Laparoscopic Treatment in Achalasia of the Cardia

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Abstract

Background: Achalasia, although a rare disease (an incidence of 1/100,000 individuals each year) is one of the common causes of motor dysphagia and is characterized by loss of peristalsis in the esophageal body and lack of relaxation of the lower esophageal sphincter.

Aim: The aim of our study was to perform a clinical, therapeutic and evolution evaluation in patients diagnosed with achalasia and operated in our department between 1997 and 2013.

Material and Methods: We performed a retrospective study using the clinical charts, operatory protocols, imagistic and video records of the 17 patients with internal achalasia operated in our clinic between 1997-2013.

Results: In the studied lot we registered a relative equal distribution by sex and a predominance of patients from the urban area. The age of patients was between 24 and 86 years (with a median of 51). Two of the cases were reoperations following earlier achalasia surgery. The technique used was the Heller operation associated with Dor fundoplication in 12 cases and Toupet fundoplication in 5 cases. The average operating time was 117.6 minutes. Three cases of iatrogenic esophageal perforation were recorded, recognized and treated during the same operation time. Postoperative evolution did not record specific technical complications. The postoperative follow-up was between 3 and 72 months.

Conclusion: The laparoscopic approach in the treatment of achalasia offers, besides the advantages of minimally invasive surgery, a good visualization of the abdominal esophagus and the gastro-esophageal junction. The cardiomiotomy, as treatment of achalasia, is usually associated with an anti-reflux procedure. The Dor fundoplication is preferred, especially in cases of iatrogenic mucosal lesions, while Toupet fundoplication, as we used in 5 cases, can have the same benefits. Achalasia is a disease with a risk for the development of dysplasia/carcinoma, which is why long-term postoperative follow-up is important.

Keywords: achalasia, lower esophageal sphincter, dysphagia, cardiomiotomy, fundoplication
video database of the 17 patients with achalasia operated in our department.

Results: We encountered an equal repartition in women and men and a predominance of urban provenience. Ages were between 24 and 86 years (with an average age of 51). There were two cases of recurrent achalasia at 2, respectively 5 years after the first operation. In all cases, Heller myotomy was used, with the addition of a Dor fundoplication in 12 cases and Toupet fundoplication in five cases, as an antireflux procedure. Mean operation time was 117.6 minutes.

There were three iatrogenic perforations of the esophageal mucosa, all of them recognized and treated in the same operative time. No postoperative complications related to the Heller-Dor/Heller-Toupet procedure were encountered. The follow-up was between 3 and 72 months.

Conclusions: Laparoscopic approach in the treatment of achalasia provides the advantages of minimally invasive surgery, but also and very important, a good visualization of the abdominal esophagus and gastroesophageal junction. Heller esocardiomyotomy is usually associated with an antireflux procedure. A Dor fundoplication is generally used, although the Toupet fundoplication may also be used with the same advantages. It is important to monitor these patients on a yearly basis, knowing the risk of dysplasia/carcinoma in achalasia.

Key words: achalasia, inferior esophageal sphincter, dysphagia, esocardiomyotomy, fundoplication

Introduction

Achalasia is one of the common causes of motor dysphagia and is characterized by aperistalsis of the esophageal body and lack of relaxation of the lower esophageal sphincter. Its pathogenesis still remains unknown although it is described as loss of inhibitory ganglia in the myenteric plexus of the esophagus.

As a consequence of the motility disorder, passage of food from the mouth to the stomach is hampered, causing dysphagia and leading to weight loss, retrosternal pain, regurgitation of undigested food, bad breath, nocturnal coughing and increased risk of aspiration pneumonia.

The diagnosis of achalasia is suggested by the barium swallow (the classic bird’s beak at distal esophagus, dilation of the esophagus and slow clearance) and upper endoscopy (end stage achalasia-esophageal stasis and dilation; pseudoachalasia) (1,2). The manometry, which is the most sensitive diagnostic test of esophageal dysmotility, confirms the diagnosis (elevated basal LES pressures, absence of esophageal peristalsis, incomplete relaxation of LES on swallowing).

Pseudoachalasia is excluded by upper endoscopy, endoscopic ultrasonography and computed tomography.

The only available therapeutic options are to loosen the LES and treat the symptoms (3).

We have three therapeutic options: surgical myotomy, pneumodilation and POEM, but minimally invasive Heller myotomy remains the gold-standard procedure in achalasia. What still remains controversial is the type of fundoplication added to the myotomy.

