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Case Series

Laparoscopic Heller's cardiomyotomy for achalasia cardia-a case series with review of literature

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ABSTRACT

Laparoscopic Heller's cardio myotomy has almost completely replaced the open procedure performed for achalasia cardia. Several studies have suggested that long-term results with surgery for achalasia cardia are better than a medical line of management. In this retrospective study, we outline our experience with laparoscopic cardio myotomy over 15 years. Also, we compare our results with those of other interventional modalities which are known therapeutic options for achalasia cardia. We identified 10 patients (4 females and 6 males) operated upon by a single surgeon from 2008 to 2023, at our institution. The age range was 16-48 years. All patients had undergone oesophago-gastro-duodenoscopy (OGD scopy), upper gastro-intestinal (UGI) manometry and contrast enhanced computerised tomography (CECT) of the lower chest and upper abdomen. The patients were analysed for age, sex, symptoms, pre-operative evaluation, the exact procedure performed (Heller's+Toupet's vs Heller's+Dorr's), morbidity/mortality and functional outcomes. They were also reviewed to examine the length of stay, length of procedure, complications and recurrent symptoms on follow-up.

Keywords: Achalasia cardia, Laparoscopic cardio myotomy, Manometry, OGD scopy

INTRODUCTION

Achalasia is a primary oesophageal motor disorder thought to be caused by autoimmune neuro-degeneration of the oesophageal myenteric plexus. Achalasia is the most well known esophageal motility disorder[1] with an annual incidence of 1 in 100,000 and prevalence of 8 in 10,000 people. The prevalence is equal in men and women.[1] However, recent studies have shown that the incidence of achalasia is increasing, particularly in South America, and varies among countries.[2] Achalasia has a bimodal age distribution, with most patients aged between either 20-40 or 60-70 years.[3] Though the disease was first described more than 300 years ago, exact pathogenesis of this condition still remains elusive.

CASE SERIES

In this retrospective study, we identified 10 patients (4 females and 6 males) with a mean age of 37.6 years (range 16-48 years), who underwent laparoscopic cardio myotomy at our institution, from 2008 to 2023; performed by a single surgeon. All patients had undergone OGD scopy, UGI manometry and contrast enhanced computed tomography (CECT) of the lower chest and upper abdomen. In all 10 patients, the performing gastroenterologist noted oesophageal dilatation, food residue in the oesophagus and resistance to passage of the scope into the stomach, across the lower oesophageal sphincter (LES) (Figure 1 A and B). After few gentle attempts at the same, after some insufflation of air, the scope could negotiate the LES with a sudden give way. In all 10 patients, the UGI manometry showed that

the LES relaxation was incomplete and revealed pan-oesophageal pressurization on wet swallows. Also, the LES pressures were significantly increased (Average-55 mms of Hg, range: 45-65 mms of Hg) (Figure 1 C and D). Thus, the diagnosis of achalasia was confirmed. Four patients presented to the surgical outpatient department (OPD) with Barium swallow X-ray already done, on the advice of their family doctor. All these 4 barium swallow films showed a dilated, characterless oesophagus with a smooth tapering towards the LES-classically called the 'bird's beak' appearance (Figure 2 A). The CECT was done to definitively rule out any pathology causing extrinsic compression of the lower oesophagus. The symptoms on presentation were dysphagia-100% (n=10), regurgitation-70% (n=7) and retrosternal pain-30% (n=3). The patients were analysed for age, sex, symptoms, pre-operative evaluation, operative approach, the exact procedure performed (Heller's+Toupet's vs Heller's+Dorr's), morbidity/mortality, and functional outcomes, length of stay, length of procedure, complications and recurrent symptoms on follow-up. The information on patient demographics is summarised (Table 1).

Five patients were on proton pump inhibitors (PPIs) pre-operatively, for variable periods ranging from 4 to 6 weeks. Duration of symptoms ranged from 3 months to 1 year. Seven patients had already undergone an average of 2 (range 1-4) separate sessions of endoscopic balloon dilatation by the gastroenterologist, prior to surgical referral. Four of these 7 patients reported an initial improvement in dysphagia, but reverted back to the original condition over 3-4 weeks. The remaining 3 patients reported no improvement in symptoms after endoscopic achalasia balloon dilatation. Only those patients with UGI manometry proven/diagnosed achalasia cardia were included in this study.

