pre-operative imaging. The group included 18 (28.5%) Type I, 6 (9.5%) Type II, 35 (55.6%) Type III, and 4 (6.3%) Type IV hiatal hernias. The prior surgical history of foregut procedures is also shown in **Table 1** and includes prior HH repair in 3 (4.8%), Nissen fundoplication in 5 (7.9%), Roux-en-Y gastric bypass in 3 (4.8%), sleeve gastrectomy in 1 (1.6%), and open repair of a Zenker's diverticulum in 1 (1.6%).

Table 2 shows the distribution of esophageal dysmotility diagnoses as determined by pre-operative HREM. Forty-three (68.3%) patients had manometry findings within normal range while 13 (20.6%) were diagnosed with EGJOO. Two patients were diagnosed with achalasia with the following distribution: 0 (0%) Type I, 1 (1.6%) Type II, and 1 (1.6%) Type III. One (1.6%) patient was diagnosed with Distal Esophageal Spasm (DES), while no patients were diagnosed with other major disorders of peristalsis including jackhammer esophagus or absent contractility. Four (6.3%) patients were diagnosed with minor disorders of peristalsis, with 3 (4.8%) with ineffective motility (IEM) and 1 (1.6%) with fragmented peristalsis.

The 13 EGJOO patients were then compared to the 43 patients with normal manometry (**Table 2**). The seven patients with achalasia and major or minor disorders of peristalsis were excluded from this analysis. **Table 3** shows differences in pre-operative demographics and

Table 1.
Demographic and Surgical History of Included Patients

	n = 63
Age	64.4 ± 11
Gender	
Male	12 (19.0%)
Female	51 (81.0%)
American Association of Anesthesiologists Class	2.4 ± 0.5
Body Mass Index	30.6 ± 4
Hiatal Hernia Type	
Type I	18 (28.5%)
Type II	6 (9.5%)
Type III	35 (55.6%)
Type IV	4 (6.3%)
Prior Foregut Surgery	11 (17.5%)
Hiatal hernia repair	3 (4.8%)
Nissen fundoplication	5 (7.9%)
Roux-en-Y gastric bypass	3 (4.8%)
Sleeve gastrectomy	1 (1.6%)
Zenker's diverticulum repair	1 (1.6%)

Table 2.
Distribution of Esophageal Motility Disorders of Patients with High-resolution Esophageal Manometry Prior to Hiatal Hernia Repair. Further Analysis Was Performed by Comparing the 43 Patients with Normal High-resolution Esophageal Manometry Versus the Thirteen Patients with Esophagogastric Junction Outflow Obstruction (Highlighted In Bold)

	n = 63	
Normal	**43 (68.3%)**	**Included in analysis**
Esophagogastric Junction Outflow Obstruction	**13 (20.6%)**	
Achalasia		
Type I	0	
Type II	1 (1.6%)	
Type III	1 (1.6%)	
Major Disorder of Peristalsis		
Distal Esophageal Spasm	1 (1.6%)	Excluded from analysis
Jackhammer esophagus	0 (0%)	
Absent contractility	0 (0%)	
Minor Disorder of Peristalsis		
Ineffective motility	3 (4.8%)	
Fragmented peristalsis	1 (1.6%)	

Table 3.
Comparison of Pre-operative Variables between Patients with Esophagogastric Junction Outflow Obstruction and Patients with Normal Findings on Pre-operative High-resolution Esophageal Manometry