Materials and Methods

This study is based on a retrospective analysis of 17 patients diagnosed with achalasia and operated in the General Surgery Department of “Prof. Dr. Agrippa Ionescu” Clinical Emergency Hospital. We evaluated the clinical and therapeutic aspects and the evolution of these patients operated between 1997 and 2013, using the clinical charts, operatory protocols, imagistic and video database.

Results

We observed an equal repartition in women and men, a predominance of urban provenience (11/17 patients). Ages were between 24 and 86 years old. The presenting symptoms were dysphagia, retrosternal pain, weight loss, regurgitation of undigested food. Associated comorbidities (9 cases): arterial hypertension, atrial fibrillation, obesity, gallstones, chronic venous insufficiency, diabetes type II.

Three diagnostic tests were used: barium swallow, upper endoscopy and manometry. We had two cases of recurrent achalasia at 2, respectively 5 years after the first operation.

As for the surgical treatment, we used the Heller myotomy in all cases, associated in 12 cases with a Dor fundoplication and in 5 cases with a posterior Toupet fundoplication. We prefer the lithotomy position with legs abducted, a 30-40° reverse Trendelenburg. The surgeon stands between the patient’s legs with the first assistant on his right and the second on his left. The trocar placement follows the Cadiere manner. A gastric tube is placed to decompress the stomach.

The laparoscopic approach follows the same principles as in open surgery. After the induction of the pneumoperitoneum (12 mmHg), we expose the GE junction, followed by the division of the phrenoesophageal ligament and the dissection of the diaphragmatic crus. Next, we mobilize the esophagus and, in case of a Toupet fundoplication, we create a wide retroesophageal space. We perform the esophageal and gastric myotomy (4-6 cm above GEJ and 2 cm on the stomach), using the Hook monopolar. Control of the integrity of the mucosa is done using a blue methylene test.

In all 17 cases we added an antireflux fundoplication - an anterior one (Dor) in 12 cases and a posterior one (Toupet) in 5 cases. Lately we prefer the posterior fundoplication. In 3 cases, the superior polar gastric vessels were sectioned in order to mobilize the great curvature of the stomach. Intraoperative incidents: 4 cases of hemorrhage (2 cases during the fundoplication with gastric hematoma and 2 cases during the incision of the phrenoesophageal ligament) and 3 cases of iatrogenic perforation of the esophageal
mucosa (recognized and treated in the same operative time by sutures; Dor fundoplication was used in these 3 cases). The mean operative time was 117.6 minutes (progressive decrease over time with the increase of the experience). No conversion to laparotomy was necessary. No significant postoperative complications and no mortality were encountered. The mean postoperative hospital stay was 4.8 days. Gastroesophageal pathologic reflux was absent in all patients. The mean clinical follow up was 17.8 months (range 3-72 months).

Discussions

The disease was first described in 1674 by Willis who described it as “food blockage in the esophagus” and treated it with a dilator made of whale bone and sponge; the term “achalasia”, a Greek word, was first used by Hurst in 1927 and means “failure of relaxation”. Achalasia is caused by loss of inhibitory ganglia in the myenteric plexus of the esophagus and gradual progression of neuronal degeneration is associated with progression of the disease from vigorous to classic achalasia. In the initial stage of the disease, the degeneration of inhibitory nerve fibers in the esophagus results in unopposed action of excitatory neurotransmitters such as acetylcholine, which leads to high amplitude non-peristaltic contractions. This stage of achalasia is known as vigorous achalasia (average amplitude of contractions in lower esophagus >40 mmHg). Progressive loss of cholinergic neurons results in dilation and low amplitude simultaneous contractions in the esophageal body; this stage of achalasia is called classic achalasia (4,5).

Many studies have attempted to determine the initiating agents that cause the disease. A genetic influence is also present as there are reports showing occurrence of disease in monozygotic twins and other first-degree relatives and also the occurrence in association with other genetic diseases such as Down’s syndrome and Parkinson’s disease. There are also incriminated environmental and autoimmune factors. (4,5). The role of the longitudinal muscles in achalasia is currently under study (6). Achalasia can be primary (idiopathic) or secondary (the cause for the degeneration of esophageal nerve fibers is known).