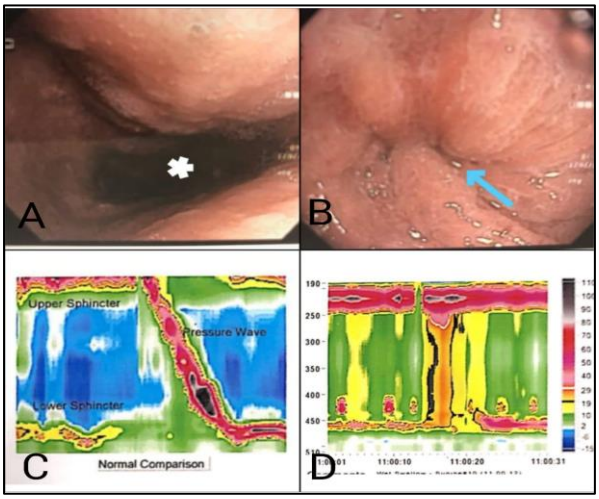

Figure 1: (A) OGD scopy shows lumen of dilated esophagus (white asterisk), (B) shows constricted esophago-gastric junction (blue arrow), (C) shows normal upper GI manometry and (D) shows upper GI manometry in achalasia patient.

Table 1: Patient demographics.

Variables	N
Total no. of patients	10
M:F	6:4
Age range (Average) (In years)	16-48 (37.6)
Mean BMI (Range)	24 (16-30)
Average duration of pre-op symptoms (Range)	8 months (3-12 months)
Average LES pressure on pre-op manometry (Range)	55 mms of Hg (45 to 70 mms of Hg)
Pre-op balloon dilatation	7 patients

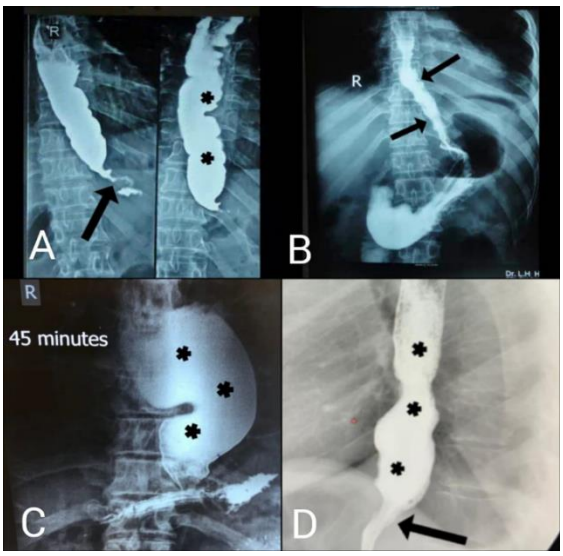

Figure 2: (A) Barium esophagogram with classical 'birds beak' appearance (black arrow) and dilated esophagus (black asterisks), (B) esophagogram in post op pt showing smooth passage of contrast across the myotomy segment (black arrows), (C) esophagogram of the end stage sigmoid achalasia pt. showing sigmoid shaped esophagus (black asterisks) and (D) post op Ba study of same pt.

At the time of writing this paper, all the patients who were past their last post-operative OPD follow-up visit, were interviewed telephonically with a standard questionnaire. Patients were asked for symptoms such as dysphagia, regurgitation and vomiting. Patients were followed up at 1-, 4- and 6-weeks post-surgery. Further evaluation was carried out after 6 months and then yearly for 2 years. Those patients who failed to physically follow up at 6 months, 1 year &/or 2 years (long term follow up) were telephonically interviewed with the same aforementioned questionnaire. Ours was a retrospective study of hospital inpatient records, OPD data and information obtained from the telephonic questionnaire.