	EGJOO (n = 13)	Normal Manometry (n = 43)	*p*-Value
Pre-operative Factors			
Gender (Male)	3 (23.1%)	8 (18.6%)	0.70
Age	67.8	63.7	0.10
ASA Class	2.6	2.4	0.08
Body Mass Index	31.6	30.3	0.18
Anemia	3 (23.1%)	3 (10.3%)	0.13
Hepatic Insufficiency	0 (0%)	0 (0%)	-
Hypertension	6 (46.1%)	17 (39.5%)	0.33
Diabetes	1 (7.7%)	2 (4.7%)	0.55
COPD	0 (0%)	3 (7.5%)	-
CAD	0 (0%)	2 (4.7%)	-
CHF	0 (0%)	1 (2.3%)	-
Anticoagulation	1 (7.7%)	5 (11.6%)	1.00
Antiplatelet	2 (15.4%)	6 (14.0%)	1.00
Immunosuppression	0 (0%)	0 (0%)	-
Current smoker	0 (0%)	1 (2.3%)	-
Smoker, Quit > 30 day	1 (7.7%)	5 (11.6%)	1.00
Any Smoker	1 (7.7%)	6 (14.0%)	1.00
GERD	12 (92.3%)	30 (69.8%)	0.15
PPI use	12 (92.3%)	33 (76.7%)	0.43
Foregut Surgery history	3 (23.1%)	6 (14.0%)	0.42
Pre-operative Symptoms			
Dysphagia	8 (61.5%)	33 (76.7%)	0.30
Regurgitation	5 (38.5%)	23 (53.5%)	0.53
Chest pain	4 (30.8%)	5 (11.6%)	0.19
Respiratory symptoms	3 (23.1%)	5 (11.6%)	0.37
Weight loss	1 (7.7%)	2 (4.7%)	0.55
Pre-operative Manometry			
Mean Basal UES pressure (mm Hg)	59.9	63.0	0.39
Mean amplitude in distal esophagus (mm Hg)	86.1	79.0	0.27
DCI (mm Hg·s·cm)	1756.1	1553.2	0.28
Distal latency (s)	6.45	6.13	0.21
% ineffective swallows	20.8%	35.2%	0.10
Mean basal LES pressure (mm Hg)	38.6	48.8	0.43
IRP (mm Hg)	22.3	3.3	< 0.001
Hiatal Hernia Type			
Type I	2 (15.4%)	13 (30.2%)	0.48
Type II	3 (23.1%)	2 (4.7%)	0.08

Table 3. Continued

	EGJOO (n = 13)	Normal Manometry (n = 43)	*p*-Value
Type III	6 (46.2%)	26 (60.5%)	0.52
Type IV	2 (15.4%)	2 (4.7%)	0.23

Abbreviations: EGJOO, esophagogastric junction outflow obstruction; ASA, American Society of Anesthesiologists; COPD, chronic obstructive pulmonary disease; CAD, coronary artery disease; CHF, congestive heart failure; GERD, gastroesophageal reflux disorder; PPI, proton pump inhibitor; UES, upper esophageal sphincter; DCI, delayed cerebral ischemia; LES, lower esophageal sphincter; IRP, idiopathic recurrent pancreatitis.

comorbidities, pre-operative symptoms, pre-operative manometry, and HH type. There were no differences in pre-operative demographics including age (67.8 vs 63.7 years), ASA Class (2.6 vs 2.4), BMI (31.6 vs 30.3), and gender ((male) 23.1% vs 18.6%) ($P > .05$). There were no differences in pre-operative comorbidities including hypertension, diabetes, coronary artery disease, and chronic obstructive pulmonary disease ($P > .05$). Additionally, no differences in use of anticoagulation, antiplatelet, or immunosuppression were present ($P > .05$). There was no difference in smoking history (7.7% vs 14.0%, $P > .05$) between the two groups. Finally, there were not differences in pre-operative gastroesophageal reflux disease (GERD) diagnosis (92.3% vs 69.8%), proton-pump inhibitor (PPI) use (92.3% vs 76.7%), and prior foregut surgical history (23.1% vs 14.0%) ($P > .05$) (**Table 3**).

Pre-operative symptoms were compared between the two groups with no differences found in rates of dysphagia (61.5% vs 76.7%), regurgitation (38.5% vs 53.5%), chest pain (30.8% vs 11.6%), respiratory symptoms (23.1% vs 11.6%), and weight loss (7.7% vs 4.7%) ($P > .05$). No differences in pre-operative HREM measurements including mean basal upper esophageal sphincter (UES) pressure, distal contractile index (DCI), distal latency, percentage of ineffective swallows, and mean basal lower esophageal sphincter (LES) pressure were identified ($P > .05$). As expected, integrated relaxation pressure (IRP) was higher in the EGJOO group (220.3 vs 30.3 mmHg, $P < .001$). There were no differences in the distribution of HH types I-IV ($P > .05$) (**Table 3**).

Table 4 shows a comparison of operative details between the EGJOO group and the group with normal manometry. Use of gastropexy (76.9% vs 32.6%, $P = .009$) was more common in the EGJOO group, with no differences in operative time (184.5 vs 218.8 minutes) or in rates of fundoplication (38.5% vs 60.5%), gastroplasty (15.4% vs 7.5%), conversion to open (7.7% vs 0%), mesh use (38.5% vs 56.1%), or robotic techniques (23.1% vs 4.7%) ($P > .05$). Notably, no patients in either group underwent myotomy. There were no differences in the number of crural sutures placed (4.6 vs 4.5) and no difference in the rate of intraoperative complications (7.2% vs 4.7%) ($P > .05$). Intraoperative complications included gastrotomy (2) and pneumothorax requiring pigtail catheter placement (1).

