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Achalasia and Other Esophageal Motility Disorders

Marco G. Patti · Fernando A. Herbella

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Abstract Achalasia, diffuse esophageal spasm, nutcracker esophagus, and the hypertensive lower esophageal sphincter are considered primary esophageal motility disorder. These disorders are characterized by esophageal dysmotility that is responsible for the symptoms. While there is today a reasonable consensus about the pathophysiology, the diagnosis, and the treatment of achalasia, this has not occurred for the other disorders. A careful evaluation is therefore necessary before an operation is considered.

Keywords Primary esophageal motility disorders · Esophageal achalasia · Diffuse esophageal spasm · Nutcracker esophagus · Hypertensive lower esophageal sphincter · Esophageal manometry · Minimally invasive surgery

Achalasia, diffuse esophageal spasm (DES), nutcracker esophagus (NE), and the hypertensive lower esophageal sphincter (HTN-LES) are considered primary esophageal motility disorders (PEMD), as they occur in the absence of an identifiable cause such as gastroesophageal reflux disease (GERD).[1] These disorders present with a specific manometric pattern, and the dysmotility is considered responsible for the symptoms. While a reasonable consensus has developed for the pathophysiology, the diagnosis, and the treatment of achalasia, this has not occurred for the other disorders. The goal of this study is to review the clinical presentation, the diagnosis, and the role of minimally invasive surgery in their treatment.

M. G. Patti (✉) · F. A. Herbella
Department of Surgery,
University of Chicago Pritzker School of Medicine,
5841 S. Maryland Ave, MC 5095, Room G-201,
Chicago, IL 60637, USA
e-mail: mpatti@surgery.bsd.uchicago.edu

Esophageal Achalasia

Evaluation

In a study of 145 patients with untreated achalasia, dysphagia was the most common symptom as it was present in 94% of patients. Regurgitation was present in 76% of patients, heartburn in 52%, and chest pain in 41%.[2] At the time of referral, 65 patients (45%) were taking acid reducing medications on the assumption that GERD was the cause of their symptoms.[2] This study shows that symptoms are less sensitive and specific for the diagnosis of GERD than commonly thought. Patients with achalasia experience heartburn because of stasis and fermentation of food in the esophagus rather than real reflux.[3]

Endoscopy is usually the first test that is performed to rule out the presence of esophagitis or a mechanical obstruction secondary to a peptic stricture or cancer. It is important to remember that the presentation and the manometric picture of achalasia can be caused by a malignancy (secondary achalasia or pseudoachalasia), mostly at the level of the gastroesophageal junction.[4] Patients with secondary achalasia are usually older, have experienced symptoms for a shorter time, and have had a greater weight loss as compared with patients with primary achalasia. When an underlying malignancy is suspected, an endoscopic ultrasound or a CT scan with fine cuts of the

gastroesophageal junction is recommended. Barium swallow shows in most cases distal esophageal tapering, and it is important to assess the diameter and the axis of the esophagus (straight versus sigmoid).

Esophageal manometry is the gold standard for the diagnosis of achalasia. Lack of peristalsis and absent or incomplete relaxation of the lower esophageal sphincter (LES) in response to swallowing are the key criteria for the diagnosis. Contrary to common belief, the LES is hypertensive in less than half of patients.[2] Recently, a new classification of esophageal achalasia has been proposed based on high resolution manometry (HRM).[5] Using HRM, Pandolfino and colleagues identified three achalasia subtypes: type I, classic, with minimal esophageal pressurization; type II, achalasia with esophageal compression; and type III, achalasia with spasm. According to their results, type II is associated to the best therapeutic response (91% with dilatation and 100% with Heller myotomy). On the other hand, a favorable response to therapy was present in only 56% of type I and 29% of type III patients.[5] The importance of the manometric pattern as predictor of treatment success has also been demonstrated by others.[6] Further studies are necessary to confirm these results.

Ambulatory pH monitoring is important in untreated patients when the diagnosis is not clear (achalasia versus GERD) and in patients who still have dysphagia after endoscopic dilatation to see if abnormal reflux is present.[2] Rather than relying on the reflux score only, it is important to examine the tracings to distinguish between true and false reflux.[3]

Treatment

The first minimally invasive myotomy for achalasia was performed in the USA in January of 1991.[7] While the initial experience was based on a left thoracoscopic approach, the technique eventually evolved into a laparoscopic myotomy with a partial fundoplication. The high success rate of this operation[8–12] has brought a shift in practice, as surgery has gradually become the preferred treatment modality for most gastroenterologists and other referring physicians.[13, 14] This remarkable change has followed documentation that a laparoscopic myotomy outperforms balloon dilatation and intra-sphincteric botulinum toxin injection.[15–18]

The technique of a laparoscopic Heller myotomy and Dor fundoplication is described elsewhere.[19] The myotomy is usually 7 to 8 cm in length and extends for 2.0–2.5 cm onto the gastric wall. After the myotomy is completed, the muscle edges are gently separated to expose the mucosa for about 40% of the circumference. The Dor is a 180° anterior fundoplication which covers the exposed mucosa. Alternatively, a posterior partial fundoplication can be used.[20]

Some areas in the treatment of achalasia are still controversial, and often there is not enough evidence to clearly support one approach versus another.