Pre-operative anaesthesia check-up was carried out in all the patients. Antibiotic prophylaxis of Ceftriaxone (1-1.5 gm intravenous), as per the hospital's antibiotic policy,

was administered just before the induction of the anaesthesia. In all the surgeries, the patient was placed in a supine Trendelenburg position with legs split up and the operating surgeon standing between the legs. The monitor was placed at the head end of the patient on the patient's left side facing the operating surgeon. Pneumoperitoneum was established by closed technique through the Veress's needle. A standard five trocar technique was used. The first optic trocar (10 mm) was inserted at the level of the junction of the upper two-thirds and lower one-third of the xiphisternum to the umbilicus line, slightly to the left of the midline. Four additional ports, right midclavicular (5mm), subxiphoid (5mm), left midclavicular (12mm) and left anterior axillary (5mm), were inserted under vision. The procedure began with an incision of the gastro-hepatic omentum and entry into the lesser sac. The dissection was then extended cephalad towards the right crus of the diaphragm. The posterior parietal peritoneum over the right crus was incised and the oesophagus dissected away from it. Due care was taken to avoid injury to the posterior vagus nerve and hepatic branches of the vagus nerve. Circumferential oesophageal mobilization was carried out after the creation of a retro-oesophageal window. The stomach was mobilized along with the upper one-third of its greater curvature up to the oesophagus. The harmonic scalpel was our preferred energy source. The mediastinal dissection was then initiated over the anterior aspect of the oesophagus and extended cephalad up to the inferior pulmonary vein. The myotomy was then started on the anterior surface of the oesophagus. The outer longitudinal muscle layer was split up using gentle opposing shearing force with blunt instruments. A curved dissector was then inserted gently into the initial myotomy and opened taking care not to injure the mucosa (Figure 3 A). This opened up the plane between the circular muscle layer and the mucosa. The harmonic scalpel then divided the inner circular layer (Figure 3 B). Due care was taken to ensure that its inactive non-vibrating blade was towards the oesophageal mucosa. This was then slowly and carefully extended cephalad for 8-10 centimetres (cm) (Figure 3 C). While performing this, great care was taken to identify and protect the anterior vagus nerve which usually crosses the anterior aspect of the oesophagus from the left to the right side (Figure 3 D). Once the proximal myotomy was done, the dissection was then extended caudad into the anterior aspect of the cardia and the anterior wall of the body of the stomach, for up to 3-4 cm. A gauze piece introduced initially was invaluable for pressure and haemostasis, along the way. In the gastric part of the dissection which is the lower end of the myotomy, it was that much more difficult to find and maintain the correct plane and had to be performed with utmost caution so as to avoid an iatrogenic perforation. Bipolar cautery can also be carefully used intermittently for haemostasis over the lips of the myotomy. Once the myotomy was complete, the exposed oesophageal and gastric mucosa was inspected carefully. Also, a 'leak test' was performed by the surgeon occluding the distal body of stomach and the anaesthesiologist pushing air through the slightly withdrawn naso-gastric tube (NGT) so as to blow up the mucosa. Saline was then instilled over it to check for leak of air bubbles, thereby definitively confirming / ruling out a mucosal perforation. If a perforation occurred, the same was suture closed using 3-0 Polydioxanone (PDS) (Figure 4 A-C). As an additional step, the lips of the myotomy were suture-fixed to the 2 crurae with 3-4 stitches of 2-0 Polypropylene on either side (Figure 3 E and F). After completion of the myotomy, an anti-reflux procedure was then performed, before concluding the operation; so as to mitigate the massive gastro-oesophageal reflux caused by the myotomy. The posterior Toupet's partial fundoplication is our preferred anti-reflux procedure because it additionally contributes in keeping the myotomy lips apart from each other. In the Toupet's wrap, the fundic lips were sutured to the 2 lips of the myotomy with 2-3 interrupted 2-0 polypropylene sutures on either side, thereby fashioning a posterior 270^0 wrap. All our patients were kept nil per oral for the first 24 hours with a NGT *in situ*, kept open and aspirated regularly. The NGT was clamped on day one of the surgery and clear liquids were started per orally. On day 2, the NGT was removed and a semisolid diet was started per orally. Patients were advised to continue this diet for 1^{st} 2 weeks. They were discharged on day 3 of surgery.