Also shown in **Table 4** are the postoperative outcomes between patients with EGJOO versus normal findings on pre-operative manometry. There were no differences in length of stay (2.8 vs 2.6 days) between the two groups. Additionally, no differences were seen in rates or readmission (7.7% vs 4.7%), postoperative complications (7.7% vs 16.3%), HH recurrence (15.4% vs 7.0%), or persistent symptoms (23.1% vs 14.0%) between the two groups ($P > .05$). The mean follow-up duration was 26 months.

Table 5 shows the long-term outcomes of the 3 (23.1%) EGJOO patients who experienced persistent symptoms postoperatively. Two of the three patients underwent HREM postoperatively which demonstrated EGJOO in both cases. The third patient did not undergo HREM but did undergo a swallow study that demonstrated esophageal dysmotility with narrowing at the gastroesophageal junction and delayed passage of a tablet. In the follow-up period, two patients underwent therapy for symptoms unrelated to EGJOO. Notably, no patients required endoscopic or surgical myotomy during the follow-up period. Additionally, all three patients with persistent symptoms and concern for postoperative EGJOO had either a history of foregut surgery (Roux-en-Y gastric bypass [RYGB] or prior HH repair) or underwent an additional foregut surgery at the time of the HH repair (Nissen fundoplication). Finally, two of the three patients experienced eventual resolution of symptoms while one patient had continued symptoms of esophageal spasms but resolution of dysphagia.

DISCUSSION

This single-institution retrospective study is, to our knowledge, the first report in the literature on the outcomes in a

Table 4.
Comparison of Operative and Postoperative Variables Between Patients Esophagogastric Junction Outflow Obstruction and Patients with Normal Findings on Pre-operative High-resolution Esophageal Manometry

	EGJOO (n = 13)	Normal Manometry (n = 43)	*p*-Value
Operative Details			
Operative time (min)	184.5	218.8	0.18
Mesh use	5 (38.5%)	25 (56.1%)	0.34
Myotomy	0 (0%)	0 (0%)	-
Fundoplication	5 (38.5%)	26 (60.5%)	0.21
Gastropexy	10 (76.9)	14 (32.6%)	0.009
Gastroplasty	2 (15.4%)	3 (7.5%)	0.58
Conversion to open	1 (7.7%)	0 (0%)	-
Robotic surgery	3 (23.1%)	2 (4.7%)	0.08
# of crural sutures	4.6	4.5	0.46
Intraoperative complication	1 (7.2%)	2 (4.7%)	0.55
Postoperative Outcomes			
LOS	2.8	2.6	0.39
Readmission	1 (7.7%)	2 (4.7%)	0.55
Complication	1 (7.7%)	7 (16.3%)	0.67
Recurrence	2 (15.4%)	3 (7.0%)	0.58
Persistent symptoms	3 (23.1%)	6 (14.0%)	0.42

Abbreviations: EJOO, esophagogastric junction outflow obstruction; LOS, length of stay.

series of HH patients with EGJOO (**Figure 1**) where a myotomy was not performed at the time of their foregut operation. This study identified a 20.6% incidence of EGJOO on pre-operative HREM prior to HH repair and identified no differences between EGJOO patients and patients with normal pre-operative manometry in terms of demographics, comorbidities, foregut surgical history, HH classification, or pre-operative symptoms. Further, postoperative outcomes including readmissions, complications, recurrence, and persistent symptoms were not different between the two groups. Finally, while three (23.1%) EGJOO patients experienced persistent symptoms after HH repair, all three had radiographically-suggested esophageal motility disorders postoperatively (**Figure 2**), and two underwent postoperative HREM demonstrating persistent EGJOO, no patients required surgical or endoscopic myotomy for control of their symptoms during the follow-up period of over two years and no patients developed HREM-proven achalasia. Importantly, of the three patients with persistent symptoms following surgery, all three had undergone additional foregut surgery either prior to, or concurrent with, their HH repair such as a RYGB or Nissen fundoplication which could have accounted for their persistent symptoms and manometric findings of persistent EGJOO. Therefore, there are no patients in this study in whom a diagnosis of primary EGJOO or achalasia was suspected in the postoperative follow-up period.