- Thoracoscopic versus laparoscopic approach

  Even though the thoracoscopic approach gave very good relief of dysphagia, some shortcomings became soon apparent: cumbersome intraoperative management (double lumen endotracheal tube, left lateral decubitus, one lung ventilation), limited exposure of the gastroesophageal junction, postoperative discomfort, and a high rate of postoperative reflux (around 60%). These problems were mostly eliminated by the laparoscopic approach (simpler anesthesia in the supine position, better exposure of the gastroesophageal junction, possibility to perform a fundoplication).[13]
- Length of the myotomy

  When the thoracoscopic approach was used, the myotomy extended onto the gastric wall for 5 mm only.[7] With the laparoscopic approach, a longer myotomy can be easily performed avoiding the risk of persistent dysphagia, and a fundoplication added. Intraoperative endoscopy to locate the squamo-columnar junction is useful until enough experience is gained. Most surgeons today extend the myotomy onto the gastric wall for 1.5 to 2.5 cm, as it has been suggested that a longer myotomy is associated to better relief of dysphagia.[21]
- Difficulty of the myotomy

  Be aware of patients that are sent for surgery after failed endoscopic therapy (pneumatic dilatation and intra-sphincteric botulinum toxin injection). In some patients, particularly after treatment with botulinum toxin, a fibrotic reaction may occur at the level of the gastroesophageal junction with obliteration of the anatomic planes.[22–24] In these patients, the myotomy is more difficult, a perforation more frequent, and the relief of dysphagia less predictable.
- Fundoplication after laparoscopic Heller myotomy

  Gastroesophageal reflux into the aperistaltic esophagus can occur after a myotomy and may cause a stricture, Barrett's esophagus, and even adenocarcinoma.[25] Because a myotomy alone is associated to reflux in 40% to 60% of patients,[13, 26] a fundoplication must be added. In a prospective and randomized trial of myotomy alone or myotomy plus Dor fundoplication, Richards and colleagues showed that postoperative reflux was present in 48% of patients after myotomy alone but in only 9% of patients when a Dor fundoplication was added.[26] Even though it has been shown that a Nissen fundoplication is the best operation for GERD,[27] this procedure creates too much resistance at the level of the gastroesophageal junction in achalasia patients who have no peristalsis.[28, 29] In a prospective

and randomized trial of Dor fundoplication versus Nissen fundoplication after Heller myotomy, Rebecchi and colleagues showed that at a 5-year follow-up 15% of patients after myotomy and Nissen fundoplication had dysphagia as compared to only 2.8% after myotomy and Dor fundoplication.[29]

A partial fundoplication, either anterior or posterior, is therefore the procedure of choice in conjunction to a Heller myotomy for achalasia. To date, there has been no direct comparison between these two procedures in terms of reflux control. While a posterior fundoplication might determine better control of reflux and keep the edges of the myotomy open, an anterior fundoplication offers the advantage of a more limited dissection and of covering the exposed mucosa.[30]

- Procedure of choice in patients with sigmoid esophagus

  In the past, it was thought that patients with a dilated and sigmoid esophagus should undergo an esophagectomy for the fear that a myotomy would not improve the esophageal emptying with persistence of dysphagia.[25] An esophagectomy, however, is associated to considerable morbidity even in the hands of experienced esophageal surgeons. For instance, Devaney and colleagues reported a 10% rate of anastomotic leak, 5% rate of hoarseness, and 2% rate of bleeding, chylothorax, and death among 93 patients who had an esophagectomy for achalasia. In addition, 46% of patients had dysphagia requiring dilatation of the anastomosis.[25] Most surgeons today feel that a Heller myotomy should be the primary treatment for achalasia, regardless of the size and shape of the esophagus, and that an esophagectomy should be reserved as last resort for patients whose dysphagia is not amenable to other treatment.[31–33] For instance, Sweet and colleagues analyzed the outcome of a Heller myotomy among 113 achalasia patients who were divided in four groups based on the size and shape of the esophagus. A logistic regression model was created to examine factors associated with fair/poor outcome. Neither the size of the esophagus, age, sex, and preoperative LES pressure affected outcome. The only factor associated to a poor outcome was the preoperative treatment with botulinum toxin.[33]
- Persistent and recurrent dysphagia after Heller myotomy