Six males (60%) and 4 females (40%) with a diagnosis of achalasia cardia with a mean age of 37.6 years (16-48 years) were included in this study. On pre-operative OGD scopy, all 10 (100%) patients had oesophageal dilatation, some food residue in the lower oesophagus and difficulty / resistance during entry across the LES into the stomach. On UGI manometry, the average LES pressure in the present series was 55 mms of Hg (range: 45-70 mms of Hg). On CECT, there were no extrinsically compressing masses. The mean body mass index (BMI) of the patient population in this study was 24 (range:16-30). The pre-operative duration of the symptoms was 6 months to 2 years. The average operating time dropped from 130 minutes for the first 4 cases to 90 minutes for the next 6 cases. The mean hospital stay was 3.1 days (range: 3-4 days). Nine patients (90%) underwent the laparoscopic Heller-Toupet procedure i.e., cardio myotomy with posterior 270-degree fundoplication. One patient (10%) underwent the laparoscopic Heller-Dorr procedure i.e., cardio myotomy with anterior 180-degree fundoplication (Figure 4 D). This patient was a 16-year-old male who had undergone pre-operative endoscopic achalasia balloon dilatation on 3 separate occasions. He had an iatrogenic oesophageal mucosal perforation which was suture closed using 3-0 PDS. In this patient, a Dorr anterior fundoplication was preferred over our routine practice of Toupet posterior fundoplication, as the anterior fundal wrap gives added cover and protection over the oesophageal suture line (Figure 4 D). This patient also additionally underwent a postoperative barium swallow on day 3, which showed resolution of the oesophageal dilatation and smooth passage of the contrast across myotomy, into stomach without extravasation (Figure 2 B). This ruled out a leak from the suture line

and only then were oral feeds started. He was discharged from hospital on day 4. Three patients had intra-op complications (Table 2). One had oesophageal perforation (as described above). One patient had surgical emphysema which settled without any further intervention, over 24 hours. One patient had port site bleeding which was stopped with external pressure. One patient (10%) had a sigmoid shaped oesophagus of advanced achalasia (Figure 2 C). He was a 48-year-old male farmer who had also undergone preoperative balloon dilatation on 2 occasions, but to no lasting relief. The 2 surgical options of total esophagectomy and laparoscopic Heller-Dorr procedure with extra 'U' stitches to correct lower oesophageal axis, were discussed with him and his family. They chose the latter option with the clear understanding that were it to fail, he would have to undergo a total esophagectomy. An additional operative step in sigmoid achalasia is taking 2-3 'U' shaped stitches on right lateral oesophageal wall so as to correct its deformed axis. Postop barium esophagogram showed correction of oesophageal axis, resolution of its dilatation and effective emptying of contrast into the stomach (Figure 2 D). Over a postoperative follow up period of 26 months, this patient continues to lead a normal life, eat normal food and be symptom free.

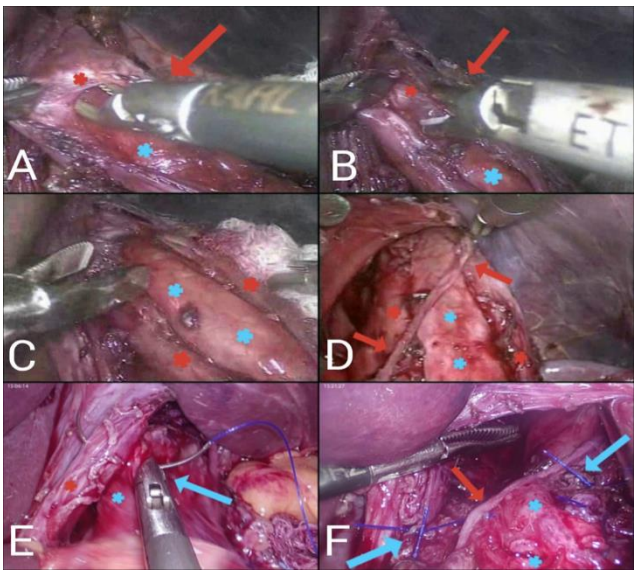

Figure 3: (A) Myotomy in progress(red arrow) shows exposed mucosa (blue asterisk), (B) musculosa (red asterisk), (C) harmonic scalpel in use during myotomy (red arrow), (D) nearly completed myotomy shows its lips (red asterisks) and (E) mucosa(blue asterisks), preserved anterior vagus nerve (red arrows) seen traversing across myotomy, myotomy lip(blue asterisk) being suture-fixed(blue arrow) to right crus(red asterisk) and (F) B/L myotomy lips suture-fixed to both crurae (blue arrows) with anterior vagus nerve seen running across(red arrow).