These findings suggest that HH repair alone is sufficient initial treatment for HH+EGJOO patients. Historically, there has been concern that an underlying primary motility disorder may be missed by HH repair alone which addresses only a secondary EGJOO cause. Although the rate of conversion from EGJOO to achalasia is unknown, this clinical progression has been documented in several recent series.[3, 4,10] In addition to these findings, significant overlap in clinical and manometric presentation have led many experts to suggest that primary EGJOO may be a precursor or variant of achalasia. Finally, several studies have recently reported a high rate of concurrent esophageal body manometric abnormalities in EGJOO patients including both major and minor disorders of peristalsis.[4, 17]

Given these findings and the knowledge that HH repair alone would not be expected to treat any underlying or developing primary motility disorders, the question of a

Table 5.
Long-Term Outcomes of Three Hiatal Hernia + Esophagogastric Junction Outflow Obstruction Patients with Persistent Symptoms following Hiatal Hernia Repair

Patient #	Age/ Gender	History	Surgery	Symptoms	Manometry	Other Diagnostics	Intervention	Resolution of Symptoms
1	59 F	Hx of RYGB, developed herniated gastric pouch	Lap HH repair, posterior cruroplasty, mesh placement, gastropexy	Persistent dysphagia, > 6 months	Yes (POD 67): EGJOO	Swallow study suggested possible concurrent cricopharyngeal dysfunction	Botox injection to upper esophageal sphincter	Resolved
2	49 F	Hx of chronic GERD and type III HH	Robotic HH repair, posterior cruroplasty, Nissen fundoplication	Persistent dysphagia and esophageal spasm, > 6 months	Yes (POD 209): EGJOO	Normal pH study; normal swallow study; normal gastric emptying study	Nitroglycerin for spasm symptoms	Persistent spasm symptoms, dysphagia resolved
3	76 F	Hx of HH repair via thoracotomy 8 years prior with recurrent HH	Robotic HH repair, posterior cruroplasty, mesh placement, gastropexy	Persistent dysphagia, > 3 months	Not performed	Swallow study showed esophageal dysmotility w/ mild narrowing at GE junction and delayed passage of tablet	None	Resolved

Abbreviations: Hx, history; RYGB, Roux-n-Y gastric bypass; GERD, gastroesophogel reflux disorder; HH, hiatal hernia; POD, postoperative delerium.

concurrent esophagomyotomy at the time of HH repair is logical. EGJOO has been successfully managed with laparoscopic Heller myotomy and Dor fundoplication, achieving complete clinical and manometric resolution.[18] Additionally, concurrent HH repair and esophagomyotomy has been described in the literature for successful management of achalasia patients with concomitant HHs. In this study by Ushimaru et al., the presence of a HH did not increase the risk of mucosal perforation during myotomy.[19] However, gastroesophageal reflux and esophageal leak are known complications of esophagomyotomy.[20–22] Our data suggest the addition of this procedure to all HH repairs in HH+EGJOO patients would be unwarranted. As evidenced in our study, 76.9% of HH+EGJOO patients had complete long-term symptomatic resolution after HH repair alone. Further, in the 23.1% of patients with persistent symptoms, none of these patients went on to require therapy targeted at LES relaxation including pharmacologic methods, Botox injection, pneumatic dilation, or surgical or endoscopic myotomy. Therefore, there is no compelling evidence in this study that any of the HH+EGJOO patients would have benefitted from esophagomyotomy at the time of their HH repair.

Several techniques are emerging which may better distinguish primary from secondary EGJOO, which could be useful in identifying patients who would benefit from esophagomyotomy at the time of HH repair. Babaei et al. recently demonstrated that pharmacologic interrogation of EGJOO patients using amyl nitrite to induce LES relaxation could be used to identify a subgroup of patients who would be expected to benefit

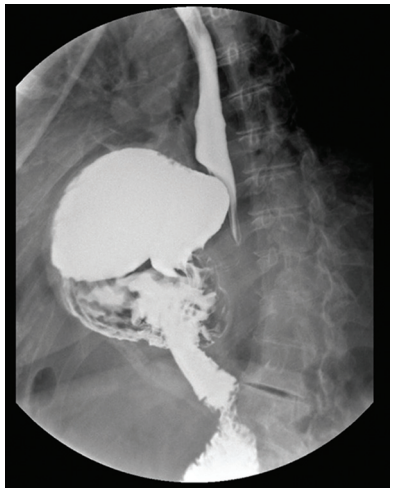

Figure 1. Pre-operative swallow study on a patient with a hiatal hernia and esophagogastric junction outflow obstruction diagnosis on underwent high-resolution esophageal manometry.