  While persistent dysphagia is usually due to a technical problem (wrong diagnosis, short myotomy, wrong configuration of the fundoplication), recurrent dysphagia after a symptom-free interval can be secondary to a variety of factors such as formation of scar tissue at the end of the myotomy and gastroesophageal reflux with a peptic stricture.[34] A careful evaluation must be performed, including a careful history, review of the operative report, barium swallow, endoscopy, esophageal manometry, and ambulatory pH monitoring.[35] After the workup is completed and a possible cause identified, dilatation or a second operation are the options to be considered to improve the swallowing status.[33, 36–38] A dilatation should be tried first as it is effective in most patients.[33, 38] A second myotomy should be the second step,[36, 37] while an esophagectomy should be the last resort when all the other therapeutic modalities have been exhausted.

Other Primary Esophageal Motility Disorders

DES, NE, and the HTN-LES are the other primary esophageal motility disorders. Overall these disorders have not been studied extensively as achalasia, and they are still poorly understood.[39]

Evaluation

Most patients with DES and HTN-LES present with dysphagia. On the other hand, chest pain is the most common complaint of patients with NE.[40] For this reason, the majority of NE patients are referred to a gastroenterologist or a surgeon after a proper workup has excluded the presence of cardiac pathology.

Due to the intermittent nature of DES, a barium swallow shows a "corkscrew" esophagus in about 30% of patients. This test can be normal in patients with NE or similar to achalasia in patients with HTN-LES.[40]

An endoscopy is usually done in patients with dysphagia. Esophageal manometry shows the following findings:[1]

- DES: the LES can be similar to achalasia or normal. Esophageal peristalsis is characterized by simultaneous contractions following more than 20% but less than 100% of wet swallows.
- NE: the LES can be similar to achalasia or normal. Esophageal peristalsis is characterized by peristaltic waves in the distal esophagus of high amplitude (>180 mmHg) and prolonged duration (>6 s).

HTN-LES: LES pressure above 45 mmHg. Peristalsis is usually normal.

Ambulatory pH monitoring is of key importance in patients with a manometric picture of DES or NE. If GERD is present, these motility patterns should be considered secondary rather than primary, and therapy should be directed towards the correction of the abnormal reflux.[41]

Treatment

Selected patients with DES who have not responded to medical therapy should be considered surgical candidates.

The surgical treatment of DES is similar to that of achalasia (myotomy and partial fundoplication). The myotomy is usually extended more proximally than in patients with achalasia. The operation, performed either by an open or a laparoscopic approach, gives very good results.[40, 42, 43] For instance, Patti and colleagues reported the results of minimally invasive surgery in 34 patients with DES.[40] Dysphagia was relieved in 80% of patients after thoracoscopic myotomy and in 86% of patients after laparoscopic myotomy. Chest pain was relieved in 75% and 80% of patients, respectively. Regurgitation was also significantly improved.[40]

In patients with NE and chest pain, the results of surgery are disappointing. Patti and colleagues reported improvement of chest pain in only six of 12 patients with NE.[40] Dysphagia was instead improved in 80% of patients. Currently, these Authors propose a myotomy only in patients whose main symptom is dysphagia, or when associated pathology such as an epiphrenic diverticulum is present. The treatment of HTN-LES is similar to that of achalasia.

Epiphrenic Diverticulum

Epiphrenic diverticulum is a pulsion diverticulum, usually located in the distal 10 cm of the esophagus. It is due to herniation of the mucosa and submucosa through the muscle layers of the esophageal wall

Most patients complain of dysphagia, and respiratory symptoms are often present due to aspiration. A barium swallow is of key importance as it shows the position of the diverticulum (more frequently on the right side), the width of the diverticular neck, and its distance from the gastroesophageal junction. Endoscopy is important to rule out a neoplastic process and for proper placement of a manometry catheter.

Most authors today feel that an epiphrenic diverticulum is always caused by an underlying esophageal motility disorder.[44–47] While conventional manometry demonstrates a motility disorder in about 70% of patients, ambulatory manometry allows determination of the underlying problem in all patients.[44] These findings support the rationale for performing a myotomy in all patients with an epiphrenic diverticulum, regardless of the findings of conventional stationary manometry. Therefore, the operation of choice is resection of the diverticulum, esophageal myotomy, and partial fundoplication. Traditionally, the operation was performed through a left thoracotomy.[46] The development of minimally invasive surgery has brought a drastic change, as today most of these diverticula are resected through a laparoscopic approach.[45, 47]

Conclusions

The evaluation and treatment of PEMD has evolved during the last 20 years. Patient's evaluation is of key importance to clearly define the motility abnormality. Minimally invasive surgery has brought a shift in the treatment algorithm, as a laparoscopic Heller myotomy with partial fundoplication is today the procedure of choice for most patients with PEMD.

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