At post-operative follow-up, 100% (n=10) of the patients reported marked improvement in their preoperative symptoms, while 2 (20%) patients described heartburn (grade I, mild, daily) at their 1st, 4th and 6th week follow up visits. There was no conversion to open and we observed no peri-operative mortality. There were no patients who underwent re-do surgeries, in the series. The 2 patients who reported mild reflux related symptoms were managed effectively to their satisfaction with a proton pump inhibitor course for 1 month. At the time of writing this paper, over a mean follow-up period of 79.4 months, all 10 patients continue to be asymptomatic. The intra and post operative details are summarised (Table 2).

Table 2: Intra and post operative details.

Variables	N
Average operating time	112 minutes
Average length of stay (range)	3.1 days (3-4 days)
Procedure performed	
Heller-Toupet	9
Heller-Dorr	1
Average follow up period	79.4 months
Post op mortality	0
Intra op morbidity	
Esophageal mucosal perforation	1
Port site bleed	1
Surgical emphysema	1
Post op morbidity	
Surgical emphysema	1
Mild reflux	2
Recurrent dysphagia	0
Conversion to open	0

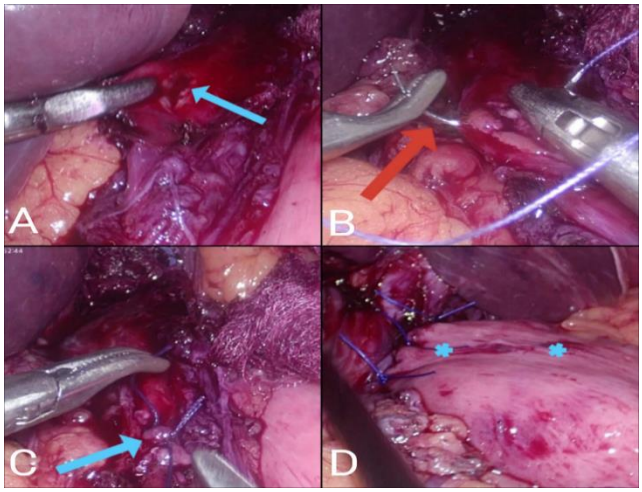

Figure 4: (A) Iatrogenic esophageal mucosal perforation (blue arrow), (B) its suture closure(red arrow), (C) complete suture closure of perforation(blue arrow) and (D) Dorr's anterior fundoplication wrap done in same pt. (blue asterisks) to cover the suture line.

DISCUSSION

Absence of oesophageal peristalsis and abnormal relaxation of LES during swallowing are features of Achalasia cardia (AC). Three subtypes of AC are

described using high-resolution manometry (HRM), according to Chicago classification.[4] All have dysfunction of esophago-gastric junction (EGJ). Differentiating characteristics are the patterns of oesophageal pressurization and contraction. Type I AC (second most common; 20-40% of cases) is described as 100% failed peristalsis (aperistalsis) with absence of periesophageal pressurization to more than 30 mm Hg. Type II AC (most common; 50-70% of cases) has 100% failed peristalsis (aperistalsis) with panesophageal pressurization to greater than 30 mm Hg. Type III AC (least common; 5% of cases) is featured by spastic contractions because of abnormal lumen obliterating contractions with/ without periods of panesophageal pressurization. As per aetiology, AC is classified into 2 subtypes-primary and secondary. Primary (Idiopathic) achalasia is of unknown aetiology. Secondary achalasia also called pseudo achalasia, is caused by gastric cardia tumours or infiltrating intestinal plexus tumours (gastroesophageal junction adenocarcinoma, pancreatic cancer, breast cancer, lung cancer, or hepatocellular carcinoma).[5] Pseudo achalasia mimics primary AC in manometry, endoscopy, and barium swallow findings. New Japanese Classification system for AC based on clinicopathological features seems to be most practical.[6] Patients are divided into 3 types based on their X-ray results: straight, S-shaped, and advanced sigmoid.