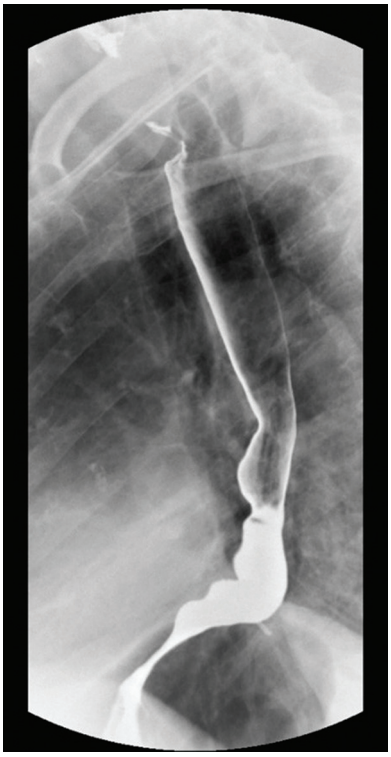

Figure 2. Hiatal hernia and esophagogastric junction outflow obstruction patient with persistent esophagogastric junction outflow obstruction after hiatal hernia repair.

from LES ablative therapies, however, the sensitivity and specificity of this test in the setting of a secondary EGJOO cause such as HH is unknown.[23] Endoscopic functional luminal impedance planimetry (EndoFLIP) technology has recently been shown to differentiate EGJOO causes and to identify EGJOO patients who are most likely to benefit from achalasia therapy based upon the measured distensibility index of the EGJ.[24, 25] Further development and widespread adoption of such techniques may eventually facilitate identification of HH+EGJOO patients who would benefit from esophagomyotomy at the time of HH repair.

Further strengthening the argument that HH repair alone is sufficient for HH+EGJOO patients is the well-established safety and clinical benefit of endoscopic therapy for both EGJOO and achalasia. In a study of 33 patients with idiopathic EGJOO, Clayton et al. demonstrated a 67% rate of subjective symptom relief following pneumatic dilation of the LES.[26] Per-oral endoscopic myotomy (POEM) has also been demonstrated in several trials to treat EGJOO with a clinical success rate greater than 93% and associated lowering of the IRP by greater than 10 mmHg on average.[27,28] POEM has also been utilized safely and effectively in patients with prior foregut surgery including RYGB and Heller myotomy and therefore represents a potential therapy for patients who develop symptoms of esophageal motility disorders after HH repair.[29–31] Given the known safety and efficacy of endoscopic techniques in the management of EGJOO and related primary motility disorders, esophagomyotomy can likely be reserved as secondary therapy for patients with persistently symptomatic EGJOO or the presumably small fraction who progress to achalasia. However, further study is required to confirm the safety and efficacy of POEM in this population.

This study was susceptible to several limitations inherent to its retrospective nature. First, the study group is heterogenous in terms of the size and type of hiatal hernia and prior and concurrent foregut procedures, making interpretation of the cause of EGJOO or persistent symptoms within specific subgroups difficult. Additionally, given the retrospective nature, no patients in this series underwent further attempts at differentiating primary from secondary EGJOO such as EndoFLIP. Finally, because pre-operative HREM was not routinely performed on every HH patient, the incidence of EGJOO in HH patients cited in this study may not be representative of the true incidence in all HH patients. Larger prospectively collected data should be collected to perform relevant subgroup analyses, study pre-operative differentiation of primary versus secondary EGJOO causes and confirm the preliminary data on surgical outcomes of HH+EGJOO patients reported in this series.

CONCLUSION

Following HH repair alone, patients with EGJOO versus normal findings on pre-operative manometry have equivalent postoperative outcomes. This study demonstrated that most HH+EGJOO patients experienced symptom resolution following HH repair alone and no patients required additional endoscopic or surgical myotomy during the follow-up period of greater than two years. No compelling evidence that HH+EGJOO patients would have benefitted from esophagomyotomy at the time of their HH repair to address an underlying primary motility disorder was identified. Further study is required to determine the optimal management of HH+EGJOO patients with persistent EGJOO following HH repair, including the appropriateness of endoscopic myotomy.

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