Manometry plays a diagnostic role in achalasia. Since the emergence of HRM (high resonance manometry), esophageal achalasia can be classified into three subtypes (7,8). In type I achalasia (classic achalasia), impaired LES relaxation but no significant pressurization within the esophageal body is observed. In type II achalasia (with compression) swallowing of water causes rapid panesophageal pressurization. This may exceed LES pressure, causing the esophagus to empty. Type III achalasia (spastic achalasia) is also associated with rapidly propagated pressurization; however, the pressurization is attributable to an abnormal lumen, obliterating contraction. HRM can be used to predict the outcome of each type of achalasia. Patients in whom HRM shows type II achalasia are more likely to respond to therapies such as pneumatic dilation and Heller myotomy compared to those with type I or III (7,8,9).

Pharmacological management plays a minor role in the treatment (3,5). Endoscopic treatment with Botox yields excellent immediate responses, but only half of all patients benefit for more than one year, so it is used to treat elderly patients or patients with high surgical risks (10,11). For endoscopic dilation immediate and short-term results have reportedly been good (12,13). Peroral endoscopic myotomy (POEM) is a novel endoscopic esophagomyotomy for achalasia that was first reported by Pasricha et al (14) in porcine models and subsequently by Inoue et al (15) in humans. POEM is performed by dissection and division of the inner circular muscle layer of the esophagus through a submucosal tunnel created endoscopically through a small proximal opening in the esophageal mucosa. The POEM technique is used in several centers with good short-term results, but long-term follow-up results are required (16). Clinical success rate > 80% has been reported in some Chinese studies for self-expanding metallic stents (17).

Minimally invasive Heller myotomy has become the gold standard procedure for achalasia. Overall success rates of laparoscopic Heller myotomy (LHM) were 47%-82% at 10 years (18).

LHM or pneumatic dilation? - the ongoing debate.

Systematic reviews and meta-analysis that have compared existing treatment methods for achalasia have found that surgery is superior to pneumatic dilation (PD) (19), but it should be noted that the first randomized controlled multicenter trial published by the European Achalasia Trial group that compared LHM and PD reported that, after 2 years of follow-up, LHM was not associated with superior rates of therapeutic success (20,21). There is a lot of debate on the choice of LHM as the primary treatment for achalasia or as a second-line treatment following the failure of nonsurgical intervention.

Esophagectomy should be reserved only for those cases in which simpler operations have failed.

However, the major adverse event after surgery is severe reflux. There is much debate on the role of fundoplication with myotomy in the reported literature (22). Intraoperative endoscopy during LHM can be used to guide the extent and adequacy of myotomy. A concomitant endoscopic examination during LHM to guide myotomy and routine fundoplication is clinically necessary, despite disagreement about the fundoplication procedure (23).

Dor fundoplication is easier to perform, does not require an extensive dissection of the GEJ, thus preserving the mechanism of a competent cardia. It does not angulate the distal esophagus and protects it after the myotomy. Instead, the Toupet fundoplication leaves the myotomy uncovered and pulls the muscle edges in opposite directions, preventing the re-approximation of the myotomy and so the occurrence of a recurrent dysphagia. There is still no evidence to support a choice. (24,25)

In summary, as stated in the recent Kagoshima consensus, despite the variations as to the length of the myotomy and the addition of an antireflux procedure, good overall long-term results suggest that these operative variations are not critical (26).
Conclusions

Esophageal achalasia is a functional disease of unclear etiology. Despite the ongoing debate and the report of the first randomized control trial, the minimally invasive surgical treatment seems to yield better results than PD.

Laparoscopic approach in the treatment of achalasia offers well-known advantages: good visualization of the distal esophagus and anterior gastric wall, faster recovery, fewer surgical site complications, aesthetic results.

Laparoscopic Heller-Dor / Heller-Toupet is a safe and effective treatment of esophageal achalasia with excellent results in terms of dysphagia resolution, providing protection from the onset of gastroesophageal reflux.

We should identify patients at risk for developing dysplasia or/and carcinoma.

The question remains whether the excellent results of myotomy are stable over time or not. We should better define when treatment is successful and identify better criteria for re-treatment.

POEM is a promising technique and is associated with good short-term results without serious complications, but long-term results are not yet available.

Our current treatment approaches reduce the discomfort of patients, but the actual cause of achalasia has not yet been identified; instead of cleaving or disrupting the muscle, we should aim for restoring its function. We need more insight into how the neurons lost in achalasia can be replaced; stem-cell transplantation may play a role as a curative treatment and improve treatment success in the future.

References