Pathophysiologically, AC is caused by loss of inhibitory ganglia in the myenteric plexus of the oesophagus.[7] In the initial stage, degeneration of inhibitory nerves in the oesophagus results in unopposed action of excitatory neurotransmitters such as acetylcholine, resulting in high amplitude non-peristaltic contractions (vigorous achalasia). Progressive loss of cholinergic neurons as time passes, results in dilatation and low amplitude simultaneous contractions in the oesophageal body (classic achalasia). Various studies have tried to zero in on agents that may cause the disease, like viral infection, other environmental factors, autoimmunity, and genetic factors. Though Chagas disease, which mimics AC, is caused by an infective agent, available evidence suggests that infection may not be a standalone cause of primary AC. A genetic basis for AC is supported by reports showing occurrence of disease in monozygotic twins, siblings and other first-degree relatives and occurrence in association with other genetic diseases such as Down's syndrome and Parkinson's disease.[7] However, larger studies are needed before arriving at definite conclusions. Currently, the disease is believed to be multi-factorial, with autoimmune mechanisms triggered by infection in a genetically predisposed individual leading to degeneration of inhibitory ganglia in the wall of the oesophagus.[7] Several studies showed a higher prevalence of autoantibodies in AC patients compared to controls. Storch et al suggested a role of autoimmunity by demonstrating a higher prevalence of anti-myenteric autoantibody in achalasia patients (64%) compared to healthy controls (7%).[8]

Due to oesophageal outflow obstruction, AC presents with clinical symptoms of dysphagia to both solids and liquids, regurgitation of indigested food and occasional chest pain, with/ without weight loss. Other less specific symptoms are heartburn, chronic cough, and aspiration pneumonia. Diagnostic delay, due to presenting symptoms mimicking those of gastroesophageal reflux disease (GERD), may run into several years.

The widespread use of HRM has permitted earlier detection of AC. It has also discerned different types of AC, which can have great prognostic and therapeutic value. Other modern day diagnostic tools like the timed barium oesophagogram (TBO) and the functional lumen imaging probe (FLIP) have also emerged to quantify severity of AC. These can then be also used to objectively measure response to therapy. Currently, AC is mainly diagnosed using HRM, endoscopy, and barium meal examination. A TBO or FLIP is used only when achalasia cannot be diagnosed. The gold standard for diagnosing AC remains HRM. In fact, HRM not only confirms AC, but also identifies different subtypes that have vastly different treatment outcomes. The Chicago classification is a guide for interpretation of HRM findings. Long-term outcomes for AC reveals that success at 5 years post treatment was best in subtype II (96% PD *vs.* 88% LHM), however success rate declined between years 2 and 5 for subtype I and II, while subtype III remained stable (48% PD *vs.* 86% LHM).[9] Older patients and achalasia subtype III were strongest predictors of clinical outcome.[9] Thus, customised treatment for a particular AC subtype could facilitate best outcomes. Use of intraoperative manometry enables calibration of the myotomy and of the fundoplication during laparoscopic Heller's myotomy (LHM). It can also identify a residual high-pressure zone which will further require an extension of myotomy in some patients. Furthermore, this would help achieve calibrated Nissen fundoplication after Heller myotomies, thus enabling better reflux control as compared to Heller myotomy and conventional Dor fundoplication.

Endoscopy is an important tool for digestive disorders, though not very sensitive for AC. Studies have shown that only one-third of patients can be diagnosed with AC using endoscopy.[10] Typically, endoscopy is used to screen patients with gastrointestinal symptoms and to rule out luminal malignancies in the oesophagus and proximal stomach. In patients with AC, barium meal esophagogram reveals oesophageal dilation, EGJ stenosis, 'beak' formation and delayed barium emptying. Studies have shown that four stages of AC can be defined as per the maximum barium diameter and shape in the oesophagus: Stage 1, ≤4 cm; stage 2, 4-6 cm; stage 3, ≥ 6 cm, with a straight oesophagus; stage 4, ≥6 cm, with a sigmoid tube (end-stage disease).[11] Acquiring static images of the oesophagus at predetermined time gaps after the ingestion of a fixed amount of barium sulphate is involved in TBO. It is an improved technique that can assess oesophageal emptying more objectively. It can be

used to evaluate success of therapy. It is simple, economical, non-invasive, repeatable, and well-tolerated by patients.[12] Notably, TBO is widely used for the preliminary evaluation of patients with suspected type I, II, or III achalasia. The FLIP is a novel catheter-based device that can be used to analyse the relationship between cross-sectional area and pressure of the lumen, measure the EGJ and dilatation index in real time, and provide supplementary information for HRM, especially for AC. FLIP has become a potential tool for diagnosis and real-time calibration of AC.[13] This device is generally suitable for patients with suspected achalasia but normal combined relaxation pressure, inconclusive HRM results, and those who cannot tolerate HRM testing.[14] In addition, FLIP can accurately predict the immediate outcome of balloon dilation and be used to guide the selection of balloon size for a single endoscopic balloon dilation.

Chest CT can be used to differentiate between primary and secondary AC. Distal oesophageal wall thickening (nodular/Lobular and asymmetrical); soft tissue masses at the gastroesophageal junction; mediastinal lymph node enlargement; and lung, liver, or bone metastases are suggestive of secondary achalasia.[15]

Endoscopy plays an important role in excluding pseudo achalasia or other mechanical obstruction that may result in symptoms similar to achalasia.[16] Endoscopy is useful in patients with recurrent symptoms post therapy to assess for reflux and possible reflux-related stricturing vs recurrence of achalasia. Barium esophagram can be complementary in patients whose manometric findings are equivocal or not classic.

Medication is usually adopted in patients who cannot, or refuse to, undergo endoscopic or surgical treatment and in those for whom endoscopic or surgical treatment has failed. Calcium channel blockers, nitrates, and PPIs are commonly used to control acid reflux; however, they provide only short-term relief and are less effective.[17] Traditional endoscopic treatment of achalasia involves injections of botulinum toxin type A (Botox) and pneumatic dilation (PD). Botox is a biological neurotoxin released by *Clostridium botulinum* that can prevent the release of acetylcholine from voluntary and involuntary muscle nerve endings.[18] It is considered an effective treatment for short-term symptomatic relief in patients with AC. This treatment is initially effective, but outcome of repeated usage is poor, and the maintenance time is short (approximately 6-9 months).[2] Pneumatic dilatation involves use of an inflated balloon with a strong stretch to destroy LES. Although short-term results of PD are adequate, long-term outcomes are poor, requiring multiple treatments.[19] Surgical treatments for achalasia include peroral endoscopic myotomy (POEM), LHM, stent implantation, and esophagectomy. The first-line surgical therapy for achalasia is LHM. It is a common treatment modality for AC, especially in adolescents and young adults. It has response rate of 90-97% with recurrent dysphagia in 3-10% of patients and GERD can occur in 2-26% of cases.[20] Post-LHM-GERD with extended myotomy can be minimised by concurrent fundoplication (posterior Toupet fundoplication at 270° is a better anti-reflux procedure than anterior Dor fundoplication at 180°). Laparoscopic approach is preferred over the thoracoscopic approach because of shorter operating time and lower probability of conversion to open myotomy. Thoracoscopic approach may be preferred in patients who have dense intraabdominal adhesions due to multiple previous surgeries. Adverse events with LHM include oesophageal perforation (1-7%) caused by inadvertent mucosal injury, recurrent dysphagia caused by incomplete myotomy (3-10%), GERD (2-26%), post vagotomy diarrhoea/dumping syndrome caused by division of the vagus nerve, and splenic injury (1-5%). In sigmoid oesophagus and type III achalasia, results of LHM are suboptimal. LHM has equal efficacy compared to PD with greater sustainability of results in young males.[20]

Recently, POEM has emerged as an alternative to LHM. Introduced in 2008, this technique is a new minimally invasive therapeutic endoscopic surgical technique that is especially suitable for patients with type III AC. This operation involves establishing a submucosal tunnel in the lower oesophagus to reach the LES' inner ring muscle bundle for myotomy while preserving the external longitudinal muscle bundle. This prevents body wall trauma and preserve the external oesophageal anatomy, while being precise and minimally invasive. One advantage of POEM over LHM is that it can be used to perform a real-time direct biopsy of muscle layers and easily control the length and location of the myotomy.[21]

Studies have shown that POEM technique has certain drawbacks that can lead to GERD because POEM is not combined with anti-reflux surgery. A 2-year follow-up study showed that POEM was not inferior to LHM combined with Dor fundoplication in controlling the symptoms of 2-year AC. However, compared with LHM patients, POEM patients were more prone to GERD. Although POEM is a promising new therapy, most endoscopists worldwide have not yet mastered this technique owing to its high technical requirements and steep learning curve.

Owing to development of endoscopic technology and the development of minimally invasive surgical operations, esophagectomy has rarely been used. Patients with surgical failure and end-stage AC can undergo esophagectomy. In 2018, international society for oesophageal diseases guidelines recommended esophagectomy for patients with persistent/ recurrent achalasia after minor invasive treatment failure and disease progression.[22] An overview of currently available and practiced therapeutic modalities for AC is summarized (Table 3).

Table 3: Various therapeutic modalities for AC in current use.

Modality of treatment	Year of origin/ indication	Salient features	Results	Advantage/s	Disadvantage/s
Pharmacotherapy	For patients who refuse endoscopic or surgical intervention	Calcium channel blockers, nitrates, and PPIs are commonly used[17]		Non interventional modality	They provide only short-term relief (if any) and are much less effective than interventional measures
Botulinum toxin type A-endoscopic injection	1980, Pasricha et al	It is a biological neurotoxin produced by Clostridium botulinum that can prevent release of acetylcholine from voluntary and involuntary muscle nerve endings	Symptomatic improvement at one month- 78.7%, but gradually decreased to 70% at 3 months, 53.3% at 6 months and 40.6% at 1 year. 2nd session required in 46.6%. Universal symptom relapse by 2 years[23]	Non operative modality	Initially effective, but the outcome of repeated usage is poor and the maintenance time is short
Pneumatic dilatation	1672 Sir Thomas Willis (using sponge attached to whale bone)	This method involves the use of an inflated balloon with a strong stretch to destroy the LES. The diameter of the balloon can range from 30 to 40 mm	A single PD resulted in a 5 year remission rate of 40% and a 10 year remission rate of 36%. Repeated dilations only mildly improved clinical response[24]	Non operative modality with adequate short-term results	Long term outcomes are poor, requiring multiple treatments
Per oral endoscopic myotomy (POEM)	2008, Inoue	New endoscopic surgical technique, involves establishing a submucosal tunnel in the lower esophagus to reach the LES inner ring muscle bundle for myotomy while preserving the external longitudinal muscle bundle; this can prevent body wall trauma and preserve the external esophageal anatomy	78% of patients maintained positive clinical response at 6 years following POEM procedure. The recurrence rate of symptoms following POEM was 22% at a 6-year follow-up[25]	Non operative modality that can be used to perform a real-time direct biopsy of muscle layers	High chances of GERD as not combined with anti-reflux surgery. Barrett's esophagus-a potential long-term sequela of postoperative reflux-may also occur if the reflux persists long term. Although POEM is a promising new therapy, most endoscopists worldwide have not yet mastered this technique owing to its high technical requirements and steep learning curve
Laparoscopic Heller myotomy	Open-1913, Ernst Heller laparoscopic-1991, Alfred Cuschieri	LHM is a common clinical treatment for achalasia, especially in adolescents and young adults. myotomy made anteriorly from 6 cm above the GEJ to 3 cm beyond, preserving cardio-oesophageal fat and the anterior vagus nerve. Post-LHM GERD with extended myotomy can be minimised by concurrent fundoplication	it has a response rate of 90-97% with recurrent dysphagia in 3-10% of patients and gastro-esophageal reflux disease (GERD) can occur in 2-26%.[20]	Minimal invasive surgical modality which gives the best long term results, post-LHM GERD can be minimised by concurrent fundoplication (posterior Toupet fundoplication at 270° is a better antireflux procedure than anterior Dor fundoplication at 180°)	Possible adverse events: oesophageal perforation (1-7%), recurrent dysphagia caused by incomplete myotomy (3-10%), GERD (2-26%), postvagotomy diarrhoea/dumping syndrome caused by accidental division of the vagus nerve, and splenic injury (1-5%). In sigmoid oesophagus and Type III achalasia, the results of LHM are suboptimal.[20]

CONCLUSION

As seen in this series, laparoscopic therapy for Achalasia cardia is feasible in an advanced setup when coupled with experience. It is the best treatment modality which gives the most favourable long-term results. A distinct advantage of LHM over POEM is the ability to concurrently perform an anti-reflux procedure which minimises post operative GERD.

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